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Letter to the Editor

Determining the combined effects of smoking and obesity on insulin resistance and inflammation

Dear Editor,

We have greatly enjoyed reading the article by Szulinska, et al[1] entitled "Evaluation of insulin resistance, tumor necrosis factor alpha, and total antioxidant status in obese patients smoking cigarettes," which was published in the previous issue of European Review for Medical and Pharmacological Sciences.

The authors[1] aimed to determine the potential influences of obesity and smoking on tumor necrosis factor alpha (TNF-α) and insulin resistance. They found that TNF-α concentrations, as well as insulin resistance levels, in obese patients significantly exceeded those observed in the control. In the group of obese patients who actively smoked, further increases in TNF-α and insulin resistance were noticed. And they observed a positive correlation between TNF-α and homeostasis model assessment-insulin resistance (HOMA-IR) in the overall population. We thank authors for the excellent data but we have some comments about the study.

Even higher waist circumference, as well as insulin concentration and HOMA-IR, were observed in the normal weight smokers, there was no information about significance and *p* value.

In this study, the results demonstrated a significant influence of smoking and obesity on HOMA-IR and TNF-α concentration. Moreover, they observed coexistence of smoking and obesity significantly aggravates the abnormalities. The hydrolytic cleavage of triglyceride (TG) by adipose triglyceride lipase (ATGL) generates fatty acids, which are used as essential precursors for lipid and membrane synthesis, and energy metabolism[2-3]. It is postulated that dysfunctional lipolysis associated with smoking due to ATGL dysfunction in macrophages could result in accumulation of TG and cholesteryl esters; attenuates the expression of the inflammatory cytokines such as interleukin-6 and TNF-α; and contributes to the pathogenesis of obesity and insulin resistance[2-5]. Further studies are required to explain this relationship in more detail.

In addition to correlation analysis, if multivariate regression analysis as well as receiver operating characteristic curve analysis were performed, the odds ratios of independent predictors could have been derived. Consequently, given the exact *p* values and the data of regression analysis; the relationships between obesity, smoking, insulin resistance and inflammation could have been understood in more detail and clearly.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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