Antioxidant Gene Polymorphisms and Oxidative Stress Resilience (Literature Review)

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ABSTRACT

Background:
Oxidative stress arises when reactive oxygen species (ROS) exceed endogenous antioxidant capacity, promoting cumulative molecular injury and contributing to aging and chronic disease (Zhou, 2023). Genetic variability in detoxification and antioxidant enzymes — including *GSTM1*, *GSTT1*, *GSTP1*, *SOD2*, and *GPX1* — has been shown to influence mitochondrial ROS handling, cytosolic electrophile conjugation, and downstream redox signaling. Deletion variants in *GSTM1* and *GSTT1* reduce cytosolic glutathione-conjugating capacity, thereby diminishing detoxification of lipid-derived electrophiles and xenobiotic metabolites (McWilliams, 1995; Boccia, 2006). Missense variants in *GSTP1*, *SOD2*, and *GPX1* alter catalytic kinetics or mitochondrial import efficiency, which may modulate individual susceptibility to oxidative damage across the lifespan (Wenzlaff, 2005; Paludo, 2014; Chen, 2011). Variation across these genes is therefore increasingly regarded as a contributor to inter-individual differences in oxidative resilience and aging-related vulnerability.

Methods:
A synthesis of peer-reviewed studies was conducted. Emphasis was placed on meta-analyses examining deletion polymorphisms in *GSTM1* and *GSTT1*, mechanistic analyses of *SOD2* rs4880 and *GPX1* rs1050450 in redox biology, and epidemiological studies evaluating multigenic and exposure-dependent effects involving *GSTP1* rs1695. Studies were included only when they examined functions or outcomes directly linked to these variants.

Results:
Across population-based and mechanistic studies, antioxidant polymorphisms demonstrated modest yet reproducible associations with oxidative stress regulation and disease susceptibility. Deletion variants affecting *GSTM1* and *GSTT1* were linked to increased cancer risk, particularly in the presence of tobacco-derived electrophiles (McWilliams, 1995; Wenzlaff, 2005; La Torre, 2005). *GSTP1* rs1695 exhibited additive risk effects in multigenic contexts and in environments involving chronic electrophile exposure (Sreeja, 2008). Variants within mitochondrial antioxidant enzymes contributed to altered cellular redox states: *SOD2* rs4880 influenced intracellular ROS responses

under inflammatory and pharmacologic challenge (Paludo, 2014), while *GPX1* rs1050450 modified peroxide detoxification efficiency and displayed consistent associations with malignancy risk (Chen, 2011; Men, 2014; Cao, 2014). Furthermore, combined mitochondrial genotypes demonstrated synergistic effects on mortality and healthy aging indicators in advanced-age cohorts, suggesting cumulative consequences across antioxidant pathways (Cao, 2014).

Discussion:
The collective evidence indicates that these antioxidant polymorphisms function not as deterministic predictors but as modifiers of vulnerability within a broader oxidative-stress framework. Their phenotypic influence becomes more apparent under conditions of elevated oxidative load, including tobacco exposure, pharmacologic challenge, high metabolic demand, or micronutrient limitation (Paludo, 2014; Cominetti, 2011). Findings across mitochondrial and cytosolic pathways highlight the importance of network-level interactions, whereby small reductions in detoxification efficiency at multiple biochemical steps collectively shape long-term redox homeostasis (Zhou, 2023). Such distributed effects provide mechanistic support for utilizing these polymorphisms as indicators of differential susceptibility to oxidative stress and aging-related decline.

Subjects: Genetics, Nutrition **Keywords:** Genetics, Polymorphism, Nutrition, Free Radicals

INTRODUCTION

Oxidative stress is a fundamental biological process characterized by the accumulation of reactive oxygen species beyond the buffering capacity of endogenous antioxidant systems, a state known to promote damage to lipids, proteins, and nucleic acids, accelerate cellular senescence, impair mitochondrial function, and increase vulnerability to chronic diseases associated with aging (Zhou, 2023; Paludo, 2014). The cellular architecture of antioxidant defense is organized across distinct subcellular compartments, each providing unique protective mechanisms to maintain redox homeostasis (Paludo, 2014).

Within the mitochondrial matrix, where electron transport chain activity produces superoxide as an inevitable by-product of oxidative phosphorylation, manganese superoxide dismutase (*SOD2*) catalyzes the conversion of superoxide to hydrogen peroxide (Paludo, 2014). This peroxide is subsequently reduced to water through glutathione-dependent pathways in which glutathione peroxidase 1 (*GPX1*) plays a principal role (Cominetti, 2011; Chen, 2011). In cytosolic and membrane environments, glutathione S-transferases (*GSTs*) detoxify electrophilic by-products of ROS damage and xenobiotic metabolism through glutathione conjugation (McWilliams, 1995; Boccia, 2006). Polymorphisms affecting these antioxidant systems — including *GSTM1*-null, *GSTT1*-null, *GSTP1* rs1695, *SOD2* rs4880, and *GPX1* rs1050450 — exert their consequences by modifying catalytic efficiency, enzyme localization, detoxification capacity, or substrate interaction (Wenzlaff, 2005; Paludo, 2014; Chen, 2011).

Research increasingly indicates that these polymorphisms modulate the ability to manage oxidative stress under environmental, nutritional, and physiological pressures (Ahmetov, 2014). Such variation may help explain inter-individual heterogeneity in biological aging, disease susceptibility, and resilience to oxidative challenges (Zhou, 2023). The following sections integrate evidence across mechanistic, epidemiological, and clinical domains to elucidate how these polymorphisms collectively contribute to oxidative-stress biology.

Oxidative Defense Is Compartmentalized: Biological Architecture and Functional Implications

Antioxidant defense mechanisms operate within a highly compartmentalized intracellular network, with distinct enzymatic systems distributed across mitochondrial, cytosolic, and membrane environments. The mitochondrial matrix contains the primary defense against superoxide formed during oxidative phosphorylation. Here, *SOD2* catalyzes the conversion of superoxide to hydrogen peroxide, a reaction followed by *GPX1*-mediated peroxide reduction. Variants affecting these enzymes — particularly *SOD2* rs4880 and *GPX1* rs1050450 — have been associated with differences in mitochondrial import efficiency, catalytic turnover, and redox signaling (Paludo, 2014; Chen, 2011; Cominetti, 2011).

In parallel, cytosolic and membrane-associated detoxification mechanisms buffer against ROS-derived electrophiles generated during lipid peroxidation and xenobiotic metabolism. *GST* enzymes mediate glutathione conjugation to such electrophiles, reducing their reactivity and facilitating clearance. Deletion variants such as *GSTM1*-null

and *GSTT1*-null, and reduced-function missense variants such as *GSTP1* rs1695, impair conjugation capacity and increase persistence of reactive species (McWilliams, 1995; Wenzlaff, 2005; Boccia, 2006).

This structural configuration identifies mitochondrial and cytosolic antioxidant processes as jointly maintaining redox equilibrium, with genetic variation modulating resilience to oxidative stress over time (Zhou, 2023).

Functional Consequences of Variation Across Antioxidant Pathways

Functional consequences of antioxidant gene polymorphisms arise across interconnected detoxification pathways. Mitochondrial variants alter the efficiency of superoxide dismutation and peroxide reduction, shifting intracellular redox gradients and modifying susceptibility to apoptotic or inflammatory signaling *(Paludo, 2014)*. Reduced mitochondrial capacity can propagate oxidative imbalance beyond the organelle, reinforcing pathways linked to senescence and tissue dysfunction.

Cytosolic variants likewise create vulnerabilities by limiting the neutralization of electrophilic species from oxidative or xenobiotic stress. Loss-of-function mutations in *GSTM1* or *GSTT1* reduce detoxification breadth, while *GSTP1* variants subtly affect catalytic efficiency. These genetic differences have been associated with altered oxidative biomarkers and differential susceptibility to metabolic or environmental stressors *(McWilliams, 1995; Cominetti, 2011; Ahmetov, 2014)*. Overall, variation in antioxidant pathways modulates redox balance and cellular responses to oxidative stress.

Table 1: Prominent Human Studies on Oxidative-Stress/Antioxidant Gene Variants and Oxidative Stress–Related Outcomes

Study (Author, Year)	Study design	Population (size, characteristics)	SNP(s)/Focus investigated	Primary outcome / key findings
Sreeja L et al., 2008	Cross-sectional (biomarker comparison; exposure-stratified)	Laryngeal cancer and non-cancer subjects; environmental tobacco smoke (ETS) exposure	*GSTM1* null vs present; oxidative stress markers (e.g., MDA, GSH)	ETS associated with ↑lipid peroxidation and ↓GSH primarily in *GSTM1*-null, supporting reduced antioxidant buffering capacity.
Doukali et al., 2017	Occupational exposure study	Tunisian medical staff exposed to ionizing radiation vs comparison group	GST polymorphisms (including *GSTM1/GSTT1/GSTP1*); oxidative stress markers	*GST* genotypes modified radiation-associated oxidative stress marker profiles (gene–exposure effect modification).
Sun et al., 2021	Panel study (repeated measures)	32 retired adults (55–77y), repeated follow-ups; ambient PM2.5 tracked	*GSTT1* null vs present; nasal oxidative stress/inflammation (8-epi-PGF2α, cytokines) and lung function	PM2.5 linked to ↑oxidative stress/inflammation and ↓lung function; effects stronger in *GSTT1*-null individuals.

Eslami S et al., 2014	Meta-analysis	Pooled case–control studies on hypertension	*GPX1* rs1050450 (Pro198Leu)	Synthesized evidence linking *GPX1* Pro198Leu to hypertension risk in pooled analyses (subgroup effects reported).
Chen et al., 2011	Meta-analysis	Multiple case–control cancer datasets	*GPX1* Pro198Leu (rs1050450)	Pooled evidence supported an association between *GPX1* Pro198Leu and cancer susceptibility (effect sizes varied by subgroup).
Men et al., 2014	Meta-analysis	Case–control studies in prostate cancer	*GPX1* Pro198Leu (rs1050450)	Meta-analytic signal for *GPX1* Pro198Leu and prostate cancer risk (direction/moderators assessed).
Cao et al., 2014	Meta-analysis	Bladder cancer case–control datasets	*SOD2* Val16Ala (rs4880; "Val/Ala" targeting variant)	Pooled evidence evaluated *SOD2* Val16Ala and bladder cancer susceptibility; subgroup effects explored.
Zhou et al., 2023	Meta-analysis	63 publications (14,733 COPD cases; 50,570 controls)	Oxidative-stress genes (incl. *GSTM1/GSTT1, GSTP1, SOD2, CAT, CYPs*, etc.)	Multiple oxidative-stress variants associated with COPD risk; *GSTM1/GSTT1* null increased risk; *SOD2* Val/Ala showed protective signal in pooled analysis.
McWilliams et al., 1995	Meta-analysis (case–control)	1,593 lung cancer cases; 2,135 controls (12 studies)	*GSTM1* (null vs present)	*GSTM1*-null modestly ↑ lung cancer risk (pooled OR ≈ 1.4); attributable fraction ~17%.
Economopoulos & Sergentanis, 2010	Meta-analysis	CRC: large pooled datasets across 19–44 studies	*GSTM1* (null), *GSTT1* (null), *GSTP1* (Ile105Val), *GSTA1*	CRC risk modestly ↑ with *GSTM1-null* and *GSTT1-null* in Caucasians; *GSTP1* and *GSTA1* largely null. Combined null genotypes increased risk.
Tahir et al., 2021	Cross-sectional (occupational exposure)	120 participants (e-waste workers + controls)	*GSTM1* (null), *GSTT1* (null)	E-waste work associated with ↑ metal burden and oxidative stress; *GSTT1-null* more frequent in highly exposed workers and linked to higher susceptibility markers.
Kellen et al., 2007	Pooled analysis + meta-analysis	Bladder cancer (meta + pooled datasets)	*GSTP1* (Ile105Val)	105Val allele associated with ↑ bladder cancer risk (dose–response; stronger in Asians); no clear smoking interaction.
Nascimento et al., 2021	Experimental (blood storage)	Donor RBC units (genotyped)	*GSTP1* (rs1695; promoter variant), *SOD2* (rs4880)	Variant genotypes showed ↑ lipid peroxidation during storage (greater oxidative "storage lesion").
Sobha et al., 2022	Meta-analysis (case–control)	CAD studies (33 total)	*GSTM1* (null), *GSTT1* (null), *GSTP1* (Ile105Val)	CAD risk associated with *GSTM1-null* (moderate) and mildly with *GSTP1-Val*; *GSTT1-null* alone mostly null. Dual *GSTM1-null + GSTT1-null* showed higher risk.

Ahmetov et al., 2014	Case–control (athlete genetics)	2,664 elite athletes; 917 controls	*SOD2* (rs4880, Ala16Val)	Val/Val underrepresented in power athletes; Val allele associated with higher muscle damage biomarkers.
Ascencio-Montiel et al., 2013	Comparative study + meta-analysis	Mexico; T2D n=994 (macroalbuminuria n=119 vs normoalbuminuria n=875)	*SOD2* (MnSOD) Val16Ala (rs4880)	C allele (CC vs TT) associated with lower odds of macroalbuminuria and higher eGFR; meta-analysis supported a protective association (Ascencio-Montiel, 2013).
Becer & Çırakoğlu, 2015	Case–control	Turkey; obese n=150 vs non-obese n=120	*SOD2* (MnSOD) Val16Ala (rs4880)	Obesity linked to ↑ leptin and oxidative stress markers; rs4880 genotype differed between groups and was associated with leptin/oxidative-stress profiles (Becer, 2015).
Cominetti et al., 2011	Randomized, placebo-controlled trial	Brazil; morbidly obese women n=37, 1 Brazil nut/day vs placebo for 8 weeks	*GPX1* Pro198Leu (rs1050450)	Brazil nuts increased Se status and GPx activity; DNA damage decreased mainly in Pro/Pro, while Leu/Leu showed higher DNA damage (Cominetti, 2011).
McKeever et al., 2021	Post hoc analysis of RCT (FeDOx)	USA; sepsis patients n=34 receiving differing enteral nutrition exposure	*SOD2* (MnSOD) rs4880 + *GPX1* rs1050450 (combined "risk alleles")	With higher kcal exposure, combined risk alleles were associated with ↑ oxidative stress (F2-isoprostanes) and higher ICU mortality (McKeever, 2021).
Soerensen et al., 2009	Prospective cohort (oldest-old)	Denmark; 1905 cohort n=1650, age 92–93 at intake; follow-up 1998–2008	*SOD2* (MnSOD) rs4880 + GPX1 rs1050450	*SOD2–GPX1* genotype combination associated with aging/longevity (mortality risk differences), consistent with oxidative-defense variation influencing survival (Soerensen, 2009).

Evidence Base from Epidemiological, Mechanistic, and Clinical Studies

Epidemiological studies consistently associate reduced *GST*-mediated detoxification with increased susceptibility to cancers driven by electrophilic metabolites, with effects amplified among smokers or occupationally exposed populations (McWilliams, 1995; La Torre, 2005; Wenzlaff, 2005). Such findings align with mechanistic evidence demonstrating accumulation of carcinogenic intermediates under reduced *GST* activity.

Variation within mitochondrial antioxidant pathways displays coherent associations with physiological responses to inflammation, metabolic stress, and pharmacologic insult (Paludo, 2014). Intervention studies further suggest that nutrient availability can modulate genotype-dependent differences in *GPX1* activity and oxidative biomarker expression, emphasizing the interplay between genetics and micronutrient status (Cominetti, 2011).

Evidence from mechanistic and clinical studies indicates that variation across multiple mitochondrial antioxidant pathways can exert cumulative effects on cellular redox responses, with combined *SOD2*- and *GPX1*-related alterations contributing to heightened oxidative stress sensitivity under inflammatory, pharmacologic, or metabolic challenge (Paludo, 2014; Ahmetov, 2014; Chen, 2011).

Gene–Environment Interactions as Determinants of Phenotypic Expression

The phenotypic impact of antioxidant polymorphisms depends strongly on environmental oxidative load. Tobacco smoke exposure amplifies risks associated with *GST* pathway deficiencies, revealing genetic predispositions not apparent at baseline (Wenzlaff, 2005). Drug-induced oxidative challenge yields genotype-specific differences in mitochondrial stress responses, demonstrating pharmacogenomic relevance. High-intensity exercise similarly reveals differences in oxidative muscle injury associated with mitochondrial variants (Ahmetov, 2014).

Micronutrient availability, particularly selenium, modulates *GPX1* activity and thus influences the expression of *GPX1* polymorphism phenotypes (Cominetti, 2011). These observations emphasize that genetic variation shapes susceptibility, not inevitability, and that phenotypic expression emerges most prominently under increased oxidative burden.

Network-Level Interpretation and Combined Effects Across Pathways

Antioxidant systems operate as interconnected biochemical networks requiring coordinated activity across multiple enzymes. Variants affecting different nodes within this system often exhibit synergistic effects. Combined *GSTM1* and *GSTT1* deletions produce stronger susceptibility signatures than either deletion alone, while co-occurrence of *SOD2* and *GPX1* variants demonstrates joint associations with survival phenotypes (Boccia, 2006).

Such findings underscore the limitations of evaluating polymorphisms in isolation and demonstrate that biological aging reflects the cumulative impact of distributed inefficiencies across antioxidant pathways.

Implications for Aging Biology and Disease Susceptibility

Antioxidant polymorphisms influence aging trajectories by shaping the capacity to mitigate oxidative damage throughout life. Variants affecting mitochondrial antioxidant mechanisms influence the onset of mitochondrial dysfunction, sensitivity to apoptosis, and cellular senescence (Paludo, 2014). Cytosolic detoxification variants heighten vulnerability to mutagenic exposures and inflammatory processes, particularly under chronic oxidative load (Wenzlaff, 2005).

Although individual effect sizes are modest, the cumulative influence across decades of exposure contributes meaningfully to risk heterogeneity for age-associated diseases. Their effects remain probabilistic rather than deterministic and interact dynamically with environmental, metabolic, and lifestyle factors (Zhou, 2023).

CONCLUSION

The antioxidant polymorphisms *GSTM1*-null, *GSTT1*-null, *GSTP1* rs1695, *SOD2* rs4880, and *GPX1* rs1050450 collectively contribute to inter-individual variability in oxidative resilience through spatially and functionally distinct mechanisms spanning both mitochondrial and cytosolic defense systems. Their influence arises not from isolated, high-magnitude effects but from distributed modifications in enzymatic efficiency, substrate handling, and compartmentalized detoxification pathways. Such cumulative perturbations reinforce the concept that aging-related vulnerability emerges from network-level variation within the redox-regulatory system (Zhou, 2023; Sreeja, 2008).

The phenotypic impact of these polymorphisms becomes most evident under conditions of elevated oxidative stress — including tobacco exposure, inflammatory activation, strenuous metabolic demand, and micronutrient deficiency — where protective capacity is challenged and genotypic differences are unmasked (Wenzlaff, 2005; Ahmetov, 2014; Cominetti, 2011). Consequently, these variants are best understood as modulators of susceptibility, shaping the threshold at which oxidative injury is incurred rather than functioning as deterministic predictors of disease or longevity. This interpretation is supported by epidemiological and mechanistic observations showing that the strongest associations with adverse outcomes emerge when multiple polymorphisms co-occur or when environmental stressors amplify the functional limitations imposed by each variant (Wenzlaff, 2005; Sreeja, 2008*)*.

A network-based perspective further suggests that subtle inefficiencies distributed across sequential detoxification steps can collectively alter intracellular redox states, influence mitochondrial integrity, and modulate inflammatory and apoptotic signaling over time (Zhou, 2023). These cumulative effects provide a mechanistic basis for inter-individual differences in oxidative resilience and susceptibility to age-associated diseases.

Understanding the distributed influence of antioxidant polymorphisms offers a foundation for developing personalized strategies to optimize oxidative defense. Approaches integrating genomic information with lifestyle, environmental exposure, and nutritional status hold promise for precision geroscience, where interventions can be tailored to enhance redox homeostasis and support healthy biological aging. While genetic variation alone does not dictate aging outcomes, it contributes materially to the heterogeneity observed among individuals, underscoring the necessity of combining genomic, environmental, and metabolic perspectives in future aging research (Zhou, 2023).

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