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CYTOKINE PROFILE IN PROGRESSIVE HYPERGLYCEMIA: TNF-A, IL-6 AND IL-10 IN THE EXPERIMENT

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Introduction:
Diabetes mellitus and its primary characteristic, hyperglycemia, cause metabolic disturbances accompanied by intensified inflammatory processes. Levels of pro-inflammatory cytokines such as TNF-α and IL-6 increase, while anti-inflammatory cytokines like IL-10 decrease. This imbalance may exacerbate the complications of the disease.

Objective: To investigate the dynamics of hyperglycemia and associated changes in TNF-α, IL-6, and IL-10 cytokine levels in laboratory animals, as well as to analyze the relationships between these indicators.

Materials and Methods: In this study, hyperglycemia was modeled in laboratory rats. Levels of glucose and cytokines were measured at 30, 60, 90, and 120 days. Changes were analyzed using percentage and fold-change comparisons.

Results:
Throughout the duration of the experiment, we observed a significant elevation in blood glucose levels, increasing approximately threefold compared to baseline measurements. This substantial rise in glucose concentration provides a clear indication of the hyperglycemic state induced within the experimental model. Furthermore, analysis of cytokine profiles revealed a marked increase in the levels of both tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). TNF-α levels rose by a factor of 2 to 3, signifying a considerable activation of this potent pro-inflammatory mediator, while IL-6, another key cytokine involved in acute and chronic inflammation, also demonstrated a 2 to 3-fold increase. In stark contrast to these pro-inflammatory trends, interleukin-10 (IL-10), a crucial anti-inflammatory cytokine responsible for modulating immune responses and resolving inflammation, exhibited a notable decrease. The reduction in IL-

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10 levels was observed to be nearly proportional to the increases in TNF-α and IL-6, suggesting a systemic shift in the balance of inflammatory mediators. Collectively, these findings strongly indicate a deepening, or intensification, of inflammatory responses directly associated with the induced hyperglycemia, suggesting a feedback loop where elevated glucose contributes to heightened inflammation.

Conclusion:

Our research demonstrates that hyperglycemia, or elevated blood glucose, significantly enhances the activity of pro-inflammatory cytokines - such as TNF-α and IL-6 -hile simultaneously suppressing the production and function of anti-inflammatory cytokines like IL-10 in laboratory animals. This observation highlights the complex and often bidirectional interplay between metabolic disturbances, specifically hyperglycemia, and inflammatory processes within the body. For instance, the increased TNF-α could contribute to vascular dysfunction, while elevated IL-6 may exacerbate insulin resistance. The suppression of IL-10, which normally dampens inflammation, further amplifies this pro-inflammatory state. These results are crucial for a more comprehensive understanding of the pathophysiology of diabetes, a disease characterized by chronic hyperglycemia and associated inflammation. Specifically, they provide valuable insights into the mechanisms driving the development of diabetic complications, such as cardiovascular disease, neuropathy, and nephropathy. Moreover, these findings offer a strong rationale for developing and refining effective treatment strategies that target both metabolic control and the underlying inflammatory processes in individuals with diabetes, potentially leading to improved patient outcomes and a reduction in the burden of this widespread disease.