D-11 Expression of Alkaline Phosphatases during Embryonic Development and Immature Stages of the Earthworm, *Eisenia andrei*

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Expression of alkaline phosphatases in developing embryo and mature worm of the earthworm, *Eisenia andrei* was investigated. The embryonic stages examined in this study appeared to have only one slow-moving form of alkaline phosphatase which had a different mobility from the intestinal alkaline phosphatases of the mature worm, suggesting that intestinal alkalline phosphatases of embryos may be different from mature forms. A surge in alkaline phosphatase activity after hatching is consistent with the expression of mature forms of intestinal alkaline phosphatase and this increase would be associated with postnatal differentiation of the intestine.

D-12 **Effects of Cortisol on Steroidogenesis and Apoptosis of Cultured Human Granulosa-luteal Cells.**

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The present study was performed to investigate whether there is direct effect of cortisol on ovarian steroidogenesis and to evaluate the hypothesis that cortisol may directly induce apoptosis of cultured human granulosa-luteal(GL) cells. After treatment with cortisol, concentrations of estradiol and progesterone secreted from GL cells into culture media were measured by chemiluminescence immuno assay(CIA) and ratio of apoptotic cells in cultured GL cells was detected by in situ apoptosis detection method. FSH treatment caused 5 fold increase in progesterone secretion compared to untreated controls, while no effect was observed in estradiol secretion. In contrast, progesterone secretion by GL cells treated with cortisol showed a dose-dependent decrease. This inhibiting action of cortisol on progesterone secretion was not overcome by co-treatment with FSH. Cortisol directly increased the number of apoptotic cells compared with controls. FSH treatment prevent the apoptotic cell death of GL cells in vitro, however, apoptosis induced by cortisol was not influenced by FSH. These results suggest that cortisol inhibit progesterone production and directly increase apoptotic cell death in cultured human GL cells.