IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 6 Ver. 2 (June. 2018), PP 05-11
www.iosrjournals.org

Smoked young adults parents smoking habits effect on respiratory parameters

Özlem Şengören Dikiş[1], Seyhan Dülger[1], Halide Kaya[1], Dilber Durmaz Yılmaz[1], Mehmet Karadağ[2], Tekin Yıldız[1], Bekir Kaplan[3]

[1.] *Health Sciences University Bursa Yuksek Ihtisas Training and Research Hospital, Department of Pulmonary Diseases, Bursa, Turkey*
[2.] *Uludag University Faculty of Medicine,Department of Pulmonary Diseases, Bursa, Turkey*
[3.] *Johns Hopkins University Bloomberg School of Public Health Environmental Health Science*
Corresponding Author: Özlem Şengören Dikiş

Objective: *We have aimed to emphasize the importance of querying the outpatients at the polyclinics on family history of cigarette smoking and exposure to cigarette smoke at childhood and/or adolescence in the home environment as well as determining their current smoking habit.****Methods:*** *The study included 72 cigarette smoking young adults within the age range of 18-40 years. Demographic details, personal and parental history of cigarette smoking were noted; the Medical Research Council (MRC) dyspnea scores were calculated.****Results:*** *Mean age of the participants was 31 years, with 63.9% being males. Daily mean cigarette smoking was 16 cigarettes/day; the mean duration of smoking was 11packs/year; and mothers of 5.6% and fathers 40.3% of the participants were smokers .At least one parent of the participants with an MRC score of 2 were smokers (****p=0,001****). While the mean FEV1 for the participants with nonsmoker parents was 3.3lt, this was 2.9lt for the participants with at least one cigarette smoking parent, and the difference was significant (****p=0,021****).****Conclusion:****Young adult cigarette smokers who had thus been exposed to cigarette smoke in early life should be accepted as a ''****high risk group****'' for COPD development and should be put under follow up control for respiratory function capacity.*

Keywords: *parents,smoker, young adult,COPD*

Date of Submission: 23-05-2018 Date of acceptance: 05-06-2018

I. Introduction

Chronic respiratory diseases and especially asthma and chronic obstructive pulmonary disease (COPD) are conditions to be investigated at any stage of human life in order to enable preventive approaches before the overt presentation of disease. Of these, COPD is particularly complicated in not being observed is some continually smoking members of the population *(*1,2,3). This suggests that the possible effects of genetic, environmental and job related factors are not yet fully identified. Morphogenesis of the lungs starts at the 5th week of gestation and progresses through 5 different stages. The last stage of alveolarisation was believed to progress until postnatal 2-4 years of age (4). However, recent studies with hyperpolarised helium-3 magnetic resonance imaging have demonstrated that alveolarisation continues through childhood, adolescence and even early adulthood (5). Hence, exposure of the individual to environmental and job related smoking , especially smoking by parents in the home ebvironment adversely affects lung development and respiratory functions of infants, children, adolescents and young adults (6,7,8,9) . Although there are many paediatric studies on the subject , only very few investigations have been made on the young adult population of 18-40 years of age (10,11,12,13,14) . It has long been known that passive exposure to cigarette smoke contributes to the development of COPD (15).

We have therefore aimed to demonstrate pulmonary function loss in individuals passively exposed to cigarette smoke at childhood as well as being passively exposed at the work place and/or by active smoking.

II. Materials and Methods

This study was designed as a prospective, observational, analytical and cross sectional inveatigation; and it was carried out with 86 young adults of 18-40 years of age meeting the inclusion criteria among a total of 345 individuals who consulted Bursa Postgraduate Training and Research Hospital Pulmonary Diseases polyclinic between January and July 2014. After recording the demographic details, the participants were queried on their parents' and their personal smoking habits.

Inclusion criteria : Being 1) 18- 40 years of age , 2) An active cigarette smoker

Exclusion criteria: Having 1) Asthma, 2) Seasonal rhinitis, 3) Acute or chronic sinusitis, 4) Upper respiratory tract infections, 5) Acute or chronic bronchitis, 7) Metabolic disorders, 8) Cardiovascular system diseases and 9) Using antidepressants.
All participants provided written informed consent and agreed to a protocol to complete a questionnaire on demographic characteristics, a questionnaire on exposure to nicotine and cigarette smoke, spirometric tests, and provide a blood specimen. The study was approved by the institutional ethics committee.

Evaluation Parameters
1.Fagerström test for nicotine dependence (FTND)
The level of nicotine dependence was measured by the Fagerström Test for Nicotine Dependence (FTND) (16) which is the most widely used measure of nicotine dependence, comprising six questions about daily cigarette consumption. It assesses the extent of cigarette smoking behaviour and the validity and reliability of the Turkish version was demosntrated by Uysal et al. in 2004 (17).
2.Pulmonary Function Test (PFT)
Spirometry was carried out by the same experienced nurse using the spirometer "Spirolab II" MIR 010, a product of Medical International Research (MIR). Measurements were in accordance with the guidelines of American Thoracic Society (ATS) and European Respiratory Society (ERS) (18). Pulmonary function parameters, such as forced expiratory volume in 1 second (FEV1), FEV1 %pred, forced vital capacity (FVC), and FEV1 /FVC%, were measured. at the baseline and post-inhaling bronchodilator . Bronchial dilation test was also included in the investigative procedure.
3.Modified Medical Research Council (MRC) Dyspnea Scale
The MRC dyspnea scale is a 5-item questionnaire based on diverse physical activities creating dyspnea perception and asks the patients to mark the activities that cause dyspnea in them (19).
4.Questions
Environmental exposure to parental tobacco smoking was defined as a positive response to the question: ''Did your father or mother ever smoke regularly during your childhood?'' Their parents were categorised as smokers in their childhood period, or as nonsmokers.

Queries were answered on cigarette smoking at the work place.

Cumulative tobacco consumption was calculated in pack-years, defined as 20 cigarettes/d/yr or equivalent.

Frequent respiratory infections during childhood were defined as a positive response to the question:''Have you has frequent respiratory infections during childhood?". Patients with a positive response were excluded from the study.

For the purposes of this study, 'definite asthma' was defined as positive responses to all three of the following questions (ATS questions 20A, 20B, and 20C3): (1) 'Have you ever had asthma?',(2) 'Do you still have it?', and (3) 'Was it confirmed by a doctor?' (20).

All questions had to be answered with either 'yes' or 'no', not with 'as far as I know'.

5. Body Mass Index (BMI)
Body mass index was calculated as measured weight (kilograms) divided by measured height squared (meter squared) (21).
6. Inflammatory Biomarkers
Plasma levels of high-sensitivity CRP, ferritine, and whole blood platelet count were measured using standard hospital assays at a central laboratory.

Statistical Analysis
Statistical analyses were performed using SPSS® for Windows®, version 18.0 (SPSS Inc., Chicago, IL). Descriptive statistics were reported as proportions, the median, the mean and standard deviation . Distribution of data was assessed by using one-sample Kolmogorov-Smirnov test. Differences between categorical variables were tested with Chi Square test. Student's t-test for continuous variables displaying normal distribution and Mann-Whitney U test for continuous variables not displaying normal distribution were used in order
to compare continuous variables in two groups. Kruskal Wallis test was used for comparing variables in 3 groups; and $p<0.05$ was accepted to represent statistical significance.

III. Results

The study included a total of 86 young adult participants comprising 63.9% males and 36.1% females with a mean age of 31 (18-40) years, and a BMI of under 25 kg/m^2 in 56.9% of the participants. All of the participants were active smokers, consuming a mean of 16 cigarettes per day, with a mean duration of 11 packages/year cigarette use. Mothers of 5.6% and fathers of 40.3% of the participants were reported to be cigarette users. None of the participants had been exposed to cigarette smoke at the work place (Table 1). The MRC value was 0 for 48.6%, 2 for 36.1% and 1 for 15.3% of the participants with, respectively, 22.9%, 81.8% and 50% of (at least one of) their parents being cigarette smokers; the parental cigarette smoking status of the participant group with MRC 2 being statistically significant (p=0,001). (Figure1). Also, the mean FNTD score was 4.63, with a score of 5 for 25% of the participants (Figure 2). Correlation between cigarette use by parents and the participant age, gender, BMI or duration of cigarette use was not demonstrable (p>0,05) (Table 2).

While the mean FEV1 value for participants with at least one cigarette smoking parent was 2.9lt, it was 3.3lt for participants with non-smoker parents, the difference being statistically significant (p=0,021). Correlation between the cigarette smoking status of the parents and the PFT parameters of the participants was not demonstrated (Table 3).

Mean ferritin level of the participants smoking 9 packages/year or below was 52.2, while this was 68.9 in the participant group smoking 10 packages/year or above; and the difference was statistically significant (p=0,026). A correlation was not found between the mean CRP value and the duration of cigarette smoking (p>0,05)(Table 4).

IV. Discussion:

Our results demonstrate that the FEV1 of young adults not exposed to cigarette smoking by parents in their childhood is significantly higher as compared to the participants whose parents were smokers. Despite the few investigations on this subject, it appears that the foundation of reduced respiratory function capacity and tendency to develop COPD is laid during the first and second decades of life, a disadvantage of childhood that continues to adulthood (14). Exposure to cigarette smoke early in life affects the development and changes of the airways more severely as compared to the mucosal sensitivity to inflammation at later decades. This creates a different clinical history than that of asthma (13).

In general clinical investigations have covered childhood asthma, lower respiratory tract infections, FEV1/FVC values at early adulthood and parental cigarette smoking (7,8,9). There are very few studies on the effects of the smoking habits of parents in the first 20 years of the lives of their offspring and the pulmonary function capacity (FEV1,FVC,FEV1/FVC) of these offsprings as cigarette smokers at early adulthood (10,11,12, 13,14). It was observed after using the MRC scale that 12.5% of the pariticipants of our study experienced dyspnea when going up a mild slope or walking at a fast pace, and that a significant percentage of these participants had been exposed at childhood to cigarette smoke of their parents in the home environment. This finding confirms that children exposed to cigarette smoke of their parents in the home environment develop adversely affected physical functions when they reach adulthood.

The simple and standardised MRC scale has been developed to assess health related life quality and is used in evaluating daily life quality of cigarette smokers (19). It is used not only for COPD patients but also for the cigarette smokers under the risk of developing COPD and shows a positive correlation between dyspnea perception and the subscales on physical function of life quality questionnaires (22) .

It has been known that elevated systemic inflammatory markers such as ferritin, CRP and platelets increase the risk of ischaemic heart disease, cancer and depression in the nonsmoker population (23,24,25). It has also been shown that increases in these markers, independently of cigarette smoking, increase the risk of cardiovascular diseases, type II diabetes mellitus, lung cancer and penumonia in COPD patients (26). In our study, it was observed that ferritin levels were increased significantly when the cumulative burden of cigarette smoking exceeded 10 packages/year, but unlike results reported by other studies, CRP and platelet levels were not affected.

The main limitation of the present study is the retrospective nature of the information about early life. The accuracy of recalling childhood asthma by adults may be related to current symptoms. However, when excluding subjects with current symptoms or asthma, our findings remained unchanged. Also, in our study the outcome measures were objective and not yet perceived which made differential recall bias less likely. A previous analysis revealed that adults reported important childhood events with high consistency regardless of symptom status. However, some random misclassification of early life factors due to nondifferential recall error is likely and will have attenuated the associations, such that the observations made may underestimate the true effects. Another problem of this study was the lack of information on potentially important factors such as childhood exposure to air pollution and childhood nutrition, which may also have contributed to underestimation of the true importance of early life disadvantages. The findings were consistent when subjects who had a history of asthma diagnosis or current respiratory symptoms were excluded. However, the findings might relate to

asymptomatic bronchoconstriction rather than to airway damage; this should be investigated in future studies (14,27).

V. Conclusion

Patients consulting the polyclinic should be questioned not only their cigrette smoking habit but also their history of exposure in the first two decades of their lives to cigarette smoking by parents in the home environment. These young adult cigarette smokers who had thus been exposed to cigarette smoke in early life should be accepted as a ‘’high risk group’’ for COPD development and should be put under follow up control for respiratory function capacity. Also, measures for stopping smoking should be intensified in this gorup of patients.

Acknowledgments
The study was not financed by any organization.
Funding sources
None declared
Conflict of interest
None declared

References

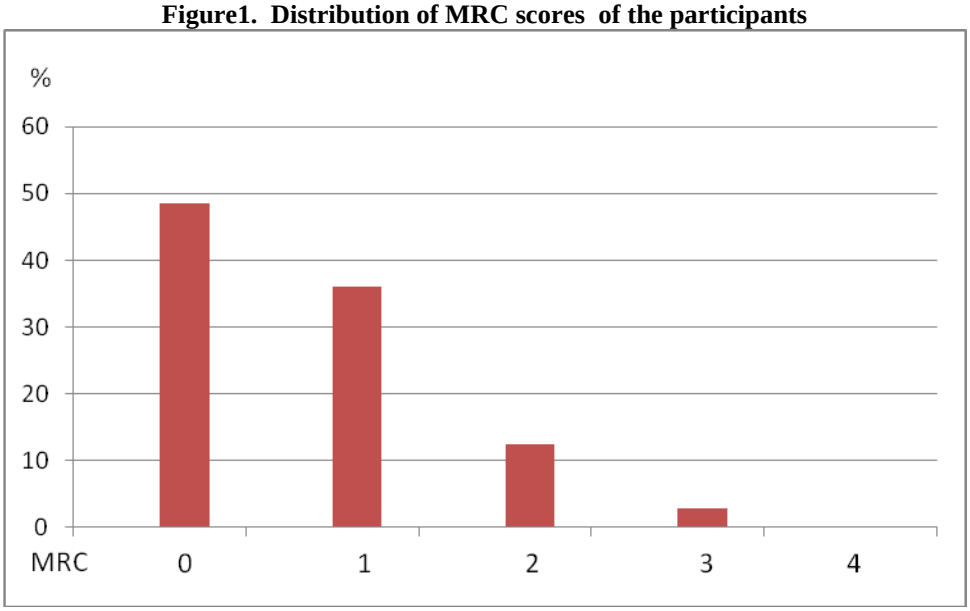
[1]. Fletcher CM,Peto R,Tinker C.The natural history of chronic bronchitis and emphysema.oxford university pres,1976.
[2]. Widysanto A, Mathew G. Bronchitis, Chronic.StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2018 Jan-.2018 Jan 12.
[3]. Divo MJ, Celli BR, Poblador-Plou B, et al; EpiChron—BODE Collaborative Group. Chronic Obstructive Pulmonary Disease (COPD) as a disease of early aging: Evidence from the EpiChron Cohort. PLoS One. 2018 Feb 22;13(2):e0193143. doi: 10.1371).
[4]. Bhattacharya S,Mariani Tj.Systems biology approaches to identify developmental bases for lung diseases. Pedatr Res 2013,73:514-522.
[5]. Butler JP,Loring SH,Patz.Evidence for adult lung growth in humans.N Eng J Med 2012; 367:244-247.
[6]. Hanrahan JP, Tager IB, Segal MR, et al.The effect of maternal smoking during pregnancy on early infant lung function. Am. Rev. Respir.Dis. 1992; 145: 1129–35.
[7]. Tager IB, Ngo L, Hanrahan JP. Maternal smoking during pregnancy.Effects on lung function during the first 18months of life.Am. J. Respir. Crit. Care Med.. 1995; 152: 977–83.
[8]. Cook DG, Strachan DP, Carey IM. Health effects of passive
[9]. smoking.9. Parental smoking and spirometric indices in children.Thorax 1998; 53: 884–93.
[10]. Tager IB, Weiss ST, Munoz A, Rosner B, Speizer FE. Longitudinal study of the effects of maternal smoking on pulmonary functionin children. N. Engl. J. Med. 1983; 309: 699–70).
[11]. Upton MN, Smith GD, McConnachie A, Hart CL, Watt GC. Maternal and personal cigarette smoking synergize to increase airflow limitation in adults. Am. J. Respir. Crit. Care Med. 2004; 169:479–87.
[12]. Upton MN, Watt GC, Davey Smith G, McConnachie A, Hart CL.Permanent effects of maternal smoking on offsprings’ lung function.Lancet 1998; 352: 453.
[13]. Guerra S, Stern DA, Zhou M,et al. Combined effects of parental and active smokingon early lung function deficits: a prospective study from birth to age 26 years. Thorax 2013; 68: 1021–8.
[14]. Svanes C, Omenaas E, Jarvis D, Chinn S, Gulsvik A, Burney P.Parental smoking in childhood and adult obstructive lung disease:results fromthe European Community Respiratory Health Survey.Thorax 2004; 59: 295–302.16.
[15]. Svanes C, Sunyer J, Plana E,et al. Early life originsof chronic obstructive pulmonary disease. Thorax 2010; 65: 14–20..).
[16]. T Yildiz, F Topçu, Y ÇELİK, L Akyildiz, G Ateş, D Durak.Effect of passive smoking on the development of chronic obstructive pulmonary disease in southeastern Turkey. Turk J Med Sci 2010; 40 (3): 349-355.
[17]. Heatherton TF, Kozlowski LT, Frecker RC, Fagerström KO. The Fagerström Test for Nicotine Dependence: a revision of the Fagerström Tolerance Questionnaire. Br J Addict. 1991;86(9):1119–2.
[18]. M. A.Uysal, F. Kadakal, Ç. Karsıdag, N.G. Bayram, Ö. Uysal, V.Yilmaz.Fagerstrom test for nicotine dependence: Reliability in a Turkish sample and factor analysis. Tüberküloz ve Toraks Dergisi 2004; 52(2): 115-121.
[19]. Pellegrino R, Viegi G, Brusasco . Interpretative strategies for lung function tests. Eur Respir J 2005;26(5):948e68.
[20]. Mahler DA, Wells CK. Evaluation of clinical methods for rating dyspnea. Chest. 1988;93(3):580–586.
[21]. Enright PL, McClelland RL, Newman AB,Gottlieb DJ, Lebowitz MD: Underdiagnosis and undertreatment of asthma in the elderly.Cardiovascular Health Study Research Group. Chest 1999; 116: 603–613
[22]. Verma BL, Kumar A, Srivastava RN. Measurement of body build based on weight/height, an index for adults in an Indian population. Indian J Public Health. 1982;26:133–43.
[23]. Roeland MM Geijer, Alfred PE Sachs, Theo JM Verheij, Huib AM Kerstjens,Marijke M Kuyvenhoven , ArnoW Hoes .Quality of life in smokers:focus on functional limitations rather than on lung function?R British Journal of General 478 Practice, June 2007.
[24]. Yousuf O, Mohanty BD, Martin SS,et al.High-sensitivity C-reactive protein and cardiovascular disease: a resolute belief or an elusive link?J Am Coll Cardiol. 2013 Jul 30;62(5):397-408.
[25]. Boen CE, Barrow DA, Bensen JT,et al. Social Relationships, Inflammation, and Cancer Survival.Cancer Epidemiol Biomarkers Prev. 2018 Feb 23. pii: cebp.0836.2017. doi: 10.1158/1055-9965.EPI-17-0836.
[26]. Naudé PJW, Roest AM, Stein DJ, de Jonge P, Doornbos B. Anxiety disorders and CRP in a population cohort study with 54,326 participants: The LifeLines study.World J Biol Psychiatry. 2018 Feb 22:1-10. doi: 10.1080/15622975.2018.1433325.
[27]. Thomsen M, Dahl M, Lange P, Vestbo J, Nordestgaard BG. Inflammatory biomarkers and comorbidities in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2012 Nov 15;186(10):982-8. doi:10.1164/rccm.201206-1113OC. Epub 2012 Sep 13.

[28]. Dratva J, Zemp E, Dharmage SC,et al. Early Life Origins of Lung Ageing: Early Life Exposures and Lung Function Decline in Adulthood in Two European Cohorts Aged 28-73 Years.PLoS One. 2016 Jan 26;11(1):e0145127.

Table 1: Demographic characteristics of the patients and paternal cigarette smoking habits

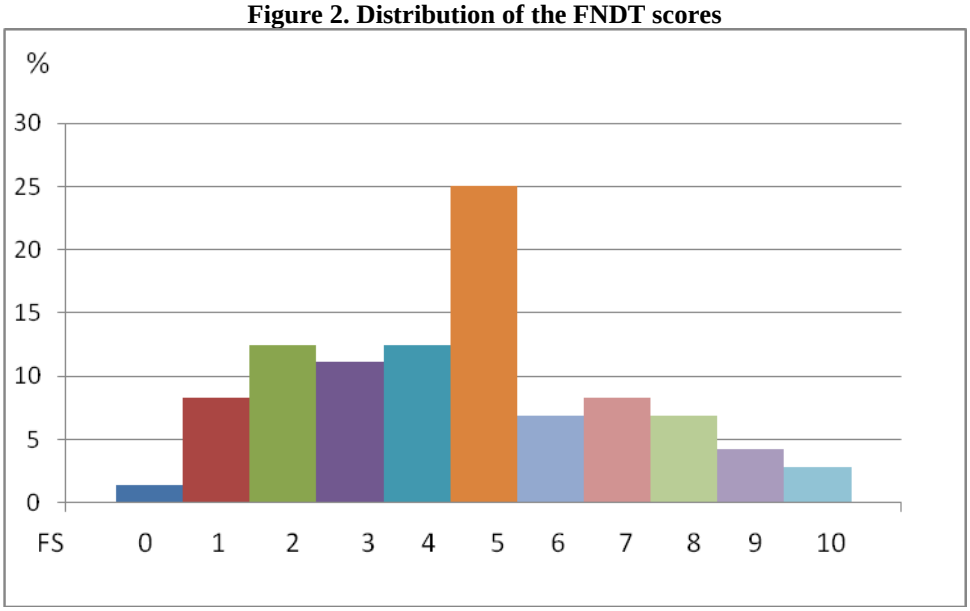
Characteristics	Numbers	Percent
Age (Years)		
≤34	46	63,9
≥35	26	36,1
Mean±SD:31.39±7.3; Median:32, The Youngest: 18, The Eldest:44		
Gender		
Male	46	63,9
Female	26	36,1
BMI		
<25,0	41	56,9
25,0-29,9	26	36,1
≥30,0	5	6,9
Mean±SD:24.6±3.5; Median:24.6;The Lowest:17.96; The Highest:35,16		
Cigarette Use		
Yes	72	100,0
No	0	0,0
Mean±SD:16,8±8,1; Median:20 ;The Lowest:2; The Highest:40		
Package/Year		
<9	37	51,4
≥10	35	48,6
Mean±SD:11,0±8,4 ;Median:8,5; The Lowest:1; The Highest:34		
Maternal Cigarette Smoking History		
Yes	4	5,6
No	68	94,4
Paternal Cigarette Smoking History		
Yes	29	40,3
No	43	59,7
Cigarette Smoking at Workplace		
Yes	0	0,0
No	72	100,0

Figure1. Distribution of MRC scores of the participants



MRC: Medical research council

Figure 2. Distribution of the FNDT scores



FNDT: Fagerström nicotine dependence test Mean±SD: 4,63±2,3; median :5, the lowest :0 ; the highest :10

Table 2: Comparison of parental cigarette smoking and some of the characteristics of the participants

Characteristic	Parental cigarette smoking				
	Yes		No		
Age (Years)	**n**	**%**	**n**	**%**	
18-24	7	43,8	9	56,3	0,759
25-34	11	36,7	19	63,3	
35-45	12	46,2	14	53,8	
Gender					
Male	17	37,0	29	63,0	0,281
Female	13	50,0	13	50,0	
BMI					
<25	19	46,3	22	53,7	0,355
≥25	11	35,5	20	64,5	
Package/Year					
≤9	14	37,8	23	62,2	0,498
≥10	16	45,7	19	54,3	
Number/Day					
≤10	9	36,0	16	64,0	0,477
≥11	21	44,7	26	55,3	
MRC					
0	8	22,9	27	77,1	**0,001**
1	13	50,0	13	50,0	
2	9	81,8	2	18,2	

Tablo 3: Comparison of the Parental smoking habit and the pulmonary function parameters (PFP) of the participants

PFP	Group	n	Mean	SD	Median	p*
FEV1 lt	PS	30	2,9	0,8	2,67	**0,021**
	PNS	42	3,3	0,7	3,42	
FEV %	PS	30	82,8	19,7	82	0,326
	PNS	42	87,0	16,1	87	
FVC lt	PS	30	3,3	0,8	3,34	0,065
	PNS	42	3,8	1,0	3,8	
FVC %	PS	30	82,5	18,1	79,5	0,946
	PNS	42	82,3	14,9	83	
FEV1/FVC	PS	30	86,4	12,5	89,0	0,186
	PNS	42	90,3	11,5	93,3	

PS: Parent smoker PNS: Parent non-smoker P* ≤ 0.05 for statistical significance

Table 4: Relationships between some acute phase protein parameters(APP) and cigarette smoking duration (package/year)

APP	Duration	n	Mean	SD	Median	p*
Ferritin	≤9	37	52,2	59,7	35,7	**0,026**
	≥10	35	68,9	46,5	62	
CRP	≤9	37	5,2	4,4	3,45	0,069
	≥10	35	9,3	20,9	3,55	
Platelet	≤9	37	336594,6	403587,8	271000	0,148
	≥10	35	255714,3	54562,7	255000	

P* ≤ 0.05 for statistical significance

Özlem Şengören Dikiş "Smoked young adults parents smoking habits effect on respiratory parameters.”."IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 6, 2018, pp 05-11.