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OXIDATIVE STRESS AND MORPHOLOGICAL ALTERATIONS IN THE UTERUS DURING CHRONIC CARBON MONOXIDE EXPOSURE

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СУРУНКАЛИ ИС ГАЗИ ТАЪСИРИДА БАЧАДОНДА ОКСИДАТИВ СТРЕСС ВА МОРФОЛОГИК ЎЗГАРИШЛАР

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ОКИСЛИТЕЛЬНЫЙ СТРЕСС И МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В МАТКЕ ПРИ ХРОНИЧЕСКОМ ВОЗДЕЙСТВИИ УГАРНОГО ГАЗА

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Резюме. *Ис гази (CO) — углеродли моддаларнинг тўлиқ ёнмаслиги натижасида ҳосил бўладиган атроф-муҳитнинг энг кенг тарқалган токсик ифлослантирувчиларидан бири. CO нинг гемоглобинга мойиллиги кислородга нисбатан 200 баравардан ортиқ юқори бўлганлиги сабабли у карбоксигемоглобин (COHb) ҳосил қилади ва қоннинг кислород ташиш қобилиятини сезиларли даражада камайтириб, тўқималарда гипоксия ривожланишига олиб келади. CO билан ўткир ва сурункали заҳарланишнинг юрак-қон томир ҳамда марказий нерв тизимига таъсири кенг ўрганилган бўлса-да, унинг паст дозали сурункали таъсири аёл репродуктив тизимига, хусусан, бачадоннинг морфологик ва биокимёвий ҳолатига қандай таъсир қилиши етарлича ўрганилмаган. Тадқиқотнинг мақсади — лаборатория ҳайвонларида сурункали ис гази таъсирида бачадон тўқималаридаги морфологик ва морфометрик ўзгаришларни, шунингдек, оксидатив стресс кўрсаткичларини ўрганиш. Тадқиқот 40 та урғочи оқ лаборатория каламушларида ўтказилган бўлиб, улар бир назорат ва уч экспериментал гуруҳга бўлинди ҳамда 30 кун давомида 50, 100 ва 200 ppm концентрацияларда CO билан таъсир кўрсатилди. Гистологик баҳолаш гематоксилин–эозин бўяш усули билан, морфометрик таҳлил эса эндометрий қалинлиги, бачадон безлари сони ва қон томир зичлигини ўлчаш орқали амалга оширилди. Оксидатив стресс малон диальдегид (МДА) миқдори ва супероксиддисмутаза (СОД) фаоллигига қараб баҳоланди. Натижалар назорат гуруҳида 420 ± 18 мкм бўлган эндометрий қалинлиги 200 ppm концентрацияда 285 ± 15 мкм гача статистик жиҳатдан ишончли камайганини ($p < 0,001$), безлар сонининг эса 28 ± 2 дан 14 ± 2 гача ($p < 0,001$) пасайганини кўрсатди. МДА даражаси назоратга нисбатан 168% га ошган, СОД фаоллиги эса 62% га камайган, бу про-/антиоксидант мувозанатнинг бузилишидан далолат беради.* ***Хулоса:*** *Сурункали ис гази таъсири бачадон тўқималарининг доза боғлиқ структуравий қайта тузилишига сабаб бўлади ва эндометрий ҳамда миометрийнинг редокс гомеостазини бузади. Аниқланган ўзгаришлар репродуктив функцияга салбий таъсир кўрсатиши мумкин ва репродуктив ёшдаги аёллар учун илгари етарлича баҳоланмаган экологик хавф омили ҳисобланади.*

Калит сўзлар: *ис гази, бачадон, эндометрий, оксидатив стресс, гипоксия, малон диальдегид, супероксиддисмутаза, репродуктив токсикология, экспериментал морфология.*

Abstract. *Carbon monoxide (CO) is one of the most prevalent toxic environmental pollutants formed during the incomplete combustion of carbon-containing materials. Owing to its high affinity for hemoglobin (more than 200 times greater than that of oxygen), CO forms carboxyhemoglobin (COHb), which significantly impairs the oxygen-carrying capacity of blood and induces tissue hypoxia. Although the systemic effects of acute and chronic CO poisoning have been extensively investigated in the cardiovascular and central nervous systems, the impact of chronic low-dose CO exposure on the female reproductive system, particularly on the morphological and biochemical state of the uterus, remains insufficiently characterized. The aim of the present experimental study was to investigate the morphological and morphometric alterations in uterine tissues, as well as oxidative stress parameters, induced by chronic exposure to carbon monoxide in laboratory animals. The study was performed on 40 female white laboratory rats divided into one control and three experimental groups exposed to 50, 100, and 200 ppm of CO over 30 days. Histological evaluation was performed using hema-*

toxylin–eosin staining, while morphometric analysis included measurement of endometrial thickness, the number of uterine glands, and vascular density. Oxidative stress was assessed by quantitative determination of malondialdehyde (MDA) and superoxide dismutase (SOD) activity. The results revealed a statistically significant dose-dependent decrease in endometrial thickness from 420 ± 18 μm in the control group to 285 ± 15 μm at 200 ppm ($p < 0.001$), accompanied by a reduction in the number of uterine glands from 28 ± 2 to 14 ± 2 ($p < 0.001$). MDA levels increased by 168% relative to control, while SOD activity decreased by 62%, indicating significant pro-oxidant–antioxidant imbalance. ***Conclusion:*** *Chronic exposure to carbon monoxide induces dose-dependent structural remodeling of uterine tissues and disturbs the redox homeostasis of the endometrium and myometrium. These changes may compromise reproductive function and represent a previously underestimated environmental risk factor for women of reproductive age.*

Keywords: *carbon monoxide, uterus, endometrium, oxidative stress, hypoxia, malondialdehyde, superoxide dismutase, reproductive toxicology, experimental morphology.*

Introduction. Environmental pollution has emerged as one of the most significant global public health challenges of the 21st century. Among the wide spectrum of anthropogenic pollutants, carbon monoxide (CO) occupies a particularly important position due to its ubiquitous presence in urban environments, industrial settings, and indoor air. CO is a colorless, odorless, and tasteless gas formed primarily during the incomplete combustion of carbon-containing materials such as coal, gasoline, natural gas, biomass, and tobacco products. According to the World Health Organization, ambient air pollution accounts for approximately 4.2 million premature deaths annually worldwide, with CO contributing substantially to the burden of chronic disease in low- and middle-income countries [1, 2].

The toxicokinetic profile of carbon monoxide is largely determined by its extraordinarily high affinity for hemoglobin — approximately 210–250 times greater than that of molecular oxygen. The resulting formation of carboxyhemoglobin (COHb) significantly reduces the oxygen-carrying capacity of blood and shifts the oxyhemoglobin dissociation curve to the left, further impairing tissue oxygen delivery. In addition to this classical hypoxic mechanism, CO directly binds to mitochondrial cytochrome c oxidase (complex IV of the respiratory chain), thereby disrupting cellular respiration even in the presence of adequate oxygen tension. This dual action establishes the molecular basis for both the acute and chronic effects of CO toxicity at the cellular level [3, 5].

Chronic exposure to low concentrations of carbon monoxide initiates a cascade of pathophysiological events, of which oxidative stress is increasingly recognized as a central mechanism. Oxidative stress is defined as a state of imbalance between the production of reactive oxygen species (ROS) — such as superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radical (•OH) — and the capacity of endogenous antioxidant defense systems, including superoxide dismutase (SOD), catalase, glutathione peroxidase, and glutathione. Excessive ROS accumulation leads to lipid peroxidation, protein carbonylation, DNA strand breaks, and ultimately cellular dysfunction or apoptotic/necrotic cell death [6, 7].

The female reproductive system, and particularly the uterus, is exquisitely sensitive to alterations in oxygen tension and redox status. The cyclic remodeling of the endometrium during the estrous and menstrual cycles, the dynamic vascular changes during the implantation window, and the metabolic demands of pregnancy all rely on a finely tuned balance between proliferation, differentiation, and apoptosis. Each of these processes is critically dependent on adequate oxygen supply, mitochondrial function, and antioxidant capacity. Disruption of this delicate equilibrium by environmental toxicants such as CO may therefore translate into structural remodeling of uterine tissues, impaired endometrial receptivity, and reduced reproductive performance [4, 8].

Existing experimental evidence suggests that chronic exposure to airborne pollutants and hypoxic conditions induces oxidative damage and morphological alterations in various reproductive organs, including the ovary, placenta, and testes [4, 9]. Moreover, recent investigations have implicated the heme oxygenase / carbon monoxide system in the pathogenesis of placental disorders such as preeclampsia, highlighting the dual nature of CO as both an endogenous signaling molecule and an exogenous toxic agent [3]. However, despite this growing body of literature, the specific effects of chronic environmental CO exposure on the morphology and oxidative status of the non-pregnant uterus remain poorly characterized. Most published studies have focused on acute high-dose exposures or on placental tissue, leaving a substantial knowledge gap regarding subchronic, low-to-moderate dose effects on uterine architecture [9, 10].

Bridging this knowledge gap is of considerable clinical and public health relevance. Women of reproductive age in many regions are routinely exposed to CO concentrations of 5–50 ppm in indoor environments (gas stoves, biomass cooking, traffic-related pollution), and occupational exposure may reach significantly higher levels. If chronic CO exposure indeed induces structural and oxidative alterations in the uterus, this would have implications for fertility, implantation, and pregnancy outcomes.

Accordingly, the present experimental study was designed to systematically evaluate the morphological, morphometric, and oxidative stress consequences of 30-day chronic CO exposure on the rat uterus across a range of clinically relevant concentrations (50, 100, and 200 ppm). Special attention was

paid to dose-response relationships in endometrial thickness, number of uterine glands, and the balance between pro-oxidant (MDA) and antioxidant (SOD) markers.

Materials and Methods. Experimental animals. The study was performed on 40 sexually mature female white laboratory rats (Wistar line) weighing 180–220 g and aged 8–10 weeks. All animals were obtained from the vivarium of Bukhara State Medical Institute and acclimatized for 7 days prior to the experiment. The animals were housed in standard polycarbonate cages (5 animals per cage) under controlled environmental conditions: temperature 22 ± 2 °C, relative humidity 50–60%, and a 12-hour light/dark cycle. Free access to standard laboratory chow and tap water was provided ad libitum. All experimental procedures were performed in compliance with the international ethical guidelines for the use of laboratory animals (Directive 2010/63/EU) and approved by the Local Ethics Committee of Bukhara State Medical Institute (Protocol No. 14/2024).

Experimental design. After acclimatization, the animals were randomly divided into four groups (n = 10 per group):

• Group I (Control) — animals kept under standard conditions without CO exposure;

• Group II — animals exposed to 50 ppm CO (4 hours daily, 30 days);

• Group III — animals exposed to 100 ppm CO (4 hours daily, 30 days);

• Group IV — animals exposed to 200 ppm CO (4 hours daily, 30 days).

Carbon monoxide exposure. CO exposure was performed in a custom-made airtight inhalation chamber (volume 0.25 m³) equipped with a continuous gas flow system. The CO concentration was monitored in real time using an electrochemical CO analyzer (MSA Altair 5X, USA) with a sensitivity of ±1 ppm and recalibrated daily. Pure CO from a certified compressed-gas cylinder (99.5%, Linde Gas) was diluted with ambient air to achieve target concentrations. Oxygen levels inside the chamber were maintained at 20–21%, and CO_2 was kept below 0.5% by means of an air-exchange system.

Tissue sampling. Twenty-four hours after the last exposure, animals were anesthetized by intraperitoneal injection of ketamine (75 mg/kg) and xylazine (10 mg/kg) and euthanized by cervical dislocation. Uterine horns were rapidly excised, washed in cold sterile saline (0.9% NaCl), and weighed. The right uterine horn was processed for histological analysis, while the left horn was homogenized for biochemical determination of oxidative stress markers.

Histological processing. Tissue samples for morphological examination were fixed in 10% neutral buffered formalin for 24 hours, dehydrated in graded ethanol solutions (70%, 80%, 95%, 100%; 12 hours each), cleared in xylene (2 hours), and embedded in paraffin (3 hours). Paraffin blocks were sectioned at 5 μm thickness using a rotary microtome (Leica RM2235, Germany). Sections were stained with hematoxylin and eosin (H&E) according to standard protocols and examined under a Leica DM2500 light microscope equipped with a digital imaging system (Leica DFC295). Histological details of the protocol are presented in Table 1.

Morphometric analysis. Morphometric measurements were performed on digital photomicrographs using ImageJ software (NIH, version 1.53e). The following parameters were assessed: (1) endometrial thickness, measured perpendicularly from the basal layer to the surface epithelium in 10 random fields per section; (2) the number of uterine glands per standardized field of view (×100 magnification); and (3) vascular density, expressed as the number of capillaries per square millimeter of endometrial stroma. All measurements were performed in a blinded manner by two independent investigators.

Biochemical analysis of oxidative stress. Uterine tissue homogenates (10% w/v in 50 mM phosphate buffer, pH 7.4) were prepared on ice using a Potter–Elvehjem homogenizer and centrifuged at 10,000 × g for 15 minutes at 4 °C. The supernatant was used for biochemical assays. Lipid peroxidation was assessed by measuring malondialdehyde (MDA) levels using the thiobarbituric acid reactive substances (TBARS) method, with absorbance read at 532 nm. The activity of superoxide dismutase (SOD) was determined using the xanthine/xanthine oxidase system based on inhibition of nitroblue tetrazolium reduction, expressed as U/mg protein. Total protein content was measured by the Bradford method.

Table 1. Histological processing protocol

Procedure	Reagent / Equipment	Duration
Fixation	10% neutral buffered formalin	24 hours
Dehydration	Ethanol (70 → 100%)	12 hours
Clearing	Xylene	2 hours
Embedding	Paraffin	3 hours
Sectioning	Microtome (Leica RM2235)	5 μm
Staining	Hematoxylin–eosin (H&E)	Standard protocol

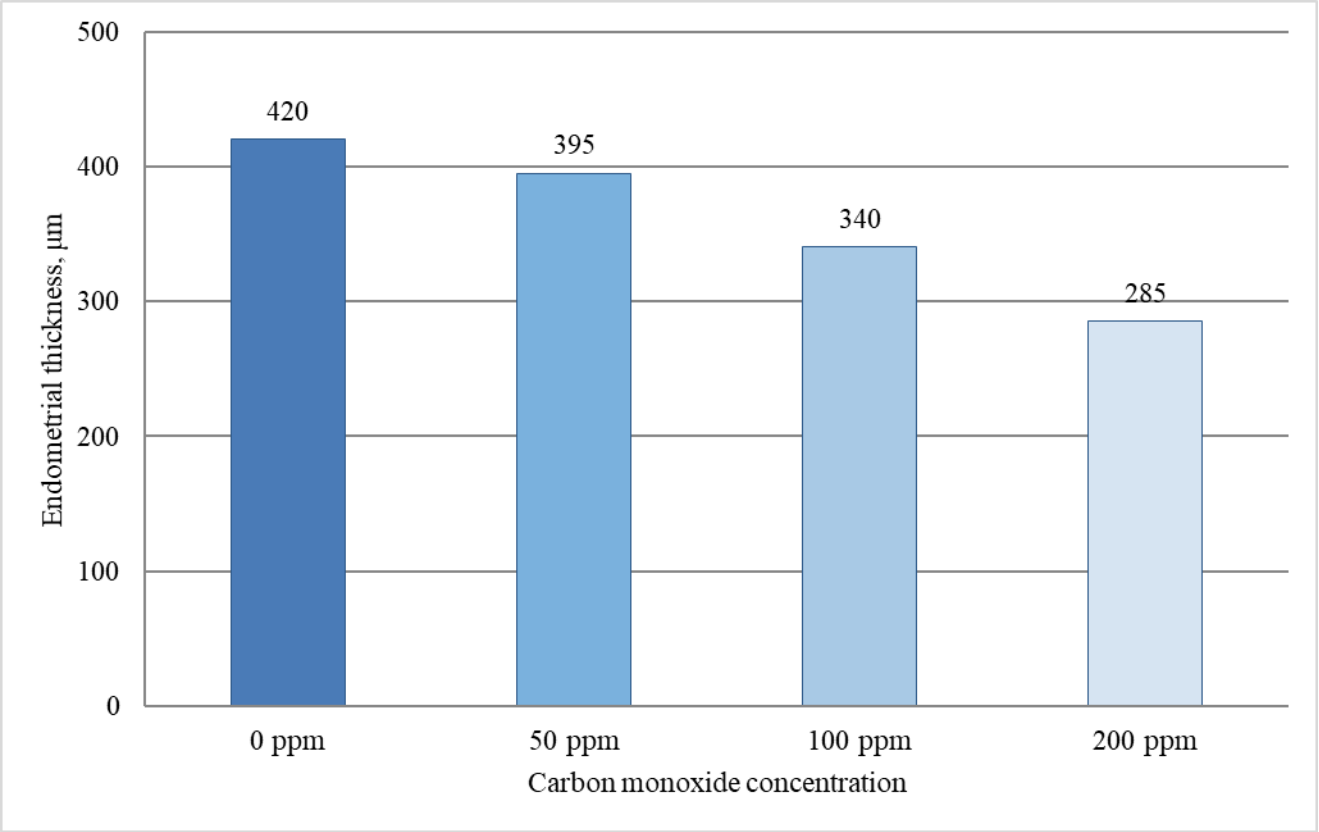

Fig 1. Dose-dependent decrease in endometrial thickness under chronic CO exposure

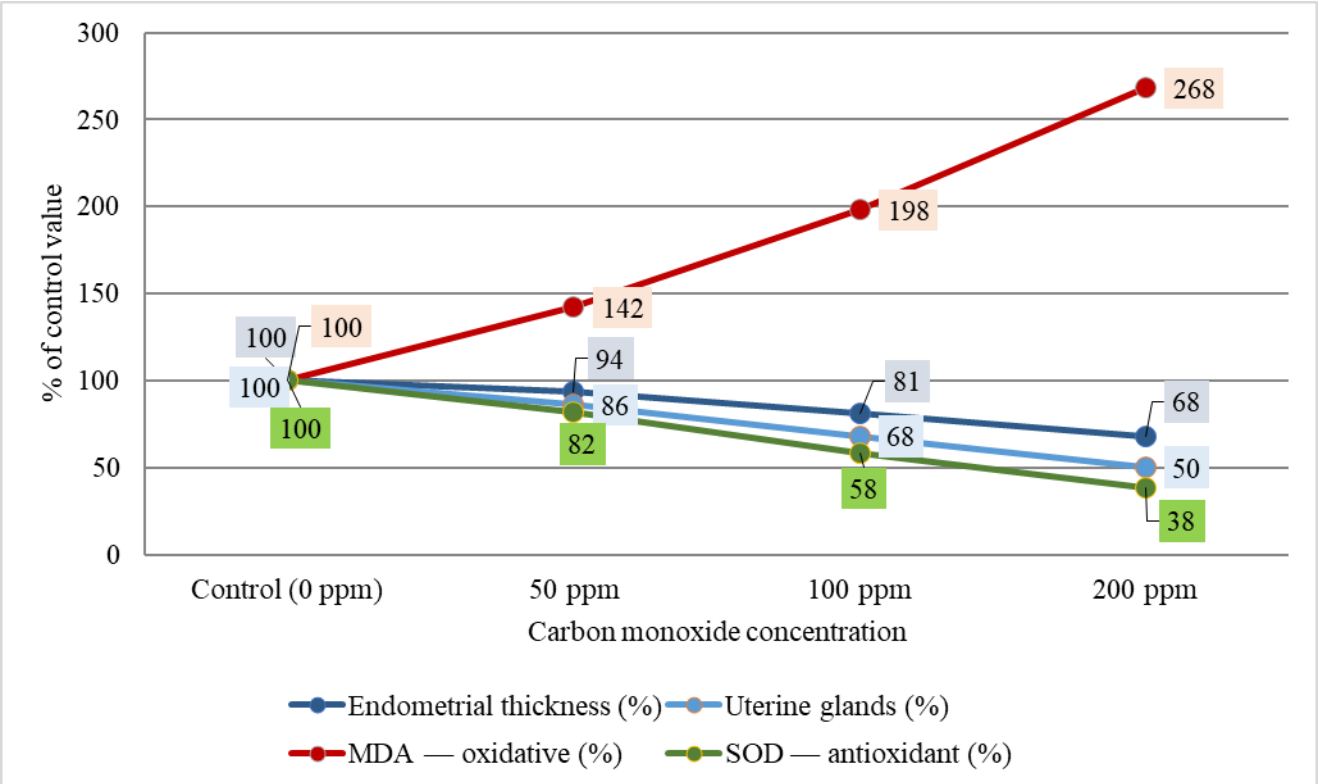

Fig 2. Comparative dynamics of morphometric and oxidative stress parameters at different CO concentrations (% of control)

Statistical analysis. All quantitative data are presented as mean ± standard deviation (M ± SD). Statistical analysis was performed using SPSS Statistics version 26.0 (IBM, USA). Intergroup differences were assessed using one-way analysis of variance (ANOVA) with Tukey's post hoc test. The Shapiro–Wilk test was used to verify the normality of data distribution. Differences were considered statistically significant at $p < 0.05$.

Results. Morphological analysis of uterine tissues demonstrated progressive structural alterations with increasing carbon monoxide concentration. In the control group (Group I), the uterus exhibited normal histoarchitecture: a clearly differentiated endometrium with a continuous columnar epithelium, well-developed and abundant uterine glands of regular shape, dense vascularized stroma, and a clearly delineated myometrium consisting of inner circular and outer longitudinal smooth muscle layers.

In animals exposed to 50 ppm CO (Group II), early adaptive changes were observed: mild congestion of stromal vessels, focal edema in the basal layer of the endometrium, and slight decrease in the height of the surface epithelium. The number of uterine glands and overall endometrial thickness were only minimally reduced, suggesting subclinical morphological changes.

In Group III (100 ppm CO), histological alterations became more pronounced. The endometrium showed marked thinning, irregular distribution of glands, and zones of stromal disorganization. The columnar epithelium was partially flattened, with focal areas of cellular vacuolization and pyknotic nuclei. Vascular changes included perivascular edema and dilation of small arterioles, consistent with chronic hypoxic injury.

Group IV (200 ppm CO) demonstrated the most severe morphological alterations. The endometrium exhibited pronounced atrophy with significant reduction in epithelial height, marked depletion of glandular structures, focal denudation of the surface epithelium, and dense fibrotic remodeling of the stroma. Sclerotic vascular changes and microhemorrhages were observed in the deep stromal layer. The myometrium showed disorganization of smooth muscle bundles and infiltration by mononuclear cells, indicating chronic inflammatory remodeling.

Quantitative morphometric analysis confirmed the histological observations and revealed a clear dose-dependent reduction in endometrial thickness across all experimental groups (Figure 1). In the control group, mean endometrial thickness was 420 ± 18 μm, corresponding to the normal morphological state of the rat uterine mucosa. Exposure to 50 ppm CO resulted in a non-significant trend toward reduction (395 ± 22 μm; $p > 0.05$ vs control), whereas exposure to 100 ppm produced a statistically significant decrease (340 ± 19 μm; $p < 0.01$). The most pronounced alteration was observed at 200 ppm, with endometrial thickness decreasing to 285 ± 15 μm — a 32.1% reduction relative to control ($p < 0.001$), indicating profound atrophic remodeling of the uterine mucosa.

A parallel analysis of the number of uterine glands revealed a similar dose-dependent pattern. The mean number of glands per standardized field decreased from 28 ± 2 in the control group to 24 ± 3 at 50 ppm ($p > 0.05$), 19 ± 2 at 100 ppm ($p < 0.01$), and 14 ± 2 at 200 ppm ($p < 0.001$), corresponding to a 50% reduction in glandular density at the highest exposure level. Together with the observed thinning of the endometrium, these findings reflect a generalized atrophic response of the uterine mucosa to chronic CO exposure.

Vascular density in the endometrial stroma demonstrated a more complex pattern. While the absolute number of capillaries per square millimeter remained relatively preserved, qualitative analysis revealed predominance of dilated and congested vessels at 100 ppm, followed by a shift toward sclerotic and stenotic vascular profiles at 200 ppm — consistent with progressive vascular remodeling secondary to chronic tissue hypoxia.

Biochemical evaluation of oxidative stress markers confirmed the histopathological findings and provided mechanistic insight into the observed structural alterations. The level of malondialdehyde (MDA) — a key marker of lipid peroxidation — increased progressively with rising CO concentration, reaching 142% of control at 50 ppm, 198% at 100 ppm, and 268% at 200 ppm ($p < 0.001$). Conversely, superoxide dismutase (SOD) activity demonstrated a parallel decrease, falling to 82%, 58%, and 38% of control values, respectively.

Table 2. Morphometric and oxidative stress parameters across experimental groups (M ± SD)

Parameter	Control	50 ppm CO	100 ppm CO	200 ppm CO
Endometrial thickness, μm	420 ± 18	395 ± 22	340 ± 19**	285 ± 15***
Number of uterine glands, n/field	28 ± 2	24 ± 3	19 ± 2**	14 ± 2***
Vascular density, n/mm²	142 ± 11	148 ± 13	136 ± 14	119 ± 12*
MDA, nmol/mg protein	2.45 ± 0.21	3.48 ± 0.28**	4.85 ± 0.34***	6.57 ± 0.42***
SOD activity, U/mg protein	18.6 ± 1.4	15.3 ± 1.5*	10.8 ± 1.2***	7.1 ± 0.9***

*Note: *p < 0.05, **p < 0.01, ***p < 0.001 vs control group (one-way ANOVA with Tukey's post hoc test).*

The integrated dynamics of all four parameters — endometrial thickness, gland number, MDA, and SOD — across CO concentrations are presented in Figure 2, illustrating the inverse relationship between morphological deterioration and oxidative stress activation.

The summary of all quantitative parameters across the four experimental groups is presented in Table 2.

Discussion. The findings of the present study provide compelling evidence that chronic exposure to carbon monoxide induces dose-dependent morphological remodeling of the rat uterus accompanied by significant disturbance of redox homeostasis. The observed combination of endometrial atrophy, glandular depletion, vascular remodeling, and pronounced pro-oxidant–antioxidant imbalance represents a coherent pathophysiological pattern consistent with chronic tissue hypoxia and oxidative injury.

The dose-dependent decrease in endometrial thickness — reaching 32.1% reduction at 200 ppm — likely reflects the synergistic action of two interrelated mechanisms. First, CO-induced systemic and local hypoxia compromises the proliferative capacity of endometrial stem and progenitor cells, which are highly dependent on adequate oxygen and nutrient supply. Second, increased ROS generation under chronic oxidative stress triggers premature apoptosis of endometrial epithelial and stromal cells, as previously demonstrated in models of uterine ageing and PCOS [6, 10].

The marked reduction in the number of uterine glands has particularly important functional implications. Endometrial glands are critically involved in the secretion of cytokines, growth factors, and nutrients required for embryo implantation and early pregnancy. The 50% decrease in glandular density observed at 200 ppm CO suggests that chronic environmental CO exposure could significantly impair endometrial receptivity, potentially contributing to subfertility, recurrent implantation failure, or early pregnancy loss in women living in highly polluted environments.

The biochemical findings — a 168% increase in MDA accompanied by a 62% decrease in SOD activity at 200 ppm — clearly demonstrate the activation of oxidative stress as a central mechanism of CO-induced uterine injury. Lipid peroxidation, reflected by elevated MDA levels, damages cellular and mitochondrial membranes, while reduced SOD activity indicates exhaustion of the first line of antioxidant defense against superoxide radicals. This pattern is consistent with previous findings in the placenta, ovary, and cochlea exposed to CO and other environmental toxicants [7, 9].

Notably, the present study extends current knowledge by simultaneously demonstrating both structural and biochemical changes in the uterus across a range of clinically relevant CO concentrations. While previous research has predominantly focused on acute CO poisoning or placental tissue, our findings underscore the relevance of subchronic, low-to-moderate exposures — levels that may be encountered in occupational settings, in households using biomass fuel, and in heavily polluted urban areas.

Several mechanistic pathways may underlie the observed effects. The heme oxygenase / CO system has been increasingly recognized as a double-edged sword: at physiological levels, endogenous CO acts as a cytoprotective signaling molecule, modulating vascular tone, inflammation, and apoptosis; in contrast, chronic excessive CO exposure overwhelms these regulatory pathways and shifts the balance toward tissue injury [3, 5]. The activation of NF-κB and HIF-1α signaling, mitochondrial dysfunction, and impaired SIRT1/NRF2 antioxidant pathways are likely candidates mediating CO-induced uterine damage [10].

The clinical and public health implications of these findings are substantial. The reproductive toxicity of CO has been historically underestimated, and current air quality standards may not adequately protect women of reproductive age. Future studies should investigate whether the structural and oxidative changes observed in animal models translate into impaired fertility, altered menstrual cycles, or adverse pregnancy outcomes in humans. The potential protective role of antioxidants, heme oxygenase modulators, and nutritional interventions also warrants further exploration.

Conclusion

Chronic exposure to carbon monoxide induces dose-dependent histological and morphometric alterations in uterine tissues, including significant thinning of the endometrium (up to 32% reduction at 200 ppm), pronounced depletion of uterine glands (up to 50% reduction), and progressive vascular remodeling. These structural changes are paralleled by activation of oxidative stress, characterized by a 168% increase in MDA and a 62% decrease in SOD activity. The findings confirm that the female reproductive system, and specifically the uterus, is a previously underestimated target of chronic CO toxicity. The observed alterations are likely mediated by a combination of tissue hypoxia, mitochondrial dysfunction, and pro-oxidant–antioxidant imbalance, and may negatively affect endometrial receptivity and reproductive function. These results provide an experimental basis for further research into the reproductive consequences of environmental CO exposure and underscore the need for stricter air quality standards aimed at protecting women's reproductive health.

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ОКИСЛИТЕЛЬНЫЙ СТРЕСС И МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В МАТКЕ ПРИ ХРОНИЧЕСКОМ ВОЗДЕЙСТВИИ УГАРНОГО ГАЗА

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Резюме. *Угарный газ (CO) является одним из наиболее распространённых токсических загрязнителей окружающей среды, образующихся при неполном сгорании углеродсодержащих материалов. Благодаря высокой аффинности к гемоглобину (превосходящей таковую кислорода более чем в 200 раз) CO образует карбоксигемоглобин (COHb), что значительно снижает кислородтранспортную способность крови и вызывает развитие тканевой гипоксии. Несмотря на детально изученные системные эффекты острого и хронического отравления CO на сердечно-сосудистую и центральную нервную системы, влияние хронического низкодозового воздействия CO на женскую репродуктивную систему, в частности на морфологическое и биохимическое состояние матки, изучено недостаточно. Целью настоящего экспериментального исследования явилось изучение морфологических и морфометрических изменений тканей матки, а также показателей окислительного стресса при хроническом воздействии угарного газа у лабораторных животных. Исследование выполнено на 40 самках белых лабораторных крыс, разделённых на одну контрольную и три экспериментальные группы, подвергавшиеся воздействию CO в концентрациях 50, 100 и 200 ppm в течение 30 дней. Гистологическая оценка проводилась с применением окраски гематоксилин–эозином, а морфометрический анализ включал измерение толщины эндометрия, количества маточных желёз и сосудистой плотности. Окислительный стресс оценивался по содержанию малонового диальдегида (МДА) и активности супероксиддисмутазы (СОД). Результаты продемонстрировали статистически значимое дозозависимое снижение толщины эндометрия с 420 ± 18 мкм в контрольной группе до 285 ± 15 мкм при концентрации 200 ppm ($p < 0,001$), сопровождавшееся уменьшением числа маточных желёз с 28 ± 2 до 14 ± 2 ($p < 0,001$). Уровень МДА повысился на 168% относительно контроля, тогда как активность СОД снизилась на 62%, что свидетельствует о выраженном про-/антиоксидантном дисбалансе. Заключение: Хроническое воздействие угарного газа индуцирует дозозависимую структурную перестройку тканей матки и нарушает редокс-гомеостаз эндометрия и миометрия. Выявленные изменения могут негативно влиять на репродуктивную функцию и представляют собой ранее недооценённый экологический фактор риска для женщин репродуктивного возраста.*

Ключевые слова: *угарный газ, матка, эндометрий, окислительный стресс, гипоксия, малоновый диальдегид, супероксиддисмутаза, репродуктивная токсикология, экспериментальная морфология.*