Prepositions to the thesis

Molecular analysis of chromosomal translocations (12;22) and (12;21) in human leukemia

1. The involvement of TEL in leukemogenesis is unique in the sense that distinct domains of the protein contribute to functionally different fusion proteins.
   Golub et al. (1996). *Mol. Cell. Biol.* **16**, 4107-4116.
   Carroll et al. (1996). *Proc. Natl. Acad. Sci. USA* **93**, 14845-14850.
   This thesis

2. TEL-AML1 negatively interferes with AML1/CBFβ dependent transcription *in vitro* as a dominant negative inhibitor rather than as dominant repressor.
   Hiebert et al. (1996). *Mol. Cell. Biol.* **16**, 1349-1355.
   This thesis

3. The observation that TEL-AML1 chimeric protein, associated with childhood pre B-cell leukemia, interacts with endogenous TEL, argues for the hypothesis that TEL has tumor suppressing activity.
   Raynaud et al. (1996). *Blood* **87**, 2891-2899.
   Wlodarska et al. (1996). *Cancer Research* **56**, 2655-2661.
   This thesis

4. The involvement of the essential nucleopore complex protein CAN/Nup214 in two distinct chromosomal translocations in myeloid leukemia could be wrongly interpreted, as it seems to suggest that aberrant nucleocytoplasmic transport is the primary affected mechanism in the leukemogenic process.
   Van Deursen et al. (1996). *EMBO. J.* **15**, 5574-5583.
   Fu et al. (1997). *Proc. Natl. Acad. Sci. USA* **94**, 1811-1815.

5. It is premature to draw the conclusion that EWS-FLI1 contains separate domains involved in transformation and transactivation, without careful analysis of the subcellular localization of the used deletion mutants.
   Lessnick et al. (1995). *Oncogene* **10**, 423-431

6. It is naive to suggest that cDNA microassay analysis will solve the problems of identifying differentially expressed genes; as with all new technology, the extraordinary amount of new data generated may raise more questions than solve problems.
   DeRisi et al. (1996). *Nature Genet.* **14**,457-460.

7. The problem for most overweight people resides between the ears, despite the absence of mutations in the hypothalamus-specifically expressed leptin receptor gene (*DB*).
   Chua et al. (1996). *Science* **271**, 994-996.
   Erickson et al. (1996). *Science* **274**, 1704-1707.
   Huszar et al. (1997). *Cell* **88**, 131-141.

8. There are no data in support of the hypothesis that alternatively spliced AML1-ETO and CBFβ-SMMYH fusion products cause a disturbance in the relative amounts of AML1/CBFβ isoforms.
   Van der Reijden et al. (1995). *Leukemia* **10**, 204-206.

9. The observation, that homopolymeric glutamine stretches when linked to a heterologous DNA binding domain modulate transcriptional activation, has no physiological relevance.
   Gerber etal. (1994). *Science* **263**, 808-811.

10. Fastfood chains in hospitals and day-time television in doctor's buildings do not serve public health education.

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Memphis, 6 May 1997.