

Propositions belonging to the thesis:

**SET and SET-CAN in human leukemia**

1. It would be worthwhile to screen patients with gastrointestinal tumors for the presence of SET-CAN fusion gene because its ectopic expression in transgenic mice results in spontaneous hyperplasia of stomach mucosa.  
*(This Thesis)*
2. The demonstration of the RNAi-induced reduction of pp32 decreases the amount of SET and results in less acetylated histone H4 conflicts with their inhibitory role on histone acetylation.  
*Brody et al. (2004) American Journal of Pathology 164:273-283*  
*Seo et al. (2001) Cell 104:119-130*
3. It has not been proven that tight regulation of the 2 isoforms of SET is important for the correct chromatin remodeling activity by the SET heterodimer.  
*Yamaguchi et al. (1999) J. Mol. Biol. 290:547-557*
4. Given that little has been done to precisely determine the differential expression pattern of the alpha and beta forms of SET ignores the possibility that this might provide further insight into the involvement of this gene in hematopoietic cell proliferation and differentiation.
5. Studies aimed at the identification of the expression signature of metastasis, as well as that of cancer stem cells will only provide key information for the development of novel therapeutic agents to cure cancer if expression profiling is done with highly purified cell populations and the correct controls.  
*Minn et al. (2005) Nature 436:518-524*  
*Weigelt et al (2005) Nat Rev Cancer 5(8):591-602*
6. Despite the opposition of the Bush administration and the long way to go to clinical application, recent generation of patient-specific human embryonic stem cells, derived from nuclear transfer of somatic cells from patients, will accelerate therapeutic-cloning studies.  
*Hwank et al. (2005) Science 308:1777-1783*

7. The poor coincidence in expression profiles between different papers using Affimetrix arrays to identify genes crucial for the development of alveolar rhabdomyosarcoma (ARMS) is more likely to reflect differences in experimental approach than the inability of expression arrays to identify such genes.  
*Khan et al. (2003) Nature Med. 7, 674-679*  
*Kappler et al. (2004) Oncogene 23, 8785-8795*  
*Wachtel et al. (2004) Cancer Research 64, 5539-5545*
8. The claim that a shortened form of the AML1-ETO fusion protein would be important for leukemogenesis in t(8;21) AML, is more based on poor experimentation than careful analysis of AML1-ETO isoforms in t(8;21) patient material.  
*Yan et al. (2004) PNAS 101:17186-17191*
9. The suggestion that SOCS4 and SOCS5 mediate turnover of the EGF receptor via ubiquitin-mediated degradation does not take into account the established role of the Cbl E3-ligase in this process.  
*Kario et al. (2005) JBC 280, 7038-7048*  
*Nicholson et al. (2005) PNAS 102, 2328-2333*
10. It is never too late to screw up an experiment (Ned Mantei).
11. No matter how old a car is in Memphis, its indicators remain brand new.