P035

The Joint Effects of human 8-oxoguanine DNA N-glycosylase 1 (*hOGG1*) Genotypes and Smoking on Endometriosis in Taiwan

王子嘉[1], 蔡佳紋[1,3], 施亮均[1,2], 應宗和[1], 謝逸憲[1], 張文馨[1,4], 黃中佑[5,6], 潘信保[5], 稅皓靄[6], 包大藹[1,2,4]

Tzu-Chia Wang[1], Chia-Wen Tsai [1,3], Liang-Chun Shih[1,2], Tsung-Ho Ying[1], Yi-Hsien Hsieh[1], Wen-Shin Chang[1,4], Chung-Yu Huang[5,6], Sin-Bao Pan[5], Hao-Ai Shui[6], Da-Tian Bau[1,2,4]

[1]Terry Fox Cancer Research Laboratory,
[2]Department of Otolaryngology, China Medical University Hospital,
[3]Graduate Institute of Basic Medical Science,
[4]Graduate Institute of Clinical Medical Science, China Medical University,
[5]Taichung Armed Forces General Hospital,
[6]Graduate Institute of Medical Sciences, National Defense Medical Center

Backgrounds: This study has two aims 1) to evaluate the association between *hOGG1* genotypic polymorphism and endometriosis risk 2) to investigate the joint effects of *hOGG1* genotype and smoking habit on endometriosis susceptibility in Taiwan. For this purpose, the well-known polymorphic variants of *hOGG1*, codon 326, was genotyped and analyzed of its association with the risk of endometriosis. **Materials and Methods:** In total, 153 patients with endometriosis and 636 non-endometriosis healthy controls were recruited and genotyped. **Results:** The results showed that the *hOGG1* codon 326 genotypes were not differently distributed between the endometriosis and non-endometriosis control groups in both genetic (P=0.6212) and allelic (P=0.4006) frequency analysis. We have further analyzed the genetic-smoking joint effects on endometriosis risk and found an obvious interaction between *hOGG1* codon 326 genotypes and smoking status. The *hOGG1* codon 326 genotypes were increased in endometriosis risk only in the smoker groups (P=0.0061), but not in the non-chewer group (P=0.0648). **Conclusion:** Our results provide the evidence that the *hOGG1* codon 326 genotype may have a joint effect with smoking on the development of endometriosis.