Histone deacetylase inhibitors suppress transdifferentiation of gonadotrophs to prolactin cells and proliferation of prolactin cells induced by diethylstilbestrol in male mouse pituitary

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Evaluation Report of Dissertation

1. Evaluation of the research purpose.

Diethylstilbestrol (DES), an estrogen agonist, increases prolactin (PRL) cells through transdifferentiation of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) cells to PRL cells in adult male mouse pituitary. This study tried to elucidate the involvement of epigenetic regulation in DES-induced transdifferentiation of FSH and LH cells to PRL cells. Therefore, the research purpose is appropriate.

2. Evaluation of the research methods.

In this study, it was designed to examine the effect of DES on the state of histone H3 acetylation by the immunohistochemical signals for acetylated histone H3 at lysine 9 (H3K9ac), H3K18ac, and H3K23ac in PRL, LH and FSH cells with HDAC inhibitors (HDACi), sodium phenylbutyrate (NaPB) or valproic acid (VPA). The research method is also valid.

3. Evaluation of the analysis, interpretation and discussion.

As a result of analyzing with the above method, it is suggested that HDACi treatment restored the level of H3K9ac expression in these cells, and inhibited DES-induced increase in PRL cells. Furthermore, NaPB and VPA also abrogated the effects of DES on the population density of both FSH and LH cells. These results indicated that the acetylation level of histone H3 plays an important role in DES-induced transdifferentiation of FSH and LH cells to PRL cells, providing a new insight to regulate differentiation of pituitary cells at molecular level.

As stated above, the dissertation will greatly contribute to the field of cell biology, and the evaluators uniformly agree that the author should be awarded a Doctor of Philosophy in Medical Science.

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