Milk-Borne Hormones: Possible Tools of Communication Between Mother and Suckling

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Summary
Early studies suggested endocrine type mother-pup interaction: 131I administered to suckling rats appeared via the urine of the suckling and mother's milk in the circulation of litter mates who were not injected with iodine; levels of thyroxin in rat milk were influenced by the status of the thyroid gland of the lactating rat. Administration of TRH (thyrotropin releasing hormone) to lactating mothers led to an appearance of unaltered hormones in the milk and stomach content of sucklings. TSH (thyroid stimulating hormone) or ACTH (adrenocorticotropic hormone) when given orogastrically to suckling rats increased thyroid hormones and corticosterone serum levels in suckling rats. Functional effects of gastrointestinal administration of insulin, bombesin (mammalian analog of gastrin-releasing peptide) and epidermal growth factor (EGF) are reviewed in detail (32 references).

Key words

Thirty years ago interesting studies were performed suggesting mother-pup interaction: 131I administered to suckling rats appeared via the urine of the suckling and mother's milk in the circulation of litter mates who were not injected with iodine; levels of thyroxin in rat milk were influenced by the status of the thyroid gland of the lactating rat. Administration of TRH (thyrotropin releasing hormone) to lactating mothers led to an appearance of unaltered hormones in the milk and stomach content of sucklings. It is very important to note that a decrease of TSH levels in the hypophysis and an increase in serum levels were detected in the suckling (Štrbák et al. 1980). TSH or ACTH (adrenocorticotropic hormone) when given orogastrically to suckling rats increased thyroid hormones (Tenore et al. 1980, Vaucher et al. 1983) and corticosterone serum levels in suckling rats (Vaucher et al. 1983). Alexandrová and Macho (1983) reported glucocorticoids in human, bovine and rat milk. Insulin caused a hypoglycaemic effect after its administration into the stomach (Mosinger et al. 1959) or into the intestinal lumen of suckling rats (Hiršová and Koldovský 1969). Orogastrically-administered bombesin (mammalian analog of gastrin-releasing peptide) to suckling rats evoked pancreatic secretion of trypsin (Pollack et al. 1989) and inhibited gastric emptying (Jiang et al. 1991).

Other studies demonstrated the presence of epidermal growth factor (EGF) in rat milk (Schaudies et al. 1990); interestingly, transforming growth factor-α (TGF-α) was not detected (Dvořák and Koldovský 1994). The presence of melatonin in human milk was
recently reported by Illnerová et al. (1993). Melatonin in human blood and milk was beyond the limit of detection during the day, whereas the melatonin concentration during the night was 280±34 pmol/l in serum and 99±26 pmol/l in milk. The presence of daily rhythm in milk suggests that melatonin fluctuations in the milk might communicate time-of-day information to breast-fed infants.


The importance of the intake of milk-borne EGF was demonstrated in studies showing that the EGF content of the GI tract of suckling rats was depleted during fasting (Schaudies et al. 1989, Grimes et al. 1992), and increased after resuckling or refeeding with rat milk substitute (RMS) to which EGF had been added (Grimes et al. 1992); administration of RMS that was not supplemented had no effect. Addition of EGF to RMS fed to sucking rats for four days normalized the protein/DNA ratio in their colon compared to sucking rats fed RMS only (Pollack et al. 1987).

Finally, recent studies suggested a protective role of presently unknown factor(s) in rat milk. Artificially-fed sucking rats that received gliadin (a substance known to provoke gluten-sensitive enteropathy in children and adults) intragastrically immediately after birth exhibited various pathological changes in the small intestine whereas similarly-treated mother-fed sucking rats appeared to be normal. Although various other factors might be involved, it is noteworthy that artificially-fed sucking rats without administration of gliadin had normal enterocyte structure (Štěpánková et al. 1989, 1990).

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**Reprint Requests**

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