REMODELLING OF THE ADRENAL ZONA FASCICULATA AND CORTICOCYTE LIPID DEPLETION AS INDICATORS OF STEROIDOGENIC STRESS UNDER MYCOBACTERIAL EXPOSURE

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Relevance. The adrenal cortex is a key effector of the stress and immune-endocrine response, and its zona fasciculata, together with the lipid content of corticocytes, serves as a morphological indicator of steroidogenic activity. Quantifying these parameters under chronic mycobacterial inflammation clarifies how the gland adapts to sustained immune stress.

Aim. To evaluate the age-dependent morphometric remodelling of the adrenal cortex — zona fasciculata thickness, corticocyte lipid content, sinusoidal calibre and immune-cell density — in rats exposed to mycobacteria.

Materials and methods. Outbred male rats aged 4 and 9 months (n=20 per group) were studied. Experimental animals received a single subcutaneous injection of Freund's adjuvant containing killed mycobacteria; intact animals served as controls. Adrenal sections (haematoxylin–eosin) were used to quantify five morphometric parameters (M±m; $p<0.05$).

Results. Mycobacterial exposure thinned the zona fasciculata and sharply reduced corticocyte lipid content, while sinusoid diameter, macrophage and lymphocyte counts increased at both ages. In 4-month animals the changes were predominantly compensatory and reversible; in 9-month animals a more destructive response prevailed, with vacuolisation and fatty dystrophy of fasciculata cells, sinusoidal dilation and hyperaemia (Table 1).

Table 1. Main morphometric parameters of the adrenal cortex (M±m)

Parameter	Control 4 mo	FA 4 mo	Control 9 mo	FA 9 mo
Zona fasciculata thickness, µm	425±18	390±17	470±20	430±19
Lipid granules in corticocytes, %	23±3	14±2	17±2	12±1.5
Sinusoid capillary diameter, µm	7.1±0.5	8.2±0.6	7.8±0.6	8.6±0.7
Macrophages / Lymphocytes, per field	1–3 / 2–5	4–6 / 6–8	2–4 / 3–6	5–7 / 6–9

Discussion. The parallel thinning of the zona fasciculata and depletion of corticocyte lipid indicate accelerated mobilisation of the cholesterol-ester substrate for glucocorticoid synthesis, i.e. functional activation rather than simple atrophy. The concomitant sinusoidal dilation and rise in macrophage and lymphocyte counts reflect an intra-adrenal immune-inflammatory reaction, in which resident macrophages participate in the local regulation of steroidogenesis. The more destructive phenotype at 9 months — vacuolisation and fatty dystrophy of fasciculata cells with hyperaemia — is consistent with a transition from a compensatory to a decompensatory phase and with the reported vulnerability of the ageing adrenal cortex to sustained inflammatory stress. Thus, zona fasciculata thickness and corticocyte lipid content act as sensitive morphometric indicators of the steroidogenic response to chronic mycobacterial inflammation.

Conclusion. Mycobacteria induce a reproducible, age-modified remodelling of the adrenal cortex; fasciculata thinning with lipid depletion reflects activated steroidogenesis, and the shift from compensation at 4 months towards decompensation at 9 months indicates reduced adaptive reserve of the older gland.

Keywords: *adrenal cortex, zona fasciculata, corticocyte lipid, morphometry, mycobacteria, steroidogenic stress.*

References

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