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Lats1 differential expression in selected human cancers

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Lats1 is a tumor suppressor gene that was studied extensively in *Drosophila melangaster*. Several studies were done to address the molecular pathway of Lats 1, yet several roles in carcinogenesis are not elucidated. Lats1 is a serine/threonine kinase similar to human myotonic dystrophy kinase (Justice *et al.*, 1995). Lats 1 is phosphorylated in a cell cycle dependent manner and was shown to modulate CDC2 and cyclin A activity (Tao *et al.*, 1999). Transgenic Lats1-/- mice develop soft-tissue sarcomas and ovarian stromal cell tumours and sensitivity to carcinogenic treatments (St John *et al.*, 1999). Lats1 was shown to play a crucial role in controlling mitosis progression by forming a Lats1/zyxin complex on mitotic apparatus (Hirota *et al.*, 2000). Overexpression of LATS1 significantly suppressed the human tumor cell growth in vitro and tumorigenicity in vivo by either G2-M arrest or apoptosis (Yang *et al.*, 2001). The kinase inactive Lats1 impaired the G1 tetraploidy checkpoint due absence of p53 induction (Iida *et al.*, 2004). Few clinical studies correlated the expression of Lats1, promoter methylation and tumor progression. We previously correlated Lats 1 overexpression with the transcription regulator CDP/Cux in selected tumors (Siam *et al.*, unpublished results). We are currently correlating these findings in clinical samples to investigate the differential expression of Lats1 in many human carcinomas. The hypermethylation of the Lats1 promoter and how this correlates with regulation of Lats1 expression by CDP/Cux p110 and/or p75 isoforms is under investigation. We have preliminary evidence suggesting that Lats1 expression and methylation status of the Lats1 promoter are distinct in selected cancers. Additionally, we are suggesting the use of Lats1 expression and the methylation status as prognostic marker for selected tumors.