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Research Article

The Glut athione 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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llutants, and

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-κB 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 vivoby reactive oxygen species (ROS), because not only are some of the most potent inducers capable of generating free radicals by redox-cycling, but H₂O₂has been shown to induce GST in plant and mammalian cells: induction of GST by ROS would appear to represent an adaptive and X

response epoxide. Class alp reneoplastic nodules te to the multidru iman tumors and k lines selec ass pi GST, although served. The ctivation, mechan stabiliza In huma ss alpha,

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 GSTMIgene in 50% of the population and deletion of the GSTTIgene in 16% of the population. The biological consequences of failure to express hGSTMI or hGSTTI protein can include susceptibility to bladder, colon, skin, and possibly lung cancer.

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

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Source: Elsevier

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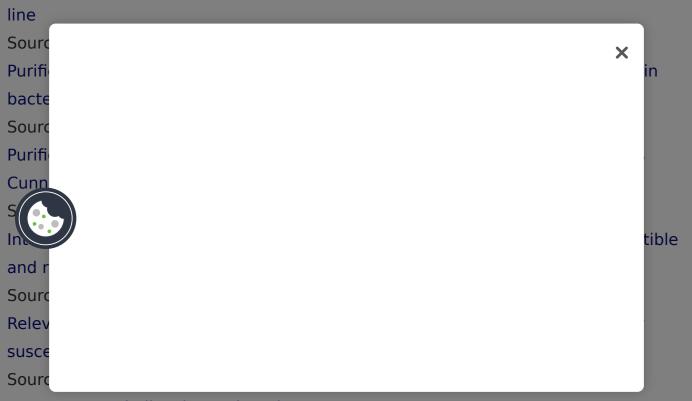
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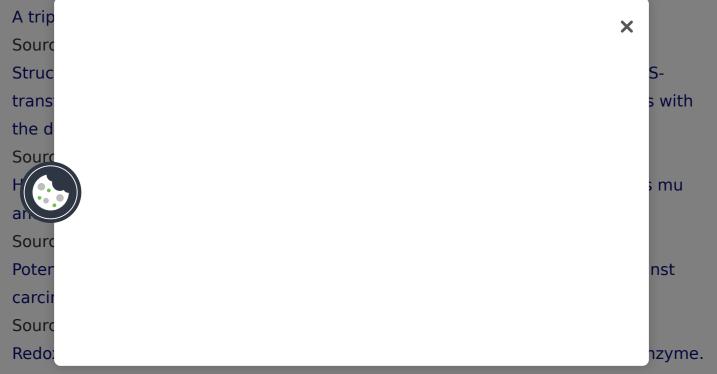
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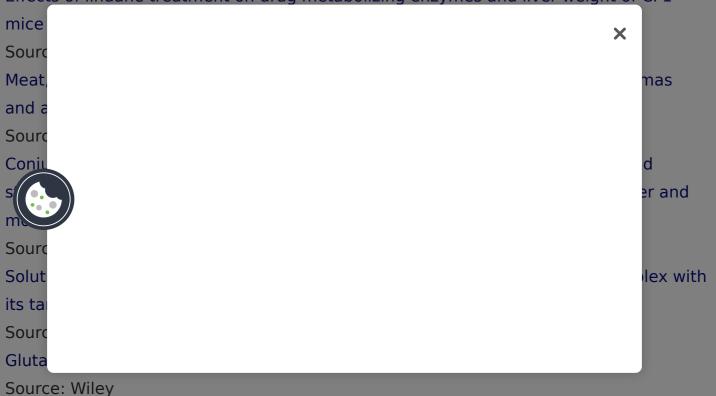
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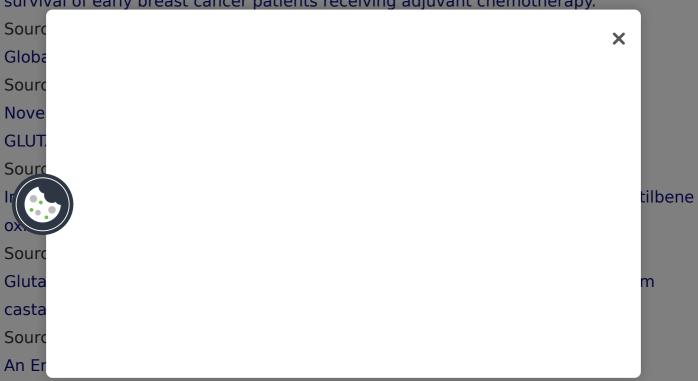
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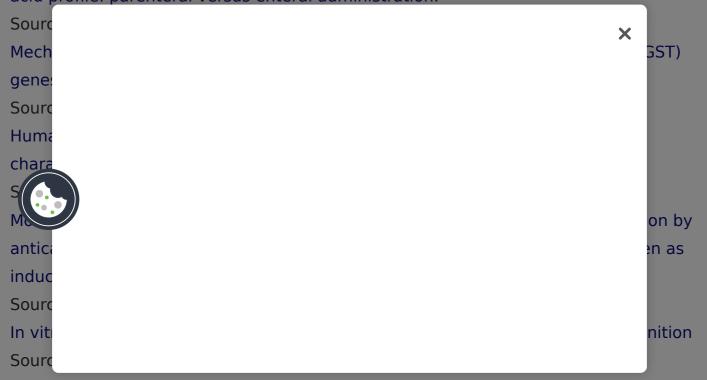
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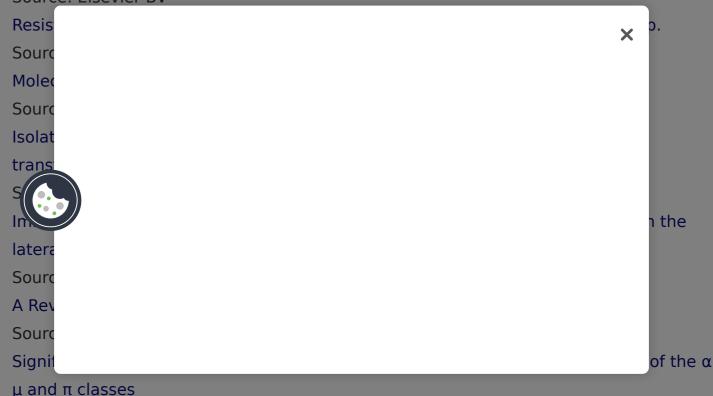
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Source: Elsevier BV

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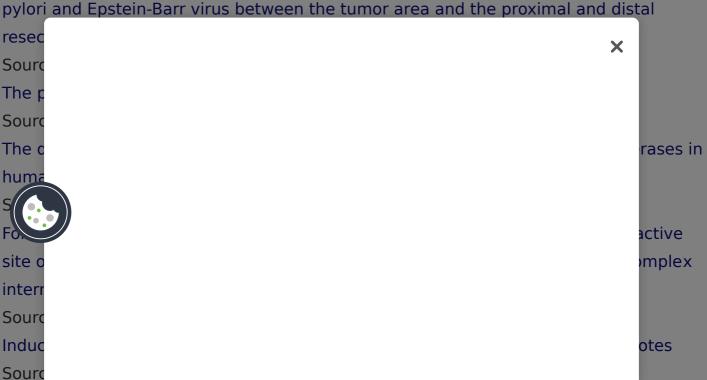
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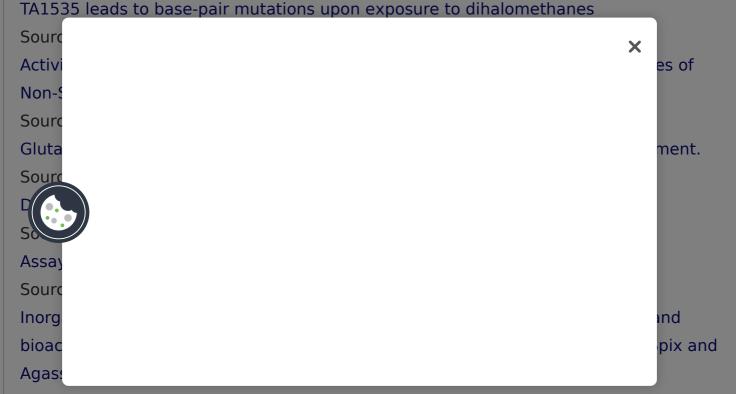
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