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# HISTOLOGICAL REMODELING OF THE THYMUS UNDER STRESS-INDUCED SECONDARY IMMUNODEFICIENCY

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#### Relevance

The increasing prevalence of psychosocial stressors, malnutrition, and environmental factors has significantly contributed to the development of secondary immunodeficiency worldwide.

The thymus, as the central organ of T-lymphocyte maturation, is one of the most stress-sensitive structures. Understanding thymic morphological damage under stress is crucial for early diagnosis, prevention, and development of therapeutic strategies in clinical immunology and pathomorphology.

#### **Objective**

To investigate stress-induced histological and cellular changes in the thymus based on experimental models simulating secondary immunodeficiency.

#### **Materials and Methods**

Experimental stress conditions were modeled in rats using immobilization, cold exposure (4–6 °C), corticosteroid injections, and sleep deprivation over 14–30 days. After euthanasia, thymic samples were collected and analyzed with:

- ➤ Hematoxylin—eosin staining,
- > PAS reaction,
- > Van Gieson method,
- ➤ Immunohistochemistry (CD3, CD4, CD8, caspase-3),
- > Light and electron microscopy.

Morphometric measurements were used to quantify cortical and medullary alterations.

#### **Results**

- ➤ Significant degenerative and apoptotic processes were revealed:
- ➤ Massive lymphocyte apoptosis and necrosis in the cortical layer;
- > Medullary lymphocyte depletion;
- ➤ Vacuolization, wrinkling, and shrinkage of Hassall's corpuscles;
- > Stromal disintegration and perivascular edema;
- ➤ Microcirculatory disorders such as congestion, hemorrhage, and stasis;
- Fatty infiltration and diffuse thymic atrophy.

The main pathogenic mechanism was linked to glucocorticoid-induced caspase-mediated apoptosis of thymocytes. Structural reduction of lymphoid reserves can predispose to infection and autoimmunity.

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