Journal of Zhejiang University SCIENCE B
ISSN 1673-1581
http://www.zju.edu.cn/jzus
E-mail: jzus@zju.edu.cn

Report:

Human cancer genetics*

LI Marilyn[1], ALBERTSON Donna[2]
([1]Medical School, Tulane University, New Orleans, LA 70112-2699, USA)
([2]Cancer Research Institute, University of California, San Francisco, California 94143, USA)
E-mail: mli2@tulane.edu; albertson@cc.ucsf.edu

Received Nov. 22, 2005; revision accepted Dec. 26, 2005

Abstract: The short report will be focused on the genetic basis and possible mechanisms of tumorigenesis, common types of cancer, the importance of genetic diagnosis of cancer, and the methodology of cancer genetic diagnosis. They will also review presymptomatic testing of hereditary cancers, and the application of expression profiling to identify patients likely to benefit from particular therapeutic approaches.

Key words: Cancer genetics, Oncogenes, Tumor suppress genes, Microarray CGH (comparative genomic hybridization), Presymptomatic testing
doi:10.1631/jzus.2006.B0164 **Document code:** A **CLC number:** Q354

Cancer is a genetic disorder. Although hereditary cancers account for only a small fraction of all tumors, most cancers are caused by a variable mix of heredity and environment that leads to accumulations of genetic alterations and then uncontrolled cell proliferations (Kops *et al*., 2005). In addition, genetic alteration is one of the a few most important biological factors that determine the diagnosis and prognosis of different cancers and dictate the treatment strategies for cancer patients. Furthermore, cancer specific therapies based on specific genetic alterations have opened a brand new era of cancer treatment (Jiang *et al*., 2003). This lecture will focus on the genetic basis and possible mechanisms of tumorigenesis, common types of cancer, the importance of genetic diagnosis of cancer, and the methodology of cancer genetic diagnosis (Pinkel and Albertson, 2005).

The workshop will also review presymptomatic testing of hereditary cancers, focusing on hereditary breast cancer genes, *BRCA1* and *BRCA2*. We will review the nature of the involved genes, and the tumor phenotypes. Genomic analyses of sporadic breast tumors and hereditary tumors will be presented. These studies highlight the variety of genomic alterations present in sporadic tumors and show the differences and similarities between sporadic and hereditary tumors. Clinical aspects of *BRCA*-mutation testing, patient management and disease prevention will be discussed.

Recent developments in tumor classification using genome-wide expression profiling on microarrays will be reviewed. Initial studies applying expression profiling to identify patients likely to benefit from particular therapeutic approaches will be discussed.

References

Jiang, G.C., Young, F., Li, M., Weissbecker, K., Price, S., Kim, K.C., Rice, J., La Russa, V.F., Safah, H., Ehrlich, M., 2003. Imtinib (STI571) provides only limited selectivity for CML cells and treatment might be complicated by silent SCR/ABL genes. *Cancer Biology & Therapy*, **2**(1):166/1-166/7.

Kops, G.J., Weaver, B.A., Cleveland, D.W., 2005. On the road to cancer: aneuploidy and the mitotic checkpoint. *Nat. Rev. Cancer*, **5**(10):773-785. [doi:10.1038/nrc1714]

Pinkel, D., Albertson, D.G., 2005. Array comparative genomic hybridization and its applications in cancer. *Nat. Genet.*, **37**(Suppl 6):S11-S17. [doi:10.1038/ng1569]

---

* Special topic report in the First Hangzhou International Symposium on the Medical and Laboratory Applications of Medical Genetics and Genomics held in Hangzhou, China, 2005