



ORIGINAL ARTICLE

Personality traits as a possible factor in the inflammatory response in the first depression episode and in recurrent depressive disorders



M. Talarowska^{a,*}, K. Bliźniewska^{a,1}, J. Szemraj^b, M. Kowalczyk^a, P. Gałęcki^a

^a Department of Adult Psychiatry, Medical University of Lodz, Lodz, Poland

^b Department of Medical Biochemistry, Medical University of Lodz, Lodz, Poland

Received 2 September 2017; accepted 14 March 2018

Available online 3 April 2018

KEYWORDS

Depressive disorders;
Personality;
Anxiety;
Neuroticism;
Inflammation

Abstract

Background and objectives: Depressive disorders are linked with an increase in the central and peripheral concentration of many pro-inflammatory cytokines, including mainly tumour necrosis factor α (TNF- α) and interleukins (ILs). The aim of the presented work is to verify whether personality traits predisposing to the occurrence of a depression episode are associated with changes in the peripheral expression of genes for selected cytokines.

Methods: 77 individuals, who met the diagnostic criteria for a depression episode were qualified to take part in the study. Personality traits was measured using selected scales of The Minnesota Multiphasic Personality Inventory (MMPI-2). Expression at the mRNA and protein level for IL-1, IL-6, IL-10, IL-12 and TNF- α were examined.

Results: A significant positive dependence was observed in the entire group examined with reference to the intensity of symptoms on the Welsh anxiety scale and the expression at the mRNA and protein level for the IL-12 gene. Analyses conducted separately for the first depressive episode group and the recurrent depression group revealed significant interrelations between the neurotic triad of the MMPI-2 test and the expression for genes IL-1, IL-10 and IL-12.

Conclusions: (1) The intensity of depression episode symptoms, measured using the neurotic triad and the Welsh anxiety scale for the MMPI-2 test, correlate significantly with the expression at the mRNA and protein level for the genes of pro-inflammatory and anti-inflammatory cytokines. (2) Anxiety as a personality trait may be a significant marker of inflammation during a depression episode.

© 2018 Asociación Universitaria de Zaragoza para el Progreso de la Psiquiatría y la Salud Mental. Published by Elsevier España, S.L.U. All rights reserved.

* Corresponding author.

E-mail address: talarowskamonika@wp.pl (M. Talarowska).

¹ Equivalent share of the authors in the compilation of this paper.

Introduction

Brain diseases belong to the most socially and economically burdening diseases in Europe. Approximately 800 billion euros are spent annually on the fight with the consequences of these diseases.¹ Among all brain diseases, more than 60% of social and economic costs are generated by mental disorders, mainly depressive disorders.²

Annual prevalence of depression in the population of adults oscillates between 6% and 12%. Based on numerous sources, it varies from 5% to even 30% among people over the age of 65.³ According to estimates of the World Health Organisation (WHO), 350 million people around the world present symptoms of depression, while depressive disorders constitute nearly 4.3% of the global burden of all diseases.⁴ Depression often accompanies other somatic diseases as a symptom. It means that approximately 10% of all adults (which corresponds to 100 million cases) show signs of depression during a year. Women suffer from depression twice as often as men.⁵

Both physical and psychological (emotional) stress increase the likelihood of occurrence of mental disorders (including depressive disorders)^{6,7} owing to the action of a series of hormonal and biochemical⁸ as well as epigenetic mechanisms, which has been confirmed in recent times.⁹ With the absence of somatic comorbidity, depressive disorders are linked with an increase in the central and peripheral concentration of many pro-inflammatory and anti-inflammatory cytokines, including mainly tumour necrosis factor α (TNF- α) and interleukins (ILs).¹⁰ Changes in the metabolism of biogenic monoamines, i.e. dopamine, noradrenaline and serotonin in mesencephalic nuclei, are considered potential ways of cytokines' impact on the aetiology of depression.¹¹ Moreover, cytokines lead to excessive secretion of cortisol – directly by means of stimulating the hypothalamic–pituitary–adrenal axis (HPA axis) and indirectly by modifying the sensitivity of glucocorticoid receptors.¹¹

A special role in the aetiology of recurrent depression is assigned to three pro-inflammatory interleukins (IL-1, IL-6 and IL-12) as well as IL-10, which is one of anti-inflammatory interleukins.^{12,13} On the other hand, TNF- α induces excessive reuptake of monoamines, stimulates pathologic hyperactivity of the HPA axis, and increases the activity of indole 2,3-dioxygenase (IDO); hence, reduces substantially the production of serotonin.^{14,15}

In one of our previous papers, we indicated that the pre-morbid personality structure (mainly anxiety as a constant feature of emotional functioning) may have a significant importance for the effectiveness of applied antidepressant pharmacotherapy.¹⁶

The aim of the presented work is to verify whether personality traits predisposing to the occurrence of a depression episode are associated with changes in the peripheral expression of genes for selected pro-inflammatory and anti-inflammatory cytokines: IL-1, IL-6, IL-10, IL-12, and TNF- α .

Material and methods

Material

Seventy-seven individuals, aged 18–64 ($M=47.96$, $SD=11.23$), meeting the diagnostic criteria for a depression episode and recurrent depressive disorders (F32.0–7.32.2, F33.0–F33.8), were qualified to participate in the experiment.^{17,18} All the examined individuals were patients of the Department of Adult Psychiatry of the Medical University of Lodz (the J. Babiński Hospital in Lodz, Poland). All the subjects were examined during their hospitalisation, and no symptoms of concurrent somatic diseases or axis I and II disorders, other than depressive episodes, were diagnosed. Inflammatory or autoimmune disorders, central nervous system traumas, and unwillingness to give informed consent were considered additional exclusion criteria. A case history was obtained from each patient using the standardised Composite International Diagnostic Interview (CIDI)¹⁹ prior to the start of the experiment.

The examined individuals were divided into two groups: the patients diagnosed with the first depression episode (ED-I, $N=25$) and the patients treated due to a recurrent episode of the disease (recurrent depression episodes group, rDE, $N=52$). Statistically significant differences were confirmed in the examined groups in terms of age ($Z=0.117$, $p=0.011$). No significant differences were found in terms of sex ($\chi^2=0.221$, $p=0.641$) between the two groups.

Depression severity was evaluated and classified using the 21-item Hamilton Depression Rating Scale (HDRS).²⁰ Intensity levels of depressive symptoms were measured with the use of the grades presented in the study conducted by Demyttenaere and De Fruyt.²¹ Each patient was examined by the same specialist (the same psychiatrist performed the HDRS test, while a clinical psychologist oversaw conducting the MMPI-2 test).

The individuals taking part in the experiment were native Poles from central Poland (not related with one another). They were selected at random without replacement sampling. Participation in the study was voluntary; all personal data and results were kept confidential. Before making a decision to participate in the experiment, the patients were informed about its purpose, assured of the voluntary nature of the experiment, and guaranteed that their personal data would be kept in secret. Written informed consent was given in accordance with the study protocol, approved by the Bioethical Committee of the Medical University of Lodz (No. RNN/272/15/KE).

Methods

The Minnesota Multiphasic Personality Inventory (MMPI-2)

The Polish version of the MMPI-2 by S. Hathaway and J. McKinley, adapted by T. Kucharski was used to evaluate the personality structure of the examined individuals.

Download English Version:

<https://daneshyari.com/en/article/8926983>

Download Persian Version:

<https://daneshyari.com/article/8926983>

[Daneshyari.com](https://daneshyari.com)