

The Effects of Parental Smoking on Anthropometric Parameters, Peak Expiratory Flow Rate and Physical Condition in School Children

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ABSTRACT

Passive smoking in children is a considerable health problem, mainly arising from parental smoking. The objectives of the present cross-sectional study were to assess the impact of passive smoking on 1) anthropometric parameters; 2) peak expiratory flow rate (PEFR); and 3) physical condition in school children. The target population included 177 children attending elementary school 5th to 8th grade. Study subjects were divided into two groups according to parental smoking habits. Body weight and height were determined using a digital weighing scale and digital stadiometer; PEFR was measured between 8 a.m. and 10 a.m. using a Peak Flow Meter; and physical condition was assessed by the 6-minute run test. Sixty-six percent of study children were exposed to passive smoking. The children of smoking parents had higher BMI [18.79 (17.50–21.13) kg/m²] than children of nonsmoking parents [17.90 (16.00–20.00) kg/m²; p=0.036]. There was no statistically significant difference in body height and weight. The children of smoking parents had statistically lower values of PEFR [M(IQR) = 84 (78–88)%, M(IQR) = 94 (89–101)%, respectively; p<0.0001] and 6-minute run test than children of nonsmoking parents [M(IQR) = 2(1–3), M(IQR)=4(3–5); respectively; p<0.0001]. The results of the present study showed that exposure of school children to passive smoking by their parents resulted in an increase of BMI, impairment of lung function, and impairment of physical condition, especially in children of both smoking parents.

Key words: children, parental smoking, antropometric parameters, peak expiratory flow rate, physical condition

Introduction

Since 1981, when a Japanese study showed that second-hand smoke caused lung cancer in nonsmokers¹, a vast number of published data have revealed that passive smoking is associated with a range of adverse health outcomes. It seems that the unfavorable impact of passive smoking is more pronounced in children, which could be due to the fact that their undeveloped systems are more susceptible to the harmful effects of second-hand smoke.

Unfortunately, the most common source of passive smoking in children is parental tobacco smoking at home. According to the World Health Organization (WHO), 43% of all children in the world are exposed to passive smoking at home by at least one smoking parent². In

Croatia, it is estimated that 73.4% of children are exposed to second-hand smoke at home³.

Passive smoking is strongly linked to a range of adverse health outcomes in children. The harmful effect of toxic agents from cigarette smoke begins even *in utero* if mother smokes during pregnancy. Available medical evidence suggest that maternal smoking in pregnancy leads to lower birth weight and birth length, and predisposes newborns to increased respiratory morbidity after birth^{4,5}. Children passively exposed to cigarette smoke are more susceptible to respiratory tract infections and other serious bacterial infections^{6,7}. It has been demonstrated that exposure to parental smoking during child-

hood is associated with significant decrease in lung function, especially peak expiratory flow rate (PEFR), forced expiratory volume in 1 second (FEV1) and forced expiratory flow^{8–13}. Peak expiratory flow monitoring is widely used to assess airway caliber because it is highly sensitive and has an accurate index of airway obstruction, is easy to perform, inexpensive and well tolerated.

Exposure to second-hand smoke adversely affects physical growth in young children¹⁴. However, it seems that not all children are equally susceptible to the harmful effects of second-hand smoke, indicating that genetic factors may determine their susceptibility to damage caused by toxic ingredients from cigarette smoke^{15,16}.

The objectives of the present study were to assess the impact of passive smoking on 1) anthropometric parameters; 2) PEFR; and 3) physical condition in school children.

Subjects and Methods

This cross-sectional study was conducted at Trilj Elementary School between September 2011 and November 2011. Trilj is a rural area of southern Croatia, with small socioeconomic differences. The target population included 177 children attending 5th to 8th grade. Forty-four children were excluded from the study: 9 for the history of asthma, 3 for the history of systemic disease with known repercussion on respiratory function (2 with neuromuscular disease and 1 with congenital cardiopathy), 17 for being diagnosed with acute respiratory tract infection during the course of the study, 7 for having refused to take part in the study, 4 children failed to complete the questionnaire, and 4 children for having admitted active smoking. Study children and their parents were asked about family smoking at home, defined as second-hand smoke exposure in this study¹⁷.

Children were divided into two groups according to the parental smoking habits: group 1 including children of smoking parents (N=88/133; 66%; 45 female) and group 2 including children of nonsmoking parents (n=

45/133; 34%; 23 female) as control group. In addition, group 1 subjects were divided into two subgroups: 1A, one smoking parent (N=49/133; 37%) 1B, both smoking parents (N=39/133; 29%).

Body weight and height were determined using a digital electronic weighing scale (range 1–150 kg) and digital stadiometer (range 70–205 cm), respectively. These data were used to calculate body mass index (BMI) of each study subject. Body weight was expressed in kilograms (kg), body height in centimeters (cm) and BMI in kg/m². PEFR was measured between 8 AM and 10 AM using a Peak Flow Meter with a scale graduated high range of 60–800 L/min. Children were instructed how to use the instrument. They were asked to take deep breath, then to exhale it by forceful expiration as fast as possible after maintaining airtight seal between lips and mouthpiece of the instrument. All measurements were performed by the same pediatrician. Every measurement was repeated three times and the best matching results were used on analysis. The results were expressed as percentage of the predicted value for age, sex and height. On assessment of physical condition, 6-minute run test (F-6 test) was used, the results being evaluated on a scale from 1 (worst) to 5 (best) according to the national guidelines for interpreting the results of F-6 test for girls and boys aged 11 to 14 years (5th to 8th graders) (Table 1)¹⁸. On teacher's request, the student takes standing position at the start line. On the start signal, the student begins to run persistently for six minutes, trying to achieve the best possible score. Testing was performed on an outdoor playground that measures 40x20 meters (one lap = 120 meters).

Data processing was performed using MedCalc software (Medisoftware, Mariakerke, Belgium). Continuous variables were described as mean and standard deviation (\pm SD) if they had normal distribution, or median and interquartile [M (IQR)] range if not. Comparisons between variables were made using Student's t-test or Mann-Whitney test. Values of $p < 0.05$ were considered statistically significant¹⁹.

TABLE 1
SIX-MINUTE RUN TEST SCORE FOR GIRLS AND BOYS, 5th to 8th GRADE

Grade	Score				
	1	2	3	4	5
Girls					
5 th	<890 m	900–999 m	1000–1099 m	1100–1149 m	>1150 m
6 th	<929 m	930–1029 m	1030–1129 m	1130–1179 m	>1180 m
7 th	<959 m	960–1059 m	1060–1159 m	1160–1209 m	>1210 m
8 th	<989 m	990–1089 m	1090–1189 m	1190–1239 m	>1240 m
Boys					
5 th	<999 m	1000–1099 m	1100–1199 m	1200–1249 m	>1250 m
6 th	<1049 m	1050–1149 m	1150–1249 m	1250–1299 m	>1300 m
7 th	<1099 m	1100–1199 m	1200–1299 m	1300–1349 m	>1350 m
8 th	<1149 m	1150–1249 m	1250–1349 m	1350–1399 m	>1400 m

m – meters

TABLE 2
BODY HEIGHT, BODY WEIGHT, BODY MASS INDEX (BMI), PEAK EXPIRATORY FLOW RATE AND F-6 TEST IN CHILDREN OF SMOKING PARENTS (GROUP 1) AND CHILDREN OF NONSMOKING PARENTS (GROUP 2)

	Height (cm) \pm SD	Weight (kg) M(IQR)	BMI M(IQR)	PEFR (%) M(IQR)	F-6 test M(IQR)
Group 1 (N=88)	157 \pm 8	45 (37–52)	18.79 (17.50–21.13)	84 (78–88)	2 (1–3)
Group 2 (N=45)	158 \pm 9	47 (41–55)	17.90 (16.00–20.00)	94 (89–101)	4 (3–5)
p	NS	NS	0.036	<0.0001	<0.0001

\pm SD – average \pm standard deviation, M – median, IQR – interquartile range, NS >0.05

TABLE 3
BODY HEIGHT, BODY WEIGHT, BODY MASS INDEX (BMI), PEAK EXPIRATORY FLOW RATE AND F-6 TEST IN SUBGROUP 1A (ONE SMOKING PARENT), SUBGROUP 1B (BOTH SMOKING PARENTS) AND GROUP 2 (NONSMOKING PARENTS)

	Group 1A, N=49	Group 1B, N=39	Group 2, N=45	P, (IA:IB)	P, (IA:II)	P, (IB:II)
Height (cm) (\pm SD)	159 \pm 9	156 \pm 7	158 \pm 9	NS	NS	0.031
Weight (kg) M(IQR)	49 (41–55)	43 (41–54)	45 (37–52)	NS	NS	NS
BMI (kg/m ²) M(IQR)	18.7 (17.6–21.2)	18.6 (17.5–21.0)	17.9 (16.0–20.0)	NS	NS	0.042
PEFR(%) M(IQR)	84 (80–90)	83 (77–86)	94 (89–101)	NS	<0.0001	<0.0001
F6-test M(IQR)	2 (2–4)	2 (1–2)	4 (3–5)	0.04	<0.0001	<0.0001

M – median, IQR – interquartile range, NS >0.05

The study was approved by the Hospital and School Ethics Committee. Diagnostic work-up was performed according to standardized procedure and in line with ethical principles and Declaration on Human Rights from Helsinki 1975 and Seoul amendments 2008. A signed informed consent was obtained from study children and their parents.

Results

Of 177 children included in the study population, 44 children failed to meet the testing criteria and were excluded from the study. Of the remaining 133 children included in the study, there were 66 (49.6%) males and 67 (50.4%) females. The children enrolled in the study were matched by age ($p=0.617$), i.e. the children in both groups were aged 12 ± 1 years. Eighty-eight (66%) children were exposed to passive smoking, while 45 (34%) children came from nonsmoking families. One parent was smoking in 49/88 (56%), while both parents were smoking in 39/88 (44%) smoking families.

There was no statistically significant difference in either body height or body weight (Table 2). However, children of smoking parents (group 1) had higher BMI [18.79 (17.50–21.13) kg/m²] than group 2 children of nonsmoking parents [17.90 (16.00–20.00) kg/m²; $p=0.036$] (Table 2).

PEFR values were statistically lower in group 1 [M(IQR) = 84 (78–88)%] than in control group 2 [M(IQR) = 94 (89–101)%; $p<0.0001$].

The median F-6 test values were statistically lower in group 1 than in control group 2 [M(IQR) = 2 (1–3) and M(IQR) = 4 (3–5), respectively; $p<0.0001$].

Children of smoking parents were divided into subgroups depending on whether one or both parents were smoking; these results are shown in Table 3. Children's weight in both subgroups was the same regardless of one or both parents were smoking. Children of both smoking parents had a statistically significant lower body height (\pm SD = 156 ± 7 cm) than control group children (\pm SD = 158 ± 9 cm). Children of both smoking parents had statistically higher BMI values [M(IQR) = 18.6 (17.5–21.0) kg/m²] than control group children [M(IQR) = 17.9 (16.0–20.0) kg/m²]. There was no difference ($p>0.05$) in PEFR values between the 1A and 1B subgroups [M(IQR) = 84 (80–90)% and M(IQR)=83(77–86)%, respectively]. F-6 test values were statistically significantly lower ($p=0.04$) in 1B than in 1A subgroup [M(IQR) = 2 (1–2) and M(IQR) = 2 (2–4), respectively].

Discussion

The results of the present study showed that exposure of school children to passive smoking by their parents resulted in an increase of BMI, impairment of lung function (i.e. decreased PEFR values), and impairment of physical condition (i.e. decreased F-6 test results), especially in children of both smoking parents.

It has already been confirmed that newborns whose mothers were smoking during pregnancy have lower BMI⁴. However, it is estimated that passive smoking in childhood is associated with higher BMI in children, which is in line with our results. The results of our study showed negative influence of passive smoking on the height of children from the families with both smoking parents. These results are in line with a previous study conducted in young athletes²⁰. Rona et al. found that

children whose parents smoked more than 10 cigarettes a day were on average by 0.6 cm shorter than those of nonsmoking parents²¹. The mechanism by which passive smoking affects growth is unknown. It has not yet been established whether the effect is indirect or direct. The most suspected substances that could play an important role in this effect are nicotine and carbon monoxide (CO). Nicotine constricts blood vessels producing a state of tissue hypoperfusion. CO, by forming carboxyhemoglobin, further hinders oxygen delivery to body tissues, which leads to tissue hypoxia. It may be hypothesized that chronic decrease in oxygen tension caused by long term exposure of children to second-hand smoke is the main factor of growth retardation. Our results are consistent with the results of other authors demonstrating that the increase in BMI resulted from increased body weight and reduced height, suggesting that it may be related to basal metabolism due to oxidative stress²². According to Wilson et al., the existence of oxidative stress in children exposed to second-hand smoke is associated with a reduced intake of antioxidants and increased consumption of the existing antioxidants²³.

Some cigarette smoke components can cause abnormal tightening of airways^{24,25}. It was also indicated by our study results, since we found that children of smoking parents had a statistically significantly lower PEF value than children of nonsmoking parents, regardless of one or both parents were smoking. This is in line with previous studies, which found passive smoking to be associated with reduced PEF⁹. Italian authors have reported lower average levels of PEF in children exposed to second-hand smoke, even after exclusion of asthmatics and those with acute respiratory problems²⁶, the same as we did. In children exposed to second-hand smoke, Cook et al. demonstrated bronchial obstruction to be the spirometric effect of second-hand smoke, with predominant involvement of small airways²⁷. Even low-level exposure to second-hand smoke in children could produce an adverse effect on lung function²⁸. These airway changes make breathing more difficult, which can restrict physical potential. The possible mechanistic aspects by which passive smoking affects lung function are unknown. Some authors consider that direct damage to respiratory epithelium leads to an inflammatory-immune process²⁹. Cigarette smoke causes mucus gland hypertrophy and hyperplasia, resulting in increased mucus production. The latter, in combination with the loss of cilia caused by cigarette smoke, leads to collection of mucus with harmful substances in the airways causing inflammation and damage to lung tissue.

Based on the results of the present study, we can conclude that children of smoking parents had a lower grade of motor skills than children of nonsmoking parents. This is consistent with previous studies conducted in children^{30,31}. Moreover, Flouris et al. showed in adult healthy nonsmokers that 1 hour of passive smoking exposure adversely affected the response to physical activity³². It seems reasonable to hypothesize that children are a more vulnerable population to the toxic effects of

passive smoking because of their physiologically undeveloped systems and unique physiologic characteristics. Although the mechanistic aspects by which passive smoking exerts adverse effects on physical condition in children remain unclear, it can be assumed that many of the substances in cigarette smoke can cause alterations in cardiorespiratory function, leading to a decrease in physical activity. In our prior work, we showed that children who were exposed to second-hand smoke at home had worse physical condition as assessed by F-6 test than children who were not exposed³¹. Since there are no available literature data on the value of F-6 test for the assessment of physical condition in school children, we could not compare our results with other authors. The 6-minute walk test and 12-minute walk/run test have been used to estimate exercise capacity in obese children and adolescents³³, while exercise testing using a treadmill or cycle ergometer has been widely used for the measurement of exercise performance in school children³⁴. However, this study showed that this simple test can be used in everyday work for the assessment of physical condition in school children.

Some potential limitations of the present study need to be considered. We did not use biomarkers of tobacco exposure. Instead, questionnaire data were used to measure exposure to cigarette smoke, which might cause misclassification because of underreporting. Results of a study by Forastiere et al. suggest that the subject's perception of a smoky environment could be surrogate indices of high passive smoking exposure in adolescents³⁵. These results indicate that nonsmokers can provide accurate description of second-hand smoke exposure. Because of organizational problems, we did not perform spirometry, but only PEF instead. Although PEF monitoring has some methodological problems, it has been successfully used to assess the effects of airborne concentrations of several pollutants on airway caliber³⁶. In addition, we lacked information on some of the potential confounders such as maternal smoking during pregnancy, the level of community pollution or other potentially air pollutants that could affected pulmonary function and any other factors that could affected anthropometric parameters. Despite these limitations, we believe that the results of our study provide additional evidence for the harmful effects of passive smoking on children's health. Hence, longitudinal larger studies could be useful to confirm our results.

In conclusion, our results show that exposure to second-hand smoke due to parental smoking has strong effects on children's physical condition and pulmonary function. In line with our findings, it could be concluded that reducing exposure of children to passive smoking would improve children's health. It might be an important argument when trying to persuade the smoking parents to quit smoking. Everyone responsible for the health of children should be aware of adverse effects of second-hand smoke exposure, emphasizing the role of educating parents on this issue.

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UTJECAJ PUŠENJA RODITELJA NA ANTROPOMETRIJSKE PARAMETRE, VRŠNI EKSPIRACIJSKI PROTOK I FIZIČKU KONDICIJU ŠKOLSKE DJECE

SAŽETAK

Pasivno pušenje u djece koja su najčešće izložena duhanskom dimu od strane njihovih roditelja je zabrinjavajući zdravstveni problem. Ciljevi našeg istraživanja bili su utvrditi utjecaj pasivnog pušenja na: 1) antropometrijske parametre (tjelesnu težinu, visinu i indeks tjelesne mase BMI), 2) vršni ekspiratorni protok (PEFR), i 3) fizičku kondiciju školske djece. Ciljana populacija je uključivala 177 djece, polaznika 5. do 8. razreda osnovne škole. Ispitanike smo podijelili u dvije skupine, ovisno o izloženosti pasivnom pušenju. Tjelesna težina i visina utvrđene su pomoću digitalne vage i digitalnog visinomjera. Za mjerenje PEFR korišten je Peak-flow meter, a za procjenu fizičke kondicije 6-minutni test trčanja (F-6 test). Šestdesetšest posto djece izloženo je pasivnom pušenju kod kuće. Statistički značajno veći BMI imaju djeca roditelja pušača [18,79 (17,50–21,13) kg/m²] od djece čiji roditelji nisu pušači [17,90 (16,00–20,00) kg/m²]; p=0,036. Nije nađena statistički značajna razlika u tjelesnoj težini niti visini između dvije skupine ispitanika. Vrijednosti PEFR su statistički niže u djece izložene pasivnom pušenju [M(IQR)=84 (78–88)%] nego u djece koja nisu izložena pasivnom pušenju [M(IQR) = 94 (89–101)%]; p=<0,0001. Djeca izložena pasivnom pušenju imaju statistički nižu ocje-

nu F-6 testa [M(IQR) = 2 (1–3)] od djece koja nisu izložena pasivnom pušenju [M(IQR) = 4 (3–5)]; $p < 0,0001$. Izloženost djece pasivnom pušenju od strane njihovih roditelja povezano je s povećanjem BMI, nižim vrijednostima PEFR i slabljenjem fizičke kondicije, što je posebno izraženo kada su oba roditelja pušači.