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Longitudinal Study of Smoking, Smoking Cessation, and Serum Biomarkers for Hypertensive Male Smokers During One Year

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Abstract

In this study, we attempt to understand the effect of smoking and its cessation on the serum biomarkers of male hypertensives (males with high blood pressure). 400 participants aged 25-70 were recruited from Iraqi specialty clinics. 300 were smokers and 100 were healthy controls. Smokers were classified based on age and smoking history into three groups, Group A (long-term), Group B (mid-term), Group C (recent), and Group D as the control. Serum levels of leptin, growth hormone (GH), beta-catenin, Endothelin-1 (ET-1), and norepinephrine were quantified using ELISA. Group A smokers had the highest mean levels of leptin (8.4±0.34 ng/mL), ET-1 (6.9±0.24 ng/mL), and norepinephrine (1140±34.32 pg/mL). They showed also the lowest levels of GH and beta-catenin (0.2±0.04 ng/mL; 0.21±0.03 ng/mL, respectively). Elevated levels of the mentioned biomarkers persisted even a year after cessation of smoking. This study emphasizes the damaging effect of smoking on some biomarkers related to the cardiovascular and endocrine systems and provides the rationale for conducting a long-term study for hypertensive smokers after cessation.

Keywords: β-catenin, Endothelin-1 (ET-1), Leptin, Smoking, Hypertension.

دراسة طولية لمدة عام واحد حول أثر التدخين والإقلاع عن التدخين على المؤشرات الحيوية في المصل لدى المدخنين الذكور المصابين بارتفاع ضغط الدم

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الخلاصة

في هذه الدراسة تأثير عملية التدخين والإقلاع عنه على بعض أعضاء الجسم من خلال بعض المؤشرات الحيوية في مصل الدم لدى الذكور المصابين بمرض ارتفاع ضغط الدم. اشتغل البحث على 400 مشترك تتراوح اعمارهم بين 25 إلى 70 سنة من عيادات و مستشفيات في التخصصات العراقية (300 مدخن و100 سليم يمثلوا الضبط). قسم المدخونون الى 3 مجموعات حسب العمر وسنة البدء بالتدخين و (أ) تدخينوا زمن طويل (ب) تدخينوا زمن متوسط (ج) تدخينوا زمن قصير و (د) ضابطة. الصمامات المتصلة بالقلب و الهرمونات (leptin, beta-catin, growth hormone, endothelin-1, noardrenaline) وقيست باستخدام الاختبار (ELISA). وجدت المجموعة (أ) أعلى مستوى من اللبتين والاندثيلين والنو أدرينالين وأدنى مستوى من هرمون النمو وبيتا كاتين. تحسن المشاركون الذين أقلعوا عن التدخين بوضوح في هذه المؤشرات بعد عام من الدراسة. النتائج تمثل وطأة التدخين على المؤشرات القلبية الوعائية والاندوكرينية وأهمية الضبط اليدوي المطول للمدخنين الذين عندهم ارتفاع ضغط الدم.

1. Introduction

Smoking is still on of the top global health concerns. Currently, there are over 1.3 billion active smokers. Tobacco use is directly linked to chronic obstructive pulmonary disease, several types of cancer, and numerous metabolic disorders. Tobacco smoke contains thousands of chemicals that impact the body, including nicotine, tar, and poisonous levels of

carbon monoxide [1-3]. Nicotine, a highly addictive chemical, has a stimulating effect on the body. This means that, after repeated use, a person will suffer from increased and chronic levels of hypertension (high blood pressure), increases to the heart rate, and increases to adrenaline production. Exposure to nicotine and the chemicals in tobacco can lead to Peripheral Artery Disease (PAD), heart attacks, and, in conjunction with other factors, results in stokes [4, 5]. Smoking is also a major contributor of oxidative stress, a condition that can lead to the formation of age-related diseases and other conditions. This can lead to wrinkling, premature skin aging, and other conditions [6-8]. Smoking's impact on hormone regulation is, in part, greatly uncharted. Smoking can adversely impact the level of several hormones, including growth hormone, thyroid hormone, and leptin. Leptin is a hormone that is released from fat cells, Lesions of the appetite and energy consciousness. Smoking also affects leve of leptin, especially in the chronic sense. While acute nicotine may cause appetite suppression, chronic smoking leads to a condition known as leptin resistance and subsequently, leptin is essentially non-functional. This paradox results in the obese smoking population, who, due to the fact that leptin is non-functional, and the smoking habit leads to an increased appetite [8-10].

Chronic smoking may suppress the secretion of growth hormones due to probable disruptions at the hypothalamic and/or pituitary levels, which could hinder tissue repair and promote muscle wasting [11-14]. The present study attempts to assess, for the first time in the Iraqi population, the smoking-related impacts and the effects of smoking cessation after a prolonged period on critical serum biomarkers in hypertensive male smokers.

2. Materials and Methodology

This study was carried out at private hospitals and clinics in Iraq. The sample consisted of 400 male participants aged between 25 and 70 years, of which 300 were hypertensive smokers and 100 were healthy non-smoker controls. The smoking cohort was classified into three categories according to their age and smoking duration as follows:

-Group A (Chronic Smokers): 100 hypertensive patients aged 55 to 70 years, with over 20 years of smoking.

-Group B (Mid-term Smokers): 100 hypertensive patients aged 40 to 55 years, with about 10 years of smoking.

-Group C (Recent Smokers): 100 hypertensive patients aged 25 to 40 years, with less than 5 years of smoking.

-Group D (Control): 100 healthy non-smokers aged between 25 and 70 years.

Blood samples were taken in the first year and repeated after one year to measure the changes (if any) in serum levels of leptin, ET-1, β-catenin, GH, and norepinephrine using the Enzyme-Linked Immunosorbent Assay (ELISA) after one year of smoking cessation for participants who quit.

3. Results

3.1 Baseline Biomarker Levels :

At baseline, Group A showed leptin, ET-1, and norepinephrine levels that were the highest among the groups and levels of GH and β-catenin that were the lowest. These results are shown in Table 1 and Figures (1-5).

Table 1- Research Categories, Age Brackets, and Initial Biomarker Data in Hypertensive Male Smokers

Group	N	(Age) (Mean±SD)	(Leptin) ng/mL (Mean±SD)	(ET-1) (ng/mL) (Mean±SD)	(β-catenin) (ng/mL) (Mean±SD)	(GH) (ng/mL) (Mean±SD)	(NE) (pg/mL) (Mean±SD)
A	100	61.96±6.00	8.4±0.34	6.9±0.24	0.21±0.03	0.2±0.04	1140±34.32
B	100	48.13±6.42	7.3±1.07	5.6±0.17	0.27±0.05	0.3±0.07	1078±21.07
C	100	36.23±6.3	5.69±1.09	5.4±0.19	0.29±0.06	0.4±0.09	1010±18.09
D	100	47.06±5.6	4.5±0.68	4.3±0.38	0.34±0.08	8.5±0.98	780±19.68
(LSD)			0.61	0.41	0.11	0.11	10.80

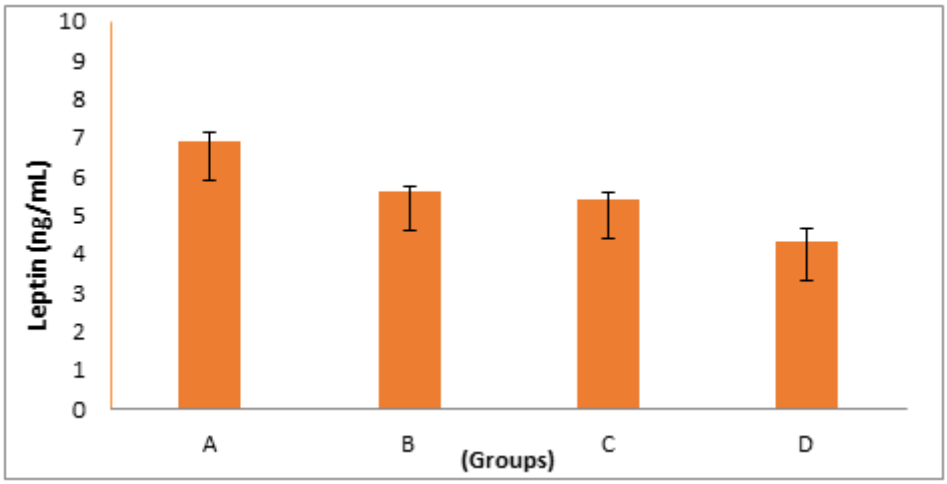

Figure -1 Leptin Serum Levels Across Different Categories of Smokers and Non-Smokers.

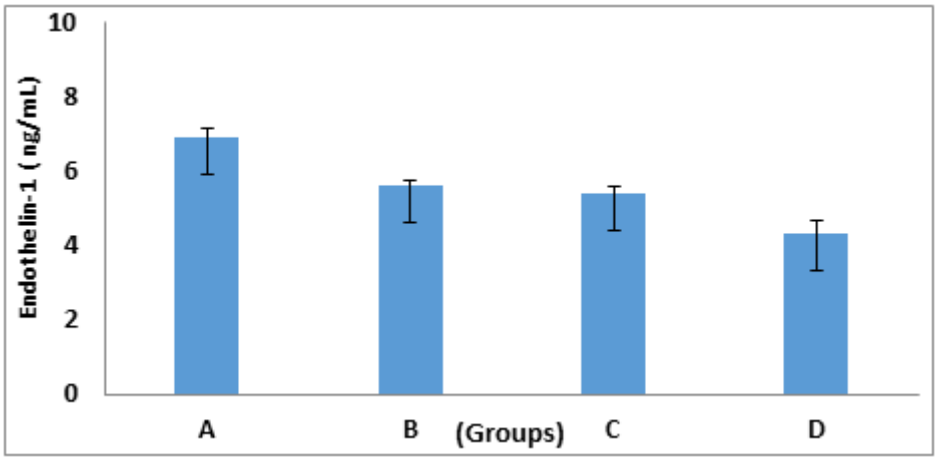

Figure -2 Endothelin-1 serum levels in the three smoker patient groups and the control group

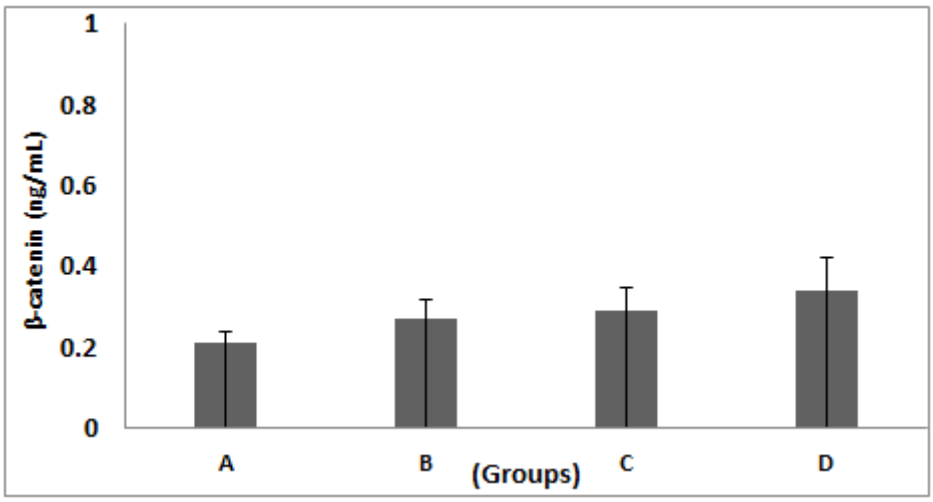

Figure -3 Levels of β-catenin in serum of smoker patients across three groups and the control group.

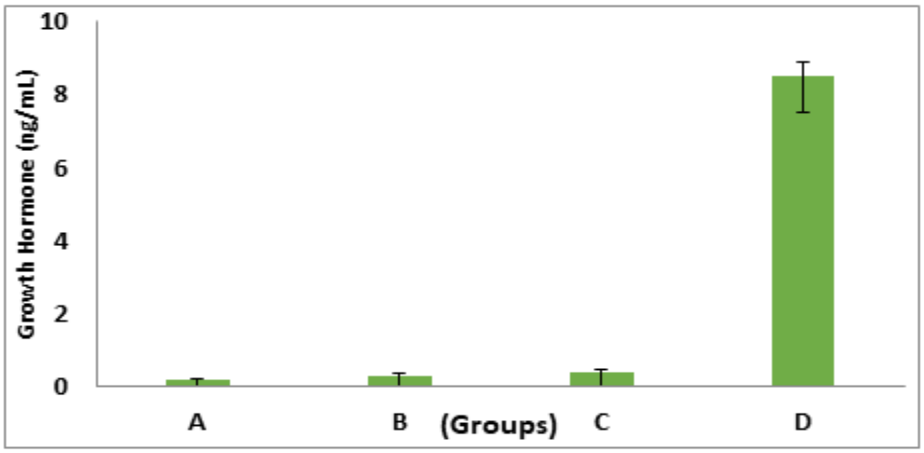

Figure -4 Control group and three smoking patient categories comparison of serum growth hormone levels.

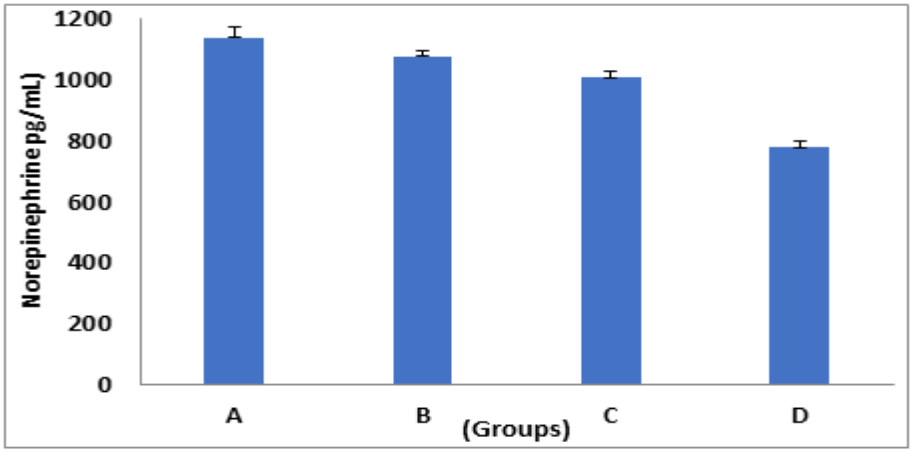

Figure -5 Serum norepinephrine levels across the three groups of smoker patients and the control group.

3.2 Post-Cessation Findings

There have been notable changes in all smoking groups after 1 year abstinence from smoking. Diminished levels of leptin and ET-1 and improved levels of GH and β-catenin were noted. The changes were captured in Table 2 and Figures (6-9).

Table 2 – Biomarker Levels in Hypertensive Male Smokers After One Year of Smoking Cessation.

Group	N	(Age) (Mean±SD)	(Leptin) (ng/mL) (Mean±SD)	(ET-1) (ng/mL) (Mean±SD)	(β-catenin) (ng/mL) (Mean±SD)	(GH) (ng/mL) (Mean±SD)	(NE) (pg/mL) (Mean±SD)
A	100	61.96±6.00	6.4±0.24	6.12±0.14	0.26±0.03	4.2±0.09	1098±44.35
B	100	48.13±6.42	5.3±0.47	5.21±0.13	0.29±0.05	5.13±0.1	1004±24.17
C	100	36.23±6.3	4.69±0.49	4.7±0.19	0.32±0.06	6.14±0.08	791±19.06
D	100	47.06±5.6	4.5±0.68	4.3±0.38	0.34±0.08	8.5±0.98	780±19.68
(LSD)			0.59	0.40	0.12	0.13	9.89

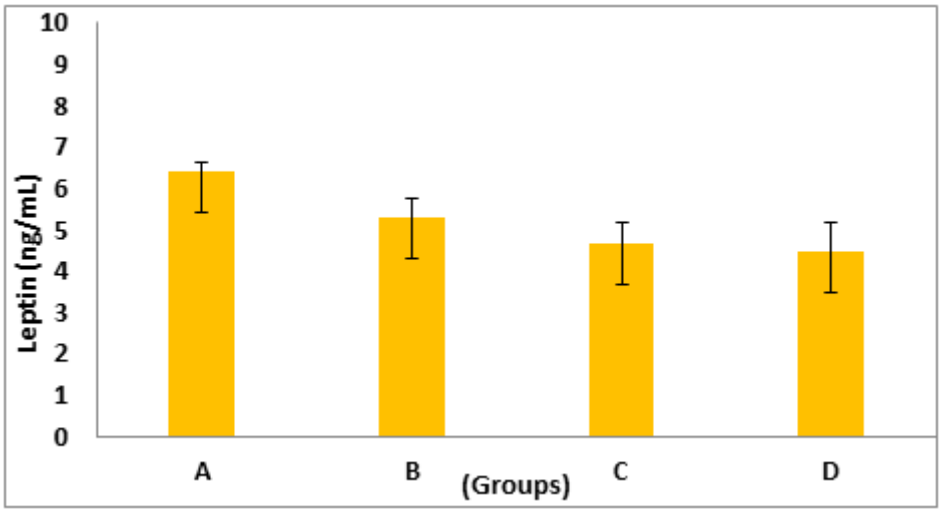

Figure -6 Leptin levels in serum from the control group and three groups of Smoking Cessation patients.

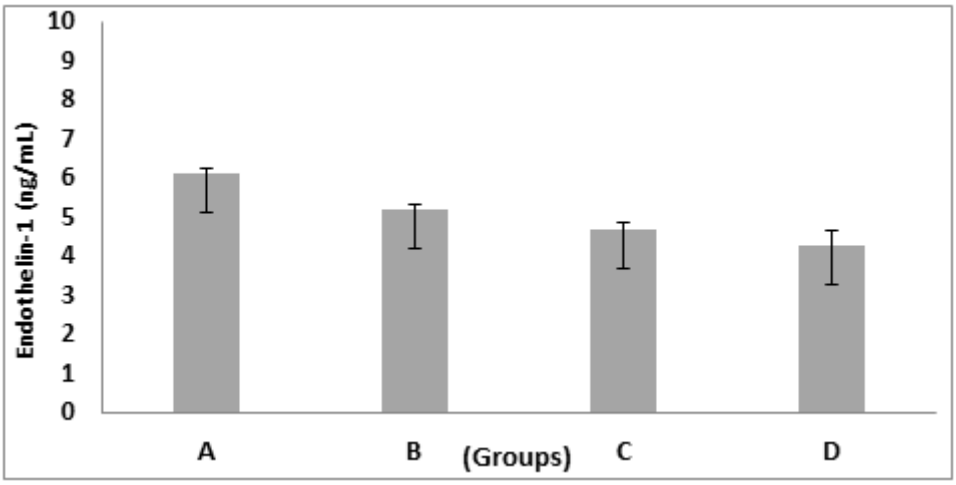

Figure --7 Serum Endothelin-1 levels for the three Smoking Cessation patient groups, and the control group.

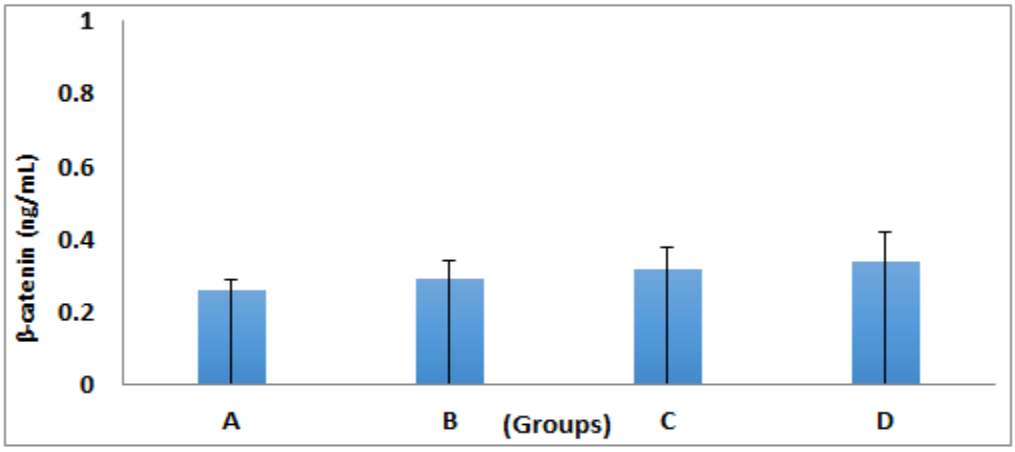

Figure -8 β-catenin serum levels across the three patient groups from Smoking Cessation and the control group.

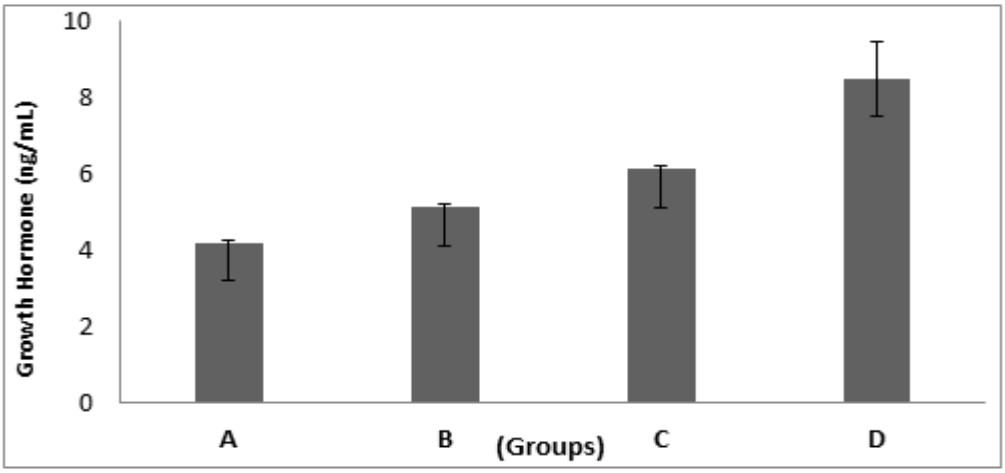

Figure -9 Serum growth hormone levels in the three patient groups from the Smoking Cessation program and the control group.

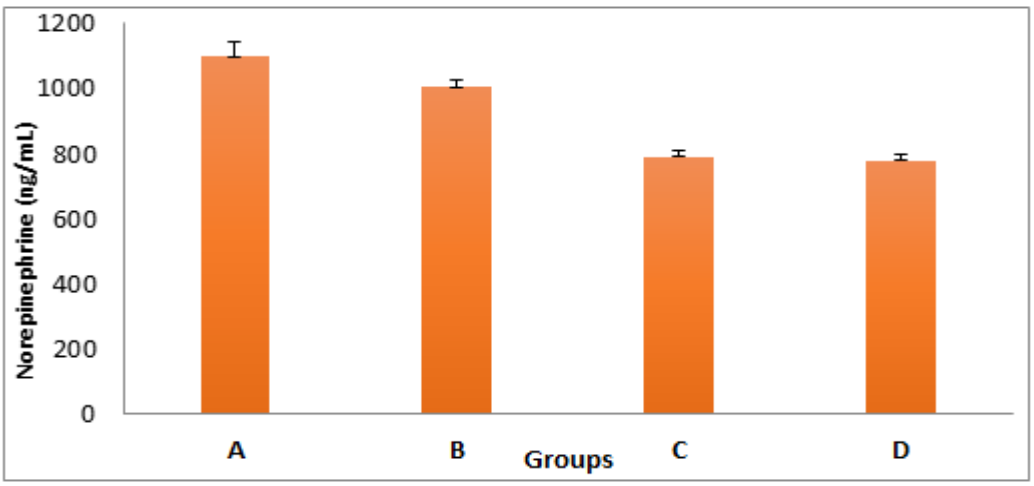

Figure -10 Serum Norepinephrine Levels of the Three Groups of Patients Engaged in Smoking Cessation and the Control Group.

4. Discussion

The smokers showed signs of leptin, ET-1, and norepinephrine, depicting a pro-hypertensive environment. Endothelial dysfunction and neuroendocrine imbalance result from chronic smoking. ET-1 is released because nicotine activates NF-κB, and ET-1 is a vasoconstrictor. Chronic smoking also alters leptin which causes increased leptin resistance and inflammation [15-18].

Aging exacerbates these effects. Among elderly, hypertensive smokers, the positive correlation between leptin and ET-1 indicates a feedback loop of vasoconstriction further potentiated by oxidative stress [19-21]. In addition, the smoking-induced suppression of GH and of β-catenin impairs vascular repair and availability of bioactive nitric oxide. Oxidative stress-mediated β-catenin degradation worsens vascular fibrosis and increases pulse wave velocity, a measure of arterial stiffness, β-catenin degradation worsens vascular fibrosis [22-30].

Increased norepinephrine (NE) and sympathetic hyperactivity are signs of chronic hypertension. The elderly have poor NE clearance leading to the hypertensive smokers (BP ≥160/100 mmHg) [31-37].

5. Conclusions and Recommendations

Smoking has an adverse effect on the homeostasis of the endocrine system and the cardiovascular system due to its effect on leptin, ET-1, Norepinephrine, GH, and β-catenin. These effects, although measurable improvement occurs within one year of smoking cessation, still do not return to normal after one year. We suggest more longitudinal studies on the hypertensive smokers after smoking cessation, and support the implementation of more extensive smoking cessation programs, as they can be of great importance to the cardiovascular risk of this population.

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Ethical Responsibilities of Authors

The authors state that the manuscript hasn't been submitted to other journals, that there is no falsification of data, and that the manuscript is compliant with ethical norms of the research integrity. All research on human subjects is in compliance with the Helsinki Declaration of 1975, and related revisions of the year 2000. Informed consents were signed by the participants. The protocol of the institution (Approval No. 2204, August 7, 2022) was sanctioned by with ethics committee. There are no studies with animals in this manuscript, conducted by any of the authors.

Disclosure and Conflict of Interest

The authors state that they have no conflicts of interest.

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