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ACTIVITY OF ENZYMES OF THE PRO-/ANTIOXIDANT SYSTEM IN BENIGN PROSTATIC HYPERPLASIA

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Background. Benign prostatic hyperplasia (BPH), which is mostly prevalent in older men, is associated with compression of the prostatic urethra and manifests as lower urinary tract symptoms. Its pathological characteristics include abnormal proliferation of stromal and glandular epithelial cells, leading to prostate enlargement. Current data highlight additional factors in the pathogenesis of this disease, notably oxidative stress and chronic inflammation.

Materials and Methods. Prostate tissue was obtained from 35 patients aged 58–72 years via transurethral resection. Prostate tissue from three patient groups was utilized. Group 1 consisted of patients with BPH (n = 7) weighing up to 40 grams (control group); Group 2 included patients with BPH (n = 14) weighing 45–98 grams; Group 3 comprised patients with benign prostatic hyperplasia and chronic inflammation (adenoprostatitis) weighing 45–98 grams (n = 14). The levels of malondialdehyde and reduced glutathione, as well as the activities of glutathione peroxidase and glutathione reductase, were determined in the tissues.

Results. In large-scale prostate hyperplasia and prostate hyperplasia associated with prostatitis, lipid peroxidation processes escalate in prostate cells, as indicated by an increase in malondialdehyde concentration. The elevation in lipid peroxidation levels is accompanied by a decrease in the activity of antioxidant defense enzymes glutathione peroxidase and glutathione reductase, alongside a reduction in the concentration of reduced glutathione. The balance within the pro-/antioxidant system is more significantly disrupted in cases of prostate hyperplasia associated with prostatitis.



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Conclusion. In large-scale prostate hyperplasia and hyperplasia associated with histologically confirmed prostatitis, lipid peroxidation processes intensify within the cells, which is confirmed by rising levels of malondialdehyde. The increase in lipid peroxidation is accompanied by a decline in glutathione peroxidase and glutathione reductase activities, as well as a decrease in the concentration of reduced glutathione. In prostate hyperplasia accompanied by adenoprostatitis, the balance in the pro-/antioxidant system is disrupted to a greater extent.

Keywords: prostate, hyperplasia, inflammation, malondialdehyde, reduced glutathione, glutathione peroxidase, glutathione reductase

INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common diseases affecting aging men, leading to lower urinary tract symptoms that can significantly impact the quality of life. BPH is widely recognized as a global public health issue, particularly among older men, with its prevalence potentially exceeding 70–90 % by the eighth decade of life (Ye *et al.*, 2024). It occurs primarily due to hyperplasia in the transitional zone, is associated with urethral compression, and can manifest as lower urinary tract symptoms, which are predominantly prevalent in older men (Wang *et al.*, 2025). Its pathological characteristics include abnormal proliferation of stromal and glandular epithelial cells, resulting in prostatic enlargement. The etiology of BPH remains unknown, although historically it has been primarily linked to androgen imbalance. Testosterone, produced by the testes, undergoes conversion into dihydrotestosterone and estradiol. Over time, the gradual accumulation of these metabolites reaches elevated concentrations, thereby disrupting the hormonal homeostasis within the prostate gland (Carson & Rittmaster, 2003).

Current data point to additional factors in the pathogenesis of BPH, notably oxidative stress and chronic inflammation (Inamura & Terada, 2024; Kaltsas *et al.*, 2025). Oxidative stress and increased production of reactive oxygen species (ROS) can cause various types of cellular damage and molecular abnormalities in hyperplasia (Kaltsas *et al.*, 2025). In the prostate gland, the hyperproduction of ROS, particularly H_2O_2 and the superoxide radical, is associated with the development of adenoma and even prostate cancer (Liou *et al.*, 2024).

Accumulating data emphasize that chronic inflammation and oxidative stress are critical yet poorly understood aspects of BPH pathogenesis (Bostanci *et al.*, 2013). Inflammatory infiltrates, often consisting of macrophages, lymphocytes, and neutrophils, are frequently detected in hyperplastic prostate tissue, and these immune cells secrete cytokines capable of exacerbating tissue damage. Concurrently, ROS generated through normal metabolic processes or increased inflammatory activity can overwhelm intrinsic antioxidant defense mechanisms, thereby promoting fibromuscular proliferation and disrupting apoptotic mechanisms (Lambeth, 2007). Although some men exhibit histological signs of BPH, they may remain asymptomatic. In many others, however, the condition progresses to distressing lower urinary tract symptoms, including frequent urination and obstructive difficulties with urination (Lee & Peehl, 2004; Forrester *et al.*, 2018).

Despite advancements in understanding the role of inflammation and oxidative stress in the development of prostatic hyperplasia, significant gaps remain in elucidating the involvement of several regulatory systems in this process, particularly the antioxidant system. For instance, biomarkers that accurately reflect local inflammatory activity or

oxidative damage, such as malondialdehyde, pro-inflammatory cytokines, or antioxidant enzymes, have not yet been validated for routine clinical use (Inamura & Terada, 2024). Understanding the mechanisms involved in the onset and progression of BPH, specifically those related to oxidative stress and inflammatory processes, will allow for better characterization and, consequently, more effective prevention and diagnosis of this pathology. In this context, we assessed oxidative stress and investigated its role in the prostate tissues of patients with adenoma and adenoprosatitis by measuring MDA and glutathione (GSH) levels, as well as by evaluating the activity of glutathione peroxidase (GP) and glutathione reductase.

The aim of the study was to investigate the level of lipid peroxidation and the activity of glutathione system enzymes in prostate tissue in benign hyperplasia alone and in benign hyperplasia accompanied by an inflammatory process.

MATERIALS AND METHODS

Study design. Prostate tissue was obtained from 35 patients aged 58–72 years via transurethral resection of the prostate. The mean age of the men was 62.4 ± 5.2 years. All patients presented with similar lower urinary tract symptoms, including dysuria, pollakiuria, urinary retention, and a weak urinary stream. The patients were divided into three groups: Group 1 (control group): patients with benign prostatic hyperplasia (BPH) and a prostate mass of up to 40 grams ($n = 7$). Group 2: patients with BPH and a prostate mass of 45–98 grams ($n = 14$). Group 3: patients with BPH and histologically confirmed chronic prostatitis, with a resected tissue mass of 45–98 grams ($n = 14$). Prostate tissue samples were collected from the three patient groups undergoing treatment at the Urology Clinic of the Danylo Halytsky Lviv National Medical University.

Ethical considerations. The study was conducted in strict compliance with the principles of medical ethics, patient rights protection, human dignity, and moral and ethical standards, in accordance with the Declaration of Helsinki, the Council of Europe Convention on Human Rights and Biomedicine, and the legislation of Ukraine. Approval was granted by the Bioethics Committee of the Danylo Halytsky Lviv National Medical University (protocol No. 7, dated June 26, 2023). All necessary measures were taken to ensure patient safety and respect for their rights, human dignity, and ethical norms.

Tissue processing and homogenization. Prostate tissue samples were cooled in physiological saline, minced, and homogenized using an FSH-2A High-Speed Homogenizer at 4,000 rpm. Homogenization was performed in a 1:9 tissue-to-buffer ratio using ice-to-0 °C-cooled 50 mM Tris-HCl buffer (pH 7.4) containing 1 mM EDTA and 250 mM sucrose. For subsequent analyses, the supernatant obtained by centrifuging the diluted homogenate at 15,000 times g for 15 minutes was used. The supernatant fraction intended for further biochemical studies was aliquoted into ultracentrifuge tubes and immediately frozen at -30 °C.

Lipid peroxidation assay. Lipid peroxidation was assessed by measuring the concentration of MDA in the supernatant fraction according to the method described by H. Nabil *et al.* (2008) and expressed in nanomoles per gram of tissue (nmol/g of tissue). The principle of the MDA determination method is based on the reaction of MDA with 2-thiobarbituric acid at high temperatures in an acidic medium. This reaction yields a colored trimethine complex with maximum absorbance at $\lambda = 532$ nm (Nabil *et al.*, 2008).

Enzyme assay. GP activity was determined by the amount of reduced glutathione, which was used to neutralize hydrogen peroxide in the glutathione peroxidase reaction, considering the dilution of the biological material in the sample and the micromolar absorption coefficient of the thionitrophenyl anion at a wavelength of 412 nm ($11.4 \text{ cm}^2/\mu\text{M}$) (Tramer *et al.*, 2004). GR activity was determined spectrophotometrically by the decrease in optical density as a result of NADPH oxidation at a wavelength of 340 nm.

Statistical analysis. Experimental data were processed by methods of variation statistics using MS Office and BioStat LE software. Inter-group differences were determined using the non-parametric Mann–Whitney U test with Bonferroni correction. P values of < 0.025 (calculated as 0.05 divided by the number of groups minus one control group) were interpreted as statistically significant.

RESULTS AND DISCUSSION

To investigate the relationship between oxidative stress and the development of BPH, we examined a number of markers that play a key role in the mechanism of oxidative stress. In particular, we determined the levels of MDA and GSH, as well as the activity of GPx and GR in prostate tissue in patients with benign hyperplasia and benign hyperplasia associated with chronic prostatitis. A statistically significant increase in MDA concentrations was revealed in patients with large-sized prostatic hyperplasia and hyperplasia accompanied by adenoprostatitis (**Fig. 1**). MDA levels were significantly elevated – 1.60-fold ($P < 0.01$) in patients with isolated prostatic hyperplasia compared to the control group. This increase was even more pronounced in patients with hyperplasia associated with adenoprostatitis, reaching 1.87-fold elevation ($P < 0.01$) compared to the control group, although the difference in MDA levels between the two patient groups was not statistically significant. This trend reflects an intensification of lipid peroxidation processes in patients of both studied groups.

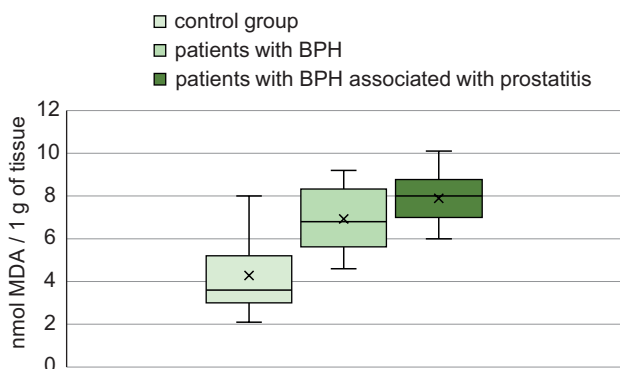


Fig. 1. Changes in MDA levels in patients with large-volume prostatic hyperplasia and prostatic hyperplasia accompanied by histologically confirmed prostatitis compared to the control group, $n = 7-14$

Measurement of reduced glutathione concentration showed that this index is also associated with the development of benign prostatic hyperplasia (**Fig. 2**). Compared to the control group, GSH levels were 2.2-fold lower ($P < 0.001$ in patients with large-volume prostatic hyperplasia, and 3.5-fold lower ($P < 0.001$) in those with hyperplasia accompanied by histologically confirmed prostatitis. The difference in GSH levels between these two patient groups was statistically significant ($P < 0.01$).

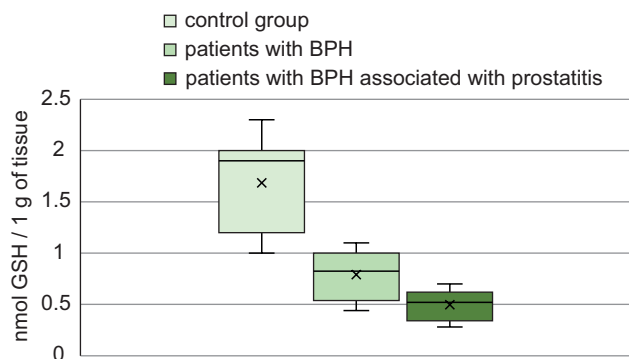


Fig. 2. Changes in GSH levels in patients with large-volume prostatic hyperplasia and prostatic hyperplasia accompanied by histologically confirmed prostatitis compared to the control group, $n = 7-14$

The development of benign prostatic hyperplasia is accompanied by a decrease in glutathione peroxidase activity (**Fig. 3**). Compared to the control group, GP activity was 1.4-fold lower ($P < 0.001$) in patients with benign prostatic hyperplasia, and 1.6-fold lower ($P < 0.001$) in those with hyperplasia accompanied by adenoprostatitis. The difference in GP activity between these two studied groups was statistically significant ($P < 0.01$).

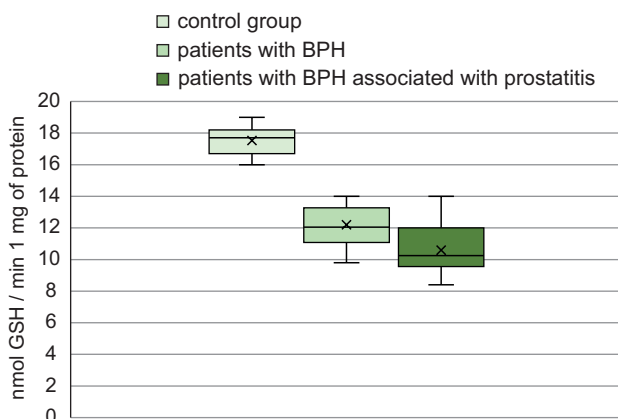


Fig. 3. GP activity in patients with large-volume prostatic hyperplasia and prostatic hyperplasia accompanied by histologically confirmed prostatitis compared to the control group, $n = 7-14$

GR activity was also reduced in both studied groups (**Fig. 4**). In large-volume prostatic hyperplasia, GR activity was 1.5 times lower ($P < 0.001$), and in hyperplasia accompanied by adenoprostatitis, it was 1.69 times lower ($P < 0.001$) compared to the control group. The difference in GR activity between these two patient groups was statistically significant ($P < 0.018$).

Thus, in the cytosolic fraction of the prostate in cases of large-scale prostatic hyperplasia and hyperplasia associated with prostatitis, lipid peroxidation increases, while the concentration of reduced glutathione and the activities of antioxidant defense enzymes (glutathione peroxidase and glutathione reductase) decrease.

Excessive production of free radicals causes direct damage to biological molecules (oxidation of DNA, proteins, lipids, and carbohydrates), as well as secondary damage due

to the cytotoxic and mutagenic properties of the metabolites that are released, in particular, during lipid oxidation (Valgimigli, 2023). Lipid peroxidation leads to the formation of a wide range of products, including aldehydes, in particular MDA. Due to its high cytotoxicity and inhibitory effect on antioxidant enzymes, MDA acts as a cancer promoter and a co-carcinogenic agent. Lipid peroxidation is a free-radical reaction involving the oxidative conversion of polyunsaturated fatty acids to MDA or other lipid hydroperoxide products.

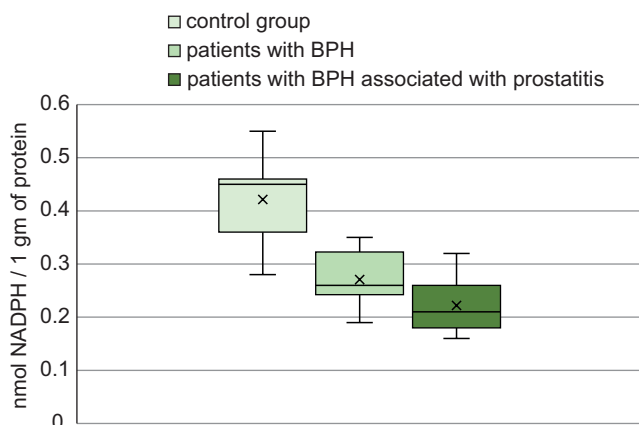


Fig. 4. GR activity in patients with large-volume prostatic hyperplasia and prostatic hyperplasia accompanied by histologically confirmed prostatitis compared to the control group, $n = 7-14$

Oxidative stress and accumulated DNA damage increase the risk of prostate cancer (Shukla *et al.*, 2020). These data suggest that chronic inflammation mediated by ROS may play an important role in DNA damage, leading to neoplastic transformation in prostate epithelial cells. The results of our study show elevated MDA levels in cytosolic adenomatous prostate tissues compared to the control group.

Among non-protein thiols, GSH plays a key role in maintaining intracellular antioxidant defense by scavenging ROS and regenerating other antioxidant molecules (Shukla *et al.*, 2020; Tsikas, 2017). In the cytoplasm, micromolar concentrations of reduced GSH effectively scavenge hydrogen peroxide. This process leads to an increase in the level of oxidized GSH (GSSG), which is converted back to GSH by glutathione reductase in an NADPH-dependent redox cycle to maintain sufficient intracellular GSH levels. It has been found that cytosolic GSH levels measured in various groups of patients with prostate adenoma were also reduced (Zabaiou *et al.*, 2017). Moreover, relatively low concentrations were observed in larger tumors. Depletion of cytosolic GSH levels may be associated with inhibition of GSH synthesis in adenomatous cells. It is known that the antioxidant activity of GSH depends on two enzymes: glutathione peroxidase (GP) and glutathione reductase (GR) (Weldy *et al.*, 2012).

GR catalyzes the reduction of oxidized glutathione (GSSG) to its reduced form, glutathione (GSH), and plays a role in maintaining glutathione in its reduced form. A decrease in GSH is therefore likely associated with the inhibition of GR activity, which prevents the regeneration of glutathione, leading to its accumulation in the oxidized form. This inhibition may also result from the suppression of genes encoding enzymes involved in glutathione synthesis, primarily glutathione synthetase, due to mutations or transcriptional inactivation. It may also be associated with a deficiency of one or more amino acids involved in its synthesis.

Cytosolic GPx activity in prostatic hyperplasia was shown to decrease with increasing prostate size. As for catalase, there are two possible explanations for the behavior of this activity. During oxidative stress, a deficiency of certain non-enzymatic antioxidants such as vitamin E, vitamin C, carotenoids, and certain trace elements like selenium was observed (Pisoschi *et al.*, 2021). The latter deficiency is most often accompanied by a decrease in GP activity.

In the prostates of older men, especially in the presence of low-grade chronic inflammation, ROS levels may remain persistently elevated, stimulating the expression of pro-inflammatory genes, promoting abnormal proliferation, and interfering with normal apoptotic control. The resulting net increase in cellular mass in the transitional zone leads to the characteristic nodules observed in patients with BPH. Given the dual nature of ROS, completely eliminating all radicals may be harmful. Instead, the goal is to finely tune their levels and restore physiological redox signaling (Kaltsas *et al.*, 2025). The non-protein thiol GSH and glutathione reductase maintain optimal cellular redox potential by inactivating H_2O_2 .

According to Sajjaboontawee *et al.* (2020), thiol/GP antioxidant defense is significantly impaired in patients with prostate diseases (Sajjaboontawee *et al.*, 2020). Patients with BPH occupy an intermediate position between the prostate cancer risk group and prostate carcinoma. These results suggest that impaired thiol-based antioxidant mechanisms play a crucial role in prostate diseases, particularly in prostate carcinoma.

In our previous studies, the activity of glutathione S-transferase was also investigated as a key component of the detoxification system and cellular antioxidant defense, further confirming the impairment of oxidative homeostasis in prostatic hyperplasia and associated inflammation (Kushynska *et al.*, 2025). The identified impairments in the antioxidant system and elevated levels of oxidative stress markers in the present work can be used as additional diagnostic criteria to assess the risk of progression of benign prostatic hyperplasia, as well as serve as a basis for the development of new therapeutic strategies aimed at restoring the physiological redox balance.

CONCLUSION

The development of benign prostatic hyperplasia is accompanied by an imbalance in the oxidative status. In large-volume prostatic hyperplasia and hyperplasia with histologically confirmed prostatitis, cellular lipid peroxidation increases, as indicated by elevated malondialdehyde levels, with these processes being more pronounced in the presence of concomitant prostatitis. The increase in lipid peroxidation is accompanied by a decrease in the activity of antioxidant defense enzymes – glutathione peroxidase and glutathione reductase – as well as a reduction in the concentration of reduced glutathione. In prostatic hyperplasia accompanied by adenoprosatitis, the balance within the pro-/antioxidant system is disrupted to a greater extent, as evidenced by higher levels of malondialdehyde, lower activities of glutathione peroxidase and glutathione reductase, and a lower level of reduced glutathione.

COMPLIANCE WITH ETHICAL STANDARDS

Conflict of interest: the authors declare that the study was conducted in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

Animal rights: this article does not include animal studies.

Human rights: all studies were conducted in accordance with the Declaration of Helsinki guidelines. Approval for the study was taken from the ethics committee of the Danylo Halytsky Lviv National Medical University (protocol No 7, dated June 26, 2023).

AUTHOR CONTRIBUTIONS

Conceptualization, [V.D.; F.R.; V.Z.]; methodology, [K.M.; O.O.]; validation, [K.M.; V.D.]; formal analysis, [K.M.; V.D.; G.N.]; investigation, [O.O.; P.O.]; resources, [F.R.; V.D.]; data curation, [O.O.; G.N.; A.M.]; writing – review and editing, [K.M.; V.D.; F.R.; V.D.]; visualization, [O.O.; P.O.]; supervision, [V.D.; F.R.; V.Z.]; project administration, [V.D.; V.Z.]; funding acquisition, [V.D.].

All authors have read and agreed to the published version of the manuscript.

REFERENCES

- Anderson, M. E. (1985). Determination of glutathione and glutathione disulfide in biological samples. In R. A. Greenwald (Ed.), *Handbook methods for oxygen radical research* (Methods in enzymology, Vol. 113, pp. 548–555). Elsevier. doi:10.1016/s0076-6879(85)13073-9
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Bostanci, Y., Kazzazi, A., Momtahan, S., Laze, J., & Djavan, B. (2013). Correlation between benign prostatic hyperplasia and inflammation. *Current Opinion in Urology*, 23(1), 5–10. doi:10.1097/mou.0b013e32835abd4a
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Carson, C., III, & Rittmaster, R. (2003). The role of dihydrotestosterone in benign prostatic hyperplasia. *Urology*, 61(4), 2–7. doi:10.1016/s0090-4295(03)00045-1
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Chughtai, B., Lee, R., Te, A., & Kaplan, S. (2011). Role of inflammation in benign prostatic hyperplasia. *Reviews in Urology*, 13(3), 147–150.
[PubMed](#) • [PMC](#) • [Google Scholar](#)
- Forrester, S. J., Kikuchi, D. S., Hernandes, M. S., Xu, Q., & Griendling, K. K. (2018). Reactive oxygen species in metabolic and inflammatory signaling. *Circulation Research*, 122(6), 877–902. doi:10.1161/circresaha.117.311401
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Grutman, A. J., Su, Z. T., & Herati, A. S. (2025). Testosterone effects on the prostate: focusing on benign prostatic hyperplasia. In B. R. Zirkin, I. Huhtaniemi, D. J. Lamb, & V. Papadopoulos (Eds.), *Leydig cells: formation, regulation and function in health and disease* (pp. 785–801). Springer. doi:10.1007/978-3-031-96376-6_31
[Crossref](#) • [Google Scholar](#)
- Inamura, S., & Terada, N. (2024). Chronic inflammation in benign prostatic hyperplasia: pathophysiology and treatment options. *International Journal of Urology*, 31(10), 968–974. doi:10.1111/iju.15518
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Kaltsas, A., Giannakas, T., Stavropoulos, M., Kratiras, Z., & Chrisofos, M. (2025). Oxidative stress in benign prostatic hyperplasia: mechanisms, clinical relevance and therapeutic perspectives. *Diseases*, 13(2), 53. doi:10.3390/diseases13020053
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Kushynska, M. E., Onufrovykh, O. K., Vorobets, D. Z., Besedina, A. S., Galyk, G. V., Pershyn, O. I., Fafula, R. V., & Vorobets, Z. D. (2025). Kinetic properties of glutathione-S-transferase in prostate gland biopsies of patients with benign prostatic hyperplasia and chronic prostatitis. *Regulatory Mechanisms in Biosystems*, 16(4), e25164. doi:10.15421/0225164
[Crossref](#) • [Google Scholar](#)
- Lambeth, J. D. (2007). Nox enzymes, ROS, and chronic disease: an example of antagonistic pleiotropy. *Free Radical Biology and Medicine*, 43(3), 332–347. doi:10.1016/j.freeradbiomed.2007.03.027
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)

- Lee, K. L., & Peehl, D. M. (2004). Molecular and cellular pathogenesis of benign prostatic hyperplasia. *Journal of Urology*, 172(5), 1784–1791. doi:10.1097/01.ju.0000133655.71782.14
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Liou, G.-Y., C'lay-Pettis, R., & Kavuri, S. (2024). Involvement of reactive oxygen species in prostate cancer and its disparity in African descendants. *International Journal of Molecular Sciences*, 25(12), 6665. doi:10.3390/ijms25126665
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Nabil, H., Moemen, L. A., & Abu Elela, M. H. (2008). Studying the levels of malondialdehyde and antioxidant parameters in normal and abnormal human seminal plasma. *Australian Journal of Basic and Applied Sciences*, 2(3), 773–778.
[Google Scholar](#)
- Pisoschi, A. M., Pop, A., Iordache, F., Stanca, L., Predoi, G., & Serban, A. I. (2021). Oxidative stress mitigation by antioxidants – an overview on their chemistry and influences on health status. *European Journal of Medicinal Chemistry*, 209, 112891. doi:10.1016/j.ejmech.2020.112891
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Raijmakers, M. T., Roelofs, H. M., Steegers, E. A., Steegers-Theunissen, R. P. M., Mulder, T. P., Knapen, M. F., Wong, W. Y., & Peters, W. H. (2003). Glutathione and glutathione S-transferases A1-1 and P1-1 in seminal plasma may play a role in protecting against oxidative damage to spermatozoa. *Fertility and Sterility*, 79(1), 169–172. doi:10.1016/s0015-0282(02)04404-7
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Sajjaboontawee, N., Supasitthumrong, T., Tunvirachaisakul, C., Nantachai, K., Snaboon, T., Reiche, E. M. V., Simão, A. N. C., & Maes, M. (2020). Lower thiol, glutathione, and glutathione peroxidase levels in prostate cancer: a meta-analysis study. *Aging Male*, 23(5), 1533–1544. doi:10.1080/13685538.2020.1858048
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Shukla, S., Srivastava, J. K., Shankar, E., Kanwal, R., Nawab, A., Sharma, H., Bhaskaran, N., Ponsky, L. E., Fu, P., MacLennan, G. T., & Gupta, S. (2020). Oxidative stress and antioxidant status in high-risk prostate cancer subjects. *Diagnostics*, 10(3), 126. doi:10.3390/diagnostics10030126
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Tramer, F., Caponecchia, L., Sgrò, P., Martinelli, M., Sandri, G., Panfili, E., Lenzi, A., & Gandini, L. (2004). Native specific activity of glutathione peroxidase (GPx-1), phospholipid hydroperoxide glutathione peroxidase (PHGPx) and glutathione reductase (GR) does not differ between normo- and hypomotile human sperm samples. *International Journal of Andrology*, 27(2), 88–93. doi:10.1046/j.1365-2605.2003.00452.x
[Crossref](#) • [PubMed](#) • [Google Scholar](#)
- Valgimigli, L. (2023). Lipid peroxidation and antioxidant protection. *Biomolecules*, 13(9), 1291. doi:10.3390/biom13091291
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Wang, G., Lv, C., Liu, Z., Huang, M., Zhang, Y., Chen, J., Hu, J., Jin, Y., & Bai, Z. (2025). Gland- and cell-level heterogeneity in the prostate: a narrative review of related diseases. *Current Urology*, 19(4), 241–246. doi:10.1097/cu9.0000000000000269
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Weldy, C. S., Luttrell, I. P., White, C. C., Morgan-Stevenson, V., Bammler, T. K., Beyer, R. P., Afsharinejad, Z., Kim, F., Chitale, K., & Kavanagh, T. J. (2012). Glutathione (GSH) and the GSH synthesis gene *Gclm* modulate vascular reactivity in mice. *Free Radical Biology and Medicine*, 53(6), 1264–1278. doi:10.1016/j.freeradbiomed.2012.07.006
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Ye, Z., Wang, J., Xiao, Y., Luo, J., Xu, L., & Chen, Z. (2024). Global burden of benign prostatic hyperplasia in males aged 60–90 years from 1990 to 2019: results from the global burden of disease study 2019. *BMC Urology*, 24(1), 193. doi:10.1186/s12894-024-01582-w
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)
- Zabaiou, N., Mabed, D., Lobaccaro, J. M., & Lahouel, M. (2016). Oxidative stress in benign prostate hyperplasia. *Andrologia*, 48(1), 69–73. doi:10.1111/and.12420
[Crossref](#) • [PubMed](#) • [Google Scholar](#)

Zhang, Y., Hu, X., & Zou, L.-Q. (2024). Flavonoids as therapeutic agents for epilepsy: unveiling anti-inflammatory and antioxidant pathways for novel treatments. *Frontiers in Pharmacology*, 15, 1457284. doi:10.3389/fphar.2024.1457284
[Crossref](#) • [PubMed](#) • [PMC](#) • [Google Scholar](#)

АКТИВНІСТЬ ЕНЗИМІВ ПРО-/АНТИОКСИДАНТНОЇ СИСТЕМИ ЗА ГІПЕРПЛАЗІЇ ПЕРЕДМІХУРОВОЇ ЗАЛОЗИ

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Обґрунтування. Доброякісна гіперплазія передміхурової залози (ДГПЗ) пов'язана з компресією простатичної уретри. Унаслідок такої компресії порушується функціонування нижніх сечовивідних шляхів, що здебільшого поширено у чоловіків старшого віку. До патологічних характеристик захворювання належить аномальна проліферація стромальних і залозистих епітеліальних клітин, що спричиняє збільшення передміхурової залози. Сучасні дані вказують на додаткові фактори в патогенезі цього захворювання, зокрема, на оксидативний стрес і хронічне запалення.

Матеріали та методи. Тканину передміхурової залози отримано від 35 пацієнтів віком 58–72 років за допомогою трансуретральної резекції. Використовували тканину передміхурової залози трьох груп пацієнтів. Група 1 – пацієнти з ДГПЗ ($n = 7$) масою до 40 г (контроль); група 2 – пацієнти з ДГПЗ ($n = 14$) масою 45–98 г; група 3 – пацієнти з доброякісною гіперплазією передміхурової залози та хронічним запаленням (аденопростатитом) масою 45–98 г ($n = 14$). У тканинах визначали вміст малонового діальдегіду, відновленого глутатіону й активності глутатіонпероксидази та глутатіонредуктази.

Результати. За гіперплазії простати великих розмірів і гіперплазії простати з простатитом у клітинах простати активізуються процеси пероксидації ліпідів, що підтверджується підвищенням концентрації малонового діальдегіду. Зростання пероксидації ліпідів супроводжується зниженням активності ензимів антиоксидантного захисту (глутатіонпероксидази та глутатіонредуктази) і концентрації відновленого глутатіону. За гіперплазії простати із простатитом більшою мірою порушений баланс у про-/антиоксидантній системі.

Висновок. За гіперплазії простати великих розмірів і гіперплазії, що супроводжується гістологічно підтвердженим простатитом, у клітинах посилюються процеси пероксидного окиснення ліпідів, що підтверджується зростанням рівня малонового діальдегіду. Зростання пероксидації ліпідів супроводжується зниженням активності глутатіонпероксидази та глутатіонредуктази, а також зниженням концентрації відновленого глутатіону. За гіперплазії простати, що супроводжується аденопростатитом, баланс у про-/антиоксидантній системі порушений більшою мірою.

Ключові слова: простата, гіперплазія, запалення, малоновий діальдегід, відновлений глутатіон, глутатіонпероксидаза, глутатіонредуктаза