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Immune Modulations by Glucocorticoids: From Molecular Biology to Clinical Research

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Message from the Guest Editors

Glucocorticoids are widely prescribed immune-suppressive drugs, but their use is limited by the severity of their side effects and the occurrence of resistance. The effects of glucocorticoids are mediated by the glucocorticoid receptor (GR), which acts as ligand-activated transcription factor, able to modulate gene transcription both positively and negatively. Traditionally, it was assumed that GRs interacting with other transcription factors, thereby inhibiting their activity, elicited the desired immune-suppressive effects and that GR binding to glucocorticoid response elements (GREs) in the DNA and subsequent transactivation of gene expression was responsible for the adverse effects. However, in recent years this view has been challenged and now appears to be a simplification. Furthermore, several mechanisms have been described that inhibit GR function and thereby cause glucocorticoid resistance. This Special Issue of *Cells* should further refine our current view of the mechanisms underlying the actions of glucocorticoids and the GR, aiming (eventually) at the development of novel immune-suppressive therapies, such as selective GR agonists and specific drug-targeting strategies.



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