UDC 618.3:618.14-006.36-002.4

DOI: 10.20333/25000136-2024-4-18-22

Catalases: oxidative stress, inflammation and carcinogenesis

E. Yu. Sergeeva, S. S. Dunaevskaya, V. V. Deulina, Yu. A. Fefelova

Prof. V. F. Voino-Yasenetsky Krasnoyarsk State Medical University, Krasnoyarsk 660022, Russian Federation

Abstract. Acute pancreatitis is a severe disease of the pancreas, the number of cases of which increases by 3.4% annually. Both acute and chronic pancreatitis can become risk factors for the development of such a malignant neoplasm of the pancreas as ductal adenocarcinoma of the pancreas, although the degree of possible danger of malignant neoplasm development in different researches varies. Search for studies was conducted using electronic databases from Google Scholar, Medline and PubMed. A total of 215 articles were reviewed and 50 articles were deemed relevant according to the inclusion criteria. Among the genes the activity of which may be modulated during the development of a malignant tumour of the pancreas is the catalase gene. Catalase is an antioxidant protection enzyme that plays a key role in ROS inactivation and prevention of oxidative stress, one of the most important pathogenetic factors in acute pancreatitis.

Key words: acute pancreatitis, catalase, oxidative stress, carcinogenesis, chronic pancreatitis, pancreas.

Conflict of interest. The authors declare the absence of obvious and potential conflicts of interest associated with the publication of this article.

Citation: Sergeeva EYu, Dunaevskaya SS, Deulina VV, Fefelova YuA. Catalases: oxidative stress, inflammation and carcinogenesis. *Siberian Medical Review*. 2024;(4):18-22. DOI: 10.20333/25000136-2024-4-18-22

Introduction

Acute pancreatitis is a severe disease of the pancreas, with an annual increase of 3.4 % [1]. After the first episode of acute pancreatitis (AP), at least 17 % of patients have a second episode of the disease, and 8 % develop chronic pancreatitis (CP) [2]. Both acute and chronic pancreatitis may be risk factors for the development of pancreatic malignancies such as pancreatic ductal adenocarcinoma (PDAC), although the degree of the possible risk of malignant neoplasm varies among different researchers [3]. There are many hypotheses explaining the pathogenetic relationship between acute pancreatitis and the development of PDAC. One of the hypotheses is based on the fact that repeated episodes of AP increase inflammation in the pancreas, which increases the risk of oncogenic mutations in the cells of this organ [4]. There is an opinion that long-term inflammation in the pancreas leads to mutations associated with downregulation of tumour suppressors, including p16, p53 and SMAD4, and upregulation of tumour promoters, in particular, K-ras, TNF-α, NF-κB genes [5-7]. Among the genes the activity of which may be modulated during the development of a malignant tumour of the pancreas is the catalase gene. Catalase is an antioxidant defence enzyme that plays a key role in ROS inactivation and prevention of oxidative stress, one of the most important pathogenetic factors in AP. Catalase converts two hydrogen peroxide molecules into one oxygen molecule and two water molecules in a two-step reaction. The gene encoding catalase, CAT, is located on chromosome 11 [8]. This review focuses on the relationship between oxidative stress, inflammation and carcinogenesis, as well as the role of catalase in the development of these processes.

Oxidative stress and inflammation

Oxidative stress results from an imbalance between the production and inactivation of particles with increased reactivity, which can be either free radicals, such as hydroxyl radical or superoxide radical, or non-free radicals, such as hydrogen peroxide. These particles can react with other molecules, oxidising them or subjecting them to other modifications [9]. Free radicals are generated in the cell during such metabolic processes as the functioning of the mitochondrial respiratory chain, β-oxidation of fatty acids, cytochrome P-450-mediated reactions, and respiratory burst during phagocytosis [10]. In AP, the cause of increased production of free radicals may be the aetiological factors of the disease, such as dietary habits, xenobiotics, bad habits. In particular, the role of smoking, one of the risk factors for AP, in increasing the activity of particles with increased reactivity has been proven [11, 12]. During inflammation, neutrophils and monocytes rush to the site of damage, transforming into macrophages. In the process of phagocytosis, neutrophils and macrophages produce ROS necessary for the effective destruction of phlogogens but are also capable of damaging macromolecules [13, 14]. When cells are damaged by oxidative damage, arachidonic acid is released from the cell membrane [15], which is extremely important for the induction of an inflammatory response, since cyclooxygenases and lipoxygenase convert arachidonic acid into inflammatory mediators, prostaglandins and leukotrienes. Inflammatory mediators contribute to a further increase in migration of neutrophils and macrophages to the area of inflammation, the release of proinflammatory cytokines, including TNF-alpha, IL-1, IL-8, the process becomes cyclic, accompanied by an increase in the levels of cytokines and chemokines, in addition, the transcription factor NF-κB is activated, resulting in increased transcription of pro-inflammatory and anti-apoptotic genes [16].

Thus, mediated ROS occurs, often associated with changes in the activity of NF-κB, STAT3, hypoxia-inducible factor-1α, kinases, growth factors, cytokines, and a number of other regulatory proteins, enzymes, up- or down-regulation of various signalling pathways, which leads to changes in cellular processes associated with inflammation [17, 18]. However, the process of inflammation itself is also accompanied by an increase in the production of molecules with increased reactivity, both

reactive oxygen species and reactive nitrogen species, which promotes oxidative stress and closes the vicious circle of pathogenesis [19, 20].

One of the factors that play an important role in the initiation of chronic inflammation is obesity. It has been shown that a decrease in catalase activity leads to lipid metabolism disorders and contributes to obesity [21]. Blockade of catalase activity, accompanied by additional effects, in particular, starvation, leads to the activation of cell death and an increase in the production of proinflammatory cytokines in mouse liver cells, induced by an increase in the level of ROS, which was accompanied by an increase in the levels of aspartate aminotransferase and alanine aminotransferase, markers of hepatocyte cytolysis [22].

However, Han et al. showed that upregulation of mitochondrial catalase leads to increased NF-κB activation, more severe inflammation, and more severe lung tissue damage in a laboratory mouse model. It has been suggested that the mediation of this hyperinflammatory response is associated with lymphocytes and macrophages with altered metabolism, which is characterised by an increase in glycolysis and an increase in ATP synthesis. Increased levels of NADH in macrophages leads to up-regulation of proinflammatory factors [23]. In addition, Mu et al. found that inhibition of catalase by 3-aminotriazole leads to increased production of ROS in peroxisomes. This results in the formation of a 4HNE-IκBα adduct that disrupts NF-κB activation by lipopolysaccharides, which leads to blockade of the production of pro-inflammatory factors and, as a result, down-regulation of inflammation [24].

It has been established that the use of an agonist of the peroxisome proliferator-activated receptor alpha (PPRα), which is a transcription factor involved in the regulation of fatty acid oxidation, in mice with alcoholic fatty liver disease, which is often combined with pancreatitis, leads to the induction of catalase activity and a decrease in the manifestations of the disease [25].

It has been shown that in severe alcoholic hepatitis there is a significant decrease in the levels of catalase and PPRα. Reactivation of the peroxisome proliferator-activated alpha receptor led to a decrease in alcohol-induced inflammation while switching the CYP2E1-dependent alcohol metabolism pathway to the CAT-dependent one. Thus, reactivation of PPRα leads to upregulation of the PPRα-CAT signalling pathway, which plays an important role in the pathogenetic correction of inflammation in alcoholic hepatitis [26].

High-mobility group box protein 1 (HMGB1) is a non-histone nuclear protein that regulates transcription and is involved in maintaining the structure of chromosomes. HMGB1 can function as a chemokine or alarmin, activating the immune system and mediating a wide range of physiological and pathophysiological processes. HMGB1 is able to interact with the glycation end products receptor and members of the Toll-like receptor family, including TLR2 and TLR4. Activation of these receptors activates the transcription factor NF-κB, which in turn leads to an increase in the production of pro-inflammatory cytokines. Zhang et al. found that when rats are kept on a diet with a high level of lipids, there is an increase in HMGB1-positive cells and Kupffer cells in the liver, which play an important role in initiating inflammation, which is accompanied by a pronounced decrease in catalase activity and an increase in the levels of glucose and cholesterol in blood plasma [27].

An important role in the pathogenesis of inflammation initiated by the respiratory syncytial virus is played by a decrease in the activity of enzymes of the antioxidant system, catalase in particular, which is due to increased degradation of the NRF2 transcription factor that controls the expression of antioxidant enzyme genes including catalase. It has been established that the intake of exogenous catalase in respiratory syncytial virus in a laboratory mouse model leads to an improvement in the condition of animals, a decrease in inflammation and airway obstruction, and a decrease in the concentration of pro-inflammatory cytokines and chemokines, including IL-1, TNF-α, IL-9, CXCL1, CCL2, CCL5 in bronchoalveolar lavage fluid [28].

Chronic inflammation which plays a key role in the pathogenesis of Crohn's disease is associated with the development of oxidative stress and imbalance of antioxidant enzymes. It is known that reduced activity of catalase is one of the features of this disease. It has been established that patients with Crohn's disease have a number of catalase gene polymorphisms associated with impaired activity of this enzyme [29].

It has been shown that in sepsis, severe kidney damage is associated with activation of the pleckstrin homology domain, which modulates the JNK/ERK signalling pathway, with a decrease in the activity of the antioxidant system, a decrease in catalase activity in particular [30].

Therefore, changes in catalase activity can play one of the most important roles in diseases whose pathogenetic basis is the development of oxidative stress and inflammation.

Oxidative stress and carcinogenesis

It is known that oxidative stress can initiate both cell death through apoptosis and the appearance of senescent (aging) cells. Senescence is a state of cells in which the cell cycle is blocked, but cell death does not occur [31]. The variant implemented – whether the cell undergoes death by apoptosis, or becomes senescent – depends on the severity of damage and on the cell type. However, both variants are a way to prevent proliferation of damaged cells and reduce the likelihood of passing the mutation of genetically unstable cells to further cell generations [32].

If a cell manages to avoid apoptosis or senescence, the excess amount of ROS inherent in oxidative stress continues to damage its targets, such macromolecules as proteins, lipids, and nucleic acids [33]. Genetic instability of cells is one of the main features inherent in the process of carcinogenesis; therefore, DNA-damaging ROS are of great importance for initiating the development of malignant neoplasms [34]. It has been shown that inhibition of catalase activity in neutrophils leads to increased death of

these cells, which is based on the accumulation of ROS and modulation of the NOX2 signalling pathway [35]. It has been established that catalase can take part in the reprogramming of tumour-associated macrophages and reduce the level of hypoxia in the tumour microenvironment, which creates the prerequisites for new approaches to the therapy of malignant neoplasms [36].

A number of genetic polymorphisms of the catalase gene, the CAT C262T variant in particular, predisposes to the development of malignant neoplasms. It can be assumed that the presence of such polymorphisms is associated with less effective inactivation of molecules with increased reactivity, which leads to DNA damage and is a trigger of carcinogenesis [37].

Interestingly, according to the results of studies by a number of authors, an increase in catalase activity in tumour cells or in cells of target organs, which reduces the level of oxidative stress, may enhance the process of metastasis [38].

It has been shown that catalase expression is reduced in prostate cancer cells (PNG), which is associated with down-regulation of the FOXO signalling pathway that plays an important role in modulating a number of cellular processes, including cell cycle arrest, cell death, and DNA repair processes [39]. Oxidative stress contributes to the progression of androgen-dependent or androgen-independent PID [40].

It has been found that an increase in catalase activity in a preclinical model of metastasis of malignant neoplasms of the mammary gland to the lungs in obesity led to inhibition of the formation of metastases in the target organ. Obesity contributes to an increase in the concentration of neutrophils in the lung tissue. An increased content of neutrophils leads to increased production of ROS by these cells, which, in turn, disrupts contacts between endotheliocytes and promotes metastasis to the lungs [41].

The NF-κB/Catalase/ATF3 signalling pathway may play an important role in the regulation of cell proliferation in oesophageal malignancies. It has been shown that inhibition of neddylation, the process of post-translational modification of proteins, during which the interaction between the ubiquitin-like protein NEDD8 and the target protein occurs, leads to autophagy of malignant cells, while the activation of the transcription factor NF-κB and the expression of catalase are blocked [42].

Gene expression profiling in acute myelogenous leukaemia cells led to the conclusion that an increase in catalase gene expression is associated with an increase in the effectiveness of treatment and a longer remission period for patients [43].

It has been established that FOXO3a, a member of the family of transcription factors involved in the induction of such cellular processes as apoptosis, proliferation, cell cycle progression, DNA damage, and carcinogenesis, is involved in the regulation of catalase activity in prostate cancer cells. Downregulation of FOXO3a in tumour cells leads to the suppression of catalase activity, which in turn promotes the progression of malignancy through the modulation of the activity of the FOXO3a-ROS signalling pathway [44].

An increase in the activity of catalase and other antioxidant enzymes can play an important role in approaches to the treatment of malignant neoplasms of the cervix associated with infection with oncogenic types of human papillomavirus, which is due to the development of chronic inflammation and an increase in the production of reactive oxygen and nitrogen species as a result of the introduction of the virus into the cell genome and expression oncoproteins, which occurs against the background of a decrease in the activity of antioxidant defence [45].

However, Bengtson et al. have shown that inhibition of catalase activity can activate proapoptotic signalling pathways triggered by reactive oxygen and nitrogen species resulting in initiation of tumour cell death [46].

Patients with colorectal malignant neoplasms showed a significant decrease in catalase activity, which reflected the severity of oxidative stress and directly correlated with the severity of the metastasis process [47].

CCAAT/enhancer-binding protein delta (BSBP) is a transcription factor that plays an ambiguous role in inflammation and carcinogenesis. There is evidence that it can act as an onco-suppressor. However, it has been found that by regulating the activity of a number of genes associated with the control of redox balance, it can contribute to the progression of glioblastoma. It has been shown that this effect is based on the modulation of catalase activity in tumour cells, which leads to blockade of apoptosis and an increase in the survival of malignant neoplasm cells [48].

It has been shown that an oestradiol analogue inhibits catalase activity by modulating the Akt/FOXO signalling pathway, which leads to accumulation of ROS in breast cancer cells resulting in cell cycle blockade in the G1 and G2/M phases and initiation of tumour cell death [49].

It has been established that the single nucleotide polymorphism rs1001179 of the catalase gene promoter is associated with an increase in the activity of this enzyme, which leads to a decrease in ROS levels in chronic lymphocytic leukaemia cells and is the cause of a more severe course and progression of the disease due to blockade of apoptosis induction [50].

Thus, oxidative stress and an increase in catalase activity during carcinogenesis can play both a procarcinogenic role and prevent the development of malignant tumours.

Conclusion

Thus, there is no doubt that one of the key antioxidant enzymes, catalase, is involved in the modulation of such important typical pathological processes as inflammation and carcinogenesis, but its role is ambiguous. Further studies are needed to understand the mechanisms of inflammation and carcinogenesis associated with changes in catalase activity better, which can help create new effective approaches to personalised treatment of diseases.

Funding

The article was written with financial support of the RSF within the framework of scientific project № 22-25-20067 and the Krasnoyarsk Regional Fund of Science and Technology Support within the framework of project № 26.

References

1. Roberts SE, Morrison-Rees S, John A, Williams JG, Brown TH, Samuel DG. The incidence and aetiology of acute pancreatitis across Europe. *Pancreatology*. 2017;(17):155–165.

2. Ahmed Ali U, Issa Y, Hagenaars JC, Bakker OJ, van Goor H, Nieuwenhuijs VB, Bollen TL, van Ramshorst B, Witteman BJ, Brink MA, Schaapherder AF, Dejong CH, Spanier BW, Heisterkamp J, van der Harst E, van Eijck CH, Besselink MG, Gooszen HG, van Santvoort HC, Boermeester MA Dutch Pancreatitis Study Group. Risk of Recurrent Pancreatitis and Progression to Chronic Pancreatitis After a First Episode of Acute Pancreatitis. *Clinical Gastroenterology* and *Hepatology*. 2016;(14):738–746.

3. Liu J, Wang Y, Yu Y. Meta-analysis reveals an association between acute pancreatitis and the risk of pancreatic cancer. *World Journal* of *Clinical Cases*. 2020;(8):4416–4430.

4. Kolodecik T, Shugrue C, Ashat M, Thrower EC. Risk factors for pancreatic cancer: underlying mechanisms and potential targets. *Frontiers in Physiology*. 2013;(4):415.

5. Kong X, Sun T, Kong F, Du Y, Li Z. Chronic Pancreatitis and Pancreatic Cancer. *Gastrointestinal Tumors*. 2014;(1):123–134.

6. Olakowski M, Bułdak Ł. Modifiable and Non-Modifiable Risk Factors for the Development of Non-Hereditary Pancreatic Cancer. *Medicina (Kaunas)*. 2022;58(8):978.

7. Alhobayb T, Peravali R, Ashkar M. The Relationship between Acute and Chronic Pancreatitis with Pancreatic Adenocarcinoma: Review. *Diseases*. 2021;9(4):93.

8. Nandi A, Yan LJ, Jana CK, Das N. Role of Catalase in Oxidative Stress- and Age-Associated Degenerative Diseases. *Oxidative Medicine and Cellular Longevity*. 2019;(2019): 9613090.

9. Uchida K. 4-Hydroxy-2-nonenal: a product and mediator of oxidative stress. *Progress in Lipid Research*. 2003;42(4):318–343.

10. Caliri AW, Tommasi S, Besaratinia A. Relationships among smoking, oxidative stress, inflammation, macromolecular damage, and cancer. *Mutation Research – Reviews in Mutation Research*. 2021;(787):108365.

11. Dickinson BC, Chang CJ, Chemistry and biology of reactive oxygen species in signaling or stress responses. *Nature chemical biology*. 2011; (7):504–511.

12. Gorgoulis V, Adams PD, Alimonti A, Bennett DC, Bischof O, Bishop C, Campisi J, Collado M, Evangelou K, Ferbeyre G, Gil J, Hara E, Krizhanovsky V, Jurk D, Maier AB, Narita M, Niedernhofer L, Passos JF, Robbins PD, Schmitt CA, Sedivy J, Vougas K, von Zglinicki T, Zhou D, Serrano M, Demaria M. Cellular Senescence: Defining a Path Forward. *Cell*. 2019;(179):813–827.

13. Galadari S, Rahman A, Pallichankandy S, Thayyullathil F. Reactive oxygen species and cancer paradox: To promote or to suppress? *Free Radical Biology and Medicine*. 2017;(104):144–164.

14. Helfinger V, Schröder K. Redox control in cancer development and progression. *Molecular aspects of medicine*. 2018;(63):88–98.

15. Saikolappan S, Kumar B, Shishodia G, Koul S, Koul HK. Reactive oxygen species and cancer: A complex interaction. *Cancer letters*. 2019;(452):132–143.

16. Kay J, Thadhani E, Samson L, Engelward B. Inflammation-induced DNA damage, mutations and cancer. *DNA Repair*. 2019; (83):102673.

17. Kawanishi S, Ohnishi S, Ma N, Hiraku Y, Murata M. Crosstalk between DNA Damage and Inflammation in the Multiple Steps of Carcinogenesis. *International Journal of Molecular Sciences*. 2017;(18) :1808.

18. Leuti A, Maccarrone M, Chiurchiù V, Proresolving Lipid Mediators: Endogenous Modulators of Oxidative Stress. *Oxidative Medicine and Cellular Longevity*. 2019;(8):107265.

19. Tallima H, El Ridi R. Arachidonic acid: Physiological roles and potential health benefits -A review. *Journal of Advanced Research*. 2018; (11):33–41.

20. Prasad S, Gupta SC, Tyagi AK. Reactive oxygen species (ROS) and cancer: Role of antioxidative nutraceuticals. *Cancer Letters*. 2017;(387): 95–105.

21. Todoric J, Antonucci L, Karin M. Targeting Inflammation in Cancer Prevention and erapy. *Cancer Prevention Research (Phila)*. 2016;(9):895–905.

22. Alnajjar KS, Sweasy JB. A new perspective on oxidation of DNA repair proteins and cancer. *DNA Repair*. 2019;(76):60–69.

23. Kay J, Thadhani E, Samson L, Engelward B. Inflammation-induced DNA damage, mutations and cancer. *DNA Repair*. 2019;(83):102673.

24. Nitta Y, Muraoka-Hirayama S, Sakurai K. Catalase is required for peroxisome maintenance during adipogenesis. *Biochimica et Biophysica Acta – Molecular* and *Cell Biology* of *Lipids*. 2020;1865(8):158726.

25. Dutta RK, Maharjan Y, Lee JN, Park C, Ho YS, Park R. Catalase deficiency induces reactive oxygen species mediated pexophagy and cell death in the liver during prolonged fasting. *Biofactors*. 2021;47(1):112-125.

26. Han W, Fessel JP, Sherrill T, Kocurek EG, Yull FE, Blackwell TS. Enhanced Expression of Catalase in Mitochondria Modulates NF-κB-Dependent Lung Inflammation through Alteration of Metabolic Activity in Macrophages. *Journal of Immunology*. 2020;205(4):1125-1134.

27. Chen X, Xu Y, Denning KL, Grigore A, Lu Y. PPARα agonist WY-14,643 enhances ethanol metabolism in mice: Role of catalase. *Free Radical Biology and Medicine*. 2021;(169):283-293.

28. Yue R, Chen GY, Xie G, Hao L, Guo W, Sun X, Jia W, Zhang Q, Zhou Z, Zhong W. Activation of PPARα-catalase pathway reverses alcoholic liver injury via upregulating NAD synthesis and accelerating alcohol clearance. *Free Radical Biology and Medicine*. 2021;(174):249-263.

29. Zhang J, Zhang L, Zhang S, Yu Q, Xiong F, Huang K, Wang CY, Yang P. HMGB1, an innate alarmin, plays a critical role in chronic inflammation of adipose tissue in obesity. *Molecular and Cellular Endocrinology*. 2017;(454):103-111.

30. Ansar M, Ivanciuc T, Garofalo RP, Casola A. Increased Lung Catalase Activity Confers Protection Against Experimental RSV Infection. *Scientific Reports*. 2020;10(1):3653.

31. Mu Y, Maharjan Y, Kumar Dutta R, Wei X, Kim JH, Son J, Park C, Park R. Pharmacological inhibition of catalase induces peroxisome leakage and suppression of LPS induced inflammatory response in Raw 264.7 cell. *Plos One does not include*. 2021;16(2):e0245799.

32. Nagarkoti S, Dubey M, Sadaf S, Awasthi D, Chandra T, Jagavelu K, Kumar S, Dikshit M. Catalase S-Glutathionylation by NOX2 and Mitochondrial-Derived ROS Adversely Affects Mice and Human Neutrophil Survival. *Inflammation*. 2019;42(6):2286-2296.

33. Iborra M, Moret I, Busó E, García-Giménez JL, Ricart E, Gisbert JP, Cabré E, Esteve M, Márquez-Mosquera L, García-Planella E, Guardiola J, Pallardó FV, Serena C, Algaba-Chueca F, Domenech E, Nos P, Beltrán B. The Genetic Diversity and Dysfunctionality of Catalase Associated with a Worse Outcome in Crohn's Disease. *International Journal of Molecular Sciences*. 2022;23(24):1588134

34. Gong M, Liang W, Lu Q, Zhang J. PHLDA1 knockdown inhibits inflammation and oxidative stress by regulating JNK/ERK pathway, and plays a protective role in sepsis-induced acute kidney injury. *Allergologia et Immunopathologia*. 2022;50(6):1-9.

35. Najafi A, Keykhaee M, Khorramdelazad H, Karimi MY, Nejatbakhsh Samimi L, Aghamohamadi N, Karimi M, Falak R, Khoobi M. Catalase application in cancer therapy: Simultaneous focusing on hypoxia attenuation and macrophage reprogramming. *Biomedicine and Pharmacotherapy*. 2022; (153):113483.

36. Barek MA, Jafrin S, Aziz MA, Islam MS. Catalase C262T genetic variation and cancer susceptibility: A comprehensive meta-analysis with meta-regression and trial sequential analysis. *International Journal* of *Biological Markers*. 2022;37(3):227-240.

37. Crist SB, Nemkov T, Dumpit RF, Dai J, Tapscott SJ, True LD, Swarbrick A, Sullivan LB, Nelson PS, Hansen KC, Ghajar CM. Unchecked oxidative stress in skeletal muscle prevents outgrowth of disseminated tumour cells. *Nature Cell Biology*. 2022; 24(4):538-553.

38. Liu Y, Ao X, Ding W. Critical role of FOXO3a in carcinogenesis. *Molecular Cancer*. 2018;17(1):104.

39. Habrowska-Gorczynska DE, Koziel MJ, Kowalska K. FOXO3a and its regulators in prostate cancer. *International Journal of Molecular Sciences*. 2021;22(22):12530.

40. McDowell SAC, Luo RBE, Arabzadeh A, Doré S, Bennett NC, Breton V, Karimi E, Rezanejad M, Yang RR, Lach KD, Issac MSM, Samborska B, Perus LJM, Moldoveanu D, Wei Y, Fiset B, Rayes RF, Watson IR, Kazak L, Guiot MC, Fiset PO, Spicer JD, Dannenberg AJ, Walsh LA, Quail DF. Neutrophil oxidative stress mediates obesity-associated vascular dysfunction and metastatic transmigration. *Nature Cancer*. 2021;2(5):545-562.

41. Chen P, Hu T, Liang Y, Li P, Chen X, Zhang J. Neddylation inhibition activates the extrinsic apoptosis pathway through ATF4-CHOP-DR5 Axis in human esophageal Cancer cells. *Clinical Cancer Research*. 2016;22(16):4145–4157.

42. Liang Y, Jiang Y, Jin X, Chen P, Heng Y, Cai L, Zhang W, Li L, Jia L. Neddylation inhibition activates the protective autophagy through NF-κB-catalase-ATF3 Axis in human esophageal cancer cells. *Cell Communication and Signaling*. 2020;18(1):72.

43 Handschuh L, Kaźmierczak M, Milewski MC, Góralski M, Łuczak M, Wojtaszewska M, Uszczyńska-Ratajczak B, Lewandowski K, Komarnicki M, Figlerowicz M. Gene expression profiling of acute myeloid leukemia samples from adult patients with AML-M1 and -M2 through boutique microarrays, real-time PCR and droplet digital PCR. *International Journal of Oncology*. 2018;52(3):656-678.

44. Tao Y, Liu S, Lu J, Fu S, Li L, Zhang J, Wang Z, Hong M. FOXO3a-ROS pathway is involved in androgen-induced proliferation of prostate cancer cell. *BMC Urology*. 2022;22(1):70.

45. Preci DP, Almeida A, Weiler AL, Mukai Franciosi ML, Cardoso AM. Oxidative damage and antioxidants in cervical cancer. *International Journal of Gynecological Cancer*. 2021;31(2):265-271.

46. Bengtson C, Bogaerts A. On the Anti-Cancer Effect of Cold Atmospheric Plasma and the Possible Role of Catalase-Dependent Apoptotic Pathways. *Cells*. 2020; 9(10):2330

47. Zińczuk J, Maciejczyk M, Zaręba K, Romaniuk W, Markowski A, Kędra B, Zalewska A, Pryczynicz A, Matowicka-Karna J, Guzińska-Ustymowicz K. Antioxidant Barrier, Redox Status, and Oxidative Damage to Biomolecules in Patients with Colorectal Cancer. Can Malondialdehyde and Catalase Be Markers of Colorectal Cancer Advancement? *Biomolecules*. 2019;9(10):637

48. Lin HY, Lim SW, Hsu TI, Yang WB, Huang CC, Tsai YT, Chang WC, Ko CY. CCAAT/Enhancer-Binding Protein Delta Regulates Glioblastoma Survival through Catalase-Mediated Hydrogen Peroxide Clearance. *Oxidative Medicine and Cellular Longevity*. 2022;2022:4081380. DOI: 10.1155/2022/4081380

49. Lebelo MT, Joubert AM, Visagie MH. Dysregulation of Catalase by a Sulphamoylated Estradiol Analogue Culminates in Antimitotic Activity and Cell Death Induction in Breast Cancer Cell Lines. *Molecules*. 2021;26(3):622.

50. Galasso M, Dalla Pozza E, Chignola R, Gambino S, Cavallini C, Quaglia FM, Lovato O, Dando I, Malpeli G, Krampera M, Donadelli M, Romanelli MG, Scupoli MT. The rs1001179 SNP and CpG methylation regulate catalase expression in chronic lymphocytic leukemia. *Cellular and Molecular Life Sciences*. 2022;79(10):521.

Author information

Ekaterina Yu. Sergeeva, Dr. Biol. Sci., Professor, Prof. V. F. Voino-Yasenetsky Krasnoyarsk State Medical University; Address: 1, Partizan Zheleznyak Str., Krasnoyarsk, Russian Federation 660022; Phone: +7 (950) 4162275; e-mail: e.yu.sergeeva@mail.ru, http://orcid.org/ 0000-0002-2089-6022

Svetlana S. Dunaevskaya, Dr. Med. Sci., Professor, Prof. V. F. Voino-Yasenetsky Krasnoyarsk State Medical University; Address: 1, Partizan Zheleznyak Str., Krasnoyarsk, Russian Federation 660022; Phone: +7 (905) 0873585; e-mail: vikto-potapenk@yandex.ru, http://orcid.org/ 0000-0003-2820-4737

Vera V. Deulina, Cand. Med. Sci., Prof. V. F. Voino-Yasenetsky Krasnoyarsk State Medical University; Address: 1, Partizan Zheleznyak Str., Krasnoyarsk, Russian Federation 660022; Phone: +7(923)2945801; e-mail: Vera_Deulin@mail.ru, http://orcid.org/0000-0001-5818-1631

Yulia A. Fefelova, Dr. Biol. Sci., Prof. V. F. Voino-Yasenetsky Krasnoyarsk State Medical University; Address: 1, Partizan Zheleznyak Str., Krasnoyarsk, Russian Federation 660022; Phone: +7(391)2283649; e-mail: fefelovaja@mail.ru, http://orcid.org/ 0000-0001-5434-7155

Received 07 February 2024
Revision Received 08 April 2024
Accepted 28 June 2024