LETTER TO THE EDITOR
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Comment on "Association between Interleukin-32 and Interleukin-17A Single Nucleotide Polymorphisms and Serum Levels with Polycystic Ovary Syndrome"

Saba Zakeri[1], Rozita Naseri[2], and Zohreh Rahimi[3]

[1] Department of Clinical Biochemistry, Medical School, Kermanshah University of Medical Sciences, Kermanshah, Iran
[2] Department of Internal Medicine, Medical School, Kermanshah University of Medical Sciences, Kermanshah, Iran
[3] Medical Biology Research Center, Kermanshah University of Medical Sciences, Kermanshah, Iran

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DEAR EDITOR

We read with great interest the article by Hesampour et al[1] published recently in the Iranian Journal of Allergy, Asthma and Immunology. Polycystic ovary syndrome (PCOS) is a common and chronic disorder of endocrine glands and is considered as a low-grade inflammatory disease that affecting around 6% of the women at the reproductive age.[2] The main cause of PCOS is not known, but studies have suggested a major role for genetic factors in susceptibility to the disease. Cytokines as cell-signaling molecules are involved in many intercellular communications such as inflammatory responses. Regarding, the role of cytokines in the regulation of inflammation, their polymorphisms through changes in the gene expression and their serum levels might play a role in the pathogenesis of PCOS.[3] Among patients with PCOS obesity is common characteristic and more than 50% of PCOS patients are overweight.[4] Also, obesity affects the secretion of the inflammatory cytokines from adipose tissue.[5] However, in the article by Hesampour et al[1] the weight and body mass index (BMI) of patients (overweight) were significantly higher than controls (normal BMI) that might affect the obtained results. So, it suggests controls and patients be BMI-matched and free of any inflammatory disease. As the authors mentioned in the introduction this syndrome occurs in women during gestational ages. The correct age ranges for occurring the syndrome is 20-45 years old. Also, in the introduction, the chance for developing PCOS in mothers and sisters of those women affected with PCOS could be 35 and 40%, respectively. The methods of detection and also the time of sampling and the day of menstrual cycle affect the level of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) that should be considered in discussing obtained FSH and LH levels. The levels of FSH and LH need to be detected in days 2-5 of the menstrual cycle. The authors should consider that the interleukin (IL)-17A rs2275913 in both patients and controls deviated from Hardy-Weinberg equilibrium [(χ^2=88.76, p<0.001 and (χ^2=30.18, p<0.001, respectively)] that could influence on the interpretation of the results and the obtained findings need to be commented with caution. It is not clear that the frequency of GC haplotype in comparison with which haplotype is different (p=0.05). Also, as the authors have mentioned in the statistical analysis the p values<0.05 were considered as statistically significant. So, this level of difference should not be considered as a significant level in discussing obtained results. The

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Corresponding Author: Zohreh Rahimi, PhD;
Department of Clinical Biochemistry, Medical School, Kermanshah University of Medical Sciences, Daneshgah Avenue, Kermanshah, P.O.Box: 67148-69914, Iran. Tel: (+98 833) 4274 882, Fax: (+98 833) 4276 471, E-mail: zrahimi@kums.ac.ir

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authors, in conclusion, have explained that the IL-32 rs9927163 (G>T) might be associated with susceptibility to PCOS among Iranian women. However, according to Table 3, this polymorphism had a protective role against PCOS. Since the frequency of T allele was lower in patients than controls and the odds ratio (OR) for T allele compared to G allele was OR=0.696 (95%CI 0.501-0.967, p=0.03), this allele could be protective against PCOS. The authors have not detected the IL-17A level in most patients and there was no available level for it among patients and controls, so serum level of this interleukin in the title could be considered as a typographic error. Further, due to presenting IL-32 level in a graph and not in a Table, the exact values of this interleukin in patients and controls are not clear and the obtained results should be commented with caution.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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