

ORIGINAL ARTICLE

Anxiety and sympathetic response to stress in allergic patients

Petra SOLARIKOVA, Igor BREZINA, Daniela TURONOVA, Jakub RAJCANI

Department of Psychology, Faculty of Arts, Comenius University, Bratislava, Slovakia

Correspondence to: Petra Solarikova, PhD., Department of Psychology, Faculty of Arts, Comenius University, Gondova Street Nr. 2, SK-814 99 Bratislava, Slovakia, E-MAIL: petra.solarikova@uniba.sk

Submitted: 2016-09-10 Accepted: 2016-10-25 Published online: 2015-11-03

Key words: **allergy; anxiety; stress; sympathetic-adrenomedullary (SAM) stress system**

Act Nerv Super Rediviva 2016; 58(3): 88–94

ANSR580316A06

© 2016 Act Nerv Super Rediviva

Abstract

OBJECTIVES: The aim of this study is to describe sympathetic responses in stress in relation to allergy and trait anxiety.

METHODS: The study was performed on 149 participants. Sample consists of allergy group (N=83) and control group (N=48). Laboratory stress was induced by Psychosocial stress test, sympathetic markers (heart rate and level of skin conductance) were monitored by system PowerLab and trait anxiety via questionnaire STAI.

RESULTS AND DISCUSSION: During the stress procedure, patients with allergy exhibited decreased heart rate (HR) during whole procedure, while skin conductance level (SCL) was observed reduced only in the recovery phase. We have recorded the occurrence of high trait anxiety in allergic patients.

CONCLUSIONS: Allergic diseases are civilizational diseases, which is necessary to pay attention to, given their enormous incidence in the population and vulnerability of allergic patients to stress. We recommend further investigation of the issue of separation anxiety from allergies. We lean toward a new view of the concept of stress, which is more dynamic and adaptive.

INTRODUCTION

The presented study deals with problems concerning abnormal functioning of Sympathetic-adrenomedullary (SAM) stress system in patients with allergy. An alarming growth of allergies requires an in-depth research of the circumstances of possible inappropriate stress reaction associated with the development of allergy. Our goal was to analyze relevant psychophysiological indicators in patients with atopy linked to their changed stress reactions.

The association between anxiety and atopic diseases was examined through several studies (Seiffert *et al* 2005; Linnet & Jenec 2001,1999). Atopy is a hereditary predisposition toward developing various types of allergies primarily including atopic dermatitis, allergic asthma, allergic rhinitis (atopic triad), but

also urticaria and some food allergies. The symptoms are manifested in hypersensitive reaction to a specific allergen, production of IgE antibodies and reactivity of smooth muscles to histamine. Atopy is a feature of allergic reactions which deteriorates their course (Gold & Kemp 2005).

There are various studies indicating a close relation between allergic asthma (AA) and anxiety. This study point mostly to their negative influence as to chronicity of the disease or worsening of the condition (Wamboldt *et al* 2003). Anxiety both as a state and as a trait may increase one's subjective feeling of having difficulty in breathing and undoubtedly represents a significant mechanism supporting the occurrence of asthma attacks (Pietras *et al* 2009; Holgate 2002). However, this is true also vice versa, meaning that asthma attacks are accompanied by a high level of

anxiety and a worse feelings. The intensity of anxiety as a state and anxiety as a trait is in relationship with the level of asthma control (Wang *et al* 2010) as well as with the level of asthma severity (Pietras *et al* 2011).

The relationship between anxiety and atopic dermatitis (AD) was also demonstrated by studies by Linnet and Jenec (2001, 1999) which also claim that psychological interventions (psychotherapy) improve not only the psychological conditions but also skin allergy symptoms, which confirms the significance of psychological treatment of people with AD. Emotional factors participate and cooperate in the occurrence of skin diseases. Anxiety represents one of the factors which play a significant role also in the etiopathogenesis of atopic dermatitis (Medansky & Handler 1981; Peluso *et al* 2009).

The character of the atopic disease belongs also to the factors which may participate in the change of psychological resilience. Having to experience the uncontrollability of the disease from the point of view of the start of acute phase and the unpredictability of the effectiveness of treatment may in atopic sufferers lead to a generally higher external control and a low self-efficacy (Buske-Kirschbaum *et al* 2008). The fact that atopic dermatitis plays in comparison to other skin diseases a significant role in the relationship with anxiety was also stated by Ginsburg *et al* (1993), who claim that there is no connection between the high anxiety and an acute or generally chronic skin disease. They compared individual dermatologic diagnoses and found out that patients with AD exhibited significantly higher scores in anxiety in comparison to patients with Pityriasis rosea or psoriasis. It is thus improbable that the anxiety in patient with AD is caused only due to increasing gravity of acute or chronic skin diseases. We assume that it is manifested through a certain as yet not specified particularity.

The strong relationship between atopic diseases and anxiety was also suggested to by Kagan *et al* (1991) who published their findings from two independent studies which revealed higher occurrence of pollinosis and social anxiety in relatives of shy children than in relatives of sociable children. The factor of social anxiety correlated with immunological vulnerability in the selected atopic diseases. These data suggest a genetic relationship between social anxiety and the selected atopic allergies.

The results of several studies indicate that personality characteristics of patients with allergic rhinitis (AR) are parallel to other atopic diseases (Buske-Kirschbaum *et al* 2008,2010; Bell *et al* 1991, Kagan *et al* 1991). The authors declare that atopic patients are generally characterized by a higher anxiety, social anxiety, fearfulness, shyness and vulnerability to stress. With regard to previous studies we assume that the personality structure of patients with AR can be characterized by increased vulnerability to stress because of which they are more prone to a permanent stress overload.

A remarkable dilemma was raised by Hashizume *et al* (2005) who mention that anxiety correlates with the synthesis of the antibody IgE and Th2 immune response. By this statement, the authors point out to a significant relationship between anxiety and immune system in patients with atopic dermatitis which confirms the connection between atopic disease and severe anxiety, whereas both phenomena show similar pathophysiology. In this case it is problematic to find out whether the pathophysiology is caused to a larger extent by the inflammatory allergic disease as such or by the dispositional trait anxiety.

A large number of works dealt with examination of the functioning of hypothalamo-hypophyseal-adrenocortical (HPA) axis and Sympathetic-adrenomedullary system (SAM) in patients with allergy (Buske-Kirschbaum *et al* 1997, 2002, 2003, 2010; Seiffert *et al* 2005; Chrousos 2009, Tran *et al* 2010; Elias 2010; Buddenkotte & Steinhoff 2010). A decreased reaction of cortisol to stress stimuli was repeatedly observed, however, only in the connection with atopic dermatitis, later also in connection with allergic asthma and in a small extent in the connection with allergic rhinitis which, so far, has been given not so much attention. Several findings concerning psychophysiological indicators were ambiguous, mainly those relating to the basal cortisol level and the changes of the SAM system.

In examination of psychophysiological reactivity to stress in patients with allergy we took into account the findings on hyperactivation of SAM system made by the team of Busko-Kirschbaum (2006) "Psycho-neuro-endocrino-immunological stress model in atopic patients". According to this model in atopic patients, the HPA axis is characterized by decreased activity, whereas the contrary is true as to the activity of the SAM system. The SAM system remains hyperactive and sends the body into allostasis.

METHODS

The research sample consisted of 149 participants of a diverse age range (18–38, $M=21.6$, $SD=3.73$) and a relatively equal number of participants as to gender (women $N=83$, men $N=66$). Almost all the participants had already completed higher education studies or were university students (98.8%), two participants were high school students and one participant was a high school graduate. From the point of view of the career orientation, the sample included participants from the field of psychology ($N=104$), other academic field ($N=14$) and from the field of technology and economics ($N=32$). Some of the participants were excluded from psychophysiological analysis for various technical reasons ($N=18$). The experimental group was constituted of participants suffering from an allergic disease ($N=83$) (the disease was in remission at the time of testing, of which in regard to gender: $W=45$, $M=38$). The control group consisted of participants

with a negative allergic anamnesis (N=48, of which: W=26, M=22).

Laboratory setting was based on the public speech which belongs among the most commonly used psychological tests in the investigation of stress (The Trier Social Stress Test). The test is remarkable for its high reliability in detection of the regulation of the HPA axis (Kudielka *et al* 2007). Social evaluation and uncontrollability have been identified as the key components. The original version consists of a fake job interview and an arithmetic task. The participant's task is to prepare for presenting oneself in a job interview (5–10 min.) and to complete an arithmetic task (5 min.). Participant accomplishes the tasks in front of an audience comprised of experts on judging one's non-verbal communication. The stress situation is intensified also by using video cameras, monitors, a microphone, lighting and a stage. We have worked out this method on the basis of the original test (Trier Social Stress Test) by applying our modification in the form of giving a speech on a socially sensitive issue (Makatsori *et al* 2004) and asking a series of general knowledge questions.

The experimental procedure is comprised of the following phases:

- Adaptation phase (10 min.): The participant was sitting at ease, having adapted himself/herself to the new situation and environment.
- Preparation phase (10 min.): The participant was given the task to prepare background papers to be able to give a speech on a previously determined subject.
- Presentation phase (15 min.): The participant was assigned to talk in front of a board of judges whereas his task was to defend his attitude on the previously determined subject, and this was followed by answering knowledge questions given to him by the board of judges, and finally he was given an arithmetic task.
- Recovery phase (45 min): During the last part of the procedure, the participant was told to be trying to quieten down and relax as much as possible.

State and trait anxiety was measured by Slovak version of the State-Trait Anxiety Inventory (STAI) which was constructed by Spielberger. The inventory is considered as the most widely used method focused on anxiety diagnostic as the current state (part X1 – S) and anxiety as a trait (part X2 – T).

The assessment of Sympathetic-adrenomedullary (SAM) stress system during the experiment was conducted via monitoring of its correlates – heart rate (HR) and level of skin conductance (SCL) by psychophysiological instrumental technique – (PowerLab Hardware with software applications LabChart, ADInstruments, Sydney, Australia).

Data on state and trait anxiety and psychophysiological data were analyzed by Kruskal-Wallis test. Statistical significance of all analyses was set to $p < 0.05$. All statistical analyses were done in IBM SPSS software.

RESULTS

H1: We assumed that patients with allergy exhibit a higher level of anxiety in comparison with a control group. During the examination of anxiety as a personality trait in the connection with allergy, a significant effect was observed ($\chi^2=4.296$, $df=1$, $p=0.038$). In allergic persons, a higher level of anxiety was recorded than in non-allergic persons (Figure 1).

Our findings demonstrate that there was no difference between groups in state anxiety before ($\chi^2=0.007$, $df=1$, $p=0.931$) and after presentation phase ($\chi^2=0.073$, $df=1$, $p=0.787$), also in current subjective experience of stress ($\chi^2=0.041$, $df=1$, $p=0.840$) measured by simple 10-point scale of perceived stress.

H2: We assumed that patients with allergy exhibit a higher level of SAM system (heart rate, level of skin conductance) in stressful laboratory conditions in comparison with a control group.

Psychosocial stress test resulted in a significant increase HR ($\chi^2=6.269$, $df=1$, $p=0.012$) in both groups.

In verifying the above stated association, the significant effect between the groups in the adaptation phase ($\chi^2=5.627$, $df=1$, $p=0.018$), in the presentation phase ($\chi^2=6.793$, $df=1$, $p=0.009$) and in the recovery phase ($\chi^2=7.944$, $df=1$, $p=0.005$) was confirmed. In allergic patients, we recorded generally lower heart rate during the whole experiment course.

During the stress procedure, patients with atopy exhibited decreased heart rate (Figure 2).

Another parameter that reflects the activity of the SAM system was electrodermal activity, in which we have specifically focused on the SCL. Significant effect between groups only appeared in the recovery stage ($\chi^2=8.969$, $df=1$, $p=0.003$), but in the opposite direction expected. The next phases did not confirm signifi-

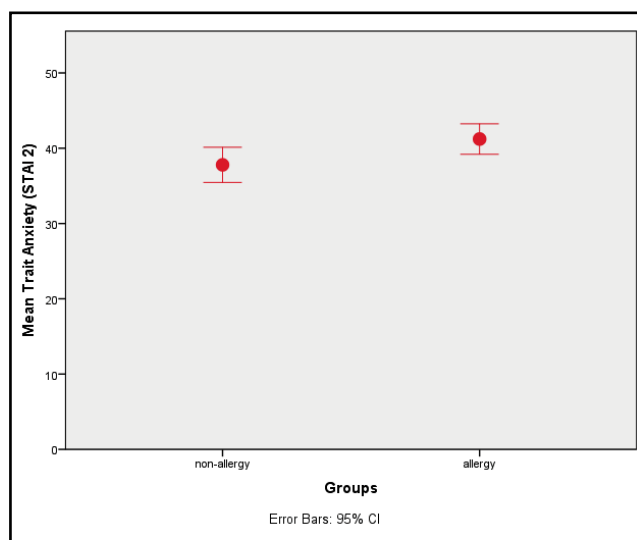


Fig. 1. Average anxiety rates in persons with allergy and non-allergic persons.

cance: adaptation phase ($x^2=0.221$, $df=1$, $p=0.639$), the preparatory phase ($x^2=0.083$, $df=1$, $p=0.773$) and stage performances ($x^2=0.049$, $df=1$, $p=0.824$) (Figure 3).

DISCUSSION

Anxiety as a personality trait is presumably considered as one of the most common correlates within the context of the stress reaction. As expected, the relationship

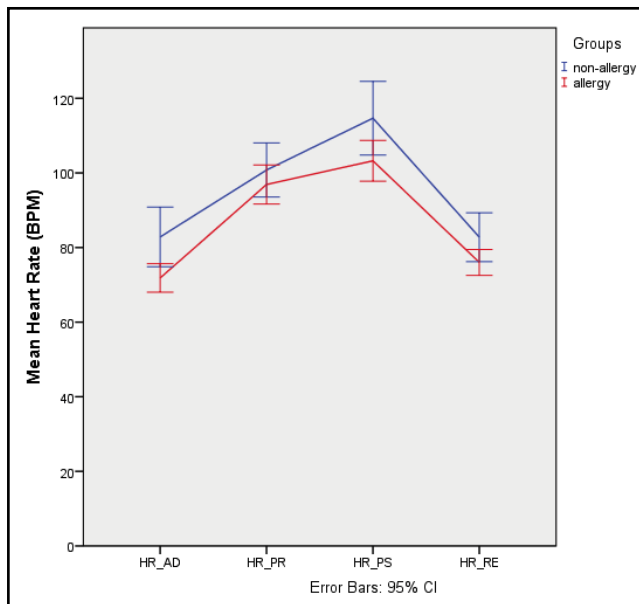


Fig. 2. Average heart rate indicators in non-allergic persons and persons with allergy (HR_AD=Adaptation phase, HR_PR=Preparation phase, HR_PS= Presentation phase HR_RE=Recovery phase).

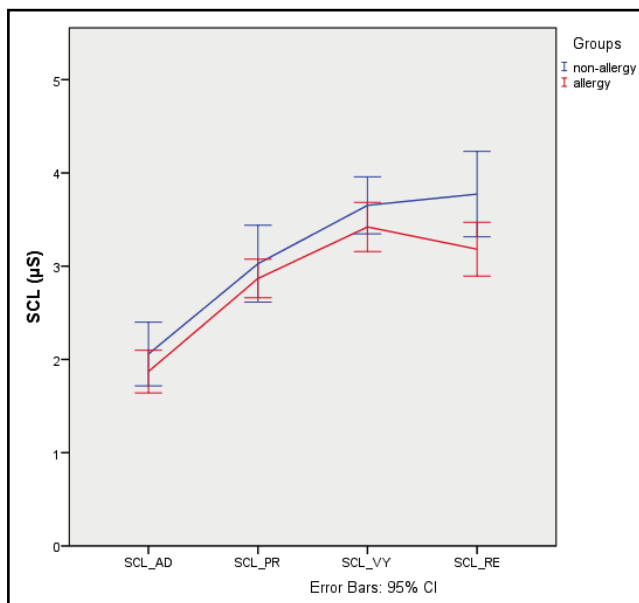


Fig. 3. Average rates of the skin conductance level (SCL) in non-allergic and allergic persons. (SCL_AD=Adaptation phase, SCL_PR=Preparation phase, SCL_PS= Presentation phase SCL_RE=Recovery phase).

between anxiety and allergy proved to be true. Similar conclusions can be found also in other studies devoted to examination of higher level of anxiety: Seiffert *et al* (2005), Linnet and Jemec (2001), Hashiro, Okumura (1998), Wierenga *et al* (1991), Scheich *et al* (1993), Ginsburg *et al* (1993), Gieler *et al* (1990), Garrie *et al* (1974), in patients with atopic dermatitis, Huovinen *et al* (2001) in patients with allergic asthma, Stauder, Kovács (2003), Buske-Kirschbaum *et al* (2008) in patients with atopic triad (AA, AD, AR). We would like to add the finding made by Radošević-Vidaček *et al* (2009), whose examinations did not confirm the association between allergic diseases and anxiety. This might be caused, apart from other things, also by the very wide variety of tests designed for examination of anxiety and blending the boundaries between the trait and condition, either current or only vaguely defined by a relatively short period of time. We would also like to mention a study where the authors point out, in general, to an important association between allergic irritation and CNS (Blais, 2008), stating that in the patients with allergic rhinitis, a higher level of anxiety, tiredness, passivity, sleep disturbances or cognitive deficits were recorded. These findings confirm the importance to explore the complex relationships between psychical insufficiency where anxiety plays an important role, along with the level of allergic irritation. The close relationship between allergic asthma and anxiety was also indicated by Wamboldt *et al* (2003) who emphasize mainly their negative effect in increasing the chronicity of the disease or worsening of the condition. Apart from that, Peluso *et al* (2009) and also Medansky and Handler (1981) expressed the opinion that anxiety is actually one of the factors playing a significant role in the etiopathogenesis of the atopic dermatitis. In older study of Garrie *et al* (1974) consider that allergies could manifest themselves in a specific pattern in comparison with another diseases. They recorded a higher level of anxiety in patients with atopic dermatitis in comparison to other acute skin diseases (psoriasis, pityriasis rosea). This is the reason why anxiety in AD might also have some other cause than just the inflammatory process followed by psychical discomfort. Ginsburg *et al* (1993) also confirm that the high anxiety is not connected only to an acute or generally chronic skin disease. Through comparison of individual dermatologic diagnoses they discovered that patients with AD exhibited significantly higher scores in anxiety as opposed to patients with „Pityriasis rosea“ or psoriasis. For this reason it is improbable that anxiety in AD is caused only due to increasing of the severity of acute or skin diseases.

In the heart rate (HR) as one of the biomarkers of the Sympathetic-adrenomedullary (SAM) system, we monitored its lowered tendency in patients with allergy in the adaptation, presentation and recovery phase of the experiment. This phenomenon is not supported by studies which present the conventional assumption that hyperarousal occurs more likely in patients with

allergy: increased HR in adult patients with atopic dermatitis as the response to mental stress (Munzel & Schandry 1990; Faulstich *et al* 1985), increased HR in patients with atopic dermatitis as well as with allergic rhinitis as the response to mental stress (Kimata, 2003). The increased HR in adult patients with AD was demonstrated not only in the laboratory-induced stress situation (TSST), but also during quiet phases (Tran *et al* 2010; Hanifin *et al* 2001). An interesting finding was presented by Seiffert *et al* (2005), who examined HR through mental test in adolescents with atopic dermatitis in remission as well as in the acute stage of the allergic disease. Despite the fact that in patients with AD, a higher heart rate was recorded during the whole course of the experimental test, the findings showed that patients with AD did not exhibit a higher reactivity to psychological stress than the healthy members of the control group. That means that, as opposed to the basal situation before the test and the recovery phase after the test, patients with allergy did not exhibit increased HR due to stress. This finding indicates that patients with AD show greater difficulties rather in balance of the sympathetic and parasympathetic nervous system during quiet conditions, than in a stress situation. The feeble reaction to the mental task might be the manifestation of a general hyperactivity of the sympathetic nervous system in patients with AD, which might cause problems to ease oneself in quiet conditions that would enable a follow-up increase in the response to the activation in stress. The findings of the team Buske-Kirschbaum *et al* do not show an increased heart rate in patients with allergy: the study by Buske-Kirschbaum *et al* (1997) does not present any differences as to heart rate in the endurance test (TSSC) in children with AD, as well as in adult persons with AD (Buske-Kirschbaum *et al* 2002) or in children with AA (Buske-Kirschbaum *et al* 2003). It is important to realize that the discrepancies in the heart rate recordings might be caused by a different design of the experimental environment in regard to basal phases or creation of burdensome conditions, different type of research sample selection as to choice of atopic diseases which are relatively widely varied etc.

We can state that our findings as regards to insufficient response of the heart rate during the performance in allergic patients support our explanation concerning the overall dysregulation of the SAM system, which may demonstrate itself in the form of hyperreaction or hyporeaction just like in case of the cortisol reactivity. This suggestion is supported by the study by Young *et al* (1998) who recorded a lower heart rate and a lower blood pressure in high-anxious participants in comparison to low-anxious participants. The hyperreactivity of the autonomic nervous system (ANS) may just be the first expression of the high anxiety, whereas this condition may be followed by a compensatory mechanism for hyporeactivity. However, the basal level of heart rate and blood pressure did not correlate with anxiety. That

is one of the reasons why it seems to be highly probable that the dysfunction of the ANS system embraces several alternatives and the individual stress responses are not coordinated with each other (Duncko *et al* 2006). The monitoring of lowered heart rate level even during restful situations in allergic patients encourages us to elaborate a more in-depth analysis of this challenging phenomenon in the future, which might not manifest itself inevitably only in stress situations but also in normal conditions. The increased activity of parasympathetic nervous system is also described in the study by Boettger *et al* (2009) which explains these contradictory viewpoints and conventionally expected hyperarousal by describing how the increased activity of the parasympathetic nervous system in atopic patients demonstrates to have anti-inflammatory effects that sooner ease the dermatitis symptoms. This might be a natural reason for the body response to the acute allergic inflammation. It is still an open question under which condition shall occur this reaction of the ANS.

The performance part in our research exhibited a response to stress by increased SCL level in allergic persons as well as in non-allergic persons which matches the common reaction. However, no difference between both groups was recorded in the response to stress and thus there was no manifestation of the high reactivity. A contrary phenomenon was observed in the recovery phase, when a significant difference between the both groups was shown where allergic persons exhibited a better ability to relax and their SCL level decreased. This is again a finding that contradicts the conventional expectation, as we would expect deteriorated recuperation in allergic persons and persistence of hyperactivation of their SAM system due to psychosomatic troubles which are typical for this disease.

As the EDA index is only rarely used in the investigation of this issue, we apply in the presentation of the findings mainly the results from other SAM biomarkers. One of the few studies devoted to a similar subject is the study by Seiffert *et al* (2005) which also focused on evaluation of the EDA in allergic persons in stress situation (TSST), however, by using other parameter (a spontaneous electrodermal response – SSCR). It was manifested that the electrodermal activity (SSCR) gained a higher level in the control group as compared to the participants with atopic dermatitis. The authors explained this finding by stating that atopic patients have a drier skin which might diminish the ability of skin to sweat. This hypothesis was also confirmed by findings made by Koehler and Weber (1992). We do not support this explanation in our findings, as our research sample consisted predominantly of atopic patients with rhinitis and their response to stress was the same as in the members of the control group.

Despite that we lean toward the explanation which, by using just one more parameter (EDA) confirms a different view on the activity of the ANS, whose dysfunction might develop in accordance with various

conditions and may manifest either in hyperactive or hypoactive manner.

CONCLUSION

There are several studies hinting to the fact that allergic diseases have a significant and in many cases also a rather specific relation with anxiety. Through our research we could also confirm the higher anxiety in allergic participants. We consider it inevitable to devote further effort to explore this dilemma postulated also by Hashizume *et al* (2005) and find the answer to the following question: Which factor – whether the allergic inflammation or anxiety as a personality trait, will dominate in the formation of the same pathophysiology which occurs in both conditions. With regard to the fact that both factors often occur together, we have to arrange our research in such a way which makes it possible to separate these factors and thus enabling us to examine this issue more deeply.

We consider controversial finding about the hypo-reactivity of the SAM system in patients with atopy, as also indicated by the postulate of the existence of opposite manifestation of the dysregulation of the autonomic nervous system. We lean toward a new view of the concept of stress, which is more dynamic and adaptive.

ACKNOWLEDGEMENT

This study was supported by grant of APVV-0496-12.

REFERENCES

- Bell IR, Jasnoski ML, Kagan J, King DS (1991). Depression and allergies: survey of a nonclinical population. *Psychother. Psychosom.* **55**: 24–31.
- Blaiss MS (2008). Pediatric allergic rhinitis: physical and mental complications. *Allergy Asthma Proc.* **29**: 1–6.
- Boettger MK, Bär KJ, Dohrmann A, Müller H, Mertins L, Brockmeyer NH, Agelink MW (2009). Increased vagal modulation in atopic dermatitis. *J Dermatol Sci.* **53**: 55–59.
- Buddenkotte J & Steinhoff M (2010). Pathophysiology and therapy of pruritus in allergic and atopic diseases. *Allergy* **65**: 805–821.
- Buske-Kirschbaum A, von Auer K, Krieger S, Weis S, Rauh W, Hellhammer D (2003). Blunted cortisol responses to psychosocial stress in asthmatic children: A general feature of atopic disease? *Psychosom Med.* **65**: 806–810.
- Buske-Kirschbaum A, Geiben A, Höllig H, Morschhäuser E, Hellhammer D (2002). Altered responsiveness of the hypothalamus-pituitary-adrenal axis and the sympathetic adrenomedullary system to stress in patients with atopic dermatitis. *J Clin Endocrinol Metab.* **87**: 4245–4251.
- Buske-Kirschbaum A, Jobst S, Wustmans A, Kirschbaum C, Rauh W, Hellhammer D (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosom Med.* **59**: 419–426.
- Buske-Kirschbaum A, Ebrecht M, Kern S, Gierens A, Hellhammer DH (2008). Personality characteristics in chronic and nonchronic allergic conditions. *Brain Behav Immun.* **22**: 762–768.
- Buske-Kirschbaum A, Ebrecht M, Hellhammer DH (2010). Blunted HPA axis responsiveness to stress in atopic patients is associated with the acuity and severeness of allergic inflammation. *Brain Behav Immun* **24**: 1347–1353.
- Duncko R, Makatsori A, Fickova E, Selko D, Jezova D (2006). Altered coordination of the neuroendocrine response during psychosocial stress in subjects with high trait anxiety. *Prog Neuropsychopharmacol Biol Psychiatry.* **30**: 1058–1066.
- Elias PM (2010). Therapeutic implications of a barrier-based pathogenesis of atopic dermatitis. *Ann Dermatol* **22**: 245–254.
- Faulstich ME, Williamson DA, Duchmann EG, Conerly SL, Brantley PJ (1985). Psychophysiological analysis of atopic dermatitis. *J Psychosom Res.* **29**: 415–417.
- Garrie EV, Garrie SA, Mote T (1974). Anxiety and atopic dermatitis. *J Consult Clin Psychol.* **42**: 742.
- Gieler U, Ehlers A, Höhler T, Burkard G (1990). The psychosocial status of patients with endogenous eczema: A study using cluster analysis for the correlation of psychological factors with somatic findings. *Hautarzt* **41**: 416–423.
- Ginsburg IH, Prystowsky JH, Kornfeld DS, Wolland H (1993). Role of emotional factors in adults with atopic dermatitis. *Int J Dermatol.* **32**: 656–660.
- Gold MS & Kemp AS (2005). Atopic disorders in childhood. *Med J Aust.* **182**: 298–304.
- Hanifin JM, Thurston M, Omoto M, Cherill R, Tofte SJ, Graeber M (2001). The eczema area and severity index (EASI): assessment of reliability in atopic dermatitis. EASI Evaluator Group. *Exp Dermatol.* **10**: 11–18.
- Hashiro M & Okumura M (1998). The relationship between the psychological and immunological state in patients with atopic dermatitis. *Journal of Dermatological Science* **16**: 231–235.
- Hashizume T, Horibe T, Ohshima A, Ito T, Yagi H, Takigawa M (2005). Anxiety accelerates T-helper 2-tilted immune responses in patients with atopic dermatitis. *Br J Dermatol.* **152**: 1161–1164.
- Holgata ST (2002). Airway inflammation and remodeling in asthma: current concepts. *Mol Biotechnol.* **22**: 179–89.
- Huovinen E, Kaprio J, Koskenvuo M (2001). Asthma in relation to personality trait, life satisfaction, and stress: a prospective study among 11 000 adults. *Allergy* **56**: 971–977.
- Chrousos, G P (2009). Stress and disorders of the stress system. *Nat Rev Endocrinol.* **5**: 374–381.
- Kagan J, Snidman N, Julia-Sellers M, Johnson MO (1991). Temperament and Allergic Symptoms. *Psychosomatic Medicine* **53**: 332–340.
- Kimata H (2003). Listening to Mozart reduces allergic skin wheal-responses and in vitro allergen-specific IgE production in atopic dermatitis patients with latex allergy. *Behav Med.* **29**: 15–9.
- Koehler T & Weber D (1992). Psychophysiological reactions of patients with atopic dermatitis. *J Psychosom Res.* **36**: 391–394.
- Kudielka BM, Hellhammer D, Kirschbaum C (2007). Ten years of research with the Trier Social Stress Test revisited. In: Harmon-Jones E, Vinkelman P, editors. *Social Neuroscience: Integrating Biological and Psychological Explanations of Social Behavior*. Guilford Press: New York, p. 56–83.
- Linnet J & Jemec GB (1999). An assessment of anxiety and dermatology life quality in patients with atopic dermatitis. *Br J Dermatol.* **140**: 268–272.
- Linnet J & Jemec GB (2001). Anxiety level and severity of skin condition predicts outcome of psychotherapy in atopic dermatitis patients. *Int J Dermatol.* **40**: 632–636.
- Makatsori A, Duncko R, Moncek F, Loder I, Katina S, Jezova D (2004). Modulation of neuroendocrine response and non-verbal behavior during psychosocial stress in healthy volunteers by the glutamate release-inhibiting drug lamotrigine. *Neuroendocrinology.* **79**: 34–42.
- Medansky RS & Handler MD (1981). Psychosomatic Dermatology. *Int J Dermatol.* **20**(1): 42–43.
- Munzel K & Schandry R (1990). Atopic eczema: Psychophysiological reactivity with standardized stressors. *Hautarzt.* **41**: 606–611.
- Peluso PR, Peluso PJ, Buckner PJ, Kern MJ, Curlette W (2009). Measuring lifestyle and attachment: an empirical investigation linking individual psychology and attachment theory. *Journal of Counseling & Development.* **87**(4): 394–403.
- Pietras T, Witusik A, Panek M, Górski P (2009). Anxiety and depression in patients with obstructive diseases. *Pol Merkur Lekarski.* **26**: 631–635.

- 34 Pietras T, Panek M, Witusik A, Wujcik R, Szemraj J, Górski P, Kuna P (2011). Analysis of the correlation between level of anxiety, intensity of depression and bronchial asthma control. *Post Dermatol Alergol*. **XXVIII**(1): 15–22.
- 35 Radošević-Vidacek B, Koščec A, Bakotić M, Macan J, Bobić J (2009). Is atopy related to neuroticism, stress, and subjective quality of life? *Arh Hig Rada Toksikol*. **60**: 99–107.
- 36 Seiffert K, Hilbert E, Schaechinger H, Zouboulis CC, Deter HC (2005). Psychophysiological reactivity under mental stress in atopic dermatitis. *Dermatology*. **210**: 286–293.
- 37 Scheich G, Florin I, Rudolph R, Wilhelm S (1993). Personality characteristics and serum IgE level in patients with atopic dermatitis. *J Psychosom Res* **37**: 637–642.
- 38 Stauder A & Kovács M (2003). Anxiety symptoms in allergic patients: identification and risk factors. *Psychosom Med* **65**: 816–823.
- 39 Tran BW, Papoiu AD, Russoniello CV, Wang H, Patel TS, Chan YH, Yosipovitch G (2010). Effect of itch, scratching and mental stress on autonomic nervous system function in atopic dermatitis. *Acta Derm Venereol*. **90**: 354–361.
- 40 Wamboldt MZ, Laudenslager M, Wamboldt FS, Kelsay K, Hewitt J (2003). Adolescents with atopic disorders have an attenuated cortisol response to laboratory stress. *J Allergy Clin Immunol*. **111**: 509–514.
- 41 Wierenga EA, Snoek M, Jansen HM, Bos JD, van Lier RA, Kapsenberg ML (1991). Human atopen-specific types 1 and 2 T helper cell clones. *J Immunol* **147**: 2942–2949.
- 42 Young EA, Nesse RM, Weder A, Julius S (1998). Anxiety and cardiovascular reactivity in the Tecumseh population. *J Hypertens*. **16**: 1727–1733.