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Invitation to Review for the Gout and Hyperuricemia

1 message

editorial@gouthyperuricemia.com <editorial@gouthyperuricemia.com> Sun, Nov 8, 2015 at 11:20 PM
To: scguo@ucsd.edu

09-Nov-2015

Dear Dr. Guo:

Manuscript ID GH-2015-0022.R1 entitled "Polymorphisms in insulin-like growth factor 1 receptorand PDZ domain containing 1 are associated with phenotype gout in Chinese Han males" with Dr. Li as contact author has been submitted to the Gout and Hyperuricemia.

I invite you to review this manuscript. The abstract appears at the end of this letter, along with the names of the authors. Please let me know as soon as possible if you will be able to accept my invitation to review. If you are unable to review at this time, I would appreciate you recommending another expert reviewer. You may e-mail me with your reply or click the appropriate link at the bottom of the page to automatically register your reply with our online manuscript submission and review system.

Once you accept my invitation to review this manuscript, you will be notified via e-mail about how to access ScholarOne Manuscripts, our online manuscript submission and review system. You will then have access to the manuscript and reviewer instructions in your Reviewer Center.

I realize that our expert reviewers greatly contribute to the high standards of the Journal, and I thank you for your present and/or future participation.

Sincerely,
Dr. Shun-Jen Chang
Gout and Hyperuricemia Associate Editor
editorial@gouthyperuricemia.com

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MANUSCRIPT DETAILS

TITLE: Polymorphisms in insulin-like growth factor 1 receptorand PDZ domain containing 1 are associated with phenotype gout in Chinese Han males

AUTHORS: Wang, Xiaomin; Li, Xinde; Liu, Shiguo; Zhou, Zhaowei; Miao, Zhimin; Chen, Ying; Li, Changgui

ABSTRACT: Objectives: Three single nucleotide polymorphisms (SNPs), rs6598541 in insulin-like growth factor 1 receptor(IGF1R) gene, rs7224610 in hepatic leukemia factor (HLF) gene and rs1967017 in PDZ domain containing 1(PDZK1) gene, were proved to be significantly associated with the risk of hyperuricemia and phenotype gout in European and Polynesia descent. However, evidence for association with gout in Chinese is adequate. Thus, we conduct this association study to identify whether variants of these three loci affected the susceptibility to gout in Chinese Han males. Methods: 932 gout patients and 1,124healthy controls were recruited for this study. The final results were obtained by sequenator. Association with gout was tested by logistic regression with false discovery rate adjusted and genotype-phenotype analysis was also conducted.

Results: Rs6598541 and rs1967017 showed a significant difference in allele frequencies between gout and control groups. Only rs1967017 showed a significant difference in genotype (rs6598541, P=0.033, OR=1.12 by allele level; P=0.099 by genotype level; rs1967017, P=0.009, OR=1.43 by allele level; P=0.015 by genotype level). Whereas rs7224610 was not replicated (rs7224610, P=0.990, OR=1.03 by allele level; P=0.802 by genotype level). With T-allele carriers of rs1967017 showing an increased risk of gout (OR=1.43, 95%CI=1.12-1.82), TT had a higher risk possibility as the recessive genotype (P=0.024, OR=1.33, 95%CI=1.08-1.65). Conclusion: We validated rs6598541 and rs1967017 to be associated with gout in Chinese Han male population and unrelated with uric acid level. It may cause gout by other ways. This study provides the new possible targets for diagnosis and treatment of gouty arthritis.