Assessment of lipid profiles of adult male athletes from two different air pollutant zones of West Bengal, India

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The study was carried out with an objective to find out the effects of air pollution on lipid profiles of trained and untrained males of West Bengal. Sample consisted of 60 sprinters, 60 footballers and 120 untrained males, subdivided into two groups from two zones namely Tollygunge and Sonarpur. SPM, RPM, SO_x and NO_x of ambient air were monitored for both zones. Height and weight of all the males was measured. Venous blood sample was drawn from the antecubital vein and the cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol and cholesterol/ HDL-cholesterol ratio were determined by standard methods. Results revealed that air pollutant concentration was significantly higher in Tollygunge than Sonarpur. Lipid profile was significantly poor in untrained males compared to either the footballers or the sprinters in both regions. However, no significant difference in lipid profile was observed when compared between footballers and sprinters, though lipid profile was better in footballers than sprinters. Alternatively, all lipid parameters of both trained and untrained males were significantly better in Sonarpur than Tollygunge. It was concluded that environmental air pollutants might influence lipid profile adversely both in trained and sedentary males. However, further study in this area is needed.

Keywords: Air pollution, cholesterol, footballers, sprinters.

THE effect of air pollution on human health has become a major concern in today's world. India is no exception. At present, in India, emissions from vehicles are the main cause of prevalence of air pollution in urban areas. The effects of poor air quality on human health are far reaching, but mostly affect the body's respiratory system and the cardiovascular system. In many countries, association of air pollution with risk factors of non-communicable diseases in children and adolescents is well documented[1–4]. Chuang *et al.*[5] observed that increased particulate matter <10 μm was associated with elevated systolic blood pressure, triglyceride, apolipoprotein B, haemoglobin A1c (HbA1c), and reduced high-density lipoprotein cholesterol which might provide a link between air pollution and progression of atherosclerotic cardiovascular diseases. Individual reactions to air pollutants depend on the type of pollutant in which a person is exposed to, the degree of exposure and the concentration of the chemicals[4].

The probable impact of air pollution on lipid disorders is considered to be an important area of study. A study on the association of blood markers of cardiovascular risk and air pollution found that PM10, but not gaseous air pollutants, is associated with blood markers of cardiovascular risk[6].

There is a growing concern regarding problems associated with exercising in polluted air. Regular exercise is advised by physicians for better health and longevity. However, people exercising in urban regions are unwittingly at risk due to the repeated contact with air pollution, it can cause cardiorespiratory disease and cancer. Athletes have been identified as a vulnerable population due to the intensity and duration of exposure to outdoor air quality. According to Folinsbee[7], exercise increases the amount of inhaled pollutants in a harmful level for healthy young individuals. The key reason for exercising persons being at special risk of acquiring disease is a significant increase in pulmonary ventilation and diffusion capacity which occurs even at low intensities[8]. The total amount of deposition of particulate matter in the lungs of exercising humans is directly related to minute ventilation, and deposition is greater during slower, deeper breathing than rapid and shallow breathing[9]. Kargarfard *et al.*[4] revealed that air pollution adversely affected cardiorespiratory fitness in young individuals during exercise. Monitoring of lipid profile in athletes can provide valuable information about their metabolic and cardiovascular status. Studies on the effects of air pollution on lipid profiles of athletes are scanty. So, an attempt has been made to determine the following. Relation of game-specific training effect on lipid profiles with respect to untrained males. If there is any association of air pollution with lipid profiles of trained and untrained males.

Two zones namely, Tollygunge and Sonarpur in West Bengal, India were selected for the study. Tollygunge is a part of core Kolkata which was included in the city of the Kolkata (core) in 1951 (Kolkata proper)[10,11].

Another area is rural–urban fringe of the city of Kolkata named as Sonarpur which is recently yearmarked by the Kolkata Metropolitan development authority as a 'Growth Centre'[12].

The air quality of these two zones was monitored for 24 h in winter during 1 November 2012 to 31 January 2013 (3 months) and suspended particulate matter (SPM), respirable particulate matter (RPM), sulphur dioxide (SO_2) and oxides of nitrogen as nitrogen dioxide (NO_2) were assessed by standard procedures described here. The average values (mean ± standard deviations) of air pollutants were then taken into account.

The equipment used for ambient air quality monitoring were respirable dust sampler (APM 460) and gas absorbing

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glass Impingers. Spectrophotometer (Spectronic Genesis), single pan balance (Mettler), oven and dessicator were used for analysis.

The APM 460 sampler first separates the coarse particles (larger than 10 μm) from the air stream before filtering it on the 0.5 μm pore-size filter allowing measurement of both SPM[13] and RPM[14].

The mass concentration of particulate matter may be calculated as follows and reported to the nearest microgram per cubic metre.

$$\text{PM } (\mu g/m^3) = \frac{(W2 - W1) \times 10^6}{V},$$

where PM is the mass concentration of particulate matter ($\mu g/m^3$); W1 the initial weight of filter paper (g); W2 the final weight of exposed filter papers (g); V the air volume sampled (m^3) and 10^6 is the conversion factor from grams to micrograms.

When SO_2 (ref. 15) from the air stream is absorbed in a sodium tetrachloro mercurate (TCM) solution at a rate of 0.3 or 0.5 LPM (litre per minute) to form a stable dichlorosulphato mercurate. The amount of SO_2 is then estimated by the colour produced when p-rosaniline hydrochloride is added to the solution. The colour is estimated by a reading from a spectrophotometer for which a calibration curve is prepared.

$$SO_2(\mu g/m^3) = \frac{\mu g\ SO_2 \text{ from curve} \times 25 \times 1000}{10 \times \text{hour} \times 60 \times \text{LPM.}}.$$

Oxides of nitrogen[16] as nitrogen dioxide are collected by bubbling air through a NaOH solution at a rate of 0.4 LPM to form a stable solution of sodium nitrate. The nitrate ion produced during sampling is determined spectrophotometrically by reacting the exposed absorbing reagent with phosphoric acid, sulphanilamide and N(1-naphthyl) ethylinediamine dihydrochloride (NEDA).

$$NO_x(\mu g/m^3) = \frac{\mu g \text{ of } NO_x \text{ from curve} \times 25 \times 1000}{10 \times \text{hour} \times 60 \times \text{LPM} \times 0.82}.$$

A total of 240 healthy, nonsmoking, nonalcoholic adult males (19–25 years) volunteered and this sample consisted of 60 football players (30 from Tollygunge and 30 from Sonarpur), 60 sprinters (30 from Tollygunge and 30 from Sonarpur) and 120 sedentary males (60 from Tollygunge and 60 from Sonarpur). Athletes were trained at that particular zone where the ambient air quality was monitored and the participants of this study (both athletes and sedentary) resided within 3 km radius of the monitoring stations. All the football players and sprinters (trained) practised regularly and had a training background of minimum 5 years. Sedentary males (control group) were healthy individuals with no athletic or sports training. Participants (subjects) were residents of those two zones for a minimum period of 5 years. All the participants belonged to the same economic status following the categorization set-up by the West Bengal Housing Board[17] and they were under same nutritional status[18]. All institutional policies concerning research on human subjects were followed. Ethical approval was taken from the competent authority.

The data collected included anthropometric parameters and lipid profiles.

Anthropometric parameters were measured as follows: standing height (cm) was measured with shoes removed, feet together. Weight (kg) was measured with shoes and jackets removed.

Lipid profiles were estimated as follows: fasting (12 h) blood sample was drawn from the antecubital vein between 8 and 9 a.m. Before the samples drawn, subjects were asked to take rest for 10 min. Within 1 h of the sample drawn, serum was centrifuged, 800 *g* for 15 min. The blood sample was analysed by spectrophotometer (Hitachi, Japan) following standard methodology[19] which was as follows.

Total cholesterol was estimated by enzymatic assay method using Ranbaxy kits. The cholesterol was estimated through cholesterol esterase and oxidase in a single reagent to determine total cholesterol in serum.

$$\text{Cholesterol esters} \xrightarrow{\text{C. esterase}} \text{Cholesterol + fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{C. oxidase}} \text{Cholesterol-3-one} + H_2O_2$$

$$2H_2O_2 + \text{4-aminoantipyrine} + \text{p-HBS} \xrightarrow{\text{Peroxidase}} \underset{\text{(red dye)}}{\text{Quinoneimine}} + 2H_2O_2$$

The intensity of red colour produced is directly proportional to the total cholesterol in the sample when read at 520 nm.

Triglycerides (TG) were estimated by enzymatic assay method using Ranbaxy make kits. This method uses a Trinders colour reaction to produce a fast linear, endpoint reaction. Triglycerides in the sample are hydrolysed by lipase to glycerol and fatty acids. The glycerol is then phosphorylated by adenosine-5-triphosphate (ATP) to glycerol-3-phosphate (G3P) and adenosine-5-diphosphate in a reaction catalysed by glycerol kinase (GK). Glycerol-3-phosphate is then converted to dihydroxyacetone phosphate (DHAP) and hydrogen peroxide by glycerophosphate oxidase (GPO). The hydrogen peroxide then reacts with 4-aminoantipyrine (4-AAP) and 3-hydroxy-2,4,6-tribomobenzoic acids (TBHB) in a reaction catalysed by peroxidase to yield a red-coloured quinoneimine dye. The intensity of colour produced is directly proportional to the triglycerides in the sample when read at 540 nm.

HDL-cholesterol (HDL-C) was estimated using PEG enzymatic method. Chylomicrons, very low-density

lipoprotein (VLDL) and low-density lipoproteins (LDL) of serum are precipitated using buffered polyethylene glycol (PEG 6000). After centrifugation, HDL remains in the supernatant. The cholesterol in the HDL fraction is then estimated as that of total cholesterol as mentioned earlier.

LDL-cholesterol was calculated using the formula

$$\text{LDL-C} = \text{Total cholesterol} - (\text{triglyceride}/5 + \text{HDL-C}).$$

C/H ratio was calculated from the value of cholesterol and triglyceride.

All the values are expressed as mean ± standard deviations (SD). Statistical package for the Social Science (SPSS) ver. 20 was used for analysis. Independent sample T test was adopted for statistical analysis of the data. Correlation coefficients among the variables were computed.

Mean values ± SD of ambient air quality data given in Table 1 revealed that all the air quality parameters (SPM, RPM, SO_2 and NO_2) were significantly higher in Tollygunge area than Sonarpur. Mean ± SD of anthropometric and lipid profiles parameters are provided in Table 2. Table 3 shows level of significance of difference in anthropometric parameters and lipid profiles between trained and untrained males of two zones whereas Table 4 revealed the level of significance of difference in those parameters of different groups between two zones. From Table 3, it is clear that TC, TG, LDL-C and TC/HDL-C showed significantly higher value and HDL-C showed significantly lower value in untrained males than both of footballers and sprinters in both regions but no significant difference was observed when compared between footballers and sprinters, though lipid profile was better in footballers than sprinters. On the other hand, studied lipid profile of both trained and untrained males showed significantly better value in Sonarpur area than Tollygunge (Table 4).

This study has been carried out to assess the lipid profile of trained and untrained adult males of two regions of West Bengal, India. Regular moderate and aerobic exercise is associated with a healthy plasma lipid profile[20] and a reduced risk of coronary artery disease and cardiovascular-related death[21,22]. Different cross-sectional[23,24] and longitudinal[25] studies suggested that aerobic exercise increases plasma levels of HDL-C and decreases triglycerides and LDL-C levels. Contradictory results have been obtained with respect to certain types of exercise demanding strenuous physical exertion[26,27]. There are several possible explanations for these discrepancies in lipid profiles of different sportspersons and sedentary individuals, including differences in the methods used to determine the levels of lipids and lipoproteins, the difference in physical fitness of the individuals tested, ethnic factors or differences in training history or the type, quantity and intensity of exercise undertaken, etc.[28]. Ruiz *et al.*[28] reported that those who practice sports involving a high level of physical exertion (volleyball and soccer players) had a less favourable lipid profile compared to control subjects. In contrast, swimmers had a more favourable lipid profile. This study showed the significantly poor lipid profile in untrained males than both of footballers and sprinters in both regions. However, no significant difference in lipid profile was observed in comparison made between footballers and sprinters, though lipid profile was better in footballers than sprinters. The difference in lipid profiles of footballers and sprinters which supported the view that any kind of chronic as well as intensive training regimen may change the lipid profile and these changes depend on the game-specific training in which the subject participates. Aerobic exercises have a positive impact on lipid and lipoprotein profiles and were effective in protecting against coronary risk factors[27]. Also, exercise reduces the level of harmful blood fats such as triglycerides and lipoprotein and increases the level of HDL.

Table 1. Level of significance of difference in air pollutant concentration between two zones

Air pollutant	Tollygunge	Sonarpur	*T*-test
SPM ($\mu g/m^3$)	269.85 ± 54.76	81.58 ± 18.28	$P < 0.01$
RPM ($\mu g/m^3$)	154.08 ± 44.72	33.28 ± 10.17	$P < 0.01$
SO_2 ($\mu g/m^3$)	11.54 ± 3.98	0.64 ± 1.53	$P < 0.01$
NO_x ($\mu g/m^3$)	86.77 ± 17.30	10.03 ± 5.28	$P < 0.01$

This study has shown that exposure to high air pollution is associated with the occurrence of poor lipid profile among trained and untrained males. In spite of belonging to the same socioeconomic and nutritional status, lipid profiles of footballers, sprinter and untrained males of Tollugunge region (high pollution) were significantly poorer than their counterparts in Sonarpur region (less pollution). There were no significant differences in height and weight among the groups. A study in Taiwan found that increased 1-year average ozone, PM and nitrogen dioxide were associated with elevated blood pressure, total cholesterol, fasting glucose and HbA1c[5]. Singh *et al.*[29] reported that traffic exhaust was positively correlated with neutrophils, lymphocytes, monocytes, ESR, CRP, S. cholesterol, LDL and S. triglycerides. Air pollution could cause dyslipidemia among people mainly in urban areas. The comparison by Tomao *et al.*[30] revealed the significant differences in average values of HDL-C and triglycerides between the exposed traffic police group and the control group. Their results suggest the possibility of an alteration in the lipid balance among asymptomatic people who are exposed to air pollution. In a study in Italy, the carboxyhaemoglobin concentration had an inverse correlation with HDL-C[31].

Athletes are at special risk of inhaling pollutants as during exercise, with increase in minute ventilation, there is a proportionate increase in the quantity of pollutants

Table 2. Age, height and weight, lipid profiles of trained and untrained males of different groups (mean ± SD)

Parameters	Tollygunge			Sonarpur		
	Footballers (n = 30)	Sprinters (n = 30)	Untrained (n = 60)	Footballers (n = 30)	Sprinters (n = 30)	Untrained (n = 60)
Age (years)	21.37 ± 3.67	22.10 ± 3.16	21.17 ± 2.96	21.87 ± 3.06	21.97 ± 3.26	21.97 ± 2.99
Height (cm)	173.82 ± 4.66	172.19 ± 5.99	172.43 ± 6.28	171.40 ± 8.46	174.92.7.78	173.56 ± 7.02
Weight (kg)	60.18 ± 3.95	61.50 ± 5.35	60.08 ± 5.38	59.33 ± 6.64	61.98 ± 4.88	61.74 ± 5.08
Training duration (year)	5.67 ± 0.60	5.61 ± 0.52	–	5.79 ± 0.53	5.74 ± 0.54	–
Cholesterol (mg dl-l)	166.6 ± 6.2	167.12 ± 6.3	173.21 ± 6.5	162.6 ± 6.12	163.12 ± 6.32	165.21 ± 6.25
Triglycerides (mg dl-l)	87.5 ± 5.32	88.23 ± 5.22	99.23 ± 5.6	84.5 ± 5.4	85.23 ± 5.25	92.23 ± 5.25
HDL-cholesterol (mg dl-l)	34.5 ± 4.7	32.12 ± 4.12	29.51 ± 4.1	36.4 ± 4.85	35.12 ± 4.12	33.51 ± 4.25
LDL-cholesterol (mg dl-l)	99.9 ± 4.12	101.21 ± 4.02	115.03.4.03	97.9 ± 3.95	99.33 ± 4.22	112.13.3.83
Cholesterol/HDL cholesterol ratio	4.83 ± 0.9	5.20 ± 0.84	5.87 ± 0.91	4.47 ± 0.76	4.64 ± 0.82	4.93 ± 0.74

Table 3. Level of significance of difference in anthropometric and lipid profiles between trained and untrained males of two zones

Parameters	Tollygunge			Sonarpur		
	F versus UM	SP versus UM	F versus SP	F versus UM	SP versus UM	F versus SP
Age (years)	NS	NS	NS	NS	NS	NS
Height (cm)	NS	NS	NS	NS	NS	NS
Weight (kg)	NS	NS	NS	NS	NS	NS
Training duration (year)	–	–	–	–	–	–
Cholesterol (mg dl-l)	$P < 0.01$	$P < 0.01$	NS	$P < 0.01$	$P < 0.01$	NS
Triglycerides (mg dl-l)	$P < 0.01$	$P < 0.01$	NS	$P < 0.01$	$P < 0.01$	NS
HDL-cholesterol (mg dl-l)	$P < 0.01$	$P < 0.01$	NS	$P < 0.01$	$P < 0.01$	NS
LDL-cholesterol (mg dl-l)	$P < 0.01$	$P < 0.01$	NS	$P < 0.01$	$P < 0.01$	NS
Cholesterol/HDL-cholesterol ratio	$P < 0.01$	$P < 0.01$	NS	$P < 0.01$	$P < 0.01$	NS

F, Footballers; SP, Sprinters; UM, Untrained males; NS, Not significant.

Table 4. Level of significance of difference in physical characteristics and lipid profiles of adult males of different groups between two zones

Parameters	TF versus SF	TS versus SS	TUM versus SUM
Age (year)	NS	NS	NS
Height (cm)	NS	NS	NS
Weight (kg)	NS	NS	NS
Training duration (year)	NS	NS	NS
Cholesterol (mg dl-l)	$P < 0.05$	$P < 0.05$	$P < 0.05$
Triglycerides (mg dl-l)	$P < 0.05$	$P < 0.05$	$P < 0.05$
HDL-cholesterol (mg dl-l)	$P < 0.05$	$P < 0.05$	$P < 0.05$
LDL-cholesterol (mg dl-l)	$P < 0.05$	$P < 0.05$	$P < 0.05$
Cholesterol/HDL-cholesterol ratio	$P < 0.05$	$P < 0.05$	$P < 0.05$

NS, Not significant; TF, Tollygunge footballers; SF, Sonarpur footballers; TS, Tollygunge sprinters; SS, Sonarpur sprinters; TUM, Tollygunge untrained males; SUM, Sonarpur untrained males.

inhaled (V_E) and a large fraction of air is inhaled through the mouth during exercise, effectively bypassing the normal nasal mechanisms for the filtration of large particles and soluble vapours[32].

From this study, it is concluded that game-specific training plays an important role contributing to variation of lipid profiles in athletes. The findings indicate the association of lipid profile with air pollutants in both trained and untrained males of West Bengal. However, this particular area needs further investigations through longitudinal studies.

Special concern regarding environmental protection issues should be taken into consideration for every individual and top priority should be given to the population under regular sports training and exercise programme.

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In vitro and *in vivo* inhibition of haemolymph juvenile hormone esterase activity by the ethanol extract of *Clerodendrum inerme* in fifth instar larva of castor semilooper, *Achaea janata* (L.)

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Juvenile hormone (JH) is a unique hormone in insects that controls growth, development, metamorphosis and reproduction. It has been well established that JH esterase is an enzyme involved in JH regulation. Results of both *in vitro* and *in vivo* studies revealed that ethanol leaf extract of *Clerodendrum inerme* significantly inhibits haemolymph JH esterase activity affecting growth and normal development of the test insect, castor semilooper (*Achaea janata*), a serious pest of oil seed crops like castor and groundnut.

Keywords: *Achaea janata*, *Clerodendrum inerme*, ethanol leaf extract, juvenile hormone.

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