

GLYCEMIC CONTROL IN SMOKERS AND RISK OF TYPE 2 DIABETES

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ABSTRACT

Chronic cigarette smoking seems to markedly aggravate insulin resistance that elevates blood glucose. Weight gain usually follows the cessation of smoking, and increasingly, fear of weight gain discourages many smokers from attempting to quit. Yet, weight gain after quitting smoking has proved so unresponsive to preventive attempt as to suggest that weight control might even be incompatible with successful smoking cessation. However, recently featured review and research on smoking and diabetes concluded the evidence that smoking is associated with the preparation for the development of Type 2 diabetes.

Keywords: Smoker, Weight gain, Type 2 diabetes.

INTRODUCTION

Type 2 diabetes (insulin resistance) develops when the body cannot maintain insulin or due to impaired insulin functions depending on age, body mass index (BMI), and adiposity, common prevalence is after the age of 40. Both genetic and environmental factors contribute to the development of diabetes, but the development of Type 2 diabetes is more likely if some or all of the following factors are also present: Physical inactivity, being overweight, family history of Type 2 diabetes, previous diabetes in pregnancy as well as recently documented literature which shows increase insulin resistance occurs normal or diabetics, hence smoking is one of the risk factors for Type 2 diabetes [1,2]. A majority of women say that the prospect of weight gain increases their reluctance to stop smoking [3].

CIGARETTE SMOKING AND TYPE 2 DIABETES

Cigarette smoking is one of the major risk factors of coronary heart disease and stroke but has no any well-documented association for Type 2 diabetes, though both have common causal factors [2-4]. However, recently featured review and research on smoking and diabetes concluded the evidence that smoking is associated with the preparation for the development of Type 2 diabetes in men and women [4-6], consistent with evidence linking smoking and insulin resistance [7-9]. Smoking cessation is often accompanied by substantial weight gain [10], and obesity is an important risk factor for the development of diabetes [11].

FREQUENCY OF SMOKE AND RISK OF TYPE2 DIABETES

Risk of Type 2 diabetes was significantly increased in cigarette smokers compared with never-smokers, despite their lower body weight (adjusting the BMI). With similar significant increase in risk in both light smokers (1-19 cigarettes per day) and heavy smokers (≥ 20 cigarettes per day), the relative risk of diabetes, adjusted for obesity, and other risk factors was significant among women who smoked 25 or more cigarettes per day compared with nonsmokers [4,6]. Contrary to the other study that follows dose-dependent risk of smoking [12].

SMOKING AND INSULIN RESISTANCE

Recent development has made the non-invasive technique to measure the Umbilicus Waist circumference, the strong marker of obesity [Centripetal fat depots] and it is considered to be one of the crucial reason for decreased insulin sensitivity [6,8] and thus Impaired glucose tolerance acutely [13] and hyperinsulinaemia in chronic

smoking. Recent study demonstrated heightened insulin level with decreased sensitivity in smokers as compared with nonsmoking control group [14] and evidence suggests that an improvement in insulin sensitivity and increase in high-density lipoprotein cholesterol occurs after cessation of smoking [15].

SMOKING CESSATION AND RISK OF DIABETES

Published literature documents revealed that smoking cessation increases insulin sensitivity and progressively amends lipoprotein profile, despite a modest increase in weight suggesting that the smoking-related risk of diabetes is reversible in individuals who quit smoking. In the long run, beneficial effects of smoking cessation outweigh the effects of weight gain; ex-smokers of ≥ 20 years' duration are no longer at increased risk of diabetes. Men who quit smoking within 5 years before screening were substantial weight than current smokers and significant weight gain was seen in subjects who quit smoking during the first 5-year follow-up. Men who quit during the 5 years before screening showed no reduction in risk compared with current smokers and the increased risk in this group was most marked in obese subjects. In addition, the increased risk was confined to those who had smoked for ≥ 30 years' [16,17].

SMOKING AND INSULIN RESISTANCE

Importantly, an improvement in insulin sensitivity and increase in high-density lipoprotein cholesterol occurs after cessation of smoking [16]. Even though smoking is associated with insulin resistance, a significant effect on HbA1c in Type 2 diabetic subjects has not been reported [17]. In Type 1 diabetic subjects, insulin requirements have also been found to be either similar [18] or increased [19] in smokers.

POSSIBLE MECHANISM OF INSULIN RESISTANCE AND WEIGHT GAIN

Mechanism of insulin resistance in smokers

The counter-regulatory hormone such as growth hormone (GH), cortisol, and catecholamine could be the culprit in insulin resistance documented among smokers [20] however, a recent study demonstrated the sensitivity of insulin is not associated with increase of counter-regulatory hormone after smoking in Type 1 diabetes [21]. On the contrary, report is on the way of debate with presentation that smoking in patients with insulin-dependent diabetes is strongly linked with secretion of GH, arginine vasopressin, and cortisol responses than in normal subjects but also enhances the counter-regulatory responses to insulin-induced hypoglycemia [22]; one of the strong reason attributed

to have role in pathogenesis of diabetic in smokers. Sonksen *et al.* [23] as well reported that secretion in excess of GH could be associated with the development insulin resistance [24].

Mechanism of post-cessation weight gain

It is hypothesized that more than one factors might have role in post-cessation weight gain as there is no any alteration in physical activity after smoking cessation, but report is emerging on the side that post-cessation weight gain reduces energy expenditure 418 kJ (100 kcal)/day, whereas energy intake increases by 627-1463 (150-350 kca)/day which is responsible for 39% and 69% variance of post-cessation weight gain [25,26].

Smoking cessation also considerably alters so food craving behavior where selectivity is preferred more toward carbohydrate-rich snack foods that is a direct source to convert fat depots hence increasing weight [27-30].

The observation that ex-smoker's heightened intake of sweets is presumably to enhance efficacy in dispelling the agitation and dysphoria that result from a functional deficiency in brain serotonin level and dopamine level [30,31]; however, weight gain is undesired health hazard to this behavior.

CONCLUSION

Considerably, the residual effects of being chronic smoker are said to be associated with increased in abdominal obesity the mechanism might be endocrine mediated, one of the major risk factors of Type 2 diabetes along with substantial weight gain during the first 3-5 years after quitting of smoking. However, in the long term, quitting smoking seems beneficial and is associated with a reduction in risk compared with current smokers. As the increased risk is seen in both men who gained weight and who did not, it suggests that the remnant effects of smoking are possibly of greater importance than the weight gain. Therefore, the suggestion is to quit smoking, a better idea than being fearful of weight gain.

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