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### ABSTRACTS – POSTERS

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#### **Telomere length maintenance in human sarcomas.**

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In order to avoid unsustainable genomic instability, all immortal cells maintain functional telomeres that cap chromosomes. Currently two mechanisms for lengthening telomeres are known: firstly the enzyme telomerase that adds (TTAGGG) repeats onto shortened telomeres and secondly the recombination-based mechanism known as the Alternative Lengthening of Telomeres (ALT). In the majority of human cancers (~90%) telomerase is active, however the proportion of tumours that utilise this enzyme varies considerable between tumour types. For example among liposarcomas 25% express telomerase, 25% ALT but the telomere maintenance mechanism (TMM) in the residual 50% is unknown. However we have shown that some liposarcomas with an unknown TMM do in fact utilise a recombination-based mechanism but lack the marker used to identify ALT+ tumours. As use of the ALT mechanism appears to be limited to tumours, it is a potential target for novel anti-cancer therapies. Yet many basic questions about the ALT mechanism and how, when and why it is activated are unanswered. Currently we are studying the role of WRN and BLM helicases (defective in Werner and Bloom syndromes respectively) in the ALT mechanism. To verify these and other observations regarding telomere length maintenance in sarcomas it is essential that we have patient derived material. Our plans to collect a panel of normal and tumour tissue samples from patients with soft tissue sarcomas will be discussed.