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Glucorticoid receptor in human cutaneous melanoma: immunohistochemical and immunofluorescence study

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GR is a nuclear receptor which, when activated by its specific ligand, can act as a transcription factor that binds to glucocorticoid response elements (GRE) or negative GRE. It affects inflammatory responses, differentiation and cell proliferation. The ligand activated glucocorticoid receptor induces a G1 cell cycle arrest or apoptosis in immature thymocytes and impairs proliferation of fibroblasts of undifferentiated mammary epithelial cells. It impairs proliferation and differentiation of neural progenitor cells in vivo and in vitro. Glucocorticoids are widely used in cancer therapy and have cell type-specific pro- or antiapoptotic effects. In melanoma, however, the antitumor activity of glucocorticoids remains an open question. A recent report demonstrated that in mouse embryo tissue and in human undifferentiated cells, cytoplasmic accumulation of GR is determined by nestin in conjunction with vimentin, copolymerised into an intermediate filament system, and that this anchoring of GR to the nestin/vimentin etheromeric complex is related to the maintenance of a high proliferation rate. The aim of this study was to analyse the expression of subcellular GR in cutaneous melanoma by immunofluorescence, immunohistochemistry and laser scanning confocal microscopy and to evaluate any effect in melanoma progression. The results will be discussed.

Keywords: Glucocorticoid receptor, human melanoma, immunohistochemistry, confocal microscopy.