Mediators of Social Anxiety

External Social Threat-Cues vs. Self-Related Negative Cognitions

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"Forty-two!" […] *"Is that all you got to show for seven and a half million years' work?"* [...] *"I checked it very thoroughly," said the computer, "and that quite definitely is the answer."*

– Douglas Adams

"*Theories are constructed to explain, predict, and master phenomena.* […] *The world is an interpretation (or model) of such scientific theories, only insofar as the sciences are true.*"

– Wikipedia

The most exciting phrase to hear in science, the one that heralds new discoveries, is not "Eureka!" but "That's funny…"

– Isaac Asimov

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Abstract

Based on a review of models and empirical findings a working model is proposed, suggesting that *self-related negative cognitions* and biased processing of *external social threat-cues* are mediators of social anxiety. Hypotheses derived from this model were tested in three experiments.

The first experiment examined whether levels of trait social anxiousness predicted fearful responding to *external social threat-cues* (angry vs. neutral and happy facial expressions) during social evaluation. Higher trait social anxiousness predisposes to an inward focus on one's fear reaction to social threat. Using this strategy was expected to enhance fearful responding to angry facial expressions. A strategy of identifying with angry faces was expected to counteract fearful responding, but was expected to fail more often with increasing levels of trait social anxiousness. To examine these hypotheses, affective modulation of the startle eye-blink was assessed in forty-four undergraduate students. This measure served as a probe into the activation of brain structures involved in the automatic evaluation of environmental threat-cues. Trait and state anxiety as well as explicit emotional responding to the stimuli were assessed with questionnaires and ratings. Processing angry faces potentiated startle amplitudes as expected. Low arousal induced by the stimuli was a probable reason, why startle potentiation to happy faces emerged instead of attenuation. Trait social anxiousness and the cognitive strategies did not influence these effects. Yet, increased trait social anxiousness predicted decreased startle latency, indicating motor hyper-responsivity, which is part of the clinical representation of social anxiety disorder (SAD). Processing facial expressions and identifying with them disrupted this association. Previous studies support that similar strategies may enhance treatment of SAD.

Individuals with SAD were expected to respond with increased arousal to *external social threat-cues.* Therefore, the *second experiment* examined whether nine individuals with SAD showed attentional (prepulse inhibition, PPI) or affective startle modulation to angry as compared to neutral and happy facial expressions. Corrugator supercilii activity was assessed as a behavioral indicator for effects of facial expressions. The remaining setup resembled the first experiment.

Facial expressions did not modulate the startle reflex, but corrugator supercilii activity was sensitive to facial valence. However, the effects were not related to trait social anxiousness. Apparently, angry facial expressions do not act as phobic stimuli for individuals with SAD.

The third experiment examined whether focusing on *self-related negative cognitions* or biased processing of *external social threat-cues* mediates relationships between trait social anxiety and anxious responding in a socially challenging situation. Inducing *self-related negative cognitions* vs. relaxation was expected to reveal a functional dependency on the supposed mediation in a multivariate assessment of criteria of the working model. Within this design, the impact of *external social threat-cues* (facial expressions and emotional words) was compared to control stimuli and context effects, using the startle paradigm.

The findings provide first evidence for full statistical mediation of the associations between trait social anxiety and self-reported anxiety as well as parasympathetic withdrawal by *self-related negative cognitions*, when thirty-six undergraduate students anticipated public speaking. Apprehensive arousal, as indicated by increased skin conductance levels and heart rate, was present in all participants. Observer ratings of behavior during public speaking matched the self-rated quality of the performance. None of these measures were correlated with trait social anxiousness. Startle amplitude correlated with state and trait social anxiety, but was no mediator. Finally, there was no affective modulation of the startle amplitude by *external social threat-cues.*

These studies advance both our current understanding of the factors that mediate social anxiety responses to situations and our knowledge of the physiological and anatomical mechanisms involved in social anxiety. Based on these findings a revised version of the working model on mediators of social anxiety is proposed in the hope it may aid further research for the ultimate goal of developing an empirically validated functional anatomical model of social anxiety.

Zusammenfassung

Sozial ängstliche Menschen richten ihre Aufmerksamkeit in sozial bedrohlichen Situationen nach innen auf *selbstbezogene negative Gedanken* (z.B. „Ich schaffe das nicht!“).Außerdem richten sie ihre Aufmerksamkeit unwillkürlich bevorzugt auf potenziell *bedrohliche soziale Umweltreize* und beurteilen diese im Vergleich zu niedrig sozial ängstlichen Kontrollpersonen besonders negativ. Einschlägige Modelle und die Fachliteratur lassen den Schluss zu, dass *selbstbezogene negative Gedanken* und die systematisch verzerrte Verarbeitung *bedrohlicher sozialer Umweltreize* Mediatoren für Zusammenhänge zwischen sozialer Ängstlichkeit und akuter Angst in sozial bedrohlichen Situationen sind. Zudem finden sich Hinweise auf Wechselwirkungen zwischen den angenommenen Mediatoren. Auf dieser Grundlage wurde ein *Arbeitsmodell zu Mediatoren sozialer Angst* erstellt. In drei Experimenten wurden von diesem Modell abgeleitete Hypothesen überprüft.

Im ersten Experiment wurde untersucht, ob erhöhte soziale Ängstlichkeit mit verstärkten Furchtreaktionen bei der automatischen Verarbeitung *bedrohlicher sozialer Umweltreize* (ärgerliche vs. neutrale und fröhliche Gesichtsausdrücke) einhergeht, die in einer sozialen Bewertungssituation präsentiert wurden. Es wurde erwartet, dass kognitive Emotionsregulationsstrategien diesen Zusammenhang *top-down* moderieren. Sozial ängstliche Menschen neigen dazu, sich im Angesicht sozialer Bedrohung auf ihre Furchtreaktion zu konzentrieren. Es wurde erwartet, dass eine alternative Strategie, nämlich sich mit einem ärgerlichen Gesichtsausdruck zu identifizieren, die Furcht reduziert. Menschen mit hoher sozialer Ängstlichkeit dürfte dies jedoch besonders schwer fallen.

Um diese Annahmen zu überprüfen wurde die affektive Schreckreflexmodulation an Studenten im Grundstudium (N = 44) untersucht. Die Lidschlussreaktion beim Schreckreflex diente dabei als Indikator für die Aktivierung von Gehirnstrukturen, die an der automatischen Entdeckung und Bewertung von bedrohlichen Umweltreizen beteiligt sind. Explizite emotionale Bewertungen der

Stimuli, soziale Ängstlichkeit und emotionales Befinden wurden in Fragebogen und Ratings erfasst.

Erwartungsgemäß war die Amplitude der Schreckreflexreaktion bei der Verarbeitung ärgerlicher Gesichtsausdrücke erhöht. Vermutlich lag das daran, dass die Stimuli nur geringe Erregung auslösten, so dass die Reaktion bei fröhlichen Gesichtern nicht verringert, sondern ebenfalls erhöht war. Unerwartet war, dass diese Effekte nicht durch die Anwendung der Emotionsregulationsstrategien verändert wurden, und dass sich kein Zusammenhang zum Grad der sozialen Ängstlichkeit zeigte. Es gab jedoch einen Zusammenhang zwischen sozialer Ängstlichkeit und der Latenz von Schreckreflexreaktionen. Dies indiziert eine erhöhte Ansprechbarkeit motorischer Systeme bei sozial ängstlichen Menschen, was dem klinischen Bild entspricht. Verarbeiteten die Versuchspersonen emotionale Gesichtsausdrücke und versuchten sie, sich mit emotionalen Gesichtsausdrücken zu identifizieren, zeigte sich dieser Zusammenhang nicht. Mit einer ähnlichen Strategie (Instruktion, die Aufmerksamkeit auf Umweltreize zu lenken) gelang es bereits, die erlebte Angst während der Expositionstherapie zu verringern und den Therapieerfolg zu erhöhen. Der bewusste Einsatz solcher Strategien könnte daher nützlich bei der Therapie sozialer Angst sein.

Patienten mit einer klinischen Diagnose „Soziale Angststörung“ (englisch: SAD) sollten auf die im ersten Experiment verwendeten *bedrohlichen sozialen Umweltreize* mit verstärkter Erregung reagieren, wenn sie diese in einer sozial bedrohlichen Situation betrachten. *Im zweiten Experiment* wurde daher untersucht, ob die automatische Verarbeitung *sozial bedrohlicher Umweltreize* (ärgerliche vs. neutrale und fröhliche Gesichtsausdrücke) bei Menschen mit SAD durch eine automatische Ausrichtung der Aufmerksamkeit (Prepulse-Inhibitition, PPI) oder verzerrte implizite Bewertung (affektive Modulation der Schreckreflexreaktion) gekennzeichnet ist. Zudem wurde die spontane Aktivität des Corrugator Supercilii über EMG erfasst. Im Sinne eines Manipulation-Checks wurde erwartet, dass ärgerliche Gesichtsausdrücke die Aktivität steigern und fröhliche sie verringern. Ansonsten entsprach die Versuchsanordnung Experiment 1.

Die Ergebnisse zeigten, dass die Schreckreflexreaktion von Patienten mit sozialer Angststörung während der Verarbeitung emotionaler Gesichtsausdrücke weder aufmerksamkeits- noch affektbezogen moduliert wurde. Bootstrapping und nachträgliche Effektstärkenanalysen zeigten, dass bei ähnlich großen Stichproben wie im ersten Experiment ebensolche Ergebnisse zu erwarten sind. Signifikante PPI-Ergebnisse wären aber erst in weitaus größeren Stichproben zu erwarten. Dies stützt die Annahme, dass niedrige Erregung, ausgelöst durch die Stimuli die Ergebnisse erklären. Signifikante Effekte der Stimuli (Ratings und spontane Aktivität des Corrugator Supercilii) traten unabhängig vom Grad der sozialen Ängstlichkeit auf. *Sozial bedrohliche Umweltreize* wirken daher offenbar bei Patienten mit SAD nicht wie phobische Stimuli. Das Arbeitsmodell zu Mediatoren sozialer Angst wurde daher im zweiten Experiment nicht bestätigt. *Im dritten Experiment* wurde untersucht, ob die verzerrte Verarbeitung *sozial bedrohlicher Umweltreize* und *selbstbezogene negative Gedanken* die Auswirkungen von sozialer Ängstlichkeit in einer sozialen Bedrohungssituation(Antizipation einer öffentlichen Rede, mediieren. Es wurde erwartet dass die experimentelle Manipulation der Intensität *selbstbezogener negativer Gedanken* (Induktion negativer Gedanken vs. Induktion von Entspannung) einen funktionalen Zusammenhang zu den vermuteten Mediatoren aufzeigen würde. Mit diesem Versuchsdesign wurde zudem untersucht, wie sich *sozial bedrohliche Umweltreize* in den unterschiedlich bedrohlichen Antizipationsphasen und je nach Grad sozialer Ängstlichkeit auswirken.
Um diese Zusammenhänge zu untersuchen, wurden Studenten im Grundstudium in einem multivariaten Ansatz untersucht, der alle Komponenten des Arbeitsmodells zu Mediatoren der sozialen Angst erfasste. Dies schloss die Erfassung von Angstsymptomen auf den drei Reaktionsebenen ein: Selbstbericht, beobachtbares Verhalten und Physiologie (Herzrate, HR) einschließlich spezifischer Indikatoren der sympathischen (Hautleitfähigkeit, SCL) vs. parasympathischen (spektrale Power der Herzratenvariabilität im Hochfrequenzband, HRV-HF) Reaktivität. Außerdem wurde erneut die affektive Modulation der Schreckreaktion durch Gesichtsausdrücke erfasst, um systematische Verzerrungen der automatischen

Informationsverarbeitung zu untersuchen. Dabei wurden emotionale Wörter und Gesichtsausdrücke mit neutralen Kontrollstimuli und der Wirkung des Kontexts verglichen.
In den Ergebnissen zeigte sich, dass die Induktion *selbstbezogener negativer Gedanken* während der Antizipation der öffentlichen Rede zu signifikant erhöhter selbstberichteter Angst und verringerter HRV-HF führte. Bei Menschen mit erhöhter sozialer Ängstlichkeit waren diese Reaktionen besonders ausgeprägt. Diese Zusammenhänge wurden vollständig durch die ebenfalls höhere Intensität *selbstbezogener negativer Gedanken* mediiert. Die Mediation erwies sich in beiden Fällen als spezifisch für soziale und unabhängig von allgemeiner Ängstlichkeit.
Des Weiteren zeigte sich, dass Versuchspersonen ihre Herzrate während der öffentlichen Rede im Vergleich zu der vom EKG abgeleiteten HR umso weniger unterschätzten, je ausgeprägter ihre soziale Ängstlichkeit war. Wenn dieser Zusammenhang um den Einfluss *selbstbezogener negativer Gedanken* bereinigt wurde, hielt er nicht stand. Dies kann als Hinweis für kognitive Mediation interpretiert werden. Im Gegensatz dazu waren Hautleitfähigkeit und Herzrate während der Antizipation der öffentlichen Rede bei allen Versuchspersonen erhöht. Das legt nahe, dass öffentliches Sprechen in den meisten Menschen gespannte Erregung auslöst. Das von Beobachtern bewertete Verhalten während der öffentlichen Rede entsprach der Selbsteinschätzung. Keines dieser Maße korrelierte jedoch mit dem Ausmaß sozialer Ängstlichkeit. Die Amplitude der Schreckreflexreaktion korrelierte zwar spezifisch mit Zustandsangst und sozialer Ängstlichkeit. Doch war sie kein Mediator für diesen Zusammenhang. *Sozial bedrohlicher Umweltreize* bewirkten außerdem erneut keine affektive Modulation der Schreckreflexreaktion.
Die Ergebnisse der drei Experimente zeigten eindrucksvoll, dass *selbstbezogene negative Gedanken* tatsächlich ein Mediator sozialer Angst sind. Im Vergleich dazu spielt die verzerrte automatische Verarbeitung *bedrohlicher sozialer Umweltreize* zumindest in ökologisch validen, sozial bedrohlichen Situationen eine vernachlässigbare Rolle.

Es konnte erstmalig die spezifische Mediation verringerter HRV-HF durch *selbstbezogener negativer Gedanken* dokumentiert werden. Dies deutet darauf hin, dass die parasympathische Kontrolle bei der Regulation emotionaler Reaktionen auf soziale Bedrohung eine wichtige Rolle spielt.

Dissoziationen zwischen Symptombereichen (Selbstbericht, Verhalten, Physiologie) sind offenbar ein charakteristisches Merkmal sozialer Angst. Dies liefert eine Erklärung, weshalb frühere Untersuchungen, in denen parasympathische Aktivität nicht erfasst wurde, nur selten physiologische Korrelate der sozialen Ängstlichkeit identifizieren konnten.

Auf der Grundlage dieser Befunde wird eine *revidierte Version des Arbeitsmodells zu Mediatoren sozialer Angst* vorgestellt. Es bietet eine strukturierte Grundlage für die Ableitung spezifischer, empirisch prüfbarer Hypothesen. Dies erlaubt eine eindeutige Interpretation komplexer Zusammenhänge auf der Grundlage eines empirisch begründeten theoretischen Konzeptes. Es besteht die Hoffnung, dass dies letztendlich zur Entwicklung eines empirisch validierten, funktional-anatomischen Modells der sozialen Angst beiträgt.

1 General Background

1.1 Social Anxiety and Social Anxiety Disorder

Social anxiety has been defined as the fear of being evaluated negatively by other individuals. Social situations typically trigger anxious apprehension in socially anxious individuals, especially when indicators for being scrutinized are present.

Social anxiety disorder (SAD) is a clinical diagnosis for individuals with extreme, persistent and disabling social anxiety. The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, APA, 2000) subsumes SAD under Axis I category „anxiety disorders“. In the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10, WHO, 1992) SAD is part of the affective disorders.

The DSM-IV-TR further suggests usage of a *generalized subtype* specifier, when fears include “most social situations” (APA, 2000, p. 451). Typically, this specifier is set when a person’s fears affect a variety of social interaction situations instead of performance situations alone (e.g. Stemberger, Turner, Beidel, & Calhoun, 1995; Turner, Beidel, & Townsley, 1992) or dependent on the number of feared social situations (for reviews, see Heimberg, Holt, Schneier, Spitzer, & Liebowitz, 1993; Hofmann, 2004).

Notably, recent studies suggest that there is no qualitative difference between individuals with social anxiety and individuals with a clinical diagnosis of SAD (e.g. Vriends, Becker, Meyer, Michael, & Margraf, 2007). In line with this view, U.S.-studies indicate that public speaking is the most commonly feared social situation in both, socially anxious individuals and the general population (Pollard & Henderson, 1988). Although it is still under debate whether Axis II category avoidant personality disorder (APD) represents a distinct entity, it has been shown that diagnostic criteria of SAD and APD are overlapping. Studies have found that both feature high levels of psychopathology, depression, poor psychosocial functioning, and high

social and trait anxiety (e.g. Turner, Beidel, & Townsley, 1992). Hence, it has been suggested to treat APD as an extreme expression of social anxiety. In sum, the current state of the debate suggests that a dimensional perspective of social anxiety is appropriate (McNeil, 2001): social anxiousness is assumed to range on a continuum from low to high levels.[1]

1.2 Epidemiology of Social Anxiety Disorder

SAD is the most common anxiety disorder and considered the third most psychological disorder in general, following major depression and alcoholism (Kessler et al., 1994). For the US, estimates of 12-month and lifetime prevalence of a DSM-IV diagnosis of SAD range between 2.8 and 7.1 %, and 5.0 and 12.1 % respectively with a 3:2 female-to-male ratio (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). However, it has been suggested that although women are more likely to have SAD, men are more likely to seek treatment, which may be explained by differences in gender roles and social expectations (Weinstock, 1999). Furthermore, differences in the particular expression of the disorder seem to vary between men and women. While eating and writing in public were more common in men, problems with using public restrooms and speaking in public were more common in women.

After an early onset with fifty percent of SAD patients reporting to remember first symptoms before age 11 and eighty percent before age 20, SAD typically takes a chronic course (Amies, Gelder, & Shaw, 1983). Therefore, comorbidity is usually considered secondary to SAD (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). Comorbidity is a common issue in more than 80 % of patients (Lepine & Pelissolo, 1996; Montgomery, 1998). An experiment with 57 patients found that comorbid diagnoses were led by lifetime depression (70 %),

[1] It should be noted that it may depend on the sensitivity of assessment tools, whether effects emerge only above a certain threshold of the intensity of trait social anxiousness. This may appear like a qualitative effect, despite the underlying continuum.

followed by panic disorder (49 %), GAD (32 %) and OCD (11 %, Van Ameringen, Mancini, Styan, & Donison, 1991). Another experiment with 123 patients found comorbid specific phobia (60.8%), agoraphobia (45%), GAD (26.9%) and lifetime panic disorder (11.6% ,Davidson, Hughes, George, & Blazer, 1993). The National Comorbidity Survey (NCS) reports comorbidity with specific phobia (37.6%), followed by major depressive disorder (37.2 %), alcohol dependency (23.9 %), agoraphobia (23.3%), posttraumatic stress disorder (15.8 %), substance dependency (14.8 %), dysthymia (14.6 %), GAD (13.3%), panic disorder (10.9%), alcohol abuse (10.9 %), and drug abuse (5.3 %, Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996). However, prevalence rates for comorbid alcoholism vary greatly (10 – 40 %) depending on definition and methods (Lepine & Pelissolo, 1998). Furthermore, most individuals with SAD also suffer from marked impairment in social and occupational domains (Hofmann & Barlow, 2002). SAD is associated with being less educated, being single, achieving a lower socio-economic status (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992), higher unemployment rates (Weiller, Bisserbe, Boyer, Lepine, & Lecrubier, 1996), more job transitions and more absenteeism (Davidson, Hughes, George, & Blazer, 1993).

Although cultural norms shape the particular presentation of SAD and prevalence rates vary across countries, SAD exists in all cultures (Heinrichs et al., 2006). Notably, the presentation of SAD is rather uniform across westernized societies. For Germany, estimates of life-time prevalence based on the DSM-IIIR, range from 11.3 % to 16.0 % (Median: 11.3 %, Perkonigg & Wittchen, 1995). In a representative survey of the German population, a 12-month prevalence of 20 % was found in the age group of 16 to 65 years (Wittchen et al., 2000). More recently, a life-time prevalence of 7 % has been reported by (Fehm, Pelissolo, Furmark, & Wittchen, 2005).

In line with the above mentioned dimensional perspective of social anxiety, subclinical social anxiety is very common. 61 % of the general population report social anxiety in singular or isolated situations. 33 % assume that their anxiety is stronger than in other individuals. 31 % think that this leads to moderate restrictions and 12 % feel severely impaired (Stein, Walker, & Forde, 1994).

1.3 Models of Social Anxiety

Although other factors also play important roles, models of social anxiety have highlighted two mediators of acute anxiety, when socially anxious individuals are confronted with social situations that are challenging to them. One is focusing on *self-related negative cognitions*; the other is biased processing of *external social threat-cues*.

1.3.1 Cognitive Models

Cognitive models have been most influential to the research on social anxiety. These models suggest that self-related negative cognitions play a key role for the development and maintenance of social anxiety. Based on features first described by Beck (1971), three types of dysfunctional cognitions have been identified at the core of social anxiety (Beck, Emery, & Greenberg, 1985): extremely high standards for social performance (“Nobody must notice my shortcomings”), conditional beliefs about social evaluation (“If I fail, nobody will accept me”) and unconditional beliefs about the self (“I’m a failure”). Later, cognitive models have extended and refined this description of cognitive features and processes (e.g. Leary & Kowalski, 1995; Reiss, 1991). Clark and Wells (1995, see Figure on p. 71 of the book) have proposed the first cognitive model specifically for social anxiety. It emphasizes the importance of an inward attentional shift, occurring when socially anxious individuals believe they are being scrutinized. Consequently, socially anxious individuals engage in a process of detailed self-monitoring, focusing on thoughts and imagery about past and expected failure in social situations (Beidel, Turner, & Dancu, 1985; Cacioppo, Glass, & Merluzzi, 1979;

Glasgow & Arkowitz, 1975; Glass, Merluzzi, Biever, & Larsen, 1982; Hope, Gansler, & Heimberg, 1989; Ingram, 1990; Spurr & Stopa, 2002; Stopa & Clark, 1993). Socially anxious individuals believe that these images are an accurate representation of their performance (Hackmann, Clark, & McManus, 2000; Hackmann, Surawy, & Clark, 1998). Increasing anxiety leads into subsequent avoidance behaviors and the application of safety behaviors. As a result, untested assumptions about expected standards and one's own performance in social situations further lead into a vicious circle of avoidance and self-fulfilling prophecies, nurturing the development and maintenance of SAD. Cognitive models have received a vast amount of empirical support see section 1.3.5, p. 33. However, no study has demonstrated experimentally that anxiety in anticipation of a socially threatening situation is cognitively mediated - one of the core assumptions of the cognitive model. As a result, some authors have recently questioned the cognitive mediation model of social anxiety (e.g. Longmore & Worrell, 2007).

Cognitive models have also argued that anxiety disorders may be associated with preferential automatic allocation of attentional resources to threatening cues (Beck, Emery, & Greenberg, 1985; Eysenck, 1992; Williams, Watts, MacLeod, & Mathews, 1997). This may trigger negative affect and has been suggested to contribute to the maintenance of social anxiety because of an association with habitual hyperarousability by disorder relevant threat-cues. Furthermore, these mechanisms have been suggested as a major route for developing SAD, due to an increasing number of false alarms to social stimuli (Bitran & Barlow, 2004).

1.3.2 Information Processing Models

The role of information processing biases has been addressed specifically in models based on the assumption that preferential processing of threatening environmental information is generally advantageous for survival. For example, the influential model by Öhman (1986) suggested that rough parallel resource-independent processing of basic stimulus features via a

so called feature detector directly activates the arousal system when an environmental stimulus signals acute threat. This is thought to lower the threshold for the detection of significant threat in the environment via a more elaborate route of processing. This slower, controlled route evaluates whether the initial response is appropriate and may lead to conscious perception of threat. Heritable preparedness for preferred processing of certain stimuli together with individual learning experiences may lead to a general or context dependent sensitization towards specific threatening cues. In the case of SAD, a specific vulnerability for preferential processing of (potentially) socially threatening environmental cues has been suspected. According to the model, individuals with SAD have a lower threshold for detecting external social threat-cues and interpret them more negatively. Therefore, ambiguous cues which would be neutral for most individuals may also induce social anxiety in individuals with SAD (see review by Heinrichs & Hofmann, 2001).

In this context, it is valuable to consider the time course of processing environmental threat-cues. Early models predicted a simple hyper vigilance towards threatening environmental stimuli (Beck & Clark, 1997; Beck, Emery, & Greenberg, 1985; Mathews & MacLeod, 1994). However, others predicted avoidance of external social threat-cues (Foa & Kozak, 1986). This conflict has been resolved in the hyper vigilance avoidance hypothesis (Mogg, Bradley, De Bono, & Painter, 1997; Mogg, Mathews, & Weinmann, 1987). In socially anxious individuals, preferential automatically processing of (potentially) socially threatening stimuli is thought to dominate the first few hundred milliseconds after stimulus onset. At later stages, when the influence of deliberative processes increases, threatening stimuli are avoided, if possible. Finally, there is empirical support for the so called specificity hypothesis, suggesting that socially anxious individuals feature information processing biases that are specific to external social threat-cues (Mathews & MacLeod, 1987).

Interestingly, Öhmann's model (1986) has been linked to a neural basis (Lang, Davis, & Öhman, 2000). In this view, fast automatic threat-detection matches Le Doux`s (1996; 2000) *low road v*ia the thalamus, activating the amygdala without conscious processing. Extensive studies in both animals (e.g. Davis & Shi, 2000) and humans (e.g. Adolphs, 2002) have confirmed the crucial role of the amygdala in processing environmental threat-cues. Brain imaging studies have further corroborated this view (e.g. Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Straube, Kolassa, Glauer, Mentzel, & Miltner, 2004; Vuilleumier, Armony, Driver, & Dolan, 2001). However, it has been shown that attention may moderate these effects (Bishop, Duncan, & Lawrence, 2004; Pessoa, Kastner, & Ungerleider, 2002). Nonetheless, tasks with subliminally presented stimuli (see Öhman, 2005) have demonstrated that information may directly activate affective reactions and behavioral tendencies without requiring attention. In line with this notion, socially anxious individuals showed increased amygdala activation during presentation of angry facial expressions in particular, when they focused their attention away from the emotion on display (Stein, Goldin, Sareen, Zorrilla, & Brown, 2002; Straube, Kolassa, Glauer, Mentzel, & Miltner, 2004; Whalen et al., 1998). Importantly, focusing attentional resources on internal information such as self-related negative cognitions as suggested by the cognitive model may not necessarily imply reduced capacity for these automatic processes (Panayiotou & Vrana, 1998).

These concepts have also been linked to Lang's fear network model (e.g. Lang, 1979, 1994). According to this view, the so-called primary motivational system may be activated via the *low road*. Threat-cue vulnerability could therefore directly mediate anxiety via automatic activation of this system.

However, the fear network model also suggests that the primary motivational system is linked to higher semantic affective information processing networks. Neuroscientific research has shown that the subcortical brain regions involved in establishing and maintaining emotional

responding partly depend on cognitive resources (Pessoa, McKenna, Gutierrez, & Ungerleider, 2002) and interact with higher-order neural systems (Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001; Lang, 1995). Therefore, automatic processing of external social threat-cues may also trigger negative cognitions via spreading activation within semantic networks. This way, anxiety could be mediated indirectly, when threat-cue vulnerability triggers negative cognitions, which in turn leads to anxiety. In the latter case, effects would match predictions of the cognitive model, but automatic processing of threat-cues would mediate or at least moderate these effects.

Finally, both cognition and threat-cue vulnerability could act independently, affecting different aspects of emotional responding (experience, behavior, and physiology). Lang's model suggests that the implicit *low-route* may particularly affect autonomic reactivity and behavioral tendencies unconsciously, while negative cognition always entails conscious information processing.

1.3.2.1 Focus: The Multi-Process Account of Startle Modulation during Affective Perception

The startle reflex is a whole body response to sudden intense stimulation like a loud burst of noise, a flash of light or a sudden tactile excitation. The first sign of this response is a fast closure of the eye, which can be quantitatively assessed via EMG of the orbicularis oculi, see Figure 1. The amplitude of the integrated EMG has been shown to vary with a number of aspects such as sensory, attentional, and emotional processes.

Therefore, pictures may act as prepulses for acoustic startle. When the startle probe is presented early after picture onset (e.g. 300ms), PPI is stronger with more arousing stimuli (Bradley, Cuthbert, & Lang, 1993). This stage of stimulus processing is considered automatic and cannot be completely suppressed.

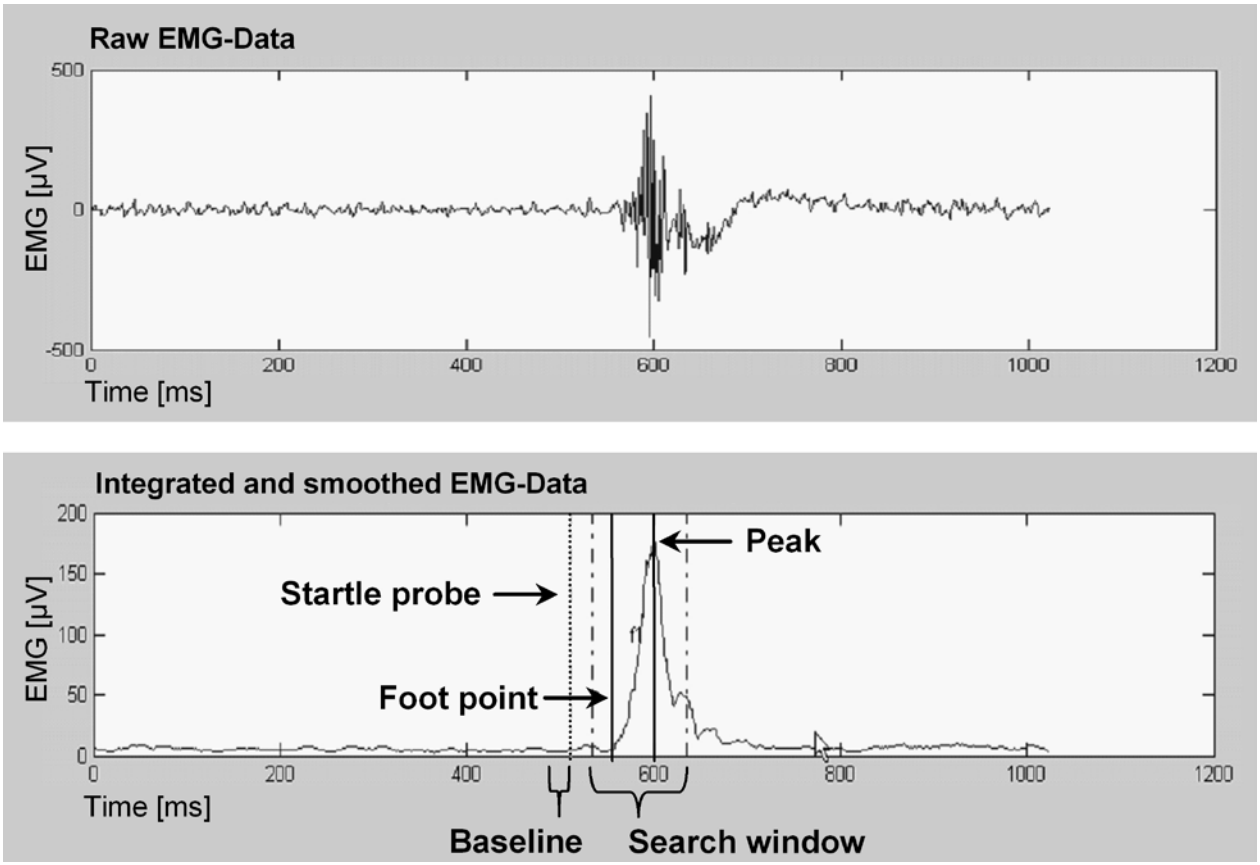

Figure 1: Example for the EMG of a startle eye-blink, recorded from sites over the left orbicularis oculi. The upper panel shows the raw signal. In the lower panel the integrated and smoothed signal is shown. *Startle probe* marks the begin of the 50 ms 100 dB(A) white noise probe, used to elicit startle reactions. The maximum of the integrated signal (*Peak*) minus the mean over a baseline of 20 ms before the onset of the startle probe is used to determine the startle amplitude (peak *search window* limits marked with dashed lines). The time from the onset of the startle probe to the *foot point* of the response is used to determine the latency of the response. The sample figure reproduces parts of the graphical user interface of the Matlab toolbox (Schulz & Alpers, 2007) used for pre-processing and scoring of EMG responses in the experiments reported below.

Extensive animal research (Davis & Shi, 2000; Davis, Walker, & Lee, 1997) and matching findings in humans with amygdala lesion (Angrilli et al., 1996) suggest that the neural basis of this reflex consists of a primary spinal circuit showing habituation. A secondary circuit

involving the central nucleus of the amygdala may be activated repeatedly without habituation and modulates the startle response.

This view matches the above-mentioned notion of a *low road* of information processing (LeDoux, 1998) and Öhman's (1986) feature detector and suggests that threat-cues are evaluated automatically (i.e. rapid, resource independent and without requiring conscious processing).

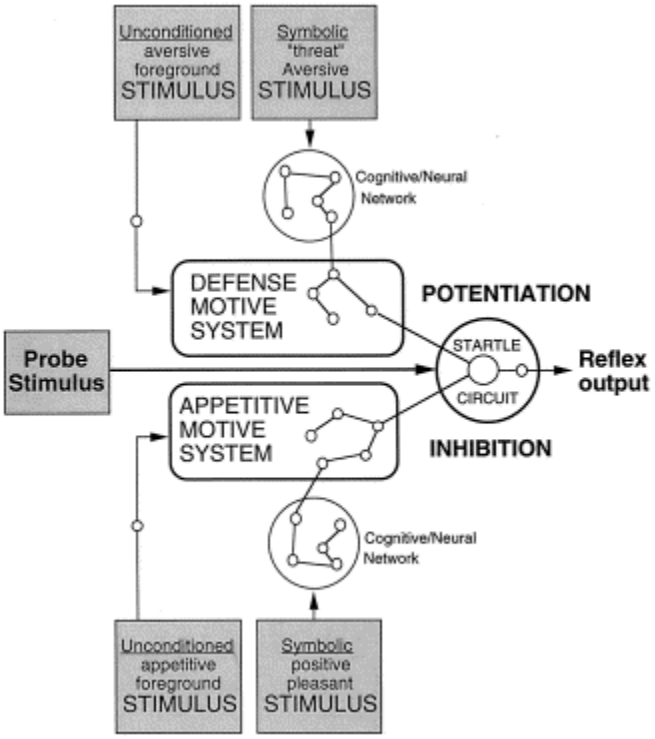

Figure 2: "Startle reflex priming. Probe stimuli (acoustic, auditory, or tactile) activate the pontine startle circuit, resulting in startle reflex output. When previous aversive unconditioned or symbolic stimuli activate the defense motive system, its direct projections to the startle circuit potentiate (augment) this reflex response. Alternatively, when the appetitive motive system is active, its projections to the startle circuit result in reflex inhibition. [Overlap within of cognitive neural networks and the two motive systems allows for interaction of bottom-up priming and top-down cognitive control.]" (reprinted with permission from Lang, Bradley, & Cuthbert, 1998, p. 1255).

Lang's (1994) motivational network model further differentiates systems for defensive (Lang, Bradley, & Cuthbert, 1997) and appetitive motivation (see Bradley, Cuthbert, & Lang, 1999) for negative and positive stimuli, respectively. Importantly, this model further suggests that upstream priming of conscious cognition may be possible because the representation of encoded information within networks involves nodes of these primary motivational systems. Similarly, top-down effects from the conscious *high road* of information processing appear possible, when spreading activation progresses to primary motivational systems; see Figure 2. Given this background, Bradley, Codispoti and Lang (2006) integrated results from several studies into a concise model describing the interaction of modulating factors in their multi-process account of startle modulation during affective perception.

When a non-startling stimulus is presented at various temporal delays before a startling probe, the blink magnitude is inhibited for up to approximately 500 ms, with peak inhibition around 150 ms after prepulse onset (Anthony & Graham, 1985). This so called prepulse inhibition (PPI, Graham, 1975) is thought to represent a low-level sensory gating mechanism, protecting selective stimulus processing (for reviews on PPI see Hackley & Boelhouwer, 1997; Thorne, Dawson, & Schell, 2005). Importantly, PPI works across different sensory modalities.

In general, PPI at short lead intervals (< 250 ms) is affected by automatic (pre-attentive) mechanisms, but at 240 ms, effects of attention allocation were significant (Heekeren, Meincke, Geyer, & Gouzoulis-Mayfrank, 2004). This may indicate that attention only affects PPI when there is sufficient time for implementing attentional mechanisms. However, sustained attentional focus may not require time to exert an influence on PPI. Instructed focus of attention as well as task relevance has shown to modulate prepulse inhibition (e.g. DelPezzo & Hoffman, 1980; Hackley & Graham, 1987; Ison & Ashkenazi, 1980). Attending the prepulse increases PPI (Filion, Dawson, & Schell, 1993). Consequently, effects of attentional focus, and the attention capturing effect of stimuli may overlap. For example, a

sustained inward attentional focus away from external visual stimuli used as prepulses (e.g. when socially anxious individuals are confronted with a socially challenging situation) may decrease PPI.

On the other hand, the automatic attention grabbing effects of emotional stimuli may counteract the PPI reducing consequences of attentional focus, thus increasing startle probes elicited at short lead intervals after picture onset. In line with this view, a recent study has shown that associations between PPI and cognition are mediated by attentional processes (Scholes & Martin-Iverson, 2009).

Affective modulation of the startle reflex has been identified as an independent but overlapping process with larger blinks elicited in aversive contexts (e.g. Greenwald, Bradley, Cuthbert, & Lang, 1998) and when aversive foreground stimuli are processed (e.g. unpleasant pictures, Vrana, Spence, & Lang, 1988). Pleasant visual stimuli reduce the startle reflex (Bradley, Cuthbert, & Lang, 1990; Cuthbert, Bradley, & Lang, 1996; Simons & Zelson, 1985). The influence of stimulus valence begins to emerge at around 500 ms after prepulse onset and increases further at least for the following 4-5 s and most likely even beyond 12 s (Smith, Bradley, & Lang, 2005; Sutton, Davidson, Donzella, Irwin, & Dottl, 1997).

In addition to startle amplitude, other parameters such as startle magnitude (i.e. amplitudes without baseline correction) and response probability have been examined, but play a minor role in the current literature. Another parameter which is less often examined than startle amplitude is the latency of startle-responding. Anatomically, this effect is sent from the nucleus reticularis pontis caudalis via the facial nucleus along the seventh (facial) cranial nerve, which innervates the orbicularis oculi muscle, causing the eye to blink. In the context of social anxiety, this measure is interesting, because it has been identified as the most sensitive indicator of the motor component of the startle response (Britt & Blumenthal, 1991; Graham & Murray, 1977; Leitner, Powers, Stitt, & Hoffman, 1981; Silverstein, Graham, &

Bohlin, 1981). In particular, shorter startle latencies have been interpreted as an indicator of hyper-responsivity of the motor system, linked to the clinical presentation of social anxiety disorder (see discussion in Britt & Blumenthal, 1993).

Importantly, recent research has suggested that startle modulation may be more specific than suggested by Lang's valence model. For example, startle attenuation primarily confirmed for strong positive stimuli depicting erotica (Lass-Hennemann, Schulz, Nees, Blumenthal, & Schachinger, 2009). Interestingly, gaze direction moderated this effect in line with the findings of Ivanonva and Allen (2001): When gaze was directed at the observer, startle eye-blink inhibition to the erotica was diminished. In contrast to the interpretation of Ivanonva and Allen (2001), this suggests that gaze direction may attract increased attention to the face. Furthermore, in the absence of sensory engagement, both positively and negatively valenced arousal increases startle amplitudes (Witvliet & Vrana, 1995). Only sufficiently arousing emotional stimuli evoke a motivational response involving appetitive or aversive behavior tendencies. For example, Cuthbert, Bradley and Lang (1996) recommend that the arousal-score assigned via SAM-ratings in the published norms of IAPS pictures should be 6 or higher (on a 9-point scale with 0 = not arousing vs. 9 = extreme arousing) to elicit affective startle modulation reliably.

Furthermore, startle modulation has been suggested to vary with the activation of systems for defensive (Lang, Bradley, & Cuthbert, 1997) or appetitive motivation (see Bradley, Cuthbert, & Lang, 1999). External cues or internal associations may activate these systems via fear memory networks. Lang (1994) has proposed that these networks may vary in associative strength across diagnoses and anxiety disorder spectrum theory has proposed a particular order following a continuum of increasing associative strength, which is associated with increasing specificity and robustness of startle potentiation: generalized anxiety disorder, agoraphobia and panic, SAD, and specific phobia (Lang, McTeague, & Cuthbert, 2005).

Finally it should be noted that attentional and affective modulation of the startle reflex are overlapping processes. Therefore summative effects may occur across the time course of information processing.

1.3.3 Integrated Models

Rapee and Heimberg (1997; Turk, Lerner, Heimberg, & Rapee, 2001) have merged aspects of cognitive, evolutionary, and information processing models. Their integrated approach also predicts that acute social anxiety emerges when a socially anxious individual enters a socially threatening situation. However, according to their model, anxious responding is not an effect of the situation per se, but is mediated by both negative cognition and biased processing of socially threatening cues, see Figure 3. Perceiving the audience directly triggers a (negative) mental representation of the self as seen by the audience. External indicators of negative evaluation further bias this mental representation. Once a negative self-image is activated, comparison against expected social standards leads to the assumption of a high risk for negative evaluation with severe consequences. This leads to behavioral, cognitive and physiological anxiety symptoms. This model is in line with the proposed assumption of an indirect effect of threat-cue vulnerability on negative cognition; see Figure 2, p. 26. However, as noted before, it remains to be tested whether threat-cues mediate negative cognitions or if they also have direct effects on anxiety symptoms. Furthermore, this model does not differentiate between automatically processed environmental information and conscious perception.

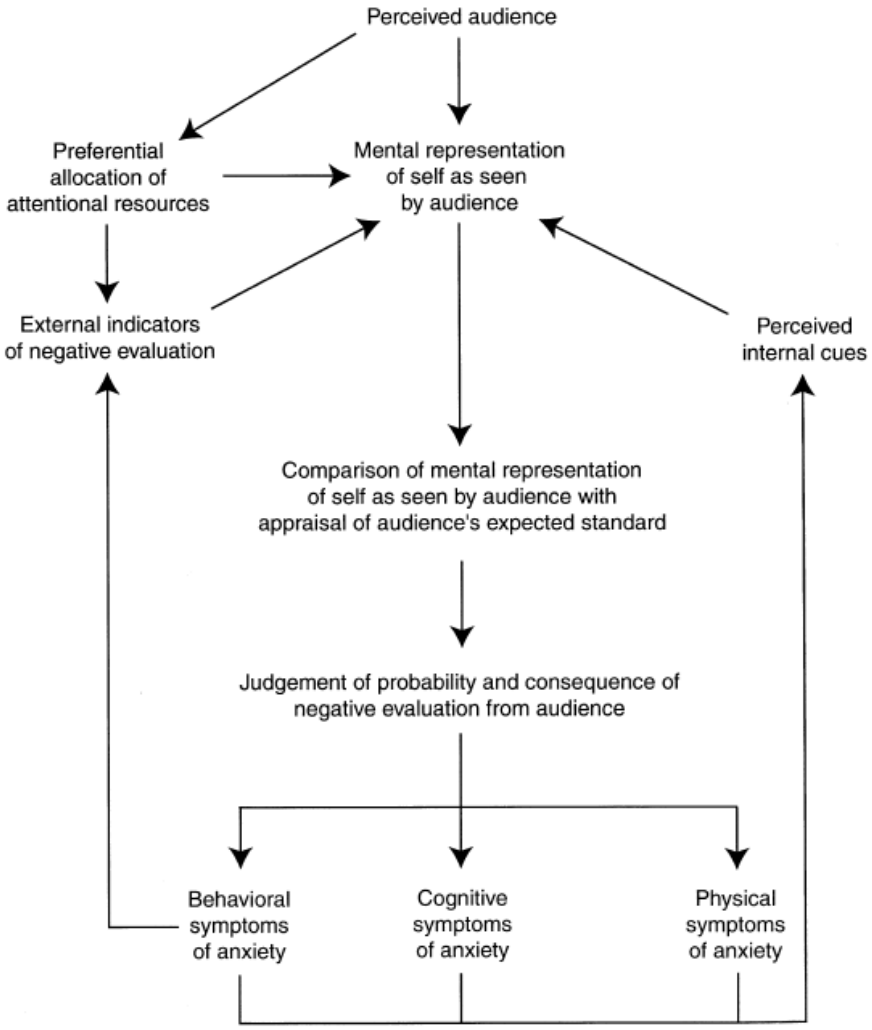

Figure 3: „A model of the generation and maintenance of anxiety in social/evaluative situations" (reprinted with permission, from Rapee & Heimberg, 1997, p. 743)

1.3.4 An Empirically Validated Model

Specific mediators and moderators of social anxiety have been identified by experimental variation of specific factors. A model recently proposed by Hofmann (2007) summarized several factors, which are presented as part of a psychological maintenance model of SAD, see Figure 4. Unrealistic social standards and a deficiency in selecting attainable social goals lead to problematic responding to socially challenging situations. Rather than perceiving reality, individuals with SAD shift their attention inward and use their negative self-perception as a source for judging the situation. They overestimate negative consequences of

their social performance and believe that they have little control over their emotions. Viewing their social skills as inadequate, they rather avoid the situation or apply safety behaviors. Finally, post-event rumination maintains future social apprehension. Despite the number of studies showing information processing bias in socially anxious individuals (see section 1.3.6, p. 38), this model has not included this pathway as a relevant factor for the elicitation of social apprehension. Furthermore, negative cognition is only indirectly addressed as an attentional shift leading into anticipatory factors that predict avoidance and safety behaviors. Finally, the role of physiological effects has not been addressed in this model, presumably due to the lack of convincing findings on specific differences between individuals with social anxiety and healthy controls; see section 1.3.5, pp. 33.

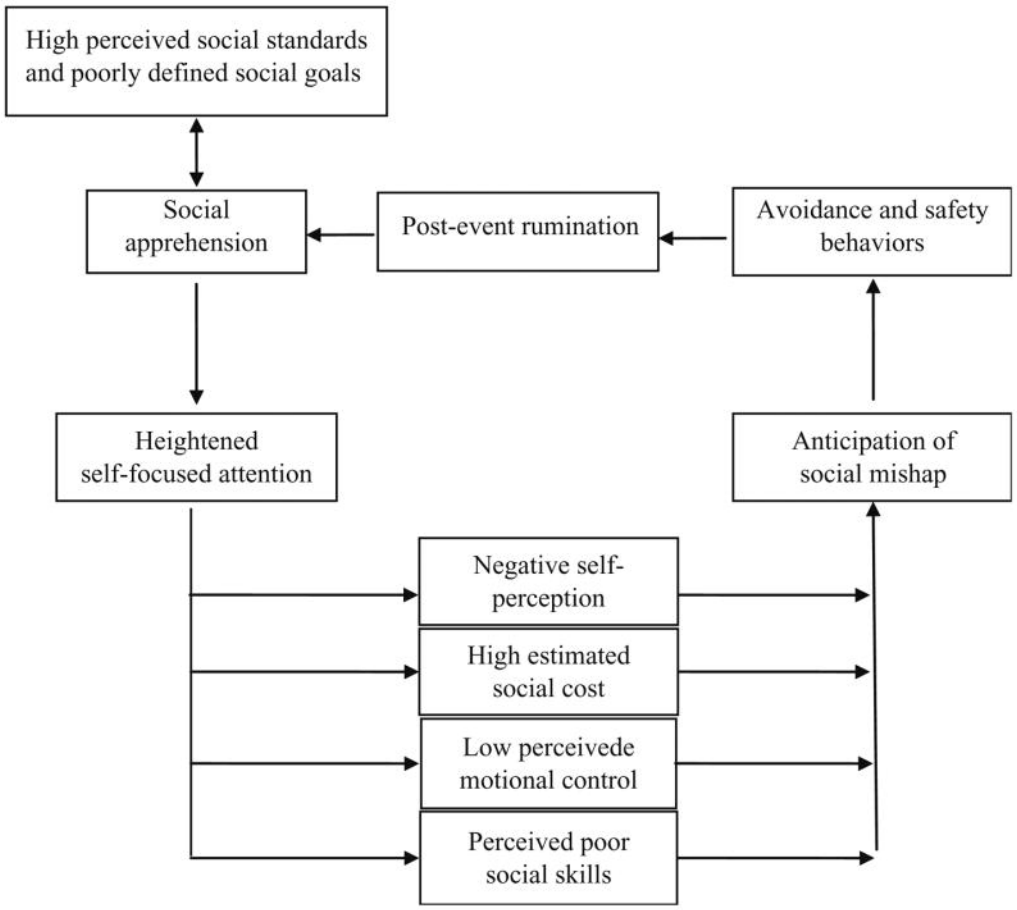

Figure 4: „Psychological factors that maintain social anxiety disorder" (reprinted with permission from Hofmann, 2007).

1.3.5 Empirical Support for Mediation of Social Anxiety by Self-Related Negative Cognitions

Self-report data has been reported in most studies on social anxiety: Socially anxious individuals report more negative and self-deprecating cognitions in socially threatening situations than healthy individuals (Clark & Wells, 1995; Hackmann, Clark, & McManus, 2000; Hackmann, Surawy, & Clark, 1998; Stopa & Clark, 1993). When being confronted with social threat, socially anxious individuals typically monitor their internal state, and experience anxiety (Hofmann & Barlow, 2002; Spurr & Stopa, 2002; Woody, 1996). Successful treatment is associated with decreased self-focused attention (Hofmann, 2000; Wells & Papageorgiou, 1998; Woody, Chambless, & Glass, 1997), which is correlated with changes in self-reported social anxiety, especially among individuals who receive cognitive behavioral interventions (Hofmann, Moscovitch, Kim, & Taylor, 2004). When healthy individuals anticipate a public speaking task with a thought protocol imitating the cognitive style of socially anxious individuals, the participants' self-reported anxiety is comparable to that of high socially anxious individuals (Hinrichsen & Clark, 2003; Vassilopoulos, 2005). Finally, when healthy controls rehearsed negative self-imagery, as compared to positive or control images, they reported higher levels of anxiety, believed that they performed worse, and reported more negative thoughts, as compared to groups rehearsing a positive self-image or a control image. Interestingly, inter-individual variations in state anxiety did not explain these associations. Therefore, negative self-imagery may be causally linked to anxiety (Hirsch, Mathews, Clark, Williams, & Morrison, 2006).

Furthermore, cognitions may impact social anxiety via context dependent set-points. In comparison to observer ratings, patients with SAD rated themselves as even more impaired in a social communication task. This bias was not present in healthy controls (Stopa & Clark, 1993) or patients with dysthymia (Norton & Hope, 2001). Importantly, it has been shown that

cues indicating that standards for performance are low alleviated this habitual bias in individuals with SAD (Moscovitch & Hofmann, 2007).

These findings are consistent with cross-sectional studies suggesting that cognitive variables, such as self-focused attention (Kashdan & Roberts, 2004), perception of emotional control (Hofmann, 2005) and evaluation of one's own performance (Perini, Abbott, & Rapee, 2006) are causally related to social anxiety. Finally, it has been shown that cognitive evaluation of the estimated cost of social mishaps partially mediates therapy success in patients with SAD (Hofmann, 2004).

The *behavior* of socially anxious individuals has been investigated via observer ratings using both free interaction with social partners and specifically tailored tasks simulating social interactions. Poor performance in these more or less standardized situations is widely used for diagnostic assessment of the severity of social anxiousness (Schulz, Meuret, Loh, & Hofmann, 2007, pp. 231 for an overview of methods). Typical symptoms used as target indicators include "entering feared social situations, degree of eye contact with an audience or individual cohort, or frequency, and duration of pauses in a conversation" (Schulz, Meuret, Loh, & Hofmann, 2007, p. 231). It should be noted that lack of or reduced behavior, like going blank in a speech, can be even more clinically informative (Marks, 1987).

In standardized speaking tests, observers rated the performance of individuals with SAD as more negative and less positive than that of controls (Stopa & Clark, 1993) or generally worse (Moscovitch & Hofmann, 2007). However, others did not find differences between observer ratings for the performance of individuals in a brief impromptu speech by individuals with SAD vs. non-anxious controls (e.g. Rapee & Lim, 1992). Increased self-focused attention has been shown to induce objectively impaired performance in individuals with SAD (Hope & Heimberg, 1988). However, others have failed to replicate this finding (Woody, 1996). Interestingly, it has been demonstrated that socially phobic individuals were able to drop

safety behaviors during public speaking with appropriate instructions (Wells, Clark, Salkovskis, Ludgate, & et al., 1995). Similar cognitive strategies improved not only the behavior but also reduced subjective levels of anxiety and perceived social threat in socially phobic individuals (Garcia-Palacios & Botella, 2003; Morgan & Raffle, 1999).

Since avoidance and security behavior is affected by self-related negative cognitions, the effect of behavioral symptoms of SAD emerge indirectly via as indicated by lower incomes, smaller social networks, being married less often and lower status in social hierarchies (Schneier et al., 1994; Wittchen et al., 2000; Wittchen, Stein, & Kessler, 1999).

Physiological indicators of social anxiety such as racing heart, blushing, sweating palms, shortness of breath are often reported by socially anxious individuals (Amies, Gelder, & Shaw, 1983) (Gorman & Gorman, 1987). Nonetheless, evidence for physiological correlates of trait social anxiousness is quite limited (reviewed by Dewar & Stravynski, 2001; and Hofmann, Heinrichs, & Moscovitch, 2004). Five major strategies have been pursued in the quest for identifying biological correlates of SAD:

First, pharmacological treatments effectively reduce symptoms of SAD, including cognition (Gorman & Gorman, 1987). However effects of drugs as diverse as serotonin reuptake inhibitors, monoamine oxidase inhibitors, and benzodiazepine modulators of the inhibitory neurotransmitter, gamma-aminobutyric-acid (GABA) are almost equipotent. Probably, they lead to „functional improvement without actually influencing the underlying neurobiological deficit, but rather by altering associated systems acting in parallel" (Dewar & Stravynski, 2001, p. 248). This limits inference on specific mediators of treatment success.

The second strategy is a logical consequence of the first one. Assessment of neurotransmitters and hormones as well as their responses to pharmacological and physiological challenge have identified adrenergic, serotonergic, and benzodiazepine functioning as potential target systems. However, so far no clear-cut association with SAD has been established. Potential

exceptions are enhanced CO_2- and pentagastrin-induced panic in socially anxious individuals (see Dewar & Stravynski, 2001). However, potential interactions with cognitive processing in SAD have not been investigated so far.

The third approach has focused on the autonomic nervous system, assessing potential indicators of social anxiety such as heart rate, blood pressure, electro-dermal activity, heart rate variability, or respiration in order to draw conclusions on presumed underlying processes. The bottom line of these studies is that there are no baseline-differences between socially anxious individuals and healthy controls. A socially threatening context or task typically elicits a strong sympathetic response, usually interpreted as task-induced distress. However, healthy controls respond in a similar range, hence general situational demand rather than social anxiety appears to drive these effects (Erdmann & Baumann, 1996; Schwerdtfeger, 2004). Excellent examples of this research are published by Mauss and colleagues (Mauss, Wilhelm, & Gross, 2003; Mauss, Wilhelm, & Gross, 2004; Westberg, Lundh, & Jonsson, 2007). Furthermore, the role of the parasympathetic branch of the nervous system has been discussed. However, findings with a suspected sub-type of SAD with a prominent blushing response to social threat suggest a stronger link to specific situational parameters (e.g. shame inducing tasks), rather than a general link to social anxiety (e.g. Gerlach, Wilhelm, & Roth, 2003). Nonetheless, there is little research on this topic. Importantly, recent research suggests that cognitive emotion regulation strategies interact with the brain-body control of parasympathetic regulation. Therefore, associations between trait-social anxiousness and self-related negative cognitions may emerge in vagal reactivity to social threat.

A fourth approach relies on the measurement of brain functioning using EEG and brain imaging techniques. The huge technical advance of the last years has led to increasing success in identifying cortical networks involved in social anxiety (see overview in Cannistraro & Rauch, 2003). Probably unsurprisingly, most of the reported brain areas are involved in

emotional information processing and are part of brain structures involved in emotion regulation (Thayer & Lane, 2000). Although it seems premature to derive clear-cut predictions whether specific systems mediate social anxiety, cognition may play an important role via emotion regulation of subcortical activity. Interactions between the medial prefrontal cortex, amygdale, insula, thalamus, and hippocampus are likely to play an important role. To discover physiological correlates of cognitive mediation of SAD may therefore require a „brain-body approach".

Finally, behavioral genetics have added a fifth perspective. Twin and family studies suggest a genetic vulnerability underlying social anxiousness (e.g. Kendler, Neale, Kessler, Heath, & Eaves, 1992; Lieb et al., 2000; Mancini, van Ameringen, Szatmari, Fugere, & Boyle, 1996; Nelson et al., 2000). Chances to develop SAD are 2 to 3 times higher for first degree relatives heritability has been estimated at around 30 % to 40 %. Interestingly, SAD and APD seem to share common genetic, but independent environmental influences (Reichborn-Kjennerud et al., 2007), a finding that further strengthens the above mentioned dimensional perspective on social anxiousness. Triggered by the pharmacological approach, symptoms of SAD have also been linked to a genetic basis (see Saudino, 2001, for an overview). Furthermore, there is indirect support for a shared genetic basis for personality variables (e.g. increased harm avoidance, reduced novelty seeking, self-directedness, and reward dependence, see Lochner et al., 2007) and the expression of SAD (e.g. Chatterjee, Sunitha, Velayudhan, & Khanna, 1997; Kim & Hoover, 1996; Marteinsdottir, Tillfors, Furmark, Anderberg, & Ekselius, 2003; Pelissolo et al., 2002). However, compared to environmental factors, genetic influences appear to play a minor role and no specific behavioral, physiological, or self-report indices of SAD have been linked to a certain gene. So far, interactions between genetic variations and self-related negative cognitions have not been investigated.

In sum, there is a wealth of evidence that self-related negative cognitions play an important role in SAD. However, despite subjective reports of somatic symptoms, there is little evidence for the role of physiological factors. Notably, no experiment has shown whether self-report, behavioral, or physiological symptoms of acute anxiety are mediated by cognition when socially anxious individuals are confronted with a socially threatening situation.

1.3.6 Empirical Support for Preferential Processing of External Social Threat-Cues in Social Anxiety

Self-report data on information processing biases is rare. This may reflect the assumption that they involve automatic processing largely without conscious awareness. However, in clinical interviews patients report that they avoid eye contact and directly looking into the face of interaction partners or people in an audience (see overview in Schulz, Meuret, Loh, & Hofmann, 2007). Notably, individuals with SAD are often not aware of these behaviors in everyday situations. Individuals with high vs. low levels of social anxiety also detected more negative audience behavior in a signal detection paradigm, where audience members were trained to show positive and negative evaluative facial signals (smiles and frowns) at irregular intervals while the participant presented a public speech (Veljaca & Rapee, 1998). Finally, systematic variation of audience behavior (friendly vs. offensive) has been shown to affect social anxiety symptoms (e.g. Pertaub, Slater, & Barker, 2002). Interestingly, when patients with SAD are specifically instructed to focus their attention externally and to notice salient social cues in their environment such as other people's reactions to them, they report significant reductions in social anxiety and negative beliefs (Wells & Papageorgiou, 1998).
Reaction times, which may be seen as a special case of *observable behavior*, have been used commonly as an indicator for information processing biases. Although there are also some published null-results (e.g. Horenstein & Segui, 1997) (Bradley et al., 1997; Kolassa & Miltner, 2006), numerous studies support the notion that socially anxious individuals have an

attention bias for socially threatening cues (for reviews see Heinrichs & Hofmann, 2001; and Hirsch & Clark, 2004). In a typical *Stroop task*, participants are required to ignore the content of stimuli but react for example to the color of words. Increased response latencies are interpreted as an index of the attention-grabbing power of task-irrelevant features such as social threat conveyed by the meaning of a word. As expected, reaction times in most Stroop tasks were slower in trials with socially threatening words (e.g. Hope, Rapee, Heimberg, & Dombeck, 1990; Mattia, Heimberg, & Hope, 1993). Stroop tasks have been criticized, because attention and response bias are confounded. The *dot-probe task* provides a cleaner measure of attention bias. In this task, two stimuli of competing salience are presented simultaneously. At stimulus offset, a dot is presented at the former location of one of the stimuli. Initially it has been assumed that gaze orienting drives this effect, but visual scan path studies have disconfirmed this view (Bradley, Mogg, & Millar, 2000). Nonetheless, response latencies in the dot-probe task are commonly interpreted as an index of attention allocation at the time of stimulus offset. At least three studies support a specific attentional bias of individuals with social anxiety in the dot-probe task, comparing neutral to social and physical threat words (Asmundson & Stein, 1994a; Lundh & Öst, 1996; Mogg, Philippot, & Bradley, 2004). A similar bias has been found with negative facial expressions (Ononaiye, Turpin, & Reidy, 2007; Pineles & Mineka, 2005).

Notably, one study demonstrated that attention bias was specifically associated with social and not general trait anxiety (Mogg & Bradley, 2002). In line with the above mentioned dimensional perspective on social anxiety, similar effects were found in individuals with subclinical levels of social anxiety, with both words (Mansell, Ehlers, Clark, & Chen, 2002) and facial expressions (Pishyar, Harris, & Menzies, 2004).

However, under high anxiety conditions (e.g. induced with a public speaking paradigm), opposite effects have been reported in socially anxious individuals (Amir, Freshman, & Foa,

2002; Mansell, Clark, Ehlers, & Chen, 1999; Yuen, 1994). The authors speculated that socially anxious individuals may be better in overriding attentional bias in such situations, because they are experts for the respective stimulus material. Furthermore, the time course of information processing may be an important moderator of effects. While Yuen (1994) used a 1000 ms delay between stimulus offset and a cue (i.e. the dot) that triggered the response in a dot probe task, others have used 500 ms (Bradley et al., 1997). However, Mansell, Clark, Ehlers and Chen (1999) compared both delay intervals and replicated effects of Yuen (1994) also with the shorter time interval.

It was further shown that once attention has been allocated, patients with SAD demonstrate greater difficulty disengaging from external social threat-cues than do non-anxious controls (Amir, Elias, Klumpp, & Przeworski, 2003). On the other hand, some data suggest that when given the option to attend to objects rather than faces, patients with SAD will attend to objects (Chen, Ehlers, Clark, & Mansell, 2002), particularly under conditions of threat of social evaluation (Garner, Mogg, & Bradley, 2006; Mansell, Clark, Ehlers, & Chen, 1999), though there has been a recent failure to replicate this finding (Sposari & Rapee, 2007).

In the so called *face in the crowd paradigm*, participants have to press a response button as soon as they detect for example an angry face in a matrix of neutral or happy faces. Highly socially anxious individuals were faster detecting angry facial expressions in this task (Gilboa-Schechtman, Foa, & Amir, 1999). Notably, at least for small matrices, the results with this task suggest parallel information processing, which is in line with Öhman's model (1986), see section 1.3.2 p. 21. Another experiment reported that physical stimulus features had to be highly controlled to replicate this effect (Schmidt-Daffy, 2006), suggesting that the effect is not very robust under real life conditions. Finally, others replicated the basic effect, but found no relationship to social anxiety (Gilboa-Schechtman, Foa, & Amir, 1999; Juth, Lundqvist, Karlsson, & Öhman, 2005).

Reaction time studies share a number of limitations. Most importantly, tasks for identifying information processing biases involve a highly standardized environment with limited ecological validity. Furthermore, these studies typically assume that effects in reaction times are caused by specific processes. However, consciously and unconsciously applied strategies may vary across individuals and even across trials of the same experiment. It is usually not possible to detect these influences from the results of reaction time tasks.

Investigations of the *visual scan path* have the advantage of assessing behavior continuously. Interestingly, when socially anxious individuals had no alternative to looking at facial expressions, they still tended to avoid emotionally meaningful areas such as the eye region. Furthermore, they demonstrated a characteristic pattern of over-scanning neutral facial areas (Horley, Williams, Gonsalvez, & Gordon, 2003). However, this experiment used sad faces only, but no angry faces. In a study comparing the visual scan-path of high vs. low socially anxious individuals to angry, happy and neutral facial expression, high FNE participants initially looked more often at emotional faces, both happy and angry ones (Wieser, Pauli, Weyers, Alpers, & Mühlberger, 2009). Another eye-tracking study using dynamic facial expressions with gaze averted or directed at the participants confirmed that gaze, directed at the observer, is a social threat-cue for socially anxious individuals, as indicated by stronger cardiac acceleration. However, rather than avoiding, high socially anxious individuals tended to fixate the eye-region longer (Wieser, Pauli, Alpers, & Mühlberger, 2009). Finally, induced social anxiety via anticipation of public speaking affected attention to angry vs. happy facial expressions in virtual reality. All participants attended more to happy facial expressions. However, increased trait social anxiousness was associated with stronger initial avoidance of emotional facial expressions. Participants who expected to give a speech were more likely to sustain attention to the happy faces (Wieser, Pauli, & Mühlberger, 2009).

Binocular rivalry is perceived as a spontaneous alternation between two perceptually incompatible images separately presented to the two eyes. It has been shown that visual and emotional salience of stimuli augments perceptual predominance in binocular rivalry (e.g. Alpers & Gerdes, 2008; Alpers & Pauli, 2006; Alpers, Ruhleder, Walz, Mühlberger, & Pauli, 2005). Notably, it has been shown, that effects in binocular rivalry task are quite robust against conscious manipulation (e.g. van Ee, van Dam, & Brouwer, 2005). Furthermore, it has been shown that under conditions of binocular rivalry angry facial expressions predominate over neutral ones (Alpers & Gerdes, 2007). In contrast to the hypothesis of a bias to angry faces, one experiment found increased dominance of neutral facial expressions relative to dominance of angry ones during anticipation of public speaking. This effect was specifically related to trait social anxiety and not general trait anxiety (Schulz, Alpers, & Hofmann, submitted). The authors suggested that a focus on ambiguous cues, away from emotionally salient facial expressions, may emerge during sustained processing of social stimuli. So far, there are no studies involving participants with SAD.

Finally, assessment of physiological reactivity offers a sophisticated way of investigating emotional responding to external social threat-cues. For example, Dimberg, Fredrikson, and Lundquist (1986) reported that individuals with high vs. low levels of public speaking fear had increased skin conductance activity to facial expressions as compared to neutral pictures (i.e. mushrooms) and no difference in their heart rate responses (for similar results, see Vrana & Gross, 2004). However, another experiment found that angry faces elicited equally enhanced skin conductance responses in both individuals with SAD and healthy controls as compared to other stimuli (Merckelbach, Hout, Hout, & Mersch, 1989).

Research on physiological effects in socially anxious individuals is complicated by the fact, that fear-eliciting threat-cues are perceptually less uniform than in most specific phobias. Furthermore, individuals with SAD often report complex interactions in a way that effects of

specific cues vary across situational context. For example, a blackboard may elicit fear in an exam, but not when used alone for drawing a picture. Several strategies have been applied to address this problem.

One strategy may be to increase individual relevance of threat-cues. For example, previously memorized descriptor sentences used to trigger individually relevant fear imagery (personal fears, social fears, and fears of physical danger) and elicited stronger autonomic responses in participants than neutral ones. The similarity to effects of phobic stimuli is underlined by the finding that panic and PTSD patients were less physiologically responsive (heart rate, skin conductance, corrugator EMG) than specific phobics and socially anxious individuals (Cuthbert et al., 2003). However, it is impossible to identify the particular role of specific stimuli with this procedure.

Furthermore, strategies to boost the salience of social threat associated with facial expressions have been used. For example conditioning angry vs. neutral facial expressions to electric shocks resulted in differential effects on skin conductance responses (Pitman & Orr, 1986). Notably, given these effects to start with, delayed habituation during exposure to angry faces was associated with increased trait anxiousness. Facial expressions have been conditioned to auditory verbal scripts (positive compliments, neutral comments and negative insults) and aversive tones (e.g. Lissek et al., 2008). Similarly, stimuli have been paired with negative arousing stimuli such as aversive odors (Birbaumer et al., 1998). However, it is questionable whether effects are related to the emotional expressions of the facial stimuli. An additional point of critique is that compound stimuli are often not ecologically valid.

Finally, the choice of dependent variables is important, as measures may vary in sensitivity depending on the processes they are thought to tap in. In particular, studies with spontaneous EMG of facial reaction have repeatedly indicated specific response characteristics of socially anxious individuals to socially threatening stimuli. A number of studies suggested specific

stronger facial reactions (i.e. increased corrugator activity to angry faces and increased zygomaticus activity to happy faces as compared to neutral ones) in individuals with high levels of social anxiousness (Dimberg, 1997; Dimberg & Christmanson, 1991; Dimberg & Thunberg, 2007). Although, this method has led to important insights, one weakness of this approach is that the underlying neural processes of these effects are not well understood. *Brain imaging* has added a unique perspective on the role of particular brain structures, which are thought to be involved in specific information processing tasks. As mentioned above, these studies provide accumulating evidence for a crucial role of the amygdala and a complex interplay with the ventral prefrontal cortex and limbic areas for threat-cues processing in socially anxious individuals. Important aspects of this research for the current works will be reported in the focus about research on social anxiety within the startle paradigm; see section 1.3.7 pp. 44. One shortcoming of brain imaging studies is their poor temporal resolution.
In general, this review of approaches suggests that attention and interpretation bias to external social threat-cues exists in socially anxious individuals. However, a major shortcoming of shared by most of these studies is that they do not differentiate between effects of automatic stimulus evaluation and response bias, which may be influenced by conscious processing of the stimuli. As already detailed above, the startle paradigm combines some unique advantages for the investigation of information processing bias in socially anxious individuals. Surprisingly, research on social anxiety disorder has rarely used the startle paradigm to investigate the role of threat-cue vulnerability.

1.3.7 Focus: Research on Social Anxiety within the Startle Paradigm

The startle paradigm has been used extensively for research on anxiety disorders. Generally, these patients feature increased baseline startle amplitudes, regular startle response habituation, valence modulation of the startle amplitude to intense emotional stimuli and

specifically potentiated startle to disorder-specific stimuli or imagery (for a review, see Grillon & Baas, 2003).

Few studies have found *baseline startle differences in patients with high vs. low social anxiety*. Social but not general trait anxiousness correlated positively with startle amplitude during baseline of an experiment on fear-potentiated startle in virtual reality (Cornwell, Johnson, Berardi, & Grillon, 2006). Anticipatory anxiety may have influenced these effects. Britt and Blumenthal (1993) reported faster startle responses in individuals with high vs. low levels of social anxiousness. This has been interpreted as an indicator for increased propensity to respond quickly to external stimuli, such as the startle probe, and may reflect the clinical presentation of socially anxious individuals which is characterized by motor symptoms (see Britt & Blumenthal, 1993). However, most studies did not find differences between individuals with high vs. low social anxiousness in baseline startle assessed without a challenging context (e.g. McTeague et al., 2009).

Literature on constructs, which are related to social anxiety, shows similar findings. Rejection sensitivity has been defined as a "disposition to anxiously expect being rejected by people, who are important to the self, an expectation developed through exposure to severe and prolonged rejection" (Downey, Mougios, Ayduk, London, & Shoda, 2004, p. 668). Eye blink startle magnitude was potentiated in people, who were high in rejection sensitivity, when they viewed rejection themes, as compared to nonrepresentational negative themes. However, no attenuation of the startle response has been found with acceptance themes, as compared to nonrepresentational positive themes (Downey, Mougios, Ayduk, London, & Shoda, 2004). Social anxiety has further been linked to low extroversion and elevated neuroticism (Eysenck Personality Questionnaire (EPQ, Eysenck & Eysenck, 1975). However, Britt and Blumenthal (1991) found no significant association between extraversion and startle response amplitudes. This study has been criticized because the intensity of the startle probe in this experiment (60

and 85 dB) may not have been sufficient to elicit differential responses. In a later experiment, Blumenthal (2001) showed that introverted individuals had larger baseline startle amplitudes using probes at 90 and 105 dB. Finally, some studies have investigated alleged precursors of social anxiety in children. In several studies, children at risk for anxiety disorders had elevated startle reflexes (Grillon, Dierker, & Merikangas, 1997; Merikangas, Avenevoli, Dierker, & Grillon, 1999). However, in temperamentally shy children, startle was not different from controls (Schmidt, Fox, Schulkin & Gold, 1999). Similarly, no significant relations were found between baseline startle and morning salivary cortisol as well as measures of shyness at age 4 (Schmidt et al., 1997). Children who respond with high motor activity and negative affect to unfamiliar stimuli are at risk for behavioral inhibition as toddlers, but no differences between high vs. low motor activity and negative affect were found in baseline startle reactivity (Schmidt & Fox, 1998).

Similar to other anxiety disorders, startle response habituation appears regular in individuals with high levels of social anxiety (e.g. Larsen, 2001). However, higher levels of extraversion and sensation seeking were associated with faster habituation (LaRowe, Patrick, Curtin, & Kline, 2006).

In an elaborate approach, Panayiotou and Vrana (1998) have further tried to differentiate whether attentional focus or content related affective activation drives modulation of the startle responses in socially anxious individuals. The *attention hypothesis* predicts reduced baseline startle when social challenge leads to an inward focus of attention, away from the environment including the startle probe. For example, as noted above, presence of an observer led to decreased startle (Blumenthal, Chapman, & Muse, 1995; Britt & Blumenthal, 1993). In contrast, the *content hypothesis* predicts increased startle due to activation of the negative motivational system by increased access to negative thought content during social challenge. Panayiotou and Vrana (1998) investigated the effects of instructed attentional self-focus in

socially anxious individuals instead of inducing information processing bias via social challenge. This led to increased startle amplitudes in individuals with high levels of social anxiousness. Importantly, this suggests that increased self-oriented attentional focus does not necessarily decrease attention to external stimuli. Furthermore, the findings support the content hypothesis. However, Panayiotou and Vrana (1998) did not directly assess the effect of their attention manipulation on cognitive content and they did not show whether their effects were mediated by cognition.

Fear potentiation of the startle response in individuals with high levels of social anxiousness was found during visual processing of socially threatening words relative to non-threatening words (Larsen, Norton, Walker, & Stein, 2002). The implication of these findings are limited, because they were based on effect size measures rather than significance testing and there was a similar increase with physical threat words. Furthermore, baseline startle response, startle response habituation and prepulse inhibition were descriptively stronger in socially anxious individuals, but not significantly different from healthy controls. This questions the specificity of the results. However, the simple display of verbal stimuli may not be arousing enough to reliably induce differential modulation of the startle response (see Cuthbert, Bradley, & Lang, 1996). Yet, a study addressing this issue by use of the comparatively much more arousing IAPS pictures (Lang, Bradley, & Cuthbert, 1999) found no specific effects in individuals with generalized high social anxiousness. Comparing socially threatening to generally threatening, neutral and positive pictures, they showed the same pattern of valence modulated startle response as healthy controls (Gros, Hawk, & Moscovitch, 2009). Importantly, anxiety disorder spectrum theory (e.g. Lang, McTeague, & Cuthbert, 2005) has suggest a less robust and less specific modulation of the startle response by fear stimuli than in specific phobia, because of reduced associative strength in fear network memory structures involving the appetitive and defensive motivational circuits thought to modulate the startle response.

Accordingly, fear potentiation of the startle response has been shown to vary systematically between anxiety diagnoses. Interestingly, when (Gros, Hawk, & Moscovitch, 2009) classified their sample according to criteria of high vs. low fear of public-speaking rather than general social anxiety, the high fear group showed reliable potentiation of the startle to external social threat-cues, while the low fear group did not. Similarly, removing individuals with a specific fear of public speaking from the sample eliminated the effect of increased startle amplitudes during cued recall of previously taught associations to brief socially threatening verbal scripts in individuals with high levels of social anxiousness (Cuthbert et al., 2003). In line with this view, startle potentiation was most specific and most pronounced in individuals with circumscribed SAD and the shortest disorder duration during imagery of narrative scripts (socially threat, survival threat, neutral, and idiographic fear scripts). Individuals with generalized SAD had potentiated startle amplitudes to a broader range of scripts (e.g. including survival threat). Finally, patients with chronic social anxiety disorder featuring comorbid depression showed attenuated startle responses, except for personal fear scripts (McTeague et al., 2009).

A different approach has investigated startle responses to conditioned stimuli, using socially relevant stimuli such as neutral faces as the CS. When aversive odors were used as the US, there was no indication for stronger conditioned responses to the now negatively conditioned neutral faces in individuals with high social anxiousness, when compared to controls. Counter-intuitively, socially anxious individuals had overall lower startle amplitudes in this experiment. However, alternative reasons for this between-subjects effect complicate its explanation (e.g. reduced arousal as indexed by skin conductance level in this group, Hermann, Ziegler, Birbaumer, & Flor, 2002). Another study showed that after being conditioned to auditory verbal scripts (positive compliments, neutral comments, and negative insults) plus appropriate facial expressions (happy, neutral, and angry), neutral faces as CS led

to expected attenuation vs. increase of the startle response only in participants with high levels of social anxiousness (Lissek et al., 2008). It seems noteworthy that verbal scripts used as CS or directly as a threat-cue also may trigger self-related negative cognitions. Therefore it is not clear whether increased startle amplitudes reflect a direct effect of the stimulus or if cognitive associations mediate the effect. Importantly, when used as a CS, it seems possible that it is rather a conditioning to fear related cognitive networks than conditioning to the mere stimulus qualities of verbal scripts.

Finally, indirect evidence for an association of social anxiousness with startle fear potentiation may be drawn from an experiment on individuals with increased harm avoidance as assessed with Cloninger's Tridimensional Personality Questionnaire (Cloninger, Przybeck, & Svrakic, 1991). Interestingly, only extraverted participants showed the typical pattern of startle modulation by emotional vs. neutral images. Subjects high in harm avoidance only showed increased startle amplitudes during presentation of unpleasant slides, whereas subjects low in harm avoidance only showed modulation to pleasant slides (Corr et al., 1995). Finally, Temple and Cook (2007) have examined valence effects of highly arousing IAPS pictures across different levels of trait anxiousness and high vs. low defensiveness. Exploratory analyses suggested that valence modulation of the startle amplitude was restricted to the subgroup with high levels in both measures.

Surprisingly, only one study so far has investigated *startle potentiation to facial expressions*, one of the most prototypic signals for regulating social interaction, in individuals with social anxiousness (see discussion in Gilboa-Schechtman, Foa, & Amir, 1999). White (2002) compared affective startle modulation to angry, neutral, and happy facial expressions across five tasks (attending to facial features in regular vs. inverted stimuli, age, angry or happy expressions) at a stimulus-onset to probe delay of 3800 ms and attentional PPI at 120 ms. Individuals with SAD displayed attenuated startle responses to regular displays as compared

to inverted angry and happy facial expressions at a stimulus-onset to probe delay of 3800 ms and attentional PPI to emotional (angry and happy) vs. neutral facial expressions at 120 ms independent of instructions in the five attention tasks. Finally, females with SAD had generally increased and delayed startle reactions, which is consistent with depressive response patterns, although no comorbid depression was diagnosed (White, 2002).

Furthermore, fear potentiated startle to facial expressions has been investigated in unselected samples. Springer et al found increased startle amplitudes during processing of angry facial expressions as compared to fearful, happy, and neutral expressions (Springer, Rosas, McGetrick, & Bowers, 2007). However, others have failed to demonstrate startle potentiation to angry vs. neutral and happy adult facial expressions (Alpers & Adolph, 2006; Schmidt-Daffy, 2006) or crying vs. smiling face images of infants (Spangler, Emlinger, Meinhardt, & Hamm, 2001). In all these studies, gaze direction of facial expressions was oriented at the participant. However, when direct gaze was compared to a gaze direction offset of 30 degrees, gaze direction emerged as a potential moderator of valence effects. Startle responses to angry (vs. happy and neutral) faces were only potentiated with gaze direction at zero degrees. At 30 degrees, startle to angry faces was slightly inhibited in comparison to neutral expressions (Ivanonva & Allen, 2001).

Furthermore, in a study comparing startle amplitudes during processing of happy, neutral, and angry facial expressions as well as positive and negative pictures, potentiation to angry and inhibition to happy faces emerged only in male participants (Hess, Sabourin, & Kleck, 2007). The authors interpreted this as an indicator for an interaction of valence and dominance. However, this claim has not been tested directly (e.g. via dominance ratings).

Finally, startle modulation to facial expressions has been examined in children. Balaban (1995) reported the expected linear relationship between startle amplitudes and angry, neutral, and happy facial expressions. Furthermore, fear potentiation of startle amplitudes to novel and

familiar pictures of angry, happy, and neutral facial expressions were investigated in 7-12 year old children with or without behavioral inhibition. However, no valence modulation or group differences were found. Exploratory analyses revealed that behaviorally inhibited children generally had attenuated startle amplitudes with novel slides, particularly happy faces, when compared to behaviorally uninhibited children. Furthermore, novel happy faces and familiar angry and neutral faces elicited larger startle amplitudes than familiar angry and neutral and novel happy faces (van Brakel, Muris, & Derks, 2006).

Interestingly, no experiment has investigated fear potentiation of the startle to specific foreground cues within a *situational challenging context*. However, situational context may be crucial for assigning individual relevance to foreground stimuli. Angry faces may for example signal a violation of social rules or expectations specifically during anticipation of public speaking (Averill, 1982) and context apparently affects startle responding. During both baseline and anticipation of public speaking, but not during a socially non-threatening counting task, a positive correlation between startle amplitude and social but not general trait anxiousness was found in virtual reality (Cornwell, Johnson, Berardi, & Grillon, 2006). Furthermore, initially increased startle during baseline in introverted participants was followed by a stronger decrease, independent of habituation, when their anxiety increased during a social encounter (Blumenthal, Chapman, & Muse, 1995). It was argued that stronger autonomic reactivity in comparison to extroverted individuals may account for a greater shift of attention focus on self-relevant cognitions in response to social threat, leading to startle inhibition. Finally, a subgroup of children with self-presentation anxiety showed both an increased cortisol response and stronger startle reactions in a stranger approach task (Schmidt, 1997).

Summing up this review of startle research in individuals with social anxiety, some considerable gaps in literature can be identified. First, no experiment has examined

interactions of fear potentiated startle, situational challenge, and social anxiety. Second, no experiment has investigated whether self-related negative cognitions mediates such associations. While this question primarily addresses whether preferential processing of threat-cues mediates the activation of related cognitive content, also reverse effects may be possible. Third, no experiment has investigated whether cognitive emotion regulation strategies affect these associations. Fourth, only one study has investigated fear potentiated startle during processing of emotional facial expressions in socially anxious individuals, although these stimuli comprise the probably most intuitive implementation of social stimuli (e.g. non-verbal negative feedback in social encounters). Since this study did not examine potential interactions with socially challenging situational contexts, effects of foreground stimuli vs. context were not examined in detail. Finally, effects of socially challenging contexts are not straightforward. While some studies found startle attenuation, others reported startle potentiation. Elaborate approaches similar to the study by Panayioutou and Vrana (1998) may be required to resolve the complementary roles of attention and affective activation.

1.3.8 A Working Model on Mediators of Social Anxiety

Integrating the models of social anxiety reviewed above with the empirical data supporting that self-related negative cognitions and vulnerability for preferential processing of external social threat-cues play an important role in individuals with social anxiety, a working model on mediators of social anxiety is proposed, see Figure 5. This model shows how the association between trait social anxiousness (*predictor*) and anxiety symptoms (*criteria*: self-report, behavior, physiology) is mediated by two presumed *mediators* (external social threat-cues and self-related negative cognitions) within a threatening social situation (*context*).

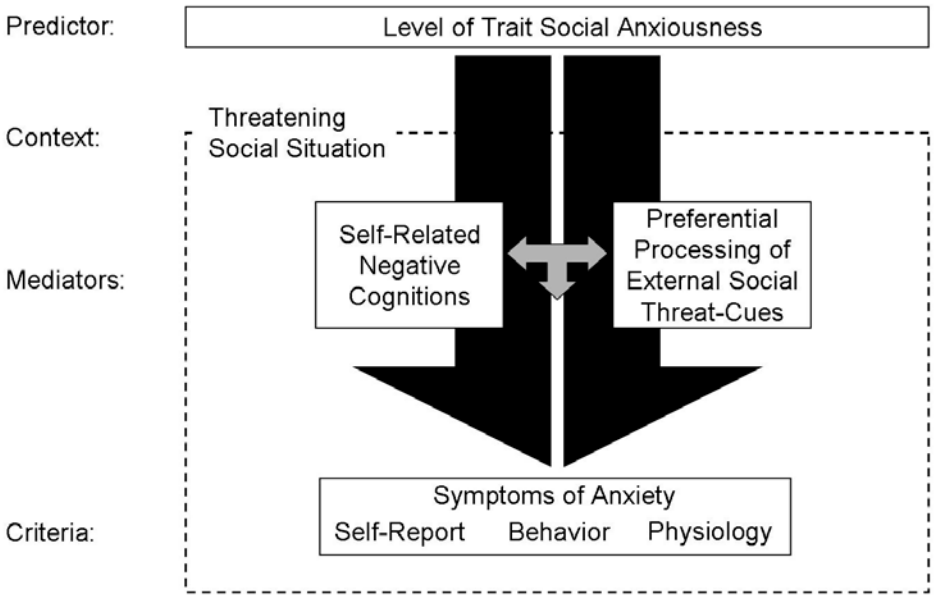

Figure 5: A working model on mediators of social anxiety: *self-related negative cognitions* and automatic *preferential processing of external social threat-cues*; see text for explanation.

In line with the dimensional perspective on social anxiety (McNeil, 2001), it is expected that increased levels of trait social anxiousness predict stronger anxiety symptoms when a socially threatening situation is present. Notably, this situational context may be a necessary precondition for a coherent emotional response across response systems (self-report, behavior, and physiology, see Lang, 1993). It should be noted that evidence for physiological characteristics of socially anxious individuals is limited, see section 1.3.5, pp. 33 and section 1.3.6, pp. 38. However, methodic shortcomings, the choice of dependent variables and examining effects without a socially challenging context may explain why previous studies have typically not found that physiology was different in individuals with high vs. low trait social anxiousness. For example, associations between trait social anxiousness and observable behavior have been found primarily in stressful social situations. This underlines the importance of investigating mediators of social anxiety within socially challenging situations connecting environmental social stimuli and self-related cognitions in context-based coherence to social anxiety.

Cognitive models highlight the importance of dysfunctional *top-down* effects of self-related negative cognitions as a potential mediator of associations between the level of trait social anxiousness and anxiety symptoms. The available literature already provides strong support for this assumption. However, so far, no experiment has directly tested the mediation hypothesis.

Information processing models emphasize the role of hereditary or acquired vulnerability of brain systems involved in the automatic detection and evaluation of external social threat-cues. A lowered detection threshold together with a more negative interpretation of external social threat-cues is assumed to mediate associations between the level of trait social anxiousness and anxiety symptoms in parallel to self-related negative cognitions.

As suggested by the multi-process account of startle modulation during affective perception (see section 1.3.2.1, pp. 24), *top-down* effects of self-related negative cognitions and *bottom-up* effects of automatic processing of external social threat-cues may interact via spreading activation within fear-networks, see Figure 2, p. 26. This potential interaction of automatic cue-processing vs. higher-order semantic processing is indicated by a grey arrow in Figure 5, p. 53.

It should be noted that for example the integrated model of social anxiety (Turk, Lerner, Heimberg, & Rapee, 2001) suggests that individual sensitivity for perceiving internal anxiety symptoms may further intensify negative cognitions. Similarly, inadequate behavior for example during public speaking may be noticed and could trigger negative self-evaluation. Although the following experiments did not focus on these aspects, they were explored, when the assessment suggested effects.

The working model on mediators of social anxiety provides a concise framework for deriving specific hypotheses about the triple interaction of trait social anxiousness, situational context, and mediators of acute social anxiety. Notably, this allows for an isolation of specific factors

for experimental manipulation in order to test whether effects are contingent with the presumed interactions between elements of the model.

1.3.9 Aim of the Experiments

The objective of the following three experiments was to examine predictions derived from the working model on mediators of social anxiety; see section 1.3.8, p. 52. In particular it was examined whether preferential processing of *external social threat-cues* and *self-related negative cognitions* mediate associations between trait social anxiousness and acute anxiety on three response domains: self-report, behavior, and physiology. As elaborated in the model, complex interactions between these mediators may exist, and effects may depend on the presence and demand characteristics of an evaluative situational context.

The first experiment focused on the question whether external social threat-cues (angry facial expressions as compared to neutral and happy ones), presented in a socially evaluative context assigning individual relevance to these stimuli, automatically induce negative affect and avoidance tendencies. Furthermore, the question was addressed whether *top-down* effects of cognitive strategies moderate the effect of external social threat-cues on anxious responding, or whether stimulus valence would affect performance of using these strategies via *bottom-up* priming. Finally, it was examined whether effects varied across different levels of trait social anxiousness.

The second experiment investigated whether individuals with a clinical diagnosis of social anxiety (SAD) respond with a particularly strong automatic negative affective reaction to external social threat-cues (angry vs. neutral and happy facial expressions). In addition, this experiment differentiated the complementary roles of automatic attention allocation and affective activation to external social threat-cues.

The third experiment examined whether focusing on self-related negative cognitions or biased processing of external social threat-cues mediates effects of trait social anxiety on state

anxiety in a socially challenging situation (anticipation of public speaking). In addition, the impact of external social threat-cues (facial expressions from experiments 1 and 2 as well as emotional words) was compared to emotional and neutral control stimuli and effects of the situational context.

2 Experiments

2.1 Experiment 1 – Startle Reflex Modulation to External Social Threat-Cues: Moderated by Cognitive Emotion Regulation?

2.1.1 Background and Hypotheses

The primary goal of this experiment was to examine *whether external social threat-cues affect* affective responding (i.e. criteria in the working model on mediators of social anxiety, see section 1.3.8 p. 52). Facial expressions are prototypical social threat-cues (Gilboa-Schechtman, Foa, & Amir, 1999), which are universally recognized (Ekman & Davidson, 1994). As elaborated above (section 1.3.2.1, pp. 24), the startle paradigm is ideally suited to examine activation in neural circuits which are involved in automatic detection of external threat-cues. At least four studies support the assumption that positive and negative facial expressions induce affective modulation of startle responses as compared to neutral ones in unselected samples (Balaban, 1995; Hess, Sabourin, & Kleck, 2007; Ivanonva & Allen, 2001; Springer, Rosas, McGetrick, & Bowers, 2007). Therefore, fear potentiated startle responses to socially threatening facial expressions (e.g. angry faces) are not only expected in individuals with social anxiety disorder, but may be moderated by information processing bias in socially anxious individuals (Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). As suggested in the working model on mediators of social anxiety, higher levels of trait social anxiousness should therefore predict increased fearful responding to external social threat-cues. One study reported affective modulation of startle amplitudes to emotional facial expressions in a comparatively small sample of patients with a clinical diagnosis of social anxiety disorder (n = 9, White, 2002). This may support the assumption that these individuals feature particularly strong effect information processing bias to external social threat-cues.

A secondary goal of this experiment was to examine potential interactions of preferential processing of external social threat-cues with cognition. Both habitual and instructed emotion regulation strategies may moderate fear potentiation of the startle response to external social threat-cues via downstream effects when cognitive networks overlap with the defensive motivational system (Lang, 1995). Affective startle modulation to imagery of threatening scenes has demonstrated that top-down modulation of the defensive motivational system is possible (Lang, Bradley, & Cuthbert, 1998). Furthermore, it has been shown that individuals are capable of regulating various aspects of emotional responding (e.g. Gross, 1998, 2002; Gross & Levenson, 1993). Finally, it has been shown that emotion regulation strategies may specifically modulate fear potentiated startle to external threat-cues. When participants were instructed to *suppress* their emotional response to unpleasant pictures, startle amplitude was attenuated, whereas the instruction to *enhance* this response led to startle potentiation (Jackson, Malmstadt, Larson, & Davidson, 2000). A similar study indicated that these instructions may primarily modulate arousal without affecting valence related differences between positive, negative, and neutral stimuli (Dillon & Labar, 2005).

In the case of social anxiety, the cognitive model of social anxiety predicts that socially anxious individuals monitor their internal state in the face of social threat (Hofmann & Barlow, 2002; Spurr & Stopa, 2002; Woody, 1996). This response matches an emotion regulation strategy of *focusing on one's internal reaction* to socially threatening stimuli (e.g. an angry facial expression). In particular, within a socially challenging context, an angry face is therefore likely interpreted as a nonverbal sign of devaluation and confrontation (e.g. Averill, 1982). In this case, both bottom-up effects of information processing bias and top-down effects of cognitive emotion regulation would link up via spreading activation of semantic networks, as suggested in Lang's (1995) model, see Figure 2, p. 26. This may enhance effects to external social threat-cues on both self-report (i.e. ratings of the emotional

properties of the stimuli) and fear potentiation of the startle response. Furthermore, associations between nodes of fear network linking angry facial expressions to social anxiety may be stronger in individuals with high vs. low trait social anxiousness (Lang, 1995). Therefore, increased levels of trait social anxiousness were expected to predict particularly strong effects of external social threat-cues on anxiety symptoms.

In contrast, a strategy to *identify with a facial expression* requires empathic responding. Furthermore, it may trigger inferences about potential reasons to show this emotional expression. This leads to an attentional focus away from one's initial reaction to the shown emotion. Notably, in the case of neutral and happy facial expressions, this does not change one's final emotional reaction (e.g. identifying with and focusing on one's reaction to a smiling face both induces happiness). However, while the default response to angry facial expressions is fear, identification induces anger.

Individuals with high trait social anxiousness have been shown to focus their attention automatically on external social threat-cues and interpret them more negatively (Heinrichs & Hofmann, 2001). Furthermore, in a socially challenging situation, they tend to focus attention inwards, which is on their fear reaction to the angry face. Therefore, it may be particularly hard for them to change their habitual response style. It was expected that this would emerge in lower ratings of successfully implementing the strategy to *identify with facial expressions* on display. In contrast, low socially anxious individuals were expected to successfully implement this strategy. This would increase the difference between individuals with low vs. high social anxiousness particularly when they try to identify with the facial expressions. Consequently, a particularly high correlation between trait social anxiousness and effects of external social threat-cues (i.e. angry facial expressions) was expected when participants tried to *identify with facial expressions*.

In addition to startle amplitude, effects of external social threat-cues and trait social anxiousness on startle latency were examined. It has been shown that increased stimulus arousal but not valence of fear imagery (Cook, Hawk, Davis, & Stevenson, 1991) as well as intense emotional pictures (Bradley, Cuthbert, & Lang, 1993) reduces startle latency elicited by acoustic probes. Therefore, generally lower latencies may emerge in responses to emotional facial expressions, as compared to neutral ones. Second, decreased startle latency has been suggested to index an increased sensitivity of the motor component of the startle response and may be linked to the hyper-responsive motor behavior featured by individuals with SAD (see Britt & Blumenthal, 1993 for a discussion). Therefore, increased trait social anxiousness was expected to predict faster startle responding in all conditions. Instructed inward focus of attention did not affect startle amplitude in a previous study (Panayiotou & Vrana, 1998). However, potential effects on startle latency were not examined. Therefore, the attention capturing effects of emotional stimuli or application of cognitive emotion regulation strategies may interact with startle latency.

Finally, affective state was assessed to control for potentially confounded effects of mood and to identify potential interactions between effects of the emotional stimuli and affective state. Personality traits such as neuroticism and extraversion may be confounded with effects of trait social anxiousness. Therefore, correlations with these variables were explored.

Experiment 1 addressed three questions derived from the working model on mediators of social anxiety (section 1.3.8 p. 52). First, do external social threat-cues such as angry facial expressions induce affective startle modulation similar to intense emotional pictures when they are presented within a socially challenging context? Second, do these effects interact with cognitive emotion regulation? Third, have individuals with higher levels of trait social anxiousness also a stronger information processing bias to external social threat-cues?

Positive answers to these three questions were expected in support for the working model on mediators of social anxiety.

2.1.2 Methods

2.1.2.1 Design and Participants

The experiment featured a 3 (Within subjects factor Facial-Expression: happy, neutral, angry) x 2 (Within subjects factor Strategy: identify, vs. react) x 2 (Between subjects factor Task-Order: Beginning with strategy identify, vs. beginning with strategy react) design.

Forty-four participants were recruited from the undergraduate psychology student pool of the University of Würzburg via postings on the experiment sign up board at the Psychology Department. The students received course credits for their participation regardless of whether they terminated the experiment or not. Inclusion criteria were normal or corrected vision and signed written informed consent, see appendix 5.1.1, p. 207. Exclusion criteria were intake of alcohol less than eight hours prior to the experiment, tinnitus, or hearing impairment.

2.1.2.2 Apparatus

Instructions, picture stimuli and startle probes were presented on a 19” flat screen CRT-monitor (Fujitsu-Siemens 19P4; 1024x768, 85 Hz) using Presentation® software (v.0.80, www.neuro-bs.com) running on a Pentium-level computer. Participants’ heads were at approx. 40 cm distance to the screen. Startle probes of 50 ms, 100 dB(A) bursts of white noise with near instantaneous rise/fall times (Lang, 1995) were presented via a SoundBlaster Audigy sound card connected to a Kenwood KA-3010 amplifier and played binaurally through Bayer Dynamik dt 901 headphones. Eye-blink startle responses were measured electromyographically (EMG) using No. 21708305 Ag/AgCl surface electrodes (GE Medical Systems) placed about 1 cm below the pupil and outer canthus of the left eye, matching sites recommended for assessing orbicularis oculi activity (see Fridlund & Cacioppo, 1986). The

raw EMG was amplified (frequency response: 19-500 Hz), digitally sampled at 1024 Hz (16 bit A/D-converter) with a Varioport system (Becker Meditec) and saved to hard disk.

2.1.2.3 Trait Measures

The German version of the Social Phobia and Anxiety Inventory (SPAI, Fydrich, 2002; English original by Turner, Stanley, Beidel, & Bond, 1989), a well-established self-report measure of trait social anxiety with excellent psychometric properties, was used to assess social anxiety and anxiety related to performance situations. See Table 1 for an overview of the internal consistency achieved for questionnaires in the experiment sample. The German version of the NEO-FFI (Borkenau & Ostendorf, 1993), which complies with the short 60-item version of the 240 item NEO-PI-R (Costa & McCrae, 1992), was used to analyze effects of personality traits with a particular focus on potential interactions of neuroticism and extraversion with social anxiety.

2.1.2.4 State Measures

The state-version of the Spielberger State Trait Anxiety Inventory (STAI-state, Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was used to examine associations with changes of state-anxiety across the experiment. The German version of the Self-Consciousness Scale (English original by Fenigstein, Scheier, & Buss, 1975; SCS-D, Heinemann, 1979) was used to control for aspects of social anxiety related to perceiving oneself as part of a social interaction. Excessive public self-consciousness may lead to increased attention to oneself as falling short of intrinsic social ideals. This may be an important component of social anxiety, because exaggerated estimation of social cost related to failing such standards has been shown to mediate SAD (Hofmann, 2004). Furthermore, levels of private and public self-consciousness may interact with perspective taking in social interactions, which has been the subject of experimental manipulation in this experiment. The Positive and Negative Affect Schedule (PANAS, Krohne, Egloff, Kohlmann, & Tausch, 1996) was used to control for

potential interactions of affective state with emotional information processing during the experiment.

Questionnaires and Subscales		Internal Consistency (Cronbach's α)
SPAI		T_0: .96
NEO-FFI	*neuroticism*	T_3: .78
	extraversion	T_3: .81
	openness to new experiences	T_3: .73
	agreeableness	T_3: .74
	conscientiousness	T_3: .77
STAI-state		T_1: .58, T_2: .49, T_3: .64
SCS-D		
	private self-consciousness	T_0: .55, T_2: .72, T_3: .70
	public self-consciousness	T_0: .59, T_2: .60, T_3: .67
	social anxiety	T_0: .77, T_2: .76, T_3: .80
PANAS		
	positive affect (PA)	T_1: .82
	negative affect (NA)	T_1: .76

Table 1: Overview of the internal consistency (Cronbach's α) achieved for questionnaires and subscales with the experiment sample, see text for details. T_0 = beginning of experiment; T_1 = at beginning of startle procedure; T_2 = after first block; T_3 = after second block; see procedure for details.

2.1.2.5 Procedure

After signed informed consent was obtained (T_0), each participant completed the SCS-D, the SPAI, and a socio demographics form. Next, the participants were introduced to the

experimental setup, including a video system for monitoring their non-verbal reactions during the experiment; see Figure 6.

Figure 6: Experimental setup from the perspective of the participants. The picture shows the monitor for stimulus presentation with a video-camera on top for behavior observation in an adjacent room, the modified keyboard for rating input and experiment control, as well as the Varioport physiology data recorder on the left foreground of the table.

To create a social evaluative context, participants were informed that the experimenter observed them on a monitor in the adjacent room and rated certain characteristics that were not further specified in detail. Next, the participants were seated in front of the screen used for stimulus presentation and were familiarized with usage of a modified keyboard for collecting ratings during the course of the experiment; see Figure 7.

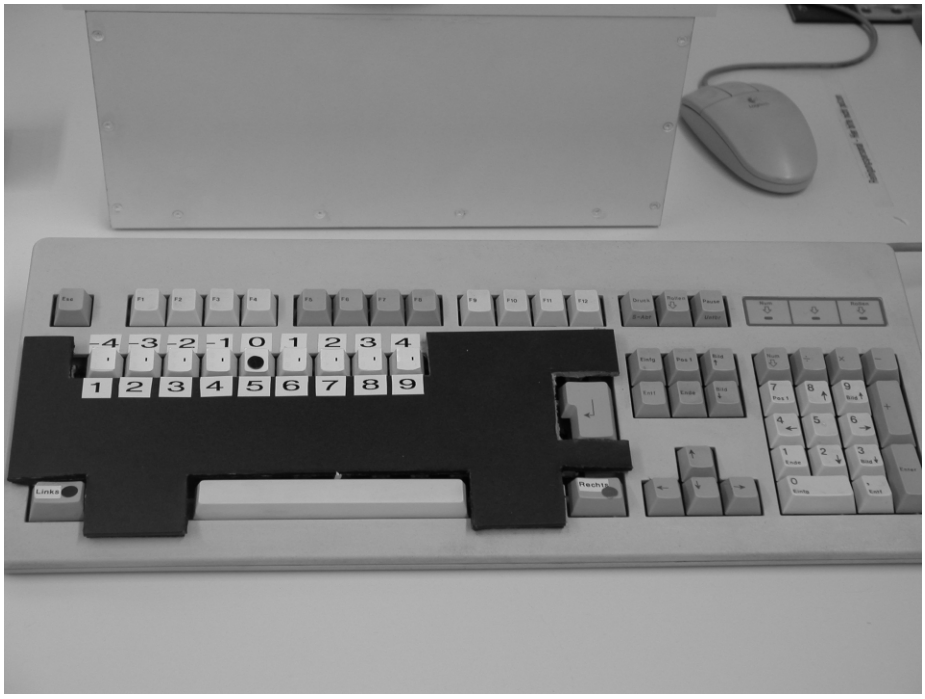

Figure 7: Close-up of the modified keyboard for rating input and experiment control.

After this, the electrodes for physiological data acquisition were attached and the participants put the headphones on. Immediately before the beginning of the startle assessment (T_1), participants were asked to fill out the PANAS and the STAI-state.

Next, two blocks followed where the participants were asked to apply one of two instructions:

Strategy to react: "In this block you should try to identify with the person shown, by focusing only on the facial expression on display and feel the emotional state of this person. Do not let other thoughts distract you."

Strategy to identify: "In this block you should attend which emotion you feel by looking at the facial expression shown. Meaning, which emotion is elicited in you, when you see somebody with this facial expression?"

See appendices 5.1.2, p. 208 and 5.1.3, p. 208 for the original German instructions.

Order of the instructions was counterbalanced across participants and randomly assigned.

Within each block, 24 stimuli (eight sets of angry, happy, and neutral facial expressions posed by eight different actors, respectively selected from the Karolinska Directed Emotional Faces

set (KDEF, Lundqvist, Flykt, & Öhman, 1998), were presented in random order. Each stimulus was presented for 8 s with a black screen as an inter stimulus interval (ISI, duration: 10 s ± 2 s). Startles were elicited at 4.5 s ± 0.5 s after stimulus onset, with fixed assignment to 3 out of 4 stimuli per category, to assess effects of the foreground stimuli on the activation of affect induced action tendencies (Lang, 1995). Before each picture, *react* vs. *identify* was presented to remind the participants of the strategy they should apply. Following each picture, three ratings on Likert-like scales were obtained: valence (-4 = extremely negative, 0 = neutral, +4 = extremely positive) and arousal (1 = not at all, 9 = extremely) of the participants' emotional response to the combination of both picture and emotion-regulation strategy, and how well the participants thought, they were able to apply this strategy (1 = not at all, 9 = perfectly). After completion of the first block (T_2), the participants completed another set of STAI-state and SCS-D forms. Next, they continued with the second block of stimulus presentation, using the other emotion regulation strategy. After completion of the second block (T_3), the participants filled out a final set of questionnaires, including STAI-state, SCS-D, NEO-FFI. After a short manipulation check interview they were fully debriefed.

2.1.2.6 Data Reduction and Analysis

EMG-recordings of the eye-blink startle responses were processed offline (digital 50 Hz notch filter to minimize AC-leakage, 4th order Butterworth lowpass-filter at 500 Hz, 4th order Butterworth highpass-filter at 20 Hz, rectified and smoothed with a 100ms moving average for amplitude scoring, and a 20ms moving average for latency scoring, see Blumenthal et al., 2005; van Boxtel, 2001) and scored using a custom MATLAB (Mathworks, Inc., Natick, MA) program (Schulz & Alpers, 2007) which implements state of the art criteria (Blumenthal et al., 2005). Responses with amplitudes equal or smaller than zero and reactions with artifacts were excluded from the analysis (0.2 %). There were no startle non-responders. Startle amplitudes were computed by subtracting the average of 20ms of data before startle-onset from the

maximum amplitude identified between 20 and 120 ms after startle-onset and averaged across all responses within cells of the experiment design. Startle amplitudes were log-transformed to achieve normal distribution across subjects. K-S tests confirmed the success of the transformation for startle amplitude within each stimulus category, all *p's* ≥ .48. K-S tests for startle latency showed that no transform was necessary, all *p*'s ≥ .08.

2.1.2.7 Statistical Analysis

A power analysis (Faul, Erdfelder, Lang, & Buchner, 2007) assuming medium effect-sizes (Cohen, 1988) for effects of the emotion regulation strategies and affective startle modulation indicated a statistical power of 90 % for a sample size of $N = 44$.

To investigate whether emotion regulation strategies differentially affected the activation of action tendencies by processing emotional facial expressions, repeated measures ANOVAs (within-subjects factor Strategy: identify, vs. react; within-subjects factor Facial-Expression: happy, neutral, angry) were computed for dependent variables startle amplitude and latency. To control for potential time effects, Block-Order (group beginning with strategy identify vs. group beginning with strategy react) was added as another factor. Planned contrasts followed, where appropriate.

Further repeated measures ANOVAs (within-subjects factor Facial-Expression: happy, neutral, angry), planned contrasts, and paired *t*-tests (two-sided) were used to examine emotional properties of the stimulus material (ratings of stimulus valence and arousal) in the current sample as well as ratings on subjective feelings of successful implementation of the emotion regulation strategies.

Pearson correlations were used to investigate associations between personality variables (NEO-FFI, SCS-D), affective state (SPAI, PANAS), and startle measures (amplitude and latency), and to examine associations with stimulus ratings (arousal, valence, and successful strategy implementation).

Finally, independent *t*-tests, Chi^2-Tests, and Pearson correlations were used for manipulation checks and to describe the sample.

2.1.3 Results

2.1.3.1 Participants

All participants identified themselves as Caucasian, their mean age was 22.27 years (*SD* = .83) and 33 (75.00 %) participants were female. Most of the participants were right-handers (*n* = 43, 98 %), had deficient but corrected vision (*n* = 22, 50 %), and all of them indicated having no hearing issues. Initial analyses indicated no gender differences in the main dependent variables. There were no differences between female and male participants with regards to age, SPAI, PANAS, SCS scales *private self-consciousness* and *social anxiety*, NEO-FFI scales *extraversion*, *openness to experiences*, *agreeableness*, *conscientiousness* and STAI-state, all *p*'s ≥ .06. The only exception was found in subscale of the SCS-D. Female participants (*M* = 3.50, *SD* = .52) scored higher on the SCS-D subscale *public self-consciousness* than male participants, *M* = 3.16, *SD* = .32, $F(1, 42) = 4.18$, $p < .047$, partial-η^2 = .09. Furthermore, female participants had higher scores on the *neuroticism* subscale of the NEO-FFI (*M* = 24.03, *SD* = 6.91) as compared to male participants, *M* = 16.91, *SD* = 4.55, $t(42) = -3.18$, $p < .01$, $d = 1.22$. However, these differences are typical for representative samples comprising both sexes. Therefore, the data were collapsed across gender and results are reported for the complete group.

2.1.3.2 Trait Measures

Participants' SPAI scores (*M* = 47.79, *SD* = 2.67, *Min* = 19.70, *Max* = 112.40, scores adjusted for comparison to the US-version of the SPAI, see Fydrich, 2002) were in a typical range for unselected student samples (e.g. Turner, Beidel, Dancu, & Stanley, 1989). Only one participant scored above a cutoff of 80, suggesting that a clinical diagnosis of social anxiety

may be warranted. 36 (81.92 %) participants scored below 60, a cutoff considered optimal for identifying non-social-phobic participants (Turner, Beidel, Dancu, & Stanley, 1989).

NEO-FFI subscale scores were comparable to the reference sample reported by Borkenau and Ostendorf (1993, p.13), see Table 2.

NEO-FFI Subscale	Sample of Experiment 1 (N = 44) M (SD)	German Reference Sample (N = 2112) M (SD)
neuroticism	22.25 (7.08)	22.1 (8.4)
extraversion	29.09 (6.48)	28.3 (6.8)
openness to new experiences	32.93 (5.78)	32.5 (6.2)
agreeableness	32.05 (5.61)	29.3 (5.9)
Conscientiousness	32.30 (5.89)	30.4 (7.6)

Table 2: Means and standard deviations for the subscales of the NEO-FFI in both the sample of experiment 1 and a German reference sample published by Borkenau and Ostendorf (1993, p.13).

2.1.3.3 State Measures

Scores on subscales of the SCS-D remained stable during the experiment, as indicated by a multivariate test with Pillay Spur-Criterion for the three assessments at T_0, T_2, and T_3, $F(6, 37) = 1.38$, $p < .25$, partial-$\eta^2 = .19$. The participants scored in a typical range for the general German population (Heinemann, 1979), see Table 3.

STAI-state assessments revealed that in comparison to age related norms (age group: 15-29 male subjects: M = 36.56, SD = 9.47 and female subjects: M = 36.96, SD = 10.27, Spielberger, Gorsuch, & Lushene, 1996) the participants had elevated scores that did not vary significantly between the beginning of the experiment ($M_{T1} = 39.23$, $SD_{T1} = 4.63$) and the assessment after the first block ($M_{T2} = 38.72$, $SD_{T2} = 4.69$, $t(43) = 1.00$, $p < .32$, $d = .11$) as

well as the assessment after the second block, M_{T3} = 39.01, SD_{T3} = 5.20, $t(43) = -.52$, $p < .61$, $d = .06$.

SCS-D Subscale	T_0	T_2	T_3
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
private self-consciousness	3.50 (0.42)	3.43 (0.51)	3.43 (0.51)
public self-consciousness	3.43 (0.50)	3.36 (0.51)	3.44 (0.54)
social anxiety	2.90 (0.75)	2.86 (0.76)	2.93 (0.81)

Table 3: Means and standard deviations for subscales of the Self-Consciousness Scale (SCS-D, German Version); T_0 = beginning of experiment; T_2 = after first block; T_3 = after second block; see procedure for details.

Importantly, there were no group differences or time by group interactions when comparing the group starting with the identification strategy vs. starting with the reaction strategy, all *p*'s ≥ .13.

Affective state as assessed with the PANAS was in a normal range (Krohne, Egloff, Kohlmann, & Tausch, 1996) immediately before the beginning of the experimental task, T_1: *positive affect*, M_{PA} = 25.66, SD_{PA} = 5.70 and *negative affect*, M_{NA} = 12.84, SD_{NA} = 2.72. There were no differences between the two groups starting with different strategies, all *p*'s ≥ .25.

2.1.3.4 Manipulation Checks

The form used for a manipulation check at the end of the experiment indicated that almost all of the participants implemented the instructions correctly (42, 95.5 %). This included two participants (4.5 %) who indicated that they were distracted sometimes during the experiment, did not affect the results. Furthermore, the group starting with the strategy to identify with the reactions reported being less focused on the task during the experiment (*M* = 2.50, *SD* = 1.50)

when compared to the groups starting with the strategy to react, $M = 3.73$, $SD = 1.28$, $t(42) = -2.92$, $p < .01$, $d = .88$. Since this variable was not correlated with startle amplitude or latency (all p's $\geq .23$), it was not necessary to further control for these effects. There were no further significant correlations between control variables (see Table 4.) and outcome measures across conditions, all p's $\geq .07$.

Items of the form used for manipulation checks	*M* (*SD*) or *n*
Vigilance (Range: 0-6)	3.11 (1.51)
Nervousness (Range: 0-6)	1.66 (1.55)
Subjective effort required for the task (Range: 0-6)	3.27 (1.25)
Corrected vision	22
Do you have any hearing issues?	0
Was your gaze focused on pictures all the time? (yes)	42
Did any of the faces look familiar to you? (yes)	8

Table 4: Means and standard deviations or frequencies for items of the manipulation check form filled out at the end of the experiment.

2.1.3.5 Effects of the Manipulation

2.1.3.5.1 Ratings of Stimulus Properties

As expected, for ratings of valence there was a main effect of Facial-Expression, $F(2, 84) = 201.27$, $p < .001$, partial-$\eta^2 = .83$. Planned contrasts revealed that happy faces were rated more positive ($F(1, 42) = 233.84$, $p < .001$, partial-$\eta^2 = .85$) and angry faces were rated more negative than neutral ones, $F(1, 42) = 66.63$, $p < .001$, partial-$\eta^2 = .61$, see Table 5. There were no effects related to Strategy, all p's $\geq .09$.

For ratings of arousal, there was also an expected main effect of Facial-Expression, $F(2, 84) = 24.86$, $p < .001$, partial-$\eta^2 = .37$. Planned contrasts further confirmed that both happy faces

($F(1, 42) = 40.58$, $p < .001$, partial-$\eta^2 = .49$) and angry faces ($F(1, 42) = 22.92$, $p < .001$, partial-$\eta^2 = .35$) were rated more arousing than neutral ones. Furthermore, happy faces were rated more arousing than angry faces, $F(1, 42) = 4.13$, $p < .048$, partial-$\eta^2 = .09$. In addition, there was a main effect of Strategy ($F(1, 42) = 17.26$, $p < .001$, partial-$\eta^2 = .29$), indicating that arousal ratings were generally lower when the participants used the strategy to identify with the displayed emotional expression.

2.1.3.5.2 Ratings of Successful Strategy Implementation

For ratings of successful implementation of the instructed emotion regulation strategy, there was also a main effect of Facial-Expression, $F(2, 84) = 9.55$, $p < .001$, partial-$\eta^2 = .19$.

Rating	Strategy	Facial-Expression		
		Angry	Neutral	Happy
		M (*SD*)	*M* (*SD*)	*M* (*SD*)
Valence	*Identify*	-1.64 (0.75)	-.64 (0.64)	1.60 (1.29)
	React	-1.71 (1.02)	-.61 (0.78)	1.92 (1.07)
Arousal	*Identify*	5.25 (1.51)	4.47 (1.43)	5.58 (1.41)
	React	5.45 (1.51)	4.31 (1.47)	5.80 (1.49)
Self-reported success of	*Identify*	5.29 (1.94)	5.21 (1.45)	6.04 (1.76)
implementing strategy	*React*	5.58 (1.56)	4.92 (1.63)	6.33 (1.52)

Table 5: Means and standard deviations for ratings of arousal (1 = not at all, 9 = extremely), valence (-4 = extremely negative, 0 = neutral, +4 = extremely positive), and subjective success of implementing the emotion regulation strategies (*identify* vs. *react*: 1 = not at all, 9 = perfectly) for the different facial expressions (angry, neutral, happy) when participants identified vs. focused on their reaction to the stimuli.

Both strategies were more successfully applied with happy faces as compared to both neutral ($F(1, 42) = 18.25$, $p < .001$, partial-$\eta^2 = .30$) and angry faces, $F(1, 42) = 7.97$, $p < .01$, partial-$\eta^2 = .16$, see Table 5. There was no difference between neutral and angry faces, $F(1, 42) = 2.08$, $p < .16$, partial-$\eta^2 = .05$.

2.1.3.5.3 Startle Eye-Blink Amplitude

For dependent variable *startle amplitude*, there were no main effects of Strategy ($F < 1$) and no interaction of Strategy by Facial-Expression, $F(2, 84) = 1.45$, $p < .24$, partial-$\eta^2 = .03$. A marginally significant effect of Facial-Expression ($F(2, 84) = 2.93$, $p < .06$, partial-$\eta^2 = .07$) did not support the expected linear relationship between stimulus valence and startle modulation.

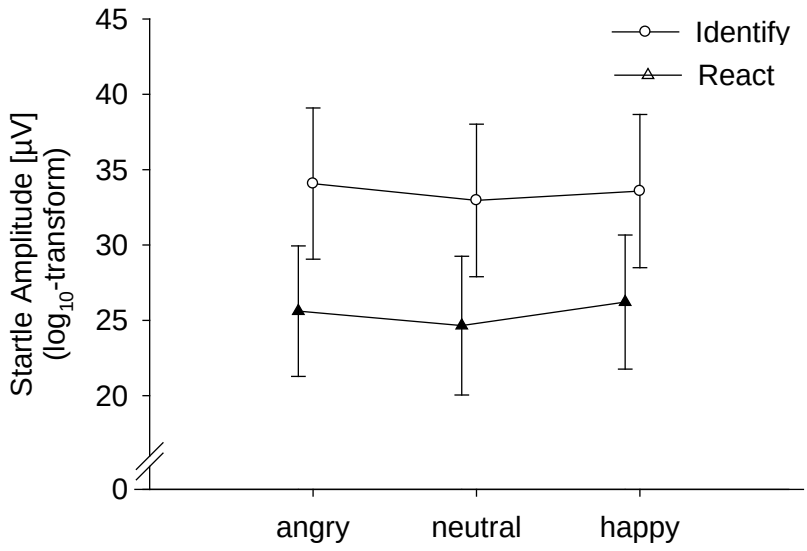

Figure 8: Means of $\log_{10}$-transformed startle amplitudes [µV] to probes presented during processing of facial expression (angry, neutral, and happy) when one of two emotion regulation strategies was applied (*identify* vs. *react*). Error bars represent standard errors of the mean.

However, there was a significant quadratic trend ($F(1, 42) = 5.60$, $p < .02$, partial-$\eta^2 = .12$), suggesting that startle amplitudes were increased during processing of both positive and negative facial expressions as compared to neutral faces; see Figure 8. Finally, a significant interaction of Strategy by Block-Order ($F(1, 42) = 95.59$, $p < .001$, partial-$\eta^2 = .70$) indicated characteristic habituation from the first ($M = 1.35$, $SD = .39$) to the second block, $M = 1.20$, $SD = .41$. It has been recommended to use intra-individual T-scores for this analysis to reduce inter-personal variance (e.g. Bonnet, Bradley, Lang, & Requin, 1995). However, an alternative analysis using this strategy did not lead to different findings.

2.1.3.5.4 Startle Eye-Blink Latency

For dependent variable *startle latency*, there were no significant main effects or two-way interactions, all *F*'s < 1; see Figure 9.

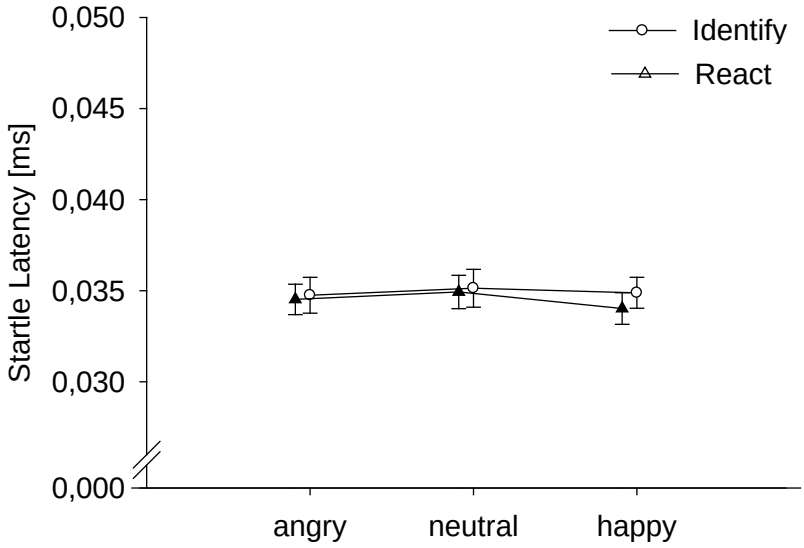

Figure 9: Means of startle latencies [ms] to probes presented during processing of facial expression (angry, neutral, and happy) when one of two emotion regulation strategies was applied (*identify* vs. *react*). Error bars represent standard errors of the mean.

Finally, the three way interaction of Strategy, Valence and Block-Order was also not significant, $F(2, 84) = 1.37$, $p < .26$, partial-$\eta^2 = .03$. Using intra-individually computed T-scores to reduce inter-personal variance for this analysis did not affect the results.

2.1.3.5.5 Associations with Trait Social Anxiousness

Higher levels of trait social anxiety (SPAI; $r = -.36$, $p < .02$), higher social anxiety as measured with the SCS-D ($r = -.37$, $p < .02$) and lower *extraversion* (NEO-FFI, $r = .36$, $p < .02$) were correlated with shorter startle latencies to neutral facial expressions when participants focused on their response to the facial expressions, see scatterplot in appendix 5.1.4, p.208. Furthermore, higher *conscientiousness* (NEO-FFI) correlated with lower startle amplitudes in all conditions, $r = -.30$ to $r = -.22$, all *p's* $< .05$.

Of note, startle amplitude and latency as well as difference scores for effects related to emotional vs. neutral facial expressions were not correlated with ratings of valence, arousal, or successful strategy implementation, all *p*'s $\geq .15$.

2.1.3.5.6 Exploratory Analyses

SPAI scores correlated significantly with negative affective state (PANAS-NA, $r = .36$, $p < .02$), social anxiety measured with the SCS-D (T_0: $r = .80$, $p < .001$, T_2: $r = .75$, $p < .001$, T_3: $r = .77$, $p < .001$,), general state anxiety at T_1 (STAI-state, $r = .41$, $p < .01$), NEO-FFI subscales *neuroticism* ($r = .61$, $p < .001$), *extraversion* ($r = -.44$, $p < .01$), and *conscientiousness* $r = -.34$, $p < .03$. In line with these findings, social anxiety as assessed with the SCS-D was positively correlated with *neuroticism* ($r = .38$, $p < .01$) and negatively correlated with *extraversion*, $r = -.40$, $p < .01$.

In addition, participants were identified with a descriptive pattern of valence related startle modulation. However, only 2 and 6 participants had the expected pattern in the conditions "identify" vs. "respond" respectively. None showed this pattern in both conditions.

Finally, we identified a subgroup of $n = 10$ participants showing arousal related startle modulation in descriptive data for both strategies (identify: $n = 22$; react: $n = 17$). In this subgroup, a significant startle potentiation was found to both angry and happy facial expressions when compared to neutral ones, $F(2, 16) = 13.30$, $p < .001$, partial-$\eta^2 = .62$. However, the amount of potentiation did not correlate with measures of social anxiety, all p's $\geq .14$.

There was no arousal related effect for dependent variable latency of the startle response, $F < 1$.

Finally, the group showing arousal related startle modulation did not differ significantly from the rest of the sample in any startle or questionnaire data, all p's $\geq .09$.

2.1.4 Discussion

Against the hypothesis, findings of Experiment 1 indicated that startle responding was not modulated by the valence of facial expressions (angry and happy vs. neutral), but startle amplitudes were increased to both positive and negative emotional facial expressions as compared to the neutral ones.

Notably, valence and arousal ratings suggest that the participants clearly associated positive vs. negative emotional responding to happy vs. angry facial expressions in comparison to neutral facial expressions. This indicates dissociations between emotional response systems (i.e. self-report vs. physiology). However, this is not the first experiment that did not find affective modulation of the startle reflex by facial expressions despite emotion-specific effects of the stimuli on other dependent variables (e.g. Alpers & Adolph, 2006; Schmidt-Daffy, 2006; Spangler, Emlinger, Meinhardt, & Hamm, 2001). Therefore, it has been speculated that facial expressions may generally not engage the defensive motivational system described by Lang, Bradley, and Cuthbert (1990). Facial expressions could be a universal safety signals,

due to their global presence, the ubiquity of social contact, and the hereditary importance of social affiliation for survival.

An alternative explanation for the findings in experiment 1 may be that facial expressions do not induce sufficient arousal to elicit affective modulation of the startle response. Similarly, pictures with arousal ratings below 6 within the standardized IAPS (Lang, Bradley, & Cuthbert, 1999) do not reliably induce affective startle modulation (Cuthbert, Bradley, & Lang, 1996). Facial expressions are processed via specialized pathways (Kanwisher, McDermott, & Chun, 1997). Therefore, a direct comparison of stimulus properties between IAPS pictures and facial expressions warrants some caution. The arousal ratings obtained in experiment 1 were on the margin of the threshold reported by Cuthbert et al. (1999). Consequently, one strategy to boost effects of facial expressions would be to increase their arousal. However, in a previous study, socially-relevant stimuli from the IAPS picture system with standardized SAM-ratings in the range suggested for reliably inducing affective startle modulation did not lead to the expected effects. Only in a subgroup of individuals with specific public speaking phobia the expected effect emerged as a trend (Gros, Hawk, & Moscovitch, 2009). Nonetheless, strategies to boost stimulus arousal should be explored in future experiments.

A third explanation is supported by recent brain imaging studies. An increasing number of more recent studies show amygdala involvement in the processing of several or even all emotional facial expressions (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Iidaka et al., 2001; Kim, Somerville, Johnstone, Alexander, & Whalen, 2003; Wang, McCarthy, Song, & Labar, 2005; Yang et al., 2002). Currently the resolution of brain imaging in these studies prevents differentiation of the individual contribution of single nuclei and substructures of the amygdala. Therefore it is not clear whether this activation involves the central nucleus of the amygdala, which is responsible for affective modulation of the startle response (Angrilli et al.,

1996). Moreover, increased activation in fMRI does not differentiate whether the underlying neurons are excitatory or inhibitory. Increased activation could therefore even indicate firing of both excitatory and inhibitory neurons. Nonetheless, these findings could explain why startle amplitudes were increased to both happy and angry facial expressions. Consequently, findings in experiment 1 may rather index the emotional salience or arousal of the stimuli than their content related threat-potential.

The lack of affective startle modulation in the current experiment complicates interpretation of findings related to the application of emotion regulation strategies. First, both strategies did not affect startle responding to angry in comparison to neutral or happy facial expressions. Second, both startle amplitude and latency were not correlated with ratings of valence, arousal, or successful strategy implementation. This is at odds with predictions derived from the working model on mediators of social anxiety (section 1.3.8 p. 52). Nonetheless, arousal ratings were generally lower when the participants used the strategy to identify with the displayed emotional expression. This suggests that strategy-use affected emotional evaluation of the stimuli on a conscious cognitive level, but did not affect the activation of the defensive motivational system via top-down modification. While this can not rule out effects of external social threat-cues in general, it indicates dissociations between conscious cognitive evaluation and automatic processing of environmental threat-cues.

The lack of affective startle modulation further complicated assessment of the hypothesis that biased processing of external social threat-cues is a mediator of social anxiety; see the working model in section 1.3.8 p. 52. However, the significant increase of startle amplitudes to both angry and happy facial expressions as compared to neutral ones was not associated with levels of trait social anxiousness. Therefore, experiment 1 did not support the working model on mediators of social anxiety.

However, increased trait social anxiety correlated with shorter startle latencies during processing of neutral pictures specifically when participants focused on their response to the facial expressions. It is unlikely that individuals with low trait social anxiousness responded with particularly strong arousal to these stimuli. Ratings of the stimuli also speak against this interpretation. Therefore, decreased startle latency featured by the participants with higher levels of trait social anxiousness may index increased sensitivity of the motor system, which governs the motor component of the startle response (Britt & Blumenthal, 1993). Processing of the more attention capturing emotional facial expressions or during application of the counterintuitive and probably more effortful emotion-regulation strategy of identifying with the facial expressions may have disrupted this effect. Notably, a similar strategy (i.e. instructions to focus attention on environmental stimuli) has been shown to decrease anxiety during public speaking exposure therapy, and had a boosting effect on therapeutic success (Wells & Papageorgiou, 1998).

Limitations of this experiment include the lack of a control condition with stimuli which have reliably induced affective startle modulation in previous studies (e.g. intense IAPS pictures). Although these effects are found in numerous studies (e.g. Lang, 1995; Lang, Bradley, & Cuthbert, 1990) this would have served well as a manipulation check to confirm that affective startle modulation could have been reliably evoked. In addition, reliance on an unselected student sample limits the generalizability of the results. Furthermore, startle modulation to different emotional stimuli only emerged on a trend level. Therefore, all interpretations related to this effect should be considered preliminary until they are replicated in future studies. Despite much support for the dimensional view on social anxiety (McNeil, 2001), individuals with clinical levels may respond with higher arousal to external social threat-cues. Above a certain threshold of trait social anxiousness, this may lead to the expected affective modulation of startle responding. Therefore, a future study should examine fear potentiation

of the startle to external social threat-cues in individuals with SAD. Finally, startle responses were not assessed in a control condition without a socially evaluative context (i.e. video-manipulation). Cornwell, Johnson, Berardi, and Grillon, (2006) have found that a positive correlation of trait social anxiety and startle amplitude depended on situational context. Interestingly, this effect disappeared when the participants focused their attention away from the internal response to emotional stimuli. Manipulations of attention focus across different levels of situational challenge may help to better understand the underlying cause of such variations.

In sum, the findings of experiment 1 provide preliminary support for fear potentiated startle modulation to facial expressions. Low arousal induced by the stimuli is a likely explanation why startle potentiation to positive and negative stimuli emerged, rather than the linear pattern which is characteristic for affective startle modulation. However, alternative explanations cannot be ruled out. Notably, startle modulation was not correlated with trait social anxiousness. Therefore, experiment 1 provides no support for mediation by external social threat-cues, as suggested by the working model on mediators of social anxiety (section 1.3.8 p. 52). Furthermore, evidence for associations between trait social anxiousness and startle latency was found. However, this effect may rather reflect motor hyper-responsivity of socially anxious individuals than biased processing of external social threat-cues. Processing emotional stimuli and the strategy of identifying with emotional expressions disrupted this association. Effects of such a strategy should be further examined as a tool during exposure therapy of social anxiety disorder. Similar to instructed focus on external stimuli, this may decrease symptoms of social anxiety and improve therapeutic success (Wells & Papageorgiou, 1998).

2.2 Experiment 2 - Attentional vs. Affective Startle Reflex Modulation to External Social Threat-Cues in Patients with Social Anxiety Disorder

2.2.1 Background and Hypotheses

Experiment 1 showed an association between trait social anxiousness and effects of external social threat-cues on startle responding. However, in contrast to the hypothesis, there was no affective modulation of the startle response (i.e. potentiation to angry and attenuation to happy facial expressions in comparison to neutral ones). On the other hand, in experiment 1 increased startle amplitudes to both angry and happy as compared to neutral facial expressions emerged. One explanation for these effects may that stimuli were only differentiated according to their arousal. Brain imaging studies suggest that this may be the case with emotional facial expression (e.g. Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006). An alternative interpretation is that the stimuli did not induce sufficient arousal to induce a differential response to angry vs. happy facial expressions. Similar findings have been reported for other positive and negative stimuli, when their arousal level was below a certain threshold (Cuthbert, Bradley, & Lang, 1996). Above this level of arousal, stimuli with the same level of valence elicited the typical affective modulation also found with highly intense positive and negative stimuli. A similar threshold might exist for the interaction of trait social anxiousness and biased information processing. Above a certain level of trait social anxiousness, the same facial expressions might elicit sufficient arousal to produce affective startle modulation.

As detailed above, affective startle modulation depends on the output of the central nucleus of the amygdala. Positron emission tomography studies show that higher dispositional negative affect correlates with an increased metabolic rate in the right amygdala (Abercrombie et al., 1998). Higher levels of trait fearfulness predict more intense fear potentiation of the startle

response (Cook, Davis, Hawk, Spence, & Gautier, 1992; Cook, Hawk, Davis, & Stevenson, 1991).

Increased levels of trait social anxiousness further predicted higher amygdala activation to threatening facial expressions (e.g. Phan, Fitzgerald, Nathan, & Tancer, 2006). Finally, research on specific phobia has shown that phobic stimuli (e.g., snakes or spiders) which are positive or non-threatening to healthy controls elicit increased amygdala activation and fear potentiated startle responses in individuals with the corresponding specific phobia (Pissiota et al., 2003). Therefore, it was expected that affective modulation of the startle response to facial expressions emerges in participants with a clinical diagnosis of social anxiety disorder.

In addition, experiment 2 aimed at differentiating whether the presumed threat-cue bias in socially anxious individuals is primarily an implicit interpretation bias or an attention bias. Prepulse inhibition (PPI) of startle amplitude is thought to index attention allocation to the stimuli, when startle responses are elicited shortly after stimulus onset (e.g. Filion, Dawson, & Schell, 1993). Consequently, PPI is stronger with more arousing stimuli (Bradley, Cuthbert, & Lang, 1993). If individuals with SAD feature attentional bias, effects should primarily emerge in PPI, and may wear off at later probe times. In addition, weak but noticeable effects may lead to increased startle amplitude to both positive and negative stimuli at later probe times. Noably, when angry facial expressions specifically attract the attention of socially anxious individuals, this would further decrease affective modulation. In contrast, interpretation bias should primarily emerge in affective modulation of startle responding elicited at later probe times. Finally, sustained inward focus of attention induced by a socially challenging context may decrease PPI (Filion, Dawson, & Schell, 1993). However, this would emerge in correlations with trait social anxiety across all types of foreground stimuli.

Only one study so far has compared PPI (probe delay: 120 ms) vs. affective startle modulation (probe delay: 3800 ms) to angry, neutral, and happy facial expressions (White, 2002). In this

study, individuals with SAD had attenuated startle responses to regular displays as compared to inverted angry and happy facial expressions at a stimulus-onset to probe delay of 3800 ms, and attentional PPI to emotional vs. neutral facial expressions at 120 ms. While this does not contradict attentional bias, it specifically supports the assumption of an implicit interpretation bias, specifically related to external social threat-cues.

In addition to fear potentiated startle, spontaneous EMG of facial reaction is a sensitive indicator of automatic emotional responding to facial emotion of interaction partners. Numerous studies have shown that people show corresponding facial reactions (e.g. Dimberg, 1982; Likowski, Mühlberger, Seibt, Pauli, & Weyers, 2008) as well as emotion contagion (Hatfield, Cacioppo, & Rapson, 1994), when they process pictures of facial expressions. Moreover, it has been shown that socially anxious individuals respond with particularly increased corrugator activity to angry faces and increased zygomaticus activity to happy faces as compared to neutral ones (Dimberg & Thunberg, 2007). Finally, modulation of spontaneous corrugator activity to emotional facial expressions has been reported as a manipulation check in previous studies on startle responding to facial expressions, in order to demonstrate that facial stimuli produce a distinct emotional response in participants (Spangler, Emlinger, Meinhardt, & Hamm, 2001). Corrugator activity after stimulus onset was expected to increase with negative and to decrease with positive stimuli. Furthermore, it was expected that higher levels of trait social anxiousness would be associated with stronger increases of corrugator activity to angry facial expression in comparison to both neutral and happy facial expressions.

Experiment 2 investigated whether patients with a clinical diagnosis of SAD feature particular strong attentional bias (PPI) and implicit affective evaluation bias (fear potentiated startle reflex) to emotional facial expressions (angry, neutral, and happy). PPI was expected to reflect attentional protection of processing more arousing stimuli. Fear potentiated startle was

expected to co-vary with the valence of stimuli (potentiation to negative vs. attenuation to positive stimuli, when compared to neutral ones). This approach details the potential role of attention vs. interpretation bias to external social-threat-cues proposed in the working model on mediators of social anxiety in section 1.3.8, p. 52.

2.2.2 Methods

2.2.2.1 Design and Participants

The design was a 3 (Facial-Expression: happy, neutral, angry) x 2 (Startle probe timing: early, vs. late) within-subjects design. Repeated assessment of the time course of corrugator supercilii EMG activity was additionally incorporated in the respective analyses (EMG-Bin: intra-individual z-scores of 500 ms bins of averaged EMG activity change from baseline [i.e. the mean of 500 ms EMG activity before stimulus onset] across the 8000 ms of stimulus presentation).

Nine patients with a principal diagnosis of social anxiety disorder (SAD) were recruited via the psychotherapeutic outpatient clinic of the Department of Psychology 1, University of Wuerzburg, Germany. When patients received a clinical diagnosis of social anxiety disorder (ICD-10, WHO, 1992), the active therapist asked them whether they would like to participate in a research experiment. A brief introductory text was presented to inform them about the experiment, see appendix 5.2.1, p.209. If they accepted this invitation, an appointment was scheduled for the experiment. It was pointed out to the patients that they could withdraw their consent to participate in the study any time without loss of any benefits they would have been entitled to otherwise. Inclusion criteria were a principal diagnosis of SAD, being 18 years of age or older, normal or corrected vision, and signed written informed consent. Exclusion criteria were intake of alcohol or drugs less than eight hours prior to the experiment, tinnitus or hearing impairment.

2.2.2.2 Apparatus

Instructions, picture stimuli, and startle probes were presented using Presentation® software (v.0.80, www.neuro-bs.com) running on a Pentium-level computer. Participants' heads were at approx. 40 cm distance to a 19" flat screen CRT-monitor (Fujitsu-Siemens 19P4; 1024x768, 85 Hz). Startle probes of 50 ms, 105 dB(A) bursts of white noise with near instantaneous rise/fall times (Lang, 1995) were presented via a SoundBlaster Audigy sound card connected to a Kenwood KA-3010 amplifier and played binaurally through Bayer Dynamik dt 901 headphones. Eye-blink startle responses were measured electromyographically (EMG) from sites over the orbicularis oculi, using No. 21708305 Ag/AgCl surface electrodes (GE Medical Systems) placed about 1 cm below the pupil and outer canthus of the left eye. Spontaneous corrugator supercilii activity was also measured via EMG using the same type of electrodes placed according to the guidelines published by Fridlund and Cacioppo (1986). The raw EMG was amplified (frequency response: 19-500Hz), digitally sampled at 1024Hz (16 bit A/D-converter) with a Varioport system (Becker Meditec) and stored to hard-disk.

2.2.2.3 Trait Measures

The German version of the Social Phobia and Anxiety Inventory (SPAI, Fydrich, 2002; English original by Turner, Beidel, Dancu, & Stanley, 1989), a well-established self-report measure of trait social anxiety with excellent psychometric properties was used to assess social anxiety and anxiety related to performance situations. See Table 6 for an overview of the internal consistency achieved for questionnaires with the experiment sample.

The German version of the NEO-FFI (Borkenau & Ostendorf, 1993; English original by Costa & McCrae, 1992) was used to examine potential associations of neuroticism and extraversion with social anxiety.

2.2.2.4 State Measures

The state-version of the Spielberger State Trait Anxiety Inventory (STAI-state, Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was used to examine associations with changes of state-anxiety across the experiment.

Questionnaires and Subscales		Internal Consistency (Cronbach's α)
SPAI		T_0: .96
NEO-FFI	*neuroticism*	T_3: .77
	extraversion	T_3:.35
	openness to new experiences	T_3:.18
	agreeableness	T_3:.52
	conscientiousness	T_3:.71
STAI-state		T_1: .94, T_2: .98, T_3: .98
SCS-D		
	private self-consciousness	T_0: .67, T_2: .89
	public self-consciousness	T_0: .65, T_2: .72
	social anxiety	T_0: .76, T_2: .67
PANAS		
	positive affect (PA)	T_1: .64
	negative affect (NA)	T_1:.92

Table 6: Overview of the internal consistency (Cronbach's α) achieved for questionnaires and their subscales with the experiment sample; see text for details. T_0 = beginning of experiment; T_1 = immediately before beginning of the startle procedure; T_2 = after completion of block 1 of the startle procedure; T_3 = after completion of block 1 of the startle procedure; see procedure for details.

The German version of the Self-Consciousness Scale (English original by Fenigstein, Scheier, & Buss, 1975; SCS-D, Heinemann, 1979) was used to control for aspects of social anxiety related to habitual self-examining. Excessive public self-consciousness may lead to increased attention to oneself as falling short of intrinsic social ideals. This may be an important component of social anxiety, because exaggerated estimation of social cost related to failing such standards has been shown to mediate SAD (Hofmann, 2004).

The Positive and Negative Affect Schedule (PANAS, Krohne, Egloff, Kohlmann, & Tausch, 1996) was used to control for potential interactions of affective state with emotional information processing during the experiment.

2.2.2.5 Procedure

After obtaining signed informed consent (T_0), all participants were asked to complete the SCS-D, the SPAI, and a socio demographics form. Next, the participants were introduced to the experimental setup, including a video system monitoring their non-verbal reactions during the experiment. To create a social evaluative context, participants were informed that the experimenter observed these responses on a monitor in the adjacent room and rated certain response characteristics. The participants were seated in front of the screen used for stimulus presentation and were familiarized with usage of a modified keyboard for collection of ratings; see Figure 7, p. 65. After this, electrodes for physiological data acquisition were attached and the participants put the headphones on. Next, all participants completed the PANAS and STAI-state (T_1).

Following, one block of 24 stimuli (eight sets of angry, happy, and neutral facial expressions, posed by eight different actors, respectively) was presented in random order. The participants were asked to look at the stimuli for the duration they were presented (8 s). A black screen served as an inter-stimulus interval (ISI, duration: 10 s ± 2 s). In the first block, startles were elicited at 4.5 s ± 0.5 s after stimulus onset, with fixed assignment to 6 out of 8 stimuli per

category, to assess effects of the foreground stimuli on the activation of affect induced action tendencies (Lang, 1995).

Following each picture, ratings of current emotional state in response to the picture (valence: -4 = extremely negative, 0 = neutral, +4 = extremely positive), rating of felt arousal (1 = not at all, 9 = extremely), ratings of „how appropriate was your response to the emotion on display?" (appropriateness: 1 = not at all, 9 = perfectly) were assessed.

After completion of a first block (T_2), the participants completed another set of STAI-state and SCS-D forms. Next, they continued with a second block that matched the first one with the only difference that startles were elicited at 120 ms after stimulus onset. After completion of the second block (T_3), the participants filled out a final STAI-state, the NEO-FFI. After a short manipulation check interview they were fully debriefed.

2.2.2.6 Data Reduction and Analysis

EMG-recordings of the eye-blink startle responses and spontaneous corrugator supercilii activity were processed offline (digital 50 Hz notch filter to minimize AC-leakage, 4th order Butterworth lowpass-filter at 500 Hz, 4th order Butterworth highpass-filter at 20 Hz, and rectified, see Blumenthal et al., 2005; van Boxtel, 2001). Startle responses were smoothed with a 100 ms moving average for amplitude scoring, and a 20ms moving average for latency scoring, corrugator supercilii EMG was smoothed with a 1000 ms moving average. Startle data was scored using a custom MATLAB (Mathworks, Inc., Natick, MA) program (Schulz & Alpers, 2007) which implements criteria specified by Blumenthal and colleagues (2005). Responses with amplitudes equal or smaller than zero and reactions with artifacts were excluded from the analysis (8 %). There were no startle non-responders. Startle amplitudes were computed by subtracting the average of 20ms of data before startle-onset from the maximum amplitude identified between 20 and 120 ms after startle-onset and averaged across all responses within cells of the experiment design. Startle amplitudes were square root

transformed to achieve normal distribution. K-S tests confirmed the success of the transformation for startle amplitude within each stimulus category, all *p*'s ≥ .81. K-S tests for startle latency showed that no transform was necessary, K-S test, all *p*'s ≥ .90. For the statistical analysis, data on corrugator supercilii activity was averaged in 500 ms bins across the 8000 ms of stimulus, and change scores to a baseline across 500 ms before stimulus onset were computed. These scores were intra-individually z-transformed to reduce inter-personal variance.

2.2.2.7 Statistical Analysis

To compare effects across the two blocks with different probe timing (within subjects factor Probe-Timing: 120 ms vs. 4.5 s ± 0.5 s), repeated measures ANOVAs were computed with for dependent variables startle amplitude and startle latency (within-subjects factor Facial-Expression: angry, neutral, and happy). A similar repeated measures ANOVA for dependent variable amplitudes of corrugator EMG assessed in the two conditions (Probe-Timing: early vs. late probe time) also included repeated measures of corrugators supercilii EMG activity (within-subjects factor EMG-Bin: Intra-individual z-scores of 500 ms bins of averaged EMG activity change from baseline [i.e. the mean of 500 ms EMG activity before stimulus onset] across the 8000 ms of stimulus presentation).

Furthermore, repeated measures ANOVAs (within-subjects factor Facial-Expression: angry, neutral, happy) and planned contrasts were used to examine effects of the experimental manipulation on dependent variables startle amplitude, startle latency, and EMG-data in the two conditions (probe timing at 120 ms vs. 4.5 s ± 0.5 s). Greenhouse-Geisser statistics are reported, when sphericity could not be assumed.

In addition, repeated measures ANOVAs (within-subjects factor Facial-Expression: angry, neutral, happy) were computed for ratings of valence and arousal, to examine the stimulus properties of the facial expressions, as rated by the experiment sample. This analysis was also

performed on ratings of how appropriate the participants felt that their response was to the facial expressions on display.

Bootstrapping was used to gain improved population estimates of parameters and distribution characteristics for effects of stimulus induced modulation of startle amplitude. This allows evaluation of distribution characteristics which may reveal subgroups that may not be apparent in data obtained with small samples (Moore & McCabe, 2005). Furthermore, it provides an impression of the probability of statistical type I vs. type II error in hypothesis testing. This approach was supplemented with post-hoc power analysis to estimate the required sample size for achieving significant startle modulation to facial expressions based on effect-sizes found with the sample of experiment 1.

Pearson correlations were used to investigate associations between personality variables (NEO-FFI, SCS-D), affective state (SPAI, PANAS), startle measures (amplitude and latency), and corrugator EMG-activity, and to examine associations with stimulus ratings (arousal, valence, appropriateness of responding).

Finally, independent *t*-tests, Chi^2-Tests, and Pearson correlations were used for manipulation checks and to describe the sample.

2.2.3 Results

2.2.3.1 Participants

Clinical assessment of the presence of SAD was completed by licensed therapists in the outpatient clinic associated with the Department of Psychology 1, University of Würzburg, Germany. The mean age of the N = 9 participants was M = 26.56 (SD = 7.91), and n = 2 (22.22 %) were female. Most of the participants were right-handers (n = 6, 67 %), about half of the sample had deficient but corrected vision (n = 4, 44 %), and all of them indicated having no hearing issues. All participants identified themselves as Caucasian. Three patients (33.33 %) had a comorbid diagnosis of depression. Two patients received tricyclic

antidepressants and one patient SSRI. One patient received an anticonvulsant as a mood stabilizer.

2.2.3.2 Trait Measures

SPAI scores (*M* = 71.87, *SD* = 25.04, *Min* = 37.05, *Max* = 120.05, scores adjusted for comparison to the US-version of the SPAI, see Fydrich, 2002) were above average, when compared to unselected samples, but below typical patient samples with clinical SAD (e.g. Turner, Beidel, Dancu, & Stanley, 1989).

NEO-FFI Subscale	Sample of Experiment 3 (*n* = 9) *M* (*SD*)	German Reference Sample (*n* = 2112) *M* (*SD*)
neuroticism	29.22 (7.25)	22.1 (8.4)
extraversion	21.13 (4.52)	28.3 (6.8)
openness to new experiences	24.56 (3.97)	32.5 (6.2)
agreeableness	27.22 (5.21)	29.3 (5.9)
conscientiousness	27.78 (6.24)	30.4 (7.6)

Table 7: Means and standard deviations for the subscales of the NEO-FFI in both the sample of experiment 2 and a German reference sample published by Borkenau and Ostendorf (1993, p.13).

Three patients scored below 60, a cutoff considered optimal for excluding non-social-phobic participants.[2] Three participants scored well above the cutoff of 80, which is commonly used as an indicator for a potential clinical diagnosis of social anxiety. NEO-FFI subscale scores

[2] All analyses were repeated without these participants. K-S-tests confirmed normal distribution of startle amplitudes, and startle latencies in this sub-sample, all p's $\geq .63$. However, no significant effects related to stimulus valence or arousal emerged, all p's $\geq .21$.

were comparable to the reference sample reported by Borkenau and Ostendorf (1993, p.13), see Table 7.

2.2.3.3 State Measures

Scores on subscales of the SCS-D remained stable during the experiment, as indicated by a multivariate test with Pillay Spur-Criterion for the two assessments at T_0 and T_2, $F(3, 4) = 2.18$, $p < .23$, partial-$\eta^2 = .62$. The participants scored in a typical range for the general German population (Heinemann, 1979) , see Table 8.

Scale	T_0-Assessment	T_2-Assessment	Mean of Assessments
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
private self-consciousness	3.67 (.52)	3.28 (.82)	3.49 (.59)
public self-consciousness	3.78 (.55)	3.63 (.71)	3.74 (.55)
social anxiety	3.69 (.78)	3.46 (.76)	3.60 (.66)

Table 8: Means and standard deviations for subscales of the Self-Consciousness Scale (SCS-D, German Version); T_0 = beginning of experiment; T_2 = after completion of block 1 of the startle procedure; see procedure for details.

STAI-state assessments suggested that in comparison to age related norms (age group: 15-29 for male subjects: M = 36.56, SD = 9.47 and female subjects: M = 36.96, SD = 10.27, see Spielberger, Gorsuch, & Lushene, 1996) the participants had elevated anxiety that did not vary significantly between (T_1 (*M* = 41.75, *SD* = 12.40), T_2 (*M* = 43.88, *SD* = 15.47; *t*(8) = -.53, *p* < .61, *d* = .15) and T_3 assessments, *M* = 42.92, *SD* = 16.56, *t*(8) = .34, *p* < .74, *d* = .06. According to comparison scores (Krohne, Egloff, Kohlmann, & Tausch, 1996), the participants scored in the normal range on both PANAS scales (*positive affect*: *M* = 25.83, *SD*

= 4.53, *negative affect*: *M* = 14.11, *SD* = 6.85) immediately before beginning with the experimental task (T_1).

There were no differences between female and male participants with regards to age, SPAI, PANAS scales, SCS scales, NEO-FFI subscales *neuroticism*, *openness to experiences*, *agreeableness*, and *conscientiousness*, STAI-state at the three measurement time points, all *p*'s ≥ .15. The comparison for NEO-FFI subscale extraversion was not possible due to missing scores from one male participant, reducing the male sample to n = 1.

Finally, the form used for a manipulation check at the end of the experiment suggested that instructions were implemented correctly. The participants indicated no other factors which may have compromised the data, see Table 9.

Items of the form used for manipulation checks	*M* (*SD*) or *n*
Vigilance (Range: 0-6)	2,56 (1.51)
Nervousness (Range: 0-6)	2 (1.58)
Subjective effort required for the task (Range: 0-6)	1,89 (1.76)
Corrected vision	4
Do you have any hearing issues?	0
How displeasing was the video observation? (Range: 0-6)	1,67 (1.80)
Was your gaze focused on pictures all the time? (yes)	7
Did any of the faces looked familiar to you? (yes)	1

Table 9: Means and standard deviations or frequencies, for items of the manipulation check form filled out at the end of the experiment.

2.2.3.4 Ratings

As expected, for ratings of valence there was a main effect of Facial-Expression $F(2, 16) = 16.59$, $p < .001$, partial-$\eta^2 = .68$. Planned contrasts revealed that happy faces were rated more positive ($F(1, 8) = 8.82$, $p < .02$, partial-$\eta^2 = .52$) and angry faces were rated more negative

than neutral ones, $F(1, 8) = 18.73$, $p < .01$, partial-$\eta^2 = .70$, see Table 10. There were no effects related to Probe-Timing, all p's $\geq .19$.

Unexpectedly, for ratings of arousal, there was no main effect of Facial-Expression, $F(2, 16) = 2.30$, $p \geq .13$, partial-$\eta^2 = .22$. There were no effects related to Probe-Timing, all p's $\geq .64$.

For ratings of appropriateness of one's emotional response to the facial expressions, there was no significant main effect of Facial-Expression ($F(1.24, 9.93) = 2.71$, $p \geq .12$, partial-$\eta^2 = .25$) and no effects related to Probe-Timing, all p's $\geq .58$.

Rating	Probe-Timing	Facial-Expression		
		angry	neutral	happy
		M (*SD*)	*M* (*SD*)	*M* (*SD*)
Valence	*120 ms*	-1.63 (1.09)	-.48 (.40)	.82 (1.24)
	4.5 ± 0.5 s	-1.46 (1.22)	-.32 (.85)	1.09 (1.05)
Arousal	*120 ms*	4.76 (2.18)	4.06 (1.19)	5.22 (1.67)
	4.5 ± 0.5 s	4.94 (1.78)	4.06 (1.06)	5.35 (1.44)
Appropriateness	*120 ms*	5.13 (2.01)	4.98 (1.70)	5.98 (1.60)
	4.5 ± 0.5 s	5.06 (1.46)	5.22 (1.46)	6.04 (1.78)

Table 10: Means and standard deviations for ratings of arousal (1 = not at all, 9 = extremely), valence (-4 = extremely negative, 0 = neutral, +4 = extremely positive), and appropriateness of one's emotional response (1 = not at all, 9 = perfectly) regarding the different facial expressions (angry, neutral, happy) when startle probes were elicited at 120 ms vs. 4.5 ± 0.5 s after stimulus onset.

2.2.3.5 Effects of the Manipulation

2.2.3.5.1 Startle Eye-Blink Amplitude

For dependent variable *startle amplitude at 120 ms*, there was no significant main effect Facial-Expression, $F < 1$, partial-$\eta^2 = .08$. For dependent variable *startle amplitude at 4.5 s ± 0.5 s*, there was also no significant main effect Facial-Expression, $F(2, 16) = 1.40$, $p < .28$, partial-$\eta^2 = .15$, see Figure 10.all other main effects and interactions, $F < 1$. Performing these analyses with intra-individually computed T-scores to reduce inter-personal variance did not affect these findings.

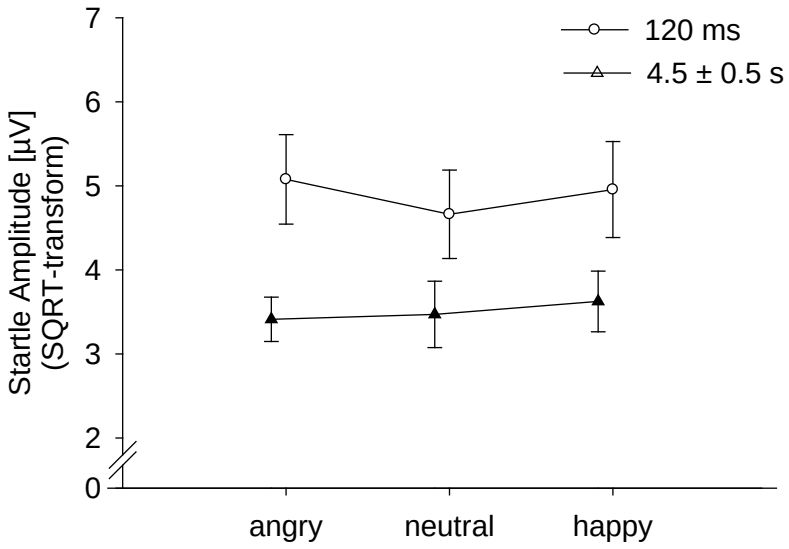

Figure 10: Means of square-root transformed startle amplitudes [µV] to probes presented early (120 ms) vs. late (4.5 ± 0.5 s) after stimulus onset during processing of facial expression (angry, neutral, and happy). Error bars represent standard errors of the mean.

2.2.3.5.2 Startle Eye-Blink Latency

For dependent variable *startle latency at 120 ms*, there was no significant main effect Facial-Expression, $F < 1$, partial-$\eta^2 = .01$, see Figure 11. For dependent variable *startle amplitude at 4.5 s ± 0.5 s*, there was also no significant main effect Facial-Expression, $F < 1$, partial-η^2 =

.06, all other main effects and interactions, $F < 1$. Performing these analyses with intra-individually computed T-scores to reduce inter-personal variance did not affect these findings.

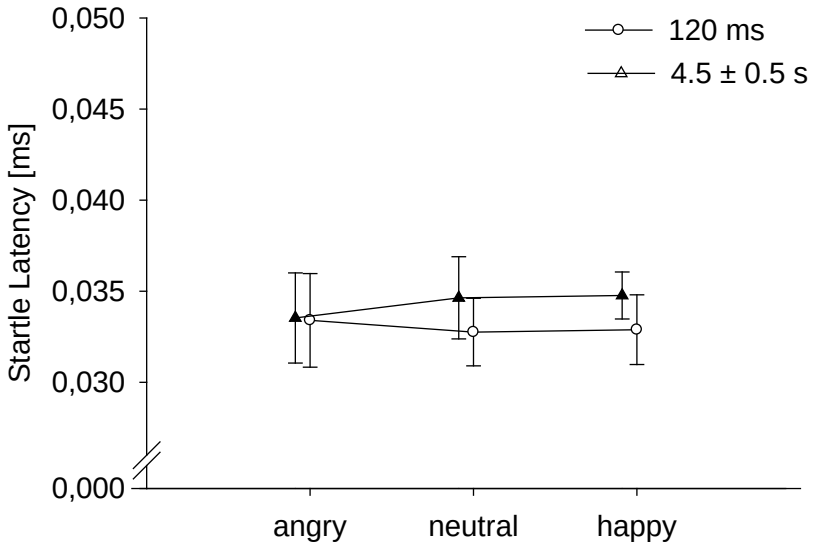

Figure 11: Means of startle latencies [ms] to probes presented early (120 ms) vs. late (4.5 ± 0.5 s) after stimulus onset during processing of facial expression (angry, neutral, and happy). Error bars represent standard errors of the mean.

2.2.3.5.3 Spontaneous EMG of the Corrugator Supercilii

The repeated measures ANOVA for bins of corrugator EMG-activity in the two conditions with early vs. late startle probes showed a significant main effect of Facial-Expression $F(2, 16) = 6.22$, $p < .01$, partial-$\eta^2 = .64$, see Figure 12 and Figure 13. Furthermore, there was a significant Facial-Expression by Probe-Timing interaction ($F(30, 240) = 5.93$, $p < .001$, partial-$\eta^2 = .43$). There was a marginally significant main effect EMG-Bin, $F(15, 120) = 1.69$, $p < .06$, partial-$\eta^2 = .17$, but no main effect of Probe timing, $F(1, 8) = 1.19$, $p \geq .30$, partial-$\eta^2 = .13$.

Planned contrasts revealed significantly increased activity to angry as compared to happy facial expressions, $F(1, 8) = 20.67$, $p < .001$, partial-$\eta^2 = .72$. In comparison to neutral faces

this effect was only marginally significant, $F(1, 8) = 4.08$, $p < .08$, partial-$\eta^2 = .34$. The comparison of neutral to happy facial expressions was not significant, $F(1, 8) = 1.76$, $p < .22$, partial-$\eta^2 = .18$.

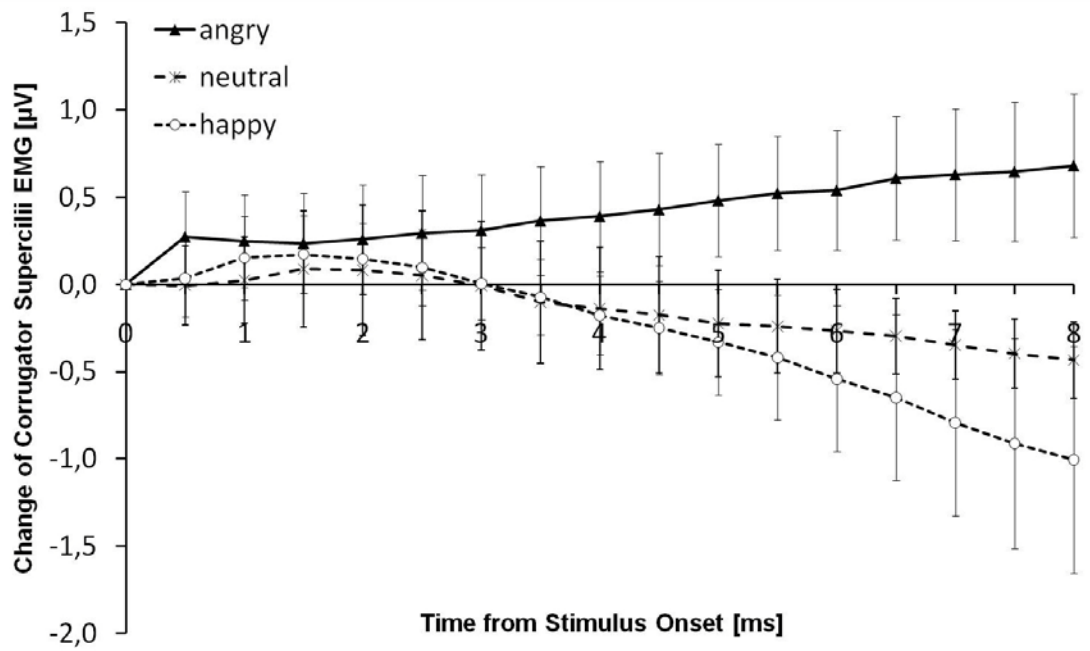

Figure 12: Means of EMG-Amplitude (change scores from baseline assessed across 500 ms of EMG data before stimulus onset) of spontaneous corrugator supercilii activity across bins of 500 ms, over 8000 ms after stimulus onset [intraindividual z-scores] during processing of facial expression (angry, neutral, and happy); data from the second block where startle probes were presented at 120 ms after stimulus onset. Error bars represent standard errors of the mean.

Beginning at 5500 ms, corrugator supercilii activity was marginally significantly reduced to happy as compared to neutral facial expressions, $F(1, 8) \geq 3.59$, $p_{5500ms} < .095$ to $p_{8000ms} < .06$, partial-$\eta^2_{5500ms} = .31$ to partial-$\eta^2_{5500ms} = .38$. Contrasting EMG activity to happy and angry facial expressions reached significance beginning at 3500 ms ($F(1, 8) = 5.56$, $p < .046$, partial-$\eta^2 = .41$), reaching a peak at 7500 ms $F(1, 8) = 18.73$, $p < .003$, partial-$\eta^2 = .70$, and slightly decreased at 8000 ms, $F(1, 8) = 17.83$, $p < .003$, partial-$\eta^2 = .68$.

Finally, a significant triple interaction of Facial-Expression, Probe-Timing, and EMG-Bin emerged, $F(1, 8) = 5.38$, $p < .049$, partial-$\eta^2 = .40$. The increase of EMG activity to angry facial expressions as compared to happy but not neutral facial expressions from 0 to 500 ms was larger in the Probe-Time condition with startle probes presented at 120 ms.

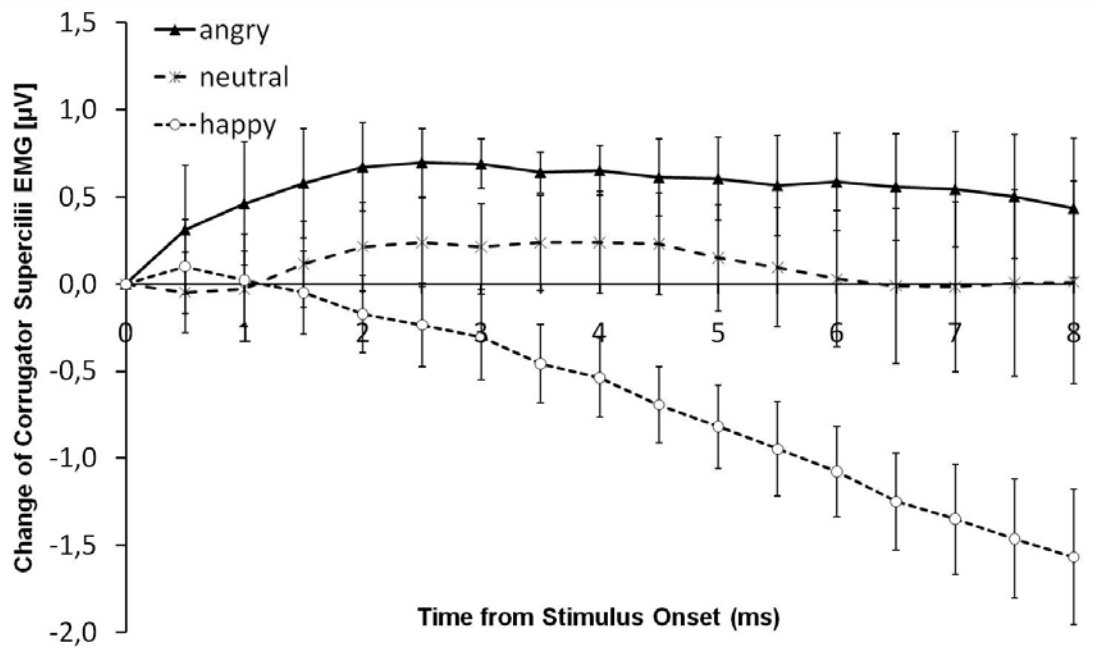

Figure 13: Means of EMG-Amplitude (change scores from baseline assessed across 500 ms of EMG data before stimulus onset) of spontaneous corrugator supercilii activity across bins of 500 ms, over 8000 ms after stimulus onset [intraindividual z-scores] during processing of facial expression (angry, neutral, and happy); data from the first block where startle probes were presented at 4.5 ± 0.5 s after stimulus onset. Error bars represent standard errors of the mean.

2.2.3.5.4 Associations with Trait Social Anxiousness

Trait social anxiety (SPAI) correlated significantly with negative affective state (PANAS-NA, $r = .67$, $p < .05$), *public self-consciousness* ($r = .72$, $p < .03$), and social anxiety measured with the SCS-D ($r = .85$, $p < .01$). Furthermore, *neuroticism* correlated significantly with *public self-consciousness* ($r = .76$, $p < .02$) and social anxiety measured with the SCS-D, $r = .68$, $p <$

.05. Finally, *public self-consciousness* and social anxiety measured with the SCS-D were correlated significantly, $r = .83$, $p < .01$.

There were no correlations between measures of stimulus related startle modulation (latency, and amplitude: difference scores angry – neutral, and angry – happy), with trait social anxiety as assessed with the SPAI (all *r*'s < .37) or with the *social anxiety* subscale of the SCS-D, all r's < .46. There were no significant correlations of affective modulation of the corrugator supercilii responses (difference scores of EMG-amplitude in response to facial expressions: angry – neutral and angry – happy) and the SPAI ($r < .42$, $p \geq .25$) or the *social anxiety* subscale of the SCS-D, all *r*'s < .55, *p*'s < .16.

2.2.3.5.5 Exploratory Analyses

Similar to Experiment 1, we explored subgroups of patients who showed the expected response patterns.

For *startle amplitude at 120 ms* 3 patients showed the expected quadratic relationship related to stimulus arousal. In this subgroup significant startle potentiation was found to both angry and happy facial expressions when compared to neutral ones, $F(2, 4) = 13.97$, $p < .02$, partial-$\eta^2 = .88$. Contrast computations confirmed a marginally significant quadratic trend, $F(1, 2) = 17.39$, $p < .05$, partial-$\eta^2 = .90$. With T-transformed scores, both effects were significant; main effect valence: $F(2,4) = 66.38$, $p < .001$, partial-$\eta^2 = .97$; quadratic trend: $F(1, 4) = 4013.13$, $p < .001$, partial-$\eta^2 \geq .99$.

For *startle amplitude at 4.5 s ± 0.5 s* only one patient showed the expected linear pattern related to stimulus valence. Interestingly, five of nine patients showed an arousal related pattern. In this subgroup, a marginally significant startle potentiation was found to both angry and happy facial expressions when compared to neutral ones ($F(2, 8) = 3.44$, $p < .08$, partial-$\eta^2 = .46$) and contrast computations confirmed the expected quadratic trend, $F(1, 4) = 11.60$, $p < .03$, partial-$\eta^2 = .74$. With T-transformed scores, both effects were significant main effect

valence: $F(2, 8) = 6.24$, $p < .02$, partial-$\eta^2 = .61$; quadratic trend: $F(1, 4) = 265.65$, $p < .001$, partial-$\eta^2 = .99$. This subgroup did not differ significantly from the rest of the sample in any startle or questionnaire data, all p's $\geq .24$. Both subgroups had no significantly elevated scores on any of the measures assessing state or trait social anxiety, all p's $\geq .30$.

2.2.3.5.6 Bootstrapping for Startle Modulation

Bootstrapping was used to determine population characteristics of the difference between responses to angry and neutral facial expressions (amplitude of startle responses elicited at *4.5 s ± 0.5 s).*

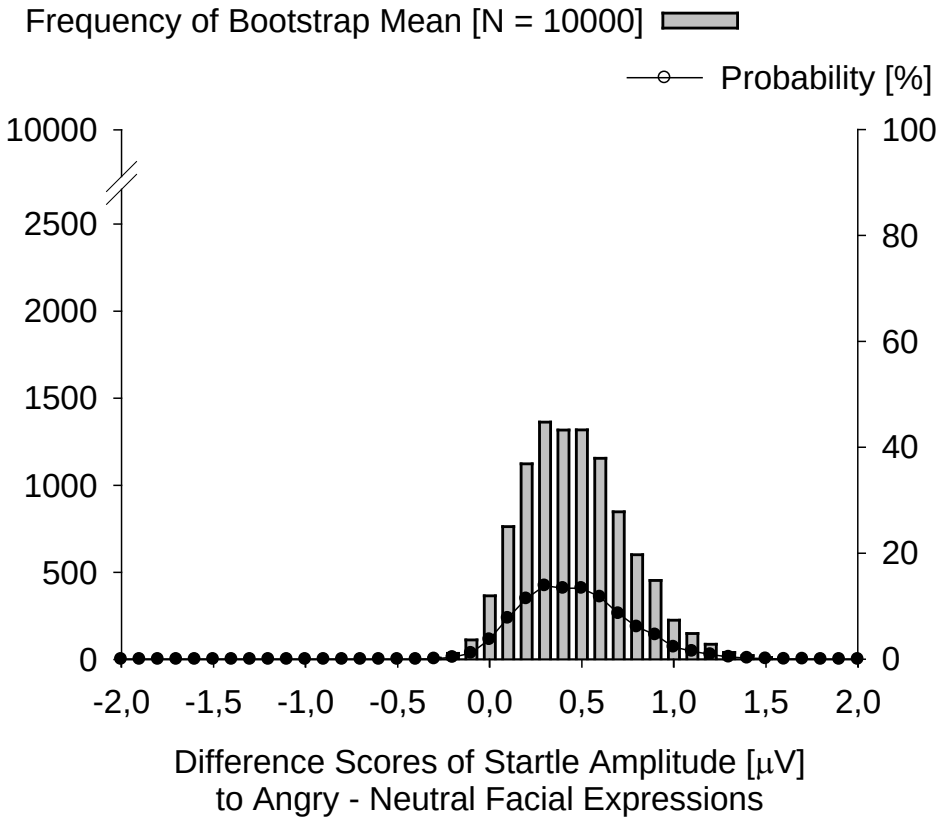

Figure 14: Histogram of the frequency and probability distribution of bootstrapping means for the difference of square-root transformed startle amplitude [µV] elicited at 4.5 ± 0.5 s after stimulus onset. Difference scores were computed for responses elicited during processing of angry minus responses elicited during processing of neutral facial expressions. Positive scores on the x-axis reflect potentiation of the response to angry facial expressions.

Using 10000 drawings of samples size n = 9, a unimodal distribution emerged with a mean bootstrap effect of *M* = .42, *SD* = .86. With an alpha set to .05, the 90% confidence interval had a lower bound of -.003 and an upper bound of .91, indicating that the effect is not significantly different form zero, see Figure 14.

Similarly, bootstrapping (10000 drawings of samples with a size of n = 9) was used to determine population characteristics of the difference between responses to happy and neutral facial expressions (amplitude of startle responses at 4.5 s ± 0.5 s).

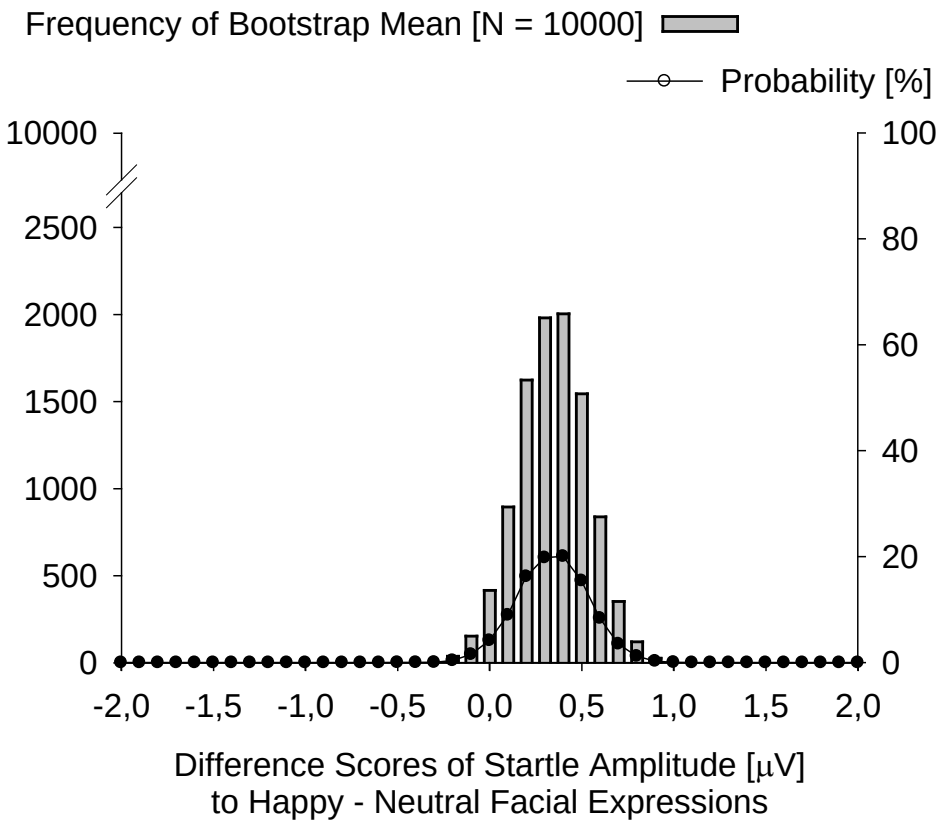

Figure 15: Histogram of the frequency and probability distribution of bootstrapping means for the difference of square-root transformed startle amplitude [µV] elicited at 4.5 ± 0.5 s after stimulus onset. Difference scores were computed for responses elicited during processing of happy minus responses elicited during processing of neutral facial expressions. Positive scores on the x-axis reflect potentiation of the response to happy facial expressions.

Again, a unimodal distribution emerged, with a mean bootstrap effect of $M = 0.29$, $SD = .57$. With an alpha set to .05, the 90% confidence interval had a lower bound of -.02 and an upper bound of .60 indicating that the effect is not significantly different from zero, see Figure 15.

2.2.3.5.7 Post-Hoc Power Analysis of Startle Modulation

Furthermore, *post hoc power analysis* of effects of facial expression on startle latency showed that a minimum sample sizes of $N_{120} = 3533$, $N_{4500} = 38$ would be required to achieve significant main effects related to facial expression when assessed at 120ms, or 4.5 ± 0.5 s respectively. Sample sizes of $N_{120} = 254$, $N_{4500} = 52$, would be required to achieve significant effects on startle amplitude.

2.2.4 Discussion

Against the hypothesis, there was no affective modulation of startle amplitudes by angry and happy vs. neutral facial expressions in experiment 2. This is at odds with the assumption that the interaction of trait social anxiousness, situational challenge, and external social threat-cues leads to increased fear potentiation of the startle response.

This raises the question whether the stimuli were appropriate to elicit significant startle modulation. As expected, ratings of valence suggest that happy and angry facial expressions were perceived as eliciting the expected emotional positive vs. negative responses. Surprisingly, ratings of arousal for neutral facial expressions were as high as ratings for positive and negative facial expressions. Probably, individuals with SAD differentiate primarily the valence of facial expressions, while they experience arousal in all social encounters. Nonetheless, affective startle modulation to happy vs. angry facial expressions may be expected, based on these ratings. On the other hand, ratings indicated that arousal was on the margin for reliably eliciting affective startle responding, when compared to effects reported for emotional pictures from the IAPS picture system (Cuthbert, Bradley, & Lang, 1996). Furthermore, Likert-like rating scales are susceptible to range bias. When endpoints of

these scales are not anchored, participants typically adjust them based on the most extreme stimuli within a given set (Ostram & Upshaw, 1968; Parducci, 1982, 1983). Anchoring endpoints of scales with more elaborate descriptions may lead to much lower arousal ratings, which may more accurately describe the potential of the stimuli to modulate startle responding.

Remarkably, spontaneous EMG activity of the corrugator supercilii varied significantly with the valence of the facial stimuli. Corrugator activity increased during processing of angry facial expressions, and decreased during processing of happy facial expressions, as compared responses to neutral faces. This indicates that the stimuli were not only rated to elicit emotional responding, but had an effect on socially relevant nonverbal behavior. This shows a clear dissociation between emotional responding to pictures of facial expressions across response systems. A potential reason for this finding may be that affective startle modulation requires involvement of the defensive motivational system proposed by (Lang, Bradley, & Cuthbert, 1998), which may depend on sufficiently arousing stimuli. In contrast, EMG responding may rely on different processing pathways, independent of an engagement of the defensive motivational system.

Additional support that the facial expressions used in this experiment have effects on emotional responding in the expected way comes from publications with valence-related modulation of the EEG (Mühlberger et al., 2009), emotional modulation of binocular rivalry (e.g. Alpers & Gerdes, 2007), and reaction time tasks (e.g. face in the crowd paradigm, see Schmidt-Daffy, 2006) using the same stimuli.

This raises the question, why startle responding did not reflect affective responding to the facial expressions. One reason may be methodic shortcomings. However, they are unlikely because significant results have been obtained with the same equipment for example in experiment 1 and previous experiments using IAPS pictures (Adolph, Alpers, & Pauli, 2006;

Alpers & Adolph, 2006). Another possibility is that faces generally do not modulate startle responding. Results from experiment 2 also showed no general potentiation of the startle amplitude during processing of emotional vs. neutral facial expressions. However, this null-finding may also reflect a type II error due to the small sample size. Post-hoc power analysis suggests that startle modulation to facial expressions may reach significance at a sample size similar to Experiment 1. Notably both descriptive data and bootstrapping used to improve population estimates for statistical parameters suggest that startle potentiation to both, angry and happy facial expressions as compared to neutral ones would emerge at both early (120 ms) and late (4.5 ± 0.5 s) probe times. On the one hand, this supports that the startle paradigm does not principally lack the necessary sensitivity to reflect differences induced by facial expressions. On the other hand, this clearly indicates that the facial stimuli used in experiments 1 and 2 do not induce affective modulation of the startle response in unselected samples and individuals with a clinical diagnosis of SAD.

On a more general level, these findings question the validity of the few reports of affective startle modulation to facial expressions (Balaban, 1995; Hess, Sabourin, & Kleck, 2007; Ivanonva & Allen, 2001; Springer, Rosas, McGetrick, & Bowers, 2007; White, 2002). Two of the studies reporting affective startle modulation to facial expressions (one conference contribution and one dissertation) were never published in peer-reviewed journals. In fact, when the current findings are included in the count, there are more published null-results than positive findings (Alpers & Adolph, 2006; Schmidt-Daffy, 2006; Spangler, Emlinger, Meinhardt, & Hamm, 2001). In addition, it remains curious, why this seemingly intuitive question was not addressed in the literature earlier and more frequently. It may be suspected, that one reason is a file-drawer effect.

It has been suggested that threatening facial expressions may not be a phobic cue for individuals with social anxiousness in the same way in which cues such as spiders induce fear

in individuals with specific phobia such as arachnophobia (Kolassa, Musial, Mohr, Trippe, & Miltner, 2005; Kolassa & Miltner, 2006). One reason may be that facial expressions do not engage the defensive motivational system suggested by Lang, Bradley, and Cuthbert (1998). Interestingly, recent brain imaging studies indicate that amygdala activity during face processing may be similarly increased during processing of fearful, angry, sad, disgusted, surprised, and happy facial expressions (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Iidaka et al., 2001; Kim, Somerville, Johnstone, Alexander, & Whalen, 2003; Wang, McCarthy, Song, & Labar, 2005; Yang et al., 2002). This in line with results from classic single cell recording studies (Rolls, 1984). Therefore, startle responding may be uniformly moderated by different facial expressions. Consequently, startle responding to facial expressions should be compared to other control stimuli and external social threat-cues.

An analysis of conditions under which previous publications found affective modulation of the startle response to facial expressions may further help identify, under which conditions facial expressions moderate startle responding. First, affective startle modulation was found when the gaze of the faces was directed directly at the participants (Ivanonva & Allen, 2001). However, this was also the case with stimuli used in the current experiments. Second, when angry but not when fearful expressions were used (Springer, Rosas, McGetrick, & Bowers, 2007). Such a direct comparison of fearful to angry facial expressions across different levels of trait social anxiousness may replicate findings by Springer, Rosas, McGetrick, and Bowers (2007) and could reveal whether such effects depend on the level of trait social anxiousness. Third, affective startle modulation emerged, when interactions with gender were factored in (Hess, Sabourin, & Kleck, 2007). However, effects of gender were examined in the current studies, but did not reveal any interactions with startle potentiation. Effects reported with infant participants (Balaban, 1995) further recommend comparisons across different age

groups. For example effects of habituation to the years of exposure to social contact may be reduced in children.

While all these studies found affective startle modulation in healthy controls, only one study reported a similar effect in individuals with a diagnosis of SAD. Notably, this effect was only present in a condition where the participants directed their attention away from the emotion of facial expressions on display (White, 2002). Therefore, inducing attentional focus away from the emotional content of facial expressions might enhance effects.

In addition, none of these studies assessed startle responding during social challenge. An internal focus of attention allocation, probably accompanied by focusing on negative cognition, due to the situational challenge may be responsible for the absence of affective startle modulation. Although, it is clearly an oversimplification to derive the potential for startle modulation from amygdala activation, a recent brain imaging study may support this argument. Neither high- nor low-anxious volunteers showed an increased amygdala response to threat distractors under high perceptual load, contrary to a strong automaticity account of amygdala function (Bishop, Jenkins, & Lawrence, 2007). This shows that a distracting context-related task may indeed be related to a lack of stimulus-related startle modulation. Therefore, a future study should compare fear potentiated startle across different levels of social challenge.

Finally, to investigate the role of threat-cue vulnerability in socially anxious individuals, there may be more appropriate „phobic" cues eliciting a distinct fear response than faces. Verbal stimuli may more directly activate semantic networks, for example related to previous experiences of failure in social situations and self-related negative cognitions. This view is supported by experiments showing startle modulation by imagery triggered via verbal scripts (McTeague et al., 2009). Furthermore, interactions of an automatically activated defensive system (Lang, Bradley, & Cuthbert, 1998) and higher cognitive processing as suggested by

the working model on mediators of social anxiety (Cuthbert et al., 2003; McTeague et al., 2009) may be particularly likely with verbal threat-cues.

The lack of startle potentiation renders comparisons between early (PPI) and late (affective) startle modulation and potential associations with trait variables. Furthermore, testing of whether preferential processing of external social threat-cues mediates associations between trait social anxiousness and anxious responding is pointless. However, associations with general startle responding (i.e. across different foreground stimuli) may emerge. In contrast to experiment 1, increased startle latency did not correlate with higher levels of trait social anxiousness and no other associations with social anxiety emerged. The small sample size and restricted range of trait social anxiousness in experiment 2 may be responsible for the absence of effects. A future study should therefore compare individuals with a wide range of high vs. low trait social anxiousness.

The small sample size is a major limitation of experiment 2 in general. However, it should be considered that the affective startle modulation to facial expressions emerged in similarly small samples in a previous study (n = 9, see White, 2002). Furthermore, post-hoc evaluation of effect size measures suggested that larger samples would not reveal affective startle modulation but potentiation of startle amplitudes to both positive and negative facial expressions as compared to neutral ones. Another limitation of the given sample is the high frequency of comorbid depression and related medication. Effects can therefore not be discerned from effects related to SAD. Importantly, the unimodal distributions of the bootstrapping means suggest that there was no subgroup of individuals that would have shown a different effect (e.g. the expected affective startle modulation). Notably, this also minimizes the possibility that individuals with comorbid depression or individuals receiving medication responded different from the rest of the sample.

In sum, finding of experiment 2 indicate that emotional facial expressions do not induce pronounced attentional or affective modulation of the startle reflex in individuals with SAD. This questions whether external social threat-cues act like phobic stimuli for individuals with specific phobia, as compared to spiders for patients with arachnophobia. If at all, the influence of automatic evaluation of facial expressions is small. Significant effects of these stimuli (ratings of stimulus properties and spontaneous corrugator supercilii) were not related to trait social anxiousness. Therefore, experiment 2 provides no support for the working model on mediators of social anxiety.

2.3 Experiment 3 – A Multivariate Approach to Investigate the Roles of External Social Threat-Cues vs. Self-Related Negative Cognitions as Mediators of Social Anxiety

2.3.1 Background and Hypotheses

While experiments 1 and 2 primarily focused on threat-cue vulnerability, the third experiment aimed at an integrative test of the mediation model of social anxiety (see section 1.3.8, p. 52). Therefore, it was a major objective of the third experiment to examine whether cognitions mediate social anxiety when socially anxious individuals are confronted with a socially challenging situation, as predicted by the cognitive model (e.g. Clark & Wells, 1995). To this end, anxious responding was assessed during anticipation of public speaking, the most commonly feared social situation by socially anxious individuals and the general population (Mannuzza, Schneier, Chapman, & Liebowitz, 1995; Pollard & Henderson, 1988). It was expected that trait social anxiety would predict increases of acute social anxiety in response to this situation. Similarly, expectations about one's performance may be affected by situational challenge (e.g. Efran & Korn, 1969). To examine the specific role of negative cognitions as a mediator, self-related negative cognitions were induced with a script developed by Hinrichsen and Clark (2003) and compared to relaxation instructions that encourage participants to focus their attention away from negative cognitions (Hudetz, Hudetz, & Reddy, 2004).[3] Therefore, greater anxiety and negative cognitions during negative anticipation than during baseline and relaxed anticipation were expected. Moreover, the amount of negative cognitions was

[3] We consider this to be a better control condition than anticipation without a task, because it actively counteracts the default reaction of socially anxious individuals to focus their attention on negative self-related cognitions when anticipating social threat.

expected to mediate task induced changes. Finally, it was predicted that these associations would be specifically related to social but not general trait anxiety.

In addition to self-report, physiological correlates of trait social anxiety were assessed (Dewar & Stravynski, 2001; Hofmann, Heinrichs, & Moscovitch, 2004). Both ICD-10 (WHO, 1992) and DSM-IV-TR (APA, 2000) classifications underline the key role of dysfunctional emotional responding to certain stimuli and situations for a diagnosis of SAD. According to the bio-informational model (Lang, 1979), emotions can be assessed within three systems involved in the expression of emotions: subjective or verbal information (i.e. reports about perceived emotions), behavioral (i.e. facial and postural expressions, speech, and paralinguistic parameters), and psychophysiological reactivity (e.g. heart rate, electro dermal activity). In the case of social anxiousness, the assumption of a consistent response pattern across response systems (e.g. Rapee, 1995) has received little support, see above. Moreover, physiological reactivity seems rather uncoupled from subjective experience (Friedman, 2007). Only few studies have assessed self-report, physiology, and behavior in socially anxious individuals within a socially challenging context. Most studies made static comparisons between subject groups, an analysis, which is oblivious to great individual variations (see critique in Dewar & Stravynski, 2001). As several researchers have noted, the within-participant design is often more sensitive to detecting coherence than is the between-participants design because it minimizes sources of between-individual variance (e.g. Lazarus, Speisman, & Mordkoff, 1963; Pennebaker, 1982; Reisenzein, 2000; Rosenberg & Ekman, 1994; Ruch, 1995). In addition, it has been noted that, conceptually, between-individual analyses might be irrelevant to the question of how tightly responses are associated (e.g. Buck, 1980; Cacioppo et al., 1992; Lacey, 1967; Stemmler, 1992). To overcome these limitations, continuous and situation-specific measurement with subjects used as their own

controls (within-subjects designs) within a multivariate design has been suggested as an optimal approach.

This approach should further include the often neglected assessment of parasympathetic influences and interactions between sympathetic and parasympathetic processes (see Porges, 1995a, p. 302). Heart rate variability in the high frequency spectrum (HRV-HF, Camm et al., 1996) is a commonly used index of respiratory sinus arrhythmia and primarily reflects parasympathetic influence on heart rate (see Grossman & Taylor, 2007, for a discussion of interpretative issues and further influencing factors). Between-subjects differences have been associated with emotional reactivity (Beauchaine, 2001; Thayer & Brosschot, 2005) and acute shifts of HRV-HF have been linked to self-regulatory efforts of emotional responding (Beauchaine, 2001; Porges, 1995b; Thayer & Lane, 2000). This suggests an inverse relationship of between-subjects' levels of HRV-HF and the level of anxiety as well as the amount of negative cognitions during anticipation of public speaking. Furthermore, higher levels of trait social-anxiety may predict stronger task induced within-individual decrease of HRV-HF. Finally, the amount of negative cognitions expected to mediate the specific association between trait levels of social anxiousness and task related HRV effects.

In addition, skin conductance level (SCL) was assessed as an index of distress related sympathetic arousal (e.g. Boucsein, 1992) and heart rate (HR) as a general indicator of physiological activation. In line with previous reports (e.g. Mauss, Wilhelm, & Gross, 2004), general stress-related activation in anticipation of public speaking was expected to lead to increased SCL and HR. However, no specific association with trait social anxiety was expected.

Finally, observable behavior was assessed, the third emotional response domain according to Lang (1993). As suggested above, the cognitive model of social anxiety predicts that increasing anxiety leads to avoidance behaviors and the application of safety behaviors. It has

been shown that observers rate the performance of individuals with SAD in standardized speaking tasks as more negative and less positive than that of controls (Stopa & Clark, 1993) or generally worse (Moscovitch & Hofmann, 2007). Therefore, it was expected that higher levels of trait social anxiousness predict poor performance in the public speaking task. Furthermore, it has been shown that increased self-focused attention led to impaired performance in individuals with SAD (Hope & Heimberg, 1988). Finally, negative anticipation was expected to further increase observable behavioral symptoms in comparison to relaxed anticipation.

The second major goal of this experiment was to examine the role of external social threat-cues as another mediator of associations between trait social anxiousness and anxiety in socially threatening situations. Furthermore, potential interactions with cognition were examined. As suggested by the mediation model of social anxiety (see section 1.3.8, p. 52), affective responding due to automatic information processing bias to external social threat-cues may directly mediate social anxiety. Furthermore, negative cognitions may be enhanced via activation of motivational defense and approach systems when semantic content is primed within associative networks (Lang, Bradley, & Cuthbert, 1998), see Figure 2, p. 26. Finally, negative cognitions may also enhance preferential automatic processing of external social threat-cues via priming of these motivational systems.

Although experiments 1 and 2 did not find the expected pattern of startle modulation to facial expressions, some indicators for a triple interaction of trait social anxiousness, situational challenge, and emotional foreground stimuli emerged. To further investigate such interactions, individuals featuring a wide range of trait social anxiousness were examined across differential levels of situational challenge (i.e. relaxed vs. negative anticipation of public speaking). In contrast to experiments 1 and 2, a more intensely challenging social situation was chosen to increase the impact of the situational context particularly in the

negative anticipation condition. Importantly, this context was expected to affect all participants to some degree. The goal of this procedure was to prevent potential floor effects in individuals with low-trait social anxiousness in the relaxed anticipation condition. As suggested by White (2002), attending to the emotion of facial expressions may attenuate affective modulation of the startle response. Furthermore, visual scan-path studies have shown that individuals with SAD over-scan facial expressions without paying particular attention to facial features related to emotion (e.g. the eye region, see Horley, Williams, Gonsalvez, & Gordon, 2003). This behavior was induced with a task that required the participants to search for strategically placed cues on the facial expression; see section 2.3.2.3, p. 117 for details. In general, similar effects as for self-reported anxiety were expected for startle amplitudes. Furthermore, negative anticipation was expected to generally potentiate startle amplitudes, especially in participants with high social anxiousness. These effects may be most prominent during processing of neutral non-social stimuli or when no foreground cues distract from the situational context, see discussions of experiments 1 and 2. This would support the assumption of a general priming of automatic threat-cue processing. Furthermore, this effect may interact with the specific content of the stimuli such that socially threatening cues elicit the strongest startle potentiation. This would suggest a stimulus specific interaction of automatic threat-cue processing and cognitive content.

Primarily, we expected these effects to emerge with prototypical facial expressions. During anticipation of public speaking, angry faces signal social devaluation. Therefore fear potentiation of the startle was expected as a combined result of threat-cue-bias in individuals with high trait social anxiousness, situational challenge, and the activation of the defensive motivational system by downstream priming effects of self-related negative cognitions (Lang, Bradley, & Cuthbert, 1998), see Figure 2, p. 26. Neutral facial expressions are ambiguous in this situation. Therefore, negativity bias in socially anxious may also lead to higher startle

amplitudes. In contrast, happy facial expressions are universally recognized as a positive social signal. Consequently, attenuation or least reduced potentiation when compared to the other facial expressions was expected. Furthermore, Springer, Rosas, McGetrick, and Bowers (2007) have shown that angry but not fearful facial expressions elicited startle potentiation in healthy controls. Fearful faces were therefore included as a facial control stimulus featuring similar valence and arousal to angry faces, but a different content. A replication of the findings of Springer, Rosas, McGetrick, and Bowers (2007) would strengthen the specificity hypothesis of threat-cue vulnerability (Mathews & MacLeod, 1987). Finally, we added houses as an additional neutral reference category with similar processing demand as faces. Similar to startle reactions elicited with no foreground stimulus presentation, these stimuli served as a relatively neutral baseline to assess effects of the anticipatory conditions (i.e. the context). In comparison to facial expressions, we further examined emotional words, which may interact more specifically with information processing of semantic content. We expected startle potentiation in all participants with general threat words, specific interactions of trait social anxiety, anticipatory context, and socially threatening words and no potentiation to non-threatening words. Finally, we examined effects on startle latency in addition to startle amplitude, because of the potential to differentiate attentional from affect-related effects of context and foreground stimuli, see section 1.3.2.1, p. 24.

Experiment 3 examined whether self-related negative cognitions mediate the relationship between trait social anxiety and anxious responding during anticipation of a socially threatening situation. In contrast to experiments 1 and 2, an experimental manipulation of the intensity of self-related negative cognitions may reveal a functional dependency of outcome measures on response domains (self-report, behavior, physiology) on the supposed mediation. Within this design, the influence of automatic information processing bias to external social threat-cues (facial expressions, emotional words) in comparison to neutral control stimuli and

effects of context were examined. Finally, interactions with increased levels of trait social anxiousness were expected to increase the effects of both mediators, as suggested in the working model on mediators of social anxiety in section 1.3.8 p. 52.

2.3.2 Methods

2.3.2.1 Design and Participants

The experiment featured a 4 (Stimulus-Category: neutral, positive, general threat, social threat) x 2 (Within-subjects factor Stimulus-Type: words, faces) x 2 (Within-subjects factor Task: baseline, negative anticipation, relaxed anticipation) x 2 (Between-subjects factor Task-Order: negative before relaxed anticipation or vice versa) design.

Thirty-six participants were recruited from the Boston University 101 undergraduate psychology student pool via postings on the experiment sign up board at the BU Psychology Department; see sign up information sheet in the appendices 5.3.1., p. 212 and 5.3.2, p.213. The students received course credits for their participation regardless of whether they terminated the experiment or not.

Inclusion criteria were 18 years of age or older, normal or corrected vision, and signed written informed consent. Exclusion criteria were smoking or intake of caffeine less than three hours before the experiment, intake of alcohol less than eight hours prior to the experiment, tinnitus or hearing impairment.

2.3.2.2 Apparatus

For a floor plan illustrating the setup of the room, see Figure 16. Instructions, picture stimuli, and startle probes were presented using Presentation® software (v.0.80, www.neuro-bs.com) running on a Pentium-level computer. Participants' heads were at approx. 40 cm distance to a 17" flat screen CRT-monitor (Best Buy, Insignia; 1024x768, 100Hz).

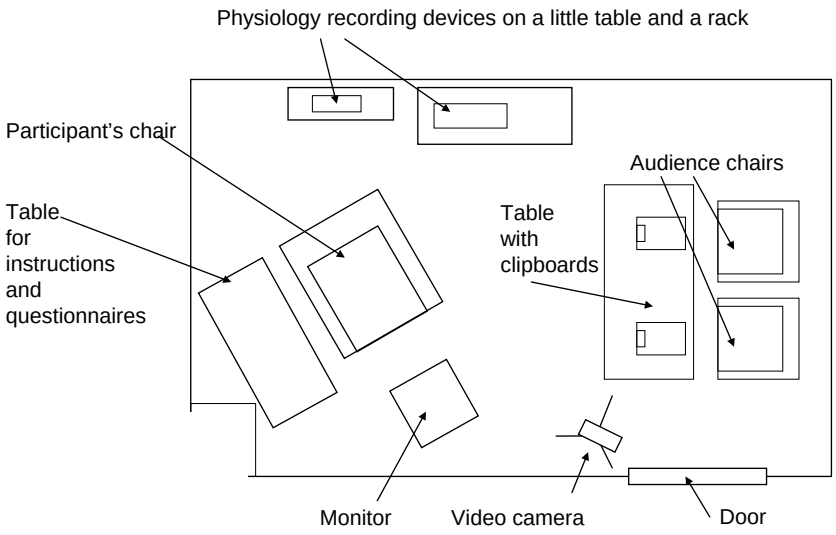

Figure 16: Floor plan of the experimental setup of experiment 3.

All psychophysiological measures were recorded with a BIOPAC MP150 system at a sampling rate of 1000 Hz. One electrode (Ag-AgCl, 8 mm) on the left clavicle served as common ground. Two electrodes (Ag-AgCl, 8 mm, on right clavicle and lower left rib cage) were used to record an electrocardiogram (EKG; analog high-pass filter: 0.5 Hz). Skin conductance level (SCL [µS]; analog low-pass filter: 10 Hz) was recorded via two electrodes (Ag-AgCl, 8 mm) on the second phalanx of index and ring finger of the non-dominant hand. Startle probes of 50ms bursts of white noise at 105 dB(A) with near instantaneous rise/fall times (Lang, 1995) were presented via a SoundBlaster 16 sound card, amplified with a Yamaha RX-750 stereo receiver and played binaurally through Etymotic ER•6 Isolator in ear headphones. Eye-blink startle responses were measured electromyographically (EMG) from orbicularis oculi, using 4 mm EL254S Ag/AgCl surface electrodes (BIOPAC Systems, Inc., 42 Aero Camino, Goleta, CA 93117) placed about 1 cm below the pupil and outer canthus of the left eye. Skin preparation, electrode placement, and analysis followed the recommendations of Blumenthal and colleagues (2005). The raw EMG was amplified (Biopac

EMG100C, gain: 5000, lowpass-filter: 5 kHz, highpass-filter 1.0 Hz), digitally sampled at 1000 Hz (16 bit A/D-converter) with a BIOPAC MP150 and stored to hard disk.

2.3.2.3 Procedure

After signed informed consent was obtained, all participants completed a set of ad hoc ratings (see appendix pp. 214) including ratings of self-reported anxiety ("How anxious do you feel at the moment? Please try to specify your current feeling on a scale ranging from 0 to 100. 0 would signify no anxiety and 100 would denote the highest possible anxiety you can imagine."). Next, they were asked to fill out the STAI-trait, SPAI, and a socio-demographics form. Next, the participants were seated in the experimental room 40 cm in front of a 17"-Monitor, which was used for presenting instructions and visual stimuli. After that, the electrodes for the psychophysiological measurement were attached. After a 2-min baseline of the physiological measures was recorded, the participants were randomly assigned to one of two anticipation conditions:

In the *negative anticipation* condition *self-related negative cognitions* were induced with a script developed by Hinrichsen and Clark (2003, p. 213). Written instructions prompted participants to remember particular social situations that did not go well to imagine, how they appeared in this situation and what impression this may have created in others. Based on this past image, participants were asked to anticipate, how they might perform in the upcoming speech, what could go wrong, what would be the worst thing that could happen, and what would happen if they made a fool out of themselves. This procedure was repeated with additional situations for the duration of the anticipatory period.

In the *relaxed anticipation* condition (Hudetz, Hudetz, & Reddy, 2004), participants listened to a pre-recorded tape instructing them to close their eyes and focus on pleasant thoughts, feelings, and images and to let go of any negative cognitions that came to mind. The

relaxation instructions included statements such as “tension flows from my body” and “I can relax at will”. This was accompanied by relaxing music and the sound of ocean waves. Following the 2-min recordings of physiological data, ratings of self-reported anxiety and sets of ad hoc ratings (see appendix 5.3.7, pp.218) were completed after 2, 8, 10, 29, 35, and 37 min of anticipation respectively. In addition, anticipation was interrupted twice (after 12 and 39 min) by a 15-min startle procedure. Following 55 min of anticipation, two confederates entered the room as an audience. Next, participants were asked to fill out the SSPS, PANAS, and STAI-state, and a camera for recording the speech was uncovered and turned on. After 30 s of preparation time, the participants were asked to speak for at least 3 min about personal strengths and weaknesses as part of an imaginary job application. In addition, the audience evaluated the speech on a rating chart, see appendix 5.3.12, p. 228. If participants stopped their speech before 3 min were over, the audience asked a standardized set of questions like e.g. „why do you think you’re the best candidate for such a position?” After 3 min of speaking, the participants were asked, if they wanted to add anything important. If the answer was no, they were permitted to end their speech, see instructions for the audience in appendix 5.3.11, p. 226.

Then, the complete procedure (anticipation, startle procedures, and another speech) was repeated with the other anticipatory protocol (negative vs. relaxed). At the end of the experiment, all picture stimuli were rated for valence, arousal, and complexity on 9-point Likert-like scales with verbal anchors derived from the original descriptions of the range of SAM-scales (Lang, Bradley, & Cuthbert, 1999) used for obtaining standardized ratings for the IAPS-picture set, which has been shown to reliably induce affective startle modulation (e.g. Lang, Bradley, & Cuthbert, 1990). See appendix for details on the rating scales and the anchor descriptions in section 5.3.10, pp. 223 and section 5.3.10.2, pp. 224. Finally, all participants

rated how pleasant they experienced the relaxed and how unpleasant they experienced the negative anticipation condition (0 = not at all; 6 = extremely), and were debriefed.

Startle Procedure. Participants wore headphones throughout the anticipation periods. At the beginning of each startle procedure four startle reactions were elicited while the screen was still blank. The first two reactions were omitted from the analysis. Next, participants were asked to direct their attention to 21 stimuli randomly drawn from 7 categories with differing threat potential (emotional words, facial expressions, and houses). Each stimulus was preceded by a 250 ms fixation cross, and followed by a 15 s (± 5 s) inter-stimulus-interval (blank screen). Startle reactions were elicited during two out of three stimulus presentations per category. During the presentation of faces and houses, startle probes were played at 3800ms ± 300 ms after stimulus onset (see White, 2002). Furthermore, visual over-scanning and an attentional focus which is not explicitly focused on the emotion of facial expressions were induced by asking the participants to search for strategically positioned cues, which were randomly distributed on the pictures. These cues were counterbalanced between features relevant vs. irrelevant for emotion recognition. For examples of stimuli and the grid overlay used for setting the cues, see appendix 5.3.9.1, p. 220. Similar search tasks have shown to induce implicit processing of the emotional facial expressions (Whalen et al., 1998). Furthermore, over-scanning of facial features mimics the typical scan-path applied by socially anxious individuals, when processing facial expressions (Horley, Williams, Gonsalvez, & Gordon, 2003). To induce a similar workload, the same task was performed with houses, a stimulus category with similar complexity and structural features to facial expressions (Kanwisher, McDermott, & Chun, 1997). Words were to be read silently and startle probes were played at 3500 ms ± 1000 ms after stimulus onset (Larsen, Norton, Walker, & Stein, 2002).

2.3.2.4 Trait Measures

The social phobia subscale of the Social Phobia and Anxiety Inventory (SPAI: Turner, Beidel, Dancu, & Stanley, 1989), a well established self-report measure with excellent psychometric properties (Turner, Stanley, Beidel, & Bond, 1989), was used to assess trait social anxiety.

Questionnaires and Subscales		Internal Consistency (Cronbach's α)
SPAI		.96
FNE		.94
STAI-trait		.49
BDI-II		.87
BCS	*private body consciousness*	.61
	public body consciousness	.72
	body competence	.75
BIS		.73
BAS	*drive*	.79
	fun seeking	.68
	reward responsiveness	.51
NEO-FFI	*neuroticism*	.76
	extraversion	.76
	openness to new experiences	.64
	agreeableness	.59
	conscientiousness	.53

Table 11: Overview of the internal consistency (Cronbach's α) achieved for questionnaires and their subscales with the experiment sample; see text for details.

An additional assessment with the fear of negative evaluation scale (FNE, Watson & Friend, 1969) was obtained to cross-validate results and to assess, whether this more efficient instrument may represent effects as well as the SPAI.

To examine if effects were specifically related to trait social anxiety, also general trait anxiety was assessed with the Spielberger State Trait Anxiety Inventory (*M* = 51.53, *SD* = 3.60, *Min* = 44, *Max* = 59, STAI-trait: Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983).

Since comorbid depression is common for individuals with social anxiety and may affect cognition and emotional information processing, the Beck Depression Inventory II was administered (BDI-II, Beck, Steer, & Brown, 1996). The BDI-II has high internal consistency (alpha = 0.93 and 0.92 in samples of college students and out-patients respectively, Beck, Steer, Ball, & Ranieri, 1996) and has been shown to be a valid indicator of depression with good diagnostic discrimination (Dozois, Dobson, & Ahnberg, 1998).

Furthermore it has been suggested that increased sensitivity towards body reactions, body consciousness, and their relation to self-concepts may lead to increased anxiety symptoms in challenging social situations. These constructs were assessed with the Body Consciousness Scale (BCS, Miller, Murphy, & Buss, 1981) and ad hoc ratings, see appendix 5.3.7, p. 218.

Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment are alleged precursors of social anxiety disorder in children. To control for these aspects, the Behavioral Inhibition System / Behavioral Approach System Scales (BIS/BAS, Carver & White, 1994) were used.

Similarly, extraversion has been related to social anxiety (e.g. Blumenthal, Chapman, & Muse, 1995; Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002). The NEO-FFI (Costa & McCrae, 1992) was used to assess personality traits and to examine potential interactions with the BIS/BAS scales (Heponiemi, Keltikangas-Jarvinen, Kettunen, Puttonen, & Ravaja, 2004).

See Table 11 for an overview of the internal consistency achieved for questionnaires with the experiment sample.

2.3.2.5 State Measures

Self-reported anxiety was assessed via ad hoc ratings using the following statement presented on a computer screen: „How anxious do you feel at the moment?" Participants rated their anxiety on a scale from 0 (no anxiety) to 100 (strongest feeling of anxiety that you can imagine). The reliability of the averaged repeated measurements was r = .81 and r = .78 within the negative and relaxed anticipation conditions, respectively. For details on further ratings see appendix 5.3.7, p. 218.

The Self-Statements During Public Speaking Scale (SSPS: Hofmann & DiBartolo, 2000) was used to measure cognitions during the preparation for the participants' speeches. The SSPS was developed to assess *self-related negative cognitions*. Specifically the negative subscale (SSPS-N) has been shown to mediate treatment change in SAD (Hofmann, Moscovitch, Kim, & Taylor, 2004) and has been shown to correlate with low expectations for success and low satisfaction in a performance situation and high self-reported anxiety in a public speaking task (Hofmann & DiBartolo, 2000). Each scale of the SSPS comprises five statements describing feelings and thoughts subjects might have about themselves in public speaking situations. Examples of negative subscale items include: "A failure in this situation would be more proof of my incapacity" and "What I say will probably sound stupid". The SSPS has excellent psychometric properties and is sensitive to change (Hofmann & DiBartolo, 2000). In the current sample, the internal consistency of the SSPS-N was .80 and .81 in the negative and relaxed anticipation conditions, respectively. To assess the amount of *self-related negative cognitions*, the negative subscale of the SSPS was used. Mood and mood changes during the experiment may also affect outcomes. Furthermore, changes in mood may be differentiated from changes in negative cognition. Therefore, we compared scores on the SSPS with scores

on the Positive and Negative Affect Schedule (PANAS, Watson, Clark, & Tellegen, 1988). Cronbach's alphas achieved with the experiment sample for PANAS subscales in the two anticipatory conditions were $\alpha_{\text{relaxed anticipation}} = .51$ and $\alpha_{\text{negative anticipation}} = .60$ for the positive, $\alpha_{\text{relaxed anticipation}} = .51$ and $\alpha_{\text{negative anticipation}} = .64$ for the negative subscale respectively. Finally, changes of state-anxiety during the experiment was assessed with ad hoc ratings (see appendix 5.3.7, p. 218), and the STAI-state (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Cronbach' alphas for STAI-state scores in the two anticipatory conditions were $\alpha_{\text{relaxed anticipation}} = .88$ and $\alpha_{\text{negative anticipation}} = .91$.

2.3.2.6 Behavioral Assessment of Public Speaking

Because there is no standardized system for assessment of speaker performance in public speaking tasks, a novel system was derived from existing systems for the assessment of social interactions (see overview in Schulz, Meuret, Loh, & Hofmann, 2007). The two person audience attending the public speeches was trained with this novel system to assess public speaking performance at fixed time intervals (i.e. every two minutes) on four dimensions (nonverbal symptoms of anxiety; content and rhetoric quality of the speech; anxiety; overall impression as a „social performer"). Scores ranged from 0 to 100. A score of 50 was marked as „standard/neutral", see appendix for details 5.3.12, p. 228. Inter-rater reliability for observer ratings obtained by the trained two-person audience during public speaking was $r = .61$.

2.3.2.7 Data Reduction and Analysis

Post-processing of the physiological data was completed with custom Matlab toolboxes. This included R-spike detection in the raw EKG, screening for artifacts and interpolation of ectopic beats and computation of the average heart rate (HR [bpm]). The RR-interval time series was prorated to equal time intervals (8 Hz) and de-trended to remove tonic shifts of sympathetic activation (10 s high-pass filter period). Finally, mean spectral power density of heart-rate-

variability in the high frequency range of 0.15-0.40 Hz (HRV-HF [ms2], see Camm et al., 1996) was derived with discrete Fourier transform (DFT; 64 s Hanning window, 50% overlap).

For the statistical analysis HR, HRV-HF, and SCL were averaged across six a priori determined 2-min epochs within each anticipatory condition. The goal of restricting the measurement to these epochs of quiet sitting was to maximize reliability and minimize artifacts and uncontrolled influences by movement or postural and respiratory variations. While such influences are problematic in general, HRV is particularly susceptible to them (Grossman & Taylor, 2007). To further avoid uncontrolled differential effects between single variables, the same measuring intervals for all physiological variables were used. To maximize comparability across response domains, each recording was followed by a rating of self-reported anxiety, which were also combined into mean scores for each anticipatory condition.

Post processing of the EMG included filtering (60 Hz notch, 20 Hz 4th order Butterworth high-pass), rectification, and smoothing (100 ms moving average for amplitude scoring, and a 20ms moving average for latency scoring). Next, scoring of amplitude and latency of the startle-blink reactions was performed with EMGpeakfind (Schulz & Alpers, 2007), a computer program which implements state of the art criteria specified by Blumenthal and colleagues (2005). Startle amplitudes were computed by subtracting the average of 20 ms of data before startle-onset (EMG-baselines) from startle magnitudes. Amplitudes equal or smaller than zero and reactions with artifacts were excluded from the analysis (15.97 %). There were no startle non-responders (Blumenthal et al., 2005). Mean startle amplitudes were square-root transformed and mean HRV-HF was $\log_{10}$ transformed to achieve normal data distribution across subjects, K-S test, all p's $\geq .22$. K-S tests for startle latency showed, that no transform was necessary, K-S test, all p's $\geq .16$.

Observer ratings of the participants' performance during public speaking were transformed to T-scores within raters to minimize effects of between session variability and averaged across the two audience members per speech. Second, for comparing performance in the two speeches of each participant, intraindividual T-scores were computed to minimize between participants variability.

Finally, all data were screened according to the recommendations by Tabachnick and Fidell (2007). Outliers in HRV-HF data were deleted from baseline in one participant, and in the negative anticipation condition from another participant.

2.3.2.8 Statistical Analysis

Repeated measures ANOVAs (within-subjects factor Task: baseline, negative anticipation, relaxed anticipation, between-subjects factor Task-Order: negative before relaxed anticipation or vice versa) were used to assess effects cognition in the two anticipatory tasks.

Further repeated measures ANOVAs were computed to examine the role of *external social threat-cues* (within-subjects factor Task: baseline, negative anticipation, relaxed anticipation; within-subjects factor Stimulus-Category: neutral, positive, general threat, social threat; within-subjects factor Stimulus-Type: words, faces; between-subjects factor Task-Order: negative before relaxed anticipation or vice versa). Planned contrasts and independent *t*-tests (two-tailed) were used to further examine effects of the experimental manipulation and possible effects of the within-subjects design such as habituation.[4]

[4] In an alternative analysis with gender added as another factor, all reported effects remained unaffected. Marginally significant effects of gender suggested lower baseline HR ($F(1, 32) = 3.95$, $p < .06$, *partial-*$\eta^2 = .11$) and SCL ($F(1, 32) = 3.36$, $p < .08$, *partial-*$\eta^2 = .10$) in men (HR: $M = 75.65$, $SD = 14.73$; SCL: $M = 14.62$, $SD = 9.27$) as compared to women, HR: $M = 77.87$; $SD = 16.08$; SCL: $M = 18.51$, $SD = 6.64$. Furthermore, there were marginally significant interactions with Task-Order, suggesting lower self-reported anxiety ($F(1, 31) = 5.70$, $p < .02$, *partial-*$\eta^2 = .16$) and HR ($F(1, 32) = 3.52$, $p < .07$, *partial-*$\eta^2 = .10$) in men in the group that began with

For tests on autonomic physiological activation, dependent variables HR, SCL, and $\log_{10}$ transforms of HRV-HF were added simultaneously in a MANOVA (within-subjects factor Task: baseline, negative anticipation, relaxed anticipation, between-subjects factor Task-Order: negative before relaxed anticipation or vice versa), because measures within this response domain are not independent. Greenhouse-Geisser statistics are reported, when sphericity could not be assumed.

Furthermore, linear regressions were used to examine if trait social anxiety (SPAI) specifically predicted anxious responding. Application of Baron and Kenny's (1986) stepwise procedure were then used to test mediation as suggested in the working model on mediators of social anxiety. The criteria for mediation are as follows: In the first step, the predictor variable has to be correlated with the outcome (path c, the *total effect*). Second, the predictor variable has to correlate with the mediator (path a). Third, the mediator has to affect the outcome variable (path b), while the influence of the predictor variable is controlled. Fourth, full mediation is present if the relationship between predictor and outcome variable, while controlling for the effect of the mediator, is zero (path c', the *direct effect*). Otherwise, there is only partial mediation. In addition, Sobel's (1982) significance test was computed to assess the *indirect effect* (path ab) of the predictor on the outcome variable through the mediator. Sobel's test can be interpreted as an index of the strength of mediation. Completion of the mediation test-sequence was only discontinued, when results (e.g. of the first step) indicated that no mediation is possible.

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relaxed anticipation, and the opposite for women in the group that started with negative anticipation. Finally, a Task by Task-Order by Gender interaction in SCL ($F(1.60, 51.28) = 2.67$, $p < .09$, *partial-*$\eta^2 = .08$) revealed higher baseline HR in women who started with relaxed anticipation, suggesting a smaller change from baseline to anticipation in this subgroup. To provide a concise account of results relevant to our theoretic rational, gender was not used as a factor in the main analysis.

Two mediation models assessed whether negative cognition (SSPS-N) mediated the relationship between trait social anxiety (SPAI) and effects of anticipation on self-reported anxiety and $\log_{10}$ transforms of HRV-HF. For these analyses, baseline corrected difference scores were used, reflecting the expected increase of self-reported anxiety and decrease of HRV-HF during negative anticipation. In a third model, affective startle modulation to *external social threat-cues* as a mediator of associations between trait social anxiousness and acute anxiety. Finally, potential top-down effects of cognition on square root transformed startle amplitudes as an anxiety symptom (i.e. on the criterion level of the working model on mediators of social anxiety) were assessed within another mediation model. Because startle amplitudes were not assessed at baseline, the analyses were performed with uncorrected scores.

Finally, Pearson correlations were used to explore associations between variables. Independent *t*-tests, Chi^2-Tests, and Pearson correlations were used for manipulation checks and to describe the sample.

2.3.3 Results

2.3.3.1 Participants

Most of the 36 participants were female (*n* = 23, 63.89 %) and Caucasian (*n* = 26, 72.22 %). Non-Caucasian participants identified themselves as Asian (*n* = 3, 8.33 %), Indian (*n* = 2, 5.56 %), Hispanic (*n* = 2, 5.56 %) or other (*n* = 3, 8.33 %). The average age was 18.83 (*SD* = .94) All participants were unmarried (*n* = 36, 100 %).

Importantly, the sample comprised a wide range of trait social anxiety (*M* = 54.48, *SD* = 23.79, *Min* = 8.80, *Max* = 99.32) as measured with the social phobia subscale of the SPAI. Overall, the sample was comparable to unselected representative student samples, (M = 54.48 vs. e.g. M = 52.45 derived from Turner, Beidel, Dancu, & Stanley, 1989). Four (11 %) participants scored above 80, a cutoff commonly used as an indicator of probable presence of

SAD. There were no significant gender difference, male vs. female: *M* = 46.68 vs. 58.90; *SD* = 5.87 vs. 5.11; $t(34) = 1.51$, $p < .14$, $d = .5$. SPAI-trait scores further correlated negatively with self-ratings of extraversion ($r = -.50$, $p < .01$) and sociability ($r = .68$, $p < .001$) during childhood.

Similarly, the sample scored in a typical range for the general population (i.e. 13 - 20) on the fear of negative evaluation scale (FNE, Watson & Friend, 1969, M = 15.34, SD = 8.65) and comprised a particularly wide range (*Min* = 0, *Max*, = 30).

STAI-trait scores indicated an elevated level of general trait anxiety in the experiment sample (*M* = 51.53, *SD* = 3.60, *Min* = 44, *Max* = 59, see age related norms in Spielberger, Gorsuch, & Lushene, 1996).

On the BDI-II, the sample scored within a minimal range (< 13), well below the cutoff of 18 commonly used as a criterion for potential presence of clinically relevant depression, *M* = 11.64, *SD* = 7.43.

The sample featured typical levels of *private body consciousness* (*M* = 3.28, *SD* = .61), *public body consciousness* (*M* = 3.44, *SD* = .73), and *body competence* (*M* = 3.30, *SD* = .80) compared to scores for 568 men and 731 women published by Miller, Murphy and Buss (1981).

Compared to scores reported for larger college groups (Carver & White, 1994; Jorm et al., 1999) the sample had relatively low scores on the BIS subscale (*M* = 13.92, *SD* = 3.21), a typical level on the BAS *Drive* subscale (*M* = 8.69, *SD* = 2.55), low scores on the BAS *fun seeking* subscale (*M* = 7.28, *SD* = 2.26), and low scores on the BAS subscale *reward responsiveness, M* = 7.47, *SD* = 1.66.

In comparison to published reference scores (Costa & McCrae, 1994), the sample had elevated levels on subscales *neuroticism* (*M* = 29.58, *SD* = 8.35), *extraversion* (*M* = 37.64, *SD* = 5.98), *conscientiousness* (*M* = 38.58, *SD* = 5.45) of the NEO-FFI, and typical scores for the

general population on subscales *openness to new experiences*, $M = 29.17$, $SD = 5.46$ and *agreeableness*, $M = 33.83$, $SD = 6.37$.

There were no significant differences in SPAI and STAI-trait, BDI-II, BCS subscales, BIS/BAS, NEO-FFI subscales, age, or ethnic origin between the two groups randomized to different Task-Order (negative before relaxed anticipation or vice versa, all p's $\geq .11$). Women had lower scores on the BIS subscale ($M_{female} = 12.78$, $SD_{female} = 2.73$; $M_{male} = 15.92$, $SD_{male} = 3.10$; $t(34) = 1.88$, $p < .01$, $d = 1.08$) and higher scores on the BCS subscale *public body consciousness*, $M_{female} = 3.66$, $SD_{female} = .70$; $M_{male} = 3.08$, $SD_{male} = .67$; $t(33) = 2.43$, $p < .02$, $d = .85$. There was no gender difference for the other BCS and BIS/BAS subscales, SPAI and STAI-trait, BDI-II, NEO-FFI subscales, age, or ethnic origin, all p's $\geq .07$.

2.3.3.2 Manipulation Checks

At the beginning of the experiment, the participants rated both, their anxiety after hearing about the experiment, $M = 24.29$, $SD = 20.06$ and their confidence that they would make a good impression on the committee (0-100) evaluating their speeches, $M = 57.11$, $SD = 21.67$. After the experiment, relaxed anticipation was rated as moderately pleasant ($M = 4.17$, $SD = 1.20$), although 7 participants reported negative associations with the relaxation protocol. Negative anticipation was rated as moderately unpleasant ($M = 3.31$, $SD = 1.49$, $t(34) = 3.51$, $p < .001$, $d = .78$) on a 0 to 6 point Likert-like scales, see Table 12, p. 133. In addition, negative cognitions (SSPS-N) were significantly enhanced during negative anticipation in comparison to relaxed anticipation, as indicated by a significant main effect Task, $F(1, 33) = 10.54$, $p < .01$, partial-$\eta^2 = .24$. There was no main effect Task-Order, differentiating the two groups either randomized to negative before relaxed anticipation or vice versa ($F(1, 33) = 10.54$, $p < .01$, partial-$\eta^2 = .24$), and there was no Task by Task-Order interaction, $F(1, 33) < 1$.

Overall, the participants reported that they were focused on their tasks for about 58.37% of the time, *SD* = 22.69.

The participants had higher scores on both scales of the PANAS during negative anticipation, PANAS-PA scores, negative anticipation: *M* = 20.63, *SD* = 3.61; relaxed anticipation: *M* = 18.57, *SD* = 4.18; $F(1, 33) = 12.16$, $p < .001$, partial-$\eta^2 = .27$, and PANAS-NA scores, negative anticipation: *M* = 21.43, *SD* = 3.92; relaxed anticipation: *M* = 18.89, *SD* = 4.71; $F(1, 33) = 28.94$, $p < .001$, partial-$\eta^2 = .47$. This suggests a generally higher level of arousal, probably complemented by ambiguous feelings. Furthermore, the participants estimated their peak heart rate during the speeches higher after negative anticipation (*M* = 57.39, *SD* = 26.09) than relaxed anticipation, *M* = 45.67, *SD* = 25.44; $F(1, 34) = 9.43$, $p < .01$, partial-$\eta^2 = .22$. Finally, self-report data collected throughout the course of the experiment suggests significant habituation. This is reflected in a decrease of reported anxiety during anticipation ($F(3, 102) = 4.11$, $p < .01$, partial-$\eta^2 = .11$), peak anxiety during the speeches ($F(1, 34) = 9.43$, $p < .01$, partial-$\eta^2 = .22$), subjective estimate of heart rate during speaking ($F(1, 34) = 12.75$, $p < .001$, partial-$\eta^2 = .27$) and a decrease of *positive* and *negative affect* as measured by the PANAS-NA ($F(1, 33) = 28.94$, $p < .001$, partial-$\eta^2 = .47$) and PANAS-PA, $F(1, 33) = 12.16$, $p < .001$, partial-$\eta^2 = .27$.

2.3.3.3 Effects of the Experimental Manipulation

2.3.3.3.1 Self-Reported Anxiety

The anticipatory tasks affected self-reported anxiety as suggested by a significant Task effect, $F(2, 66) = 7.78$, $p < .001$, partial-$\eta^2 = .19$. Planned contrasts showed that self-reported anxiety was significantly elevated during negative anticipation ($F(1, 33) = 5.31$, $p < .03$, partial-$\eta^2 = .14$), but not during relaxed anticipation ($F(1, 33) = 1.61$, $p < .21$, partial-$\eta^2 = .05$), when compared to baseline, see Figure 17. The direct comparison between negative and relaxed anticipation was significant, $F(1, 33) = 23.29$, $p < .001$, partial-$\eta^2 = .41$. There was no main

effect differentiating the two groups randomized to different Task-Order, $F(1, 33) < 1$. The Task by Task-Order interaction was marginally significant, $F(2, 66) = 2.43$, $p < .10$, partial-$\eta^2 = .07$. Contrast computations revealed that self-reported anxiety decreased significantly from the first ($M = 29.77$, $SD = 23.01$) to the second anticipation ($M = 22.91$, $SD = 17.54$), as indicated by a significant Task by Task-Order interaction, $F(1, 33) = 5.72$, $p < .02$, partial-$\eta^2 = .15$.

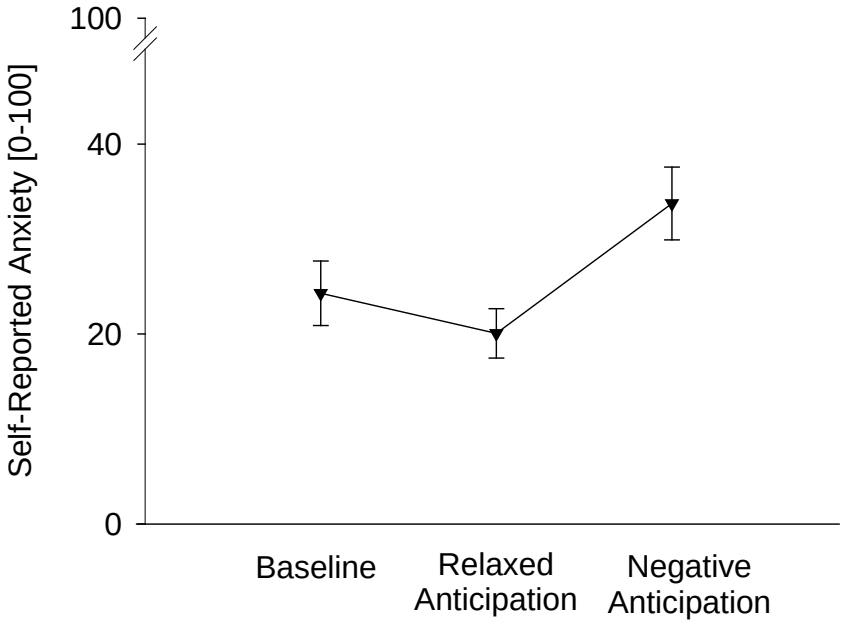

Figure 17: Means of self-reported anxiety (rating scale from 0 = no anxiety to 100 = extreme anxiety) at baseline and during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

Another repeated-measures ANOVA comparing retrospective ratings of subjective anxiety during speaking between the task conditions (task: negative anticipation, relaxed anticipation) corroborated this with a significant main effect task; $F(1, 35) = 5.66$, $p = .02$, partial-$\eta^2 = .14$. As expected, anxiety ratings were higher after negative anticipation ($M = 56.36$, $SD = 28.58$) than after relaxed anticipation ($M = 46.69$, $SD = 23.09$).

Similarly, the amount of negative cognitions (modified SSPS-N) was significantly higher after negative anticipation (M = 9.43, SD = 5.15) than after relaxed anticipation (M = 7.31, SD = 5.05; $F(1, 34) = 11.19$, $p = .01$, partial-$\eta^2 = .25$), while there was no significant difference between negative (M = 16.31, SD = 3.68) and relaxed anticipation (M = 16.00, SD = 4.04) in the amount of positive cognitions, modified SSPS-P; $F(1, 34) = 1.73$, $p = .53$, partial-$\eta^2 = .01$. There was no significant main effect of Task-Order or two-way interaction of Task and Task-Order, all F`s ≥ 1.

State anxiety as assessed with the STAI-state was higher after negative (M = 44.81, SD = 9.81) than relaxed anticipation, M = 42.03, SD = 8.79; $F(1, 34) = 16.30$, $p < .001$, partial-η^2 = .32. There was no significant main effect of Task-Order or two-way interaction of Task and Task-Order, all F's < 1.

2.3.3.3.2 Participant Ratings

Anxiety remained at an increased level which was significantly higher than the baseline assessment obtained immediately before the first anticipation phase, i.e. at completion of setup; $F(1, 31) = 5.87$, $p = .01$, partial-$\eta^2 = .16$; all other p's $\geq .19$, see Table 12. As mentioned above, there was also a significant difference between the two anticipatory conditions with higher anxiety during negative anticipation. Somatic awareness also increased from baseline to the rating obtained immediately before the first anticipation phase, $F(1, 31)$ = 9.74, $p = .01$, partial-$\eta^2 = .24$; all other p's $\geq .43$. The participants spent more time thinking about the speech during negative as compared to relaxed anticipation, $F(1, 32) = 38.26$, p = .001, partial-$\eta^2 = .55$; all other p's $\geq .22$. Finally, retrospective ratings of peak anxiety after holding the public speeches were higher after the negative as compared to relaxed anticipation condition, $F(1, 32) = 4.62$, $p = .04$, partial-$\eta^2 = .13$; all other p's $\geq .13$.

Ad hoc rating	Before reading the consent form *M* (*SD*)	Baseline *M* (*SD*)	At completion of setup *M* (*SD*)	Relaxed Anticipation *M* (*SD*)	Negative Anticipation *M* (*SD*)
Self-reported anxiety (0-100)	20.66 (20.53)	24.29 (20.06)	32.42 (21.69)	19.71 (15.70)*	32.98 (22.90)
Confidence to make a good impression (0-100)	--	57.11 (21.67)	61.97 (20.92)	54.35 (21.83)	52.30 (24.57)
Awareness of bodily symptoms (0-100)	--	36.97 (27.61)	52.17 (24.56)	40.57 (24.23)	43.47 (25.44)
Evaluation of awareness (1 to -6)	--	3.11 (.99)	5.75 (10.92)	3.83 (6.00)	3.19 (.86)
Thinking about speech during task (%)	--	--	--	22.51 (20.93)*	53.40 (24.96)
Time being focused on task (%)	--	--	--	55.07 (27.82)	61.66 (25.78)
Number of cues detected	--	--	--	20.72 (6.37)	21.19 (5.68)
Estimated number of negative facial expressions	--	--	--	5.47 (1.12)	5.38 (1.47)
Estimated number of negative words	--	--	--	5.28 (1.05)	5.65 (1.18)
Time of thinking about the content of the speech (%)	--	--	--	20.00 (30.19)	15.89 (24.46)
Valence of thoughts about job application	--	--	--	2.64 (2.03)	2.44 (2.12)
Peak anxiety during speaking (0-100)	--	--	--	46.69 (23.09)*	56.36 (28.58)
Impression made on the Committee (0-100)	--	--	--	50.42 (25.43)	43.69 (27.44)
Estimation of max. heart rate during speaking (bpm)	--	--	--	83.47 (14.66)	85.64 (22.79)

Table 12: Means and standard deviations for ad hoc ratings obtained via self-ratings of the participants at three different baselines and during relaxed vs. negative anticipation of public

speaking; see procedure for details; * marks significant differences between the two latter conditions, $p < .05$.

2.3.3.3.3 Biased Evaluation of Somatic Cues

To assess estimation bias of self-rating of somatic cues, the difference between EKG-derived heart-rate and estimated peak heart-rate during public speaking was computed. Trait social anxiousness predicted reduced underestimation of self-rated peak heart rate after both negative ($R^2 = .12$, $\beta = -.34$, $t(1, 34) = -2.10$, $p < .043$) and relaxed anticipation of public speaking, $R^2 = .12$, $\beta = -.34$, $t(1, 34) = -2.11$, $p < .042$. This association was not significant anymore, after partialling out the effect of *self-related negative cognitions*, all p's $\geq .11$. No association was found between estimation bias and general trait anxiety, STAI-trait, all p's $\geq .61$.

2.3.3.3.4 Behavior Ratings

In addition to the main effects of Task (Negative vs. Relaxed Anticipation; see Table 13), there was a Task x Task-Order interaction for dependent variable *Overall Impression, F*(1, 29) = 12.20, $p < .01$, partial-$\eta^2 = .30$. While the group beginning with the negative anticipation improved substantially from the first ($M = 54.91$, $SD = 5.51$) to the second speech ($M = 58.39$, $SD = 4.78$), the group beginning with relaxed anticipation changed minimally from the first ($M = 56.38$, $SD = 6.61$) to the second speech, $M = 55.39$, $SD = 4.78$. All other main effects or interactions were not significant, $p \geq .05$.

Notably, public speaking performance was not correlated with trait social anxiousness (SPAI and FNE), general trait anxiousness (STAI-trait), self rated anxiety during anticipation, and the amount of *self-related negative cognitions* during anticipation (SSPS-N), all p's $\geq .06$. However, higher self-rated peak anxiety during speaking was associated with reduced content and rhetoric quality during both relaxed ($r = .34$, $p < .045$) and negative anticipation ($r = .39$, $p < .02$).

Observer Ratings for different aspects of public speaking performance	Relaxed Anticipation *M* (*SD*)	Negative Anticipation *M* (*SD*)	Task-Effect $F(df, df_{error})$, p, partial-η^2
Nonverbal Symptoms of Anxiety	43.79 (7.33)	43.53 (6.58)	$F(1, 34) = .23, .64, .01$
Content and Rhetoric Quality	57.13 (4.91)	56.30 (5.95)	$F(1, 34) = 1.06, .31, .03$
Anxiety	44.41 (6.68)	44.38 (7.19)	$F(1, 34) = .014, .91, .01$
Overall Impression	56.93 (5.97)	55.62 (6.01)	$F(1, 29) = 3.80, .06, .12$

Table 13: Means and standard deviations for observer ratings obtained during public speaking (T-scores, 50 marks average performance) at baseline and during relaxed vs. negative anticipation of public speaking. The last column indicates whether ratings were significantly different in the two anticipatory conditions.

Furthermore, higher self-rated peak anxiety during speaking was associated with higher observer ratings of anxiety during negative anticipation, $r = .37$, $p < .03$. Finally, higher self-rated confidence to make a good impression was associated with higher observer ratings of content and rhetoric quality during both negative ($r = .36$, $p < .03$), and relaxed anticipation, $r = .41$, $p < .01$.

2.3.3.3.5 Autonomic Measures

A multivariate test using the Pillay-Spur criterion with measures of autonomic physiological activation as dependent variables (SCL, HR, and $\log_{10}$ transforms of HRV-HF) showed a significant main effect Task, $F(6, 27) = 3.09$, $p < .02$, partial-$\eta^2 = .41$. There was no significant difference between the two groups randomized to different Task-Order, $F(3, 30) = 1.18$, $p < .34$, partial-$\eta^2 = .11$. However, there was a significant Task by Task-Order interaction, $F(6, 27) = 3.23$, $p < .02$, partial-$\eta^2 = .42$. This multivariate test was followed up

with univariate analyses and planned contrasts to examine task-effects in each dependent measure, for descriptives see Table 14, p. 139.

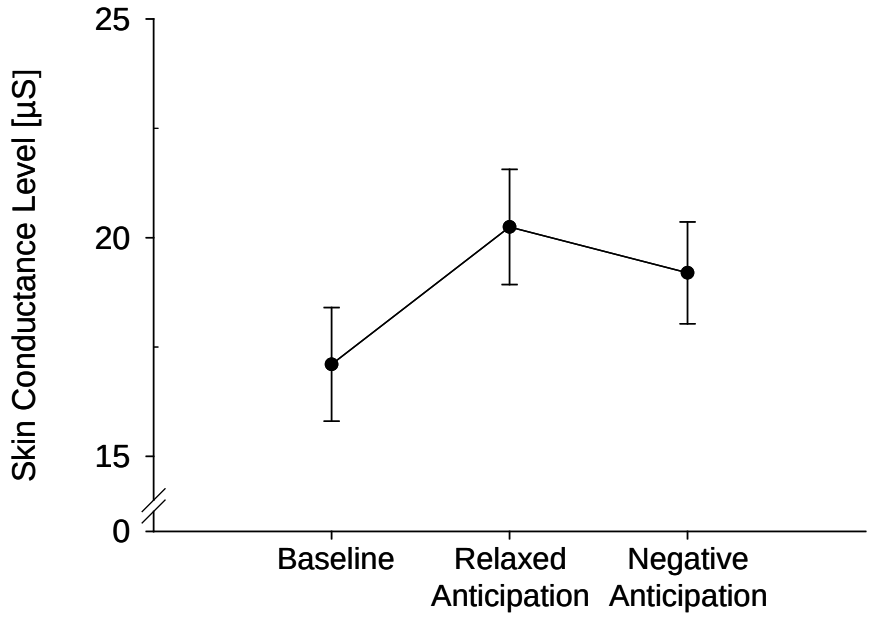

Figure 18: Means of skin conductance level [µS] at baseline and during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

The Task effect was significant for SCL ($F(1.53, 51.94) = 4.92$, $p < .01$, partial-$\eta^2 = .13$) and planned contrasts showed that SCL was elevated during both negative ($F(1, 34) = 6.16$, $p < .02$, partial-$\eta^2 = .15$) and relaxed anticipation ($F(1, 34) = 5.55$, $p < .02$, partial-$\eta^2 = .14$), in comparison to baseline, see Figure 18. There was no significant difference between negative and relaxed anticipation, $F(1, 34) = 1.68$, $p < .20$, partial-$\eta^2 = .05$. The two groups randomized to different Task-Order were not significantly different, $F(1, 34) < 1$. There was no Task by Task-Order interaction, $F(1.53, 51.94) = 1.75$, $p < .19$, partial-$\eta^2 = .05$.

Similarly, the Task effect was significant for HR, $F(2, 68) = 9.75$, $p < .001$, partial-$\eta^2 = .22$. Planned contrasts showed that HR was significantly higher during both negative ($F(1, 34) = 15.38$, $p < .001$, partial-$\eta^2 = .31$) and relaxed anticipation ($F(1, 34) = 9.05$, $p < .01$, partial-$\eta^2 = .21$) in comparison to baseline, see Figure 19.

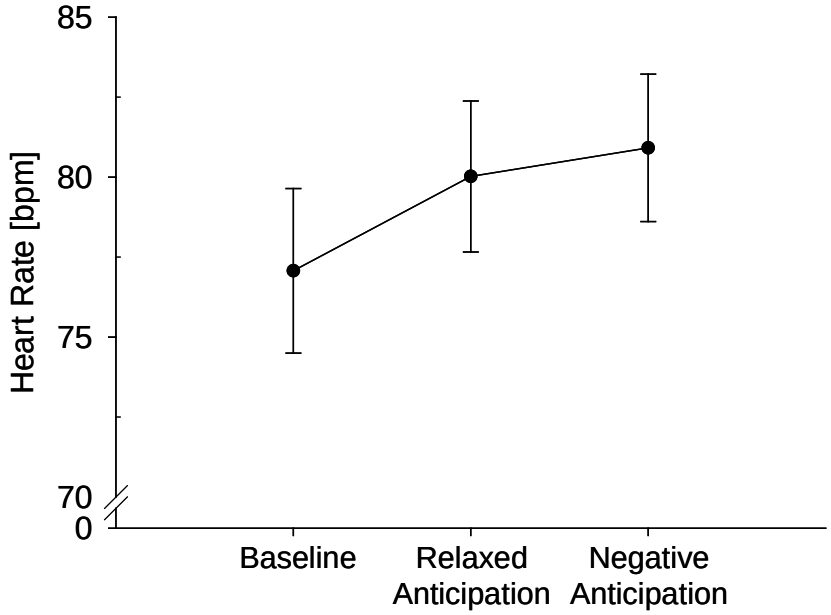

Figure 19: Means of heart rate [bpm] at baseline and during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

There was no significant difference between negative and relaxed anticipation, $F(1, 34) = 1.13$, $p < .30$, partial-$\eta^2 = .03$. The two groups randomized to different Task-Order were not significantly different, $F(1, 34) = 2.34$, $p < .14$, partial-$\eta^2 = .07$. However, there was a significant Task by Task-Order interaction, $F(2, 68) = 4.27$, $p < .02$, partial-$\eta^2 = .11$. Contrast computations revealed that self-reported anxiety decreased significantly from the first ($M = 81.68$, $SD = 13.96$) to the second anticipation ($M = 79.25$, $SD = 13.95$), as indicated by a significant Task by Task-Order interaction, $F(1, 34) = 11.17$, $p < .01$, partial-$\eta^2 = .25$. Finally, there was a significant Task effect for $\log_{10}$ transformed HRV-HF, $F(1.51, 48.41) = 7.10$, $p < .01$, partial-$\eta^2 = .18$.

Planned contrasts showed that HRV-HF was significantly decreased during negative anticipation ($F(1, 32) = 9.58$, $p < .01$, partial-$\eta^2 = .23$) and marginally significantly decreased during relaxed anticipation ($F(1, 32) = 3.27$, $p < .08$, partial-$\eta^2 = .09$), when compared to baseline, see Figure 20.

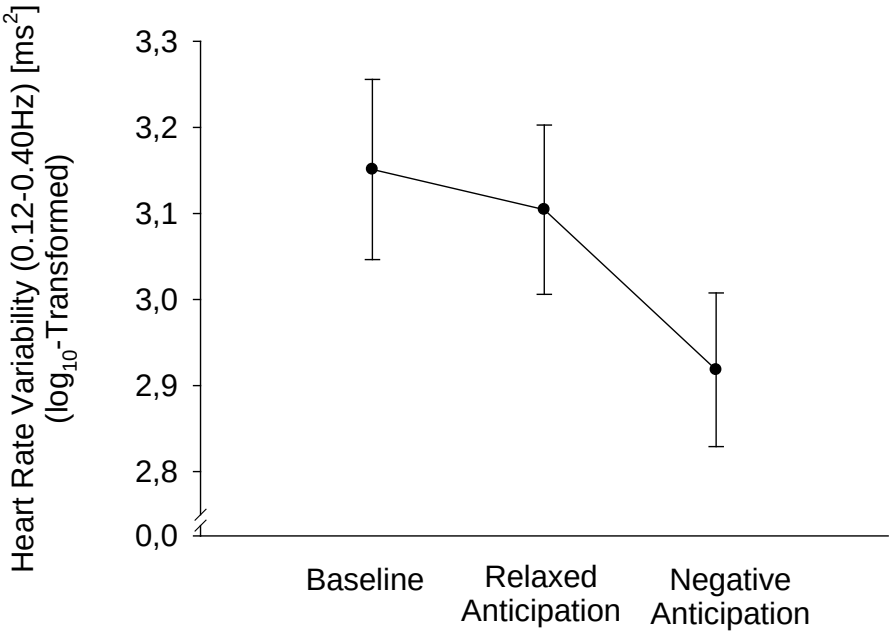

Figure 20: Means of $\log_{10}$-transforms of heart rate variability power spectral density [ms^2] in the high frequency band (0.12 – 0.40 Hz) at baseline and during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

Consequently, HRV-HF was significantly lower during negative than during relaxed anticipation, $F(1, 32) = 7.44$, $p < .01$, partial-$\eta^2 = .19$. A marginally significant main effect Task-Order indicated that HRV-HF was generally higher in the group beginning with negative anticipation, $F(1, 32) = 3.60$, $p < .07$, partial-$\eta^2 = .10$. There was no Task by Task-Order interaction, $F(1.51, 48.41) < 1$.

Autonomic Measures	Baseline *M* (*SD*)	Relaxed Anticipation *M* (*SD*)	Negative Anticipation *M* (*SD*)
SCL	17.10 (7.80)	19.20 (6.98)	20.24 (7.90)
HR	77.07 (15.43)	80.02 (14.17)	80.91 (13.82)
HRV-HF (0.15 – 0.4 Hz)	3.04 (.68)	2.94 (.60)	2.83 (.60)

Table 14: Means and standard deviations for autonomic measures (skin conductance level [SCL, µSi], heart rate [HR, bpm], $\log_{10}$-transforms of heart rate variability in the high frequency range [HRV-HF, ms^2]) at baseline and during relaxed vs. negative anticipation of public speaking.

2.3.3.3.6 Stimulus Properties

For ratings of valence there was a main effect of Stimulus-Type ($F(1, 35) = 44.13$, $p < .001$, partial-$\eta^2 = .56$), indicating that words were rated more negative ($M = -.97$; $SD = .44$) than facial expressions, $M = -.26$; $SD = .53$. There was no main effect of Valence or Stimulus-Type by Valence interaction, all F's < 1.

For ratings of arousal there was a main effect of Stimulus-Type ($F(1, 35) = 6.58$, $p < .02$, partial-$\eta^2 = .16$), indicating that words were rated more arousing ($M = 3.49$; $SD = 1.37$) than facial expressions, $M = 3.00$; $SD = 1.38$. There was no main effect of Valence ($F(2, 70) = 1.15$, $p < .32$, partial-$\eta^2 = .03$) or Stimulus-Type by Valence interaction, $F < 1$.

Finally, for ratings of complexity, there was no main effect of Stimulus-Type ($F(1, 34) = 1.64$, $p < .21$, partial-$\eta^2 = .05$), main effect of Valence ($F < 1$), or Stimulus-Type by Valence interaction, $F(2, 68) = 2.81$, $p < .07$, partial-$\eta^2 = .08$.

Rating	Facial-Expressions			Words		
	happy	afraid	angry	no threat	general threat	social threat
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Valence (*n* = 36)	-.19 (.69)	-.27 (.65)	-.32 (.72)	-.98 (.72)	-.86 (.71)	-1.08 (.57)
Arousal (*n* = 36)	3.88 (1.52)	3.98 (1.52)	4.14 (1.35)	4.43 (1.48)	4.53 (1.48)	4.51(1.42)
Complexity (*n* = 35)	4.33 (1.11)	4.25 (1.34)	4.49 (1.12)	4.49 (.99)	4.75 (1.10)	4.43 (.95)

Table 15: Means and standard deviations for ratings of valence, arousal, and complexity for the different facial expressions (angry, neutral, and happy), and words (no threat, general threat, and social threat). Scales ranged from 1 to 9 for extremely low to extremely high arousal and complexity and from -4 to +4 for extremely negative to extremely positive ratings of valence.

When compared to houses (*M* = -.14; *SD* = .62), facial expressions were rated equally arousing (*t*(35) = 1.12, *p* < .27, *d* = .21), but words were rated more arousing, *t*(35) = 7.09, *p* < .001, *d* = 1.54. Arousal ratings for houses (*M* = 3.06; *SD* = 1.25) were similar to ratings of facial expressions (*t*(35) =.52, *p* < .61, *d* = .32), but words were rated more negative, *t*(35) = -2.48, *p* < .02, *d* = .05. Finally, complexity ratings for houses (*M* = 3.37; *SD* = 1.07) were similar to both ratings for faces, *t*(34) = -1.08, *p* < .19, *d* = .19) and words, *t*(35) = .10, *p* < .92, *d* = .01. See Table 15 for descriptive data on the stimulus properties.

2.3.3.3.7 Startle Eye-Blink Amplitude

For dependent variable *startle amplitude*, there was a significant main effect Stimulus-Type (*F*(1, 31) = 27.46, *p* < .001, partial-η^2 = .47), indicating higher startle amplitudes when words

were read silently (M = .52; SD = .25), as compared to searching cues on the facial expressions, M = .46; SD = .26, see Figure 21. There was no main effect of Valence (F(2, 62) = 1.23, p < .30, partial-η^2 = .04) and no mean effect of Task (negative vs. relaxed anticipation, F(1, 31) < .01, $p \geq .95$, partial-η^2 < .01) or Valence by Task interaction, F(2, 62) = .78, $p \geq$ .46, partial-η^2 = .03, see Table 16. Furthermore, there was no Stimulus-Type by Task interaction, (F(1, 31) = 1.24, $p \geq .28$, partial-η^2 = .04), Stimulus-Type by Valence interaction, (F(1, 62) = 1.21, $p \geq .31$, partial-η^2 = .04), or three-way interaction of Stimulus-Type, Valence, and Task, F(2, 62) = .58, $p \geq .56$, partial-η^2 = .02. However, a significant interaction of Task by Task-Order, (F(1, 31) = 20.89, p < .001, partial-η^2 = .40), indicated a significant decrease of startle amplitudes from the first to the second anticipation. Furthermore, a significant three-way interaction of Task, Stimulus-Type, and Task-Order (F(1, 31) = 5.89, p < .03, partial-η^2 = .16), suggested a different time course of habituation for words vs. facial expressions in the two groups beginning with different anticipatory tasks. In the group beginning with the relaxation procedure, the decrease of startle amplitudes from the first to the second anticipation period was greater in responses to words than to facial expressions. In contrast, the group beginning with negative anticipation showed stronger reductions to facial expressions.

Despite the non-significant Valence effect, startle responding to words and facial expressions was further examined in separate analyses, because different pathways and brain structures may have been involved in the processing of these different stimulus categories. However, the only effects yielding significance were a general decrease across time, as indicated by interactions of Task by Task-Order, facial expressions: F(1, 31) = 12.51, p < .001, partial-η^2 = .29; words: F(1, 31) = 26.50, p < .001, partial-η^2 = .46.

In a final analysis, startle amplitudes were collapsed across all types of stimuli to boost reliability. There was no significant difference between square-root transforms of startle

amplitude during negative and relaxed anticipation, $F(1, 33) = 1.35$, $p < .25$, partial-$\eta^2 = .04$, see Table 16, p.143. Furthermore, there was no difference between the two groups randomized to different Task-Order ($F(1, 33) = 1.73$, $p < .20$, partial-$\eta^2 = .05$), but a significant Task by Task-Order interaction ($F(1, 33) = 23.26$, $p < .001$, partial-$\eta^2 = .41$), suggesting a significant decrease of startle amplitude from the first ($M = .60$, $SD = .30$) to the second anticipation ($M = .44$, $SD = .23$).

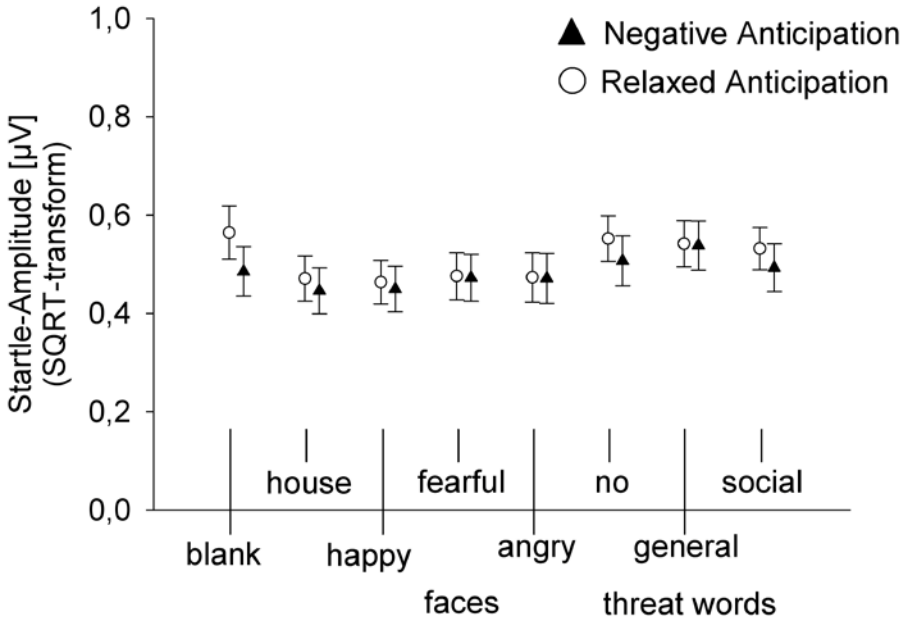

Figure 21: Means of square-root transformed startle amplitudes [µV] during processing of facial expression (angry, fearful, and happy), words (social threat, general-threat, and no-threat), and control stimuli (blank screen and houses) during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

As in experiment 1, participants who descriptively showed the expected response patterns to facial expressions ($n = 6$) and words ($n = 8$) were identified. However, only one participant showed this response pattern for both faces and words. In addition, these individuals had no significantly elevated scores on any of the measures assessing state or trait social anxiety, all p's $\geq .10$. Finally, there were no significant differences between the two anticipatory

conditions in these subgroups, all *p*'s ≥ .06. Using intra-individual T-scores for the analysis of startle responding to reduce inter-personal variance did not affect the findings.

Measures by Response Domain	Negative Anticipation *M* (*SD*)	Relaxed Anticipation *M* (*SD*)
Startle Eye-Blink		
Startle Amplitude	.50 (.29)	.54 (.27)

Table 16: Means and standard deviations for square-root transformed amplitudes of startle eye-blink responses [µV] collapsed across all stimulus categories during relaxed vs. negative anticipation of public speaking.

2.3.3.3.8 Startle Eye-Blink Latency

For dependent variable *startle latency*, there was no significant main effect Stimulus-Type ($F(1, 20) = 2.88$, $p < .10$, partial-$\eta^2 = .13$), Task ($F(1, 20) = .20$, $p < .66$, partial-$\eta^2 = .01$), or Valence, $F(3, 60) = 1.98$, $p < .13$, partial-$\eta^2 = .09$, see Figure 22. There were no significant two-way interactions of Stimulus-Type and Valence ($F(3, 60) = 2.40$, $p < .08$, partial-$\eta^2 = .11$), Valence and Task ($F(3, 60) = 1.65$, $p < .19$, partial-$\eta^2 = .08$), or Stimulus-Type and Task, $F(1,20) = .14$, $p < .71$, partial-$\eta^2 = .01$. The three-way interaction of Task, Valence, and Stimulus-Type was not significant, $F(3,60) = 1.70$, $p < .18$, partial-$\eta^2 = .08$. Finally, there were no significant main effects or interactions of Task-Order, all *p*'s ≥ .15.

Similar to startle amplitude, another analysis was computed with startle latencies collapsed across all types of stimuli to boost reliability. Startle latency was marginally significantly lower during relaxed as compared to negative anticipation $F(1, 28) = 3.68$, $p < .07$, partial-$\eta^2 = .12$, see Table 17, p. 144. There was no difference between the two groups randomized to different Task-Order ($F(1, 28) = 1.35$, $p < .26$, partial-$\eta^2 = .05$) and no Task by Task-Order,

$F(1, 28) = 2.05$, $p < .16$, partial-$\eta^2 = .07$. Using intra-individual T-scores for the analysis of startle responding to reduce inter-personal variance did not affect the findings.

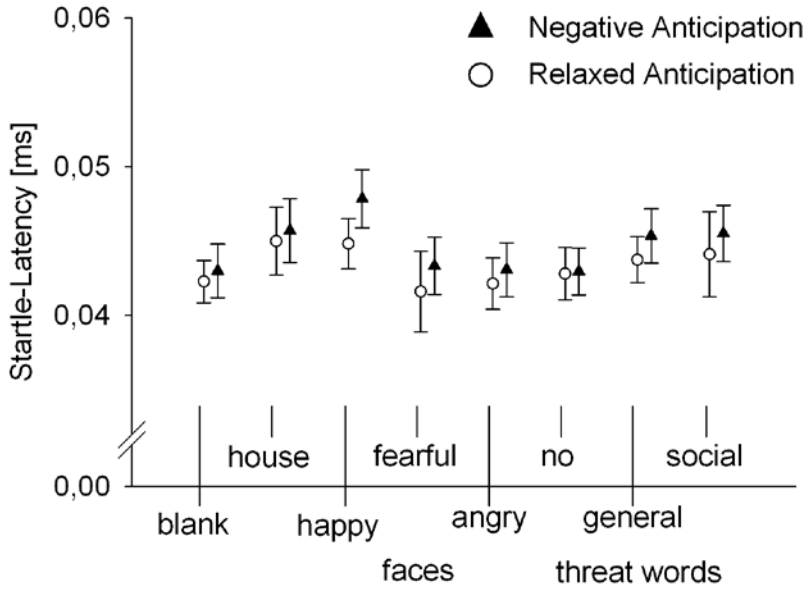

Figure 22: Means of startle latencies [ms] during processing of facial expression (angry, fearful, and happy), words (social threat, general-threat, and no-threat), and control stimuli (blank screen and houses) during relaxed vs. negative anticipation of public speaking. Error bars represent standard errors of the mean.

Measures by Response Domain	Negative Anticipation *M* (*SD*)	Relaxed Anticipation *M* (*SD*)
Startle Eye-Blink		
Startle Latency	.046 (.009)	.044 (.007)

Table 17: Means and standard deviations for latencies of startle eye-blink responses [ms] collapsed across stimulus categories during relaxed vs. negative anticipation of public speaking.

2.3.3.3.9 Associations with Startle Amplitude

Because there was no stimulus-related modulation of startle responding, no further steps of the planned test for mediation by biased processing of *external social threat-cues* were performed.

Due to the same reason, startle amplitudes to different stimuli were collapsed across stimulus categories for the following analyses. During negative anticipation, there were significant correlations of startle amplitude with negative cognitions (SSPS-N, $r = .34$, $p < .05$), self-reported anxiety ($r = .40$, $p < .02$), FNE ($r = .42$, $p < .02$, $n = 31$), BAS subscale *reward responsiveness* ($r = .38$, $p < .02$, $n = 35$) and NEO-FFI scale *extraversion,* $r = -.36$, $p < .04$, $n = 25$.

During relaxed anticipation, there were significant correlations of startle amplitude and negative cognitions (SSPS-N, $r = .47$, $p < .01$), FNE ($r = .56$, $p < .001$, $n = 30$), BDI-II ($r = .37$, $p < .03$, $n = 34$), and NEO-FFI scales *neuroticism* ($r = .40$, $p < .02$, $n = 34$) and *extraversion,* $r = -.51$, $p < .01$, $n = 34$.

Ratings indicated that words were perceived as more threatening than the facial expressions. This was reflected in higher startle amplitudes to words. Therefore, correlations with the difference between startle amplitudes with words and startle amplitudes with facial expressions were explored. However, none of the questionnaires or rating scales correlated significantly with this measure, all $r < \pm .27$, all p's $\geq .12$.

2.3.3.3.10 Associations with Startle Latency

Similarly, latencies of startle response to different stimuli were collapsed across stimulus categories due to the absence of significant differences between the categories. During negative anticipation, a significant correlation with the BAS subscale *reward responsiveness* emerged during both, relaxed ($r = -.36$, $p < .03$) and negative anticipation ($r = -.39$, $p < .03$). Furthermore, startle latency correlated with the NEO-FFI subscale *openness* (*r*

= .34, $p < .05$). No further correlations with trait and state measures or autonomic measures emerged, all p's $\geq .05$.

2.3.3.3.11 Mediation Analyses

Trait social anxiety specifically predicted the amount of negative cognitions (SSPS-N), self-reported anxiety, and square-root transformed startle-amplitude during both negative and relaxed anticipation, but not at baseline (Table 18).[5] In contrast, trait social anxiety and general trait anxiety ($R^2 = .15$, $\beta = -.39$, $t(1, 34) = -2.41$, $p < .02$) predicted $\log_{10}$ transformed HRV-HF at baseline. Finally, HR and SCL were neither related to trait social anxiety nor to general trait anxiety. See appendix 5.4, p. 229, for scatterplots of significant correlations between SPAI and outcome measures.

In two mediation models, it was examined if negative cognition (SSPS-N) mediated the relationship between trait social anxiety (SPAI) and effects of anticipation on self-reported anxiety and $\log_{10}$ transformed HRV-HF (Figure 23). Step 1 of Baron and Kenny's (1986) procedure showed that trait social anxiety predicted the increase of self-reported anxiety ($R^2 = .13$, $\beta = .36$, $t(1, 33) = 2.21$, $p < .03$) and the decrease of HRV-HF ($R^2 = .24$, $\beta = -.50$, $t(1, 33) = -3.25$, $p < .01$) from baseline to negative anticipation (path c in Figure 23 A and B).

In addition, specificity of this effect was confirmed with an analogous analysis with predictor SPAI-trait, all t's < 1.98. Step 2 (path a in Figure 23 A and B) was established for both mediation models: trait social anxiety (SPAI) predicted the amount of negative cognitions (SSPS-N) during negative anticipation, $R^2 = .57$, $\beta = .76$, $t(1, 33) = 6.65$, $p < .001$. Step 3 (path b in Figure 23 A and B) confirmed that negative anticipatory cognitions (SSPS-N) mediated the relationship between trait social anxiety (SPAI) and the increase of self-reported

[5] There were only low and non-significant correlations between trait social anxiety and amplitude of EMG-baselines, confirming that the effect is not a result of signal strength.

anxiety from baseline to negative anticipation (R^2 = .24, β = .50, t(2, 31) = 1.09, p < .05) as well as the decrease of HRV-HF from baseline to negative anticipation, R^2 = .35, β = -.49, t(2, 31) = -2.20, p < .04. Full mediation was shown by Step 4 (path c' in Figure 23 A and B), which confirmed that the *direct effect* of trait social anxiety on the outcome variables, while controlling for the effect of the mediator was no longer significant (self-reported anxiety: R^2 = .24, β = -.01, t(2, 31) = -.02, p < .99; HRV-HF: R^2 = .35, β = -.13, t(2, 31) = -.58, p < .57). This was corroborated by significant *indirect effects,* self-reported anxiety: 0.36; Sobel *z-value* = 1.99, p < .05; HRV-HF: -0.37; Sobel *z-value* = -2.12, p < .03.

	Baseline R^2, β *t (df)*	Negative Anticipation R^2, β *t (df)*	Relaxed Anticipation R^2, β *t (df)*
Self-Report Measures			
Self-Reported Anxiety	.11, .33, 1.99 (1,33)	.42, .65, 4.91 (1,33)***	.18, .43, 2.67 (1,33)**
SSPS-N	--	.57, .76, 6.65 (1,33)***	.28, .53, 3.63 (1,34)***
Autonomic Measures			
SCL	.05, .21, 1.28 (1,34)	.02, .13, .76 (1,34)	.03, .16, .96 (1,34)
HR	.01, -.08, -.46 (1,34)	.01, -.02, -.14 (1,34)	.01, -.03, -.17 (1,34)
HRV-HF	.13, .36, 2.24 (1,33)*	.01, .08, .47 (1,33)	.07, .27, 1.60 (1,32)
Startle Eye-Blink			
Startle Amplitude	--	.20, .45, 2.95 (1,34)**	.24, .49, 3.19 (1,33)**

Table 18: Parameters for separate linear regression analyses of criteria (self-reported anxiety, negative cognitions [SSPS-N], skin conductance level [SCL], heart rate [HR], $\log_{10}$-transforms of heart rate variability in the HF-range [HRV-HF], square-root transforms of startle eye-blink amplitude) on predictor trait social anxiety (SPAI) at baseline and during negative and relaxed anticipation of public speaking; * p < .05, ** p < .01, *** p < .001.

The third model on the presumed associations of trait social anxiousness and biased processing of *external social threat-cues* was not tested due to the lack of startle modulation, disconfirming the first step of the mediation test (i.e. an association between predictor and criterion).

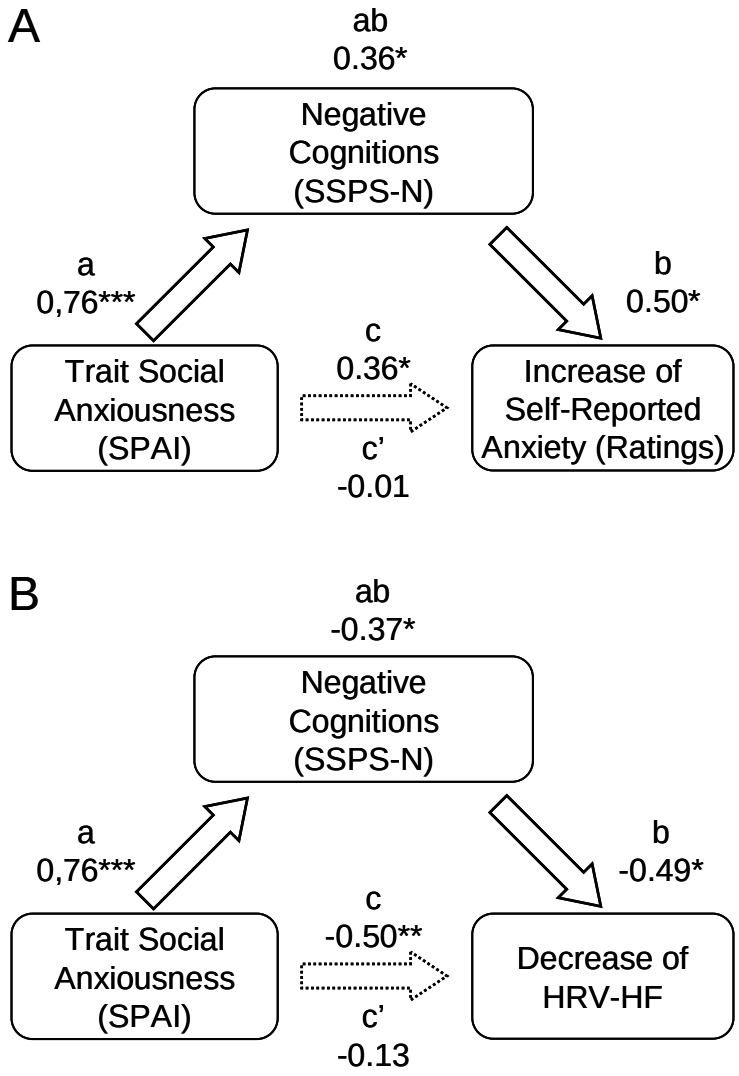

Figure 23: Results of the hypothesized mediation models: A) negative cognitions (SSPS-N) as a mediator between trait social anxiety (SPAI) and increases of self-reported anxiety from baseline to negative anticipation; B) negative cognitions (SSPS-N) as a mediator between trait social anxiety (SPAI) and decreases of $\log_{10}$-transforms of heart rate variability in the HF-range (HRV-HF) from baseline to negative anticipation; standardized regression coefficients, * $p < .05$, ** $p < .01$, *** $p < .001$.

In a final model, cognitive mediation of square-root transformed startle amplitudes was investigated. As reported above, trait social anxiety (SPAI) predicted startle amplitude during both, negative and relaxed anticipation. Hence requirements of Step 1 in Baron and Kenny's (1986) procedure were met. In Step 2, it was shown that trait social anxiety predicted the amount of negative cognitions (SSPS-N) not only during negative but also relaxed anticipation, $R^2 = .53$, $\beta = .53$, $t(1, 34) = 3.63$, $p < .001$. However, in Step 3 of Baron and Kenny's (1986) procedure, negative cognitions were not predictive of startle amplitude during negative ($R^2 = .20$, $\beta = -.01$, $t(2, 32) = .02$, $p < .98$) or relaxed anticipation, $R^2 = .30$, $\beta = .30$, $t(2, 32) = 1.71$, $p = .10$. Finally, Step 4 showed that marginally significant effects of trait social anxiety persisted while controlling for the effect of negative cognition during negative ($R^2 = .20$, $\beta = .46$, $t(2, 32) = 1.89$, $p < .07$) and relaxed anticipation, $R^2 = .30$, $\beta = .33$, $t(2, 32) = 1.94$, $p < .06$.

Furthermore correlations of startle amplitude with other measures were explored. Significant correlations were found with negative cognitions (SSPS-N) during both negative ($r = .34$, $p < .05$) and relaxed anticipation ($r = .47$, $p < .01$) as well as with self-reported anxiety during negative anticipation only, $r = .40$, $p < .02$.

2.3.4 Discussion

In experiment 3 a core assumption of the cognitive model of social anxiety was examined (Clark & Wells, 1995; Rapee & Heimberg, 1997), namely, that self-related negative cognitions mediate the relationship between trait social anxiety and anxious responding during anticipation of a socially threatening situation (i.e. public speaking). Results of experiment 3 provide strong support for this hypothesis. Consistent with previous reports (Hinrichsen & Clark, 2003; Vassilopoulos, 2005), self-reported anxiety increased significantly from baseline to anticipation of public speaking, when participants engaged in self-related negative cognitions. Furthermore, self-reported anxiety but not general trait

anxiety predicted higher intensity of negative cognition (SSPS-N) and self-reported anxiety. The mediation analyses with the procedures described in Baron and Kenny (1986) and Sobel (1982) revealed, that this heightened anxiety response was fully mediated by the amount of self-related negative cognitions during anticipation (SSPS-N). This supports a central predication of the cognitive model. As expected, the participants had significantly reduced negative cognitions during relaxed anticipation. Greater trait social anxiety predicted both higher intensity of negative cognitions (SSPS-N) and higher self-reported anxiety during relaxed anticipation. This finding suggests an interaction between trait social anxiety and situational context, regardless of the nature of the anticipatory task. However, self-reported anxiety did not increase significantly from baseline during relaxed anticipation. This finding shows that manipulating anticipatory cognitions successfully affected the emergence of anticipatory anxiety.

Participants were specifically encouraged to assess their feelings when rating self-reported anxiety, as opposed to assessing their cognitions (SSPS-N). Although these are standard methods to measure emotional response and cognitions, these measures may be subject to rater biases such as a halo effect. The physiological measures are unlikely to be affected by these biases. Interestingly, the results of heart rate variability in the high frequency spectrum (HRV-HF) mirror the findings of self-reported anxiety when examining task-induced changes from baseline. Increased self-reported anxiety during negative anticipation was matched by a significant decrease of HRV-HF. Moreover, higher levels of trait social anxiousness predicted stronger decrease of HRV-HF. This relationship was fully mediated by self-related negative cognitions (SSPS-N). Although there was also a significant decrease of HRV-HF in the relaxed anticipation condition, it was significantly smaller. A positive correlation between general trait anxiety and baseline HRV-HF suggests a general deficiency in emotional reactivity to situational demands (Beauchaine, 2001; Thayer & Brosschot, 2005). At the same

time, there was a positive correlation between baseline HRV-HF and trait social anxiety, suggesting flexibility of emotional reactivity in socially anxious individuals. During anticipation of public speaking, this relationship reversed. In line with the hypothesis, trait social anxiety predicted a deficient parasympathetic regulatory response to anticipation of public speaking, which was especially pronounced during negative anticipation. Taken together, these findings suggest that these effects hinge on a triple interaction of trait, situational context, and cognition, which is in line with the DSM-IV definition of social anxiety disorder (APA, 2000).

Consistent with previous results (Mauss, Wilhelm, & Gross, 2004), further evidence for physiological correlates of social anxiety was limited. Heart rate (HR) and skin conductance level (SCL) increased from baseline to both negative and relaxed anticipation. HR and SCL were not correlated with trait social anxiety or general trait anxiety. First, this suggests that all participants were activated and had increased sympathetic arousal in anticipation of public speaking (Erdmann & Baumann, 1996; Schwerdtfeger, 2004). Second, it further underlines the specific association of parasympathetic reactivity and social anxiety. Interestingly, longitudinal studies have linked low heart period variability in response to cognitive stressors to behavioral inhibition in infants and children (Kagan, Reznick, & Snidman, 1987), a predictor of later development of SAD (Hayward, Killen, Kraemer, & Taylor, 1998). In adults, HRV-HF decreases in response to situational demands were associated with deficiencies in emotional regulation (Thayer & Lane, 2000). However, in contrast to these studies, a deficient parasympathetic response emerged, rather than sympathetic dis-inhibition by parasympathetic feed-forward loops. Notably, other studies have also failed to show sympathetic hyperarousability in socially anxious individuals (Brown, Chorpita, & Barlow, 1998; Mauss, Wilhelm, & Gross, 2004). Taken together, these findings open up an interesting perspective for future studies on correlates of emotion regulation as potential precursors in the

etiology of the negative cognitive style associated with SAD. In this pursuit it will be important to disambiguate the relative contribution of sympathetic vs. parasympathetic reactivity in response to social threat.

To complete the multivariate assessment of anxiety, observer ratings of the public speaking performance were compared across experimental conditions and to self-assessments. Observer ratings of the overall impression as a social performer were generally lower in the negative anticipation condition. This confirms what physiology and self-rated anxiety suggests: Negative anticipation of public speaking effectively moderated public speaking performance. Increased self-rated peak anxiety during speaking and decreased confidence to make a good impression on the audience was correlated with observer-ratings of low content und rhetoric quality in both anticipation periods. These effects were not related to high trait social anxiousness. This suggests that speakers were aware of noticeable differences, but not because they had elevated trait social anxiety, but likely due to other individual and situational factors.

Interestingly, evaluating internal cues or at least one's cognitions about them appears to play an important role for social anxiety. Generally, participants in experiment 3 underestimated their heart-rate, when they were asked to indicate, what they thought their peak heart-rate had been during public speaking. Interestingly, with increasing levels of trait social anxiousness, this bias was reduced. This could indicate better accuracy, probably related to increased attention to somatic cues. People with SAD tend to believe that their own internal somatic experience is a good indicator of outward appearance (Mansell & Clark, 1999; Mellings & Alden, 2000; Wells & Papageorgiou, 2001) and possibly misperceive and exaggerate their physiological activation (e.g. Mulkens, de Jong, Dobbelaar, & Bogels, 1999; Steptoe & Vögele, 1992). However, scores on the body conscious scales (BCS) did not indicate a generally higher ability to perceive somatic cues in individuals with increased trait social

anxiousness. Furthermore, reduced estimation bias correlated with increased negative cognitions during anticipation of public speaking. Finally, the association between trait social anxiousness and estimation bias was gone after partialling out the effect of self-related negative cognitions. Therefore, the increased ratings may rather reflect cognitive bias than increased accuracy. Notably, biased evaluation of internal somatic cues may be a stabilizing factor of SAD, because it is impossible to disprove the accuracy of the factually less biased estimations of heart rate via reality checks (e.g. hear-rate biofeedback).

Findings with the startle paradigm indicate that threat-cue vulnerability was not increased in highly socially anxious individuals. There was no difference in startle responding across anticipatory tasks (negative vs. relaxed). Therefore, cognitive content apparently had no moderating downstream effect on implicit processing of environmental cues. Furthermore, neither affective modulation nor generally increased responses to emotional stimuli emerged in responses to facial expressions and verbal cues in comparison to control cues. Startle amplitudes were increased to words as compared to facial expressions. First, this shows that floor or ceiling effects do not explain the absence of affective startle modulation.

Second, these findings match the effects found in valence and arousal ratings of the stimuli. In contrast to experiments 1 and 2, the Likert-like scales used for these ratings were anchored with elaborate descriptions of the extreme end-points of the scales. As predicted, this led to much lower arousal ratings for facial expressions, when compared to ratings obtained without detailed verbal anchors. This suggests, that range bias may mislead interpretation of Likert-like ratings when they are compared to standardized stimulus sets comprising more extreme stimuli (e.g. the IAPS-picture set Lang, Bradley, & Cuthbert, 1999). Importantly, significant differences found with the anchored ratings matched the effects obtained with the startle paradigm; see the comparison of words vs. faces. This recommends further usage of the texts used for anchoring the ratings, see appendix 5.3.10, p. 223.

Similar to previous results (Panayiotou & Vrana, 1998) trait social anxiousness but not general trait anxiousness predicted generally increased startle amplitudes in our sample. In the negative anticipation condition, startle amplitude correlated with self-reported anxiety. There were no correlations of startle responding with HR or SCL. These findings parallel effects found for self-reported anxiety and HRV-HF. However, startle amplitude was not different in the relaxed vs. negative anticipation condition and startle response was not mediated by cognition. These findings raise doubt whether the interpretation of (Panayiotou & Vrana, 1998) fits to the current findings, as sympathetic arousal and cognitive content were unrelated to startle responding. Nonetheless, increased activation of the central nucleus of the amygdala may be responsible for increased startle amplitudes. Probably compensatory effects of prefrontal control or other brain structures involved in the neural circuitry of emotion processing are responsible that these effects were not stronger. Notably, this could have downstream effects on parasympathetic withdrawal, which may only kick in, when cognition and dysfunctional emotion regulation link up. Considerably, this post-hoc interpretation is only tentative and needs to be tested within the logic of functional-anatomical models of brain-body interactions involved in emotion regulation. Recent brain imaging studies have only started to reveal the framework for such investigations (Ahs, Sollers, Furmark, Fredrikson, & Thayer, 2009; Wager et al., 2009a; Wager et al., 2009b). The available data support that the amygdala and parasympathetic control as indexed by HRV may play a central role within these networks.

Lack of startle measurements at baseline complicates the interpretation of these results. Cornwell, Johnson, Berardi, & Grillon (2006) found similar positive correlations between startle amplitude and fear of negative evaluation not only during anticipation of public speaking in a virtual reality task (r = .46) but also during baseline (r = .40). Interestingly, they did not find such a correlation when the participants performed a non-social demanding

cognitive task. Probably, task-effort disrupted the association between trait social anxiousness and startle-amplitude in this experimental condition. Redirecting attentional focus away from emotional stimuli has been shown to curtail threat processing in low-anxiety individuals. High trait anxiety was associated with the persistence of threat processing unless working memory resources were exhausted (Dvorak-Bertsch, Curtin, Rubinstein, & Newman, 2007). Furthermore, effortful evaluation and appraisal may moderate amygdala-mediated emotion processing (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003) and focusing attention on a different effortful task may eliminate emotion related amygdala activation (Pessoa, McKenna, Gutierrez, & Ungerleider, 2002). Distraction and working memory load may therefore play an important moderating role for associations between social anxiety and startle responding.

Experiment 3 has a number of limitations. First, only self-related negative cognitions were considered as a mediator of social anxiety. Other potential mediators such as estimated social cost (Hofmann, 2004) may be even more specific to social anxiety. Second, Task-Order by Task interactions indicated a general decrease in all measures except SCL and HRV-HF from the first to the second anticipation of public speaking. Although one might expect that depending on the first task different carry over effects to the second anticipation might occur or that post processing of the first speech might affect the second anticipation period, these effects were principally consistent. In addition, the two groups randomized to different Task-Order were largely equivalent. Therefore, the most parsimonious interpretation is habituation or a reduction of overall arousal due to reduced uncertainty about the procedure in the second task. Although such effects should be further explored in future studies there was no indication that this compromised our main results. Third, HRV-HF is only a reliable indicator of parasympathetic activation when assessed under controlled conditions. We addressed this issue by using only data uncompromised by known sources of artifact. Furthermore, measuring shifts of HRV-HF is more reliable than baseline assessment of vagal tone (e.g.

Grossman & Taylor, 2007). Without controlling for the influence of respiratory parameters, it cannot be ruled out that faster breathing may have inflated HRV-HF decreases (Camm et al., 1996). Controlling for the influence of both tidal volume and respiration rate may further improve estimation of vagal activity from HRV-data (Ritz & Dahme, 2006; Schulz, Ayala, Dahme, & Ritz, in press). Fourth, although some studies have suggested that gender might be an important factor in social anxiety disorder, there were only minor differences between men and women, unrelated to social anxiety. Yet, gender effects may be stronger in individuals with a clinical diagnosis of social anxiety disorder. Finally, we used an experimental manipulation to encourage or discourage participants from engaging in negative thinking. Under the assumption that all other conditions were held constant between the two anticipatory conditions, the findings suggest that cognitions cause changes in self-reported anxiety and psychophysiology. Although other variables may be involved in this effect, this finding implies a functional role of this mediator. Such claims of causality remain to be tested in future studies with appropriate models and experiment designs (e.g. Steyer, 1992).

In sum, these findings substantiate the notion that self-related negative cognitions mediate effects of trait social anxiety on subjective and psychophysiological correlates of social anxiety when anticipating social threat. This supports a primary assumption of cognitive models of social anxiety (Clark & Wells, 1995; Rapee & Heimberg, 1997). Interestingly, preliminary evidence also emerged for cognitive mediation of a negative evaluation bias regarding somatic internal cues (heart rate). In contrast, experiment 3 provides no evidence for the assumption that biased processing of external social threat-cues is a mediator of social anxiety. The discovery of an association between trait social anxiousness and cognitive mediation of decreased HRV-HF during social challenge suggests an important role of deficient emotion regulation to social stress. A general association between higher trait social anxiousness with increased startle amplitudes suggests that amygdala activation may play an

important role in this complex dynamic. Effects on SCL, HR, and public speaking performance (self- and observer-ratings) were not related to trait social anxiety and self-related negative cognitions. The accumulating evidence for such systematic dissociations across emotional response systems should therefore encourage a revision of prevalent models of social anxiety.

3 General Discussion

3.1 Mediators of Social Anxiety: Integration of Findings and Outlook

The objective of this thesis was to identify and examine mediators of associations between trait social anxiousness and acute social anxiety in socially challenging situations. Based on a review of established models of social anxiety and empirical findings, two mediators of principal importance were identified: *negative self-related cognitions* and a specific information processing bias for *external social threat-cues*. In three experiments, predictions derived from a working model on mediators of social anxiety (section 1.3.8, p. 52) were examined.

3.1.1 Self-Related Negative Cognitions: Synopsis

Altogether, the findings of the three experiments provide strong evidence for cognitive mediation of self-reported and physiological symptoms of social anxiety.

First, induced negative self-related cognitions during anticipation of public speaking led to a significant increase of self-reported anxiety as compared to relaxed anticipation. Higher levels of trait social anxiousness predicted even more intense self-reported anxiety. This is in line with a dimensional view of social anxiety (McNeil, 2001), adopted in the working model. Notably, the intensity of negative cognitions fully mediated the association between trait social anxiousness and self-reported anxiety. This is the first study reporting full statistical mediation of anxiety symptoms in socially anxious individuals when being confronted with socially threatening situations. Furthermore, active manipulation of cognitive mediation via inducing negative self-related cognitions vs. relaxation suggests a causal role of this mediator.

In experiment 1, a correlation between trait social anxiousness and startle latency indicated motor hyper-responsivity of socially anxious individuals, which is part of the clinical presentation of SAD (e.g. Beidel, Turner, & Dancu, 1985). Notably, processing of emotional

facial expressions or focusing attention away from one's internal response to facial expressions disrupted this effect. Both findings suggest that strategies of focusing attention away from negative self-related cognitions may be a valuable tool for the therapy of SAD. Similar approaches (i.e. instructions to focus attention on environmental stimuli) have already proven to increase favorable effects of public speaking exposure therapy (Wells & Papageorgiou, 1998).

Socially anxious individuals also featured a decreased estimation bias of perceived heart rate. As discussed above (discussion of experiment 3, p. 149), this may rather reflect cognitive bias than increased perceptual accuracy. It seems impossible to disprove the accuracy of factually less biased estimations of heart rate via reality checks (e.g. heart-rate biofeedback). Therefore, biased evaluation of somatic cues should be investigated as a potential factor for the maintenance of SAD.

Furthermore, similar to previous reports (e.g. Mauss, Wilhelm, & Gross, 2004), dissociations across emotional response domains (self-report, observable behavior, physiology, Lang, 1993) emerged. This adds to the accumulating evidence that may be characteristic for social anxiety. First, performance in the public speaking task as assessed by both observer ratings and self-report was not related to trait levels of social anxiousness. There were no interactions with negative self-related cognitions. Second, all participants were activated to a similar level before holding a public speech, as indicated by increased heart-rates (HR) and skin-conductance levels (SCL). Induced negative self-related cognitions did not further increase HR and SCL. In contrast, changes in self-reported anxiety were paralleled by parasympathetic withdrawal, as indexed by increased heart-rate variability in the high frequency spectrum (HRV-HF). Again, the intensity of negative cognitions mediated the association between trait social anxiousness and the HRV-HF decrease.

Notably, this is the first study revealing a link between trait social anxiousness, negative cognitions and HRV-HF. This finding may explain why previous studies have largely failed in identifying physiological correlates of SAD. The same level of sympathetic arousal as indexed by SCL and HR may have more negative consequences, when the compensatory effect of parasympathetic activation is reduced. These findings match predictions derived from functional anatomic models of brain-body interactions in the domain of emotion regulation (Benarroch, 1993; Benarroch, 1997; Berntson, Sarter, & Cacioppo, 1998; Friedman, 2007). In these models, reduced HRV-HF has been suggested as a peripheral indicator of dysfunctional emotion regulation, for example in response to social threat. To identify causal dependencies between emotion regulation and social anxiety, future studies should examine temporal associations between the activation of specific brain structures involved in emotion regulation (Thayer & Lane, 2000), self-reported anxiety, and parasympathetic withdrawal in individuals with different levels of trait social anxiousness. First approaches in this direction have been reported lately (Ahs, Sollers, Furmark, Fredrikson, & Thayer, 2009; Wager et al., 2009a; Wager et al., 2009b).

3.1.2 External Social Threat-Cues: Synopsis

None of the three experiments found the expected pattern of startle potentiation to negative and startle attenuation to positive facial expressions as compared to neutral ones. Similarly, processing of verbal threat-cues did not lead to affective startle modulation. It is unlikely that methodic reasons such as a lack of sensitivity of the startle method or the question whether the stimuli used in experiments 1-3 were appropriate to elicit behaviorally relevant emotional reactions are responsible for the lack of findings, see discussions of experiments 1-3. This is supported by findings in experiment 2, where significant effects of positive vs. negative facial expressions emerged in spontaneous facial EMG activity of the corrugator supercilii. Yet, these effects were not related to trait social anxiousness.

On the other hand, startle amplitudes were elevated to both angry and happy facial expressions, as compared to neutral ones in experiment 1. Interpretation of effect size in experiment 2 further suggests that similar findings may emerge in larger samples of patients with SAD. These findings match results reported with low-arousing emotional pictures (Cuthbert, Bradley, & Lang, 1996). Therefore, the most likely reason for the absence of affective startle modulation is insufficient arousal induced by the stimuli. This interpretation is corroborated by low arousal ratings obtained for the facial expressions when scale-limits were anchored with descriptions matching highly arousing pictures (Lang, Bradley, & Cuthbert, 1999) inducing affective startle modulation reliably. In contrast to these findings, startle amplitudes were not significantly different across positive and negative emotional facial expressions, words, and neutral control stimuli in experiment 3. The main difference in this experiment was higher contextual social threat (anticipation of public speaking) than in the first two experiments (video observation). This has induced significant arousal in all participants, probably accompanied by increased preparatory apprehension and working memory demand. It is likely, that this has decreased the figure-ground contrast between situational threat and effects of emotional foreground stimuli. This may have further limited the capability to elicit affective startle modulation by weak foreground stimuli. Again, ratings with scales anchored as described above support this interpretation. When these ratings indicated a significant difference between the emotional properties of stimuli (valence, arousal), effects on startle amplitudes were also significant. Notably, these differences were not related to trait social anxiousness or cognitive mediation. Altogether, this suggests that external social threat-cues may elicit affective startle responding when sufficiently arousing cues with large valence contrasts are used. However, ecologically valid socially challenging situations may rather attenuate effects of automatically processed external social threat-cues. Furthermore, facial expressions are most likely not the kind of external social threat-cues,

which elicit remarkable fear in these situations. Therefore the current findings suggest consistently across three experiments with different levels of social challenge and participants with trait social anxiousness ranging from low-levels to levels associated with a clinical diagnosis of social anxiety disorder (SAD) that threat-cue vulnerability plays a minor role as a mediator of social anxiety.

Nonetheless, the abundance of findings in brain imaging studies that have even found specific correlations of amygdala activation to threatening facial expressions and social anxiousness (see also Phan, Fitzgerald, Nathan, & Tancer, 2006; Stein, Goldin, Sareen, Zorrilla, & Brown, 2002), raises the question why this activation does not mediate startle modulation. It may be necessary to differentiate the activation within single nuclei of the amygdala to clarify whether these reports reflect activation in the central nucleus of the amygdala being responsible for affective startle modulation. In the meantime, a final comparison of the published studies reporting affective startle modulation to facial expressions with experiments reporting null-findings (see introductions and discussions of experiments 1-3) may reveal conditions under which affective startle modulation to facial expressions may be most likely:

1) Individuals with extreme levels of trait social anxiousness (Heinrichs & Hofmann, 2001; McNeil, 2001)
2) Homogeneity of individuals in the sample with regards to feared cues, ideally sharing a specific fear for example regarding angry interaction partners (Cuthbert et al., 2003; Gros, Hawk, & Moscovitch, 2009)
3) Threat-cues which are capable of evoking strong emotional responses (e.g. dynamic film clips or even live presentations by trained actors; suggested by experiment 3)
4) Stimuli with individual relevance to the participants (e.g. a teacher from a previously failed oral exam, see discussion in Gilboa-Schechtman, Foa, & Amir, 1999)

5) High perceived dominance of the person enacting a facial expression (Hess, Sabourin, & Kleck, 2007)
6) Gaze directed at the observer (Ivanonva & Allen, 2001), probably with strong indicators of emotional activation in the eye region (Horley, Williams, Gonsalvez, & Gordon, 2003)
7) Attention not focused on the emotion of the facial expressions (Whalen et al., 1998; White, 2002)
8) Low workload, cognitive effort, background arousal, and binding of attentional resources (Dvorak-Bertsch, Curtin, Rubinstein, & Newman, 2007)
9) Related to point 7 and 8, a context should be created that maximizes the figure-ground contrast between external social threat-cues and context (experiment 3)

Considering these conditions, the following critical question may be asked: Given that affective startle modulation emerges under these circumstances, is this relevant for SAD? For a potential answer it may be considered that attentional and interpretation biases in socially anxious individuals to similar cues as used in the reported experiments 1-3 (e.g. angry facial expressions and socially threatening words, see Heinrichs & Hofmann, 2001) have been typically found in highly controlled laboratory settings without a realistic social context eliciting social anxiety. Furthermore, in these tasks the response behavior was ecologically irrelevant (e.g. pressing a button in response to detecting a dot). Similarly, the above list of conditions suggested for expecting affective startle modulation appears highly artificial. This further corroborates the conclusion that biased processing of external social threat-cues and in particular of emotional facial expressions is not a mediator of anxiety when individuals with increased levels of trait social anxiousness enter ecologically valid socially challenging situations.

3.2 Revised Working Model on Mediators of Social Anxiety

An integration of results found in experiments 1-3 with previously published findings suggests some revisions to the working model on mediators of social anxiety presented in section 1.3.8, p. 52. Most importantly, the current results confirm that associations between trait social anxiousness (predictor) and anxiety symptoms (criteria) are mediated by the intensity of self-related negative cognitions in socially challenging situations (context). The revised working model takes the dissociations found across emotional response systems into account: Both increased self-reported anxiety and parasympathetic withdrawal is predicted by the level of trait social anxiousness. This association is mediated by self-related negative cognitions. Future studies should further differentiate the contribution of internal attentional focus vs. cognitive content to mediation of social anxiety. Skin conductance level as an indicator of sympathetic arousal and heart rate as an indicator of general activation increased by situational demand characteristics that are independent from social anxiety. Furthermore, observer ratings of behavior during public speaking, and the self-rated quality of the performance were influenced by individual characteristics independently from social anxiousness but moderated by situational demand characteristics. Finally, positive estimation bias of peak heart rate (i.e. an underestimation as compared to EKG-derived heart rate) during public speaking was reduced in individuals with higher levels of trait social anxiousness. Notably, the increased ratings may rather reflect cognitive bias moderated by the intensity of self-related negative cognitions than increased accuracy of evaluating internal somatic cues, see discussion of experiment 3. Nonetheless, the intensity of noticeable somatic cues may influence their evaluation and interact with cognitive mediation of social anxiety. The uncertain role of biased evaluation of somatic cues should receive specific attention in future studies.

Although the null-findings in experiments 1-3 may not be considered as final evidence, several arguments have been discussed above, suggesting that preferential processing of external social threat-cues is not a mediator of social anxiety, at least in ecologically valid challenging social situations. Therefore, this mediator is removed from the revised model. Startle responding may further be considered on the criterion level of the model. In an arousing social situation such as public speaking, higher levels of trait social anxiousness predicted increased startle amplitudes (experiment 3). Self-related negative cognitions did not interact with this association. Previous findings suggest that similar associations also emerge without a socially threatening situational context. However, a non-social distracting and effortful task disrupted the association between trait social anxiousness and augmented startle amplitude (Cornwell, Johnson, Berardi, & Grillon, 2006). High workload or capacity exhausting attention binding may therefore be strategies to interfere with this association. It remains curious that startle amplitude correlated with self-reported anxiety only when self-related negative cognitions were induced. Increased amygdala activation may indicate increased activity of the defensive motivational system (Lang, Bradley, & Cuthbert), and bottom-up priming of associated fear networks. Notably, cognitive schemata are only susceptible to priming effects when they are activated (Bower, 1981). Therefore, (weak) bottom-up effects may only emerge in anxiety ratings, when semantic networks are activated that overlap sufficiently with the defensive motivational system. Inducing self-related negative cognitions may have led to such a wide-spread activation of overlapping schemata. Finally, increased levels of trait social anxiousness were associated with decreased startle latency when situational challenge was rather mild, when the participants were not focused on an effortful task or when they processed emotional facial expressions (experiment 1). Descriptively, individuals with SAD had even shorter startle latencies to all facial expressions (experiment 2).

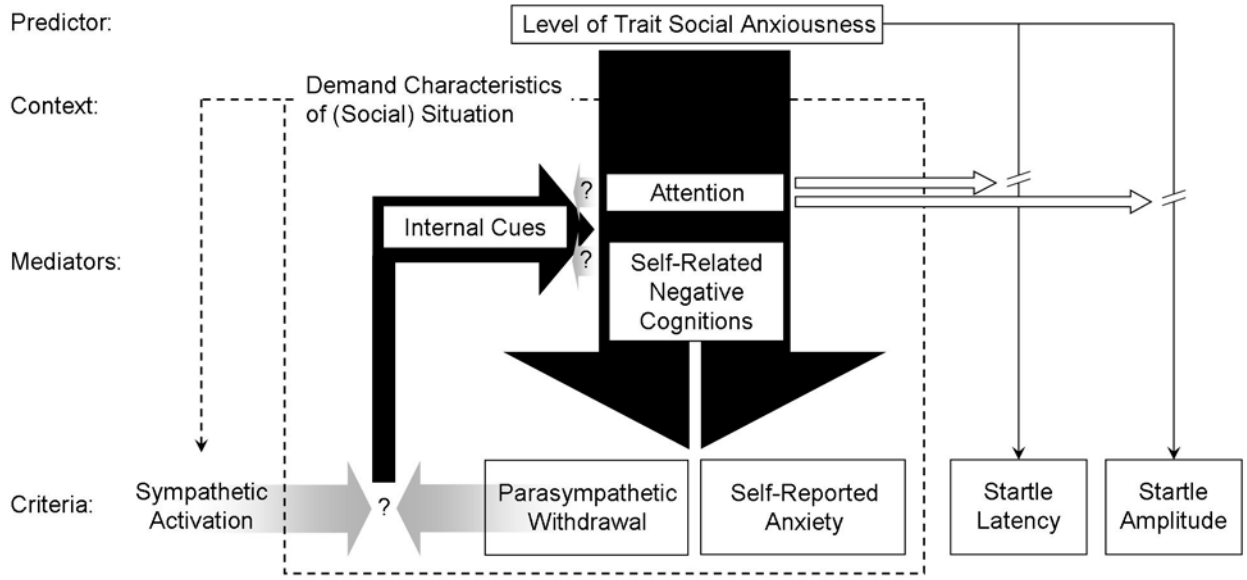

Figure 24: Revised working model on mediators of social anxiety; see text for explanation.

Shorter startle latency has been interpreted as an index for increased motor-responsivity, which is part of the clinical presentation of SAD (e.g. Beidel, Turner, & Dancu, 1985). Variations across task conditions and experiments suggest interactions with situational challenge (experiment 3), emotion regulation (experiment 1), and processing of emotional foreground stimuli (experiment 1). Despite the tentative nature of these post-hoc explanations, it is proposed to examine probable effects of attentional focus on startle responding.

The revised working model on mediators of social anxiety allows empirical testing of more specific hypotheses than the original model. Furthermore, established models may be refined by an integration of implications from this model. Most importantly, dissociations across emotional response domains (Lang, 1993) should be added to traditional models. Furthermore, the roles of information processing biases to external and internal threat-cues may be revised. Finally, cognitive mediation may be added to empirically validated models of social anxiety (e.g. Hofmann, 2007).

Altogether, research based on instances of the working model on mediators of social anxiety may comprise a valuable tool for deriving testable hypotheses and for sorting out and

interpreting complex interactions of predictors, context, potential mediators, and multivariate measures for model criteria.

3.3 Limitations of the Approach

A number of limitations of the approach pursued in this dissertation should be acknowledged. First, the working model on mediators of social anxiety relies substantially on the dimensional perspective on social anxiety disorder (McNeil, 2001). Future research is needed to examine whether qualitative differences between individuals with low vs. high levels of social anxiousness exist. For example, behavioral genetics may reveal such differences (e.g. Arbelle et al., 2003; Brocke et al., 2006; Canli & Lesch, 2007; Lesch et al., 1996).

Considering this point, it may be fatal that linear statistics are the basis for mediation modeling. Examination of characteristics of parameter distributions and appropriate transfer functions for fitting nonlinear data provide adequate solutions for some of the issues related to this approach. Considerable methodic care is required when complex interactions are interpreted. Configuration-based methods (e.g., Lienert, 2008) may be a valuable extension to this linear approach, because complex qualitative associations between variables may be detected. In sum, the hypotheses, which can be tested within a mediation model of social anxiety, are likely too limited for advancing such models to the ultimate goal of developing a functional anatomical model of social anxiety. Finally, statistical mediation does not allow causal interpretation. One solution for this issue is to interpret effects within a given theoretic background. Furthermore, methodic approaches for establishing causal evidence may be used (e.g. Steyer, 1992).

Limitations of the three experiments are mentioned in the respective discussions. In sum, it should be noted that the findings of experiment 1 and 3 are based on student samples. In experiment 2, the sample comprised of individuals with a clinical diagnosis of SAD. But sample size was small. Thus generalization of the results to the general population or to

clinical samples warrants caution. Nonetheless, the findings are in line with the presumed dimensional view on social anxiousness. Therefore, extrapolation of results to more extreme levels of social anxiousness may at least be useful for deriving new hypotheses.

None of the experiments examined effects of external social threat-cues without a socially challenging context. Biased processing of threat-cue may therefore emerge in different situational contexts. Other outcome variables and different methods may reveal different results. For example, assessing affective modulation of approach reflex circuits such as the post-auricular reflex (Benning, Patrick, & Lang, 2004) may reveal dysfunctional information processing to positive stimuli. Similar to depression, it has been suggested that individuals with SAD may rather feature a lack of positive than increased negative emotions when being confronted with social threat. This is an alternative explanation for differential attentional effects found with positive vs. negative facial expressions. Both assessments of startle and post-auricular reflex suffer from the fact that only discrete time points can be assessed namely the moment, in which the startle probe is presented. Continuous response assessment may be useful to overcome this disadvantage. This would allow assessment of dissociations and interactions between central and peripheral emotional reactivity across time.

3.4 Conclusion

The experiments presented in this dissertation provide strong support for the role of *self-related negative cognitions* as a mediator of social anxiety, while preferential processing of *external social threat-cues* appears to play a negligible role, at least in ecologically valid socially challenging situations. Based on these results, a revised version of the working model on mediators of social anxiety has been proposed. This model addresses dissociations of anxiety symptoms across response systems, which have been partially reported in previous publications and were confirmed by the current findings. In particular, the new discovery of a specific association between self-related negative cognitions and decreased HRV-HF suggests

an important role of parasympathetic control during emotional responding to social distress. This finding can explain why previous studies have largely failed in finding physiological correlates of social anxiety. They have largely neglected parasympathetic effects. Furthermore, this finding is in line with current functional-anatomical models on brain-body interaction during emotion regulation. Future studies should examine the interplay of central and peripheral components involved in emotion regulation of socially anxious individuals. To this end, the revised model on mediators of social anxiousness provides a concise framework for empirical research on social anxiety. The model allows straightforward interpretation based on a sound theoretic concept. Ultimately, this may aid the development of an empirically validated functional-anatomical model of social anxiety.

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5 Appendix

5.1 Materials: Experiment 1

5.1.1 Informed Consent Form (German)

Name des Versuchs- Instruktion und Einverständniserklärung

Liebe(r) Untersuchungsteilnehmer/in,

vielen Dank für die Teilnahme an diesem Experiment! Deine Aufgabe wird es sein,
zunächst eine Reihe von Gesichtern auf dem Bildschirm aufmerksam zu betrachten. Zur Erholung gibt es zwischendurch eine kurze Pause. Beginn und Ende der Pause werden deutlich angekündigt. Du kannst in der Pausenzeit somit entspannt abschalten.
Während der Untersuchung messen wir die Augenlidbewegungen. Dazu werden 2 kleine Messfühler unterhalb des Auges und einer am Schlüsselbein angebracht. Es ist ausgeschlossen, dass dadurch Schmerzen oder gar eine Gefährdung entstehen, da lediglich die körpereigenen Ströme gemessen werden. Es wird also kein elektrischer Strom auf den Körper übertragen! Zusätzlich zur Messung der Augenlidbewegung findet während des gesamten Versuchs eine Videoaufzeichnung der Gesichtsmuskelaktivität statt, die der Versuchsleiter im Nebenraum auswertet.
Die Untersuchung wird ungefähr eine Stunde dauern.

Alle Daten werden anonym verarbeitet und absolut vertraulich behandelt.

Während des Versuchs sind in unregelmäßigen Abständen laute Töne zu hören. Falls dein Hörvermögen eingeschränkt ist oder gesundheitliche Probleme im Zusammenhang mit dem Gehör bestehen (z.B. Hörstürze, Tinitus, Trommelfellschädigung, häufige Ohrenschmerzen, etc.), darfst Du **nicht** an dem Versuch teilnehmen.

Die Teilnahme an der Untersuchung ist völlig freiwillig. Du kannst – ohne Angabe von Gründen - die Teilnahme jederzeit abbrechen, ohne dass Dir dadurch irgendwelche Nachteile entstehen.

Falls Du weitere Fragen hast, wende Dich bitte an den/die – Versuchsleiter/in.

Einverständniserklärung

Ich habe die oben aufgeführten Informationen gelesen und verstanden. Ich bin darüber informiert worden, dass ich jederzeit aus der Untersuchung ausscheiden kann, ohne dass mir persönliche Nachteile entstehen.
Ich erkläre mich einverstanden, dass meine Daten zu Forschungszwecken verwendet werden.

Ich erkläre hiermit, dass
- bei mir keine Hörschäden vorliegen
- mein Hörvermögen nicht vermindert ist
- ich nicht an Problemen mit den Ohren / dem Gehör leide

Würzburg, den ..... Unterschrift .....

Unterschrift des/der Versuchsleiters/-in: .....

5.1.2 Instructions for Strategy "Identify" (German)

„In diesem Block sollst du versuchen, Dich mit der gezeigten Person zu identifizieren, indem Du Dich nur auf den gezeigten Gesichtsausdruck konzentrierst und den emotionalen Zustand der Person empfindest. Lass Dich nicht durch andere Gedanken ablenken."

5.1.3 Instructions for Strategy "React" (German)

„In diesem Block sollst Du Dich darauf konzentrieren, welche Emotion Du bei dem gezeigten Gesichtsausdruck empfindest. Also, welche Emotion wird in Dir ausgelöst, wenn du jemanden mit diesem Gesichtsausdruck siehst?"

5.1.4 Scatterplot for Correlation of Startle Latency vs. Trait Social Anxiousness

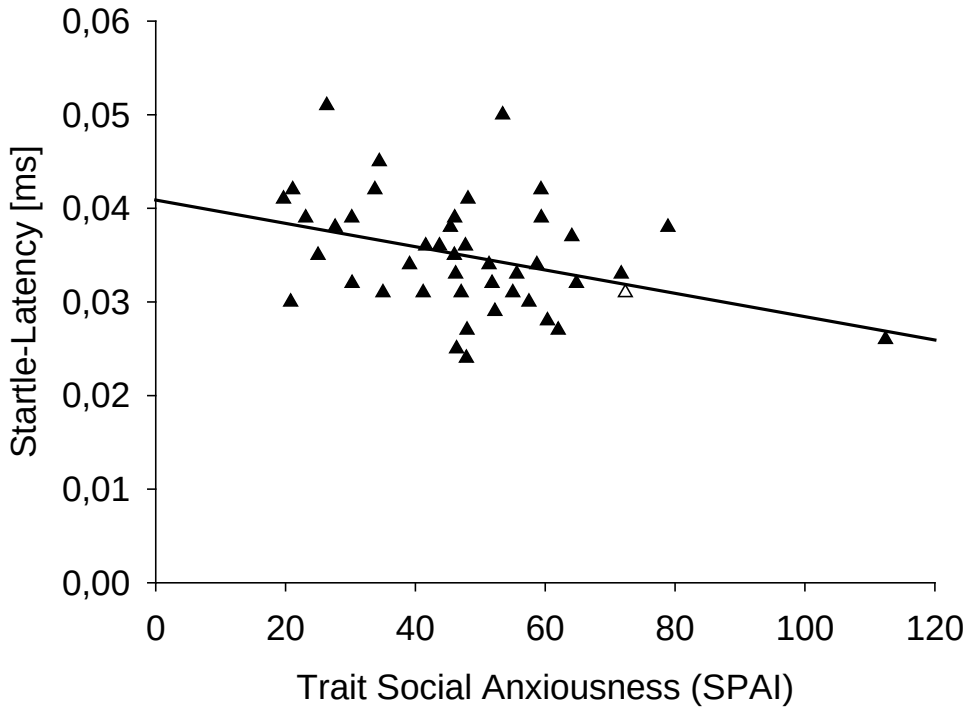

Figure 25: Scatterplot of the relationship between startle latency [ms] and trait social anxiousness as assessed with the SPAI, including linear trend (black line).

5.2 Materials: Experiment 2

5.2.1 Invitation to Participate (German)

Sehr geehrte Patientin, sehr geehrter Patient der Hochschulambulanz!

Wir laden Sie ein, an einer wissenschaftlichen Untersuchung teilzunehmen bei der es darum geht, wie unterschiedliche Gesichtsausdrücke auf verschiedene Menschen wirken.

Ihre Aufgabe wird es dabei lediglich sein, eine Reihe von Gesichtern auf dem Bildschirm aufmerksam zu betrachten. Während der Untersuchung messen wir unter anderem die Augenlidbewegungen. Während des Versuchs sind in unregelmäßigen Abständen laute Töne zu hören. Daher dürfen Sie leider **nicht** an dem Versuch teilnehmen, falls Ihr Hörvermögen eingeschränkt ist oder gesundheitliche Probleme im Zusammenhang mit dem Gehör bestehen (z.B. Hörsturz, Tinnitus, Trommelfellschädigung, häufige Ohrenschmerzen, etc.).

Die Untersuchung wird ungefähr eine Stunde dauern. Alle Daten werden anonym verarbeitet, auch wenn Sie in der Ambulanz behandelt werden.

Der Ansprechpartner für diese Untersuchung ist Frau Diplom-Psychologin Nina Steinhäuser (*Telefon*) und Herr Diplom-Psychologe Stefan Schulz (schulz@psychologie.uni-wuerzburg.de).

5.2.2 Informed Consent Form (German)

INSTITUT FÜR PSYCHOLOGIE DER UNIVERSITÄT WÜRZBURG
Lehrstuhl für Psychologie I
(Biologische Psychologie, Klinische Psychologie und Psychotherapie)
Prof. Dr. Paul Pauli

Versuchstitel: SPA-WS03/04 **Code-Nr.:_____**

Versuchsinformation und Einverständniserklärung

Liebe(r) Untersuchungsteilnehmer/in,

Mit diesem Schreiben möchten wir sicherstellen, dass Sie bereits zu Beginn der Untersuchung eine Vorstellung davon bekommen, wofür Sie sich mit Ihrer Teilnahme entscheiden.

Bei dieser Untersuchung werden Sie jeweils am Beginn und Ende des Versuchs Fragebogen erhalten. Außerdem werden Sie die Aufgabe bekommen, eine Reihe von Gesichtern auf dem Bildschirm aufmerksam zu betrachten. Dieser Teil des Experiments besteht aus zwei Abschnitten. Zur Erholung gibt es zwischendurch eine kurze Pause, in der Sie weitere Fragebogen bekommen. Beginn und Ende der Pause werden deutlich angekündigt, denn die Pause soll auch der Entspannung dienen.

Während der beiden Untersuchungsabschnitte am PC messen wir verschiedene physiologische Reaktionen ihres Körpers. Dazu werden Ihnen zwei kleine Messfühler unterhalb des Auges, zwei weitere an der Stirn, zwei am Handgelenk und einer am Schlüsselbein angebracht. Es ist ausgeschlossen, dass dadurch Schmerzen oder gar eine Gefährdung entstehen, da lediglich die körpereigenen Ströme gemessen werden.

Zusätzlich findet während der Untersuchungsabschnitte am PC eine Videoaufzeichnung Ihrer Gesichtsmuskelaktivität statt, die der Versuchsleiter im Nebenraum auswertet.

Während des Versuchs werden in unbestimmten Abständen laute Töne zu hören sein. Falls Ihr Hörvermögen eingeschränkt sein sollte oder gesundheitliche Probleme im Zusammenhang mit den Ohren bestehen (z.B. Hörstürze, Tinnitus, Trommelfellschädigung, häufige Ohrenschmerzen, etc.), wenden Sie sich bitte jetzt an den Versuchsleiter.

Die Untersuchung dauert insgesamt ungefähr 1,5 Stunden.

Die Teilnahme an der Untersuchung ist völlig freiwillig. Sie können die Teilnahme ohne Angabe von Gründen jederzeit abbrechen, ohne dass Ihnen dadurch irgendwelche Nachteile entstehen. Die Daten dienen ausschließlich Forschungszwecken. Sie werden vertraulich behandelt und ohne Namensnennung unter einem Code abgespeichert Eine Weitergabe der Daten an Dritte erfolgt nicht. Diese Einverständniserklärung wird getrennt von den erhobenen Daten aufbewahrt.

Falls Sie weitere Fragen haben, wenden Sie sich bitte an den/die – Versuchsleiter/in.

Einverständniserklärung

Ich habe die oben aufgeführten Informationen gelesen und verstanden. Ich bin darüber informiert worden, dass ich meine Einwilligung jederzeit widerrufen kann, ohne dass mir persönliche Nachteile daraus entstehen. Ich erkläre mich einverstanden, dass meine Daten anonymisiert zu Forschungszwecken verwendet werden. Ich bin damit einverstanden an der Untersuchung teilzunehmen. Ich erkläre hiermit, dass

- bei mir keine Hörschäden vorliegen ☐ richtig ☐ falsch
- mein Hörvermögen nicht vermindert ist ☐ richtig ☐ falsch
- ich nicht an Problemen mit Ohren / dem Gehör leide ☐ richtig ☐ falsch

Vorname:…….. Name:……

Würzburg, den ..... Unterschrift .....

Unterschrift des/der Versuchsleiters/-in: .....

Vielen Dank für Ihre Teilnahme an diesem Experiment!

5.3 Materials: Experiment 3

5.3.1 Experiment Sign-up Sheet 1

Psychological Department – Experimental Participation

Experiment Number: _____ Numbers of hours of credit: 3.

Experimenter's name:
Dipl.-Psych. Stefan M. Schulz

Experimenter's office and phone number:
Center for Anxiety and Related Disorders (CARD) 648 Beacon St. 4th floor, room 402
phone: 617-353-9610, email: cardstudy@gmx.net

Location of Experiment:
Center for Anxiety and Related Disorders (CARD) 648 Beacon St. 4th floor, room 402

Brief Description of experiment:
The study investigates differences in the processing of emotional stimuli during anticipation of a public speech. Different cognitive strategies are expected to influence behavioral, cognitive, and somatic reactions in anticipation of and during public speaking.

Restrictions: Please check your eligibility before making appointments.

Inclusion criteria:

- You experience considerable distress in anticipation of and during public speaking (e.g. in class).
- 18 years of age or older.
- Normal or corrected vision.
- Signed written informed consent.

Exclusion criteria:

- Smoking or intake of caffeine less than three hours before the experiment.
- Intake of alcohol less than eight hours prior to the experiment.
- Tinnitus or hearing impairment.

Please read this information carefully. In the space below, clearly print your name, course and section number, I.D. number, and phone number or email address next to the day, date, and time of your choice. PLEASE BE SURE TO FILL OUT AN EXPERIMENTAL PARTICIPATION APPOINTMENT CARD, which you must take with you to the experiment and have signed by the experimenter, before you take it to your PS 101 instructor. You will be responsible for appearing on time or notifying the experimenter in advance if you cannot attend.

IF YOU HAVE TO CANCEL, OR RESCHEDULE YOUR APPOINTMENT PLEASE USE "cardstudy@gmx.net"

5.3.2 Experiment Sign-up Sheet 2

Psychological Department – Experimental Participation

Experiment Number: _____ Numbers of hours of credit: 3 .

Experimenter's name:

Dipl.-Psych. Stefan M. Schulz

Experimenter's office and phone number:
Center for Anxiety and Related Disorders (CARD) 648 Beacon St. 4th floor, room 402
phone: 617-353-9610, email: cardstudy@gmx.net

Location of Experiment:
Center for Anxiety and Related Disorders (CARD) 648 Beacon St. 4th floor, room 402

Brief Description of experiment:
The study investigates differences in the processing of emotional stimuli during anticipation of a public speech. Different cognitive strategies are expected to influence behavioral, cognitive, and somatic reactions in anticipation of and during public speaking.

Restrictions: Please check your eligibility before making appointments.

Inclusion criteria:

- You generally feel comfortable speaking before a group
- 18 years of age or older.
- Normal or corrected vision.
- Signed written informed consent.

Exclusion criteria:

- Smoking or intake of caffeine less than three hours before the experiment.
- Intake of alcohol less than eight hours prior to the experiment.
- Tinnitus or hearing impairment.

Please read this information carefully. In the space below, clearly print your name, course and section number, I.D. number, and phone number or email address next to the day, date, and time of your choice. PLEASE BE SURE TO FILL OUT AN EXPERIMENTAL PARTICIPATION APPOINTMENT CARD, which you must take with you to the experiment and have signed by the experimenter, before you take it to your PS 101 instructor. You will be responsible for appearing on time or notifying the experimenter in advance if you cannot attend.

IF YOU HAVE TO CANCEL, OR RESCHEDULE YOUR APPOINTMENT PLEASE USE "cardstudy@gmx.net"

5.3.3 Informed Consent Form

Informed Consent Form

This is the Informed Consent Form for the study entitled: “Anticipatory Processing in Social Anxiety and Startle Modulation by verbal vs. nonverbal Evaluation.” to be conducted at the Center for Anxiety and Related Disorders at Boston University.

Purpose

The goal of this study is to investigate differences in the processing of emotional stimuli in subjects with vs. without social anxiety. You will get the opportunity to practice holding a speech in front of a small audience. Different cognitive strategies during the anticipation of this speech may influence your behavioral, cognitive and somatic reactions.

A comparison of cognitive, behavioral, and somatic measures across participants with vs. without social anxiety, different stimuli, and different situations may help to understand how and why people with social anxiety have specific problems in social performance situations like e.g. a public speech.

Procedures

The maximum overall duration of the experiment is less than three hours.

As part of the experimental procedure, you will be asked to engage in two different tasks prior to holding a short speech in front of a two-person audience. One is a strategy that is considered to lower speech anxiety, while the other one may increase it. A video camera will record your speeches for later analysis of your performance.

In order to measure your somatic reactions during the course of the experiment, the study uses psychophysiological measures. Therefore sensors, specific for each measure, have to be attached to your body in order to record heart rate, galvanic skin response, and the electromyogram of your eye blink and your forehead respectively.

You will also be asked to fill out some questionnaires and answer questions concerning your mental status with a special focus on social anxiety as well as your current feelings and thoughts. However, you are free to omit any answer to specific items or questions during the experimental procedure or in the questionnaires.

Furthermore you will be asked to watch a series of stimuli (words and human faces) on a computer screen and rate certain qualities of these stimuli. During the presentation of the stimuli you will also hear some loud bursts of noise played on headphones that might be slightly uncomfortable or annoying. However they are not dangerous or harming in any way.

If you have questions about any of these measures, or the procedure, please feel free to ask the experimenter about more details.

Risk and Discomforts

There are no risks associated with the interviews or psychological tests other than possible discomfort involved in answering some of the questions.

The speech task is typically anxiety provoking to subjects with social anxiety and the engagement in cognitive processes characteristic for individuals with social anxiety is expected to increase anxiety in anticipation and during the speech.

The potential risks to participants during the psycho physiological assessments and startle test are minimal. There may be some mild discomfort during electrode attachment, and in rare cases, participants have complained that the electrode paste produced transitional erythrodermia. In addition, some subjects responded negatively to the startle stimuli (a 105 dB(A), 50ms burst of white noise) and have described the

sounds as "startling" or "annoying". However, the acoustic parameters proposed for this study pose no risk to participants and identical stimuli have been employed in hundreds of similar studies internationally and may occur for much longer times in everyday life, e.g. at a subway station or at an airport. However, such noise exposure usually is considerably more extended in daily life.

Benefits

Results provide empric data for improvements in the theroretic understanding of information processing in social anxiety. This knowledge can further help to understand how and why people with social anxiety have specific problems in social performance situations like public speaking.

Alternatives

Your alternative is not to participate in this research study or to withdraw from it at any time without penalty or loss of benefits to which you would be otherwise entitled.

Confidentiality

All data with identifying information will be stored in locked files. Data being analyzed will be identified by subject codes and identifying information will be removed. Information from this study may be combined with study data form other projects. This will not affect your privacy or confidentiality of your data. No identifying information will be revealed in the presentation or publication of any result from the project or any combination with data form another project. Any others working on the data will be educated about the importance of strictly respecting subjects' rights to confidentiality.

General information

You may contact Dipl.-Psych. Stefan M. Schulz at CARD, 648 Beacon St., 6th floor, Room 402 during weekdays from 9 a.m. to 5 p.m. (phone: 617-353-9610 email: smschulz@bu.edu), or Dr. Joanne Palfai (Director, Academic Affairs) at the Department of Psychology, 64 Cummington Street, (phone: 617-353-2064 email: jpalfai@bu.edu) for any further questions or concerns regarding the study.

Consent

I understand that my participation in the study is voluntary. Refusal to participate will involve no penalty or loss of benefits to which I would be otherwise entitled, and I may discontinue participation at any time without penalty or loss of benefits to which I would be otherwise entitled. I have received a copy of this consent statement.

_____

Name of Participant (please print)

_____	_____	_____
Signature of Participant	Place	Date
_____	_____	_____
Signature of Person Obtaining Consent	Place	Date

5.3.4 Instructions for Inducing Self-Related Negative Cognitions

STEPS FOR PREPARATION

During the next 10 minutes, we would like you to prepare for the speech by following the steps below.

Please spend a few minutes on each of the steps and make sure you go through all of them in the order in which they are given.

Please make sure you follow all of the steps.

1. Try to think of a particular social situation that you felt did not go well, where you felt uncomfortable or felt that others formed an unfavorable impression of you.
2. Try to imagine how you appeared in that situation. How do you think you looked to others?
3. Now, try to imagine how you are going to appear in the speech you are about to give. Try to think about how you will appear to others. What will they see?
4. Try to analyze in as much detail as possible what could go wrong while you are giving the speech.
5. Try to anticipate the worst thing that could happen while you are giving the speech.
6. Try to think about what you would do if you made a fool of yourself.

If you have finished the task before the end of the 10-min period, please go back to the beginning, and try to think of another social situation that you felt did not go well.

It is very important that you try to stay focused on this task for the whole time!

5.3.5 Tape Recorded Instructions for Relaxation - Part 1

You will now get some instructions that may help you to deepen your state of relaxation.
After a few minutes only the music will continue.

If you want you may close your eyes.

You may think about short statements – like
"Tension flows from my body" or "I can relax at will."
You can find your own statements and repeat them in your mind from time to time.

You are calm and quiet.
Thoughts are passing by
Like some clouds in the sky.
They may come and go.
You feel only your body
Everything else is far away.

Please draw your attention away from stress by focusing on something pleasant, for example lying on the beach and listening to ocean waves.

5.3.6 Tape Recorded Instructions for Relaxation - Part 2

Go through your body now, and seek for tension in specific muscles.
Try to relax these muscles and calm down

Feel for example, how your arms become heavy and warm.
How your legs become heavy and warm.

You are calm and quiet.
Thoughts are passing by
Like some clouds in the sky.
They may come and go.
You feel only your body
Everything else is far away.

Your breathing goes slowly and steady.
Inhale …. And exhale…

"Tension flows from my body"

I am calm and quiet.
Thoughts are passing by
Like some clouds in the sky.
They may come and go.
I feel only my body
Everything else is far away.

5.3.7 Set of Ad Hoc Ratings

Self-reported anxiety: How anxious do you feel at the moment? Please try to specify your current feeling on a scale ranging from 0 to 100. 0 would signify no anxiety and 100 would denote the highest possible anxiety you can imagine.

Confidence to make a good impression: How confident are you that you will be able to make a good impression on the committee? (From 0 = absolutely impossible to 100 = perfectly)

Awareness of bodily symptoms: How aware are you about bodily sensations at the moment? (From 0 = not at all to 100 = extremely)

Evaluation of awareness: How positive or negative is your appraisal of this degree of awareness (from 1 = extremely positive to 6 = extremely negative)

Thinking about speech during task: Estimate how much of the past 10 minutes you spent thinking about your future speech. Type in your estimation from 0 to 100 % of the time.

Time being focused on task: How long during the 10-minute preparation period did you focus on your task? Please consider that it is much more important for the validity of this research, to provide a honest answer than trying to please the experimenter. Type in your estimation from 0 to 100 % of the time.

Thinking about a job-situation during the task: Did you think about a job interview or similar situation during the preparation period? Estimate how much time during the preparation period you thought about it. Type in your estimation in percent of the complete time (from 0 minutes = 0 to 10 minutes = 100):

Peak anxiety during Speaking: What was your peak anxiety during the speech? (From 0 = no anxiety to 100 = strongest feeling of anxiety that you can imagine):

Impression made on the committee: How good an impression do you think you made on the committee? From 0 = extremely poor to 100 = outstanding)

Estimation of maximum heart rate during speaking: Estimate your highest heart rate during the speech task. (A typical resting heart rate is between 60 and 80 beats per minute.) Type in your estimation in beats per minute.

5.3.8 Ad Hoc Ratings Obtained by the Experimenter

SPBU Date: _____ Time: _____ Subject-Nr.:_____

Ad Hoc Rating

1st Set: *apply after obtaining informed consent*

Rating

1. How anxious do you feel at the moment?_____
(From 0 = no anxiety to 100 = strongest feeling of anxiety that you can imagine)

2. How confident are you that you will be able to make a good impression on the committee?_____
(From 0 = absolutely impossible to 100 = perfectly)

3. How aware are you about bodily sensations at the moment?_____
(From 0 = not at all to 100 = extremely)

4. How positive or negative is your appraisal of this degree of awareness?_____
(from 1 = extremely positive to 6 = extremely negative)

2nd Set: *obtaining after 3 minute speech preparation (1st SPEECH)*

Rating

1. How anxious do you feel at the moment?_____
(From 0 = no anxiety to 100 = strongest feeling of anxiety that you can imagine)

2. How confident are you that you will be able to make a good impression on the committee?_____
(From 0 = absolutely impossible to 100 = perfectly)

3. How aware are you about bodily sensations at the moment?_____
(From 0 = not at all to 100 = extremely)

4. How positive or negative is your appraisal of this degree of awareness?_____
(from 1 = extremely positive to 6 = extremely negative)

3rd Set: *obtaining after 3 minute speech preparation (2nd SPEECH)*

Rating

1. How anxious do you feel at the moment?_____
(From 0 = no anxiety to 100 = strongest feeling of anxiety that you can imagine)

2. How confident are you that you will be able to make a good impression on the committee?_____
(From 0 = absolutely impossible to 100 = perfectly)

3. How aware are you about bodily sensations at the moment?_____
(From 0 = not at all to 100 = extremely)

4. How positive or negative is your appraisal of this degree of awareness?_____
(from 1 = extremely positive to 6 = extremely negative)

5.3.9 Stimuli

5.3.9.1 Picture Stimuli

Figure 26: Exemplary angry facial expression with cue for example under the left alar wing of the nose (In the experiment, colored versions of the pictures were used in experiment).

Figure 27: Exemplary fearful facial expression with cue for example under the right eye (In the experiment, colored versions of the pictures were used in experiment).

Figure 28: Exemplary happy facial expression with cue for example on the right check (In the experiment, colored versions of the pictures were used in experiment).

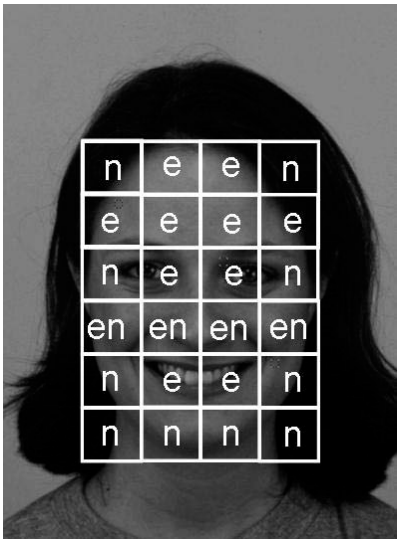

Figure 29: Exemplary facial expressions with grid-overly used for random allocation of cues, counterbalanced between facial areas more (e) less (en), or not relevant (n) for emotion detection.

Figure 30: Exemplary house stimulus with cues for example on the upper and lower ends of the left middle edge of the building (In the experiment, colored versions of the pictures were used in experiment).

5.3.9.2 Word Stimuli

15 socially threatening and 15 generally threatening emotional words were selected from MacLeod, Mathews, and Tata (1986) 15 non-threatening words obtained from a study by Asmundson and Stein (1994b) were used as stimuli for the fear-potentiated startle trials, see Table 19.

Non-threatening words	General threat words	Social threat words
Mild	Injury	Criticized
Normal	Ambulance	Embarrassed
Friendly	Emergency	Failure
Quiet	Disease	Stupid
Honorable	Cancer	Foolish
Fair	Fatal	Inferior
Gentle	Mutilated	Indecisive
Sensible	Coffin	Lonely
Alert	Death	Hated
Respectable	Paralyzed	Humiliated
Observant	Coronary	Incompetent
Cautious	Harm	Worthless
Typical	Violence	Ridiculed
Kind	Fracture	Insecure
Modern	Corpse	Ashamed

Table 19: Non-threatening, general and social threat words used as stimuli in experiment 3.

The threatening and non-threatening words have been used in the past to show consistent attentional differences between patients with panic disorder, social anxiety disorder and in healthy controls (Asmundson & Stein, 1994b; Beck, Stanley, Averill, Baldwin, & Deagle, 1992; Hope, Rapee, Heimberg, & Dombeck, 1990; MacLeod, Mathews, & Tata, 1986). In addition, the physical threat, social threat and non-threatening word sets were matched for length and frequency of use in the English language (Kucera & Francis, 1967).

5.3.10 Anchored Ratings of Stimulus Properties

5.3.10.1 Ratings of Picture Stimuli

Now you will again see the pictures from the experiment.
We ask you to rate the impression they made on you.
Please note that the same keys are used for two different scales: One ranges from 1 to 9, while the other ranges from –4 to +4.
Please look at the keyboard now and make yourself familiar with the key assignment.

The first question will always be, how “happy vs. unhappy” you felt while watching a picture.
The happy extreme could also be described as feeling pleased, satisfied, contented, or hopeful. If you felt like this, please press the key marked with –4.
The other extreme could be described as extremely unhappy, or annoyed, unsatisfied, melancholic, despaired, or bored. In case you felt like that, press the button marked with +4
If you felt completely neutral, neither happy nor sad, press the center key marked with 0.
The other keys permit you to make more finely graded ratings of how you feel in reaction to the pictures.

-4	-3	-2	-1	0	+1	+2	+3	+4
extremely unhappy				neutral				extremely happy

Next we ask you to rate the stimulus on the excited vs. calm dimension.
At one extreme of the scale you felt stimulated, excited, frenzied, jittery, wide-awake, aroused. If you felt completely aroused, press the key marked with 9.
At the other end of the scale, you felt completely relaxed, calm, sluggish, dull, sleepy, unaroused. If you felt completely calm, press the left key marked with 1.
The other keys again permit you to make more finely graded ratings of how you feel in reaction to the pictures.

1	2	3	4	5	6	7	8	9
extremely calm								extremely excited

The third question is about the impression of complexity you get.
If you think a picture was extremely complex, detailed, multifaceted, intricate, embroidered, rich press the key marked with 9.
The other end of the scale could be described as extremely simple, sketchy, one-sided, boring, artless, inornate. If you get this impression of the picture, press the left key marked with 1.
Once again, the other keys permit you to make more finely graded ratings of how you feel in reaction to the pictures.

1	2	3	4	5	6	7	8	9
extremely simple								extremely complex

If you have any questions about how to rate the pictures please ask the experimenter now.

How do you feel when you watch this picture?

-4	-3	-2	-1	0	+1	+2	+3	+4
extremely unhappy				neutral				extremely happy

How do you feel when you watch this picture?

1	2	3	4	5	6	7	8	9
extremely calm								extremely excited

What rating of complexity would you give this picture?

1	2	3	4	5	6	7	8	9
extremely simple								extremely complex

5.3.10.2 Ratings of Word Stimuli

Now you will again see the words from the experiment.
We ask you to rate the impression they made on you.
Please note that the same keys are used for two different scales: One ranges from 1 to 9, while the other ranges from –4 to +4.
Please look at the keyboard now and make yourself familiar with the key assignment.
While the first two questions are very similar to the questions about the pictures, questions 3 and 4 are different. Please read these instructions carefully.

The first question will always be, how “happy vs. unhappy” you felt while reading the word.
The happy extreme could also be described as feeling pleased, satisfied, contented, or hopeful. If you felt like this, please press the key marked with –4.
The other extreme could be described as extremely unhappy, or annoyed, unsatisfied, melancholic, despaired, or bored. In case you felt like that, press the button marked with +4

If you felt completely neutral, neither happy nor sad, press the center key marked with 0.
The other keys permit you to make more finely graded ratings of how you feel in reaction to the pictures.

-4	-3	-2	-1	0	+1	+2	+3	+4
extremely unhappy				neutral				extremely happy

Next we ask you to rate the stimulus on the excited vs. calm dimension.
At one extreme of the scale you felt stimulated, excited, frenzied, jittery, wide-awake, aroused. If you felt completely aroused, press the key marked with 9.
At the other end of the scale, you felt completely relaxed, calm, sluggish, dull, sleepy, unaroused. If you felt completely calm, press the left key marked with 1.
The other keys again permit you to make more finely graded ratings of how you feel in reaction to the words.

1	2	3	4	5	6	7	8	9
extremely calm								extremely excited

The third question is about the impression of complexity you get.
If you think a word describes something extremely complex, detailed, multifaceted, intricate, embroidered, rich press the key marked with 9.
The other end of the scale could be described as extremely simple, sketchy, one-sided, boring, artless, inornate. If you get this impression of the word, press the left key marked with 1.
Once again, the other keys permit you to make more finely graded ratings of how you feel in reaction to the words.

1	2	3	4	5	6	7	8	9
extremely simple								extremely complex

How do you feel when you read this word?

-4	-3	-2	-1	0	+1	+2	+3	+4
extremely unhappy				neutral				extremely happy

How do you feel when you read this word?

1	2	3	4	5	6	7	8	9
extremely calm								extremely excited

What rating of complexity would you give the contents of the word?

1	2	3	4	5	6	7	8	9
extremely simple								extremely complex

5.3.11 Instructions for Audience Behavior during Public Speaking

1. **Ask subject to remove headphones**.
2. **Introduce** yourself and the confederate:

Experimenter provides a situational framing: *"Hi. We are here to simulate the job interview situation now (again). We will pose as the committee that will observe and evaluate your speech. <last name> <first name> <last name> will (again) take the role of the chairman."*
Chair: *Hello <Mr. / Mrs.> <last name>. My name is <first name> <last name>, and this is <first name> <last name>. First of all there is some paperwork to do. Please fill out these standardized questionnaires now.*

3. Assistant hands over clipboard with **STAI-State, PANAS and SSAPS**.
4. After subject has finished the questionnaires:

Chair: *"Now, please fill out this speech preparation sheet. You have one minute for each of the 3 sections, and we will tell you each time, when you should switch to the next section.*

5. After 3 minutes, the chair asks the subject to **put the notes aside** and answer one set of **ad hoc ratings**.

I will start the video camera now to record your speech for later analysis.

6. Chair **turns on Video-Camera**.

Chair: *"Please take a moment to focus on your job application now. In your speech you should tell us about your personal characteristics and why you would be the best candidate for this position.*
Try to speak the full 10 minutes if possible. However, after a minimum of 3 minutes you may end the speech by holding up the STOP-sign on the table next to you if you experience significant distress.
You may begin as soon as you feel ready."

Instructions for the committee members:

- During the first 3 minutes, remain silent until the subject stops talking for more than 20 seconds. Then the chair alerts the subject to the remaining time, as with the phrase "You still have time, please continue…."
- Should the subject remain silent for another 10 seconds, the chair asks questions until the end of the 3-minute period.
- The phrasing of these questions is left to the chair's discretion; it may also be solely oriented on the subject's previous statements. Typical questions in this context are: *Why do you think that you are the best applicant for this position? What other experiences have you had in this area? What about your studies identifies a special aptitude and motivation for this position? Where else did you apply? Why? What would you do, if your application here would not succeed?*
- **Stop asking questions after 3 minutes.**
- 10 minutes later, or after the subjects has put up the stop sign the committee thanks the subject for the speech

Nonverbal behaviour:

- Generally be friendly, polite and comforting.
- Keep interaction with the subject on a minimum.
- Do not reinforce interaction (e.g. questions, jokes, etc. during the experiment), but answer questions straightforward.
- Explain every step you do, especially during the attachment of the electrodes.

During the speech:

- Be attentive and interested.
- Make eye contact, but try to not interact. If you realize, the subject asks for nonverbal feedback, look away.
- Do not cross legs, fumble, tap, play around with pen etc.
- Do not frown.
- If the subjects asks something during the speech. Give a short answer and ask the subject to continue the speech. If the subject asks if he/she can stop the speech, say: “Please continue as long as you have something to say”.
- Try to find 2 or 3 comfortable but reputable seating positions that you can alternate.
- Do not lean forward when the speech is interesting and lean backward when it is boring.
- Occasional reinforcement e.g. by nodding are ok, but try to keep it low level.

7. **Turn off the video-camera** and **cover it** with the grey cloth
8. The experimenter attaches the **headphones** to the subject’s ear and asks if they **seal** the ear
9. The experimenter **asks the subject to press the ENTER-button** to go on with the procedure.
10. The **committee leaves the room**.

5.3.12 Rating System for Behavioral Assessment of Public Speaking

Committee-Rating Date_____ Time _____ Subject-No. ___ Rater: _____

Time: 0 – 1st minute – Rating 1:	**Speech A**	**Speech B**
How strong do you perceive the subject's bodily and nonverbal symptoms of anxiety? (From 0 = not at all to 100 = extremely)		
How would you rate the contents and rhetorical aspects of the speech? (From 0 = horrible to 100 = perfect)		
How would you rate the overall anxiety of the subject as a speaker? (From 0 = no anxiety [subject seems perfectly comfortable] to 100 = extremely severe)		

Time: 2nd – 3rd minute – Rating 2:	**Speech A**	**Speech B**
How strong do you perceive the subject's bodily and nonverbal symptoms of anxiety? (From 0 = not at all to 100 = extremely)		
How would you rate the contents and rhetorical aspects of the speech? (From 0 = horrible to 100 = perfect)		
How would you rate the overall anxiety of the subject as a speaker? (From 0 = no anxiety [subject seems perfectly comfortable] to 100 = extremely severe)		

Time: 4th – 5th minute – Rating 3:	**Speech A**	**Speech B**
How strong do you perceive the subject's bodily and nonverbal symptoms of anxiety? (From 0 = not at all to 100 = extremely)		
How would you rate the contents and rhetorical aspects of the speech? (From 0 = horrible to 100 = perfect)		
How would you rate the overall anxiety of the subject as a speaker? (From 0 = no anxiety [subject seems perfectly comfortable] to 100 = extremely severe)		

Overall impression	**Speech A**	**Speech B**
How would you rate the subject as a social performer in general? (From 0 = horrible to 100 = perfect)		

5.4 Scatterplots of Correlations

5.4.1 Self-Rated Anxiety vs. Trait Social Anxiousness

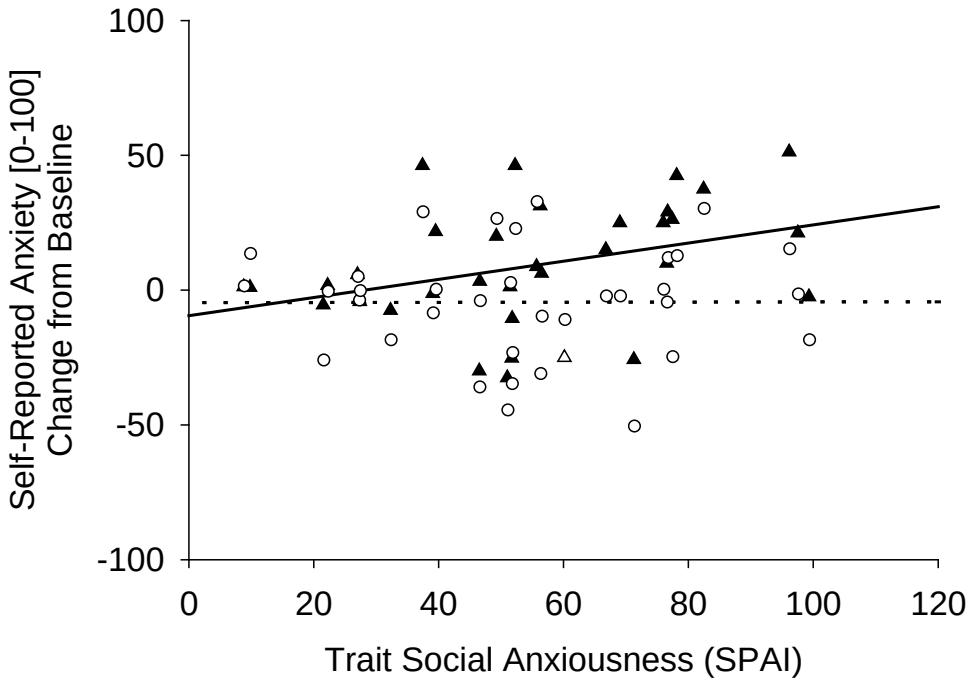

Figure 31: Scatterplot of the relationship between self-reported anxiety [rating: 0-100] and trait social anxiousness as assessed with the SPAI, showing change scores from baseline to negative (black triangles, black line marks linear trend) vs. relaxed anticipation (white circles, dashed line marks linear trend) of public speaking.

5.4.2 HRV-HF vs. Trait Social Anxiousness

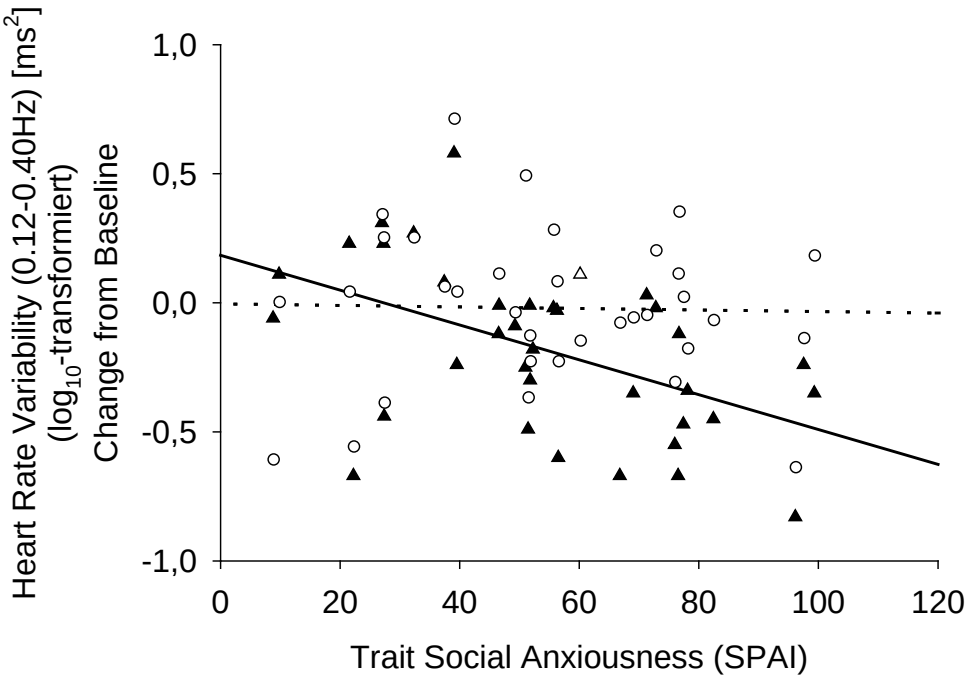

Figure 32: Scatterplot of the relationship between power spectral density of heart-rate variability in the high frequency spectrum [ms^2] and trait social anxiousness as assessed with the SPAI, showing change scores from baseline to negative (black triangles, black line marks linear trend) vs. relaxed anticipation (white circles, dashed line mark linear trend) of public speaking.

5.4.3 Startle Amplitude vs. Trait Social Anxiousness

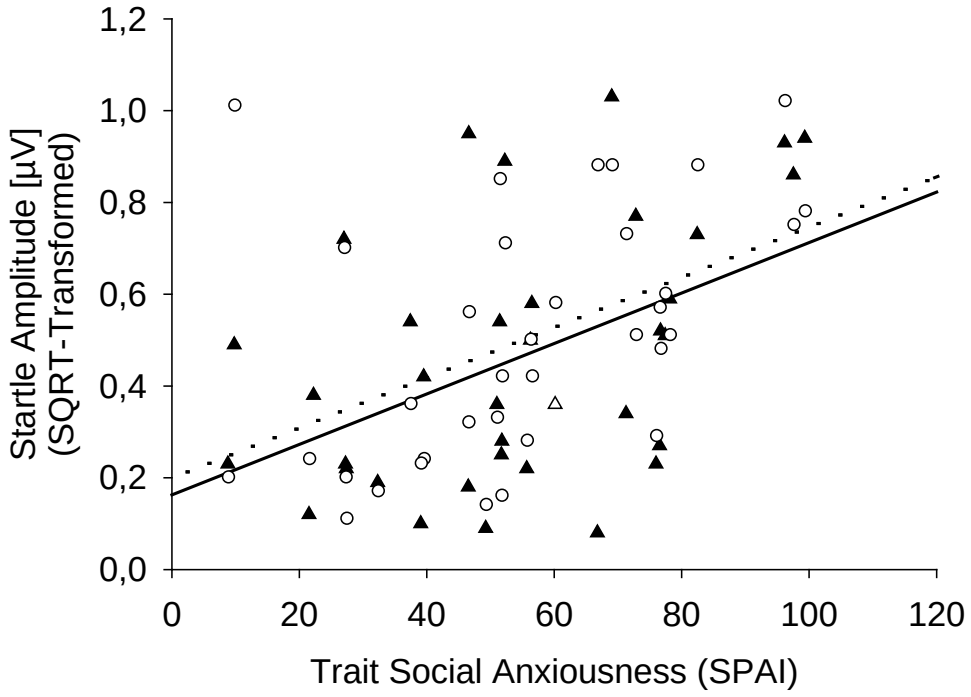

Figure 33: Scatterplot of the relationship between startle amplitude [µV] and trait social anxiousness during negative (black triangles, black line marks linear trend) vs. relaxed anticipation (white circles, dashed line mark linear trend) of public speaking and trait social anxiousness as assessed with the SPAI.

5.5 Lebenslauf (CV)

Persönliche Angaben

Name:	Stefan Manuel Schulz
Anschrift:	Semmelstraße 16, 97070 Würzburg
Geburtsdatum:	17.03.1974
Geburtsort:	Augsburg
Familienstand:	verheiratet
Staatsangehörigkeit:	deutsch

Abschlüsse

09.07.1993	Abitur
13.05.1997	Diplom-Vorprüfung für Psychologen
03.01.2003	Diplom-Prüfung für Psychologen

Bildungsweg und wissenschaftlicher Werdegang

1980 – 1984	Grundschule: Parkschule Stadtbergen
1984 – 1993	Gymnasium bei St. Stephan, Augsburg
01.04.1995 – 03.01.2003	Studium an der Bayerischen Julius-Maximilians-Universität Würzburg, Hauptfach Psychologie. Diplomarbeit: Kongruenzeffekte zwischen motorischen Reaktionen und die Wirkung emotionaler Information im Spannungsfeld von Annäherung und Vermeidung
01.02.2003 - 31.05.2004	Nebenberuflicher Wissenschaftlicher Mitarbeiter am Lehrstuhl für Psychologie I, Universität Würzburg
15.12.2003 – 31.05.2006	Promotionsstipendiat der Gottlieb Daimler- und Karl Benz-Stiftung, Ladenburg
10.06.2004 – 24.05.2006	J-1 Research Scholar bei Prof. Dr. Stefan G. Hofmann am Center for Anxiety and Related Disordes (CARD), Dept. of Psychology, Boston University, Boston, USA
01.08.2006 – 30.09.2006	Nebenberuflicher Wissenschaftlicher Mitarbeiter am Lehrstuhl für Psychologie I, Universität Würzburg
01.10.2006 – 28.02.2007	Wissenschaftlicher Mitarbeiter im Projekt „Ein internetbasiertes Programm zur Prävention von psychischen Belastungen bei Patienten mit implantierbarem Kardioverter-Defibrillator (ICD)“
Seit 01.03.2007	Nebenberuflicher Wissenschaftlicher Mitarbeiter am Lehrstuhl für Psychologie I, Universität Würzburg. Inhaltliche Betreuung der interdisziplinären Research Training Group 1253/1: Processing of affective stimuli: from the molecular basis to the emotional experience

Ersatzdienst

1.10.1993 – 28.02.1995	Ulrichsheim, Behindertenwohnheim der Ulrichswerkstätten, Augsburg

Wissenschaftliche Auszeichnungen

Wintersemester 1997/98	Erster Preis im Internetseminar „Werbepsychologie“ für die Konzeption eines Werbespots, ausgestrahlt im Februar 1998, Institut für Psychologie Lehrstuhl für Kognitive Ergonomie / Ingenieurpsychologie, Humboldt Universität Berlin
22.09.2003	Förderpreis der Vereinigten Stipendien- und Preisstiftung der Universität Würzburg
12.02.2008	Stipendium der Deutschen Gesellschaft für Psychophysiologie und ihren Anwendungen (DGPA) zur Teilnahme an der Spring School 2008 „Biopsychology of Emotions“, Kloster Seeon, 27.-30. März, 2008
August 2008	Förderbeihilfe der Jubiläumsstiftung zum 400-jährigen Bestehen der Universität Würzburg für einen Forschungsaufenthalt am Center for Anxiety and Related Disordes (CARD), Dept. of Psychology, Boston University, Boston, USA
August 2008	Student Travel Award der Society for Psychophysiological Research (SPR) zur Teilnahme am 48th Annual Meeting der SPR, Austin, Texas, USA, 1.-5.10.2008

Organisation wissenschaftlicher Konferenzen

29.09.2009 – 10.02.2009	Summerschool der RTG 1253/1, Benedictushöhe, Retzbach
08.03.2009 – 11.03.2009	Springschool der RTG 1253/1 - Cognition and Emotions - Forced Choice between Siamese Twins?, Kloster Bronnbach
19.10.2008 – 22.10.2008	Summerschool der RTG 1253/1 - Biopsychology of Emotions, Kloster Plankstetten
10.04.2008 – 13.04.2008	5th Würzburg Brain and Behaviour Days - Comparative Research on Emotion Processing - A joint venture of RTG 1253/1: Emotions and GRK 1156: Synaptic & behavioral plasticity, Kloster Bronnbach
06.10.2007 – 09.10.2007	Summerschool der RTG 1253/1 - Methods of Affective Neuroscience, Kloster Bronnbach
27.04.2007 – 29.04.2007	4th Würzburg Brain and Behaviour Days, Schloß Zeilitzheim

Lehre

WS 2003/04	Experimentelles Praktikum zur Klinischen Psychologie - Hauptstudium - Universität Würzburg – Unter Leitung von Prof. Dr. Georg W. Alpers
SS 2004	Praktikum Neuropsychologie - Hauptstudium - Universität Würzburg – Unter Leitung von Prof. Dr. Georg W. Alpers
Sommer 2005	Einführung und Training zu psychophysiologischen Messverfahren - Graduate Level, Boston University
WS 2007	Blockveranstaltung: Grundlagenseminar Kommunikation, ZTW – Co-Trainer, Leitung: Dr. Amina Özelsel
SS 2007	Kolloquium für Doktoranden der RTG 1253/1 "Verarbeitung emotional relevanter Reize: Von den molekularen Grundlagen zur Empfindung" - Hauptstudium - Universität Würzburg - gemeinsam mit Prof. Dr. Paul Pauli Blockveranstaltung: 1st Interdisciplinary Exchange Day – iNED – GK-Emotions

	Blockveranstaltung: Grundlagenseminar Rhetorik, ZTW – Co-Trainer, Leitung: Dr. Katrin Schimmel
WS 2007/08	Kolloquium für Doktoranden der RTG 1253/1 "Verarbeitung emotional relevanter Reize: Von den molekularen Grundlagen zur Empfindung" - Hauptstudium - Universität Würzburg - gemeinsam mit Prof. Dr. Paul Pauli Tutorium: Wissenschaftliche Präsentation - Hauptstudium - Universität Würzburg
SS 2008	Kolloquium für Doktoranden der RTG 1253/1 "Verarbeitung emotional relevanter Reize: Von den molekularen Grundlagen zur Empfindung" - Hauptstudium - Universität Würzburg - gemeinsam mit Prof. Paul Pauli Seminar Translationale Klinische Psychologie - Hauptstudium - Universität Würzburg
WS 2008/09	Seminar für Doktoranden und assoziierte Mitglieder des GRK 1253/1 "Emotions" und des Promotionskollegs "Biopsychologie" der Universitäten Würzburg und Bamberg - gemeinsam mit Prof. Dr. Paul Pauli
SS 2009	Seminar für Doktoranden und assoziierte Mitglieder des GRK 1253/1 "Emotions" und des Promotionskollegs "Biopsychologie" der Universitäten Würzburg und Bamberg

5.6 Publikationsliste (Publications)

5.6.1 Buchkapitel (Book Chapters)

Schulz, S. M., Meuret, A. E., Loh, R., & Hofmann, S. G. (2007), *Social Phobia*. In M. H. Herson and J. C. Thomas (Eds.), Comprehensive Handbook of Interviewing, Volume I – Interviewing Adults (pp. 223-237). Sage Publications, Thousand Oaks, California.

5.6.2 Veröffentlichungen in Fachzeitschriften (Papers in Peer-Reviewed Journals)

Hofmann, S. G., Spira, J. L., **Schulz, S. M.**, Heering, S., Murphy, J., Muench, F., & Bufka, L. F. (submitted). Autonomic Arousal in Generalized Anxiety Disorder with and without Depression. International Journal of Psychophysiology.

Crössmann, A.*, **Schulz, S. M.***, Kühlkamp, V., Ritter, O., Neuser, H., Schumacher, B., Bauer, W., Pauli, P. (submitted). A Randomized Controlled Trial on Secondary Prevention of Anxiety and Distress in a German Sample of Patients with an Implantable Cardioverter Defibrillator (ICD). Psychosomatic Medicine. [* equal author contribution]

Schulz, S. M., Ayala, E., Dahme, B., & Ritz, T. (2009). A MATLAB toolbox for correcting within-individual effects of respiration rate and tidal volume on respiratory sinus arrhythmia during variable breathing. *Behavior Research Methods*, 41 (4), 1121-1126.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2008), Negative Self-Focused Cognitions Mediate the Effect of Trait Social Anxiety on State Anxiety. *Behaviour Research and Therapy*, 46, 438-449.

Hofmann, S. G., **Schulz, S. M.**, Meuret, A. E., Moscovitch, D. A., & Suvak, M. (2006), Sudden Gains During Therapy of Social Phobia. *Journal of Consulting and Clinical Psychology*, 74 (4), 687-697.

Neumann, R., Hess, M., **Schulz, S. M.**, & Alpers, G. W. (2005). Automatic behavioral responses to valence: Evidence that facial action is facilitated by evaluative processing. *Cognition and Emotion*, 19, 499-513.

5.6.3 Konferenzbeiträge (Conference Proceedings)

Schulz, S. M., Murphy, J., Spira, J. L., Gevirtz, R., Heering, S., Muench, F., Pauli, P., Alpers, G. W., & Hofmann, S. G. (2009). Augmenting CBT for GAD with biofeedback vs. PMR yields similar effects. To appear in the proceedings of the *49th Annual Meeting of the Society for Psychophysiological Research (SPR*), October 21-24, Berlin.

Neumann, R., **Schulz, S. M.**, Alpers, G. W., Lozo, L. (2009, September) Automatische mimische Reaktionen auf subliminal präsentierte Emotionsausdrücke: Imitation oder Evaluation? To appear in the proceedings of the *12. Tagung der Fachgruppe Sozialpsychologie*, Luxemburg.

Schulz, S. M., Murphy, J., Spira, J. L., Gevirtz, R., Heering, S., Muench, F., Alpers, G. W., Pauli, P., & Hofmann, S. G. (2009). Biofeedbacktraining respiratorischer Sinusarrhythmie zur Unterstützung kognitiver Verhaltenstherapie (CBT) bei generalisierter Angststörung. Poster presented at the *35. Arbeitstagung Psychophysiologie und Methodik*, Leipzig.

Schulz, S. M., Murphy, J., Spira, J. L., Heering, S., Muench, F., Gevirtz, R., Alpers, G. W., Pauli, P., & Hofmann, S. G. (2009). Kognitive Verhaltenstherapie für generalisierter Angststörung wirkt unabhängig vom Entspannungstraining: PMR vs. Biofeedbackgestützte

Atementspannung. Poster presented at the *27. Tagung für Klinische Psychologie und Psychotherapie der Fachgruppe Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie (DGPs)*, Zürich.

Webb-Murphy, J., Spira, J., Gevirtz, R., Hofmann, S., **Schulz, S. M.** & Muench, F. (2009). Comparison of relaxation techniques for group cognitive behavioral therapy for generalized anxiety disorder. Poster session presented at the *Annual meeting of the Association for Applied Psychophysiology and Biofeedback*, Albuquerque, NM.

Schulz, S. M., & Alpers, G. W. (2009). Reaktionszeit als Momentaufnahme komplexer Prozesse... - sind kontinuierliche Maße überlegen? In A. B. Eder, K. Rothermund, S. R. Schweinberger, M. C. Steffens, H. Wiese (Eds.), *Tagungsprogramm und Abstracts der 51. Tagung experimentell arbeitender Psychologinnen*, (p. 44), Lengerich: Pabst.

Hofmann, S. G., Murphy, J., Spira, J. L., **Schulz, S. M.**, Heering, S., & Muench, F. (2008, November). Augmentation of CBT for generalized anxiety disorder with biofeedback of cardiac vagal tone. In A. E. Meuret (Chair), Novel biobehavioral and technology-supported treatment approaches in anxiety disorders. Paper presented at the *42nd annual convention of the Association for Behavioral and Cognitive Therapies.* Orlando, FL.

Schulz, S. M., Alpers G. W., Gerlach A. L., Dahme B., & Ritz T., (2008), RsaToolbox - A Matlab toolbox for controlling individual effects of breathing frequency and volume on respiratory sinus arrhythmia. *Psychophysiology*, 45, S43.

Schulz, S. M., Gerdes B. M., Alpers, G. W. (2008). Positive Faces, Words, and Scenes facilitate approach. Poster presented at the *XXIX International Congress of Psychology ICP 2008*, Berlin.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2008). Mediators of Social Anxiety: Negative Self-Focused Cognition vs. Threat-Cue Vulnerability. Poster presented at the *2008 DGPA Spring School "Biopsychology of Emotions"*, Kloster Seeon.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2008). Social Anxiety and the Mediating Power of Negative Thought. In C. S. Hermann, & B. A. Sabel (Eds.), *Tagungsband Psychologie und Gehirn 2008 - Magdeburg* (p. 46), GCC, Calbe.

Schulz, S. M., Alpers, G. W., Pauli, P. (2008). How the internet may bridge a supply gap for patients with implantable cardioverter defibrillators (ICD). In P. Warschburger, W. Ihle, G. Esser, Seelisch gesund von Anfang an, Programm und Abstracts des *26. Symposiums der Fachgruppe Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie* (p. 52), 1. - 3. Mai 2008 in Potsdam.

Schulz, S. M., Baumeister, J., Alpers G. W., Crössmann., A., Neuser, H., Puppe, F., & Pauli, P. (2007). An internet-based intervention to reduce cardiac fear in patients with implantable cardioverter defibrillator. *German Journal of Psychiatry* 11, S14.

Schulz, S. M., Baumeister, J., Crössmann., A., Alpers, G. W., Neuser, H., Puppe, F., & Pauli, P.(2007). www.icd-forum.de – Ein internetbasiertes Programm für Patienten mit implantiertem Cardioverter Defibrillator. In H. Eschenbeck, U. Heim-Dreger, & C. Kohlmann (Eds.), *Gmünder Hochschulreihe Nr. 29* (p. 127), Pädagogische Hochschule Schwäbisch Gmünd.

Schulz, S. M., & Alpers, G. W. (2007). EMGpeakfind - A MATLAB-toolbox for scoring startle eye-blink, and other EMG data. In E. Wascher, M. Falkenstein, G. Rinkenauer, & M. Grosjean (Eds.), *Psychologie und Gehirn 2007* (p. 117). Available at: http://hdl.handle.net/2003/24421

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2007). Negative Kognitionen als Mediator sozialer Angst. Poster presented at the *5. Workshopkongress für Klinische Psychologie und Psychotherapie, 25. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der Deutschen Gesellschaft für Psychologie (DGPs)*, Tübingen.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2007). Mediatoren subjektiver und physiologischer Komponenten von sozialer Angst in einer Bewertungssituation. In K. F. Wender, S. Mecklenbräuker, G. D. Rey, & T. Wehr (Eds.), *Beiträge zur 49 Tagung experimentell arbeitender Psychologen* (p. 114), Lengerich: Pabst.

Conzelmann, A., Weyers, P., Mucha, R., Gerdes, A., Jacob C., Romanos M., Bähne C., **Schulz, S. M.**, Lesch K.-P., Warnke A., Alpers G. W., Pauli, P. (2006). Emotionale Defizite bei ADHS: Startle-Reflex und subjektive Bewertungen bei affektiver Bildverarbeitung und deren Bezug zu genetischen Markern. In F. Lösel, D. Bender (Eds.), *45. Kongress der Deutschen Gesellschaft für Psychologie* (p. 56), Lengerich: Pabst.

Conzelmann, A., Weyers, P., Mucha, R., Jacob, C. P., Gerdes, A., **Schulz, S. M.**, Bähne, C. G., Lesch, K.-P., Alpers, G. W., Pauli, P. (2006). Emotional deficits in ADHD as indicated by ratings and startle response modulation and its relation to genetic markers. In G. W. Alpers, H. Krebs, A. Mühlberger, P. Weyers & P. Pauli (Eds.), *Wissenschaftliche Beiträge zum 24. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie* (pp. 17). Lengerich: Pabst.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2006). Schreckreflex-Modulation durch Soziale Ängstlichkeit. In F. Lösel, D. Bender (Eds.), *45. Kongress der Deutschen Gesellschaft für Psychologie* (p. 323), Lengerich: Pabst.

Schulz, S. M., Hofmann, S. G., & Alpers, G. W. (2006). Plötzliche Symptomreduktion (Sudden Gains) im Verlauf der Therapie Sozialer Phobie. In G. W. Alpers, H. Krebs, A. Mühlberger, P. Weyers & P. Pauli (Eds.), *Wissenschaftliche Beiträge zum 24. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie* (p. 191). Lengerich: Pabst.

Schulz, S. M., Gerdes, A., & Alpers, G. W. (2006). Emotionale Gesichter, Wörter und Szenen erleichtern unterschiedliche Komponenten valenzkompatibler motorischer Reaktionen. . In H. Hecht, S. Berti, G. Meinhardt & M. Gamer (Eds.), *Beiträge zur 48. Tagung experimentell arbeitender Psychologen* (pp. 305). Lengerich: Pabst.

Walker, S., Stangier, U., Steffens, M. C., Hofmann, S. G., **Schulz, S. M.** (2006). Implicit self-evaluation during socially anxious and depressive mood: Are there specific dysfunctional differences? Poster presented at the *36th Congress of the EABCT*, Paris.

Schulz, S. M., Alpers, G. W., Hofmann, S. G. (2005), Cognitive augmenting of social anxiety vs. relaxation in anticipation of public speaking, *Psychophysiology*, 42, S114.

Schulz, S. M., Alpers, G. W., Pauli, P. (2004). Facing emotional expressions with startle reflex, *Psychophysiology*, 41, S77.

Schulz, S. M., Alpers, G. W., & Pauli, P. (2004). Schreckreflex-Modulation durch emotionale Gesichtsausdrücke. In T. Rammsayer, S. Grabianowski & S. Troche (Eds.), *44. Kongress der Deutschen Gesellschaft für Psychologie: 100 Jahre Deutsche Gesellschaft für Psychologie* (p. 312). Lengerich, Berlin: Pabst Science.

Neumann, R., Alpers, G. W., & **Schulz, S. M.** (2003). The latency of facial action differentiates the valence of processed information: Evidence that facial action is facilitated by evaluative processing. *Journal of Psychophysiology*, 17(3), 170.

Schulz, S. M., Neumann, R., & Alpers, G. W. (2003). Interoceptive skills affect automatic processing of motor valence information in appraisals of attractiveness. *Journal of Psychophysiology*, 17(3), 178.

5.6.4 Eingeladene Vorträge (Invited Lectures)

Schulz, S. M. (1. März 2008). Vortrag beim Arzt-Patientenseminar von Universitätsklinik Würzburg und Biotronik: Leben mit Herzrhythmusstörungen: Möglichkeiten der implantierbaren Defibrillatortherapie und Umgang mit Angststörungen

Schulz, S. M. (28. Mai 2008). Vortrag zur Ärztefortbildung Klinikum Rothenburg: Leben mit Herzrhythmusstörungen -Therapie mit implantierbaren Defibrillatoren

Schulz, S. M. (2007). Rede, Vortrag, Referat - ein Horror!! ...oder was denken Sie? - Wie Gedanken mit sozialer Angst zusammenhängen. Einladung zum Forschungskolloquium des Instituts für Psychologie, TU-Chemnitz, 12. Juni, 2007. Leitung: Prof. Dr. Peter Sedlmeier.

5.6.5 Weitere Fachvorträge (Further Scientific Presentations)

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2008), Social Anxiety and the Mediating Power of Negative Thought. Talk presented at the *34. DGPA Jahrestagung "Psychologie und Gehirn"*, Magdeburg

Schulz, S. M. (2006), Social Anxiety - Subjective Feeling vs. Objective Body Reaction. Talk presented at the *Annual Meeting of Fellowship Holders of the Gottlieb Daimler- and Karl Benz-Foundation*, Lichtenfels, Germany.

Neumann, R., Alpers, G. W., **Schulz, S. M.**, (2003). The latency of facial action differentiates the valence of processed information: evidence that facial action is facilitated by evaluative processing. Symposium presented at the *29. Arbeitstagung "Psychophysiologische Methodik (APM)"* 2003, Würzburg, Germany.

Alpers, G.W., **Schulz, S. M.**, Gerdes, A., Mühlberger, A., Pauli, P. (2003). Ein Psychophysiologischer Attraktivitätsdetektor. Paper presented at the *Annual Mindlab Workshop, DaimlerChrysler Research Center Ulm*, Germany.