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Daytime Functioning and Quality of life in Chronic Insomnia: A Multi-Method, Multi-Level Approach

Simon David Kyle
Master of Arts (First Class Hons.) in Psychology
0207803

Submitted for the degree of Doctor of Philosophy in Medicine

University of Glasgow Sleep Centre
Department of Psychological Medicine
Faculty of Medicine
University of Glasgow
Scotland

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Thesis Overview

Insomnia disorder is characterised by difficulties with initiating and/or maintaining sleep. Similar to most psychiatric and mental health conditions, insomnia is defined according to subjective complaint, and achieves disorder ‘status’ when associated daytime functioning impairment is present. Yet, ironically, it is these two cornerstones of insomnia disorder, combined, that have achieved relatively minimal attention in the literature. That is, perhaps surprisingly, the subjective experience, and impact of insomnia, at least from the patient (‘expert’) perspective, has been under-researched. Night-time symptoms and sleep parameters have typically been the target of both treatment and non-treatment (clinical, epidemiological, mechanistic) research.

In this thesis, a multi-method, multi-level approach is adopted to better understand the daytime experience of those with chronically disturbed sleep. First, a brief overview (chapter one) of insomnia is provided to familiarise the reader with the ‘problem of insomnia’. A narrative review (chapter two) then sets the scene in relation to the assessment and measurement of health-related quality of life (HRQoL) and daytime functioning. This work reveals several inadequacies and limitations of existing work, and outlines a prospective research agenda.

Chapter three describes the first ever phenomenological study carried out in primary insomnia patients. Here, two qualitative methodologies, focus groups and audio-diaries, are combined to help better understand the proximal and distal impairments attributed to chronic sleep disturbance. Chapter four builds on this work by describing the creation of
two new clinical scales, developed to quantify, in both valid and novel ways, the impact of poor sleep on aspects of daytime functioning and insomnia-related quality of life.

Chapter five combines the aforementioned qualitative and questionnaire approaches to explore the experience of an effective behavioural intervention for insomnia, sleep restriction therapy (SRT). The application of these refined methods provided insight into the effects of SRT on both sleep and daytime functioning, but also permitted exploration of treatment-related issues - such as adherence, side-effects, and mechanisms of action - that have otherwise been difficult to probe using traditional quantitative methodologies.

Chapter six tackles the issue of objective daytime impairment, typically assessed using computerised reaction time tasks. Through ‘mining’ an existing brain and behavioural database, and applying an algorithm to select poor and normal sleepers, it was possible to investigate cognitive functioning at two broad stages of processing – event-related potentials generated from the scalp-recorded electroencephalogram (EEG), and performance output using neuropsychological testing. The results provide some interesting hypotheses concerning possible cognition-arousal and -effort interactions. Importantly, as a by-product of this work, a methodological template for the future standardized assessment of brain and behavioural function in insomnia is considered.

Finally, chapter seven synthesises the results of each preceding experimental chapter, with particular emphasis on how this work will advance research, measurement and understanding of insomnia-related functioning. Immediate clinical implications and relevance to other areas of insomnia research are also briefly considered.
“When you lose sleep, you lose the better part of yourself. You’re not all there—as insomniacs know, as we say with the terms we use for ourselves: zombies, the living dead, nobody home. It seems ironic that sleep is feared as the loss or disappearance of the self, when it may actually be the way we become most fully ourselves, maintain the continuity of past and present selves, retain our identities through time and change, become our most creative, intelligent, and alive. Sleep is how we manage to be all there. You might even say, I sleep, therefore I am.”

Greene (2008, p.48)
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>List of Tables</td>
<td>x</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xii</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>xiv</td>
</tr>
<tr>
<td>List of Abbreviations</td>
<td>xv</td>
</tr>
<tr>
<td><strong>Chapter 1 – The Problem of Insomnia: A Brief Overview</strong></td>
<td>17</td>
</tr>
<tr>
<td>1.1. Insomnia: defining features</td>
<td>18</td>
</tr>
<tr>
<td>1.2. Risk factors and precipitants</td>
<td>21</td>
</tr>
<tr>
<td>1.3. Insomnia assessment</td>
<td>24</td>
</tr>
<tr>
<td>1.4. Insomnia costs: economic and psychobiological considerations</td>
<td>27</td>
</tr>
<tr>
<td>1.5. Contemporary models of insomnia</td>
<td>29</td>
</tr>
<tr>
<td>1.5.1. The 3P (predisposing, precipitating, perpetuating) model: a general framework</td>
<td>30</td>
</tr>
<tr>
<td>1.5.2. Neurocognitive model</td>
<td>31</td>
</tr>
<tr>
<td>1.5.3. Cognitive model</td>
<td>31</td>
</tr>
<tr>
<td>1.5.4. Psychobiological Inhibition/Attention-Intention-Effort (PIM/AIE) Model</td>
<td>32</td>
</tr>
<tr>
<td>1.5.5. Beyond dualistic tendencies: towards an integrative psychobiological insomnia model</td>
<td>33</td>
</tr>
<tr>
<td>1.6. Evidence-based treatment of insomnia</td>
<td>35</td>
</tr>
<tr>
<td>1.6.1. Pharmacotherapy</td>
<td>36</td>
</tr>
<tr>
<td>1.6.2. Cognitive Behavioural Therapy for insomnia (CBT-I)</td>
<td>38</td>
</tr>
<tr>
<td>1.6.3. What constitutes effective treatment?</td>
<td>41</td>
</tr>
<tr>
<td><strong>Chapter 2 – Insomnia and Health-Related Quality of Life</strong></td>
<td>43</td>
</tr>
<tr>
<td>2.1. Abstract</td>
<td>44</td>
</tr>
<tr>
<td>2.2. Introduction</td>
<td>45</td>
</tr>
<tr>
<td>2.3. Insomnia: daytime consequences and associated morbidity</td>
<td>46</td>
</tr>
<tr>
<td>2.4. Quality of Life (QoL) and Health-related Quality of Life (HRQoL)</td>
<td>49</td>
</tr>
</tbody>
</table>
2.5. Does insomnia negatively affect HRQoL? .......................... 52
2.6. Does improving insomnia also improve aspects of HRQoL? ................. 61
  2.6.1. Cross-sectional & uncontrolled studies .................................. 62
  2.6.2. Prospective controlled trials .............................................. 66
2.7. Reflections on existing insomnia-HRQoL treatment literature ................. 71
2.8. Beyond generic measurement: future directions ............................ 75
2.9. Conclusion ........................................................................... 81
  2.9.1. Practice points ................................................................. 82
  2.9.2. Research agenda ............................................................. 82
  2.9.3. Going forward ............................................................... 83

Chapter 3 - Daytime Phenomenology in Primary Insomnia ..................... 84
  3.1. Abstract ............................................................................. 85
  3.2. Introduction ....................................................................... 86
  3.3. Method .............................................................................. 91
    3.3.1. Participants ................................................................. 91
    3.3.2. Procedure ..................................................................... 92
    3.3.3. Focus Groups ............................................................. 92
    3.3.4. Audio-diary reports .................................................... 93
    3.3.5. Framework for qualitative analysis ................................. 93
  3.4. Results ............................................................................. 95
    3.4.1. Participant characteristics ........................................... 95
    3.4.2. Interpretative analysis .................................................. 95
      3.4.2.1. ‘Just struggle through’ ............................................. 96
      3.4.2.2. ‘Isolated, feeling like an outsider’ ............................. 100
      3.4.2.3. ‘Insomnia as an obstruction to the desired self’ .......... 104
  3.5. Discussion ........................................................................ 109

Chapter 4 - The Development of Two New Measurement Approaches to Assessing Insomnia-Related Quality of Life and Daytime Functioning: the Glasgow Sleep Impact Index (GSII) and the Daytime Functioning and Sleep Attribution Scale (DFSAS) ..................................................... 117
  4.1. Abstract ........................................................................... 118
  4.2. Introduction ....................................................................... 119
4.3. The Daytime Functioning and Sleep Attribution Scale (DFSAS)................. 125
  4.3.1. Background & aims ......................................................... 125
  4.3.2. Methods................................................................. 127
    4.3.2.1. Participants...................................................... 127
    4.3.2.2. Measures and procedures..................................... 129
  4.3.3. Results........................................................................ 129
    4.3.3.1. Item generation and face validity............................. 129
    4.3.3.2. Discriminant validity.......................................... 130
    4.3.3.3. Concurrent validity............................................ 133
    4.3.3.4. Relationship with sleep quality and sensitivity to treatment outcome......................................................... 134
    4.3.3.5. Internal consistency............................................. 137
    4.3.3.6. Sensitivity and specificity...................................... 137
    4.3.3.7. Relationship between DFSAS parts 1 and 2............... 138
  4.4. Glasgow Sleep Impact Index (GSII)..................................... 139
    4.4.1. Background & aims.................................................. 139
    4.4.2. Scale development & pilot ....................................... 141
    4.4.3. Results....................................................................... 145
      4.4.3.1. Participant demographics.................................... 145
      4.4.3.2. Rank content analysis (content validity)................ 145
      4.4.3.3. Relationship between ranks.................................. 147
      4.4.3.4. Concurrent validity.......................................... 148
      4.4.3.5. Relationship with sleep and treatment outcome sensitivity....................................................... 149
  4.5. Discussion....................................................................... 150
    4.5.1. DFSAS........................................................................ 150
    4.5.2. GSII.......................................................................... 154
    4.5.3. Limitations................................................................... 158
    4.5.4. Concluding remarks and future directions..................... 159

Chapter 5 - The Integration of Quantitative and Qualitative Methodologies to Investigate the Patient Experience of Sleep Restriction Therapy (SRT) for Insomnia................................................................. 162
  5.1. Abstract........................................................................... 163
  5.2. Introduction.................................................................... 164
  5.3. Methods......................................................................... 170
    5.3.1. Participants............................................................ 170
    5.3.2. Treatment protocol.................................................. 171
    5.3.3. Screening instruments.............................................. 173
    5.3.4. Outcome questionnaire measures............................... 173
      5.3.4.1. Sleep................................................................. 173
      5.3.4.2. Daytime functioning.......................................... 175
<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.3.4.3. Treatment-related process measures</td>
<td>176</td>
</tr>
<tr>
<td>5.3.5. Qualitative methodologies</td>
<td>178</td>
</tr>
<tr>
<td>5.3.5.1. Audio-diaries</td>
<td>178</td>
</tr>
<tr>
<td>5.3.5.2. Semi-structured interviews</td>
<td>178</td>
</tr>
<tr>
<td>5.3.6. Data preparation and statistical analysis</td>
<td>179</td>
</tr>
<tr>
<td>5.3.6.1. Outcome variables</td>
<td>179</td>
</tr>
<tr>
<td>5.3.6.2. Audio-diaries &amp; Semi-structured interviews</td>
<td>180</td>
</tr>
<tr>
<td>5.4. Results</td>
<td>182</td>
</tr>
<tr>
<td>5.4.1. Participant demographics</td>
<td>182</td>
</tr>
<tr>
<td>5.4.2. Sleep outcomes</td>
<td>183</td>
</tr>
<tr>
<td>5.4.3. Daytime functioning outcomes</td>
<td>186</td>
</tr>
<tr>
<td>5.4.4. Relationship between sleep and daytime functioning changes</td>
<td>188</td>
</tr>
<tr>
<td>5.4.5. Treatment side-effects</td>
<td>190</td>
</tr>
<tr>
<td>5.4.6. Self-report adherence</td>
<td>193</td>
</tr>
<tr>
<td>5.4.7. Qualitative results</td>
<td>194</td>
</tr>
<tr>
<td>5.4.7.1. Audio-diaries</td>
<td>194</td>
</tr>
<tr>
<td>5.4.7.2. Interviews</td>
<td>203</td>
</tr>
<tr>
<td>5.5. Discussion</td>
<td>217</td>
</tr>
<tr>
<td>5.5.1. Sleep-related changes</td>
<td>217</td>
</tr>
<tr>
<td>5.5.2. Side-effects and daytime functioning/HRQoL</td>
<td>220</td>
</tr>
<tr>
<td>5.5.3. Adherence</td>
<td>224</td>
</tr>
<tr>
<td>5.5.4. Implications for clinical practice and future research</td>
<td>227</td>
</tr>
<tr>
<td>5.5.5. Limitations</td>
<td>229</td>
</tr>
<tr>
<td>5.5.6. Concluding remarks</td>
<td>230</td>
</tr>
</tbody>
</table>

Chapter 6 - Auditory P300 and neuropsychological performance in poor and normal sleepers: A controlled comparative study 231

6.1. Abstract                                                          232
6.2. Introduction                                                      233
6.3. Method                                                           239
6.3.1. Participants                                                   239
6.3.2. Materials & Procedure                                          240
6.3.2.1. Neuropsychological assessment                                240
6.3.2.2. Oddball task                                                 243
6.3.2.3. EEG/ERP acquisition                                          243
6.3.2.4. Analyses                                                     244
6.4. Results                                                          246
6.4.1. Participant characteristics                                    247
6.4.2. Neuropsychological testing                                    246
6.4.3. Oddball task (ERPs)                                            248
List of tables

Table 1.1 - Insomnia classification according to ICSD-2 and DSM-IV............. 19

Table 1.2 - Essential features of primary insomnia phenotypes in ICSD-2, redrawn from Schutte-Rodin et al. (2008)......................................................... 39

Table 1.3 - Main CBT-I components, taken from Morin et al. (2006b)........... 39

Table 2.1 – Instruments used to assess HRQoL and QoL in insomnia populations................................................................. 53

Table 2.2 – Insomnia treatment studies assessing Health-Related Quality of Life as an outcome variable.............................................................. 63

Table 3.1 - Participant demographics for those participating in focus groups and sub-set completing audio-diaries................................................................. 95

Table 3.2 - Superordinate and sub-themes from IPA........................................... 96

Table 4.1 – Required scale characteristics to meet existing measurement needs.... 123

Table 4.2 - Demographics for primary insomnia (PI) patients and normal sleepers (NS). Parentheses represent the standard deviation.............................. 128

Table 4.3 - Mean DFSAS item values for PI patients and normal sleepers (NS)... 132

Table 4.4 - Treatment sensitivity for DFSAS parts 1 and 2 in PI patients........... 135

Table 4.5 - Responder analysis for DFSAS parts 1 and 2. Median (Mdn) values are presented with the interquartile range (IQR) in parentheses.......................... 137

Table 4.6 - Sensitivity and specificity of DFSAS in identifying NS and PI patients................................................................................................. 138

Table 4.7 - Content domains from GSII rank content analysis........................ 146

Table 4.8 - VAS ratings (mm) and spend (£) for generated ranks for all patients... 148

Table 4.9 - VAS scores for GSII ranks collected over the course of intervention and at follow-up................................................................. 149

Table 4.10 - Responder analysis for the GSII. Median (Mdn) values are presented with the interquartile range (IQR) in parentheses................................. 150
Table 4.11 - Measurement needs and subsequent fulfilment by developed scales................................................................. 151

Table 5.1 - SRT session content................................................................................................................................. 172

Table 5.2 - Time-line of assessment and data collection points.............................................................................. 177

Table 5.3 – Participant demographics.......................................................... ........................................................................ 182

Table 5.4 - Treatment effects and post-hoc comparisons for sleep diary and questionnaire variables ................................................................. 184

Table 5.5 - Treatment effects and post-hoc comparisons for daytime functioning and HRQoL variables ........................................................................ 187

Table 5.6 - Relationship between changes in daytime functioning variables and sleep variables (baseline to follow-up).................................................................................................................. 189

Table 5.7 - Side-effect frequency and interference for entire sample .......... 191

Table 5.8 - Group mean scores for adherence to SRT guidelines on weekdays and weekends................................................................. 194

Table 5.9 - Major themes from thematic analysis of audio-diary entries ........ 195

Table 5.10 - Major themes from thematic analysis of semi-structured interviews.. 203

Table 6.1 – IntegNeuro standard neuropsychological test descriptions (taken and modified from Paul et al., 2006).............................................................................................................................. 241

Table 6.2 - Participant demographics.......................................................................................................................... 246

Table 6.3 - Mean (not transformed) scores (SD) for neuropsychological tests for poor sleepers (PS) and normal sleepers (NS)........................................................................................................ 247

Table 6.4 - Mean P300 amplitude and latency for both groups at each midline electrode site................................................................. 248

Table 7.1 - Key findings from thesis........................................................................................................................... 262
List of Figures

**Figure 2.1** - Deviations in SF-36 scores for Insomnia, Depression, and Congestive Heart Failure, relative to hypertension reference group. Scores plotted from Katz & McHorney (2002)………………………………………… 60

**Figure 4.1** - Copy of the Daytime Functioning and Sleep Attribution Scale (DFSAS)…………………………………………………………………………... 126

**Figure 4.2** - Boxplots of DFSAS part 1 scores for PI patients and normal sleepers (NS)………………………………………………………………………… 131

**Figure 4.3** - Boxplots of DFSAS part 2 scores for PI patients and normal sleepers (NS)……………………………………………………………………….. 131

**Figure 4.4** - Radar plot of mean item (1-12) DFSAS part 1 scores for patients with primary insomnia (PI) and normal sleepers (NS)…………………… 132

**Figure 4.5** - Radar plot of mean item (1-12) DFSAS part 2 scores for patients with primary insomnia (PI) and normal sleepers (NS)…………………… 133

**Figure 4.6** - DFSAS part 1 mean (± standard error) scores over the course of intervention……………………………………………………………………… 135

**Figure 4.7** - DFSAS part 2 mean (± standard error) scores over the course of intervention……………………………………………………………………… 136

**Figure 4.8** - Copy of the Glasgow Sleep Impact Index (GSII)…………………… 143

**Figure 4.9** - Follow-up ‘closed’ version of the GSII with patient example……… 144

**Figure 4.10** - Boxplots of VAS (mm) ratings for GSII ranks 1, 2 and 3, for all patients………………………………………………………………………… 147

**Figure 5.1** - Session time-line……………………………………………………… 172

**Figure 5.2** - Relationship between GSII rank 2 change scores and ISI change scores………………………………………………………………………… 190

**Figure 5.3** - Graphical representation of % of sample reporting each listed side-effect………………………………………………………………………… 191

**Figure 5.4** - Scatterplot of relationship between PSQI change scores and number of experienced side-effects…………………………………………….. 192
Figure 5.5 - Histogram showing distribution of global adherence ratings............ 193

Figure 5.6 - CBT-I treatment targets. Taken from Edinger & Means (2005) and modified (red lines) based on present study findings................................. 220

Figure 6.1 - P300 latencies (mean plus standard error) for poor and normal sleepers.......................................................... 249
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## List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AASM</td>
<td>American Academy of Sleep Medicine</td>
</tr>
<tr>
<td>AD</td>
<td>Audio-Diary</td>
</tr>
<tr>
<td>AIE</td>
<td>Attention-Intention-Effort pathway</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
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<td>APSS</td>
<td>Associated Professional Sleep Societies</td>
</tr>
<tr>
<td>ARAS</td>
<td>Ascending Reticular Activating System</td>
</tr>
<tr>
<td>BAI</td>
<td>Beck Anxiety Inventory</td>
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<tr>
<td>BDI</td>
<td>Beck Depression Inventory</td>
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<td>BRID</td>
<td>Brain Resource International Database</td>
</tr>
<tr>
<td>BZRA</td>
<td>non-Benzodiazepine Receptor Agonists</td>
</tr>
<tr>
<td>CAP</td>
<td>Cyclic Alternating Pattern</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive Behavioural Therapy</td>
</tr>
<tr>
<td>CBT-I</td>
<td>Cognitive Behavioural Therapy for Insomnia</td>
</tr>
<tr>
<td>CGI</td>
<td>Clinical Global Impressions Scale</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
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<tr>
<td>CPAP</td>
<td>Continuous Positive Airway Pressure</td>
</tr>
<tr>
<td>DASS</td>
<td>Depression Anxiety and Stress Scales</td>
</tr>
<tr>
<td>DBAS</td>
<td>Dysfunctional Beliefs and Attitudes about Sleep scale</td>
</tr>
<tr>
<td>DFSAS</td>
<td>Daytime Functioning and Sleep Attribution Scale</td>
</tr>
<tr>
<td>DSM-IV</td>
<td>Diagnostic and Statistical Manual of Mental Disorders, 4th Edition</td>
</tr>
<tr>
<td>DSM-IV-TR</td>
<td>Diagnostic and Statistical Manual of Mental Disorders, Text Revision</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
</tr>
<tr>
<td>EORTC-QOL-C30</td>
<td>European Organization for Research and Treatment of Cancer Quality of Life Questionnaire</td>
</tr>
<tr>
<td>ERP</td>
<td>Event-Related Potentials</td>
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<td>ES</td>
<td>Effect Size</td>
</tr>
<tr>
<td>ESS</td>
<td>Epworth Sleepiness Scale</td>
</tr>
<tr>
<td>FACT-G</td>
<td>Functional Assessment of Cancer Therapy – General</td>
</tr>
<tr>
<td>FFS</td>
<td>Flinders Fatigue Scale</td>
</tr>
<tr>
<td>FG</td>
<td>Focus Group</td>
</tr>
<tr>
<td>fMRI</td>
<td>functional Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>FOSQ</td>
<td>Functional Outcomes of Sleep Questionnaire</td>
</tr>
<tr>
<td>FSS</td>
<td>Fatigue Severity Scale</td>
</tr>
<tr>
<td>GABA</td>
<td>Gamma-AminoButyric Acid</td>
</tr>
<tr>
<td>GSES</td>
<td>Glasgow Sleep Effort Scale</td>
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<td>GSII</td>
<td>Glasgow Sleep Impact Index</td>
</tr>
<tr>
<td>HD-16</td>
<td>Hotel-Dieu 16</td>
</tr>
<tr>
<td>HRQoL</td>
<td>Health Related Quality of Life</td>
</tr>
<tr>
<td>ICSD-2</td>
<td>International Classification of Sleep Disorders, Second Edition</td>
</tr>
<tr>
<td>IIS</td>
<td>Insomnia Impact Scale</td>
</tr>
<tr>
<td>IL-6</td>
<td>Interleukin-6</td>
</tr>
<tr>
<td>IPA</td>
<td>Interpretative Phenomenological Analysis</td>
</tr>
<tr>
<td>IQR</td>
<td>Interquartile Range</td>
</tr>
<tr>
<td>IRQoL</td>
<td>Insomnia-Related Quality of Life</td>
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<tr>
<td>ISI</td>
<td>Insomnia Severity Index</td>
</tr>
<tr>
<td>LORETA</td>
<td>Low Resolution Electromagnetic Tomography</td>
</tr>
<tr>
<td>M</td>
<td>Mean</td>
</tr>
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<td>MANOVA</td>
<td>Multivariate Analysis of Variance</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
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<tr>
<td>Mdn</td>
<td>Median</td>
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<tr>
<td>MOS</td>
<td>Medical Outcomes Study</td>
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<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
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<tr>
<td>NAW</td>
<td>Number of Awakenings</td>
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<td>NHP</td>
<td>Nottingham Health Profile</td>
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<td>NIH</td>
<td>National Institutes of Health</td>
</tr>
<tr>
<td>NREM</td>
<td>Non-Rapid Eye Movement sleep</td>
</tr>
<tr>
<td>NS</td>
<td>Normal Sleepers</td>
</tr>
<tr>
<td>OISQ</td>
<td>Occupational Impact of Sleep Questionnaire</td>
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<tr>
<td>OSA</td>
<td>Obstructive Sleep Apnea</td>
</tr>
<tr>
<td>PG1</td>
<td>Patient-Generated Index</td>
</tr>
<tr>
<td>PHQ-9</td>
<td>Patient Health Questionnaire</td>
</tr>
<tr>
<td>PI</td>
<td>Primary Insomnia</td>
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<tr>
<td>PIM</td>
<td>Psychobiological Inhibition Model</td>
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<tr>
<td>PS</td>
<td>Poor Sleepers</td>
</tr>
<tr>
<td>PSG</td>
<td>Polysomnography</td>
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<tr>
<td>PSQI</td>
<td>Pittsburgh Sleep Quality Index</td>
</tr>
<tr>
<td>QALY</td>
<td>Quality Adjusted Life Years</td>
</tr>
<tr>
<td>Q-LES-Q</td>
<td>Quality of Life Enjoyment and Satisfaction Questionnaire</td>
</tr>
<tr>
<td>QoL</td>
<td>Quality of Life</td>
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<tr>
<td>QOLI</td>
<td>Quality of Life of Insomniacs Questionnaire</td>
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<tr>
<td>QoLI</td>
<td>Quality of Life Inventory</td>
</tr>
<tr>
<td>QSOQ</td>
<td>Quebec Sleep Questionnaire</td>
</tr>
<tr>
<td>RCT</td>
<td>Randomised Controlled Trial</td>
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<tr>
<td>RDC</td>
<td>Research Diagnostic Criteria</td>
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<tr>
<td>REM</td>
<td>Rapid Eye Movement sleep</td>
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<tr>
<td>ROC</td>
<td>Receiver Operating Characteristic curve</td>
</tr>
<tr>
<td>RT</td>
<td>Reaction Time</td>
</tr>
<tr>
<td>SAQLI</td>
<td>The Calgary Sleep Apnea Quality of Life Index</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic Blood Pressure</td>
</tr>
<tr>
<td>SCN</td>
<td>Suprachiasmatic Nucleus</td>
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<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<tr>
<td>SE</td>
<td>Sleep Efficiency</td>
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<tr>
<td>SF-36</td>
<td>Short-Form Health Survey 36</td>
</tr>
<tr>
<td>SIP</td>
<td>Sickness Impact Profile</td>
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<tr>
<td>SOL</td>
<td>Sleep Onset Latency</td>
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<tr>
<td>SPHERE</td>
<td>Somatic and Psychological Health Report questionnaire</td>
</tr>
<tr>
<td>SQ</td>
<td>Sleep Quality</td>
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<tr>
<td>SRAS</td>
<td>Sleep Restriction Adherence Scale</td>
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<tr>
<td>SRT</td>
<td>Sleep Restriction Therapy</td>
</tr>
<tr>
<td>SWS</td>
<td>Slow-Wave Sleep</td>
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<tr>
<td>TAU</td>
<td>Treatment As Usual</td>
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<tr>
<td>TST</td>
<td>Total Sleep Time</td>
</tr>
<tr>
<td>UGSC</td>
<td>University of Glasgow Sleep Centre</td>
</tr>
<tr>
<td>VAS</td>
<td>Visual Analogue Scale</td>
</tr>
<tr>
<td>VLPO</td>
<td>Ventrolateral Preoptic Nuclei</td>
</tr>
<tr>
<td>WASO</td>
<td>Wake-time After Sleep Onset</td>
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<tr>
<td>WLQ</td>
<td>Work Limitations Questionnaire</td>
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</tbody>
</table>
Chapter 1:

The Problem of Insomnia: A Brief Overview
1.1. Insomnia: defining features

The core symptoms of insomnia, specified in major disease and sleep disorder classification manuals (ICSD-2, 2005; DSM-IV, 1994; ICD-10, 1992), correspond to difficulties with initiating and/or maintaining sleep, or non-restorative sleep (i.e. poor quality sleep). To achieve disorder ‘status’, sleep disturbance must not be simply a function of restricted sleep opportunity (i.e. curtailment), or environmental perturbation (such as noise, bed partner snoring etc.). Importantly, the diagnosis of insomnia disorder is made only when impairment in daytime functioning is present, which is linked (attributionally) to night-time sleep difficulties. Daytime impairments may be measured with reference to isolated symptoms, such as fatigue and concentration (Research Diagnostic Criteria - Edinger et al., 2004; ICSD-2, 2005), but also to more global dysfunction; for example, in areas of social and occupational functioning (DSM-IV, 1994). One additional marker of insomnia severity refers to the frequency and length (persistence) of insomnia symptoms, which, for an insomnia diagnosis, is usually set at greater than or equal to three nights per week (Lichstein et al., 2003; Ohayon & Reynolds III, 2009), being present for at least a one month period (Edinger et al., 2004; DSM-IV, 1994). It is notable that there is currently no ubiquitous cut-off for daytime functioning severity, though a score of at least 2 (range: 0-4) on the ‘daytime interference’ question (item 5) from the Insomnia Severity Index (ISI; Morin, 1993) has been recommended by some (Morin & Espie, 2003).

It is important to note that there are two main diagnostic manuals for categorizing insomnia, which vary with respect to the level of symptomatic detail and sub-type classification. For example, the International Classification of Sleep Disorders manual
(ICSD-2, 2005) identifies several different sub-types of insomnia disorder; whereas the DSM-IV presents a more narrow focus on just a few (see table 1.1).

Table 1.1 - Insomnia classification according to ICSD-2 and DSM-IV.

<table>
<thead>
<tr>
<th>DSM-IV insomnia categories</th>
<th>ICSD-2 insomnia categories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary insomnia</td>
<td>Adjustment insomnia (acute insomnia)</td>
</tr>
<tr>
<td>Insomnia related to (Axis I or II category)</td>
<td>Psychophysiological insomnia</td>
</tr>
<tr>
<td></td>
<td>Paradoxical insomnia</td>
</tr>
<tr>
<td></td>
<td>Idiopathic insomnia</td>
</tr>
<tr>
<td></td>
<td>Inadequate sleep hygiene</td>
</tr>
<tr>
<td>Sleep disorder due to general medical condition, insomnia type</td>
<td>Insomnia due to mental disorder</td>
</tr>
<tr>
<td>Sleep disorder due to substance abuse, insomnia type</td>
<td>Insomnia due to medical condition</td>
</tr>
<tr>
<td></td>
<td>Insomnia due to drug or substance</td>
</tr>
<tr>
<td></td>
<td>Insomnia not due to substance or known physiological condition, unspecified</td>
</tr>
<tr>
<td></td>
<td>(Nonorganic Insomnia, NOS)</td>
</tr>
<tr>
<td></td>
<td>Physiological (organic) insomnia, unspecified</td>
</tr>
<tr>
<td></td>
<td>Behavioral insomnia of childhood</td>
</tr>
</tbody>
</table>

One prominent difference is that DSM-IV does not distinguish between primary insomnia sub-types, unlike the ICSD-2 where there are four specified sub-types. DSM-IV tends to focus on primary insomnia as an exclusionary diagnosis, where insomnia disorder is present in the absence of additional psychiatric, medical, sleep or substance abuse
pathology. Conversely, ICSD-2 has specific characteristic inclusion criteria for each sub-type (see table 1.2 for a description of sub-type features).

Table 1.2 - Essential features of primary insomnia phenotypes in ICSD-2, redrawn from Schutte-Rodin et al. (2008).

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Essential features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychophysiological Insomnia</td>
<td>The essential features of this disorder are heightened arousal and learned sleep-preventing associations. Arousal may be physiological, cognitive, or emotional, and characterized by muscle tension, “racing thoughts,” or heightened awareness of the environment. Individuals typically have increased concern about sleep difficulties and their consequences, leading to a “vicious cycle” of arousal, poor sleep, and frustration.</td>
</tr>
<tr>
<td>Paradoxical Insomnia</td>
<td>The essential feature of this disorder is a complaint of severe or nearly “total” insomnia that greatly exceeds objective evidence of sleep disturbance and is not commensurate with the reported degree of daytime deficit. Although paradoxical insomnia is best diagnosed with concurrent PSG and self-reports, it can be presumptively diagnosed on clinical grounds alone. To some extent, “misperception” of the severity of sleep disturbance may characterize all insomnia disorders.</td>
</tr>
<tr>
<td>Idiopathic Insomnia</td>
<td>The essential feature of this disorder is a persistent complaint of insomnia with insidious onset during infancy or early childhood and no or few extended periods of sustained remission. Idiopathic insomnia is not associated with specific precipitating or perpetuating factors.</td>
</tr>
<tr>
<td>Inadequate Sleep Hygiene</td>
<td>The essential feature of this disorder is insomnia associated with voluntary sleep practices or activities that are inconsistent with good sleep quality and daytime alertness. These practices and activities typically produce increased arousal or directly interfere with sleep, and may include irregular sleep scheduling, use of alcohol, caffeine, or nicotine, or engaging in nonsleep behaviours in the sleep environment. Some element of poor sleep hygiene may characterize individuals with other insomnia disorders.</td>
</tr>
</tbody>
</table>

Numerous epidemiological studies have been conducted to assess insomnia symptom and disorder prevalence rates (Ohayon, 2002). Estimates naturally depend on the definition used, but prevalence of insomnia symptoms is estimated to affect one third of the population, and insomnia disorder (core sleep symptoms and daytime impairment) affects approximately 5-10% (Roth, 2007). Of note, few epidemiological studies have implemented strict diagnostic criteria in relation to both classification manuals (i.e. ICSD-2 & DSM-IV), or recorded data on whether the individual actually endorses a sleep problem/complaint. A recent important study in the field by Ohayon & Reynolds III (2009) employed both DSM-IV and ICSD-2 criteria to assess prevalence rates in a large sample of
individuals (25,579), aged 15 and over, residing in seven European countries. It was found that 34.5% of the sample reported at least one difficulty at the ‘symptom’ level (i.e. sleep initiation and/or maintaining difficulties or non-restorative sleep). Just less than ten percent (9.8%) were found to meet insomnia at the criterion level; that is, reporting both night-time symptoms and daytime consequences. After excluding those not scoring ‘positive’ for the complaint of insomnia, it was found that 6.6% of the general population met criteria for DSM-IV insomnia disorder. When further broken down, based on DSM-IV criteria, 3.3% of the sample met classification for primary insomnia. Most of the specialised ICSD-2 insomnia sub-types had a prevalence of less than 1% in the general population, and the prevalence of Psychophysiological Insomnia, the most common primary insomnia subtype, was found to be 1.4%. These results reasonably approximate previous epidemiological studies (e.g. Morin et al., 2006a; NIH state-of-the-science conference statement, 2005; Ohayon, 2002), though an important issue that arose was that a large number of individuals who, although reporting an insomnia complaint, failed to be categorized by the classification systems, suggesting future refinement of criteria and measurement may be necessary.

1.2. Risk factors and precipitants
Several risk factors have been associated with increased insomnia prevalence. Among the most strongly supported are: increasing age, being female, shift work, and co-morbid medical and psychiatric disorders (Roth, 2007). Psychiatric disorders are particularly pronounced in those with insomnia; with estimates suggesting 40% of all patients with insomnia experience a co-occurring psychiatric condition (Ford & Kamerow, 1989).
Specifically, depression and anxiety have been found to be highly prevalent in those with an insomnia diagnosis compared to those without (Taylor et al., 2005). Moreover, those with insomnia syndrome, relative to both good sleepers and individuals with insomnia symptoms, demonstrate higher scores on questionnaire measures of depression, anxiety, neuroticism, extraversion, arousal predisposition, and stress perception, as well as a tendency towards emotion-oriented coping (LeBlanc et al., 2007). Another intriguing finding from this latter study by LeBlanc and colleagues was that the group with insomnia symptoms also tended to score higher than good sleepers on measures of depression, anxiety, and neuroticism; suggesting a possible linear trend with increasing insomnia ‘severity’.

In a recent study of good sleepers ($n=464$), measured at three time-points over the course of a one-year period, many of these aforementioned factors (recorded at baseline) were also found to be involved in the onset of insomnia symptom ‘incident’ cases, the onset of insomnia syndrome ‘incident’ cases, and new (first time) onset of an insomnia syndrome (Beaulieu-Bonneau et al., 2007a). Natural evolution studies of this nature have the capacity to substantially advance our understanding of the aetiology, development and maintenance of chronic insomnia. For example, similar prospective longitudinal work by the same group, over a three year period, also revealed that insomnia disorder tends to persist long-term, with little deviation in severity (Morin et al., 2009).

Clinically, patients often ‘anchor’ the onset of sleep disturbance to significant life events. In support of this, Bastien and colleagues (2004a) systematically examined precipitating
factors of insomnia in a sample of 345 patients presenting at a sleep disorders clinic. They discovered that events relating to family, health, and work or school, were most frequently associated with the onset of sleep disturbance. Of note, across all recorded events, the majority (65%) were considered to be negative in nature (e.g. loss of job, bereavement).

A few studies have reported increased familial susceptibility to developing insomnia. For example, Dauvilliers et al. (2005) found that 73% of a sample of primary insomnia patients \((n=77)\) had a positive family history for familial insomnia, compared with just 24% in a normal-sleeping control group. A recent population-based study similarly found that those with a past (or current) history of insomnia had a greater likelihood of a positive family history for insomnia, relative to good sleepers that had never experienced insomnia before (Beaulieu-Bonneau et al., 2007a). Moreover, monozygotic twins have also been found to have higher rates of insomnia concordance, within pairs, in comparison with dizygotic twin pairs (Watson et al., 2006).

Although candidate genes have not yet been reliably identified, there are several possible targets relating to sleep homeostasis, circadian timing, and general arousal/de-arousal regulation (Riemann et al., 2010); all of which appear to be worthwhile investigating, and particular so given the high heritability of sleep EEG power spectra \((\delta, \theta, \alpha, \sigma \text{ bands};\) Ambrosius et al., 2008) as well as conventional PSG sleep parameters (Tafti, 2009). It is worth mentioning that a very recent paper (Deuschle et al., in press) has, for the first time, revealed an association between primary insomnia and a serotonin transporter length polymorphism \((5\text{-HTTLPR short allele}).\) This association remained robust after controlling
for those with a lifetime incidence of affective disorder. Such a finding needs replicated but is intuitively appealing given the greater occurrence of this genotype within other stress-related psychiatric conditions (which are frequently related to insomnia) and suggestions of enhanced cortisol reactivity to experimental psychosocial stress in healthy individuals with this genotype (Way & Taylor, in press).

1.3. Insomnia assessment

Insomnia is typically diagnosed according to subjective report; that is, through clinical interview, prospective sleep diary completion, and retrospective questionnaire assessments. Assessments probe sleep and functioning symptoms, but also beliefs and attitudes about sleep, pre-sleep arousal, and applied sleep effort – constructs considered important markers of insomnia, particularly the psychophysiological phenotype (Espie & Kyle, 2009). Polysomnography (PSG), the gold standard for objectively measuring sleep, is only indicated in circumstances where insomnia is suspected to be related to other sleep disorder pathology, such as periodic leg movements and sleep-related breathing disorders (Reite et al., 1995).

There are at least two main reasons for this conservative use of PSG. Firstly, it has been known for several decades that individuals with insomnia tend to (as a group) misperceive sleep: significantly underestimating total sleep time and overestimating time taken to fall asleep compared with PSG recordings (Carskadon et al., 1976). This notwithstanding, insomnia patients report statistically significant differences in sleep parameters compared with normal sleepers, such as reduced total sleep time and sleep efficiency, and greater
number of awakenings; these differences, however, are typically disproportionate to the subjective complaints of patients (e.g. Feige et al., 2008). Indeed, the core defining feature of the paradoxical insomnia sub-type (see table 1.2) is gross sleep misperception, when a substantial mismatch occurs between subjective report and objective recordings (PSG or actigraphy).

Finally, PSG is an expensive procedure, and given the high prevalence of insomnia disorder in the general population, coupled with limited clinical/treatment insight, overnight laboratory-recording of sleep is not cost-effective, at least in insomnia clinical practice. It is worth pointing out though that PSG is recommended in research as a screening tool and as an outcome measure in efficacy studies (though not as the primary dependent variable; Buysse et al., 2006).

The lack of clinical necessity for objective sleep recordings, however, does not mean that PSG has had little impact on furthering our understanding of pathophysiologic mechanisms relevant to insomnia disorder. For example, power spectral (microstructure) analysis of EEG amplitude during wake, non-REM sleep, and REM sleep states provides information on level of cortical arousal/excitability. A number of studies report evidence for increased power, both relative and absolute, in fast EEG rhythms (sigma, beta, or gamma frequencies) during wakefulness, NREM sleep or REM sleep, compared with normal sleepers (Merica et al., 1998; Freedman, 1986; Perlis et al., 2001a; Perlis et al., 2001b; Krystal et al., 2002; Buysse et al., 2008). These alterations tend to occur in the absence of conventional PSG abnormalities (when compared with controls), and are thought to
account, in part, for the subjective experience of being awake during objectively scored sleep (e.g. Borkovec et al., 1981). Indeed, in the Perlis study (2001b), it was found that increased beta power during NREM sleep was associated with greater subjective-objective sleep discrepancies. Similarly, slow wave spectral power in the delta range (0.5-4Hz), a marker of homeostatic drive for sleep (or sleep intensity), has been found to be both reduced (Merica et al., 1998; Krystal et al., 2002) and increased (Buysse et al., 2008*) in PI patients relative to controls. Variability in methods, small sample sizes, and new findings concerning sex differences (Buysse et al., 2008), suggests more work in this area is required.

Another emerging technique to study the microarchitecture of sleep is the cyclic alternating pattern (CAP). CAP can be thought of as a measure of sleep instability (Terzano et al., 2002), represented as periodic ‘activation’ features of non-REM sleep, involving delta bursts, K-complexes, K-alpha complexes, and other arousal-related events, which occur in defined clusters (so-called phase A types: of which there are three) interspersed with background EEG activity (phase B type). Alternatively, a period of non-CAP (> 60 seconds in duration in the absence of phase A type) is characterised by the absence of activation patterns, and therefore is considered a marker of sleep stability (Terzano et al., 2002). In one study, untreated (placebo) individuals with primary insomnia were found to spend more time in CAP, have a greater CAP rate (% of CAP time spent in NREM sleep), and a higher number of phase A1 and A2 types, compared with normal sleepers (Terzano et al., 2003). Interestingly, sleep quality assessed using a 100mm visual analogue scale (VAS) correlated, negatively, and more strongly, with CAP rate than conventional PSG

* Spectral differences were only found for female insomnia patients
parameters. A recent study also found that total CAP rate, and, in particular, CAP rate for sleep stages 1 and 2, was higher in individuals with paradoxical insomnia relative to normal sleepers (Parrino et al., 2009). It is argued that CAP rate may be related to the misperception of sleep and reports of poor sleep quality in those with insomnia. This technique appears promising for understanding possible microstructural abnormalities not otherwise measureable using conventional PSG parameters.

1.4. Insomnia costs: economic and psychobiological considerations

A handful of studies have estimated both direct (e.g. health-care utilization, physician consultations) and indirect costs (downstream consequences, e.g. work productivity) of insomnia (Martin et al., 2004). Perhaps the most comprehensive study to date on cost impact was carried out by Daley and colleagues (2009a) in the province of Quebec, Canada. A random sample \( n=948 \) of residents were classified, according to strict diagnostic criteria, into good sleepers, those with insomnia symptoms, and those with insomnia syndrome (disorder). Assessments were made for use of health-care services, products to treat sleep disturbance, accidents, insomnia-related work absenteeism and productivity (presenteeism). Objective data were also obtained through a government register regarding recorded health-related consultations. The total estimated cost of insomnia for that particular region (when extrapolated) was $6.6 billion (Canadian) dollars. In particular, insomnia-related absenteeism, reduced work-related productivity, and use of alcohol as a sleep aid were the three biggest contributors. The average annual cost per-person with insomnia syndrome was $5,010; $1,431 for those with insomnia symptoms;
and just $421 for good sleepers. This study underscores the economic costs induced by both chronic and transient symptoms of sleep disturbance.

Insomnia also appears to have general health costs. It has been two decades since Ford & Kamerow (1989) published their seminal finding that insomnia is a risk factor for the development of subsequent psychopathology (depression). This relationship has been repeatedly found in several investigations (for reviews see Riemann & Voderholzer, 2003 and Pigeon & Perlis, 2007), suggesting that insomnia may be an independent predictor of depression. Indirect mounting evidence also supports a role for insomnia in the perpetuation of depression. For example, Pigeon and colleagues (2008) show, in a large sample of elderly individuals undergoing treatment for major depression, that persistent insomnia at baseline is significantly associated with poorer (depression) treatment response. Moreover, specifically targeting insomnia within the context of standard treatment for depression - using both cognitive behavioural therapy (Manber et al., 2008) and Eszopiclone (Fava et al., 2006) - subsequently leads to improvements in sleep but also potentiates the anti-depressant effect beyond monotherapy.

Associations between insomnia and cardiovascular morbidity have similarly been reported for some time, though possible confounders have limited conclusions regarding causality (Bonnet & Arand, 2007). An important study by Vgontzas et al. (2009) has recently advanced the field in terms of identifying a robust link between insomnia and hypertension. Using PSG recordings (one night) in a large random population sample \((n=1,741)\) it was found that those meeting criteria for insomnia disorder, and who also (objectively) slept
less than five hours, had a greatly increased risk of experiencing hypertension. This was not the case for individuals without insomnia symptoms and who also slept less than five hours (short sleepers). Importantly, this insomnia-hypertension relationship held after controlling for several major confounders, including depression and sleep apnea. Recent experimental data showing attenuated systolic blood pressure (SBP) day-to-night dipping, and elevated night-time SBP in normotensive primary insomnia patients compared with normal sleepers, provides one possible pathway for this link between sleep disturbance and hypertension risk (Lanfranchi et al., 2009). Another interesting finding from this study was that insomnia patients showed a trend towards increased beta activity in NREM sleep, and beta activity (across the whole group) was positively associated with night-time SBP.

**1.5. Contemporary models of insomnia**

Over the years, several ‘single factor’ as well as multi-factorial models have been put forward to account for the aetiology and maintenance of chronic insomnia. These ‘single factor’ accounts have tended to focus on specific, focused abnormalities, which have some degree of support in the existing literature, and include: stimulus dyscontrol and instrumental conditioning (Bootzin, 1972); altered sleep homeostasis (Pigeon & Perlis, 2006); alterations in the circadian parameters involved in the timing of sleep (Lack & Wright, 2007); physiological hyperarousal preventing the de-aroused state necessary for sleep (Bonnet & Arand, 1997; Richardson, 2007); and dysfunctional cognitive processes surrounding sleep and sleep-related daytime functioning (e.g. Harvey, 2002). Whilst these ‘models’ are well formulated, it is likely that multi-component perspectives are required to capture the heterogeneity of insomnia symptoms, associated sub-types, and insomnia
development/trajectory. Although it is outwith the scope of this brief review to describe each multi-component model in detail, it is worth outlining some of the main accounts, which are inclusive and encompassing with regard to associated characteristics of insomnia disorder.

1.5.1. The 3P (predisposing, precipitating, perpetuating) model: a general framework

The main framework for most working models of insomnia was set out by Spielman et al. (1987a) in the form of the 3P model. This stress-diathesis conceptualization outlines how chronic insomnia may develop over time; proposing, as a first step, that acute sleep disturbance occurs as a consequence of both predisposing (e.g. altered neurotransmission, trait arousal, genetic susceptibility, ruminative personality etc.) and precipitating factors (life-stressors such as occupational stress, emotional and health problems). Perpetuating factors refer to maladaptive sleep practices, which interact with experienced insomnia symptoms, and are aimed at coping with the consequences of poor sleep during the day (e.g. drinking coffee to improve alertness) or directly trying to increase the probability of ‘achieving’ sleep (e.g. extending time in bed). After the precipitant resolves, most individuals will return to the default mode of sleep automaticity, but in those with a predisposition for sleep disturbance, combined with the continued practice of maladaptive perpetuating behaviours, sleep disturbance may become chronic.

Thus a main assumption of this model is that sleep disturbance may, over time, become dislocated from the precipitating trigger (Ebben & Spielman, 2009). Such a model is intuitively appealing because it suggests that treatment should target, specifically,
perpetuating factors involved in the maintenance of insomnia. Indeed, this is exactly what cognitive behavioural therapy for insomnia (CBT-I) attempts to do; with an emphasis on correcting maladaptive coping strategies, behaviours and sleep-related dysfunctional beliefs and attitudes (Espie & Kyle, 2009).

1.5.2. Neurocognitive Model

Perlis and colleagues (1997) extend this behavioural perspective, acknowledging that acute insomnia is initially precipitated by life stress, is similarly maintained by maladaptive coping strategies (for example, extending time in bed), but that, importantly, the associated wakefulness becomes classically conditioned in terms of arousal (somatic, cognitive and cortical). It is argued that increased cortical arousal (as measured by fast rhythms in the EEG) at sleep-onset, during sleep, and middle of the night awakenings, subsequently disrupts sleep initiation and maintenance through enhanced sensory/information processing and attenuated mesograde amnesia. These altered cognitive parameters may subsequently help explain sleep-state misperception. A later addition to this model also includes the possibility that sleep-related objects (bed, pillow etc.) become conditioned stimuli for cortical arousal (Perlis et al., in press A) and hence contribute to the perpetuation of continued sleep disturbance.

1.5.3. Cognitive Model

Harvey (2002) describes a (maintenance) model of insomnia which focuses primarily on dysfunctional cognitive processes, based upon a large body of work from the anxiety disorders literature. Because it is arguably the first model to give equal attention to both
daytime and night-time factors, it is worth outlining some of its main features. Harvey argues that individuals with insomnia tend to excessively worry about sleep and catastrophize about the consequences of not getting adequate sleep (in relation to impact on health and daytime functioning). Ensuing negatively toned cognitive activity, typically about sleep but also other (negatively valenced) life issues, coupled with the application of maladaptive safety behaviours, results in elevated autonomic arousal and emotional distress. As a consequence of this heightened stress state, individuals with insomnia tend to monitor for sleep-related threat cues (internal and external) to confirm that they have not slept and that functioning is adversely affected. Pre-existing dysfunctional beliefs exacerbate the situation. Otherwise innocuous cues are subsequently misinterpreted as evidence for sleep and daytime deficits, ‘tricking’ the individual into overestimating both the level of sleep and daytime impairment. This serves to cause further worry and concern about not sleeping, which may, through feedback mechanisms, increase anxiety and cognitive load, leading to the enhanced possibility of a ‘real’ deficit occurring in both sleep and daytime functioning. The main assumption of this model is that cognitive processes have a causal role in the maintenance of an insomnia state.

1.5.4. Psychobiological Inhibition/Attention-Intention-Effort (PIM/AIE) model

Espie (2002) and Espie and colleagues (2006) take a starting point of normal sleep for their model of insomnia. They acknowledge that normal sleep is governed by two oscillatory processes - a self-sustained oscillating circadian rhythm and an ‘hourglass’ sleep homeostat - rendering the (adaptive) sleep process automatic, involuntary and, hence, not under direct control. Acute stressful life events can however create both physiological and
psychological ‘over-arousal’, which interacts negatively with normal sleep-wake regulation, leading to acute sleep disturbance. For most individuals, the ‘plasticity’ of the sleep system accommodates such transient disruptions, without any lasting chronic modifications. However, it is argued that the development of acute to chronic insomnia, where the defining feature is a fundamental difficulty in inhibiting wakefulness, is precipitated by three related cognitive processes (Espie et al., 2006). Attending to sleep-related stimuli, explicitly intending to sleep, and applying voluntary effort to the sleep onset process, all represent an attempt to control sleep, an otherwise automatic process. These attempts have the opposite effect: preventing de-arousal by failure to reach a level of inhibitory sufficiency. Factors relevant to this resultant ‘sleep effort syndrome’ (Espie, 2007), include enhanced sleep-preoccupation, affect dysregulation, sleep-incompatible conditioning, dysfunctional beliefs and expectations about sleep, and enhanced focus on the consequences of poor sleep.

1.5.5. Beyond dualistic tendencies: towards an integrative psychobiological insomnia model

Although not strictly a new ‘model’, it would be fair to say that in the last few years a new perspective has been put forward to explain insomnia across a number of different ‘levels’; with particular emphasis on underlying neurobiology. For example, in the most up-to-date account, Riemann and colleagues (2010) synthesise work on the hyperarousal concept of insomnia, focusing on hyperarousal across autonomic, neuroendocrine, neuroimmunological, electrophysiological and neuroimaging parameters. They also draw on extensive theorizing by Perlis and colleagues (2007; 2009; Pigeon & Perlis, 2007) in relation to possible neurobiological abnormalities relevant to the features of insomnia; as
well as cutting-edge work on rodent models of stress-induced insomnia (Cano et al., 2008). The development of testable hypotheses has been aided largely by recent understandings on the neurobiology of normal sleep-wake regulation (Saper et al., 2005a; Schwartz & Roth, 2008).

The perspective outlined is a further update of the Neurocognitive account, originally set out by Perlis et al. (1997). Although at a speculative stage regarding possible mediators of insomnia aetiology and development, it is proposed that a genetic arousal predisposition may render certain individuals at greater risk of developing insomnia via altered neurobiology and neurochemistry. In particular, modified levels of several neurochemicals, including orexin, monoamines (histamine, dopamine, norepinephrine, serotonin), adenosine, and the stress hormone cortisol, may, during acute periods of stress, disrupt arousal-related (ascending reticular activating system; ARAS) and/or sleep-promoting (ventrolateral preoptic nuclei; VLPO) components of the ‘flip-flop’ sleep switch. Inputs to the VLPO from various limbic structures may also directly impact the capacity to de-arouse adequately, overcoming homeostatic sleep pressure (Saper et al., 2005b). This, of course, would normally represent a typical adaptive response to stress. However, the subsequent development of chronic ‘arousal’, post-acute phase, possibly more likely in those with a genetic predisposition for arousability and/or increased stress responsivity, interacts with maladaptive sleep practices, resulting in both circadian and homeostatic dysregulation. This dysregulation may, over time, induce chronic changes in the sleep-wake system, perhaps reflected in cortical hyperarousal, arousal across other physiological parameters, and difficulties in inhibiting wakefulness. Recent work by Seugnet et al. (2009) in which
Drosophila melanogaster were artificially selected and bred over many generations to create insomnia-like characteristics (sleep initiation and maintenance difficulties, and daytime impairment), helps one conceive of an inherited sleep-system with reduced plasticity and/or an altered stress reactivity threshold (Perlis et al., in press A).

Although this perspective focuses predominantly on neurobiology, it clearly acknowledges that insomnia is a psychobiological disorder, with psychological and neurobiological abnormalities likely to be highly inter-related (Perlis et al., 2009). This seems to be a more convincing integrative account of insomnia and its associated features, rather than just a pure physiological hyperarousal perspective (see Bonnet & Arand, 2010). Indeed, in an inclusive depiction, physiological changes are paralleled with cognitive-behavioural features, which are likely to interact at multiple levels, capturing maintenance and development factors relevant to the original neurocognitive model, Spielman’s 3P framework, Espie’s PIM/AIE formulations, and Harvey’s cognitive model. It will be important to investigate insomnia at each ‘level’, and ultimately to understand how genetic, biological, and psychological factors interact to determine the course of sleep disturbance over time.

1.6. Evidence-based treatment of Insomnia

It is important to note, firstly, that the majority of patients with insomnia do not actually seek treatment for their sleep difficulties (Morin et al., 2006a; Bartlett et al., 2008). Although only a small amount of work has been carried out to understand why this is the case, it appears likely that a perception of sleep as not being viewed important by the
medical profession (Stinson et al., 2006); a poor understanding and/or recognition of sleep disturbances by health-care providers; and limited public knowledge of available treatments (Ancoli-Israel & Roth, 1999), may mediate, to some extent, this phenomenon.

1.6.1. Pharmacotherapy

A number of pharmacological agents exists for the treatment of insomnia symptoms, most of which primarily work via their agonistic effects on gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the central nervous system (CNS). Specifically, the most common class of hypnotics, benzodiazepines (e.g. temazepam), and the newer non-benzodiazepine receptor agonists (zolpidem, eszopiclone; BZRAs or ‘z drugs’) modulate GABA$_A$ receptors, facilitating the inhibitory effects of GABA on overall CNS arousal, while similarly inhibiting norepinephrine activity (Mitchell & Wienshenker, in press). BZRAs are considered more effective due to their greater selectivity for the alpha 1 sub-unit, thus enhancing activity in terms of sedation and limiting more generic effects involved in the interaction with other subunits (Nutt & Stahl, in press). These compounds also tend to have shorter half-lives than the original benzodiazepines, reducing the likelihood of carry-over effects the next day; though available data on side-effects and comparable efficacy do not allow for clear conclusions to be made (e.g. Dundar et al., 2004; Krystal, 2009).

Although demonstrating effectiveness (moderate to large effects) in terms of improving major indicators of sleep continuity and quality in those with insomnia, positive effects of BZRAs/benzodiazepines have yet to be reliably demonstrated beyond active administration
Indeed, as it stands, there is not enough evidence to suggest or recommend that BZRAs/benzodiazepines are useful for the long-term management of chronic insomnia beyond four weeks of treatment (NIH state-of-the-science conference statement, 2005). Some recent studies report improvements during extended/intermittent use of hypnotics over 6 (Walsh et al., 2007) and 12 month periods (Ancoli-Israel et al., 2005), but again no adequate data exist indicating maintained benefits long after treatment cessation. Concerns about long-term hazardous side-effects and tolerance issues strongly argue against long-term prescription: safety and efficacy must be documented (Kripke, 2000).

The only other class of sleep-promoting agents, licensed and approved for the treatment of insomnia, are melatonin receptor agonists, acting on M1/M2 receptor sites in the Suprachiasmatic Nucleus (SCN) of the hypothalamus. Endogenous melatonin is secreted by the pineal gland under the control of the SCN; absence of light input to the SCN leads to a rise in melatonin from early evening onwards, which is paralleled by an increase in sleepiness and a decrease in core body temperature – suggesting melatonin is involved in the regulation of the sleep/wake rhythm, and hence, has somnogenic properties (Sateia et al., 2008; Arnedt & Skene, 2005).

Ramelteon® is the only approved melatonin agonist on the (US) market and has been investigated in a few trials. Although demonstrating low potential for abuse, and limited side effects (i.e. similar to placebo) in comparison with GABA-mediated hypnotics, Ramelteon appears to have only moderate effects on sleep latency, and little/no impact on WASO and TST (Sateia et al., 2008). Circadin®, an extended-release melatonin agonist
was also recently approved for use in the UK (and other parts of Europe). Circadin® is only indicated for short-term use (< 1 month) in over 55s – owing to documentation of melatonin deficiency in elderly individuals with insomnia (e.g. Haimov et al., 1994). Data are limited, but there are some beneficial reports in terms of sleep continuity parameters, sleep quality, and daytime functioning, and the safety profile appears encouraging (e.g. Luthringer et al., 2009; Wade et al., 2007). More multi-centred trials with long-term follow-ups are required to determine the role of exogenous melatonin in the management of chronic insomnia.

A number of other drugs are also used ‘off-label’ to treat insomnia, including sedative anti-depressants, anti-neuroleptics and atypical (second generation) anti-psychotics; though it is important to note, this is not evidence-based practice. Indeed, the most frequently prescribed hypnotic in the US is the sedative anti-depressant, trazadone, yet only one randomized trial of this therapy has been conducted with insomnia patients, with unconvincing results (Krystal, 2009). The uncovering of mechanisms involved in basic sleep-wake regulation has led to the development of a number of novel compounds, several of which are currently under investigation within clinical trials. These include orexin antagonists, novel melatonin compounds, histamine receptor antagonists, and specific serotonin (5HT) antagonists - which may help lead to the development of safer and more effective pharmacotherapies for insomnia (Sullivan & Guilleminault, 2009).
1.6.2. Cognitive Behavioural Therapy for insomnia (CBT-I)

Given that maladaptive behaviours and cognitive processes are thought to underlie the maintenance of insomnia, it is intuitive that therapy targets these factors directly (Edinger & Means, 2005). Cognitive behavioural therapy for insomnia (CBT-I) is an evidence-based treatment modality, containing a number of supported techniques to improve sleep. Practice parameters set out by the American Academy of Sleep Medicine (AASM) recommend and endorse the following single components: paradoxical intention therapy, stimulus control therapy, sleep restriction therapy, progressive muscular relaxation, and biofeedback (Chesson et al. 1999; Morgenthaler et al., 2006). In addition, two multi-component CBT approaches are also supported. Indeed, most outcome research has focused on multi-component, multisession CBT interventions. See table 1.3 for a description of the main components that make up a typical CBT-I programme.

Table 1.3 - Main CBT-I components, taken from Morin et al. (2006b).

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stimulus Control Therapy</strong></td>
<td>A set of instructions designed to re-associate the bed/bedroom with sleep and to re-establish a consistent sleep-wake schedule: (1) go to bed only when sleepy; (2) get out of bed when unable to sleep; (3) use the bed/bedroom for sleep only (no reading, watching TV, etc.); (4) arise at the same time every morning; (5) no napping.</td>
</tr>
<tr>
<td><strong>Sleep Restriction Therapy</strong></td>
<td>A method designed to curtail time in bed to the actual amount of sleep time. For example, if a patient reports sleeping an average of 6 hours per night, out of 8 hours spend in bed, the initial recommended sleep window (from lights out to final arising time) would be restricted to 6 hours. Periodic adjustments to this sleep window are made contingent upon sleep efficiency, until an optimal sleep duration is reached.</td>
</tr>
<tr>
<td><strong>Relaxation Training</strong></td>
<td>Clinical procedures aimed at reducing somatic tension (e.g. progressive muscle relaxation, autogenic training) or intrusive thoughts at bedtime (e.g. e.g. imagery training, mediation) interfering with sleep.</td>
</tr>
<tr>
<td><strong>Cognitive Therapy</strong></td>
<td>Psychological methods aimed at challenging and changing misconceptions about sleep and faulty beliefs about insomnia and its perceived consequences. Other cognitive procedures may include paradoxical intention or methods aimed at reducing or preventing excessive monitoring of and worrying about insomnia and its correlates/consequences.</td>
</tr>
<tr>
<td><strong>Sleep Hygiene Education</strong></td>
<td>General guidelines about health practices (e.g. diet, exercise, substance abuse) and environmental factors (e.g. light, noise, temperature) that may promote or interfere with sleep. This may also include some basic information about normal sleep and changes in sleep patterns with aging.</td>
</tr>
<tr>
<td><strong>Cognitive-Behavioural Therapy (CBT)</strong></td>
<td>A combination of any of the above behavioural (e.g. stimulus control, sleep restriction, relaxation) and cognitive procedures.</td>
</tr>
</tbody>
</table>
CBT-I has a large and extensive evidence-base (spanning over 20 years) in treating insomnia as a primary disorder, and mounting evidence for the successful improvement of insomnia symptoms in the context of co-occurring conditions (Riemann & Perlis, 2009; Morin et al., 2006b; Edinger et al., 2009). Two reviews carried out by the American Academy of Sleep Medicine (AASM) taskforce revealed that CBT improves sleep parameters in approximately 70% of insomnia patients (Morin et al., 1999b; Morin et al., 2006b). Effectiveness studies in primary care settings similarly report strong CBT-I effects, including those where patients are already on prescription hypnotics (Espie et al., 2007; Morgan et al., 2003). Studies have also demonstrated maintenance of sleep improvements long after active treatment – indeed, up to two years follow-up (Morin et al., 1999a; Espie et al., 1989). Maintenance of gains is important given the persistent and chronic nature of insomnia (cf. Morin et al., 2009), explaining why CBT is the treatment of choice for the management of chronic insomnia. This was appropriately summarized by the recent National Institutes of Health (NIH) state-of-the-science conference statement (2005):

“… [CBT] has been found to be as effective as prescription medications are for brief treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT, in contrast to those produced by medications, may last well beyond the termination of active treatment” (p.1052)

Direct comparisons between CBT and pharmacotherapy reinforce this conclusion: treatment gains are similar during the acute treatment phase, but medicated individuals tend
to regress after treatment cessation, compared with CBT-treated individuals who maintain, or improve on, established gains (e.g. Morin et al., 1999b; Jacobs et al., 2004; Sivertsen et al., 2006a). Despite strong evidential support and demonstrations of cost-effectiveness (e.g. Morgan et al., 2004), access to CBT-I remains limited. Thus, as Espie (2009) notes:

“The challenge for CBT is no longer to prove its credentials, but to punch its weight. For at least a decade, CBT should have been a contender as the treatment of first choice for insomnia. In reality, however, it has had very little impact on the high volume of insomnia patient care. Indeed, it has amounted to little more than a patchy cottage industry.”

(p.1549)

Solutions to making CBT-I more available are currently being formulated and tested. These include: innovative health care models (Espie, 2009); telephone consultations (Bastien et al., 2004b); condensed brief CBT-I interventions (e.g. Edinger & Sampson, 2003; Germain et al., 2007); bibliotherapy (Mimeault & Morin, 1999); DVD and television broadcasts (Van Straten et al., 2009); and internet programmes (Ritterband et al., 2009; Vincent & Lewycky, 2009). This work has important implications for how CBT research and evidence can be translated into everyday clinical practice.

1.6.3. What constitutes effective treatment?

It is abundantly clear that CBT-I is an effective treatment for improving night-time symptoms of insomnia – several systematic and meta-analytic reviews support this perspective (for a synthesis see Riemann & Perlis, 2009). However, none of these reviews report on (perhaps) the major concern of insomnia patients – functional impact/impairment, the main determinant of treatment-seeking. Surely, ‘effective’ interventions should also be
defined in terms of how they affect how a person feels and functions? The same is also true for pharmacotherapy research. Indeed, often in these trials the focus is on documenting a lack of daytime impairment/side-effects as a consequence of treatment initiation (compared with placebo), rather than actually attempting to demonstrate positive effects on daytime functioning and related aspects of quality of life.

There are signs, however, that this exclusive focus on night-time symptoms is changing to a more thorough investigation of disorder, and treatment impact, on the ‘whole patient’. This thesis represents a step towards better understanding and measuring insomnia-related daytime functioning and quality of life. Central to this work is the notion that insomnia patients, and their narratives, are powerful sources of data, which have the potential to help better understand and clarify several related aspects of insomnia, particularly those concerning disorder impact and treatment effectiveness.

The next chapter will now outline, firstly, what is currently known about the daytime consequences and associated morbidity of insomnia. The review will then turn to the more global constructs of Health-Related Quality of Life (HRQoL) and Quality of Life; with particular emphasis on definition, measurement and treatment effects, in insomnia populations. On the basis of this thorough review, a prospective research agenda is set out for the insomnia field as a whole, in relation to insomnia and HRQoL. Some of these important research objectives are subsequently addressed and explored in the following chapters of this thesis.
Chapter 2:

Insomnia and Health-Related Quality of Life

The contents of this chapter have been published as follows:

2.1. Abstract

Health-related Quality of Life (HRQoL) has become an important construct in contemporary medicine and health care, permitting assessment of disorder burden and evaluation of interventions on various aspects of functioning, in a standardized manner. Here we review literature on the measurement of HRQoL in insomnia populations, and the extent to which insomnia treatment improves domains of HRQoL. It is concluded from the relatively small literature that insomnia impacts on diverse areas of HRQoL, and that both pharmacological and non-pharmacological interventions can produce, to varying degrees, improvements in domains spanning physical, social and emotional functioning. Limitations of the current literature are identified; with particular emphasis on measurement and conceptual shortcomings. Suggestions are made in relation to improving the quality of future research, and how to further shed light on the impact of insomnia - and treatment thereof - on both HRQoL and global quality of life.
“They call this a “sleep disorder”, but it’s actually an all-day disorder. Insomnia is not just something that happens to the night, it happens to the day, the whole day, and if it’s chronic insomnia, it happens to many days. *A half-life of ruined days.*”

*Greene (2008, p28)*

**2.2. Introduction**

Despite Insomnia being recognised as a ‘24-hour disorder’ in both the major sleep nosologies (ICSD, 2005; DSM-IV), historically, there has been less interest in the daytime aspects of insomnia compared with night-time symptoms and sleep parameters. Recent recommendations from leading researchers in the field (Buysse et al., 2006; Morin, 2003; NIH state-of-the-science conference statement, 2005) encourage further investigations into the waking consequences and correlates of insomnia. Indeed, clinical research in general, across a wide spectrum of illnesses, has moved towards a more holistic approach; looking beyond proximal symptoms, and viewing the patient within their wider psychosocial context (Armstrong et al., 2007).

The purpose of this review is to: a) give a brief overview of the known daytime consequences and morbidity associated with insomnia; b) review work on the definition and measurement of quality of Life (QoL), or more specifically, health-related quality of Life (HRQoL), as it relates to insomnia; and finally, c) outline a prospective research agenda, focusing on further understanding and measuring the extent to which insomnia, and its treatment, impacts HRQoL and individual QoL.
2.3. Insomnia: daytime consequences and associated morbidity

Impairment in daytime functioning attributed to disturbed and/or poor quality sleep, features as one of the core diagnostic criteria for insomnia disorder (ICSD-2, 2005; DSM-IV; Edinger et al., 2004). Clinician reports of patient consultations (Moul et al., 2002), and cross-sectional (Roth & Ancoli-Israel, 1999) and prospective questionnaire studies (Buysse et al., 2007; Levitt et al., 2004), reveal that individuals with insomnia report consistent decrements in mood and cognitive abilities (concentration, memory, attention), coupled with elevated levels of anxiety, fatigue and physical pain/discomfort, relative to normal sleepers. Such impairments persist in those diagnosed with primary insomnia (PI), after screening and excluding co-morbid pathology (Buysse et al., 2007; Kyle et al., in press; Lichstein et al., 1997; Orff et al., 2007; Varkevisser et al., 2005). Evidence for subjective (and objective) sleepiness, on the other hand, remains equivocal (Riedel & Lichstein, 2000; Sanford et al., 2006), with surveys revealing approximately 25% of PIs report excessive daytime sleepiness (Day et al., 2001).

Although reviews of earlier studies failed to find reliable unequivocal differences in objective, neuropsychological functioning (Riedel & Lichstein, 2000; Fulda & Schulz, 2001), more recent controlled work, using sensitive measures that vary in task complexity and cognitive load, appear to be isolating and capturing specific impairments in attention and vigilance (Varkevisser & Kerkhof, 2005; Edinger et al., 2008a; Altena et al., 2008a). The heterogeneity in findings may be explained by both methodological (e.g. sample size and composition, assessment tools, time of testing), and theoretical factors (e.g. ‘negative cognitive set’, compensatory effort, and cortical arousal; Altena et al., 2008a; Harvey,
2002; Bastien et al., 2003; Winkelman et al., 2008). Further work is needed to tease out the contributing roles of each. Interestingly though, a recently presented meta-analysis (Fortier-Brochu et al., 2008) of all studies assessing neuropsychological performance in well-defined PIs, revealed significant impairments (small to moderate effect sizes) in aspects of attention, episodic and working memory, and executive functioning domains. Other groups have focused on the known relationship between sleep and consolidation of newly encoded memory traces. Specifically, PIs show an attenuation of the normal overnight sleep enhancement effect on tasks probing declarative (Backhaus et al., 2006) and procedural memory performance (Nissen et al., 2006), relative to normal sleeping controls.

Recent structural and functional imaging work may also shed light on the neural underpinnings of the cognitive dysfunction experienced by insomnia patients. For example, Riemann and colleagues (2007) reported decreased bilateral hippocampal volume in a small sample of PIs (n=8) relative to controls, and, more recently, Altena et al. (2010) found decreased gray matter volume in the left orbitofrontal cortex† and parietal cortices (specifically the precuneus), in a larger sample (n=24) of elderly individuals with primary insomnia. Furthermore, the first published functional MRI study in PIs revealed hypoactivation of medial and inferior prefrontal regions during a verbal fluency task – a pattern which reversed/normalised post-behavioural therapy (Altena et al., 2008b). Although this imaging work is still in its infancy (and issues of causal ordering remain to be resolved), such atrophy, and functional alterations, may map onto reported daytime impairments in mood, memory, and reduced cognitive flexibility. Daytime event-related

† gray matter density significantly and strongly (negatively) correlated with subjective insomnia severity
potential (ERP) data, though relatively sparse, also point to potential impairments in aspects of attention and processing speed (e.g. Bruder et al, 1991; Szelenberger & Niemcewicz, 2001); though again, more work in this area using sophisticated paradigms, and large, well-defined samples is required. Recent ERP studies assessing sensory and cognitive processing pre-sleep, during sleep, and on awakening (Bastien et al., 2008; Devoto et al., 2005), over multiple nights, are beginning to tease out the relationship between sleep quality and cortical arousal, which may have implications for better understanding daytime insomnia phenomenology (Turcotte & Bastien, 2009).

Large survey and population-based studies further reveal a number of increased morbidity markers in those suffering from insomnia, including: increased rates of health care utilization (physician visits, medication prescriptions) and chronic health problems (Leger et al., 2002; Simon & Vonkorff, 1997; Hatoum et al., 1998), elevated work absenteeism rates, reduced work productivity, and greater frequency of motor and non-motor accidents (Roth & Ancoli-Israel, 1999; Leger et al., 2006; Daley et al., 2009b). Such impairments may be moderated, in part, by co-occurring illness; however, workplace studies controlling for both mental and physical co-morbidities still reveal significant negative effects of insomnia on objective absenteeism, self-report work efficiency (Leger et al., 2006), and work disability pension claims (Sivertsen et al., 2006b). Longitudinal epidemiological studies also indicate that isolated sleep disturbance, measured at time point one, can independently predict the development of a new depressive episode 1-3 years later (for a review see Riemann & Voderholzer, 2003). Recent work also confirms insomnia as a predictor of future clinical anxiety (Neckelmann et al., 2007).
Crucially, perceived impact on daytime functioning serves as an important factor in driving help-seeking behaviour among individuals with insomnia, rather than simply perceived sleep loss (Stepanski et al., 1989). For example, Morin and colleagues (2006a) found, in a large epidemiological study, that four out of five of the most commonly cited reasons for seeking a sleep-related consultation with a health professional, were daytime consequences of fatigue, psychological distress, physical discomfort, and reduced work productivity. Thus, once a threshold of noticeable daytime dysfunction is reached, individuals feel motivated to seek medical advice – ultimately with the hope that successful treatment will restore the particular functional impairment back to ‘normal’ status (Henry et al., 2008).

2.4. Quality of Life (QoL) and Health-related Quality of Life (HRQoL)

In recent years there has been a shift towards assessing the overall impact of illness on aspects of QoL, through the measurement of HRQoL (Testa & Simmons, 1996). QoL and HRQoL have become well established terms in the medical and health literature – indeed, a PubMed search reveals that published work with the term ‘Quality of Life’ in the title or abstract has risen more than fourfold in the last ten years (1998-2008: 62,641), relative to the previous decade (1988-1998: 14,428). This has occurred mainly because of the recognition that objective changes in pathology rarely correlate with, or predict improvements in, functional capacity or patient experience, and that what the individual desires when seeking treatment is, put simply, a return to pre-illness well-being. Similarly, with increasing life expectancy and medical technology advancement, the emphasis has shifted to chronic illness management (the quality of life) rather than simply the extension (quantity) of life. HRQoL has thus become a variable that can help policy makers decide
on which treatments should get resources and service provision, relative to competing others (through cost-effective analyses and health technology assessment).

One effect of this increased attention to quality of life measurement has been the tendency, criticised in recent reviews and commentaries (Gill & Feinstein, 1994; Moons et al., 2006 Dijkers, 2007), for researchers to include QoL and HRQoL scales in intervention and epidemiological studies without paying much attention to the concept they are purporting to measure. That is, QoL has become an ‘umbrella term’ (Feinstein, 1987) for a number of different concepts and definitions. The distinction between QoL and HRQoL is an important one, yet much of the health/medical literature seems to use these two terms interchangeably. QoL is widely regarded as a complex phenomenon: some argue it encompasses both objective and subjective indices of well-being (Cummins, 2000); whereas others suggest it is a purely subjective impression of ‘life satisfaction’ (Moons, 2006). Factors relevant to quality of life may thus range from emotional functioning and happiness, through to material well-being and education; it is therefore difficult to measure, not least with a single generic instrument. The World Health Organization (Herrman et al., 1993) defines QoL as:

“An individual’s perception of their position in life, in the context of the culture and values in which they live and in relation to their goals, expectations, standards, and concerns” (WHOQoL study group)

A more contemporary definition proposed by Ruta et al. (2007), based on seminal work by the economist-philosopher, Amartya Sen, views QoL in terms of a ‘gap hypothesis’:
“Quality of Life is the gap between what a person is capable of doing and being, and what they would like to do and be; in essence it is the gap between capability and expectations” (p. 402)

Inherent in both these approaches is the importance of the individual in the assessment of QoL. The role of relativism and subjectivity in QoL assessment is perhaps best illustrated by the ‘disability paradox’ (Albrecht & Devlieger, 1999): individuals who may have society-defined functional/health impairment (e.g. cancer sufferers, amputees, the physically disabled), can report satisfactory, or in some cases, enhanced, quality of life (Moons et al., 2006). Thus, health is just one of the many components implicitly factored into the quality of life equation. Simply put, having poorer health status does not necessarily mean that one has a lower quality of life, than say someone in impeccable health (Car & Higginson, 2001).

HRQoL assessment, on the other hand, is concerned with isolating the impact of disease or illness on prominent aspects of functioning – ‘…the radiating impact of pathology on the patient’s wider world’ (Armstrong et al., 2007, p578). In clinical medicine the ‘pathology’ has both immediate, proximal symptoms (for example, in the case of insomnia, an increased sleep latency, or reduced total sleep time), and more ‘downstream’, distal consequences (such as reduced work performance, and social impairment). It is these latter, more psychosocial, variables that are the target of HRQoL assessment, capturing impairment relevant to patients’ everyday functioning. Because health impact is easier to quantify than global QoL, most generic HRQoL instruments typically focus on similar
aspects of functioning: covering physical, psychological (emotional) and social well-being (Wood-Dauphinee, 1999). Subsumed under these functioning domains are isolated symptoms, such as mood, memory, and fatigue. The various ‘levels’ of measurement have been likened to a pyramid (Spilker, 1990): the top of which can be thought of as overall subjective well-being/quality of life; the second level representing a collection of functional domains; and the third, foundation level, consisting of a number of isolated symptoms. Disease-specific HRQoL scales will tend to be tailored to aspects of impaired functioning that are most salient within a particular population, covering a mix of global functioning domains and relevant symptoms (e.g. fatigue, pain, and physical functioning in cancer patients). Table 2.1 provides descriptions of the scales used to assess HRQoL and QoL in insomnia populations.

2.5. Does insomnia negatively affect HRQoL?

Given the reported daytime symptoms attributed to poor sleep (ICSD-2, 2005; DSM-IV, 1994; Edinger et al., 2004) it is reasonable to assume that individuals suffering from a chronic sleep problem may have a somewhat reduced ‘downstream’ HRQoL. Indeed, about two decades ago, the first studies began to appear focusing on the relationship between insomnia and HRQoL. Rombaut and colleagues (1990) created what they call the Quality of Life of Insomniacs (QOLI) questionnaire, a 52-item scale designed from the amalgamation of three other questionnaires (Leeds sleep evaluation questionnaire, the Jenkins sleep evaluation scale, and the Psychological well-being index) and 22 additional items, to assess functioning across five broad domains: quality of sleep, physical well-being, mood and mental state, and social and professional/work relationships.
<table>
<thead>
<tr>
<th>Concept measured</th>
<th>Instrument</th>
<th>Brief description</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generic Health status/HQoL</td>
<td>SF-36</td>
<td>36 items covering 8 dimensions of functioning: physical functioning, physical role limitation, bodily pain, vitality, mental health, emotional role limitation, social functioning, health perception. Can calculate dimension scores, and two component summary scores for mental and physical well-being. 12 and 8-item versions are also available.</td>
<td>Appears sensitive to insomnia impairment and treatment Extensive normative data and disease norms available</td>
</tr>
<tr>
<td></td>
<td>NHP</td>
<td>38 items assessing impact of illness across 6 dimensions (sleep, energy, pain, physical mobility, social isolations, emotional reactions). Combined weighted values of individual items make up total dimension scores (0-100).</td>
<td>Simpistic dichotomous response format. Focus on extreme ill-health</td>
</tr>
<tr>
<td></td>
<td>SIP</td>
<td>136 yes/no items grouped into 12 categories (body care and movement, ambulation, mobility, social interaction, alertness behaviour, emotional behaviour, household management, recreation and pastimes, communication, eating, work, sleep and rest). Items are completed with reference to ‘today and because of health’. Global profile score, category scores, and summary physical and psychosocial scores, can be calculated.</td>
<td>Exhaustive number of items</td>
</tr>
<tr>
<td>Quality of Life</td>
<td>QoLI</td>
<td>Based on a model of ‘life satisfaction’, covering 17 domains (health and non-health) identified from the literature as being important for overall ‘life satisfaction’: health, self-regard, philosophy of life, standard of living, recreation, learning, creativity, social service, civic action, love relationship, friendships, relationships with children, relationships with relatives, home, neighbourhood, community.</td>
<td>Total score is based on domains only regarded as important and relevant by respondents.</td>
</tr>
<tr>
<td></td>
<td>Q-LES-Q (short form)</td>
<td>Contains 16 item-domains (health &amp; non-health). 14 of these single item-domains make up the total score (physical health, mood, work, social relations, ability to function in daily life, ability to get around physically, household activities, family relationships, leisure, sexual drive, economic status, living or housing situation, vision, overall sense of well-being).</td>
<td>Sensitive to treatment outcome in depression and anxiety.</td>
</tr>
<tr>
<td>Disease-specific HQoL</td>
<td>QOLI</td>
<td>52-item scale encompassing three questionnaires (Leeds Sleep evaluation questionnaire, Jenkins Sleep Evaluation Scale, Psychological well-being index) plus additional item questions, grouped into five domains: quality of sleep, quality of waking, physical well-being, mood and mental state, and relationships.</td>
<td>Varying response formats</td>
</tr>
<tr>
<td></td>
<td>HD-16</td>
<td>16-item scale, covering five core domains (physical role, energy, cognitive, social, and psychological well-being). Global and domain scores can be calculated.</td>
<td>Items generated by insomnia patients and experts</td>
</tr>
</tbody>
</table>

HD-16 = Hotel Dieu-16; Q-LES-Q = Quality of life enjoyment and satisfaction questionnaires; QOLI = Quality of life of insomniacs questionnaire; QoLI = Quality of life inventory; NHP = Nottingham health profile; SF-36 = Short-Form health survey; SIP = Sickness impact profile.
The scale therefore measures both sleep and non-sleep variables. The initial pilot study revealed good discriminant properties between untreated insomnia patients and normal sleeping controls, with the patient group showing statistical impairments on each domain. However, the scale has not been widely used since; only a few early studies have employed it (Kelly et al., 1993; Goldenberg et al., 1994), or variants of it (DeSouza, 1996). This is likely to be because of poor face validity (i.e. items are not grounded in the words of individuals with insomnia), the simultaneous evaluation of both sleep and daytime variables, varied response formats, and the exhaustive number of items. Nevertheless, the limited data on the QOLI do indicate sensitivity to impairment in a number of HRQoL domains, relative to normal sleepers.

The bulk of published studies focusing on HRQoL and insomnia have used a generic health status measure, the Medical Outcomes Study short-form health survey 36 (SF-36; Ware & Sherbourne, 1992). The SF-36 is a generic instrument, initially designed for assessment of health status across different disease states. Eight dimensions (36 items) assess aspects of functioning (emotional role limitations, energy and vitality, social functioning, physical functioning, physical role limitations, bodily pain, mental health) and perceived general health, yielding two component summary scores for mental and physical well-being. Scores range from 0 to 100 - with lower scores indicating greater impairment in health status, or HRQoL. Short forms of the SF-36, the SF-12 and SF-8 have also been published and validated.

The first studies to use this instrument with insomnia populations consistently demonstrated lower scores on all domains, relative to normal sleepers (Hatoum et al., 1998; Zammit et al., 1999; Hajak & Sine, 2001). Several large survey studies have also
now reported a graded trend with insomnia severity; that is, those with more ‘mild’ or occasional insomnia symptoms show greater impairment on all eight domains relative to normal sleeping controls, whereas individuals (typically) satisfying criteria for insomnia disorder score significantly lower than both groups (Hatoum et al., 1998; Leger et al., 2001; Schubert et al., 2002; Katz & McHorney, 2002; LeBlanc et al., 2007*). Such associations continue to hold after controlling for both physical (Schubert et al., 2002; Katz & McHorney, 2002) and mental health co-morbidities (Leger et al., 2001; Katz & McHorney, 2002). Although these studies differ in terms of what they classify as ‘mild’ or ‘severe’ insomnia, the linear pattern between HRQoL and insomnia severity consistently emerges.

More recently, Dixon and colleagues (2006) analyzed baseline SF-36 data (n=209) collected during a randomized trial of Cognitive Behavioural Therapy (CBT) to reduce hypnotic intake (cf. Morgan et al., 2003), and compared them with UK normative values. In this study, the authors stratified domain scores by age, creating three groups (30-49, 50-69, 70-100 yrs), and compared them with their corresponding age-matched reference values. Similarly, it was found that those meeting minimum criteria for insomnia disorder (DSM-IV), in the ‘young’ category (ages 30-49), had significantly lower scores on all domains of the SF-36; the middle category (50-69 yrs) were impaired on all but two domains (emotional role limitation, physical role limitation); and the elderly category were significantly impaired in four out of the eight domains (pain, vitality, mental health, physical functioning), relative to reference values. The high degree of co-morbidities, particularly chronic illness and pain interference (anxiety and depression scores were in the normal to mild ranges), may mediate, to some extent, the magnitude of impaired dimensions.

* LeBlanc et al. used the SF-12 in their Quebec population-based study
In contrast to the more clinical- and effectiveness-based approach of Dixon et al., Walsh and colleagues (2007) collected SF-36 data in the context of a multi-centred RCT of nightly Eszopiclone for a six-month period, on a large sample \(n=830\), 21-64 yrs of well-defined PIs (sleep parameter inclusion and DSM-IV criteria). Comparison of pre-treatment SF-36 scores with US normative reference values, revealed significant impairments in vitality, social functioning and the overall mental health summary component. The more selective decrements are likely to be explained by the stringent level of screening/exclusion and the nature of recruited versus referred populations (e.g. Stepanski et al., 1989; Davidson et al., 2009).

In a similar vein, Omvik and colleagues (2008) recently published baseline data on daytime functioning and QoL variables from a randomized trial of CBT versus zopiclone (cf. Sivertsen et al., 2006a). SF-36 scores from 46 elderly individuals (>55 yrs: mean age 60.9 yrs) with DSM-IV criteria insomnia, who had undergone rigorous screening (including PSG), failed to reveal any significant evidence of impairment relative to normative values. The authors do not, however, report data for each of the eight dimensions; instead using the mental health and physical health component summary scores for comparison. This may obscure differences at the dimension level. Nonetheless, data collected on another generic instrument, the Quality of Life Inventory (QoLI; Frisch et al., 1992), in the same sample, also revealed scores within the normal range. The QoLI is based on a conceptual model of general ‘life satisfaction’, asking patients to rate the importance of 17 pre-determined domains deemed important for an enjoyable life, and thus covers a wide-range of items not necessarily specific to health. It is perhaps not surprising that the composite score of this measure failed to detect insomnia-relevant impairment. It is worth pointing out, however, that the key strength
of this scale is that composite scores are calculated only on items that are rated as important/relevant by the individual.

Another study (Lichstein et al., 2001a), again focusing on older adults (58+ years), compared well-defined PIs ($n=82$), normal sleepers ($n=61$), and those with ‘secondary’ insomnia ($n=46$) on dimensions of the SF-36. Only one difference was found for the comparison between PIs and normal sleepers; with PIs scoring lower on the vitality dimension. Those with ‘secondary’ insomnia were significantly impaired on seven of the eight dimensions (except role-emotional) relative to both the PI group and normal sleepers. The reduced sensitivity of the SF-36 to HRQoL impairment in elderly individuals with insomnia, especially those with primary insomnia, seems to be a recurrent finding. It is unclear what may be mediating this trend. Perhaps elderly individuals, who have had their sleep problem for a long period of time, adapt and ‘recalibrate’ in a way that limits the impact of insomnia on aspects of functioning. Conversely, normative reference values for this age group may be low anyway, given the high level of co-morbidity and reduced functional abilities associated with normal ageing - subsequently obscuring potential differences.

Researchers have also used a variety of other tools to assess HRQoL. For example, Philip et al. (2006) compared those with insomnia ($n=986$), and normal sleepers ($n=586$), on the Nottingham Health Profile (NHP; Hunt et al., 1980). The NHP, similar to the SF-36, measures salient areas of health functioning, comprising 38 yes/no statements under six main domains: energy level, pain, sleep, social isolation, emotional reactions, and physical abilities. Individuals with insomnia were characterized according to DSM-IV symptom criteria, and had to report difficulties at least three
times per week for the last three months; daytime functioning was not assessed. Significant differences were observed on all domains, with the insomnia group evidencing significant impairment. This pattern of impairment was similarly found in a selected sub-group \((n=442)\) of the insomnia sample, screened for both organic sleep complaints (using the Epworth Sleepiness Scale; ESS) and depressive symptoms.

Leger and colleagues (2005) recently published the Hotel Dieu 16 (HD-16), a disease-specific measure designed to detect quality of life disturbance relevant to insomnia. Items for the HD-16 were initially generated from interviews with 20 patients and through expert consensus opinion. Factor analysis on an initial list of 43 items resulted in the final selection of 16 items, subsumed under five categories: physical role; energy, will to do things; cognitive (concentration, attention, memory); social (relationships with others); and psychological well-being. Good sleepers \((n=391)\), individuals with ‘mild’ insomnia \((n=422)\), and individuals with ‘severe’ \((n=240)\) insomnia, were selected from SOFRES, a French polling institute. The ‘mild’ insomnia group were defined as those with ‘occasional’ sleep difficulties; whereas those in the severe group had at least two insomnia complaints for the last month, and suffered impaired daytime functioning as a consequence. Initial screening excluded those with depressive or anxiety profiles; those with other medical co-morbidities were not identified or excluded. A linear trend was again found, similar to studies using the SF-36: the mild insomnia group scored significantly lower on all dimensions, and global score, relative to good sleepers; individuals with severe insomnia scored significantly poorer than both groups, again on each dimension and total score. Thus, \textit{prima facie}, the HD-16 does look sensitive to the functional impairments experienced by those with insomnia, and has good face validity given that items are grounded in words of sufferers. A further
strength is the ability to configure a total score, which may be useful in outcome studies, as well as clinical settings. Despite being a relatively brief measure, however, dimension calculation looks to be a rather complex and arduous process (based on weighted coefficients from the factor analysis), and so this may preclude it from being used on a regular basis in clinical settings. Further studies are required to assess psychometric properties and sensitivity to change.

The HD-16 was principally devised because of the absence of a widely used insomnia-specific HRQoL instrument, as compared with, for example, the sleep apnea field, where there are three - the Functional Outcomes of Sleep Questionnaire (FOSQ; Weaver et al., 1997), the Sleep Apnea Quality of Life Index (SAQLI; Flemons & Reimer, 1998), and the Québec Sleep Questionnaire (QSQ; Lacasse et al., 2004). The implication being: generic measures like the SF-36 may not pick up impairments relevant to those with insomnia (Moul et al., 2004). Nevertheless, the main strength of the SF-36 is that it permits comparisons with other illnesses; which of course is vital in order to document the relative burden of insomnia. In this context, Katz & McHorney (2002) found, in a cross-sectional analysis of data from the medical outcomes study (MOS), that those with mild and severe insomnia (defined in terms of frequency of symptoms over a 4-week period) scored significantly lower on all domains of the SF-36 relative to a mild hypertension reference group. More importantly, however, the magnitude and distribution of HRQoL decrements of the severe insomnia group were comparable to individuals with clinical depression and congestive heart failure (see figure 2.1), even after controlling for 16 co-morbid conditions, and various other demographics. This pervasive nature of insomnia fits well with what patients report: sleep disturbance has a knock-on effect on nearly every aspect of daily functioning.
Kyle et al., in press), whereas many other conditions are more selective in their consequences.

Figure 2.1 - Deviations in SF-36 scores for Insomnia, Depression, and Congestive Heart Failure, relative to hypertension reference group. Scores plotted from Katz & McHorney (2002).

PF = physical functioning; RP = role-physical; BP = bodily pain; GHP = general health perception; VT = vitality/energy; SF = social functioning; RE = role-emotional; MH = mental health.

One final line of evidence to support the view that disturbed sleep can have a bearing on aspects of HRQoL comes from studies looking at the additive effect of poor sleep on existing conditions. In a recent study, ‘poor sleep’ (measured using the PSQI) was found to be a significant independent predictor of mental and physical health component scores (from the SF-36) in patients with multiple sclerosis (Merlino et al., 2009). Fortner et al. (2002), again using the PSQI, categorized breast cancer patients into ‘good’ and ‘bad’ sleepers; finding that ‘bad’ sleepers scored lower on six (role-physical, bodily pain, vitality, social functioning, role-emotional, and mental health) of the eight SF-36 dimensions. Other studies using more concrete definitions of insomnia
reveal a similar pattern. For example, Caap-Ahlgren & Dehlin (2001) reported that Parkinson’s disease patients with insomnia had impaired HRQoL on every dimension of the SF-36, relative to those without insomnia symptoms. Rumble et al. (2005) also demonstrated lower scores in lung cancer patients meeting criteria for insomnia on the ‘global health/quality of life’ 2-item sub-scale scale from the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ C30; Aaronson et al., 1993), a cancer-specific HRQoL questionnaire, relative to a control group of cancer patients without insomnia.

Overall, cross-sectional studies highlight the pervasive impact of insomnia on aspects of functioning and HRQoL, and this relationship holds, to varying degrees, after controlling for co-morbidities. Well-screened PIs appear to have more selective impairments relative to those individuals in the population-based and clinical studies, which might be expected given the reported (and unreported) co-morbidities in the latter groups. There is some evidence to suggest that individuals with co-morbid insomnia experience greater/entrenched daytime functioning impairments relative to PI patients (Lichstein et al., 2001a). Crucially, both disease-specific and generic instruments appear to be sensitive to HRQoL impairments.

2.6. Does improving insomnia also improve aspects of HRQoL?

Because health-related functional impairments are prevalent within insomnia populations, and enshrined in the diagnostic criteria, treatment should ultimately target and alleviate such impairments. That is, improving sleep should improve functioning (this of course is based on the notion that impaired sleep is causally related to reduced HRQoL). Surprisingly, very few controlled studies (see table 2.2) have included
assessments of HRQoL (see Krystal, 2007, for a review of more general daytime parameters), and only two have specified HRQoL as the primary outcome measure.

Table 2.2 specifies the following: all controlled studies of insomnia treatment that include a HRQoL or QoL measure; two additional uncontrolled trials of insomnia treatment, specifying HRQoL as one of the primary outcome variables of interest; and finally two large RCTs of CBT-I in those with insomnia and cancer (selected only as examples of how treating insomnia can modify HRQoL variables pertinent to the co-occurring illness). All papers were sourced by searching major search engines (PubMed, ISI Web of Knowledge, ScienceDirect), and through hand-picking citations from existing published articles.

2.6.1. Cross-sectional & uncontrolled studies

Leger and colleagues (1995) conducted a cross-sectional study, selecting patients with insomnia who had been on zopiclone for at least twelve months, and compared them with a matched group of good sleepers, on a study-specific QoL instrument. ‘Quality of life’ was assessed using questions probing five core domains: work, relationships, safety, domestic activities, and leisure activities. The groups did not differ in terms of sleep disturbance, except that the Zopiclone patients had ‘occasional’ difficulties falling asleep. It was also found that both groups had comparable scores for each QoL domain. Similarly, using a cross-sectional design, Zammit and colleagues (1999) compared recruited treated insomnia patients with a matched group of untreated patients, on the SF-36. They, however, found no differences across dimensions between groups. The use of a non-validated questionnaire in the Leger et al. study, and the poor
<table>
<thead>
<tr>
<th>Study</th>
<th>Methods/intervention</th>
<th>Sample size/characteristics</th>
<th>Instrument used</th>
<th>Sleep outcome</th>
<th>HRQoL outcome</th>
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<tr>
<td>Uncontrolled</td>
<td>Multi-national randomized trial of zolpidem 10mg 5 nights/week (remaining 2 nights were given placebo) versus nightly use, for 14 days.</td>
<td>789 PIs (DSM-IV) free from medication at intake</td>
<td>SF-36</td>
<td>Both discontinuous and continuous groups had similar numbers (59 v. 65%) rating 'much improved' or 'very much improved' on the CGI-II Improvements on all SF-36 dimensions were similar between groups. No within-subject statistical testing reported.</td>
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<td>Hajak et al. 2002</td>
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<td>Three sub-scales from the SIP (social interactions, alertness/ intellectual functioning, and recreation) and four 'sub-scales' from the RAND-36 (general health; 'problems at work', 'social occupation', and 'feeling')</td>
<td></td>
<td>Both groups had significantly improved SOL, WASO, TST, and SE, relative to baseline Composite ratings on both scales significantly improved at 9-month follow-up, for both group and individual CBT.</td>
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<tr>
<td>Verbeek et al. 2006</td>
<td>Compared individual CBT with group CBT</td>
<td>32 PIs (Individual CBT) versus 74 with mix of PI and co-morbid insomnia (group CBT)</td>
<td></td>
<td>Both groups had significantly improved SOL, WASO, TST, and SE, relative to baseline Composite ratings on both scales significantly improved at 9-month follow-up, for both group and individual CBT.</td>
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<td>Goldenberg et al. 1994</td>
<td>Multi-national RCT of zopiclone 7.5mg versus placebo taken nightly for 2 weeks, and on demand for 6 weeks thereafter</td>
<td>231 received zopiclone; 227 received placebo. Participants had to report two insomnia symptoms for inclusion, and were excluded for co-occurring illness or CNS affecting medication.</td>
<td>QOLI</td>
<td>Zopiclone group had significantly greater improvements in the sleep domain 2 month follow-up: both groups improved on the psychological well-being domain and overall score, but treatment group had significantly greater improvements on activity, social, and work/profession domains.</td>
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<tr>
<td>Walsh et al. 2000</td>
<td>RCT of zolpidem 10mg versus placebo (3-5 nights per week for 8 weeks)</td>
<td>163 PIs meeting DSM-IV criteria</td>
<td>SF-36</td>
<td>Treatment group significantly improved on patient global ratings and diary measures of SOL, TST, NAW, and sleep quality No significant group differences at any time point (4,8 weeks) Small but significant improvement on the Physical component summary score for Valerian compared with placebo group at 4-week assessment point.</td>
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<tr>
<td>Morin et al. 2005</td>
<td>Randomized placebo controlled trial of valerian-hops and diphenhydramine</td>
<td>184 'mild' insomniacs, experiencing initiation and/or maintenance problems for between 2.4 times per week for at least a month.</td>
<td>SF-36</td>
<td>Both treatments had a mild hypnotic effect at two-week assessment relative to placebo Significant CBT improvements on global health/quality of life scores for Valerian compared with placebo group at 4-week assessment point.</td>
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<td>Savard et al. 2005</td>
<td>RCT of CBT versus wait-list control</td>
<td>57 females who had undergone treatment for breast cancer, and reported significant insomnia for 6 months (DSM-IV/ICSD-2 and standard quantitative criteria)</td>
<td>Sub-scale assessing health/global quality of life' taken from the EORTC-CQLQ-C30</td>
<td>Significant CBT improvements on measures of SOL, WASO and SE post-treatment relative to wait-list controls Significantly greater improvements on global health/quality of life' scores for the CBT treated patients at post-treatment. Pooled data for both groups revealed maintenance of improvements at 12 months, compared to baseline.</td>
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<td>Scharf et al. 2005</td>
<td>RCT of Eszopiclone 1mg, 2mg or placebo nightly for 2 weeks.</td>
<td>231 Elderly PIs (age range 66-86) meeting DSM-IV criteria</td>
<td>Q-LES-Q</td>
<td>Significant treatment affects for 2mg on SOL, WASO and TST relative to placebo 2mg treatment group demonstrated improvements in five domains (physical health, mood, household activities, leisure time activities, and medication)</td>
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<td>Study</td>
<td>Design/Intervention</td>
<td>Participants</td>
<td>Measures</td>
<td>Results</td>
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<td>Dixon et al. 2006</td>
<td>RCT of group CBT versus 'no additional' treatment in a general practice clinical setting (Morgan et al., 2003)</td>
<td>209 hypnotic users meeting diagnostic criteria (DSM-IV) for insomnia</td>
<td>SF-36</td>
<td>CBT group had significantly decreased PSQI global score and lower percentage of group on hypnotics, compared with controls. CBT group significantly improved on physical functioning, emotional role limitation, and mental health, relative to control group (6 month time adjusted means).</td>
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<td>Espie et al. 2007</td>
<td>RCT of group CBT versus TAU in a clinical setting</td>
<td>201 individuals meeting DSM-IV/ICSD-2 and quantitative criteria for insomnia</td>
<td>SF-36</td>
<td>CBT group significantly improved on measures of SOL and WASO at 6 months relative to TAU group. At 6 months: significant improvements in energy/vitality and mental health dimensions (small ES) for CBT versus TAU.</td>
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<td>Walsh et al. 2007</td>
<td>Multi-centred RCT of Eszopiclone 3mg versus placebo, nightly for six months</td>
<td>830 PIs</td>
<td>SF-36</td>
<td>SOL, WASO, TST significantly improved relative to placebo at six months (medium to large ES). Vitality (small to moderate ES), social functioning, physical functioning, and bodily pain (small ES) were all significantly improved relative to placebo group at 6 months.</td>
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<td>Espie et al. 2008</td>
<td>Pragmatic RCT of CBT versus TAU</td>
<td>150 cancer patients undergoing active cancer treatment, and reporting significant insomnia (meeting standard quantitative criteria for at least three nights in last three months, and daytime functioning impairment).</td>
<td>FACT-G (functional assessment of cancer therapy). Sub-scales physical, emotional, social and functional well-being</td>
<td>CBT group significantly improved on measures of SOL, WASO and SE (large ES), relative to TAU. At 6 months, CBT group significantly improved on the physical and functional domains (large ES).</td>
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<td>Szeftling et al. 2008</td>
<td>Randomized trial of three-component CBT (stimulus control, sleep hygiene, relaxation) versus sham biofeedback</td>
<td>47 hypnotic-dependent older adults</td>
<td>SF-36</td>
<td>CBT significantly improved on SOL, WASO and SE (medium to large ES) relative to sham control group. Neither group improved on any dimension of the SF-36, at 4 weeks.</td>
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<td>Omvik et al. 2008</td>
<td>RCT of Zopiclone 7.5mg versus group CBT versus pill placebo for 6 weeks (Sivertsen et al., 2006)</td>
<td>46 elderly (mean age = 55) adults meeting criteria for PI</td>
<td>SF-36 &amp; QoL</td>
<td>Post-treatment: CBT significantly improved relative to placebo and zopiclone groups on (objective) WASO and SWS; both treatment groups had improved objective SE compared with placebo (PSG). CBT group more improved on objective (WASO, SE, SWS) and subjective (WASO) parameters relative to zopiclone group at 6 months. No evidence of improvements between or within groups, post-treatment, or at 6 months follow-up (placebo group not included in analyses).</td>
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CBT, Cognitive Behavioural Therapy; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; EORTC QLQ-C30, European Organization for Research and Treatment of Cancer Quality of Life Questionnaire; ES, Effect Size; FACT-G, Functional Assessment of Cancer Therapy – General; CGI-II, Clinical Global Impressions Scale; ICSD-2, International Classification of Sleep Disorders, 2nd Edition; ISI, Insomnia Severity Index; NAW, Number of Awakenings; NHP, Nottingham Health Profile; PI, Primary Insomnia; PSG, Polysomnography; PSQI, Pittsburgh Sleep Quality Index; Q-LES-Q, Quality of Life Enjoyment and Satisfaction Questionnaire; QOLI, Quality of Life of Insomniacs Questionnaire; QoLI, Quality of Life Inventory; RCT, Randomised Controlled Trial; SE, Sleep Efficiency; SF-36, Short-Form Health Survey; SIP, Sickness Impact Profile; SOL, Sleep Onset Latency; SWS, Slow-Wave Sleep; TAU, Treatment As Usual; TST, Total Sleep Time; WASO, Wake-time After Sleep Onset.
characterization of patients in the Zammit et al. investigation, coupled with the cross-sectional nature of both papers, means these data do not provide strong enough evidence to help elucidate the relationship between HRQoL and treatment of insomnia.

As part of a multi-national study, Hajak et al. (2002) compared continuous versus non-nightly zolpidem in a large sample of individuals with insomnia. Both groups had a similar number of ‘responders’, as determined by the clinical global impression improvement score, and SF-36 improvements were also similar for both groups across all dimensions (non-significant group x time effect). However, there was no appropriate placebo/control group, and within-group analyses were not reported on the dimensions; it is therefore hard to tell if and how treatment affected specific domains of HRQoL.

Verbeek et al. (2006) reported data from both individual (n=32) and group CBT (n=74) on primary outcomes of both sleep and HRQoL parameters. Across treatment modalities, sleep-onset latency (SOL), wake time after sleep-onset (WASO), sleep efficiency (SE), and total sleep time (TST), significantly improved and remained robust at the 9 month post-treatment assessment. To sample HRQoL domains relevant to those with insomnia, the authors selected items probing four domains (‘general health’, ‘problems at work’, ‘social occupation’, and ‘feeling’) from the RAND-36, a practically identical scale to the SF-36, and three sub-scales (social interactions, alertness/intellectual functioning, and recreation) from the 136-item sickness impact profile (SIP; Bergner et al., 1981). Both treatment groups showed comparable improvements in ‘global’ scores of the RAND-36 and SIP, with the SIP demonstrating the most robust effects, at 9-month follow-up. The authors, however, did not detail

*The Rand-36 is an exact replica of the SF-36 in terms of content, but has different scoring algorithms for the Bodily pain and General Health sub-scales.*
scores for each subscale, in either the RAND-36 or SIP, making it impossible to identify what specific HRQoL components were most sensitive to the CBT intervention. The lack of a control group also prevents ruling out nonspecific effects; it is notable that the intermediate phase before receiving therapy (i.e. between baseline and post-waitlist) had significant positive effects on both sleep and HRQoL measures. The authors believe sleep hygiene advice, provided at the first screening interview, may account for this, although the current literature on sleep hygiene recommendations for insomnia would perhaps argue against this interpretation (cf. Stepanski & Wyatt, 2003). A relevant control group seems essential for an accurate interpretation of HRQoL outcomes; the placebo impact of sham CPAP, for example, has been documented in OSA patients, where ‘active’ and control groups can show comparable improvements across a number of functioning and HRQoL domains (Atkeson & Basner, 2008).

2.6.2. Prospective controlled trials

A number of prospective controlled trials have included a validated instrument to assess QoL or HRQoL domains. Goldenberg et al. (1994) conducted a multi-national RCT of zopiclone versus placebo. Two hundred and thirty one patients were randomized to receive 14 days of zopiclone (and on demand for six weeks thereafter) and 227 received placebo. All patients completed Rombaut et al.’ QOLI. At two weeks, both groups showed improvements in the psychological well-being component and overall global ‘quality of life’ score, but the zopiclone group demonstrated significantly greater improvements in the sleep, activity, social, and work/profession domains. Relative improvements remained for activity, sleep and social domains at the two-month follow-up.
Walsh et al. (2000) conducted an 8 week RCT of zolpidem 10mg versus placebo (3-5 nights per week) in 163 PIs. Although sleep parameters improved significantly post-treatment in the experimental group, there was no corresponding change on any dimension of the SF-36. Dixon and colleagues, in their analysis of SF-36 outcome data from 209 individuals on hypnotics (with continuing insomnia) randomized to either CBT or control, found simultaneous improvements in sleep and HRQoL, relative to a control group. PSQI global scores significantly decreased, as too did the percentage of hypnotic users in the experimental group. Time-weighted mean adjustments across the 6 month follow-up period revealed significant improvements in physical functioning, emotional role limitation, and mental health, relative to the control group. On closer inspection however, these ‘improvements’ do appear to be largely mediated by declining functioning in the control group (e.g. up to a 16 point decrease for the emotional role dimension). Regardless, the data show an overall tendency for the CBT group to improve, albeit marginally, and the control group to experience further impairment. CBT improvements, in this particular population, might then be argued to act as an important buffer, preventing further (likely) decrements in HRQoL.

More recently, Soeffing et al. (2008) reported data from a randomized trial of a three-component cognitive behavioural intervention (stimulus control, relaxation, and sleep hygiene) or sham biofeedback (psychological placebo), in a small sample of hypnotic-dependent older adults (n=47). At post-treatment, significant effects were found for the CBT treated group in terms of subjective sleep parameters (SOL, WASO and SE; medium to large effects) but there was no corresponding improvement in SF-36 scores (mean dimension/component scores were not reported). Indeed, there was no improvement in any of the other daytime assessments probing sleepiness, fatigue,
anxiety or mood. The rather early post-treatment assessment could perhaps undermine improvements, particularly using the SF-36 instrument which has a 4-week recall period.

Espie and colleagues (2007) conducted an RCT of CBT versus treatment as usual (TAU) in general practice, using trained nurses as CBT therapists. At the 6 month follow-up, small to moderate effect sizes were found for SOL and WASO relative to controls; and a medium effect size (ES) for improvement in sleep efficiency. Sleep improvements were also accompanied by small, but significant improvements in the energy/vitality and mental health subscales of the SF-36, relative to the TAU group. Importantly, though, these follow-up improvements are still substantially lower than normative reference values, which again is likely to be mediated by the ‘real world’ clinical context.

Walsh and colleagues (2007), in their large RCT of Eszopiclone versus placebo, assessed HRQoL and daytime functioning as primary outcome measures. Changes in sleep parameters were significantly larger in the treatment group for SOL, WASO and TST (medium to large ES) relative to the placebo group at the 6 month follow-up, and 50% scored ≤ 7 on the Insomnia Severity Index (ISI; compared with 19% in the placebo group). In terms of HRQoL, SF-36 domains of vitality, social functioning, physical functioning, and bodily pain were all significantly improved relative to the placebo group at six months (small to moderate ES for the vitality domain, and small ES for the rest of the domains). Although the authors report ‘no change’ in terms of sleep variables during the discontinuation period (i.e. sleep gains were maintained), they do not report data on HRQoL parameters. This is likely to be because of the short 2-week
interval, coupled with the SF-36 recall period; nevertheless, it would be interesting to see if these improvements are maintained beyond ‘active’ treatment.

In the original study by Sivertsen et al. (2006a), the authors showed that PI patients treated with CBT improved in both objective and subjective sleep parameters, relative to a zopiclone treatment group, and that these relative improvements remained robust at 6-month follow-up. In a recent follow-up report on daytime functioning measures from this trial, Omvik and colleagues (2008) failed to find any evidence of impairment (at baseline) relative to normative values, using the SF-36 and the QoLI. Not surprisingly, then, they also failed to find a significant group interaction effect, with neither treatment improving mental or physical health component scores (a possible ceiling effect), at post-treatment or follow-up. Again, however, they did not report dimension values, and so subtle component effects may be masked. One interesting finding, when groups were collapsed, was a significant association between pre-post increases in QoLI scores and increases in slow-wave sleep. This does point to a potential relationship between objective sleep and relevant quality of life variables.

Another study (Scharf et al., 2005) used a generic QoL measure, the Quality of Life enjoyment and satisfaction questionnaire (Q-LES-Q; Endicott et al., 1993), comprising 16 separate dimensions (both health and non-health). Scharf and co-workers reported significant improvements in five domains (physical health, mood, household activities, leisure time activities, and medication) in a group of elderly PIs treated with Eszopiclone 2mg, relative to a placebo group. These were accompanied by improvements in SOL, WASO and TST compared with placebo. Final assessment was,
however, short at two-weeks, and the authors do not present baseline or post-treatment means, preventing comparison with norms or other clinical groups.

Morin et al. (2005) conducted a randomized-placebo controlled trial of valerian-hops and diphenhydramine, on a group of ‘mild’ insomniacs (n=184; experiencing difficulties in initiating and/or maintaining sleep for between 2 and 4 times per week, in the last month). Relatively mild hypnotic effects were found with both treatments, particularly at the two week assessment. In the comparison between valerian and placebo at four weeks, the valerian group showed a small but significant relative improvement on the physical component summary of the SF-36. Dimensions scores were not presented and the mental health component summary failed to reveal any improvements. The limited and short-lived improvements in sleep may account for the marginal improvements in HRQoL, coupled with the fact that patients were not required to self-report daytime dysfunction on study entry, perhaps creating a ceiling effect, and subsequently making it harder to detect noticeable changes in functioning post-intervention.

In line with contemporary conceptualizations of insomnia as a potentially ‘co-occurring’ phenomenon, and not merely a secondary ‘nuisance’ symptom (Stepanski & Rybarczyk, 2006; Lichstein, 2006), recent studies have examined the impact of treating insomnia within the context of other disorders on aspects of HRQoL. For example, Espie and colleagues (2008) recently conducted a pragmatic RCT of CBT versus treatment as usual (TAU) in cancer patients (n=150), who had completed active cancer treatment but also reported significant insomnia. At 6 months follow-up, the CBT group evidenced significant improvements in measures of SOL, WASO, and SE, with corresponding
large effects, relative to the TAU group. The authors also included a measure of cancer-related Quality of Life, the Functional Assessment of Cancer Therapy-General (FACT-G; Cella et al., 1993), assessing physical, emotional, social and functional domains of the cancer experience. At 6 months the CBT group demonstrated significant improvements in both the physical and functional domains (large effect sizes). The potential limitation of this scale, in the context of insomnia and HRQoL, is the inclusion of sleep items, which interestingly feature only in the subscales that showed statistical improvements. The authors also note that changes in SE and changes in statistically significant HRQoL domains were low. Results from a similar study (Savard et al., 2005), adopting an efficacy approach, comparing CBT with a wait-list control, also demonstrated strong improvements in sleep parameters. The authors included the ‘global health/quality of life’ sub-scale from the EORTC QLQ-C30, which revealed significant and enduring improvements in treated patients from baseline to 12 month follow-up. Other sub-scales of this measure were not reported (including social, emotional, physical and cognitive functioning).

2.7. Reflections on existing insomnia-HRQoL treatment literature

From the limited treatment studies it is clear that improving sleep, in some cases, can lead to statistical improvements in aspects of HRQoL. However, what is far from clear is whether these improvements are clinically meaningful: do they really matter to the patient? For the most part, improvements are small and/or fall short of normative values, though this may be dependent on the particular population under investigation. Follow-up assessments have typically been short, with most occurring about 6 months post-treatment - it is possible that improvements in functioning become apparent well after sleep parameters have stabilized, particularly with CBT. The converse may also
occur in those treated with hypnotics: initial improvements may not be sustained after discontinuation. Relationships between sleep parameter changes and HRQoL improvements are rarely assessed (or reported), and no study has yet documented adequate improvement in HRQoL in primary insomnia patients, using Cognitive Behavioural techniques.

Moreover, to our knowledge, no comparative controlled study of CBT versus hypnotics exists that demonstrates superior HRQoL outcomes in favour of a particular treatment modality (only one relevant RCT [Sivertsen et al., 2006a; Omvik et al., 2008] has included a HRQoL measure). Comparative treatment studies (and intervention studies more generally), probing changes in HRQoL, are important because of their implications for cost-effectiveness and utility models. HRQoL scores are typically used in the calculation of Quality Adjusted Life Years (QALYs) – a weighted product of life expectancy and quality of remaining life years – which can then be combined with intervention costs to produce a cost-utility ratio. This cost/QALY ratio (i.e. the difference between the costs of two interventions divided by the difference in QALYs gained) then helps establish costs per QALY for a particular insomnia intervention, relative to, say, non-treatment, competing insomnia treatments, and other interventions for other common medical problems (Martin et al., 2004). Inexpensive interventions (i.e. low cost per QALY) are subsequently prioritized in terms of resource allocation, over more expensive ones. With the contemporary focus on the burden of insomnia it is clear that future intervention research must include, and pay attention to, the HRQoL concept, in order to document associated morbidity as well as utility of treatment (Morgan et al., 2004; Morin, 2004).
However, as a field we also need to strive for a more sophisticated understanding and conceptualization of QoL and HRQoL, as it relates to insomnia. From the studies reviewed, only a select few actually defined what they were attempting to measure (and these were typically cross-sectional or scale development/validation studies). Moreover, many refer to assessing or improving ‘quality of life’ when they were actually measuring aspects of HRQoL, using a generic health status measure. It may well be true that treating insomnia does improve ‘quality of life’, but where specialised measures such as the SF-36, NHP, SIP, or even the HD-16 are used, it may be more appropriate to emphasise the measurement of health-related QoL.

The key strength of generic measures, like the SF-36, is the ability to compare scores across disease states and with normative community reference values, in a standardized manner. Such data, of course, are vital for cost-effectiveness analyses when, for example, comparing treatments, and assessing whether patients return to ‘normal’ status. The sacrifice, however, is the poor specificity to a particular disorder. The inclusion of non-specific domains may dampen the sensitivity of a measure to detect change (Hill et al., 1996); this is especially true when only summary scores are reported. Future studies should report both overall summary scores and dimension scores, particularly in light of literature questioning the independence of the mental and physical health components and their representation of individual profile scores (e.g. Taft et al., 2001). Indeed, for these reasons it is recommended that intervention studies include both disease and generic HRQoL measurement – with the former typically being more sensitive to change than the latter (Guyatt, 1997). This approach has achieved some success in the sleep apnea field (Reimer & Flemons, 2003); and is the
recommended perspective by recent insomnia expert consensus workgroups (Buysse et al., 2006).

Currently, there is no widely used insomnia-specific measure; attempts to pilot and develop new instruments, and investigate the validity/sensitivity of the recently published HD-16, should be considered an important research goal. In the absence of specific measures, researchers need to consider using validated scales that tap dimensions relevant to the insomnia experience. Qualitative work from the Pittsburgh group (Carey et al., 2005) and more recently our own group (Kyle et al., in press; also chapter three of this thesis), reveal the nature of insomnia-related functional impairments - specifically in aspects of cognition, occupational functioning, social/interpersonal relationships, and limitations in goal attainment. It is interesting that the SF-36 does not adequately assess any of these dimensions; even the social functioning scale of the SF-36 simply probes the extent and frequency of interference without detailing what that interference may be. In the case of insomnia this interference is likely to be multi-factorial – from rescheduling activities, failing to schedule or commit in the first place, cancelling at the last minute, or not enjoying social interaction when present.

Another related issue, again concerning the SF-36, is the construct validity of the energy/vitality domain. This dimension resonates best among sleep-disordered patients, particularly individuals with insomnia, capturing fatigue-related symptoms common to sleep loss/fragmentation. This however is the problem; the dimension may simply be measuring fatigue. Scale development and evaluation studies typically use the vitality sub-scale as a check for concurrent validity, revealing high correlations with
instruments such as the fatigue severity scale (e.g. Kleinman et al., 2000). The danger of course being, that studies (cross-sectional and treatment) may confuse assessing or improving HRQoL impairment, with modifications in levels of fatigue. This may prove problematic when, for example, collapsing scores into a physical component summary, subsequently leading the author to conclude that ‘x treatment improves HRQoL’. A solution to this issue is to always document all measured dimensions, and include, and correlate, validated measures of fatigue with the vitality dimension, to see if there is a substantial overlap in variance.

A final note on generic measurement concerns the explicit focus on ‘health’ and ‘illness’ state terminology. The SF-36 probes the limiting impact of ‘health’ on aspects of functioning; it is, after all, a health status measure. It is not clear if those with insomnia, particularly primary insomnia, actually consider poor/disturbed sleep to represent a change or variation in health state (it is not uncommon for an individual with PI to proclaim, when searching for an underlying causal factor for why they cannot sleep, that they are ‘healthy’ and otherwise have a good life). In comparison with other disorders, that perhaps have a more direct health-link, insomnia impairment may be more difficult to document reliably, and thus show improvements post-intervention, using generic health status tools. This may explain HRQoL discrepancies between clinical, pragmatic studies, and those where PIs are recruited through media adverts.

**2.8. Beyond generic measurement: future directions**

Many questions remain unanswered about the nature of HRQoL in insomnia. The more basic ones, such as what are the predictors/mechanisms of HRQoL impairment and subsequent improvement will become apparent as researchers increasingly investigate
insomnia as a 24-hour disorder. Some possible targets include: sleep parameters; daytime symptoms like fatigue, mood, and neurocognitive impairment; dysfunctional beliefs; cognitive biases; and objective markers of the stress system. Longitudinal studies on the natural evolution of insomnia, and the transition between states (i.e. good sleepers >>> acute insomnia >>> chronic insomnia), will also prove helpful in better defining causal relations between insomnia and HRQoL.

In relation to assessment and measurement, the scope of this review, a prospective research agenda should be initiated on adapting and creating new instruments that adequately probe the insomnia experience. One potential avenue, in addition to generic measurement, is to adopt a supplemental modular approach (Aaronson, 1989) to functional HRQoL assessment. That is, to consider important domains that are commonly reported to be affected by individuals with insomnia, but are not necessarily dealt with comprehensively by generic instruments. For example, occupational functioning is cited as a potential daytime consequence in the diagnostic criteria, yet little work has actually focused specifically on this domain. Encouragingly, David & Morgan (2006) recently created and validated the Occupational Impact of Sleep Questionnaire (OISQ), a scale that probes the impact of sleep quality on a number of work-related areas. Initial data collected on the OISQ shows it has good discriminant ability to distinguish between PIs and good sleepers, and, more recently, significant correlations were found with global sleep quality (as measured by the PSQI) and sleepiness (ESS) in a Dutch sample of office workers (Verster et al., 2008).

In this context, two recent pharmacotherapy studies are also worth mentioning (Walsh et al., 2007; Erman et al., 2008). Walsh and colleagues assessed work limitations, using
the work limitations questionnaire (WLQ; Lerner et al., 2001), in an RCT of Eszopiclone versus placebo. Baseline data showed lower scores on all domains of the instrument (time demands, physical demands, mental-interpersonal demands, output demands, and work productivity loss) relative to normative values, and somewhat comparable scores to those suffering from clinical depression. Importantly, all domains showed a relative improvement in the treatment group versus placebo, across the six-month treatment phase. Erman et al. similarly assessed workplace functioning in PI patients \(n=752\) taking part in a randomized controlled trial of Zolpidem versus placebo. The authors analyzed data from two components of the WLQ, the ‘time’ and ‘output’ sub-scales. Again, baseline values were more impaired (approximately three times greater) than normative healthy controls. Significant effects of treatment emerged after just 12 weeks, which were sustained at the 6-month follow-up, and improvements were related to global sleep changes. These data are important because they not only highlight the burden of insomnia on the workplace beyond traditional measures of absenteeism - confirming qualitative reports (Kyle et al., 2008) and population-based studies (e.g. Daley et al., 2009b) - but also demonstrate the ability to achieve simultaneous improvements in sleep and occupational functioning. Cognitive behavioural interventions should include a similar assessment of occupational functioning in future treatment trials.

As well as the development and use of relevant instruments, researchers should also consider the merit of applying new methods and approaches to assessing insomnia-related impairment. For example, the use of ecological momentary assessment (Buysse et al., 2007; Levitt et al., 2004) to collect daily diary ratings of common insomnia symptoms might provide a new way to track daytime outcomes during treatment, and
post-treatment, as well as establishing relationships with nightly sleep parameters. There is no reason why this method cannot be adapted to assess, for example, social functioning, or subjective workplace performance. A ‘significant other’ perspective (qualitative and/or quantitative) would also be a useful source of information to gauge how sleep, and appropriate treatment, affects domains of HRQoL. To our knowledge no study has used this resource to probe daytime functioning and insomnia specifically. This also raises the issue of relationship functioning and insomnia. It has been reported that individuals with insomnia have less satisfying interpersonal relationships (Roth & Ancoli-Israel, 1999), and although it is unlikely to follow a simple cause-and-effect model (for a review, see Troxel et al., 2007), sleep quality may exert a mediating effect. It would be interesting to see if treating insomnia can also improve marital/relationship quality; certainly, improvements in marital satisfaction have been reported in sleep apnea patients after initiation of CPAP therapy (McFadyen et al., 2001).

Global quality of life has been viewed pragmatically in the literature as a multi-dimensional construct, assessed using commonly reported domains deemed important. The likelihood, however, is that QoL means different things for different people - a uniquely personal perception (Gill & Feinstein, 1994) - making it difficult to accurately measure at the group level, and especially through modular assessment. One rather simple, but valid method might be to ask individuals to rate their quality of life on a Likert item or visual analogue scale. It could be argued that on making this judgment, individuals are factoring in all the domains/life areas that are important to them. This single-item format has proved sensitive when documenting, for example, how many people within a sample of individuals with insomnia report a ‘very good’, ‘good’, or ‘poor’ quality of life, compared to those without sleep-complaints (Roth & Ancoli-
A similar approach could be tested alongside generic and modular assessment in treatment studies; single item visual-analogue scales have been shown to be valid, reliable and responsive to change in patient groups (e.g. DeBoer et al., 2004).

A related methodological approach would be to tailor items asking patients to report whether they think their QoL has *improved* as a result of treatment; instead of simply trying to demonstrate *reductions in functional impairments*, as generic measures typically do. The Patient Global Impression improvement scale, developed from the Clinical Global Impressions scale (Guy et al., 1976), is used for a similar purpose, allowing patients to report the extent to which their overall illness symptoms have improved post-treatment. Such an instrument could be modified to assess perceived improvements in focused areas of functioning as well as subjective perceptions of global quality of life.

It may also be worthwhile directing efforts towards gathering basic qualitative data on the individual experience of treatment. We (Kyle et al., in press) and others (Carey et al., 2005; Fox et al., 2007) have successfully applied qualitative methods to understanding the daytime insomnia experience, revealing novel insights on the nature of functional impairments. Similar methodologies (semi-structured interviews, audio-diaries) could be applied to assessing treatment outcomes, as well as the treatment process itself. The SAQLI (Flemons & Reimer, 1998) includes a section looking at negative side-effects of CPAP treatment in OSA patients; it is not yet clear what, if any, impairments in HRQoL are incurred *during* CBT for insomnia (stimulus control and sleep restriction would be likely candidates for study). While clinicians may be familiar
with how sleep improvements influence patient well-being, systematic study of the
patient narrative may prove helpful in understanding the treatment process (side effects,
locus of improvement, mechanisms of change, adherence), and the net impact of
treatment on daytime functioning and subjective quality of life (at different time-
points).

The importance of measuring items that are relevant, not just to the disorder, but to the
individual has been noted in the QoL and HRQoL literature (Gill & Feinstein, 1994;
Carr & Higginson, 2001; Bilsbury & Richman, 2002), and, more recently, by sleep
researchers (Reimer & Flemons, 2003; Omvik et al., 2008). We are currently piloting a
mixed method, patient-centred approach (Ruta et al., 1994; Kyle et al., 2009) to the
assessment of insomnia-related quality of life (IRQoL) at the Glasgow Sleep Centre.
By IRQoL we mean ‘the impact of insomnia on aspects of life that are most salient to
the individual, as reported by the individual’. Specifically, patients are asked to
generate, using their own words, the most important areas of their life that are affected
by sleep disturbance. These areas are then ranked in terms of importance, and rated in
relation to extent of interference over a defined time interval. Items can be assessed pre
and post-treatment, within- and between-subjects. This idiographic approach permits
assessment of areas that are important to the individual, which is likely to be a lot more
sensitive to change given that we are enhancing the signal (relevance) and reducing the
noise (non-relevance) associated with generic instruments. Moreover, it has been
commented previously (Buysse et al., 2006; Moul et al., 2004; Reimer & Flemons,
2003) that the major obstacle to creating an insomnia-specific QoL questionnaire is the
high degree of co-morbidity. This is certainly the case when conducting pragmatic
effectiveness trials, or in clinical settings, where other illnesses may mediate scores on
HRQoL instruments. The strength of our individualized approach is that it emphasizes the attribution to sleep, and, therefore, it should be theoretically possible to isolate the subjective impact of an intervention on sleep only, even in the context of other illnesses.

2.9. Conclusion

It is clear that insomnia does have a measurable negative impact on domains of HRQoL, and that these impairments are not simply limited to obvious domains, like vitality and energy, but also extend to other aspects of mental, social, and physical functioning. Comparisons with other illnesses, linear trends with insomnia severity, and additive effects of insomnia beyond a primary/co-occurring illness, all support and strengthen this perspective. Although research into HRQoL is in its infancy within the insomnia literature, there are already emerging data that successful treatment can improve functioning across a number of domains. Of course, to what extent group mean improvements are important to individual patients, and their daily lives, remains an unanswered question. Future research in this area should attempt to approach the issue of how insomnia treatment improves functioning and individual quality of life, by using new innovative methods and instruments. Such a task is not a trivial undertaking; the impact of insomnia on the individual (Kyle et al., in press) and society (Daley et al., 2009a) is very real, and authors have suggested that the pathway between insomnia and depression, for example, may be mediated, in part, through reduced HRQoL (e.g. Taylor, 2008).

In order to improve the quality of future research, outcome studies should include a generic measure of HRQoL as well as other measures that are relevant to the insomnia experience, and have proven sensitivity. When reporting results, authors should specify
baseline scores and their relationship with normative values, detail each profile score as well as component/global summary scores, and make some attempt at investigating the relationship between sleep improvements and HRQoL changes (i.e. correlation analysis and/or responders versus non-responders, based on ISI category change, or quantitative sleep variables).

2.9.1. Practice points

- HRQoL and QoL are not synonymous constructs.
- Insomnia negatively affects diverse aspects of HRQoL
- The limited treatment data tentatively suggest that successfully improving sleep, using both pharmacological and non-pharmacological interventions, can lead to significant improvements in domains of HRQoL.
- There is a great need to standardize measurement use and reporting of scores to facilitate future comparisons and meta-analyses across studies and treatment modalities.
- HRQoL measurement contributes to cost-effectiveness models and therefore plays a pivotal role in service provision and development.

2.9.2. Research Agenda

- Include measures of HRQoL in future cross-sectional, natural evolutional and interventional studies.
- Compare different symptom (initiating, maintenance, mixed) and diagnostic (idiopathic, psychophysiological) sub-types of insomnia on measures of HRQoL.
- Compare different treatment modalities on HRQoL primary outcomes within the same study.
Consider assessing domains that are under-researched, yet part of the insomnia experience, such as social, occupational and relationship functioning.

Follow-up HRQoL outcomes for longer durations and consider using new informative methods that capture the patient perspective e.g. qualitative research methodologies.

Investigate relationships between sleep parameters (objective and subjective) and HRQoL dimensions.

Identify non-sleep predictor variables of HRQoL impairment and change.

Consider the effect of the insomnia treatment process on HRQoL.

Measure the impact of treating insomnia in the context of co-morbid illness (e.g. cancer, depression, pain), on HRQoL.

Develop and validate measures of insomnia-related quality of life that are grounded in the words of patients.

2.9.3. Going forward

This review clearly indicates that many fundamental research questions are yet to be addressed surrounding the nature, measurement, and modification of HRQoL and functional parameters. The experimental chapters of this thesis will now explore several important related themes from the aforementioned research agenda, specifically those with a focus on understanding the daytime experience and associated impairments of chronically disturbed sleep; development of novel measures to capture functional and quality-of-life impairment; and the integration of mixed methodologies to assess the patient experience of behavioural treatment for insomnia.
Chapter 3:

Daytime Phenomenology in Primary Insomnia

The work presented in this chapter has been accepted for publication as follows:

Kyle, S.D., Espie, C.A. & Morgan, K. ‘...Not just a minor thing, it is something major, which stops you from functioning daily’: Quality of Life and Daytime Functioning in Insomnia. *Behavioral Sleep Medicine*, in press.
3.1. Abstract
According to diagnostic manuals, insomnia is a 24-hour disorder, impairing important aspects of daytime functioning. There is, however, little published work describing the impact of insomnia on important areas of functioning, or indeed the experience of living with chronically disturbed sleep on a daily basis. We recruited 11 volunteers with persistent insomnia to take part in one of three focus group discussions, exploring the typical daytime consequences of poor sleep and impact on quality of life (QoL). A sub-sample (n=8) were also asked to keep an audio-diary for seven days – appraising sleep quality and subsequent daytime functioning. Interpretative Phenomenological Analysis (IPA) of transcripts produced three superordinate themes: ‘just struggle through’, ‘isolated, feeling like an outsider’, and ‘insomnia as an obstruction to the desired self’. Participants described daily difficulties with cognitive, emotional and physical functioning; this had the cumulative effect of reducing work performance and social participation, as well as limiting life aspirations. Participants also described feeling isolated because of their disorder; this was precipitated by a lack of understanding from others, and experiences with health care providers. Important novel data were generated on the proximal and distal impact of insomnia, indicating that chronically disturbed sleep can seriously limit overall QoL.
3.2. Introduction

Insomnia disorder is estimated to affect 10% of the adult population (Morin et al., 2006a) and is characterized by difficulties with initiating AND/OR maintaining sleep, or non-restorative sleep (DSM-IV-TR, 2000). Importantly, the major diagnostic nosologies (ICSD-2, 2005; DSM-IV-TR, 2000) acknowledge that insomnia is a 24-hour disorder, specifying, to warrant ‘disorder’ status, that sleep disturbance must result in some form of daytime dysfunction or distress e.g. in social, occupational, or other important areas of functioning. This is especially important considering that daytime distress is often the catalyst leading sufferers to seek treatment; not merely disturbed sleep during the night (e.g. Morin et al., 2006a).

Several large survey studies demonstrate that individuals with insomnia report deficits in numerous domains. For example, Roth and Ancoli-Israel (1999), in their analysis of the 1991 National Sleep Foundation telephone survey (n=1000), found that individuals with insomnia report greater fatigue and mood disturbance, as well as impairments in cognitive abilities, physical well-being, and the ability to accomplish certain daily tasks, relative to normal sleepers. Moreover, level of impairment was related to insomnia severity: with the ‘chronic insomnia group’ exhibiting greater impairment than the ‘occasional insomnia group’ (and both showing greater decrements than their normal sleeping counterparts). Reports in clinical samples (e.g. Moul et al., 2002) and well-screened individuals with primary insomnia (PI), with no other co-morbid pathology, corroborate this pattern of daytime dysfunction (e.g. Buysse et al., 2007).

In recent years, there has been a shift towards assessing the overall impact of insomnia on QoL, through the measurement of health-related quality of life (HRQoL). HRQoL
can be conceptualized as the distal, downstream effects of illness – “...the radiating impact of pathology on the patient’s wider world” (Armstrong et al., 2007, p578); in essence, the limitations imposed on a person through disease or illness. HRQoL instruments have thus tended to cover a broad range of quality of life domains e.g. physical, mental (emotional) and social functioning (Bowling, 2005).

Early studies assessing HRQoL in insomnia populations, using the generic Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992) revealed impairments across all domains relative to normal sleepers (e.g. Zammit et al., 1999; Hatoum et al., 1998). More recent studies controlling for anxiety and depression (Leger et al., 2001), and various medical co-morbidities (Katz & McHorney, 2002; Philip, et al. 2006), confirm that poor sleep can independently impair HRQol, and that severity of sleep disturbance is linked to magnitude of impairment (e.g. Leger et al., 2001). Final evidence that poor sleep impairs HRQoL comes from treatment studies (pharmacological and non-pharmacological) demonstrating improvements in a number of HRQoL domains post-intervention, in parallel with improvements in sleep (e.g. Walsh et al., 2007; Espie et al., 2007).

Generic HRQoL measures [such as the SF-36 and Nottingham Health Profile (NHP; Hunt et al., 1981)] are useful for several reasons: they permit cross-disease comparisons, comparisons with normative community values, assessment of treatment outcome, and a measure of cost-effectiveness for a particular intervention. However, in the context of insomnia, such measures are not without limitations: they are not developed specifically for use with insomnia populations, and so may not fully reflect impairment typical of insomnia; and largely fail to capture the uniqueness of the
individual, being “…applied deductively, from the construct to the individual, rather than inductively - from individual to construct” (Bilsbury & Richman, 2002, p.8). As a direct result, standardized generic HRQoL measures may miss salient, important issues for an individual, while at the same time include redundant irrelevant items. Therefore, reliance on existing questionnaires as a gauge of how insomnia impacts on an individual’s daytime functioning and overall quality of life, may underestimate (1) the breadth of areas that are impaired by poor sleep; and (2) the extent to which these areas limit one’s enjoyment of life i.e. the relative importance of a domain for a particular individual.

Two recent insomnia expert panels (NIH state-of-the-science conference statement, 2005; and the Pittsburgh consensus work groups, Buysse et al., 2006) called for greater attention and research into the consequences and correlates of insomnia, with special emphasis on waking function. Given the limited nature of existing measurement (using non-specific pre-selected domains) it is possible that insomnia has implications for factors that are not so obviously “health-related”, like family relationships, career aspirations, performance/productivity at work, or even just general life expectations (Reimer & Flemons, 2003). As a result such factors may have escaped thorough investigation. Similarly, although diagnostic criteria for insomnia specify ‘impairments in social, occupational, or other important areas of functioning’ (DSM-IV-TR), little is known about the constituent components which underlie these super-ordinate impairments, with the majority of published work merely asking an individual to rate the extent to which a global area of functioning is affected. Therefore, the existing literature has largely neglected nature of impairment. Understanding the daytime experience of insomnia and how it impacts individual QoL is crucial to fully
characterizing insomnia disorder, and its societal impact, as well as forming targets for
treatment outcome.

The present study sought to characterize, using qualitative methodologies, the daytime
experience of insomnia. Qualitative methods were chosen because they allow
individuals to describe, in their own words, the experience of illness, “reaching the
parts other methods cannot reach” (Pope & Mays, 1995). To date there has been little
published work on illness experience, from the patient perspective – “we’re more like
objects than subjects, really: we have no speaking part in this literature, no voice”
(Greene, 2008, p17). Indeed, to our knowledge there are only two qualitative studies,
using focus groups, which explored experiences of insomnia (Carey et al., 2005; Green
et al., 2008). The study by Carey and colleagues revealed some interesting data on the
daytime aspects of insomnia disorder; however, half the sample also had a diagnosis of
insomnia and depression, and the purpose was primarily to create items for a new self-
report metric (content analysis), rather than explore the full impact of insomnia on
domains of functioning and QoL. Similarly, Green et al. (2008) conducted focus group
discussions in a very small sample of individuals with insomnia (n=6). Again, the
primary aim was to generate items to help develop a questionnaire, with emphasis on
insomnia management; little information was provided concerning sample
characteristics in terms of diagnostic criteria and co-morbidities, and there were
inconsistencies in focus group methodology between the two conducted groups. In
sum, there is a dearth of ‘bottom-up’ research aimed specifically at investigating
daytime experiences in those with primary insomnia.
In the present study, within an Interpretative Phenomenological Analysis (IPA) framework, we utilized focus groups to explore, in detail, the daytime experience of insomnia, and how sufferers believe poor sleep impacts upon QoL. To further gain an “insider’s perspective” (Conrad, 1987) on the relationship between sleep quality and daytime functioning, we asked a subset of our focus group participants to complete a prospective one-week audio-diary.
3.3. Method

3.3.1 Participants

We recruited 11 volunteers (9 female, 2 male: mean age = 38; range 20-64), meeting DSM-IV-TR (American Psychiatric Association, 2000), and Research Diagnostic Criteria (RDC; Edinger, et al., 2004) for primary insomnia. Recruitment was achieved through newspaper adverts, posters in Dental/GP surgeries and public libraries, and by contacting GP-referred patients on the Glasgow Sleep Centre waiting list.

Inclusion criteria included the following:

- Either difficulties initiating AND/OR maintaining sleep, or non-restorative sleep, on at least three nights per week for the last month.

- Sleep disturbance results in some form of daytime impairment, indicated by the presence of at least one associated daytime symptom, according to research diagnostic criteria (Edinger et al., 2004).

- A score of $\geq 15$ (clinical insomnia, moderate severity) on the Insomnia Severity Index (ISI; Morin, 1993) and a score $> 5$ (maximum sensitivity and specificity for insomnia; Backhaus et al., 2002) on the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

- Sleep disturbance is not due to substance abuse, another mental or sleep disorder, or a general medical condition (assessed by structured interview, based on Morin & Espie, 2003; see Appendix A).

Subjects were excluded if they did not satisfy any of the above criteria, if they were currently receiving psychological treatment for their sleep problem, or taking sleep medication more than 3 nights per week.
3.3.2. Procedure

Approval to conduct the study was obtained from the local NHS ethics committee. Interested respondents to advertisements/posters, scoring within the desired ranges on the screening questionnaires, were invited to meet with the researcher at the Sleep research laboratory to complete a short screening interview. After screening, those meeting inclusion/exclusion criteria were invited to attend the focus group discussion and subsequent audio-diary component.

3.3.3. Focus Groups

Three focus group discussions (n=11; two groups contained 4 participants, and one group, 3) were facilitated by the researcher, according to recommended guidelines (Kitzinger, 1995). Groups took place in a meeting room at the University of Glasgow Sleep Centre, during the evening. On arrival, participants were instructed on the ground rules: aim is to encourage discussion; answers are neither right nor wrong; one person to speak at a time, if possible; that the group is being recorded; and everything that is said within the group is confidential. Discussions were recorded using an Olympus® digital dictaphone (Model: DS-30), and the facilitator made informal notes on emergent themes. The group was facilitated with minimal direction, allowing the group to explore issues they deemed important. Central topics were addressed using open-ended, fixed questions (see Appendix B) across the three groups, tapping into how insomnia affects QoL variables, as well as the daily relationship between sleep and next day functioning. Supplementary questions were used to probe areas of interest, and were modified in light of each transcription to inform subsequent groups. Group discussions lasted between 1 – 1 ½ hours.
3.3.4. Audio-diary reports

Eight participants from the eleven focus group attendees were selected, based on random allotment, to take part in the audio-diary component of the study. Prospective audio-diaries have recently been successfully pilot ed as a tool to investigate the ‘social context’ of normal sleep in both middle-aged women and couples (e.g. Hislop et al., 2005). They have the strength of capturing rich qualitative data in the context of participants’ own environment, are less prone to recall biases than traditional retrospective measures, and provide insights into respondents’ thoughts and experiences, proximal to the event. This is the first application within a sleep-disordered population.

Participants were briefed (see Appendix C) on how to operate the hand held digital dictaphone (Olympus© WS-200s) and semi-structured guidelines for diary entries. On awakening (within approx 15-20 mins.) participants were asked to describe their experience of the sleep period. In the evening, approximately two hours before going to bed (to avoid creating unnecessary pre-sleep anxiety), they were requested to describe relevant feelings and experiences during the course of the day and their relationship (if any) with sleep quality. Guidelines were purposely left open (see Appendix D), allowing participants to report information they deemed interesting and relevant.

3.3.5. Framework for qualitative analysis

Interpretative phenomenological analysis (IPA: Smith, 1996) formed the framework for both design and analytical approach. IPA is concerned with understanding lived experiences, and, in particular, the value and meaning participants assign to such experiences (Reid et al., 2005). IPAs exploratory nature, coupled with recruitment of
small, homogenous samples (‘experts’ in the phenomenon) renders it a useful method for investigating the impact of illness/disease on patient groups. It is also interpretative in the sense that it acknowledges the role of the researcher’s own ideas, conceptions and understandings in making sense of participants’ subjective accounts. That is, the researcher attempts to make sense of the participant’s interpretation of their own world, rather than trying to make objective statements about experiences or a specific population (Smith & Osborn, 2003).

Analysis was conducted according to methods described by Smith & Osborn (2003). Focus groups and audio-diaries were transcribed verbatim, including pauses, laughter and false starts. Transcription generated ~38,600 words for focus group discussions, and ~17,600 words for audio-diary entries. Transcripts were analyzed individually by first noting significant words or phrases in the margins. On a second review of the transcripts, initial notes were transformed into emergent themes. Themes were then compared across focus groups and individual audio-diaries, looking for common recurrent themes as well as deviant/contradicting cases. This resulted in the generation of superordinate themes and corresponding sub-themes. A sleep psychologist, with special expertise in daytime aspects of insomnia and IPA methodology, checked themes and supporting quotes for transparency (a credible account as opposed to a true account); any discrepancies were reviewed and discussed.
3.4. Results

3.4.1. Participant characteristics

Mean scores (SD) for the 11 participants on the ISI and PSQI were 19.4 (2.8) and 13.4 (2.9), respectively (see table 3.1). Nine of the participants had difficulties with both initiating and maintaining sleep, and the remaining two had difficulties with maintaining sleep only. The audio-diary sub-group (n=8) was composed of 6 females and 2 males (mean age = 36; range 20-54), scoring 19.8 (3.2) on the ISI and 12.8 (3.4) on the PSQI.

Table 3.1 - Participant demographics for those participating in focus groups and sub-set completing audio-diaries.

<table>
<thead>
<tr>
<th></th>
<th>Focus Groups (n=11)</th>
<th>Audio-Diaries (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>38 (20-64)</td>
<td>36 (20-54)</td>
</tr>
<tr>
<td>Gender</td>
<td>9 Female/ 2 Male</td>
<td>6 Female / 2 Male</td>
</tr>
<tr>
<td>ISI</td>
<td>19.4 (2.8)</td>
<td>19.8 (3.2)</td>
</tr>
<tr>
<td>PSQI</td>
<td>13.4 (2.9)</td>
<td>12.8 (3.4)</td>
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</tbody>
</table>

3.4.2. Interpretative analysis

Interpretative analysis revealed three superordinate, but inter-related, themes: ‘just struggle through’, ‘isolated, feeling like an outsider’, and ‘insomnia as an obstruction to the desired self’. Within each main theme, several more specific sub-themes were identified (see table 3.2).

Direct participant quotes are presented to support themes along with focus group (FG) and audio-diary (AD) identifiers. Pseudonyms are used to protect the identity of participants.
### Table 3.2 - Superordinate and sub-themes from IPA.

<table>
<thead>
<tr>
<th>Superordinate themes</th>
<th>Sub-themes</th>
</tr>
</thead>
<tbody>
<tr>
<td>‘Just struggle through’</td>
<td>Cognitive</td>
</tr>
<tr>
<td></td>
<td>Emotional/Psychological</td>
</tr>
<tr>
<td></td>
<td>Physical</td>
</tr>
<tr>
<td>‘Isolated, feeling like an outsider’</td>
<td>Lack of understanding</td>
</tr>
<tr>
<td></td>
<td>Labelling &amp; Scepticism</td>
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<tr>
<td></td>
<td>Coping strategies</td>
</tr>
<tr>
<td>‘Insomnia as an obstruction to the desired self’</td>
<td>Vocational functioning</td>
</tr>
<tr>
<td></td>
<td>Social impact/exclusion</td>
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<td></td>
<td>Good sleep/remove insomnia</td>
</tr>
</tbody>
</table>

3.4.2.1. ‘Just struggle through’

The focus groups captured the most salient daytime consequences of disturbed sleep, and these were reinforced, prospectively, by the audio-diaries. At a general level, participants reported impairments in several areas of functioning; this created the feeling of “only running at half level or below” (Susan, FG 2), and as a consequence participants commonly viewed the day “as a struggle” or an “effort”.

**Cognitive**

Participants described how poor sleep has a detrimental impact on their cognitive abilities during the day, “you can’t think straight, you can’t think the same” (Alison, FG 1). This inability to ‘think the same’ reflected impairments in concentration, attention and memory:
“I need to read quite a lot at work, you find you’ve read a page and you have no clue what it said, you have to go back and re-read it” (Rachel, FG 1)

Lapses in attention and concentration were often related to being ‘clumsy’ or nearly being involved in car accidents (prompting one participant to seek treatment). The audio-diaries were able to capture instances of these concentration ‘lapses’:

“I’ve just decided to fill in that form that you gave me, and I’ve just realized that I put down my daughter’s date of birth instead of my own…that is because I’m so tired. I’m obviously not thinking straight” (Alison, AD)

**Emotional/psychological**

Participants talked about how poor sleep, especially several days of poor sleep, can impact upon mood and emotional well-being.

“I find my mood only kinda starts to be affected after a couple of nights of not having a great sleep...like I try [and] just be laid back as possible, but I do find, eventually, it starts to wear you down, and then it’s always my boyfriend that gets it...I start to get really snappy, and that’s not like my temperament or anything like that, that’s not me at all” (Susan, FG 2)

Susan’s assertion - ‘that’s not me at all’ - conveys, rather nicely, a conflict that participants battle with on a regular basis: the ‘me’ versus the ‘me after sleeping poorly’. Group members discussed how ‘irritable’, ‘moody’, and ‘agitated’ they became after a ‘bad’ night, yet maintained this is not what they are normally ‘like’.
This dissonance also extended to the social sphere, with participants expressing concerns about how others may perceive them during daily interactions. Not performing to others, as well as own personal expectations, cognitively and socially, seemed to be a source of worry and anxiety for group members:

“There’s the anxiety of how people are looking at me today...am I behaving alright? How am I relating to people today? It puts an extra load of worry, apart from the sleeplessness itself there’s another pattern of worry, you know, how you are carrying yourself” (Helen, FG 3)

“I’m feeling guilty because I’m not performing to the standard that I would like to think I would normally and it makes me feel lazy” (Kate, FG 2)

Audio-diaries also proved extremely useful for tracking modifications in mood and getting a real-time snapshot:

“I feel quite low tonight, quite weepy, and I’m sure it’s just because I’m absolutely knackered” (Rachel, AD)

Physical

The physical consequences of poor sleep were present from the moment of waking. This was reported as a “sick”, “drained” feeling, or overall “fuzziness”. There was a real sense that participants had been involved in a struggle or “some epic battle” (James, AD), throughout the course of the night, often feeling more “exhausted, or “knackered” than when retiring to bed:
“I’ve had about four hours sleep, I feel really really sick, like really nauseous, and my legs feel like jelly, I feel like I’ve got the flu actually, like I just, my legs just feel like I wouldn’t be able to walk on them” (Emma, AD)

Persistent fatigue throughout the day was conceptualized as feeling “sluggish” or “absolutely shattered”, described by one participant as being like “stuck in a bubble that you can’t quite get out off” (Kate, FG 2).

Some individuals also voiced concerns about the long-term impact of chronic insomnia on their physical health and well-being. This was often reported as problems with vision and bodily pain, but there was a general consensus of a ‘slump’ in health, reflected in a susceptibility to cold/viruses:

“You always feel as though you’re just on the edge of getting a cold” (James, FG 3)

Although separated for clarity here, emotional, physical and cognitive impairments typically interacted with each other, creating, as one lady described, a “weight on your shoulders” (Mary, FG 1). This ‘weight’ translated into deficits in several areas of functioning and overall quality of life:

“because it’s [insomnia] built up over a week or so many years or whatever, it kind of grinds you down, it does affect every single part of your day…and it’s not just a minor thing, it is something major which just, you know, it stops you from functioning daily” (Mary, FG 1)
3.4.2.2. ‘Isolated, feeling like an outsider’

There was a general feeling among group members that their sleeping problem made them feel different from others, this was captured in the theme ‘isolated, feeling like an outsider’.

**Lack of understanding**

This perceived isolation was, in part, driven by a lack of understanding and sympathy from others, including friends and family:

“I feel very isolated about, basically, that nobody can conceive what it’s like, no one understands what it’s like, because they once had a bad night’s sleep and so they ‘know what it’s like’ and they ‘just got over it’...so it’s something obviously lacking in me ” (James, FG 3)

The perception by others that those with insomnia should just ‘get over it’ not only infuriated participants but also made them feel somehow responsible for their own sleep difficulties; this appeared to further perpetuate their isolated position and engender feelings of self-blame and embarrassment:

“I feel embarrassed even to discuss about my sleeplessness, why I’m so tired, why I’m dull, why I’m not performing maybe to my friends expectations, you know, cos to the world it is a problem you can sort out” (Helen, FG 3)
Labelling & Scepticism

One group discussed their reluctance to speak to people, even friends, about their sleep pattern, because of anticipated labelling:

“SEAN: you don’t really tell people that you haven’t slept for more than one night
ELAINE: cos they think you’re a freak
KATE: or they think you’re a liar
SEAN: or if you say ‘oh I’ve got insomnia’, everyone thinks ‘och you’re just being a hypochondriac, stop being silly’...” (FG 2)

This ‘labelling’ theme also came through strongly when participants discussed encounters with the medical profession. It seemed that sleep not being taken seriously as a separate entity, a single problem in isolation, was a common experience. Many felt that a diagnostic label of depression was being thrust upon them, despite their protests to the contrary, “I’m not depressed, it [insomnia] just makes me depressed after a couple of nights” (Alison, FG 1):

“I don’t think I’ve really seen a doctor who’s believed that it’s a separate kind of entity, and it’s not linked to depression”(Laura, FG 3)

One man even felt that doctors were sceptical of him and his sleep problem, as if he was acting as a malingerer, failing to reveal other symptoms that might truly account for the disturbance:
“I thought that doctors thought that I wasn’t telling them the truth, as in like: ‘well if you’re not depressed, there must be something else, but if you’re not prepared to tell us that then just go away and you’ll come back eventually’” (James, FG 3)

The failure of the medical profession to identify the ‘real’ underlying cause of sleep disruption created a sense of hopelessness/helplessness among participants; continually striving to understand what may have precipitated their sleep disturbance.

“You see everyone else, how do they manage? They’ve got lives, no worries, I’ve no worries I’ve got nothing to worry about, I’ve got a good life.” (Alison, FG 1)

Despite desperate attempts to establish an underlying aetiology or precipitant, participants were unable to imagine how their sleep pattern could ever be improved, becoming almost resigned to the fact that it may never be successfully treated:

“… if you’re ill, you take a pill or something, if you’re depressed you can do things… or if you have all these other mental health issues, you know, you can get counselling or you can work with the issue, I can’t envisage what would happen…you know, for it to change…” (James, FG 3)

An exchange between three participants highlighted the ingrained nature of this ‘felt isolation’; while others may suffer from sleep problems, they’re in the ‘fortunate’ position of having a reason, something tangible that can explain the disturbance, and ‘legitimize’ their suffering:
RACHEL: “Women with small children, with babies, they’re good to talk to, cos they don’t sleep either...”

ALISON: “you feel they get sympathy because they’ve got a baby.”

MARY: “in a way that’s funny... they’ve got a reason that another person is causing the problem, whereas we don’t really have a problem... so it’s harder”

RACHEL: “and you know in another year they’ll be fine...they’ll be back to their two minutes and snoring on the pillow” (FG 1)

Importantly, the focus group forum was viewed favourably by the participants, nearly all of whom had never met someone with a similar problem:

“it’s just so hard sometimes, for not to have other people to talk to about it, cos you know, unless they are experiencing it themselves, they don’t really understand it...so just something like this is good, cos there’s other people you can reflect, you know, what you’re feeling” (Mary, FG 1)

Coping strategies

To avoid becoming overwhelmed by the consequences of their insomnia and isolated ‘status’, participants adopt a number of strategies to help them manage and cope during the day. The fear of labelling and stigma meant many would not use sleep as an excuse (e.g. for missing work or avoiding social contact); instead, they were forced into playing the role of an ‘actress’ - ‘put an act on, put a face on it’ (Alison, FG 1)

“this week I was at a meeting, and I was exceptionally tired, em, and I tend to over compensate so that people won’t know that I haven’t slept and that I’m tired, so I tend to talk too much, it’s probably a load of nonsense” (Laura, FG 3)
Others had to structure their day differently/change activities to try and mask, or at least attenuate, the impact of poor sleep:

“Everybody comes into the office about nine or just after nine, I find I’m in from 8 because if I come in at nine and then everybody else is really cheery and things like that, I’m just, it makes me really annoyed .. so if I’m in at eight, then at least by the time they all come in at nine, I’m maybe wakened up a bit, and then I’m a bit more social”

(Rachel, FG 1)

Such well developed “coping mechanisms” (James, FG 3) extended also to dealing with cognitive impairment (e.g. using memory aids, having set schedules) and staving off sleepiness (e.g. splashing water on face to increase alertness). There was a real sense that participants had to ‘regroup’ at several points during the course of a day; this reinforced the idea that life was often a “struggle”, and that a continuous battle was being fought – an approach described by one lady as “retreat, come back in and attack it again, different front” (Elaine, FG 2)

3.4.2.3. ‘Insomnia as an obstruction to the desired self’

The last major theme, ‘insomnia as an obstruction to the desired self’, captured how the specific consequences of disturbed sleep – that we all experience from time to time – can culminate in deficits in important areas of functioning, and restrict life goals, when experienced frequently.

Vocational functioning
In particular, participants felt that their ability to perform and fulfil work/university roles is adversely affected by their insomnia. In extreme cases this meant a reduction in hours, but more generally, participants reported impairments in work performance:

“my [work] performance has dropped big time, I’ve always been good at multi-tasking, and I feel the more I go without sleep, I just cope with less at the one time” (Elaine, FG 2)

Impairments in learning/retaining information were particularly noticeable for those in full-time education, and could be captured through audio-diary entries:

“…my last class was just really, I don’t know…I just can’t concentrate on anything, I’m just so tired, and just want to fall asleep, so I never learnt much in educational studies today…just because I had such a crap sleep last night” (Susan, AD)

Social impact/exclusion

Not surprisingly, given the reported impairments in mood and fatigue, participants frequently described a negative impact on social activities and meeting with friends. This tended to be expressed as having to cancel or avoid committing to social fixtures, because of the unpredictability of poor sleep and subsequent effects on functioning:

“I had planned to go to Edinburgh today but that I had to cancel, my friend came over later on, and I decided against going out because I just felt too tired, I find this all very frustrating, I’m a pretty sociable person...so it doesn’t suit me at all” (Laura, AD)
Others would make the effort to attend social gatherings, but risk being tired and/or leaving early:

“because I’m so used to not sleeping, I just get by anyway, but by the end of the night I might be the first one home” (Kate, FG 2)

“…today my sleep really did impact my day, I was really really slow, and couldn’t keep up with anyone’s jokes at university, eh I was just kinda like unresponsive” (Sean, AD)

Participants also voiced concerns that sleep, indirectly through fatigue and reductions in energy, affected keeping in touch with friends. This was a largely ‘self-imposed’ exclusion from social activities, with participants making the ‘choice’ not to call/answer phone calls:

“I’m terrible for em, I don’t get away with it if I’m not in the house myself, but if I’m in the house myself I let the answer machine pick up because I just canny be bothered with the conversation” (Rachel, FG 1)

**Good sleep/remove insomnia**

The greatest insight into the limiting and obstructive nature of insomnia was best achieved when participants discussed how their life would change if their insomnia were to cease. The majority of participants described predicted improvements in lifestyle (including retraining for a preferred occupation, taking up hobbies, enhanced social life), and importantly, enjoyment of life.
“I certainly have lots of regrets about my life and things that I haven’t done and haven’t achieved... and I put it down to the chronic insomnia... so if I could establish a sleep pattern again, I would take on a lot of challenges in my life, I would go overseas to work for example, and socially I would change my social situation as well.. I would definitely change a lot” (Laura, FG 3)

Thus, in a way, it appeared that insomnia was acting as an obstruction, preventing participants from attaining goals, otherwise achievable if they were good sleepers:

“I don’t really like the job I’m in at the moment, and I just...to apply for a job and go for interview it’s just too much of a task...I feel just plod along and do what I’m doing...I just feel I could make a life changing difference, a new job...” (Alison, FG 1)

Participants realized that because poor sleep has a knock-on effect on many aspects of functioning, then several diverse areas would improve simultaneously:

“I feel as if my life would be fuller...if I was getting a proper sleep every night, then my concentration would be better, so my uni work would be better, so I’d probably get a better degree, I’d probably phone my friends a bit more because I’d have time to spend on the phone..I feel as though I’d go out and do things more” (Susan, FG 2)

Interestingly, such changes could actually be tracked with the audio-diaries. Even from waking ‘a good sleep’ could be conveyed not only in content of words but also tone:
“It’s Wednesday morning and I had a fantastic night’s sleep… I literally jumped out of bed to get ready to go out” (Sean, AD)

Sleeping well often then translated into a reversal, an almost ceasing of symptoms (‘condition on-condition off’ nature):

“I’m pleased to say I did have a much better day today, I managed to do everything that I had planned to do, I was able to concentrate a lot more, felt in a much happier mood, physically I had the energy to go out for a walk and enjoy chatting with friends etc., and all this is basically down to having a less disturbed night’s sleep” (Laura, AD)

It is important to note, however, that a small minority of participants didn’t feel that improvements in sleep would necessarily lead to dramatic alterations/improvements in functioning. Having lived with sleep disturbance for a number of years, and as a result, adapting and adjusting to its consequences through compensatory strategies, it appeared that life, after the removal of insomnia, would simply be much less of an effort. This ‘recalibration’ meant that although major transformations were not predicted, participants would definitely notice alleviation of forced effort:

“Well I don’t think, personally, it probably wouldn’t have any big life changes because I just do try and not let it stop me doing as much as possible... so I would just do things more willingly” (Rachel, FG 1)
3.5. Discussion

The goal of this study was to explore the daily experience of insomnia, with particular emphasis on functioning and individual QoL. Interpretative phenomenological analysis of focus group and audio-diary transcripts produced three superordinate themes: ‘just struggle through’, ‘isolated, feeling like an outsider’, and ‘insomnia as an obstruction to the desired self’.

Participants reported, in their own words, daily decrements in cognitive, emotional and physical functioning because of disturbed sleep. This supports existing work in those with PI, using both self-report retrospective and prospective rating scales (e.g. Moul et al., 2002; Buysse et al., 2007; Orff et al., 2007). The failure of people with insomnia to show unequivocal objective cognitive deficits, yet report enduring subjective impairment, continues to perplex the field (Orff et al., 2007). One potential explanation is that individuals with insomnia can exert enough ‘compensatory effort’ to overcome performance deficits during testing. Interestingly, our participants consistently reported “struggling through the day” or putting in extra “effort” to complete tasks; thus, it may be that such “compensatory mechanisms” are sufficient to minimize any ‘obvious’ performance decrements (Orff et al., 2009; Espie & Kyle, 2008). It is intuitive also that putting in extra ‘effort’ in an attempt to maintain functioning during the day may exacerbate feelings of fatigue, poor concentration and memory, perhaps contributing to inflated subjective-objective discrepancies (Varkevisser et al., 2007).

Alterations in mood and increased irritability were frequently reported by participants after several days of poor sleep. Compared to normal sleepers, individuals with PI typically show elevated scores - but below clinical thresholds - on measures of
depression, worry and anxiety (e.g. Buysse et al., 2007). Moreover, it has been documented that insomnia assessed at time point one can independently predict the development of depression at time point two (e.g. for a review see Riemann & Voderholzer, 2003). Interestingly, participants frequently reported feeling ‘depressed’, ‘weepy’ or ‘low’ after a couple of nights of poor sleep, and made strong causal attributions to their disturbed sleep. It is intuitive that the limiting daytime consequences of insomnia, such as impairment in social functioning, may also precipitate the onset of a depressive episode – participants were certainly frustrated at having to cancel/avoid social contact. Although such a hypothesis may be hard to test, practically and ethically, audio-diaries do appear to be a particularly sensitive tool for capturing how an individual feels, and thinks, after sleeping poorly. Future studies using this methodology, over a longer duration, may provide better insights into the link between insomnia and mood disturbance.

Participants further described how insomnia impacted upon physical functioning and health. Cross-sectional data suggest associations between insomnia and increased medical consultations, as well as more frequent health problems, relative to normal sleepers (e.g. Simon & VonKorff, 1997). Causality is, however, difficult to establish. Immune functioning would appear to be an obvious link, and although alterations in immune markers (i.e. elevated IL-6, lower counts of lymphocyte subpopulations) have been documented in those with PI, the clinical significance of these alterations remain unknown (Burgos et al., 2006; Savard et al., 2003). Further studies using larger samples and repeated measurement points are needed to determine if insomnia is associated with significant immune alterations at baseline, and if so, whether these (and subjective reports of well-being) ‘normalize’ after successful treatment.
During focus group interactions, participants expressed feelings of ‘isolation’ because of their insomnia. This was attributed to a lack of understanding and sympathy from others, as well as sleep disturbance not being taken seriously as a single, important issue. Previous qualitative work supports the notion that those with insomnia feel that others, including medical staff, do not realize how serious sleep disturbance and its consequences can be: insomnia as a ‘hidden disorder’ (Carey et al., 2005) and ‘an invisible weight’ (Greene, 2008). Participants frequently felt that others believed they should ‘get over it’, ‘stop being silly’, or that there must be some other deep-rooted issue, like depression. This led some to look inward, questioning whether they were doing something ‘wrong’, or that poor sleep was somehow self-inflicted (‘something lacking in me’). This not only amplified perceptions of isolation but also created a sort of ‘felt stigma’ (Scambler, 1988) among participants, choosing to conceal their sleeping problem from others. Intriguingly, similar experiences of ‘felt scepticism’ and ‘rejection’ have been reported by patients with other conditions, such as chronic fatigue syndrome and chronic pain, where there are no obvious exposed or ‘objective’ symptoms (Dickson et al., 2008; Glenton, 2003).

Not being taken seriously forced participants to adopt a hopelessness/helplessness approach to their insomnia; possibly accounting for the recent finding that individuals with PI fail to seek treatment primarily because of the perception of insomnia as a “benign, trivial problem or that one should be able to cope with alone” (Stinson et al., 2006). Importantly, participants found the group format helpful for discussing and sharing experiences. Given that most of the sample had never met anyone with similar sleep difficulties, the chance to reflect and exchange experiences was viewed positively, and some argued that this facility may have helped had it been available at the onset of
their sleep difficulties. This does raise the issue whether targeted social support groups could prove useful in ameliorating insomnia-associated concerns and ‘felt stigma’, potentially removing barriers to treatment. Patients taking part in a group cognitive behavioural intervention (CBT) for insomnia have certainly reported great value from meeting others with similar difficulties (Green et al., 2005).

Of note, and perhaps in direct contrast to how others perceive insomnia (at least as far as participants were concerned), chronic insomnia was reported to be a “major thing”, affecting several important areas of functioning. For example, participants frequently described impairments in vocational/occupational functioning. Existing data on insomnia and the workplace have largely focused on absenteeism, with mixed results (e.g. Leger et al., 2006; Phillip et al., 2006). In the current study very few participants reported taking days off/missing lectures due to sleep disruption; instead they described “struggling through the day” with subsequent effects on productivity and performance. Only recently have studies documented measurable impairments in workplace functioning, using rating scales (e.g. David & Morgan, 2006; Erman et al., 2008); our sample revealed the nature of this impairment. Specifically, work output, completion of tasks on time, effort, and learning were all impacted. Interestingly, our participants reported awareness of compensatory techniques to minimize vocational impairment, allowing themselves to “gauge” their way through the day. Such techniques included completing work at home, scheduling tasks to optimize cognitive and social performance, forced physical activity (going for a walk, making tea, rubbing eyes, reading text) to stave off fatigue and sleepiness, and off-setting ‘heavier’ duties to another day. Given that the workplace appears particularly sensitive to the consequences of insomnia, and plays an important role in prompting sufferers to seek
medical treatment (Henry et al., 2008), there is a great need for future intervention studies to include occupational outcome measures.

Similarly, participants complained that insomnia has a detrimental impact on social functioning. Again this domain has received little attention in its own right, usually being assessed as a construct within overall HRQoL measurement. Studies have documented lower scores on the social functioning sub-scale of the SF-36 in those with PI relative to normative reference values (e.g. Walsh et al., 2007). However, this component sub-scale simply asks two questions relating to extent and frequency of social ‘interference’, failing to capture what this ‘interference’ actually is. Focus groups and audio-diaries revealed an inability to plan social events because of sleep instability, self-imposed social inclusion due to lack of enthusiasm/energy, and reduced responsiveness when in social situations.

Participants found it difficult to think of areas of functioning that escape impairment. This does fit with questionnaire studies demonstrating decrements in every aspect of HRQoL relative to controls (e.g. Leger et al., 2001), as well as cross-disease comparisons indicating comparable, and more wide-ranging, HRQoL impairments than patients with depression and congestive heart failure (e.g. Katz & McHorney, 2002). Indeed, it is this enduring, all encompassing nature of insomnia that is characteristically reported by patients during consultations. Participants felt the burden of insomnia prevented them from achieving goals/aspirations in life e.g. travelling, working full-time, training/applying for a preferred vocation, performing in university/college courses, and taking up hobbies. Importantly and not surprisingly, there was great diversity in the areas that individuals reported to be affected, as well as differences in
the relative importance of each area. Thus, it would appear crucial that assessments of functioning and QoL take into account those aspects that are important to the individual. Previous studies using standardized measures may not have been sensitive enough to detect initial daytime distress, and therefore improvement, post-intervention. This may explain why improvements in sleep are not always closely mirrored by improvements in daytime functioning (e.g. Omvik et al., 2008). Qualitative, patient-centred methodologies in combination with both disease-specific and generic outcome measures may provide a better indication of treatment success in improving daytime functioning.

This study has to be viewed within the context of several limitations. Firstly, it is worth pointing out that our participants, through volunteering to take part in a qualitative study focusing on daytime functioning, may over-represent those with severe and pronounced functional impairments. That being said, many of our themes and sub-themes can be linked/related to issues in the existing literature, including qualitative/quantitative, and clinical/non-clinical studies. Importantly, because we adopted an IPA method to explore participants’ experience of insomnia, our conclusions can only be attributed to the 11 participants within the study - and not taken as representative of all individuals with PI. Moreover, traditional exemplary methods for IPA are semi-structured interviews and other idiographic approaches e.g. diaries (Smith, 1999); we opted to use focus groups given that previous literature (Carey et al. 2005) suggests insomnia can be an isolating and misunderstood condition, and therefore creating a supportive environment was a key methodological consideration. Focus groups also tend to overcome power imbalances between researcher and participants (a potential problem when conducting face-to-face interviews), and capitalize on shared experiences through everyday communication (Kitzinger, 1995). We do realize, however, the limitations of
focus group methodology when attempting to explore personal, individual experience, particularly problems concerning participant and issue dominance; nevertheless, such influences can be attenuated through effective group facilitation. Comparing and contrasting our findings with future studies that utilize other qualitative methods (such as semi-structured interviews) would be informative. Finally, interpretation is central to IPA, and thus the beliefs and role of the researcher, when running focus groups and analyzing transcripts, inevitably influenced interpretation of data.

A number of research possibilities are worth mentioning that could extend and build upon the present study. For example, we included participants of working age only, it would be interesting to investigate, qualitatively, daytime functioning in elderly adults with insomnia, given there is some indication in the literature that this group may have less severe HRQoL impairments (see Kyle et al., 2010). Qualitative inquiry applied to insomnia in the context of other medical and psychiatric illness, may also be a worthwhile endeavour; perhaps helping to uncover the intimate reciprocal relations between insomnia and co-occurring conditions. One final implication of our research concerns measurement of functional impairment. It is clear that future tools assessing daytime functioning and quality of life in insomnia need to include items that adequately tap domains relevant to patients with insomnia, as revealed in the present study. Indeed, in Glasgow we are piloting a measure which goes one step further: asking patients to generate, in their own words, relevant insomnia-related impairments, which are then converted into a quantifiable metric (Kyle et al., 2009). Such an approach may be a particularly sensitive way to evaluate simultaneous changes in both sleep and functioning.
In summary, the current study highlights that individuals with persistent insomnia encounter difficulties and impairments on a daily basis and across many aspects of functioning. A lack of empathy and understanding from others, coupled with the inadequacy of the medical profession at getting to the ‘source’ of sleep difficulties, created anxiety, isolation and ‘felt stigma’ amongst participants. Insomnia was described as limiting, affecting QoL variables such as work performance, careers, life aspirations and social functioning. It is important that policy-makers and health care providers understand that insomnia is a serious condition with deleterious consequences, and attempts be made to integrate effective and sustainable treatments, such as cognitive behavioural therapy (Espie et al., 2007; Morgan et al., 2003), into everyday clinical practice.
Chapter 4:

The Development of Two New Measurement Approaches to Assessing Insomnia-Related Quality of Life and Daytime Functioning: the Glasgow Sleep Impact Index (GSII) and the Daytime Functioning and Sleep Attribution Scale (DFSAS)
4.1. Abstract

Daytime dysfunction and quality of life (QoL) impairment are clearly important and salient consequences of poor sleep in those with insomnia. The surprising lack of suitable tools to assess these two constructs limits conclusions that can be drawn regarding which (and if) treatments are effective in improving both sleep and non-sleep aspects of insomnia. Existing measurement approaches to functional impact tend to rely on non-specific generic tools, non-validated scales, or single scale items; in short, they are sub-optimal at best and misleading at worst. In this chapter, two new measurement approaches to assessing and understanding the downstream effects of insomnia on functioning and QoL are explored. The Daytime Functioning and Sleep Attribution Scale (DFSAS) and the Glasgow Sleep Impact Index (GSII) capture impairment in (1) common everyday daytime functional domains, and (2) individual-specific insomnia-related impairment, respectively. Preliminary results indicate these measures to have good psychometric properties; including, high internal consistency, strong face, construct, and discriminant validity, and sensitivity to change, post-behavioural treatment. Further work should investigate test-retest reliability, and assess sensitivity to change in the context of large randomised controlled trials of both psychological and pharmacological interventions for insomnia. The described novel attempts to capture and measure functional impairment in insomnia, it is hoped, will stimulate others to focus on the fundamental measurement of the insomnia experience.
4.2. Introduction

A number of studies indicate that insomnia negatively impacts several domains of daytime functioning (e.g. Kyle, Espie & Morgan, in press; Buysse et al., 2007; Alapin et al., 2000), and this, of course, is reflected in contemporary disease/disorder classification manuals (DSM-IV, 1994; ICSD-2, 2005; ICD, 1992; RDC, Edinger et al., 2004). Both qualitative and quantitative research also highlight that the functional impact of sleep disturbance is what drives treatment-seeking behaviour for many patients (Henry et al., 2008; Morin et al., 2006a).

Several authors have commented on the need to better understand the waking consequences of insomnia (Buysse et al., 2006; Orff et al., 2007), as well the urgency to develop tools that are sensitive to impairments experienced by patients (e.g. Riedel & Lichstein, 2000; Moul et al., 2004; Gradisar et al., 2007; Krystal, 2007). With the documented mixed literature on neurocognitive performance in those with insomnia (Riedel & Lichstein, 2000; Fulda & Schulz, 2001; NIH state-of-the-science conference statement, 2005; Shekleton et al., 2010), coupled with conflicting reports surrounding post-interventional changes in daytime parameters (Omvik et al., 2008; Means et al., 2000), it seems increasingly important to ‘home-in’ on, and develop measures to adequately capture, self-report functioning.

Accordingly, there has been increased emphasis on scale development. Gradisar et al. (2007) created the 7-item Flinders Fatigue Scale (FFS) to probe components of fatigue, often the most frequently reported daytime symptom by those with insomnia. Preliminary data indicate good discriminant validity, high internal consistency, and sensitivity to treatment outcome; suggesting the FFS may be a useful measure in outcome studies involving insomnia populations. Secondly, Leger and colleagues
(2005) developed the Hotel Dieu 16 (HD-16), a measure designed to assess ‘Quality of Life’ in those with insomnia, owing to the lack of an existing widely used disease-specific tool. Domains cover the following categories: physical role, energy, cognitive, social and psychological functioning. Strengths of this measure include: appropriate item generation (based on patient interviews and then factor analysis); graded discriminant validity across ‘severe insomniacs’, ‘mild insomniacs’ and normal sleepers; and dimensional as well as global score calculation. The HD-16, however, has several limitations; specifically concerning translational issues, complex score derivation, and lack of available data on test-retest reliability, concurrent validity, and sensitivity to treatment outcome. Indeed, to the best of our knowledge, no single published study has used the instrument, other than the original validation paper.

In a recent thorough review of the literature on health-related quality of life (HRQoL) in insomnia, Kyle and colleagues (2010; also chapter two of this PhD thesis) outline the need to develop measures that adequately capture QoL impairment relevant to patients. They highlight that the majority of studies assessing HRQoL or the more global ‘Quality of Life’ construct, have used generic instruments, such as the Short Form Health Survey 36 (SF-36; Ware & Sherbourne, 1992) - a health status tool. Reliance on generic and non-specific measures in insomnia outcome research may be obscuring real treatment effects (Omvik et al., 2008; Soeffing et al., 2008), and hence leading to classic type II error conclusions. Kyle et al. also suggest that more attention be paid to the underlying constructs that tools are supposed to measure, such as daytime functioning, Quality of Life (QoL) and Health-related quality of life (HRQoL) - which are typically used interchangeably, indiscriminately and without definition in most of the insomnia, and indeed health literature. For example, ‘daytime functioning’ has usually been
assessed by administering scales that measure various discrete constructs e.g. the Beck Depression Inventory (BDI; Beck et al., 1961) for depression, the Beck Anxiety Inventory (BAI; Beck et al., 1988) for anxiety, and the Fatigue Severity Scale (FSS; Krupp et al., 1989) for fatigue. The problem with this approach is that, firstly, those with primary insomnia are excluded for co-morbid psychopathology, and so there is limited scope for change within the sub-clinical category; and secondly, daytime impairment can vary within an individual, so that only some domains are affected (above some personal ‘interference threshold’), while others remain unaffected.

In a similar vein, Krystal (2007) reviewed work on general daytime impairments in insomnia, covering isolated symptoms and domains of functioning. Although it was concluded that (1) daytime impairment reliably exists in those with insomnia, and (2) both pharmacological and non-pharmacological treatments can improve daytime symptoms, much of the reviewed studies used non-validated, non-specific questionnaires, or just single items, to assess daytime symptoms/functioning. The message from both review papers is clear: the field needs to pay more attention to the measurement of non-sleep variables, particularly aspects of subjective functioning and quality of life, importantly, as they relate to insomnia patients.

Other than the HD-16, there are only two scales claiming to assess aspects of functioning specific to insomnia: the Quality of Life of Insomniacs questionnaire (QOLI; Rombaut et al., 1991) and the Insomnia Impact Scale (IIS; Hoelscher et al., 1993). The QOLI is a 52-item measure made up of three existing questionnaires (the Psychological Well-Being Index, the Leeds Sleep Evaluation Questionnaire, and the Jenkins Sleep Evaluation Questionnaire) plus an additional 22 items. Although the
QOLI has been used in a handful of studies, with some evidence of treatment-outcome sensitivity (e.g. Goldenberg et al., 1994), it has a number of limitations. These include: a lack of adequate data on psychometric properties; an exhaustive number of items covering both sleep and non-sleep domains; and varied response formats. The IIS is a 40-item measure assessing sleep-related impairments in functional, cognitive, social, occupational, emotional and physical domains. Again basic data are lacking on this scale; no paper has been published documenting its psychometric properties (the initial validation study remains a conference abstract); items cover non-daytime functioning domains; ratings are made with reference to ‘experience’ or ‘belief’, making it unclear what construct the scale assesses; and only one research group continue to use it (Soeffing et al., 2008; Means et al., 2000; Ustinov et al., in press).

A limitation of the IIS, and a challenge for all measures designed to probe functioning relevant to insomnia, is the loaded nature of items linking poor sleep to impaired functioning. For example, the IIS asks participants to rate their agreement with 40 statements, with reference to the ‘past two weeks’, such as: ‘I have problems concentrating and I make foolish errors after a poor night of sleep’. Wording of items in this manner may, for both poor and normal sleepers, lead to artificially elevated scores by (1) priming the PI patient’s negatively toned cognitive set (Harvey, 2002); and (2) prompting the normal sleeper to reflect on a rare occasion when they did experience disturbed sleep, and hence, conclude that ‘poor sleep does in fact cause me to have problems with concentration and to make foolish errors’. Of course, it is also possible that normal sleepers struggle to recollect any significant episode of poor sleep, creating floor effects and inflating between group differences (Riedel & Lichstein, 2000). Further, even if sleep and subjective functioning were to improve in a group of
individuals with insomnia, post-intervention, the very nature of the wording of these items and the response context (i.e. ‘experience’ or ‘belief’), could still generate high scores (indicative of impairment), as the ‘reformed’ poor sleeper recounts prior or occasional current episodes of sleep disturbance. It is interesting that treatment effects have never been documented using this scale despite quite robust improvements in sleep (Soeffing et al., 2008; Means et al., 2000).

Clearly, what is needed (see table 4.1) is some measure of daytime functioning that limits the effect of sleep- or insomnia-related priming, and which permits the collection of appropriate normative data by which to then gauge whether a patient has returned to ‘normal’ levels of functioning. The attribution for poor sleep in accounting for functional impairment is, of course, also very important; for two main reasons. Firstly,

Table 4.1 – Required scale characteristics to meet existing measurement needs.

<table>
<thead>
<tr>
<th>Required scale characteristics for use with insomnia patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Assessment of concerns relevant to each individual patient</td>
</tr>
<tr>
<td>2. A measure which permits ranking of concerns in order of personal importance</td>
</tr>
<tr>
<td>3. Some measure of the attribution for poor sleep in causing daytime impairment</td>
</tr>
<tr>
<td>4. Coverage of items that are commonly reported by insomnia patients as a group</td>
</tr>
<tr>
<td>5. Brief measure that can be used in both clinical and research settings</td>
</tr>
<tr>
<td>6. Can be completed by normal and poor sleepers with limited sleep-related priming</td>
</tr>
<tr>
<td>7. Can be completed by patients with additional health conditions</td>
</tr>
</tbody>
</table>
several cognitive processes have been investigated as being relevant to the development and maintenance of insomnia (Harvey, 2002; Harvey et al., 2005; Espie, 2002; Morin, 1993). For example, the tendency to catastrophize about the consequences of poor sleep, perhaps coupled with underlying maladaptive sleep-beliefs, may render some individuals with increased sleep-related threat detection, monitoring, and enhanced sleep pre-occupation (Harvey, 2002). It is proposed that these individuals are likely to misinterpret non-significant innocuous cues as evidence of sleep-related impairment, fuelling daytime anxiety and dysfunction via enhanced cognitive load and compensatory safety behaviours. Being able to identify those with a markedly high (mis-) attribution for poor sleep in causing daytime impairment may help in tailoring interventions for an individual, ultimately enhancing the probability of treatment response.

Secondly, the co-occurring nature of insomnia (e.g. Sarsour et al., in press) has been put forward as the biggest challenge to developing scales for specific use with insomnia patients (e.g. Reimer & Flemons, 2003; Buysse et al., 2006). The ability to partial out the effect of insomnia on everyday functioning, in the context of co-morbid pathology, would be desirable, and have particular relevance in the clinical context. Above all though, assessment must reflect concerns and values of individual insomnia patients.

This chapter describes the background, creation and development of two new tools, from differing theoretical perspectives, that may address the void in current functional assessment of insomnia. The Daytime Functioning and Sleep Attribution Scale (DFSAS) explores, in a two part format, the level of daytime interference across 12 commonly reported symptoms (part 1), and the perceived extent to which the
impairment is caused by poor sleep (part 2). The Glasgow Sleep Impact Index (GSII), conversely, asks patients to generate their own items. Specifically, in this patient-centred measure, items unique to each individual are ranked on importance and subsequently rated in relation to perceived impact.

4.3. The Daytime Functioning and Sleep Attribution Scale (DFSAS)

4.3.1. Background & Aims

There is a need for a scale that can be used by both individuals with sleep disturbance and normal sleepers. Secondly, items need to have good face validity i.e. reflect concerns and experiences relevant to patients and their everyday lives. Thirdly, there is a necessity to tease out the perceived impact of poor sleep in contributing to daytime impairment in (1) those with primary insomnia and (2) patients who suffer from additional co-morbidities beyond sleep disturbance.

With these needs in mind we created the Daytime Functioning and Sleep Attribution scale (DFSAS; see figure 4.1), a 12-item, two-part scale. Part 1 asks participants to rate 12 impairment-related aspects of daily functioning (e.g. ‘Difficulty concentrating and focusing on things’) in terms of how much of a problem (0-3) it has been in the past two weeks. Part 2 then asks participants to re-rate each individual item, but this time in relation to how much poor sleep was responsible (0-4) in generating the impairment. Thus, part 1 (range: 0-36) is a general assessment of everyday functional limitations and problems, and part 2 (range: 0-48) is a measure of how much the respondent believes that poor sleep accounts for the experienced impairment.
Figure 4.1 - Copy of the Daytime Functioning and Sleep Attribution Scale (DFSAS).

<table>
<thead>
<tr>
<th>Name:</th>
<th>PART 1</th>
<th>Date:</th>
</tr>
</thead>
</table>

_Please complete Part 1 before moving on to Part 2._

Please rate each item below on how much of a problem it has been for you, in the past two weeks. Circle the most appropriate response.

1. **Difficulty concentrating and focusing on things:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

2. **Feeling irritable:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

3. **Fatigue or tiredness:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

4. **Feeling ‘down in the dumps’/ low mood:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

5. **Not performing at work or study as well as you would like to:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

6. **Feeling tense or anxious:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

7. **Difficulty remembering things:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

8. **Lack of energy and motivation:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

9. **Aches and pains:**
   - No problem at all
   - Only a very slight problem
   - Somewhat of a problem
   - A very big problem
   - Not at all  
   - Only slightly  
   - Moderately  
   - To a large extent  
   - Entirely

10. **Avoiding or cancelling social activities:**
    - No problem at all
    - Only a very slight problem
    - Somewhat of a problem
    - A very big problem
    - Not at all  
    - Only slightly  
    - Moderately  
    - To a large extent  
    - Entirely

11. **Feeling sleepy during the day:**
    - No problem at all
    - Only a very slight problem
    - Somewhat of a problem
    - A very big problem
    - Not at all  
    - Only slightly  
    - Moderately  
    - To a large extent  
    - Entirely

12. **Lack of desire for physical intimacy or sex:**
    - No problem at all
    - Only a very slight problem
    - Somewhat of a problem
    - A very big problem
    - Not at all  
    - Only slightly  
    - Moderately  
    - To a large extent  
    - Entirely

Many things can determine the way we feel and behave. In your opinion, how much was poor sleep responsible for your answers in part 1, in the past two weeks? Please circle the most appropriate response for each item.

- Not at all
- Only slightly
- Moderately
- To a large extent
- Entirely
4.3.2. Methods

4.3.2.1. Participants

To investigate basic statistical properties of the scale, including discriminant validity, internal consistency, and sensitivity and specificity, we combined three samples of highly screened individuals with primary insomnia (PI). PI patients who completed the scale were from the following University of Glasgow Sleep Centre (UGSC) studies: those taking part in a condensed sleep restriction intervention ($n=18$; see chapter five); those taking part in a mechanistic study ($n=12$) investigating adherence to stimulus control treatment (Crawford, unpublished); and finally, nine participants meeting criteria for Psychophysiological insomnia (ICSD-2), taking part in an ongoing NIH-funded study. Thus, number of patients with insomnia totalled thirty nine. Normal sleepers ($n=31$) were recruited from the same aforementioned NIH study.

All PI patients met basic criteria for Primary Insomnia according to research diagnostic criteria (RDC; Edinger et al., 2004; DSM-IV, 1994). That is, they all had difficulties with initiating and/or maintaining sleep, or non-restorative sleep, lasting for at least a one-month period. All subjects underwent an initial screen using the UGSC brief screen protocol (see Appendix E). This records basic information on severity and frequency of insomnia symptoms, co-morbid medical/psychiatric/sleep difficulties, and medication use. Those satisfying the brief screen then received a thorough phone interview using the UGSC screening questionnaire, based on a template set out by Morin & Espie (2003; see Appendix A), to exclude those with affective/psychiatric disorder, those with sleep-disruptive medical co-morbidities, and to assess sleep pattern/symptoms to rule out occult sleep disorders. In keeping with diagnostic criteria of insomnia as a 24-hour disorder, participants also had to report experiencing at least
one daytime functional impairment, attributed to disturbed sleep (RDC; Edinger et al., 2004). Participants were all aged between 18 and 65, and recruited through media adverts and GP referral letters. Additionally, those patients in the NIH study (n=9) all received an overnight PSG screening assessment to (objectively) rule out occult sleep disorder pathology, and also met criteria for Psychophysiological insomnia based on ICSD-2 (2005) classification. Two (5%) out of the 39 patients were on sleep-promoting hypnotic medications at the time of scale completion.

Normal Sleepers (NS) had no complaint of sleep disturbance, failed to meet DSM-IV criteria for insomnia, did not use sleep-promoting hypnotics, and reported the absence of medical or psychiatric illness (ascertained using the UGSC brief screening protocol). Additionally, all NS were required to score ≤ 5 on the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

Participant demographics are presented in table 4.2. The mean (SD) age of the PI group was 43.9 ± 13.1 years; and 31.5 ± 13.5 years for normal sleepers. This difference was statistically significant \[ t(68) = 3.88, p<.001 \]. As expected, mean PSQI scores were

<table>
<thead>
<tr>
<th></th>
<th>PI (n=39)</th>
<th>NS (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>43.9 (13.1)</td>
<td>31.5 (13.5)</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td>27♀/12♂</td>
<td>18♀/13♂</td>
</tr>
<tr>
<td><strong>PSQI</strong></td>
<td>12.86 (2.6)</td>
<td>2.97 (1.4)</td>
</tr>
<tr>
<td><strong>ISI</strong></td>
<td>17.5 (3.9)</td>
<td>N/A</td>
</tr>
</tbody>
</table>

*Table 4.2 - Demographics for primary insomnia (PI) patients and normal sleepers (NS). Parentheses represent the standard deviation.*
higher for the patient group (12.86 ± 2.6) compared with normal sleepers (2.97 ± 1.4); this difference proved highly significant [$t(59.9) = 20.13, p<.001$]. Both groups had a greater female-to-male ratio, which was slightly more pronounced in the patient group, though a chi-square test for independence (with Yates continuity correction) indicated no significant association between gender and group status [$X^2 (1, n=70) = .52, p=.473$]. Mean (SD) ISI scores for PIs (17.5 ± 3.9) were in the ‘clinical moderate insomnia’ range.

4.3.2.2. Measures and procedures

Participants completed both the PSQI and the DFSAS to permit investigation into associations between global sleep quality and daytime functioning. PI patients also completed the Insomnia Severity Index (ISI; Morin, 1993) to investigate relations between insomnia severity and daytime impairment. Those patients taking part in the sleep restriction intervention (see chapter five) completed the Short-Form Health Survey 36 (SF-36; Ware & Sherbourne, 1992) and Occupational Impact of Sleep Questionnaire (OISQ; David & Morgan, 2006), permitting assessment of concurrent validity. Sensitivity to change could also be assessed from this treatment study as patients completed the DFSAS at post-treatment and at 3 months follow-up.

4.3.3. Results

4.3.3.1. Item generation and face validity

After several reviews of audio-diary and focus-group transcripts, collected during a previous qualitative investigation on daytime functioning and quality of life in insomnia (Kyle, Espie & Morgan, in press; see chapter three, table 3.2), several item-domains were identified to capture everyday functional impairments relevant to those with
insomnia. Discussions between the authors (researchers and clinicians) and one independent researcher not affiliated with the study, coupled with exhaustive reading of the literature (see Kyle et al., 2010), resulted in the agreement and acceptance of 12 items. Response formats were modelled on the widely used Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), for part 1, and the Flinders Fatigue Scale (FFS; Gradisar et al., 2007), for part 2. The time reference period, ‘in the past two weeks’, was chosen to parallel that of the Insomnia Severity Index, and the typical assessment period for subjective sleep diary parameters (10-14 days).

4.3.3.2. Discriminant validity

The ability of the DFSAS to discriminate insomnia patients (n = 39) from good sleepers (n = 31) was investigated. The mean DFSAS part 1 total score for insomnia patients was 17.12, SD = 5.67; and 6.16, SD = 3.06 for good sleepers. Mean DFSAS part 2 total score was 23.81, SD = 9.00, for patients; and 5.53, SD = 6.03 for good sleepers. Thus, both parts 1 [t(60.6) = 10.33, p < .001] and 2 [t(67) = 9.58, p < .001] readily discriminated the groups (figures 4.2 & 4.3 indicate the distribution of scores for both groups).

Because of group discrepancies in terms of age, we also conducted univariate analyses controlling for the influence of this variable. This revealed that age was not significantly related to group differences for either DFSAS part 1 [F(1, 67) = 1.42, p = .24, partial eta squared = .021] or 2 [F(1, 66) = .227, p = .64, partial eta squared = .003], and that group effects, partialling out age, remained robust (both p < .001).

Mean scores for individual DFSAS items (parts 1 & 2) also discriminated the groups. Item (mean) scores and related statistical significance are detailed in table 4.3 and graphically represented in figures 4.4 and 4.5.
Figure 4.2 - Boxplots of DFSAS part 1 scores for PI patients and normal sleepers (NS).

Figure 4.3 - Boxplots of DFSAS part 2 scores for PI patients and normal sleepers (NS).
Table 4.3 - Mean DFSAS item values for PI patients and normal sleepers (NS).

<table>
<thead>
<tr>
<th>Item</th>
<th>PI</th>
<th>NS</th>
<th>t</th>
<th>p</th>
<th>PI</th>
<th>NS</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.64</td>
<td>0.61</td>
<td>7.46</td>
<td>&lt; 0.001</td>
<td>2.59</td>
<td>0.47</td>
<td>10.03</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>2</td>
<td>1.46</td>
<td>0.61</td>
<td>5.22</td>
<td>&lt; 0.001</td>
<td>2.08</td>
<td>0.33</td>
<td>7.68</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>3</td>
<td>2.13</td>
<td>0.87</td>
<td>8.17</td>
<td>&lt; 0.001</td>
<td>3.31</td>
<td>0.97</td>
<td>9.96</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>4</td>
<td>1.28</td>
<td>0.52</td>
<td>4.38</td>
<td>&lt; 0.001</td>
<td>1.79</td>
<td>0.30</td>
<td>6.09</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>5</td>
<td>1.45</td>
<td>0.45</td>
<td>5.53</td>
<td>&lt; 0.001</td>
<td>2.21</td>
<td>0.37</td>
<td>7.84</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>6</td>
<td>1.21</td>
<td>0.52</td>
<td>4.62</td>
<td>&lt; 0.001</td>
<td>1.44</td>
<td>0.30</td>
<td>5.19</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>7</td>
<td>1.33</td>
<td>0.52</td>
<td>5.32</td>
<td>&lt; 0.001</td>
<td>1.67</td>
<td>0.23</td>
<td>6.53</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>8</td>
<td>1.92</td>
<td>0.71</td>
<td>7.45</td>
<td>&lt; 0.001</td>
<td>2.77</td>
<td>0.97</td>
<td>7.32</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>9</td>
<td>0.92</td>
<td>0.23</td>
<td>4.39</td>
<td>&lt; 0.001</td>
<td>0.62</td>
<td>0.30</td>
<td>1.51</td>
<td>0.136</td>
</tr>
<tr>
<td>10</td>
<td>0.92</td>
<td>0.10</td>
<td>4.75</td>
<td>&lt; 0.001</td>
<td>1.23</td>
<td>0.10</td>
<td>4.50</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>11</td>
<td>1.81</td>
<td>0.90</td>
<td>5.99</td>
<td>&lt; 0.001</td>
<td>2.92</td>
<td>1.07</td>
<td>7.24</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>12</td>
<td>1.13</td>
<td>0.13</td>
<td>4.83</td>
<td>&lt; 0.001</td>
<td>1.14</td>
<td>0.00</td>
<td>5.56</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Figure 4.4 - Radar plot of mean item (1-12) DFSAS part 1 scores for patients with primary insomnia (PI) and normal sleepers (NS).
4.3.3.3. Concurrent validity

Given the lack of a widely used measure to assess functioning specific to insomnia, we chose the SF-36 to investigate concurrent validity with DFSAS part 1 scores. Although not a directly comparable measure, the SF-36 was chosen because it has been shown to be sensitive to insomnia impairment, as well as to change, post-behavioural and pharmacological intervention (for a review, see Kyle et al., 2010; chapter two of this thesis). Concurrent validity was also assessed using the OISQ, a measure of sleep-related occupational impairment. Spearman correlational analyses revealed moderate negative associations between part 1 scores and the following dimensions of the SF-36: vitality ($\rho=-.51, n=18, p<.05$), general health ($\rho=-.54, n=17, p<.05$), emotional role limitation ($\rho=-.43, n=18, p=0.07$), mental health ($\rho=-.41, n=18, p=0.09$) and social
functioning \((\rho = -.40, n = 18, p = .10)\). The OISQ was found to be strongly and positively associated with DFSAS part 1 scores \((\rho = .76, n = 16, p < .01)\).

4.3.3.4. Relationship with sleep quality and sensitivity to treatment outcome

To investigate associations between functional impairment and sleep quality/insomnia symptoms, correlational analyses were carried out between DFSAS scores and both PSQI and ISI values for the patient group. Associations between PSQI and part 1 scores were weak \((\rho = .19, n = 38, p > .05)\). Relationships between PSQI and part 2 scores were weak to moderate \((\rho = .31, n = 38, p > .05)\). Conversely, associations between global ISI scores and DFSAS parts 1 \((\rho = .49, n = 38, p < .01)\) and 2 \((r = .61, n = 38, p < .001)\) were significant and moderate to large in strength.

Based on data from 18 patients taking part in the sleep restriction intervention (see chapter five), we assessed the sensitivity of the DFSAS to change over time with successful treatment. The DFSAS was completed at baseline, post-treatment (4 weeks), and 3-months follow-up. Repeated measures ANOVA were used to assess time effects, and post-hoc paired \(t\) tests investigated specific mean differences. Finally, responder analyses were also carried out to determine whether those who responded to therapy, in terms of overall insomnia severity, had lower scores on the DFSAS compared with non-responders to treatment.

Mean Insomnia Severity Index (ISI) scores for the total sample indicated significant time effects, with a baseline average of 17 decreasing to 10 at post-treatment, which remained at this level at 3 months follow-up (see chapter five). ANOVA for DFSAS part 1 indicated a main effect of time \([F(2, 26) = 12.42, p < .001]\). Post hoc \(t\)-tests (see
Table 4.4 revealed significant improvements at post-treatment, relative to baseline, and a further enhancement effect at 3 months; with values being significantly lower (improved) compared with both baseline and post-treatment scores (see figure 4.6).

Table 4.4 - Treatment sensitivity for DFSAS parts 1 and 2 in PI patients.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th></th>
<th>Post-treatment</th>
<th></th>
<th>Follow-up (3 months)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td>M (SD)</td>
</tr>
<tr>
<td>DFSAS Part 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>16.93</td>
<td>5.24</td>
<td>11.36*</td>
<td>4.99</td>
<td>8.64**&lt;a&gt;</td>
<td>4.41</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DFSAS Part 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>23.21</td>
<td>10.96</td>
<td>18.79</td>
<td>12.08</td>
<td>14.60*</td>
<td>10.36</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Asterisks indicate post-treatment changes from Baseline: *p<.05, **p<.01
a. contrast with post-treatment mean sig. at p<.05

Figure 4.6 - DFSAS part 1 mean (± standard error) scores over the course of intervention.
Part 2 scores similarly evidenced a main effect of time over the assessment points \([F(2, 26) = 4.56, p<0.05]\). Paired \(t\) tests indicated significant mean reductions at 3 months relative to baseline, though between-subject variability remained high (see figure 4.7).

Figure 4.7 - DFSAS part 2 mean (± standard error) scores over the course of intervention.

A treatment responder analysis was also carried out to compare responders versus non-responders. Those showing at least a six point change on the ISI were considered to be treatment responders (Yang et al., 2009). Twelve (66.6%) patients were found to be in the ‘responder’ group; the remaining six were classified as non-responders.

Significant responder versus non-responder effects were found for DFSAS part 1 \([U = 6.50, z = -2.79, p<.01]\) and a trend was present for part 2 \([U = 14.00, z = -1.91, p=.056]\), with responders having substantially lower scores (indicative of less impairment; see table 4.5)
Table 4.5 - Responder analysis for DFSAS parts 1 and 2. Median (Mdn) values are presented with the interquartile range (IQR) in parentheses.

<table>
<thead>
<tr>
<th></th>
<th>Responders (n=12)</th>
<th>Non-Responders (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mdn</td>
<td>IQR</td>
</tr>
<tr>
<td>DFSAS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part 1</td>
<td>8.00</td>
<td>(7-10)</td>
</tr>
<tr>
<td>Part 2</td>
<td>11.00</td>
<td>(7-18)</td>
</tr>
</tbody>
</table>

4.3.3.5. Internal consistency

Cronbach's $\alpha$ was 0.91 for DFSAS part 1 and 0.94 for part 2. When the PI patients were investigated separately, Cronbach’s $\alpha$ remained high at 0.81 for part 1 and 0.89 for part 2. Items were then systematically removed to ascertain the stability of the DFSAS, reflected in the item-deletion alpha (Broomfield & Espie, 2005). For part 1, specifically for the patient group, item-deletion alphas remained high (mean $\alpha = 0.80$, range 0.77-0.84). Item-deletion alphas similarly remained high for Part 2 (mean $\alpha = 0.88$, range 0.87-0.89).

4.3.3.6. Sensitivity and specificity

Both sensitivity (the probability that an individual with the condition will be correctly classified as having the condition) and specificity (the probability that a person without the condition will be properly classified as not having the condition) were calculated for DFSAS parts 1 and 2 using receiver operating characteristic (ROC) curves. The area under the curve was 0.945 ($p<.001$) and 0.954 ($p<.001$) for parts 1 and 2, respectively. A cut-off $\geq 10$ on part 1 correctly identified 87.2% of PI patients and 87.1% of normal sleepers (see table 4.5). In relation to part 2, a cut off $\geq 13$ correctly identified 92.3% of PI patients and 86.7% of normal sleepers.
Table 4.6 - Sensitivity and specificity of DFSAS in identifying NS and PI patients.

<table>
<thead>
<tr>
<th></th>
<th>PI</th>
<th>NS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Using DFSAS part 1 cut-off score of ≥ 10:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct</td>
<td>34</td>
<td>27</td>
</tr>
<tr>
<td>Incorrect</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Using DFSAS part 2 cut-off score of ≥ 13:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct</td>
<td>36</td>
<td>26</td>
</tr>
<tr>
<td>Incorrect</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

4.3.3.7. Relationship between DFSAS parts 1 and 2

Because DFSAS items reflect those daytime impairments associated with poor sleep (as reported qualitatively by patients), it was predicted there would be a relationship between magnitude of impairment (part 1) and attribution for poor sleep as being responsible for the impairment (part 2). Accordingly, correlational analyses were carried out revealing a positive association between scores on part 1 and scores on part 2 for PI patients ($r = .60, n=39, p < .001$).
4.4. Glasgow Sleep Impact Index (GSII)

Having presented an approach grounded in psychometric theory, where the construct under investigation is pre-determined, we decided next to investigate the utility and practicability of an individualised patient-centred measure that recruits the expertise of the patient when assessing insomnia-related quality of life impairment.

4.4.1. Background & Aims

Kyle et al. (2010) in their review of the insomnia and quality of life literature point out that in order to claim measurement of the ‘quality of life’ construct, concerns and values relevant to each individual patient must be considered. The main criticism being, that generic measurement with, for example, the SF-36 takes little account of domains of functioning that are salient or specific to the individual with insomnia. The net effect is that these measures may be less sensitive to picking up treatment effects because of the neglect of issues important to patients, coupled with the inclusion of non-relevant items. Of course, this is a problem that, to some extent, pervades nearly all research founded on traditional psychometric (nomothetic) ideology: ultimately the aim is to make rules and predictions about the group on some ‘global’ construct (for example, ‘depression’).

Alternative approaches to symptom measurement have existed in Psychiatry and Clinical Psychology for some time, perhaps starting with Monte Shapiro’s (1961) personal questionnaire. Dissatisfied with multi-item scales that fail to reflect the illness experience, or intensity of symptoms relevant to each individual patient, Shapiro developed a system to assess clinical change using the words of patients to describe their current illness state. In a second stage, statements are constructed, again based on the patient narrative, which describe desired states of ‘improvement’ and ‘recovery’.
Over time, these states are compared and contrasted to monitor clinical change (in a number of symptom domains) within the individual patient. The strength of this idiographic, patient-centred approach to measurement is the ability to track aspects of illness that are otherwise difficult to do using psychometric theory, aided by recruiting the patient ‘expert’ into the process. The construct to be measured is defined by the individual; not the other way round, as is typically the case in psychometric research (Bilsbury & Richman, 2002). Such patient-centred and clinical staging methodologies have recently gained renewed momentum in the psychiatric research community (Bilsbury & Richman, 2002), in the health-related quality of life measurement literature (Carr & Higginson, 2001), and for assessment of outcomes in primary care (e.g. Paterson, 1996).

From our early qualitative work with insomnia patients (Kyle, Espie & Morgan, in press; chapter three of this thesis), it was clear that although common domains of sleep-related impairment were expressed at the group level, each individual assigned different values of importance to particular domains of impairment, within the context of their own daily lives. We therefore wanted to create a measure that would allow patients with insomnia to describe impairments that were most important and relevant to them, and hence capture *insomnia-related quality of life impairment*, which could subsequently be evaluated within both clinical and research settings. To do this we modified the standard patient-generated index (PGI; Ruta et al., 1994), an individualised measure of quality of life.

In the standard PGI, participants are asked to write down, in a structured template, the five most important areas of their life affected by a particular health condition (e.g.
They are then asked to rate the areas on a 0-100 scale, with scores ranging from ‘the worst you can imagine’ to ‘exactly as you would like it to be’. Finally, participants prioritise each generated item by assigning points (out of 60) to the area(s) considered most important to eradicate. There is also an additional box referring to ‘all other areas of your life’, which is similarly rated and assigned points. Overall score is calculated by multiplying ratings by the weighted sum of the ‘points’ value, per item, and combining them to give a score between 0 and 100. The PGI is based on a model that considers QoL to be the gap between reality and expectations: the more pronounced the gap, the poorer the current state of QoL (and vice versa). Hence the aim is for reality to be as close to expectations as possible (Ruta et al., 1994).

### 4.4.2. Scale development & pilot

We initially made only small modifications to the standard PGI, using the aforementioned three-part format, including item generation, rating, and point assignment sections. For the ‘list areas’ section, participant instructions were to “write down the five most important areas of your life that are affected because of poor sleep”. A list of domains was provided as a ‘trigger’ list to prompt and aid participants in item generation (as in the initial PGI). Subsequently, we informally piloted the scale on a small sample of participants who took part in a previous qualitative study (Kyle, Espie & Morgan, in press; chapter three), to gain feedback on face validity, ease of completion, and practicability.

Observations from this pilot indicated three important limitations of the initial measure: (1) participants could not always generate five areas; (2) participants tended to copy areas directly from the ‘trigger’ list, particularly if they were unable to generate them
independently (a previously documented problem; Patel et al., 2003), and (3) the section ‘all other areas of your life’ was confusing to patients and often left incomplete. Based on this feedback we made several modifications to the scale (see figure 4.8), so that participants were now asked to generate three items (stage 1), rank the three items in terms of importance (1-3; stage 2), rate how ‘bothered’ they had been by the impairment in the past 2 weeks on a 100mm visual analogue scale (VAS; stage 3), and, finally, ‘spend an imaginary £60’ on getting rid of impairments of choice (stage 4). This final version of the scale was designed to allow participants to generate items without a trigger list, so fully in their own words; and also to enable collapsing of ranks (1-3) across the group, permitting within-subject analyses of change, yet at the same time retaining individual patient concerns.

This collapsing of generated ranks (and therefore scoring) differs from the original PGI, where sensitivity is typically assessed using a so-called ‘blind’ version, in which ranks (patient concerns) are newly generated each time the PGI is completed (i.e. participants are not shown their previous responses). Although this ‘blind’ format does take account of the fluctuating nature of ‘quality of life’ (so participants can insert new domains on re-completion), responsiveness to change has been poor, likely because of the inconsistency in rated items (Witham et al., 2008). We wanted to introduce some experimental stability over what ranks/items are rated; thus when re-administering the scale, in say a treatment context, original ranks are inserted by the researcher/clinician and the patient is asked to re-rate them – a so-called ‘closed’ format (see figure 4.9). A further related idea was that in future studies these generated areas of individual-specific concerns could be inserted into a sleep diary and measured on a daily basis, in a similar manner to quantitative sleep parameters assessed throughout the intervention period.
Figure 4.8 - Copy of the Glasgow Sleep Impact Index (GSII).
Figure 4.9 - Follow-up ‘closed’ version of the GSII with patient example.

**PART 1: Areas**

Dear participant, below are the three most important things affected by your sleep, that you filled in and rated previously.

- **Concentration on activities (easily distracted)**
- **Efficiency at work (running out of energy)**
- **Social life (get tired too early)**

**PART 2: ratings**

Can you please now give each of these areas a score, depending upon how bothered you were by it, in the past two weeks. Place a vertical mark through the line (+) wherever your personal rating falls.

- Very bothered
- Not bothered

- Very bothered
- Not bothered

- Very bothered
- Not bothered
Based on the premise that quality of life impairment (above an individual personal threshold) drives treatment-seeking behaviour in those with insomnia, the ultimate aim of the GSII is to document a reduction in quality of life interference in parallel with clinically significant changes in sleep.

### 4.4.3. Results

#### 4.4.3.1. Participant demographics

The 39 primary insomnia patients that completed the DFSAS also completed the GSII at the same time (see table 4.2 for demographics).

Based on data from PI patients, we assessed the following properties of the GSII: content of generated domains/ranks; how VAS ratings and 'spend money' scores compared across the three most important generated life areas (within-subjects); concurrent validity with SF-36 dimensions and the Occupational Impact of Sleep Questionnaire (OISQ; David & Morgan, 2006); relationships with global measures of insomnia symptoms/sleep quality; and sensitivity to treatment outcome during a sleep restriction intervention.

#### 4.4.3.2. Rank content analysis (content validity)

The scale was completed appropriately by nearly all patients; missing data was small. Of the 39 patients involved in the three studies, three were not administered the scale by the relevant researcher, leaving 36 completed GSII questionnaires. Out of these 36, two patients failed to enter data for one of the ranks; hence, missing data was 2 (1.85%) out of a possible 108 rank entries.
We carried out a quantitative content analysis (Hsieh & Shannon, 2005) of the 106 remaining generated domains, across all participants and their three specified concerns. Each item was given a code; codes were subsequently grouped into global categories. The frequency of each category, reflected as a percentage of the total number of generated items, is reported in table 4.7. Although likely to be highly inter-related, the most commonly reported categories were: ‘occupational functioning’, ‘cognitive functioning’, ‘mood’, ‘energy’ and ‘social functioning’. There was no clear pattern in terms of generated domains and rank importance i.e. areas of functioning concern tended to be equally distributed across ranks 1, 2 and 3.

*Table 4.7 - Content domains from GSII rank content analysis.*

<table>
<thead>
<tr>
<th>Category</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupational Functioning</td>
<td>15.10%</td>
</tr>
<tr>
<td>Cognitive Functioning</td>
<td>15.10%</td>
</tr>
<tr>
<td>Energy</td>
<td>11.30%</td>
</tr>
<tr>
<td>Mood</td>
<td>11.30%</td>
</tr>
<tr>
<td>Social Functioning</td>
<td>8.50%</td>
</tr>
<tr>
<td>Motivation</td>
<td>7.50%</td>
</tr>
<tr>
<td>Outlook</td>
<td>6.60%</td>
</tr>
<tr>
<td>Family/Partner Relationships</td>
<td>6.60%</td>
</tr>
<tr>
<td>Health</td>
<td>6.60%</td>
</tr>
<tr>
<td>Tired/Fatigue</td>
<td>4.70%</td>
</tr>
<tr>
<td>Appearance</td>
<td>2.80%</td>
</tr>
<tr>
<td>Happiness</td>
<td>1.90%</td>
</tr>
<tr>
<td>Sleepy</td>
<td>1.90%</td>
</tr>
<tr>
<td>Anxiety</td>
<td>1.90%</td>
</tr>
<tr>
<td>Confidence/Self-esteem</td>
<td>1.90%</td>
</tr>
</tbody>
</table>
4.4.3.3. Relationship between ranks

Ranks and ratings were collapsed across all patients, creating global group mean VAS scores for ranks 1, 2, and 3. Lower VAS scores are indicative of greater perceived impairment in the particular area of concern. These mean scores were investigated using repeated-measures ANOVA, revealing a significant main effect of rank \( F(2, 62) = 18.44, p < .001 \). Post-hoc paired \( t \) tests indicated a graded trend across the ranks (see figure 4.10 and table 4.8), so that rank 1 scores were significantly lower than both ranks 2 \( t(31) = -2.89, p < .01 \) and 3 \( t(31) = -5.74, p < .001 \), and rank 2 scores were statistically lower than rank 3 values \( t(31) = -3.32, p < .01 \). This analysis essentially confirmed the ordering of generated rank importance.

*Figure 4.10* - Boxplots of VAS (mm) ratings for GSII ranks 1, 2 and 3, for all patients.
Scores on the ‘spend’ category further supported this pattern (table 4.8), with participants opting to place the highest ‘monetary’ value on eradicating rank 1 (£34.20, SD=14.6), then rank 2 (£16.50, SD=9.7) and finally, rank 3 (£9.35, SD=8.0).

Table 4.8 - VAS ratings (mm) and spend (£) for generated ranks for all patients.

<table>
<thead>
<tr>
<th>VAS score (mm)</th>
<th>Level of 'spend' (£)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rank 1</td>
<td>22.94 (12.2)</td>
</tr>
<tr>
<td>Rank 2</td>
<td>30.69 (16.1)</td>
</tr>
<tr>
<td>Rank 3</td>
<td>41.34 (19.2)</td>
</tr>
</tbody>
</table>

4.4.3.4. Concurrent validity

Relations between GSII rank scores and both SF-36 and OISQ scores, collected at baseline during the aforementioned sleep restriction study (n=18), were investigated using correlation analyses.

Rank 1 was found to be significantly associated with emotional role limitation (rho=.56, n=17, p<.05) and moderately associated with social functioning (rho=.41, n=17, p=.099).

Ranks 2 and 3 were both significantly (negatively) associated with occupational functioning as measured by the OISQ (r =-.59, n=16, p<.05; and rho=-.55, n=16, p<.05). Rank 2 was strongly associated with emotional role limitation (rho=.70, n=18, p<.01) and moderately associated with mental health (r =.43, n=18, p=.07) and social functioning (rho=.37, n=18 p=.13). Rank 3 was similarly positively associated with social functioning (rho=.59, n=18, p<.05), and moderately associated with emotional
role limitation ($\rho=.40, \ n=18, \ p=.10$), mental health ($\rho=.37, \ n=18, \ p=.13$), and vitality ($\rho=.37, \ n=18, \ p=.13$).

4.4.3.5. Relationship with sleep and treatment outcome sensitivity

Interestingly, ranks 1 and 2 were weakly and non-significantly associated with PSQI and ISI values. Rank 3, however, was negatively (moderately) associated with both PSQI ($r=-.40, \ n=32, \ p<.05$) and ISI ($r=-.42, \ n=32, \ p<.05$) values.

The GSII was completed over the course of the sleep restriction intervention, at baseline, post-treatment and 3 months follow-up. Repeated measures ANOVA revealed main effects of time for rank 1 [$F(2, 26) = 5.31, \ p<.05$] and rank 2 [$F(2, 28) = 3.56, \ p<.05$], but not rank 3 [$F(2, 28) = 0.92, \ p=.410$]. Post-hoc $t$ tests indicated that although mean group scores for ranks 1 and 2 increased (improved) from baseline to post-treatment, between-subject variability was high, and hence both failed to reach statistical significance. However, both ranks 1 and 2 achieved statistical significance at the three month assessment point relative to baseline values (see table 4.9).

Table 4.9 - VAS scores for GSII ranks collected over the course of intervention and at follow-up.

<table>
<thead>
<tr>
<th></th>
<th>Baseline M (SD)</th>
<th>Post-treatment M (SD)</th>
<th>Follow-up M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSII</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rank 1</td>
<td>34.14 (17.96)</td>
<td>53.14 (22.58)</td>
<td>58.00* (22.85)</td>
</tr>
<tr>
<td>Rank 2</td>
<td>39.47 (17.72)</td>
<td>49.93 (24.77)</td>
<td>57.20* (19.96)</td>
</tr>
<tr>
<td>Rank 3</td>
<td>50.87 (19.01)</td>
<td>54.60 (22.66)</td>
<td>58.80 (24.43)</td>
</tr>
</tbody>
</table>

* significant at $p <.05$, relative to baseline
Finally, those categorized as ‘responders’ to sleep restriction therapy, based on ISI values, were found to have significantly higher (improved) scores for ranks one \( [U = 13.50, z = -2.11, p < 0.05] \), two \( [U = 6.00, z = -2.81, p < 0.01] \) and three \( [U = 4.00, z = -2.92, p < 0.01] \), relative to non-responders (indicating less impairment; see table 4.10).

*Table 4.10 – Responder analysis for the GSII. Median (Mdn) values are presented with the interquartile range (IQR) in parentheses.*

<table>
<thead>
<tr>
<th></th>
<th>Responders</th>
<th>Non-Responders</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mdn</td>
<td>IQR</td>
</tr>
<tr>
<td>GSII</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rank 1</td>
<td>63.50</td>
<td>(33-73)</td>
</tr>
<tr>
<td>Rank 2</td>
<td>67.00</td>
<td>(51-78)</td>
</tr>
<tr>
<td>Rank 3</td>
<td>66.00</td>
<td>(59-78)</td>
</tr>
</tbody>
</table>
4.5. Discussion

In the present study, two new brief scales, the Glasgow Sleep Impact Index (GSII) and the Daytime Functioning and Sleep Attribution Scale (DFSAS), were developed to help refine measurement of insomnia-related impairment in both clinical practice and research settings (see table 4.11). The DFSAS was created to address the need for a scale that is based on commonly reported insomnia-symptom domains; can be completed by both patients and non-patients; and includes (as a separate section) some reference to poor sleep attributions in causing daytime impairment. The GSII, on the other hand, was created as an individualised measure for specifically targeting daytime and quality of life impairments reported by insomnia patients. This tool addresses the need for a scale that records items relevant to each patient in their own unique vocabulary, capturing individual meaning, relevance and importance.

Table 4.11 - Measurement needs and subsequent fulfilment by developed scales.

<table>
<thead>
<tr>
<th>Required scale characteristics for use with insomnia patients</th>
<th>Met by...</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Assessment of concerns relevant to each individual patient</td>
<td>GSII</td>
</tr>
<tr>
<td>2. A measure which permits ranking of concerns in order of personal importance</td>
<td>GSII</td>
</tr>
<tr>
<td>3. Some measure of the attribution for poor sleep in causing daytime impairment</td>
<td>GSII/DFSAS</td>
</tr>
<tr>
<td>4. Coverage of items that are commonly reported by insomnia patients as a group</td>
<td>DFSAS</td>
</tr>
<tr>
<td>5. Brief measure that can be used in both clinical and research settings</td>
<td>GSII/DFSAS</td>
</tr>
<tr>
<td>6. Can be completed by normal and poor sleepers with limited sleep-related priming</td>
<td>DFSAS</td>
</tr>
<tr>
<td>7. Can be completed by patients with additional health conditions</td>
<td>GSII/DFSAS</td>
</tr>
</tbody>
</table>

4.5.1. DFSAS

The DFSAS, based on common patient descriptors, was found to have high internal consistency, good discriminant validity, and, using a cut-off of 10 (part 1), was able to
correctly identify 87% of insomnia patients and 87% of normal sleepers. Furthermore, DFSAS (part 1) demonstrated fair concurrent validity; correlating moderately with SF-36 dimensions tapping vitality/energy, emotional well-being, social functioning and general health (domains that are typically the most sensitive to insomnia impairment and treatment; Kyle et al., 2010). Given the global coverage of symptoms in the DFSAS, these moderate correlations are not surprising; suggesting that our new scale, as predicted, is likely to be tapping into a more global daytime functioning construct, over and above isolated dimensions. Interestingly, the DFSAS was strongly related to the OISQ, accounting for approximately 58% of the variance in scores. The DFSAS items probe a range of daytime symptoms, including items relating to work productivity, concentration, memory, and fatigue, all of which are likely to be important for optimal workplace functioning.

A particular strength of the DFSAS is the ability to collect normative data on healthy normal sleepers, in a non-loaded fashion. This proved useful when assessing sensitivity to change in PI patients during the sleep restriction intervention, where improvements were found to be robust and graded over the three assessment points. Indeed, final 3 month follow-up mean scores (part 1) were only approximately 2 scale points higher than those observed for normal sleeping controls. Although a small uncontrolled trial, such data suggest that (1) a brief behavioural intervention can improve daytime functioning, and (2) these end-point scores are not markedly dissimilar from values indicative of normal functioning. Clearly, the DFSAS needs to be piloted in large randomised controlled trials of behavioural and pharmacotherapy interventions before any definite conclusions can be made.
Part 2 of the DFSAS provides an index of the attribution for poor sleep in accounting for experienced daytime impairment, recorded in part 1. Part 2 was found to have high internal consistency, good discriminant validity, good sensitivity and specificity, and, as predicted, was associated with magnitude of reported daytime impairment. This attributional approach to measurement of daytime impairment has yet to be fully explored, but one can envisage several important implications. For example, those scoring particularly high on questions relating to the subjective role of poor sleep in accounting for experienced daytime impairment may require focused cognitive therapy to challenge/alleviate maladaptive beliefs on the link between sleep and functioning (cf. Harvey, 2002). That is, those who have pronounced sleep-related cognitive distortions and maladaptive views on sleep, which may relate to the misperception of both subjective sleep and impact on daytime functioning (‘daytime misperception’; Orff et al., 2007), could be identified as a specific sub-group. It is interesting that the ‘non-responders’ to sleep restriction treatment (see table 4.5) have substantially higher scores on part 2 than responders, and also notably higher scores than baseline values for the whole group. These individuals, perhaps, may not be predicted to respond to a single behavioural intervention, instead requiring additional cognitive restructuring components to target sleep-daytime attributions (Harvey et al., 2007). Due to the small numbers available to assess treatment response, these findings are necessarily preliminary and require further investigation.

Secondly, DFSAS items probe everyday functional limitations, and are therefore likely to be relevant to other patient groups. It would be interesting to administer the scale to other patient groups and compare, firstly, the relative magnitude of everyday functional impairments, and secondly, the role of sleep in its perceived causation. This could
prove particularly interesting in disorders where sleep disturbance is prevalent (for example, depression, chronic pain, and cancer populations) to give a better understanding of the role of poor sleep in contributing to impairment among other disease-relevant contributors. The DFSAS provides a means by which to assess poor sleep attributions in accounting for impairment, and how these may change post-intervention.

4.5.2. GSII

The GSII, a patient-centred measure, was developed to quantify *insomnia-related quality of life impairment* at the individual level. That is, life domains considered important and salient by the individual, which are negatively affected by insomnia, are recorded and subsequently rated. By administering a ‘closed’ version (where originally generated items are inserted into a follow-up GSII), participants can re-rate their three originally generated ranks, permitting assessment of change over time.

The scale was well received by patients. Although not systematically recorded, conversations with patients suggested they enjoyed having their own input in the measurement process, which, according to some, ‘made sense’. Moreover, completion rates were high. This was important to document given the original PGI has often been poorly completed when not interview-administered (Ruta et al., 1994, 1999).

Content analysis of generated domains validated the presence of impairments cited in diagnostic classification manuals, such as difficulties with fatigue, energy, motivation, mood, and cognitive functioning, as well as more ‘downstream’ disruption, related to social, occupational, and relationship functioning (ICSD-2, 2005; DSM-IV, 1994).
Importantly though, additional categories of impairment emerged that have not been described or investigated in the literature, with perhaps the exception of some recent qualitative work (Carey et al., 2005; Kyle et al., in press). These included: happiness, outlook, confidence/self-esteem, and appearance. It is interesting that many of these additional categories represent a shift in focus away from specific symptoms; instead capturing highly personal and difficult-to-define constructs, which only have meaning and relevance for the individual patient. Overall, the breadth of generated domains underscores the pervasiveness of insomnia-related impairment, supporting earlier work using the SF-36 indicating that insomnia patients have comparable, but more wide-ranging, functional impairments than those with depression and congestive heart failure (Katz & McHorney, 2002).

Another emergent finding from this content analysis was that occupational functioning was the most frequently cited area impacted by poor sleep. This is perhaps not surprising given that work-life represents a large percentage of our waking activity. What is surprising, though, is the small amount of literature investigating the relationship between insomnia and workplace functioning. It is fair to say that the majority of published literature has assessed the relationship at a rather blunt, macro-level; typically in large epidemiological studies reporting on absenteeism rates, and (non-validated) measures of work productivity (e.g. Philip et al., 2006; Daley et al., 2009b). Encouragingly, the inclusion of the Work Limitations Questionnaire (WLQ; Lerner et al., 2001) in two recent large randomized trials of hypnotic medication for insomnia revealed that work-related impairments can undergo improvements in parallel with sleep (Walsh et al., 2007; Erman et al., 2008). The development of the OISQ by David & Morgan (2006) also looks a promising addition to the field, with a particular
focus on the impact of sleep quality on workplace functioning. Of interest, ranks 2 and 3 from the GSII were significantly (negatively) associated with scores on the OISQ, indicating that those with lower rank scores (reflective of greater impairment) also tended to experience greater occupational difficulties.

It is also worth mentioning that although ‘fatigue’ is regarded as the most commonly associated daytime symptom of insomnia (e.g. Lichstein et al., 1997; Riedel & Lichstein, 2000) no single participant actually used this term when describing insomnia-related impairments. ‘Tired’, ‘energy’ and ‘motivation’, often synonyms for fatigue, were used more frequently. Perhaps a seemingly trivial point, but daytime functioning scales, such as the Fatigue Severity Scale (a recommended and often-used measure in insomnia research), tend to be composed of items which include the term ‘fatigue’. This inclusion may subsequently fail to capture ‘fatigue’-related impairment in a meaningful way for patients. Such data parallel work in the pain literature by De Souza & Frank (2000). These authors interviewed 11 patients with chronic pain to gain ‘insider’ accounts of how pain is perceived and understood by those who have it. Interestingly, patients reported less than half of the pain descriptors cited in the widely used McGill Pain Questionnaire. This finding, coupled with our content analysis, reinforces the need for an understanding of, and appreciation for, personal semantics when describing health-related impairments. The distinction between ‘sleepiness’ and ‘fatigue’ at the beginning of the recently developed Flinders Fatigue Scale (Gradisar et al., 2007) indicates, to some extent, awareness of such important related issues in the field of sleep medicine.
Concurrent validity of the GSII was assessed through correlating ranks with SF-36 dimensions. Similar to the DFSAS, GSII ranks tended to be moderately associated with dimensions covering aspects of emotional well-being and social functioning. This is perhaps not surprising given that ‘social functioning’ and ‘mood’ were in the top five most frequently generated categories of the GSII. This could also suggest there is a strong affective component involved in rating insomnia-related quality of life impairment. Indeed, recent work with a large number ($n=160$) of primary insomnia patients indicates that higher levels of depression (as measured with the BDI) predicted impairments in three factors capturing fatigue, physical health and mental health (Beaulieu-Bonneau et al., 2007b).

Interestingly, from this study, levels of depression predicted impairments in these factors to a greater degree than both objective and subjective measures of sleep. This may, therefore, explain why in the present study standard self-report measures of sleep quality and global insomnia severity tended to correlate poorly with GSII generated ranks. Alternatively, the poor relationship between insomnia severity and the GSII may be explained by the nature of what patients are asked to report in the GSII. Participants are asked to write down the three most important areas of their lives affected by poor sleep. Given that each generated rank is salient, important and therefore relevant to each (untreated) patient, one would perhaps not expect these values to vary in a linear fashion with scores on a pre-determined measure of insomnia severity.

In a recent review, Krystal (2007) made the important point that “successful therapy should improve difficulties identified prior to initiating treatment” (page 69). He was in fact directing his comments towards clinicians within everyday clinical practice, where
quantitative measurement is not necessarily the arbiter of success. The GSII, however, could fulfil this very role in both clinical and research settings; eliciting patient-relevant concerns in a systematic fashion. Although requiring replication within a larger controlled study, preliminary data from the small number of PI patients who took part in the sleep restriction intervention reveals significant improvements in the two most importantly generated patient concerns at follow-up. Furthermore, when dividing the group into responders and non-responders (a more sensitive and appropriate analysis for patient-centred measures; Martin et al., 2007), based on global insomnia severity, it was found that responders had significantly higher (improved) scores for all three ranks compared with non-responders. Our modified format and collapsing of generated ranks, across the entire group, may account for the superior sensitivity and responsiveness of the GSII, relative to the often poor response rate reported in work with the original and revised PGI (Witham et al., 2008; Patel et al., 2003; Martin et al., 2007).

Finally, although not used for score derivation, the ‘spend’ section (part 4) of the GSII may be particularly useful within the clinical context. If, for example, a patient decides to ‘spend’ all, or most, of the allocated ‘money’ on getting rid of a specific problem (e.g. impairment in work performance), then the therapist/clinician may tailor the intervention accordingly. This may involve elements of cognitive restructuring to minimize distress, behavioural experiments to test out unhelpful beliefs, and the development of coping strategies aimed at reducing functional impact.

4.5.3. Limitations

This work has several limitations. Firstly, for scale development studies, the sample size was small. This is particularly important when reviewing our data on concurrent validity and within-group treatment effects, which are based on a small number of
participants. Concerning the sample of normal sleepers, in the analysis of DFSAS properties, it should be noted that they significantly differed in age compared to insomnia patients. Although this age discrepancy is often reported in insomnia research – as sleep disturbance becomes more prevalent with age – our convenient sampling of normal sleepers represents a limitation of the present study. Importantly, however, this discrepancy did not mediate the research findings, in that group effects remained robust after controlling for the influence of age. Future validation work with the DFSAS should recruit matched samples across the age range.

Moreover, both scales are yet to be assessed for test-retest reliability, restricting their immediate applicability. The DFSAS and GSII should be completed by a sub-set of individuals twice within a stable time-period. In this regard, it would also be interesting to assess the stability of generated quality of life impairments, which could be achieved by administering a ‘blind’ version of the GSII, where participants are asked to generate items on two separate occasions. Such information (‘index of rank change’) may provide important insights on the conceptualisation/re-conceptualisation of insomnia-related quality of life impairment over time.

4.5.4. Concluding remarks and future directions

Moul and colleagues (2004), in their review of self-report insomnia measures, state that “progress in insomnia research will depend in part on advances in questionnaire design” (p.194). Here, we provide preliminary data on two new questionnaires developed to assess both common and individual-specific impairments relevant to insomnia patients. Both have excellent face validity because they are, in different ways, based on the words of patents. One can envisage the DFSAS being used to (1) highlight the magnitude of impairment in those with insomnia relative to normal sleepers; (2)
‘flag-up’ those individuals who may have ‘daytime misperception’ and hence be responsive to a more daytime-focused intervention; and (3) track daytime functioning changes and poor sleep attributions over the course of treatment in those with insomnia as a primary disorder, as well as in those where sleep disturbance is a co-occurring phenomenon.

The GSII is likely to be useful for (1) the clinician working with individual patients, encouraging engagement in the therapeutic process, and the tailoring of interventions based on relevant concerns; and (2) in trials of insomnia treatment to fully capture, and be able to document changes in, concerns of each and every individual patient, yet at the same time permit group-level analysis.

What still needs to be determined, in larger validation and controlled intervention studies, are psychometric indices of reliability and responsiveness across different treatment modalities. Relationships between both scales and objective/subjective sleep parameters, as well measures of affect, should be investigated in future work. One final possibility, alluded to earlier in the chapter, is that patient-generated concerns from the GSII could be inserted into a prospective sleep diary and assessed over the course of treatment. In this way, daily fluctuations in both insomnia-related quality of life impairment and nightly sleep parameters could be tracked prospectively. A similar ecological momentary assessment methodology has been recently piloted in insomnia, though with pre-determined scale items (Levitt et al., 2004; Buysse et al., 2007). Sensitivity is likely to be increased by the inclusion of patient-specific concerns, adding another dimension to the assessment of insomnia-related outcomes.
Overall, this work presents a first step in considering alternative ways to capture and measure insomnia-related quality of life and daytime functioning impairments.
Chapter 5:

The Integration of Quantitative and Qualitative Methodologies to Investigate the Patient Experience of Sleep Restriction Therapy (SRT) for Insomnia
5.1. Abstract

Although clearly effective in improving subjective measures of sleep, several important aspects of cognitive behavioural therapy for insomnia remain poorly understood and under-researched. To investigate the patient experience of sleep restriction therapy (SRT) for insomnia we carried out a mixed-method study employing sleep and daytime functioning questionnaires, prospective qualitative audio-diaries, and post-treatment semi-structured interviews, to gain insights from a number of different but complementary perspectives. Eighteen individuals with primary insomnia participated in a four week sleep restriction therapy group treatment. Questionnaire measures indicated strong treatment effects concerning subjective sleep parameters and daytime functioning/HRQoL variables. Audio-diaries and interviews provided rich accounts of side-effects associated with acute sleep restriction implementation; changes to sleep parameters, daytime functioning, and perceptions of sleep; and general challenges surrounding treatment implementation and adherence. This work has important implications for the delivery of SRT, particularly concerning awareness of possible ‘adverse events’ and likely implementation challenges. Findings also pave the way for testable hypotheses concerning possible mechanisms of action involved in sleep restriction treatment. This study highlights the insights that can be achieved through a pragmatic mixed-method approach.
5.2. Introduction

Cognitive behavioural therapy for insomnia (CBT-I) is widely accepted as an effective intervention for improving insomnia symptoms. Moreover, CBT-I is a standard, recommended treatment (Chesson et al., 1999; Morgenthaler et al., 2006), and is commonly regarded as the treatment modality of choice (Espie & Kyle, 2009; see Riemann & Perlis, 2009 for a review of published meta-analyses). There still remains, however, a lack of available CBT-I and behavioural sleep medicine specialists to deliver appropriate treatment. Indeed, it is a common quip at scientific meetings that the only patients gaining access to CBT-I, are those enrolled in randomised trials (Espie, 2008). Ongoing work attempts to widen access to CBT-I, and behavioural sleep medicine more generally (Espie, 2009; Perlis & Smith, 2008).

Although improvements in sleep are commonly reported post CBT-I and up to 2 years follow-up, there is a paucity of work documenting changes in daytime parameters and health-related quality of life (HRQoL; Morin, 2004, Morin, 2003; Riemann & Perlis, 2009). One recent review paper indicates that only a handful of randomised trials have included a measure of HRQoL (Kyle et al., 2010; see chapter two of this thesis). Given that, by definition, insomnia is associated with disruption to patients’ daily lives (DSM-IV; ICSD-2; Kyle et al., in press; Carey et al. 2005), and that this complaint typically acts as the principal motivator for treatment-seeking behaviour (Stepanski et al., 1989; Morin et al., 2006a), the lack of measurement of daytime functioning parameters in trials is an important omission. Nevertheless, the small amount of data that does exist, tentatively suggests that behavioural therapy and pharmacotherapy can improve domains of HRQoL and daytime functioning (Kyle et al., 2010; Krystal, 2007), though much work is still to be done to determine reliable indices of change.
Equally remarkable, no study has asked patients about their experience of CBT-I. That is, similar to the dearth of qualitative data on the subjective experience of insomnia, there too remains a gap in the literature describing the patient perspective on treatment. Such fundamental work is long overdue, and has potentially important implications.

Firstly, although there are a number of plausible explanations as to how CBT-I exerts its therapeutic effect (Spielman et al., 1987b; Pigeon & Perlis, 2006; Morin, 1993; Morin & Espie, 2003; Edinger et al., 2008b; Edinger & Means, 2005), there are few experimental studies that shed light on candidate mechanisms. Tracking the subjective narrative account of treatment, longitudinally, may prove fruitful in confirming, as well as raising additional, factors associated with CBT-I response.

Secondly, it is often presumed that psychological therapies, in contrast to pharmacological interventions, are devoid of ‘side-effects’ (Nutt & Sharpe, 2008; Berk & Parker, 2009). This may be naïve, particularly in the context of the behavioural treatment of insomnia. Stimulus control and sleep restriction therapies can lead to significant (acute) decrements in total sleep time, and, therefore, possibly induce daytime dysfunction over and above baseline difficulties. Such impairments, if they exist, could be particularly pronounced in those with elevated levels of sleep misperception, and those who fail to ‘respond’ to sleep restriction after several days. The presence of ‘side-effects’ are yet to be described systematically, beyond anecdotal report (Spielman et al., 1987b; Hoelscher & Edinger, 1988; Greene, 2008). Fittingly, in their recent review, Riemann & Perlis (2009) asserted “the question of adverse events has not been properly addressed up to now in research on psychological/behavioural methods, possibly taken it for granted that no such risks exist…research on adverse
events for psychological treatments needs to be intensified” (p.213). The NIH state-of-the-science conference statement (2005) came to similar conclusions: ‘there is no evidence that such treatment produces adverse effects, but thus far, there has been little, if any, study of this possibility’. (p.1056)

If negative consequences of behavioural therapy do exist, they need to be documented, and their time course evaluated, to further alert practitioners and prospective patients prior to initiation of treatment. The lack of investigation of CBT-I side-effects can be contrasted with approaches in other areas of medicine, such as pharmaceuticals where there tends to be rigorous recording of ‘adverse events’ during active therapy. Adverse event profiles are then used as cautionary notes to inform patients when initiating treatment. This is also the case for sleep apnea patients undergoing continuous positive airway pressure (CPAP) therapy, where side-effects have been associated with drop-out rates and non-adherence during clinical trials (Haynes, 2005). The importance of monitoring side-effects in sleep apnea patients is further underlined in the Calgary Sleep Apnea Quality of Life Index (SAQLI; Flemons & Reimer, 1998), which contains a sub-scale specifically inquiring about CPAP adverse events. Such information may subsequently guide CPAP titration.

A third benefit of prospective qualitative monitoring may be a contribution to our understanding of adherence to behavioural instructions. To date, there is only a handful of published studies investigating adherence to stimulus control and/or sleep restriction, using a variety of adherence measures – from session attendance and therapist ratings, to sleep diary data. Nevertheless, this small literature indicates that: (1) consistency of bed/rising times, though not necessarily sleep reduction, may be predictive of outcome
(Riedel & Lichstein, 2001); (2) questionnaire ratings of retrospective global adherence moderately relate to outcome (Vincent et al., 2008); and (3) pre-treatment sleepiness, dysthymia, lower self-efficacy and perceived barriers to treatment engagement, are all associated with reduced adherence and implementation of behavioural guidelines (Vincent & Hameed, 2003; Vincent et al., 2008; Perlis et al., 2004; Bouchard et al., 2003). Crucially, although questionnaire data indicate that behavioural aspects of CBT-I strongly relate to outcome at one year follow-up, they remain among the least liked and used components of CBT-I (Harvey et al., 2002; Vincent & Lionberg, 2001). Qualitative data tracking patients’ implementation of behavioural instructions could shed light on factors relevant to therapy adherence, and potentially help refine the future delivery of CBT-I.

Fourthly, as already stated, post-interventional functioning/HRQoL has been inadequately investigated. Indeed, given the small and mixed literature on functional assessment and outcome, some authors have questioned whether improving sleep in those with insomnia will in fact modify self-reported daytime functioning (e.g. Means et al., 2000; Omvik et al., 2008; Horne, 2010). Explanations for this apparent incongruence include concerns with measurement (Kyle et al., 2010), and theoretical interpretations surrounding physiological arousal (e.g. Horne, 2010; Bonnet & Arand, 1997) and dysfunctional cognitive processes (e.g. Semler & Harvey, 2006; Espie et al., 2006; Orff et al., 2007). One potential way to shed light on sleep and functioning (treatment) relationships is to simply ask participants to describe, in their own words, the impact of insomnia treatment on domains of functioning.
Qualitative methodologies are increasingly being recognised for their ability to explore topics that are poorly dealt with using conventional quantitative tools. Some recent examples in medicine and health care include: understanding adherence/non-adherence to medical regimes (PLoS Medicine editors, 2007; Tolmie et al., 2003); experience of illness (e.g. chronic pain; Osborn & Smith, 1998); the phenomenology of emotional (blunting) side-effects of anti-depressants (Price et al., 2009); development and refinement of QoL measurement (Hawker, 2009); factors that prompt/delay patients in seeking medical advice (e.g. breast cancer patients; Burgess et al., 2001); and how to improve patient recruitment into clinical randomised controlled trials (Donovan et al., 2002).

Although slow to filter into sleep medicine, perhaps given the infancy of the field, qualitative work has recently appeared in the sleep apnea literature. For example, using case study methodology and phenomenographic analysis, Brostrom and colleagues (2008) tracked a single male patient (and partner) through OSA diagnosis and subsequent CPAP management, interviewing at four different time points - pre-CPAP, 2, 3, and 6-months post-treatment. Such data provided valuable insights into the impact of untreated OSA on sleep and daytime functioning; the treatment process and related issues of compliance and side-effects; and finally, quality of life and health-related improvements, post-intervention. How CPAP initiation was ‘negotiated’ in the patient-partner relationship was also an important emergent theme from their analysis. The same group also used qualitative methods to good effect in a recently published study focusing on CPAP adherence (Brostrom et al., in press). Semi-structured interviews (qualitative content analysis) with apnea patients provided important information on
inhibitory and facilitatory factors related to CPAP adherence, spanning biological, psychological and social domains.

In light of the aforementioned potential benefits of qualitative inquiry, we adopted a mixed-method design to improve understanding of the implementation, experience, and impact of a condensed sleep restriction intervention. Sleep restriction therapy (SRT) was chosen because of its strong relationship with outcome, relative ease of administration, and reliance on adherence to prescriptive guidelines. The treatment ‘journey’ was investigated using (1) audio-diaries, to capture in vivo proximal reflections, and (2) post-treatment face-to-face semi-structured interviews, to provide a global experiential account. In addition to exploring the subjective experience of sleep restriction, our design permitted the evaluation of self-reported changes in daytime functioning, forming a secondary aim. Although single-component sleep restriction (Friedman et al., 2000) and sleep compression (Lichstein et al., 2001b) interventions have proven successful in terms of modifying sleep parameters, comprehensive assessment of daytime functioning impairments is lacking. Thus, in keeping with a ‘stepped care’ model of CBT-I delivery (Espie, 2009), we also sought to evaluate whether our brief intervention could improve daytime as well as (predicted) sleep outcome variables.
5.3. Methods

5.3.1. Participants

Individuals meeting research diagnostic criteria (RDC; Edinger et al., 2004; DSM-IV, 1994) for primary insomnia (PI) were recruited into the study. Participants, therefore, reported difficulties with initiation and/or maintenance of sleep, lasting for at least a one-month period. Individuals were excluded if they had a co-morbid active psychiatric disorder, or medical disorder that was related to sleep disturbance (i.e. participants could have co-morbid medical ailments but they had to show temporal separation, and attributional independence, from the insomnia disorder). Additionally, in keeping with diagnostic criteria of insomnia as a 24-hour disorder, participants had to report at least one daytime impairment attributed to disturbed sleep (RDC; Edinger et al., 2004).

Participants were aged between 18 and 65, and recruited through two avenues: (1) those completing non-interventional University of Glasgow Sleep Centre (UGSC) projects; and (2) those responding to local and national adverts, seeking individuals with sleep disturbance to take part in UGSC research. Finally, those taking prescription sleep medication were accepted into the study only if they maintained a stable regime i.e. weekly intake was less than or equal to three days per week, and they had not undergone a modification to treatment strategy in the four weeks prior to treatment initiation.

The study was approved by Greater Glasgow & Clyde NHS local ethics committee.
5.3.2. Treatment protocol

Treatment was conducted in groups of three or four, and delivered by the researcher. Following a seven-day baseline period, treatment took place over four weeks, comprising two group sessions and two individualised phone conversations (see figure 5.1 for session time-line). Sleep restriction therapy content (see table 5.1) was based on Morin & Espie (2003) and the Glasgow Sleep Centre protocol for CBT (Espie et al., 2007; Espie et al., 2008). Specifically, calculated sleep window was based on the average total sleep time for the baseline period, and was positioned according to the individual schedule of each patient (accounting, also, for circadian factors). If the patient felt that their calculated window was unachievable, then there was some negotiation to try to facilitate adaptation and adherence.

The sleep window was titrated in the following ways: if sleep efficiency for the week was ≥ 90%, then the sleep window was increased by 15 minutes; if sleep efficiency was < 85 %, then the sleep window was decreased by 15 minutes; and if values fell between 85 and 89% there was no change to the schedule. Downward adjustments were not made until the end of the second week. The minimum possible sleep window duration was set at 5 hours.

Prior to attending session 1, participants were sent a seven-day sleep diary to complete each morning. They also completed baseline questionnaires (see below) covering aspects of sleep and daytime functioning. SRT was delivered using PowerPoint© presentation slides (Appendix F), and calculations were completed using handbooks and sleep-efficiency grids.
Figure 5.1 - Session time-line.

Baseline: 7-day diary

Session 1, Sleep Centre: Treatment rationale & instructions
(~ 45-60 minutes)
(keep audio-diaries)

Session 2, Sleep Centre: Review & planning
(~ 30-45 minutes)

Session 3: weekly phone call to monitor progress/titrade
(~ 5-10 minutes)

Session 4: weekly phone call to monitor progress/titrade
(~ 5-10 minutes)

Final meet, Sleep Centre: semi-structured interview & debriefing

3 month follow-up: complete questionnaire pack

Table 5.1 - SRT session content.

<table>
<thead>
<tr>
<th>Session</th>
<th>Content</th>
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<tbody>
<tr>
<td>Session 1</td>
<td>Participants attended the UGSC in the evening and received treatment rationale. Treatment covered the following areas: sleep need &amp; variability, sleep homeostasis &amp; timing, calculation of total sleep time, setting 'threshold' &amp; 'rising' times, sleep efficiency, motivation to change, and frequently asked questions in relation to treatment implementation.</td>
</tr>
<tr>
<td>Session 2</td>
<td>Participants again attended the UGSC. The purpose of this session was to review progress, calculate average sleep efficiency, and then set schedule for the forthcoming week.</td>
</tr>
<tr>
<td>Session 3 &amp; 4</td>
<td>Brief weekly phone calls to monitor/review weekly progress, and assist with sleep window modifications.</td>
</tr>
<tr>
<td>Session 5</td>
<td>Final meet at the UGSC to review progress, provide final instructions on how to continue to use SRT and titrate sleep window as required. Participants also took part in a semi-structured interview with the first author.</td>
</tr>
</tbody>
</table>
5.3.3. Screening instruments

All subjects underwent an initial screen using the Glasgow Sleep Centre brief screen protocol (see Appendix E). This records basic information on severity and frequency of insomnia symptoms, co-morbid medical/psychiatric/sleep difficulties, and medication use. Those satisfying the brief screen then received a thorough phone interview using the Glasgow Sleep Centre screening interview schedule, based on a template set out by Morin & Espie (2003; see Appendix A), to exclude those with affective/psychiatric disorder, those with sleep-disruptive medical co-morbidities, and to assess sleep pattern/symptoms to rule out occult sleep disorder pathology.

5.3.4. Outcome questionnaire measures

The following measures were completed at baseline, post-treatment (four weeks), and three-month follow-up. As well as constituting an outcome variable, sleep diaries were completed each day throughout the treatment phase to guide implementation and titration. See table 5.2 (p177) for a tabulated guide to the timing of measures and assessment points over the course of the intervention.

5.3.4.1. Sleep

Sleep Diary. The sleep diary used was based on a template set out by Espie (1991) and Morin & Espie (2003), assessing the previous night’s sleep parameters and sleep quality (see Appendix G). The main variables of sleep-onset latency (SOL), number of awakenings (NAW), wake time after sleep-onset (WASO), total sleep time (TST), sleep efficiency (SE), and subjective sleep quality (SQ) were extracted.
**Insomnia Severity Index** (ISI; Morin, 1993). The ISI is a seven-item scale assessing the severity of insomnia night-time symptoms and interference with daytime functioning, in the previous two weeks. Scores range between 0-28, and four clinical cut-offs have been identified: 0-7 (no insomnia problem); 8-14 (sub-clinical insomnia); 15-21 (clinical insomnia, moderate severity); and 22-28 (clinical insomnia, severe). The ISI has satisfactory internal consistency (Cronbach’s alpha = .76-.78; Bastien et al., 2001) and is a recommended measure for use in outcome research with insomnia populations (Buysse et al., 2006).

**Pittsburgh Sleep Quality Index** (PSQI; Buysse et al., 1989). The PSQI is a 19-item questionnaire that assesses sleep quality over the preceding month. The questionnaire yields a global score of ‘sleep quality’, in addition to seven individual component scores: subjective sleep quality; sleep latency; sleep duration; sleep efficiency; sleep disturbances; use of sleeping medication; and daytime dysfunction. The global score ranges from 0 to 21, with higher scores being indicative of poorer sleep quality. The PSQI has high internal consistency (Cronbach’s alpha = .85), and a score of greater than five achieves maximum sensitivity and specificity for insomnia (Backhaus et al., 2002).

**Glasgow Sleep Effort Scale** (GSES; Broomfield & Espie, 2005). The GSES is a 7-item scale (range 0-14) assessing the application of voluntary effort towards sleeping. Preliminary data indicates that it has satisfactory internal consistency (Cronbach’s alpha = .77) and reliably discriminates between normal sleepers and primary insomnia patients (Broomfield & Espie, 2005).
5.3.4.2. Daytime functioning

**Occupational Impact of Sleep Questionnaire** (OISQ; David & Morgan, 2006). The OISQ is a 24-item scale (range 0-96) assessing the impact of sleep quality on various aspects of work-related tasks and productivity (see Appendix H). The tool appears to discriminate between those with insomnia and good sleepers (Morgan & David, 2006), has high internal consistency (Cronbach’s alpha = .95), and has also been shown to correlate, positively, with global PSQI scores (Verster et al., 2008).

**Glasgow Sleep Impact Index** (GSII; Kyle et al., unpublished). The GSII was developed (see chapter four) to quantify insomnia-related impairments relevant to each individual patient. Participants write down in their own words the three most important aspects of their life impacted by poor sleep. These are subsequently ranked (1-3), rated on a 100mm visual analogue scale, and then assigned a sum of ‘money’ based on the patient’s desire to eradicate the problem (i.e. prioritise). A ‘closed’ version is administered at post-treatment and follow-up, where original generated items are inserted by the researcher/clinician to ensure stability in the scoring of identified ranks.

**Short-Form Health Survey 36** (SF-36; Ware & Sherbourne, 1992). The SF-36 is a generic health status tool, assessing functioning across eight core domains: physical functioning (PF), role-physical limitations (RP), mental health (MH), role-emotional limitations (RE), vitality and energy (VT), social functioning (SF), bodily pain (BP), and general health (GH) perception. Each dimension has a satisfactory to high level of internal consistency (Cronbach’s alpha = ≥.76; Jenkinson, Coulter & Wright, 1993). The SF-36 is recommended as an outcome measure in insomnia research (Buysse et al.,
2006), and appears sensitive to change post-behavioural and pharmacological intervention (e.g. Espie et al., 2006; Dixon et al., 2006; Walsh et al., 2007).

Daytime Functioning and Sleep Attribution Scale (DFSAS; Kyle, et al., unpublished). The DFSAS was developed (see chapter four) to assess impairments in a range of symptoms commonly associated with insomnia disorder (part 1). Part 2 of the scale asks participants to rate each item again, though this time in relation to how much poor sleep was responsible for the impairment reported in part 1 (poor sleep attribution). Preliminary data indicates good discriminant validity and high internal consistency (Cronbach’s alpha = .81 for part 1, and .89 for part 2).

5.4.3.3. Treatment-related process measures

Side-effects checklist and interference scale (unpublished). This scale asks patients to check, from a list, those symptoms experienced as a consequence of sleep restriction therapy (see Appendix I). Owing to the lack of an existing tool specific for CBT-I ‘adverse events’, we generated items spanning a mix of somatic, cognitive and emotional domains that may be perturbed by sleep deprivation and alterations to sleep timing. Checked symptoms are rated in terms of their interference with daytime functioning (0-4), following a similar format to existing published side-effects questionnaires used in pharmacological assessment (e.g. antidepressants; Uher et al., 2009). Additional space at the end of the scale also allows patients to qualitatively report domains of impairment not listed. The scale was completed during session 2, one week after the commencement of treatment.
Sleep Restriction Adherence Scale (SRAS; unpublished). The SRAS was created for the present study (see Appendix J), owing to the lack of available instruments to assess adherence to behavioural components of CBT-I. The scale is roughly based on the Medical Outcomes Study general adherence scale (MOS-A; Kravitz et al., 1993). Although previous work has modified this scale for use with insomnia patients (e.g. Vincent et al., 2008), we made further modifications to make it more relevant to sleep restriction therapy, and to probe adherence at a ‘local’ level. The 5-item SRAS assesses self-report global adherence, and adherence to set ‘threshold’ and ‘rising’ times on both weekends and weekdays (1-6 Likert item response format). The SRAS was completed at 3-months follow-up. Total adherence score can be calculated for items 2-5 (range: 4-24).

Table 5.2 - Time-line of assessment and data collection points.

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
<th>Post-treatment</th>
<th>Follow-up (3 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Diary</td>
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<td>Sleep Diary</td>
<td>Sleep Diary</td>
<td>Sleep Diary</td>
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<td>Sleep Diary</td>
</tr>
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<td>-</td>
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<td>ISI</td>
</tr>
<tr>
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<td>Audio-Diary</td>
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<td>-</td>
<td>PSQI</td>
<td>PSQI</td>
</tr>
<tr>
<td>GSES</td>
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<td>-</td>
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<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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<td>GSII</td>
</tr>
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<td>-</td>
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<td>-</td>
<td>OISQ</td>
<td>OISQ</td>
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<tr>
<td></td>
<td>Semi-structured</td>
<td>Interview</td>
<td>Sleep Diary</td>
<td></td>
<td></td>
<td>SRAS</td>
</tr>
</tbody>
</table>
5.3.5. Qualitative methodologies

5.3.5.1. Audio-diaries
Handheld dictaphones (Olympus© WS-200s) were utilised to track subjective experiences during the first week of treatment. Participants were asked to make two entries per day, according to open-ended semi-structured guidelines (see Appendix K). In brief, on awakening (within ~ 30 minutes), participants described their experience of implementing treatment instructions for the preceding sleep period. Similarly, in the evening, approximately 2 hours before going to bed, participants reflected on the course of the day and how their previous night’s sleep (with reference to their new schedule) affected their ability to function, for better or worse. Evening entries were completed sufficiently prior to the sleep-onset period to avoid creating excessive sleep preoccupation, or pre-sleep anxiety, proximal to sleep-initiation. Guidelines were purposely kept open to facilitate recording of relevant and interesting topics.

The study rationale was presented to participants as an investigation into the experience of sleep restriction. There was no priming in terms of what information we were particularly interested in; rather, it was emphasised that we wanted to know as much as possible about the experience of SRT implementation, from the individual perspective.

5.3.5.2. Semi-structured interviews
Four weeks after treatment initiation (session 5), participants were interviewed by the researcher on their overall experience of treatment. The interview format and schedule was based on published guidelines (Smith & Osborn, 2003) and consisted of open-ended fixed questions, with supplementary prompts (Appendix L) - again permitting
exploration of additional interesting and relevant topics. Core questions covered the following areas:
- understanding and expectations
- implementation of sleep schedule
- impact on sleep and daytime functioning

5.3.6. Data preparation and statistical analysis

5.3.6.1. Outcome variables

Dependent variables were screened for extreme outliers and assessed for normality using histograms and boxplots. Extreme outliers were replaced with the respective group mean (this was done for only two data points for two variables), and logarithmic transformations performed to correct skewed distributions. The trial aspect of the study was viewed as an intention-to-treat analysis; hence, the last observation carried forward (LOCF) method was used to impute missing data values. Imputation was almost exclusively confined to the three patients who were lost between post-treatment and follow-up assessment points.

Changes in sleep diary variables (SOL, NAW, WASO, TST, SE, SQ), sleep-related questionnaires (ISI, PSQI, GSES), and daytime functioning measures (GSII, DFSAS, SF-36, OISQ), across the three time points, were assessed using repeated measures ANOVA. Multivariate statistics (Wilks’ Lambda) are reported because they provide the most conservative way of interpreting factor effects when sphericity assumptions are violated (Pallant, 2007). Significant main effects were followed up using paired t-tests. Finally, correlational analyses were conducted to investigate relationships between changes in sleep and daytime functioning. Partial eta squared ($\eta^2$) and Cohen’s $d$
(Cohen, 1988) are reported to provide an indication of effect size (ES) magnitude for repeated measures ANOVA, and baseline to follow-up changes, respectively.

5.3.6.2. Audio-diaries & semi-structured interviews

Audio-diaries and interviews were transcribed verbatim, including pauses, laughter and false starts. Audio-diaries and interviews were analysed separately, and according to the framework of thematic analysis (Braun & Clarke, 2006). Thematic analysis is a flexible method for locating patterns (themes) within qualitative data sets, and is applied, to varying degrees, within major established analytic approaches (such as grounded theory and IPA). Recent structured guidelines (Braun & Clarke, 2006) establish thematic analysis as a flexible method in its own right, unconstrained by theoretical and epistemological underpinnings. Applications of this method in the medical and psychological-based literature include investigations of self-monitoring of blood glucose levels in type 2 diabetes (Peel et al., 2007) and the psychosocial consequences of developmental prosopagnosia (Yardley et al., 2008).

Analysis was a recursive process involving several stages (Braun & Clarke, 2006). First, transcripts were read through several times to gain a sense of the whole phenomenon under investigation. On subsequent readings of individual transcripts, significant words and/or phrases were highlighted, and bullet points entered in the margins, creating preliminary coding schemes. After several further reviews of the transcripts, initial notes and codes were collated into themes. Themes were compared across individuals, looking for common recurrent themes as well as inconsistencies and contradicting cases. Themes were refined throughout the analytic process via
discussions with another insomnia researcher (also a clinician) and from feedback during data presentation at lab seminars.

Given that the focus was on generating descriptions of the Sleep Restriction Therapy experience, emphasis was initially placed on identifying themes at the semantic or explicit level (Braun & Clarke, 2006). These were then subsequently related to, and interpreted in light of, contemporary literature on the behavioural management of insomnia, as well as additional questionnaire findings from the present study.
5.4. Results

5.4.1. Participant demographics

The intention was to run 6 treatment groups of 3-4 participants each, allocating one half to the qualitative component. Twenty three insomnia patients were initially enrolled into the study, but five failed to complete the treatment phase (see table 5.3 for participant demographics). Reasons for cessation included: falling pregnant ($n=1$); side-effects were too impairing ($n=2$); and, for the remaining two, reasons could not be ascertained.

Participant demographics for those completing all treatment sessions ($n=18$) are also presented in table 5.3. The mean age of the sample was 41.9 (13.2), with a range of 18-64 years. Five (28%) participants were male (13 female), and the average insomnia duration was 17 (14.4) years. Three participants had problems initiating sleep only, three had difficulties with maintaining sleep only, and the remaining 12 suffered from both initiation and maintenance difficulties. Finally, 2 participants (11%) were on prescribed sleep-promoting hypnotics at treatment intake. Additionally, three participants were lost to follow-up (3 months).

Table 5.3 – Participant demographics.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>PI patients enrolled ($n=23$)</th>
<th>PI patients completing treatment ($n=18$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>40.2 (13.0)</td>
<td>41.9 (13.2)</td>
</tr>
<tr>
<td>Gender</td>
<td>5 male / 18 Female</td>
<td>5 male / 13 female</td>
</tr>
<tr>
<td>Insomnia Duration (yrs)</td>
<td>14.9 (13.8)</td>
<td>17.0 (14.4)</td>
</tr>
<tr>
<td>Insomnia Sub-type:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initiating</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Maintaining</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Mixed</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>Medication</td>
<td>5/23 (22%)</td>
<td>2/18 (11%)</td>
</tr>
</tbody>
</table>
5.4.2. Sleep outcomes

Table 5.4 provides an indication of main effects of time, post-hoc comparisons between assessment points, and related statistical significance.

Sleep Onset Latency (SOL)

There was a significant change over time in mean subjective SOL. As is evident from table 5.4, mean SOL for the group decreased significantly by 21 minutes, from 41 minutes pre-treatment to 20 minutes at the post-treatment assessment. This improvement was sustained, and remained significant, at 3-month follow-up (ES=0.80).

Total Sleep Time (TST)

Subjective TST remained similar from baseline (320 minutes) to post-treatment (323 minutes). At 3 months, mean TST significantly improved, relative to baseline figures, by 47 minutes (ES=0.67).

Number of awakenings (NAW)

Number of nightly awakenings significantly decreased from baseline (2.97) to post-treatment (1.42). This effect was maintained at 3-months (M=1.69; ES=0.69).

Wake time after Sleep-Onset (WASO)

A significant effect of time was again found with mean WASO values. Mean WASO of 72 minutes at baseline significantly reduced by 43 minutes to 29 minutes at post-treatment. These improvements were sustained and remained significant at 3 month follow-up (ES=1.06).
Table 5.4 - Treatment effects and post-hoc comparisons for sleep diary and questionnaire variables.

<table>
<thead>
<tr>
<th>Diary</th>
<th>Baseline M (SD)</th>
<th>Post-treatment M (SD)</th>
<th>Follow-up (3 months) M (SD)</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOL (mins.)</td>
<td>41.12 30.63</td>
<td>19.70** 13.84</td>
<td>21.27** 17.30</td>
<td>(2, 16)</td>
<td>7.18</td>
<td>.006</td>
<td>0.473</td>
</tr>
<tr>
<td>TST (mins.)</td>
<td>319.79 75.31</td>
<td>323.07 48.80</td>
<td>367.08*** 65.56</td>
<td>(2, 16)</td>
<td>6.12</td>
<td>.011</td>
<td>0.433</td>
</tr>
<tr>
<td>NAW</td>
<td>2.97 2.20</td>
<td>1.42*** 1.18</td>
<td>1.69*** 1.44</td>
<td>(2, 16)</td>
<td>13.28</td>
<td>&lt;.001</td>
<td>0.624</td>
</tr>
<tr>
<td>WASO (mins.)</td>
<td>71.97 55.64</td>
<td>29.03*** 35.91</td>
<td>26.30*** 25.22</td>
<td>(2, 15)</td>
<td>10.94</td>
<td>.001</td>
<td>0.593</td>
</tr>
<tr>
<td>SE (%)</td>
<td>64.20 14.44</td>
<td>85.25*** 10.52</td>
<td>80.88*** 13.85</td>
<td>(2, 16)</td>
<td>19.15</td>
<td>&lt;.001</td>
<td>0.705</td>
</tr>
<tr>
<td>Sleep Quality (0-4)</td>
<td>1.42 0.63</td>
<td>2.13*** 0.65</td>
<td>2.23** 0.87</td>
<td>(2, 16)</td>
<td>11.69</td>
<td>.001</td>
<td>0.594</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Baseline</th>
<th>Post-treatment</th>
<th>Follow-up (3 months)</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISI</td>
<td>17.19 3.76</td>
<td>9.67*** 5.15</td>
<td>10.07*** 6.08</td>
<td>(2, 16)</td>
<td>16.00</td>
<td>&lt;.001</td>
<td>0.667</td>
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<tr>
<td>PSQI</td>
<td>12.63 2.96</td>
<td>7.97*** 2.02</td>
<td>7.82*** 3.36</td>
<td>(2, 15)</td>
<td>21.25</td>
<td>&lt;.001</td>
<td>0.739</td>
</tr>
<tr>
<td>GSES</td>
<td>9.28 3.27</td>
<td>5.31*** 2.52</td>
<td>5.61*** 3.48</td>
<td>(2, 16)</td>
<td>13.96</td>
<td>&lt;.001</td>
<td>0.636</td>
</tr>
</tbody>
</table>

Asterisks indicate post-hoc significant changes from Baseline: *p<.05, **p<.01, ***p<.001
a. contrast with post-treatment mean sig. at p<.05
Sleep efficiency (SE)

Mean SE significantly increased by an average of 21% from 65% at baseline to 86% post-treatment. Three month average SE (81%) values remained highly significant relative to pre-treatment assessment, with a net improvement of 17% (ES=1.18).

Sleep Quality (SQ)

Small but significant improvements were recorded for subjective sleep quality, from baseline to post-treatment. These benefits were statistically maintained at 3-month follow-up (ES=1.07).

Insomnia Severity Index (ISI)

Baseline mean ISI values (17.2) indicated the presence of clinical insomnia of moderate severity, according to scale cut-offs. Post-treatment reductions in ISI scores were significant (mean of 9.7 – ‘sub-clinical insomnia’), and were maintained at 3 month follow-up (ES=1.41). In terms of clinical significance, 6/18 (33%) participants scored in the ‘no insomnia’ range (‘remitters’) at post-treatment. The number of ‘remitters’ at follow-up increased to 8/18 (44%). Treatment ‘response’ rates were calculated based on recently published minimally important difference data for the ISI (Yang et al., 2009). The number of individuals evidencing a change of at least six ISI scale points at post-treatment was 66.6% (12/18). This rate of treatment response remained the same at follow-up (66.6%).

Pittsburgh Sleep Quality Index (PSQI)

Similar to the ISI, mean PSQI scores significantly decreased from baseline (13) to post-treatment (8). This improvement was also sustained at the three month assessment point (ES=1.52).
Glasgow Sleep Effort Scale (GSES)

Mean GSES score changes between baseline (9) and post-treatment (5) were highly significant. Again this pattern remained robust at three-month follow-up (ES=1.09).

5.4.3. Daytime functioning outcomes

Table 5.5 provides an indication of main effects of time, post-hoc comparisons between assessment points, and related statistical significance.

Daytime Functioning and Sleep Attribution Scale (DFSAS)

Mean scores on part one of the DFSAS significantly reduced from baseline (18) to post-treatment (12) – see table 5.5. Comparisons between post-treatment and 3-month follow-up indicated further reductions in associated daytime impairment, which again reached statistical significance, above and beyond post-treatment effects. Effect size magnitude for baseline to follow-up change was large (ES=1.42). Concerning part 2, the attributional component, repeated measures ANOVA indicated a main effect of time. Mean values significantly improved post-treatment, which were found to be most robust at follow-up (ES=0.99).

Glasgow Sleep Impact Index (GSII)

Both GSII ranks 1 and 2 indicated main effects of time. Post-hoc testing for rank 1 revealed significant effects at post-treatment and follow-up, with effects being most pronounced at final assessment (ES=1.21). Rank 2 similarly demonstrated improvements across the three assessments, though effects achieved significance only at the 3-month follow-up (ES=1.04). There was no within-subjects effect for rank 3, with mean values remaining quite stable over the assessment points.
Table 5.5 - Treatment effects and post-hoc comparisons for daytime functioning and HRQoL variables.

<table>
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<tr>
<th></th>
<th>Baseline</th>
<th>Post-treatment</th>
<th>Follow-up (3 months)</th>
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<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>9.94***a</td>
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<tr>
<td>Part 2</td>
<td>24.75</td>
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<td>18.94*</td>
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<td></td>
<td>14.39**</td>
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<td></td>
<td>(2, 16)</td>
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<tr>
<td></td>
<td>8.63</td>
<td>.003</td>
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<td></td>
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<tr>
<td>Rank 1</td>
<td>31.41</td>
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<td>SF-36</td>
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</tr>
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<td>VT</td>
<td>39.72</td>
<td>12.85</td>
<td>46.48*</td>
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<td>BP</td>
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<td>23.83</td>
<td>78.89</td>
</tr>
<tr>
<td></td>
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<td>26.86</td>
</tr>
<tr>
<td>GH</td>
<td>63.82</td>
<td>16.73</td>
<td>66.47</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>18.77</td>
</tr>
</tbody>
</table>

Asterisks indicate post-hoc significant changes from Baseline: *p < .05, **p < .01, ***p < .001

a. contrast with post-treatment mean sig. at p < .05

PF = physical functioning; SF = social functioning; RP = role-physical limitations; RE = role-emotional limitations; MH = mental health; VT = vitality/energy; BP = bodily pain; GH = general health perception.
**Occupational Impact of Sleep Questionnaire (OISQ)**

The OISQ was completed by 16 (89%) working participants in the study. Mean OISQ values significantly improved at post-treatment by, on average, 16 points. This effect remained robust and significant at three month follow-up (ES=0.74).

**Short-Form Health-Survey 36 (SF-36)**

Three dimensions of the SF-36, ‘vitality/energy’ (VT), ‘role-emotional limitations’ (RE) and ‘mental health’ (MH), evidenced main effects of time, with moderate effect sizes. Both RE (ES=0.48) and MH (ES=0.67) dimensions achieved statistical significance at 3 months (relative to baseline values). For the Vitality dimension, both post-treatment and 3 month values (ES=0.82) indicated significant improvements relative to baseline levels. There was no effect of time or trends for the other five dimensions.

**5.4.4. Relationship between sleep and daytime functioning changes**

To determine whether changes in daytime functioning were associated with improvements in sleep symptoms, correlation analyses were carried out on those variables demonstrating significant improvements over the course of the intervention period and follow-up. Change scores (baseline to follow-up) were calculated for each variable of interest, and standardised so that positive values indicated relative improvement on all variables. Correlation coefficients and an indication of significance level are presented in table 5.6.

Improvements in insomnia severity, as measured by the ISI, were positively and significantly associated with improvements in daytime functioning (see table 5.6), across several measures, including DFSAS (part 1), GSII rank 2 (see scatterplot, figure
5.2) and the vitality/energy dimension from the SF-36; all ≥ .67 in strength. Likewise, PSQI improvements were similarly associated with improvements in aspects of functioning; specifically scores on the GSII rank 2, DFSAS and Vitality (all > .74). Sleep diary variables of WASO, number of awakenings, and SOL tended to be weakly (and non-significantly) associated with daytime functioning improvements. TST, SE and Sleep Quality, however, tended to be strongly and significantly related to improvements in functional outcomes across the DFSAS, GSII ranks 1 and 2, the OISQ, and the vitality dimension from the SF-36.

Table 5.6 - Relationship between changes in daytime functioning variables and sleep variables (baseline to follow-up).

<table>
<thead>
<tr>
<th></th>
<th>ISI</th>
<th>PSQI</th>
<th>SOL</th>
<th>No. Awak.</th>
<th>WASO</th>
<th>TST</th>
<th>SE</th>
<th>SQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>DFSAS (1)</td>
<td>.831**</td>
<td>.855**</td>
<td>.011</td>
<td>-.026</td>
<td>.085</td>
<td>.595**</td>
<td>.578*</td>
<td>.494*</td>
</tr>
<tr>
<td>GSII rank 1</td>
<td>.475</td>
<td>.350</td>
<td>.361</td>
<td>.598*</td>
<td>-.218</td>
<td>.384</td>
<td>.392</td>
<td>.650*</td>
</tr>
<tr>
<td>GSII rank 2</td>
<td>.674**</td>
<td>.633**</td>
<td>.282</td>
<td>.232</td>
<td>.031</td>
<td>.576*</td>
<td>.655**</td>
<td>.643**</td>
</tr>
<tr>
<td>OISQ</td>
<td>.455</td>
<td>.418</td>
<td>.021</td>
<td>.066</td>
<td>.326</td>
<td>.604*</td>
<td>.357</td>
<td>.196</td>
</tr>
<tr>
<td>MH</td>
<td>.354</td>
<td>.586</td>
<td>.069</td>
<td>.054</td>
<td>.073</td>
<td>.288</td>
<td>.381</td>
<td>.422</td>
</tr>
<tr>
<td>VT</td>
<td>.715**</td>
<td>.748**</td>
<td>-.037</td>
<td>.150</td>
<td>.147</td>
<td>.643**</td>
<td>.589**</td>
<td>.669**</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
5.4.5. Treatment side-effects

As presented in table 5.7 and figure 5.3, ≥ 50% of participants reported sleep restriction-related side-effects, in eight out of the twelve listed domains. The three most commonly reported symptoms were ‘fatigue/exhaustion’ (100%), ‘extreme sleepiness’ (94%), and ‘reduced energy/motivation’ (89%). Subsequent ratings of the extent of ‘side-effect’ interference revealed that ‘fatigue/exhaustion’, ‘extreme sleepiness’, ‘feeling irritable’ and ‘changes in hunger/appetite’, interfered ‘somewhat’ to ‘much’ (mean ratings 2-3) with everyday functioning (table 5.7).
Table 5.7 - Side-effect frequency and interference for entire sample.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>% of sample reporting symptom</th>
<th>Daytime functioning interference rating (0-4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Mood</td>
<td>61</td>
<td>1.55</td>
</tr>
<tr>
<td>Fatigue/exhaustion</td>
<td>100</td>
<td>2.56</td>
</tr>
<tr>
<td>Extreme sleepiness</td>
<td>94</td>
<td>2.58</td>
</tr>
<tr>
<td>Feeling agitated</td>
<td>50</td>
<td>1.78</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>33</td>
<td>1.17</td>
</tr>
<tr>
<td>Headache/migraine</td>
<td>72</td>
<td>1.31</td>
</tr>
<tr>
<td>Euphoria/intense increase in mood</td>
<td>39</td>
<td>1.29</td>
</tr>
<tr>
<td>Reduced motivation/energy</td>
<td>89</td>
<td>1.88</td>
</tr>
<tr>
<td>Changes in hunger/appetite</td>
<td>72</td>
<td>2.00</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>22</td>
<td>1.00</td>
</tr>
<tr>
<td>Dizziness</td>
<td>28</td>
<td>1.40</td>
</tr>
<tr>
<td>Feeling irritable</td>
<td>61</td>
<td>2.09</td>
</tr>
</tbody>
</table>

Figure 5.3 - Graphical representation of % of sample reporting each listed side-effect.
Six (33%) participants added, qualitatively, additional domains of impairment. These spanned the categories of: pain/discomfort, temperature regulation, word-finding difficulties, social interaction impairment, illness, ‘hangover’-like effects, problems with concentration, and fatigue at unusual times.

**Figure 5.4** - Scatterplot of relationship between PSQI change scores and number of experienced side-effects.

![Figure 5.4](image)

Exploratory correlation analyses were carried out between number of experienced side-effects, relative daytime interference ratings for side-effects [i.e. (interference score for checked symptoms/total possible score for checked symptoms)*100], and sleep improvements (baseline to post-treatment). Interestingly, it was found that a higher frequency of side-effects was associated with a greater magnitude of change on the PSQI ($r = .51, n=17 \ p<.05$; see figure 5.4) and, to a lesser (non-significant) degree, sleep efficiency ($r = .44, n=18, \ p=.065$) and ISI ($r = .33, n=18, \ p=.185$) values. Other
measures of sleep continuity were weakly and non-significantly related to side-effect frequency. Greater relative daytime interference ratings were significantly related to both improvements in PSQI scores ($r = .54, n=17, p<.05$) and reductions in sleep effort, as measured by the GSES ($r = .48, n=18, p<.05$).

5.4.6. Self-report adherence

Fifteen (83.3%) participants completed the adherence questionnaire at 3-months follow-up. The sample mean for ‘global adherence’ to sleep restriction instructions, post-treatment phase, was 3.87 (1.41), reflecting adherence ‘a good bit of the time’ (range 1-6); see figure 5.5 for distribution of scores. Subsequent ratings of specific ‘threshold’ and ‘rising time’ adherence, across both weekdays and weekends, also indicated similar levels of adherence behaviour (see table 5.8); with mean ratings falling between the two categories of ‘some of the time’ and ‘a good bit of the time’.

Figure 5.5 - Histogram showing distribution of global adherence ratings.
Correlations between ‘total score’ adherence ratings and sleep diary/questionnaire parameters (in terms of change scores) were found to be small and non-significant.

Table 5.8 - Group mean scores for adherence to SRT guidelines on weekdays and weekends.

<table>
<thead>
<tr>
<th></th>
<th>Rising Time</th>
<th>Threshold Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weekdays</td>
<td>4.00 (1.71)</td>
<td>4.29 (1.44)</td>
</tr>
<tr>
<td>Weekends</td>
<td>3.43 (1.87)</td>
<td>3.86 (1.83)</td>
</tr>
</tbody>
</table>

5.4.7. Qualitative results

At the outset of the study, the intention was to recruit approximately 50% (~12) of the total sample into the qualitative component of the trial. Participants were initially selected based on random allotment, within each treatment group, but because of time constraints and recruitment difficulties all participants within the final few treatment groups were selected to take part. Hence, 14 (78%) participants (4 male; 10 female) completed both audio-diaries and interviews, which was slightly more than predicted from the outset, though within standard limits for qualitative research.

5.4.7.1. Audio-diaries

In week one of SRT, participants, in total, recorded 179 diary entries. Transcription produced ~ 29,100 words of data. Thorough analysis of both morning and evening entries revealed three major themes, each with respective sub-themes/categories (see table 5.9). Direct quotes are presented to support generated themes and pseudonyms are used to protect patient identities.
Table 5.9 - Major themes from thematic analysis of audio-diary entries.

<table>
<thead>
<tr>
<th>Theme</th>
<th>Sub-themes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime side-effects: ‘it’s made it worse’</td>
<td>On awakening</td>
</tr>
<tr>
<td></td>
<td>Throughout the course of the day</td>
</tr>
<tr>
<td>Adjustment to new sleep schedule</td>
<td>Challenges to adherence</td>
</tr>
<tr>
<td></td>
<td>Coping strategies to help facilitate adherence</td>
</tr>
<tr>
<td>Evolving changes to the sleep experience</td>
<td>‘Unusual’ feelings of sleep pressure</td>
</tr>
<tr>
<td></td>
<td>Changes to sleep quality: could this be working?</td>
</tr>
</tbody>
</table>

**Daytime side-effects: ‘it’s made it worse’**

Participants described, on the whole, feeling and functioning worse during week one of therapy, relative to pre-treatment. A reduction in sleep opportunity typically translated into less total sleep time, which had implications for the remainder of the day. Although impairment perhaps indicated that the therapy instructions were being followed and possibly beginning to work - in the sense that sleep pressure was being applied - this was clearly not without consequences. Indeed, these consequences were conveyed on awakening, proximal to the sleep-period, with common references to ‘zombie’ or ‘hangover’-like states:

“...I feel like I’m drunk at the moment, my head’s quite swimming, and em not thinking very straight at all, I find it quite hard to write in this bit of paper too.” [Bill, 68-70]

“I feel really groggy, in fact...I can hardly concentrate just now...got quite a sore head, em, pain in the back of my head just now, from [being] really tired...” [Jim, 41-42]
“In terms of sort of quality of sleep, em I actually feel this morning as if I’ve got a hangover, and I didn’t drink anything last night, em I’ve got a headache and actually I feel quite sick.” [Jennifer, 23-25]

Sleep restriction therapy negatively impacted on numerous daytime functioning domains. On an elementary level, participants reported feeling exhausted and fatigued, experiencing difficulties with concentration and memory, as well as depressed mood. These symptoms, combined, had the net effect of impairing aspects of job performance and ability/effort to interact socially. Furthermore, 5/14 patients (36%) made reference to impaired driving abilities at least once during the course of the week.

“Hi, it’s Sunday night, about ten o’clock and I’m absolutely exhausted, I’ve had a really bad day and never left the house the whole day, just felt so bad, em, I don’t know if I can stay up till one o’clock…I just feel totally, at the moment, terrible” [Gillian, 58-60]

“I had about four hours, four hours thirty minutes last night, and I felt the thing that affected me today was my memory, my job entails a lot of analysis of patient speech on the spot, which I was able to do, but often I have to back up what I see and explain it to the families, and I couldn’t remember the specific example of what the patient was saying to back up my hypothesis of their diagnosis.” [Hannah, 220-224]

“Woke up bright and breezy, half six, Tuesday morning, raring to go, got into the car…and within twenty minutes I was absolutely exhausted, so bad that I swear I was nearly falling asleep all the way to work…it was torture, I was cross-eyed, em, eyes drooping, driving…” [Sarah, 122-126]
“The restrictive programme has affected my ability to function, it’s made it worse for me working, it’s been very hard at work to focus and be as sharp as you should be” [Bill, 187-188]

Adjustment to new sleep schedule

This theme captured the practical experience of implementing set threshold and rising times. Participants reported a number of issues that made adhering (rigorously) to the programme incredibly challenging, and which frequently led to non-adherence. Such factors included: spending extended amounts of time on own, particularly at weekends; running out of activities to do during extra hours (boredom); inability to stave off sleepiness until set bed time; fear of disrupting partner by entering the bedroom after they had initiated sleep; staying out late, drinking and socialising; feeling pressure to ‘perform’ when going to bed so late, and having a short period of time to obtain adequate sleep; and, finally, no indication of re-bound sleep over several nights of adherence, in parallel with accumulating daytime difficulties.

“When I went to bed last night I was conscious of thinking ‘oh god, you’ve got to be up in five hours’ and that felt like a bit of pressure and I think it took me just a little bit longer to sleep because of that.’ [Sarah, 51-53]

“I’m sitting at my computer and I’m listening to some nice quiet, soothing music. To try and keep me awake, AC/DC is going on, hopefully that will do the trick, keep me going for another hour or so, sit and ponder life, quite lonely because you don’t have anyone with you, even the dog’s went to his bed.” [Bill, 256-259]
“...this regime would be quite easy to do if you didn’t have any social life, em, as soon as you’re going out late, or drinking, or whatever, it does sort of seem to go to pot a bit…I’ll certainly restart it again tonight…I think that’s the difficulty come the weekend, it all goes a bit haywire, so whether that’s undoing any good that it’s done through the week, em, I possibly would think so…” [Jennifer, 196-198]

“Em, just talking about the experience of implementing my new bedtime routine, I feel it’s just been really negative so far, and although I can understand that it probably will work eventually, at the moment I just feel really bad…I’ve had about an hour and a half to two hours sleep last night, and just feel really bad today, and already just worrying about how on earth I’m gonna stay up till one o’clock this morning…” [Gillian, 65-71]

A sub-theme captured strategies that patients put in place to facilitate adjustment and promote adherence to sleep restriction instructions. These again were numerous and varied, but included the following: sleeping in a separate room from partner; partner staying up late to accompany and motivate the participant to adhere; scheduling activities or making modifications to activity levels to promote alertness, and to fend off sleep prior to set threshold time; reducing alcohol intake and late nights out; and seeking out others experiences of sleep restriction via the internet for additional reassurance/motivation.
“I made a point of staying with a friend to keep me awake for a while tonight...they’re off work...it’s made me stay awake and not be tempted to go to bed earlier than I should do.” [Jim, 55-57]

“ It’s only when you stop you realise you’re tired, so if you’re having a dance or whatever then you feel fine, sitting talking to someone I suppose it’s fine, it’s only when you stop talking and you just sit there quietly, eh that’s when it, that’s when you really feel tired, as long as I’m doing something, I’m okay, but just sitting would be a ‘no-no’ right now (laughs)” [Bill, 224-230]

“ I’ve watched more television in the last five days than I’ve watched in years at this time of night, however, I think that’s what keeps me awake” [Maria, 71-72]

“ I put more lights on than I normally would cos normally I would have it sort of quite dark in the lounge but em I reckon more light is probably a good thing, just to keep me awake - and I think it’s working” [Sarah, 39-41]

“from about 6 o’clock to 8.15, which is now, I’ve been really tired, like fighting the tiredness, I sat down on the couch…and I think my couch is going to have to be a no-go zone area because I get too comfy and I just want to nap. I closed my eyes for a second when I was watching the news, but it was literally just a second and I was awake and got a fright…I thought ‘I really can’t nap’…so I’m going to do a few chores to keep myself up…” [Hannah, 48-54]
“...I did do a bit of checking late at night last night on the internet for other people who have done this sleep restriction thing, and most of them seem to think it was worth persevering (laughs), some said it only took a couple of nights till they slept for the time they were allowed to be in bed, at least one said it took longer than that, so I’m hopeful that I’m not the only person who takes so long to sleep the amount of time I’m allowed” [Jane, 80-84]

Evolving changes to the sleep experience

Finally, participants reflected on how sleep quality, feelings of sleep pressure, and views on sleep, were beginning to change. These comments were made, in nearly all cases, towards the end of the first week as improvements in sleep became apparent. Changes in sleep pressure were conveyed with reference to the perceived ability to sleep longer than usual (past set rising time), and, in particular, ‘unusual’ feelings of ‘tiredness’ and involuntary napping.

“didn’t do much on Saturday, went to the cinema, and was really tired and thought I was going to fall asleep for the first time ever, half-way through, em a movie...went out for a walk and came back in again, and I was absolutely fine” [Lisa, 81-83]

“this afternoon I was very dosey and sort of quite tired em because I had sort of struggled a little bit with sleep last night, em, I got about three and a half hours all in, and em, this morning found it quite hard to get up...which [is] a little bit unusual for me” [Jim, 46-48]
“I got really tired about three o’clock but not the usual tired that I get - just can’t be bothered doing anything - but more I could fall asleep at my desk and my eyes really gritty tired” [Lisa, 49-50]

“these napping situations, it’s not like I’m sitting down wanting to nap but it’s just that I’m tired and I’m watching the telly and I’m drifting off and that’s so unlike me” [Hannah, 118-119]

“I’ve been sort of fighting myself to stay awake, eh for most of the day” [Jim, 88-89]

This pressure was linked to self-reported improvements in sleep, particularly reduced sleep latencies and less frequent and lengthy awakenings. It was thus towards the end of week one that patients were beginning to realise that the programme may actually be working; that the acute exhaustion and increased wake time may be a worthwhile endeavour.

“Sunday morning, I think it’s day 4, and eh, feel quite good, quite a good sleep last night, but eh I knew I would because I was so tired yesterday, em, so that’s a good sign, pretty much slept right through, and eh, yeah, feeling quite good” [David, 46-48]

“It’s eh Friday morning, I managed to sleep right through the night last night, didn’t wake up at all, and woke up this morning, before the alarm for the first time this week, so I feel a lot brighter and don’t feel as groggy or kind of spaced out this morning” [Lisa, 54-56]
“I’ve got to admit, despite being up twice during the night, I’m having a deeper sleep than I normally get and that’s been the same for the last couple of nights, although it’s shorter, it’s actually deeper” [Maria, 60-62]

“I do feel that the quality of my sleep last night was better than before I started the programme” [Sarah, 142-143]

“I’m feeling quite good today, and I had a good sleep last night, em, I didn’t wake up at all during the night, and I went out like a light at twelve thirty which was very unusual for me, but I think it was because the whole night I was craving sleep” [Hannah, 144-145]

“I still feel sleep deprived, em, and every time I walk past my bedroom, I just look longingly at my bed, I just want to be in there, this is ridiculous, Saturday morning watching breakfast news, before seven o clock, is there any need? Eh, anyway, all in all, if only I woke up once, it’s maybe not that bad” [Sarah, 53-56]

“..had a pretty good night, again woke up a couple of times, as per usual, but, you know, felt as if perhaps the quality of my sleep is better than it used to be.” [Sarah, 157-158]

“I did have a good sleep last night, but I still took a...nearly an hour to get to sleep, but I wasn’t really, I wasn’t lying there fretting or anything, it’s almost like being semi-comatose, em and then I woke twice, em and didn’t go to the bathroom which was
really good, and then I woke up finally at em seven o’ clock, I reckon it was pretty good sleep” [Louise, 48-53]

“In general I just feel I’ve been a lot [more] tired...a lot more tired this week than usual and could probably have slept more than what I’ve restricted myself to em despite em oversleeping twice, em, fingers crossed that’s a good sign” [Jim, 129-131]

5.4.7.2. Interviews

The same 14 participants also took part in a face-to-face interview with the researcher at the post-treatment assessment point. Verbatim transcription produced ~ 66,400 words of data. Following strict coding, a number of themes were generated, capturing impressions and experiences of sleep restriction therapy (see table 5.10 for a summary of main themes).

Table 5.10 - Major themes from thematic analysis of semi-structured interviews.

<table>
<thead>
<tr>
<th>Themes</th>
</tr>
</thead>
<tbody>
<tr>
<td>‘At the end of my tether’</td>
</tr>
<tr>
<td>‘This is a sleep restriction programme’</td>
</tr>
<tr>
<td>Adherence &amp; adjustment</td>
</tr>
<tr>
<td>‘I actually want to go to bed now’</td>
</tr>
<tr>
<td>Daytime functioning: a thermometer for success?</td>
</tr>
</tbody>
</table>
'At the end of my tether’

Participants discussed why they had decided to take part in the sleep restriction intervention; descriptions tended to be dominated with references to daytime and quality of life impairments, as key catalysts.

“…just my mood and em kinda temperament and it affected my everyday life, I thought, quite badly…and I thought things could be better if my sleep was better, and it seems to be working.” [Ross, 6-7]

“I got it into my head that I was a bad sleeper, and I thought ‘oh god, I’m still really young, I have to make a change or else this is going to be me’, and I don’t want this to be me…I had a wee bit of a fright about a few months ago, I didn’t fall asleep at the wheel, I would say I did, but it was almost too quick…I was just really tired, and I remember the music was blasting, window was down, and I must have felt drowsy, and then for a second my head was down and then I woke up, and I touched the kerb with the car, and I think I got a fright and I just thought I have to do something about this” [Hannah, 17-20/24-27]

“…because I was at the end of my tether, em, I felt that it was interfering in not just my personal / social life, but it was also interfering in work life” [Sarah, 11-13]

Related to this, it emerged that patients had tried a range of other treatments, including medication/herbal strategies; that had, for the most part, failed to effectively alleviate insomnia symptoms.
“I’ve tried herbalism…yeah just trying herbs and things, potions, but as I say I think it’s a whole lot of rubbish…somebody told me it would work, it worked for them, so you go and try it, it didn’t work.” [Bill, 41-43]

“I’ve only ever tried sleeping pills, and obviously sort of watching the diet and stuff like that, and eh people have recommended sort of various herbal medicines, which I… kinda had never worked for me, nothing else has worked for me, at all really.” [David, 26-28]

“I’d tried herbal medicine, I tried Chinese medicine, I tried… (laughs) which was sort of boiling up a lot of sticks, I’ve tried, (laughs)…tasted absolutely disgusting, and it were no good at all…I tried homeopathy…I mean I looked up sort of you know, homeopathic doctors, I tried herbal stuff, I have tried, I don’t really, I mean I have some sleeping pills but I don’t think they are very successful, I remember years ago having temazepam which I reckon were pet pills, I mean I don’t sleep at all with those…” [Jane, 23-28]

‘This is a sleep restriction programme’

Initial subjective impressions of SRT were that it was ‘logical’, and ‘made sense’; yet, for some, it did still feel ‘counter-intuitive’, relative to how they have typically tried to cope with their insomnia (i.e. extending time in bed/’catch-up’ sleep). Others, although again finding it logical, thought it seemed ‘too simple’ to be effective (‘insomnia is a chronic problem’). This perceived ease or simplicity failed to translate into actual experience, as the first 1-2 weeks of the programme were described as incredibly difficult, and, by some, as ‘hell’ and even ‘torture’. As noted in audio-diary entries,
participants similarly reflected on the negative side-effects encountered in the early stages of the programme, particularly relating to extreme fatigue and sleepiness, impairing nearly all aspects of daytime functioning, including subjective driving ability. These impairments were of greater magnitude than pre-treatment functioning levels.

“…telling somebody with insomnia ‘stay up late’ is like turkey’s voting for Christmas, don’t be ridiculous” [Sarah, 432-433]

“…the sort of limited amount of sleep that you were giving us in the first night sounded pretty horrific, and it was, the first week was really tough, eh, but I think I could see the sense then, but I didn’t see it immediately you know, it didn’t hit me immediately that it was going to work, that sort of came in the second week’ [David, 16-19]

“as the week went on you realise it was quite a task and it was quite an inconvenience to people, it’s got other issues attached to it I guess, and by the time you’ve done a week then you realise, eh, it is a sleep restriction programme.” [Bill, 63-65]

“ the first few days, I remember, I was on a real buzz about it, because I thought this is really going to work, I’m loving it, and I just, I got out of bed with a spring in my step, and then about the third day, I was just like ‘oh my god, this is hell’, I want more sleep, and it was by that point that I would’ve wanted to break the rules, and sleep in a wee bit or go for a nap.” [Hannah, 89-91]

“it was torture, like one of these reality TV programmes when they sort of torture people [laughs], it was absolutely awful because I knew I had a lot of things on that
week, work wise...and various things that I wanted to do...I knew I was going to be completely shattered when I was doing them, and I was, basically." [David, 39-39]

“...that week one needs to come with a health warning” [Sarah, 154]

“It’s depriving yourself of sleep at night and being constant during the day; it’s quite a hard cycle” [Maria, 271-272]

“driving was a nightmare, and I’ve never ever had an issue with driving before” [Bill, 84]

Adherence and adjustment

Adherence to set rising and threshold times was affected by a number of variables. These included, but were not restricted to, the following: experiencing pressure to sleep in such a condensed period, coupled with concern for next-day-functioning; external fluctuating stressors and commitments; actual felt impact of restricted sleep opportunity on daytime functioning; and boredom associated with extra hours of wakefulness. Weekend adherence was particularly difficult for participants, being adversely impacted by: socialising/alcohol consumption; the prospect of being alone and awake for such a lengthy period of time; and the awareness of returning to work at the beginning of the week (catastrophizing about possible daytime consequences and coping).

“if you don’t sleep and you know it takes you a while to get to sleep, you’re just concerned ‘oh my god, I’ve only got like three hours left, I better get to sleep’, you know, this type of thing, whereas if you had that extra hour that’s maybe an hour for
you to chill out, get to sleep type thing, and then the rest is for sleeping…” [Jennifer, 327-330]

“I suppose rationally and logically, it did seem a good idea, but then I think the first week of actually doing it, I felt worse, and so you kinda, there’s a temptation there to think ‘och this is not working’, like ‘give up now’” [Lisa, 17-19]

“…some nights when I was really just so tired, I went to bed earlier, or if I knew I had to do something the next day…I was slightly concerned, when I was really tired, about driving, I really did feel that driving was a danger” [Jane, 145-148]

Despite these obstacles, participants developed strategies to help promote adherence, such as refraining from going out/socialising late at night; not allowing self to relax in comfortable positions prior to ‘threshold time’ (to prevent ‘dosing’/napping); keeping active and setting chores; and negotiations/discussions with partner to facilitate adjustment.

“my husband was determined I was sticking at it, you know, cos he, when I was sitting at night-time, and I was like almost falling asleep…he kept shouting at me ‘get up’, ‘wake up, don’t go to sleep’, you know it was kinda like that, but I felt it very difficult to get up and do anything, I was actually too tired to” [Gillian, 135-138]

“a couple of parties and things… I was still aware that I wasny gonna get too drunk…or stay up too late, cos I wanted to kinda keep my sleeping pattern, cos I didn’t
want to break it, because it was beginning to kinda, it was beginning to kinda form, so I didn't really want to start messing about with it” [Ross, 45-49]

Another factor that emerged was that non-adherence (mostly sleeping-in and napping) contributed to sleep-onset problems the subsequent evening, when attempting to ‘restart’ the programme. This ‘experimental feed-back’ acted as a negative reinforcer, helping to reduce non-adherence and motivate continued implementation of treatment instructions.

“…there were times where I napped and then regretted it because it had an effect” [Hannah, 95]

“…there’s been a few mornings where you’re em, oh you’re thinking, especially like Saturday and Sunday, ‘there’s just no way I want to get out of my bed’, but em, then you think ‘well no’, especially at the weekend cos then if I lie long this morning then I don’t get to sleep tonight, it’s just going to start the whole thing again, that’s, I suppose, the motivation, the fact that you think ‘well if this makes you sleep five hours through the night, then just get up’” [Lisa, 273-278]

‘I actually want to go to bed now’

For the majority of participants significant changes to sleep tended to occur in the second or third week of treatment. These related to commonly-measured sleep parameters, such as reduced sleep latency, decreased wake-time after sleep-onset, and decreased number of awakenings. Changes to sleep, however, also extended to more ‘subjectively’ expressed aspects of sleep, such as quality and depth of sleep,
predictability that sleep will happen, and unusual feelings of sleep pressure and ‘craving’. Interestingly, for many participants, this ‘paradigm shift’ to ‘looking forward’ to going to bed reduced sleep anxiety/distress and pre-occupation, both at sleep-onset and during awakenings.

“I’m sleeping longer, and going into a deeper sleep, I think, when I wake during the night it’s only…it’s less frequent, and it’s easier to get back over again...previously if I woke up during the night I’d be worried about trying to get back over again, and I’d be thinking about it, but because you’re so tired by this sort of programme then you actually get back over much quicker and it seems to work” [David, 62-66]

“...now, with the light off, I compose myself for sleep and then I’ll say to myself ‘right, I’ll give myself a wee mindless job to do in my head, right think this or something’, and I find it difficult to concentrate on it which is quite good because that’s, you know, it shows that I’m not really able to...I’m not really fully conscious, but before I used to find myself anxiously looking at the clock to see what time it was” [Louise, 138-142]

“...I feel a lot more confident like, and my sleeping like...I don’t worry about ‘am I going to get to sleep’, I just go to bed and pretty much hope for the best, I’m not always looking at the clock and stuff.” [Becky, 122-124]

“I just, I suppose I feel a bit of a weight’s taken off my shoulder, em, I almost feel liberated from the constant thinking off ‘am I going to sleep?’ ” [Sarah, 406-407]
“I would say number one is that I actually want to go to bed now, which is really good for me, before it was something like a chore that I felt like I had to do, or never got enough of, and now I look forward to bed which is a completely new experience for me” [Hannah, 124-126]

“I think I’ve altered my, I just seem to have gone onto a different plain when it comes to my attitude to sleep, em, another thing though I’ve realised is that em it’s possibly true that I just don’t need 8 hours of sleep or even seven and a half hours sleep or even seven, possibly I only need about six and a half hours sleep.” [Louise, 78-81]

“you’re so knocked that you don’t have the anxiety to be anxious at that time of night, really, I’m so looking forward to going to my bed, when I get to whichever hour my times up, I say ‘Yes, times up, going to bed’ [laughs] so that’s probably the highlight of the day” [Bill, 167-170]

“I’m not concerned, I’m not worried about not sleeping, because I know that when I go to bed I will sleep” [Sarah, 188-189]

“.normally if I’ve got something important the next day, then that’s the sort of thing that would keep me awake, but as I say I’m so tired by the time it gets to quarter past twelve or whatever, then em I don’t even worry about the following day, I know I’m going to sleep” [David, 114-118]
“it’s a different kind of tiredness it’s more of a…there’s periods during the day when you think I could put my head down on the desk and fall asleep, whereas before it was go away and leave me alone and just don’t annoy me” [Lisa, 298-299]

Indeed, even for those few participants who felt they hadn’t benefited from the sleep restriction programme, they did, however, describe changes to their attitude towards sleep, particularly sleep need. Having encountered side-effects during restriction, they tended to have a ‘response-shift’ to their own sleep duration; engendering ‘this could be worse’ phenomenon, which seemed to relieve some pre-occupation/concern with sleep.

“I think with just looking at this overall, I sort of think well okay if I only sleep for four hours it’s no big deal, I seem to function okay, I mean I will maybe get to the point in the weekend where if I have maybe a couple of extra hours, that’s enough sort of thing, and it seems to recharge the batteries, and you know, you can carry on, so it’s maybe, maybe I came in to this thinking ‘oh my god, I don’t sleep properly’ and blahdy blah, but actually I just think well that is probably what’s normal for me..” [Jennifer, 200-205]

For most participants, improvements were considered to be ongoing, and their perceived-capacity to obtain more sleep, and build on gains, was a prime motivator for continuing with the programme (after formal monitoring ceased).

“It’s the first time, as I say, that I’ve actually had a positive experience from something like this, em, and after ten years of sort of just dealing with it and being told there’s nothing much that can be done about it, it’s given me a slightly sort of
difference...attitude towards it...that there might be a, you know, some sort of light at the end of the tunnel with it, and I also appreciate that I might not become an eight or nine hour-a-night person, If I can quite simply stabilise at five to six, or even seven hours, then for me that’s gonna have a huge impact on me personally” [Jim, 238-242]

“I just have a very positive feeling about it, and I know that it’s early days and you’ve got to remember that it’s such a long time since I’ve had a good nights sleep, that it won’t change overnight, you know, and so I’ve got to keep working at it...so, and I think it’s the most positive thing I’ve done or felt is going to work, medication never really worked” [Gillian, 221-224]

Daytime functioning: a thermometer for success?

Although improvements in functioning tended not to be as robust as changes to sleep, at least at the time of interview, many participants reported positive modifications to aspects of daily living, such as having more energy, being more organised, being less anxious, enhanced coping skills, adopting a more positive outlook, and even comments by significant others concerning physical appearance. These improvements were typically just evolving 1-2 weeks prior to interview, in a slight time-lag behind sleep symptom changes.

“...I do think having the sleep sorted out has helped me [to] be calmer and more relaxed” [Louise, 179-180]

“I’ve got much more energy now than I had before” [Sarah, 242-243]
“see when you’re not sleeping you just don’t want to do anything, you’re just sitting, you don’t want to really talk to people, but now I’m going about, going out more and stuff, so good.” [Becky, 185-187]

“much more sort of…quite motivated for work and stuff, eh, it definitely has had a very positive effect on my sort of daily life” [David, 87-88]

“I think now I’m more likely if I’ve planned something for after work, to do it, em, I’m meant to, I’ve paid for most of this year west Dunbartonshire council money for a gym membership, that I [‘ve] never been in, em, so I’m now back three times a week!” [Lisa, 185-187]

“my sister has commented em that I don’t look like a panda anymore…with my big black eyes” [Lisa, 236-237]

“I do feel like a normal sleeper, which is bizarre and great, this has been huge for…like driving, I feel like I’m calmer you know I don’t get road rage, I don’t…I’m not as bad as what I was, I’m taking less risks in the car, I’m not in a rush, because I’m not as anxious, I’m not on edge, I’m just a bit more chilled I think, and I think the sleep helps to centre yourself…” [Hannah, 353-357]

“[it’s] bizarre thinking well I actually have to get out of bed, and I could still sleep another couple of hours, but I’ve actually also slept quite a few hours so that’s quite liberating, I do feel I’ve got more energy” [Sarah, 407-409]
Impact on functioning was considered a thermometer by many to gauge therapy success. The continuation of a journey, an ongoing treatment process, was often made with reference to obtaining future improvements in aspects of functioning.

“ If I can build it up to six and a half I think, six and a half hours, I’m gonna feel like I can function, right, so I guess I’m thinking in my head that’s where my functioning range is, six and a half hours is gonna allow me to function, eh, if I can better that then I’m hoping that’s where the energy and the zest for life is going to come from” [Bill, 325-329]

“I think...[I] still need that extra bit of sleep, which might come just the next couple of weeks, because I felt quite irritable this morning and that was because I didn’t have enough sleep” [Ross, 297-298]

“I’ve had some mornings where I felt really good, you know, and feeling the positiveness coming back again, thinking this is really going to work and that hasn’t left you know, I still think there’s room for improvement in that, so that’s what I’m heading for” [Gillian, 120-123]

Those individuals who considered SRT to have been unsuccessful in terms of improving night-time sleep, or that only reported minimal improvements (at least up until the interview), voiced that functioning was detrimentally affected during times of adherence (both acutely and throughout the four-week period). In extreme cases this meant a subsequent shift towards baseline sleep schedules, accompanied by a return to
‘normal’ levels of functioning; while others voiced determination to commit to the programme despite negative daytime experiences.

“I got quite excited, cos I felt although I wasn’t sleeping long, that I felt that I had slept better, but then I think that was three nights in all the three and a half weeks that I’d done it, that I felt like that, otherwise some nights I felt sort of always semi-awake, other nights I slept really badly, so it was only three nights that I felt ‘oh I really slept soundly’, so that was rather disappointing…I haven’t done it since the grandchildren are staying because I need to be on sparkling form all day with them” [Jane, 164-168/172]

“I was alright the first sort of few nights, and then I started feeling a wee bit down about it actually, I remember I said that on the dictaphone as well, that I started thinking hmm I can’t sort of comply with this and I can’t really…I don’t think I can do it properly, you know, that sort of thing, and I don’t like doing things sort of half-heartedly” [Jennifer, 138-140/145-146]

“I actually do feel worse during the day than what I previously did, whilst I’ve managed to recover some of the sleep that I wasn’t previously getting, em, I feel more exhausted during the day than what I did previously, em certainly in the mornings, and in the mid afternoons, em, I am not as alert as I used to be, em unless I’m actually working on a project or have a group of people to work with” [Jim, 173-176]
5.5. Discussion

The aim of this mixed-method study was to investigate, using both quantitative and qualitative methodologies, the patient experience of sleep restriction therapy for insomnia. Sleep-diaries, questionnaires, audio-diaries and semi-structured interviews were combined in an attempt to understand the impact of SRT on sleep and functioning, but also to gain an ‘insider’s perspective’ (Conrad, 1987) on treatment implementation and process.

5.5.1. Sleep-related changes

All major sleep diary variables significantly improved from baseline to post-treatment (excluding TST), and these improvements were maintained at three months. By three months, TST demonstrated a gain of 47 minutes, on average, relative to baseline values. Overall, sleep changes were comparable (and for some variables, larger) in terms of magnitude, to those obtained in full CBT interventions (Riemann & Perlis, 2009; Smith et al., 2002) and previously published trials of sleep restriction therapy (e.g. Friedman et al., 2000). Moreover, global insomnia disorder severity (assessed using the ISI) indicated mean values for the group to be in the ‘sub-clinical insomnia’ range at post-treatment and follow-up, relative to baseline levels in the ‘clinical moderate’ range. The clinical significance of these changes was confirmed with 44% and 67% of patients being classified as ‘remitters’ and ‘responders’, respectively, at three month follow-up.

Semi-structured interviews with patients, post-treatment, supported these changes in nightly sleep parameters; specifically concerning reduced sleep-onset latencies, decreased number of awakenings (also reported as a reduced ability to remember awakenings), and decreased amounts of wake-time during the night. Interviews also
revealed changes in subjective sleep quality; with participants providing rich
descriptions of sleep as being ‘deeper’ and ‘more efficient’, as well as reporting
dampened mentation at sleep-onset, and during middle-of-the night awakenings. Such
‘qualitative’ changes and increased predictability of sleep are likely related to the
harnessing of homeostatic pressure and circadian re-alignment; both desired targets of
sleep restriction therapy (Perlis & Pigeon, 2006; Spielman et al., 1987b; Cervena et al.,
2005; Edinger & Means, 2005).

Importantly, participants described simultaneous modifications to how they perceived
sleep. The feeling of ‘craving’ sleep – as a consequence of it being ‘denied’/restricted -
represented a significant shift from how participants typically viewed the sleep-onset
and sleep period. In turn, this led participants to describe having reduced anxiety and
worry when initiating sleep, and during middle-of-the-night awakenings. A recent
study by Spiegelhalder and colleagues (2010) found, somewhat surprisingly, that
increased sleep-related attentional bias (measured using a visual dot-probe paradigm)
prior to sleep, was subsequently positively associated with markers of improved sleep
continuity (greater sleep efficiency, slow wave sleep, and total sleep time, as well as
reduced awakenings). The authors interpret these findings as suggestive of a
homeostatic craving for sleep in PI patients. Sleep restriction may work, in part, then
by modifying - through partial sleep deprivation - the evolved threat value that sleep
and associated bedtime routine have come to represent in those with primary insomnia.
Although speculative, this notion could also relate to the natural variability of ‘good’
and ‘poor’ nights in PI patients (e.g. Perlis et al., in press B), where a good night has
been found to follow one to three nights of poor sleep: an eventual build up of sleep
pressure may help to defuse sleep-related anxiety, associated arousal, and selective attention, ultimately restoring sleep automaticity.

The knock-on effect of craving sleep, in terms of reduced pre-sleep anxiety and arousal, and increased nightly sleep predictability, may well relate to the robust decreases in the application of ‘sleep effort’ (as measured by the GSES), a concept central to recent cognitive models of psychophysiological insomnia (cf. Espie et al., 2006; Broomfield & Espie, 2005). Furthermore, it was also interesting that some patients underwent an adjustment process with respect to their sleep need during the first week. Functioning worse than they previously had done, a few patients began to feel perhaps their sleep problem was not as problematic or obstructive as once thought – which seemed to provide some level of reassurance, despite no obvious or immediate improved modifications to sleep.

To build on Edinger & Means’ (2005) description of ‘pathways’ implicated in CBT response (see figure 5.6), the present study suggests, based on participant descriptions, that SRT may have secondary or parallel effects on both ‘inhibitory factors’ (particularly conditioned arousal) and ‘cognitive factors/dysfunctional beliefs’, thought to be important in the aetiology and maintenance of insomnia [see additional red (dash) lines superimposed on figure 5.6]. This, of course, requires additional empirical testing in the context of isolated component intervention studies.
5.5.2. Side-effects and Daytime Functioning/HRQoL

Functioning fluctuated over the course of the four-week period. Responses from our newly developed side-effects checklist indicated that at least one half of participants experienced eight of the 12 listed symptoms, as a consequence of sleep restriction therapy during week one. Fatigue, extreme sleepiness, and reduced motivation/energy were the most commonly experienced difficulties, and, along with irritability and changes to hunger/appetite, negatively interfered with daytime functioning (to the greatest degree). In vivo diary reflections corroborated these accounts, with participants describing, at length, impairments in occupational performance, social functioning and everyday duties, citing exacerbated levels of fatigue and sleepiness and their subsequent downstream effects on cognition, as the main culprits. Of particular prominence, more than one-third of our sample described, during diary entry recordings, concerns with driving ability (these were again discussed during interviews). With numbers of this magnitude, it is clear that SRT is not an intervention with just mild and trivial effects, and it remains to be determined if such a treatment approach (at least acutely) is
associated with a ‘spike’ in automobile accidents (as reported with hypnotic use: Gustavsen et al., 2008; Engeland et al., 2007). Clearly the risk exists. It would be interesting to qualitatively track patients assigned to active hypnotic therapy to compare patient narratives and experiences across treatment modalities.

According to a recently published 6-month study on the efficacy of nightly Eszopiclone 3mg in those with primary insomnia (Walsh et al., 2007), adverse event rates were as follows for Eszopiclone versus placebo: somnolence (*8.8% v 3.2 %), myalgia (*6.0% v. 2.9 %) and headache (15.0% v. 15.0%). Another study by Scharf et al. (2005), over a more similar time-period to the present study, revealed the following rates for Eszopiclone 1mg, 2mg and placebo: somnolence (6.9% and 3.8%, vs. 8.8%) and headache (15.3 % and 15.2%, vs. 15%). It is abundantly clear that rates in the present study are substantially higher, and it is important to note that the above recorded adverse events in the pharmacological studies usually include multiple events from a single patient. Also of interest, two out of the five individuals who discontinued treatment did so because ‘side-effects’ (presumably) outweighed any sleep gain/benefits. CBT-I treatment studies rarely report (and/or record) reasons for attrition, perhaps because they are often difficult to ascertain, but if approximately 10% (in this study) drop-out due to an exacerbation of baseline impairments, this represents a significant challenge for CBT uptake and therefore effectiveness.

It is also worth pointing out that in the current study the minimum sleep window was set at five hours; other CBT-I programmes set a bottom limit of as little as four hours (e.g. Perlis et al., 2001c), which might be expected to produce more pronounced impairments.

* significantly different from placebo
than those reported here. One also has to remember that about 25% of insomnia patients may have high pre-existing excessive sleepiness levels (Day et al., 2001); these individuals may be a particularly high-risk group during the acute sleep restriction period.

To the best of our knowledge, side-effects encountered during CBT have not been adequately described or investigated in previous literature. Perlis and colleagues (2004) in their randomised trial of modafinil as an adjunctive to CBT, indicated that during week one of treatment, sleepiness, as measured by the Epworth Sleepiness Scale (ESS), approached pathological levels (ESS = ~10.5 versus a baseline of ~7); an effect that was in fact attenuated in the CBT plus modafinil group. Similarly, an abstract presented at the 2003 meeting of the Associated Professional Sleep Societies (APSS), in Chicago, by Fortier-Brochu and colleagues, reported on weekly questionnaire data (assessing daytime functioning symptoms) collected during the course of CBT-I. Results indicated a worsening of symptoms during the early stages of CBT, including elevated levels of fatigue, sleepiness and irritability – which coincided with the introduction of sleep restriction and stimulus control components. The present study is the first to reveal, in-depth, the descriptive nature and magnitude of experienced ‘side-effects’, that were attributed to the programme. Interviews suggested that for the majority of participants, side-effects sub-sided 1-2 weeks into treatment initiation, as sleep parameters began to stabilise and improvements in sleep quality evolved.

One intriguing finding was that number of checked side-effects positively correlated (moderate strength) with ISI, PSQI, and sleep efficiency change scores, from baseline to post-treatment. Side-effect daytime interference ratings were also significantly
associated with PSQI and GSES change scores (again moderate strength). It may be that side-effects provide an index of adherence to set threshold and rising times and hence mediate this relationship; the association with decreased sleep effort gives further credence to this argument. Of note, however, this ‘side-effect-outcome’ account runs partially counter to the findings of Perlis et al. (2004), who found that blocking/attenuating sleepiness using modafinil did not interfere with CBT-I efficacy; concluding that sleepiness may not be essential for CBT outcome. This finding, of course, does not rule out additive effects of modafinil properties beyond simply its stimulant action, such as the blockade of dopamine transporters (Volkow et al., 2009) and possible mood-enhancing effects, as well as increased levels of activity. Interestingly, the authors also reported a tendency for this group to be more adherent to bed-time instructions. Future studies must examine the mechanisms of sleep restriction and predictors of response.

With respect to daytime functioning outcomes, significant improvements were observed on the DFSAS (parts 1&2), GSII (ranks 1&2), OISQ, and three domains of the SF-36 (role emotional, mental health and vitality/energy) – all with moderate to large effect sizes. Overall, improvements tended to be most robust at three months than post-treatment, which may reflect the overlap between questionnaire reference period and experienced side-effects during the acute stages of treatment. This is an important consideration when assessing CBT gains directly following the ‘active’ phase. From semi-structured interviews, participants were beginning to notice improvements in domains of functioning, three to four weeks into the programme, describing positive changes in energy levels, fatigue, but also aspects of work life and social functioning. To add credence to these improvements being related to the intervention itself, strong
relationships were found between changes in sleep symptoms and daytime improvements – an association rarely found (or left unreported) in insomnia research (Means et al., 2000; Omvik et al., 2008; Kyle et al., 2010). Intriguingly, changes were most pronounced on our newly developed scales (DFSAS, GSII), which may suggest enhanced sensitivity of these measures – both based on words/experiences of insomnia patients – in detecting daytime difficulties relevant to those with insomnia disorder.

5.5.3. Adherence

Our use of qualitative methodologies provided insights into how participants adjusted and adapted to their new sleeping schedule. Audio-diary entries were particularly interesting because we were able to track experiences over time, from day 1, and gain access to moments of adherence and, importantly, non-adherence. Recent work by Vincent and colleagues (2008) revealed that perceived barriers to sleep restriction and stimulus control treatment engagement (measured with a non-validated questionnaire post-CBT) predicted self-report adherence; here we were able to capture the nature of encountered barriers, in real time. Specifically, negative impact on functioning, an inability to stave off sleepiness prior to bedtime, and boredom and loneliness during extra hours were all prominent reasons for non-adhering. Such factors also affected those who were adherent, representing general challenges of sleep restriction therapy implementation.

Side-effects interacted with going to bed late, so that impaired daytime functioning not only made staying up until a late set bed time incredibly difficult, but also created pressure and anxiety for participants when attempting to initiate sleep. They were faced with both the prospect of only having x hours to ‘obtain’ sleep, and the experience of continued, and possibly enhanced, impairments the following day. Thus, for some, the
normal catastrophizing about daytime consequences (Wicklow & Espie, 2000) was in fact exacerbated during the early stages of sleep restriction, which discouraged rigorous adherence. It would seem important, therefore, to identify those who do not respond in the first few days of treatment, to prevent complete or partial disengagement from treatment instructions. Indeed, a few participants, during interviews, did indicate that if there were to be increased therapist contact during active treatment, it would be in the first week.

In this regard, it would also be a worthwhile research endeavour to extend the early work by Perlis and colleagues (2004), to determine if additional stimulant therapy can attenuate daytime impairments, improve adherence, and (possibly) enhance outcome. Of course, activation therapies may not necessarily be restricted to pharmacological intervention. For example, some components of fatigue interventions used with cancer patients undergoing chemotherapy could also be applied, such as specific exercise activities (Escalante & Manzullo, 2009). Interestingly, during interviews and audio-diary entries, participants described a number of counter-measures they had developed, which included keeping active, engaging in discussion, and setting chores, all with the primary aim of improving alertness to promote adherence.

Another, perhaps unsurprising, finding from audio-diaries and interviews, was that adherence was most difficult on weekends compared with weekdays. Weekends tended to be viewed (prior to SRT) as designated time for catch-up sleep and rest. Participants commented on how ‘long’ and ‘lonely’ weekends had become, particularly because of a lack of stimulation and interaction during Saturday and Sunday mornings. Socialising also negatively impacted adherence and effectiveness through three main routes: firstly,
staying out late tended to shift bed time and the sleep window forward; secondly, proximal ‘stimulation’ too close to set bed time created ‘over arousal’, and hence sleep initiation more difficult; and finally alcohol intake negatively interfered with sleep quality. It would seem important to discuss, more thoroughly, weekend adherence with patients during CBT-I sessions. Emphasis could be placed on the importance of organising social activities in advance of weekends to help put in place a routine which supports adherence to threshold and rising times, and lessens the associated boredom (Vincent et al., 2008).

It should be noted that we did not find any evidence of a relationship between our questionnaire measure of adherence at 3 months and magnitude of outcome (pre-treatment to follow-up). The use of a non-validated scale, small sample, and time-frame (reference to adherence ‘in the last three months’) may all account for the null effects. Nevertheless, it is important to consider the possibility that rigorous adherence to sleep restriction is not necessarily (linearly) associated with outcome. Patients classified as ‘remitters’, for example, may no longer feel the need to rigorously implement set times; they are, after all, now considered to be in the normal sleeping range (Harvey et al., 2002). Moreover, patient narratives from the present study indicate other secondary effects of sleep restriction, such as reduced pre-sleep anxiety and sleep pre-occupation, increased sleep predictability, and even a ‘response shift’ to insomnia severity. Perhaps these parallel effects also contribute unique variance to explaining outcome. It is interesting that the few studies to assess relations between behavioural adherence and outcome tend to find either quite weak to modest associations (e.g. Vincent et al., 2008; Vincent & Hameed, 2003) or that sleep window consistency may be a better predictor than sleep reduction per se (e.g. Riedel & Lichstein, 2001). Perhaps stabilising set bed
and rising times, with a mild sleep reduction, as in fixed sleep restriction studies with elderly participants (e.g. Hoch et al., 2001), may be enough to obtain clinical improvements without inducing significant daytime disruption. It seems important for the field to investigate (1) conventional and modified (compression; Lichstein et al., 2001b) sleep restriction interventions; (2) predictors of adherence; and (3) the development of standard ways to assess adherence, including the use of objective measures such as actigraphy.

5.5.4. Implications for clinical practice and future research

There are several implications for clinical practice arising from this work. Firstly, patients need to be made fully aware of how difficult the sleep restriction programme can be, as well as the possible risks/dangers associated with the acute stages. Our participants’ initial perceptions of the programme when explained – although positive and logical – were, typically, far removed from the reality of implementation. Perhaps vignettes from real participants, like those in the present study, would give the prospective CBT-I patient an insight into some of the challenges involved.

This also raises the issue of whether the ‘expert patient’ (Department of Health, 2001) may have a role in treatment preparation, as a way of reducing attrition and enhancing adherence. That is, in addition to vignettes, participants (‘graduates’) who had previously taken part in CBT-I (and benefited) could speak with prospective patients about the treatment experience and implementation. This could be in the form of a recorded video clip, for example, shown to patients during CBT-I sessions. A similar approach is currently used in some respiratory clinics to inform sleep apnea patients of the benefits of CPAP therapy, prior to initiation (e.g. Wiese et al., 2005). Finally,
supplementary motivational interventions may also have a place in the behavioural management of insomnia; preliminary data suggests motivational interviewing, for example, is effective in improving CPAP adherence (Aloia et al., 2004). Application to CBT-I is worthy of investigation.

Although admittedly a small sample, results indicate that a brief sleep restriction intervention can, in isolation, improve aspects of functioning and sleep. This supports other published controlled studies that have used similar brief CBT-I packages (two sessions), documenting substantial treatment gains (Edinger & Sampson, 2003; Germain et al., 2007). Though more work is required, this literature and the present findings suggest that a brief (low-resource) behavioural intervention may be delivered as a first line treatment, in an attempt to widen access to evidence-based non-pharmacological insomnia treatments (Espie, 2009).

This work also has important implications for research. For example, the finding that SRT modifies sleep-related anxiety, arousal and perceptions, demands greater attention in future work, with a range of assessments. In particular, serial measurement of attentional bias and self-report questionnaire measures of pre-sleep mentation and applied sleep effort, over the course of sleep restriction, would be a worthwhile endeavour. Furthermore, does sleep restriction modify physiological arousal prior to sleep and also during the day? And what is the relationship, if any, with daytime functioning outcomes? It would also be informative to assess objective (cognitive) functioning throughout the intervention period, helping to establish whether acute restriction is associated with measurable impairments (as our qualitative reports suggest), and also whether therapy reverses possible baseline neurobehavioural deficits.
5.5.5. Limitations

The results of the present study have to be viewed in the context of several limitations. In relation to the diary and questionnaire data, particularly in light of the strong results, it needs to be borne in mind that this was an uncontrolled study, and that non-specific and other placebo-related effects cannot be ruled out without the inclusion of an adequate control group. Similarly, procedural aspects of the treatment protocol may have impacted results in two important ways. Firstly, audio-diary entries may have acted as a proxy for therapist contact, enhancing motivation/support to adhere with the programme, and ultimately moderating outcome. Secondly, post-treatment interviews were conducted by the therapist (also the researcher: SK), which may have created ‘demand characteristic’ behaviour on the part of interviewees. On this point, however, it was made explicit to participants that we were interested in their experience, ‘good or bad’, and that all information was interesting and relevant.

We also did not use polysomnography (PSG) to diagnose our patients as having primary insomnia. Although reliance on subjective self-reports to ‘diagnose’ sleep disturbance is standard for PI populations (Buysse et al., 2006), it remains possible that some of our participants experienced (significant) sleep state misperception, which may have exacerbated experienced side-effects during the intervention period.

Moreover, although not systematically recorded, the sample was largely white, middle class, and well-educated; these may be important factors when considering aspects of SRT implementation, motivation and adherence. Such potential moderating variables should be investigated in future trials. The generality of the findings may, therefore, be
restricted. In addition, several of the questionnaires used in the present study were non-validated and require further testing with larger samples of individuals with insomnia.

Finally, numerous statistical tests were performed, increasing the likelihood of type one error outcomes. Because of the small sample size and a predominant focus on integrating measures of participant ‘experience’, it was considered appropriate to not adjust the alpha level, balancing the likelihood of type I and type II errors. Nevertheless, further supplementary analysis of sleep and daytime functioning outcome data, applying the Bonferroni correction within each variable \( \alpha / 3 = 0.017 \), indicates that all carried out comparisons remain robust at the adjusted level, except for two variables (baseline to post-treatment comparisons for DFSAS part 2 and the SF-36 vitality dimension).

5.5.6. Concluding remarks

The present study provides novel data on the implementation and experience of sleep restriction therapy for insomnia. The triangulation of quantitative and qualitative methodologies gives considerable credence to the findings. This data has several important implications concerning SRT mechanisms of action, experienced side-effects and adverse events, factors impacting adherence and, finally, perceptions of benefit. This work underlines the value of using mixed methodologies to explore poorly understood and/or under-researched phenomena.
Chapter 6:

Auditory P300 and Neuropsychological Performance in Poor and Normal Sleepers: A Controlled Comparative Study
6.1. Abstract

Individuals suffering from insomnia typically report daytime difficulties with aspects of cognition. There are however mixed findings concerning the ‘objectivity’ of their subjective complaint. On the whole, previous studies assessing neurocognitive variables have recruited small and poorly defined samples, and differed in the range and sensitivity of measures used to probe cognition. To further understand the relationship between sleep disturbance and daytime information processing, we compared the largest investigated sample to date of poor sleepers (PS; n=58), meeting DSM-IV symptom criteria for primary insomnia, with 59 well-matched normal sleepers (NS) on several measures of neuropsychological performance and event-related potentials (ERPs) during an auditory oddball task. We found no evidence of performance impairment in the PS group, relative to NS, on any domain of the cognitive test battery. Results from the oddball task revealed no differences in terms of P300 amplitude, but the PS group had significantly reduced P300 latencies, specifically at Cz and Pz (p<.05). Taken together, our findings point towards increased arousal and/or applied task effort; both of which may help maintain behavioural performance on relatively ‘simple’ neuropsychological tests. The field would benefit from directing research efforts towards assessing the moderating effects of sleep, arousal, and effort on task performance. The utility of an international standardized database to probe aspects of insomnia is also considered.
6.2. Introduction

Cognitive impairment features as a potential daytime consequence of insomnia in the major sleep disorder classification manuals (i.e. ICSD-2, 2005; DSM-IV-TR, 2000). Telephone surveys, qualitative studies, clinician reports, and prospective diary investigations, have all documented that individuals with insomnia report deficits in different facets of cognition; including memory, concentration, attention, and executive functioning (e.g. Roth & Ancoli-Israel, 1999; Carey et al., 2005; Moul et al., 2002; Buysse et al., 2007). These isolated, proximal symptoms may culminate in more ‘downstream’ consequences, such as impaired work performance (e.g. Erman et al., 2008; Daley et al., 2009b; Kyle et al., 2008), and increased risk of being involved in road traffic accidents (e.g. Leger et al., 2006).

However, studies utilising objective measures of cognitive functioning, typically through computerised assessment and traditional neuropsychological testing, are inconclusive (Riedel & Lichstein, 2000; Fulda & Schulz, 2001; Orff et al., 2007). Some cognitive models of insomnia have instead explained the subjective-objective discrepancy in terms of elevated pre-occupation with sleep, and misattributions and monitoring for sleep-related consequences, rather than there actually being any real deficit (e.g. Semler & Harvey, 2006; Harvey, 2002). More recently, however, a meta-analysis of all studies assessing neuropsychological performance in insomnia, revealed small to modest effect sizes for impairments in attention, episodic and working memory, and executive functioning domains (Fortier-Brochu et al., 2008). Such data suggest that deficits do exist, but that methodological factors may be obscuring their identification. A recent systematic review of the neurobehavioral literature came to
similar conclusions: people with insomnia show performance impairments (more often than not) on tasks assessing attention and working memory (Shekleton et al., 2010).

It has been suggested that many of the early studies assessing objective performance in insomnia suffered from a number of design limitations, such as small and poorly characterised samples, and limited and/or insensitive test batteries (Edinger et al., 2008a; Shekleton et al., 2010). Others have also suggested that the absence of deficits may be explained by the poor sleeper’s ability to exert ‘compensatory effort’ during testing, potentially masking performance decrements (e.g. Bastien et al., 2003; Varkevisser et al., 2007; Orff et al., 2007; Espie & Kyle, 2008).

One way to assess alterations in cortical function, in the absence of recorded behavioural output, is through the measurement of event-related potentials (ERPs). ERPs are small amplitude fluctuations in the scalp-recorded EEG, elicited by either an external physical stimulus or an internal psychological event. ERPs thus provide high temporal resolution on the timing of sensory and cognitive processing. A number of positive and negative components have been identified and associated with specific brain processes. For example, early components occurring within 100 ms post-stimulus presentation reflect early sensory processing; components peaking at about 100 ms relate more to attentional processes (e.g. encoding, selective attention); and later components occurring 300-600 milliseconds post-stimulus typically index higher order cognitive processing, such as memory updating and detection of semantic deviance (Colrain & Campbell, 2007; Banich, 2004).
The most studied ERP phenomenon is a late positive component peaking at about 300 ms (referred to as the P300 or P3b) in response to stimulus change, across a number of sensory modalities (Duncan et al., 2009). P300 is an endogenous component, which is typically elicited during discrimination tasks (so-called oddball paradigms), where high probability (‘background’) stimuli are interspersed with low probability (‘target’) stimuli. The P300 wave therefore occurs when the subject detects the presence of a rare ‘target’ tone (in the case of an auditory task). The memory trace is much less formed for such stimuli compared with the frequently presented ‘background’ tones, and thus the generated centro-parietal wave is thought to reflect attentional processing and hippocampal-mediated updating of working memory (e.g. Linden, 2005; Polich, 2007).

Relatively consistent findings from the sleep deprivation literature reveal decreased amplitude and increased latency of the target P300 compared with healthy normal sleeping controls (e.g. Lee et al., 2003). Thus the speed at which the brain evaluates and updates the deviant or rare stimulus, and the amount of processing resources allocated to carrying out this operation, appears to be compromised under the load of sleep deprivation. Similar alterations have also been reported in sleep apnea patients (Rumbach et al., 1991; Sforza & Haba-Rubio, 2006), in healthy subjects during continuous sleep restriction protocols (sleep time reduced by one third of habitual total sleep time; Cote et al., 2008), and the transition from wake-to-sleep, reflecting changes in level of arousal (cf. Colrain & Campbell, 2007, for a review of sleep and ERPs). It is also worth pointing out that P300 abnormalities have been documented in a number of psychopathologies; including individuals with depression, schizophrenia, various sub-types of dementia, traumatic brain injury, and childhood developmental disorders (see Duncan et al., 2009, for a clinical review).
To the best of the author’s knowledge, there are approximately 11 published ERP studies using insomnia samples. Most have been interested in assessing aspects of ‘hyperarousal’ at sleep onset, during NREM sleep, on awakening, or during the day; predominantly focusing on earlier components of attention and sensory processing (Bastien et al., 2008; Yang & Lo, 2007; Regestein et al, 1993; Wang et al., 2001; Milner et al., 2009), though not exclusively (e.g. Devoto et al., 2003; Devoto et al., 2005).

This latter work by Devoto and colleagues (2005) is particularly worth mentioning because they specifically focused on P300 measurement during an oddball task, immediately pre- and post-sleep, in a small number of individuals with primary insomnia (n=7) and matched normal sleepers. The study was carried out over several nights permitting distinction between ‘good’ and ‘bad’ nights. Results indicated increased P300 amplitudes in the patient group relative to controls when pre and post sleep measurements were averaged for a ‘poor’ night of sleep, supporting earlier pilot data based on laboratory recordings the morning after a self-defined ‘poor’ night of sleep (Devoto et al., 2003). The data, overall, were interpreted as evidence for increased arousal, present only for poor nights of sleep (i.e. state hyperarousal). Such results conflict with compromised P300 parameters reported in healthy individuals during sleep deprivation.

Because oddball performance was measured prior to sleep, and immediately on awakening, results may not be relevant to the full daytime period, but instead might reflect an acute triggered threat-detection state directly proximal to experiencing, or anticipating experiencing, poor sleep. It is notable that increases in P300 amplitude have been reported in patients with anxiety disorders (e.g. Enoch et al., 2008).
Moreover, group differences in the Devoto study were only found at electrode site $F_z$; inconsistent with the topographical distribution of the evoked P300. Instead such a finding may be suggestive of a frontal measured P3a, representing inhibition of novel stimuli (Colrain, 2005). This study, therefore, awaits replication.

A small number of studies report on the potentially negative impact of insomnia symptoms on daytime cognitive processing. For example, Anderer and co-workers (2003) found significantly delayed latency and decreased amplitude of the P300 in postmenopausal woman with insomnia (prior to undergoing hormone replacement therapy), relative to healthy postmenopausal controls. In a secondary analysis, using low resolution electromagnetic tomography (LORETA) to localize scalp electrical activity, Anderer et al. (2004) reported decreased source strength of standard (N1, P2) and target (N2, P300) components. Similarly, Szelenberger & Niemwicz (2001), again using LORETA, found reduced source density during the continuous attention task across both early and late attentional components (i.e. P1, N1, N2, P3) in 14 individuals with primary insomnia (PIs), with differences particularly pronounced in orbitofrontal, medial prefrontal, and anterior cingulate regions. Moreover, Bruder and colleagues (1991) reported, in a group of depressed individuals, significant positive associations between P300 latency (during an audiospatial task) and sleep item scores on the Hamilton rating scale for depression i.e. longer latencies for those with greater sleep disturbance. In contrast, however, Sforza & Haba-Rubio (2006) found no evidence of altered P300 amplitude or latency during pre- and post-sleep recordings, in a well-screened sample of PIs relative to healthy controls.
In summary, this small literature tentatively points to potential information processing deficits in those with insomnia, at least at the cortical level. Importantly though, available studies suffer from small and/or diluted groups, and/or the failure to assess both ERPs and neuropsychological variables within the same sample. In collaboration with the Brain Resource International Database (BRID) the present study sought to (1) better understand the impact of sleep disturbance on information processing by comparing a relatively large sample of poor sleepers (meeting putative symptom criteria for DSM-IV Primary Insomnia) with a well-matched group of normal sleepers; and (2) as a by-product of this approach, consider the merits of a standardized database to probe specifics aspects of insomnia. Group differences were investigated on behavioural measures assessing a number of neuropsychological variables, as well as latency and amplitude of the late P300 component during an auditory oddball paradigm.

Based on the mixed and small literature this study was necessarily exploratory in nature, but we hypothesized a number of possible outcomes: (1) poor sleepers would show impairment on both neuropsychological tests and the P300 component; (2) poor sleepers would not show overt performance deficits but would have impaired measures of P300 amplitude and latency; (3) poor sleepers would not display neuropsychological impairment and have P300 markers suggestive of ‘arousal’ (i.e. increased amplitude and/or decreased latency); and finally (4) there would be no alterations on any neurocognitive dependent measure between poor and normal sleepers.
6.3. Method

6.3.1. Participants

We identified 58 participants from the normative BRID, meeting our defined criteria for poor sleeper (PS). The BRID is an international collaborative effort which uses standard uniform brain and behavioural measurements, and exclusion/inclusion criteria, across approximately fifty different labs (Gordon, 2003; Gordon et al., 2005). BRID represents an integrative neuroscience platform, collecting data using a number of methods, including: self-report questionnaires, quantitative EEG, ERPs, fMRI, and genetic swab analysis. The normative healthy dataset currently has over 2000 participants, all of whom have undergone a standard thorough protocol, and have been excluded for major medical, psychiatric, neurological, and substance abuse disorders. Specifically, exclusion criteria are determined using BRID personal history and screening instruments, which include the Somatic and Psychological Health Report questionnaire (SPHERE-12; Hickie et al., 1998) and the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001), as well as additional items to assess medical and psychiatric history. All participants give informed consent to have their (anonymised) data uploaded to a centralised database.

As part of the screening/informational procedure, participants complete a basic (non-validated) sleep questionnaire (see Appendix M). To be included in our PS group, participants had to indicate that they experienced difficulties ‘initiating’ and/or ‘maintaining’ sleep at least 3-4 times per week in the last month. Participants were also required to respond below a certain criterion (1-2 times per week, or less) on questionnaire items assessing symptoms of sleep-disordered breathing and restless legs syndrome. Thus, participants met symptom criteria for DSM-IV primary insomnia, free from psychiatric and medical co-morbidities. None of the sample reported taking sleep-
promoting hypnotics. Our matched normal sleepers (NS; n=59) reported no difficulty with sleep initiation, maintenance, or sleepiness during the day. For each poor sleeper, a normal sleeper was selected and matched, as much as possible, in terms of age, gender, and years of education.

6.3.2. Materials & Procedure

6.3.2.1. Neuropsychological assessment

All participants within the BRID complete the touch screen IntegNeuro™ test battery, assessing performance in a number of cognitive functions – including sensori-motor, memory, attention, executive functioning, and language domains. Subjects complete ten tasks in a sound attenuated room, taking a total of ~50 minutes. Tests are administered using pre-recorded, automated task instructions (.wav files via headphones). An IBM touchscreen records participant responses and .wav files record spoken answers in tasks requiring a verbal response. The battery has been validated against more traditionally used measures of neuropsychological functioning, revealing highly significant correlations (Paul et al., 2005).

Immediately prior to testing, participants complete information pertaining to basic demographics, and also record their sleep duration for the previous night. As part of the screening procedure, data are also recorded on depression, anxiety and stress, using the depression, anxiety, and stress scales (DASS; Lovibond & Lovibond, 1995). We selected four broad domains for analysis that have some support in the existing literature for being impaired in those with insomnia: attention and motor speed, verbal fluency, executive functioning, and verbal learning and memory functioning (see table 6.1 for a description of tests used).
Table 6.1 – IntegNeuro standard neuropsychological test descriptions (taken and modified from Paul et al., 2006).

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choice Reaction Time</td>
<td>One of four circles was illuminated on the screen. Immediately following presentation, the subject is required to touch the illuminated circle as quickly as possible. Twenty trials were administered with a random delay between trials of 2–4 seconds. The dependent variable was the mean reaction time (ms) across trials.</td>
</tr>
<tr>
<td>Sustained Attention/Working Memory</td>
<td>A series of letters (B, C, D or G) were presented to the subject, one-by-one, on the computer screen (for 200 milliseconds), separated by an interval of 2.5 seconds. If the same letter appeared twice in a row (target letters), the subject was asked to press buttons with the index finger of each hand. Speed and accuracy of response were equally stressed in the task instructions. There were 125 stimuli presented in total, 85 being non-target letters and 20 being target letters. The dependent variable was mean reaction time (ms) to target stimuli and number of errors.</td>
</tr>
<tr>
<td>Executive Maze</td>
<td>The subject was presented with a grid (8 × 8 matrix) of red circles on the computer screen. The object of the task was to identify the hidden path through the grid, from the beginning point at the bottom of the grid to the end point at the top. The subject navigates around the grid by pressing arrow keys on the touchscreen interface. The subject was presented with one tone (and a red cross at the bottom of the screen) if they made an incorrect move, and a different tone (and a green tick at the bottom of the screen) if they made a correct move. The purpose of the task was therefore to assess how quickly the subject learned the route through the maze and their ability to remember that route. The trial ended when the subject completed the maze twice without error or after 10 minutes had elapsed. Dependent variables were time to complete full maze for the first time (secs) and number of trials in total to complete the task (maze completion twice without errors).</td>
</tr>
<tr>
<td>Stroop Task</td>
<td>Participants were presented with colour words printed in incongruent ink and they were required to name the colour of the ink rather than read the word. In a second part participants were this time asked to name the word and ignore the background colour. Total number of correct trials was the dependent variable for both parts.</td>
</tr>
</tbody>
</table>
### Switching of Attention

This modified version of the Trail Making Test consisted of two parts. The first required the connecting of numbers in ascending sequence (1-2-3- etc.). On the first test (switching of attention 1), 25 numbers, in circles, were placed on the touchscreen and the subject was instructed to press them in the correct order. This tests the basic ability to hold attention on a simple task. The second test (switching of attention 2) required the connecting of numbers and letters in an ascending but alternating sequence (1-A-2-B etc.). The numbers 1–13 and the letters A–L were presented in circles on the touch-screen. Dependent variables of interest were time taken to complete both parts 1 and 2.

### Verbal Fluency

Subjects were required to generate words beginning with the letters F, A, and S. Sixty seconds were allotted for each letter and proper nouns were not allowed. The total number of correct words generated across the three trials was the dependent measure. In a second related test animal word generation was assessed by asking subjects to name as many animals as possible within sixty seconds. Total number of animals generated was the dependent measure.

### Verbal Learning and Memory

Participants were read a list of 12 words, which they were asked to memorize. The list contained 12 words from the English language. Words were closely matched on concreteness, number of letters and frequency. The list was presented four times in total and the subject was required to recall as many words as possible after each presentation. The subject was then presented with a list of distractor words and asked to recall them. The subject was then asked to recall the 12 words from the original list. The dependent variable was the number of words correctly recalled across the four learning trials, learning rate over the trials, number of intrusions, words recalled from the distractor list, and short and long-term delay recall.

### Digit Span

Subjects were presented with a series of digits (for example, 4,2,7…) presented individually for 500 ms and separated by a one second interval. The subject was then immediately asked to enter the digits on a numeric keypad on the touchscreen, either in forward order or backwards (reverse digit span task). The number of digits in each sequence was gradually increased from 3 to 9. The dependent measure was the maximum number of digits the subject recalled (forward and backwards) without error.
6.3.2.2. Oddball task

The oddball task is completed as part of the LabNeuro test battery. In the auditory oddball task, participants are presented binaurally, via headphones, with a series of high and low tones, at 75 dB, and lasting for 50ms, with an inter-stimulus interval (ISI) of 1 second. Rise and fall times of the tones was 5 ms. Participants were instructed to press designated buttons with the index finger of each hand in response to ‘target’ tones (presented at 1000 Hz). They were asked not to respond to ‘background’ tones (presented at 500 Hz). The task took place in a sound, temperature and light controlled room. Participants were given a brief practice session to clarify the distinction between target and background stimuli. Speed and accuracy were stressed equally in the task instructions. There were 280 background and 60 target tones presented in a quasi-random order, with the only constraint being that two targets could not appear consecutively. Duration of task was six minutes and reliability of the task, similar to the neurocognitive test battery, has been documented (Williams et al., 2005).

6.3.2.3. EEG/ERP acquisition

A QuickCap (Neuroscan) was used to acquire EEG data from electrode sites according to the 10-10 international system. Data were recorded relative to the average of A1 and A2 (mastoid) electrode sites. EEG data were screened visually for artefacts, normal variants and changes in alertness. For the oddball task, ERP data were extracted from EEG recordings. Conventional ERP averages were formed at each recording site in relation to each target stimulus.

All target stimuli with a correct button response (only) were included in the target average. Before averaging, each single-trial waveform was filtered at 25Hz with a
tukey (cosine) taper to 35Hz, above which frequency no signal was passed. For the
target stimuli waveforms, the peak (amplitude and latency) of the P300 component was
identified (relative to a pre-stimulus baseline average of -300 to 0 milliseconds) at each
site. ERPs were scored using an automated algorithm, which were then validated by
experienced scorers. The algorithm uses pre-determined latency windows as a guide to
determining component peaks; specifically, 270-550 ms post-stimulus for the P300
component (e.g. Williams et al., 2000). Between group effects were assessed in terms
of latency (msec) and amplitude (µV) of the target P300 at electrode sites Fz, Cz, and
Pz (elicited P300 is maximal over midline scalp sites; Duncan et al, 2009). We also
extracted behavioural responses (button press) for each participant, in terms of average
reaction time (RT) to target stimuli.

6.3.2.4. Analyses

Student $t$ tests and non-parametric equivalents were used to compare groups on
demographic variables of interest, including age, education, sleep duration, stress,
depression, and anxiety. Gender distributions were assessed using a 2x2 chi-square test.

Dependent variables were screened for extreme outliers and assessed for normality
using histograms. Extreme outliers (>3 SD above the mean) were replaced with the
respective group mean, and logarithmic transformations performed to correct skewed
distributions. Replaced outliers made up less than 5% of data points for any single
dependent variable.

The four domains from the neuropsychological test battery were investigated separately
using one-way (between-groups) MANOVA models. Given the small amount of
published work investigating P300 parameters in insomnia, univariate analyses were conducted at each midline electrode site (Fz, Cz, Pz) for both amplitude and latency. To balance likelihood of type I and II errors, the Bonferroni correction method was applied within each domain (i.e. $\alpha = .05/3 = .017$). Effect sizes (Cohen’s $d$; Cohen, 1988) were also calculated. Behavioural reaction time (RT) data for target stimuli were assessed using univariate ANOVA.
6.4. Results

6.4.1. Participant characteristics

As expected, poor and normal sleepers had practically identical mean age, years of education, and gender distributions (all non-significant; see table 6.2). PS reported significantly less sleep duration for the night prior to testing, relative to the control group of normal sleepers \( t(115) = -3.98, p<.001 \), and significantly greater scores for depression \( U = 1301, z = -2.30, p<.05 \), stress \( U = 1129, z = -3.22, p<.01 \) and anxiety \( U = 1363, z = -2.05, p<.05 \) scales of the DASS (see table 6.2). These small but reliable differences emerged despite subjects within the ‘normal’ database scoring well below clinical cut-offs.

Table 6.2 - Participant demographics. Presented are group means and standard deviations (in parentheses). N.B. Median scores are presented for depression, anxiety and stress (DASS) scales, with the accompanying interquartile range in parentheses.

<table>
<thead>
<tr>
<th></th>
<th>PS (n=58)</th>
<th>NS (n=59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (SD)</td>
<td>38.0 (14.1)</td>
<td>38.3 (14.2)</td>
</tr>
<tr>
<td>Gender</td>
<td>41 female/17 male</td>
<td>42 female/17 male</td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>14.0 (3.0)</td>
<td>14.4 (2.6)</td>
</tr>
<tr>
<td>Sleep Duration (hrs)</td>
<td>***6.2 (1.4)</td>
<td>7.2 (1.2)</td>
</tr>
<tr>
<td>Depression *2.00 (0-3)</td>
<td>1.00 (0-2)</td>
<td></td>
</tr>
<tr>
<td>Anxiety *1.00 (0-2)</td>
<td>0.00 (0-1)</td>
<td></td>
</tr>
<tr>
<td>Stress **3.00 (2-5)</td>
<td>1.00 (0-4)</td>
<td></td>
</tr>
</tbody>
</table>

***p<.001, **p<.01, *p<.05
6.4.2. Neuropsychological testing

MANOVA did not reveal any group differences (or trends) for the domains of verbal fluency \[F(2, 107) = .574, p = .565; \text{Wilks’ Lambda} = .99; \text{partial eta squared} = .01\], attention and motor speed \[F(7, 94) = 1.40, p = .214; \text{Wilks’ Lambda} = .91; \text{partial eta squared} = .09\], verbal learning and memory \[F(6, 51) = 1.06, p = .401; \text{Wilks’ Lambda} = .89; \text{partial eta squared} = .11\] or executive functioning \[F(4, 100) = 1.37, p = .248; \text{Wilks’ Lambda} = .948; \text{partial eta squared} = .05\]. Thus, no group differences in performance were present on any test of the battery (see table 6.3 for group means and standard deviations for each test).

*Table 6.3 - Mean (not transformed) scores (SD) for neuropsychological tests for poor sleepers (PS) and normal sleepers (NS).*

<table>
<thead>
<tr>
<th></th>
<th>PS</th>
<th>NS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Verbal Fluency</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Animal (words)</td>
<td>23.4 (6.9)</td>
<td>22.2 (5.3)</td>
</tr>
<tr>
<td>FAS (words)</td>
<td>15.2 (4.7)</td>
<td>14.5 (3.7)</td>
</tr>
<tr>
<td><strong>Attention &amp; Motor Speed</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Choice RT (ms)</td>
<td>705.4 (90.7)</td>
<td>691.8 (91.5)</td>
</tr>
<tr>
<td>Sustained att. (no. errors)</td>
<td>1.26 (1.6)</td>
<td>1.86 (1.7)</td>
</tr>
<tr>
<td>Sustained att.(ms)</td>
<td>474.6 (86.5)</td>
<td>506.8 (89.2)</td>
</tr>
<tr>
<td>Switching att. (numbers; secs)</td>
<td>19.5 (5.3)</td>
<td>20.8 (4.4)</td>
</tr>
<tr>
<td>Switching att. (mixed; secs)</td>
<td>46.6 (15.8)</td>
<td>48.0 (15.9)</td>
</tr>
<tr>
<td>Digit span (words)</td>
<td>6.2 (1.4)</td>
<td>6.4 (1.3)</td>
</tr>
<tr>
<td>Reverse digit span (words)</td>
<td>4.9 (1.4)</td>
<td>4.8 (1.4)</td>
</tr>
<tr>
<td><strong>Executive functioning</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop Word (no. correct)</td>
<td>18.6 (2.3)</td>
<td>18.4 (3.4)</td>
</tr>
<tr>
<td>Stroop Colour (no. correct)</td>
<td>11.7 (3.8)</td>
<td>11.8 (4.5)</td>
</tr>
<tr>
<td>Total no. of trials to complete maze</td>
<td>9.0 (4.1)</td>
<td>9.0 (3.9)</td>
</tr>
<tr>
<td>Maze completion time (first time; secs.)</td>
<td>213.5 (155)</td>
<td>184.4 (115)</td>
</tr>
<tr>
<td><strong>Verbal Learning and Memory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Memory recall (trials 1-4)</td>
<td>33.1 (5.3)</td>
<td>33.1 (4.6)</td>
</tr>
<tr>
<td>Learning rate</td>
<td>0.99 (0.55)</td>
<td>1.24 (0.48)</td>
</tr>
<tr>
<td>Number of intrusions (1-4)</td>
<td>1.40 (1.7)</td>
<td>1.16 (1.4)</td>
</tr>
<tr>
<td>Distracter list (no. of words)</td>
<td>4.8 (1.8)</td>
<td>5.1 (1.4)</td>
</tr>
<tr>
<td>Delayed recall (short delay; words)</td>
<td>8.3 (2.5)</td>
<td>7.9 (1.9)</td>
</tr>
<tr>
<td>Delayed recall (long delay; words)</td>
<td>7.9 (2.6)</td>
<td>7.7 (2.5)</td>
</tr>
</tbody>
</table>
6.4.3. Oddball task (ERPs)

One hundred and ten of the original 117 participants completed the auditory oddball task (54 poor sleepers, 56 normal sleepers). In terms of P300 amplitude (see table 6.4), no group differences were present at site $F_z$ [$F(1, 104) = .739, p = .375; \text{Cohen’s } d = 0.11$], $C_z$ [$F(1, 101) = .312, p = .578; \text{Cohen’s } d = 0.12$], or $P_z$ [$F(1, 103) = .244, p = .623; \text{Cohen’s } d = 0.10$]. For P300 latency, univariate analyses at each electrode site revealed that latencies were significantly reduced in PS compared with NS at $C_z$ [$F(1, 101) = 6.53, p = .012; \text{Cohen’s } d = 0.50$] and $P_z$ [$F(1, 103) = 5.52, p = .021; \text{Cohen’s } d = 0.46$], though not $F_z$ [$F(1, 104) = 2.16, p = .144; \text{Cohen’s } d = 0.29$]. Thus, after Bonferroni correction, latency group differences remained statistically significant at electrode site $C_z$ (see table 6.4 and figure 6.1).

Table 6.4 - Mean P300 amplitude and latency for both groups at each midline electrode site.

<table>
<thead>
<tr>
<th></th>
<th>PS</th>
<th>NS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amplitude (µV)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$F_z$</td>
<td>8.7 (7.4)</td>
<td>7.5 (6.4)</td>
</tr>
<tr>
<td>$C_z$</td>
<td>9.2 (8.4)</td>
<td>8.3 (7.7)</td>
</tr>
<tr>
<td>$P_z$</td>
<td>14.3 (7.5)</td>
<td>13.6 (6.7)</td>
</tr>
<tr>
<td><strong>Latency (msec.)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$F_z$</td>
<td>335.9 (24.6)</td>
<td>343.0 (24.7)</td>
</tr>
<tr>
<td>$C_z$</td>
<td>334.0* (28.6)</td>
<td>347.8 (26.5)</td>
</tr>
<tr>
<td>$P_z$</td>
<td>346.0* (24.5)</td>
<td>357.0 (23.5)</td>
</tr>
</tbody>
</table>

* sig. at $p < .05$

a sig. after Bonferroni adjustment
There were no differences in terms of behavioural reaction time (button press to target tones) between the two groups \(F(1, 107) = .343, p=.559;\) Cohen’s \(d= 0.11\): poor sleepers M=335.2, SD=41.0); and normal sleepers M=339.7, SD=40.6.
6.5. Discussion

We found no direct evidence of neuropsychological impairment across several cognitive domains in poor sleepers meeting DSM-IV symptom criteria for primary insomnia using a battery of standardised tests. This parallels a number of studies that have failed to find objective evidence to corroborate the typical subjective daytime complaint of cognitive dysfunction in insomnia (e.g. Orff et al., 2007; Varkevisser et al., 2007; Omvik et al., 2008; Riedel & Lichstein, 2000; Fulda & Schulz, 2001). Our poor sleepers did not differ from normal sleepers on domains of attention and motor speed, verbal fluency, executive functioning, or verbal learning and memory.

Such data may accord with recently published work indicating that impairments in performance, in \textit{complaining patients}, only become apparent in ‘complex’ load-intensive tasks, invoking a series of parallel cognitive processes (e.g. Edinger et al., 2008a; Altena et al., 2008a). In more ‘basic’ tasks, like the ones used in the present study, the poor sleeper may be able to mobilise adequate compensatory effort to mask performance decrements. The ability to compensate for the effects of sleep loss on task performance during sleep deprivation studies has been relatively well described at the neural level. Although contingent on task type, a compensatory system which involves stronger activation and/or additional recruitment of regions in the prefrontal cortex and parietal lobes may assist in maintenance of performance (e.g. Drummond et al., 2000; Drummond et al., 2004). Although preliminary, recent work also indicates that similar neural compensation may occur in insomnia. PI patients were shown to demonstrate increased cerebral activation in task-specific neural networks, including the right middle frontal gyrus, relative to normal sleepers during a working memory (n-back) task, despite no evidence of overt performance impairment (Orff et al., 2009).
In terms of the oddball task, there were no group differences in P300 amplitude. Interestingly though, we did find a significant group effect for peak latency; with the PS group evidencing reduced latencies at sites Cz and Pz (the typical scalp distribution for the P300 component). Although latency differences were small, they are similar in magnitude to differences recently reported between patients with major depression and healthy controls - though in the opposite direction - from the very same database (Kemp et al., 2009). Indeed, effect sizes \( d \) were found to be in the moderate range for Pz and Cz. There was, therefore, no evidence of a slowing or reduction in brain processing capacity during (context) updating of rare tones, as has been reported in the sleep restriction literature (e.g. Cote et al., 2008) and in previous studies recruiting patients with insomnia symptomatology (e.g. Anderer et al., 2003, 2004; Bruder et al., 1991). Instead, the pattern of data may be explained by at least two possible mechanisms, which could also help understand neuropsychological performance.

### 6.5.1. Arousal

Firstly, reduced latencies may indicate increased levels of ‘arousal’, or ‘arousability’, within the sample of poor sleepers. This would be in line with a number of studies (using active and passive oddball paradigms) that report increased levels of cortical arousal across both early (Bastien et al., 2008; Milner et al., 2009; Regestein et al., 1993) and late ERP components (e.g. Devoto et al., 2003, 2005) - typically reflected in increased amplitude. This is the first study to suggest a reduction in P300 peak latency. Although often negatively related, P300 amplitude and latency may be differentially affected by aspects of arousal. For example, increased heart rate and body temperature in healthy individuals is associated with shortened latencies, but not increased amplitude (for a review see Polich & Kok, 1995). This is particularly relevant to
insomnia as both heart rate and body temperature have been reported to be increased in patients compared with normal sleepers (for a review see Bonnet & Arand, 2010). Although speculative, alterations to these physiological parameters may have contributed to a shortening of P300 latency in the present sample. The P300 finding may, therefore, reflect ‘hyperarousal’ in both autonomic and central nervous systems - consistent with contemporary perspectives on insomnia (Riemann et al., 2010). The inability to document altered P300 amplitudes, as reported by Devoto and colleagues (2003, 2005), may relate to differences in sample characteristics concerning measures of arousal. It should also be mentioned that Devoto et al. (2003, 2005) only found group differences for the ‘worst’ night of sleep. We were unable to ascertain from this sample of individuals whether sleep the night before testing was considered ‘poor’ quality.

Nevertheless, and although requiring replication, our P300 finding tentatively supports the notion that documented hyperarousal and enhanced sensory processing at sleep-onset and during NREM sleep (e.g. Nofzinger et al., 2004; Perlis et al., 2001b; Milner et al., 2009; Bastien et al., 2008), may extend right across the 24-hour period. Interestingly, anxiety disorder patients, who are typically considered to be dysfunctionally aroused, also demonstrate increased P300 amplitudes and reduced latencies (Enoch et al., 2008; Hanatani et al., 2005). Indeed, in the former of these studies, greater P300 amplitudes were associated with enhanced performance on a digit symbol task assessing attention and working memory (though only for anxious patients; Enoch et al., 2008). Alternatively, it might be that reduced P300 latencies, indicative of speed of stimulus evaluation, and perhaps a reflection of arousal, may help to maintain performance in those with insomnia in certain controlled tasks. Related to this, elevated ‘arousal’ has been proposed, in a recent study, as the explanation for why complaining
PI patients performed significantly faster (RT) on a simple test of vigilance (Altena et al., 2008a), and showed impairments only in tasks demanding greater cognitive load, compared with good sleepers. This apparent ‘inverted U’ performance curve, where hyperarousal may be facilitatory on some tasks, and obstructive when cognitive load is manipulated in a systematic fashion, could lead to a masking of any obvious impairment during tests typically sensitive to sleep loss (Horne, 2010).

Reduced whole brain gamma-aminobutyric acid (GABA) levels in PIs (Winkelman et al., 2008), though speculative and requiring further investigation, may lead to attenuated inhibitory action on aspects of alertness, emotional reactivity, and ultimately cognitive functioning. In relation to possible GABA abnormalities, it is notable that stimulation of GABAergic receptors in healthy controls, through the administration of GABA_A receptor agonists (e.g. barbiturates, benzodiazepines), leads to an attenuation of P300 amplitude (Watson et al., 2008), and a lengthening of latency (Fowler & Mitchell, 1997). This, of course, reflects a general dampening of cortical information processing; conversely, therefore, enhanced cortical arousal may lead to, and be reflected by, a shortening of latencies. To fully conclude an increased cortical arousal account, with respect to evoked potentials, earlier components of attention (N1, P2) to both target and non-target stimuli will require to be recorded several times throughout the course of the day in patients with PI.

6.5.2. Effort

A second plausible explanation is that reduced P300 target latencies may be a marker of increased effort or motivation. Recent work in sleep deprivation provides some support for this account. Hsieh and colleagues (in press) sleep deprived 24 healthy volunteers
and randomised half to receive monetary incentives for improved accuracy on a modified flanker task. After each trial, points would appear on the screen conveying to participants how well they had performed. Participants were told that total number of obtained points would be converted into currency. P300 was recorded during the Flanker task. Two important findings emerged. It was found that subjective effort level decreased in the group with sleep deprivation plus ‘no incentives’, relative to when the same group completed the task after a regular night of sleep. Conversely, there was no recorded decrement (sleep deprived versus rested) in subjective effort in the sleep deprived plus ‘incentives’ group. Thus the usual decrease in subject effort induced by sleep deprivation was attenuated with the provision of incentives. Importantly, P300 latencies were also found to be significantly reduced in those who were offered monetary rewards (compared to the ‘no incentives group’), suggesting that the effects of sleep deprivation on attentional processes involving stimulus categorization and evaluation can be attenuated by the deployment of effort/motivation.

Additional evidence for the effects of motivation on P300 parameters comes from work by Carrillo-de-la-Pena & Cadaveira (2000). In this study, healthy participants completed the auditory oddball task (similar to the present study) twice. Directly after run 1, participants were instructed to do as well as they could in a next subsequent run. They were also told that their results would be compared with their peer group (classmates). Motivational instructions resulted in an increase in amplitude and a trend towards decreased P300 latency. A repeat of the study one year later in the same subjects (without motivational instructions) ruled out a simple repetition explanation for the findings. It is possible, however, that motivational instructions concerning peer comparison may have induced anxiety-arousal responses, subsequently modifying P300
parameters. Nevertheless, in light of this work, combined with evidence from sleep deprivation, and preliminary neural compensation imaging data in insomnia patients (Orff et al., 2009), reduced latencies in the present study may indicate that participants are ‘working harder’ to try to maintain performance. From previous qualitative work by our group (Kyle et al., in press) it is clear that patients with primary insomnia report deploying more effort to complete daily tasks; whether or not this translates to controlled performance testing and notable modifications to ERP parameters, remains unclear. No subjective measure of effort was included in the present study but future research should consider investigating both subjective and objective markers of cognitive effort during testing as well as naturalistic conditions.

6.5.3. Limitations

Conversely, a general inability to observe deficits in the poor sleeper group may also relate to several intrinsic limitations of the study. Although the selected poor sleepers met questionnaire criteria for insomnia symptoms, we cannot be sure they have a self-defined ‘problem’ with sleeping (i.e. have a clinical complaint). Furthermore, we do not, for example, have data on duration of sleep disturbance (beyond a minimum of one month) or self-reported daytime impairment attributed to significant sleep disturbance. Importantly, however, mean age, gender bias towards females, reduced prior night sleep duration, and significantly increased scores – despite an enforced ceiling for clinical cut-offs – on depression, stress and anxiety scales, all mirror the characteristics of a primary insomnia sample (e.g. Orff et al., 2007; Morin et al., 2003).

Concerning severity of insomnia, it is worth mentioning that a recent study (Fernandez-Mendoza et al., in press), assessing the largest number of insomnia patients to date ($n=$
116; duration ≥1 year), failed to find evidence for impairment across a number of domains (attention, visual memory, word fluency, psychomotor speed) compared with normal sleepers (n=562). The strength of this study, however, was that all participants underwent PSG recording, allowing further sub-grouping. Four groups were evaluated in terms of performance: insomnia patients with normal objective sleep duration (≥6 hours; n=65); insomnia patients with < 6 hours objective sleep duration (n=51); normal sleepers with ≥ 6 hours of sleep duration (n=343); and finally normal sleepers with < 6 hours sleep duration (n=219). The comparison between objectively ‘short sleeping’ insomnia patients, normal sleeping healthy controls, and short sleeping healthy controls, revealed impairments in insomnia patients (compared with both groups) in processing speed, set-switching attention, and short-term visual memory. It might be, therefore, that objective impairment is most pronounced in (objectively) short sleeping individuals with insomnia (see, also, Edinger et al., 2008a), though this remains to be documented at the subjective level. The sample investigated in the present study, similar to many other studies assessing performance in insomnia, may have included a heterogeneous group in terms of ‘severity’, preventing documentation of ‘true’ performance deficits.

Another major limitation of the present study is that we cannot be sure that time of testing was similar for all participants, and therefore circadian effects in terms of alterations to vigilance and alertness, may have confounded both neuropsychological (Schmidt et al., 2007) and ERP (Polich & Kok, 1995) findings. Finally, our ‘bottom-up’ statistical approach, using univariate analyses to assess ERP parameters, may be considered liberal. Given the small literature in this field, it was thought best to take a pragmatic view to balancing type I and II errors. However, applying the Bonferroni correction and calculating effect size magnitude, while still not the most conservative
approach, minimises the opportunity for misleading conclusions. It should also be noted that adjusting the alpha level within both amplitude and latency variables separately, as compared with adjusting across the six comparisons, may potentially contribute to an increased likelihood of type I error outcomes. This latter more conservative approach, however, also increases the likelihood of type II error conclusions (Perneger, 1998), and hence adjusting within the variables separately was considered the most reasonable approach.

6.5.4. The role of a standardized database in insomnia research

This is the largest investigated sample in terms of ERPs and primary insomnia symptomatology. The standardization of methods and inclusion/exclusion criteria across several international labs made it possible to select large and extremely well-characterised and matched samples, thus minimizing the influence of extraneous variables on task performance. In spite of these obvious benefits, the present study lacks specificity to insomnia because the database was not set up to investigate sleep disturbance. It is however worth reflecting, briefly, on the benefits of such a method and how it could be applied to insomnia research.

The insomnia field has struggled for years to compare results of investigations because of poor characterisation of patients and reporting of statistics, as well as heterogeneity of measures used to assess and monitor outcomes. Recent expert consensus work groups have tried to overcome some of these issues with the creation and publication of standard research diagnostic criteria and assessment procedures (Edinger et al., 2004; Buysse et al., 2006). It may be worthwhile going one step further, to create a system where data is collected by participating labs using existing standard assessment
procedures and dependent measures, as well as additional uniformly agreed instruments/protocols.

The ability to investigate a large number of individuals with insomnia, who have undergone a standard protocol, may prove important in confirming existing hypotheses concerning insomnia mechanisms and treatment, but also elicit new insights and discoveries. Some potential topics, out of many, that may benefit from such a database approach include: a better understanding of insomnia sub-types, their pathogenic features and responsiveness to different treatment modalities; prospective monitoring of insomnia as a risk factor for psychopathology and medical co-morbidity; consequences of insomnia in terms of integrated measures of brain structure, function and behaviour; how treatment modifies associated morbidity of insomnia; candidate genes linked to insomnia susceptibility, or specific sub-types of insomnia; and finally, powerful psychometric assessment of existing and newly developed insomnia scales and instruments. Concerning this latter point on psychometrics, it is worth mentioning that a recent multi-lab collaboration gathered and compared (retrospective) data on the Dysfunctional Beliefs and Attitudes about Sleep (DBAS; Morin et al., 2007) scale, on over 1000 patients with various sub-types of insomnia disorder (Carney et al., 2010). A *prospective* standardised approach to data collection would ensure greater comparability among participating labs, bolstering the power to address many important questions surrounding pathogenic features involved in insomnia.

**6.5.5. Future directions and concluding remarks**

Given the heterogeneity of daytime (and night-time) symptoms in insomnia, particularly hyperarousal versus hypoarousal, research should further delineate the mediating roles
of both effort and arousal (and their interaction) in explaining task performance. Multiple ERP recordings/paradigms and cognitive assessments across nights, pre- and post sleep (e.g. Bastien et al., 2008; Devoto et al., 2005; Sforza, & Haba-Rubio, 2006; Turcotte & Bastien, 2009), and at several time-points throughout the day (Varkevisser & Kerkhof, 2005), will further aid in elucidating relationships between sleep quality, arousal, and daytime performance.

Tasks found to be sensitive to insomnia impairment in recent studies, particularly those tapping, and challenging, attentional and executive functioning systems (Edinger et al., 2008a; Fernandez-Mendoza et al, in press; Altena et al., 2008a), should be used in future studies, including intervention research, to confirm sensitivity and possible predictor variables of change. Individual variability in terms of susceptibility and resilience to the effects of sleep loss should also be investigated in future work (so-called ‘trototypes’; Van Dongen et al., 2005). The collection of stress system markers, such as cortisol (Lee et al., 2007), prior to and during testing, may also help in understanding moderators of cognitive performance in those with insomnia. Finally, greater attention should be paid to the clinical complaints of patients: do current neuropsychological tests adequately capture the subjective self-report? A recent study using ethnographic observational methods, at home and in the work-place, suggests that many of the problems patients report are not covered by common testing procedures (Fox et al., 2007). It would seem important, therefore, to also consider how laboratory tests can better approximate ‘real-world’ functioning (e.g. Leufkens et al., 2009).

In sum, the current study did not reveal any evidence of cognitive impairment in a large sample of poor sleepers meeting PI symptom criteria, relative to normal sleepers. P300
latencies were however significantly reduced in the PS group, specifically at electrode site Cz. Our data may be interpreted as evidence for daytime cortical hyperarousal, and/or applied task effort, both of which may help to maintain cognitive performance and behavioural output during simple tasks designed to reveal gross brain abnormalities. Research groups should consider the merits of creating a large standardized database to further probe specific aspects of insomnia, particularly neurocognitive phenomena.

Acknowledgements

The author would like to acknowledge the support of the Brain Resource International Database (under the auspices Brain Resource; www.brainresource.com) for use of the normative data set. Thank you also to the individuals who gave their time to take part in the study.
Chapter 7:

General Discussion
7.1. Thesis findings: a synthesis

This thesis set out to explore daytime functioning and quality of life in individuals with chronic insomnia, from a number of different perspectives. Historical neglect of investigating, and measuring, insomnia-related functioning has been commented on by leading researchers in the field (e.g. Buysse et al., 2006; Morin, 2003; Riemann & Perlis, 2009). The body of work presented here attempts to further research and understanding in this important area; specifically through the application of several methodological approaches, employed across a number of different ‘experiential’ levels.

Table 7.1 - Key findings from thesis.

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Key Findings &amp; Insights</th>
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</table>
| 2       | Review and clarification of issues relevant to HRQoL measurement in insomnia  
Outlining of extensive research agenda and parameters to help standardise reporting of HRQoL scores |
| 3       | In-depth description of the limiting nature of insomnia  
First use of audio-diaries with insomnia patients  
Novel insights into aspects of insomnia illness experience |
| 4       | Development of two new scales to assess insomnia-related daytime functioning and quality of life impairment  
Preliminary demonstration of good psychometric properties and treatment responsiveness  
Consideration of alternative approaches to understanding and measuring impairment in insomnia patients |
| 5       | First application of qualitative methods to assess experiences of CBT-I  
First comprehensive documentation of CBT-I side-effects  
Effectiveness of brief behavioural intervention on both functioning and sleep  
Shed light on possible mechanisms of SRT and barriers to adherence in real time |
| 6       | Largest investigated sample in terms of ERPs and insomnia symptomatology  
First report of reduced P300 latencies in poor sleepers compared with normal sleepers  
Consideration of a database method that may help standardise insomnia assessment |

Table 7.1 details the key findings from each chapter. The utilization of novel methodologies, even within each study, generated new insights surrounding, in particular, the impact of chronically disturbed sleep; development of alternative approaches to measuring functional impact; how daytime functioning and sleep
quality/perceptions fluctuate over the course of behavioural treatment; and how patterns of objective neuropsychological performance may be influenced and explained by moderating factors. Furthermore, additional important findings emerged from this work, specifically concerning: general aspects of the insomnia illness experience; implementation of, and adherence to sleep restriction therapy for insomnia; and newly identified pathways associated with treatment response.

Results from each chapter informed and complemented subsequent data collection. For example, reviewing the insomnia-HRQoL literature revealed several inadequacies with current approaches to defining, measuring, and framing HRQoL, as applied to individuals with insomnia. To fundamentally gauge important daytime issues relevant to insomnia patients, focus-groups and audio-diaries were combined in a further study. Transcripts and phenomenological analysis then helped shape and guide novel scale development - recruiting features of both traditional nomothetic assessment and idiographic patient-centred measurement.

Given the insights obtained from the early phenomenological study (chapter three), qualitative techniques - audio-diaries and semi-structured interviews - were again utilized to probe the patient experience of sleep restriction therapy for insomnia. The newly created scales were also assessed for responsiveness in this study, providing methodological triangulation alongside sleep-related assessments, prospective qualitative diaries, and post-treatment interviews. Staying within the broad domain of daytime functioning, a next step was to investigate neuropsychological performance and P300 parameters in poor and normal sleepers. Earlier qualitative research from chapter three complemented this work: helping to formulate hypotheses that may possibly
explain neuropsychological and ERP findings. Finally, the database approach uncovered in this last experimental chapter may have particular relevance to the insomnia field; providing a method to collect data in a standardized manner across the various ‘levels’ of brain and behavioural measurement. Such a database approach may not only help further our understanding of insomnia pathophysiology but could also herald in a ‘personalised medicine’ perspective on the management of insomnia phenotypes.

7.2. Limitations: some reflections on methods and design

As this thesis demonstrates, there are clearly a number of strengths to applying qualitative, patient-centred approaches in combination with commonly-used quantitative designs. However, it is also worth discussing some potential limitations to such an approach, specifically surrounding the present body of work.

Typical of qualitative research, results are based on a small number of participants and hence unlikely to be representative of all individuals with primary insomnia. It is important to note, though, that qualitative research does not seek to be generalisable; instead focusing more on the idiographic perspective. This is why the fusion of mixed methods can be informative, strengthening perspectives on a given topic (Whitley, 2007). The incongruence between these two methodological approaches may also be of important value, perhaps suggesting a need for refinement in current assessment practices. In this regard, it will be interesting to re-visit individual accounts of patients who qualitatively voiced experiencing ‘improvement’ or ‘no improvement’ (or a worsening of symptoms) during sleep restriction therapy (chapter five), and compare,
on a case-by-case basis, subjective narratives with questionnaire and sleep diary measures.

One often-cited limitation of qualitative research is the inherent reliance on the primary researcher. The quality of data generated, particularly in a face-to-face context, is contingent on the ability of the researcher to engage with participants, making them feel comfortable enough to share their experiences in a ‘valid’ way. The researcher also inevitably influences results, because questions, and therefore analysis, can be constrained by prior knowledge, experience, and beliefs about the phenomenon under investigation. A further relevant limitation relates to possible participant self-censorship during interviews because of an established relationship with the researcher. In this case (chapter five), the author assumed the role of both therapist and interviewer/researcher; thus, if possible, it may be desirable in future work for another member within the research team to conduct interviews with patients, limiting demand characteristic behaviour on the part of the interviewee.

The central role of the researcher and emphasis on participants’ subjective experience in qualitative research is often considered a trade-off for the enhanced validity and insights of obtained findings. Analysis and results must be credible, with transparent stages of analysis and supporting quotations (Braun & Clarke, 2006). All quotations in the present thesis were independently checked for credibility. Further checks may have enhanced credibility, such as asking participants to read over findings (so-called ‘member-checking’), or requesting additional researchers to code and thematically analyse transcripts independently, and subsequently compare findings. There are also, however, limitations to these approaches that may not necessarily lead to increased
rigour; instead perhaps reflecting a naïve notion of ‘technical essentialism’ (Barbour, 2001, 2003).

The decision to use qualitative or mixed methodologies should be fundamentally determined by the research question; and not a hierarchy of evidence or methods (O’Cathain et al., 2009; Barbour, 2003). Clearly, if one wishes to primarily investigate whether sleep restriction therapy is effective in improving sleep, as a single component intervention, relative to say a simple educational pamphlet, then such a question is perhaps better addressed within the context of a randomised controlled trial. Conversely, if the topic is poorly understood, under-explored, and involves highly personal factors, such as beliefs and lived experience, then qualitative patient-centred methodologies are likely best placed to provide a foundation knowledge base to aid future hypothesis testing. Of note, the GSII reflects an approach which bridges both qualitative and quantitative paradigms; recording patient-relevant concerns in unique personal vocabulary, but still allows for the investigation of treatment effects at the level of the group. More emphasis should be placed on this type of integration in future health services research.

Additional salient caveats to drawing conclusions based on the present thesis findings relate to sample size and the need for more elaborate psychometric testing (chapter four), as well as sample composition issues (chapter six). Ideally, both the DFSAS and GSII would have been completed by a larger number of participants to thoroughly assess psychometric properties, including factor structure and test-retest reliability. Nevertheless, although small numbers, both PI patients and normal sleepers were well-defined, adding some credence to the preliminary findings. Future studies with larger
and more heterogeneous samples may help in addressing questions concerning possible insomnia sub-type differences and relationships with nightly sleep parameters. Finally, it may be important, also, to collect scale data on insomnia patients presenting for treatment in the primary care setting; the majority of participants taking part in the present research were recruited through study adverts, further limiting the generalisability of results.

Concerning chapter six, focusing on neurobehavioural and ERP parameters, it is important to bear in mind that our defined ‘poor sleepers’ may not necessarily have a problem with sleep. It is well documented that individuals with ‘poor sleep’, at least according to sleep diaries, may not consider sleep to be a significant issue and hence never feel the need to seek treatment (i.e. non-complaining poor sleepers; McCrae et al., 2003). Thus, the findings may not be generalisable to complaining primary insomnia patients. Despite this, the strengths of the database method were considered important to investigate, with potential for application to the insomnia field. This could be something that the newly formed European Insomnia Network of labs may wish to consider in their quest to elucidate mechanisms involved in insomnia.

7.3. Clinical implications and future directions

From this body of work, several ‘practice points’ emerge in relation to the clinical management of insomnia. First, it is clear that the daytime consequences of insomnia are pervasive and distressing for patients; and their amelioration should be considered a major indicator of intervention success. Both the GSII and DFSAS, though early in development, may be useful clinical tools to capture daytime functioning and quality of life impairment relevant to insomnia. Second, prior to treatment initiation, clinicians
should be aware that some patients may feel particularly isolated by their insomnia, and may have experienced negative clinician-patient interactions during previous treatment-seeking attempts. This is important because patients (chapter three) often reported being mislabelled as ‘depressed’, something which frustrated and isolated them. The prospect of initiating a ‘psychological’ therapy, then, may be viewed as a threatening concession to ‘the problem lies with me and my mind’, particularly for those trying to justify sleep disturbance as the main, real, underlying malady. This, of course, could subsequently impact treatment uptake of, and adherence to, cognitive behavioural ‘talking’ therapies. For these individuals, group therapy may be a preferred option, if available, providing a supportive forum to share experiences.

Possible dangers, potency of side-effects (and their likely time-course), and potential implementation difficulties, should be thoroughly discussed with patients prior to the initiation of sleep restriction therapy. This is likely to lead to enhanced safety awareness and adherence to behavioural guidelines. Providing individuals with ‘expert patient’ narratives may help in preparation for behaviour change, and also function as supportive material during challenging periods of sleep restriction implementation.

The findings from this thesis have wider implications for the insomnia-HRQoL research agenda. There has been a tendency for some authors to conclude (prematurely) that improving subjective sleep in insomnia patients may not be accompanied by improvements in functioning and HRQoL (e.g. Horne, 2010; Omvik et al., 2008). The likelihood, however, is that the field is only now paying due attention to the daytime complaints and experiences of patients; this is borne out in the historical poor characterisation and measurement of functioning. Similar issues have also plagued the
objective functioning literature, until recently, with subjective impairments rarely being corroborated by objective computerised testing. In the last 2-3 years there have been several important studies, with improved methodological rigour and assessment procedures, which have furthered our understanding of possible insomnia-related consequences (Shekleton et al., 2010; Riemann et al., 2007; Altena et al., 2008a, 2008b, 2010; Fernandez-Mendoza et al., in press; Edinger et al., 2008a). Hence, the argument advanced is that, similar to continued progress in elucidating abnormal sleep processes in insomnia patients (i.e. increased CAP rate, altered ERP parameters, and elevated cerebral metabolism), greater research emphasis is required to refine our knowledge and measurement of daytime experiences in those with chronically disturbed sleep.

Naturally, the characterisation of ‘the problem’ is an essential starting point and, as this thesis demonstrates, can be achieved effectively within a qualitative framework. It will be important to monitor how these aspects of the insomnia illness experience change during, and long after, effective treatment. This is where qualitative research, particularly methodologies we have piloted and used in the present body of work, can be informative. For example, do patients report a change in ‘sleeper identity’ after insomnia treatment? That is, do they now endorse being a ‘normal’ or ‘good’ sleeper? Intervention studies might show a reduction in quantitative sleep diary values, but are these important and meaningful to patients? Preliminary data from semi-structured interviews (chapter five) suggest a tentative ‘yes’, but do functioning and sleep change in the long-term, or is it more that patients cope better with poor sleep and adapt to functional impact? Essentially, does CBT-I work by engendering a ‘cognitive homeostatic’ (Ruta et al., 2007) response? These questions are difficult to probe in any
great detail with questionnaire measures, instead requiring systematic analysis of the patient perspective.

Qualitative work may also help in the design of testing procedures to objectively quantify functional impairments. A recent up-to-date thorough review of the neurobehavioural literature came to this very conclusion (Shekleton et al., 2010): subjective impairments in insomnia are not revealed by tasks sensitive to sleep loss, clearly there should be greater phenomenological focus on self-reports. In this regard, an important next step will be to re-visit the qualitative data collected in the present thesis, and focus exclusively on how individuals describe impairments in cognition and to what extent these are captured by existing measurement protocols. One salient requirement is for an ecologically valid task to capture memory deficits experienced by insomnia patients. For example, patients tend to report impairments in remembering to carry out delayed intentions, such as forgetting to complete particular chores. Whereas commonly used tasks tend to focus exclusively on encoding and working memory deficits, it seems that patients are more likely to experience prospective memory difficulties, a largely executive functioning-mediated phenomenon, which places particular demand on attentional shifting (e.g. Groot et al., 2002). A paradigm to capture this type of prospective remembering (e.g. Rendell & Henry, 2008) would be worthwhile pursuing.

Related to this, a poster presented at the 2007 international meeting for the Association of Professional Sleep Societies (APSS) described the application of ethnographic methods, involving direct observation of people in their homes and workplace, to achieve a better insight into experienced functional impairment (Fox et al., 2007). This
work revealed that several aspects of patients’ daytime complaints are not covered by existing ‘gold standard’ neuropsychological tests. Innovative application of ‘bottom-up’ methodologies like those piloted by Fox and colleagues hold considerable promise in aiding the future refinement of testing procedures. One final way to enhance validity of both objective and subjective measures would be to systematically ask patients to comment on whether assessments capture their experience in a relevant way. This approach has proved fruitful, for example, in the early design stages of new HRQoL scales (Paterson, 2004), and has recently been suggested as an important next step in the design and refinement of instruments to assess the poorly understood construct of ‘non-restorative’ sleep (Vernon et al., in press).

Concerning measurement of subjective functioning, there are a number of promising directions. For example, what are the best predictors of change on our newly developed quality of life and functional impairment scales? If primary insomnia is a problem of hyperarousal, does treatment, firstly, reduce arousal parameters (Bonnet & Arand, 2010), and do these changes map onto perceptions of sleep quality and subsequent daytime functioning/HRQoL improvements? It will be important to assess factors relevant to the insomnia syndrome as well as the individual patient. In light of the present ERP findings (chapter six), and others’ (e.g. Devoto et al., 2005; Bastien et al., 2008), it will be important to assess relationships between self-report measures of functioning, neuropsychological testing and evoked potentials; testing out the concept of an ‘inverted U’ performance curve more thoroughly (Horne, 2010). Indeed, this is where a prospective standardized database may be most useful. Despite the heterogeneity of insomnia phenotypes, sub-types, and, unsurprisingly, research findings, authors still tend to make grand conclusions about ‘what insomnia is’. The ability to
record extensive data, spanning self-report sleep, PSG-recordings, cognitive process measures, DNA polymorphisms, subjective and objective functioning variables, and structural/functional imaging, for a single individual, may help to identify clusters of patients with similar characteristics; paving the way for a better understanding of the mechanisms involved in the genesis of ‘the insomnias’, and ultimately in the tailoring of treatments. Although a lofty task, the Brainnet database discussed in chapter six suggests an integrative approach is feasible and worthwhile.

Methods developed and applied within this thesis have research implications beyond daytime functioning and HRQoL measurement. These are worth touching on, briefly. For example, what are the thoughts, behaviours, and experiences of those with acute (adjustment) insomnia, experienced in the context of a life stressor? It is argued that the transition from acute to chronic insomnia may be mediated by maladaptive sleep-disruptive cognitive processes and behavioural practices; there are, however, little data on this. Collecting real time audio-diary entries, longitudinally, or post-acute insomnia episode reflections (semi-structured interviews), might help further understanding of relevant factors (and their time course) that contribute to a return to normal sleep or alternatively the development of chronic sleep difficulties. Such topics are important yet poorly understood and under-investigated, and would benefit from a mixed method approach.

Another interesting angle could be to investigate subjective descriptions of the sleep period itself in insomnia patients. Proximal reflections immediately on awakening could prove informative in understanding, for example, dream phenomenology. Feige et al. (2008) recently reported the intriguing finding that REM sleep time was positively
related to greater objective-subjective sleep discrepancies; perhaps indicating that dream activity reflects waking concerns of patients, leading to the subjective perception of being awake. There is preliminary evidence for REM sleep hyperarousal in PI patients, as indexed by absolute and relative cerebral metabolic rate of glucose (Germain et al., 2007). Very few studies have systematically looked at dream content in insomnia, but subjectively recorded accounts from patients, after forced or natural awakenings (at home and/or in the lab), over several nights, may help clarify if there is a robust link between what patients experience/think about during the day (audio-diary entries) and subsequent sleep mentation. Qualitative research would be a methodological asset here.

7.4. Concluding remarks

The body of work presented in this thesis addresses the important concepts of daytime functioning and quality of life as applied to insomnia. The development and application of mixed methodologies paved the way for insights that arguably would have been unobtainable using conventional quantitative and psychometric procedures. The emergent findings described within have significant implications for insomnia clinical practice and research. Developed methodological approaches may have further utility within the field of sleep medicine and research.
Appendices

Appendix A: University of Glasgow Sleep Centre interview schedule

University of Glasgow Sleep Research Group
Sleep Interview schedule

ID 
Date:

Age:

Marital status: married; single; divorced; widowed

Occupation:

Does this involve working nightshifts?

Retired: Yes; No

Years of Education:

Gender: Male/Female

1. How long have you had your sleep problem (number of years/months)?

   i) Did this start before you were 10 years old?

   ii) If yes to i), was there any identifiable precipitant to this?

   iii) If yes to i) have your experience of sleeping difficulties been persistent since this time?

2. How many nights per week do you have difficulties sleeping?

3. How much sleep do you get on an average night?

4. Do you have difficulty falling asleep at night?

5. On average, how long (hours/minutes) does it take you to fall asleep each night?

6. Do you have difficulty staying asleep?
7. On average, how long in total (hours/minutes) are you usually awake at night (after you have been asleep for the first time)?

8. Are you ever awake all night (not intentionally)?

   If yes, how often?

   After such nights, do you nap the next day?

9. Occasionally, do you find that you get what you would consider a 'normal' night's sleep?

   If yes, how often?

10. On average, what time do you wake up in the morning?

11. On average, how long (hours/minutes) do you wake before you are required to?

12. How would you rate the quality of your sleep

    Very good  Fairly good  Fairly bad  Very bad

13. Other than difficulties in sleeping, is there anything that prevents you getting as much sleep as you would like e.g. having a young child, a partner who snores?

14. Do you experience any of the following in the day time due to your sleep problem?

    Fatigue/malaise  Yes/no

    Attention, concentration, or memory impairment  Yes/no
Social/vocational dysfunction or poor school performance \hspace{1cm} Yes/no

Mood disturbance/irritability \hspace{1cm} Yes/no

Daytime sleepiness \hspace{1cm} Yes/no

Motivation/energy/initiative reduction \hspace{1cm} Yes/no

Proneness for errors/accidents at work or while driving \hspace{1cm} Yes/no

Tension, headaches and/or GI symptoms in response to sleep loss \hspace{1cm} Yes/no

Concerns or worries about sleep \hspace{1cm} Yes/no

15. \hspace{1cm} Do you feel that you spend a disproportionate amount of time thinking and worrying about your sleep? \hspace{1cm} Yes/no

16. \hspace{1cm} Do you have difficulty falling asleep in bed at the desired bedtime or during planned naps, but fall asleep with relative ease during monotonous activities e.g. watching TV or reading when not intending to sleep? \hspace{1cm} Yes/no

17. \hspace{1cm} Do you find that you sleep better away from home than at home? \hspace{1cm} Yes/no

18. \hspace{1cm} Do you experience persistent thoughts in bed when trying to sleep that seem to come out of the blue? \hspace{1cm} Yes/no

19. \hspace{1cm} Do you experience ‘racing’ thoughts in bed that prevent you falling asleep as they are impossible to control? \hspace{1cm} Yes/no

20. \hspace{1cm} Do you find it almost impossible to relax enough in order to fall asleep at bedtime? \hspace{1cm} Yes/no

21. \hspace{1cm} Does pain or physical discomfort interrupt your sleep at night? \hspace{1cm} Yes/no

\hspace{1cm} If yes, does taking pain medication help this? \hspace{1cm} Yes/no

21. \hspace{1cm} Do you think that pain is the main cause of your sleeping problem?
22. Do any other physical health problems interrupt your sleep at night? Please give details.

23. Have you suffered from any physical health problems in the past that have disrupted your sleep? Yes/No

24. When did your current sleeping difficulties occur in relation to these physical health problems?

25. Have you felt particularly ‘down’ or ‘sad’ or ‘empty’ about things in the past two weeks?
   □ Never □ Sometimes □ Usually □ Always

26. Does your doctor know about this?

27. Are these low feelings a change from what is normal for you?

28. When did these feelings start?

29. In the past two weeks:
   Have you lost pleasure in your usual activities? Yes/No
   Has your appetite changed? Yes/No
   Have you lost or gained weight? Yes/No
   Have you lacked energy? Yes/No
   Have you felt agitated? Yes/No
   Have you felt slowed down? Yes/No
   Have you thought about harming yourself? Yes/No
   Have you felt excessively guilty or worthless? Yes/No
Has your concentration been poor? Yes/No

30. Are you currently being treated for depression? If yes, please give details.

31. Are you currently suffering from emotional or mental health problems (if not mentioned above)? Yes/No
   Please provide some details

32. Have you suffered from emotional or mental health problems in the past?

33. When did your current sleeping difficulties occur in relation to these mental health difficulties?

34. Please provide details of any medication that you are currently taking (or have been taking in the past three months) for sleep difficulties or any other purpose.

35. Are you currently receiving psychological help for sleeping difficulties? Yes/no

36. Do you drink alcohol? Yes/No
   If yes, what do you usually drink?
   How many of these alcohol drinks do you usually have per day (if not daily, how many in a week)?
   Do you feel that your alcohol use disrupts your sleep?
37. Do you use illicit drugs? Yes/No
   If yes, what do you usually use?
   How much do you have in a day (if not daily, in a week)?
   Do you feel that your drug use disrupts your sleep?

38. Do you find that you fall asleep at the ‘wrong’ time?
    □ Never □ Sometimes □ Usually □ Always

39. In what way do you feel that it is wrong?

40. Do you have difficulties waking up at a set time in the morning (or would you if you are currently not required to) in order for example to be ready to leave for work at for 8.15 am? Yes/No
   If yes, in what way (e.g. keep falling asleep again, feel you need to sleep more due to a bad night)?

41. If you had no social constraints (e.g. work) is there particular times that would optimise your chances of having a good sleep? Yes/No
   If yes, what would these be?
   Sleep at.......... Get up at.......... 

42. Do you snore during your sleep?
    □ Never □ Sometimes □ Usually □ Always

43. If you do, how do you know? For example, has your partner been awoken?

44. Do you hold your breath, have breathing pauses or stop breathing in your sleep?
    □ Never □ Sometimes □ Usually □ Always
If you do, how do you know? For example, has your partner noticed this?

45. Do you think breathing difficulties may be the main cause of your sleeping problem? Why do you think this is the case?

46. Do you fall asleep unintentionally or have to fight to stay awake during the day?

☐ Never  ☐ Sometimes  ☐ Usually  ☐ Always

47. When you wake up in the morning do you feel unrefreshed and groggy?

☐ Never  ☐ Sometimes  ☐ Usually  ☐ Always

48. Please indicate how likely you are to fall asleep in the following situations. Use the scale below to indicate how likely you are to fall asleep in each situation:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

<table>
<thead>
<tr>
<th>Situation</th>
<th>Record chance of dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
</tr>
<tr>
<td>Watching TV</td>
<td></td>
</tr>
<tr>
<td>Sitting, inactive in a public place (e.g. a theatre or a meeting)</td>
<td></td>
</tr>
<tr>
<td>As a passenger in a car for an hour without a break</td>
<td></td>
</tr>
<tr>
<td>Lying down in the afternoon to rest when circumstances permit</td>
<td></td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td></td>
</tr>
<tr>
<td>Sitting quietly after lunch without alcohol</td>
<td></td>
</tr>
<tr>
<td>In a car, while stopped for a few minutes in the</td>
<td></td>
</tr>
</tbody>
</table>
49. Do you experience repeated, uncontrollable leg jerks or leg twitches during your sleep?
   □ Never  □ Sometimes  □ Usually  □ Always

50. Do you think such uncontrolled limb movements may be the main cause of your sleeping problem?

51. Do you experience restless or “crawling” feelings in your legs at night which go away if you move your legs?
   □ Never  □ Sometimes  □ Usually  □ Always

52. Do you think such restless legs may be the main cause of your sleeping problem?

53. Do you experience excessive sleepiness and sudden muscle weakness?

54. Do you experience any of the following:
   - nightmares
   - inability to perform voluntarily movements before going to sleep, or upon wakening
   - grinding/clenching teeth
   - sleepwalking/talking
   - night terrors (waking up screaming or with an intense feeling of terror)
   How often?

55. Do you feel you want to sleep at the wrong time? – too early? Too late?

56. Do you feel that you have any other difficulties related to sleeping that have not been covered in this interview?

57. Are you physically well at present?

58. Are you attending your GP for any reason at present?
Appendix B: Focus group core questions

Focus Group Questions

Core questions

➢ What daytime effects do you experience after a poor night’s sleep?

➢ Can you give some examples of how these affect important areas of your life?
   Such as:
   (a) relationships
   (b) work
   (c) social life

➢ What things in your life remain unaffected after a poor night’s sleep?

➢ How do you cope with sleeping poorly so you can get through the day?

➢ How would things be different if your insomnia were to stop tomorrow?
Appendix C: Participant instructions for operating dictaphone (audio-diary)

**Dictaphone Instructions**

To turn on:

- On the back of the Dictaphone.
- Pull the ‘HOLD’ button downwards
- Then, push the ‘POWER’ button to the right (‘on’)

To record:

When you are ready to speak:

- On the side of the Dictaphone, at the top, press the red ‘REC’ button. A red light will come on during recording.
- Speak into the recorder (hold approx. 6 inches from mouth).
- Please do not move the Dictaphone when recording.

If you feel you have to pause during the diary entry for any reason press the ‘REC’ button for a second time. A flashing ‘PAUSE’ message will appear on the screen. To continue recording press the ‘REC’ button.

- When you feel you have covered everything, press the ‘STOP’ button located on the side of the recorder.

To turn off (after each diary entry):

- On the back of the Dictaphone
- Push the ‘HOLD’ button upwards
- Then finally pull the ‘POWER’ button to the left (‘off’)

Each time you record an entry, this will be stored as a single file. The number of files recorded can be read from the two numbers in the centre of the screen.

**IMPORTANT:** Please do not press any other buttons on the Dictaphone, and if you have any problems or difficulties call me on 0141 232 7699.

MANY THANKS.
Appendix D: Audio-diary entry guidelines

Morning Instructions
(1.) Paper Sleep Diary
Dear Participant, every morning when you get up could you please complete the paper sleep diary for the previous night. For example, if starting on a Monday morning you would fill in the information for the Sunday night.

(2.) Audio Diary
As well as the details put in the paper diary we would also like to know how YOU FEEL about your sleep. Can you please take a few minutes to speak into the recorder and describe what last night’s sleep was like from your point of view.....

Please feel free to record for as long as you want and include any information that you think is relevant or interesting.

THANK YOU!
Evening Instructions

Dear Participant,
Approximately two hours before going to bed can you take a few minutes to think about today – what happened and how did you feel....

Can you please now take a few minutes to speak into the recorder and describe any events or feelings that you experienced today that you think were due to the way you slept last night....
If you had a good sleep last night can you describe how things were different today from when you have a bad night’s sleep....

Please feel free to record for as long as you want and include any information that you think is relevant or interesting.

THANK YOU!

-------------------------------------------------------------------------------------------------------------------------------
Appendix E: Glasgow Sleep Centre brief screen protocol

**Source**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>How did you find out about the University of Glasgow Sleep Centre?</td>
<td></td>
</tr>
<tr>
<td>Why have you contacted us?</td>
<td></td>
</tr>
<tr>
<td>Method of initial contact (mobile, email, office phone)?</td>
<td></td>
</tr>
</tbody>
</table>

**Personal**

<table>
<thead>
<tr>
<th>Personal Details</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Name:</td>
<td></td>
</tr>
<tr>
<td>Date of Birth:</td>
<td></td>
</tr>
<tr>
<td>Age:</td>
<td></td>
</tr>
<tr>
<td>Telephone:</td>
<td></td>
</tr>
<tr>
<td>Address:</td>
<td></td>
</tr>
<tr>
<td>Alternative Telephone:</td>
<td></td>
</tr>
<tr>
<td>When is a good time to call?</td>
<td></td>
</tr>
<tr>
<td>What GP practice do you attend, and who is the GP you normally see?</td>
<td></td>
</tr>
</tbody>
</table>

**Sleep**

<table>
<thead>
<tr>
<th>Sleep Details</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have difficulty sleeping at the moment? (Y/N)</td>
<td></td>
</tr>
<tr>
<td>Have you always been a poor sleeper? (Y/N)</td>
<td></td>
</tr>
<tr>
<td>How long have you had a sleep problem? (yr)</td>
<td></td>
</tr>
<tr>
<td>Do you have difficulty falling asleep? (Y/N)</td>
<td></td>
</tr>
<tr>
<td>How many nights per week do you have difficulty falling asleep? (out of 7)</td>
<td></td>
</tr>
<tr>
<td>How long does it normally take you to fall asleep? (min)</td>
<td></td>
</tr>
<tr>
<td><strong>Do you have a difficulty with waking up during the night? (Y/N)</strong></td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td><strong>How many nights per week do you have a difficulty with waking up during the night? (out of 7)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>How long are you normally awake during the night, in total? (min)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>What time do you normally go to bed? (time)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>What time do you normally get up? (time)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>How long do you normally sleep? (hr/min)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Do you have any other difficulties with your sleep (e.g. restless legs, breathing problems, sleep walking)?</strong></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Do you work shifts, night shifts?</strong></th>
</tr>
</thead>
</table>

| **Roughly, how many units of alcohol do you drink per week?**  
(Remember: One standard (175ml) glass of wine = 2 unit  
One pint of standard lager = 2.3 units  
Spirit & Mixer = 1 unit) |
<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Does your sleep disturbance affect how you feel and function during the day (e.g. fatigue, sleepiness, concentration, memory, mood, motivation, irritable, work/social functioning etc.). If yes, specify most salient.</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Health</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Do you keep in good physical health? (Y/N)</strong></td>
</tr>
<tr>
<td><strong>What physical health problems do you have (if applicable)?</strong></td>
</tr>
<tr>
<td><strong>What medicines do you take for your physical health? (if applicable)</strong></td>
</tr>
<tr>
<td><strong>Do you keep in good mental health? (Y/N)</strong></td>
</tr>
<tr>
<td><strong>What mental health problems do you have (if applicable)?</strong></td>
</tr>
</tbody>
</table>
What medicines do you take for your mental health? (if applicable)

Notes

For Office Use

Enquiry taken by:
At (time):
On (date):

Information sent:
☐ [study name] ☐ [study name] ☐ [study name] ☐ [study name]
☐ [study name] ☐ [study name] ☐ [study name] ☐ [study name]
☐ [study name] ☐ [study name] ☐ [study name] ☐ [study name]

On (date):
Appendix F: Treatment PowerPoint slides

Bed restriction Therapy

How much sleep do I need?
- Everyone’s sleep needs are different, but people with insomnia often have very irregular sleep.
- This makes it difficult to know how much sleep on average you are getting.
- Use your diary to work out an average.

Working out your average sleep time

<table>
<thead>
<tr>
<th>Day</th>
<th>Amount Slept</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6 hr 30 min</td>
</tr>
<tr>
<td>2</td>
<td>3 hr 30 min</td>
</tr>
<tr>
<td>3</td>
<td>5 hr 15 min</td>
</tr>
<tr>
<td>4</td>
<td>6 hr 15 min</td>
</tr>
<tr>
<td>5</td>
<td>4 hr</td>
</tr>
<tr>
<td>6</td>
<td>6 hr</td>
</tr>
<tr>
<td>7</td>
<td>5 hr 30 min</td>
</tr>
</tbody>
</table>

Total amount slept = 37 hrs or 2220 mins

Avg sleep time = 5 hrs 17 mins or (347 mins)
= 5hrs 15 mins, to nearest 1/4 hr

Step 1

Getting your sleep into a regular pattern

- ‘Anchor’ your sleep around a morning rising time, e.g. 7am. We call this a ‘set’ rising time.
- Minus your average sleep time e.g. 5 1/4 hrs.
- This gives your ‘threshold time’ e.g. 01:45am; after this you can go to bed.

- You can adjust to preferred threshold and rising time e.g. threshold 00:45, then rising time would be 6am.
- Important to keep a minimum of 5 hours sleep time!

E.g.
Average sleep time: 4hr 30 minutes
Set rising time: 7:00 am
Threshold time: 7:00am – 5hrs=2:00am

289
Steps 2 & 3

Improving your ‘sleep efficiency

• Sleep efficiency is simply how much of the time you spend in bed is spent sleeping
  e.g. Go to bed: 11:00pm  
       Get to sleep: 12:30am
       Wake up: 6:30am
       Time asleep: 6 hours
       Time in bed: 7.5 hours
  
       Sleep efficiency = 6 / 7.5 x 100 = 80%

Step 4

Bed Restriction

Aim: To sleep for at least 90% of the time you spend in bed

✓ Step 1: Work out your current average sleep
✓ Step 2: Decide on a rising time
✓ Step 3: Work out your threshold time
✓ Step 4: Calculate current sleep efficiency

• Now you must stick to your pattern seven days a week!

When will I start to get more sleep?

• Once you are sleeping 90% of your time in bed
  - Increase your time in bed by 15 minutes
• Stay with this pattern for at least a week
• Make sure that you keep your sleep efficiency at or above 90%
• We will review your progress each week for the next 4 weeks
What do I do with this extra evening time?
- Continue with normal daily tasks, whatever you want to do
- But do not fall asleep before ‘threshold time’

What if I take sleeping pills?
- Continue as normal unless you feel you are able to get through the night without them
- Safety: do not take risks.

Can I nap during the day?
- Napping can reduce the effectiveness
- Only take a short nap (15-20 mins) if you are struggling to stay awake during the day.
- Safety: avoid taking risks

Can I really make the change?
- Bed restriction is tough
- Surgical treatment of sleep pattern
- Work hard and stay positive

The Wheel of Motivation

Step 5

Making these changes
- Changes can be very difficult to make
- ‘Old habits die hard!’
- Weekends can be difficult
- Try to stick with the programme and stay motivated
- Replace: ‘I am never going to get a good night’s sleep’ with ‘This is hard to break, but I am going to keep on trying’

Your New Routine
- Stay up until your threshold time
- Turn out the light when you get into bed
- Get up in the morning at your set time
- Follow this programme 7 days/nights a week!
Appendix G: Seven-day sleep diary

SLEEP DIARY

Name ___________________________________ Day 1: _______________________

The sleep diary is designed to provide a record of your sleep pattern as well as how you feel on awakening. Please complete one column of the diary each morning, soon after you wake up. Take a few minutes to do this, trying to be as accurate as you can. It is your best estimate that we are looking for, but try not to get into the habit of clockwatching at night.

<table>
<thead>
<tr>
<th>MEASURING THE PATTERN OF YOUR SLEEP</th>
<th>DAY 1</th>
<th>DAY 2</th>
<th>DAY 3</th>
<th>DAY 4</th>
<th>DAY 5</th>
<th>DAY 6</th>
<th>DAY 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Did you nap at any point yesterday? If yes, how long for (minutes)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. At what time did you rise from bed this morning?</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. What time did you wake up at this morning?</td>
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<td></td>
<td></td>
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<tr>
<td>4. At what time did you go to bed last night?</td>
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</tr>
<tr>
<td>5. What time did you switch off the light at intending to go to bed?</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>6. How long did it take you to fall asleep (minutes)?</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>7. How many times did you wake up during the night?</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>8. How long were you awake during the night (in total)?</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>9. About how long did you sleep altogether (hours/minutes)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>10. How much alcohol did you have last night?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Did you take sleeping pills to help you sleep last night? If so, how many?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MEASURING THE QUALITY OF YOUR SLEEP</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How rested / restored do you feel this morning?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>not at all</td>
<td>moderately</td>
</tr>
<tr>
<td>2. How alert do you feel this morning?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>not at all</td>
<td>moderately</td>
</tr>
<tr>
<td>3. How would you rate the overall quality of your sleep last night?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>poor</td>
<td>very good</td>
</tr>
</tbody>
</table>
Appendix H: Occupational Impact of Sleep Questionnaire (OISQ)

Name ___________________________ Date ___________________________

**Occupational Impact of Sleep Questionnaire**

*Quality of sleep can influence our ability to perform in the workplace.* The following questions relate to ways in which your work performance may have been affected by your sleep **during the past 4 weeks**. Please indicate (✓) how often each item applied to you. Answer all the questions.

**During the past 4 weeks, how often did the quality of your sleep make it difficult for you to:**

<table>
<thead>
<tr>
<th></th>
<th>All of the time</th>
<th>Most of the time</th>
<th>Some of the time</th>
<th>A little bit of the time</th>
<th>Never/Not Applicable</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Wake up for work on time?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Arrive at work on time?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Work the required number of hours?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Get going easily at the beginning of the workday?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Start on your job as soon as you arrive at work?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Do your work without stopping to take breaks or rests?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Keep working effectively during the afternoon?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Maintain your stamina throughout the day?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Keep to a routine or schedule?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Think clearly when working?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please continue over page
During the past 4 weeks, how often did the quality of your sleep make it difficult for you to:

<table>
<thead>
<tr>
<th></th>
<th>All of the time</th>
<th>Most of the time</th>
<th>Some of the time</th>
<th>A little bit of the time</th>
<th>Never/Not Applicable</th>
</tr>
</thead>
<tbody>
<tr>
<td>11. Keep your mind on your work?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Do work carefully?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>13. Concentrate on your work?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Work without losing your train of thought?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Easily read or use your eyes when working?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Speak with people in-person, in meetings or on the phone?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Control your temper around people when working?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Help other people to get work done?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Handle the workload?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. Work fast enough?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21. Finish work on time?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. Do your work without making mistakes?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Feel you have done what you are capable of doing?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24. Gain satisfaction from your work?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix I: Side-effects checklist

**Symptom Checklist**

Dear participant, can you go through each item below and indicate whether taking part in the actual bed restriction programme resulted in any of the following unwanted symptoms. Can you also rate to what extent they interfered with normal functioning (i.e. how you felt before starting treatment).

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Did you experience…</th>
<th>IF YES, to what extent did it interfere with everyday functioning. Please circle the most appropriate response.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Mood</td>
<td>(Y / N)</td>
<td>Not at all</td>
</tr>
<tr>
<td>Fatigue/Exhaustion</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Extreme Sleepiness</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Feeling Agitated</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Bodily Pain</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Headache/Migraine</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Euphoria/Intense Increase in mood</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Reduced Motivation/Energy</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Changes in Hunger/Appetite</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Blurred Vision</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
<td>Not at all</td>
</tr>
<tr>
<td>Feeling irritable</td>
<td></td>
<td>Not at all</td>
</tr>
</tbody>
</table>

If you experienced any other unwanted symptoms that are not listed above can you please write them in the box below:

---

295
Appendix J: Sleep Restriction Adherence Scale (SRAS)

<table>
<thead>
<tr>
<th>Sleep Restriction therapy</th>
<th>None of the time</th>
<th>A little of the time</th>
<th>Some of the time</th>
<th>A good bit of the time</th>
<th>Most of the time</th>
<th>All of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I followed the sleep scheduling programme...</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>I went to bed at my calculated ‘threshold time’ on:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) weekdays</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>(b) weekends</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>I got up at my calculated ‘rising time’ on:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) weekdays</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>(b) weekends</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
Appendix K: Sleep restriction therapy audio-diary guidelines

Audio-diary

Morning Instructions

(1) Paper Sleep Diary
Dear Participant, every morning when you get up could you please complete the paper sleep diary for the previous night. For example, if starting on a Monday morning you would fill in the information for the Sunday night.

(2) Audio-Diary
As well as the details put in the paper diary we would also like to know how you feel about your sleep and new bed restriction programme. Can you please now take a few minutes to speak into the recorder and describe:

- the experience of implementing your new bed-time and rising time…
AND
- what was the quality of your sleep like from your point of view, how do you feel this morning?

Please feel free to record for as long as you want and include any information that you think is relevant or interesting.

Thank you!

Please turn over…………….
Evening Instructions

Approximately two hours before going to bed can you take some time to think about today – what happened and how did you feel…

Can you please now take a few minutes to speak into the recorder and describe:

(1)
- how your day has gone, were there any events or feelings you experienced today because of the quality of your sleep last night….

AND

(2)
- how your new bed restriction programme affected your ability to function, for better or for worse….

Please feel free to record for as long as you want and include any information that you think is relevant or interesting.
Appendix L: Post-sleep restriction interview (core) schedule

Semi-structured Interview

- Can you explain what made you decide to take part in the treatment study?

**Treatment expectations/credibility**

- What were your initial thoughts about Bed Restriction when it was first explained to you?

Possible supplementary prompts: in terms of.... making sense?.... Potential for improvements in your sleep?

**Experience of treatment**

- How does bed restriction compare to treatments that you have tried in the past?

- Can you describe the experience of putting your new sleep-waking schedule into practice? And any obstacles you encountered?

- What impact did bed restriction have on how you felt during the day when you first started the treatment programme?

Prompts: can you describe any unwanted symptoms that you experienced during the programme? How do you think you will get on with the programme after the study has finished?

**Impact of treatment on insomnia and functioning/sleep**

- What effect has bed restriction had on your sleep?

Prompts: In terms of – quality, duration, time to get to sleep, awakenings? Can you recall when you started to notice changes in your sleep?

- Can you describe if, and how, bed restriction has made a difference to how you function/feel during the day? Compared to how you felt before you started the treatment programme.

- What effect, if any, has the treatment had on important areas of your life?

Prompts: such as work performance, or social/ family relationships? What have others (friends/family) said about changes either in your sleep or how you have been feeling/behaving during the day?
Appendix M: Sleep history from BRID protocol

Only complete this form if you answered YES to the following question in the CORE section.

“In the last month, have you experienced, or have you been told about any of the following sleep symptoms - difficulty in falling asleep at night; frequent night awakenings; breathing difficulties, snorting, gasping or loud snoring?”

During the last month how often have you had, or been told about, the following symptoms?

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
</table>
| 1. Snorting or gasping | □ Never  
  □ Rarely, less than once per week  
  □ 1-2 times per week  
  □ 3-4 times per week  
  □ 5-7 times per week  
  □ Don't know |
| 2. Loud snoring | □ Never  
  □ Rarely, less than once per week  
  □ 1-2 times per week  
  □ 3-4 times per week  
  □ 5-7 times per week  
  □ Don't know |
| 3. Breathing stops, choke or struggle for breath | □ Never  
  □ Rarely, less than once per week  
  □ 1-2 times per week  
  □ 3-4 times per week  
  □ 5-7 times per week  
  □ Don't know |
| 4. Frequent awakenings | □ Never  
  □ Rarely, less than once per week  
  □ 1-2 times per week  
  □ 3-4 times per week  
  □ 5-7 times per week  
  □ Don't know |
| 5. Tossing, turning or thrashing | □ Never  
  □ Rarely, less than once per week  
  □ 1-2 times per week  
  □ 3-4 times per week  
  □ 5-7 times per week  
  □ Don't know |
<table>
<thead>
<tr>
<th></th>
<th>Difficulty falling asleep</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ Never</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Rarely, less than once per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 1-2 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 3-4 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 5-7 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Don’t know</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Legs feel jumpy or jerky</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>□ Never</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Rarely, less than once per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 1-2 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 3-4 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 5-7 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Don’t know</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Morning headaches</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ Never</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Rarely, less than once per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 1-2 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ 3-4 times per week</td>
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</tr>
<tr>
<td></td>
<td>□ 5-7 times per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>□ Don’t know</td>
<td></td>
</tr>
</tbody>
</table>
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Espie, C.A. (2008). Treating insomnia with CBT: should we step up to stepped care? Keynote lecture delivered at the 22nd annual meeting of the Association for Professional Sleep Societies (APSS), June 9-12, Baltimore, USA.


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