

***KIR* haplotypes defined by segregation analysis in 59 Centre d'Etude Polymorphisme Humain (CEPH) families**

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In the original version of the manuscript, we failed to discuss and cite a key study closely related to ours (Norman et al. 2004). This study described and analyzed data from 27 of the 57 families used in our study. Thus, our study extends that of Norman et al. by including 30 additional families. We view our manuscript as a source of reference material as opposed to the analytical synthesis provided by

the Norman et al. study. We sincerely apologize for our omission and wish to correct the manuscript as follows:

1. Norman et al. 2004 should be included as one of the citations for the sentence below:

Human populations can vary remarkably in the frequencies of *KIR* genes and haplotypes, possibly a reflection of selection pressures conferred by population-specific diseases (Cook et al. 2003; Crum et al. 2000; Denis et al. 2005; Frassati et al. 2006; Gendzekhadze et al. 2006; Jiang et al. 2005; Kulkarni et al. 2008b; Middleton et al. 2007; Norman et al. 2004; Norman et al. 2001; Rajalingam et al. 2002; Toneva et al. 2001; Velickovic et al. 2006; Whang et al. 2005; Witt et al. 1999; Yawata et al. 2002).

2. A statement referring to the families used in the study should have read as follows:

In order to more fully characterize the *KIR* locus in Caucasians, we typed for the presence/absence status of 16 *KIR* genes (Martin and Carrington 2007) in members of 57 CEPH families of European descent and determined haplotypes based on segregation analysis (Figure 1). These families included one Amish family, ten French families, and forty six families from Utah. *KIR* data from 27 of the 46 Utah families used in the present study were previously reported by Norman et al. 2004. Our study includes an additional 30 families and thus is an extension of this previous work.

Norman PJ, Cook MA, Carey BS, Carrington CVF, Verity DH, Hameed K, Ramdath DD, Chandanayingyong D, Leppert M, Stephens HAF, Vaughan RW (2004) SNP haplotypes and allele frequencies show evidence for disruptive and balancing selection in the human leukocyte receptor complex. *Immunogenetics* 56:225–237 doi:10.1007/s00251-004-0674-1

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