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Original article

Effects of overweight and smoking on aerobic capacity, autonomic and pulmonary function in physically inactive middle-aged male

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Background: A higher body mass index (BMI) can lead to smoking, some people believe that smoking can help them to lose their weight and also quit smoking can cause weight gain. The effects of overweight and smoking have not been studied in physically inactive middle aged males, a vulnerable group in gaining weight and being at greater risk of *developing* cardiovascular diseases (CVD).
Objective: This study aimed to determine the effects of overweight and smoking on aerobic capacity, autonomic and pulmonary function in physically inactive middle aged male subjects.
Methods: Seventy-two physically inactive subjects were divided according to normal BMI nonsmokers (18.5 – 22.9 kg/m^2, n = 18), normal BMI smokers (n = 18), overweight BMI nonsmokers (23.0 - 24.9 kg/m^2, n = 18) and overweight BMI smokers (n = 18). All subjects were measured for body composition, heart rate variability (HRV). Pulmonary function and maximal oxygen consumption (VO_{2max}) were measured by spirometry and cycle ergometer, respectively. Normality in the data was tested by using the Kolmogorov Smirnov test. Two-way ANOVA was used to determine the effects of overweight BMI and smoking.
Results: Overweight and smoking aggravated body composition imbalance, autonomic dysregulation and diminished VO_{2max}. Overweight decreased force vital capacity (FVC), while smoking diminished forced expiratory volume in one second (FEV1), FEV1/FVC ratio and fat-free mass. This study also found interaction of visceral fat and low frequency (LF) value in physically inactive subjects.
Conclusion: Overweight is as hazardous as regular smoking on VO2 max, pulmonary function. Overweight and smoking also decreased fat-free mass, increased visceral fat as well as autonomic dysfunction. It can be related tentatively to the development of CVD in physically inactive middle-aged males.

Keywords: Overweight, smoking, aerobic capacity, autonomic function, pulmonary function.

The prevalence of smoking and overweight body mass index (BMI) is of major public health concern.[1] Epidemiological studies reported that by 2030 more than 38.0% of the world's population are overweight.[2] Approximately 30.0% of daily male smokers are overweight or pre-obese men try to give up smoking and are unwilling to quit. [3]

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A higher BMI can lead to increased smoking, and greater nicotine consumption, quitting smoke causes weight gain.[1] The effects of smoking and overweight BMI is a complicated, multifactorial, along with premature death [2] and direct estimates of the causal interaction are helpful.[4]

Heart rate variability (HRV) is a simple method for evaluating autonomic balance.[5] Autonomic imbalance has been reported in various conditions, with some studies showing that obesity, overweight and smoking exert autonomic dysregulation. [6 - 9] Aerobic capacity is used to determine the ability to remove and utilize oxygen from circulating blood to active muscle while performing intense exercises or physical

activities.[10] Pulmonary function is a contributing to the enhancement of aerobic performance. [10] Toxic substances from smoking have been demonstrated not only progressively worsening of pulmonary function but also declining aerobic capacity.[11]

Potential effect of BMI on smoking has been complex due to the belief that smoking cessation cause weight gain, some people still smoke to control their weight.[1] Middle-aged male experienced weight gain due to hormonal changes, leading to reduce basal metabolic rate and subsequently weight gain and overweight.[12] Current smokers in middle-aged males who were unwilling to quit, the incidence of cancer and deaths was higher.[13] Therefore, the research and education on smoking and smoking should target in middle and older aged males, a vulnerable group to gaining weight and are more likely to smoke.[12, 14] Previous studies mostly focused on either obesity or overweight compared with cigarette smoking in early and late adolescence. Such as Mackey and his colleagues found that obesity and overweight were as hazardous as heavy and light smoking, respectively, but there was no interaction between BMI and smoking status in early and late adolescent.[15]

The study has not addressed the effects of overweight and smoking on heart rate variability, pulmonary function as well as aerobic capacity in middle aged male individuals who have fewer physical activities and less exercise.[16] This study sought to explore the potential causal effect of overweight BMI and smoking on aerobic capacity, autonomic and pulmonary function in physically inactive male subjects aged 36 to 55 years, in order to intensify public health concern and promote the importance of healthy physical fitness.

Materials and methods

Recruitment of subjects:

Inclusion and exclusion criteria:

This cross sectional study was conducted in accordance with an approved protocol from the Naresuan University's Institutional Review Board (IRB no.0112/62) following the Declaration of Helsinki. The study was carried out in the research laboratory at the Department of Physical Therapy, Faculty of Medical Health Science, Naresuan University, Phitsanulok, Thailand. The number of subjects was calculated according to a previous study by Kim DJ, *et al.*[17] In order to show a significant difference at alpha, error of 5.0% and power of 80.0%, with 10.0% nonresponse rate, 18 subjects in each group were needed in this study. Informed consent was obtained from all of the subjects before the test and after a full explanation of the research study.

Inclusion criteria were as follows: all subjects in the 36 - 55 year age group, with normal and overweight BMI according to the Asian Pacific criteria[18], performed moderate or vigorous intensity exercise for less than 30 minutes 5 days per week or 25 minutes 3 days per week[19] by using the international physical activity questionnaire.[20] The physically inactive subjects, who could read and write Thai well, were taken as the study population and divided into the four groups: normal BMI nonsmokers (18.5 – 22.9 kg/m^2, n = 18 per group), normal BMI smokers (18.5 – 22.9 kg/m^2, n =18), overweight BMI nonsmokers (23 - 24.9 kg/m^2, n = 18) and overweight BMI smokers (23 - 24.9 kg/m^2, n = 18). The nonsmoking subjects had never smoke or had been ex-smoker before, while daily smokers had smoked regularly for at least 25 - 30 days per month for at least a year.[21]

Subjects who were underlying cardiovascular, pulmonary and neurological diseases, as well as comorbidities were excluded from this study. Subjects who had taken alcoholic beverages, caffeine or stimulating drugs within the previous 48 hours, and those with musculoskeletal disease also were excluded.[22]

Anthropometric measurements

Body composition measurements were taken by bioelectrical impedance analysis (BIA) (HBF 375, Omron Healthcare Co. Ltd., Kyoto, Japan) under conditions according to the standardized protocol.[23] All of the subjects abstained from eating and drinking for > 4 hours, taking caffeine, chocolate, alcohol and nicotine for > 48 hours, strenuous exercise for > 24 hours, and diuretics within 7 days before the test. They also had to empty their bladder 30 minutes before the test.[24] The subjects stood on a measurement platform with their knees and back straight while looking straight ahead. All of the subjects raised their arms to a horizontal level in front of them and extended their elbows out straight to form a 90° angle to the body. They remained still and did not move until the measurement was completed.[23]

ECG recording and HRV analysis

After resting in supine position, the electrodes were placed on the participants' wrists in a limb lead position on the right arm (RA) with a reference electrode

placed on the left leg (LL). A lead II ECG recorded all of the subjects for 20 min using 200 sampling-Hz frequency (Power lab and Lab chart V.8, AD Instrument, Sydney, Australia).[25] All of the subjects were instructed to breathe normally during the ECG recording, while lying in supine position in a controlled environment which was carried out in a room with controlled temperature. Frequency domain analyses were carried out in accordance with the guidelines determined by the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology.[26] The frequency domains had three major oscillatory components that detected total power (0 - 0.4 Hz); a high frequency (HF, 0.15 - 0.40 Hz) band component representing cardiac parasympathetic activity, low frequency (LF, 0.04 - 0.15 Hz) band representing sympathetic activity and LF/HF, the mixture of sympathetic activation and parasympathetic modulation. [26]

Pulmonary function

The pulmonary function test was performed using a spirometer (Spiro Master PC-10, Chest M.I., Inc, Tokyo, Japan). All of the subjects were familiarized with the instrument and the protocols and in accordance with the American Thoracic Society criteria.[27] A breathing tube was inserted into the subjects' mouth before instructing them to inhale deeply and rapidly in order to access functional residual capacity, prior to starting forced exhalation. The highest level of forced vital capacity (FVC), forced expiratory volume in 1 second (FEV1) and %FEV1/ FVC were taken independently from the curves in order to determine pulmonary function parameters.[28] All of the subjects were recorded in three trials, and the best value was used in the analysis.

Maximal oxygen consumption

The cycle ergometer (Monark 828E, *Monark Exercise AB, Vansbro*, Sweden) program was set according to submamimal Astrand-Ryhming test protocol, with a warm-up period of two minutes at gravitational level (0.0% weight).[29] The test was performed initially at 1 Kilo Pond intensity for six minutes, with the heart rate kept at between 120 and 170 beats/minute. Heart rate (HR) was recorded throughout each stage of the test.[30] The final two minutes of the test determined the correct maximum oxygen consumption ($VO_{2\,max}$) during the mean steady stage of the HR. $VO_{2\,max}$ was calculated from the following formula: Corrected VO_{2max} (L/min) = VO_{2max} (Nomogram) X Age Factor.[29]

Statistical analysis

All of the data were expressed as mean ± standard deviation (SD). All of the statistical analyses were performed using SPSS program. The data distribution was analyzed by the Kolmogorov Smirnov test. Two-way ANOVA was used to determine aerobic capacity, pulmonary and autonomic function values between nonsmokers and smokers, after stratifying all of the subjects according to their BMI. *P*- value < 0.05 was considered statistically significant difference.

Results

Demographic data

Descriptive data for the normal and overweight smokers and nonsmokers are shown in Table 1. The BMI and weight in overweight nonsmokers and smokers were higher than those in smokers and nonsmokers of normal weight ($P < 0.05$). There was no significant difference in age and height among the groups. Cardiovascular parameters [HR, and systolic and diastolic blood pressure] in the normal weight nonsmokers and smokers also were not significantly different ($P > 0.05$). There was no significant difference in duration or frequency of smoking and nicotine dependence level among the groups ($P > 0.05$).

Body composition analysis

The main effect on body composition by BMI and smoking showed significant differences. Body fat and visceral fat percentages in the overweight BMI ($f = 21.148$, $P = 0.001$, $f = 32.364$, $P = 0.001$) and smoking groups ($f = 15.435$, $P = 0.001$, $f = 20.565$, $P = 0.001$) were significantly higher when compared with normal BMI and the nonsmoking groups. There was no significant difference of fat free mass in overweight group, compared with normal BMI group ($f = 1.903$, $P = 0.172$). Smokers showed significantly higher visceral fat along with lower fat free mass ($f = 19.172$, $P = 0.001$), when compared with nonsmokers (Table 2). There were no effects of interaction between BMI and smoking on body fat percentage ($f = 2.558$, $P = 0.114$), and fat free mass ($f = 0.064$, $P = 0.802$), but the effects of interaction with smoking were shown on visceral fat ($f = 6.052$, $P = 0.016$).

Table 1. General characteristics of the participants.

Parameters	NWN n =18	OWN n =18	NWS n =18	OWS n =18	*P* for BMI	*P* for smoking	*P* for interaction
Age (year)	43.72 ± 6.84	44.83 ± 6.97	45.17 ± 7.13	43.89 ± 6.75	0.495	0.431	0.902
Height (Centimeter)	168.77 ± 5.86	167.51 ± 5.34	167.78 ± 5.58	166.17 ± 7.02	0.413	0.533	0.755
Weight (Kilogram)	63.45 ± 4.82	68.51 ± 5.28	62.06 ± 5.15	66.94 ± 6.18	0.001	0.249	0.948
BMI (Kg/m^2)	22.20 ± 0.76	24.39 ± 0.65	22.01 ± 0.83	24.23 ± 0.75	0.001	0.289	0.789
HR (BPM)	72.28 ± 6.86	73.44 ± 8.08	73.78 ± 5.75	76.16 ± 7.89	0.274	0.194	0.706
SBP (mmHg)	108.11 ± 7.07	110.83 ± 6.69	110.56 ± 6.37	112.78 ± 8.26	0.203	0.266	0.758
DBP (mmHg)	71.38 ± 6.37	73.05 ± 7.16	73.44 ± 6.84	75.11 ± 5.65	0.451	0.102	0.746
Smoking measures	N/A	N/A	20.22 ± 6.38	17.83 ± 5.92			
Smoking duration (years)							
Current smoking frequency (cigarettes/day)	N/A	N/A	13.05 ± 2.58	12.17 ± 2.79			
Nicotine dependence level	N/A	N/A	6.25 ± 1.43	5.44 ± 1.21			

NWN (normal-weight nonsmokers), OWN (overweight nonsmokers), NWS (normal-weight smokers), OWS (overweight smokers), BMI (body mass index), Kg/m^2 (kilogram per square meter), HR (heart rate), BPM (beats per min), SBP (systolic blood pressure), DBP (diastolic blood pressure), mmHg (millimeter of mercury), N/A (not applicable)

Table 2. Bioelectrical body composition analysis.

Parameters	NWN n =18	OWN n =18	NWS n = 18	OWS n = 18	*P* for BMI	*P* for smoking	*P* for interaction
TBF (%)	16.40 ± 3.57	23.12 ± 3.76	22.14 ± 4.74	26.42 ± 4.18	0.001	0.001	0.114
VF (%)	6.19 ± 2.26	11.33 ± 2.51	10.19 ± 3.04	12.51 ± 2.63	0.001	0.001	0.016
FFM (%)	34.31 ± 3.48	32.60 ± 3.42	27.65 ± 3.71	26.18 ± 4.13	0.172	0.001	0.802

NWN (normal-weight nonsmokers), OWN (overweight nonsmokers), NWS (normal-weight smokers), OWS (overweight smokers), TBF (total body fat), VF (visceral fat), FFM (fat free mass), % (percentage)

Table 3. Pulmonary function and maximal oxygen consumption.

Parameters	NWN n = 18	OWN n = 18	NWS n = 18	OWS n = 18	*P* for BMI	*P* for smoking	*P* for interaction
FEV1 (%predict)	89.78 ± 12.63	87.78 ± 10.45	80.89 ± 10.58	76.84 ± 10.12	0.181	0.001	0.564
FVC (%predict)	95.66 ± 7.31	90.55 ± 8.67	93.44 ± 8.24	91.23 ± 7.84	0.023	0.988	0.715
FEV1/FVC (%)	93.77 ± 9.94	96.31 ± 10.14	85.26 ± 8.67	83.10 ± 7.02	0.818	0.001	0.215
VO_{2max} (ml/kg/min)	37.15 ± 7.48	31.20 ± 5.36	30.18 ± 6.64	28.82 ± 5.57	0.016	0.002	0.123

NWN (normal-weight nonsmokers), OWN (overweight nonsmokers), NWS (normal-weight smokers), OWS (overweight smokers), FEV1 (forced expiratory volume in one second), FVC (forced vital capacity), FEV1/FVC (forced expiratory volume in 1 second/forced vital capacity), VO_{2max} (maximal oxygen consumption), % (percentage), ml/kg/min (milliliters per kilogram per minute)

Table 4. Heart rate variability analysis.

Parameters	NWN n = 18	OWN n = 18	NWS n = 18	OWS n = 18	*P* for BMI	*P* for smoking	*P* for interaction
SDNN (ms)	68.03 ± 10.79	55.48 ± 15.43	52.76 ± 12.78	47.66 ± 14.05	0.006	0.001	0.238
RMSSD (ms)	56.74 ± 10.38	48.42 ± 14.11	42.01 ± 11.17	36.81 ± 12.74	0.022	0.001	0.588
LF (n.u.)	41.98 ± 13.82	61.01 ± 13.77	63.25 ± 11.95	67.20 ± 12.16	0.001	0.001	0.016
HF (n.u.)	62.40 ± 11.72	53.36 ± 14.62	50.57 ± 10.54	45.93 ± 11.18	0.019	0.001	0.445
LF/HF	0.71 ± 0.29	1.13 ± 0.33	1.28 ± 0.37	1.51 ± 0.31	0.001	0.001	0.195

NWN (normal-weight nonsmokers), OWN (overweight nonsmokers), NWS (normal-weight smokers), OWS (overweight smokers), SDNN (standard deviation of N to N interval), RMSSD (root mean square of successive difference), LF (low frequency), HF (high frequency), LF/HF (ratio of low and high frequency), ms (millisecond)

Pulmonary function and aerobic capacity among the various groups

There was significant effect of overweight BMI on FVC (f = 5.448, P = 0.023), whereas no observed significantly in FEV1 and its ratio (f = 1.826, P = 0.181, f = 0.053, P = 0.818). The FEV1 and FEV1/FVC ratio (f = 16.091, P = 0.001 and f = 27.546, P = 0.001, respectively) were lower in smokers than in nonsmokers. The differences were not observed in FVC by smoking (f = 0.018, P = 0.988). There were no effects of interaction between overweight BMI and smoking on FEV1 (f = 0.335, P = 0.564), FVC (f = 0.135, P = 0.715) or its ratio (f = 1.547, P = 0.215).

The 2-way ANOVA revealed the main effects of overweight BMI and smoking on VO_{2max}, which were significantly lower in the overweight BMI and smoking groups, compared with the normal BMI and nonsmoking groups (f = 6.158, P = 0.016; f = 10.107, P = 0.002, respectively). There was no effect of interaction observed on VO_{2max} (f = 2.436, P = 0.123) as shown in Table 3.

Heart rate variability

The time and frequency domain of HRV analyses are presented in Table 4. A statistically significant difference between overweight BMI and smoking was found in SDNN (f = 7.956, P = 0.006; f = 13.628, P = 0.001) and RMSSD (f = 5.538, P = 0.022; f = 21.013, P = 0.001). There were no effects of interaction on SDNN (f = 1.419, P = 0.238) and RMSSD (f = 0.296, P = 0.588).

There was a significant effect of overweight BMI and smoking on LF (f = 14.158, P = 0.001; f = 20.247, P = 0.001) and the LF/HF ratio (f = 19.656, P = 0.001; f = 31.614, P = 0.001, respectively). HF was higher in the overweight BMI and smoking groups than in the normal BMI and nonsmoking groups (f = 5.745, P = 0.019, f = 11.368, P = 0.001 respectively). Interactive effects of overweight BMI and smoking on LF/HF ratio and HF were not observed (f = 1.709, P = 0.195; and f = 0.589, f = 0.445 respectively). However, there was an effect of interaction between overweight BMI and smoking on the LF ratio (f = 6.095, P = 0.016) (Table 4).

Discussion

The major findings of this study are as follows: overweight BMI and smoking developed body composition imbalance by increasing body fat and visceral fat percentages, and aggravated autonomic imbalance by decreasing SDNN and RMSSD, HF and increasing LF and the LF/HF. Furthermore, smoking also decreased FEV1 and FEV1/FVC, VO_{2max} and fat free mass. Higher BMI increased fat accumulation, FVC and decreased VO_{2max}, but was not statistically significant in FEV1, FEV1/FVC. This study found the effect of interaction between overweight BMI and smoking on visceral fat, the LF value in physically inactive subjects.

HRV is a predictor of morbidity and mortality rate form cardiovascular disease (CVD). [5] SDNN reflects all the components responsible for both the sympathetic and parasympathetic nervous systems, while RMSSD represents an estimate of parasympathetic activity. [5] The main effects of smoking and overweight BMI on autonomic function by reducing SDNN and RMSSD. LF and the LF/HF ratio were increased, meanwhile, HF was lower by smoking and overweight, associated with decreased parasympathetic activity.[5] This study also found that overweight BMI and smoking increased body fat percentages, suggesting that body composition imbalance by increased BMI and smoking caused autonomic dysfunction.[31] This finding was associated with a previous study, with the reduction of HRV being correlated with increased fat mass in obese, overweight groups.[6, 32] A possible explanation for this may be increased fat deposition leads to the release of more oxidative stress and inflammatory cytokines in blood circulation, and is a contributing factor of autonomic dysregulation.[33]

The health and physical fitness of middle-aged adults, who will be the future elderly, are important for not only those individuals concerned, but also society as a whole.[34] The study found that smoking showed the effects of interaction with overweight BMI on visceral fat percentage as well as the LF value, indicating increased sympathetic activity in physically inactive smokers. It was suggested, therefore that fat in the visceral organs plays a role in autonomic modulation in physically inactive subjects.[10] Visceral adiposity is harmful to health and a contributing factor in developing cardiovascular disease (CVD): therefore, a cigarette smoking program or health promotion for middle-aged males should control fat in the visceral organs to prevent diseases.[35]

Furthermore, the findings found that smoking decreased pulmonary function by diminished FEV1, and the ratio of FEV1/FVC. Also, fat-free mass was

lower in smokers associated with decreased aerobic capacity which are important to body performing physical activities and exercise in daily life.[35] Overweight BMI also shows unfavorable effects on FVC and visceral fat, suggesting that increased BMI in middle-age males also increased risk of pulmonary impairment. Our study suggested that not only smoking caused respiratory function to decline but also increased BMI in middle-aged males resulting in decreased pulmonary function. Thus fat accommodation especially in visceral organ by smoking and overweight BMI in middle-aged males caused pulmonary function and physical fitness decline. [35]

All of the findings in this study demonstrated that overweight and smoking decreased aerobic capacity, autonomic function and pulmonary function which diminished in performing daily physical activity. Importantly, smoking behavior in physically inactive subjects developed body composition imbalance by decreasing fat- free mass and increasing body fat, especially fat accommodation in visceral organs. It can be a relative factor to develop cardiovascular diseases.[10] Therefore, further investigation is needed to warrant clinical application, and further studies into the evaluation and promotion of overweight and smoking should focus on body composition imbalance along with quitting smoke.

The study has some limitations, however. There was a small sample size, which might have affected interaction outcomes. Because the study was cross-sectional in nature, it is not possible to explain the long term effects of smoking and overweight. Further studies should be conducted with larger sample sizes, using a prospective cohort study to determine effects.

Conclusion

Increased visceral fat in physically inactive males caused by cigarette smoking and overweight can be related tentatively to the development of CVD in the future. Therefore, these results suggest that ceasing smoking and controlling body composition, especially reducing visceral fat, decrease the risk of CVD in physically inactive males.

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Conflicts of interest

The authors declare no conflict of interest.

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