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ORIGINAL ARTICLE

Effect of air pollution and time of day on performance, heart rate hematological parameters and blood gases, following the YYIRT-1 in smoker and non-smoker soccer players

Effet de la pollution atmosphérique et de l'heure de la journée sur les performances, les paramètres hématologiques, la fréquence cardiaque et les gaz de sang, suite au YYIRT-1 chez des footballeurs fumeurs et non fumeurs

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KEYWORDS
Environment;
Smoking;
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Summary
Objectives. — This study aimed to investigate the effect of air pollution and time of day on performance, heart rate hematological parameters and blood gases, following the Yo-Yo intermittent recovery test level 1 in smoker and non-smoker soccer players.
Methods. — Twenty-two soccer players were divided into two groups; (smoker: $n = 11$) and (non-smoker: $n = 11$). Each participant completed the test at 08:00 h and 18:00 h in two different areas (i.e. Polluted and non-polluted area) with a recovery period ≥ 72 h in between.
Results. — Our results showed that at rest, both groups revealed a significant diurnal variation on oral temperature, heart rate, and white blood cell count with an acrophase at 18:00 h and on red blood cells with a significant decrease at 18:00 h in the two areas ($P < 0.05$). Following the

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exercise, both groups presented a diurnal fluctuation on maximal oxygen uptake in non-polluted area with higher values at 18:00 h. However, this variation was disappeared in polluted area for smoker group. Moreover, maximal heart rate, hematological parameters and blood gases were significantly affected ($P < 0.05$) upon two times of day, except partial pressure of oxygen in non-smoker subjects and platelet count within smoker subjects. Besides, this effect was more accentuated in the evening in most parameters. In addition, 60 min of recovery appeared not sufficient for smoker group to attenuate the rest values (i.e. Heart rate, white blood cells) upon two times of day.

Conclusion. — Intermittent recovery test under high concentrations of pollutants leads a significant alteration of performance and physiological parameters in healthy and smoker athletes especially in the evening. Furthermore, results from the field may help to guide athletes and contribute to public health recommendations on exercise and pollution.

MOTS CLÉS
Environnement ;
Tabagisme ;
Heure de la journée ;
Exercice

Résumé

Objectifs. — Cette étude a pour objectif d'étudier l'effet de la pollution de l'air et de l'heure de la journée sur les performances, les paramètres hématologiques et les gaz de sang suite au Yo-Yo intermittent recovery test level-1 chez des joueurs de football fumeurs et non fumeurs.

Méthodes. — Vingt-deux joueurs de football ont été divisés en deux groupes (fumeur : $n = 11$) et (non fumeur : $n = 11$). Chaque participant a complété le test à 08:00 h et 18:00 h dans deux régions différentes (Polluée et non polluée) avec une période de récupération ≥ 72 h entre les sessions.

Résultats. — Nos résultats ont montré une variation diurne de la température orale, la fréquence cardiaque, et du nombre de globules blancs avec une acrophase observée à 18:00 h et une diminution significative des globules rouges à 18:00 h dans les deux régions ($p < 0,05$) chez les deux groupes. De même, les deux groupes ont présenté une fluctuation diurne au niveau de la consommation maximale d'oxygène dans la région non-polluée avec des valeurs plus élevées à 18:00 h. Cependant, cette variation a disparu dans la région polluée pour les fumeurs. De plus, la fréquence cardiaque maximale, les paramètres hématologiques et les gaz de sang étaient significativement affectés ($p < 0,05$) le matin et l'après-midi, à l'exception la pression-veineuse en O_2 chez les non-fumeurs et le nombre de plaquettes chez les fumeurs. En outre, cet effet était plus accentué l'après-midi pour la majorité des paramètres mesurés. De plus, 60 minutes de récupération ne semblent pas suffisantes pour que les athlètes fumeurs atténuent leurs valeurs de repos.

Conclusion. — Une exposition aiguë à des fortes concentrations des polluants lors d'un exercice intermittent entraîne une altération significative de la performance et des paramètres physiologiques chez les athlètes fumeurs et non fumeurs. Cependant, cet effet semble être plus aggravé chez les athlètes fumeurs surtout l'après-midi. Par conséquent, ils doivent bien planifier leurs programmes d'entraînement en tenant compte du cadre spatio-temporel de leurs activités afin de minimiser les risques d'exposition à la pollution.

1. Introduction

Pollution is the introduction of harmful substances or products into the environment. The short-term effects of air pollution on athletic performance and athletes' health have been studied in a several studies. Some reports aim at looking at the effects of increased levels of air pollution on athletic performance as measured by common physiological parameters [1,2]. Thus, exercising in polluted air led an alteration on physical and cognitive performance [3,4]. Likewise, Das et al. [5] showed higher values in agility, leg muscle power and handgrip strength in less air pollutant zones than higher air pollutant zones in soccer players. In fact, during exercise, even at relatively low intensities, inhaled air is taken in predominantly through the mouth, and there is a major increase in minute ventilation and diffusion capacity, and the total amount of ultrafine particulate matter deposited in the respiratory tract of humans during moderate exercise is about five times than at rest [6]. Indeed, athletes are more vulnerable to cardiovascular diseases in particular during competition period. Soccer is a high intensity intermittent activity and the Yo-Yo intermittent recovery test (YYIRT) is one of the main tests used frequently during training sessions [7]. Studies in this field showed that

YYIRT induce a significant increase of muscle damage and lipid profile, and oxidative stress markers in soccer players that can be accentuated under polluted conditions [1,8].

On the other hand, the physiological effects of nicotine, tar and carbon monoxide on the respiratory, cardiovascular systems, blood parameters and performance of athletes have been well studied [9,10]. Several studies showed a potent relation between smoking and physical activity [9,11]. In this context, a significant alteration was observed with tobacco consumption with a decrease of maximum oxygen consumption (VO_{2max}), anaerobic threshold, oxygen pulse, a higher heart rate, ventilation, respiratory rate, cardiac output and pulse pressure [11]. The exact mechanisms of this alteration are probably multifactorial. Furthermore, studies in the field showed a circadian variability in some physiological parameters [12,13]. Nevertheless, no one was interested in the exogenous effect of air pollution on smoker population on performance and physiological responses.

Self-sustained endogenous timers ''biological clocks'' permit organisms to anticipate daily environmental rhythms, and adjust their physiology and behavior consequently. In this context, several studies exhibited a significant correlation between time of day and air pollutant concentrations. Thus, the air pollution effect on health seems to be a TOD dependent [14,15]. Some studies have highlighted in daily variation of some pollutants (peak in the afternoon ~16:00). Likewise, pollutants characterized by circannual fluctuations (i.e. CO, NO_2, O_3) with a significant increase in the cold months of the year [15]. Cakmak et al. [16] illustrated an alteration in performance with a lower aerobic fitness score in athletes after exposure to high concentration of ozone.

Because of limited findings in this field, it is critical to investigate the effect of air pollution at two different times of day on physical performance and physiological functions in smoker and non-smoker athletes following a maximal intermittent recovery test. We hypothesized that pollution lead a significant alteration of cardiovascular, hematological responses and circulatory system following the YYIRT-1 especially in smokers and non-smoker upon two times of day.

2. Materials and methods

2.1. Participants

Twenty-two soccer players were divided into two groups (i.e. Smokers (SG) and non-smokers (NSG), all characteristics of participants were mentioned in the Table 1. Subjects were selected based on their chronotype and on the basis of their answers to Horne and Ostberg [17], self-assessment questionnaire. They had an intermediate chronotype (i.e. Sleep duration between 22:30 ± 1:00 and 07:00 ± 1:00 h). After receiving a thorough explanation of the protocol, participants gave written consent to participate in this study. They had a soccer experience of minimum 9 years and participated in regular training at least 4 day per week. They did not consume nutritional supplements, caffeine, drugs, or alcoholic beverages and they didn't have any history of chronic, cardiovascular or pulmonary disease. The choice of the smoking group is based on their level of tobacco consumption (at least 11 cigarettes per day for at least 3 years) [18].

2.2. Experimental design

During the week before the experiment, the participants were familiarized with the equipment and the experimental procedures, to minimize learning effects during the course of the study. Each group completed identical test sessions in the morning (07:00—09:00 hours) and in the evening (17:00—19:00 hours) in two areas with a recovery period ≥ 72 h in between and each two tests in different areas were interspersed by one week. The two experimental areas (polluted and non-polluted) were determined on the basis of the environmental protection agency (ANPE) of Tunisia based on their level of air pollution on each area and the characteristics of each area were mentioned in the Table 2. The test procedure was randomized in order to avoid familiarization. The experimental design is illustrated in Fig. 1. Subjects participated in test sessions at an air temperature between 27—28 °C and with 53—56% relative humidity at all sessions.

Each session began with 15 min sitting, the weight and body composition of subjects were recorded using bioelectrical impedance scale to the nearest 0.1 kg (Tanita, Tokyo, Japan) calibrated in accordance with the manufacturer's guidelines by one trained technician. From these two variables, the body mass index (BMI; $kg.m^{-2}$) was then calculated and recorded.

Oral temperature was measured with a digital clinical thermometer (Omron, Paris, France; accuracy ± 0.1 °C) inserted sublingually for at least 3 min at 08:00 h and 18:00 h after a 15-min rest in a seated position. After that, the YYIRT-1 was performed accompanied by a blood sample collections and heart rate (HR) measurement before, immediately and 60 min after exercise. The heart rate was measured using an electronic blood pressure arm (Microlife, W 90, Paris) [19]. Reliability was found to be good to excellent for both time-domains heart rate variability (ICC: 0.77 and 0.8 for HR_{rest} and HR_{max} respectively). The test—retests were performed and the values were recorded during the

Table 1 Characteristics of smoker and non-smoker athletes.

Characteristics	Non-smokers (n = 11)Mean ± SD	Smokers (n = 11)Mean ± SD	P
Age (year)	21.82 ± 0.51	22.09 ± 0.29	NS
Height (cm)	178.00 ± 0.02	176 ± 0.02	NS
Body mass (kg)	73.97 ± 2.82	73.18 ± 1.72	NS
BMI (kg/m^2)	23.57 ± 0.75	23.25 ± 0.52	NS

NS: no significant difference.

Table 2 Characteristics of the two environments, including polluted and non-polluted area.

	Non-polluted area	Polluted-area	
	08:00 h and 18:00 h	08:00 h	18:00 h
Altitude (m)	12	8	8
Relative humidity (%)	53–56	53–56	53–56
Temperature (°C)	27–28	27–28	27–28
CO (ppm)	1	18	19
NO_2 (ppb)	5	25	26
SO_2 (ppb)	12.5	83	90
PM_{10} ($\mu g/m^3$)	9	89	90
O_3 (ppb)	4	27	60
AQI	< 50	> 200	> 200

ppb: par per billion; ppm: part per million; $\mu g/m^3$: micrograms per cubic meter air; CO: carbon monoxide; NO_2: nitrogen dioxide; O_3: ozone; SO_2: sulfur dioxide; PM_{10}: particulate matter; AQI: air quality index (good: < 50; moderate: 51–100; unhealthy for sensitive groups: 101–150; unhealthy: 151–200; very unhealthy: 201–300 and hazardous: 300–500).

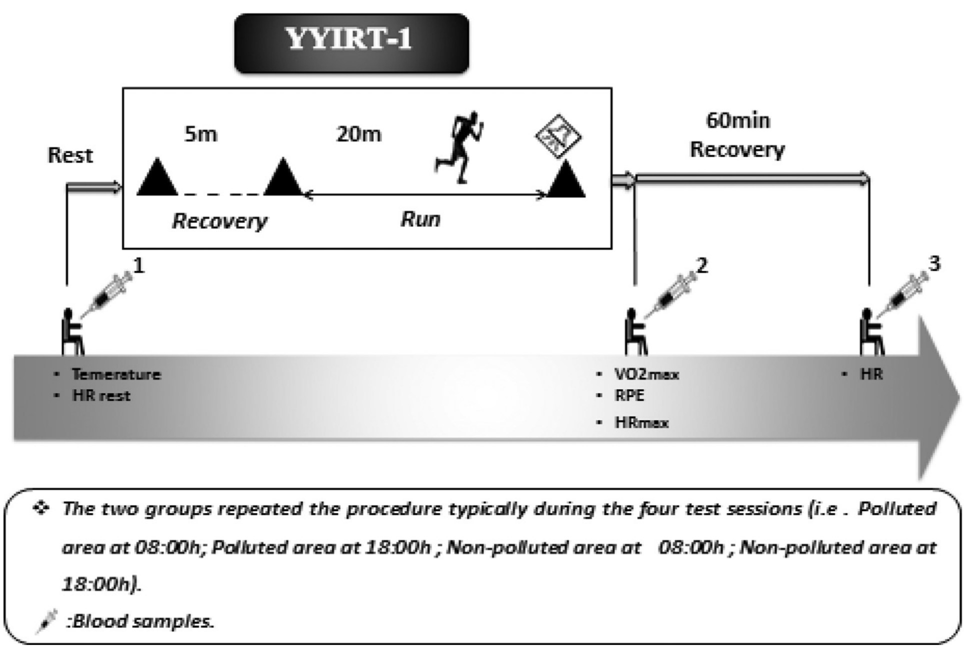

Figure 1 Schematic representation of study design.

familiarization and pre-protocol evaluation sessions at the same time of the day.

Before the morning test sessions, only one glass (150 to 200 ml) of water was allowed, to avoid postprandial thermogenesis and possible acute effects [1]. After the morning sessions, a restricted food intake was allowed. Before the evening test sessions, participants had the same standard isocaloric meal at 12:00 h. After that meal, only water was allowed ad libitum.

The subjects were instructed to not perform strenuous activity on the day before and during the experimental period. And smokers were asked not to smoke at least 2 h prior to the test to exclude the acute effect of smoking on the airways [18]. The study design protocol is in accordance with the Declaration of Helsinki for human experimentation and was approved by the Ethics Committee of the Medical Faculty of the University of Tunisia.

2.3. Exercise protocol

2.3.1. The Yo-Yo Intermittent Recovery Test Level-1 (YYIRT-1)

All participants were familiar with the testing procedures as part of their usual fitness assessment program. Before starting the test, subjects are asked to stay on the starting line waiting for the first signal and they are asked to keep pace with the speed of the race. According to Krustrup et al. [20], the Yo-Yo tests level-1 consisted on 20-m shuttle runs performed at increasing velocities until exhaustion, each running bout is interspersed by a 10-s active recovery period where the subject jogs around a marker placed 5 m behind the starting line. Audio cues of the yo-yo endurance test were recorded on a CD player with no obstructions. Participants are strongly encouraged verbally throughout the trial and they carried out a warm-up period consisting of the first four running bouts in the test. The test was considered ended when the participant failed to reach the front line in time. The total distance covered during the test (including the last incomplete shuttle) was considered as the testing performance. Each subject's maximal oxygen uptake (VO_{2max}) was determined from the equation developed by Bangsbo et al. [21]: VO_{2max} (ml·kg^{-1}·min^{-1}) = 0.0072 × Dist YoYoIRT1 + 41.03. Reliability of the field tests considered in this study is reported elsewhere [20].

2.3.2. Rate of perceived exertion (RPE)

Before performing exercise testing, the Ratings of Perceived Exertion (RPE) [22], scales were explained to each participant by trained practitioners. After that, during exercise testing, the overall perception of muscle fatigue (RPE), and physical stress were assessed. RPE is considered as a valid method to quantify training in response to high-volume, intermittent exercise.

2.4. Study location

The two regions of the city were chosen according to the level of their air quality, Sfax (south east of Tunisia) is one of the most polluted areas in Tunisia [23] however Kerkennah (in the south east of Tunisia) and is known by the purity and cleanness of its air. The air of Sfax is predominantly affected by industrial emission, particularly steel plant and motor traffic because of a high proportion of diesel trucks and cars, leading to elevated concentration during stagnant conditions. Data were collected from the environment protection agency of Tunisia city.

2.5. Blood samples

Venous blood samples were taken from venous catheters at rest, at the end of exercise and after 60 min recovery, using vacuum blood collection system, for blood gases, after heparin of syringe, approximately (2 ml) blood was sampled, for determination of partial pressure of oxygen (P_vO_2) (mmHg), partial pressure of carbon dioxide (P_vCO_2) (mmHg), potential of hydrogen (PH) and bicarbonate levels (HCO_3^-) (mmol/L). Resting blood samples were obtained while the subject was in a supine position following insertion of the catheters. Then the needle was bent to prevent the air enters the saying and the lid was covered with a plastic and transformed to the blood laboratory. Blood gas and acid-base measurements were made using a blood gas analyzer (Medica easy blood gas; Analyzer Bedford, MA 01730-1413 USA). The result was printed on special sheets. And for the hematological parameters 5 ml of blood was sampled for a full blood test which included red blood cells count (RBC) ($10^6/\mu l$), hemoglobin count (Hb) (g/dl), platelets (PLT) ($10^3/\mu l$), white blood cells count (WBC) ($10^3/\mu l$), neutrophils (NEUT) (%), and lymphocytes (LYM) (%). The blood specimens were measured using the SFRI hematology system (HEMIX 5-60).

2.6. Data analysis and statistics

The normality of the distribution of the variables was tested using the Shapiro-Wilk *W*-test. All statistical tests were processed using STATISTICA Software (Stat-Soft, Maisons-Alfort, France) and expressed as mean ± SD in the text, tables and figures. Independent t-tests were used to compare the smoker and non-smoker groups regarding anthropometric parameters and baseline measures. A tow way ANOVA with repeated measure [2 (time of day) × 2 (pollution)] was used for VO_{2max}, RPE, and temperature to evaluate the effect of pollution exposure and time of day in each group. Data of HR and blood measurement collection were analyzed in each group using Three-way ANOVA [2 (pollution) × 2 (time of day) × 3 (Time of measurement)] to inspect the effect of pollution and time of day in each group than the effect of exercise and the recovery period on these parameters. The Bonferroni post-hoc test was performed whenever significant effects or a significant interaction were found using ANOVA. To assess the data practical significance, effect sizes were calculated as partial eta-squared (η_p^2). The test—retest reliability was expressed by interclass correlation coefficients (ICCs). The level of statistical significance was set at $P < 0.05$.

3. Results

Concerning the characteristics of SG and NSG, no significant difference was observed ($P > 0.05$) (Table 1). The distribution of environmental pollution levels (under clean and polluted air conditions) during the study period is depicted in Table 2. The altitude, ambient temperature and humidity of both testing conditions were identical, but the pollutants density and PSI of the polluted air environment were much higher than those in the clean air atmosphere.

3.1. Oral temperature

Oral temperature, present significant diurnal variation in both groups ($P < 0.001$) with acrophase observed at 18:00 h in the two areas (i.e. PA and NPA) without significant pollution effect ($P > 0.05$). Moreover, no significant difference between SG and NSG was observed ($P > 0.05$) (Table 3).

3.2. The Yo-Yo intermittent recovery test level-1

The ICC of YYIRT-1 showed a high reliability (ICC. 0.75). Likewise, a significant diurnal variation of VO_{2max} was observed in NSG ($F = 8.9$, $\eta_p^2 = 0.47$, $P < 0.001$) in the two areas, with increasing values about 18:00 h. The same variation was observed in SG only in NPA ($F = 12.5$, $\eta_p^2 = 0.55$, $P < 0.05$) and it disappeared in PA. Concerning the pollution effect, both groups were significantly affected regarding VO_{2max} levels upon tow TOD, ($F = 30$, $\eta_p^2 = 0.58$, $P < 0.001$), with a percentage variation (3.14% at 08:00 h and 3.19% at 18:00 h) for NSG and (3.1% at 08:00 h and 3.08% at 18:00 h) for SG (Fig. 2).

Regarding RPE scores, both NSG and SG presented a significant diurnal variation in the two areas ($P < 0.001$) with higher values about 18:00 h compared to 08:00 h. However, no significant pollution effect was observed ($P > 0.05$) (Fig. 3).

Table 3 Comparison of all baseline measures (Mean ± SD) in smoker and non-smokers in all test sessions.

	Non-polluted area				Polluted area			
	08:00 h	*t* within groups	18:00 h	*t* within groups	08:00 h	*t* within groups	18:00 h	*t* within groups
Temperature (°C)								
Non-smokers	36.46 ± 0.19	−1.15	37.59 ± 0.1[**]	0.3	36.55 ± 0.17	−0.49	37.49 ± 0.13[**]	−0.15
Smokers	36.75 ± 0.15		37.55 ± 0.11[**]		36.65 ± 0.14		37.52 ± 0.12[**]	
HR_{rest} (bpm)								
Non-smokers	65.00 ± 1.35	−1.05	66.91 ± 1.28[**]	−0.69	64.82 ± 1.23	−1.35	67.55 ± 0.62[**]	−0.61
Smokers	66.82 ± 1.08		68.09 ± 1.13[**]		67.00 ± 1.04		68.36 ± 1.19[**]	
RBC(10^6/μl)								
Non-smokers	5.14 ± 0.13	−2.16	4.84 ± 0.14[**]	−2.67	5.12 ± 0.15	−2.11	4.82 ± 0.14[**]	−2.82
Smokers	5.44 ± 0.07[*]		5.27 ± 0.08[*,**]		5.46 ± 0.06[*]		5.29 ± 0.09[*,**]	
Hb (g/dl)								
Non-smokers	16.21 ± 0.23	−0.01	15.87 ± 0.26	−0.64	15.34 ± 0.24	−1.79	15.25 ± 0.20	−1.94
Smokers	16.22 ± 0.27		16.14 ± 0.32		16.95 ± 0.25		15.8 ± 0.21	
WBC(10^3/μl)								
Non-smokers	6.77 ± 0.13	−3.82	6.97 ± 0.17[**]	−3.69	6.72 ± 0.13	−4.08	7.02 ± 0.16[**]	−3.65
Smokers	7.59 ± 0.17[*]		7.96 ± 0.21[*,**]		7.57 ± 0.16[*]		7.97 ± 0.21[*,**]	
LYM (%)								
Non-smokers	23.70 ± 1.34	−6.49	27.24 ± 1.14	−4.07	31.18 ± 0.79	−2.83	32.32 ± 1.74	−2.19
Smokers	33.97 ± 0.84[*]		33.08 ± 0.87[*]		34.19 ± 0.7[*]		36.35 ± 0.59[*]	
NEUT (%)								
Non-smokers	52.43 ± 0.33	−0.32	52.37 ± 0.69	0.52	52.43 ± 0.33	0.07	52.63 ± 0.54	1.09
Smokers	52.65 ± 0.57		51.83 ± 0.77		52.37 ± 0.64		51.69 ± 0.66	
PLT (10^3/μl)								
Non-smokers	203.82 ± 10.99	1.54	204.09 ± 11.12	1.52	204 ± 10.87	1.61	203.91 ± 10.68	1.54
Smokers	185.64 ± 4.11		185.82 ± 4.49		185.09 ± 4.37		185.82 ± 4.75	
P_vO_2 (mmHg)								
Non-smokers	43.36 ± 0.74	0.61	43.91 ± 0.56	1.4	44.00 ± 0.66	2.01	44.00 ± 0.83	2.07
Smokers	42.82 ± 0.46		42.91 ± 0.42		42.36 ± 0.45		42.09 ± 0.37	
P_vCO_2 (mmHg)								
Non-smokers	47.50 ± 1.86	−0.25	47.53 ± 1.62	−0.34	47.32 ± 1.97	−0.37	47.42 ± 1.88	−0.27
Smokers	48.09 ± 1.34		48.22 ± 1.16		48.15 ± 1.04		48.01 ± 0.98	
HCO_3^- (mm/l)								
Non-smokers	24.99 ± 0.42	1.96	25.08 ± 0.55	1.92	25.08 ± 0.63	1.85	24.81 ± 0.82	1.41
Smokers	23.88 ± 0.38		23.84 ± 0.34		23.65 ± 0.44		23.52 ± 0.41	
pH								
Non-smokers	7.32 ± 0.01	0.22	7.33 ± 0.02	0.4	7.31 ± 0.02	−0.23	7.31 ± 0.02	−0.46
Smokers	7.32 ± 0.01		7.32 ± 0.01		7.31 ± 0.02		7.32 ± 0.01	

P_vO_2: partial pressure of oxygen; P_vCO_2: partial pressure of carbon dioxide; HR: heart rate; PH: potential of hydrogen; HCO_3^-: bicarbonate; RBC: red blood cells count; Hb: hemoglobin count; PLT: platelets; WBC: white blood cells count; NEUT: neutrophils; LYM: lymphocytes.

[*] Significant difference with NSG at $P < 0.05$.

[**] Significant difference with (08:00 h) at $P < 0.05$.

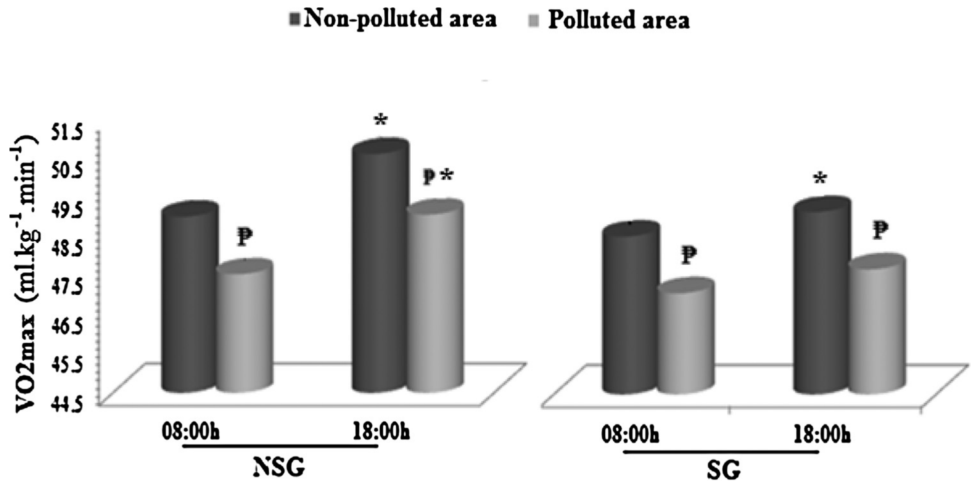

Figure 2 VO_{2max} measures (means ± SD, $n = 22$) registered in two areas at two times of day. *: significant difference with 08:00 h at $P < 0.05$; Ᵽ : significant difference with non- polluted area at the same time of day at $P < 0.05$.

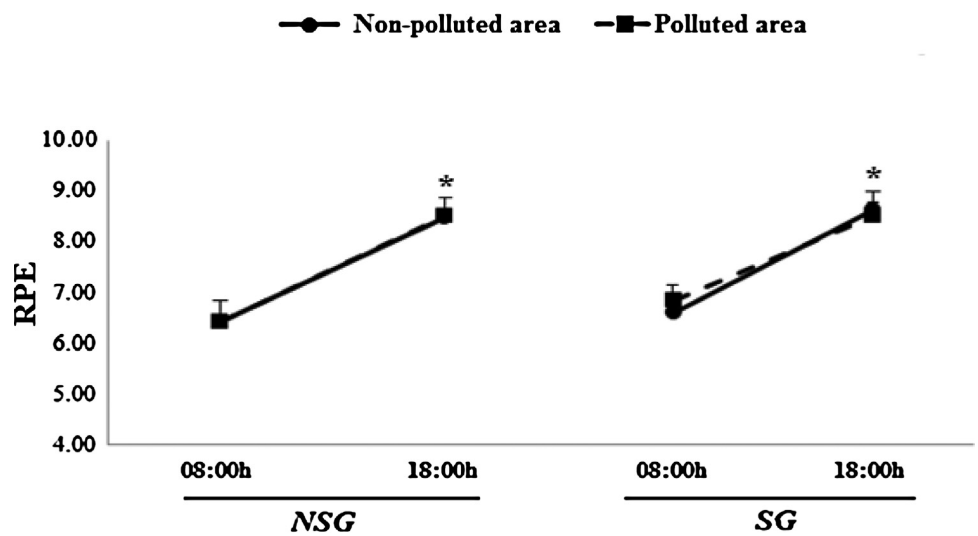

Figure 3 RPE scores (means ± SD, $n = 22$) registered in two areas at two times of day. *: significant difference with 08:00 h at $P < 0.05$.

3.3. Heart rate

No significant difference was observed ($P > 0.05$) between SG and NSG regarding HR_{rest}, likewise it increased significantly at18:00 h compared to 08:00 h in the two areas. Furthermore, both groups were significantly affected by pollution following the YYIRT-1 upon two TOD ($P < 0.05$). In fact, for NSG, HR_{max} was significantly higher in polluted area compared to non-polluted area measures ($F = 6.16$; $\eta_p{}^2 = 0.38$; $P < 0.05$). However, in SG presented significantly lower values of HR_{max} in polluted area ($F = 6.34$, $\eta_p{}^2 = 0.38$, $P < 0.05$) upon two times of day (Fig. 4). Likewise, 60 min of recovery are insufficient for both SG and NSG to attenuate the rest values in polluted area upon two times of day with high difference observed at 18:00 h ($P < 0.001$). Besides, SG appeared unable to recover resting values ($P > 0.05$) (Table 5).

3.4. Hematological parameters

The difference between the two groups (i.e. SN and NSG) of all parameters at baseline was illustrated in Table 3. In fact, a significant difference was observed in all test sessions regarding WBC, LYM and RBC ($P < 0.05$) with higher values in SG compared to NSG. No significant difference ($P > 0.05$) was observed concerning other parameters (i.e. PLT, Hb, NEUT). Likewise, most hematological parameters presented no significant change between the two TOD for both SG and NSG ($P > 0.05$). Nonetheless, a significant diurnal variation was observed with low values of WBC count ($P < 0.01$) and high values of RBC count at 18:00 h for both groups in the two areas ($P < 0.01$) (Table 3).

Following the YYIRT-1 in polluted area, both groups are affected regarding hematological parameters upon two times of day ($P < 0.01$). In fact, RBC and Hb decrease significantly ($P < 0.05$) in PA compared to NPA following the YYIRT-1. However, WBC, LYM and NEUT increase significantly

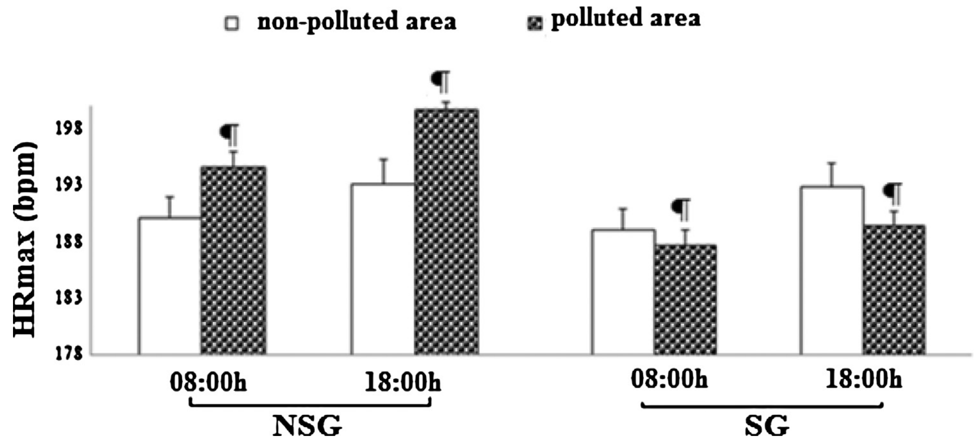

Figure 4 Maximal heart rate measures for both groups (HRmax) (means ± SD, $n = 22$) registered in two areas at two times of day. ₱ : significant difference with non- polluted area at the same time of day at $P < 0.05$.

under polluted air ($P < 0.05$). PLT were affected by pollution ($F = 22.02$, $\eta_p^2 = 0.68$; $P < 0.05$) upon two TOD only in NSG presenting a significant increase ($P < 0.05$) compared to non-polluted area. Moreover, the percentage of variation was higher in SG compared to NSG, concerning most of hematological parameters (i.e. RBC, Hb, WBC, LYM) (Table 4). Likewise, 60 minutes of recovery are insufficient to recuperate the WBC in PA ($P < 0.001$) for SG upon two times of the day in particular at 18:00 h. Concerning NEUT, values continue to increase even 1 h late in both groups under PA and NPA (Table 5).

3.5. Blood gases

Concerning blood gas parameters (i.e. P_vCO_2, P_vO_2, HCO_3^- and PH) at rest, no significant difference was observed in SG compared to NSG. Likewise, NSG and SG revealed no significant time of day effect in all blood gases ($P > 0.05$). However, they were significantly affected by pollution in both groups during the two TOD. Following the YYIRT-1, NSG and SG presented lower values of PH and HCO_3^- and higher values of P_vCO_2 in PA compared to NPA upon two TOD. Regarding P_vO_2 measures, only SG was affected ($F = 17.91$, $\eta_p^2 = 0.68$, $P < 0.001$), with a significant decrease upon the tow time sessions compared to NPA. Moreover, The amount of decline was higher in SG: For HCO_3^- (−19.23% vs. −10.58% at 08:00 h and −18.96% vs. −15.05% at 18:00 h), for P_vCO_2 (+7.45% vs. +6.85% at 08:00 h and +7.97% vs. +7.08% at 18:00 h) and for PH (−0.54% vs. −0.33% at 08:00 h and −0.64% vs. 0.33% at 18:00 h) (Fig. 5). Likewise, 60 minutes of recovery appeared not sufficient for both smoker and non-smoker athletes to attenuate rest values of P_vCO_2 and P_vO_2 in all test sessions, and HCO_3^- values in SG at 18:00 h (Table 5).

4. Discussion

Our results revealed that, the two groups were affected significantly by pollution regarding maximum oxygen uptake upon two TOD, presenting a significant decrease when exposing to high concentration of pollutants and the magnitude of change differs from smoker to non-smoker subjects. The reason for this lower performance could be the result of the deterioration in oxygen distribution and lung function that occurs during exercise under polluted air, which is agree with some reports in this field [24,25]. This decrease of VO_{2max} in polluted area seems to be explained by the amount of carbon monoxide (CO) inhaled from the respiratory tract during exercise, altering the ability of hemoglobin to carry oxygen and as a result requiring muscle will be unable to fulfill the energy requirements of the exercise. Additionally, the decrease was markedly observed in SG compared to NSG upon two TOD (47.52 vs. 47.09 (ml kg^{-1} min^{-1}) at 08:00 h) and (49.04 vs. 47.71 (ml kg^{-1} min^{-1}) 18:00 h). In this context, some reports mentioned that subjects who have recently smoked experience presented cardiovascular changes that adversely affect oxygen availability at the tissue level, and their blood component present already elevated level of carbon monoxide which is the accumulate major component of tobacco smoke [10]. Moreover, smokers resulting in high level of carboxyhemoglobin (COHb) as a result of the combination of CO with the oxygen-transmitting hemoglobin of the blood. This can explain the difference between the groups. Furthermore, the diurnal variation of VO_{2max} disappeared in SG. In this context, some pollutants such as ozone presented a diurnal pattern peaking around evening hours [26], this could explain in part the higher depletion of performance observed at this time of day. Previously, Cakmak et al. [16], have shown that ozone concentration at evening, led an alteration in performance in athletes by decreasing macrophage activity, and depleting airway antioxidant defenses.

Our results showed higher values of HR_{max} under polluted air upon two TOD (190.09 bpm vs. 194.55 bpm) at 08:00 h and (193.09 bpm vs. 199.55 bpm) at 18:00 h in NSG. The increase in HR_{max} could be linked to a fall in oxygen levels in the blood when breathing in the polluted air environment, which stuck oxygen transportation to the active muscles [27]. Recent studies confirmed the relationship between HR and exposing to air pollution for healthy subjects with an increase in HR_{max} and pulse pressure [24,28]. Regarding smoker group, HR_{max} was significantly lower in polluted area compared to non-polluted area during two TOD (188.91 bpm vs. 187.55 bpm) at 08:00 h and (192.64 bpm vs. 189.27 bpm)

Table 4 Variation of hematological parameters following the YYIRT-1 under polluted and non-polluted air in both groups upon two times of day.

	Non-Smokers (mean ± SD)		Within-area, %	*F*-values	Smokers (mean ± SD)		Within-area, %	*F*-values
	Non-polluted area	Polluted area			Non-polluted area	Polluted area		
RBC ($10^6/\mu l$)								
08:00 h	5.61 ± 0.12	5.5 ± 0.09*	−1.96	$F = 8.84$, $\eta_p^2 = 0.46$	5.70 ± 0.04	5.54 ± 0.04*	−2.79	$F = 7.27$, $\eta_p^2 = 0.42$
18:00 h	5.38 ± 0.1	5.25 ± 0.07*	−2.44		5.55 ± 0.04	5.43 ± 0.08*	−2.38	
Hb (g/dl)								
08:00 h	16.62 ± 0.2	16.27 ± 0.2*	−2.21	$F = 8.6$, $\eta_p^2 = 0.46$	17.11 ± 0.19	16.63 ± 0.18*	−2.97	$F = 17$, $\eta_p^2 = 0.62$
18:00 h	16.14 ± 0.2	16.02 ± 0.2*	−0.94		17.26 ± 0.17	16.33 ± 0.15*	−5.75	
WBC ($10^3/\mu l$)								
08:00 h	9.13 ± 0.14	9.36 ± 0.18*	+2.42	$F = 16.46$, $\eta_p^2 = 0.59$	9.65 ± 0.11	9.96 ± 0.08*	+3.11	$F = 6.51$, $\eta_p^2 = 0.39$
18:00 h	9.3 ± 0.17	9.52 ± 0.18*	+2.37		9.70 ± 0.12	10.10 ± 0.09*	+3.93	
LYM (%)								
08:00 h	30.3 ± 1.52	34.98 ± 1.36*	+4.21	$F = 29,69$ $\eta_p^2 = 0.74$	36.23 ± 0.49	38.96 ± 0.51*	+6.87	$F = 13.89$, $\eta_p^2 = 0.58$
18:00 h	34.6 ± 1.23	35.37 ± 1.49*	+0.19		36.43 ± 0.54	39.73 ± 0.71*	+8.06	
NEUT (%)								
08:00 h	61.87 ± 0.75	64.69 ± 0.94*	+4.21	$F = 8.15$, $\eta_p^2 = 0.44$	56.24 ± 0.62	58.81 ± 0.64*	+4.37	$F = 63.16$ $\eta_p^2 = 0.86$
18:00 h	62.24 ± 1.87	65.90 ± 0.99*	+5.58		55.96 ± 0.68	60.04 ± 0.6*	+6.79	
PLT ($10^3/\mu l$)								
08:00 h	251.82 ± 13.85	271.55 ± 14.18*	+7.26	$F = 22.02$, $\eta_p^2 = 0.68$	221.82 ± 10.43	215.09 ± 9.97	−3.10	$F = 0.76$, $\eta_p^2 = 0.07$
18:00 h	251.45 ± 13.77	271.27 ± 14.15*	+7.27		221.64 ± 11.03	213.91 ± 11.2	−3.74	

RBC: red blood cells count; Hb: hemoglobin count; PLT: platelets; WBC: white blood cells count; NEUT: neutrophils; LYM: lymphocytes.

* Significant difference with non-polluted area at $P < 0.05$.

Table 5 Physiological responses of both smoker (SG) and non-smoker (NSG) athletes after 60 min of recovery in all tests sessions.

	Non-polluted area						Polluted area					
	08:00 h			18:00 h			08:00 h			18:00 h		
	Rest	60' recovery	*P*	Rest	60' recovery	*P*	Rest	60' recovery	*P*	Rest	60' recovery	*P*
HR (bpm)												
NSG	65 ± 1.35	70.27 ± 2.02	> 0.05	66.91 ± 1.28	72.45 ± 1.52	> 0.05	64.82 ± 1.23	73.4 ± 2.8*	0.014	67.55 ± 0.62	74.8 ± 2.63*	0.048
SG	66.82 ± 1.08	73.18 ± 2.29*	0.0000	68.09 ± 1.13	75.55 ± 2.13*	0.0000	67 ± 1.04	74.27 ± 2*	0.0000	68.36 ± 1.1	75.82 ± 2.1*	0.00000
RBC ($10^6/\mu l$)												
NSG	5.14 ± 0.13	5.16 ± 0.13	> 0.05	4.84 ± 0.14	4.97 ± 0.08	> 0.05	5.12 ± 0.15	5.14 ± 0.13	> 0.05	4.82 ± 0.14	4.88 ± 0.12	> 0.05
SG	5.44 ± 0.07	5.48 ± 0.07	> 0.05	5.27 ± 0.08	5.28 ± 0.12	> 0.05	5.46 ± 0.06	5.39 ± 0.07	> 0.05	5.29 ± 0.09	5.31 ± 0.08	> 0.05
Hb (g/dl)												
NSG	16.21 ± 0.23	16.02 ± 0.26	> 0.05	15.87 ± 0.26	15.47 ± 0.23	> 0.05	15.34 ± 0.24	14.93 ± 0.22	> 0.05	15.25 ± 0.2	14.59 ± 0.2	> 0.05
SG	16.22 ± 0.27	16.17 ± 0.27	> 0.05	16.14 ± 0.32	16.2 ± 0.27	> 0.05	16.95 ± 0.25	16.11 ± 0.29	> 0.05	15.8 ± 0.21	16.08 ± 0.29	> 0.05
WBC ($10^3/\mu l$)												
NSG	6.77 ± 0.13	6.69 ± 0.13	> 0.05	6.97 ± 0.17	6.73 ± 0.17	> 0.05	6.72 ± 0.13	6.8 ± 0.11	> 0.05	7.02 ± 0.1	6.75 ± 0.14	> 0.05
SG	7.59 ± 0.17	7.40 ± 0.15	> 0.05	7.96 ± 0.21	7.46 ± 0.15*	0.0000	7.57 ± 0.16	7.25 ± 0.1*	0.003	7.97 ± 0.2	7.46 ± 0.1*	0.0000
LYM (%)												
NSG	23.7 ± 1.34	19.32 ± 1.5	> 0.05	27.24 ± 1.36	23.95 ± 2.59	> 0.05	31.18 ± 0.79	24.64 ± 1.2	> 0.05	32.32 ± 1.74	26.1 ± 2.31	> 0.05
SG	33.97 ± 0.84	34.42 ± 0.83	> 0.05	33.08 ± 0.87	33.53 ± 0.77	> 0.05	34.19 ± 0.7	35.01 ± 0.68	> 0.05	36.35 ± 0.59	36.99 ± 0.49	> 0.05
NEUT (%)												
NSG	52.43 ± 0.33	71.36 ± 1.8*	0.0000	52.37 ± 0.69	71.18 ± 1.9*	0.0000	52.43 ± 0.3	72.55 ± 2*	0.0000	52.63 ± 0.5	72.55 ± 2*	0.0000
SG	52.65 ± 0.57	59.18 ± 1.2*	0.0000	51.83 ± 0.77	59.09 ± 1.2*	0.0000	52.37 ± 0.64	60.6 ± 1.1*	0.0000	51.69 ± 0.66	61.8 ± 1.03*	0.0000
PLT ($10^3/\mu l$)												
NSG	203.82 ± 109	197.27 ± 10.5	> 0.05	204.09 ± 11	197.1 ± 10.76	> 0.05	204 ± 10.87	198.09 ± 10	> 0.05	203.9 ± 10.6	197.6 ± 10.7	> 0.05
SG	185.64 ± 4.1	187.73 ± 3.8	> 0.05	185.82 ± 4.4	185.55 ± 4.47	> 0.05	185.09 ± 4.3	186.9 ± 6.4	> 0.05	185.82 ± 4.7	186.18 ± 4.8	> 0.05
P_vO_2 (mmHg)												
NSG	43.36 ± 0.74	40.8 ± 0.9*	0.0005	43.91 ± 0.5	39.9 ± 1.35*	0.0000	44 ± 0.66	41.09 ± 1.3*	0.0000	44 ± 0.28	41.91 ± 1.3*	0.0000
SG	42.82 ± 0.46	41 ± 0.47*	0.0002	42.91 ± 0.42	41.27 ± 0.82*	0.005	42.36 ± 0.45	40.36 ± 0.2*	0.0007	42.09 ± 0.37	41 ± 0.57*	0.001
P_vCO_2 (mmHg)												
NSG	47.5 ± 1.86	44.7 ± 1.32*	0.001	47.53 ± 1.6	44.9 ± 1.3*	0.007	47.32 ± 1.9	46.04 ± 1.2*	0.0000	47.42 ± 1.8	46.1 ± 1.4*	0.0005
SG	48.09 ± 1.34	45.5 ± 0.68*	0.001	48.22 ± 1.1	45.5 ± 0.6*	0.004	48.15 ± 1	47.9 ± 0.49*	0.0000	48.01 ± 0.98	47.46 ± 0.3*	0.0000
HCO_3^- (mm/l)												
NSG	24.99 ± 0.42	26.5 ± 1.03	> 0.05	25.08 ± 0.5	27.16 ± 1.04	> 0.05	25.08 ± 0.6	26.8 ± 1.38	> 0.05	24.81 ± 0.82	26.25 ± 1.34	> 0.05
SG	23.88 ± 0.38	24.26 ± 0.9	> 0.05	23.84 ± 0.34	23.72 ± 0.01	> 0.05	23.65 ± 0.44	22.55 ± 0.7	> 0.05	23.52 ± 0.41	22.2 ± 0.51*	0.029
pH												
NSG	7.324 ± 0.01	7.324	> 0.05	7.327	7.326	> 0.05	7.307 ± 0.01	7.303	> 0.05	7.309 ± 0.01	7.318 ± 0.01	> 0.05
SG	7.32 ± 0.01	7.324	> 0.05	7.318	7.323	> 0.05	7.313 ± 0.01	7.306	> 0.05	7.32 ± 0.01	7.306	> 0.05

P_vO_2: partial pressure of oxygen; P_vCO_2: partial pressure of carbon dioxide; HR: heart rate; PH: potential of hydrogen; HCO_3^-: bicarbonate; RBC: red blood cells count; Hb: hemoglobin count; PLT: platelets; WBC: white blood cells count; NEUT: neutrophils; LYM: lymphocytes.

* Significant difference with rest values at $P < 0.05$.

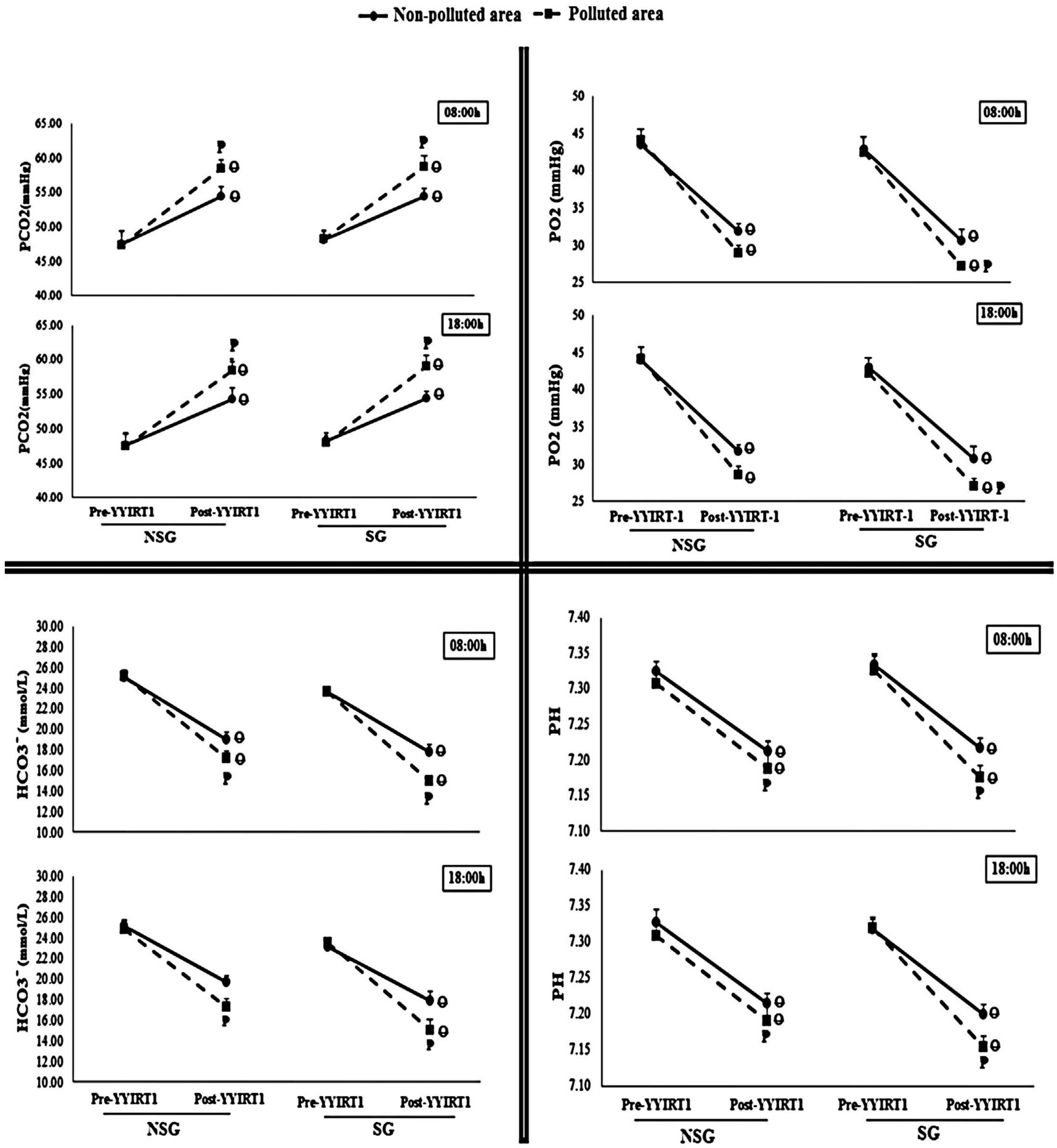

Figure 5 P_vCO_2, P_vO_2, HCO_3^- and pH values (means ± SD, $n = 22$) of both groups (NSG and SG) registered in two areas pre-, post- and 60 min after YYIRT at two TOD (08:00 h and 18:00 h). θ: significant difference with pre-YYIRT-1 at the same time of day at $P < 0.05$; Ᵽ : significant difference with polluted area at $P < 0.05$.

at 18:00 h especially in the evening (−.72 at 08:00 h % vs. −1.76 at 18:00 h %). In fact, it has been established by several studies that smoking blunts HR elevation during progressive exercise [29], as a result of down-regulation of β-adrenergic receptors of the cardiovascular system caused by chronic smoking [9]. Several studies suggests that acute and chronic active smoking generates marked disruptions in the normal autonomic nervous system functioning characterized by increased sympathetic drive, and reduced heart rate variability and parasympathetic modulation [30]. This mechanism seems to be more accentuated under pollutant stressors, indeed exposure to some pollutant while exercising induce alteration in cardiac autonomic control resulting in reduced vagal modulation. Thus, the decreased HR_{max} values in response to the YYIRT-1 under polluted air in smoker adaptation could be explained by the incapacity of the cardiac muscle to adapt the amplified rhythm of transferring oxygen to other required tissues. In our study, there were no signs of unbalanced HR recovery observed in both groups. However, the HR decline during recovery in smokers' athletes under polluted area in the evening didn't attenuate the rest values. Thus, smoking was negatively related with HR alteration after intermittent maximal exercise. More studies are needed to more explain the exact mechanism responsible of these alterations under polluted area upon two times of day.

For both groups, all hematological parameters presented a significant increase following the YYIRT-1 under polluted and clean air upon two times of day. Our results are in agreement with those of Kilic-Toprak et al. [31] investigating the effect of YYIRT-1 on hemorheological parameters in female volleyball players, they attribute this change to the exercise induced acute hemoconcentration. In fact, the increment in the number of erythrocytes and leukocytes in response to stressful stimuli including exercise is a well-known phenomenon, which may partly be explained by altered hemodynamic conditions. That is increased flow and shear forces within the circulation would be expected to lead to recruitment of sequestered RBC in various circulatory beds and of WBC from the marginal pool [31].

Furthermore, concerning pollution effect, NSG presented a significant alteration on RBC and Hb post-exercise upon two times of day with lower values in polluted area compared to NPA. And the same effect was observed in SG concerning RBC, Hb and HTC levels. In this context, Syd-BOm et al. [32] reported a relationship between exposure to particulate matter and changes in hemoglobin concentration and red blood cell count. These fine particulate contaminants in polluted air have a destructive effect on corpuscles, particularly when inflow to the lungs is more demanding and rigorous. Nikolić et al. [33] resolved that the decrease in hemoglobin was caused by increased peripheral sequestration of red blood cells, rather than generalized hemodilution.

Moreover the magnitude of the decrease was more accentuated in SG than NSG regarding RBC (−1.96% vs. −3.14% at 08:00 h and −2.44% vs. −5.39% at 18:00 h) and Hb levels (−2.21% vs. −2.41% at 08:00 h and −0.94% vs. −2.81% at 18:00 h). In fact smokers presented highest levels of RBC and Hb at rest, which is associated with blood viscosity and thrombus formation [34], it has been established that increased level of RBC and hematocrit in smokers slows blood velocity and increase the risk of intravascular clotting during exercise [35], and this fact will be more aggravated under polluted air.

In the other hand, we observed that WBC, LYM and NEUT were significantly increased by pollution during the two TOD in both groups. In fact, following increased exposure to air pollutants can cause probably the leukocytosis by tissue destruction, which increases antibodies production. Previous studies reported significant effects on WBC counts after exposure to lower doses of air pollution, some observed increases in total WBC or neutrophil counts [28,36], while others have found drops in total WBC, neutrophil or monocyte counts [37,38]. Since the major differences in study design it is difficult to compare the outcomes of these studies. Likewise, this alteration was more accentuated at evening hours (Table 3) and this could be related to the circadian rhythm of RBC count seen at rest that decreased significantly at 18:00 h. In fact, the increase of coagulation activity, and the decrease in fibrinolytic activity about 18:00 h could reflect in part the drop of RBC count explained by the increases myocardial oxygen demand, while its supply may decrease [13]. It also may be correlated with the amount of pollutant increased at this time of day.

Also our finding showed higher amount of increase in WBC and LYM in SG compared to non-smoker group after exercising under polluted air upon two times of day. In fact, our results indicated at baseline measures that smoker athletes had significantly higher WBC count at rest compared to non-smokers ($P < 0.05$) in all test session (Table 1). These results are consistent with other published reports [39], and it may be linked to an increase in blood flow, which recruits leukocytes from the marginal pool or hormonal changes. It has been suggested that inflammatory stimulation of the bronchial tract by pollution contaminants induces an increase in inflammatory markers in the blood and also it has been suggested that nicotine may induce an increase in blood lymphocyte counts [25,40]. Moreover, the increase in WBC count in such a context is likely to reflect WBC activation generated by inflammatory mechanisms. This change was established by other studies investigating the effect of exposure to high concentration of pollutants in rat models [41,42]. They reported that intra tracheal instillation of particulate air pollution induces alterations in leukocyte flow properties through trapping of monocytes in the microcirculation [41] and a rise in plasma viscosity after combustion-derived and manufactured nanoparticles exposure [42] which is correlated with increased WBC numbers in circulatory blood. This fact, probably impair microcirculatory flow which increases cardiovascular risk. Therefore, we conclude that higher heart rate in polluted air already mentioned in our results can be due to activated WBC.

Taking into account the physical aptitude of the studied population while comparing our results with the literature concerning the hematological variation after exposure to polluted air during exercise. A study in this field [43] has suggested that there is a linear relationship between increasing in physical activity and decreasing in hematological risk factors, they reported that in Healthy subjects increased levels of physical activity were associated with reduced RBC, WBC, platelet counts and plasma neutrophil elastase concentration. In our study subjects are all well-trained individuals (4 times/week for more than 9 years) and it is suggested that this fact help them to resist in such a stressful situation compared to untrained subjects which may have more serious hematological risks under pollution, so further investigations are needed to test the hypothesis and elucidate hematological effects of pollution in non-trained subjects following intermittent exercise in order to clarify underlying mechanisms and determine to what extent this effect could pose a risk for. In fact our results showed that smoker subjects presented already higher WBC and RBC at rest compared to non-smoker athletes; This partly reflects the wide range of alteration caused by pollution observed in smoker compared with non-smokers, so we conclude that even they have a good physical aptitude, smoking chronically can be a potent factor to induce hematological risks for athletes.

Concerning platelet counts, only NSG were affected by pollution, presenting significantly higher PLT numbers post-exercise compared to NPA measures, which is agreeing with previous studies [8,44]. They attribute this fact to the perturbation of the autonomic nervous system, and translocation of fine particles or its constituents into the circulation and these pathways may result in systemic oxidative stress and inflammation, inducing an increase in circulating platelet counts during exercise. Concerning smoker groups, our results can not be compared to the literature because of limited studies in this field.

Our findings showed also a significant increase of P_vCO_2 and decrease of PH, P_vO_2 and HCO_3^- following the YYIRT. This alteration was significantly accentuated in polluted area for both groups upon two TOD excepting for P_vO_2 levels in NSG. The increase of P_vCO_2 levels in polluted area can be explained by the amount of carbon monoxide inhaled from the air to the respiratory tract that alters the ability of hemoglobin to carry oxygen. Hemoglobin combines with CO to form carboxyhemoglobin [25]. Then, the decline in HCO_3^- and PH could be related to the high level of P_vCO_2.

Additionally, the amount of change under polluted air was more important within smoker group (Fig. 5). Indeed, smoker subjects presented already high levels of COHb derived from the combination of hemoglobin and the CO released from smoke and increased levels of COHb in blood causes tissue hypoxia, this is correlated with upheaval of the acid-base balance in smoker athletes following exercise.

Furthermore, our results showed that the pollution effect was more aggravated at evening hour (18:00 h) for both groups, and this alteration was observed in recovery period when blood gases (i.e. P_vO_2, P_vCO_2, HCO_3^-) didn't attenuate rest values in SG (Table 5). This fact may be attributed to the diurnal variation of some pollutant. Indeed, several studies have reported that most of pollutant concentrations were higher at evening compared to morning hours; it was the case in our study, which present higher values of pollutant at 18:00 h compared to the morning (Table 2). On the other hand, little is known about the diurnal variations in the carbon monoxide diffusing capacity of the lung (DLCO), which is one of the essential indicators of pulmonary function [16]. In this context, Medarov et al. [45] have shown a pattern of circadian rhythm of DLCO (unfortunately not measured in our study) with values higher at evening and this could explain in part the alteration on acid-base balance at this time of day.

5. Conclusion

In conclusion, physiological and blood parameters in smoker and non-smoker athletes were markedly impacted when undertaking exercise under polluted air at two different times of day and the impact was more higher in smoker group especially in the evening. Thus, on the basis of these results, coaches, athletes and medical staff must take precaution regarding the harmful effect of intermittent maximal exercise practice or competitions under polluted air especially for smoker soccer athletes. Regularly diagnostics are recommended for smoker's athletes in order to minimize the risk of cardiovascular diseases and they should well plan their training sessions taking into account spatial and temporal conditions.

5.1. Study limitations

To the best of authors' knowledge, this is the first study to investigate the combined effect of air pollution on performances, cardiovascular, hematological parameters and blood gases on soccer players following the YYIRT-1 at two different times of day. Though, a study like the present one has some limitations. First, the participants were well-trained individuals and it remains unclear whether the results can be applied to sedentary subjects. Second, because of the limited sample amounts, other important inflammatory (Interleukin-6) and oxidative stress (malondialdehyde) markers were not examined.

5.2. Practical recommendations

This approach can be an encouragement for coaches and athletes especially smokers who trained outdoors, to sensitize them of the several risks of pollution and tobacco consumption. Although the effect of smoking and pollution during exercise is unknown, training and competition in the morning under polluted area appears to minimize the damage and soreness after performing intermittent maximal exercise and minimize or to avoid physiological risk face to such problem.

Disclosure of interest

The authors declare that they have no competing interest.

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