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Kinetic properties of glutathione-S-transferase in prostate gland biopsies of patients with benign prostatic hyperplasia and chronic prostatitis

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The molecular and stromal mechanisms associated with the pathogenesis of prostate hyperplasia as a metabolic disorder are not yet fully understood, but it is believed that the etiology of hyperplasia is influenced by a number of factors, including aging, hormonal changes, metabolic syndrome, dietary factors, inflammation, oxidative stress, and, more recently, suppression of apoptosis in prostate tissue. A number of studies emphasize the disruption of pro/antioxidant status in the development of prostate hyperplasia. The aim of the research is to study the activity and kinetic characteristics of glutathione-S-transferase (GsT) in prostate biopsies from patients with benign hyperplasia and hyperplasia with chronic prostatitis. The prostate tissue (biopsies) from two groups of patients were used. Group 1 consisted of patients with benign prostatic hyperplasia (n = 14); group 2 consisted of patients with benign prostatic hyperplasia and chronic prostatitis (n = 14). It was shown that GsT activity in the soluble fraction of prostate biopsies from patients with benign hyperplasia with chronic prostatitis was 1.60 times higher than in patients without chronic prostatitis. It was found that the kinetics of the GsT reaction is consistent with the patterns of a zero-order reaction in the range of 0–3 min: in this time interval, the dependence graph of the product formation on the incubation period was practically linear. It was shown that the value of V_0 in patients with prostatic hyperplasia with chronic prostatitis was almost twice as high as that in patients without chronic prostatitis. Over the entire range of GSH concentrations, enzyme activity in samples from patients with inflammation was higher than in patients without chronic prostatitis. Similar changes were also observed with an increase in the concentration of 1-chloro-2,4-dinitrobenzene in the incubation medium at a constant GSH concentrations. Calculation of the kinetic parameters of GsT activity shows that the maximum reaction rate of accumulation of the optically active dinitrobenzene conjugate, determined by GSH, was 1.44 times higher in patients with chronic prostatitis compared to the first group. The affinity constant for GSH in this group of patients was 2.28 times higher compared to patients without chronic prostatitis. When interpreting the obtained kinetic parameters determined by GSH, it is shown that GsT activity in patients with benign prostatic hyperplasia without chronic prostatitis is reduced both due to a decrease in the enzyme reaction rate (V_{max} decreases) and due to a increase in the enzyme's affinity for GSH (KGSH decreases).

Keywords: prostatic hyperplasia; chronic prostatitis; glutathione-S-transferase; oxidative stress; malondialdehyde.

Introduction

Prostate gland diseases are an important problem in urology and andrology, since chronic prostatitis is one of the most common diseases in men of reproductive age (Roehrborn & McConnell, 2002; Ercan et al., 2019). Overall, chronic prostatitis and benign prostatic hyperplasia (BPH) are serious health problems, and the incidence of prostate-related diseases is expected to increase with longer life expectancy. BPH is the growth of both epithelial and stromal cells in the transitional zone and periurethral areas of the prostate. It affects mostly older men. The diagnosis of BPH is histological and requires tissue examination; its prevalence exceeds 70% in men aged 60 and over 90% in men aged 70. The histological presence of BPH is not necessarily accompanied by clinical symptoms (Roehrborn, 2008; Chughtai et al., 2011; Kim et al., 2016).

Chronic prostatitis often results from long-standing, recurring inflammation of the prostate. Inflammation of the prostate gland may be an important factor influencing its growth and symptom progression. Prostate stromal cells play a key role in triggering the inflammatory response by activating CD4+ lymphocytes. Although the molecular and stromal mechanisms involved in the pathogenesis of this metabolic disorder are not yet fully understood, it is believed that the etiology of hyperplasia is influenced by a number of factors, including aging, hormonal changes, metabolic syndrome, dietary factors, inflammation, oxidative stress, and, more recently, suppression of apoptosis in prostate tissue (Roehrborn & McConnell, 2002; Aleksandra et al., 2015; Ercan et al., 2019). Various growth factors and cytokines are involved in the inflammatory process. Both chronic and acute inflammation can lead to proliferation in prostate tissue through various mechanisms, including oxidative stress. Both tissue damage and oxidative stress can cause compensatory cell proliferation, resulting in hyperplastic growth (Chughtai et al., 2011).

Inflammation of the prostate gland can lead to the formation of free radicals, including nitric oxide (NO) and various reactive oxygen species. Both macrophages and neutrophils are sources of these free radicals, which can induce hyperplastic changes through oxidative stress that damages tissues and DNA. One of the characteristic features of such reactions is the release of arachidonic acid from cell membranes. This process is accompanied by the formation of new reactive oxygen species. These reactions can lead to the conversion of arachidonic acid into prostaglandins with the participation of cyclooxygenase enzymes. Prostaglandins are recognized as an important factor in the regulation of prostate cell proliferation (Sugar, 2006).

According to some authors, the development of benign prostatic hyperplasia is accompanied by a violation of the oxidative status due to an increase in MDA levels, depletion of GSH concentration, and a decrease in the activity of antioxidant enzymes. These results allow us to understand part of the etiology of BPH associated with oxidative stress (Zabaiou et al., 2016). To assess the state of oxidative stress in BPH, it was proposed to measure cytosolic levels of malondialdehyde (MDA), glutathione (GSH) and cytosolic enzymatic activity of superoxide dismutase, catalase, glutathione peroxidase, and glutathione-S-transferase (Zabaiou, 2016).

According to other data, glutathione-S-transferase (GsT) deserves the most attention, as it is overexpressed in patients with BPH

(Konwar et al., 2010). Normally, prostate tissue is protected against oxidative stress by antioxidant enzymes such as superoxide dismutase and glutathione-S-transferase (GsT)-P1 (Naber & Weidner, 2000). GsT (EC 2.5.1.18) is one of the important enzymes in the metabolism of xenobiotics. GsTs are divided into three classes according to their cellular localization: cytosolic, mitochondrial, and microsomal. Among them, cytosolic GsTs are probably the most studied and widely expressed in various cell types (Chatterjee & Gupta, 2018; Singh & Reindl, 2021).

Each class of GsTs is characterized by specific properties. However all GsTs isoforms perform common functions – they participate in the transport of hydrophobic compounds (such as aromatic hydrocarbons, porphyrins, steroid hormones) and ensure the detoxification of organic peroxides and xenobiotics (Strange et al., 2021). The mechanism of detoxification involves the nucleophilic addition of the SH group of glutathione to the electrophilic center of the toxic molecule. This results in the formation of a glutathione conjugate, which usually has lower toxicity and greater solubility in water, facilitating its removal from the cell and the body as a whole (Dann et al., 2004). The substrates for these enzymes are chemical carcinogens, certain endogenous compounds formed during oxidative stress, and various drugs. Experimental data suggest that increased expression of isoenzymes of this family may be one of the factors contributing to the resistance of cancer cells to antitumor drugs (Piaggi et al., 2010; Wang et al., 2015).

Studies have shown that electrophilic substances formed as a result of oxidative stress are associated with various diseases, such as cancer, Alzheimer's disease, Parkinson's disease, schizophrenia, diabetes, atherosclerosis, and diseases associated with aging (Grimsrud et al, 2007; Zimniak, 2008; Zhou et al., 2008; Butterfield et al., 2010). GsTs play an important role in the inactivation of these endogenous electrophiles, which means that GsT deficiency may increase the risk of these diseases. The development of BPH is accompanied by a disturbance in oxidative status, in particular due to an increase in MDA levels and changes in the activities of antioxidant enzymes (Zabaiou et al., 2016).

The aim of this study is to study the activity and kinetic characteristics of glutathione S-transferase in prostate biopsies from patients with benign hyperplasia and hyperplasia with chronic prostatitis.

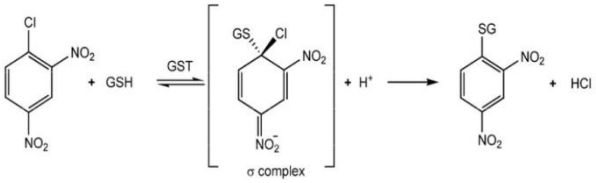
Materials and methods

Prostate tissue (biopsies) from two groups of patients undergoing treatment at the urology clinic of the Lviv regional clinical hospital were used. Group 1 – patients with BPH (n = 14); Group 2 – patients with benign prostatic hyperplasia and chronic prostatitis (n = 14). The age of patients ranged from 58 to 72, and the average age was 62.4 ± 5.2. The study was carried out in compliance with the principles of medical ethics and protection of patients' rights, human dignity and moral and ethical standards, in accordance with the principles of the Helsinki Declaration of Human Rights, the Council of Europe Convention on Human Rights and Biomedicine, and the legislation of Ukraine; permission of the Bioethics Committee of Danylo Halytskyi Lviv National Medical University. Measures were taken to ensure patient safety, respect for their rights, human dignity, and moral and ethical standards in accordance with the principles of the Helsinki Declaration on Human Rights, the Council of Europe Convention on Human Rights and Biomedicine, and the relevant laws of Ukraine.

The tissues were cooled in physiological solution, crushed and homogenized using an FSH-2A HTGH Speed Homogenizer at 4000 rpm, adding cooled to 0 ºC 50 mM Tris-HCl buffer (pH 7.4) containing 1 mM EDTA and 250 mM sucrose in a tissue-buffer mass ratio of 1:9. The supernatant obtained by centrifuging the diluted homogenate at 15 000 g for 15 min was used in the studies. The supernatant fraction, which was intended for further biochemical studies, was divided into parts, transferred to ultracentrifuge tubes, and immediately frozen in a freezer at a temperature of –30 ºC.

Lipid peroxidation was assessed by the concentration of malondialdehyde (MDA) in the supernatant fraction using the method (Nabil et al., 2008) and expressed to grams of tissue. The principle of the MDA determination method is that at high temperatures in an acidic environment, it reacts with 2-thiobarbituric acid, forming a colored trimethylene complex with a maximum absorption at λ = 532 nm (Nabil et al., 2008).

GsT activity was determined in the soluble fraction of the prostate gland. The total protein content in the samples was determined by the Lowry method using a kit manufactured by Simko Ltd (Ukraine). GsT activity was determined by the rate of enzymatic formation of the optically active dinitrobenzene (glutathione-S) conjugate in the reaction of glutathione reduction with 1-chloro-2,4-dinitrobenzene. The aqueous solution of the formed product has a maximum light absorption at a wavelength of 340 nm (Habig et al., 1974).



In a 10 mm cuvette with the test sample containing 2.5 mL of 0.1 M potassium phosphate buffer (pH 6.5), 0.03 mL of a 300 mM solution of reduced glutathione and 0.1 mL of a sample containing 50 μg of protein were added. To a cuvette with a control sample containing 2.5 mL of 0.1 M potassium phosphate buffer (pH 6.5), 0.03 mL of 300 mM reduced glutathione solution was added, and instead of the protein fraction, 0.1 mL of 0.1 M potassium phosphate buffer (pH 6.5) was added. The reaction was initiated by adding 0.2 mL of 0.015 M 1-chloro-2,4-dinitrobenzene to both cuvettes, and after 3 minutes, the optical density at a wavelength of 340 nm was measured for the test sample against the control. The enzymatic activity of GsT was calculated based on the extinction coefficient of the complex (9.6 $mM^{-1}cm^{-1}$) and expressed in μmol GSH per min per 1 mg of protein.

The kinetic parameters of the glutathione-S formation reaction (the maximum instantaneous reaction rate V_0, the maximum (plateau) amount of reaction product formation P_{max}, and the characteristic reaction time (half-saturation period)) were determined as described in the article (Kosterin & Karahim, 2020). The kinetic properties of GsT were studied in a standard incubation medium, which was modified according to the concentration of the substrate – GSH or 1-chloro-2,4-dinitrobenzene. The apparent kinetic parameters characterizing the reaction catalyzed by GsT (the apparent affinity constant for GSH or 1-chloro-2,4-dinitrobenzene and the maximum reaction rate) were determined in the Lineweaver-Burk coordinates {1/V on 1/S}, where S is the substrate concentration and V is the reaction rate at a given reagent concentration. The research results were processed using generally accepted methods of variational statistics with the use of ANOVA.

Results

Malondialdehyde (MDA) is considered the most informative marker of oxidative stress during the onset and progression of prostate hyperplasia, as elevated levels signal increased lipid peroxidation and impaired antioxidant defenses. It has been shown that the MDA level in patients with BPH with chronic prostatitis increases 1.52 times ($P < 0.05$) compared to patients without chronic prostatitis (Fig. 1). Inceased level of MDA indicates an intensification of oxidative processes.

It is known that GsTs are key detoxification systems that inactivate toxic products of lipid peroxidation. It has been established that GsT activity was 1.60 times higher ($P < 0.05$) in the soluble fraction of prostate biopsies from patients with BPH with chronic prostatitis than in patients without chronic prostatitis (Fig. 2).

In order to study the characteristics and mechanism of GsT functioning, the maximum instantaneous reaction rate (V_0), the maximum (plateau) amount of reaction product formation (P_{max}), and the characteristic reaction time (τ) were determined. To find these kinetic parameters of the GsT-catalyzed reaction, the dynamics of accumulation of the reaction product were studied. For this purpose, the soluble fraction of prostate biopsies was incubated in a standard incubation

medium for different periods of time (1–5 min). The experimental data showed that the kinetic curves of glutathione-S formation in the soluble fraction of the prostate gland of patients with BPH and hyperplasia with chronic prostatitis tend to saturate (Fig. 3).

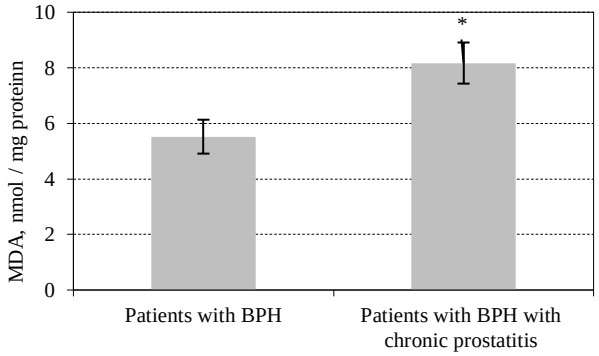


Fig. 1. MDA concentration in prostate biopsy tissue of patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

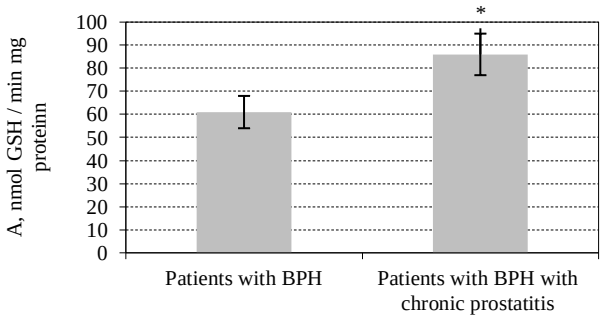


Fig. 2. GsT activity in the soluble fraction of prostate biopsies from patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

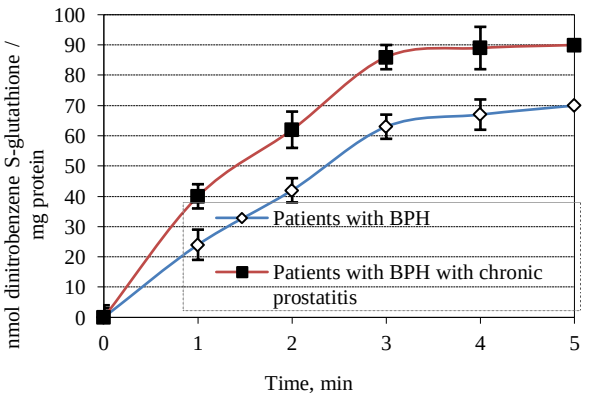


Fig. 3. Dynamics of glutathione-S accumulation in the soluble fraction of prostate biopsies from patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

Analysis of the results obtained allows us to conclude that the kinetics of the GsT reaction is consistent with the patterns of a zero-order reaction in the range of 0–3 min. In this time interval, the dependence graph of the reaction product formation on the incubation period is practically linear. Therefore the incubation time of homogenates and, accordingly, the enzyme reaction was 3 min in further experiments. As can be seen in Fig. 3, over the entire range of time, the amount of conjugated product formation in patients with chronic prostatitis is higher than in patients with hyperplasia without chronic prostatitis. By linearizing the obtained data in the coordinates {P/t on P}, the main kinetic characteristics of the accumulation of the optically active conjugate were calculated (Fig. 4).

As can be seen from the data in Table 1, the values of the kinetic parameters of accumulation of the optically active conjugate in the soluble fraction of the prostate gland of patients in both study groups differ significantly. In the absence of a significant difference in the value of P_{max} of accumulation of the optically active conjugate, we have shown that the value of V_0 in patients with BPH with chronic prostatitis is almost twice ($P < 0.01$) as high as this value for patients without chronic prostatitis. Based on these data, we assume that in the prostate gland of patients with an inflammatory process, the accumulation of optically active conjugate occurs more actively (the characteristic reaction time is 2.08 times ($P < 0.001$) shorter), but is characterized by the same capacity.

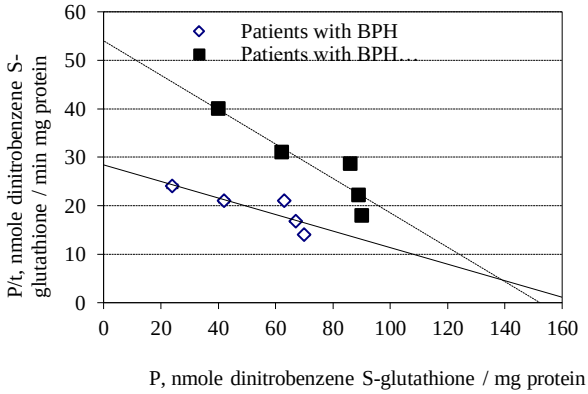


Fig. 4. Linearization of glutathione-S accumulation curves in prostate gland homogenates of patients with BPH and BPH with chronic prostatitis [P/t; P] (mean ± SD, n = 14; r > 0.9)

Table 1
Kinetic parameters of glutathione-S accumulation in the soluble fraction of the prostate gland in patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

Kinetic parameters	Patients with BPH	
	without chronic prostatitis	with chronic prostatitis
V_0, nmol glutathione-S / min per mg of protein	27.40 ± 0.42	54.25 ± 5.36 **
P_{max}, nmol glutathione–S / mg protein	167.12 ± 20.15	152.22 ± 18.44
τ, min	5.92 ± 0.54	2.85 ± 0.44 ***

Note: V_0 – maximum instantaneous reaction rate, P_{max} – maximum (plateau) amount of reaction product, τ – characteristic reaction time (half-life period); changes are significant compared to values in patients without chronic prostatitis ** – $P < 0.01$, *** – $P < 0.001$.

An important characteristic of GsT is the dependence of enzyme activity on the substrate concentration in the incubation medium, which is determined by the value of the apparent affinity constant for the substrate. The soluble fraction of the prostate gland was incubated in a medium containing reaction substrates – GSH in a concentration range from 5 to 300 mM and 1-chloro-2,4-dinitrobenzene from 50 to 200 mM.

An increase in the GSH content in the incubation medium (at a constant concentration of 1-chloro-2,4-dinitrobenzene) leads to a monotonic increase in GsT enzymatic activity with a plateau (Fig. 5). The enzyme activity in samples from patients with chronic prostatitis is higher over the entire range of GSH concentrations studied than in patients without inflammation. Similar changes are also observed with an increase in the concentration of 1-chloro-2,4-dinitrobenzene in the incubation medium at a constant GSH concentration (Fig. 6).

To elucidate the possible mechanism of change in GsT activity, the main kinetic parameters of accumulation of the optically active dinitrobenzene conjugate in the soluble fraction of the prostate gland were determined. By linearizing the obtained concentration dependencies in the Lineweaver-Burk coordinates, the main kinetic parameters of glutathione-S accumulation in the soluble fraction of the prostate gland of patients with BPH with chronic prostatitis were determined (Fig. 7 and 8). As can be seen from the presented curves, the

dependencies {1/V; 1/[GSH]} differ in the slope tangent and intersect the abscissa and ordinate axes at different points. This dependence corresponds to a mixed type of enzyme inhibition. A similar nature of inhibition was also found for 1-chloro-2,4-dinitrobenzene. The obtained values of the apparent affinity constant for GSH and 1-chloro-2,4-dinitrobenzene are in the millimolar concentration range, which is consistent with the data of other researchers.

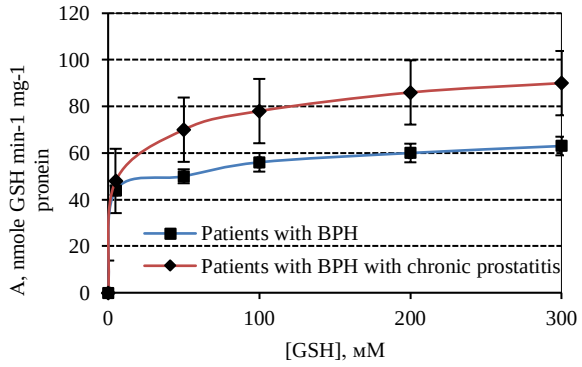


Fig. 5. Concentration dependence of GSH on GsT activity in the soluble fraction of the prostate gland of patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

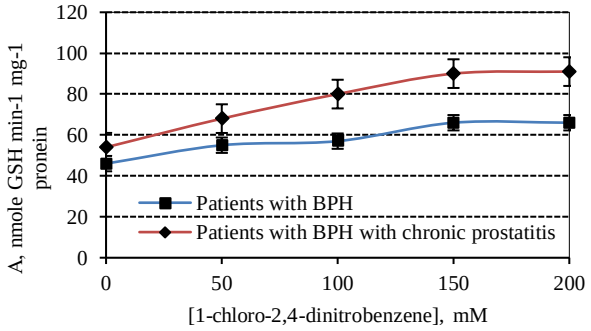


Fig. 6. Concentration dependence of 1-chloro-2,4-dinitrobenzene on GsT activity in the supernatant fraction of the prostate gland of patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

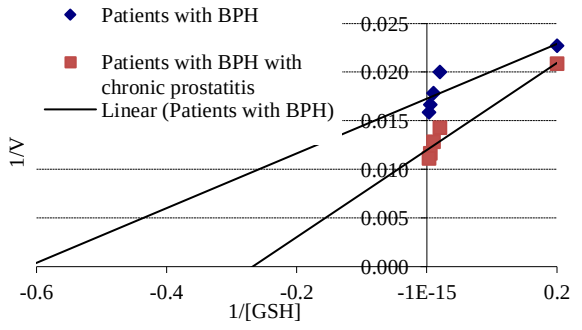


Fig. 7. Linearization of the concentration curves shown in Figure 5 in the Lineweaver-Burk coordinates, where V is GsT activity (n = 14; r > 0.95)

It was shown that the maximum reaction rate of accumulation of the optically active dinitrobenzene conjugate (determined by GSH) is 1.44 times ($P < 0.05$) higher in patients with chronic prostatitis compared to the first group. The affinity constant for GSH in this group of patients is 2.28 times ($P < 0.001$) higher compared to patients without inflammation. When interpreting the obtained kinetic parameters determined by GSH, it is shown that GsT activity in patients with BPH without chronic prostatitis is reduced both due to a decrease in the enzyme reaction rate (V_{max} decreases) and due to a increase in the enzyme's affinity for GSH (KGSH decreases). Similarly, the maximum reaction rate of accumulation of the optically active dinitrobenzene conjugate (determined by 1-chloro-2,4-dinitrobenzene) is 1.52 times ($P < 0.05$) higher in patients with chronic prostatitis compared to the first group. The affinity constant for 1-chloro-2,4-dinitrobenzene in patients with chronic prostatitis is 5.42 times ($P < 0.001$) higher than in patients without inflammation.

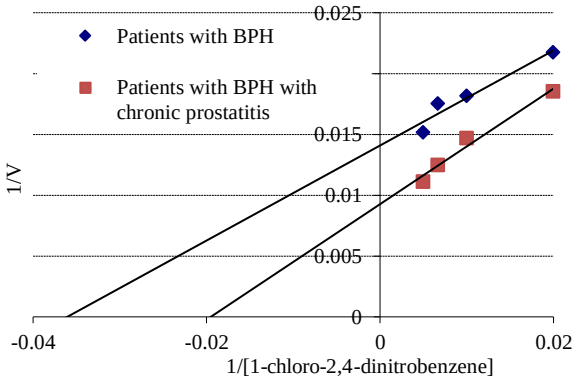


Fig. 8. Linearization of concentration curves shown in Figure 6 in Lineweaver-Burk coordinates, where V is GsT activity (n = 14; r > 0.95)

Table 2
Kinetic properties of GsT in the soluble fraction of the prostate gland in patients with BPH and BPH with chronic prostatitis (mean ± SD, n = 14)

Kinetic parameters	Patients with BPH	
	without chronic prostatitis	with chronic prostatitis
V_{max}, nmol GSH/min per mg protein (according to GSH)	58.5 ± 5.3	84.1 ± 8.3*
K_{GSH}, mM	1.62 ± 0.16	3.70 ± 0.42***
V_{max}, nmol GSH/min per mg protein (by 1-chloro-2,4- dinitrobenzene)	70.9 ± 8.3	107.5 ± 9.3*
K1-chloro-2,4-dinitrobenzene, mM	9.5 ± 1.2	51.6 ± 5.4***

Note: V_{max} – initial maximum enzyme activity, KGSH, K – 1-chloro-2,4-dinitrobenzene – Michaelis constant for substrates; changes are significant compared to the values in the control group * – $P < 0.05$, ** – $P < 0.01$, *** – $P < 0.001$.

Discussion

The increased level of MDA is main marker of the oxidative stress in prostate hyperplasia (Zabaiou et al., 2016; Ercan et al., 2019). The biological role of GsT is determined by its ability to detoxify a wide range of endogenous and exogenous toxic compounds by conjugating them with glutathione (Lv et al., 2023). In our previous studies it was shown that sperm GsTs play key role in susceptibility of spermatozoa to oxidative damage and maintaining sperm antioxidant status (Vorobets et al., 2018; Fafula et al., 2019). In present study we studied the the activity and kinetic characteristics of glutathione S-transferase in prostate biopsies from patients with benign hyperplasia and hyperplasia with chronic prostatitis.

Glutathione S-transferase catalyzes the conjugation of reduced glutathione with 1-chloro-2,4-dinitrobenzene (CDNB), which is a universal model substrate for most GsT isoforms. GsTs are known to catalyze the nucleophilic addition of glutathione (non-protein thiol, GSH, tripeptide γ-Glu-Cys-Gly) to electrophilic molecules of various toxic substrates, including xenobiotics (RX + GSH → RSG + HX). This process is carried out using hydrogen from reduced glutathione, to which this enzyme has a high affinity. Given this, changes in the concentration of GSH in the incubation medium likely affect the rate of the reaction catalyzed by GST. Dinitrophenols have electrophilic centers that make the molecule susceptible to nucleophilic attack. GST promotes the nucleophilic addition of GSH to the aromatic ring of dinitrophenol. Since dinitro groups are strong electron acceptors, the ring becomes activated for attack by GSH thiolate. This reaction results in

the formation of a glutathione conjugate of dinitrophenol (the molecule becomes less reactive and is more easily excreted) (Lv et al., 2023).

Hyperplastic changes in the prostate gland are associated with increased activity of glutathione metabolism enzymes, in particular GsT. Increased GsT activity is considered a protective mechanism aimed at neutralizing oxidants and lipid peroxidation products that accumulate under conditions of structural and functional tissue changes. The obtained results, which indicate an increase in GsT activity in the prostate tissue in hyperplasia, are consistent with previous studies. However, the nature and degree of activation of different GsT isoforms may depend on the stage of the pathological process, hormonal status, and individual genetic characteristics, which requires further study (Bostwick et al., 2007; Zabaiou et al., 2016).

In this regard, GsT deserves the most attention, as it is overexpressed in patients with BPH (Konwar et al., 2010). On the other hand, there is evidence that the development of BHP is accompanied by a disturbance in oxidative status due to an increase in MDA levels, depletion of GSH concentration, and a decrease in the activity of all antioxidant enzymes studied. According to the authors, these results provide insight into the etiology of benign prostatic hyperplasia associated with oxidative stress (Zabaiou et al., 2016).

A growing number of studies link the diverse biological activity of GsT to various diseases. GsT has been shown to be overexpressed in many tumor tissues. High expression of GsT mediates cellular resistance to anticancer drugs through various mechanisms, which mainly include metabolic detoxification, MAPK signaling pathway regulation, DNA repair, autophagy, and glycolytic processes. In addition, a recent study has shown that high GsT expression in lung fibroblasts promotes the progression of pulmonary fibrosis by catalyzing S-glutathionylation of proteins in lung fibroblasts (Lv et al., 2023).

Conclusion

It has been found that in patients with benign prostatic hyperplasia with chronic prostatitis, the accumulation of optically active conjugate occurs more actively compared to patients without chronic prostatitis, but is characterized by the same capacity. When interpreting the obtained kinetic parameters determined by GSH, it is shown that GsT activity in patients with benign prostatic hyperplasia without chronic prostatitis is reduced both due to a decrease in the enzyme reaction rate (V_{max} decreases) and due to a increase in the enzyme's affinity for GSH (KGSH decreases).

No potential conflicts of interest relevant to this article were reported.

References

Aleksandra, R., Iwona, R., Tomasz, M., Marcin, S., Barbara, D., Anna, L., & Laszczyńska, M. (2015). Metabolic syndrome and benign prostatic hyperplasia: Association or coincidence? Diabetology and Metabolic Syndrome, 7, 94.

Alnasser, S. M. (2024). The role of glutathione S-transferases in human disease pathogenesis and their current inhibitors. Genes and Diseases, 12(4), 101482.

Bathige, S. D., Umasuthan, N., Saranya Revathy, K., Lee, Y., Kim, S., Cho, M. Y., Park, M. A., Whang, I., & Lee, J. (2014). A mu class glutathione S-transferase from Manila clam *Ruditapes philippinarum* (RpGSTμ): Cloning, mRNA expression, and conjugation assays. Comparative Biochemistry and Physiology C: Toxicology and Pharmacology, 162, 85–95.

Bostwick, D. G., Meiers, I., & Shanks, J. H. (2007). Glutathione S-transferase: Differential expression of alpha, mu, and pi isoenzymes in benign prostate, prostatic intraepithelial neoplasia, and prostatic adenocarcinoma. Human Pathology, 38(9), 1394–1401.

Butterfield, D. A., Bader Lange, M. L., & Sultana, R. (2010). Involvements of the lipid peroxidation product HNE in the pathogenesis and progression of Alzheimer's disease. Biochimica et Biophysica Acta, 1801, 924–929.

Chatterjee, A., & Gupta, S. (2018). The multifaceted role of glutathione S-transferases in cancer. Cancer Letters, 433, 33–42.

Chughtai, B., Lee, R., Te, A., & Kaplan, S. (2011). Role of inflammation in benign prostatic hyperplasia. Reviews in Urology, 13(3), 147–150.

Dann, A. T., Kenyon, A. P., Seed, P. T., Poston, L., Shennan, A. H., & Tribe, R. M. (2004). Glutathione S-transferase and liver function in intrahepatic cholestasis of pregnancy and pruritus gravidarum. Hepatology, 40(6), 1406–1414.

Ercan, M., Alp, H. H., Kocaturk, H., Bakan, N., & Gul, M. (2019). Oxidative stress before and after surgery in benign prostatic hyperplasia patients. Andrologia, 51, e13326.

Fafula, R. V., Paranyak, N. M., Besedina, A. S., Vorobets, D. Z., Iefremova, U. P., Onufrovych, O. K., & Vorobets, Z. D. (2019). Biological significance of glutathione S-transferases in human sperm cells. Journal of Human Reproductive Sciences, 12(1), 24–28.

Grimsrud, P. A., Picklo, M. J., Griffin, T. J., & Bernlohr, D. A. (2007). Carbonylation of adipose proteins in obesity and insulin resistance: Identification of adipocyte fatty acid-binding protein as a cellular target of 4-hydroxynonenal. Molecular and Cellular Proteomics, 6, 624–637.

Habig, H. W., Pabst, M. J., & Jacoby, W. B. (1974). Glutathione S-transferases. Journal of Biological Chemistry, 249, 7130–7139.

Kim, E. H., Larson, J. A., & Andriole, G. L. (2016). Management of benign prostatic hyperplasia. Annual Review of Medicine, 67, 137–151.

Konwar, R., Manchanda, P. K., Chaudhary, P., Nayak, V. L., Singh, V., & Bid, H. K. (2010). Glutathione S-transferase gene variants and risk of benign prostate hyperplasia in a North Indian population. Asian Pacific Journal of Cancer Prevention, 11(2), 365–370.

Kosterin, S. O., & Karakhim, S. O. (2020). Biochemical kinetics. Naukova Dumka, Kyiv.

Lv, N., Huang, C., Huang, H., Dong, Z., Chen, X., Lu, C., & Zhang, Y. (2023). Overexpression of glutathione S-transferases in human diseases: Drug targets and therapeutic implications. Antioxidants, 12(11), 1970.

Mayer, R. J., & Ofial, A. R. (2019). Nucleophilicity of glutathione: A link to Michael acceptor reactivities. Angewandte Chemie International Edition, 58(49), 17704–17708.

Mazari, A. M. A., Zhang, L., Ye, Z.-W., Zhang, J., Tew, K. D., & Townsend, D. M. (2023). The multifaceted role of gluta-thione S-transferases in health and disease. Biomolecules, 13(4), 688.

Naber, K. G., & Weidner, W. (2000). Chronic prostatitis – an infectious disease? Journal of Antimicrobial Chemotherapy, 46(2), 157–161.

Piaggi, S., Raggi, C., Corti, A., Pitzalis, E., Mascherpa, M. C., Saviozzi, M., Pompella, A., & Casini, A. F. (2010). Glutathione transferase omega 1-1 (GSTO1-1) plays an anti-apoptotic role in cell resistance to cisplatin toxicity. Carcinogenesis, 31(5), 804–811.

Roehrborn, C. G. (2008). BPH progression: Concept and key learning from MTOPS, ALTESS, COMBAT, and ALF-ONE. BJU International, 101(S3), 17–21.

Roehrborn, C., & McConnell, J. (2002). Etiology, pathophysiology, epidemiology and natural history of benign prostatic hyperplasia. In: Walsh, P. et al. (Eds.). Campbell's Urology. 8th ed. Saunders, Philadelphia. Pp. 1297–1336.

Samiec, P. S., Dahm, L. J., & Jones, D. P. (2000). Glutathione S-transferase in mucus of rat small intestine. Toxicological Sciences, 54(1), 52–59.

Singh, R. R., & Reindl, K. M. (2021). Glutathione S-transferases in cancer. Antioxidants, 10, 701.

Strange, R. C., Spiteri, M. A., Ramachandran, S., & Fryer, A. A. (2001). Glutathione-S-transferase family of enzymes. Mutation Research, 482(1–2), 21–36.

Sugar, L. M. (2006). Inflammation and prostate cancer. Canadian Journal of Urology, 13(S1), 46–47.

Vorobets, M. Z., Fafula, R. V., Besedina, A. S., Onufrovych, O. K., & Vorobets, D. Z. (2018). Glutathione S-transferase as a marker of oxidative stress in human ejaculated spermatozoa from patients with pathospermia. Regulatory Mechanisms in Biosystems, 9(2), 287–292.

Wang, Z., Liang, S., Lian, X., Liu, L., Zhao, S., Xuan, Q., Guo, L., Liu, H., Yang, Y., Dong, T., Cao, J., & Liu, S. (2015). Identification of proteins responsible for adriamycin resistance in breast cancer cells using proteomics analysis. Scientific Reports, 5, 9301.

Zabaiou, N., Mabed, D., Lobaccaro, J. M., & Lahouel, M. (2016). Oxidative stress in benign prostate hyperplasia. Andrologia, 48(1), 69–73.

Zhou, C., Huang, Y., & Przedborski, S. (2008). Oxidative stress in Parkinson's disease: A mechanism of pathogenic and therapeutic significance. Annals of the New York Academy of Sciences, 1147, 93–104.

Zimniak, P. (2008). Detoxification reactions: Relevance to aging. Ageing Research Reviews, 7(4), 281–300.