Summary

The present thesis describes the complex relationship between insomnia, psychiatric comorbidity and other psychosocial factors. It highlights the importance of psycho-diagnostics in the evaluation of chronic insomnia. Furthermore, it describes how personality and coping research might attribute to an understanding of different phenotypes of insomnia as well as support the enhancement of quality of life in patients with insomnia and might guide the prediction of treatment results.

Chapter 1 is the general introduction of this thesis and describes the background of our study and research questions. The pathophysiology, nosological classification and treatment of insomnia and the relationship with personality traits and psychiatric disorders are discussed.

In **chapter 2** "The role of personality traits in insomnia" we reviewed studies on insomnia and personality and concluded that neuroticism, internalization and anxious personality traits characterize insomniacs in general. However, due to a lack of longitudinal data and other methodological issues, such as small sample sizes and lack of psychodiagnostics it was difficult to conclude whether these personality traits are predisposing for insomnia. Specific differences in personality traits might just as well be a consequence of insomnia. In addition, certain personality traits may be related to the response to (cognitive) behavioral treatment. We also stressed that personality should not be viewed as a single predisposing factor, but should be assessed as a part of a larger group of interacting psychological and physiological factors involved in the predisposition to and perpetuation of chronic insomnia. For future research we recommended longitudinal studies including different psychosocial variables and clear psychodiagnostic assessment of the research population.

In **chapter 3** "Subjective sleep characteristics in insomnia with and without psychiatric comorbidity" we discussed differences between subjective sleep diary variables between three groups. We found that patients with psychiatric comorbidity do not necessarily show more severe subjective complaints. Patients with comorbid anxiety disorders showed a sleep efficiency that was significantly higher than patients with primary insomnia. Also they showed a sleep efficiency-score that was more than 20% higher than patients with comorbid mood disorders. While subjective total bedtimes were comparable, the total sleep time was on average 2 hours longer in patients with comorbid anxiety disorders than in patients with comorbid mood disorders. We also found evidence that the main problem in patients with comorbid anxiety disorders involves sleep fragmentation. These results show that tailored CBT-I treatment may vary according to the presence and type of psychiatric comorbidity. Patients with comorbid anxiety disorders might benefit more from sleep restriction while patients

with primary insomnia and comorbid depressive disorders might show better results through stimulus control procedures.

In **chapter 4** "General quality of life in insomnia with and without psychiatric comorbidity" we examined psychosocial factors and sleep variables and their possible contribution to a higher quality of life in insomniacs. We found that the association of these variables with general quality of life (QOL) varies according to the presence of psychiatric comorbidity. Remarkably, the ISI-score was not associated with QOL in primary insomniacs. On the other hand, fatigue was negatively associated with QOL and seems to be more important for general well-being than subjective sleep diary measures such as sleep efficiency or total sleep time in both groups. High discrepancies in social support and low Extraversion are associated with lower QOL in primary insomniacs. Including an assessment of these factors during intake procedures might guide more tailored treatments in which (social) activation might enhance QOL in these patients. In patients with comorbid insomnia, insomnia severity (ISI-score) was negatively associated with QOL as well as the anxiety and depressive symptoms (HADS-score). This stresses the importance of adding CBT-I elements in the treatment of psychiatric disorders in patients who also suffer from insomnia.

Chapter 5 "Psychiatric comorbidity and coping predict cognitive behavioral treatment effect" focused on the prediction of CBT-I treatment effect by examining personality traits, coping and social support. We found that the presence of a comorbid psychiatric disorder predicts worse treatment results. In our results we found a trend that patients who are following current treatment for their comorbid psychiatric disorder might benefit more from CBT-I than patients who are not engaging in current psychological treatment for the comorbid psychiatric disorder. Also, we found that high refocus on planning independently predicts worse CBT-I treatment results. If a comorbid psychiatric disorder is present, we suggest that it is better to integrate cognitive behavioral treatment for insomnia with psychological treatment for the comorbid psychiatric disorder and not exclusively treat the insomnia problem. Adding specific treatment strategies focusing on the decrease of controlling behavior and thoughts around sleep en promoting acceptance of the sleep problem might gain better treatment results in patients with high refocus on planning.

In **chapter 6** "Phenotypes of sleeplessness: stressing the need for psychodiagnostics in the assessment of insomnia" we used cluster analysis to distinguish different phenotypes in patients with insomnia. Our findings stress the need for psycho-diagnostic procedures next to a sleep-related diagnostic approach, especially in younger insomnia patients. We identified three clusters. The "moderate insomnia with low psychopathology"-cluster was characterized by relatively normal personality traits, as well as normal levels of anxiety and depressive symptoms in the presence of moderate insomnia severity. The "severe insomnia with moderate psychopathology"-

cluster showed relatively high scores on the Insomnia Severity Index and scores on the sleep log that were indicative for severe insomnia. Anxiety and depressive symptoms were slightly above the cut-off and they were characterized by below average self-sufficiency and less goal-directed behavior. The “early onset insomnia with high psychopathology”-cluster showed a much younger age and earlier insomnia onset than the other two groups. Anxiety and depressive symptoms were well above the cut-off score and the group consisted of a higher percentage of subjects with comorbid psychiatric disorders. This cluster showed a “typical psychiatric” personality profile. Specific treatment suggestions are given based on the three phenotypes.

In **Chapter 7** we discussed the implications of our findings for clinical practice. We concluded that there are strong personality and subjective sleep differences in patients with and without psychiatric comorbidity. Also, there are other factors correlated with general quality of life depending on the presence of psychiatric comorbidity. Therefore, the term insomnia disorder is much too broad and we suggested the introduction of the term comorbid insomnia. Especially in younger patients with insomnia, psychodiagnostics are very important. These patients often show more comorbid psychiatric disorders. Patients with comorbid psychiatric disorders show less positive treatment results after CBT-I and we recommend a parallel treatment of the psychiatric disorder when starting with CBT-I. Based on our results we conclude that patients with anxiety disorders might benefit more from sleep restriction and that extra attention should be given towards stimulus control in patients with insomnia and comorbid mood disorders. Adding new promising techniques such as acceptance commitment therapy (ACT) and mindfulness-based treatment might enhance treatment effect in specific groups of patients who respond less well to CBT-I. Future research should focus on the benefits of implementing tailored interventions based on type of psychiatric comorbidity and cognitive coping style.