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Original article

Effect of cigarette smoking on insulin resistance risk

Risque d'insulino-résistance chez les fumeurs

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Abstract

Objectives. – Smoking is one of the main risk factors for cardiovascular disease (CVD). The mechanism(s) of the effects of smoking on CVD are not clearly understood; however, a number of atherogenic characteristics, such as insulin resistance have been reported. We aim to investigate the effects of cigarette smoking on insulin resistance and to determine the correlation between this parameter with smoking status characteristics.

Study design. – This study was conducted on 138 non-smokers and 162 smokers aged respectively 35.6 ± 16.0 and 38.5 ± 21.9 years. All subjects are not diabetic.

Methods. – Fasting glucose was determined by enzymatic methods and insulin by chemiluminescence method. Insulin resistance (IR) was estimated using the Homeostasis Model of Assessment equation: HOMA-IR = [fasting insulin (mU/L) × fasting glucose (mmol/L)]/22.5. IR was defined as the upper quartile of HOMA-IR. Values above 2.5 were taken as abnormal and reflect insulin resistance.

Results. – Compared to non-smokers, smokers had significantly higher levels of fasting glucose, fasting insulin and HOMA-IR index. These associations remained significant after adjustment for confounding factors (age, gender, BMI and alcohol consumption). A statistically significant association was noted between the smoking status parameters, including both the number of cigarettes smoked/day and the duration of smoking, and fasting insulin levels as well for HOMA-IR index. Among smokers, we noted a positive correlation between HOMA-IR index and both plasma thiocyanates and urinary cotinine.

Conclusion. – Our results show that smokers have a high risk to developing an insulin resistance and hyperinsulinemia, compared with a matched group of non-smokers, and may help to explain the high risk of cardiovascular diseases in smokers.

Keywords: Cigarette smoking; Glucose; Insulin; HOMA-IR; Insulin resistance risk

Résumé

Objectifs. – Le tabagisme est l'un des principaux facteurs de risque des maladies cardiovasculaires. Le(s) mécanisme(s) des effets du tabagisme sur les maladies cardiovasculaires ne sont pas encore élucidés. L'objectif de notre travail était d'étudier les effets du tabac sur la résistance à l'insuline et déterminer la corrélation entre ce paramètre avec le nombre de cigarettes fumées/jour ainsi qu'avec l'ancienneté de l'exposition.

Population d'étude. – Notre travail a été mené auprès de 138 fumeurs et 162 non-fumeurs âgés respectivement de 35,6 ± 16,0 et 38,5 ± 21,9 ans. Tous les sujets ne sont pas diabétiques.

Méthodes. – La glycémie été déterminée par une méthode enzymatique et l'insuline par chimiluminescence. La résistance à l'insuline (IR) a été estimée en utilisant l'équation suivante : HOMA-IR = [insuline à jeun (mU/L) × glycémie à jeun (mmol/L)]/22,5. Les valeurs supérieures à 2,5 sont considérées comme anormales et traduisent la résistance à l'insuline.

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Résultats. – Par rapport aux non-fumeurs, les fumeurs avaient des valeurs significativement plus élevées de la glycémie à jeun, de l'insuline à jeun et de l'index HOMA-IR. Après ajustement aux facteurs confondants (âge, sexe, IMC et consommation alcoolique), ces résultats demeurent significatifs. Une association significative a été notée entre les paramètres du statut tabagique (le nombre de cigarettes fumées/jour et l'ancienneté de l'exposition) et l'insulinémie ainsi qu'a avec l'HOMA-IR. Par ailleurs, nous avons retrouvé chez les fumeurs, une corrélation positive entre l'HOMA-IR et les deux marqueurs biologiques du tabagisme (thiocyanates plasmatiques et cotinine urinaire).

Conclusion. – Nos résultats montrent que les fumeurs avaient un risque élevé de développer une résistance à l'insuline expliquant ainsi le risque important de maladies cardiovasculaires chez eux.

Mots clés : Tabagisme ; Glycémie ; Insulinémie ; HOMA-IR ; Risque d'insulino-résistance

1. Introduction

Smoking is one of the main risk factors for coronary heart disease, stroke and sudden cardiac death, both in active smokers and passive smokers. In 2000, 1.69 million residents worldwide dead of coronary heart disease caused by smoking [1]. By 2020, smoking will cause 8.4 million deaths and will be the largest health problem. The leading causes of death from smoking are cardiovascular diseases (1.69 million deaths), chronic obstructive pulmonary disease (0.97 million deaths) and lung cancer (0.85 million deaths). Stopping smoking at age 60, 50, 40 or 30 years leads to a gain of about three, six, and nine or ten years in life expectancy, according to the study among male British doctors followed-up for 50 years. Although, many pharmacological actions of cigarette smoking and nicotine have been demonstrated [2], the mechanisms for the relationship between smoking and cardiovascular disease have not been clarified. Smoking contains more than 2000 chemical materials, including nicotine, polycyclic aromatic hydrocarbons, trace metal ions, pro-oxidants and reactive oxygen species. High insulin concentrations and different degrees of insulin resistance have been found in smokers and in contrast, smoking cessation would significantly increase the insulin sensitivity. Many mechanisms have been proposed to explain the effects of smoking on glucose metabolism, but none has been satisfactory [3].

The aim of this study is to investigate the effect of cigarette smoking on insulin resistance and to determine the correlation between this parameter and smoking status characteristics.

2. Materials

2.1. Study design

2.1.1. Population

The study was performed on 300 voluntary subjects without endocrinological or psychiatric diagnoses and matched for age: 138 non-smokers (62 men and 76 women) aged 38.47 ± 21.91 years and 162 current smokers (145 men and 17 women) aged 35.55 ± 16.03 years. This study was approved by the local ethical committee and all subjects were of Tunisian origin. Written informed consent was obtained from all voluntary adult participants and from the parents of minors.

2.1.2. Samples

After a 12-hour overnight fasting, venous blood for each patient was drawn in tubes containing lithium heparinate and into tubes containing EDTA/K_3 (Becton Dickinson, NJ, USA) and immediately centrifuged. Urine samples were obtained from the smokers and non-smokers. These samples were either used on the same day or frozen at –20 °C until they were required for analysis. All the subjects were tested for urinary cotinine.

2.2. Methods

2.2.1. Smoking questionnaire

Subject information and cigarette smoking outcome data were collected in a structured interview. The available data were limited to the classification of smoking to three categories: never, former, or current. The majority of current and former smokers were able to provide information on the number of cigarettes they smoked and the duration of smoking. All subjects were questioned about their sociodemographic characteristics, including age, gender, education and employment.

2.2.2. Laboratory analysis

Fasting glucose, creatinine, and urinary cotinine levels were determined by the enzymatic colorimetric method on the Konelab 30^{TM} analyzer (Thermo Electron Corporation). Cotinine was expressed as micrograms per micromol of creatinine in urine. Insulin was determined using chemiluminescence (Elecsys 2010^{TM} Roche diagnostics). SCN^- levels were determined using selective electrodes (Ionometer Seven Multi S80^{TM}, Mettler Toledo, Schwerzenbach, Switzerland) and expressed as milligrams per liter in plasma.

2.3. Data measures

Insulin resistance (IR) was estimated using the Homeostasis Model of Assessment equation:

HOMA-IR = [fasting insulin (mU/L) × fasting glucose (mmol/L)]/22.5. IR was defined as the upper quartile of HOMA-IR. Values above 2.5 were taken as abnormal and reflect insulin resistance.