Subjective and physiological reactivity to flight in people with fear of flying

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Anxiety Sensitivity moderates the relationship of changes in physiological arousal with flight anxiety during in vivo exposure therapy

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ABSTRACT

Physiological sensations and discomfort constitute the major symptoms reported by aviophobics. Anxiety sensitivity (AS) seems to moderate the relationship between self-reported somatic sensations and flight anxiety, and AS 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.

In fifty aviophobics participating in Cognitive Behaviour Group Therapy (CBGT), flight anxiety, somatic sensations and autonomic nervous system reactivity were assessed during a guided return flight. Results indicate that physiological reactivity interacted with AS. Changes in heart rate and parasympathetic activity were more strongly associated with changes in reported flight anxiety for high AS participants, and less for participants low on AS. Results did not indicate a moderating effect of AS on the relationship between self-reported somatic sensations and flight anxiety.

Our results suggest that therapy for flight phobia might benefit from addressing the physical effect of anxiety, by means of cognitive restructuring and exposure to interoceptive stimuli, particularly in aviophobics high in AS.

INTRODUCTION

Taking a flight is common practice for many people in the western world, but not for all. Up to 40% of the general population in industrialized countries experience mild fear before or during flight (Curtis, 1998; Depla, Ten Have, van Balkom, & de Graaf, 2008; Van Gerwen, Diekstra, Arondeus, & Wolfger, 2004). Some 7% of all people experience serious interference in daily life and social functioning due to fear of flying (FOF). Most symptoms reported by aviophobics are related to bodily experiences (Roth, 2005; Van Gerwen, Spinhoven, Van Dyck, & Diekstra, 1999).

Fear of flying is a heterogeneous problem and can be conceptualized both as a situational phobia as well as the expression of other non-situational phobias with or without agoraphobia. Flying phobics can fear accidents, have complaints of acrophobia and claustrophobia, report panic attacks in anticipation of flights, want to be in control over the situation or are afraid to loose control over themselves. Social anxiety can be part of FOF as well (Van Gerwen, Spinhoven, Diekstra, & Van Dyck, 1997). In general, aviophobics with agoraphobia are more concerned about panic and its consequences, whereas aviophobics without agoraphobia in general report more concern about external aspects of flying like crashing (McNally & Louro, 1992).

Fear in general is often described by physical discomfort as sweating, heart racing and muscle tension. During flight one is exposed to sudden loud and strange noises, unexpected movements during turbulence, vibration, acceleration and pressure changes. All of these can lead to physical discomfort as well. While some people just notice these bodily responses, others might misinterpret these signals as danger signals. Anxiety sensitivity (AS) can be viewed as a key moderator between the experience of these bodily responses and anxiety. Anxiety sensitivity is the tendency to fear anxiety-related bodily sensations, based on the belief that the sensations have harmful consequences (Reiss, 1991). Although AS is most strongly related to panic, generalized anxiety disorder and posttraumatic stress disorder, specific phobia is also significantly associated with elevated AS (Naragon-Gainey, 2010). The meta-analysis of Naragon-Gainey indicated a correlation with a medium effect size between AS and blood/injection/injury and animal phobias, while the correlation between physical confinement (claustrophobia) and the fear of bodily harm with AS was large. Especially the physical subcomponent of the latter two showed a rather large correlation with AS.

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Several studies link FOF with elevated levels of AS. Rivas and Tortella-Feliu (2000) assessed 523 non-clinical participants and found that participants with FOF had an elevated AS score, while a higher intensity of FOF was associated with a higher AS. Vanden Bogaerde and De Raedt (2008) performed a moderator analyses on questionnaire data of 160 students and concluded that AS moderates the relationship between somatic sensations and flight anxiety. Somatic sensations predicted flight anxiety in individuals with high AS, while this was not the case for students with low AS. The same authors corroborated these findings in a second more ecological valid study. Anxiety and somatic symptoms of 54 aviophobics and 49 controls without FOF were measured just before take-off on a regular line flight (Vanden Bogaerde & De Raedt, 2011). Results again showed the same moderating effect of AS on the relationship of somatic symptoms with flight anxiety. Furthermore, flight phobics had in general higher levels of AS than the control participants. While the 2008 study used a non-clinical student sample not controlled for a concurrent panic disorder, the 2011 study found similar results with a clinical sample of flight phobics without a concurrent panic disorder or anxiety disorder that was primary to the fear of flying.

Interestingly the moderating effect of AS on the relationship of bodily sensations with flight anxiety has only been studied by means of questionnaires and verbal report. Although the focus of AS lies on the experience of bodily sensations, up till now only one experimental study combined AS, FOF and actual physiological measurements (Busscher, van Gerwen, Spinhoven, & de Geus, 2010). Here measurements of AS and self-reported anxiety of 127 aviophobics were combined with measures of autonomic nervous system reactions to a neutral video and a anxiety provoking flight video. Although changes in Heart Rate (HR) and Respiratory Sinus Arrhythmia (RSA, a measure of parasympathetic activity) were correlated with changes in self-reported anxiety, AS did not moderate this association. Flight phobics who are afraid of anxiety-related bodily sensations did not report more distress than phobics who score low on this trait, even when they show stronger physiological responses. This is contra intuitive and not in line with research on AS and interoceptive awareness in other domains of anxiety related disorders. For instance, Sturges and Goetsch (1996) found that women high on anxiety sensitivity were significantly more accurate at heartbeat perception than women low on AS, although absolute heart rate did not differ across groups. Accurate perception of changes in pulse transit time and several other measures of sympathetic activity were consistently related to higher levels of AS in a study by Richards and Bertram (2000). In a review combining these and other studies by Domschke et al (2010), enhanced interoceptive awareness was characteristic of high AS individuals. The weighted mean effect size (Cohen's d) for

the relationship between AS and heartbeat perception was .63, indicating a medium to large effect. Individuals high in AS are generally more accurate perceivers of interoceptive processes associated with anxiety compared to individuals low in AS. Given the fact that high AS individuals are more accurate perceivers, that is better perceivers of anxiety related arousal, one would expect higher levels of self-reported anxiety in these high AS individuals when arousal is indeed elevated in anxiety provoking situations.

The aim of this study was to investigate to what extent flight phobics who score high on AS and who react with an increase in physiological arousal to phobic stimuli report a higher flight anxiety than aviophobics who score low on AS, even when these individuals show a concordant increase in physiological arousal. First, we tried to replicate the findings of our colleagues (Vanden Bogaerde & De Raedt, 2011) regarding the moderating effect of AS on self-reported somatic sensations and flight anxiety. Next, we tried to extend their findings by including measurements of autonomic nervous system reactions induced by real flight into our analyses.

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METHOD

Participants

The 50 participants in this study were aviophobics who participated in a treatment program for fear of flying at the VALK foundation in Leiden, The Netherlands. The VALK Foundation is a collaborative venture by the Leiden University, Amsterdam Airport Schiphol, KLM, Transavia.com, Martinair and ArkeFly, specialized in treating fear of flying (FOF). The treatment program starts with a diagnostic assessment during the first visit in Leiden, followed by individual therapeutic sessions and a two day cognitive-behavioral group treatment (CBGT) as described in detail elsewhere (Van Gerwen, Spinhoven, & Van Dyck, 2006; Van Gerwen, Spinhoven, Diekstra, & Van Dyck, 2002). Most participants were self-referrals, some were referred by health care agencies, health professionals and company health programs. Airline personnel were excluded from this study. Other reasons for exclusion were current use of cardioactive medication like β blockers and a concurrent panic disorder of such severity according to the treating clinician that it would seriously interfere with the treatment of fear of flying. 79 individuals with aviophobia were considered eligible and participated in this study. Inclusion criteria for this study were complete data on all essential questionnaires (ASI, SUD, VAFAS) and complete data of all physiological variables (HR, RSA, PEP) during both flights. The security check at the airport appeared to be a major barrier for the physiological measurements.

The ambulatory measurement device and attached electrodes required a physical padding of all participants. After security screening 19% of the recording devices did not record all variables properly. Physiological data of two participants was lost due to equipment failure. One flight was cancelled due to adverse weather, excluding another 2 participants. Finally, ten participants were excluded from analyses because of incomplete data on the relevant questionnaires. This left 50 phobic clients (22 men) with an average age of 38,4 (S.D. = 10,6). Extensive missing value analysis on all physiological data and all questionnaire data available revealed no systematic differences between the fifty remaining participants and the 29 participants with incomplete data, with only small effect sizes for differences between both groups on questionnaire data (η^2 < .01). The largest effect size on the physiological variables was found for differences in HR during taxi-out on the first flight: η^2 = .014.

INSTRUMENTS

Physiological recordings

Heart Rate (HR), Respiratory Sinus Arrhythmia (RSA) and the Pre-Ejection Period (PEP) were recorded using the VU-AMS (version 4.6, Vrije Universiteit Amsterdam, The Netherlands; www.vu-ams.nl). The VU-AMS is a light-weight ambulatory device that records the impedance cardiogram (ICG) and electrocardiogram (ECG) continuously in freely moving subjects by means of six Ag-AgCl electrodes attached to the torso region (De Geus, Willemsen, Klaver, & van Doornen, 1995; Willemsen,G.M.H., De Geus, Klaver, Van Doornen, & Carrol, 1996). The apparatus has an inbuilt vertical accelerometer, which output can be used to select movement free periods for analysis. The RSA is a measure of parasympathetic control (Berntson et al., 1994), whereas PEP is considered a measure of sympathetic cardiac control (Sherwood et al., 1990). HR can be viewed as the resultant of both control mechanisms. In general, stimulation of the parasympathetic system will decrease the heart rate, while stimulation of the sympathetic system will increase the heart rate and the force of contraction. Scoring of these variables was automatic, followed by visual inspection of the impedance and respiratory signal from the entire recording. Details on scoring of these variables, recording methodology, reliability and validity are describes elsewhere (De Geus et al., 1995; Goedhart, Kupper, Willemsen, Boomsma, & de Geus, 2006; Goedhart, van der Sluis, Houtveen, Willemsen, & de Geus, 2007; Houtveen, Groot, & de Geus, 2006; Riese, 2003; Willemsen,G.M.H. et al., 1996). Briefly, from the ECG (sampling rate 1000 Hz) the HR was obtained from the time between two adjacent R waves. PEP was defined from the ECG and ICG as the time interval from

the Q-wave onset, the onset of the electromechanical systole, to the B-point (from the ICG), which signals opening of the aortic valves (Sherwood et al., 1990; Willemsen,G.M.H. et al., 1996). RSA was obtained from the ECG and thorax impedance derived respiration signals by subtracting the shortest 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, van Beek, & Wientjes, 1990). When no phase-related acceleration or deceleration was found, the breath was assigned a RSA score of zero. Our focus on cardiac parameters reflects three major considerations: measurements needed to be as non-invasive as possible, they needed to respond to changes in psychological state over a time scale of a few minutes and they needed to be reliable in an ambulatory setting. The HR, PEP and RSA measures are uniquely qualified to meet these demands (Goedhart et al., 2006; Willemsen,G.M.H. et al., 1996).

QUESTIONNAIRES

All questionnaires were administered in the Dutch language.

Visual Analogue Flight Anxiety Scale (VAFAS)
The one-tailed visual analogue flight anxiety scale was used at initial diagnostic assessment and after the second flight to examine to what extent participants were anxious about flying. The scale ranges from 0 ("no flight anxiety") to 10 ("terrified or extreme flight anxiety") (Nousi, Van Gerwen, & Spinhoven, 2008a).

Flight Anxiety Situations (FAS) questionnaire
This 32-item self-report inventory administered at initial assessment and after the second flight assesses anxiety related to flying experienced in different flight or flight related situations on a five point Likert scale. The questionnaire consists of three subscales: (a) an Anticipatory Flight Anxiety Scale, containing 14 items that pertain to anxiety experienced when anticipating a flight, (b) an In-Flight Anxiety Scale, containing 11 items measuring anxiety experienced during a flight and (c) a Generalized Flight Anxiety Scale, containing seven items assessing anxiety experienced in connection with airplanes in general (Nousi et al., 2008a; Van Gerwen et al., 1999). The internal consistency of the subscales of the FAS in the present study was good to excellent, Cronbach's Alpha ranging from .86 to .95.

Flight Anxiety Modality (FAM) questionnaire
The FAM is a 23 item self-report inventory that was used to assess the symptoms by

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which flying related anxiety was expressed at initial assessment and after the second flight. Each symptom is rated on a 5-point Likert-type scale. The questionnaire consists of two subscales: (a) a Somatic Modality scale, pertaining to physical symptoms and (b) a Cognitive Modality scale, related to the presence of distressing cognitions (Nousi et al., 2008a; Van Gerwen et al., 1999). The internal consistency of the two subscales of the FAM in the present study was good at the initial diagnostic assessment phase, Cronbach's Alpha respectively .85 and .90, and acceptable to excellent after the second flight, Cronbach's Alpha respectively .76 and .94.

Anxiety Sensitivity Index (ASI)

The Dutch version of the Anxiety Sensitivity Index (Reiss, 1986; Vancleef, 2006) was administered once during the initial diagnostic assessment to assess the degree to which participants are concerned about possible negative consequences of anxiety related sensations. The 16 self-report items are rated on a 5-point Likert-type scale. For the operationalization of anxiety sensitivity (AS) we used the total score on the ASI. Cronbach's Alpha in the present study was .83.

Subjective Units of Discomfort (SUD)

The Subjective Units of Discomfort scale was verbally administered to each participant individually during taxi-out flight 1 to examine to what extent participants were feeling anxious. The one-tailed scale ranges from 1 ("totally relaxed") to 10 ("extremely anxious") (Wolpe, 1973).

Procedures

Anxiety sensitivity, flight phobia (FAS, FAM and VAFAS) and related psychopathology were assessed during the initial diagnostic assessment by questionnaires on fears and phobias in general and fear of flying in particular, followed by a semi-structured interview by a fully qualified clinical psychologist. Written informed consent was obtained from all participants at this stage as well. The local medical ethics committee approved the research protocol. At the end of this assessment phase the therapist proposed a treatment plan based on individual needs. The ensuing individualized preparation phase consisted of one to four therapeutic sessions covering relaxation and breathing techniques, psychological factors involved in fear and anxiety, and coping skills. Acrophobia, claustrophobia, traumatic social events and traumatic transportation accidents were addressed if applicable.

All participants started CBGT five weeks after initial assessment. Upon arrival for the second day of CBGT six electrodes were attached and connected to the ambulatory monitoring device. The second day of CBGT is focused on exposure, with at the end a guided return flight within Europe. Both flights were regular commercial flights with a flying time of around one hour. On average a CBGT group consisted of eight patients, a fully qualified psychotherapist and a pilot. The experimental data collection focussed on these two flights.

During taxi-out of the first flight a SUD was verbally administered to each participant individually. Using a visual display of the output of an inbuilt vertical accelerometer of the ambulatory monitoring device in combination with a log kept by the therapist and accompanying pilot we identified artefact-free periods that lasted 5 minutes each during taxi-out flight 1 and taxi-in flight 2. After disembarkation, some thirty minutes after taxi-in of flight 2 participants filled out questionnaires on flight phobia (FAM, FAS and VAFAS). Thereafter, the electrodes and the ambulatory recording device were removed.

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Data analysis
The first aim of this study was to replicate the findings of Vanden Bogaerde and De Raedt (2011) regarding the moderating effect of AS on self-reported somatic sensations and flight anxiety. The SUD score during taxi-out of the first flight was used as dependent variable, analogue to the Visual Analogue Scale (VAS) measurement used by Vanden Bogaerde and de Raedt. In addition, we repeated the analysis with the VAFAS scores after the second flight as an alternative dependent variable. Anxiety sensitivity (AS) and the somatic modality subscale (FAM-after flight) were used as independent variables. The product of the FAM somatic modality subscale scores with the AS scores was added to the regression models to test for an interaction between somatic sensations and anxiety sensitivity.

The second aim of this study was to extend the findings of Vanden Bogaerde and De Raedt (2011) by including measurements of autonomic nervous system reactions induced by real flight into the analyses. In order to do so we created change scores for each of the three autonomic variables (HR, RSA and PEP) that reflected the reactivity to both flights. Change scores were defined as the value during taxi-in of the second flight minus the value during taxi-out of the first flight. Flight anxiety after the second flight (VAFAS) was again used as dependent variable. In addition we also assessed the flight anxiety change scores (VAFAS after exposure minus VAFAS at diagnostic assessment). Negative change scores reflect higher reactivity for HR, and lower reactivity for RSA and PEP. Physiological

change scores, AS scores and the product of the physiological change scores with the AS scores were used as independent variables in multiple regression, to test the hypotheses that AS moderates the relationship of physiological reactivity with flight anxiety and change in flight anxiety.

All independent variables were standardized to eliminate multicollinearity problems and to be able to report the correct regression coefficient B (Aiken & West, 1991). RSA was first log (ln) transformed to obtain normal distributions.

RESULTS

Clinical characteristics

As shown in table 1, scores on the VAFAS and all FAM en FAS (sub-) scales at the assessment phase were in line with the established norms for these questionnaires (Nousi et al., 2008a). Post flight scores were slightly above post treatment scores reported by Nousi et al. (2008b) for 251 participants who underwent the same therapy previously. Differences between scores can be explained by time of measurement, as Nousi et al. collected data three months after treatment, while our participants filled out questionnaires 30 minutes after the second flight. Anxiety sensitivity scores were slightly above scores reported for 160 participants with specific phobia by Naragon-Gainey (2010) in a meta-analyses on AS and anxiety disorders, but very much in line with scores reported by Vanden Bogaerde and De Raedt (2011) with 54 flight phobics. Table 2 shows pre- and post-exposure physiological variables. Eta square (η^2), being the effect size statistic for repeated measures ANOVA, showed a large effect for all measures. By convention, η^2 values of .01, .06, and .14 are interpreted as small-, medium-, and large effect sizes, respectively.

Regression Analyses

We started with multiple regression analyses without physiological variables in an effort to replicate the findings of Vanden Bogaerde and De Raedt (2011). We were unable to reproduce their outcome. Results indicated no interaction effects and no main effect for AS and somatic sensations on the SUD score during taxi-out of the first flight. Only with flight anxiety (VAFAS) after flight instead of the SUD as dependent variable a main effect for somatic sensations showed up t(46) = 2.77, p = .008), but still no effect for AS nor an interaction effect was seen (table 3).

Table 1. Measures of flight-related anxiety and somatic complaints at pre- and post-treatment.

	Assessment score		After second flight		
	Mean	SD	Mean	SD	Effect size (η^2)
FAS					
Anticipatory anxiety	43.0	8.5	25.8*	15.5	.510
In-flight anxiety	37.4	8.4	19.5*	7.0	.807
Generalized flight anxiety	13.2	4.9	8.9*	2.3	.444
Sum score	104.1	18.1	60.0*	21.6	.748
FAM					
Somatic complaints	26.6	9.2	15.9*	4.0	.647
Cognitive complaints	23.9	7.8	11.7*	5.3	.732
AS	33.1	9.4	-	-	
VAFAS	7.9	1.3	2.1*	1.7	.904

* Assessment score differ from post-flight score at *P < .001
FAS = Flight Anxiety Situations Questionnaire, FAM = Flight Anxiety Modality Questionnaire, VAFAS = Visual Analogue Flight Anxiety, AS = Anxiety Sensitivity, as measured by the Anxiety Sensitivity Index (ASI) questionnaire.

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Table 2. Mean and S.D. of the three physiological variables HR, RSA and PEP before and after the exposure flights.

	Taxi-out 1		Taxi-in 2		
	Mean	SD	Mean	SD	Effect size (η^2)
HR	97.7	15.0	84.6**	12.4	.719
RSA	28.3	18.7	33.4*	21.1	.154
PEP	87.3	15.0	91.8*	16.8	.204

* Pre-flight (Taxi-out 1) score differ from post-flight (Taxi-in 2) score at * P < .005 and ** P < .001
HR = Heart Rate, RSA = Respiratory Sinus Arrhythmia, PEP = Pre-Ejection Period.

Table 3. Regression analyses of flight anxiety (VAFAS) on Anxiety Sensitivity (AS), Somatic Sensations (FAM) and their interaction.

	B	SE	t	p	Zero-order correlation
Step 1					
Constant	2.080	.217	9.582	<.001	-
AS-assessment	.076	.233	.325	.747	.182
FAM-after exposure	.655	.233	2.804	.007	.412
Step 2					
Constant	2.077	.229	9.086	<.001	-
AS-assessment	.070	.270	.260	.796	.182
FAM-after exposure	.655	.236	2.774	.008	.412
Interaction AS-FAM	.008	191	.042	.967	.085

VAFAS = Visual Analogue Flight Anxiety, FAM = Flight Anxiety Modality Questionnaire (somatic subscale), AS = Anxiety Sensitivity, as measured by the Anxiety Sensitivity Index (ASI) questionnaire.

Next, the hypothesis was tested that phobics who score high on anxiety sensitivity in combination with a heightened physiological arousal to flight exposure show a higher flight anxiety than aviophobics who score low on AS, even when these individuals show a concordant elevated physiological arousal. Physiological reactivity to flight predicted flight anxiety after the flight ($F_{(7, 42)}$ = 3.00, p = .012, R-Square = .333) and pre- to post flight changes in flight anxiety ($F_{(7, 42)}$ = 7.46, p < .001, R-Square = .554). A main effect for physiological reactivity emerged for both anxiety variables. Higher HR reactivity to flight was associated with less flight anxiety after exposure and a stronger decrease in flight anxiety over both flights. In addition, lower RSA reactivity was associated with less flight anxiety after exposure and a stronger decrease in flight anxiety over both flights (tables 4 and 5) [1].

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[1] Analyses over both flights separately produced similar results.

Table 4. Regression analyses of flight anxiety (VAFAS) on Anxiety Sensitivity (AS), physiological measures (HR, RSA and PEP) and their interaction.

	VAFAS After exposure				
	B	*SE*	*t*	*p*	Zero-order correlations
Step 1					
Constant	2.080	.211	9.880	<.001	-
AS-assessment	.341	.226	1.507	.139	.182
HR Reactivity	.846	.273	3.097	.003	.410
RSA Reactivity	.433	.233	1.859	.070	.040
PEP Reactivity	.025	.266	.096	.924	-.268
Step 2					
Constant	2.193	.266	9.718	<.001	-
AS-assessment	.590	.256	2.305	.026	.182
HR Reactivity	1.000	.312	3.204	.003	.410
RSA Reactivity	.562	.248	2.272	.028	.040
PEP Reactivity	.305	.350	.872	.388	-.268
Interaction AS-HR	-.013	.292	-.045	.964	-.009
Interaction AS-RSA	.589	.287	2.051	.047	.193
Interaction AS-PEP	.110	.313	.352	.726	-.059

VAFAS = Visual Analogue Flight Anxiety, AS = Anxiety Sensitivity, as measured by the Anxiety Sensitivity Index (ASI) questionnaire, HR = Heart Rate, RSA = Respiratory Sinus Arrhythmia, PEP = Pre-Ejection Period.

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Post-hoc simple slope analyses revealed that in the group of high AS participants changes in HR significantly predicted changes in flight anxiety (B = .761, $t(46)$ = 2.00, p = .05), in such a way that decreased fear was accompanied by the highest HR reactivity. This relationship was not significant for the group of low AS participants (B = .211, $t(46)$ = .510, p = .612) (fig 1 left panel). For the group of high AS participants, changes in RSA did significantly predict changes in flight anxiety (B = 1.38, $t(46)$ = 3.15, p = .003) whereas this relationship was not significant for participants in the low AS group (B = -.186, $t(46)$ = -.507, p = .614) (fig 1 right panel).

Table 5. Regression analyses of changes in flight anxiety (VAFAS) on Anxiety Sensitivity (AS), physiological measures (HR, RSA and PEP) and their interaction.

	VAFAS Reactivity				
	B	SE	t	p	Zero-order correlation
Step 1					
Constant	-6.021	.194	-31.08	<.001	-
AS-assessment	.515	.208	2.471	.017	.263
HR Reactivity	.803	.251	3.197	.003	.320
RSA Reactivity	.867	.214	4.043	<.001	.279
PEP Reactivity	-.091	.245	-.370	.713	-.298
Step 2					
Constant	-5.829	.188	-31.01	<.001	-
AS-assessment	.861	.213	4.037	<.001	.263
HR Reactivity	1.046	.260	4.021	<.001	.320
RSA Reactivity	1.030	.206	4.993	<.001	.279
PEP Reactivity	.424	.291	1.457	.153	-.298
Interaction AS-HR	.500	.243	2.054	.046	.211
Interaction AS-RSA	.895	.239	3.743	.001	.204
Interaction AS-PEP	.305	.261	1.169	.249	-.128

VAFAS = Visual Analogue Flight Anxiety, AS = Anxiety Sensitivity, as measured by the Anxiety Sensitivity Index (ASI) questionnaire, HR = Heart Rate, RSA = Respiratory Sinus Arrhythmia, PEP = Pre-Ejection Period.
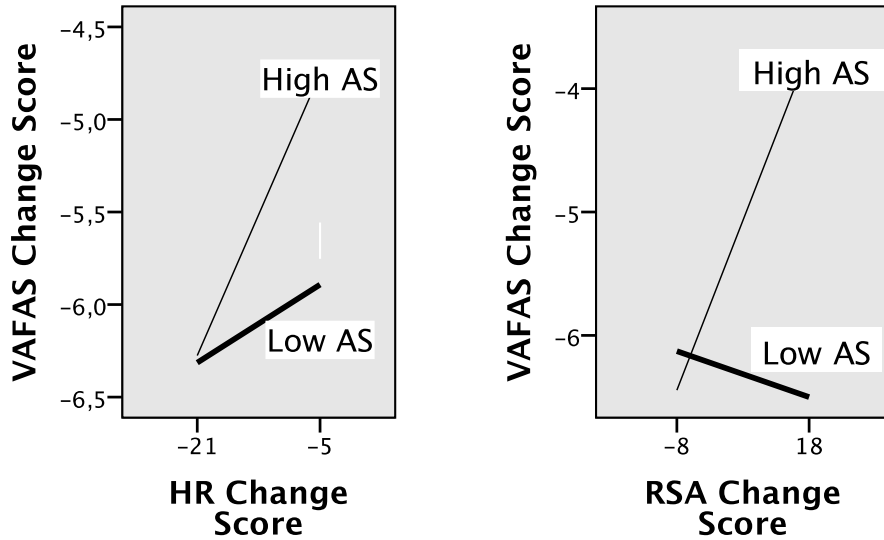
Figure 1. Simple slope regression lines for HR change scores, RSA change scores and flight anxiety change scores (VAFAS), for low AS and high AS aviophobics. A negative VAFAS change score implies decreased flight anxiety from pre- to post-measurement. A negative HR change score implies higher HR reactivity to flight, and a positive RSA change score implies a higher parasympathetic reactivity. FAM = Flight Anxiety Modality Questionnaire. VAFAS = Visual Analogue Flight Anxiety Scale. HR = Heart Rate. RSA = Respiratory Sinus Arrhythmia.

DISCUSSION

This study explored the relationship between anxiety sensitivity, somatic sensations, physiological arousal and fear of flying. In fifty aviophobics participating in CBT to overcome fear of flying, cardiac autonomic responses were assessed during two guided exposure flights. Results indicate that physiological reactivity predicted flight anxiety after the exposure as well as changes in reported flight anxiety. Furthermore, physiological reactivity interacted with anxiety sensitivity. Changes in heart rate and parasympathetic activity were more strongly associated with changes in reported flight anxiety for high AS participants, and less for participants low on anxiety sensitivity.

Flight phobia has been linked to elevated levels of anxiety sensitivity (Naragon-Gainey, 2010; Vanden Bogaerde & De Raedt, 2008; Vanden Bogaerde & De Raedt, 2011). In our sample of aviophobics, AS scores were very much in line with these findings as well as with AS scores previously found for participants with specific phobia (Rivas & Tortella-Feliu, 2000). According to Clark's cognitive approach to panic (Clark, 1986), normal autonomic anxiety responses are catastrophically misinterpreted as much more dangerous than they really are. This misinterpretation leads to increased fear and anxiety, which leads to even more bodily sensations, creating a vicious cycle. Anxiety sensitivity is thought to be the moderating key element in this relationship between somatic sensations and anxiety, the driving force between awareness of the aversive bodily sensations and the misinterpretation as danger signals (Reiss, 1991). Up till now only questionnaires and verbal report have been used to assess this relationship with aviophobics. This study is the first to include actual physiological arousal.

Our results indicate that the addition of physiological markers of arousal strengthens the model of cognitive misinterpretation of bodily sensations, in such a way that a higher HR reactivity was associated with a stronger reduction in flight anxiety for all participants, but more so for aviophobics who score high on AS. Individuals high in AS are known to be better perceivers of heartbeat activity than persons low on AS (Domschke et al., 2010). In view of our results it seems that changes in heartbeat influences anxiety in participants who are susceptible to interpret normal bodily sensations in a threatening manner. Interestingly, low parasympathetic reactivity to flight further contributed to a decrease in flight anxiety after exposure. Increased parasympathetic activity when exposed to frightening stimuli was seen in some dental phobics and blood phobics, and resembles a passive coping response, characterized by increased sympathetic activity paired to a concurrent increase in parasympathetic activity (Bosch et al., 2001; Bosch, de Geus,

Veerman, Hoogstraten, & Nieuw Amerongen, 2003; Sarlo, Palomba, Angrilli, & Stegagno, 2002). High AS has been associated with accurate perception of several measures of sympathetic activity (Richards & Bertram, 2000), but to our knowledge there is no research linking parasympathetic (RSA) changes to interoceptive awareness. Our results indicate that a sympathetically driven HR reactivity paired to low parasympathetic reactivity to actual flight was associated with decreased flight anxiety. The lack of interaction between AS, FOF and actual physiological reactivity to phobic video stimuli previously reported by the same authors (Busscher et al., 2010) might have been caused by the low ecological validity of the used stimuli. The current study used in vivo exposure during two actual flights.

We were unable to reproduce the moderating effect of AS on the relationship between self-reported somatic sensations and flight anxiety as reported by Vanden Bogaerde and De Raedt (2008, 2011). In their most recent study they asked flight phobics and control participants just before take-off, while already seated in the airplane, to indicate how anxious they were using a Visual Analogue Scale (VAS), and their bodily sensations using the FAM somatic subscale. The VAS normally measures state anxiety (Davey, Barratt, Butow, & Deeks, 2007) and not flight anxiety. To replicate their analyses we used the SUD value during taxi-out of the first flight, which is comparable to their VAS. We also performed the regression analyses with the VAFAS, a true measure of flight anxiety. Although a positive correlation existed between somatic sensations and flight anxiety, no moderating effect of anxiety sensitivity emerged on the relationship of somatic sensations with either the SUD or VAFAS. It seems that flight phobics who are afraid of anxiety-related bodily sensations do not report more flight anxiety or distress than phobics who score low on this trait, even when they report more somatic sensations. Vanden Bogaerde and De Raedt (2011) used the combined sample of aviophobics (n = 54) and controls (n = 49) in their moderator analyses. We performed our study only in aviophobics seeking treatment. The difference in results might be partly due to the inclusion of control participants in their analyses.

Although AS generally is seen as a stable dispositional variable, cognitive behavioural therapy (CBT) is efficacious in reducing AS. In a meta-analytic review large effect sizes for CBT in reducing AS in clinical and at risk samples are reported (Smits, Berry, Tart, & Powers, 2008). Likewise, a meta-analytic review on AS and anxiety disorders shows that changes in AS correspond with changes in anxiety symptoms (Olatunji & Wolitzky-Taylor, 2009). In their review on interoceptive sensitivity Domschke et al (2010) remark that regardless of the accuracy of the interoceptive perception, individuals high in AS show heightened

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reactivity to these sensations. Moreover, they conclude that behavioural and cognitive interventions, especially cognitive restructuring, exposure to interoceptive stimuli and biofeedback using heart rate, are effective in modifying a putative stable trait as AS.

We did not measure perception of arousal during the flight, nor did we measure cognitive misinterpretation in real-time. Consequently, we have to be prudent with therapeutically recommendations. However, it seems reasonable to assume that, although not measured, participants most probably were able to perceive their (increased) heartbeat. As changes in heartbeat were associated with anxiety reduction, more focused exposure to these physiological symptoms might be a clinical implication of the present findings. The fact that this association was more pronounced in participants high on AS can be interpreted in various ways, because neither perception nor interpretation of physical symptoms has been assessed in real time in the present study. However, persons with elevated levels of AS are known to habitually attend to bodily sensations and to interpret normal autonomic anxiety responses as much more dangerous than they really are. Possibly, cognitive interventions in addition to exposure to bodily sensations might be particularly useful in those participants who are more likely to attend to these sensations and to interpret them catastrophically (Oakes & Bor, 2010; Wells & Papageorgiou, 2001; Wild, Clark, Ehlers, & McManus, 2008). Evidently, future research assessing perception and interpretation of bodily sensations in participants varying in anxiety sensitivity during actual flight seems warranted.

The strength of the current study is the use of a relatively large sample of true aviophobics seeking treatment, in combination with in vivo exposure and actual psycho-physiological measurements. To our knowledge only a few studies used comparable numbers of true aviophobic participants in real live exposure but without including psychophysiological measurements [e.g., Van Gerwen et al (2003, 2006), Howard et al (1983)]. Burger (2011) took ECG recordings from 24 aviophobic individuals during a fear of flying seminar including exposure in a simulator and two actual flights. Many other studies on fear of flying used limited number of participants [(Holmes et al (1979) n=5; Haug et al (1987) n=10; Ekeberg et al (1989, 1990, 1990) n between 13 and 34; Wilhelm and Roth (1997a, 1998b, 1998a) in a series of publications based on the same experiment n=28 women], lacked in-vivo exposure [Busscher et al (2010); McNally and Louro (1992); Wilhelm and Roth (1997b); Bornas et al (2006a, 2006b, 2007)], made use of non-clinical participants (Vanden Bogaerde & De Raedt, 2008), or used a combination of low numbers, students and no in vivo exposure [Bornas et al (2004, 2005) n=15].

This study also has several shortcomings. Aviophobics generally score high on AS, and did so in this experiment. This limits the range of scores and hence predictive power. The anxiety sensitivity questionnaire was administered only once, at the very first beginning of the therapy, while the FAM, FAS and VAFAS were taken at the beginning and once more at the end. Although AS is regarded as a relatively stable trait, as mentioned before CBT is known to be efficacious in reducing AS. The individual therapeutic sessions and CBGT were not specifically aimed at reducing AS, nevertheless AS might have been reduced as a side effect in the course of the experiment. Another limitation is the high study drop-out due to equipment failure, the security screening before the flights and incomplete questionnaire data. Extensive missing data analyses however showed no systematic differences between completers and the lost participants. Moreover, the VAFAS was only administered at initial diagnostic assessment and after the second flight and consequently change scores on the VAFAS may also reflect the effect of the individual preparation phase and the first day of group treatment preceding the exposure flights. Also, the semi-structured interview used during assessment was not validated to verify diagnosis of simple phobia. A further limitation of the current study, which precludes conclusions about cause and effect relationships, lies in the correlational design. Longitudinal and controlled trials could further clarify the relationship of AS, somatic sensations, physiological reactivity and fear of flying.

In conclusion, we found a positive correlation between somatic sensations and flight anxiety, but no proof that AS moderates this relationship. The addition of physiological markers of anxiety, especially HR, strengthens the model of cognitive misinterpretation of bodily sensations. A stronger reactivity in HR was associated with a stronger reduction in flight anxiety for all aviophobics, but more so for participants high on AS. Results indicate that CBT for FOF might benefit from addressing somatic sensations and the physical effect of anxiety, by means of cognitive restructuring and exposure to interoceptive stimuli, particularly in aviophobics high in AS.

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