results in the French study[1] cannot be ruled out as a reason for the neutral results observed in the meta-analysis. Also, the heterogeneity of the population (ie, *MMR* gene) is not taken into account when a meta-analysis is performed and as shown by Talseth-Palmer *et al*[3] this can drastically affect the observed results.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1 Houlle S, Charbonnier F, Houivet E *et al*: Evaluation of Lynch syndrome modifier genes in 748 MMR mutation carriers. *Eur J Hum Genet* 2011; **19**: 887–892.
2 Wijnen JT, Brohet RM, van Eijk R *et al*: Chromosome 8q23.3 and 11q23.1 variants modify colorectal cancer risk in Lynch syndrome. *Gastroenterology* 2009; **136**: 131–137.
3 Talseth-Palmer BA, Brenne IS, Ashton KA *et al*: Colorectal cancer susceptibility loci on chromosome 8q23.3 and 11q23.1 as modifiers for disease expression in lynch syndrome. *J Med Genet* 2011; **48**: 279–284.

Reply to Talseth-Palmer *et al*

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Following the publication of our article entitled 'Evaluation of Lynch syndrome modifier genes in 748 MMR mutation carriers'[1] in which we reported that in MMR mutation carriers 8q23.3 and 11q23.1 polymorphic alleles were not significantly associated with an increased colorectal cancer (CRC) risk, Talseth-Palmer *et al*[2] indicated that we did not correctly report their results by indicating: 'During the submission of this study Talseth-Palmer *et al* reported that in *MLH1* carriers, but not in *MSH2* carriers, the 11q23.1 *CC* and 8q23.3 *AC* genotypes were associated with an increased risk, but this significant association detected in 373 Australian mutation carriers was not found in 311 Polish mutation carriers analysed in the same study'. Their study was indeed performed in two distinct samples of MMR mutation carriers, originated from Australian and Polish families, respectively.[2] As indicated in Figure 1C of their article, the variation in CRC risk according to the 11q23.1 *CC* genotype was not statistically significant in the Polish sample cohort, but only a trend was observed (log-rank P=0.1336; Wilcoxon P=0.1109, and tware P=0.117). Moreover, the variation in CRC risk according to the 8q23.1 genotype was significant only in the Australian sample whereas no results are reported for the combined sample or the Polish sample, likely pointing to non-significant results. Therefore, our comment is appropriate. Moreover, the combination of the Australian and Polish MMR mutation carrier performed in their study amounts to a meta-analysis using pooled data from two different populations. Finally, all significant differences reported were restricted to *MLH1* mutation carriers and no results were reported for *MSH2* mutation carriers or for all subjects, which also raises questions on the real impact of the 8q23.3 and 11q23.1 genotypes on the CRC risk in MMR mutation carriers. Therefore, the title of their article 'Colorectal susceptibility loci on chromosome 8q23.3 and 11q23.1 as modifiers for disease expression in Lynch syndrome' appears too broad. We do not agree with their conclusion suggesting that 8q23.3 and 11q23.1 genotyping might have a clinical utility in *MLH1* mutation carriers.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Association study of the single nucleotide polymorphisms of *PARK2* and *PACRG* with leprosy susceptibility in Chinese population

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Leprosy is a chronic infectious disease caused by *Mycobacterium leprae*, affecting both the skin and peripheral nerves. It has long