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Chapter 1

General introduction
Fear is functional. However, not all fear is realistic nor is it functional in every situation. When railroads began to be used people were afraid of their speed (Whittall 1882), but few people today can imagine that the speed of travel is in itself dangerous. Even those afraid of flying do not use speed as an argument. Travel is generally safe, and journeys undertaken at a slow tempo are not safer than those at high speed. Walking and cycling in traffic are not safer than driving a car, depending on how you make comparisons. But certainly for longer distances, if one compares the safety statistics per distance travelled, speed is more likely to be a safety-enhancing factor than a risk. From the standpoint of survival, if you have a long distance to travel it is better to use an automobile than a bicycle, and even better an airplane, if you want to arrive in one piece.

Safety is a key element of an airline service; the perception of insecurity might influence flight behaviour in general. “An accident to an airline is like a product defect to a manufacturer, which results in lower product demand and sales revenue” (Wong and Yeh 2003 p. 471). Besides local effects for the airline involved, accidents might in general temporarily influence passenger numbers due to a sense of insecurity regarding air travel. Wong and Yeh (2003) reported an average of 5.6% decline in passenger flow in the 2.5 months following serious accidents with rival airlines in the same Taiwanese market. Gigerenzer (2004) analysed data from the U.S. Department of Transportation and reported a 16% decline in passenger miles flown in the three months after the 911 twin tower tragedy. At the same time road traffic miles driven on interstate highways increased by 5.3%, resulting in an extra 317 road crashes with 353 fatalities. Regrettably, the act of terrorism cost 266 passengers and crewmembers (as well as many more people at the scenes of the attacks) their lives. However, the number of lives lost due to avoiding the perceived risk of flying shortly after the 911 attacks was higher than the total number of passengers and crew killed in the four fatal flights. Unfortunately, logical arguments and statistics about the safety of air travel do not assuage the fears of those suffering from aviophobia.

Assessment of anxiety disorders is based mainly on a patient’s own verbal report, sometimes supported by written questionnaires. The diagnostic reliability of these assessments is a profound problem (Lang 2014). Moreover, even though physiological activation and physiological symptoms are central to these disorders, physiological measures are not used in their diagnosis (Roth 2005). Fear in general is often described in terms of physical discomfort as sweating, heart racing and muscle tension. As many as
eight out of ten symptoms experienced by individuals with aviophobia during flight are related to bodily sensations (Thyer and Himle 1987; Roth 2005). Self-report of physiological discomfort is used prominently to diagnose fear of flying, and is also often included as one of the main measures of treatment effectiveness (Van Gerwen et al. 1999). Reliance on subjective report alone may neglect important supplementary information available through psychophysiological measures, because within the domain of anxiety disorders there is a substantial discordance between subjective and physiological reactivity (Lang and McTeague 2009).

Imagine a doctor not performing a physical examination when you come to an appointment with stomach pain. You describe how you feel and the doctor asks some questions according to a standardized protocol. Does s/he have all the available information? Or would listening to the heart and lungs lead to another diagnosis? Sometimes heart failure, for example, manifests itself as a stomach problem. For an optimal diagnosis one must use all available information. Why, then, does this usually not happen in the case of anxiety disorders? Why are diagnoses nearly always based on self-report?

This thesis examines, from various angles, individual differences within a relatively large group of people with serious fear of flying (FOF) who applied for treatment to overcome this phobia. Psychophysiology is one of these angles. An important goal of this thesis is to explore added value of psychophysiological measures for the diagnosis and treatment of fear of flying. Physiological anxiety responses may augment diagnosis for this disorder, and also make it possible to track improvement during therapy. A further expectation is that measurement of physiological reactivity will lead to better prediction of treatment outcomes.

Outline of this introduction
The introduction describes a number of relevant background topics. It starts with the question of why we should trouble ourselves, and more importantly why we should bother patients, with measuring physiological variables. Then follows a review of the pros and cons of several psychophysiological measures. Next, this chapter provides general information on fear of flying and its prevalence, etiology and treatment, and concludes with an overview of the empirical studies that form the backbone of this dissertation (chapters 2 to 7).
PSYCHOPHYSIOLOGY

Lang’s tripartite model states that the emotion of fear is expressed in three loosely coupled domains: affective language, overt behaviour, and physiological reactivity of the autonomic nervous system (ANS) (Lang and McTeague 2009; Lang 2014). In this dissertation, affective language is defined as self-reported data gained by use of questionnaires and structured interviews. Behaviour is not treated as an independent measure, but as a dependent measure in the empirical studies in this dissertation.

Of the three domains of the tripartite model the physiological is the least used. Analysis of psychophysiological variables is regarded as difficult for the researcher and bothersome for the patient. If indeed these variables are studied, it is mostly done in a well-controlled environment, a laboratory. And no matter how neat it is to control all disturbing variables, a lab is very different from real life. Moreover, in psychophysiological research it often happens that the participants are first year students. This makes it very difficult to generalize the results to clinical samples.

Ecologically valid research requires ambulatory measurements in naturalistic settings with real-life stressors and actual patients. Patients are people who apply for therapy and might be willing to tolerate some concurrent scientific research, but only if it does not interfere with therapeutic goals and does not become a nuisance. The participants in the research for this dissertation were highly anxious aviophobics who came for therapy. Needless to say, we did our utmost not to add procedures that would increase their anxiety. The feasibility of ANS research with these patients severely restricted our choice of physiological parameters. Measurements needed to be as non-invasive as possible, and measures needed to respond to changes in psychological state over a time scale of a few minutes. The next section, therefore, focuses on ANS functionality and the pros and cons of several measures that capture ANS activity in a naturalistic environment.

The main function of the ANS is to ensure homeostasis. It does so by responding adaptively to changes in the internal and external environment. Furthermore, the ANS can anticipatory prepare for threats to homeostasis, even in the absence of actual activity. An ANS flight-fight or freeze response can also be triggered by a phobic stressor, i.e. a subjective experience of stress without an actual objective danger present. ANS executes control by modulated activation of the sympathetic- and parasympathetic-nervous systems. Historically, a simple bipolar reciprocal model of autonomic control has been assumed, in which increased activity of one branch is associated with decreased
activity of the other. This one-dimensional model has been refined into a bivariate model of autonomic space with sympathetic activity along one axis and parasympathetic activity along the other axis, providing a multitude of combinations of sympathetic and parasympathetic activity (Berntson et al. 1994). Responses to psychological stress are highly individual, with some people showing an increase in sympathetic activity, others a parasympathetic withdrawal, and still others a combination of these responses, or even a coactivation in both branches (Quigley and Barrett 2014). On the other hand, within-individual response patterns to psychological stress seem relatively stable (Berntson et al. 1994). Non-invasive assessment of sympathetic activity and parasympathetic activity is possible by looking at the innervated organs. Heart rate variability (HRV) is the only reliable non-invasive measure currently available to capture parasympathetic reactivity. Several non-invasive measures are available to capture sympathetic reactivity.

Generally, careful consideration is needed when selecting physiological measures to capture ANS activity. Not all physiological measures capture fear intensity variations along the whole continuum. For example, Aue et al. (2012) exposed 18 spider phobic participants and 18 nonphobic participants (all female) randomly to pictures of animals (spider, snake, bird) and recorded subjective fear estimates together with several physiological measures. Skin conductance mirrored only extreme levels of fear, respiratory measures distinguished phobic from nonphobic fear but did not differentiate fear levels within phobic participants; only HR captured fear intensity variations from extremely low levels of fear to considerable phobic fear. HR seems to be a sensitive measure that captures fear intensity at both extremes of the fear continuum (Kreibig 2010; Wilhelm and Grossman 2010; Aue et al. 2012).

The heart is dually innervated. HR is the resultant of sympathetic and parasympathetic control on the intrinsic rate of the cardiac pacemaker. HR itself does not reveal the sympathetic and parasympathetic ANS cardiac activity. Different patterns of reciprocal activation, co-activation and co-inhibition in sympathetic and parasympathetic control on the heart have been found. During rest HR is tonically inhibited by parasympathetic (vagal) control, slowing the intrinsic cardiac pacemaker rate (100-150) to the normal resting heart rate of a healthy adult (60-80). Heart rate increases during inspiration and decreases during expiration, promoting an efficient exchange of oxygen in the lungs. This phasic inhibition and excitation of the heart is brought about primarily by rapid vagal control coupled to the respiratory cycle. Heart Rate Variability (HRV) in the time domain can easily be obtained from the electrocardiogram (ECG) by taking the root mean square of differences (RMSSD) in the interbeat interval between consecutive R-waves. HRV in
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the frequency domain is indexed by HF (0.15 - 0.4 Hz) power obtained via spectral power analysis by fast Fourier transformation (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996; Friedman et al. 2002).

A “pure” respiratory sinus arrhythmia (RSA) can be derived when combining ECG data with respiratory signals by subtracting the shortest interbeat interval (IBI) during HR acceleration in the inspirational phase from the longest IBI during deceleration in the expirational phase (i.e., the peak-through method) (Grossman et al. 1990). RMSSD, HF power and RSA are highly correlated (Friedman et al. 2002). RSA is theoretically considered to be the soundest measure of cardiac vagal control with substantial evidence supporting its validity (Goedhart et al. 2007). Greater RSA reflects greater parasympathetic cardiac control. Higher vagal tone and greater vagal withdrawal during challenge have been associated with a greater ability to engage and disengage with environmental demands, and reduced HRV and vagal tone have been associated with anxiety and less adaptive emotion regulation (Friedman 2007; Mathewson et al. 2013; Chalmers et al. 2014).

The pre-ejection period (PEP) is a reliable and valid non-invasive measure to index changes in sympathetic cardiac control under naturalistic and ambulatory conditions (Goedhart et al. 2006). PEP is defined as the onset of the electromechanical systole (the onset of the Q wave on an electrocardiogram (ECG)) to the opening of the aortic valve (represented by the B point on an impedance cardiogram (ICG)). Shortening of the PEP reflects greater sympathetic control. Alternatives to PEP currently in use are salivary alpha-amylase (sAA), LF/HF power ratio in the frequency domain and galvanic skin response. Salivary collection is a non-intrusive measure that can be performed relatively easily in a naturalistic setting, although the procedure disrupts whatever is going on. The time lag between increased sympathetic activity and SAA secretion is unclear and may be at least 10 minutes. Salivary alpha-amylase secretion is under concurrent sympathetic and parasympathetic control and therefore cannot be used to index either input selectively (Bosch et al. 2011; Nagy et al. 2015).

The LF/HF ratio is still up for debate and seems to correlate poorly with other measures of sympathetic activity (Lien et al. 2015). A third alternative to PEP sometimes used to index sympathetic activity are electrodermal measures like skin conductance responses (or galvanic skin responses). However, electrodes are often placed on the palm of the hand, thereby restricting participants severely in their normal routines. Moreover, movement of the hand affects signal quality, thus hampering interpretation. The PEP is currently
the measure of choice in psychophysiological stress research in real life settings (Lien et al. 2015). A disadvantage of this measure is the laborious visual scoring of the entire recording. Scoring of the PEP requires identification of the onset of the Q wave, which can be ambiguous. Finally, PEP is known to be sensitive to preload and afterload effects that occur mainly during head-up tilting and changes of posture from supine to sitting to standing (Houtveen et al. 2005).

**AVIOPHOBIA**

Fear is a normal response to a genuine danger, anxiety a normal response to anticipated events. Fear and anxiety are not different aspects, but differ in the dimension of threat imminence (Barlow 1991). Fear and anxiety become problematic when excessive and disproportional to the actual or envisaged danger or when temporary anxiety turns into chronic anxiety. A phobia is a type of anxiety disorder, defined by persistent fear of an object or situation (American Psychiatric Association 2013). Aviophobia (fear of flying, FOF) is a persistent marked fear during or in anticipation of flying. It is disproportional to the actual danger, and this anxiety, fear or avoidance causes clinically significant distress or impairment.

Fear of flying is a heterogeneous phenomenon and often includes, or is a combination of, acrophobia, claustrophobia, fear of relinquishing control, fear of losing control over oneself, fear of a crash, and panic and social phobias (Van Gerwen et al. 1997; Oakes and Bor 2010a). Although classified as a specific phobia, also known as simple phobia, fear of flying is far from simple in view of its heterogeneous and compound nature. In general, aviophobics with agoraphobia are more concerned about panic and its consequences, whereas aviophobics without agoraphobia generally report more concern about external aspects of flying, like crashing (McNally and Louro 1992).

In the Netherlands the lifetime prevalence of anxiety disorders in the period 2007-2009 was reported to be 19.6%, and the 12-month prevalence 10.1 %, based on DSM-IV criteria. For specific phobias a lifetime prevalence of 7.9% was reported, and a 12-month prevalence of 5.0%. Between 1996 and 2007-2009 the 12-month prevalence figures did not change (de Graaf et al. 2012). The lifetime prevalence rate for a specific phobia is about half as high in men as in women (Grant and Odlaug 2015; Adolph et al. 2016), although men might underreport a specific phobia. Alcohol abuse might be a masculine masking strategy (Bekker and van Mens-Verhulst 2007). Fredrikson et al. (1996) reported
increased aviophobia as a function of age in women but not in men. It is estimated that more than a third of all people find flying difficult and distressing. Fear of flying is a debilitating disorder affecting 10% - 15% of the general population in the western world (Oakes and Bor 2010a; Ekeberg et al. 2014). Nearly all of these people either avoid flying, or fly with the help of medication, drugs or alcohol. Aviophobic people experience serious interference in daily life and social functioning due to their fear of flying.

Fear of flying is mostly an individual problem that can cause social or professional impairment (Van Gerwen et al. 1997). Besides, it reduces air travel and can have negative financial repercussions for the airline industry. A fearful passenger’s failure to embark can negatively influence on-time performance, as safety regulations stipulate that luggage is not allowed to travel without its owner on board. A passenger wishing to disembark after doors have closed, or even after pushback, causes even more delay. Psychiatric issues during flight constitute 3.5% of in-flight medical incidents, most of which are caused by acute anxiety (Naouri et al. 2016).

Some studies report that people with fear of flying might be inclined to pay more for flight attributes that alleviate fear, like non-stop flights, scheduled carriers and home carriers (Fleischer et al. 2012), or pay more for flights and airlines that are perceived as safer (Fleischer et al. 2015; Koo et al. 2015). Participants in these studies did not actually book a flight or pay for their choice. To my knowledge no research on actual booking behaviour with risk/price trade-offs has been published. Empirical research on actual risk/price trade-off behaviour is needed (Savage 2012).

ETIOLOGY OF AVIOPHOBIA

Several pathways to the onset of phobia are known. The first is direct learning through classical conditioning of aversive events. Twelve (71%) out of 17 aviophobic participants without agoraphobia reported a threatening event during a flight (e.g. severe turbulence) as an etiological factor (McNally and Louro 1992). None of the 17 aviophobic participants with agoraphobia in the study cited exposure to threatening events as the cause of their phobia. Wilhelm and Roth (1997) reported that 17 (46%) of 37 aviophobics without a comorbid panic disorder had unpleasant (24%) and even life-threatening (22%) flight experiences. In this study, for the 21 control participants without aviophobia the numbers were 5% respectively 9%. Schindler et al. (2016) reports that 15 (50%) of 30 aviophobic participants and 16 (53%) of 30 healthy controls reported frightening events
in the air. These numbers are in sharp contrast with percentages found in an amazingly large sample of 2001 self-referred highly phobic adults who applied for a fear of flying treatment program (Nousi et al. 2008). Of all these people 85.6% flew before applying for treatment and reported uneventful flights, 5.7% flew before treatment and reported an event (5.4%) or even traumatic flight (0.3%), while 8.7% had no previous experience with flying. These numbers substantiate that direct conditioning cannot be the only mechanism in the development of fear of flying. In any case, it is unlikely that all people with a fear of flying have had a serious incident or accident; the number of these negative events is simply too low to account for the high prevalence of aviophobia. Moreover, a significant proportion of the healthy controls mentioned previously reported frightening events during flight without developing aviophobia.

The discrepancy in reported frightening events might partly be due to nomenclature. Although people with fear of flying may not have experienced flight-related incidents or accidents, they still may have thought that they were in a threatening situation. Pilots classify a discontinued approach (a “go-around”), a non-event that breaks the routine. However, unexpected roaring of the engines, pitch changes and associated acceleration, and vestibular effects might scare a passenger. A passenger can interpret a non-event as (extremely) dangerous and life-threatening and thus experience it as traumatic. A person’s appraisal of a situation is the key to the resulting emotion (Frijda 1986; Lazarus 1993). Emotionally intense events tend to be remembered very well (McNally 2016). A problem in research on the conditioning of FOF is that people who develop FOF will probably remember a frightening instance associated with flight, whereas controls may have no recall of negative flight experiences; for them the experiences did not linger or result in a lasting aversion to flight. Retrospective recall might even favour remembrance of events that never occurred to explain current emotional distress (McNally 2016). Finally, individual differences in associative learning may contribute to the development of aviophobia, and explain why some individuals develop fear of flying after an aversive event associated with flying, and others do not. For example, Vriends et al. (2012) reported a slightly stronger conditioning effect to phobia-unspecific frightening pictures and frightening words among 33 aviophobic participants compared to 39 non-aviophobic control participants.

A second pathway to developing aviophobia is indirect learning through observation (vicarious learning, [Rachman 1977]). Published reports include a limited number of experimental studies supporting observational fear learning in toddlers and young children (see LoBue and Rakison 2013 page 286 for details), and one study with adults with
fear of flying (Schindler et al. 2016). Here 11 (37%) of 30 aviophobic participants reported that a family member or other important person suffered from past or present fear of flying. However, only two patients (6%) reported that watching somebody in their family undergoing a strong fear reaction during flight triggered their own phobia. Also familial resemblance of fear of flying may be partly attributable to a shared genetic sensitivity to anxiety disorders, the heritability of which is known to be substantial (Hettema et al. 2001).

The third pathway through which a fear of flying can be acquired is transmission of information and instruction (Rachman 1977). People with anxiety disorders have an attentional bias for threat cues (LoBue and Rakison 2013; Sarapas et al. 2017). These threat cues are omnipresent in the media. Aviophobic participants without agoraphobia reported significantly more often (71%) than aviophobic participants with agoraphobia (18%) that verbal and media information figured in the development of their fear of flying (McNally and Louro 1992). Schindler et al. (2016) found that a significantly higher percentage of patients with fear of flying (70%), compared to non-aviophobic controls (37%), felt influenced by media information about crashes and other flight incidents. Only three of these patients (9%) reported informational learning as triggers of their phobia; the majority of patients reported the influence of the media information after the beginning of their phobia. Informational learning might be an important factor in the maintenance of fear of flying.

A fourth pathway to aviophobia might be non-associative learning. This pathway to the onset of phobia is an extension of the previous three paths, with the addition that some fears are non-associative and are based on innate fear cues specific to evolutionarily threats which do not require prior association with aversive stimuli (Gray and McNaughton 2003). Fear and avoidance of certain stimuli have in the past proven beneficial to the survival of the species (Seligman 1971). A false negative fear reaction might result in death, while false positive fear reactions, in isolation, have few consequences: better safe than sorry. However, when too many false positives cumulatively begin to impair daily functioning there is a problem (Adolphs 2013). Phobias can be seen as instances of highly “prepared” learning, and the dangers natural to a species (spiders, snakes, heights) lead easily to phobia (Seligman 1971).

The four pathways do not necessarily work in isolation, although people differ in sensitivity to different pathways to develop aviophobia (Schindler et al. 2016). Individual risk factors seem to play a crucial role in the onset, development and maintenance of
anxiety. These risk factors include personality characteristics like neuroticism, a genetic vulnerability factor related to many anxiety disorders (Middeldorp et al. 2005; Smoller et al. 2008). An enhanced genetic liability can contribute to the development of anxiety disorders and specific phobias. Phobias in general are moderately heritable with an estimated heritability ranging from 20% to 40% (Hettema et al. 2001). Twin studies on the heritability of specific phobia are rare; a review by Houtem et al. (2013) included only 10 studies and found a mean heritability for specific phobias of 25%, range 0-33.

Stressful life events might enhance conditionability and increase vulnerability for developing aviophobia (Wilhelm and Roth 1997). Schindler et al. (2016), who reported that about 50% of aviophobics and controls indicated frightening events in the air, reported a significant difference between aviophobics (60%) and controls (19%) in reported stressful life events at the time of their frightening flight experience. Memory recall of distressing events appears to play a significant role in the acquisition and maintenance of aviophobia. Unexpected or inexplicable events during flight are quite common and many passengers have distressing moments during flight. Most people forget these unsettling experiences and do not develop a conditioned fear of flying in reaction to these events.

Fear is embedded in a network of causal relationships with cognitive processes. Motivation, attention and memory are part of an adaptive response to a threatening stimulus. Rumination about fear and increased expectation can be associated with increased vigilance and attention to potentially dangerous stimuli (Adolphs 2013). People generally overestimate the consequences of fearful stimuli (Seligman 1971), and people with anxiety disorders have an additional bias to rate stimuli in general as more frightening or negative (LoBue and Rakison 2013). Threat cues seem more salient to anxious individuals (Sarapas et al. 2017), and threat cues are abundant during flight. Before and during flight passengers are constantly reminded of safety and safety aids (e.g. the safety briefing before flight pointing out emergency exits and life vest locations, seat belt signs illuminated during turbulence, dimming of lights during start and landing, et cetera). The mere availability of a physical safety aid may paradoxically elicit, rather than mitigate anxiety, even more so in individuals hyper vigilant for phobic cues (Blakey and Deacon 2015).
TREATMENT OF AVIOPHOBIA

Although fear of flying is highly prevalent, only a small percentage of people with this phobia face their fear and apply for therapy. The preferred treatment method is cognitive-behavioural therapy combined with in-vivo exposure (Van Gerwen et al. 2004; Oakes and Bor 2010b; Pearl and Norton 2016). The complex and heterogeneous nature of fear of flying calls for interventions known to be effective for similar anxiety disorders. Therapy often tackles the multitude of underlying phenomena with a combination of providing information, cognitive restructuring, relaxation training and graded exposure, both in-vitro and in-vivo (Van Gerwen et al. 2004; Oakes and Bor 2010b). Imaginal exposure, virtual reality and computer-assisted exposure can be part of in-vitro exposure therapy and have comparable efficacy for fear of flying (Rus-Calafell et al. 2013). Although non-exposure treatments for specific phobia do outperform no treatment, the magnitude of their effect is only slightly greater than that of placebo versus no treatment (Wolitzky-Taylor et al. 2008). Exposure seems to be a necessary component in the treatment of aviophobia. In-vivo exposure for specific phobia yields better short-term results than imaginal exposure and virtual reality exposure (Wolitzky-Taylor et al. 2008). However, stand-alone in-vivo exposure therapy is associated with high dropout rates and low treatment acceptance (Choy et al. 2007). Cognitive interventions before exposure enable patients to take part in flight-exposure; this would not otherwise be possible (Oakes and Bor 2010b).

Most treatment facilities and programs are run in close co-operation with a domestic airline and end the treatment with a “graduation” flight. Treatment is often group-based, consisting mostly of 5 to 10 participants, and conducted over one or two days. A few facilities provide individual treatment and a number of shorter sessions if desired or required (Van Gerwen et al. 2004). Efficacy of treatment is defined as a reduction in self-reported anxiety from pre- to post-treatment, or partaking the graduation flight or a post-treatment flight. Acclaimed success rates of therapy range from 67 to 96% (Van Gerwen and Diekstra 2000; Van Gerwen et al. 2004; Oakes and Bor 2010b).

The multicomponent group-treatment for fear of flying may include therapeutic ingredients that are unnecessary for individual participants, but that favour transdiagnostic treatment gains (Deacon 2013). Comorbidity across anxiety disorders is high (Pearl and Norton 2016). Many anxiety disorders have much in common, like attentional biases, inaccurate threat beliefs, the use of safety behaviours, escape and avoidance. The multi-component transdiagnostic fear of flying treatment packages will
tackle most of these components, and might greatly improve quality of life as well as alleviate fear of flying.

CONTEXT OF THE EMPIRICAL STUDIES IN THIS DISSERTATION

Aviophobic participants in the empirical studies were individuals who applied for treatment to overcome their fear of flying. Most participants were self-referrals. Health care agencies, health care professionals and company health programs referred a minority of participants. Inclusion criteria for all studies were a good understanding of the Dutch language and no flight scheduled within 5 weeks of start of treatment. Airline personnel were excluded from the studies. Other reasons for exclusion were current use of cardioactive medication like β blockers, and pharmacotherapeutic medication. Participants followed a highly standardized treatment program for fear of flying at the VALK foundation in The Netherlands. This institution is a joint enterprise of the Section of Clinical Psychology of Leiden University with Amsterdam’s Schiphol Airport and several Dutch airlines. It specializes in fear-related problems, especially fear of flying. The fear of flying treatment program starts with a thorough diagnostic assessment, including pre-treatment phobia and flight-anxiety measurements, followed by a maximum of four individual 1-hour therapeutic sessions, covering general information on factors relevant to fear and anxiety, relaxation and breathing techniques, and coping skills. Claustrophobia, acrophobia, traumatic transportation accidents and traumatic social events were addressed where applicable. Participants started a two-day cognitive-behaviour group treatment (CBGT), lasting 20 hours in total, five weeks after diagnostic assessment. CBGT groups consisted of a minimum of five to a maximum of eight participants, a therapist and an airline pilot. The first day of group treatment focused on psycho-education and technical information on flying. The second day focused on exposure and included two flights in a full motion cabin flight simulator normally used for flight safety training for cabin crew. The day ended with in vivo exposure during guided return flights of at least one hour each on a commercial airliner. Three months after CBGT participants were invited to attend an optional single three-hour follow-up session. Details of the therapeutic protocol have been published elsewhere (Van Gerwen et al. 2002, 2006). Written informed consent was obtained from all participants previous to the diagnostic process. The local medical ethics committee approved the research protocol for the studies in this thesis.
All empirical studies in this thesis were part of a longitudinal study on fear of flying, and five out of six studies utilized data from the same sample of aviophobic participants. The study described in chapter 3 utilized a different sample of aviophobic and nonphobic participants. The number of participants in the separate studies depended on the research question. Some studies required more stringent criteria than others. For example, inclusion criteria for the study mentioned in chapter 4 were complete data on all essential questionnaires at the start and at the end of treatment, in combination with complete data of all physiological variables (HR, RSA, PEP) during both flights. The security checks at the airports were a major challenge for the physiological measurements. The electrodes of the ambulatory measurement device required physical patting down of all participants. After security screening not all recording devices recorded all variables properly, a factor that excluded participants with missing data from the chapter 4 study. Nevertheless, all these participants were included in the study mentioned in chapters 6 and 7, because research questions of these studies allowed analyzing strategies that deal with partially missing data without having to exclude participants.

EMPIRICAL STUDIES IN THIS DISSERTATION

At the end of the 19th century James (1884) and Lange (1885) developed the theory that stimuli from the environment lead to physical reactions like rapid heartbeat, muscle tension, etc. Emotions are the result of sensing these reactions. With phobic people, exaggerated subjective arousal may arise from exaggerated physiological arousal during exposure to anxiety-related stimuli. On the other hand, the primary deficit in phobics may not be exaggerated physiological arousal, but a tendency to focus attention on bodily sensations and/or overinterpret these signals as danger signals. Phobic individuals may be prone to interpret normal bodily sensations in a threatening manner in this more psychological approach. Chapter 2 examines the relationship between subjective and physiological arousal in individuals with fear of flying and controls without aviophobia when confronted with flight-related stimuli. The physiological perspective predicts a higher reactivity in phobics than non-phobics to flight-related stimuli and, within the group of phobics, a significant correlation between physiological reactivity and the amount of self-reported fear. The psychological perspective predicts a weaker concordance between subjective and physiological arousal, which may be limited to individuals who score high on anxiety sensitivity.
Chapter 3 thoroughly investigates an aspect of a relatively new treatment for aviophobia. Virtual Reality (VR) techniques are emerging for the treatment of many disorders, including phobias. An important element of VR therapy for phobic disorders is that the exposure is done gradually to more anxiety-arousing situations, with therapists continuously monitoring the anxiety level of a patient. This can be done using Subjective ratings of Anxiety (SUD), behavioral observations or physiological measures. The latter have the advantage of being more objective. However, physiological measures require a baseline measurement because of individual variation. One often-used procedure is to obtain a physiological baseline recording when the patient is placed in a neutral VR world, i.e. a VR world that should not include phobia-related stressors. Nevertheless, even without a phobia-related stressor, it is not clear whether the experience of being placed in a Virtual Environment (VE) in itself causes some level of anxiety. Chapter 3 explores whether it is possible to create a truly neutral world that causes no anxiety. This ‘real’ neutral world could then be used as baseline for further VR research.

Chapter 4: Anxiety sensitivity is the tendency to fear anxiety-related bodily sensations, based on the belief that the sensations have harmful consequences (Reiss 1991). Individuals with high anxiety sensitivity are prone to interpret normal bodily sensations as threatening, whereas those with low anxiety sensitivity experience these sensations as unpleasant but non-threatening. Anxiety sensitivity has been identified as a vulnerability factor for flight phobia. In this study we examined whether AS moderates the effects of somatic sensations and autonomic nervous system reactivity on flight anxiety induced by real flight. If so, this might have implications for interventions during CBT.

Some cognitive coping strategies seem more adaptive than others, while the use of certain maladaptive cognitive coping strategies has been linked to psychopathology. People with aviophobia who seek treatment are found to have a dispositional tendency to use maladaptive strategies, including avoidance behaviour, to cope with their anxiety. Almost no studies of anxiety problems have been performed to find whether psychological interventions may change the use of specific coping strategies, and if so, whether these changes predict subsequent reductions in anxiety. Chapter 5 describes research with aviophobic participants who were followed from pre-treatment to long-term follow-up, 41 months later. We expected that participants, who at the start of treatment, more often used maladaptive strategies than adaptive strategies would profit less from treatment. Furthermore, we expected that targeted interventions during CBT would reduce the predominant use of maladaptive strategies and enhance the use of more adaptive coping strategies. Finally, we hypothesized that in particular a reduction
of the maladaptive coping style during treatment would predict a better long-term therapy outcome.

Next to cognitive interventions, exposure is the second basis of cognitive behavior therapy. According to emotional processing theory, exposure to a feared stimulus activates a ‘fear network’, and activation of this fear network is seen as a necessary condition for improvement of phobias and other anxiety disorders. Successful emotional processing during exposure is subsequently indicated by within-session and between-session habituation of fear responses. Chapter 6 deals with the question of whether fear activation and habituation during exposure are predictive of short-term and long-term therapy results. A second goal was to see whether the prediction of therapy outcome would be improved by adding measurements of physiological reactivity to self-reporting of anxiety.

Chapter 7: The tripartite model of Lang (2014) states that the emotion of fear is expressed in three loosely coupled domains: affective language, overt behaviour, and physiological reactivity. Emotion can be seen as the organizing process that coordinates these different systems to prepare the individual for optimal and effective response to challenges. When the individual is faced with increasing demands the coordinated co-activation of multiple response systems would seem even more important. Response coherence would thus be expected to increase with increasing emotional intensity. Conversely, adaptive emotional functioning could be indicated by proper and progressive response coherence upon increasing demands. If so, the magnitude of coherence could be an indication of progress during therapy. In this study we first examined whether synchronous change in subjective and physiological reactivity over repeated exposures increased from watching a flight video (low intensity) through simulated flight (medium intensity) to actual flight (high intensity). Second, we assessed whether the magnitude of synchronous change predicted short- and long-term treatment outcome. Based on the assumption that successful treatment of anxiety disorders should be indicated by synchronous change, we expected participants with more synchronous change in the two response systems during treatment to show lower flight anxiety at the end of treatment and three years after treatment than participants with less synchronous change during treatment; we also expected them to have engaged in more actual flights.

Finally, chapter 8 summarizes and discusses the main findings and their clinical implications, and presents directions for further research.
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