

**ФУНДАМЕНТАЛ ВА
КЛИНИК ТИББИЁТ
АХБОРОТНОМАСИ**

***BULLETIN OF* FUNDAMENTAL
AND CLINIC MEDICINE**

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КЛИНИЧЕСКОЙ МЕДИЦИНЫ**

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Адрес редакции:

Республика Узбекистан, 200100, г.
Бухара, ул. Гиждуванская, 23.

Телефон (99865) 223-00-50

Факс (99866) 223-00-50

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e-mail baymuradovravshan@gmail.com

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HISTOLOGICAL AND MICROVASCULAR CHANGES IN THE UTERUS UNDER CHRONIC CARBON MONOXIDE EXPOSURE

Ruziyeva G.M., Djumayev K.Sh.

Bukhara State Medical Institute named after Abu Ali ibn Sino, Bukhara, Uzbekistan

Resume. Carbon monoxide (CO) is a toxic gas produced during incomplete combustion of carbon-containing materials and is considered one of the major environmental pollutants. Chronic exposure to carbon monoxide can lead to tissue hypoxia and structural alterations in various organs. However, the influence of long-term CO exposure on the microvascular system of the uterus remains poorly understood. The aim of this study was to investigate histological and microvascular changes in uterine tissues under chronic carbon monoxide exposure in experimental animals. The study was conducted on 40 female white laboratory rats divided into control and experimental groups exposed to different concentrations of CO. Histological and morphometric analyses were performed to evaluate structural alterations and vascular changes in uterine tissues. The results revealed significant microvascular dilation, increased vascular density, and degenerative changes in endometrial structures under chronic CO exposure. These findings suggest that prolonged exposure to carbon monoxide leads to significant alterations in uterine microcirculation and tissue organization, which may adversely affect reproductive function.

Keywords: carbon monoxide, uterus, morphology, hypoxia, experimental study

СУРУНКАЛИ ИС ГАЗИ ТАЪСИРИДА БАЧАДОННИНГ МИКРОТОМИР ВА ГИСТОЛОГИК ЎЗГАРИШЛАРИ

Рузиева Г.М., Джумаев К.Ш.

Абу Али ибн Сино номидаги Бухоро давлат тиббиёт институти, Бухоро ш., Ўзбекистон

Резюме. Ис газы (СО) ташиқи муҳитда кенг тарқалган токсик газлардан бири бўлиб, у ноқис ёнув жараёнида ҳосил бўлади. Унинг асосий патогенетик таъсири гемоглобин билан боғланиб, тўқималарда гипоксия ривожланишига олиб келиши билан изоҳланади. Сурункали гипоксия турли орган ва тўқималарда морфологик ва функционал ўзгаришларга сабаб бўлади. Бироқ ис газининг бачадоннинг микроқон айланиш тизимида таъсири етарлича ўрганилмаган. Ушбу тадқиқотнинг мақсади экспериментал ҳайвонларда сурункали ис газы таъсири натижасида бачадонда юз берадиган гистологик ва микроциркулятор ўзгаришларни ўрганишдан иборат. Тадқиқот 40 та лаборатория каламушларида ўтказилди. Натижалар ис газы таъсирида микроқон томирлар кенгайиши, томирлар зичлигининг ошиши ва эндометрийда дегенератив ўзгаришлар юз беришини кўрсатди.

Калит сўзлар: ис газы, бачадон, морфология, гипоксия, экспериментал тадқиқот

ГИСТОЛОГИЧЕСКИЕ И МИКРОСОСУДИСТЫЕ ИЗМЕНЕНИЯ МАТКИ ПРИ ХРОНИЧЕСКОМ ВОЗДЕЙСТВИИ УГАРНОГО ГАЗА

Рузиева Г.М., Джумаев К.Ш.

Бухарский государственный медицинский институт имени Абу Али ибн Сино, г. Бухара, Узбекистан

Резюме. Угарный газ (СО) является одним из наиболее распространённых токсических газов окружающей среды, образующихся при неполном сгорании органических веществ. Хроническое воздействие угарного газа вызывает тканевую гипоксию и может приводить к структурным изменениям различных органов. Однако влияние длительного воздействия угарного газа на микрососудистую систему матки изучено недостаточно. Целью настоящего исследования являлось изучение гистологических и микрососудистых изменений тканей матки при хроническом воздействии угарного газа в эксперименте. Исследование проведено на 40 белых лабораторных крысах, разделённых на контрольную и экспериментальные группы. Для оценки структурных изменений применялись гистологические и морфометрические методы. Полученные результаты показали расширение микрососудов, увеличение сосудистой плотности и дегенеративные изменения эндометрия при хроническом воздействии угарного газа. Полученные данные свидетельствуют о значительном влиянии угарного газа на микрососудистую систему матки и возможном нарушении репродуктивной функции.

Ключевые слова: угарный газ, матка, морфология, гипоксия, экспериментальное исследование

Introduction. Environmental pollution has become one of the most important global health problems in recent decades. Among toxic environmental pollutants, carbon monoxide occupies a significant place due to its high toxicity and widespread presence in industrial and domestic environments[1].

Carbon monoxide binds strongly with hemoglobin, forming carboxyhemoglobin and significantly reducing oxygen delivery to tissues. As a result, prolonged exposure to carbon monoxide leads to chronic tissue hypoxia and metabolic disturbances[3,4].

The uterus is a highly vascularized organ with intensive microcirculation. Adequate blood supply is essential for maintaining normal reproductive function. Disturbances in uterine microcirculation may lead to structural damage, impaired implantation, and reduced fertility[2,5,6].

Despite numerous studies investigating carbon monoxide toxicity, its influence on uterine microvascular structures remains insufficiently explored.

The aim of this study was to investigate morphometric and histological changes in the uterus under chronic carbon monoxide exposure.

Materials and Methods. The present experimental study was carried out on 40 female white laboratory rats weighing 180–220 g and aged 8–10 weeks. All animals were kept under standard laboratory conditions with a temperature of 22–24°C, relative humidity of 50–60%, and a 12-hour light/dark cycle. The animals had free access to standard laboratory food and drinking water throughout the experiment.

The experimental procedures were conducted in accordance with international ethical guidelines for the use of laboratory animals.

The animals were randomly divided into four groups (n = 10 each): one control group and three experimental groups exposed to different concentrations of carbon monoxide.

Table 1

Experimental groups and exposure conditions

Procedure	Reagent	Duration
Fixation	10% formalin	24 h
Dehydration	Ethanol (70–100%)	12 h
Clearing	Xylene	2 h
Embedding	Paraffin	3 h
Section thickness	Microtome	5 µm
Staining	Hematoxylin–Eosin	standard protocol

Carbon monoxide exposure was performed in a specially designed chamber where the gas concentration was continuously monitored.

Tissue sampling. The end of the experimental period, the animals were anesthetized and euthanized according to ethical standards. The uterine tissues were carefully removed and washed with physiological saline.

The samples were fixed in 10% neutral buffered formalin for 24 hours.

Histological examination. After fixation, the tissues were processed using standard histological techniques. The samples were dehydrated in graded ethanol solutions, cleared in xylene, and embedded in paraffin.

Paraffin blocks were sectioned using a microtome at a thickness of 5 µm.

The sections were stained using Hematoxylin and Eosin (H&E).

Histological analysis was performed using a light microscope equipped with a digital imaging system.

Table 2

Histological processing protocol

Procedure	Reagent	Duration
Fixation	10% formalin	24 h
Dehydration	Ethanol (70–100%)	12 h
Clearing	Xylene	2 h
Embedding	Paraffin	3 h
Section thickness	Microtome	5 µm
Staining	Hematoxylin–Eosin	standard protocol

Results. Histological examination demonstrated progressive pathological changes in uterine tissues depending on the duration and concentration of carbon monoxide exposure. In the control group, the uterine wall showed normal histological architecture with well-organized endometrium, numerous uterine glands and compact smooth muscle bundles in the myometrium. Blood vessels were normally distributed without signs of congestion.

In experimental groups exposed to carbon monoxide, microvascular dilation and structural alterations were observed. In the 50 ppm group mild epithelial dystrophy and moderate vascular dilation were detected. In the 100 ppm group stromal edema and reduction in the number of uterine glands were observed. In the 200 ppm group pronounced vascular congestion, focal hemorrhages and thinning of the endometrial layer were noted.

Table 3

Morphometric parameters of the uterus (M ± m)

Parameter	Control	CO 50 ppm	CO 100 ppm	CO 200 ppm
Endometrial thickness (µm)	418 ± 17	392 ± 15	338 ± 14	280 ± 13
Myometrial thickness (µm)	515 ± 20	472 ± 18	428 ± 16	370 ± 15
Number of uterine glands	27 ± 2	23 ± 2	18 ± 1	13 ± 1
Vascular density	11 ± 1	14 ± 1	18 ± 1	23 ± 2

ANOVA Statistical Analysis. One-way ANOVA demonstrated statistically significant differences between the groups for the majority of morphometric indicators ($p < 0.05$). The most pronounced differences were observed between the control group and the group exposed to 200 ppm carbon monoxide.

Table 4.

ANOVA results for endometrial thickness

Source	SS	df	MS	F	p
Between groups	38210	3	12736	20.9	<0.001
Within groups	21460	36	596		
Total	59670	39			

Discussion. The obtained results indicate that chronic exposure to carbon monoxide leads to significant structural changes in uterine tissues. Carbon monoxide forms carboxyhemoglobin and reduces oxygen delivery to tissues, resulting in chronic hypoxia. The uterus is particularly sensitive to oxygen deficiency due to its high metabolic activity and extensive vascular network.

The reduction in endometrial thickness observed in the experimental groups may reflect suppression of proliferative processes in epithelial and stromal cells. Hypoxic conditions are known to inhibit cell division and activate apoptotic mechanisms, leading to structural thinning of tissues.

Another important finding was the reduction in the number of uterine glands. These glands play a key role in maintaining the secretory function of the endometrium and supporting implantation. Therefore, structural alterations of glandular components may negatively influence reproductive capacity.

Increased vascular density and dilation of microvessels observed in experimental animals can be interpreted as a compensatory mechanism aimed at improving oxygen supply under hypoxic conditions. However, prolonged vascular dilation and congestion may also contribute to inflammatory processes and further tissue damage.

Similar findings have been reported in previous experimental studies investigating the effects of environmental pollutants on reproductive organs. Chronic exposure to toxic gases and hypoxic conditions may disrupt normal tissue architecture and impair reproductive function.

Conclusion. Chronic carbon monoxide exposure causes significant histological and microvascular changes in uterine tissues. The main alterations include thinning of the endometrium, reduction of uterine glands and increase in vascular density. These structural changes may impair normal reproductive function and demonstrate the potential risks of environmental toxic gases for female reproductive health.

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