CORRIGENDUM

Genetic variations and heterosexual HIV-1 infection:
analysis of clustered genes encoding CC-motif
chemokine ligands

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Since the publication of this paper, the authors have noticed that *CCL3* intron 3 is incorrectly used in the abstract, sixth and eleventh paragraphs, and that references 17 and 18 are irrelevant to the study cohort. The correct paragraphs and replacement references appear below.

Abstract

Several CC-motif chemokine ligands (CCLs) can block HIV-1-binding sites on CC-motif chemokine receptor 5 (CCR5) and inhibit viral entry. We studied single-nucleotide polymorphisms (SNPs) in genes encoding three CCR5 ligands (*CCL3* (MIP-1 α), *CCL4* (MIP-1 β) and *CCL5* (RANTES)) along with an adjacent gene encoding a CCR2 ligand (*CCL2* (MCP-1)) to identify candidate markers for HIV-1 infection and pathogenesis. Analyses of 567 HIV-1 serodiscordant Zambian couples revealed that rs5029410C (in *CCL3* intron 2) was associated with lower viral load (VL) in seroconverters, adjusted for gender and age (regression $\beta = -0.57 \log_{10}$, $P = 4 \times 10^{-6}$). In addition, rs34171309A in *CCL3* exon 3 was associated with increased risk of HIV-1 acquisition in exposed seronegatives (hazard ratio = 1.52, $P = 0.006$ when adjusted for VL of the initially seropositive partner and genital ulcer/inflammation). SNP rs34171309 encodes a conservative Glu-to-Asp substitution. Five neighboring SNPs in tight linkage disequilibrium with rs34171309 all showed similar associations with HIV-1 acquisition. How these multiple *CCL3* SNPs may alter the occurrence or course of HIV-1 infection remains to be determined.

Sixth paragraph

Of the few reports on CCL gene variants and HIV-1 disease progression,^{13,21–24} none of them addressed the association

between CCL variants and HIV-1 VL. By linear regression, we tested the effect of the minor SNP allele on chronic-phase VL in index partners and the set-point VL taken at 6 months after the imputed infection date in seroconverters.¹¹ Carriage of one copy of the C allele of rs5029410 in *CCL3* intron 2 was strongly associated with lower VL in the seroconverters (regression $\beta = -0.57 \log_{10}$, $P = 4 \times 10^{-6}$) with adjustment for age and gender. However, this SNP showed no association with VL in index partners ($\beta = 0.05 \log_{10}$, $P = 0.46$). Index partners did not receive antiretroviral therapy, but their average duration of infection was much longer than that of seroconverters. Thus, the SNP variant might exert its effect only early in HIV-1 infection rather than later.

Eleventh paragraph

We did not adjust *P*-values for the number of tests implied by the number of SNPs eligible for analysis, because a number of SNPs within each gene are in high LD with each other and the number of independent tests would actually be considerably lower. Only the strong association of rs5029410C (*CCL3* intron 2 variant) would have withstood even conservative Bonferroni correction. The false discovery probability (*Q* value) for rs5029410C and rs34171309A was 0.0001 and 0.03, respectively.

References

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18. Kempf MC, Allen S, Zulu I, Kancheya N, Stephenson R, Brill I, et al. Enrollment and retention of HIV discordant couples in Lusaka, Zambia. *J Acquir Immune Defic Syndr* 2008; 47:116–125.

The authors would like to apologise for these errors.