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CHANGES IN LIPID PEROXIDATION MARKERS AND ANTIOXIDANT STATUS IN LUNG ADENOCARCINOMA

In the present article, the dynamics of blood lipid peroxidation (LPO) markers and the antioxidant system (AOS) are investigated in patients with lung adenocarcinoma (LA) at different disease stages (I–IV). The study included 40 patients with histologically verified LA and 40 healthy donors. In blood serum, the concentrations of malondialdehyde (MDA), diene conjugates (DC), triene conjugates (TC), and Schiff bases (SB) were determined. In erythrocyte hemolysates, the activity of glutathione peroxidase (GPx), glutathione reductase (GR), glutathione S-transferase (GST), and the level of reduced glutathione (GSH) were measured.

In LA patients, a pronounced increase in LPO markers (MDA, DC, SB) and a decrease in GPx activity and GSH levels were revealed compared with the control group. A stage-dependent pattern was established: MDA levels were highest at stage I (a 3.4-fold increase), followed by a decline by stage IV. The concentration of DC (a primary LPO product) was elevated at the early stages, whereas secondary and terminal products (TC and SB) showed a progressive increase from stage I to stage IV (SB exceeding control values by 30.7-fold at stage IV). GPx activity was reduced at all stages, and GSH levels remained consistently decreased. GR activity exhibited a non-linear pattern.

The development of lung adenocarcinoma is accompanied by a profound imbalance in pro-/antioxidant homeostasis, manifested by enhanced LPO and depletion of antioxidant defenses. A specific stage-related dynamics of LPO markers is demonstrated: a marked rise in primary and secondary products at early stages, followed by a shift in the marker profile at advanced stages. The antioxidant system displays a phased response, with signs of partial compensation at stage III and decompensation at stage IV of the disease.

Keywords: lung adenocarcinoma; lipid peroxidation; oxidative stress; antioxidant system; malondialdehyde

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Introduction. Lung cancer remains one of the most pressing challenges in contemporary oncology, with persistently high incidence and mortality worldwide. Among histological variants, lung adenocarcinoma (LADC) is the predominant form of non-small cell lung cancer [6,10,15,33], underscoring the importance of elucidating the molecular mechanisms of its development.

Oxidative stress—arising from an imbalance between the generation of reactive oxygen species (ROS) and the activity of the antioxidant system (AOS)—is regarded as a key driver of carcinogenesis. Intensification of free-radical oxidation damages critical biomolecules, foremost the lipids of cellular membranes. Lipid peroxidation (LPO) initiated by ROS leads to membrane destabilization [18,19,28,35], impairment of barrier and receptor functions [16,21,26,35], and the formation of highly toxic secondary products [14,18,31,33] such as malondialdehyde (MDA) and conjugated dienes (CD). These compounds not only exacerbate cellular dysfunction but also possess mutagenic and carcinogenic potential, thereby promoting malignant transformation and tumor progression [4,27,31,32].

In response to enhanced LPO, a multilevel antioxidant defense is activated. However, during aggressive tumor growth, compensatory mechanisms may become insufficient, leading to aggravation of oxidative damage and disease progression [3,8,13,19].

Despite extensive research on the role of oxidative stress in cancer, the stage-dependent interplay between LPO markers and AOS status in lung adenocarcinoma remains insufficiently characterized. Establishing correlations between levels of LPO products, activities of antioxidant enzymes, and the extent of tumor spread is of both fundamental and applied significance. Such studies may deepen understanding of the molecular underpinnings of LADC progression, identify novel prognostic biochemical markers, and substantiate the rationale for antioxidant-oriented interventions within combined treatment strategies.

Study objective: To evaluate changes in the concentrations of primary and secondary LPO markers and in the activities of antioxidant system enzymes in blood from patients with lung adenocarcinoma across stages I–IV

Materials and Methods. The study was conducted in 2025 at the Laboratory of Precancerogenesis and Malignant Tumors, Department of Epidemiology of Chronic Non-Communicable Diseases, Yakut Science Center of Complex Medical Problems, in collaboration with the Yakut Republican Oncology Center (Yakutsk, Russian Federation).

We examined 40 patients with histologically confirmed lung adenocarcinoma and 40 apparently healthy volunteers matched to the patient group by age, sex, and ethnicity. Major exclusion criteria for controls were: presence of any oncologic disease, severe comorbid conditions, and use of medications with pronounced antioxidant properties.

The study complied with the Declaration of Helsinki and its subsequent amendments and was approved by the Local Ethics Committee of the Yakut Science Centre of Complex Medical Problems (Protocol No. 52, 24 March 2021). Baseline characteristics of participants are presented in Table 1.

Fasting venous blood served as the study material. Serum concentrations of lipid peroxidation (LPO) markers were determined by spectrophotometric methods: malondialdehyde (MDA) [2], conjugated dienes (CD) [22] and conjugated trienes (CT) [23], and Schiff bases (SB) [9]. In erythrocyte hemolysate, the activ-

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ities of glutathione peroxidase (GPx) [7], glutathione reductase (GR) [24], glutathione S-transferase (GST) [11], and the level of reduced glutathione (GSH) [20] were measured.

Statistical analysis was performed using SPSS version 23. Normality of the data distribution was assessed with the one-sample Kolmogorov–Smirnov test. Between-group differences were evaluated using Student's t-test (for normally distributed data) or the Mann–Whitney U test (for non-normal distributions). Differences were considered statistically significant at $p < 0.05$

Results. Comparison of LPO markers between patients with lung adenocarcinoma and the control group showed a statistically significant increase in most LPO indices (MDA, CD, SB), along with reduced GPx activity and lower GSH levels in the patient group (Table 2).

We identified a stage-dependent pattern for MDA levels (Table 3). At stage I, the MDA concentration was highest (2.61 ± 0.39 μmol/L), 3.4-fold above the control. At stages II and III, MDA remained elevated (3.0- and 2.8-fold above control, respectively), whereas at stage IV a statistically significant decrease was observed (1.5-fold above control). The sharp rise in MDA at early stages likely reflects active lipid peroxidation, whereas the subsequent decline may be attributable to depletion of oxidizable substrates, systemic intoxication, or activation of alternative pathways for the disposal of lipid peroxidation products.

The level of conjugated dienes (primary LPO products) was highest at stage I (1.5-fold above control). At subsequent stages, CD values stabilized at levels only slightly exceeding the control, indicating active initiation of free-radical processes at the early phase of carcinogenesis. Secondary LPO products—conjugated trienes and Schiff bases—increased progressively from stage I to stage IV, showing marked elevations at advanced stages (SB levels at stages III and IV were 20.9- and 30.7-fold above control, respectively). This pattern indicates the accumulation of deep, largely irreversible damage to lipids and proteins as the tumor progresses.

Against the background of intensified LPO, the antioxidant system exhibited phased changes—from compensation to exhaustion (Table 4).

Table 1

Characteristics of the examined patients with lung adenocarcinoma and the control group

Parameter	Patients (n = 40)	Controls (n = 40)
Sex, M/F (n)	34/6	34/6
Ethnicity, Sakha/ Russian (n)	34/6	34/6
Mean age (years)	66.4 ± 1.1	65.5 ± 1.4
Stage I (n)	3	-
Stage II (n)	6	-
Stage III (n)	15	-
Stage IV (n)	16	-

Table 2

Lipid peroxidation (LPO) markers and antioxidant system (AOS) parameters in patients with lung adenocarcinoma and in the control group

Parameter	Patients (n = 40)	Controls (n = 40)	p-value
MDA, μmol/L	1.79 ± 0.15	0.76 ± 0.14	< 0.001
CD, arbitrary units (a.u.)	1.04 ± 0.05	0.89 ± 0.05	0.012
CT, a.u.	0.41 ± 0.15	0.22 ± 0.02	0.603
SB, a.u.	0.33 ± 0.08	0.016 ± 0.001	< 0.0001
GPx, U/mL	9.44 ± 0.68	11.72 ± 0.27	0.003
GST, U/mL	1.56 ± 0.13	1.66 ± 0.10	0.194
GR, U/mL	4.75 ± 0.49	3.66 ± 0.20	0.646
GSH, μmol/L	2.38 ± 0.15	3.08 ± 0.13	< 0.001

Note: The p-value was calculated using the Mann-Whitney U test.

Table 3

Lipid peroxidation markers in patients according to disease stage

Parameter	Control (n = 40)	Stage I (n = 3)	Stage II (n = 6)	Stage III (n = 15)	Stage IV (n = 16)
MDA, μmol/L	0.76 ± 0.14	2.61 ± 0.39**	2.24 ± 0.40**	2.11 ±0.22***	1.17 ± 0.17*
CD, a.u.	0.89 ± 0.05	1.36 ± 0.00	0.91 ± 0.19	1.12 ± 0.06**	0.98 ± 0.06
CT, a.u.	0.22 ± 0.02	0.09 ± 0.00*	0.14 ± 0.03	0.24 ± 0.03	0.71 ± 0.36
SB, a.u.	0.016 ± 0.001	0.035 ± 0.000*	0.057 ± 0.012***	0.31 ± 0.07***	0.46 ± 0.16***

Note: p-values were calculated using the Mann-Whitney U test: * – $p < 0.05$; ** – $p < 0.01$; *** – $p < 0.001$.

Table 4

Antioxidant defense parameters in patients according to disease stage

Parameter	Control (n = 40)	Stage I (n = 3)	Stage II (n = 6)	Stage III (n = 15)	Stage IV (n = 16)
GPx, U/mL	11.72 ± 0.27	9.24 ± 4.56	7.93 ± 0.43***	8.67 ± 1.09*	10.76 ± 1.12
GST, U/mL	1.66 ± 0.10	1.45 ± 0.47	1.50 ± 0.26	1.51 ± 0.14	1.65 ± 0.28
GR, U/mL	3.66 ± 0.20	2.68 ± 0.00	4.05 ± 0.93	5.93 ± 0.92*	4.24 ± 0.70
GSH, μmol/L	3.08 ± 0.13	2.40 ± 0.17	2.28 ± 0.19*	2.30 ± 0.29**	2.50 ± 0.26

Note: p-values were calculated using the Mann-Whitney U test: * – $p < 0.05$; ** – $p < 0.01$; *** – $p < 0.001$.

Glutathione peroxidase (GPx) activity was reduced at all stages, with the greatest suppression at stages II and III (32.3% and 26.0% below control, respectively). Glutathione S-transferase (GST) activity remained relatively stable, showing no statistically significant differences from the control group. Glutathione reductase (GR) activity exhibited a non-linear pattern: a decrease at stage I, followed by an increase to a peak at stage III, and a subsequent decline at stage IV. The level of reduced glutathione (GSH) was consistently decreased across all disease stages.

Discussion. The present study revealed pronounced disturbances in lipid peroxidation (LPO) and antioxidant defense in patients with lung adenocarcinoma, supporting the central role of oxidative stress in the pathogenesis of this disease.

The increase in core LPO markers (MDA, conjugated dienes, Schiff bases) together with decreases in GSH level and GPx activity corroborates the concept that intense tumor growth is associated with induction of free-radical processes and depletion of antioxidant potential.

The stage-dependent behavior of MDA proved most informative. The peak concentration at stage I—3.4-fold above control—likely reflects a phase of active initiation of free-radical reactions. This interpretation is consistent with the findings of Zheng et al. (2024), who associated elevated MDA levels with early stages of cancer, underscoring its potential as a biomarker of tumor progression [35]. The subsequent decline at stage IV can plausibly be explained by depletion of peroxidation substrates, overall metabolic depression, and systemic intoxication in the terminal phase, in line with the observations of Jomova et al. (2023), who noted altered oxidative-stress profiles at advanced stages of cancer [14].

Analysis of stage-wise dynamics of LPO products clarifies the cascade of oxidative reactions. Elevated conjugated dienes at stage I indicate active initiation of free-radical oxidation at early carcinogenesis. This is consistent with findings by Lei et al. (2021), who emphasized the role of early oxidative damage, evidenced by accumulation of characteristic metabolites at initial stages of oncogenesis [18]. Stabilization of conjugated dienes at later stages, accompanied by substantial increases in conjugated trienes and Schiff bases, indicates efficient conversion of primary LPO products into secondary and terminal species. Progressive accumulation of Schiff bases clearly reflects the deepening of oxidative stress as the disease advances [2].

The antioxidant response to intensified LPO was phased. Persistent reductions in GSH and GPx activity at all stages appear to represent a fundamental defect characteristic of a deficit in the key peroxide-detoxifying system. This observation aligns with Barartabar et al. (2023), who reported that tumor growth is frequently accompanied by depletion of antioxidant reserves, thereby amplifying oxidative stress [4]. The nonlinear pattern of GR activity conforms to the classical transition from compensatory to decompensatory phases, culminating in exhaustion of antioxidant reserves at the terminal stage [17]. This is consistent with Chaudhary et al. (2023), who discussed how dysregulation of antioxidant-enzyme activity can aggravate oxidative injury and promote cancer progression [8].

In sum, these findings underscore a profound imbalance in pro-/antioxidant homeostasis associated with lung adenocarcinoma. The stage-specific dynamics of LPO markers—an early surge in primary and secondary products with a subsequent shift in the marker profile at later stages—provide valuable insight into the molecular mechanisms underpinning tumor progression. Moreover, the phased antioxidant response, with signs of compensation at stage III and decompensation at stage IV, points to the potential of therapeutic strategies aimed at mitigating oxidative stress, which could enhance the effectiveness of existing treatments and improve patient outcomes, as suggested by recent reviews on the role of antioxidants in cancer therapy [29].

Conclusions. The development of lung adenocarcinoma is accompanied by a profound imbalance in pro-antioxidant homeostasis, manifested by intensified lipid peroxidation (LPO) and depletion of antioxidant defenses.

A stage-specific pattern of LPO markers was identified: a sharp rise in primary and secondary products at early stages, followed by a shift in the marker profile at advanced stages.

The antioxidant system exhibits a phased response, with signs of compensation at stage III and decompensation at stage IV.

The authors declare that there is no conflict of interest.

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DIAGNOSTIC AND TREATMENT METHODS

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THE USE OF STEM CELLS IN OSTEOPLASTY OF JAW DEFECTS: A CELL - ENGINEERING APPROACH

The use of stem cells in osteoplasty of jaw defects is one of the most promising areas of modern cellular engineering regenerative medicine. Traditional osteoplasty methods have a number of limitations, from the risk of infections and pain to the limited amount of available graft. In this regard, stem cells open up new possibilities for creating biologically active structures capable of stimulating osteogenesis and restoring complex structures of the maxillofacial region. The review systematizes current data on the use of periodontal ligament (PDLSC), dental pulp (DPSC) and jawbone (JBMSC) stem cells in osteoplasty of jaw defects. Their morphological and molecular characteristics, osteogenic potential, interaction with the microenvironment of the defect, as well as integration with biomaterials and growth factors are considered. Special attention is paid to the results of preclinical and clinical studies confirming the safety and effectiveness of cellular therapies aimed at restoring the cement–periodontal ligament–bone complex and improving the osseointegration of implants. In addition, the work analyzes existing preclinical models of jawbone defects in small and large animals, providing an experimental basis for evaluating the effectiveness of cellular engineering structures and developing safe protocols for clinical use. The importance of DPSC and JBMSC exosomes as biologically active factors enhancing osteogenic differentiation and tissue regeneration is noted. The obtained data emphasize the high prospects of using stem cells from the oral cavity for bone tissue regeneration, the development of new biocompatible materials and individualized therapeutic strategies. The presented review can serve as a scientific basis for creating effective, safe and clinically justified approaches to the treatment of maxillofacial defects and improving the results of implantation therapy.

Keywords: stem cells, osteoplasty, jaw defects, tissue engineering, PDLSC, DPSC, JBMSC, bone regeneration

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