AS15-01 - ANTIDEPRESSANT ACTION: ROLE OF GLUCOCORTICOID RECEPTOR

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Our research has contributed to the understanding of the mechanism underlying HPA axis and inflammation hyperactivity iin depression. We have proposed an explanatory model centred around the glucocorticoid receptor (GR), that is, one of the most important receptors and transcription factors governing the stress response. Glucocorticoid hormones, like cortisol in humans and corticosterone in rodents, are the final output of the HPA axis, and the main hormones involved in the stress response. By binding to the GR (and to the mineralocorticoid receptor, MR), cortisol effects its cellular actions, including the negative feedback regulation of the HPA axis (by which stress-induced activation of the HPA axis is followed by a rapid return to normal functioning), and the restrain of the inflammatory response (which maintains a physiological control on excessive immune processes). In this talk I will review our clinical and cellular study implicating a reduced function of the GR (glucocorticoid resistance in key depression-related phenomena, such as the increased HPA axis and inflammation, the reduction in neurogenesis, and the therapeutic action of antidepressants.

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