Type A Behavior Pattern, Impulsiveness, Risk Propensity, and Empathy as Predictors of Dyspnea and Number of Infections in Men with Chronic Obstructive Pulmonary Disease: A Cross-Sectional Study

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Background: Stress and psychological factors can induce dyspnea in patients with chronic obstructive pulmonary disease (COPD). The aim of this study was to assess selected elements of the clinical presentation of COPD in the context of the severity of type A pattern of behavior, impulsiveness, and tendency for empathy.

Material/Methods: This was a cross-sectional study. The study group consisted of 179 men with COPD and the control group consisted of 31 healthy male smokers. In all patients, the number of infectious exacerbations over the past year, the result on the dyspnea scale (MRC), and the FEV1-to- predicted FEV1 ratio was assessed. The A pattern of behavior was measured using the Type A scale. To measure impulsivity, risk propensity, and empathy, the IVE impulsivity questionnaire was used.

Results: An increase in the number of infectious exacerbations was associated with an increased score on the Type A scale, an increase in risk propensity, and a decrease in impulsivity score. Increased severity of dyspnea was associated with an increase in Type A behavior pattern score and an increase in the risk propensity score.

Conclusions: Type A behavior pattern and risk propensity are independent predictors of the number of infections in the last year and of the subjective severity of dyspnea among men with COPD and healthy male smokers.

MeSH Keywords: Empathy • Impulsive Behavior • Pulmonary Disease, Chronic Obstructive

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Background

Epidemiology and clinical characteristics of chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) develops in about 15% of smokers [1]. It is estimated that around 3 million people die because of COPD every year. The mediators produced by inflammatory cells, mainly neutrophils, as well as irreversible bronchoconstriction and bronchial hypertrophy, emphysema, and accompanying dynamic pulmonary hyperinflation cause severe dyspnea [2]. Up to 40% of patients with COPD who visit primary care have moderate-to-severe breathlessness [3].

Factors affecting quality of life in COPD patients

Comorbidity of COPD, especially respiratory failure, pulmonary hypertension, and right heart failure, additionally deteriorate the quality of life in the course of COPD [4,5].

Stress and psychological factors may induce attacks of dyspnea in patients with COPD and negatively affect quality of life. On the other hand, the disease itself, with chronic dyspnea, is a very strong stressor [5,6]. Although several biological markers of COPD exacerbation have been recognized, the relationship between the experienced level of stress and the variables that characterize the clinical picture of the disease is not fully understood [7]. Both the effect of a severe somatic disease on the quality of life and the influence of psychological variables on the clinical presentation of the disease are taken into account [6,8]. The experienced stress level is a modulator of dyspnea in the course of COPD, which means that the disease is to some little extent a psychosomatic illness. Thus, the temperamental and personality variables, not only genetic factors, have a certain effect on the clinical picture of COPD [9,10]. On the other hand, persistent dyspnea, infectious exacerbations, feelings of danger and anxiety associated with dyspnea attacks in the course of exacerbations, the use of drugs that act on central nervous system receptors (e.g., theophylline) may also to some extent influence the development of temperament and personality in the course of COPD [11]. Additionally, respiratory insufficiency and chronic hypoxia of the central nervous system can affect the cognitive and emotional processes in COPD patients [9,11].

Type A behavior pattern

The inborn temperament traits and strong influence of social learning develop the style of behavior. The style of behavior is a group of human behavioral traits conditioned by the product of the biological, temperamental, and social learning-related factors [12]. One of the known unhealthy behavior styles is the so-called type A behavior pattern, which predisposes to the development of cardiovascular diseases, especially ischemic heart disease [12,13]. This construct was developed by Friedman and Rosenman [14]. It is defined as an unhealthy lifestyle and behavior that arises from the interaction of personality factors, and is a result of the imposition of social learning processes on the temperamental traits. The type A pattern of behavior means a constant struggle to achieve as many goals as possible in a short time, and its basic elements are anger, rivalry, and hostility [12,13]. Initially, type A behavior pattern was associated with ischemic heart disease, and later it was found to be a non-specific risk factor for many other diseases [12,14].

Study rationale

The question arises whether the tendency for aggression and irritation typical of type A behavior pattern in COPD patients may be relevant to the clinical presentation of the disease (12). This question is important because nicotine dependence is also a common risk factor for coronary heart disease and COPD. Impulsivity, risk propensity, and empathy are important psychological variables moderating the response to stress [15]. It should be assumed that psychological stress-modifying stress variables may be correlated with clinical elements of COPD and with the behavioral pattern.

The aim of the present study was to assess the risk level of COPD and selected aspects of its clinical picture in the context of the severity of type A behavior pattern, impulsiveness, and tendency for empathy.

Material and Methods

Study participants

The study sample was selected from patients in a pulmonary clinical ward and a clinic in a tertiary level hospital. The group qualified for the study consisted of 179 men aged 45–70 years diagnosed with COPD, all of whom were former or current smokers. The control group consisted of 43 healthy male smokers aged 45–70 years.

Ethical considerations

This study was conducted in accordance with the Declaration of Helsinki. The research protocol was approved by the Bioethics Committee of the Medical University of Lodz (RNN/130/14/KE, 15th July 2014).

Inclusion/exclusion criteria

Patients with respiratory failure (i.e., with hypoxemia, hypercapnia, or both) were excluded *ad hoc* because of possible

Table 1. Levels of the analyzed variables in the patient groups.

	Control (N=43)		Group A (N=43)		Group B (N=33)		Group C (N=28)		Group D (N=32)		F	p
	M	SD	M	SD	M	SD	M	SD	M	SD		
Age	55.88	6.92	57.19	7.20	56.00	6.89	57.04	7.42	57.50	7.80	0.377	0.825
Type A behavior pattern	0.40	0.14	0.42	0.12	0.62***	0.13	0.42†	0.12	0.61*,**,##	0.13	26.924	<0.001
Empathy	11.02	3.55	9.56	2.21	8.39*	2.41	9.96	3.19	9.22	2.54	3.873	<0.01
Impulsivity	11.33	2.32	10.28	2.58	14.33***	2.75	10.00#	2.54	14.06*,**,##	2.50	22.399	<0.001
Propensity to risk	9.21	2.09	9.33	2.18	11.09***	2.30	10.11	1.77	13.06*,**,#,##	1.83	20.906	<0.001

Control – smokers without COPD diagnosis, M – mean, SD – standard deviation, F – F statistics, p –probability in the F-test, or Welch test, depending on the fulfilment of assumption concerning homogeneity of variance between groups; * p<0.01 *vs.* Control; ** p<0.001 *vs.* Group A; # p<0.001 *vs.* Group B; ## p<0.001 *vs.* Group C.

cognitive dysfunctions [16]. Patients with cancer, systemic connective tissue diseases, past acute coronary episode during the last 6 months, and those experiencing intensive stress due to mourning, surgery, or job loss, were excluded from the study.

Study procedures

In all patients, the number of infectious exacerbations over the past year, the actual FEV1-to-predicted FEV1 percentage ratio, and the severity of clinical symptoms were assessed. The severity of dyspnea was assessed using the modified Medical Research Council dyspnea scale, scored from 0 to 4, in which a higher result indicates greater severity of clinical symptoms (mMRC). Based on these data, the patients were divided into A, B, C, and D COPD groups, as suggested by the Global Initiative for Chronic Obstructive Lung Disease [17,18]:

- Group A: 0 or 1 exacerbation and mMRC 0–1 (low risk, few symptoms);
- Group B: 0 or 1 exacerbation and mMRC ≥2 (low risk, many symptoms);
- Group C: ≥2 exacerbations and mMRC 0–1 (high risk, few symptoms);
- Group D: ≥2 exacerbations and mMRC ≥2 (high risk, many symptoms).

The pattern of behavior (A) was measured using the Type A scale standardized by Zygfryd Juczyński [19]. This is a unidimensional questionnaire in which the result approximating 1 corresponds to pattern A, whereas a near-zero result corresponds to behavior pattern B. To measure impulsivity, risk propensity, and empathy, the IVE impulsivity questionnaire was used [15]. This questionnaire measures 3 personality variables: empathy, impulsiveness, and venturesomeness (propensity to risk).

Statistical analysis

The statistical analysis was performed using SPSS Statistics 24 PL software (IBM, USA). Mean values and standard deviations were used to characterize the continuous variables. Due to the sample size (N=179), the boundary theorem was used and the hypothesis of the normal distribution of continuous variables was abandoned. The hypothesis of homogeneity of variance of continuous variables in the selected groups was verified by Levene's test. Comparisons between more than 2 groups were made by analysis of variance (F-test) with the post hoc Tukey test. If the assumption of homogeneity of variance in the analyzed groups was not fulfilled, Welch's test was used with the post hoc T3 Dunnett's test. The relationship between 2 continuous variables was assessed using Pearson's correlation coefficient. Linear regression models were constructed for prediction of the annual number of infectious exacerbations and for prediction of the severity of dyspnea. An analysis of residuals was performed and the models were rated for meeting the criteria of homoscedasticity and independence between observations (Durbin-Watson test). Collinearity was assessed using the tolerance coefficient. The results are expressed as the linear directional regression parameters B with standard error (SE). The effect of the individual explanatory variables was evaluated using the standardized Beta parameter. The level of statistical significance was α=0.05.

Results

Comparisons between the COPD risk groups

The studied groups did not differ significantly in demographic variables, as presented in Table 1.
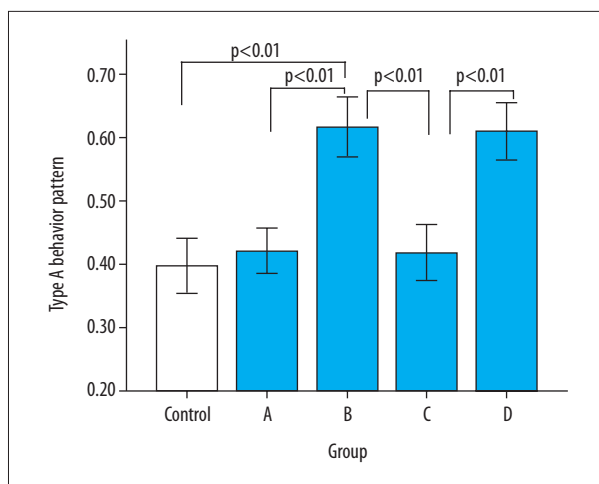
Figure 1. Comparison of type A behavior pattern scores among the ABCD COPD risk groups. Presented as mean values (bars) with 95% confidence intervals (whiskers). p – probabilities in the post hoc tests.

In COPD risk group B, the average score on the Type A scale was significantly higher than in the control group and group A. Group C was characterized by a lower average score of type A behavior pattern than in group B. In group D, the result was significantly higher than in the control group and groups A and C (Table 1, Figure 1).

Intensity of the empathy dimension was lower in COPD risk group B than in the control group (Table 1). People from COPD risk group B were characterized by higher impulsivity than were healthy smokers, and those from group D, who had higher impulsivity than healthy smokers and patients from COPD risk group A. The severity of the Risk Propensity dimension was higher in group B and in group D as compared to healthy smokers from COPD risk group A.

Correlations

The correlations between the tested variables were found to be interesting. The number of infections in the past year correlated positively with the score on the dyspnea scale, Type A Pattern of behavior, impulsivity and propensity to risk (Table 2). The results in the MRC Dyspnea Scale also correlated positively with pattern A, impulsivity, and risk propensity, and negatively with empathy (Table 2). Behavior pattern A was correlated positively with impulsivity and risk propensity (Figures 2, 3), whereas empathy was correlated negatively with impulsivity and positively with propensity to risk, and impulsivity was positively correlated with propensity to risk. It is noteworthy that in the case of Type A pattern of behavior, impulsivity, and risk propensity, the values of Pearson correlation coefficient for dyspnea were about twice as high as those for the number of infections (Table 2).

Predicting the number of infectious exacerbations

In the linear regression model, the increase in infectious exacerbations in the past year was associated with an increase in the type A pattern of behavior scale result, an increase in the score on the IVE propensity to risk scale, and a decrease in IVE impulsivity. The strongest effect among the aforementioned relationships was associated with the type A behavioral pattern. There was no correlation between the number of infections and age or the IVE empathy scale score.

Predicting the severity of dyspnea

The increase in dyspnea was associated with an increase in the type A pattern of behavior score and an increase in the score on the IVE propensity to risk scale. The strongest effect among the aforementioned relationships was associated with the type A behavioral pattern. There was no correlation between the severity of dyspnea symptoms and age or IVE impulsivity and empathy results (Table 3).

Table 2. Pearson correlation coefficients between the selected continuous variables in the analyzed sample.

	Age	mMRC	Infections	Type A behavior pattern	Empathy	Impulsivity
mMRC	0.045					
Infections	0.009	0.458**				
Type A behavior pattern	−0.013	0.700**	0.399**			
Empathy	0.034	−0.187*	−0.081	−0.119		
Impulsivity	0.015	0.514**	0.152*	0.595**	−0.241**	
Propensity to risk	0.056	0.510**	0.369**	0.392**	−0.216**	0.532**

mMRC – modified Medical Research Council dyspnea severity scale; Infections – number of infectious exacerbations in the year preceding the study; * p<0.01 (two-tailed); ** p<0.05 (two-tailed).
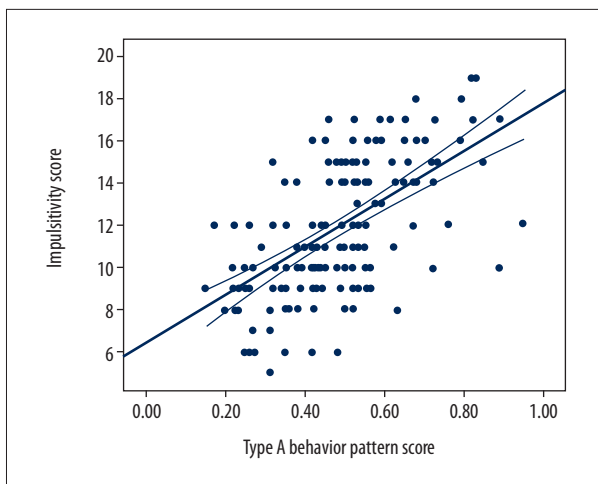
Figure 2. Scatter plot showing the correlation between the type A behavior score and IVE impulsivity score (N=179, r=0.595, p<0.05).
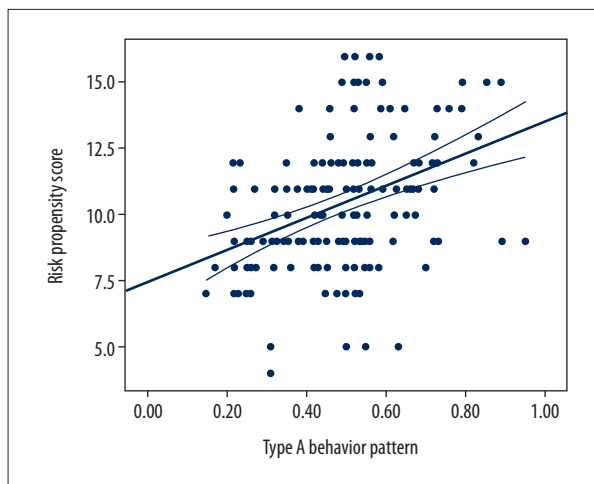

Figure 3. Scatter plot showing the correlation between the type A behavior score and IVE risk propensity score (N=179, r=0.392, p<0.05).

Table 3. Parameters of the linear regression models predicting the number of exacerbations in the year preceding the study and the severity of dyspnea in the analyzed sample. Presented as parameter B with the standard error (SE) and the standardized beta parameter (a measure of the effect size).

	Number of infectious exacerbations				
	r^2=0.511, df=5, F=12.200, p<0.001				
	B	SE	Beta (β)	t	p
(Constant)	−0.661	0.725		−0.913	0.363
Age	0.001	0.010	0.000	0.002	0.999
Type A behavior pattern	3.030	0.567	0.438	5.340	<0.001
Empathy	−0.010	0.025	−0.026	−0.376	0.708
Impulsivity	−0.108	0.032	−0.301	−3.350	0.001
Propensity to risk	0.156	0.035	0.352	4.492	<0.001
	Dyspnea severity (mMRC)				
	r^2=0.749, df=5, F=44.251, p<0.001				
	B	SE	Beta (β)	t	p
(Constant)	−2.431	0.607		−4.004	<0.001
Age	0.007	0.008	0.040	0.793	0.429
Type A behavior pattern	4.400	0.475	0.584	9.254	<0.001
Empathy	−0.025	0.021	−0.060	−1.152	0.251
Impulsivity	0.005	0.027	0.013	0.189	0.850
Propensity to risk	0.125	0.029	0.259	4.291	<0.001

r^2 – determination coefficient; df – number of degrees of freedom; t – student t statistics; p – probability in the test; mMRC – modified Medical Research Council.

Study limitations

A shortcoming of our research is our failure to include women, as well as the value of the flow volume loop measurement results and other clinical parameters characterizing the clinical presentation of chronic obstructive pulmonary disease, primarily the CAT scale. On the other hand, the purpose of the study was to answer the question of whether personality variables are predictors of dyspnea and the number of infections in men who have COPD. It would also be worthwhile to compare the obtained results with a control group of healthy non-smokers, which was lacking in our study. On the other hand, in the test adaptation manual in Polish, there are norms for the general population, which makes it possible to compare our results to general population standards. Also, it would be interesting to analyze the 3 factors identified in the type A behavior pattern construct by its creators: factor H (involvement in a specific activity), factor J (the involvement of the subject in professional work), and factor S (the measure of behavior dynamics) [12,20,21]. The type A scale developed by Juczyński does not differentiate these factors, considering only the tested subject's position on the continuum between behavior pattern A and behavior pattern B. This test, in our opinion, is less useful than the original 3-factor tool developed by the originators of the type A behavior pattern concept [22].

Discussion

Importance of the study

COPD risk levels B and D are characterized by high severity of dyspnea, which means, at least theoretically, lower quality of life. As clearly demonstrated in the present study, the patients from B and D risk groups had higher scores in type A pattern of behavior, impulsivity, and risk propensity dimensions compared to those from the control group and the type A behavior risk group. The question arises of whether dyspnea is due to the personality predisposition or to the personality that affects the feeling of breathlessness. Nowobiliski et al. demonstrated that anxiety as a personality trait is a predictor of dyspnea in patients with bronchial asthma. It was also shown to affect disease severity in men but not in women [23]. In addition to anxiety, the predictors of severity of dyspnea included body mass index and value of forced expiratory volume in one second (FEV 1) [24,25]. In our own research, we have shown that the dimensions of temperament according to the concept developed in Poland by Strelau (in the regulatory temperament theory), and especially the low activity dimension, are associated with the severity of asthma and shortness of breath [24,25]. The personality structure, through the limbic system and the autonomic nervous system, influences the reactivity of the internal organs, including bronchospasm [26].

On the other hand, central nervous system activity may be associated with the profile of secreted mediators and hormones, affecting both the sensation of dyspnea and bronchospasm, which are measurable by spirometry. It is known that proinflammatory cytokines favor the development of depression, which can be measured using psychometric tools [27]. Topp et al. studied personality according to the Big Five model, and the severity of symptoms in COPD patients using the COPD Assessment Test (CAT) psychometric tool. The authors demonstrated that neuroticism, agreeableness, conscientiousness, depression, and anxiety were correlated with CAT scores in COPD patients [28]. Benzo et al., in turn, demonstrated that high emotional intelligence is associated with better functioning in COPD patients [29]. Interestingly, in our work, empathy was lower than in the control group only in the group of people with B risk level (i.e., those with many clinical symptoms but a small number of infections and with previously good spirometry results). Thus, risk group B includes subjects with "good" physiological parameters and "bad" subjective well-being. This "hypochondriac" profile of group B indicates a self-focusing attitude and experiencing subjectively severe symptoms, which may be associated with low empathy and a certain "narcissistic" trait in that group.

Clinical implications

Type A pattern of behavior was significantly higher in the B and D risk groups compared to the controls and risk group A (i.e., in those patients who had a large number of subjective complaints). Once again, that dimension turned out to be a non-specific marker of clinical symptoms in somatic diseases [12,13]. Persons with type A pattern of behavior tend to be competitive, achievement-oriented, impatient, and speak quickly. They respond to misfortunes with stress and somatic symptoms, often including severe dyspnea. This is confirmed indirectly by our results showing that highly symptomatic COPD risk levels are characterized by more severe type A pattern of behavior than in the control group and COPD risk group A. Our results clearly indicate that type A pattern of behavior can no longer be regarded as a specific marker of ischemic heart disease [12,13], but rather is a risk factor for psychosomatic disorders and the severity of subjective symptoms in somatic (partially psychosomatic) conditions, including ischemic heart disease and obstructive diseases such as asthma and COPD [11,23,24].

Therefore, it is not surprising that the severity of dyspnea correlates positively with type A pattern of behavior, impulsiveness, and propensity to risk. The tendency for violent actions can induce dyspnea by both psychogenic and neurophysiological mechanisms. For example, cold air hyperventilation associated with experienced stress, in combination with bronchial hyperreactivity characteristic of obstructive diseases, may

induce bronchospasm [30,31]. Such a contraction is associated with the subjective feeling of breathlessness experienced by the patient. The negative correlation between the severity of dyspnea and empathy is probably related to the fact that people with low level of empathy do not receive support from their social microenvironment. This can mean a sense of loneliness and concentration on own ailments [32]. The number of infections is also positively correlated with type A pattern of behavior, impulsiveness, and propensity to risk. These dimensions, on the one hand, signify a tendency for conflicting relationships with people, which may be associated with increased susceptibility to infections. People with COPD are particularly susceptible to viral and bacterial infections due to epithelial barrier damage and chronic bronchitis [33]. On the other hand, it is known that people with COPD tend to have a small network of social relationships. Perhaps it should be investigated whether the discussed dimensions and the number of infections in the past year correlate, for example, with the number of pack-years smoked or other clinical variables.

The constructed linear regression models are matched to the empirical data (p<0.05 in the F tests). It should also be noted that both in the case of prediction of dyspnea on the mMRC scale and of the number of infectious exacerbations in the past year, the coefficient of determination R^2 is higher than 0.4. This means that from the statistical point of view, these models are suitable for predicting the severity of dyspnea and the number of infections based on the age and scores on the scales of Type A behavior pattern, impulsivity, risk propensity, and empathy [34]. Contrary to expectations, the increase in impulsivity was demonstrated to be associated with fewer infectious exacerbations, irrespective of type A pattern of behavior and other IVE dimensions. This means that the psychological construct of impulsivity cannot be equated with type A behavior pattern, although the definition of the latter includes the elements of impulsive behaviors. The results also indicate that the impulsivity psychological construct is constructed differently than the constructs of type A behavior pattern or risk propensity. It also has a lower predictive validity for prediction of dyspnea and onset of infection in COPD than the constructs of type A behavior pattern and risk propensity (Table 3).

The relationship between the investigated psychological variables

We found that impulsiveness, propensity to risk, and type A behavior pattern are positively correlated with each another, indicating that they probably have a single underlying factor. These results correspond to the general population standards in Poland. It is notable that the mean impulsivity results of healthy smokers were within the 6th of standard ten range compared to the population used to normalize IVE test results, propensity to risk within the 7th of standard ten range, and empathy around the

5th of standard ten. In view of the above, healthy smokers in our group were characterized by higher impulsivity and higher risk propensity than the population on which the test results were normalized [15]. This gives a certain psychological profile of healthy smokers as more impulsive and risk-taking than healthy non-smokers of similar age. As shown in a study conducted by Choi et al., smokers are characterized by a higher level of neuroticism dimension compared to the control group and the lower level of conscientiousness dimension according to the Big Five personality model [35]. It is therefore possible to develop a psychological profile of a smoker that considers multiple variables [1,4,5]. The psychological profile of the smoker may potentially enable the selection of appropriate interventions in cognitive-behavioral therapy to quit smoking. The lack of standards for type A behavior pattern in the standardization manual makes it impossible to compare our results to the results obtained for the general population in Poland [19]. The average score of 0.4 for healthy smokers (Table 1) suggests a type intermediate between type A and B patterns of behavior in that group. Possibly, it is the smokers with pattern A who develop ischemic heart disease and COPD, which we have demonstrated with certainty for risk groups B and D of COPD patients (Table 1).

Conclusions

In this study, we assessed 179 men with COPD. The control group consisted of 43 healthy male smokers. In all patients, the number of infectious exacerbations over the past year, the results on the dyspnea scale (MRC) and the FEV1-to-predicted FEV1 ratio were assessed. The A pattern of behavior was measured using the Type A scale. To measure impulsivity, risk propensity, and empathy, we used the IVE impulsivity questionnaire

An increase in the number of infectious exacerbations was associated with an increased score on the Type A scale, an increase in risk propensity, and a decrease in impulsivity scores. Increased severity of dyspnea was associated with an increase in Type A behavior pattern score and an increase in risk propensity score.

All things considered, the following conclusions were reached:
1. COPD risk groups according to GOLD differ not only in terms of the severity of symptoms and the risk of exacerbations, but also, among others, in the levels of impulsivity, propensity to risk, empathy and Type A behavior pattern.
2. Type A behavior pattern and risk propensity are independent predictors of the number of infections in the past year and of the subjective severity of dyspnea among men with COPD and healthy male smokers.

Conflict of Interest

None.

References:

1. Kim DK, Hersh CP, Washko GR et al: Epidemiology, radiology, and genetics of nicotine dependence in COPD. Respir Res, 2011; 12: 9

2. Barnes PJ: Cellular and molecular mechanisms of chronic obstructive pulmonary disease. Clin Chest Med, 2014; 35(1): 71–86

3. Müllerová H, Lu C, Li H, Tabberer M: Prevalence and burden of breathlessness in patients with chronic obstructive pulmonary disease managed in primary care. PLoS One, 2014; 9(1): 70–80

4. Margaritopoulos GA, Antoniou KM, Wells AU: Comorbidities in interstitial lung diseases. Eur Respir Rev, 2017; 26(143): pii: 160027

5. Dury R: COPD and emotional distress: not always noticed and therefore untreated. Br J Community Nurs, 2016; 21(3): 138–41

6. Clark AJ, Strandberg-Larsen K, Masters Pedersen JL et al: Psychosocial risk factors for hospitalisation and death from chronic obstructive pulmonary disease: A prospective cohort study. Chronic Obstr Pulm Dis, 2015; 12(2): 190–98

7. Feng W, Wu X, Li S et al: Association of serum Galectin-3 with the acute exacerbation of chronic obstructive pulmonary disease. Med Sci Monit, 2017; 23: 4612–18

8. Chen Q, Wu C, Gao Y et al: A clinical study on the role of psychosomatic therapy in evaluation and treatment of patients with chronic obstructive pulmonary disease complicated with anxiety-depression disorder. Int J Clin Exp Med, 2015; 8(9): 16613–19

9. Kahraman H, Orhan FO, Sucakli MH et al: Temperament and character profiles of male COPD patients. J Thorac Dis, 2013; 5(4): 406–13

10. Du Y, Xue Y, Xiao W: Association of IREB2 gene rs2568494 polymorphism with risk of chronic obstructive pulmonary disease: A meta-analysis. Med Sci Monit, 2016; 22: 177–82

11. Tabała K, Wrzes M, Patryk S, Józef K: Personality traits, level of anxiety and styles of coping with stress in people with asthma and chronic obstructive pulmonary disease – a comparative analysis. Psychiatr Pol, 2016; 50(6): 1167–80

12. Rosenham RH: Do environmental effects on human emotions cause cardiovascular disorders? Acta Physiol Scand Suppl, 1997; 640: 133–36

13. Richards SH, Anderson L, Jenkinson CE et al: Psychological interventions for coronary heart disease. Cochrane Database Syst Rev, 2017; (4): CD002902

14. Friedman M, Ulmer D: Treating type A behavior and your heart. London, UK: Michael Joseph, 1985

15. Jaworowska A: IVE impulsivity questionnaire. Impulsiveness. Venturesomeness. Empathy. Polish Standardization. Warsaw: Psychological Tests Laboratory of the Polish Psychological Association, 2011

16. Andrianopoulos V, Gloeckl R, Vogiatzis I, Kenn K: Cognitive impairment in COPD: should cognitive evaluation be part of respiratory assessment? Breathe, 2017; 13(1): e1–9

17. Global Initiative for Chronic Obstructive Lung Disease. Pocket Guide to COPD Diagnosis, Management and Prevention. A Guide for Health Care Professionals, 2017

18. Gupta N, Pinto LM, Morogan A, Bourbeau J: The COPD assessment test: A systematic review. Eur Respir J, 2014; 44(4): 873–84

19. Juczyński Z: Measurement tools in health promotion and psychology. Warsaw: Psychological Tests Laboratory of the Polish Psychological Association, 2009

20. Witusik A, Pietras T: Personality-related and behavioral factors in clinical presentation of bronchial asthma. Piotrków Trybunalski: Naukowe Wydawnictwo Piotrkowskie, 2011

21. Friedman HS, Hall JA, Harris MJ: Type A behavior, nonverbal expressive style, and health. J Pers Soc Psychol, 1985; 48: 1299–315

22. Friedman HS, Kern ML: Personality, well-being, and health. Annu Rev Psychol, 2014; 65(1): 719–42

23. Nowobilski R, Furgał M, Czyż P et al: Psychopathology and personality factors modify the perception of dyspnea in asthmatics. J Asthma, 2007; 44(3): 203–7

24. Panek M, Pietras T, Witusik A et al: 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. Physiol Behav, 2015; 149: 269–78

25. Panek M, Jonakowski M, Zioło J et al: Identification of relationships between interleukin 15 mRNA and brain-derived neurotrophic factor II mRNA levels with formal components of temperament in asthmatic patients. Mol Neurobiol, 2017; 54(3): 1733–44

26. Stahl SM: Stahl's essential psychopharmacology. Neuroscientific Basis and Practical Applications. 4th ed. Cambridge, United Kingdom: Cambridge University Press, 2013

27. Schmidt FM, Schröder T, Kirkby KC et al: Pro- and anti-inflammatory cytokines, but not CRP, are inversely correlated with severity and symptoms of major depression. Psychiatry Res, 2017; 239: 85–91

28. Topp M, Vestbo J, Mortensen EL: Personality traits and mental symptoms are associated with impact of chronic obstructive pulmonary disease on patients' daily life. J Chronic Obstr Pulm Dis, 2016; 13(6): 773–78

29. Benzo RP, Kirsch JL, Dulohery MM, Abascal-Bolado B: Emotional intelligence: A novel outcome associated with wellbeing and self-management in chronic obstructive pulmonary disease. Ann Am Thorac Soc, 2016; 13(1): 10–16

30. Nair P, Martin JG, Cockcroft DC et al: Airway Hyperresponsiveness in asthma: Measurement and clinical relevance. J Allergy Clin Immunol Pract, 2017; 5(3): 649–659.e2

31. Page C, O'Shaughnessy B, Barnes P: Pathogenesis of COPD and asthma BT – pharmacology and therapeutics of asthma and COPD. In: Page CP, Barnes PJ (eds.), Cham: Springer International Publishing, 2017; 1–21

32. Miller JG: Physiological mechanisms of prosociality. Curr Opin Psychol, 2018; 20(Suppl. C): 50–54

33. Leung JM, Tiew PY, Mac Aogáin M et al: The role of acute and chronic respiratory colonization and infections in the pathogenesis of COPD. Respirology, 2017; 22(4): 634–50

34. Szymczak W: Fundamentals of statistics for psychologists. Academic handbook Second edition (revised). Warszawa: Wydawnictwo Difin, 2010

35. Choi J-S, Payne TJ, Ma JZ, Li MD: Relationship between personality traits and nicotine dependence in male and female smokers of African-American and European-American samples. Front Psychiatry, 2017;8: 122