

An Analysis of the Thickness of Abdominal Muscles during Forceful Expiration and Pulmonary Function in Teenage Smokers and Nonsmokers

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Abstract. [Purpose] The purpose of this study was to examine the effects of smoking on teenagers' internal oblique (IO) and transverses abdominis (TrA) expiratory muscles and their pulmonary function. [Subjects] A total of 30 healthy teenagers (15 smokers; 15 nonsmokers) voluntarily participated in the study. [Methods] The subjects were instructed to maintain an upright standing posture with their scapulars against the wall. Measurements were then taken to determine the thickness of their right IO and their right TrA while they were at rest and in a state of forced expiration using a 7.5 MHz linear probe of an ultrasonic imaging system. The measured thickness was converted into the percentage of change in muscle thickness (PCMT) and the relative contribution ratio (RCR) using a calculation formula, and then the data were analyzed. [Results] No significant differences were found between the two groups in the thickness, PCMT, and RCR of both the IO and TrA muscles, while there were significant differences in the forced expiratory volume at one second (FEV1) and the peak expiratory flow (PEF). [Conclusion] This study examined teenage smokers whose duration of smoking was relatively short. The two groups did not show significant differences in the thickness of both the IO and TrA muscles. However, based on the forced expiratory volume at one second (FEV1) and PEF measurements, the smokers showed greater decreases in pulmonary function than the nonsmokers.

Key words: Smokers and nonsmokers, Pulmonary function, Abdominal muscles

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INTRODUCTION

Many smokers start smoking in their teens, and teenage smokers are 16 times more likely to carry the smoking habit into adulthood than nonsmoking teenagers because the earlier people begin smoking, the sooner they become habitual smokers¹⁾. Smoking increases the risk of lung cancer by causing DNA damage in lung cells²⁾, and it also decreases the ability to exchange gases in the alveoli by irritating the airway mucosa, which raises bronchial tube contraction and mucus secretion³⁾. Smoking particularly causes serious harm to the cells and tissues of teenagers' bodies, which are not yet fully developed, and it increases oxidative stress, which promotes chronic diseases and aging, inhibits the growth of brain cells and bone marrow, and leads to memory impairment and anxiety⁴⁾.

Respiration performs the function of exchange between oxygen and blood via the lungs, vital parts of the body's ventilation system⁵⁾. The muscles involved in the stability of the trunk and respiration are divided into the inspiratory

muscles and the expiratory muscles. The main inspiratory muscles include the diaphragm and the external intercostals, which are recruited during normal respiration. The accessory muscles of inspiration include the sternocleidomastoid, the scalenes, the trapezius, the pectoralis major, the pectoralis minor, and the serratus anterior, which are recruited during deep, forced, or labored respiration. In comparison, the main muscles involved in expiration do not act during normal respiration, while the accessory muscles, including the rectus abdominis, the transverse abdominis, the internal/external oblique, and the internal-intercostals, act during deep or strong respiration^{6, 7)}.

Based on ultrasonic measurement of young men, Kim and Kim⁸⁾ reported that the thickness of the IO and TrA muscles measured while the subjects were at rest significantly increased when the thickness was measured while the subjects were in a state of forceful expiration. They also found significant differences in the PCMT of the TrA and in the RCR of both the IO and the TrA. These results were obtained from adults who had smoked for 56 months by only measuring the thickness of their respiratory muscles, not by taking their vital capacity into account. No research has measured the vital capacity of teenagers whose duration of

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smoking is relatively short, 14 months on average.

Thus, this study aims to determine the effects of smoking on teenagers' IO and TrA expiratory muscles by measuring the thickness of the muscles and by examining the teenagers' lung capacity.

SUBJECTS AND METHODS

A total of 30 subjects (15 smokers, 15 nonsmokers) participated in the research. Their average age, height, weight, and body mass index (BMI) were 13.03 years, 170.73 cm, 61.87 kg, and 18.07 kg/m², respectively. The smokers' average smoking period was 17.53 months. Prior to the experiment, a pulmonary function test using a spirometer (SP-260 pneumotacho sensor, Schiller, Switzerland) was performed on all prospective subjects to examine their pulmonary functions. Using a maximal-effort expiratory spirogram, the forced vital capacity (FVC), forced expiratory volume at one second (FEV1), and FEV1/FVC ratio were measured in order to determine whether the subjects had obstructive or restrictive pulmonary disease. Their airway resistance was then measured based on the peak expiratory flow (PEF).

Changes in the thickness of the IO and TrA were analyzed using ultrasonography (ESAOTE Europe B.V., Netherlands) while the subjects were resting and in a state of forceful expiration. Ultrasonography was conducted by referring to previous studies. Ultrasonographic imaging was obtained using a 7.5 MHz linear probe, with the midpoint of the transducer placed along the midaxillary line in the transverse plane just above the right iliac crest. All subjects maintained an upright standing posture during both resting and forceful expiration states, with their scapulars fixed on the wall during expiration to prevent movement of the trunk during ultrasonographic imaging. The changes in the thickness of the IO and TrA of both the smokers and nonsmokers while resting and in a state of forceful expiration were analyzed using a paired t-test. The homogeneity of the subjects' general characteristics, the percentage of change in muscle thickness (PCMT), and the relative contribution ratio (RCR) while resting and in a state of forceful expiration were calculated using the following formula and were compared using an independent t-test. The significance level, α , was set to 0.05. $PCMT = [(muscle\ thickness\ during\ forceful\ expiration - muscle\ thickness\ during\ resting) / muscle\ thickness\ during\ resting] \times 100$. $RCR = [PCMT\ of\ IO\ (or\ TrA) / (PCMT\ of\ IO + PCMT\ of\ TrA)] \times 100^{(8)}$.

RESULTS

The thicknesses of the subjects' IO and TrA muscles were measured while they were at rest and while they were in a state of forceful expiration, and then the results were compared. According to the results, both smokers and nonsmokers did not show significant differences in the thickness of the muscles during forceful expiration. No significant differences were found between smokers and nonsmokers in the PCMT and RCR of both the IO and TrA. In terms of vital capacity, the subjects did not show differences in the FVC1 and FEV1/FVC, while they revealed significant differences in the FVC (for both groups, $p=0.03$) and PEF (for both groups, $p=0.00$) (Table 1).

DISCUSSION

Smoking decreases FEV1 and lung diffusing capacity and causes lung growth disorders in children or teenagers⁽⁹⁾, and it has been reported that smoking increases the risk of bronchial infection to the level of the risk experienced by chronic lung disease patients⁽¹⁰⁾. It also increases airway resistance and causes ventilation impairments, which decreases FVC and PEF to between 25% and 75%⁽¹¹⁾. This study also found that teenage smokers showed decreased vital capacity in terms of FEV1 and REF compared with nonsmokers.

Regarding the thickness of the respiratory muscles, not many studies have reported cases of musculoskeletal dysfunction resulting from smoking⁽⁸⁾, but such cases did show decreased oxidative enzymes, slow recovery of phosphocreatine after exercise, early appearance of lactate, a high level of oxidative stress, and fiber-type redistribution in COPD patients resulting from long-term inactivity⁽¹²⁻¹⁶⁾. Moreover, it has been reported that smoking decreases the cross-sectional area of type I and type II muscle fibers of the vastus lateralis of non-COPD smokers, warning against skeletal muscle damage caused by smoking⁽¹⁷⁾.

Teenage smokers, whose average duration of smoking was relatively short, did not show significant differences in respiratory muscle imbalance in comparison with teenage nonsmokers, but the teenage smokers revealed clear differences in terms of decreased pulmonary function. Considering that smoking causes lung growth disorders and decreased respiratory function in teenagers, antismoking campaigns are extremely necessary at this point. In addition, research showed that a significant increase in the IO's contribution during forceful expiration in healthy subjects

Table 1. Comparison of pulmonary function during forceful expiration

PF	Smoker	Nonsmoker		(unit: L)
FVC (L)	4.38±0.99	4.83±1.15	-1.10	> 0.05
FEV1 (L/sec)	3.74±0.65	4.33±0.72	-2.48	< 0.05*
FEV1/FVC (%)	88.33±10.98	91.20±8.74	-0.83	> 0.05
PEF (L/sec)	5.89±1.62	7.73±1.56	-4.08	< 0.05*

PF: pulmonary function, FVC: forced expiratory vital capacity, FVC1: forced expiratory volume in 1 sec, FVC1/FVC: forced expiratory vital capacity in 1 sec as a percentage of FVC, PEF: peak expiratory flow

in their twenties without musculoskeletal disorders, such as LBP, indicates that smoking affects the lumbar stabilizers' recruitment patterns⁸⁾. This finding emphasizes the necessity of calling teenage smokers' attention to the fact that continuation of smoking habits from adolescence to adulthood may cause an imbalance in their respiratory muscles.

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