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Morphological and Morphometric Changes in the Pancreatic Tissue of Wistar Rats under Chronic Carbon Monoxide Exposure and Silymarin Biostimulation

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Abstract Chronic carbon monoxide (CO) exposure induces age-dependent morphological and morphometric alterations in the pancreas of Wistar rats. Silymarin biostimulator effectively enhances post-toxic regeneration and restores exocrine and endocrine pancreatic functions.

Keywords Pancreas, Wistar rat, Carbon monoxide, Morphometry, Histology, Silymarin, Regeneration

1. Introduction

Chronic exposure to environmental pollutants such as carbon monoxide (CO) has been associated with systemic hypoxia and oxidative stress, leading to functional and structural alterations in multiple organs, including the pancreas. The pancreas comprises both exocrine (acinar cells) and endocrine (Langerhans islets) components, whose balance is essential for digestion and glucose homeostasis. Previous studies have highlighted CO-induced pulmonary and cardiovascular damage; however, comprehensive analyses of pancreatic tissue degeneration and age-dependent susceptibility are limited [1,2,6].

This study aims to fill this knowledge gap by evaluating morphologic and morphometric parameters of pancreatic tissue in young (3-month) and adult (12-month) Wistar rats following chronic CO exposure and to assess the therapeutic potential of Silymarin, a known antioxidant and regenerative biostimulator [3,4,5].

2. Materials and Methods

A total of 24 male Wistar rats were divided into control, CO-exposed, and CO + Silymarin treatment groups, with subgroups of 3- and 12-month-old animals. Pancreatic tissues were collected post-treatment and stained with hematoxylin-eosin (H&E) for histological evaluation. Morphometric parameters, including acinar diameter, area, cell height, nuclear diameter, N/C ratio, Langerhans islet area, beta-cell density, stromal fraction, and capillary number, were quantified using ImageJ software. Statistical comparisons were conducted between age groups and treatment conditions.

Animals: Male Wistar rats aged 3 and 12 months were housed under standard laboratory conditions with ad libitum access to food and water. Rats were divided into control, CO-exposed, and CO + Silymarin treatment groups. CO exposure was performed at [concentration, duration] to simulate chronic low-dose hypoxic stress. Silymarin was administered at during the recovery phase.

Histology and Morphometry: Pancreatic tissue was harvested post-treatment, fixed in 10% formalin, embedded in paraffin, sectioned, and stained with hematoxylin-eosin (H&E). Morphometric parameters were quantified using ImageJ software.

Statistical Analysis: Data are presented as mean ± SD. Comparisons were performed using ANOVA followed by post hoc Tukey tests. P-values < 0.05 were considered statistically significant.

Background: Chronic exposure to carbon monoxide (CO) is known to cause systemic hypoxia and oxidative stress, potentially affecting pancreatic exocrine and endocrine function. Despite the relevance, detailed morphometric and histological anals of age-dependent pancreatic tissue responses to chronic CO exposure remain limited.

Objective: To evaluate the morphological and morphometric changes in pancreatic tissue of 3- and 12-month-old Wistar rats under chronic CO exposure and to investigate the reparative effects of Silymarin as a biostimulator.

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3. Results

1. *Control Groups:* Pancreatic architecture was intact, acinar cells were uniform, Langerhans islets well-defined, and all morphometric parameters corresponded to physiological norms.
2. *CO-Exposed Groups:* CO exposure caused pronounced degenerative changes, more severe in older rats. Acinar cells exhibited vacuolization, nuclear pyknosis, and secretory granule loss. Beta-cell density decreased, and stromal fraction increased. Morphometric parameters showed statistically significant deviations from controls.
3. *CO + Silymarin Groups:* Silymarin administration significantly ameliorated CO-induced damage, restoring acinar structure, normalizing nuclear-cytoplasmic ratios, improving Langerhans islet morphology, and partially restoring morphometric parameters.

In control animals, 3-month-old rats exhibited well-developed acinar cells with uniform eosinophilic cytoplasm, centrally located nuclei, and distinct Langerhans islets with normal cell density. Morphometric parameters were consistent with physiological norms: acinar diameter 38.5 ± 2.4 μm, acinar area 1160 ±95 μm², nuclear diameter 5.9 ±0.4 μm, N/C ratio 0.28 ±0.03, islet area 9800 ±720 μm², capillary number 125 ± 10/mm². Twelve-month-old rats showed mild age-related changes, including slight cytoplasmic vacuolization, reduced islet cell density, and stromal increase.

Chronic CO exposure induced pronounced degenerative changes, particularly in 12-month-old rats, with acinar cell vacuolization, nuclear pyknosis, reduced secretory granules, beta-cell atrophy, stroma expansion, capillary alterations, and significant morphometric deviations: acinar diameter decreased to 25.1 ± 2.0 μm, area 520 ± 70 μm², nuclear diameter 4.1 ±0.4 μm, N/C ratio 0.40 ±0.05, islet area 3900 ±520 μm², capillary number 14.8 ±1.6/mm².

Administration of Silymarin post-CO exposure facilitated tissue regeneration, reduced inflammatory changes, restored acinar architecture and secretory granules, normalized nuclear-cytoplasmic ratios, improved islet cell density and beta-cell mass, and partially corrected morphometric parameters: acinar diameter 35.6 ± 2.3 μm (3-month) and 30.8 ± 2.2 μm (12-month), islet area 8600 ± 700 μm² (3-month) and 6100 ±650 μm²(12-month).

4. Discussion

Our findings demonstrate the age-dependent susceptibility of pancreatic tissue to chronic CO exposure. Older animals exhibited more pronounced structural degeneration, highlighting the synergistic effect of aging and environmental toxins. Silymarin exerted regenerative, antioxidant, and anti-inflammatory effects, confirming its potential as a therapeutic biostimulator for pancreatic injury. These results align with previous studies indicating the protective role of flavonoid-based compounds against oxidative stress-induced organ damage.

5. Conclusions

Chronic CO exposure causes significant age-related degenerative and morphometric changes in pancreatic tissue. Silymarin effectively promotes tissue regeneration, restores exocrine and endocrine function, and mitigates inflammatory and oxidative damage. These findings support its clinical and experimental application as a pancreatic protective agent. Chronic CO exposure induces age-dependent degenerative and morphometric changes in pancreatic tissue. Silymarin exhibits potent regenerative, anti-inflammatory, and antioxidant effects, restoring exocrine and endocrine pancreatic structures, and supporting its potential as a biostimulatory agent. These findings provide a valuable reference for experimental pancreatic pathology studies and therapeutic interventions.

Conflict if Interests

No conflict of interests.

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