ORIGINAL RESEARCH

Cigarette smoking exacerbates health problems in young men

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Abstract

Purpose: To examine the hypothesis that smoking exacerbates health problems in young male smokers (age range, 18.6-22.8 yr; mean, 19.4 yr).

Methods: 1169 subjects were recruited, 25.41 % were smokers (2-15 cigarettes daily). All subjects were examined for body mass index, blood pressure, exhaled carbon monoxide content (carboxyl hemoglobin), blood hematology and biochemistry.

Results: Data for WBC ($P<0.001$), hemoglobin ($P=0.001$), hematocrit ($P=0.004$), MCV ($P=0.001$), MCH ($P=0.003$), COHB% ($P<0.001$), albumin/globulin ($P<0.001$) and triglyceride ($P<0.001$) were higher for smokers than non-smokers, while total-bilirubin ($P<0.001$), total protein ($P<0.001$) and globulin ($P<0.001$) were markedly lower. The results of WBC ($r=0.164$, $P<0.004$), COHB% ($r=0.958$, $P<0.001$), gamma glutamyl transpeptidase ($r=0.159$, $P=0.006$), alkaline-phosphatase ($r=-0.154$, $P=0.008$) and triglyceride ($r=0.144$, $P<0.001$) were closely correlated with number of cigarettes smoked daily. Investigation of associations with illness revealed that young smokers had an increased risk of hypertriglyceridemia to young non-smokers (adjusted ORs, 2.124; 95% CIs, 1.414-3.190), hyperglycemia (adjusted ORs, 1.980; 95% CIs, 0.803-4.901), neutrophilia (adjusted ORs, 1.947; 95% CIs, 1.248-3.037), RBC macrocytosis (adjusted ORs, 1.929; 95% CIs, 1.137-3.275), hyperchromia (adjusted

ORs, 1.844; 95% CIs, 1.412-2.407) and polycythemia (adjusted ORs, 1.314; 95% CIs, 0.805-2.145) (all $P<0.05$ for linear trends).

Conclusion: The findings emphasize the importance of increasing surveillance of diseases exacerbated by smoking and reducing smoking in the young to prevent cardiovascular illnesses, metabolite disorders and other clinical diseases.

Cigarette smoking poses a serious public health risk and associated with many illnesses. Smoking-associated cancer and other illnesses lead to over 440,000 deaths annually in the U.S.^{1, 2} Previous studies have demonstrated that cigarette smoking may have numerous adverse effects, including inducing organ injury, for example chronic obstructive pulmonary disease (COPD),³ ischemic heart disease,⁴⁻⁶ atherosclerosis,^{7,8} hepatitis,⁹ and pancreatitis.¹⁰ Smoking can also exacerbate infections such as asthma,⁸ hypertension,¹¹ and tuberculosis (TB).¹² Moreover, smoking stimulates the production of inflammatory factors including interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor- α

(TNF- α)⁹ and increases fibrinolytic activity in the blood.¹³ Smoking is also associated with cell injury and autoimmune diseases including rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, Grave's hyperthyroidism and primary biliary cirrhosis.¹⁴⁻¹⁶ Additionally, accumulated damage from continuous cigarette smoking exhausts cellular defense and repair functions, increases the risk of carcinoma,^{9, 17, 18} reduces life span by an average of 7 yr and reduces disease-free life by 14 yr.¹⁹ Moreover, smoking during pregnancy increases the rate of respiratory infection and asthma and reduces body weight in newborn infants.^{20, 21} Recent reports have identified a growing prevalence of lung cancer and respiratory disease in adult and elderly patients,²²⁻²⁴ particularly smokers who started at an early age.

Few studies have assessed the influence of smoking on blood biochemistry in young males. This study thus elucidates the potential biohazards associated with smoking in young males through physical and blood biochemical analysis.

Methods

Ethics approval for this study was obtained from the Ethical Review Committee of Yuanpei University for studies involving human subjects. The investigation compared the results of physical examinations in 1169 volunteer male students attending a university in northern Taiwan (age range, 18.6-22.8 yr; mean age, 19.4 yr). Of the subject group, 25.41% were currently smoking at least 2 cigarettes daily for at least eight months prior to the examination. All subjects were examined for body mass index (BMI), blood pressure, exhaled CO content (carboxyl hemoglobin) and blood content via hematological and biochemistry tests. The hematological test profiles comprised complete blood count (CBC), including WBC, RBC and platelet count, hemoglobin (Hb) level and hematocrit (Hct), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV) and WBC differential count

(DC) including neutrophil% (Neut%), lymphocyte% (Lym%) and mixed granulocytes% (MDX%). Biochemistry tests included liver function test (LFT), kidney function test (KFT), blood lipids and blood glucose (BG) test. Among these tests, LFT included total bilirubin (T-Bili), total protein (TP), albumin (Alb), globulin (Glo), albumin/globulin ratio (A/G), alkaline phosphatase (Alk-P), aspartate aminotransferase (AST), alanine aminotransferase (ALT) and gamma glutamyl transpeptidase (GGT); KFT included blood urea nitrogen (BUN), serum creatinine (Scr) and uric acid (UA); lipid tests included total cholesterol (TC) and triglyceride (TG).

Tests were performed as follows: BMI was calculated as kilograms of body weight divided by height in meters. Systolic and diastolic blood pressure (mmHg) was measured twice at ten minute intervals. Blood pressure was measured on the right arm using a sphygmomanometer, and a mean value was recorded. Exhaled CO was measured by a Micro CO meter (MCO2, Micro Medical Ltd, Rochester, Kent, UK) with sensitivity of 1-100 parts per million (ppm, by volume) CO or via corresponding saturation of carboxyhemoglobin concentration (COHb%).^{25, 26} All blood samples were obtained from the forearm following fasting and collected using EDTA treated vacuum tubes and plain vacuum tubes (Becton Dickinson, Franklin Lakes, NJ, USA). The CBC items were counted according to the principles of electrical impedance measurements and multi-angle polarized scatter separation (MSPSS) using fully automated cell counters (Abbott Diagnostics Division, Santa Clara, CA, USA).^{27, 28} Biochemical data other than BG were measured by standard procedures using commercially available kits (Wako Chemical Co., Osaka, Japan) on a Hitachi 7150 Autoanalyzer (Hitachi Ltd. Tokyo, Japan), and BG was measured via a glucose oxidase method using the Beckman Glucose Analyzer (Beckman Instruments, Irvine, CA). Table 1 lists the defined reference values for all tests, and table 2 displays clinical definitions of hypertriglyceridemia, hyperglycemia, neutrophilia, RBC macrocytosis, hyperchro-

mia, polycythemia, renal malfunction, obesity, hypercholesterolemia, hypertension, hepatitis and hyperuricemia. These specific disorders were chosen for analysis owing to their apparent association with cardiovascular disease and organic injuries.

The statistical significance of the descriptive statistical data was analyzed by the independent Student *t* test and Chi-square test, and logistic regression with adjustments for potential confounders was used to calculate the adjusted odds ratios (ORs) and 95% confidence intervals (CIs). All data were processed via the statistical software package SPSS13 (Chicago, IL, USA). The level of statistical significance was set to a *P* value less than 0.01 or 0.05.

Results

Data of biomedical validation

Of the 1169 male subjects enrolled in this study, 25.41 % were smokers (the sample contained 872 non-smokers and 297 smokers, 1: 0.34, with mean age 19.4 years old). Smokers were defined as those currently smoking 2-15 cigarettes daily and who had sustained this smoking habit for at least eight months prior to the investigation. WBC ($P<0.001$), Hb ($P=0.001$), Hct ($P=0.004$), MCV ($P=0.001$), MCH ($P=0.003$), COHb% ($P<0.001$), A/G ($P<0.001$) and TG ($P<0.001$) were higher for smokers than for non-smokers, but T-Bili ($P<0.001$), TP ($P<0.001$) and Glo ($P<0.001$) were lower for smokers than for non-smokers (Table 1).

Correlation with dose-response of cigarette smoked

For 297 smokers who had smoked 2-15 cigarettes daily for at least eight months before the examination, the results for WBC ($r=0.164$, $P<0.004$), COHb% ($r=0.958$, $P<0.001$), GGT ($r=0.159$, $P=0.006$), Alk-P ($r=-0.154$, $P=0.008$) and TG ($r=0.144$, $P<0.001$) were closely correlated with the intensity of their smoking habit (Fig 1).

Prevalence of and increasing risk of developing illnesses among smokers

The test data exceeding the upper limits of reference values are evaluated in various illnesses (listed in Table 2). Those illnesses, in order of frequency, included hyperchromia (39.10%), hyperuricemia (35.58%), renal malfunction (25.84%), hypertension (21.99%), obesity (18.05%), hypercholesterolemia (16.68%), hepatitis (13.60%), hypertriglyceridemia (9.41%), neutrophilia (7.78%), polycythemia (7.02%), RBC macrocytosis (5.30%) and hyperglycemia (1.71%) (Fig 2). Smokers exhibited the risks of these illnesses, as detailed below.

Association of hypertriglyceridemia and smoking

Hypertriglyceridemia was observed in 9.41% of subjects (14.81% of smokers vs 7.57% of non-smokers; adjusted ORs, 2.124; 95% CIs 1.414-3.190) (Table 2). Of the 110 cases of triglyceridemia, Hb ($P=0.049$), MCV ($P=0.028$), MDX% (mainly monocytes) ($P=0.005$), COHb% ($P<0.001$), A/G ($P=0.001$) and TG ($P=0.025$) were higher in smokers than non-smokers, but Neut% ($P=0.032$), T-Bili ($P=0.035$), TP ($P<0.001$), Glo ($P<0.001$) and TC ($P=0.023$) were considerably lower in smokers than in non-smokers.

Association of hyperglycemia and smoking

Hyperglycemia was observed in 1.72% of subjects (2.69% of smokers vs 1.38% of non-smokers; adjusted ORs, 1.98; 95% CIs, 0.803-4.901) (Table 2). For the 20 cases of hyperglycemia, COHb% was higher for smokers than non-smokers ($p=0.002$).

Association of neutrophilia and smoking

Neutrophilia was observed in 7.78% of subjects (11.78% of smokers vs 6.42% of non-smokers; adjusted ORs, 1.947; 95% CIs, 1.248-3.037) (Table 2). For the 91 subjects with neutrophilia, Lym% ($P=0.025$) and COHb% ($P<0.001$) were higher in smokers than non-smokers, but Neut% ($P=0.013$) and T-Bili

TABLE 1. Clinical, laboratory, and hemodynamic characteristics of smokers versus non-smokers.

Test items	Reference values	Mean $\pm$ SD		medium	significant (2-tailed) <i>p</i> value
		Non-smokers <i>n</i> =872(74.59%)	Smokers <i>n</i> =297(25.41%)		
Body height		171.33 $\pm$ 5.97	170.77 $\pm$ 5.75	171.20	0.151
Body weight		68.33 $\pm$ 15.21	66.95 $\pm$ 13.21	64.90	0.134
BMI	19-24	23.24 $\pm$ 4.79	22.97 $\pm$ 4.78	22.20	0.376
Blood pressure					
Systolic	<135 mmHg	116.81 $\pm$ 13.37	117.84 $\pm$ 13.05	118	0.241
Diastolic	<85 mmHg	74.22 $\pm$ 10.21	74.83 $\pm$ 9.67	74	0.351
CBC					
WBC	5.0-9.0 $\times 10^3$ /cumm	7.11 $\pm$ 1.75	7.81 $\pm$ 1.78	7.10	<0.001*
RBC	3.8-5.3 $\times 10^6$ /cumm	5.25 $\pm$ 0.46	5.24 $\pm$ 0.47	5.20	0.655
Hb	12-16 g/dL	15.77 $\pm$ 1.08	16.02 $\pm$ 1.10	15.90	0.001*
Hct	38-48%	47.10 $\pm$ 2.91	47.68 $\pm$ 2.98	47.10	0.004*
MCV	82-99 fl	89.90 $\pm$ 6.73	91.49 $\pm$ 7.19	90.90	0.001*
MCH	27-32 pg	30.21 $\pm$ 2.47	30.77 $\pm$ 2.86	30.80	0.003*
MCHC	32-36%	33.59 $\pm$ 2.00	33.62 $\pm$ 1.57	33.80	0.769
Platelet	15-40 $\times 10^3$ /cumm	267.70 $\pm$ 55.94	261.71 $\pm$ 56.35	263.00	0.113
WBC DC					
Neutrophil%	50-75%	58.11 $\pm$ 8.57	57.08 $\pm$ 9.00	58.40	0.084
Lymphocyte%	25-45%	31.75 $\pm$ 7.68	32.30 $\pm$ 7.68	31.50	0.292
Middle cell%	5-12%	10.11 $\pm$ 3.31	10.60 $\pm$ 4.48	9.70	0.084
COHb%	<1%	0.23 $\pm$ 0.26	2.26 $\pm$ 1.17	0.32	<0.001*
Liver function tests					
T-Bili	0.3-1.2 mg/dl	0.87 $\pm$ 0.43	0.72 $\pm$ 0.40	0.74	<0.001*
Total-protein	6.7-8.3 g/dl	7.61 $\pm$ 0.34	7.46 $\pm$ 0.36	7.60	<0.001*
Albumin	3.5-5.0 g/dl	4.64 $\pm$ 0.20	4.62 $\pm$ 0.22	4.64	0.223
Globulin	1.5-4.0 g/dl	2.97 $\pm$ 0.28	2.84 $\pm$ 0.27	2.90	<0.001*
A/G ratio	1.0-1.9	1.58 $\pm$ 0.16	1.64 $\pm$ 0.16	1.60	<0.001*
Alk-P	40-128 IU/L	88.49 $\pm$ 23.74	86.48 $\pm$ 21.06	86.00	0.171
AST	8-38 IU/L	20.03 $\pm$ 13.82	20.24 $\pm$ 14.58	16.80	0.824
ALT	4-40 IU/L	26.99 $\pm$ 25.24	27.64 $\pm$ 34.47	18.20	0.765
GGT	7-55 IU/L	23.69 $\pm$ 17.41	25.78 $\pm$ 16.95	19.40	0.069
Kidney function tests					
BUN	8-20 mg/dl	12.68 $\pm$ 2.67	12.84 $\pm$ 2.49	12.40	0.356
Scr	0.5-1.2 mg/dl	1.14 $\pm$ 0.15	1.15 $\pm$ 0.10	1.14	0.147
UA	2.5-7.0 mg/dl	6.68 $\pm$ 1.48	6.54 $\pm$ 1.34	6.50	0.122
Lipids					
Total cholesterol	50-200 mg/dl	172.74 $\pm$ 31.21	173.64 $\pm$ 32.59	168.00	0.676
Triglyceride	50-150 mg/dl	84.39 $\pm$ 50.87	109.18 $\pm$ 143.65	70.00	<0.001*
Blood sugar					
Fasting glucose	70-110 mg/dl	89.27 $\pm$ 14.42	91.00 $\pm$ 14.09	88.00	0.069

Significant at <0.01 level (2-tailed)

Abbreviations: SD: standard deviation; BMI, body mass index; CBC, complete blood count; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; Hct, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; DC, WBC differential count; Neut%, neutrophil; Lym%, lymphocyte; MDX%, mixed granulocytes; COHb, carboxyl hemoglobin; T-Bili, total bilirubin; A/G, albumin/globulin; Alk-P, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma glutamyl transpeptidase; BUN, blood urea nitrogen; Scr, serum creatinine; UA, uric acid.

FIGURE 1. Linear regression of levels of WBC, CoHb%, Alk-P, GGT and TG were based on level of cigarette consumption. X-axis - daily cigarette consumption; Y-axis - concentration of test items.

TABLE 2. Relationships with various illnesses among non-smoking subjects, smoking subjects and their outcomes.

Cases	Non-smokers <i>n</i> =872	Smokers <i>n</i> =297	Odds ratio	95% CI*
Hypertriglyceridemia TG >150 mg/dL	66 (7.57%)	44 (14.81%)	2.124	1.414-3.190
Hyperglycemia Blood glucose ≥126 mg/dL	12 (1.38%)	8 (2.69%)	1.98	0.803-4.901
Neutrophilia WBC>10.0×10 ⁶ /cumm	56 (6.42%)	35 (11.78%)	1.947	1.248-3.037
RBC macrocytosis MCV > 99 fl	38 (4.36%)	24 (8.08%)	1.929	1.137-3.275
Hyperchromia Hb >16 gm/dL or MCH > 32 pg	308 (35.32%)	149 (50.17%)	1.844	1.412-2.407
Polycythemia Hct >55% or RBC>5.5×10 ⁶ /cumm	57 (6.54%)	25 (8.42%)	1.314	0.805-2.145
Renal malfunction Scr >1.2 mg/dL	218 (25.00%)	84 (28.28%)	1.183	0.881-1.590
Obesity BMI >27	154 (17.66%)	57(19.19%)	1.107	0.790-1.551
Hypercholesterolemia T-Chol>200 mg/dL	147 (16.86%)	48 (16.16%)	0.951	0.666-1.358
Hypertension systolic pressure ≥135 mmHg or diastolic pressure ≥85 mmHg	194 (22.25%)	63 (21.21%)	0.941	0.683-1.297
Hepatitis ALT >40 IU/L	123 (14.11%)	36 (12.12%)	0.840	0.565-1.250
Hyperuricemia UA > 7.0 mg/dL	325 (37.27%)	91 (30.64%)	0.743	0.561-0.986

*95%CI: 95% confidence interval

($P=0.005$) were markedly lower among smokers than non-smokers.

Association of red blood cell macrocytosis and smoking

RBC macrocytosis was observed in 4.45% (8.08% of smokers vs 4.36% of non-smokers; adjusted ORs, 1.929; 95% CIs, 1.137-3.275) (Table 2). Analysis of 62 RBC macrocytosis data revealed that MCV ($P=0.044$), MCH ($P=0.015$), Lym% ($P=0.003$), COHb% ($P<0.001$), A/G ($P=0.002$) and Alk-P ($P=0.027$) were higher in smokers than non-smokers, but Neut% ($P=0.011$), T-Bili ($P=0.005$), and Glo ($P=0.005$) were considerably lower in smokers than in non-smokers.

Association of hyperchromia and smoking

Hyperchromia was observed in 39.10% of all participants (50.17% of smokers vs 35.32% of non-smokers; adjusted ORs, 1.844; 95% CIs, 1.412-2.407) (Table 2). In the 457 subjects with hyperchromia, WBC ($P=0.001$), MCV ($P<0.001$), MCH ($P<0.001$), MDX% ($P=0.036$), COHb% ($P<0.001$), A/G ($p=0.001$) and TG ($P=0.013$) were higher among smokers than non-smokers, but PLT ($p=0.038$), Neu% ($P=0.039$), T-Bili ($P<0.001$), TP ($P<0.001$) and Glo ($P<0.001$) were markedly lower in smokers than non-smokers.

FIGURE 2. Percentage of subjects, X axis, with hyperchromia, hyperuricemia, renal malfunction, hypertension, obesity, hypercholesterolemia, hepatitis, hypertriglyceridemia, neutrophilia, polycythemia, RBC macrocytosis and hyperglycemia. Blank rectangles - percentages of non-smokers (left number in parentheses) Grey rectangles - percentages of smokers (right number in parentheses) among all subjects.

Association of polycythemia and smoking

Polycythemia was noted in 7.02% of all subjects (8.42% of smokers vs 6.54% of non-smokers, adjusted ORs, 1.314; 95% CIs, 0.805-2.145) (Table 2). Among the 82 polycythemic subjects, COHb% was higher in smokers than non-smokers ($P<0.001$), but Neut% ($P=0.044$), TP ($P=0.024$), and Glo ($P=0.018$) were considerably lower in smokers than non-smokers.

Association of renal malfunction and smoking

Some 25.84% of subjects exhibited renal malfunction, (28.28% of smoker vs 25.00% of non-smokers; adjusted ORs, 1.183; 95% CIs, 0.881-1.590) (Table 2). Data for all 304 subjects with kidney malfunction dis-

played that WBC ($P=0.005$), Hb ($P=0.025$), MCV ($P=0.001$), MCH ($P<0.001$), COHb% ($P<0.001$), A/G ($P=0.002$) and TG ($p=0.045$) were higher among smokers than non-smokers, while T-Bili ($P=0.012$), TP ($P=0.001$), Glo ($P<0.001$) and Alk-P ($P=0.031$) were lower among smokers than non-smokers.

Association of obesity and smoking

Obesity was existed in 18.05% of subjects (19.19% of smokers vs 17.66% of non-smokers; adjusted ORs, 1.107; 95% CIs, 0.790-1.551) (Table 2). Of the 211 obese subjects, WBC ($P=0.010$), Hb ($P=0.022$), Hct ($P=0.022$), Lym% ($P=0.037$), COHb% ($P<0.001$), and TG ($P=0.046$) were higher in smokers than non-

smokers, but body height ($P=0.018$), body weight ($P=0.001$), BMI ($P=0.016$), TP ($P=0.040$) and Glo ($P=0.014$) were lower among smokers than non-smokers.

Association of hypercholesterolemia and smoking

Regarding the 16.68% of patients who were hypercholesterolemic, the prevalence of the condition among smokers did not differ from that among non-smokers (16.16% vs 16.86%; adjusted ORs, 0.951; 95% CIs, 0.666-1.358) (Table 2). However, for the patients with hypercholesterolemia, WBC ($P=0.001$), Hb ($P=0.001$), Hct ($P=0.016$), MCH ($P=0.027$), COHb% ($P<0.001$), A/G ($P=0.025$), and TG ($P=0.003$) were higher among smokers than non-smokers, but the values for T-Bili ($P=0.023$), TP ($P=0.001$) and Glo ($P<0.001$) were lower for smokers than non-smokers.

Association of hypertension and smoking

Hypertension was noted in 21.99% of participants (21.21% of smokers vs 22.25% of non-smokers; adjusted ORs, 0.941; 95% CIs, 0.683-1.297) (Table 2). Among the 257 subjects with hypertension, WBC ($P<0.001$), Hb ($P<0.001$), Hct ($P=0.011$), MCV ($P<0.001$), MCH ($P<0.001$), MCHC ($P=0.018$), COHb% ($P<0.001$), A/G ($P=0.005$) and TG ($P=0.019$) were higher among smokers than non-smokers, while body height ($P=0.043$), T-Bili ($P=0.015$), TP ($P=0.004$), Glo ($P<0.001$) and Alk-P ($P=0.036$) were lower among smokers than non-smokers.

Association of hepatitis and smoking

Hepatitis was observed in 13.60% of all subjects (12.12% of smokers vs 14.11% of non-smokers; Adjusted ORs, 0.840; 95% CIs, 0.565-1.205) (Table 2). Although the prevalence of subjects with ALT values exceeding 40 IU/L (the upper limit of reference value) did not differ between smokers and non-smokers, the values of subjects with hepatitis for Hb ($P=0.017$), COHb% ($P<0.001$) and A/G ($P=0.033$) were higher in

smokers than non-smokers. Meanwhile, TP ($P<0.001$) and Glo ($P<0.001$) were lower among smokers than non-smokers.

Association of hyperuricemia and smoking

Finally, the test results revealed hyperuricemia in 35.58% of subjects (30.64% of smokers vs 37.27% of non-smokers; adjusted ORs, 0.743; 95% CIs, 0.561-0.986) (Table 2). Data for all 416 patients revealed higher MCH ($P=0.015$), COHb% ($P<0.001$), A/G ($P<0.001$), TG ($P=0.018$) and BG ($P=0.030$) for smokers than for non-smokers, while Neut% ($P=0.042$), T-Bili ($P=0.047$), TP ($P<0.001$) and Glo ($P<0.001$) were lower among smokers than non-smokers.

Limitations

The limitations of this work include difficulty in differentiating the effects of secondhand or environmental tobacco smoke for non-smokers, the possibility of diseases unrelated with smoke and the possibility of uncontrolled confounding factors. This study also indicated that certain smoke-exacerbated diseases were more likely to affect females than males (data not shown). Future studies will be required to further analyze these limitations and findings.

Discussion

Main findings

Cigarette smoking has numerous adverse health effects and is reportedly the second leading cause of death worldwide.²⁹ This study found a 25.41% prevalence of smoking among young men, close to previous studies of college students in US³⁰ and middle east countries.^{31,32} Of all areas, smoking affected hematological values, including increasing the number of white blood cells, hemoglobin content (Hb, MCH and CoHb%) and RBC volume (MCV and Hct) (Table 1). Among these, white blood cell count and monoxycarboxyl hemoglobin content (COHB%) exhibited

positive dose dependence on cigarette-consumption (Fig 1). For biochemistry, TG level was increased (up to 24.78 mg/dL) by smoking (Table 1) and with GGT was positively and dose-dependently associated with smoking (Fig 1). Conversely, some tests related to liver function, including T-Bili, TP, Glo and A/G were negatively correlated with smoking (Table 1), and Alk-P level was inversely and dose-dependently correlated with smoking (Fig 1). Regarding illness, subjects suffered from hyperchromia, hyperuricemia, renal malfunction, hypertension, obesity, hypercholesterolemia, hepatitis, hypertriglyceridemia, neutrophilia, polycythemia, RBC macrocytosis and hyperglycemia (39.10%, 35.58%, 25.84%, 21.99%, 18.05%, 16.68%, 13.60%, 9.41%, 7.78%, 7.02%, 5.30%, 7.71%, respectively) (Fig 2). Furthermore, this study demonstrated that smoking increased the risks of hypertriglyceridemia, hyperglycemia, neutrophilia, RBC macrocytosis, hyperchromia and polycythemia in young males (Table 2).

Cigarette smoke contains about 4,000 substances, among which carbon monoxide (CO) and tars are the main toxic substances.³³ CO can diffuse rapidly across alveolar capillaries, bind firmly to Hb (with binding ability 200-250 times greater than that of oxygen) forming COHb, and is a leading cause of tissue hypoxia.^{22, 34} The reference level of COHb% in the blood is < 1%, but can increase by 5% per pack smoked daily, representing an increase of 10-15% in a heavy smoker.^{35, 36} This study demonstrated that, in smokers, COHb% was raised (Table 1), was closely correlated with cigarette dosage (Fig 1), and was high in illnesses described in Fig. 2. Besides high COHb%, smokers were more likely to suffer RBC macrocytosis hyperchromia and polycythemia (Table 2). As previously reported, RBC macrocytosis, hyperchromia and polycythemia were associated with cardiovascular diseases,^{6, 37} hepatitis⁹, pulmonary disease³⁸ and renal glomerular cell injury.³⁹ As in previous studies, this investigation found that T-Bili, TP and Glo levels were all markedly lower in smokers than non-smokers, as were hepatitis and renal malfunction. Alk-

P was inversely corrected to cigarette consumption (Fig 1). However, the mechanisms by which varying protein levels are associated with these illnesses in smokers are poorly understood. Furthermore, smokers had higher WBC than non-smokers (Table 1), which was dose-dependent, and exhibited an increased risk of neutrophilia (Table 2, Fig 1) which is an indicator of tar induced inflammation^{33, 40-41}.

Hypertriglyceridemia is an important cardiovascular risk factor,⁴² and is strongly associated with cigarette smoking among the aged.⁴³ This study found high levels of blood TG in young smokers (Table 1), and a positive correlation between measured concentration and dose of cigarette consumption (Fig 1). Among hypertriglyceridemia subjects, smoker monocyte, Hb level, RBC volume (MCV) and CoHb% were higher in smokers than in non-smokers. Monocytes are the main cells that damage vascular cells and cause atherosclerosis when they penetrate the endothelial cells of blood vessels and transform into foam cells.⁴⁴ Thus, large amounts of monocytes would be a exacerbation factor for smokers with hypertriglyceridemia. Moreover, high Hb, MCV and CoHb% concentration could increase vascular loading and blood viscosity in long term smokers. Young male smokers are expected to face an increased risk of developing cardiovascular disease, especially atherosclerosis.^{7, 45, 46} Although fasting blood glucose levels did not differ between smokers and non-smoker, smokers had an increased risk of suffering hyperglycemia, as previously report in patients with type 2 diabetes mellitus.⁴³ However, no difference was noted in blood cholesterol concentrations between smokers and non-smokers. Smokers did not have an increased risk of hypercholesterolemia (Table 2). On the other hand, smokers with hypercholesterolemia had higher WBC, Hb, MCH, Hct content, COHb% and TG than did non-smokers with hypercholesterolemia. This phenomenon demonstrated that young male smokers with a long history of hypercholesterolemia had a strongly increased risk of suffering metabolic syndrome and cardiovascular diseases.⁴³

Obesity and hypertension are risk factors closely associated with cardiovascular diseases.^{47, 37} While smoking is allegedly helpful in controlling weight,⁴⁸ the present study failed to find any difference in BMI and blood pressure between young smokers and non-smokers (Table 1). However, subsequent analysis of the findings for subjects with both hypertension and obesity revealed that smokers had higher WBC, Hb, Hct, MCH (hypertension only), MCV (hypertension only), COHb% and TG, all of which were predicted to be vital factors exacerbating health in young men.

Finally, this investigation demonstrated that young smokers suffered disproportionate health risks compared with non-smokers, particularly with regard to cardiovascular disease, metabolic disorders, RBC disorders and inflammations. If present rates of smoking continue, by 2025, the death toll from smoking is expected to reach 10 million globally.²⁹ Reducing or curtailing smoking among young men is an important way to reduce the risk from primary and secondary clinical diseases.

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