Glutathione S-Transferase Polymorphisms and Differential Detoxification of Environmental Pollutants in Humans (Literature Review)

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ABSTRACT

Background:
Humans in industrialized environments are continuously exposed to mixtures of pesticides, industrial solvents, combustion-related air pollutants and heavy metals. Glutathione S-transferases (GSTs) are phase II enzymes that conjugate glutathione to electrophilic toxicants, limiting oxidative damage and facilitating excretion (Ghelli et al., 2021; Singh et al., 2012). Common deletion polymorphisms in *GSTM1* and *GSTT1* ("null genotypes") and the functional *GSTP1* variant rs1695 (Ile105Val) are highly prevalent and can markedly reduce GST activity (de Oliveira et al., 2014; Sirivarasai et al., 2013). These variants are widely used in personalized genetic reports to estimate individual capacity for detoxification of herbicides, pesticides and metals.

Methods:
A narrative literature review was conducted, focusing on human studies that examined polymorphisms in *GSTM1*, *GSTT1* and *GSTP1* in relation to exposure to pesticides, industrial chemicals, combustion-related air pollutants and heavy metals. Original epidemiological and clinical studies as well as meta-analytic work were included if they reported associations between genotype and exposure biomarkers, intermediate endpoints such as oxidative stress, genotoxicity or inflammatory markers, or clinically relevant non-malignant outcomes including respiratory and neurological disorders (Singh et al., 2012; Barrón Cuenca et al., 2019).

Results:
Across agricultural, industrial and community cohorts, *GSTM1* and *GSTT1* null genotypes have been consistently associated with higher pesticide-induced DNA damage, stronger biomarker responses after formaldehyde and particulate exposure, and greater genotoxicity in pesticide-exposed populations (Singh et al., 2012; Ghelli et al., 2021; Barrón Cuenca et al., 2019). Individuals with these deletions or reduced-function *GSTP1* variants show altered distribution of mercury between blood compartments and more pronounced inflammatory responses to a given blood lead level (de Oliveira et al., 2014; Sirivarasai et al., 2013). In occupational and clinical settings, *GSTM1*/*GSTT1* double-null

genotypes have also been linked to increased susceptibility to toluene di-isocyanate–induced asthma and greater vulnerability in progressive myoclonus epilepsy (Lee et al., 2022; Ercegovac et al., 2015).

Discussion:
Human data support the concept that common *GST* polymorphisms generate substantial inter-individual differences in detoxification of pesticides, industrial chemicals and heavy metals. Individuals with fully functional *GSTM1*, *GSTT1* and *GSTP1* alleles appear better able to neutralize these agents, whereas those with null or low-activity variants accumulate higher internal doses and exhibit amplified oxidative and inflammatory responses for the same exposure (Singh et al., 2012; Ghelli et al., 2021). Although *GST* genotypes do not deterministically predict disease risk, they provide a mechanistic basis for personalized advice, such as reducing solvent and pesticide exposure, preferring lower-residue foods when GST genes are impaired, and supporting antioxidant defenses, especially in people with combinations of null alleles (de Oliveira et al., 2014; Sirivarasai et al., 2013).

INTRODUCTION

Modern life entails unavoidable contact with environmental chemicals, including agricultural pesticides, industrial solvents, combustion-derived particles and toxic metals in air, water and food (Gómez-Martín et al., 2015; Barrón Cuenca et al., 2019). Once absorbed, many of these compounds or their reactive metabolites can damage DNA, lipids and proteins, contributing to oxidative stress–driven pathophysiology in multiple organs (Singh et al., 2012).

Glutathione S-transferases (GSTs) form a key component of the enzymatic defense system that mitigates such damage. These phase II enzymes catalyze the conjugation of reduced glutathione to electrophilic xenobiotics, increasing their water solubility and promoting biliary or urinary excretion (Ghelli et al., 2021; Singh et al., 2012). Among cytosolic GSTs, the mu (*GSTM1*), theta (*GSTT1*) and pi (*GSTP1*) isoforms are particularly relevant in the metabolism of pesticides, volatile organic compounds and reactive metabolites of heavy metals (de Oliveira et al., 2014; Sirivarasai et al., 2013).

Genetic variation in *GSTM1*, *GSTT1* and *GSTP1* is common and includes whole-gene deletions (null genotypes) and functional missense variants. Individuals lacking *GSTM1* or *GSTT1* activity, or carrying lower-activity *GSTP1* variants, may therefore be less efficient at detoxifying environmental chemicals, potentially resulting in higher internal exposure and stronger downstream biological effects (Singh et al., 2012; de Oliveira et al., 2014).

Role of GST Genes in Detoxification and Impact of Polymorphism

Role of *GSTM1*, *GSTT1* and *GSTP1* in detoxification and impact of polymorphisms *GSTM1* encodes a mu-class GST that preferentially conjugates bulky aromatic compounds and some organophosphate metabolites, whereas *GSTT1* encodes a theta-class enzyme active toward smaller halogenated and epoxide compounds (Singh et al., 2012; Ghelli et al., 2021). *GSTP1* encodes a pi-class GST highly expressed in the lung and other epithelial tissues, where it detoxifies reactive intermediates from combustion products and endogenous oxidative stress; together, these enzymes convert a broad spectrum of electrophilic toxins into less reactive glutathione conjugates that are subsequently excreted (Ercegovac et al., 2015; de Oliveira et al., 2014). Unlike many polymorphisms involving single-nucleotide changes, the most clinically relevant variants in *GSTM1* and *GSTT1* are homozygous gene deletions that produce "null" genotypes with complete absence of enzyme activity, while the *GSTP1* rs1695 Ile105Val substitution typically reduces catalytic efficiency and thermal stability for many substrates (Ghelli et al., 2021; Singh et al., 2012; de Oliveira et al., 2014). Because these three enzymes have overlapping but distinct substrate specificities, combinations of null and reduced-activity alleles can substantially narrow the range of xenobiotics that are efficiently detoxified; individuals with two functional copies of *GSTM1* and *GSTT1* and a wild-type *GSTP1* allele generally have the highest GST-mediated detoxification capacity, whereas carriers of one or more null or low-activity alleles have compromised capacity (Gómez-Martín et al., 2015; Ercegovac et al., 2015).

Table 1. Key *GST* Variants and Their Impact on Detoxification of Chemicals

Study (Author, Year)	Study Design	Population (Size, Characteristics)	SNP(s)/Focus Investigated	Primary Outcome / Key Findings
Santillán-Sidón et al., 2020	Cross-sectional (biomonitoring)	Agricultural workers with blood organochlorine pesticide (OCP) measures (Mexico)	*GSTM1* null; *GSTT1* null; GST enzyme activity	Higher OCP burden and lower GST activity at higher OCP concentrations; *GSTM1/GSTT1* null genotypes associated with higher OCP levels (Santillán-Sidón et al., 2020).
Sirivarasai et al., 2013	Population study (clinical trial label; biomonitoring analysis)	n=924 adults; environmental lead exposure (Thailand)	*GSTM1* null; *GSTT1* null; *GSTP1* Val105Ile	Higher blood lead associated with ↑ hs-CRP and ↑ systolic BP; *GST* variants/nulls linked to stronger lead-associated inflammatory response, with largest effects in combined null patterns (Sirivarasai et al., 2013).
Saad-Hussein et al., 2017	Case–control	51 pesticide-exposed workers vs 50 controls (Egypt)	*GSTP1* Ile105Val; XRCC1 (399/194)	Pesticide exposure associated with higher comet-assay DNA damage; GSTP1 Ile/Ile (and XRCC1 Arg/Arg) showed greater DNA damage (Saad-Hussein et al., 2017).
Wong et al., 2008	Cross-sectional (occupational exposure)	Pesticide-exposed fruit growers vs controls (Taiwan)	*GSTP1* Ile105Val ± DNA-repair variants	Susceptible *GSTP1* (± DNA-repair) genotypes associated with higher pesticide-related DNA damage (Wong et al., 2008).
Yohannes et al., 2022	Cross-sectional (pediatric biomonitoring)	n=140 lead-exposed children near mine area (Zambia)	*GSTM1; GSTT1* null; *GSTP1* Ile105Val	*GSTT1* null associated with higher blood Pb/Cd; *GSTP1* Ile/Val linked to higher Pb-toxicity susceptibility; combined *GSTP1* Ile/Val + GSTT1 null showed stronger risk (Yohannes et al., 2022).
Ghelli et al., 2021	Occupational exposure study	Hospital workers with formaldehyde exposure	*GST* polymorphisms (focus on oxidative stress susceptibility)	Formaldehyde exposure associated with oxidative stress biomarker differences; GST genetic variation examined as susceptibility modifiers (Ghelli et al., 2021).
Lee et al., 2022	Occupational exposure study	Workers exposed to toluene diisocyanate (TDI)	*GSTM1* null genotype	*GSTM1* null evaluated as a modifier of exposure–lung function relationships in TDI-exposed workers (Lee et al., 2022).
Barrón et al., 2019	Observational (biomonitoring)	Humans (farming/chemical exposure context)	*GSTM1* null, *GSTT1* null	Assessed whether *GSTM1/GSTT1* null genotypes relate to pesticide metabolite levels and/or susceptibility markers in an exposed setting. (PubMed)
Sharma et al., 2019	Case–control / observational	Humans (disease risk)	Xenobiotic genes (incl. *GSTM1/GSTT1/GSTP1*)	Assessed organochlorine pesticide exposure in relation to disease risk with detox-gene interaction analyses. (PubMed)

Aliomrani et al., 2017	Observational	Humans	*GST* polymorphisms	Examined *GST* polymorphisms in a chemical exposure context (biomarkers or disease risk). (PubMed)
de Oliveira et al., 2014	Observational	Humans	*GST* polymorphisms	Evaluated *GST* variation in relation to chemical exposure/detox outcomes. (PubMed)
Singh et al., 2012	Observational	Humans	*GST* polymorphisms	Investigated *GST* variation and a chemical exposure / detoxification phenotype or risk endpoint. (PubMed)
Pandiyan et al., 2023	Observational	Humans	Detoxification genes (incl. *GSTs*)	Investigated *GST* variation and chemical exposure biomarkers/outcomes. (PubMed)
Glass et al., 2018	Observational (gene–exposure)	Humans (agricultural setting)	Detox/oxidative-stress variants (incl. *GSTs*)	Evaluated whether genetic variants modify organophosphate-related neurotoxicity outcomes in farmers. (PubMed)
Valeeva et al., 2020	Observational	Humans	Detoxification genes (incl. *GSTs*)	Examined *GST*-related variants in relation to chemical exposure biomarkers/outcomes. (PubMed)
Bushra et al., 2020	Observational / pharmacogenetic	Humans (treatment context)	*GSTP1* (and related detox genes)	Assessed *GSTP1*-related variation in relation to drug/xenobiotic handling and clinical outcomes (relevant as a chemical detoxification phenotype). (PubMed)
Silva et al., 2024	Observational	Humans	Detoxification genes (incl. *GSTs*)	Addressed *GST* (and related) variants in relation to chemical exposure susceptibility and/or biomarker differences. (PubMed)
Sun et al., 2021	Observational (air pollution interaction)	Humans (ambient pollutant context)	*GSTT1* null (± other GSTs)	Evaluated effect modification by *GSTT1* deletion for pollution-related outcomes (gene–environment interaction).
Tahir et al., 2021	Observational	Humans	Detoxification genes (incl. *GSTs*)	Evaluated *GST*-related genetic susceptibility in a chemical exposure setting. (PubMed)
Ghelli et al., 2021	Observational	Humans (environmental exposure)	*GST* polymorphisms	Evaluated *GST* variants in relation to chemical/metal exposure biomarkers and/or susceptibility. (PubMed)
Ahluwalia et al., 2018	Observational	Humans	*GST* polymorphisms	Assessed *GST* variation in a chemical exposure context (biomarkers and/or risk endpoints). (PubMed)
Ercegovac et al., 2015	Observational	Humans	*GST* polymorphisms	Tested associations of *GST* variation with chemical-related biomarkers/outcomes.

Goodrich el al., 2011	Observational	Humans	Detoxification genes (incl. GSTs)	Assessed GST-related polymorphisms and chemical exposure endpoints. (PubMed)
White et al., 2007	Observational	Humans	Detoxification genes (incl. *GSTs*)	Evaluated GST-related variants with chemical exposure biomarkers/risk endpoints. (PubMed)
Gómez-Martín et al., 2015	Observational	Humans (children; farming community context)	*GSTM1* null, *GSTT1* null (± other detox genes)	Reported distribution of GST null genotypes and evaluated their relevance for susceptibility in a high-exposure setting.
Padiyan et al., 2023	Hospital-based cross-sectional	360 participants	*GSTT1*, *GSTM1* and *CYP2E1* genes	The study suggests that genetic variations in detoxification genes, together with pesticide exposure–related oxidative stress in farm workers, may contribute to the development of cancer.

Environmental Exposure Contexts and Human Evidence for *GST*-Dependent Susceptibility

Humans encounter environmentally harmful substances through multiple routes, including inhalation of indoor/outdoor air contaminants, dermal contact with chemicals during product use, and ingestion of residues or contaminants in foods (Ghelli et al., 2021; Sun et al., 2021; de Oliveira et al., 2014). Exposure patterns differ by geography and lifestyle, but common categories include industrial chemicals/solvents, herbicides/pesticides, and heavy metals, each of which can generate electrophilic species requiring glutathione-dependent conjugation (Lee et al., 2022; Gómez-Martín et al., 2015; Sirivarasai et al., 2013).

Herbicides and Pesticide

In industrialized societies, herbicide and pesticide exposure occurs predominantly via ingestion of residues on plant-based foods and inhalation of aerosolized sprays, with dermal exposure contributing during handling. Human biomonitoring studies demonstrate that *GST* genotype modifies biological responses to pesticide exposure. In organophosphate-exposed cohorts, *GSTM1* null status is associated with greater DNA strand breaks in peripheral blood lymphocytes compared with *GSTM1*-positive individuals at comparable exposure levels (Singh et al., 2012). In populations exposed to mixed pesticide formulations (herbicides, fungicides and insecticides), higher genotoxicity endpoints (e.g., micronuclei) have been reported, and effects are accentuated in carriers of *GSTM1/GSTT1* deletions (Barrón Cuenca et al., 2019). High frequencies of *GSTM1/GSTT1* null genotypes in pediatric cohorts living in intensive farming areas further indicate that a substantial fraction of children may have reduced enzymatic reserve for processing pesticide residues (Gómez-Martín et al., 2015).

Industrial Chemicals and Solvents

Reactive industrial chemicals and solvent-like compounds represent an important exposure domain, with relevance to occupational settings and, to a degree, indoor environments. Human studies indicate that *GSTM1* and *GSTT1* null genotypes can modify susceptibility to reactive chemicals that challenge glutathione-dependent pathways. For example, *GSTM1/GSTT1* null genotypes occur more frequently in individuals with toluene diisocyanate–induced asthma than in exposed individuals without asthma, supporting a gene–environment interaction for a potent respiratory sensitizer (Lee et al., 2022). Formaldehyde exposure has similarly been associated with biomarker changes consistent with stress responses, with stronger effects among carriers of *GSTM1* or *GSTT1* null genotypes (Ghelli et al., 2021).

Combustion-Related Air Pollutants

Combustion processes generate complex mixtures of particulate matter and reactive constituents that can overwhelm detoxification defenses. In a panel study of older adults, short-term PM2.5 increases were associated with lung function impairment and inflammatory biomarker changes, with substantially stronger effects among participants with *GSTT1* null genotype (Sun et al., 2021). These findings are consistent with the interpretation that *GSTT1* contributes to buffering effects of inhaled pollutant mixtures in real-world exposure scenarios.

Heavy Metals from Diet and Indoor Legacy Sources

Heavy metal exposure remains environmentally relevant through dietary intake and legacy indoor contamination. Dietary methylmercury exposure is commonly associated with fish consumption, and genotype can influence internal distribution of mercury species. In a fish-eating population, polymorphisms in glutathione-related genes — including *GSTM1, GSTT1* and *GSTP1* — were associated with altered partitioning of mercury between plasma and whole blood and with genotype-dependent distribution of inorganic versus methylmercury fractions (de Oliveira et al., 2014). Lead exposure is still encountered through legacy sources, particularly dust from older housing materials such as now-banned lead-based paint. In adults with varying environmental lead exposure, blood lead was associated with inflammatory markers and blood pressure, with stronger associations among individuals with *GSTM1/GSTT1* null or variant *GSTP1* genotypes (Sirivarasai et al., 2013).

Synthesis

Across exposure domains, a consistent pattern emerges reduced GST function, particularly via *GSTM1/GSTT1* deletions, tends to be associated with stronger biomarker responses at comparable exposure levels. This supports the use of GST genotype as a biologically plausible modifier of susceptibility to environmentally harmful substances (Singh et al., 2012; Ghelli et al., 2021).

GST Polymorphisms, Susceptibility, and Non-Malignant Outcomes

Beyond intermediate biomarkers, *GST* genotypes have been associated with clinically relevant non-malignant outcomes in settings where environmental chemicals and oxidative or inflammatory pathways are central. The *GSTM1/GSTT1* double-null genotype has been linked to increased susceptibility to toluene diisocyanate–induced asthma (Lee et al., 2022). In addition, *GSTM1, GSTP1* and *GSTT1* polymorphisms have been investigated in progressive myoclonus epilepsy, where genotype patterns were associated with disease vulnerability and biomarker profiles (Ercegovac et al., 2015). These observations support the broader interpretation that functional *GST* activity contributes to resilience against environmental and endogenous stressors, while combined deficits can lower physiological reserve.

Health and Lifestyle Implications

The evidence that polymorphisms in *GSTM1*, *GSTT1* and *GSTP1* modify detoxification of environmental toxicants has direct implications for preventive strategies and exposure management. Individuals with functional alleles at all three loci generally exhibit lower biomarker responses (e.g., DNA damage indices or inflammatory mediators) for a given pesticide, solvent or air-pollution exposure than carriers of null or reduced-activity variants, indicating a wider physiological safety margin (Singh et al., 2012; Ghelli et al., 2021; Sun et al., 2021). In contrast, carriers of *GSTM1/GSTT1* null genotypes or lower-activity *GSTP1* variants can be considered to have a reduced "detoxification reserve," reflected in higher genotoxicity and/or more pronounced inflammatory responses at comparable exposure levels (Barrón Cuenca et al., 2019; de Oliveira et al., 2014; Sirivarasai et al., 2013).

From an applied perspective, these genotype-dependent differences support a tiered approach to exposure reduction. For herbicides and pesticides, strategies that reduce dietary residue intake — such as thorough washing of fruits and vegetables, peeling when appropriate, and preferential selection of lower-residue (or organic) options when feasible — are consistent with observed gene–environment interactions in human biomarker studies (Gómez-Martín et al., 2015; Singh et al., 2012). For industrial chemicals and solvents, limiting inhalation and skin contact — particularly in indoor environments where cleaning agents, sprays or solvent-containing products are used — aligns with evidence of genotype-dependent biomarker responses to reactive chemical exposures; when use is unavoidable, reducing contact time and improving ventilation, and using gloves or other protective measures, are rational precautions (Ghelli et al., 2021; Lee et al., 2022). For air pollution, individuals with reduced GST function may benefit from additional avoidance strategies during high-PM events (e.g., limiting outdoor exertion, improving indoor filtration), given stronger biomarker and lung function responses observed in susceptible genotypes (Sun et al., 2021).

For heavy metals, *GST* genotype information can complement standard exposure-control recommendations. Evidence that glutathione-related genotypes influence mercury distribution and lead-associated inflammation suggests that individuals with reduced *GST* function may derive particular benefit from avoiding high-mercury fish where relevant and minimizing indoor exposure to legacy lead sources, including dust associated with older lead-based paints (de Oliveira et al., 2014;

Sirivarasai et al., 2013). Nutritional strategies that support metal handling — such as adequate calcium intake to reduce lead absorption and sufficient zinc to support metallothionein-mediated binding of certain metals — are biologically plausible adjuncts for individuals with reduced *GST* capacity, although intervention data remain limited (Sirivarasai et al., 2013).

Overall, *GST* genotyping should be interpreted as a modifier of vulnerability rather than a deterministic predictor. In individuals with favorable genotypes, standard public-health guidance regarding pesticide, solvent and metal exposure is generally sufficient. In those with one or more null or low-activity alleles, the same guidance may carry greater relevance because baseline detoxification capacity is lower and exposure-reduction strategies may yield proportionally greater benefit (Singh et al., 2012; Ghelli et al., 2021).

CONCLUSION

Common polymorphisms in *GSTM1*, *GSTT1* and *GSTP1* are important determinants of how efficiently individuals detoxify environmentally harmful substances, including pesticides, reactive industrial chemicals/solvents, combustion-related pollutant mixtures and heavy metals. Null alleles in *GSTM1* and *GSTT1* and lower-activity *GSTP1* variants (including rs1695 Ile105Val) reduce the capacity to conjugate electrophilic metabolites and are associated with stronger biomarker responses and altered internal handling of exposures such as pesticide mixtures, PM2.5, mercury from fish consumption and lead from legacy indoor sources (Singh et al., 2012; Sun et al., 2021; de Oliveira et al., 2014). Human studies further indicate that carriers of these variants — particularly individuals with combined *GSTM1/GSTT1* deletions or multi-locus low-activity profiles — show higher biomarkers of DNA damage, oxidative stress and inflammation under comparable exposure conditions, and in some contexts demonstrate increased susceptibility to non-malignant outcomes such as occupational asthma and neurological vulnerability (Singh et al., 2012; Lee et al., 2022; Ercegovac et al., 2015).

From a personalized prevention perspective, *GST* genotype information provides a mechanistically grounded basis for tailoring recommendations on exposure reduction and nutritional support. Individuals with fully functional *GST* genes may tolerate typical background exposures within regulatory limits, although general exposure-minimization principles remain advisable. In contrast, individuals with impaired *GST* function represent a sizeable subgroup for whom avoiding unnecessary pesticide and solvent use, improving indoor and outdoor air quality, selecting foods with lower chemical burdens (including attention to high-mercury fish where relevant), and reducing contact with legacy lead sources may yield disproportionate benefit (de Oliveira et al., 2014; Sirivarasai et al., 2013).

Future work should refine quantitative risk estimates by integrating *GST* polymorphisms into exposure–response models and should evaluate whether genotype-tailored interventions measurably reduce exposure biomarkers or improve health outcomes in real-world settings.

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