Establishment and characterization of immortalized porcine 11β HSD1-hepatocytes

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**EP1: Adrenal cortex**

**Introduction**

Glucocorticoid, known as cortisol, is a steroid hormone essential to the maintenance of homeostasis, and is released in response to stress and low blood glucose concentration. It is converted from cortisone by 11β hydroxysteroid dehydrogenase type 1 (11β HSD1). The liver plays a major organ in metabolism, has numerous functions, mostly consists of hepatocytes, and is a principal target of cortisol. In murine model, it was observed that too much cortisol or overexpression of 11β HSD1 induced obesity and the insulin resistance that accompanies metabolic syndrome.

**Materials and Methods**

In our previous study, 11β-HSD1-transgenic (TG) fibroblasts were established, and then the porcine model was generated by SCNT using those fibroblasts. Hepatocytes overexpressing 11β-HSD1 obtained from liver of this porcine model and, in vitro cultured. However, primary hepatocytes show short life span or low proliferation rate. To overcome these problems, SV40 large T antigen, oncogene, was transduced into primary 11β-HSD1-TG hepatocytes and those cells were immortalized.

**Results**

Immortalized 11β-HSD1-TG hepatocytes shows restored morphology, more rapid proliferation rate, and more expression of 11β-HSD1 than primary ones. Immortalized 11β-HSD1-TG hepatocytes increase the expression of gluconeogenic genes including G6Pase and PEPCK by cortisol treatment.

**Conclusions**

These immortalized cells maybe be useful for studying traits and potential pharmacotherapeutic drugs for metabolic disorders induced by overexpression of 11β-HSD1 in hepatocytes.

**Reference**