



Identification and association of relationships between selected personal and environmental factors and formal components of temperament and strategies of coping with stress in asthmatic patients

Michał Panek^{a,*}, Tadeusz Pietras^b, Andrzej Witusik^c, Łukasz Wieteska^d, Beata Małachowska^e,
Łukasz Mokros^{b,f}, Wojciech Fendler^e, Janusz Szemraj^d, Piotr Kuna^a

^a Department of Internal Medicine, Asthma and Allergy, Medical University of Lodz, 22 Kopcińskiego Str., 90-153 Lodz, Poland

^b Department of Pneumology and Allergology, Medical University of Lodz, 22 Kopcińskiego Str., 90-153 Lodz, Poland

^c Department of Psychology, The Jan Kochanowski University of Humanities and Sciences in Kielce, Branch in Piotrków Trybunalski, 114/118 Słowackiego Str., 97-300 Piotrków Trybunalski, Poland

^d Department of Medical Biochemistry, Medical University of Lodz, 6/8 Mazowiecka Str., Lodz 92-215, Poland

^e Department of Pediatrics, Oncology, Hematology and Diabetology of Medical University of Lodz, 36/50 Sporna Str., Lodz 91-738, Poland

^f Students Research Group at the Department of Internal Medicine, Asthma and Allergy, Medical University of Lodz, 22 Kopcińskiego Str., 90-153 Lodz, Poland

HIGHLIGHTS

- The genetic and environmental factors affected stress coping by asthmatic patients.
- The psychopathological variables correlated with stress coping by asthmatic patients.
- Temperamental traits depend on environmental and psychopathological variables.
- Tth1111 polymorphic form of NR3C1 gene determined perseverance.
- The increase in TGFβ1 expression led to a decrease in patients' emotional reactivity.

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ABSTRACT

Background: Personal and environmental factors might have an impact on strategies of coping with stress and temperamental traits according to the Regulatory Theory of Temperament in asthmatic patients. They can modify the clinical picture, the course of a disease and effectiveness of treatment. Personal variables are key factors in determining formal characteristic of behavior and effective management method in asthmatic patients.

Aim of study: The aim of the study was to identify selected personal and environmental factors, as well as factors inducing attacks and asthma exacerbations or maintaining them in a complex of personal traits of patients.

Methods: Two hundred and eighty one participants were included in the study. Of this number 122 subjects were healthy volunteers and 159 were asthmatic patients. In all the subjects the authors applied the Formal Characteristic of Behaviour – FCZ-KT – Temperament Inventory, Coping Inventory for Stressful Situations (CISS), Beck Depression Inventory, State-Trait Anxiety Inventory and Borg Rating of Perceived Exertion (RPE) Scale. Genotyping of polymorphic forms of NR3C1 gene was conducted with PCR-RFLP and PCR-HRM methods. Expression of TGFβ1 gene was measured with the use of qRT-PCR.

Results: The authors confirmed a significant influence of personal and environmental factors, such as: age, height, body weight, sex, asthma exacerbations, drugs administered by patients, allergy and psychopathological variables on strategies of coping with stress by asthmatic patients (Task-Oriented Coping, Emotion-Oriented Coping, Avoidance-Oriented Coping, distraction seeking, social diversion). Temperamental traits (Briskness, Perseverance, Sensory Sensitivity, Emotional Reactivity, Endurance, Activity) depend on age, sex, body weight, genetic predispositions and they are modified by asthma exacerbations, allergy, drugs administered by patients, depression and anxiety (state and trait). The authors confirmed a correlation between Tth1111 polymorphic form of NR3C1 gene and perseverance ($p = 0.0450$). It was noted that an increase in the TGFβ1 expression level led to a decrease in the patients' emotional reactivity ($p = 0.0212$).

* Corresponding author.

E-mail addresses: michalmp@poczta.onet.pl (M. Panek), tadeusz.pietras@umed.lodz.pl (T. Pietras), awitusik@ujk.edu.pl (A. Witusik), l.wieteska@yahoo.com (Ł. Wieteska), b.e.malachowska@gmail.com (B. Małachowska), sage.mkrs@gmail.com (Ł. Mokros), wojciech.fendler@umed.lodz.pl (W. Fendler), janusz.szemraj@umed.lodz.pl (J. Szemraj), piotr.kuna@umed.lodz.pl (P. Kuna).

Conclusions: Strategies of coping with stress and temperamental traits according to the Regulative Theory of Temperament in asthmatic patients are determined by personal and environmental factors.

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1. Introduction

Asthma is a heterogeneous disease with complex etiopathogenesis, where it is neither easy nor direct to identify relationships between groups of risk factors predisposing for a disease and somatic as well as psychic symptoms. Both personal (genetic characteristics, sex, body weight, temperament, personality) and environmental factors affect the development and expression of variable bronchial obturation. Heterogeneity of asthma, incomplete gene penetrance, differentiated protein expression, complex gene–gene and gene–environment interactions make it difficult to make a precise presentation of molecular mechanisms of induction of psychopathological disorders and behavioral changes [1,2,3,4].

In asthma, according to a current biopsychosocial paradigm, a psychic factor plays an important role in the etiopathogenesis of a disease [1,4,5,6]. Although paroxysmal bronchospasm and dyspnea are clearly induced by inflammation, asthma is a psychosomatically conditioned disease [7,8]. Chronicity and paroxysmal nature of symptoms, impaired social and family functioning of the sick person, disturbed human relationships, school absenteeism and less intense professional activity might greatly decrease the mood and the quality of life of patients [1,4]. Lack of life success and professional promotion stigmatize asthmatic patients, which inhibits the process of formation of mature personality. It can be particularly observed in children. Thus, the disease contributes to lower self-esteem, which intensifies their anxiety and mood swings [1,4]. Strong stress associated with a paroxysm of dyspnea induced by various factors (genetic, environmental) intensifies hyperventilation which together with bronchial overreactivity intensifies a bronchial contraction and a dyspnea paroxysm [1,4,9,10,11]. Temperament, which is an essential factor modeling an individual response to stress (stressor – dyspnea), might change the clinical picture and the course of asthma [12,13,14]. This formal characteristic of behavior (temperament) is one of the personality dimensions conditioned biologically at the level of congenital and acquired properties of the central nervous system [12,13]. Temperament is genetically conditioned at the DNA level (deoxyribonucleic acid) and through biological mediating factors it affects the limbic system, which codetermines the complex of personality traits (psychoticism – P, extraversion – E, neuroticism – N) – the PEN theory (Eysenck's theory of superfactors) [12,13,14]. Scientific studies showed that temperament induces negative effects of extreme stress which is a recognized factor of affective and personality disorders [12,13,14,15,16,17]. Numerous laboratory tests confirmed an impact of many genetic determinants and inflammatory proteins on a change of the structure of PEN factors and the personality taxonomy. Researchers discovered that a number of glucocorticoid receptors (GR) in cells as well as their function is reduced in patients with mood disorders [18]. Expression of GR is determined and regulated by glucocorticoid receptor gene *NR3C1* (nuclear receptor subfamily 3, group C, member 1 gene) [19,20,21]. They also observed that there is a relationship between haplotypes of *NR3C1* gene and depression and anxiety [22]. It is known that inflammatory mediators are important risk factors of mood disorders and in the course of somatic inflammatory diseases intensified mood disorders correlate with concentration of many inflammatory markers: C-reactive protein (CRP), IL-1, IL-2, IL-5, IL-6, IL-12, IL-13, TNF- α , and Interferon- α [23,24,25,26,27,28,29,30,31]. It was reported that chronic inflammation, which can be observed in asthma, through inflammatory mediators (cytokines) contributes to secondary downregulation of Cyclic adenosine monophosphate Response Element-Binding protein (CREB), Tyrosine Receptor Kinase (TRK) protein and release of Brain-Derived Neurotrophic Factor (BDNF) in frontal

lobes and the limbic system, which results in damage to the hippocampus and a decrease in the level of monoamines in the brain [23,24,25]. Also some drugs, such as theophylline, used in asthma therapy correlate with an increase in the level of cyclic Adenosine MonoPhosphate (cAMP) but do not correlate with an increase in the activity of CREB protein, which might result in excitation of the limbic system and an increased catecholamine release. In consequence, a patient might demonstrate significant clinical changes in the behavior and mood, starting with fits of panic and finishing with suicidal thoughts [32]. According to the monocyte–T-lymphocyte hypothesis the immune system plays a key role in determining someone's personality and temperament, through synthesis, release and uptake of monoamines in the central nervous system [25,33,34]. Decreased or increased expression of synaptic plasticity, neurogenesis, and neuromodulation through basal ganglia, the frontal cortex and activity of the hypothalamus–pituitary axis (HPA) affect the limbic system and influence its various activations [26,27,35,36,37,38]. Thus, the complex of PEN traits and personality (temperament) is conditioned by many factors [39].

The aim of the study was to identify selected personal factors (polymorphic forms of *NR3C1* gene, expression of Transforming Growth Factor β 1 (TGF β 1) Messenger RNA (mRNA), age, sex, body weight), environmental factors (allergy, smoking nicotine, asthma exacerbations induced by external factors) and factors inducing attacks and asthma exacerbations or maintaining them (level of asthma control, level of depression, level of anxiety, level of dyspnea, drugs, allergens, active and passive cigarette smoking) in a complex of personal traits of patients as well as check if and to what extent they influence six temperamental factors and ways of reacting to stress in asthmatic patients.

2. Material and methods

2.1. Ethical approval

The study was approved by the local Ethics Committee (Consent of Research Review Board of the Medical University of Lodz, Łódź, Poland; no. RNN/133/09/KE). At the beginning of the study, the participants were invited to attend voluntarily and prior to enrolment, written informed consent was obtained from every patient.

The study was performed by two investigators who were unaware of the participants' phenotypes. Two hundred and eighty one participants were included in the study. Of this number 122 subjects were healthy volunteers and 159 were asthmatic patients. The mean age in the group of healthy volunteers was 44.45 years and in the group of patients the mean age was 50.40 years. Table 1 presents a detailed characteristic of the study groups.

Table 1

Detailed statistics of the control group and the group of asthmatic patients, bearing information on the size, sex, age and basic spirometric parameters. N – size, % – percentage, iv – independent value, SD – standard deviation. Detailed presentation in the text. The author's own study.

Variable	Asthmatic patients	Healthy subjects
Number of patients	159/281 (56.58%)	122/281 (43.42%)
N/N in total (%)		
Males	46/122 (37.70%)	60/159 (37.74%)
N/N in total (%)		
Patients' age	50.40 \pm 15.70 years	44.45 \pm 16.28 years
Mean \pm SD		
FEV1 iv	72.27 \pm 20.78%	95.03 \pm 13.36%
Mean \pm SD		

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