BIOCHEMISTRY

Deoxyribonucleic Acid Content of Basal Cells of Mouse Epidermis

Studies on mouse-ear and body-skin epidermis have led Gelfant[1,2] to the conclusion that there are two distinct populations of epidermal cells, one with a G_2 period of a few hours, the other with a G_2 period as long as five days. Bullough[3] has commented on the work of Gelfant and stated that his results are statistically inadequate. In view of the general uniformity in length of the G_2 period in mammalian cells studied so far[4] of approximately 2–4 h, the existence of a population of cells with an unusually long G_2 period is of some importance. It seemed worth while, therefore, to determine the distribution of DNA levels in epidermal cells, using a densitometric method, since a pronounced peak would be found at the tetraploid level if a sizeable population of cells were in the G_2 state.

Pieces of skin were removed from the ears and backs of hairless mice immediately after they were killed by cervical fracture. Care had been taken not to stress the animals during the previous week[5]. The skins were left for 24 h at 2° C in a solution of 0·5 per cent acetic acid. At the end of this time the epidermis was peeled off the underlying connective tissue and soaked in acetic/methanol (1 : 3) for 30 min. The basal cells were then gently scraped off the keratin layer on to glass coverslips, squashed in a press, air-dried, and stained by the Feulgen method[6], using pararosanaline dye (Chroma). A Barr and Stroud integrating microdensitometer was used to measure the absorption of the cells at 5500 Å. Leucocytes from a mouse were smeared on the coverslips prior to staining in order to establish the diploid DNA-level. This level did not alter if leucocytes were initially exposed to acetic acid in the same way as the skin.

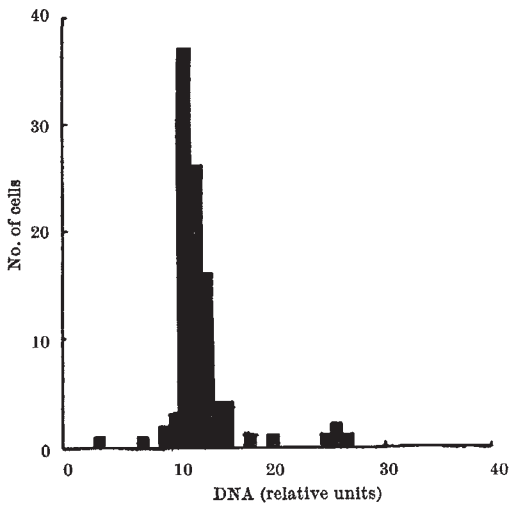

Fig. 1. DNA content of basal cells of ear epidermis

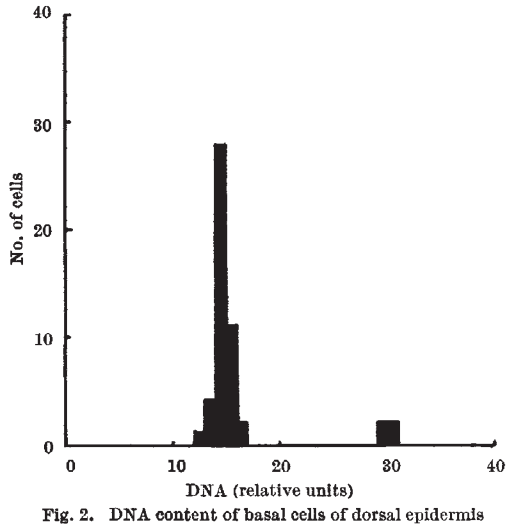

Fig. 2. DNA content of basal cells of dorsal epidermis

Histograms are shown of the DNA-levels of the basal cells of the thin epidermis of the ear in Fig. 1 (100 cells), and the multilayered epidermis of the dorsal skin in Fig. 2 (50 cells). The cells in both cases were almost entirely diploid. Scanning several hundred other cells by eye failed to reveal more than an occasional cell with a tetraploid amount of DNA. Similar distributions of DNA-levels were found in basal cells obtained from the skin of hirsute (random bred Hall Institute) mice. Since there is normally a slow turnover of basal cells[7,8], it is to be expected that some cells would be in the G_2 phase. However, no evidence was found of a large population in this phase. Hypodiploid nuclei were evident and could be obtained in quantity by more vigorous scraping of the keratin layer. A reduction in the DNA content of epidermal cells undergoing keratinization has been reported[9].

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Progressive Hyperglycaemia in Experimental Obesity of Albino Rats

The coincidence of two hereditary metabolic disorders, diabetes and obesity[1,2], is quite common although their interrelationship is still a matter of speculation as there is no proof that obesity causes diabetes. On the other hand, the importance of various physiological conditions, such as obesity, puberty or pregnancy, in precipitating diabetes cannot be overlooked. Few experimental observations record their coincidence. Mayer *et al.*[3] showed the connexion between obesity and diabetes in an obese-hyperglycaemic strain of mice. Long[4] mentioned that only with great difficulty was a temporary diabetes-like state induced in hypothalamic obesity in albino rats by partial pancreatectomy. Except for the observation of Katsuki *et al.*[5], who induced obesity and hyperglycaemia by gold thioglucose injection in intact mice, there is no recorded evidence that in intact animals hyperglycaemia results from obesity. While investigating the metabolic character of obese albino rats, we had already noticed that the fasting blood sugar was significantly high in these animals in comparison to control animals[6].

An investigation into the carbohydrate metabolism of obese albino rats, in which obesity was induced and maintained for a longer period, was therefore undertaken to detect any correlation between obesity and hyperglycaemia.

Thirty-six male albino rats of this Institute's colony were divided between two equal groups and maintained on a standard stock diet and a 40 per cent fat diet, respectively. Both groups were treated with 'P.Z.' insulin as recorded earlier[6]. Glucose tolerance tests were performed periodically on the surviving rats of the control and obese groups at 7, 10 and 12 months. At 10 months, when the fasting blood glucose-level was high, blood