An Improved Dry Mounting Autoradiography Technique of A Whole Body Section of A Fetal Rat and Its Application to Cell Kinetics of Endocrine Organs

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A dry mounting autoradiographic technique was improved in order to observe the distribution of radioactivity in a whole body section of a rat. Distribution of 3H-thymidine in fetal rat was investigated with this improved autoradiographic technique. The thymus displayed intense labeling in a fetal rat in which the growth rate was rapid. It is, therefore, reasonable that there are considerable number of thymic cells engaged in cell division. The outerzone of the adrenal cortex and the testis displayed a moderate degree of synthetic activities. There were few labelled cells in the innerzone of the adrenal cortex. The observations seem to indicate the inward displacement of cells (cells derived from the outerzone move into the innerzone), supporting the centripetal cell displacement theory. The ovary showed a lower degree of synthetic activities than the testis did. The difference of labelling index between the ovary and the testis may be partly due to the difference of hormonal activities.—The testis begins to function before birth, while the ovary is hormonally quiescent in fetal life.

Determination of β-Hydroxy-β-Methylglutaryl Coenzyme A (HMG-CoA) Reductase Activity in the Liver of Male and Female Rats, The Effect of Enzyme Inducers on the Circadian Rhythm

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It is generally accepted that β-hydroxy-β-methylglutaryl coenzyme A (HMG-Co A) reductase is the rate-limiting enzyme of cholesterol biosynthesis, the activity of the reductase exhibiting circadian rhythm with peak level at midnight and the nadir at noon.

We have studied the effects of repeated administration of phenobarbital (P) spironolactone (S), and diazepam (D) on the activity of the HMG-Co A reductase at night and noon.

S.D. rats, 5 weeks, were kept under lighting conditions (light on 6:00 a.m. to 6:00 p.m., light off 6:00 p.m. to 6:00 a.m.) with food and water available ad libitum. The rats were administered 0.8 mg/100 g B.W. of P (i.p.), 10 mg/100 g B.W. of S (oral), 5 mg/100 g B.W. of D (oral) in drug treated groups and 1 ml/100 g B.W. of saline (oral) in control group at 11:00 a.m. for four days.

D.L. (3-14C) HMG anhydride was prepared from the acid by the method of Goldfarb et al., D.L. (3-14C) HMG-Co-Co A was prepared from the anhydride by the method of Hilz et al. and purified by chromatography on Whatman No. 3 MM paper with n-butanol-acetic acid-H2O (5: 2: 3) as the developing solvent

The rats were stunned by a blow on the head and then decapitated at 12:00 a.m., or 12:00 p.m. preparation of microsomes was performed by the following procedure; 1 g liver was homogenized with 4 ml 50 mM phosphate buffer (pH 7.4, containing 250 mM NaCl, 30 mM EDTA, 1.0 mM Dithiothreitol), centrifuging for 15 min at 10,000 Xg, the supernatant fraction was centrifuged at 100,000 Xg for 60 min, and resulting pellet was used as microsomes.

Assay of HMG-Co A reductase activity as performed by the modification of the method reported by Shapiro et al.

The reductase activities were compared between male and female, at noon and at night. The activities at noon and night in female were higher.
than male rats by 40–60%, and at night higher than noon by 16–18 fold in both sexes.

In P treated groups, the reductase activity increased at noon to 180% and 140% for male and female respectively compared with respective control values. At night the activity of male was not altered significantly, although that of female increased 140%.

In S treated groups, the activity at noon increased to 140% and 180%, in male and female respectively, but decreased at night to 38% and 36%, in male and female respectively.

In D treated groups, the activity of male decreased to 80% and 60%, at noon and night respectively. In female the activity decreased to 40% but at noon increased 2 fold that of control value.

**Plasma Pancreatic Glucagon in the Patients with Various Liver Diseases**


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Plasma pancreatic glucagon concentrations were determined in the patients with various liver diseases. After overnight fasting, the patients and normal subjects received an intravenous infusion of 30 g of 1-arginine over a period of 30 min. Blood was withdrawn before and 15, 30, 60, 90, 120 min after the start of the infusion.

Plasma pancreatic immunoreactive glucagon (IRG) was determined by the radioimmunoassay with antiserum 30 K.

In the patients with acute hepatitis and liver cirrhosis, plasma IRG concentration in the basal state was almost three times greater than that observed in the control subjects. In the patients with acute hepatitis, chronic hepatitis and liver cirrhosis, plasma IRG response to arginine was significantly greater than in the control subjects.

In the patients with liver cirrhosis, the prolonged disappearance curve of injected exogenous glucagon was observed.

Correlation between the maximum concentration of IRG after arginine infusion and liver function tests in the patients with chronic liver diseases was studied.

The correlation between response of IRG and concentration of serum albumin was significant. But the correlation between response of IRG and another liver function tests was not significant.

**Plasma Prolactin and Breast Cancer**

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Plasma human prolactin concentrations were measured using a commercially available radioimmunoassay kit (CIS) in 46 patients with breast cancer and 42 female hospital controls. Mean plasma human prolactin levels in female controls 15.20 ± 8.05 ng/ml.

In 12 patients with primary breast cancer receiving radical mastectomy and postoperative irradiation (aged 30 to 71 years, mean 48.3 years), mean plasma human prolactin levels were 16.92 ± 12.05 ng/ml.

In 34 patients with advanced breast cancer (aged 33 to 60 years, mean 46 years), mean plasma human prolactin were 26.49 ± 26.72 ng/ml.

In 13 patients who received oophorectomy (aged 33 to 55 years, mean 43.3 years), mean plasma human prolactin levels were 35.62 ± 37.39 ng/ml.

In 17 premenopausal patients (aged 31 to 53 years, mean 41.9 years) mean plasma human prolactin levels were 19.24 ± 10.74 ng/ml.

In 11 postmenopausal patients (aged 52 to 72