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STROMAL REMODELLING AND CELLULAR REDISTRIBUTION IN REGIONAL LYMPH NODES FOLLOWING CHRONIC COMBINED PARTICULATE AND TOBACCO-SMOKE INHALATION

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Relevance. Fine particulate matter (PM2.5) and tobacco smoke are among the leading global environmental risk factors, and their combined inhalation places a dual burden on the immune system. While the effects of each agent on the lung have been widely studied, the fate of the connective-tissue framework and the effector-cell composition of the draining lymph nodes under their combined action is far less understood. Because inhaled particulates accumulate in lung-associated lymph nodes and remodel their architecture with age, the capsule, trabeculae and medullary effector compartment represent an informative but underexplored read-out of chronic injury. This thesis focuses on stromal remodelling and the redistribution of the cellular population rather than on the follicular apparatus alone.

Aim. To characterize the changes in capsule thickness, the cortex-to-medulla balance and the effector-cell composition (macrophages, plasmocytes, reticular cells) of tracheobronchial and mediastinal lymph nodes in rats of three ages under combined PM2.5 and tobacco-smoke exposure, and to assess their biocorrection with Althaea officinalis L.

Materials and methods. Outbred white rats aged 6, 12 and 18 months were allocated to control, combined-exposure and biocorrection (Althaea officinalis infusion, 100 mg/kg orally) groups. Lymph nodes were fixed in 10% neutral buffered formalin, paraffin-embedded, sectioned at 4–5 µm and stained with haematoxylin and eosin. Capsule thickness, cortex and medulla areas and the counts of small, medium and large lymphocytes, macrophages, plasmocytes and reticular cells within a standardized region of interest were quantified in QuPath v0.6.0, and lymphocyte density was computed.

Results. Combined exposure produced pronounced remodelling of the external framework: capsule thickness increased from 21.4, 24.099 and 28.599 µm in controls to 31.2, 35.801 and 42.7 µm — a rise of 45.8%, 48.6% and 49.3% — indicating compensatory stromal sclerosis that deepened with age. In parallel, the cortex contracted (by 37.2–56.4%) while the medulla area increased relatively,

reflecting a shift of the parenchymal balance away from the B-cell-rich cortex. The cellular composition was redistributed: small lymphocytes fell by 31.2%, 38.7% and 46.4%, whereas macrophages (from 24 to 58, 67 and 79), plasmocytes (from 19 to 31, 39 and 47) and reticular cells rose steadily, and lymphocyte density declined from 1325 to 884, 731 and 650 $\times 10^3$ cells/mm^2. This reciprocal pattern - a falling lymphocyte pool against an expanding mononuclear-phagocyte and plasma-cell compartment - is characteristic of oxidative-stress- and cytokine-driven chronic injury. Biocorrection reduced capsule thickness toward normal (24.301, 27.2 and 31.199 μm), improved the cortex-to-medulla balance and partially restored the cellular profile; at 18 months, for example, small lymphocytes recovered from 294 to 476 and macrophages fell from 79 to 41. The incomplete normalization at the oldest age indicated an age-limited reparative capacity.

Conclusion. Chronic combined PM2.5 and tobacco-smoke inhalation drives progressive capsular thickening and a shift of the lymph-node cellular composition from a lymphocyte-dominated toward an effector-cell-dominated profile, most marked at 18 months. Althaea officinalis biocorrection attenuates stromal remodelling and partially restores the cellular balance, acting as a protective and partially reparative agent whose efficacy declines with age.

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