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Research Article

# The Glutathione S-Transferase Supergene Family: Regulation of GST and the Contribution of the Isoenzymes to Cancer Chemoprotection and Drug Resistance Part I

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## Abstract

The glutathione S-transferase (GST) supergene family consists of several isoenzymes with different properties. The GST supergene family members are involved in the metabolism of carcinogens and other xenobiotics. Evidence indicates that the supergene family members contribute to the detoxification of carcinogens and other xenobiotics. The supergene family members are involved in the metabolism of carcinogens and other xenobiotics. The supergene family members are involved in the metabolism of carcinogens and other xenobiotics.

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A description of the mechanisms of transcriptional and posttranscriptional regulation of GST isoenzymes is provided to allow identification of factors that may modulate resistance to specific noxious chemicals. The most abundant mammalian GST are the class alpha, mu, and pi enzymes and their regulation has been studied in detail. The biological control of these families is complex as they exhibit sex-, age-, tissue-, species-, and tumor-specific patterns of expression. In addition, GST are regulated by a structurally diverse range of xenobiotics and, to date, at least 100 chemicals have been identified that induce GST; a significant number of these chemical inducers occur naturally and, as they are found as nonnutrient components in vegetables and citrus fruits, it is apparent that humans are likely to be exposed regularly to such compounds. Many inducers, but not all, effect transcriptional activation of GST genes through either the antioxidant-responsive element (ARE), the xenobiotic-responsive element (XRE), the GST P enhancer I(GPE), or the glucocorticoid-responsive element (GRE). Barbiturates may transcriptionally activate GST through a Barbie box element. The involvement of the Ah-receptor, Maf, Nrl, Jun, Fos, and NF- $\kappa$ B in GST induction is discussed. Many of the compounds that induce GST are themselves substrates for these enzymes, or are metabolized (by cytochrome P-450 monooxygenases) to compounds that can serve as GST substrates, suggesting that GST induction represents part of an adaptive response mechanism to chemical stress caused by electrophiles. It also appears probable that GST are regulated in vivo by reactive oxygen species (ROS), because not only are some of the most potent inducers capable of generating free radicals by redox-cycling, but H<sub>2</sub>O<sub>2</sub> has been shown to induce GST in plant and mammalian cells: induction of GST by ROS would appear to represent an adaptive response

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mu, and theta GST. The molecular basis for the variation in class alpha GST is not



known. Absence of certain class mu and theta GST can be attributed to deletion of the GSTM1 gene in 50% of the population and deletion of the GSTT1 gene in 16% of the population. The biological consequences of failure to express hGSTM1 or hGSTT1 protein can include susceptibility to bladder, colon, skin, and possibly lung cancer.

Key Words: glutathione S-transferases chemoprotection enzyme induction adaptive response antioxidants drug resistance population polymorphisms carcinogenesis.

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