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CHRONIC CARBON MONOXIDE EXPOSURE AND UTERINE REMODELING

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Resume. In recent years, the problem of the impact of adverse environmental factors on reproductive health has become increasingly relevant. One of the most common toxic agents is carbon monoxide (CO), which is formed during incomplete combustion of carbon-containing substances and is widely found in urban areas, industrial production and domestic sources. 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, reproductive organ.

Introduction

The uterus is a highly sensitive organ to hypoxic and metabolic influences, since its functional state directly depends on adequate blood supply, hormonal balance and regulation of cell proliferation[1]. Despite the available data on the systemic effects of carbon monoxide, issues related to its effects on the morphological and morphometric state of the uterus remain insufficiently studied. In particular, changes in the thickness of the endometrium, the state of the glandular apparatus, and tissue architectonics during prolonged exposure to CO require clarification[2].

Of particular interest is the study of morphometric indicators, which make it possible to quantify the degree of structural disorders and identify early signs of pathological changes[3,4]. In addition, in modern conditions, the importance of finding effective methods for correcting identified disorders is increasing, aimed at reducing the severity of oxidative stress, improving microcirculation and restoring regenerative processes in tissues[5,6].

The aim of this study was to compare the morphological and morphometric changes in the uterus during chronic exposure to carbon monoxide, as well as to substantiate approaches to their correction.

Materials and Methods

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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
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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

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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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