1.1 Introduction

Mental, physical and social health aspects are tightly interwoven. Stress may severely disrupt physiological balances, resulting in stress-related health impairments in the long term. Stress-related disorders are an increasing concern for western societies due to absenteeism, reduced productivity and an increased use of health care systems, posing a considerable burden on industry and social systems.

However, not every person exposed to stress turns ill. Some individuals are easily affected by minor stressors, whereas others are surprisingly resilient. Physiological mechanisms underlying stress resilience, in turn, are poorly understood to date.

Despite its principal role in reproduction, testosterone, the main male sex hormone, has been implicated in stress resilience in males: Generally, the major endocrine stress axis, the hypothalamic-pituitary adrenal axis secreting the stress hormone cortisol, and the hypothalamic-pituitary gonadal axis, secreting testosterone, antagonistically affect one another in the long run. Testosterone levels are furthermore negatively linked with classic stress-related health alterations, such as obesity, diabetes and depression.

The role of testosterone in the acute response to stress, however, is not clear. Contrary to the common belief that stress and stress hormone secretion principally inhibit testosterone levels, testosterone levels may in fact acutely rise during challenge situations in males. Of interest, in animals, testosterone increases have been related to a high social status, aggressive behaviours, and proactive stress coping.

Another source impacting on stress resilience and stress vulnerability are experiences early in life, such as poor as compared to high quality of parental bonding. Whereas evidence accumulates that early life stress may increase cortisol secretion, it is unclear whether early life stress may similarly affect stress-related testosterone secretion.

The acute testosterone response to stress and its physiological, psychological and social correlates have not been scrutinized in men to date. The present work studies changes in testosterone levels in response to acute social stress, and examines associations with social status, physiological and subjective stress responses, aggression, early life factors, and health-related variables.

1.2   Outline of this thesis

Chapter 2 provides the **theoretical background** for this work. Section 2.1 outlines basic principles of testosterone (T) physiology, including T biosynthesis and biological actions of T over the life span. Section 2.2 highlights the association between T and men's health, including its relation to body composition, strength, obesity, libido, osteoporosis, and mortality. The section ends with a remark on potential adverse side effects of T administration. Section 2.3 presents psychobiological aspects of T, including a short section on its associations with sexual activity, exercise and cognition, followed by five larger sections covering the association between T and stress, social status, aggression, stress coping and paternal bonding. The latter provide the basis for this work's research hypotheses.

Chapter 3 presents the **research hypotheses** of this thesis. Hypotheses are clustered into five sets, each containing several subhypotheses on the same topic. Set I contains hypotheses on manipulation checks regarding the stress induction method and the occurrence of hemoconcentration (section 3.1). Set II presents hypotheses on stress-related T increases and its potential physiological mechanisms (section 3.2). Set III refers to hypotheses on the association between social status, stress-related T alterations, aggression and quality of life (section 3.3). Set IV contains hypotheses on the relation between social status, stress coping and health outcomes (section 3.4). Set V refers to hypotheses on parental bonding, stress-related T alterations, aggression, stress responses and health outcomes (section 3.5).

Chapter 4 specifies the **methods** used to test research hypotheses outlined in chapter 3. The chapter provides information on the recruitment of participants and study exclusion criteria (section 4.1), study participants (section 4.2), the general setting of the study (section 4.3), questionnaire measures (section 4.4), anthropometric and cardiovascular measures (section 4.5), assessment of blood and saliva parameters, calculation of stress-related increases and arithmetic correction for hemoconcentration (section 4.6) and statistical procedures (section 4.7).

Chapter 5 presents **results** of this thesis. First, results on manipulation checks are shown demonstrating that the stress induction method was efficient (section 5.1). The next

section presents that T levels increased in response to stress, whereas increases were unrelated to hemoconcentration and stress responses (section 5.2). Then, results on associations of social status with T increases and aggression (section 5.3), and associations of social status, stress responses and health outcomes (section 5.4) are shown. Finally, associations of parental bonding, T responses, aggression and health outcomes (section 5.5) are presented.

Chapter 6 gives a **discussion** of this work's findings. Sections 6.1-6.5 discuss results with regard to the research questions outlined in chapter 3 and integrate the results into the existing body of knowledge. Limitations of the study as well as implications for theory and practice are presented. Sections 6.6 and 6.7 give a more general discussion on social status and paternal bonding and their potential importance for mechanisms underlying T increases as well as their relation to health outcomes. The final section summarizes key findings and draws general conclusions from this work.