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CARBON MONOXIDE-INDUCED UTERINE DAMAGE

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Abstract
Carbon monoxide (CO) is one of the most common toxic gases formed during incomplete combustion and poses a serious threat to both human and animal health. The main pathogenic mechanism of carbon monoxide is associated with its high affinity to hemoglobin, leading to tissue hypoxia. Chronic hypoxia may cause structural and functional alterations in various organs and tissues. However, the effect of carbon monoxide on the female reproductive system, particularly on uterine tissues, remains insufficiently studied. The aim of this study was to investigate morphological and morphometric changes in the uterus under chronic exposure to carbon monoxide in experimental animals.

Keywords: Carbon monoxide, uterus, morphology, hypoxia, experimental study, reproductive organ.

Introduction

The female reproductive system is particularly sensitive to hypoxic conditions and oxidative stress [1]. The uterus plays a crucial role in reproductive physiology, including implantation, embryo development, and maintenance of pregnancy. Normal uterine function depends on adequate blood supply, balanced cellular proliferation, and structural integrity of the endometrium and myometrium [4].

Disturbances in oxygen supply and increased oxidative stress may lead to structural remodeling of uterine tissues and impairment of reproductive function.

Several experimental studies have demonstrated that environmental toxicants and hypoxic conditions may induce oxidative stress and morphological alterations in reproductive organs[3,5]. However, the influence of chronic carbon monoxide exposure on oxidative stress processes and structural changes in uterine tissues remains insufficiently investigated[2]. Understanding the mechanisms underlying these changes is important for evaluating the potential reproductive risks associated with environmental exposure to toxic gases[6].

The aim of the study was to investigate oxidative stress and morphological alterations in uterine tissues during chronic carbon monoxide exposure in experimental animals.

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

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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.
- Histological processing protocol

Results

Morphological analysis demonstrated progressive structural alterations in uterine tissues with increasing carbon monoxide concentration.

The presented graphs reflect morphological changes in the uterine endometrium during chronic exposure to carbon monoxide, and both indicators show a clear dose-dependent negative trend.

The graph shows a change in the number of uterine glands, which also decreases sequentially as the concentration of carbon monoxide increases. In the control group, the number of glands is about 28, which corresponds to the normal histoarchitectonics of the endometrium. At 50 ppm, the indicator decreases to about 24, which indicates an initial violation of the glandular apparatus. At 100 ppm, the number of glands decreases to 19, reflecting deeper morphological changes and, probably, a reduction in the functional activity of the endometrium. At 200 ppm, the number of glands reaches a minimum

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value of about 14, which indicates a pronounced depletion of the glandular component and may be associated with a violation of secretory function and a decrease in endometrial receptivity.

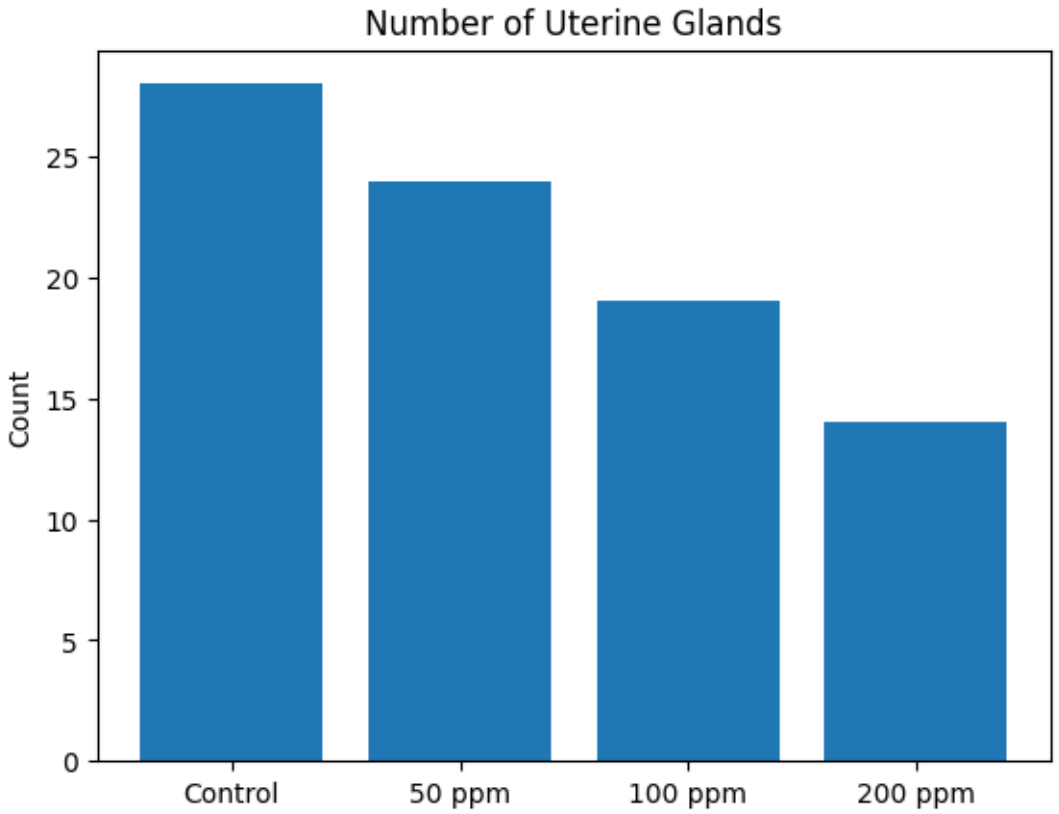

Figure 1. Changes in uterine gland number.

Taken together, both graphs demonstrate that chronic exposure to carbon monoxide leads to progressive thinning of the endometrium and a decrease in the number of uterine glands, and the changes are dose-dependent.

Conclusion

Thus, Chronic carbon monoxide exposure causes significant histological and morphometric changes in uterine tissues, including thinning of the endometrium and reduction in glandular structures. These data confirm the negative effect of CO on the morphofunctional state of the uterus, probably mediated by hypoxia, impaired microcirculation and increased oxidative stress, which in the future may lead to a decrease in reproductive function.

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