



CONFERENCE 2007

ABSTRACTS

Leiomyosarcoma and gastrointestinal stromal tumours share common chromosomal imbalances

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Leiomyosarcoma (LMS) and gastrointestinal stromal tumours (GISTs) are malignant tumours arising from the mesenchyme. Until relatively recently GISTs were classified as smooth muscle tumours such as LMS and leiomyomas, however they are now recognised as a separate entity arising from the interstitial cells of the Cajal. LMS falls into a category of STS which lack tumour specific genetic alterations and frequently show complex karyotypic changes. GISTs, however, have gain of function mutations in the proto-oncogene *c-kit* (CD117) which encodes a receptor tyrosine kinase KIT, resulting in constitutive activation of KIT. Mutations within exon 11 of *c-kit* are the most common, and are the target of drug treatment with the tyrosine kinase inhibitor imatinib mesylate. Genetic aberrations including gene amplifications and deletions play an important role in tumour development and progression. In the present study comparative genomic hybridisation (CGH) was used to detect chromosomal gains and losses allowing a genome wide screen for genetic abnormalities in order to determine genetic similarities and differences within a series of LMS and GIST cases. Tissue fluorescence *in situ* hybridisation (FISH) was used to confirm and further refine the regions identified by CGH and protein expression of candidate genes residing within the target areas identified by CGH and FISH was analysed by immunohistochemistry.