**Genomics views: Xenobiotic metabolizing enzymes and cancer risks**

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**Abstract**

Epidemiological studies have estimated that approximately 80% of all cancers are related to environmental factors. Individual cancer susceptibility can be the result of several host factors, including differences in metabolism, DNA repair, altered expression of tumor suppressor genes and proto-oncogenes, and nutritional status. In fact, xenobiotic metabolism is the principal mechanism for maintaining homeostasis during the body's exposure to xenobiotics. Most xenobiotics that enter in the body are subjected to metabolism that functions primarily to facilitate their elimination. Metabolism of certain xenobiotics can also result in the production of electrophilic derivatives that can cause cell toxicity and transformation. The balance of xenobiotic absorption and elimination rates in metabolism can be important in the prevention of DNA damage by chemical carcinogens. Thus the ability to metabolize and eliminate xenobiotics can be considered one of the body's first protective mechanisms. However, there are marked species differences in the way mammals respond to xenobiotics, which are due in large part to molecular differences in xenobiotic metabolizing enzymes and has been related to the enzymatic polymorphisms involved in activation and detoxification of chemical carcinogens and that can impact drug therapy and cancer susceptibility. This paper focus the member of the cytochrome P450 family of enzymes (Cyp2D6), glutathione *S*-transferases (GSTT1 and GSTM1 ) and *N*-acetyltransferases (NAT1 and NAT2) on genetic polymorphisms involved in the metabolism of endocrine disruptors potentially related to cancer development