**MET** mutation and familial gastric cancer

J D Chen, S Kearns, T Porter, F M Richards, E R Maher, B T Teh

**Methods**

To examine these patients for germline **MET** gene mutations within the juxtamembrane and tyrosine kinase domains, polymerase chain reaction (PCR) was carried out in exons 14 and 16 to 21 in a 50 µl reaction volume containing 50 ng DNA, 20 mmol/l Tris-HCl (pH 8.4), 50 mmol/l KCl, 1.5 mmol/l MgCl₂, 0.2 µmol/l each primer, 0.2 mmol/l dATP, dGTP, dCTP, dTTP each, and two units of Taq DNA polymerase (GIBCO-BRL, Life Technologies). Amplification was carried out in a programmable thermal cycler (GeneAmp PCR system 9700, Perkin-Elmer) at the following settings: after a denaturation at 94°C for five minutes, samples were amplified for 35 cycles at 94°C for 30 seconds, 55-58°C for 30 seconds, and 72°C for 45 seconds, with a final extension at 72°C for 10 minutes. After amplification, all the PCR products were subjected to purification using Microcon YM-100 column (Amicon, Millipore) and direct sequencing using ABI PRISM BigDye Terminator cycle sequencing ready reaction kit (PE Applied Biosystems).

**Results**

We did not find any **MET** mutation in the juxtamembrane or tyrosine kinase domains in a large series of familial gastric cancer cases, suggesting that **MET** gene mutations are an infrequent cause of gastric cancer susceptibility in the sample studied. To date, germline **CDH1** mutations have only been described in familial diffuse gastric cancer. The **MET** gene mutation (P1009S), reported by Lee et al,™ was found in a Korean patient with intestinal gastric cancer (J-H Lee, personal communication); it is possible that this mutation may be ethnic specific and not found in other populations (16 of our cases were white and two were from the Indian subcontinent). The mutation may also be specific to intestinal type of gastric cancer rather than the diffuse type, which was found in 5/6 of our families in which the histology was available.

**Conclusion**

In conclusion, our findings suggest that further investigations are required to identify susceptibility genes that account for the majority of **E-cadherin** negative gastric cancer families.


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