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#### The sleep of the child – the parent's stressor? A study within the ABIS project

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Abstract	3
Introduction	5
Foreword	7
Diabetes	9
Stress	12
The concept	12
Allostasis and allostatic load	13
Basic endocrinology of stress response; implications for allostatic load.	14
Psychological appraisal	16
Evolutional implications of psychological appraisal for allostatic load	18
Balancing environmental and mental	19
Measuring psychological stress	20
The situation	21
The response	21
The duration	22
Operationalising it	22
Stress in the ABIS study	23
Measures	23
Associations to other measures	24
Attachment theory	25
Sleep	27
Aims	28
Hypotheses	29
Methods	33
Procedure	35
Participants	35
Measures	36
Central measures	36
Sleep measures	36
Stress measures	37
Peripheral measures	39
Child temperament	39
Social support dissatisfaction	39
Both parents born outside of Sweden	40
Single parenthood	40
First child	40
Parental education	40
Parental age at birth of child	40
Night feedings	41
Ethical considerations	41
Statistical analyses	42
Results	43
Associations	45

Child / parental sleep quality and parenting stress45Background factors and sleep/stress46Child temperament46Social support dissatisfaction46Both parents born outside of Sweden47Single parents48Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Background factors and sleep/stress46Child temperament46Social support dissatisfaction46Both parents born outside of Sweden47Single parents48Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Child temperament46Social support dissatisfaction46Both parents born outside of Sweden47Single parents48Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Social support dissatisfaction
Both parents born outside of Sweden47Single parents48Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Single parents48Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Firstborn child48Parental education48Parental age49Child gender50Life events and parenting stress50
Parental education48Parental age49Child gender50Life events and parenting stress50
Parental age
Child gender
Life events and parenting stress
Longitudinal patterns
Discussion
Associations
Child / parental sleep quality and parenting stress
Background factors and sleep
Background factors and stress
Between sleep variables
Patterns
Sleep patterns
What do the patterns really tell us?
How do they vary over time?
What does that mean for this thesis?
Stress patterns
Measures: methodology and concepts
Stress
Sleep
Night wakings
Sleep quality
Child temperament
Summary and conclusions71
Acknowledgments
References
Appendix

## Abstract

Poor sleep and chronic stress are important factors detrimental to physical and mental health. This is no less true for children than for adults. Therefore, investigating sleep and stress patterns in early life is important. Since children live in a close relationship to their parents, the sleep and stress patterns of the parents is likely to influence those of the children. In this thesis, the relationships between parent-reported child sleep quality, self-reported parent sleep quality, and parenting stress as measured by the Swedish Parenting Stress Questionnaire (SPSQ) have been investigated. Several background factors have been tested for associations to parent and child sleep quality and parenting stress, and their possible involvement in the associations between sleep and stress measures has been investigated. The hypotheses were that child sleep, parental sleep and parenting stress show concurrent intermeasure associations and longitudinal intrameasure stability, which should also generate longitudinal intermeasure associations. The participants were parents of about 10000 children in the ABIS study, born in south-east Sweden in the years 1997-99. Questionnaires were gathered at birth and at 1, 3 and 5 years and data analyzed statistically The hypotheses were supported: sleep and stress measures showed strong concurrent associations and longitudinal stability. However, parental sleep quality seems to explain most of the child sleep-parenting stress association.

All background factors except child gender showed some level of association to sleep and stress measures at least at some age. No background factor had any effect on the associations between sleep and stress measures when included in logistic regression. Our data does not support the hypothesis that night feedings condition the child to night wakings. A possible predictor of persistent sleep problems is found in uncertainty about the cause of night wakings.

To conclude, parent-perceived child sleep quality has a connection to parenting stress which in our data is mainly explained through parental sleep quality. This is important to consider when advising parents that complain about their child/ren's sleep quality.

# Introduction

### Foreword

The ABIS study, which provides the data material for the current thesis, is a prospective cohort study with the purpose of generating hypotheses as to the aetiology of juvenile diabetes. While this thesis does not directly concern diabetes, it does investigate a possible pathway contributing to the development of diabetes in children, namely psychological stress. In order to show how the current study is connected to diabetes research, I will start by describing, in brief, basic theory about diabetes aetiology, then turn to theory about psychological stress, including current thinking about what psychological stress entails, the physiology of the stress response, human life stress and the measurement of stress.

In order for psychological stress to contribute to the development of child diabetes, it is the child that must be stressed. However, we do not as yet have measures of psychological stress in the children themselves in ABIS, but only in the parents. Since a previous ABIS study has shown a relationship between psychological stress in parents and diabetes-related autoantibodies in children, there may be a link between such stress in parents and in their children. This link may be explained by attachment theory, wherefore I will also describe the basics of this theory and how it applies in this study.

One important stressor for parents is the sleep of their child. In the ABIS questionnaire, questions about the sleep of the child are included. Poor sleep may in itself be a factor in both stress and diabetes. However, we do not have any objective data on child sleep. Instead, we have data on the parental perceptions of child sleep. I have analysed these data in an attempt to shed some light on the connections between parental perceptions of child sleep and parenting stress.

After describing the fundamentals of theories of diabetes, psychological stress, attachment and sleep, I will describe the hypotheses of the current study, before I present and discuss my methods, results and conclusions.

## Diabetes

Diabetes is the name of a serious disease characterised by hyperglycaemia caused by deficiencies in the ability of the body to regulate blood glucose by means of insulin. Symptoms include excessive polyuria, thirst, fatigue and weight loss, sometimes to production of ketone bodies which may lead to pain in the stomach, nausea, deep breathing, and gradually unconsciousness. Sometimes other symptoms occur, eg vision blurring. These symptoms may or may not be present in adults, but are usually pronounced, sometimes severe in children. Diabetes in children and adolscents, Type 1 diabetes, cannot be cured but requires a life-long intensive treatment with daily insulin injections, special diet, regular blood glucose measurements et cetera. In spite of this heavy treatment, it leads to life-threatening both acute and late complications. Diagnosis of diabetes rests partly on symptoms but chiefly on

measurements of blood/plasma glucose. For an asymptomatic person, it takes repeated such measurements or an oral glucose tolerance test (OGTT)<sup>1</sup>. In children, diagnosis is based on blood/plasma glucose measurement without OGTT, due to the common severity of symptoms in children, and as children almost without exception have high or very high blood glucose values at diagnosis.

Traditionally, diabetes is categorised into different subgroups according to the nature of the insulin deficiency. This may be a lack of insulin secretion, which in turn may be caused by a number of factors, or relative insulin deficiency in combination with insulin resistance, i.e. an insufficient response to insulin by the target cells.

Type 1 Diabetes (T1D) is caused by the destruction of pancreatic  $\beta$ -cells, in which insulin is produced. This destruction is usually due to an autoimmune process. At least 10% of all diabetes cases are T1D and the type is increasing all over the world. The incidence is highest in Finland, with Sweden as second highest incidence in the world. There is a large increase in incidence in most countries<sup>2</sup>.

The cause or causes of the  $\beta$ -cell destruction is as yet unknown, but is thought to be a combination of genetic and environmental factors, resulting in an autoimmune process. The onset of the disease is often preceded by increased levels of autoantibodies such as IAA, IA-2A and GADA seen in up to 95% of the newly-diagnosed children<sup>3</sup>. Research is being carried out on environmental factors that can be connected to an increase in such autoantibodies. One such factor, which the current thesis is centred about, has been found to be psychological stress<sup>4</sup>.

The aetiology of Type 2 Diabetes (T1D) is considered to be a combination of gradually insufficient insulin secretion and insulin resistance acquired through life-style factors, notably low physical activity and/or overweight. Also different forms of stress may lead to insulin resistance, which thus increases the need for insulin. Thus when insulin resistance is combined with a decrease of insulin production (although not as pronounced as in Type 1 diabetes) there will be relative lack of insulin leading gradually to the same or similar symptoms and signs as in Type 1 diabetes. This categorisation of diabetes into Types 1 and 2 has been challenged by e.g. Wilkin<sup>5</sup>, on the grounds that there is no clear clinical distinction between the two – Type 1 cases frequently present insulin resistance and Type 2 cases  $\beta$ -cell destruction. Wilkins has instead proposed the accelerator hypothesis, in which he names three accelerators which combine to cause both types of diabetes: 1) an intrinsically high rate of  $\beta$ cell apoptosis; 2) insulin resistance and 3) a genetic predisposition to autoimmunity. Without the third accelerator, the progress is slower and autoimmunity does usually not develop during the patient's life-time, but nevertheless the disease follows the same basic pattern.

Ludvigsson has proposed a further hypothesis, the  $\beta$ -cell stress hypothesis<sup>4</sup>, which may be seen as an extension of the accelerator hypothesis. It focuses on the plight of the pancreatic  $\beta$ -cells, the very insulin producers.  $\beta$ -cell stress occurs when too high demands are placed on the insulin-producing cells, due to insulin resistance, or increased insulin demand due not only to rapid weight gain, but also to other factors such as puberty, trauma, infections, low physical activity, increased glucose consumption, or psychological stress. An additional complication is that stressed  $\beta$ -cells may present antigens such as GAD<sup>6</sup>, which may trigger or aggravate an autoimmune response in individuals genetically predisposed to autoimmune reactions, or with an imbalance of the immune system for other reasons.

Thus viewed, diabetes becomes a process facilitated by a number of identifiable factors. Some genetic factors predisposing to autoimmunity are already known, such as certain HLA-types, especially HLA DR3 and/or 4, as well as HLA DO 2 and/or 8, and then environmental factors also contribute to the development of autoimmunity. Hygiene is discussed as a possible cause of imbalance of the immune system, and certain virus infections may play a role for initiating the autoiummune process, as well as early nutritional factors. However, in this research project the focus has been on how stress may influence the process sometimes ending in diabetes. Thus, insulin resistance is caused by e.g. the above-mentioned life-style factors of too much food and too little exercise. Are there other possible causes of insulin resistance? Indeed, infections may induce it<sup>7</sup>, as well as elevated stress hormone levels<sup>8</sup>, which is one reason why psychological stress is a possible factor in the aetiology of diabetes. A more thorough presentation of the connection between stress and diabetes will be presented further on..

A successful struggle against diabetes, then, should rest on research on several possible aetiological factors. Life-style factors such as overweight are good candidates, since they can be dealt with by straightforward and not too costly therapeutic approaches such as prescribing diet and exercise (which of course does not guarantee their success). Psychological stress is another life-style factor, which may be ameliorated by a variety of more or less costly methods.

Thus, in researching e.g. stress, in itself a worthy cause, one may also contribute to the progress of diabetic research. In this thesis, I will investigate an aspect of stress which, using the same data material, has previously been shown to have a connection to the occurrence of diabetes-associated autoantibodies. The aspect in question is parenting stress, which seems to have a connection to autoantibodies not in the parents themselves (on which there are no such data in the material), but in their children<sup>9</sup>.

## Stress

#### The concept

What is understood by the word "stress"? Merriam-Webster<sup>10</sup> gives us the following explanation (not showing entries related to linguistics):

Etymology: Middle English stresse stress, distress, short for destresse. Constraining force or influence: as

- a) a force exerted when one body or body part presses on, pulls on, pushes against, or tends to compress or twist another body or body part; especially: the intensity of this mutual force commonly expressed in pounds per square inch
- b) the deformation caused in a body by such a force
- c) a physical, chemical, or emotional factor that causes bodily or mental tension and may be a factor in disease causation
- d) a state resulting from a stress; especially: one of bodily or mental tension resulting from factors that tend to alter an existent equilibrium <job-related stress>
- e) strain, pressure <the environment is under stress to the point of collapse Joseph Shoben>

Let us leave items a and b, which both deal with mechanics rather than with life sciences, and look closer at c and d. Both of these involve "bodily or mental tension", but in c, stress is a factor which causes such tension, while in d, stress is the state of tension itself.

This duality of perspective accurately reflects the state of stress research which is reviewed by Monroe,<sup>11</sup> according to whom most theories in operation about stress can be divided roughly into two branches, one of which may be called stimulus-oriented and the other response-oriented. The stimulus-oriented theories correspond with item c above in maintaining that stress is something that arises in the environment and influences an organism in one way or another. On the response-oriented branch, corresponding with item d, stress is the state arising as a response of the organism to something in the environment which is then termed a *stressor*.

It may be regarded as a mere matter of semantics if it is the stimulus or the response that is called stress. However, as Monroe points out, both views fail to capture an essential element of stress, namely its transactionality.

While we may regard stress as a rather modern phenomenon, it is certainly not so. Although modernity has brought on a new host of factors that influence our stress, the phenomenon itself has ancient evolutionary roots, as a systemic adaptation to ensure the survival of an organism in a changing environment.

What happens in an organism during a stressful episode is basically this: some change in the environment catches the organism's attention. The organism *perceives* the change as threatening/challenging or benign/irrelevant (this is what Lazarus & Folkman called *primary appraisal*<sup>12</sup>). If the change is appraised as a threat or challenge, the organism takes stock of the behavioural systems at its disposal (*secondary appraisal*<sup>12</sup>) and activates the (hopefully) appropriate one to meet it. As a result, if the organism is successful, the environment changes again (e.g. the threat is neutralised by fighting or fleeing or the challenge is met by e.g. successful courting or food seeking) or the change is appraised differently (e.g. the threat turned out to be false alarm on closer inspection or feelings of hunger are suppressed in the absence of food sources). As a consequence of the changed internal or external environment, the systems are deactivated.

Without environmental change, no stress. Without organism response, no stress. Both views presuppose that transactions occur between organism and environment. Hence, to fully capture the nature of stress, one must consider the stress process in terms of "external challenges and perceptions of the challenges, coping resources and perception of coping resources, and the dynamic interplay of these over time".<sup>11</sup>

To this end, two conceptual developments have emerged.<sup>11</sup> One concerns the concepts of allostasis and allostatic load, the other deals with the psychological appraisal of environmental challenges.

#### Allostasis and allostatic load

One of the most fundamental driving forces of evolution in organisms is the preservation of homeostasis, namely the keeping of certain key conditions in the body (e.g. temperature, salinity, oxygenation) within a range that allows the survival of the organism.<sup>13, 14</sup> In a changing environment, as noted above, the organism must have access to a repertoir of different behaviours to ensure homeostasis. These behaviours include changes in the body – such as faster breathing to keep up blood oxygenation during bodily exertion – which under other circumstances would be detrimental to homeostasis. In order to remain unchanged, the organism must change – like Alice in the Looking-glass<sup>15</sup>, it runs only to remain in the same place.

This state of constant adaptive change has been referred to as *allostasis*.<sup>16</sup>, <sup>17</sup> Of course, different environmental conditions impose different levels of challenge on an organism ranging from no to severe challenge, and thus different allostatic conditions impose different strains on the organism. Normally, straining allostatic conditions change back into less straining ones when the challenge is met or the threat averted and is then adaptive, beneficial rather than harmful. However, if the allostatic condition is constantly activated or insufficiently deactivated the benefits of the stress response turns into harmful strain. The strain that an allostatic condition imposes on an organism during inadequate activation (too much or too little) is termed *allostatic load*.<sup>17</sup>

# Basic endocrinology of stress response; implications for allostatic load

The most remarkable feature of the stress response is its uniformity in the face of such a diversity of stressors. The first to bring this observation into the awareness of the scientific community was Hans Selye, often called the father of stress research, who coined the term "general adaptation syndrome" (GAS) for the stress response<sup>18</sup>. This uniformity means that from physiological stressors such as blood loss or infection, through clear and present dangers like a charging tiger, to the vague psychological feeling of something being wrong, the same basic mechanism is activated. Of course, different stressors activate it differently, to different degrees in different parts of the system, but all in all the homogeneity is great enough that "stress response" is a useful concept for research.

The most salient physiological changes in the stress response is the change in metabolic and cardiovascular functions, optimising them for the "fightor-flight" response. To keep the muscles and the brain going in order to overcome the challenge, blood glucose and oxygenation must be kept at homeostatic levels in the face of an increasing utilisation energy and oxygen. This means that breathing and pulse must increase and that energy stored as fat, glycogen and protein must be made available to the hardworking muscle and brain cells in the form of glucose. One important event in this process is the suppression of insulin action by, which otherwise facilitates the storage of energy, i.e. the opposite pathway of energy mobilisation. This suppression includes both "insulin antagonism" and control of insulin secretion by both sympathetic and hormonal signalling.<sup>14</sup>

The most prominent hormones involved in the regulation of the stress response are adrenaline (epinephrine), noradrenaline (norepinephrine) and cortisol, which can be said to constitute two axes in the activation of the stress response. One axis runs through the sympathetic nervous system (the Sympatho-Adrenomedullary axis or SAM), resulting in the secretion of noradrenaline from sympathetic synapses on various organs and adrenaline from adrenal medulla as a response to sympathetic nerve activation. Cortisol, on the other hand, is released from the adrenal cortex by the activation of the HPA (Hypothalamic-Pituitary-Adrenocortical) axis by means of hormonal control.<sup>14</sup> It has also been suggested by Henry<sup>19</sup> that the SAM axis be divided into two; one utilising noradrenaline and testosterone and responsible for the fight response (anger), the other utilising adrenaline to induce the flight response (fear).

The SAM axis is quick in activation and deactivation – adrenaline is rapidly cleared by active transport into the liver, giving it a plasma half-life of less than 2 minutes<sup>20</sup> – while the HPA axis is somewhat slower to start and its effects longer-lasting, the plasma half-life of cortisol being 1-2 hours<sup>20</sup>. However, while activated, both axes exert their respective allostatic load. The actions of the two systems are summarised below, adapted from Vander's *et al* book<sup>14</sup>.

Actions of the Sympatho-Adrenomedullary axis

- Increased glycogenolysis in muscle and liver
- Increased triacylglycerol breakdown in adipose tissue
- Decreased skeletal muscle fatigue
- Increased pulse and breathing
- Diverting blood to skeletal muscle
- Increased blood coagulability
- Decreased insulin- and increased glucagon secretion

Actions of stress levels of cortisol (HPA axis)

- Stimulation of metabolic mechanisms to increase blood/plasma levels of glucose and other energy sources
- Inhibition of glucose uptake by most cells except brain cells, by "insulin antagonism"
- Enhanced vascular reactivity to sympathetic nerve stimulation
- Inhibition of immune responses (long-term stress)
- Changes in CNS pertaining to memory formation and stress reactivity

These actions are adaptive in the short run, but if they e.g. take place too often or are sustained for too long, they may increase the allostatic load. For example, both axes are involved in converting stored energy into energy available to cells. Frequent mobilisation of energy stores and subsequent refilling is costly in terms of energy expenditure and thus increases the allostatic load<sup>21, 22</sup>. The insulin antagonism included in this process also places a great allostatic load on insulin-producing cells, the consequences of which will be discussed further on.

Furthermore, the increased cardiovascular activity, induced by the sympathetic axis and partly facilitated by the HPA axis, places greater mechanical stress on the involved tissues<sup>22, 23</sup> as well as contributing to coronary artery atherogenesis<sup>22, 24</sup>.

The immunosuppression associated with long-term stress<sup>22, 25</sup> results in a greater allostatic load by leaving the organism more susceptible to infection. However, it also appears that recurring stress may facilitate the development of autoimmune diseases, including diabetes<sup>22, 25, 26</sup>. The changes in the central nervous system (CNS; see below) may perhaps not in themselves be considered an increase in allostatic load, but as we shall see, they increase the frequency and intensity of future stress responses with ensuing higher allostatic load. Also, these are not the only effects of prolonged stress-levels of glucocorticoids on the CNS; many studies have pointed to their role in damage to several brain structures, e.g. hippocampus and prefrontal cortex.<sup>22, 27</sup>

These are not the only ways in which the stress response results in allostatic load. For an accessible and extensive review, see Sapolsky<sup>22</sup>.

#### Psychological appraisal

When selecting from the behavioural repertoir to meet a perceived challenge, the organism has to appraise the challenge in order to make an appropriate selection. The cognitive level of this appraisal in humans may range from pure reflex without even subconscious cognition, through subconscious appraisals emerging into consciousness only as gut-feelings, to fully conscious analysis of the problem at hand<sup>12</sup>. While reflexes are normally more or less genetically encoded, cognitive appraisal must rely on experiences of similar, previous situations. These experiences, in turn, are based on the appraisal and outcome of the previous situation. As mentioned above, Lazarus and Folkman introduced the concept of *primary* and *secondary* appraisal, terms which they stress are not to be taken as indications of precedence neither temporally nor in importance<sup>12</sup>.

During primary appraisal, the sensory input pertaining to the situation is given cognitive and emotional meaning. This process seems to be dependent on the hippocampus, essential to the formation of declarative memories, and amygdala, which plays a central role in the formation of emotion.<sup>28</sup> Sensory input is filtered through these regions and emerges as concepts loaded with information about the nature of the stimuli that evoked them: dangerous or benign? Potentially rewarding or uninteresting? Hippocampus and amygdala are both closely connected to the anterior cingulate gyrus, which is active in animals engaged in selecting a behavioural strategy, and its associated regions<sup>28</sup>. This could then be interpreted as the neurophysiological correlate of secondary appraisal. All these regions are also connected to the bed nuclei of the stria terminalis, which in turn exerts control over the hypothalamus and thereby the HPA axis.<sup>28</sup>

HPA axis activation includes the release of glucocorticoids from the adrenal cortex (cortisol in humans). These are lipophilic and therefore pass the blood-brain barrier. In the CNS, cortisol exerts a negative feedback on its own release by inhibiting the release of corticotropin-releasing hormone in the hypothalamus<sup>14</sup>. However, other regions of the brain are also rich in cortisol receptors.<sup>28</sup>

There are two types of cortisol receptors: type I and type II. Type I are more sensitive and are activated by normal cortisol levels, whereas type II are 10-20 times less sensitive than type I and need stress-levels of cortisol for activation<sup>21, 28</sup>. Hippocampus and amygdala are two regions rich in type II receptors (the rat hippocampus has about equal numbers of type I and II receptors, while the rat central amygdala has about ten times more type II than type I<sup>28</sup>.) The changes resulting from activation of type II receptors include alteration of the expression of specific genes, resulting in an amygdala sensitised to sensory cues indicating impending stress<sup>28</sup>. It appears, then, that cortisol changes the way we appraise the events of our world.

In this, individual temperament plays a part as well as previous experience, since individuals of different temperament<sup>29, 30</sup>, commitments and beliefs<sup>12</sup> have an innate tendency to perceive and therefore appraise similar situations in somewhat different ways. Personality differences such as temperament are to a great extent genetically heritable<sup>31</sup>, possibly via genetic polymorphism of neurotransmitters, such as dopamine and serotonin, and/or their receptors and transporters, although the nature and extent of such genetic influence on personality remains a topic of investigation<sup>32, 33</sup>. Still, it is reasonable to believe that different

neurotransmittor activity levels in the individual brain colour the brain's perception of the situation.

Thus the individual psychology (whether genetically inherited or not) has a large role to play in the stress process. If a perceived environmental challenge is appraised as potentially rewarding rather than threatening, the choice of behavioural response will result in a condition which is likely to impose a much lower allostatic load on the organism.

# Evolutional implications of psychological appraisal for allostatic load

So when the perception of a situation is appraised to require it, the brain responds by shifting the body into a new allostasic condition in order to preserve homeostasis. How well, then, does the perception and the following psychological appraisal correspond with the objective challenge? Given that it must be evolution which has shaped the processes underlying the perceptions, they should be maximised to assure the survival and procreation of the organism. However, what assures survival varies with the circumstances. To take an extreme example, a tendency to perceive the charge of an enraged tiger as an opportunity for petting is not likely to result in the survival of the organism, while a prevailing tendency to perceive the sigh of the wind in the trees as the stealthy paws of a hunting tiger is likely to impose a severe allostatic load on the organism, which in the long run could eliminate the organism from the gene pool as effectively as the flesh-and-blood tiger itself would, albeit not as speedily. It may be through the direct ill-health of the organism, but also through reduced fertility by processes like reduced testicular function, as studied by e.g. Sapolsky on baboons<sup>22, 34</sup>.

In the evolutionary history of humanity, we have most likely spent a significant amount of the time under the threat of large predators<sup>35, 36</sup>. Under such circumstances, choosing a higher allostatic load over the possible event of an actual tiger – a "rather safe than sorry" approach – may outweigh a relaxed "no worries" attitude in terms of survival value. Physiologically, this may be mediated at least partly by the above described effects of cortisol on the CNS, namely to sensitise the amygdala to potential stressors and thus keep the HPA axis alert in stressful times and climes<sup>21</sup>. Furthermore, if the HPA axis were to become desensitised by repeated activation, this would effectively render the organism incapable of being stressed, which would have disastrous consequences for the

organism, by far outweighing the allostatic load of the more frequently activated stress response<sup>21</sup>.

From this perspective, humans may be predicted to have a tendency to perceive situations as more threatening, or at least challenging, than they actually are. We have evolved an imagination which has helped us to see problems before they occur – even if they never would have; we have the ability to see patterns that are not even there. The evolutionally brief episode of civilisation, where the tiger threat and similar challenges can be regarded as history, cannot be expected to have had any profound impact on the deeply rooted genetic foundation of these systems.

In the light of evolution, then, expecting a consistently very high correspondence between the human perception of a challenge and the objective gravity of the environmental conditions thus perceived – i.e. that stress has a purely environmental source – seems an untenable position. Likewise untenable is expecting a consistently very low correspondence – i.e. that stress has a purely mental source – since this would undermine the ability to respond adequately to actual environmental challenges. Thus the most fruitful approach if one wishes to investigate stress should be to study the interplay between the environmental and the mental; between the situation itself and our appraisal of and reaction to it.

#### Balancing environmental and mental

While it appears most reasonable to regard stress as a transactional phenomenon, this much may be said when balancing the environmental against the mental: The nonexistence of environmental challenges is no guarantee for the non-arising of a stress response in any organism capable of mentalising, since the psyche is capable of conjuring up threats where none exist. On the other hand, the nonexistence of a mental representation of a threat *on any cognitive level* must be taken as a guarantee against the arising of a stress response, even in the event of an actual threat, since it is the CNS which activates the stress response. Thus, the mental perception but not the environmental presence of a stressor must be a necessary condition for a stress response. Monroe and Kelley go so far as to take this as "almost axiomatic"<sup>37</sup>.

At the same time, as discussed above, an organism in which perceived threats and actual threats are more or less a random match must be seen as highly maladaptive. Therefore, if an organism that appears well adapted to its natural environment perceives a stressor in such an environment, then there should be a high probability of the presence of an actual stressor. In other words, if somebody is stressed, we can neither presume nor preclude the possibility that there exists an actual stressor in his or her environment, which means that the situation is still an interesting point of investigation. However, regardless of the actual situation, the stress response is real and should be dealt with as such. The question that then arises is: How can we measure an individual's stress level?

### Measuring psychological stress

If we want to accurately quantify psychological stress in individuals, we need to answer the question of what is a greater and what is a lesser level of stress. We might try a dichotomous approach, dividing a population into groups of stressed/not stressed, or we might try to create a variable that grows higher as an individual becomes more stressed. But whichever approach we take, we must somehow operationalise our concepts of psychological stress, in other words assign values to different individual stress profiles.

How we do that, of course, depends on what is understood by stress. If stressors are seen as something environmental; a factor exogenous to the stressed organism, then we may identify the stressors and then quantifying the extent to which they are present in the organism's environment. If stressors are conceptualised as the mental representation of a threat or challenge, then it becomes the more difficult matter of assessing the state of mind of the organism, which may be done at a psychological level, tapping into the individual mental representations of the situation, at a biological level by measuring the stress hormone levels, or both (in animals, the mental representations are of course quite inaccessible). In order to measure stress as a transactional process between environment and mind, it would be necessary to look at both environment and individual response, and how their interactions develop over time. The issue of time is important to the concept of transactionality. Acute stressors, for example, are not in themselves likely to create as much allostatic load in the long run as chronic stressors, although a traumatic experience such as loss of a partner may give rise to subsequent chronic stressors such as economic hardship.

This may lead us to seeing three ways of tapping into the process of the stress response: 1) The situation itself, 2) The individual response to the situation and 3) The duration of the stressful situation.

#### The situation

This is the environmental approach to measuring stress and may be broadly categorised into two classes: Life events and Daily hassles. Life events is perhaps the oldest approach to measuring stress and is based on the assumption that stress is a reaction to circumstances that demand adaptation of the individual<sup>38</sup>. Greater change in the environment would then result in greater stress. Measuring life events is usually done using checklists.

It is natural to assume that different types of situations have different impact on the individual stress levels. However, attempts to create indices of events weighted after different perceived magnitude have not enhanced predictivity<sup>37, 38</sup>. This may be interpreted in at least two ways: either the weighting has been inadequately operationalised, failing to capture an existing, objective relative stressfulness of different types of situations, or it may be that appraisal has a much greater impact on individual stress levels than the situation itself.

Daily hassles, on the other hand, is according to Monroe<sup>37</sup> actually more related to the appraisal approach discussed below. These are the day-to-day minor annoyances that arise at work, at home, in relationships et cetera. Some studies<sup>39, 40</sup> suggest that not only do these seemingly minor events better predict aversive health outcomes – especially if paired with a shortage of positive daily occurences termed *uplifts*<sup>40</sup> – and Kanner *et al* also propose that the aversive effects of life events may be mediated by a changed pattern of daily hassles: "divorce might create a whole collection of unusual minor demands ... which did not have to be dealt with previously."<sup>40</sup>

#### The response

This may be described as the psychological and/or biological approach to measuring stress. The biological aspect of the stress response is perhaps the easiest to measure, since it may be done by measuring levels of cortisol and other stress hormones, but they only yield a snapshot of the state of the individual, which may be confounded by individual differences and circadian rhythmicity.

The psychological approach focuses on the individual's appraisal of the situation. If it is assessed as threatening or challenging in primary appraisal, we may expect a stress response. Monroe<sup>37</sup> notes that appraisal measures, just as biological measures, are useful for making associations to outcomes

of psychological stress such as physical or mental illnesses, although not in unravelling the aetiology of stress.

#### The duration

Stressors and stress responses can also be broadly divided into chronic and acute. Both are characterised by activation of the same mechanisms, the difference is in duration and it is usually only chronic stress that is regarded as systematically detrimental to the individual's health<sup>41, 42</sup>. A stress response which is soon deactivated is not likely to affect the organism negatively.

That said, it is not as simple as it may sound to draw a line between acute and chronic stress. When does a stressor begin and when does it end? For how long may the effects of the stressor persist after the original factor has been eliminated from the environment? Is repeated, brief exposure more stressful than a single, long exposure even if the total time is the same? And how many repetitions and/or how long exposure does it take to start generating adverse effects? There are probably no clear and objective cutoff values to be had, since so many different factors come into play, not least appraisal.

#### Operationalising it

These various approaches are operationalised in somewhat different ways. The biological response approach, as noted above, can be assayed in a biochemical analysis, while the other approaches rely on information given by the subject him- or herself. Life events are usually measured by checklists. One problem in this regard is the fallacy of individual memory when trying to determine the exact timing of an event, which may have implications when trying to determine its influence on the present state. Another is in differential interpretation by subjects: just what constitutes a "serious" disease, for example?

For quantifying the stressfulness of psychological appraisal, Monroe<sup>37</sup> describes the following types of approaches:

- Ad-hoc single item measures, in which a single situational factor which is simply rated as more or less positive/negative and important/unimportant,
- Multiple-item scales, in which the respondent answers several questions that taken together form a more complete picture of the individual's perceptions,

- Investigator-based approaches with interviews by a trained professional and
- Life event scales, to the extent that they make provisions for assessing the subject's perceptions about the events.

The most sensitive method is probably the investigator-based. Ad-hoc single item measures are very blunt and also share the methodological shortcoming of multiple-item scales, as with all self-report measures: that items are interpreted differently by different subjects. Operationalising psychological stress by these methods would yield a variable dependent partly on the strength of the respondent's replies, partly on the number of items measured. This may also pose a weakness: high values in one part of the measure may be concealed by average or low values in others, even with an interview approach<sup>37</sup>. In some scales, such as the SPSO<sup>43</sup> (see below), factor analysis has identified a number of subscales, tapping into related but somewhat distinct dimensions of the stress experience. Perhaps this technique may serve to highlight specific stress that would otherwise remain hidden in the general? When it comes to duration, Baum *et al*<sup>42</sup> divide stress into three components, whose durations may differ: Event, threat and response. Each of these may be dichotomised as chronic or acute, and if all three are chronic, the total stress is seen as "perfect chronic" and vice versa. Baum et al give the example of victims of disasters or other traumatising events, whose reactions may far outlast the actual event and in a minority of cases cause chronic stress ranging from subclinical levels to severe posttraumatic stress disorder (PTSD). Thus, habitually characterising a stressful event of clearly limited duration as an acute stressor may be misleading. Taking into account the persistence of the individual's reaction to the event gives more information about the chronicity and therewith of the potentiality for negative health impact of the stressor.

#### Stress in the ABIS study

#### Measures

In the ABIS questionnaire, items are included that lend themselves to estimating the stress level of the respondent. One is a yes/no question about if the respondent has experienced something which s/he perceives as a major life event since the birth of the child, followed by a brief checklist of life events. Another is two items asking about social support and feelings of confidence, at one time-point followed by a multiple-item scale intended to measure social support. Finally, a multiple-item scale called the Swedish Parenting Stress Questionnaire (SPSQ) was included to measure the stressfulness of the parenting situation of the respondent. For reasons explained later, the measure primarily used in this study was the SPSQ. This means that the approach used is the psychological appraisal approach. The checklist only contains items which are not specifically related to parenthood, and parenthood as a life event has occurred to 100% of the participants, since that was an inclusion criterion to start with. The restriction to the appraisal approach will of course influence the course of the discussion. In the light of the above outline of stress theory, I hope to have made it clear that to the extent that the SPSQ succeeds in capturing the respondents' psychological appraisal of the stressfulness of their parenting situations (further discussed in the methods section), we can take this as an adequate measure of the parenting stress level of the respondent.

#### Associations to other measures

In previous ABIS studies, high levels of psychological stress in the parents have been associated to other measures in the children, notably increased levels of autoantibodies<sup>9</sup> and obesity<sup>44</sup>.

Sepa<sup>9</sup> found that both high parenting stress and experiences of a serious life event were associated with an increase of tyrosine phosphatase autoantibodies (IA-2As) in the child, independent of family history of diabetes. IA-2As are considered one of the best markers of the autoimmune process that leads to diabetes.<sup>9</sup> In Koch's study<sup>44</sup>, children from families that reported stress in at least 2 of the 4 domains assessed had significantly higher odds ratios for obesity.

By which mechanism, then, would psychological stress in the parents induce physiological changes in their child? The most likely candidate is of course by causing psychological stress in the child, which in turn results in the observed physiological changes. One theory which might be able to explain how and why psychological stress in parents may be echoed by their children is attachment theory, which is briefly described in the following section.

## Attachment theory

If we are investigating child health, why then is it relevant to attempt to measure the stress level of the parent? Attachment theory gives us a possible answer to that question. The following description of attachment theory is based in its entirety on Broberg's *et al* work<sup>45</sup>.

According to attachment theory, infants and to a decreasing degree older children have throughout our evolutionary history been completely at the mercy of a caregiver for survival. This has driven the evolution of an attachment system, which is a primary motivational system for triggering attachment behaviour in the child (e.g. crying or smiling) which in turn triggers the caring system in the present caregiver, prompting him or (usually) her to respond to the child's expressed needs (e.g. comfort, food or play).

The neonate does not discriminate between persons; any competent adult may respond to the child's needs and thus turn off the attachment system. With time, however, the attachment system becomes more selective and hierarchical and the attachment behaviour is directed towards a few caregivers, ranked from primary and down. If a secure attachment relation has developed, the selected caregivers may function as secure bases from which the child can safely explore its surroundings (an important activity for the development of the child) and a safe haven to return to in the event of a perceived threat.

At the same time, the child's limited experience of the world and mentalising capability makes it difficult for the child itself to discriminate between real and imaginary threats. It may for example perceive a twisted, fallen branch as a coiled snake, cancel its exploratory activities and seek the caregiver's aid, or it may fail to recognise the hostility in a growling dog and try to play with it, at the risk of being bitten. The latter may of course prove fatal, while the former is merely detrimental if the caregiver's response is consistently inadequate (e.g. unnecessarily fearful), driving the establishment of a pattern where the child withdraws more or less entirely from exploring for fear of evoking the caregiver's distressing response. In order to learn to correctly identify threats, the child is dependent on the judgment of a more experienced and trustworthy person, namely the caregiver. The caregiver of the dog-loving child recognises the danger and sharply calls the child away to avoid its coming to harm, while that of the snake-fearing child will pick up the branch to show it for what it is, so that the child can go on exploring with a new lesson learnt.

For this system to work, children need a highly evolved sensitivity to the moods and signals of the caregiver; an inborn capacity to read the facial expressions and body language of the caregiver. From infancy, the child constructs internal working models with the function of anticipating and interpreting the behaviour and emotions of the caregiver and arrange the child's attachment behaviour, emotions and cognitions accordingly. These models are constructed from concrete experiences of the caregiver's response in situations with perceived threats. If the child frightened by the branch in the above example is met with irritation, it will develop a lower sense of worth than if met with understanding.

While a measure of parenting stress cannot tell us exactly how a caregiver interacts with a child, it gives us a clue. A stressed parent may care for the child in the most competent way, but the sensitivity of the child to the caregiver's mood may pick up the signal that although all its worldly needs are being met, there's something wrong. Since parenting stress deals exclusively with stress in relation to the role of the caregiver, it may be expected that this stress is expressed in parenting situations, i.e. in physical proximity to the child – perhaps not always, but at least in situations where there is a conflict between the needs of parent and child, or where the parent feels unable to meet the needs of the child.

Thus the idea that parenting stress is in turn a stressor to the child is based on sound theory. We cannot test it as a hypothesis within the ABIS material for lack of a measure of child stress, but we will assume it in this study. As we have seen in the section on stress theory, the putative stress induced in the child has the potential to influence the child's health negatively, including as a possible contributing factor to the development of diabetes.

## Sleep

Sleep is one of the cornerstones of a sound physical and mental health. This statement is supported both by everyday experience and by science. Several studies indicate that disturbed sleep not only increases the risk for conditions such as lowered glucose tolerance at least in adults <sup>46, 47</sup> and obesity in both adults and children <sup>48, 49</sup>, which in turn are related to development of diabetes, but sleep disturbances also negatively influence cognitive and emotional functions<sup>50-52</sup> and is a probable factor in elevated chronic stress levels<sup>53</sup>.

Sleep is a complex phenomenon and disturbances therein may be associated with many factors. Physiological problems such as sleep apnoea or pain are likely to cause sleep disturbances, but they may also arise out of psychosocial factors such as crowded living<sup>54</sup>, ethnical background<sup>54, 55</sup>, birth order<sup>54</sup>, and family stress<sup>56, 57</sup>.

Sleep disturbance in children appears to be rather common: 25-30% of all children are estimated by Ward & Mason<sup>58</sup> to have some sort of sleep disturbance. In most cases these disturbances disappear of their own, nevertheless they seem to have some degree of stability over time (r = 0.29 according to Gregory & O'Connor <sup>59</sup>) – i.e. young children with sleep disturbances. The predominant sleep disturbances in children consist of unspecified difficulties in falling and staying asleep – dyssomnias – or parasomnias such as sleep terrors, nightmares and sleepwalking <sup>58</sup>.

Night-wakings may actually not be a problem for the child, even though it may be one for the parents, causing undue concern and/or sleep disruption on their part if the child does not soothe itself. Brief night wakings are normal <sup>58</sup> and occur 5-8 times in the typical sleep pattern even of a tenyear-old <sup>60</sup>, usually without being noticed by neither child nor parent. In infants, McKenna <sup>61</sup> has proposed that night wakings are a necessary component of normal brain development.

As described above, cortisol is highly involved in the human stress response. Since it is also an important regulator of the circadian sleep-wake cycle, it should come as no surprise that sleep and stress are highly connected to each other – cortisol being but one of many probable connections<sup>53</sup>. Regardless of the reason for elevated average cortisol levels

in a human, it results in both increased allostatic load (see the section on stress theory) and disturbed sleep.

For a parent, few problems are so stressful as their children's sleep problems. Having researched child sleep literature helped relieving my own stress at my infant daughter's sleep refusal, but nothing could entirely counter the frustration and feelings of helplessness in the face of yet another cry in the dark. In earlier research, parenting stress has already been shown to have an association to child sleep disturbances<sup>62, 63</sup>. This association could perhaps be viewed as an inter-individual connection between sleep and stress.

## Aims

Could it be said that sleep, stress and health are all equally fundamental cogwheels in the human machinery? If so, then turning one will influence all the others and none can be said to have primacy in a causal chain of event; for instance, one might as well say that poor sleep causes stress which in turn causes ill-health, as that ill-health causes stress which causes poor sleep, or any other arbitrary order. In addition, other cogwheels are in operation, including relations to other human individuals. Thus viewed, the state of a human being deserves to be assessed in a holistic perspective. One must, however, remember not to exaggerate the possibilities of such a perspective: the position of one of the cogwheels does not enable us to extrapolate the position of all others. It is necessary to take several pieces of the puzzle together to give us a better chance of guessing the whole picture.

On these grounds, the scope of this thesis is an investigation of the relationships between a few of the cogwheels, first and foremost the sleep of parent and child, as rated by the parent, and stress in the parent, as measured by the Swedish Parenting Stress Questionnaire (SPSQ). Also, some possible other factors are taken into consideration, such as if the parents were born outside of Sweden (a situation which may serve as a stressor), child temperament and the parent's satisfaction with her/his social support. In the end, a possible model for how the different cogwheels fit together is presented.

## Hypotheses

The hypotheses are summarily listed at the end of this section. The following is a description of the reasoning behind them.

What should we expect when we investigate the associations between child and parental sleep quality and parenting stress? Since our data are all based on a questionnaire taken by a single respondent each, they are definitely related in the sense that in each case, they are the outcomes of a single person's perceptions. All these perceptions are formed by the same brain, working according to its own, personal pattern – it is the same person who has rated her/his own sleep quality and that of the child as well as the items composing the SPSQ.

Considering the prominence of feedback loops in the human brain and its tendency towards post-hoc rationalisations, all these perceptions may be related in a bidirectional manner. Perceived child sleep may be influenced by the respondent's sense of own sleep quality as well as the other way round, and sleep and stress measures should influence each other, while they are all in turn related to other perceptions, such as social support and child temperament.

Thus when performing statistical analyses on our data, each and every measure may be designated as a dependent variable; they should all be inter-dependent. The question is not which variable is the dependent, but rather to which degree they depend on each other. This is the reason for the choice of statistical method outlined in the Methods section.

That parental sleep and parenting stress should be highly interdependent would come as no surprise, considering the large number of studies indicating the connection between sleep and stress. Also, as noted above, child and parental sleep should interdepend to a large degree. But may child sleep quality also be directly connected to parenting stress? It is possible that a perceived poor child sleep quality increases feelings of incompetence ("It's my fault she doesn't sleep") and restriction in your personal life ("Why can't he sleep so I can get some time to myself?"), as well as putting strain on the relationship to the other parent ("It always seems to be up to me to tackle the bedtime hassle") – all of which may be considered components of parenting stress. It may also be a source of worry that the child's health might be compromised for lack of sleep. This is the central hypothesis of this thesis: that we will see that child sleep, parental sleep and parenting stress will all show interdependence. To what degree remains to be seen.

It may also be expected that each of these phenomena will show intrameasure stability over time. Individual sleep patterns should not change haphazardly and parenting stress should rest on underlying, stable factors such as parent and child personality, socioeconomic status and family situation. This is a further hypothesis investigated in this thesis. If both of these hypotheses are validated, then it would be expected that one phenomenon at an early age may have some influence on another phenomenon at a later age, if nothing else by influencing said other, longitudinally stable phenomenon at the earlier age. The question is: could there be something else? Could for example the memory of poor child sleep at an earlier age still induce stress at a later age? This seems unlikely, yet it is something to take into account when analysing our data. If there is some such connection, then we should be able to see a connection between e.g. earlier child sleep and later parenting stress, which should remain even when controlling for the concurrent connections between child sleep and parenting stress at earlier ages.

To summarise, the main hypotheses of this thesis are:

- Concurrent child sleep quality, parental sleep quality and parenting stress all influence each other.
- Child sleep, parental sleep and parenting stress show some stability over time.
- As a result of the combination of the above, we should observe associations between these phenomena across ages.

In addition to these hypotheses, some additional questions arose during the progress of the work concerning the connection between the above phenomena and other factors, based on previous research. The following factors were investigated for possible associations with reporting poor sleep quality and high parenting stress:

- Uncertainty about the cause of night wakings at an age of one year
- High child temperament ratings
- Both parents having been born outside of Sweden
- Single parenthood
- Dissatisfaction with social support
- Firstborn child (at least at early ages)
- Low parental educational level

And finally:

- Night feeding at an age of 1 year was investigated for possible association with more frequent night wakings at higher ages.
- High parental age was investigated for possible association with high parenting stress, at least in the Role restriction subscale (see Methods section), and to possibly have some effect on sleep measure reports, without any prediction about direction.
- Child gender was believed to have no association to stress or sleep
- The occurrence of any life event was investigated for possible association with a higher risk for subsequently reporting parenting stress.

# **Methods**
# Procedure

The data on which this thesis is based come from the ABIS project (All Babies In South-east Sweden). Out of 21700 children born in that area between October 1, 1997, and October 1, 1999, 17055 (78.6%) became part of the project after informed consent by the parents of the children. The aim of this project is to investigate possible environmental factors in the development of Type I diabetes and other immune-mediated diseases. The cohort has been followed from birth and longitudinally at regular intervals: 1, 2.5-3, and 5-6 years of age. For convenience, the time-points will henceforth be referred to as ages 0 (birth), 1, 3 and 5. At each timepoint, biological samples and questionnaires were collected. Questionnaires were filled out by either of the parents at regular well-child clinic check-ups, in which 99% of Swedish parents participate, at the above-mentioned time points. The questionnaires were filled out either at the clinic or at home. No reminders were used.

# Participants

16070 parents handed in questionnaires at age 0 (birth), 11090 at age 1, 8805 at age 3, and 7443 at age 5. Some additional questionnaires were handed in after birth, making the total number of participants 16468. Questionnaires were consecutively entered into the questionnaire data base with no specific selection criteria. More than 90% of the questionnaires were filled in by the mother. The follow-up cohorts were all representative of the age 0 cohort concerning parental age and educational level<sup>44</sup>. Questionnaire data were excluded if the reported age of the child fell outside certain ranges. These were at age 1: 8-18 months (70 cases excluded), at age 3: 24-48 months (133 cases excluded) and at age 5: 49-76 months (44 cases excluded.) Hence 11020 cases at age 1, 8672 cases at age 3 and 7399 cases at age 5 were analysed. The exact numbers in each analysis varied due to internal dropout.

# Measures

The central phenomena of this thesis are child sleep, parental sleep and parenting stress. They are described and discussed in the next section. Some additional phenomena were hypothesised to have an influence on the sleep-stress complex. These are child temperament, child gender, firstborn child, social support satisfaction, parental age and education, parental origin of birth and single parenthood.

The measurement of all these phenomena will be described in the following section. For descriptive statistics of the distribution of these measures, see Results and Appendix.

### **Central measures**

#### Sleep measures

#### Child Sleep Quality

This was assessed at age 1 and up with the question "How would you rate the quality of your child's night sleep?" with answers on a Likert scale ranging from *Very good* to *Very poor*. The Likert scale was changed from year 3 on: a 5-point response scale was used at age 1 but 6-point scales at ages 3 and 5. The reason for this was to force a choice between slightly better or slightly worse, avoiding the undefinable middle value. By choosing the one and two endpoint values respectively, we attempted to study the groups consisting of parents who have reported their sleep as at least worse than just a little poor. Thus, *poor sleep* was defined as a value of 5 at age 1 and a value of 5-6 at ages 3 and 5.

#### Number of Wakings per Night

This was assessed at each age with the question "How many times does your child usually wake up at night?" Options were *Never*, 1, 2, 3, 4, 5 *times*, and 6 *times or more*. *Many* wakings was defined as  $\geq$ 4 at age 1;  $\geq$ 3 at age 3; and  $\geq$ 2 at age 5.

#### Parental Sleep Quality

This was assessed with the question "How do you sleep yourself at night?" This item was included in the questionnaires at ages 3 and 5 but not at age 1. The scales and definition of *poor sleep* were identical with those for the children at the same time points.

#### Bedtime

The time of putting the child to bed was assessed with the question "At about what time in the evening do you put your child to bed for the night?" Options were given in full clock hours, from 4 pm to 12 pm or later. The results were categorised into Early (17-18; below the 5:th or lower possible percentile), Normal (19-21) and Late (22 or later; above the 95:th or higher possible percentile).

#### Risetime

The time when the child woke up/was roused was assessed with the question "At about what time in the morning do you take your child out of bed / your child rise?" Options were given in full clock hours, from 4 am to 12 am or later. By the same standards as for *Bedtime*, the results were categorised into *Early* (4-5), *Normal* (6-8) and *Late* (9 or later).

#### Number of Night Hours in Bed

The time that the child spent in bed was defined as the hour difference between bedtime and risetime. Note that this does not inform us on how many hours were spent in actual sleep. By the same standards as for *Bedtime*, the results were categorised into *Few* (5-9), *Normal* (10-12) and *Many* (13-15) *Night Hours in Bed*.

#### Cause of Wakings

What the parents believed to be the cause of night wakings was assessed at age 1 with the question "If your child tends to wake up at night, what do you believe is the usual cause?". The options were "Hungry"; "Seems to be in pain"; "Worried"; "Woken by sibling"; "Woken by parent" and "Noise" and multiple choices were possible. These options were then dichotomised into *Specific causes*, represented by hunger, sibling, parent or noise and the *Unspecific causes* worry or pain. The idea behind this dichotomisation was that "*Seems* to be in pain" (italics added) and the single, unspecified word "Worried" seem to hint at a greater uncertainty about why the child wakes up, and that this uncertainty may be a cause of stress or worry that could make the parent more prone to reporting poor sleep.

#### Stress measures

To measure parenting stress, an adaptation of the Parenting Stress Index (PSI<sup>64</sup>) was used, namely the Swedish Parenting Stress Questionnaire (SPSQ) The SPSQ has been validated for Swedish conditions<sup>43</sup> and

consists of 34 items with answers on a 6-point Likert-type response scale, ranging from *Strongly disagree (1)* to *Strongly agree (6)*. The items are divided into five subscales: *Incompetence* (11 items), *Role Restriction* (6 items), *Spouse Relationship Problems* (5 items), *Social Isolation* (7 items), and *Health* (4 items). Examples of items in the different subscales are (my translation): "Being a parent is harder than I thought" (*Incompetence*), "The needs of the child/ren usually dominate my life" (*Role Restriction*), "Having children has brought me and my spouse closer together" (*Spouse Relationship Problems*, reversed item), "I feel alone and without friends" (*Social Isolation*), and "During the past half year, I've felt more tired than usual" (*Health*). SPSQ has shown good internal reliability (Chronbach's alpha  $\geq 0.65$  for all subscales<sup>43</sup>, and in our cohort  $\geq 0.88$  for the scale as a whole at different time points<sup>44</sup>).

The Role restriction subscale originally contains seven items. However, one of these concerns a change in sleep patterns and is therefore a likely confounder when analysing relations between sleep and stress. Hence, this item was excluded in the analyses.

In the age 1 and 3 questionnaires, all subscales were included, while *Social Isolation* and *Health* were excluded at age 5, chiefly because of space priorities. Therefore, only *Incompetence, Role restriction* and *Spouse relationship problems* were included in the analyses of this thesis. Mean values were calculated at all ages, for each of the three included subscales if one or less items were missing and also for all three combined, if five or less items were missing. Since these are not continuous variables, a dichotomised variable was created based on the mean value. *High parenting stress* was defined as a mean value above the 95<sup>th</sup> percentile. For convenience, when the term *stress* is used alone in this article, *parenting stress* is intended unless otherwise specified. For cut-off values and distribution, see table 5 in Results.

Other stress measures were possible. At ages 3 and 5, a brief life events checklist was included in the questionnaires, covering the time from the child's birth and onward. This measure was also analysed, but was not found to provide any additional information. As previously discussed, life events measure the occurrence of acutely stressful events and thus in themselves probably cause little in the way of long-term stress effects. However, they may be the source of subsequent chronic stress such as economic hardship. If this has happened in the ABIS participants, it should also be reflected in the SPSQ measures. Therefore, a brief analysis of the possible association between these measures was performed.

The quantity and quality of social support and some items reflecting parental worries about the welfare of the child were also assessed. These could be used as stress measures. However, in this thesis only social support is used, but not as a direct stress measure. It is considered a peripheral measure and is described in more detail below.

Koch *et al* used a composite measure derived from the same data set, which included SPSQ, life events, parental worries and social support<sup>44</sup>. This measure was also tried but not found to add clarity nor depth to the current context.

### **Peripheral measures**

### Child temperament

The "Fussy-Difficult" subscale from the Child Characteristic Questionnaire  $(CCQ^{65})$  was included in the age 3 questionnaire to assess child temperament. This instrument consists of seven questions with Likert scales between 1-7, where higher values indicate a more *difficult* temperament. The mean value of the seven items was calculated and a *difficult* temperament was defined in paper 1 as a mean above the 90:th percentile, i.e. of 4.5 or higher. A stricter cut-off value of 5 or more was also tested and yielded similar (and even somewhat stronger) results. The measure showed a good internal consistency in our data (Chronbach's alpha = 0.83).

### Social support dissatisfaction

A measure of social support was included in the age 5 questionnaire in the form of ten items assessing two aspects of support: a) the quantity of social support available from family, friends and neighbours, concerning parenting, emotional and general issues, and b) the perceived quality of the support received. The measure was derived from Crnic et al<sup>66</sup> and has also been used by Östberg and Hagekull<sup>63</sup>. A 5-graded Likert scale ranging from very *satisfied* to very *dissatisfied* was used and a mean value calculated if one or no item was missing. Values above the 95<sup>th</sup> percentile were defined as *dissatisfaction with social support*. The measure showed a reliability of  $\alpha = 0.88$  (10 items).

### Both parents born outside of Sweden

This was measured by the questions "Were You born in Sweden?" and "Was the child's father born in Sweden?". The options were: Yes / No / Don't know. Based on these data, the respondents were divided into two categories: those where none of the parents were born in Sweden and those where at least one parent was born in Sweden.

### Single parenthood

This was measured at ages 0, 3 and 5 with the question "Which is your family situation?" Options were: Single / Cohabiter / Married. Cohabiter and Married were grouped into a single category called Not single. Also, in the above-mentioned life events checklist, one of the questions concerned the occurrence of a divorce.

### First child

Whether or not the child was the first-born to the parents was assessed at age 0 by the question "Does the child have any siblings?" Options were "Yes", "No" and "Don't know". The latter option was recoded as missing value, while the answer "No" was interpreted as the child being the first-born to the parents.

### Parental education

This was assessed by the question "Which is your level of education?" The options were "Elementary School;" "High School, practical program;" "High School, theoretical program;" "Folk High School;" "College 1-3 yrs;" and "College/University, 3.5 yrs or more." Three categories were defined depending on the parent's level of *theoretical* education: *None* (Elementary School and High School, practical program); *Some* (High School, theoretical program and Folk High School); and *Higher* (College 1-3 yrs and College/University, 3.5 yrs or more.)

### Parental age at birth of child

This measure was derived from the birth dates of the responding parent and the child, which was provided by their civic registration numbers. Categorising people into groups based on age easily becomes arbitrary, such as using five- or ten-year intervals starting from majority age. Therefore, a categorisation based on what life phase a person can be expected to be in was sought. The Swedish Statistical Central Bureau has introduced two age definitions based on statistical occupational status<sup>67</sup>: Entry age, defined as the age group in which 50% of the population has employment, and Establishment age, which was defined at 75% employment. This allows the definition to fluctuate between years; see table A2 in Appendix for the age definitions pertaining to our data. Parents were thus assigned to three categories: Youth, Entry age and Establishment (Est.) age. Youth was defined as the group younger than Entry age. Quartiles were also used to divide the parent population into age categories tested in analyses, see table A1 in Appendix for age quartiles.

### Night feedings

The amount of night feeding was measured at age 1 by the question "How often does the child eat at night?" Options were: Never; 1; 2; 3; 4; 5; and 6 times or more.

# Ethical considerations

Parental consent was given after oral and written information about the ABIS study. Prospective participants were also offered the opportunity to watch a video about the project before the child was born. Active return of a completed age 0 questionnaire and/or biological samples was considered as informed consent.

The ABIS project and the current study were approved by the Research Ethics Committees of the Faculty of Health Science at Linköping University, Sweden, and the Medical Faculty at the University of Lund, Sweden.

As this is a project involving a large number of healthy children, too young to give their own informed consent, and who probably will earn no therapeutic benefits from participating, the ethics of the ABIS study has been regarded as so important that these questions have been studied in a number of publications <sup>e.g. 68, 69, 70</sup>. In summary one may say that the majority of parents are very positive to participation in the study. Furthermore, participation in the ABIS study seems to have had a calming effect on the great majority of the participants and very few have become more worried<sup>71</sup>

# Statistical analyses

Statistical tests were performed with the statistical software SPSS for Windows v15.0, by SPSS Inc. Odds Ratios (ORs) were obtained and are presented with 95% confidence interval in parentheses. Note that only  $\chi^2$  values significant at the <0.001 level are considered statistically significant due to the large sample size. Significances at the <0.01 and <0.05 levels (indicated in the tables by a superscribed figure 1 and 2, respectively) should be regarded as strong and weak statistical trends rather than significant.

Crude ORs were obtained for the three central measures across the three age groups 1, 3 and 5 by two-by-two crosstabulation, using  $\chi^2$  tests to obtain *p*-values. Thereafter, ORs were adjusted for all other measures through logistic regression, using the Wald statistic for *p*-values (within age 1, where only the two measures of child sleep and parenting stress were available, logistic regression was not meaningful). Longitudinal difference in distributions within measures were tested using the sign test. Hypothetically, all associations may be bidirectional (see Introduction  $\rightarrow$ Hypotheses). Therefore, in the logistic regressions, one measure at a time was designated as the dependent variable, including all other concurrent and preceding measures as independent variables. The reference group for each variable was the entire population outside the focal group (e.g. if *high parenting stress* values *below* the 95<sup>th</sup> percentile).

After analysing the associations between the central measures, the peripheral measures were included in the analyses by the same method: first crude two-by-two crosstabulation, thereafter logistic regression.

# Results

Ass	<b>50c.</b> <sup>3</sup>	Crude ORs	Adjusted ORs
1	u, d	3.6 (2.4-5.3)	n/a
2	u d	22.7 (16.6-31.1)	20.1 (13.9-29.1) 20.3 (14.0-29.3)
3	u d	10.4 (6.3-17.3)	16.2 (8.1-32.5) 16.5 (8.1-33.3)
4	u, d	4.5 (3.5-5.8)	4.5 (3.2-6.3)
5	u d	4.3 (3.2-5.6)	4.5 (3.1-6.7) 4.5 (3.0-6.6)
	6	12.6 (7.6-20.9)	6.4 (3.5-11.7)
	7	15.7 (7.8-31.5)	13.1 (4.9-35.2)
	8	6.9 (5.4-8.9)	6.8 (4.9-9.3)
	9	25.1 (19.5-32.3)	22.9 (17.6-29.9)
	10	20.5 (15.4-27.2)	9.9 (6.7-14.6)
	11	14.7 (11.1-19.5)	5.5 (3.7-8.2)
	12	5.2 (3.4-7.9)	2.7 (1.6-4.5)
	13	3.6 (2.2-5.8)	$2.5(1.3-4.7)^{1}$
	14	3.8 (2.7-5.2)	$1.8(1.1-2.8)^2$

Table 1. Odds ratios for associations in Figure 1a and 1b.

 $^{1} p < 0.01$   $^{2} p < 0.05$  All others: p < 0.001

Note that values in the Crude ORs column show odds ratios for the unnumbered paths in Figure 1a (please consult figure 1b for numbers).

*p*-values for logistic regression derive from Wald statistic, for crosstabulation from  $\chi^2$  test.

<sup>3</sup>The "u" (up) and "d" (down) refers to the direction of the logistic regression in Figure 4b. For paths 1u, 2u, and 3u, child sleep is the designated dependent variable, whereas for 1d, 2d, and 3d it is parental sleep. For 4u and 5u it is parental sleep, whereas for 4d and 5d it is parenting stress.

#### Key to figures 1 a & b

C Sleep = Poor Child Sleep P Sleep = Poor Parental Sleep P Stress = High Parenting Stress



**Figure 1a.** Crude odds ratios for observed associations between child sleep, parental sleep and parenting stress in age groups 1, 3 and 5. Thickness of arrows indicates strength of association by odds ratio intervals; see key. \* This association is p < 0.05, all other associations (arrows) are p < 0.001.



**Figure 1b.** Adjusted odds ratios for observed associations between child sleep, parental sleep and parenting stress in age groups 1, 3 and 5. Thickness of arrows indicates strength of association by odds ratio intervals; see key. Arrows "u" (up) and "d" (down) indicate which variable is dependent in each separate analysis for the concurrent paths 1-5, see Table 3. Odds ratios and p-values for each association number are also given in Table 3.

## Associations

### Child / parental sleep quality and parenting stress

In testing the main hypotheses of this thesis, odds ratios were obtained for the associations between child and parental sleep quality and parenting stress, first two by two in crosstabulation, second through binary logistic regression to adjust for all concurrent and preceding measures, as described in Methods above. Adjusted odds ratios for the associations that remained significant after logistic regression are shown in table 1, along with the crude odds ratios for the same associations.

Figure 1a shows all associations found significant before adjusting, while figure 1b shows remaining associations after adjusting. As seen from the tangle of figure 1a, more or less all measures showed interconnectedness, just as hypothesised. The measures show stability over time as well as associating concurrently, and indeed association between measures over time is also observed. One exception is that child sleep quality and parenting stress at age 5 show no significant association even before adjusting, which is not in accordance with hypothesis. It may be an effect of the small number of cases reported with poor child sleep at age 5. Figure 1b, on the other hand, shows us that contrary to hypothesis, adjusting eliminates the significance of the direct association between child sleep quality and parenting stress even at age 3, leaving only an indirect association through parental sleep quality. However, the longitudinal associations *between* measures have all but disappeared.

The crude odds ratio between poor child sleep and high parenting stress (Combined scale) within the group which did *not* report poor parental sleep at age 3 is significantly high at 4.1 (2.1-7.9, p < 0.001). However, adjusting within the same group for poor child sleep and high parenting stress at age 1, the OR for the Combined scale sinks to 2.7 (1.0-7.4, p = 0.053). Including any one of the background factors (i.e. the peripheral measures: child temperament, child gender, firstborn child, social support satisfaction, parental age and education, parental origin of birth and single parenthood) in the binary logistic regressions had no significant impact at all on the odds ratios presented in table 1.

### **Background factors and sleep/stress**

Tables 2-4 show the odds ratios for associations between background factors and child sleep, parenting stress, and parental sleep, in that order. Table 2 is mostly identical with the lower half of table 3 in paper 1, but contains a few errata. Table 3 includes odds ratios for the Combined three scales as well as for each of the three subscales included at all ages.

#### Child temperament

High child temperament rating was strongly associated with poor child and parental sleep quality and high parenting stress, see Appendix for exact odds ratios. At age 3, when child temperament was assessed, the association with Combined stress had an OR of 4.8 (3.9-6.0). In the subscales, it was strongest for Incompetence: OR 5.4 (4.3-6.7), next for Role restriction: 3.1 (2.4-3.9), then for Spouse relationship problems: 2.2 (1.7-2.9), all at p < 0.001. There were also significant associations to Combined stress and all subscales at ages 1 and 5, with odds ratios ranging from 2.0 to 4.2, p < 0.001 (specific data not shown), except for Spouse relationship problems at age 5, which had an OR of 1.6, p < 0.05. The pattern of association when using a stricter percentile is identical but with somewhat higher odds ratios.

Child temperament was also associated to sleep variables at all ages. At age 3, the OR for many night wakings was 3.2 (2.4-4.4), for poor child sleep quality it was 4.4 (3.1-6.1), and for poor parental sleep quality it was 2.9 (2.3-3.6), p < 0.001 for all three.

#### Social support dissatisfaction

This measure also showed significant or trends towards associations to all sleep and stress measures at all ages. At age 5, when it was assessed, the odds ratios were (p < 0.001 unless otherwise specified): for many night wakings 2.4 (1.5-3.8), poor child sleep quality 2.4 (1.1-5.4, p < 0.05), poor parental sleep quality 3.2 (2.4-4.2), Combined stress 10.8 (8.3-14.0), Incompetence 7.0 (5.3-9.2), Role restriction 6.1 (4.6-8.1) and Spouse relationship problems 7.0 (5.3-9.3).

	1	Age 1		Age 3	Age 5	
Background factor	MW	LSQ	MW	LSQ	MW	LSQ
Foreign-born parents	2.1 (1.4-3.2)	4.7 (2.8-8.0)*	ns	ns	ns	ns
Father of Est. age	2.1 (1.4-3.3)	$1.4(1.0-1.9)^2$	ns	ns	ns	ns
Father of Entry age	0.45 (0.28-0.70)	) ns	ns	ns	ns	ns
Mother No Theor. Edu.	ns	$1.4(1.1-1.9)^{2*}$	ns	$1.5(1.1-2.1)^{1}$	ns	ns
Father No Theor. Edu.	ns	$1.4(1.1-2.0)^{2*}$	ns	ns	ns	ns
Mother High Theor. Ed.	ns	ns	ns	$0.61 (0.45 - 0.83)^1$	$0.69 (0.49-0.96)^{2*}$	ns
Significant at $(\chi^2)$ :	$^{1}p < 0.01$ $^{2}$	p < 0.05 else: p <	0.001		* Errata for pa	per 1

Table 2: Odds ratios between background factors and many night wakings (MW) / low child sleep quality (LSO).

Table 3:	Odds	ratios	for	associations	between	background	factors and	parenting stress.

		SPSQ		SPS	Q scale	
Measure		at age	Combined	Incompetence	Role restriction	Spouse rel prob
	0	1	2.5 (1.5-4.3)	ns	ns	6.2 (3.9-9.9)
Single parent at age	3	3	4.1 (3.0-5.5)	$1.5(1.0-2.3)^2$	$1.5(1.1-2.2)^2$	45.5 (34.0-60.9)
	5	5	4.1 (3.1-5.5)	ns	$1.5(1.0-2.1)^2$	28.5 (21.5-37.8)
		1	$1.3(1.1-1.5)^{1}$	ns	1.6 (1.3-1.9)	$1.2(1.0-1.5)^2$
Mother of establishment a	ge	3	1.4 (1.2-1.8)	ns	1.6 (1.3-1.9)	ns
		5	ns	ns	$1.4(1.2-1.8)^{1}$	ns
		1	1.5 (1.2-1.8)	$1.4(1.1-1.7)^{1}$	1.7 (1.5-2.1)	1.4 (1.1-1.6) <sup>1</sup>
Mother's age above 3 <sup>rd</sup> qua	artile	3	1.6 (1.3-2.0)	ns	1.7 (1.4-2.1)	ns
		5	$1.3(1.1-1.7)^2$	ns	1.6 (1.3-2.1)	ns
		1	ns	ns	ns	$0.65(0.49-0.87)^{1}$
Father of establishment ag	ge	3	$2.0(1.2-3.3)^{1}$	ns	$1.8(1.1-2.9)^2$	$0.68 (0.48-0.95)^2$
		5	ns	ns	$1.9(1.1-3.3)^2$	ns
Eather's age above 2rd que	rtila	3	$1.4(1.2-1.8)^{1}$	$1.3(1.0-1.6)^2$	ns	ns
Tamer's age above 5 qua	luie	5	ns	ns	$1.3(1.0-1.7)^2$	ns
Both parents born outside	Sweden	1	$1.8(1.1-2.8)^2$	2.3 (1.5-3.6)	$1.7(1.1-2.7)^2$	ns
Significant at $(\chi^2)$ : <sup>1</sup> n	< 0.01	2n < 0	05 else: $n < 0$	001		

P : 0.01	P \ 0.00	

factors / cause of wakings and pare	ental sleep qual	ity.
	Poor parental	sleep quality
Measure	Age 3	Age 5
At least one unspecific cause of waking	1.5 (1.2-1.8)	1.5 (1.2-1.8)
Multiple causes, at least one unspecific	$1.4(1.1-1.8)^2$	ns
Mother No Theoretical Education	$1.3(1.1-1.6)^{1}$	ns
Father No Theoretical Education	$1.2(1.0-1.5)^2$	ns
Single parent at age 3	1.9 (1.4-2.6)	ns
Single parent at age 5	$1.7 (1.2-2.5)^1$	$1.6(1.2-2.2)^{1}$
Significant at $(x^2)$ : ${}^{1}n < 0.01$ ${}^{2}n < 0.05$	else: $p < 0.001$	

Table 4: Odds ratios for associations between background

Significant at  $(\chi^2)$ :  $p^{2} < 0.01$   $p^{2} < 0.05$  else: p < 0.001

### Both parents born outside of Sweden

There were associations between two foreign-born parents and child sleep / parenting stress variables but only at age 1, see tables 2 and 3. The dropout within the group where both parents were born outside of Sweden was somewhat higher than for the cohort as a whole, especially between ages 0 and 1 (see table A3 in Appendix). The cases who dropped out from this group from age 3 to 5 had a higher risk for Spouse relationship problems at age 3: OR 5.2 (1.0-26.5, p < 0.05); the

corresponding odds ratio for the entire cohort was a weaker but significant 1.4 (1.1-1.7, p < 0.01). The dropouts within this group were not significantly different from the remainers in any other of the measures in this study.

### Single parents

Parents who reported singlehood at each age also had increased ORs for high Combined stress at the same age, see table 3 (note that those who reported singlehood at age 0 were tested against parenting stress at age 1, since family status was not assessed at age 1). However, the absolutely major part of these odds ratios seem to derive from the subscale spouse relationship problems. The role restriction subscale only shows a weak trend at age 3 and 5 and incompetence only at age 3. No significant associations were found to child sleep variables other than higher odds ratios for reporting *no* night wakings, see paper 1, table 4. Singlehood at ages 3 and 5 was associated with poor parental sleep at the same age, weaker for age 5 (see table 4). Note that there is also an association between poor parental sleep quality at age 3 and single parent at age 5. However, adjusting for single parenthood at age 3, this association becomes non-significant.

Since singlehood was associated with both parental sleep quality and parenting stress, which in turn were associated with each other, a binary logistic regression was performed on these measures (including singlehood and stress at age 1), whereupon all associations remained significant at about the same levels (data not shown).

### Firstborn child

Parents who had no children before the child participating in the ABIS study showed a weak trend towards reporting many night wakings at ages 1 and 3 and poor child sleep quality at age 1 (ORs below 1.5, p < 0.05). They also had a higher risk for high Incompetence and Role restriction stress at age 1: ORs of 1.7 (1.4-2.1) and 1.5 (1.2-1.8) respectively, p < 0.001, but a weak trend towards *lower* risk for high Incompetence stress at age 5, OR 0.75 (0.60-0.94, p < 0.05)

### Parental education

Parental education showed a weak trend in associating with child sleep measures, such that parents with a purely practical education had a somewhat increased and mothers with higher theoretical education a somewhat decreased risk for reporting sleep disturbances at certain ages, see table 2.

Education also showed a very weak association with parenting stress. The only significant results were that mothers with higher theoretical education had a slight trend towards *lower* risk and those with a practical (i.e. non-theoretical) education a correspondingly slightly *higher* risk for high stress in the Spouse relationship problems subscale at all ages (data not shown), and that if the father had a higher theoretical education, there was a slight increase in risk for high Role restriction stress at age 1 of OR 1.3 (1.0-1.5, significant at < 0.05).

Parents with no theoretical education also showed a very weak trend towards poor parental sleep quality at age 3, see table 4.

Looking at the results for father's education in tables 2 and 3, bear in mind that it is usually the mother who has answered the questionnaire. If we look at education within the group where the father is the respondent, then at age 3, fathers with purely practical (i.e. no theoretical) education had a trend towards increased risk for reporting high Role restriction and Spouse relationship problems, odds ratios of 4.0 (1.3-11.9, p < 0.01) and 3.9 (1.1-13.2, p < 0.05) respectively, while the Combined stress was not significant. At age 5, the same fathers had a trend towards reporting many night wakings, OR 2.7 (1.0-6.9, p < 0.05) but nothing else significant.

#### Parental age

There were some associations between father's age and child sleep measures at age 1, especially night wakings – see table 2. More associations were found between both mother's and father's age and parenting stress at all ages, especially in the Role restriction subscale, see table 3. Generally, higher age increased the risk for high parenting stress, with the exception of Spouse relationship problems for fathers of establishment age (although *not* for fathers above the 3<sup>rd</sup> age quartile). As with father's education, looking only at the group where the respondent at age 3 is the father, the OR for fathers above the 3<sup>rd</sup> age quartile and high Role restriction at the same age is an astounding 16.6 (2.1-132, see crosstable in table A5 in the Appendix for exact numbers), while at age 5 and for establishment age fathers, no significance was found.

### Child gender

No significant associations whatever were found between child gender and higher risk for neither poorer sleep reported nor high parenting stress.

### Life events and parenting stress

There were some associations between Life events and SPSQ: in the Combined subscale, reporting any life event at ages 3 or 5 was associated with an OR of about 2 for also reporting high stress at the same age, significant at <0.001. In the Spouse relationship subscale, the OR:s were even around 3, but when eliminating all those who had reported a divorce as life event, these OR:s turned non-significant (exception: at age 5, those who reported a life event other than divorce had an OR of 1.55 (p < 0.01) for high stress in this subscale the same year). This elimination also had the effect of lowering the OR:s in the Combined scale somewhat (by around 0.3), but had little effect on the other subscales. When comparing means in SPSQ values between the groups which had/had not reported any life events, however, they differred very little (usually less than 0.1).

# Longitudinal patterns

Table 5 shows the distributions of night wakings and sleep quality, including cut-off values, as well as for parenting stress, social support satisfaction and child temperament. Due to non-normality of distribution, both mean/standard deviation and quartiles are shown. Table 6 (from paper 1) shows the odds ratios within and between child sleep measures within and across ages.

Figure 2 (also from paper 1) shows the percentage distributions of the child sleep measures in table 1. The general decrease in number of night wakings and improvement of child sleep quality between ages, which is apparent from figure 2, is significant at < 0.001 (sign test). Parental sleep quality does not change significantly from age 3 to age 5, as seen in table 5. Figure 3 contains boxplots showing the distributions of SPSQ values on the three scales used and the combined scale, for each age group. From table 1 and figure 3 both, we see that a marked decrease in overall stress occurs in the Incompetence, Spouse relationship problems and Combined subscales from age 1 to age 3, then it remains more or less the same from age 3 to age 5. In the Role restriction subscale, a steady decrease is evident from age 1, over age 3 to age 5. These decreases are statistically significant at < 0.001 (sign test), except for Incompetence and Spouse relationship problems, for which a very slight *inc*rease, too slight to be visible in figure 3, occurs from age 3 to 5 (significant at < 0.05 and 0.01, respectively).

Table 5: Distributions and cut-off value	s of dichc	tomised s	leep mea	sures, par	enting stre	ss, social sı	upport satisfa	action and	child temp	erament.	
	Age			Std.			Quartiles			Cutoff	N/% over
Measure	group	z	Mean	Dev	Min	1st	Median	3rd	Max	value	cut-off <sup>1</sup>
	1	10767	1.27	1.24	0	0	1	2	$^{+9}$	4	592 / 5.5
Wakings per night	с	8678	0.77	0.75	0	0	1	1	<del>6</del> +	ŝ	230/2.7
	5	7379	0.49	0.57	0	0	0	1	$^{+9}$	7	187/2.5
	1	10743	1.98	1.02	-	1	7	ε	5	5	202/1.9
Parent-rated child sleep quality	б	8681	1.79	0.93	-	1	7	7	9	5	174/2.0
	5	7379	1.48	0.76	1	1	1	7	9	S	63 / 0.9
Colf ana and ana and a loss and iter	б	8678	2.31	1.20	1	1	6	ε	9	S	566 / 6.5
sent-reported parential steep quanty	5	7350	2.29	1.23	1	1	7	б	9	5	516/7.0
	1	10845	2.86	0.48	1.00	2.55	2.82	3.18	6.00	3.73	565/5.3
SPSQ, Incompetence	ю	8458	2.27	0.71	1.00	1.73	2.18	2.73	5.73	3.55	407 / 4.8
	5	7177	2.29	0.72	1.00	1.73	2.18	2.73	5.45	3.55	343 / 4.8
	1	10787	3.93	0.99	1.00	3.33	4.00	4.67	6.00	5.50	636 / 5.9
SPSQ, Role restriction	б	8710	3.84	0.99	1.00	3.17	3.83	4.50	6.00	5.40	437 / 5.0
	5	7330	3.66	0.99	1.00	3.00	3.67	4.33	6.00	5.20	365 / 5.0
	1	10783	2.93	0.58	1.00	2.60	3.00	3.20	6.00	4.00	552 / 5.2
SPSQ, Spouse relationship problems	б	8373	2.29	1.01	1.00	1.60	2.00	2.80	6.00	4.20	414/4.9
	5	6669	2.30	1.00	1.00	1.60	2.00	2.80	6.00	4.20	336/4.8
	1	10778	3.16	0.50	1.43	2.83	3.13	3.48	5.36	4.00	566/5.3
SPSQ, Combined	ю	8658	2.71	0.68	1.00	2.22	2.65	3.13	5.43	3.91	429 / 5.0
	5	7287	2.67	0.68	1.00	2.17	2.61	3.13	5.35	3.73	350/4.8
Social support satisfaction	5	7002	1.67	0.70	1.00	1.10	1.40	2.00	5.00	3.10	376 / 5.4
Child temperament	с	8735	3.25	0.90	1.00	2.57	3.14	3.86	6.86	4.50	796/9.1
<sup>1</sup> Cut-off values for SPSQ variables wei	e defined	as a mea	n above th	ie 95 <sup>th</sup> pe	rcentile. H	owever, the	exact perce	ntage of th	e group de	efined with	high stress
varied due to the non-continuity of the 1	nean of L	ikert scal	e items.								

longitudinally	
y and	
concurrently	
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child	
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and	
between	
s ratios	
Odds	
Table 6:	

	Å	ge 1	Ag	e 3	Age	5
Sleep measure	MW	LSQ	MW	LSQ	MW	LSQ
1	n/a	26.5 (22.0-31.9)	9.8 (7.1-13.5)	6.3 (4.2-9.3)	5.5 (3.7-8.3)	4.3 (2.1-9.0)
MW at age 3	9.8 (7.1-13.5)	6.4(4.7-8.6)	n/a	60.2 (42.8-84.7)	14.3 (9.4-21.9)	8.9 (4.3-18.8)
4)	5.5 (3.7-8.3)	2.8 (1.9-4.2)	14.3 (9.4-21.9)	10.7 (6.5-17.6)	n/a	27.1 (15.9-46.1)
1	26.5 (22.0-31.9)	n/a	6.4 (4.7-8.6)	7.1 (5.0-10.1)	2.8 (1.9-4.2)	5.2 (2.9-9.6)
LSQ at age 3	6.3 (4.2-9.3)	7.1 (5.0-10.1)	60.2 (42.8-84.7)	n/a	10.7 (6.5-17.6)	15.7 (7.8-31.5)
4)	6 4.3 (2.1-9.0)	5.2 (2.9-9.6)	8.9 (4.3-18.8)	15.7 (7.8-31.5)	27.1 (15.9-46.1)	n/a
Multiple causes of waking	3.0 (2.4-3.6)	3.2 (2.7-3.7)	3.4 (2.4-4.7)	3.0 (2.1-4.5)	$1.8(1.2-2.8)^{1}$	ns
At least one unspecific d:o	4.1 (3.4-5.0)	4.7 (4.0-5.5)	3.2 (2.3-4.4)	2.7 (1.8-3.8)	1.8 (1.3-2.5)	$1.9(1.1-3.5)^2$
Cimition of $(n^2)$ . $1_n > 0$	$01  2_m > 0.05  a1_{co.1}$					

Significant at  $(\chi^2)$ :  ${}^1p < 0.01 {}^2p < 0.05$  else: p < 0.001



Figure 2: Bar graphs showing the percentage distributions of child sleep measures across age. Parentrated sleep quality is divided into two panels due to the difference in grades on the Likert scales.



# Discussion

## Associations

### Child / parental sleep quality and parenting stress

These associations are the ones around which the main hypotheses of paper 2 and this thesis are centred. For a lengthier discussion on these associations, see paper 2.

As seen from figure 1a and 1b, the multitude of associations is much reduced when put through a binary logistic regression. Most of the main hypotheses are supported, but the most important exception is that the direct association between poor child sleep quality and high parenting stress at age 3 is eliminated (recall that at age 5, none such existed to begin with and at age 1, adjusting was not possible). Perhaps parents who see their children's sleep as problematic are not so much stressed for the sake of their child, but for the quality of their own sleep?

This latter hypothesis, however, is not supported if we look only at the group which did not report poor parental sleep at age 3 and obtain the crude odds ratio between poor child sleep and high parenting stress at that age; this OR was significantly high. Yet adjusting for poor child sleep and high parenting stress at age 1 within this group, the association between poor child sleep and high parenting stress at age 3 again became nonsignificant. The association between poor child sleep quality and high parenting stress when the parent does not sleep poorly at age 3 may to a large extent be a spillover of age 1 high parenting stress associated with unassessed poor parental sleep at age 1. It is also possible that high parenting stress caused by other factors, including personality (see discussion in paper 2) may predispose parents towards rating their child's sleep quality as poor. We cannot exclude perceived poor child sleep quality as a parenting stressor in its own right on the basis of our data, nor is there any theoretical reason for doing so, but it seems to be subordinate to the stressor of poor parental sleep quality.

As reviewed by Meerlo *et al*<sup>53</sup>, not only total sleep deprivation as in laboratory conditions, but also restricted and/or interrupted sleep, such as may occur normally in the life of a parent, appears to have an impact on stress levels – at least if the lack of sleep is combined with physical and/or

cognitive demands, which would also be the case for a parent. Of special interest for this thesis is that sleep deprivation may increase *perceived* exhaustion during physical exertion, rather than influencing physiological parameters such as heart rate and metabolic rate<sup>53, 72</sup>. Furthermore, sleep deprivation may enhance negative and decrease positive emotional perceptions of daily events<sup>53,73</sup>. And as has already been argued, perceptions are of great importance in shaping a stress response. This also leads us to the question if the parents' general outlook on life -i.e.personality - may be a factor in both perceived sleep quality and parenting stress, as has already been suggested by some studies<sup>74, 75</sup>, and how this may have influenced the associations between sleep and stress. However, personality was not assessed in ABIS so this remains speculation. Thus to reduce parenting stress for parents who complain about their child's sleep, it may pay more to look at the parents' sleep than at the child's. As Sundelin & Thunström point out<sup>76</sup>, these parents are often too fatigued to seek help. This, of course, does not eliminate the need to investigate if the child indeed has a problematic sleep pattern causing excess fatigue during daytime, such as in sleep apnoea – or if, as for the vast majority, the "problem" is merely a part of a natural development and will pass with time.

### **Background factors and sleep**

In paper 1, the associations between several background factors and reported sleep quality were investigated. The strongest impression when looking at these analyses was that there are many factors which have a weak influence on parent-reported sleep variables. This is not limited to those factors presented in the paper; there were numerous other factors which were significantly but slightly associated with a difference in sleep pattern.

This is not surprising. Sleep is a complex phenomenon which is likely to be associated with a vast range of environmental factors, both psychosocial and physical. This makes it difficult to define any risk groups for poor parent-reported sleep quality. The strongest associations found between a background factor and sleep variables were child temperament, social support dissatisfaction, and when both parents were born outside of Sweden. Regarding temperament, and social support dissatisfaction, see discussion in paper 2. As discussed in paper 1, the association between parental birthplace and child sleep quality may be a result of a more stressful situation, having immigrated and trying to adapt to a new society, which may sensitise parents to sleep disturbances. It may also be a question of different cultural sleep norms and habits. Since we have no data concerning at which age nor from which country the parents came to Sweden, which should also be of importance, drawing any firm conclusions about this is impossible without further investigation. Please note the erratum concerning the number of cases where both parents were born abroad. The association to low sleep quality, however, was affected only by an increase in OR, as can be seen comparing table 5 in the results section to table 3 in paper 1 (there was no change in OR for many night wakings).

Another erratum concerns the level of theoretical education, for which parents with a purely practical education had a weak trend towards lower child sleep quality at age 1, and mothers with higher theoretical education a likewise trend towards fewer night wakings at age 5, trends which were overlooked in paper 1. However, these trends are too weak to draw any conclusions and will not be discussed further.

Single parenthood was not associated to child sleep ratings (see paper 1), but to parental sleep, as seen in table 4. This is not surprising: a single parent has no access to relief from the other parent in dealing with night wakings, but must bear the full burden alone. Researching the literature, no other studies have been found that investigate this perhaps rather taken-forgranted relationship further.

The trend for parents of firstborn children towards reporting many night wakings and poor sleep is weak in comparison to other associations found and will not be discussed further. For discussion on dissatisfaction with social support, please see paper 2.

### **Background factors and stress**

The strong association between child temperament and parenting stress is likely to be bidirectional, since we deal with parental perceptions, and perhaps to covary with other factors as well, such as parent personality (see discussion in paper 2).

As with sleep measures, the group in which both parents were born outside of Sweden had higher risk for high stress, especially in the Incompetence subscale, but only at age 1. This may perhaps be taken as support for the idea that this group has a more stressful situation while adapting to Swedish culture (see discussion in paper 1).

While parental age showed little association to sleep measures, there were indeed associations to higher stress, and especially to Role restriction stress,

as hypothesised. Odds ratios were higher for mothers both of establishment age and of an age above the 3<sup>rd</sup> quartile. The latter was higher than establishment age, and the associations were slightly stronger in the older age group. Perhaps there is something to the hypothesis that older parents are more sensitive to especially Role restriction stress, making it worthy of further investigation. One study has investigated parenting stress in older mothers and found that 0%, 22%, and 6%, in the groups aged in their 30s, 40s, and 50s, respectively, had a parenting stress above the  $80^{\text{th}}$  percentile<sup>77</sup>. Lower maternal education level was weakly associated to higher parenting stress in the Spouse relationship problems subscale at age 1. This may be a reflection of parents with higher education being more gender-aware and therefore sharing child-care and household chores more equally. When it comes to father's age and education, the associations found may be due to a correspondence between father's and mother's age and education, since it is usually the mother who has answered the questionnaire. However, isolating the father-respondents above the  $3^{rd}$  age quartile, these may be at an even greater risk – of the 11 fathers who reported high Role restriction stress at age 3 (i.e. a mean above 5.40 on items with a scale up to 6), 10 were above the 3<sup>rd</sup> age quartile (35 years), while of the 144 fathers below the 3<sup>rd</sup> age quartile, only one had reported high stress. As for the father-respondents without theoretical education, these had an increased risk for high Role restriction and Spouse relationship problems stress at age 3. However, at ages 1 and 5, no similar pattern was seen, and the percentage of father-respondents at age 3 was rather lower than at the other ages, so caution is advised in drawing any conclusions from this association.

Single parents had very high values on the Spouse relationship problems subscale, which of course is natural – if there had been no such problems, then the parent should usually not be single. There was a trend towards high stress for single parents in the other subscales as well, but perhaps less than might have been expected in such a stressful situation. Kazak and Linney argued that single parent women "feel competent in their parenting abilities"<sup>78</sup>. Perhaps being single simply forces a parent to cope? Since our ultimate study objects are the children, it would be interesting and perhaps more rewarding to directly study the children's stress levels in connection to single parenthood.

### **Between sleep variables**

One question about child sleep is the role of night feedings; some studiesseem to indicate that this habit conditions the child to wake up for feeding<sup>79, 80</sup>. The present data do not support this hypothesis, especially when analysing the group whose number of reported night feedings at age 1 was identical with the reported number of night wakings; in other words those who were fed each time they woke up at night. This should be the ideal way of conditioning a child to wake up for feeding, but this group was not significantly different from any other group with respect to reported sleep patterns (see discussion in paper 1).

One finding which was not discussed in paper 1 was the associations between late bedtime and poor sleep quality/many wakings, especially at age 3 (OR:s of 3.7 and 3.3 respectively). Quite possibly, a late bedtime is just another expression of sleep difficulties in the child – perhaps and indicator of sleep resistance. Another possibility is that the child is routinely put to bed at a later hour and that this habit is detrimental for the child's sleep. However, this is deemed to be a less likely explanation for the association.

The association between reporting unspecific and/or multiple causes of waking at age 1 and poor sleep quality *at all ages* may be interesting for identifying risk groups for persistent perceived sleep problems in a clinical setting. One hypothesis is that reporting singular, specific causes of waking is an indicator that the parents know how to cope with the sleep disturbances, which should lower the risk for reporting sleep disturbances at later ages. This is also supported by the association between multiple and/or unspecific causes of waking reported and SPSQ values (especially the Incompetence and Role restriction subscales), since one factor in all psychological stress, according to appraisal theory, is a lack of coping ability.

The association between reported night wakings and sleep quality is discussed under the heading Measures: methodology and concepts  $\rightarrow$  Sleep below.

### Patterns

#### **Sleep patterns**

#### What do the patterns really tell us?

The first article of this thesis describes the current sleep patterns of some 10,000 Swedish children. While these patterns show us what is statistically normal in this population, they do not tell us what constitutes healthy sleep. If, as suggested by McKenna<sup>61</sup>, night wakings in infants are not only normal, but necessary for their development, then judging sleep quality in infants only by number of night wakings must be utterly misleading. In later ages as well, brief night wakings appear normal and usually go undetected by the parents. It is only when too many wakings result in parental arousal and their consequent sleep disruption that they become perceived as a problem – a problem which is, perhaps, the parents' more than the child's. Modern society imposes demands on adults that do not rhyme well with lack of sleep.

Thus if we want to discuss the healthiness of existing sleep patterns, we must decide on which perspective to take; that of the actual physiology of human beings, evolutionally still perhaps best adapted to stone-age living conditions, or that of fitting this physiology into our current, tightly scheduled environment. Modern society and our biological make-up are both realities that cannot be disregarded. One may argue that the healthiest option would be to remold society to suit our bodies rather than the other way round, but from a clinical point of view, that is not an issue. The issue, instead, is how to best handle the maladies that arise from a collision between biology and culture.

To do so, understanding the nature of the collision is necessary; to study in what way the demands of our society discords with those of our bodies. Therefore, it would be of high interest to establish *biologically* healthy sleep norms, for our children as well as for adults. This, however, is beyond the scope of the ABIS data material, which instead yields information about the existing sleep patterns, as influenced by

contemporary Swedish cultural and social norms and demands and as perceived by the parents, the carriers of said norms.

#### How do they vary over time?

Most sleep disturbances seem to pass with age. As seen in table 5 and figure 2, the distributions of night wakings and sleep quality shift strongly with increasing age towards the "good quality" and "no wakings" end of the spectra.

Nevertheless, the opposite pattern also exists. As seen in table 6 (from paper 1), the odds ratios are high for poor sleepers at a young age to be reported with poor sleep at later ages as well, whereas they are low for a sound or at least non-poor young sleeper to turn into an older poor sleeper (see paper 1).

However, we must still bear in mind that we are looking at *parent-reported* sleep quality. Persistently poor reported sleep quality may well be a trait in the child, but it may also be one in the parent. Social and individual norms for what defines sleep quality, as already noted, may not correspond to the as-yet elusive objective biological norms, and subjective norms may continue to operate over the years, resulting in poor sleep quality being reported even if the child's sleep pattern is biologically sound.

### What does that mean for this thesis?

A possible theoretical implication of the attachment theory is that how the parent perceives the child, including its sleep, and how the parent reacts to that perception, will have some influence on how the child perceives itself and its situation. This perception on the part of the child may be a source of psychological stress with all its physiological consequences, as outlined in the theory section.

Thus by studying parental perceptions, we study a factor which has great potential to affect the well-being of the child during an extended period of time. While we cannot say how close to objective patterns nor how biologically healthy the patterns are, nor how persistent any biologically problematic sleep patterns may be, we can investigate the associations of sleep pattern perceptions to background factors that may contribute to the formation of the perceptions, as well as to outcomes in terms of e.g. psychological stress and/or disease (which may of course be background factors in their own right).

#### **Stress patterns**

The decrease in overall stress with increasing age which is seen in figure 3 may perhaps be taken as a sign that the parents are getting to know the new child, so that it stresses them less. This is especially so for the Incompetence and Spouse relationship problems subscales, which diminish the most after age 1 and then not at all (but rather increases, significantly but too slightly to allow for any conclusions).

For the Role restriction subscale, the pattern is slightly different. Firstly, the initial decrease is lesser but on the other hand continues at the same pace after age 3. Secondly, the means on the Role restriction subscale are distributed markedly higher than those of the other scales. If the subscales indeed measure the underlying mental constructs that they are assumed to (see discussion on stress measures below), this should mean that in this population, the most stressful thing about parenthood is that you can't live your life like before having children.

Perhaps this is telling of Swedish society, which is perceived by many as very individualistic. The high age of first-time parents in Sweden may also be a factor here, if higher age can be assumed to mean a more settled life with habits that are compromised by the arrival of children. It is beyond the scope of this thesis to further discuss this finding in itself, but it is an interesting indication for anyone who wishes to work towards alleviating parenting stress in Sweden. It is also the reason for the hypothesis that older parents have higher Role restriction means, which is discussed above under the heading Associations .

In our data, the means for the Combined scale are 3.16 (SD 0.50) at age 1 and 2.71 (SD 0.68) at age 3. At these ages, the two omitted subscales of Health and Social isolation were also included in the questionnaires, and the means for the total five scales were comparable to those for the three Combined scales.



In Östberg's & Hagekull's data<sup>63</sup>, the mean was slightly lower: 2.50 (SD 0.55). In comparing these, one must take into account that Östberg & Hagekull used a five-graded Likert scale while a six-graded scale was used

in the ABIS questionnaires, and that the children in Östberg's and Hagekull's study were of an age from 6 months to 3 years (mean age in months 21.4, SD 9.3), covering the entire span of our two groups. Also, Östberg & Hagekull used the full five subscales as opposed to the present three.

The "middle value" of "neither agree nor disagree" is at 3 on a five-grade Likert scale and at the non-selectable 3.5 on a six-graded scale. In relation to the endpoints and the middle value, Östberg's & Hagekull's mean is located at a point along the scale below the middle value, comparable to our two means (see figure 4). As already noted, our means separate significantly into one slightly higher for age 1 and one slightly lower for age 3. One explanation for this may be that it reflects the stressfulness of a new situation compared to a desensitisation which should occur as the parents get used to the new child. It would be interesting to see if the difference between ages 1 and 3 would be significant with a five-graded scale as well.

### Measures: methodology and concepts

### Stress

What type of stress is measured by the SPSQ? Looking back at stress theory, we see that the instrument is of the type described by Monroe<sup>37</sup> as a *psychological appraisal approach*. It does not deal with life events, other than parenthood, an event which has naturally occurred to all ABIS participants.

SPSQ also does not measure acute, but chronic stress. It also does not give any information about the physiological state of the participant in terms of stress system activation, nor directly about psychological stress in the child (although stress in the parent may be taken as a proxy for stress in the child, as supported by attachment theory).

The information that SPSQ does provide is the parent's appraisal of the stressfulness of the parenting situation. It includes both primary appraisal (items assessing the stressfulness of the situation, e.g. "Since I had children, I have almost no time to myself") and secondary appraisal (items assessing the ability of the parent to cope with the situation, e.g. "I need help to cope with my parenting").

There is no clear-cut distinction between the two types of appraisal<sup>12</sup>, hence no question by itself can be said to assess only the one or the other. Still, many questions, such as those translated above, seem inclined towards one of the two ends of the spectrum, while some items, such as "Having become a parent, I get less help and support from my spouse/cohabiter than I expected", seem to tap into both primary appraisal (the stressfulness of disappointment in a relationship) and secondary appraisal (less resources at one's disposal to cope with the situation).

As described in the Methods section, there are five subscales in the SPSQ instrument, but only three are included in these analyses: Incompetence, Role Restriction and Spouse Relationship Problems. How do these differ? According to Östberg *et al*<sup>43</sup>, the names for the subscales derive from the "assumed underlying [mental] constructs". Incompetence then refers to the parent's assumed sense of her/his own lack of competence at parenting, Role Restriction to the parent's assumed sense of being constrained in

her/his personal interests by the parenting role and Spouse Relationship Problems refers to the parent's assumed sense of how the relationship to the other parent is working with respect to caring for children. To verify the assumptions that these underlying mental constructs are assessed by the instrument, further research would be needed. In the current thesis, however, the subscales will be treated as three different aspects of parenting stress that do reflect these assumptions.

In the ABIS questionnaires, a six-graded Likert scale was used for SPSQ, whereas in the original scale by Östberg *et al*, a five-grade scale was used<sup>43</sup>. The idea of a six-grade scale is to force a choice between slightly more or less agreement to the statement of the item, as opposed to the "neither agree nor disagree" value of 3 on a five-graded scale. The implications of this change is discussed above under the heading of Patterns  $\rightarrow$  Stress patterns.

The definition of "High" parenting stress as those above the 95<sup>th</sup> percentile may be questioned. The reason for this choice of dichotomisation is that we see very few respondents reporting stress at the very high end of the scale. At age 1, only 5 respondents had a Combined stress value of 5 or higher. Yet as can be seen from table 6, all cut-off values were above the intermediate value of 3.5, the lowest cut-offs being for Spouse relationship problems at ages 3 and 5 (3.55 and higher). Thus all parents defined with "high stress" have a stress score which is at least on the more stressed than non-stressed side of the scale. As argued by van Eck<sup>81</sup>, even minor stressful events or mood fluctuations have an impact on cortisol secretion. Thus it seems reasonable to regard stress levels even slightly on the negative side of a scale as a possible detrimental factor, at least if the stressor can be assumed to be chronic, as in the case of parenting stress.

Comparing the life events question and SPSQ for measuring psychological stress, we see that the occurrence of any life event is somewhat associated with a higher risk for high stress. However, looking at the population at large, the occurrence of a life event does not increase the mean SPSQ value by any noteworthy degree. Thus the occurrence of life events, representing acute stress, may perhaps result in increase the chronic type of stress which SPSQ represents in a small group (see the section on stress in the introduction), but in no way can life events be said to be predictive of parenting stress. This does not preclude the possibility of life events having a greater effect on other types of psychological stress.

### Sleep

There is as yet no consensual definition of sleep quality nor method for assessing it. Should we look at the physiological state of a subject, should we observe the sleeper directly and assess the amount of tossing and turning, or should we ask how he/she feels? Some experimental studies induce suppression of slow-wave or REM sleep and find detrimental effect on physical and/or mental well-being and performance<sup>47, 50, 52, 82</sup>, but the findings from these experimental conditions cannot be directly translated to the conditions of every-day sleep patterns. Total suppression of e.g. slow-wave sleep may be considered poor sleep quality, but does this occur in bed at home? If night wakings occur between sleep cycles, without interrupting REM or slow-wave sleep, does this have any effect on physical or mental well-being?

Probably, as with all investigations involving a psychological dimension, we should investigate all these different aspects of sleep if we want as clear an answer as possible. Whether this can be done depends on circumstance. The circumstances of the ABIS study – a questionnaire with thousands of respondents - do not leave room for much depth in the way of sleep assessment and certainly eliminate the possibility of physiological investigation or direct observation. The assessment of parental, subjectively rated sleep quality consists of a single question at ages 3 and 5, while child sleep quality is assessed by two questions at ages 1, 3 & 5: one simple question about sleep quality and one about night wakings. Previous studies comparing objective sleep measures to parent-report have shown a weak correlation between them when it comes to sleep quality (night wakings and time spent in sleep), but a stronger one when it comes to sleep schedules. Sadeh *et al*<sup>83</sup> reported that parents overestimated the time their child spent in actual sleep while underestimating the numbers of night wakings, giving correlations of r = 0.41 and 0.60, respectively (p < 1000.001). If we assume that these figures apply more or less to the parents in the ABIS study as well, then we cannot assume that parent-rated poor child sleep is an accurate indicator of objective poor child sleep. That is one reason why this thesis is concerned primarily with parental perceptions, not with objective sleep.

#### Night wakings

Since the number of night wakings reported corresponds well with the child sleep quality assessment (see table 6 in Results), the night wakings measure was assumed to be a very influential factor when the parents

assess the sleep quality of their child. Night wakings, regardless of cause, is a sleep pattern trait which is easily and directly observable by the parents. Considering the high correlation between the two measures, in addition to the question about parental sleep quality being directly comparable to that of child sleep quality, the decision was made to focus solely on the sleep quality question in the statistical analyses of associations between sleep quality and parenting stress in paper 2 and this thesis. However, some points deserve to be made about the assessment of night wakings.

On the basis of distribution, as described in the Results section, and for the purposes of statistical analysis, the cut-off value for many night wakings was set at four or more at age 1, three or more at age 3 and two or more at age 5. In the above-mentioned study by Sadeh, a group of 66 infants 7-26 months old, who were referred to a clinic for sleep disturbances, had a parent-reported average of 3.37 wakings per night.<sup>83</sup> The diagnostic condition for disturbed sleep set by Sadeh was 2 or more wakings per night and less than 90% of time in bed spent in actual sleep. Sadeh's subjects were comparable in age to our age 1, so our statistical lower limit for disturbed sleep is rather higher (4 wakings per night) than Sadeh's diagnostic criterion (2 wakings per night). Our data show that about 66% of the children at age 1 had 1 or fewer wakings per night and about 86% had 2 or fewer. In light of the parent-reported prevalence of sleep disturbance of 20-30%, perhaps a reasonable clinical limit is at 2 or 3 wakings per night – N.B. in conjunction with other criteria. For the purpose of statistical analysis, however, by focusing on the group reported with so many night wakings, we try to ascertain that we are indeed looking at a group with a number of reported night wakings that may be considered problematic.

### Sleep quality

When asking the respondent of a questionnaire study about his or her sleep quality, or that of the child, what will determine how the respondent answers? The number of night wakings has already been argued to be an important factor, as supported by the present data. How well rested you feel or the child appears to be should be another.

With our single quantitative question, we cannot qualify the respondent's reasons for judging sleep quality as poor or good. What we can say, however, is that a respondent who places him- or herself or his/her child at the poor or very poor end of a scale of sleep quality, does so for some

reason. Whether it is based on wakings, feelings of non-restedness or both is not really an issue in this study. It is the *perceived* sleep quality that is assessed, not any objective sleep measures. Since our stress measure is one of psychological appraisal, the perceived sleep quality should be of high importance regardless of objective sleep quality.

Certainly, it is possible to deny that you sleep poorly while the fact is that you never get a good night's rest, and a *de facto* sleep deficiency, even if denied, is likely to influence your feelings of stress at a physiological level<sup>53</sup>. Thus deciding whether a person sleeps well or poorly on the basis of a single question would be clinically irresponsible. But in statistical analysis, taken as a part of a whole situation as perceived by a single respondent, this single question about sleep quality serves its useful purpose.

### **Child temperament**

The same criticism as for the definition of high stress may be levelled at the definition of "difficult" temperament at the 90<sup>th</sup> percentile and is answered in the same way, see above. Also, the reason for using the 90<sup>th</sup> instead of the 95<sup>th</sup> percentile is that temperament was seen as a more peripheral measure, thus less strict definitions were needed. Yet the stricter definition was also tested and yielded similar results, as has already been noted.
## Summary and conclusions

To summarise, this study investigates the role of parent-perceived child sleep quality as a parenting stressor. To this end, the existing perceived sleep patterns were first described along with their relationship to certain background factors that might serve as covariates in the sleep-stress complex of associations. The same was done for stress patterns, and finally, the associations between perceived sleep quality for both child and parent to parenting stress were analysed, longitudinally and concurrently. The background factors were also included in these analyses to see if they had any effect on the complex of associations. The hypotheses were that child and parental sleep quality and parenting stress are all interassociated concurrently, and as an effect of this and of the hypothesised intrameasure stability also longitudinally.

The conclusions that may be drawn from this study concerning the main hypotheses is that they are mostly supported: poor child and parent sleep quality and parenting stress are all interassociated, but the direct connection between poor child sleep and parenting stress seems to be weaker than, perhaps to some extent subordinate to, that between poor parental sleep and parenting stress. These associations seem independent of other factors.

A clinical implication of this is that when trying to help parents with complaints about their child's sleep, it is important to try to improve the sleep of the parents. This should help in reducing parenting stress that may otherwise persist into later years, with possible negative outcomes for parent and child alike.

Further conclusions generated by this study are that parent-rated child temperament and dissatisfaction with social support may be regarded as strong parenting stressors (the opposite directions of high stress  $\rightarrow$  temperament /dissatisfaction are also likely). Single parenthood, high parental age, immigrant status and firstborn children show age- and/or subscale-specific associations to high parenting stress. Night feeding does not seem to condition children to more night wakings. Uncertainty about the cause of night wakings may be a marker for persistent child (and parent) sleep disturbances.

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measures that fall into ordinal or interval scales. Due to non-normality of distribution, The only change over time is that these measures become even more tightly centred respectively, and as a result, the number of night hours in bed is centred around 11. both mean/standard deviation and quartiles are shown. From this table, we see that bedtime and risetime are highly centred around the clock hours 20:00 and 07:00, Table A1 shows the distribution of demographic and non-dichotomised sleep around these values.

Table A1: Distributions of d	emograph	ic and no	n-dichot	omised s	sleep m	easures.			
	Age			Std			Quartiles		
Measure	group	N	Mean	Dev.	Min	$1^{st}$	Median	$3^{\rm rd}$	Max
	1	10635	12.09	2.47	8	12	12	12	18
Child's age (months)	б	8550	33.16	5.34	24	30	32	36	48
	5	6741	64.50	3.47	49	61	65	67	76
	1	10794	19:58	00:54	17	19:00	20:00	20:00	24
Bedtime	б	8700	19:46	00:45	18	19:00	20:00	20:00	24
	5	7382	19:53	00:37	18	20:00	20:00	20:00	23
	1	10788	07:16	00:52	4	07:00	07:00	08:00	11
Risetime	б	8692	07:03	00:45	4	07:00	07:00	01:00	12
	5	7379	06:58	00:39	4	07:00	07:00	07:00	10
	1	10780	11:17	00:55	S	11:00	11:00	12:00	15
Night hours in bed	б	8670	11:17	00:49	8	11:00	11:00	12:00	14
	S	7379	11:04	00:42	8	11:00	11:00	11:00	14
Mother's age (years)	0	14337	29.59	4.63	15	26	29	33	48
Father's age (years)	0	14244	32.09	5.49	16	28	31	35	99
Number of night feedings <sup>1</sup>	1	3190	1.50	0.91	1	1	1	2	9
<sup>1</sup> Excluding those who repor	ted having	g quit nig	ht feedin	gs, i.e. a	value	of zero ()	N = 7484).		

## Appendix

Table A2. Entry/Est. age

by year an	a gender	•	
	Ye	ear	
	97	98-99	1
Women	22/35	22/30	
Men	22/27	21/26	

Table A2 shows the cut-off values for the age definitions of Entry age and Establishment age, see methods section.

Table A3 shows the number and percent of the different age groups which had positive values for diverse dichotomised background variables and the night feeding dichotomisations. It also shows percent missing values within the total age 0 cohort. For ages 1, 3 and 5, a large number of the missing values were due to longitudinal dropout (except for the measure of Both parents born outside of Sweden. which was only assessed at age 0), so for these ages, the percent missing within each age group is included within parentheses.

Table A4: N and % within age group 1 reporting each cause of waking.

Cause of waking	Ν	%
Hungry	2757	24.9
Worried	4886	44.1
Woken by parent	469	4.2
Seems in pain	343	3.1
Woken by sibling	359	3.2
Noise	213	1.9

Table A3: Count and valid percent of categorical peripheral measures with positive values and % missing values. % missing *within* age groups 1, 3 & 5 are in parentheses.

Measure	Age	N	%	% Missing
	0	489	3.1	3.1
Both parents born	1	243	2.3	2.6
outside of Sweden <sup>1</sup>	3	171	2.0	2.9
	5	147	2.0	2.6
The man and ont is	1	460	5.4	$48.0(33.4)^2$
the fether	3	290	3.3	47.0 (0.9)
the father	5	651	8.8	55.3 (1.1)
	0	187	1.6	26.9
Single parents	3	435	5.0	46.9 (0.8)
	5	481	6.7	55.2 (1.0)
	0	7717	48.2	2.8
Cialo	1	5203	48.1	34.2 (2.5)
GIRIS	3	4133	48.3	48.0 (2.7)
	5	3482	48.0	55.9 (2.4)
First child	0	4712	39.2	27.0
Mothers of est. age	0	6528	45.5	12.9
Fathers of est. age	0	12904	90.6	13.5
Any night feedings	1	3190	29.9	35.2 (3.8)
Fed at each waking	1	2020	18.8	34.9 (3.3)

<sup>1</sup> This is an erratum for paper 1, in which this % was 2.4. <sup>2</sup> The high percentage is because respondent at age 1 was assessed at age 3. Within age 3, % missing is 2.8.)

Table A4 shows the number and percent within age group 1 reporting each of the options for cause of waking at age 1. Missing values is not applicable for this measure. Table A5 shows the crosstable for older fathers vs high age 3 Role restriction stress, when the father is the respondent.

		Father's a 3 <sup>rd</sup> qu	age above Jartile	
		Yes	No	Total
Polo restriction at ago 3	Yes	10	1	11
Note restriction at age 5	No	86	143	229
Total		96	144	240

Table A5: Crosstable of Role restriction vs older fathers within age group 3; the father is the respondent.

Figure A1 shows the valid percentage distribution of maternal and paternal education at the birth of the child. Percent missing are 3.1 (mothers) and 4.6 (fathers). The categories in figure 1 may be categorised further according to level of theoretical education, see Methods. The percentages of these categories are: Higher theoretical education – mothers 31.7%, fathers 24.6%; Some theoretical education – mothers 32.7%, fathers 23.0%; No theoretical education – mothers 35.5%, fathers 52.4%.

