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Review article

Oxidative Stress-Related Genetic Polymorphisms in Nasopharyngeal Carcinoma: Emphasis on Gpx-1 and MPO Variants in Jordanian Population

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Abstract: Nasopharyngeal carcinoma (NPC) is a distinct head and neck malignancy characterized by marked geographic variation and multifactorial etiology involving Epstein-Barr virus (EBV) infection, environmental exposures, and host genetic susceptibility. Oxidative stress has emerged as a central biological mechanism linking chronic inflammation and genomic instability to NPC pathogenesis. Antioxidant enzymes such as glutathione peroxidase-1 (GPx-1) and pro-oxidant inflammatory enzymes such as myeloperoxidase (MPO) regulate intracellular redox balance. This review summarizes the role of oxidative stress pathways in carcinogenesis and outline current knowledge regarding the role of antioxidant and pro-oxidant enzyme polymorphisms particularly GPx-1 and MPO in NPC development, with special emphasis on finding from a recent Jordanian case-control study that demonstrated significant association between GPx-1 variants (1416T>TC and 1458C>T) and MPO variants (611G>A and 736G>GA) with increased NPC risk. Regional genetic data from Jordan and comparative international studies are discussed, highlighting the potential for oxidative stress related polymorphisms to serve as biomarkers to carcinogenesis. Evidence from international and regional literature is discussed to contextualize these findings and explore implications for genetic screening and targeted preventive strategies.

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Introduction

NPC arises from epithelial cells lining the nasopharynx and represents a biologically and epidemiologically unique subtype of head and neck cancer [1]. Although globally uncommon, NPC displays high incidence in Southeast Asia, North Africa, and parts of the Middle East, including Jordan, making it an important regional health concern [2]. Genetic predisposition plays a pivotal role in NPC susceptibility, particularly genes involved in xenobiotic metabolism, immune regulation, and oxidative stress control [3]. Imbalance between reactive oxygen species (ROS) and antioxidant defense mechanisms is called oxidative stress which lead to genetic instability that increase cancer risk [4]. Over forty single-nucleotide polymorphisms (SNPs) have been linked to NPC risk worldwide [5], but data from Middle Eastern populations remain limited. The Jordanian study summarized in this review represents one of the first focused investigations of oxidative stress-related genes in NPC within this population.

Epidemiology and Clinical Significance of NPC

NPC originates from the epithelial lining of the nasopharynx and constitutes a biologically distinct subgroup of head and neck cancers [6]. Unlike most squamous cell carcinomas of the upper aero-digestive tract, NPC exhibits marked geographic clustering, with the highest incidence in Southeast Asia, southern China, North Africa, and parts of the Middle East, including Jordan [7]. This uneven distribution suggests strong interactions between environmental exposures (such

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as dietary nitrosamines and smoking), viral infection, and host genetic background [3]. NPC is classified as keratinizing carcinoma (squamous cell carcinoma), non-keratinizing carcinoma (differentiated and undifferentiated variants), and basaloid squamous cell carcinoma. Non-keratinizing subtype is the endemic form, primarily found in Asia [8]. Within Jordan, NPC represents a clinically important malignancy characterized predominantly by undifferentiated histological subtypes as reported in the Jordanian cohort in which 91.43% of tumors belonged to this category. Male predominance was also observed, with approximately 78% of cases occurring in men.

Oxidative stress arises from excessive production of ROS relative to cellular antioxidant capacity, resulting in oxidative modification of lipids, proteins, and nucleic acids [9]. These molecular lesions promote mutagenesis, chromosomal instability, altered signaling pathways, and malignant transformation [10]. In NPC, chronic inflammation, viral infection, and immune responses, may further amplify ROS generation in nasopharyngeal tissues, accelerating oncogenic processes.

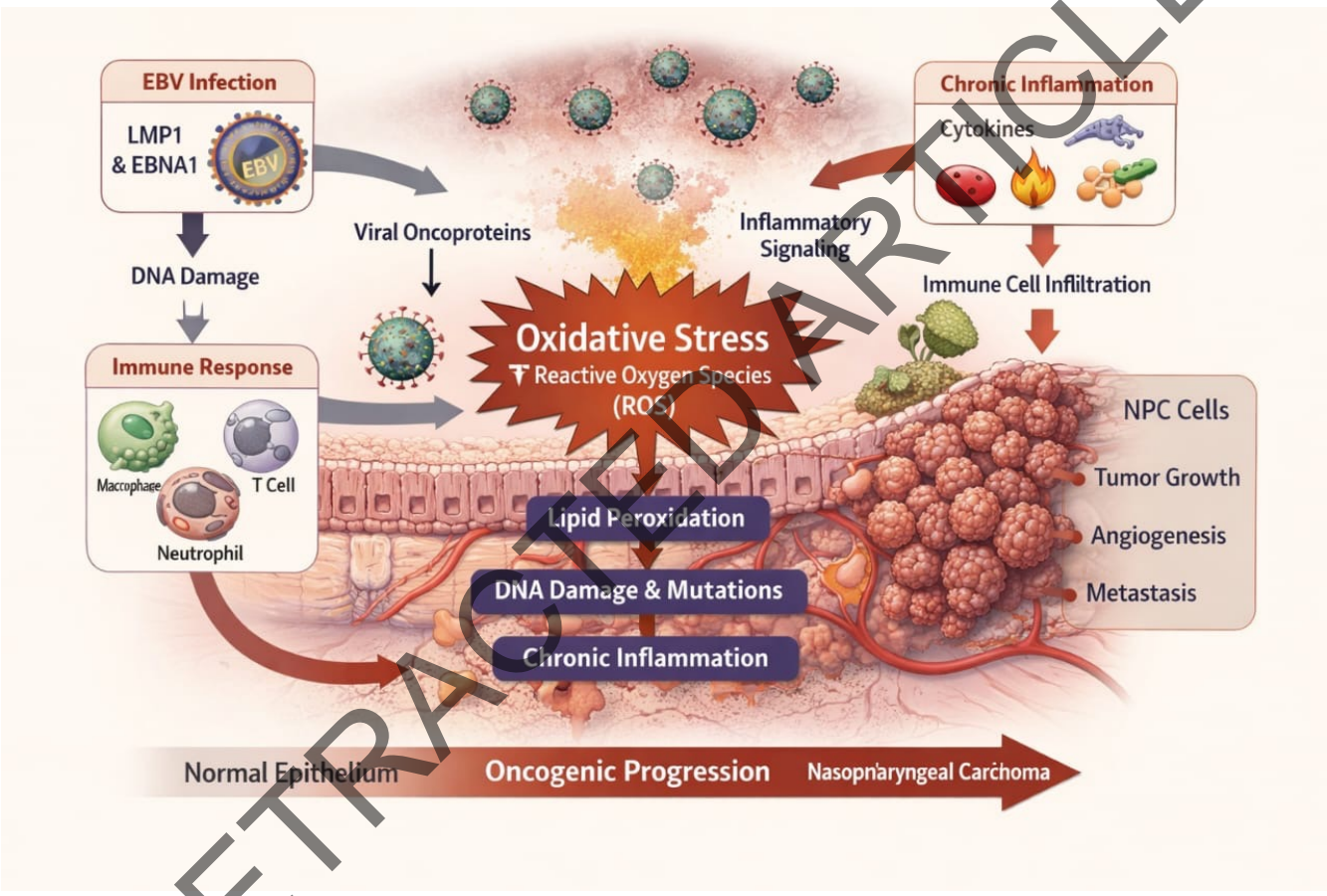

Figure 1. Oxidative stress driven oncogenic progression in NPC.

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This schematic illustrates the interplay between EBV infection, chronic inflammation, and host immune responses in promoting oxidative stress within nasopharyngeal epithelium. EBV latent oncoproteins (e.g., LMP1 and EBNA1) stimulate intracellular reactive oxygen species (ROS) production and induce DNA damage. Concurrently, chronic inflammation and cytokine release recruit immune cells such as macrophages, neutrophils, and T lymphocytes, which further amplify ROS generation through inflammatory signaling pathways. The cumulative oxidative stress leads to lipid peroxidation, genomic instability, and mutation accumulation, thereby accelerating oncogenic transformation. Persistent redox imbalance promotes tumor growth, angiogenesis, and metastatic potential, ultimately driving the progression from normal epithelium to NPC].

Two enzyme systems are central to this balance; GPx-1, a selenium-dependent antioxidant enzyme that detoxifies hydrogen peroxide and lipid hydro peroxides, thereby protecting cellular membranes and genomic DNA [11]. The second enzyme is MPO, a heme-containing enzyme in neutrophils that generates hypochlorous acid for antimicrobial defense but can promote tissue injury and mutagenesis when

overactive [12, 13]. Functional polymorphisms in either system may tilt redox homeostasis toward persistent oxidative damage, thereby increasing cancer susceptibility [14].

Beyond GPx-1 and MPO, multiple antioxidant and pro oxidant genes contribute to redox homeostasis and influence cancer susceptibility [15]. Genetic variation in these genes may modulate individual oxidative stress responses and modify cancer risk across populations, these genes include: (1) Catalase, breaks down hydrogen peroxide; polymorphisms in its promoter region can alter enzyme activity and influence cancer risk [16]. (2) Superoxide dismutase, convert superoxide radicals to hydrogen peroxide; SNPs such as SOD2 Val16Ala have been linked to altered ROS levels and cancer susceptibility in other malignancies [17]. (3) Peroxiredoxins, reduce peroxide levels and regulate redox signaling; their expression is affected by viral oncogenes and oxidative stress in NPC cell models [18]. (4) Nuclear factor erythroid 2 related factor 2 (Nrf2), master regulator of antioxidant response controlling transcription of numerous detoxification and antioxidant enzymes [17]. (5) Glutathione peroxidase 4 (GPx-4), protects against ferroptosis, a form of oxidative cell death; EBV infection induces GPx-4 expression in NPV, promoting progression and therapy resistance [19]. This expanding network of redox related genes highlights that genetic predisposition to oxidative imbalance is multi genic and may interact with environmental and viral factors to modify NPC susceptibility and progression.

EBV, Oxidative Stress, and NPC

EBV infection is a hallmark of nonkeratinizing NPC and contributes to tumorigenesis through inflammation, genomic instability, immune modulation, and metabolic reprogramming [20]. Recent studies demonstrate that EBV actively drives oxidative stress, creating a redox environment that promotes oncogenic progression and therapy resistance. EBV oncoprotein LMP1 induces high ROS levels through upregulation of NADPH oxidases and triggers a redox resetting in infected NPC cells characterized by increased ROS accumulation and enhanced antioxidant defenses via Nrf2 signaling. This redox shift is associated with EBV reactivation and contributes to radiosities and tumor recurrence [21].

Markers of oxidative DNA damage such as (8-hydroxy-2-deoxyguanosine) are elevated in NPC patient samples and correlate with both EBV DNA levels and poor clinical outcomes, underscoring a direct link between viral infection and oxidative genomic stress [21]. Additionally, EBV nuclear antigen EBNA1 has been shown to regulate oxidative stress response proteins like superoxide dismutase and peroxiredoxin, increasing ROS and potentially facilitating NPC metastasis [18]. This evidence supports a model in which EBV not only establishes latent infection but also reprograms host redox mechanisms to favor carcinogenesis, linking viral biology, inflammation oxidative stress, and genetic susceptibility.

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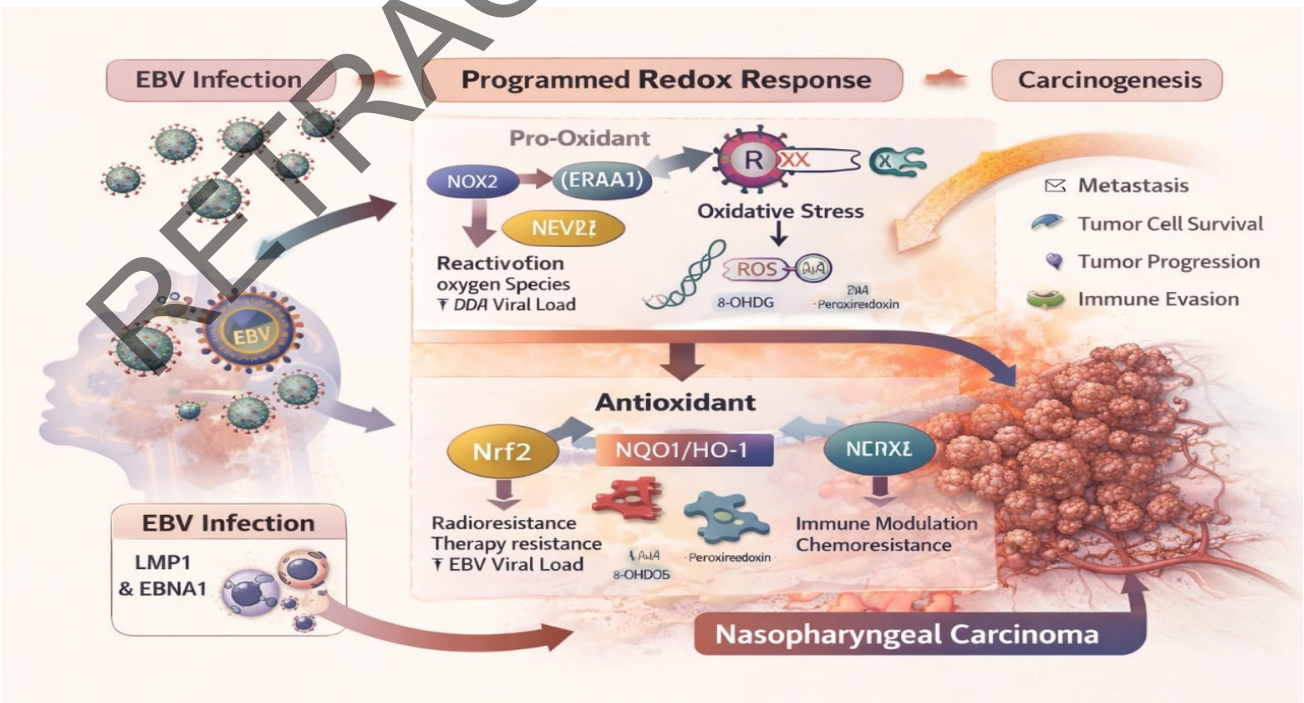

Figure 2. EBV mediated reprogramming of host redox mechanisms in NPC.

This schematic illustrates how EBV infection reshapes host cellular redox homeostasis to promote carcinogenesis in nasopharyngeal epithelial cells. Following infection, EBV latent oncoproteins (including LMP1 and EBNA1) stimulate pro-oxidant pathways such as NADPH oxidases (e.g., NOX2), leading to increased production of reactive oxygen species (ROS). Elevated ROS induces oxidative DNA damage (e.g., 8-OHdG formation), genomic instability, and activation of oncogenic signaling pathways]. (Adapted from author.

Concurrently, EBV activates antioxidant defense systems through redox-sensitive transcription factors such as Nrf2, upregulating downstream targets (e.g., NQO1, HO-1, and peroxiredoxins). This "redox reprogramming" establishes a balanced but persistently elevated oxidative state that supports tumor cell survival under stress conditions, enhances resistance to radiotherapy and chemotherapy, and facilitates immune modulation [22]. The combined pro-oxidant and antioxidant adaptations create a tumor-permissive microenvironment that accelerates malignant transformation, tumor progression, immune evasion, and metastasis, ultimately contributing to the development and maintenance of NPC.

Tumor Microenvironment and Oxidative Stress in NPC

The tumor microenvironment (TME) critically influences NPC progression by integrating malignant cells with immune cells, stromal fibroblasts, endothelial cells, and extracellular matrix. Interaction between these components creates a pro-oxidant and inflammatory niche that promotes tumor growth, immune evasion, and metastasis [23]. Chronic inflammation within the TME drives persistent ROS production, which not only induces direct DNA damage but also modulates cell signaling pathway promoting malignant transformation and survival. ROS in the TME is generated by cancer cells, infiltrating inflammatory cells (neutrophils and macrophages), and stromal fibroblasts, leading to genomic instability and metastatic behavior through cytoskeleton remodeling and mitophagy pathways [24].

In NPC specifically, the coexistence of tumor-infiltrating lymphocytes with EBV-infected epithelial cells fosters a distinct TME that enables immune evasion while supporting oxidative stress driven oncogenesis. Studies indicate that EBV products such as LMP1 and EBNA1 can further alter oxidative state of NPC cells and their surroundings, increasing ROS via NADPH oxidases and modifying antioxidant pathways [25]. The combination of an inflammatory microenvironment and oxidative stress underscores the importance of redox regulation as determinant of disease progression and therapeutic resistance in NPC, linking extracellular signals directly to oncogenic pathways and DNA damage responses.

Genetic Polymorphisms as Modifiers of Cancer Risk

Genetic polymorphisms (defined as DNA variants present in at least 1% of a population) can influence enzyme activity, expression levels, or protein stability [26]. While many are silent, nonsynonymous single nucleotide polymorphisms (SNPs) may directly modify protein structure and catalytic efficiency, thereby altering disease susceptibility [27]. GPx-1 polymorphisms are particularly notable because the gene in located on chromosome 3p21.3 and includes functional variants previously associated with reduced enzymatic activity and elevated cancer risk [28]. Meta-analysis have confirmed that GPX-1 polymorphisms such as 1050450 are associated with overall cancer risk in pooled analysis, including head and neck cancers, suggesting broader importance of oxidative defense variation. Similarly, MPO polymorphisms at chromosome 17q22 can influence inflammatory responses and oxidative burden by altering enzyme production or activity [29].

GPx-1 Polymorphisms in NPC

GPx-1 polymorphisms have been linked to several malignancies, including colorectal, bladder, and lung cancers in diverse populations, supporting the biological plausibility that compromised peroxide detoxification promotes malignant transformation [30]. The Jordanian data extend these observations to NPC and highlight population specific genetic risk profiles; the study involved 40 NPC patients and 18 controls.

In the Jordanian NPC cohort, sequencing revealed two major nonsynonymous variants; 1416T>TC (Isoleucine→Threonine) associated with a 1.68-fold increased NPC risk (OR = 1.68; P = 0.004). And 1458C>T (Prolin →Leucine; P200L) displayed a stronger

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association (OR = 2.03; P = <0.001). Structural modeling suggested that these substitutions may destabilize the enzyme's hydrophobic core or disrupt protein folding, ultimately reducing antioxidant efficiency and heightening vulnerability to ROS-mediated DNA damage.

MPO Polymorphisms in NPC

MPO gene variation has shown inconsistent association with cancer across populations, acting either as a risk or protective factor depending on tumor type and genetic background [31]. The Jordanian NPC study identified two nonsynonymous variants; 611G>A (Glycine→Arginine) OR = 1.54; P = 0.013. And 763G>GA (Glycine→Serine) OR = 1.89; P = 0.001. Both variants were predicted to influence catalytic activity or protein folding, potentially enhancing MPO-mediated ROS production and chronic inflammatory signaling in nasopharyngeal tissues.

Gene Environment virus Interactions in NPC

NPV development can be conceptualized as a multistep process, where none of the factors alone is sufficient, but together they create a permissive oncogenic environment:

Dietary Nitrosamines; Consumption of salt preserved fish and other nitrosamine rich foods has been strongly associated with NPC risk. Nitrosamines generate DNA adducts and oxidative damage, individuals carrying polymorphisms in detoxification and oxidative stress related genes may have reduced capacity to neutralize carcinogens increasing susceptibility to DNA damage [3].

Salt preserved fish and processed foods contain nitrosamines which require metabolic activation to become DNA damaging intermediates. MPO produced by neutrophils during chronic inflammation generates strong oxidants such as hypochlorous acid. MPO activity may enhance nitrosamine activation, increase oxidative and chlorinated DNA damage, and amplify inflammatory ROS production [32].

Tobacco Smoke; Cigarette smoking increases ROS and nitrosamine exposure in the nasopharyngeal epithelium, polymorphisms in oxidative stress regulating genes may modulate the extent of oxidative DNA damage in exposed individuals, this decreases the ability to neutralize hydrogen peroxide and lipid peroxides generated by tobacco exposure leading to, increased oxidative damage, higher mutation burden, and enhanced carcinogenic susceptibility [33].

Viral Load and Host Immune Genetics; Circulating EBV DNA is a validated biomarker for NPC diagnosis and prognosis, host immune gene variants may influence viral persistence and viral load levels, high EBV viral load combined with reduced antioxidant capacity may result in persistent oxidative DNA damage, genomic instability, and enhanced malignant transformation [34].

Genetic Susceptibility to Cancer in Jordan

Genetic epidemiology studies in Jordan have increasingly highlighted polymorphisms in metabolic and inflammatory genes such as MTHFR, CYP450, IL-1, HRAS, and GSTP1 as contributors to cancer risk, emphasizing the role of detoxification and immune pathways in tumor development [35]. However, NPC specific genetic investigations remain scare. Given Jordan's unique environmental exposures and ethnic composition, the identification of GPx-1 and MPO variants linked to NPC represents a major step toward defining regional molecular risk profiles.

Clinical and Public Health Implications

The consistent association of antioxidant and pro-oxidant related gene polymorphisms with NPC in Jordan support several translational possibilities; Risk stratification through genetic screening in high incidence families or endemic regions, Preventive strategies emphasizing antioxidant status, and personalized surveillance for individuals carrying high risk alleles. The study concluded that these polymorphisms may serve as future biomarkers for NPC susceptibility, although functional assays and larger population-based studies remain essential before clinical implementation.

Conclusion

Accumulating evidence implicates oxidative stress as a driving force in NPC pathogenesis. Findings from Jordan demonstrate that nonsynonymous variants in GPx-1 and MPO significantly increase NPC risk, reinforcing the biological importance of redox regulation in carcinogenesis. These data expand the global understanding of NPC genetics and emphasize the need for region specific molecular epidemiology studies to guide precision oncology initiatives.

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References

1. Johnson, D.E., Burtness, B., et al. Head and neck squamous cell carcinoma. Nature reviews Disease primers. **2020**. 6(1): 92.
2. Bray, F., Laversanne, M., et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: a cancer journal for clinicians. **2024**. 74(3): 229–263.
3. Tsang, C.M., Lui, V.W.Y., et al. Translational genomics of nasopharyngeal cancer. in *Seminars in cancer biology*. 2020. Elsevier.
4. Juan, C.A., Pérez de la Lastra, J.M., et al. The chemistry of reactive oxygen species (ROS) revisited: outlining their role in biological macromolecules (DNA, lipids and proteins) and induced pathologies. International journal of molecular sciences. **2021**. 22(9): 4642.
5. Bei, J.-X., Jia, W.-H., and Zeng, Y.-X. Familial and large-scale case–control studies identify genes associated with nasopharyngeal carcinoma. in *Seminars in cancer biology*. 2012. Elsevier.
6. Mody, M.D., Rocco, J.W., et al. Head and neck cancer. The Lancet. **2021**. 398(10318): 2289–2299.
7. Hamid, G.A. Epidemiology and outcomes of nasopharyngeal carcinoma. Pharynx-Diagnosis and Treatment. **2021**.
8. Liu, H., Tang, L., et al. Nasopharyngeal carcinoma: current views on the tumor microenvironment's impact on drug resistance and clinical outcomes. Molecular cancer. **2024**. 23(1): 20.
9. Lushchak, V.I. Free radicals, reactive oxygen species, oxidative stress and its classification. Chemico-biological interactions. **2014**. 224: 164–175.
10. Valko, M., Rhodes, C., et al. Free radicals, metals and antioxidants in oxidative stress-induced cancer. Chemico-biological interactions. **2006**. 160(1): 1–40.
11. Li, D., Ding, Z., et al. Reactive oxygen species as a link between antioxidant pathways and autophagy. Oxidative Medicine and Cellular Longevity. **2021**. 2021(1): 5583215.
12. Handy, D.E. and Loscalzo, J. The role of glutathione peroxidase-1 in health and disease. Free Radical Biology and Medicine. **2022**. 188: 146–161.
13. Kargapolova, Y., Geißen, S., et al. The enzymatic and non-enzymatic function of myeloperoxidase (MPO) in inflammatory communication. Antioxidants. **2021**. 10(4): 562.
14. Pandey, M.K., Von Suskil, M., et al., Cancer on fire: role of inflammation in prevention and treatment, in *Current advances for development of functional foods modulating inflammation and oxidative stress*. **2022**, Elsevier. p. 605–626.
15. Pei, J., Pan, X., et al. Research progress of glutathione peroxidase family (GPX) in redoxidation. Frontiers in pharmacology. **2023**. 14: 1147414.
16. Rajaraman, P., Hutchinson, A., et al. Oxidative response gene polymorphisms and risk of adult brain tumors. Neuro-oncology. **2008**. 10(5): 709–715.
17. Smail, S.W., Ismail, B.A., et al. Antioxidant and oxidative enzymes, genetic variants, and cofactors as prognostic biomarkers of COVID-19 severity and mortality: A systematic review. Frontiers in molecular biosciences. **2025**. 12: 1700263.
18. Cao, J.Y., Mansouri, S., and Frappier, L. Changes in the nasopharyngeal carcinoma nuclear proteome induced by the EBNA1 protein of Epstein-Barr virus reveal potential roles for EBNA1 in metastasis and oxidative stress responses. Journal of virology. **2012**. 86(1): 382–394.
19. Yuan, L., Li, S., et al. EBV infection-induced GPX4 promotes chemoresistance and tumor progression in nasopharyngeal carcinoma. Cell Death & Differentiation. **2022**. 29(8): 1513–1527.
20. Rjoub, Y. and Rahaifeh, S.A.A. Epstein-Barr Virus and Nasopharyngeal Carcinoma: A Review on Virology, Oncogenic Mechanisms, and Clinical Implications. Electronic Journal of Medical Research. **2025**. 1(2): 14–22.
21. Hu, J., Li, Y., et al. Targeting Epstein-Barr virus oncoprotein LMP1-mediated high oxidative stress suppresses EBV lytic reactivation and sensitizes tumors to radiation therapy. Theranostics. **2020**. 10(26): 11921.
22. Zhao, Z., Liu, W., and Luo, B. Role of oxidative stress in the Epstein–Barr virus lifecycle and

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tumorigenicity. Future Virology. **2023**. 18(7): 465–477.
23. Jiang, J. and Ying, H. Revealing the crosstalk between nasopharyngeal carcinoma and immune cells in the tumor microenvironment. Journal of Experimental & Clinical Cancer Research. **2022**. 41(1): 244.
24. Liu, J., Zhan, X., et al. Mitochondrial proteomics of nasopharyngeal carcinoma metastasis. BMC medical genomics. **2012**. 5(1): 62.
25. Huang, S., Tsao, S., and Tsang, C., Interplay of viral infection, host cell factors and tumor microenvironment in the pathogenesis of nasopharyngeal carcinoma. Cancers. 2018; 10 (4): 106. Epub 2018/04/05. https://doi.org/10.3390/cancers10040106 PMID: 29617291.
26. Azizzadeh-Roodpish, S., Garzon, M.H., and Mainali, S. Classifying single nucleotide polymorphisms in humans. Molecular Genetics and Genomics. **2021**. 296: 1161–1173.
27. Chiarella, P., Capone, P., and Sisto, R. Contribution of genetic polymorphisms in human health. International Journal of Environmental Research and Public Health. **2023**. 20(2): 912.
28. Ravn-Haren, G., Olsen, A., et al. Associations between GPX1 Pro198Leu polymorphism, erythrocyte GPX activity, alcohol consumption and breast cancer risk in a prospective cohort study. Carcinogenesis. **2006**. 27(4): 820–825.
29. Daiyasu, H. and Toh, H. Molecular evolution of the myeloperoxidase family. Journal of molecular evolution. **2000**. 51(5): 433–445.
30. Chen, J., Cao, Q., et al. GPx-1 polymorphism (rs1050450) contributes to tumor susceptibility: evidence from meta-analysis. Journal of Cancer Research and Clinical Oncology. **2011**. 137: 1553–1561.
31. Hawkins, C.L. and Davies, M.J. Role of myeloperoxidase and oxidant formation in the extracellular environment in inflammation-induced tissue damage. Free Radical Biology and Medicine. **2021**. 172: 633–651.
32. Lazim, N.M. and Abdullah, B., Risk factors and etiopathogenesis of nasopharyngeal carcinoma, in *An Evidence-Based Approach to the Management of Nasopharyngeal Cancer*. **2020**, Elsevier. p. 11–30.
33. Khan, A.Q., Rashid, K., et al. Reactive oxygen species (ROS) in cancer pathogenesis and therapy: An update on the role of ROS in anticancer action of benzophenanthridine alkaloids. Biomedicine & Pharmacotherapy. **2021**. 143: 112142.
34. Ahmed, N., Abusalah, M.A.H.A., et al. Updates on Epstein–Barr Virus (EBV)-associated nasopharyngeal carcinoma: emphasis on the latent gene products of EBV. Medicina. **2022**. 59(1): 2.
35. Al-Eitan, L.N., Al-Ahmad, B.H., and Almomani, F.A. The association of il-1 and HRAS gene polymorphisms with breast cancer susceptibility in a jordanian population of arab descent: A genotype–phenotype study. Cancers. **2020**. 12(2): 283.

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