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CHANGES IN CYTOKINE PROFILE OF MACROPHAGES INDUCED BY CHRONIC STRESS IN INTACT AND TUMOR-BEARING RATS

Background. The prolonged psychoemotional stress results in the individual adaptive response ensured by the coordinated interactions among the nervous, endocrine, and immune systems. A key role in this process is played by the interaction between stress hormones and effector cells of innate immunity, particularly macrophages (Mph), which constitute the first line of host defense. **The aim** of this study was to experimentally investigate the effects of stress hormones on the cytokine spectrum produced by Mph of the intact and tumor-bearing rats. **Materials and Methods.** Wistar rats with transplanted Guerin carcinoma were used in a study. To induce experimental chronic stress, animals received dexamethasone or adrenaline (0.5 mg/kg body weight). Plasma levels of TNF-α, IL-6, and IL-10 were determined using ELISA. **Results.** In the intact animals, prolonged exposure to stress hormones resulted in significant alterations in the macrophage cytokine profile. Adrenaline administration was accompanied by a transient increase in TNF-α ($p < 0.05$) along with a simultaneous decrease in IL-10 ($p < 0.05$); subsequently, TNF-α production was suppressed against a marked increase in IL-10 synthesis. Dexamethasone induced an increased TNF-α production at later time points. In rats bearing Guerin carcinoma, the most pronounced changes in TNF-α and IL-10 production were observed following dexamethasone administration: on day 14, TNF-α levels significantly increased, followed by a sharp decline accompanied by the elevated IL-10 levels, reflecting Mph polarization from the M1 to the M2 phenotype. This indicates the development of immunosuppression and correlates with the active tumor growth. A significant increase in IL-6 production was observed on day 14 under both adrenaline and dexamethasone exposure. Subsequently, adrenaline suppressed IL-6 production, whereas dexamethasone stimulated it, which may indicate the development of glucocorticoid resistance and paradoxical enhancement of inflammation. **Conclusion.** The prolonged exposure to stress hormones may create conditions favorable for tumor progression through suppression of innate immune effectors and development of immunosuppression, as well as through paradoxical enhancement of inflammatory responses.

Keywords: chronic stress, Guerin carcinoma, macrophages, cytokines.

The relationship between stress and carcinogenesis has become an increasingly relevant area of research. Stress has been shown to exert a significant modulatory effect on the functioning of individual physiological systems as well as the organism as a whole, increasing not only the risk of malignant neoplasms but also potentially adversely affecting treatment outcomes. Prolonged psychoemotional

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stress leads to the formation of an individual adaptive response mediated by coordinated interactions among the nervous, endocrine, and immune systems. A crucial role in this process is played by the interactions between stress hormones and effector cells of innate immunity, particularly macrophages (Mph), which provide the first line of host defense.

The main mediators of stress are glucocorticoids and catecholamines. Generally, stress hormones exert immunosuppressive effects by inhibiting inflammatory processes. However, recent studies indicate the existence of more complex mechanisms. Under prolonged stress exposure, immune cell receptors may lose sensitivity to regulatory signals, leading to the paradoxical responses accompanied by enhanced inflammation and development of autoimmune disorders [1, 2].

Prolonged exposure to glucocorticoids and catecholamines results in substantial alterations in Mph functional activity. Stress hormones induce a shift in macrophage polarization from the proinflammatory M1 phenotype to the anti-inflammatory M2 phenotype. This shift is associated with the suppression of cytotoxic and phagocytic activities [3—5]. The changes in Mph polarization are accompanied by alterations in cytokine production, characterized by the decreased synthesis of proinflammatory cytokines (TNF-α, IL-1β, and IL-6) and increased production of anti-inflammatory cytokines (IL-10). In certain cases, prolonged cortisol exposure may lead to glucocorticoid resistance, characterized by a transition from immunosuppression to insensitivity to further hormonal regulation. One mechanism of short-term cortisol-mediated anti-inflammatory action involves modulation of the transcription factor NF-κB, which regulates the expression of proinflammatory cytokines (TNF-α, IL-1, IL-6), chemokines (e.g., CXCL8), and adhesion molecules (e.g., ICAM-1). NF-κB is considered a key mediator of inflammatory responses; its downregulation reduces transcription of proinflammatory genes and suppresses inflammation. However, prolonged cortisol exposure may reduce Mph receptor sensitivity or receptor density, resulting in the sustained production of the proinflammatory cytokines despite the elevated cortisol levels and leading to uncontrolled systemic inflammation [6—9].

Therefore, chronic stress can modulate immune responses toward either suppression or uncontrolled activation. This becomes particularly critical in the setting of tumor growth. Macrophages, as key effectors of antitumor immunity, play significant roles in angiogenesis, metastasis, and immunosuppression [10, 11]. In our previous studies, the prolonged exposure to adrenaline and dexamethasone caused profound impairment of the innate immune effectors, including natural killer cells and Mph. Initially, a transient activation phase was observed, followed by sustained suppression of activity. Under prolonged stress hormone exposure, Mph exhibited M2-like characteristics: decreased cytotoxic activity, increased arginase activity, and reduced NO production [12].

Based on these findings, the present study aimed to investigate experimentally the effects of stress hormones on the cytokine spectrum produced by Mph of intact rats and tumor-bearing animals.

Materials and Methods

Animals. The study was carried out on male Wistar rats, 2.5 months old, weighing 180—200 g, bred at the vivarium of R.E. Kavetsky Institute of Experimental Pathology, Oncology and Radiobiology of the National Academy of Sciences of Ukraine (IEPOR NASU). The animals were kept in the standard vivarium conditions with natural lighting and provided with food and water ad libitum. The rats were handled and kept in accordance with the standard international rules of biological ethics and the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes [13]. The permission to conduct research was approved by the Bioethics Commission of the IEPOR NASU (protocol No. 5 dated 06.05.2025). All animals underwent a preliminary 10-day quarantine before being involved in the study. After the adaptation period, the animals were weighed and divided into groups.

Tumor strain. Guerin carcinoma cells were obtained from the Bank of Cell Lines from Human and Animal Tissues of the IEPOR NASU. Tumor cell transplantation (1×10^6 cells in physiological solution) was performed subcutaneously in the pelvic region of the back.

Agents. Dexamethasone (i/v solution 4 mg/mL, "Lekhim-Kharkiv", Ukraine) and adrenaline (i/v solution 1.82 mg/mL, "Zdorovya", Ukraine) were used as stress hormones and administered at a dose

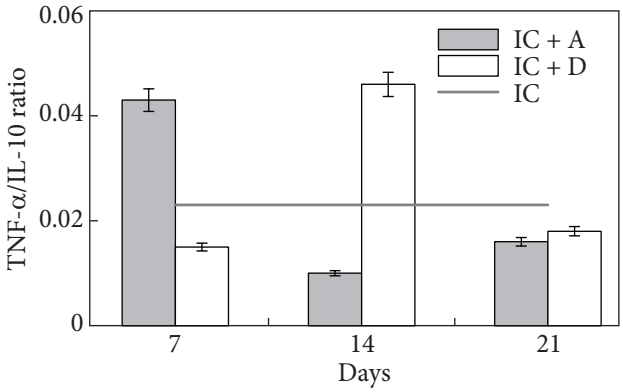

Fig. 1. Dynamics of the TNF-α/IL-10 ratio in the blood plasma of the intact Wistar rats under prolonged exposure to stress hormones

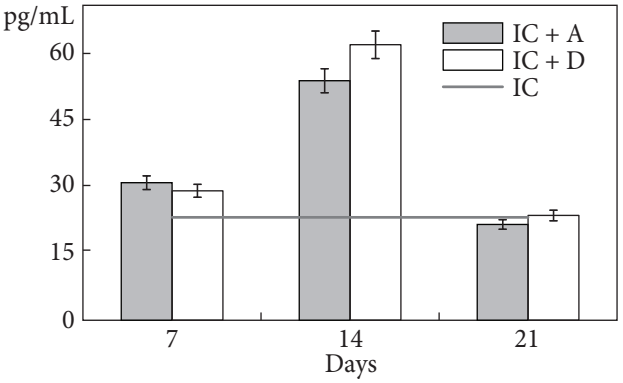

Fig. 2. Dynamics of the IL-6 content in the blood plasma of intact Wistar rats under prolonged exposure to stress hormones

of 0.5 mg/kg of body weight. The agents were administered for 12 days, starting from the 2nd day after the transplantation of Guerin carcinoma, every other day.

Experimental design. The study included the determination of changes in the production of pro- and anti-inflammatory cytokines by Mph of the intact and tumor-bearing Wistar rats in the setting of prolonged exposure to stress hormones. The doses and schedules of drug administration to intact rats and animals with tumors were the same.

6 groups were formed: "Intact control, IC" — intact rats injected with physiologic solution ($n = 15$); "IC + A" — intact rats injected with adrenaline ($n = 15$); "IC + D" — intact rats injected with dexamethasone ($n = 15$); "Tumor growth control, TGC" — rats with Guerin carcinoma injected with physiologic solution ($n = 15$); "Adrenaline" — rats with tumors injected with adrenaline ($n = 15$); "Dexamethasone" — rats with tumors injected with dexamethasone ($n = 15$).

The peripheral blood samples from experimental animals were taken, placed in heparin tubes (InterVacTechnology, Estonia), and spun at 1,500 *g* for 10 min. Plasma was collected, frozen, and stored at –20 °C.

The levels of cytokines IL-6, IL-10, and TNF-α in the peripheral blood plasma were measured on the 7th, 14th, and 21st days of the experiment.

Assessment of cytokine levels. Cytokine levels in the blood plasma samples were measured using commercial test systems Rat TNF-α ELISA Kit; Rat IL-6 ELISA Kit; and Rat IL-10 ELISA Kit (FineTest, China) according to the instructions of the manufacturer. The immunoenzyme reactions were read out at λ = 450 nm using an automatic StatFax 2100 reader (USA). The levels of cytokines (pg/mL) in rat blood plasma were determined using a calibration curve.

Statistical processing of the results was performed using the standard methods of variational statistics with GraphPad Prism 8.0.1 (Graphpad Software Inc., USA). The significance of the differences between the groups was assessed using Student's *t*-test and was considered significant at $p < 0.05$.

Results and Discussion

The administration of adrenaline and dexamethasone to the intact rats was associated with changes in Mph polarization and corresponding alterations in cytokine production. The TNF-α/IL-10 ratio reflects the direction of Mph polarization. Fig. 1 shows the dynamics of changes in the levels of the TNF-α/IL-10 ratio in the blood plasma of intact Wistar rats under prolonged exposure to stress hormones.

On day 7, adrenaline administration induced a transient increase in TNF-α levels (19.1 ± 1.8 pg/mL vs 10.3 ± 1.5 pg/mL in controls, $p < 0.05$), accompanied by a decrease in IL-10 (289.0 ± 37.3 pg/mL vs 439.7 ± 39.4 pg/mL, $p < 0.05$). Subsequently, TNF-α production was suppressed while IL-10 levels significantly increased. Dexamethasone stimulated TNF-α production on day 14 (22.2 ± 4.2 pg/mL vs 10.3 ± 1.5 pg/mL in controls, $p < 0.05$), whereas IL-10 remained at control levels. By day 21, cytokine profiles in both hormone-treated groups corresponded to the M2 phenotype.

A significant increase in IL-6 levels was observed on day 14 under both adrenaline (53.3 ± 3.1 pg/mL)

and dexamethasone (61.3 ± 7.5 pg/mL) exposure compared with controls (22.9 ± 1.4 pg/mL, $p < 0.05$). Thereafter, IL-6 levels returned to control values (Fig. 2).

Thus, data on the production of pro- and anti-inflammatory cytokines by intact rat Mph indicated that prolonged exposure to stress hormones is accompanied by significant changes in the functional activity of these cells. The observed changes imply the development of immunosuppression that could negatively affect the formation of an antitumor immune response.

In rats bearing Guerin carcinoma, similar dynamics of TNF-α and IL-10 production were observed, with more pronounced effects following dexamethasone administration (Fig. 3). On day 14, TNF-α levels significantly increased; by day 21, TNF-α levels decreased below those of both intact controls (2.2-fold) and tumor-bearing controls (2.1-fold) ($p < 0.05$), indicating a shift toward M2 polarization and systemic immunosuppression.

The IL-6 production in tumor-bearing rats increased significantly on day 14 following both adrenaline and dexamethasone administration (Fig. 4). Subsequently, adrenaline reduced IL-6 to the TGC group levels, whereas prolonged dexamethasone exposure maintained the elevated IL-6 levels. On day 21, the IL-6 levels exceeded those of intact rats by 1.6-fold, TGC by 3.5-fold, and adrenaline-treated animals by 2.3-fold ($p < 0.05$ for all comparisons). Such data most likely indicate a paradoxical increase in the inflammatory process under the influence of glucocorticoids, which may result in the development of autoimmune diseases or a more profound immunosuppressive state.

Notably, dexamethasone-treated animals demonstrated accelerated tumor growth. Fig. 5 shows data on the dynamics of growth of Guerin carcinoma in experimental rats of different groups. As can be seen from the presented data, by day 14 of tumor growth, the size of the primary tumor node was small and nearly identical in rats of all groups (the mean tumor weight ranged from 0.28 to 0.39 g), which may indicate the preservation of antitumor immune responses at this time point. Subsequently, in the TGC and Dexamethasone groups, a significant acceleration of tumor growth was observed (6.13 ± 1.02 g and 4.67 ± 0.45 g, respectively), coinciding with the marked suppression of

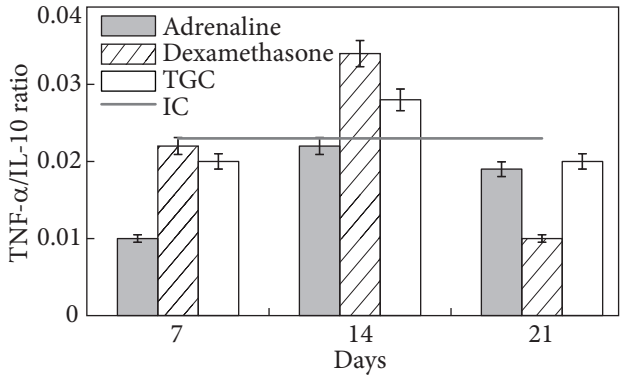

Fig. 3. Dynamics of the TNF-α/IL-10 ratio in the blood plasma of Wistar rats with Guerin carcinoma under prolonged exposure to stress hormones

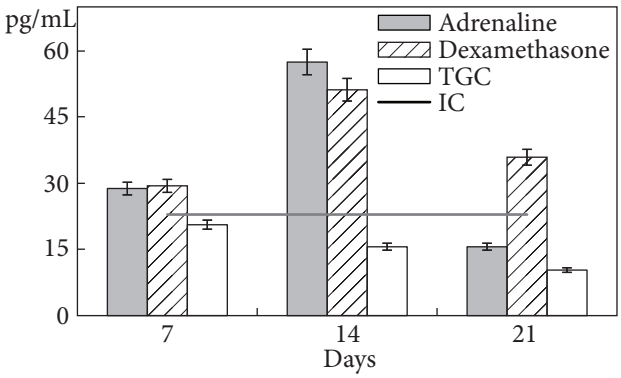

Fig. 4. Dynamics of the IL-6 content in the blood plasma of Wistar rats with Guerin carcinoma under prolonged exposure to stress hormones

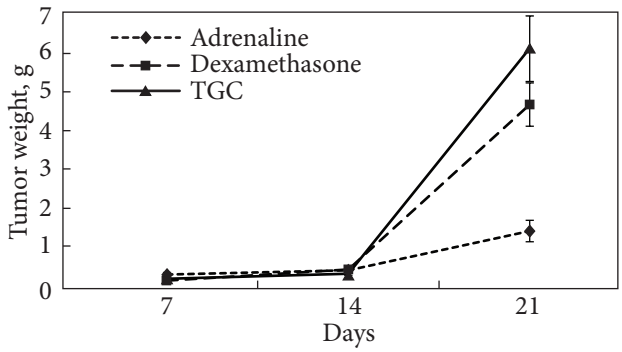

Fig. 5. Dynamics of Guerin carcinoma growth under conditions of modeled chronic stress

Mph activity and the development of an immunosuppressive state.

Thus, prolonged exposure to adrenaline and dexamethasone led to significant changes in the functional activity of Mph as one of the main effectors of natural antitumor resistance. However, the action of these agents differed somewhat. The activation of Mph in the intact rats under the influence of adrenaline occurred at earlier time points:

already on day 7, a significant increase in TNF-α production was observed, whereas the significant activation under dexamethasone was detected on day 14 of the experiment. This effect may be attributed to the functional characteristics and localization of the respective hormonal receptors. Surface membrane adrenergic receptors mediate a more rapid response than intracellular glucocorticoid receptors, whose activation involves gene transcription and is therefore slower [14, 15].

At the same time, the most pronounced changes in TNF-α and IL-10 production in both intact rats and tumor-bearing animals occurred under dexamethasone exposure: on day 14, a significant increase in TNF-α levels was observed, followed by their sharp decline against a background of rising IL-10 levels. This pattern reflects the dynamics of Mph polarization from the pro-inflammatory M1 phenotype toward the anti-inflammatory M2 phenotype. Such a process indicates the formation of immunosuppression, which correlated with active tumor growth on day 21.

Noteworthy are the changes in the production of another pro-inflammatory cytokine, IL-6. A significant increase in IL-6 production in rats with Guerin carcinoma was observed under the influence of both adrenaline and dexamethasone on day 14 of the experiment. Subsequently, adrenaline contributed to the suppression of IL-6 production. In contrast, prolonged dexamethasone exposure was accompanied by stimulation of IL-6 production: on day 21, the IL-6 levels exceeded those of intact rats by 1.6-fold, control rats with Guerin carcinoma by 3.5-fold, and rats treated with adrenaline by 2.3-fold ($p < 0.05$ for all groups). This may indicate the development of glucocorticoid resistance and paradoxical enhancement of the inflammatory process upon glucocorticoid exposure [6—9]. According to the literature, since IL-6 is a key activator of the STAT3 signaling pathway, its hyperproduction creates conditions favorable for tumor progression [16—18], which we also demonstrated in the Guerin carcinoma model.

In addition, the more pronounced immunomodulatory effect of dexamethasone may be associated with the differences in its pharmacodynamic profile from cortisol and the greater potency in influencing Mph. In particular, dexamethasone has a significantly higher affinity for glucocorticoid receptors than cortisol. On the one hand, this results in more pronounced suppression of the Mph production of pro-inflammatory cytokines (such as TNF-α, IL-1β, and IL-6) and expression of inducible nitric oxide synthase (iNOS), along with the increased production of anti-inflammatory cytokines (such as TGF-β1, IL-4, and IL-10) and enhanced arginase activity [19, 20]. On the other hand, the higher affinity and binding strength of dexamethasone to glucocorticoid receptors may provoke a loss of receptor sensitivity. As a consequence, a paradoxical enhancement of inflammation may develop even after discontinuation of the agent, which we observed when assessing IL-6 production dynamics in rats with the experimental tumor process.

Thus, the use of adrenaline and dexamethasone to model chronic stress–induced immunosuppression has demonstrated that prolonged hormonal imbalance may create favorable conditions for the accelerated tumor progression through both the suppression of the activity of innate immune effectors and the formation of immunosuppression, as well as the development of a paradoxical inflammatory response.

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ЗМІНИ ЦИТОКІНОВОГО ПРОФІЛЮ МАКРОФАГІВ, ІНДУКОВАНІ ХРОНІЧНИМ СТРЕСОМ У ІНТАКТНИХ ЩУРІВ ТА ПУХЛИНОНОСІЇВ

Стан питання. Наслідком тривалого психоемоційного навантаження є формування індивідуальної адаптивної реакції, яка забезпечується координованою взаємодією нервової, ендокринної та імунної систем. Ключову роль у цьому процесі відіграє взаємодія між гормонами стресу та клітинами-ефекторами природної імунної відповіді (зокрема, макрофагами), які забезпечують першу лінію захисту організму. **Мета** даної роботи — експериментально дослідити вплив гормонів стресу на спектр цитокінів, продукованих макрофагами інтактних щурів та тварин з модельним пухлинним процесом. **Матеріали та методи.** Дослідження проводили на щурах лінії Wistar; як модель пухлинного росту використана карцинома Герена. Для створення експериментальної моделі хронічного стресу тваринам вводили дексаметазон або адреналін (0,5 мг/кг ваги). Рівень цитокінів TNF-α, IL-6 та IL-10 в плазмі периферичної крові щурів визначали за допомогою ELISA. **Результати.** В інтактних тварин тривалий вплив стресорних гормонів призводив до значних змін спектру цитокінів, продукованих макрофагами. Введення адреналіну супроводжувалося транзиторним підвищенням вмісту TNF-α ($p < 0.05$) з одночасним зменшенням вмісту IL-10 ($p < 0.05$); в подальшому відбувалося пригнічення продукції TNF-α на тлі суттєвого зростання синтезу IL-10. Дексаметазон обумовлював збільшення продукції TNF-α в більш пізні терміни. У щурів з карциномою Герена найбільш виражені зміни в продукції TNF-α та IL-10 спостерігали при дії дексаметазону: на 14-ту добу — суттєве збільшення рівнів TNF-α, в подальшому — їх стрімке зниження на тлі зростання IL-10, що відображає динаміку поляризаційного стану макрофагів від M1 до M2 фенотипу. Такий процес свідчить про формування стану імуносупресії та корелює з активним ростом новоутворення. Суттєве зростання рівнів продукції IL-6 відбувалося під впливом адреналіну та дексаметазону на 14-ту добу експерименту. В подальшому адреналін сприяв пригніченню його продукції, дексаметазон — її стимулюванню, що може бути ознакою формування стану глюкокортикоїдної резистентності та парадоксального посилення запального процесу під впливом глюкокортикоїдів. **Висновки.** Тривалий вплив гормонів стресу може створювати сприятливі умови для прискорення пухлинного процесу за рахунок як пригнічення активності ефекторів неспецифічного імунітету та формування стану імуносупресії, так і формування парадоксальної реакції підсилення запального процесу.

Ключові слова: хронічний стрес, карцинома Герена, макрофаги, цитокіни.