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LIPIDS IN MUCOSAL EPITHELIUM OF THE INTESTINE OF MICE FED ON ZN-DEFICIENT DIET

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THE loss of appetite and growth retardation associated with sterility, oesophageal parakeratosis, aplocia and impaired wound healing have been frequently reported in mammals under Zn-deficient conditions[1]. The causes of most of the symptoms have been related to depression in the activities of many Zn-dependent enzymes[2]. However growth retardation and anorexia in such animals point toward malfunctions of alimentary canal besides other factors. An attempt has been made to study the absorption rate of lipids under Zn-deficient conditions through perfusion experiments[3].

Lipids have been associated with the inhibitory effect on gastric secretion and stomach emptying process when they are present in mucosal epithelium of the intestine[4,5]. Cytochemical localization of lipids in mucosal epithelial cells, therefore, can provide us the morphological evidence for such a phenomenon. This paper reports the effect of severe Zn-deficiency on the cytochemically detectable lipids in mucosal epithelium of the intestine of mice.

Twenty male mice, *Mus musculus* of Lacca strain weighing 18–20 g, were equally divided into two groups. The animals in the first group (ZD) were fed *ad libitum* on semisynthetic diet containing: EDTA-treated casein: 30% sucrose: 51% corn oil: 8% mineral mixture[6]: 4% vitamin mixture[6]: 5% methionine: 0.8% and agar agar 1.2%. The analysis of this feed by atomic absorption spectroscopy has shown that it contains 0.5–1 ppm of Zn. The animals in the second group (ZS) that served as the control were fed on Zn-supplement diet which was identical to Zn-deficient diet except that $ZnSo_4$ was added to the mineral mixture to raise the Zn content to 100 ppm. Triple distilled deionized water was given to the animals *ad libitum*.

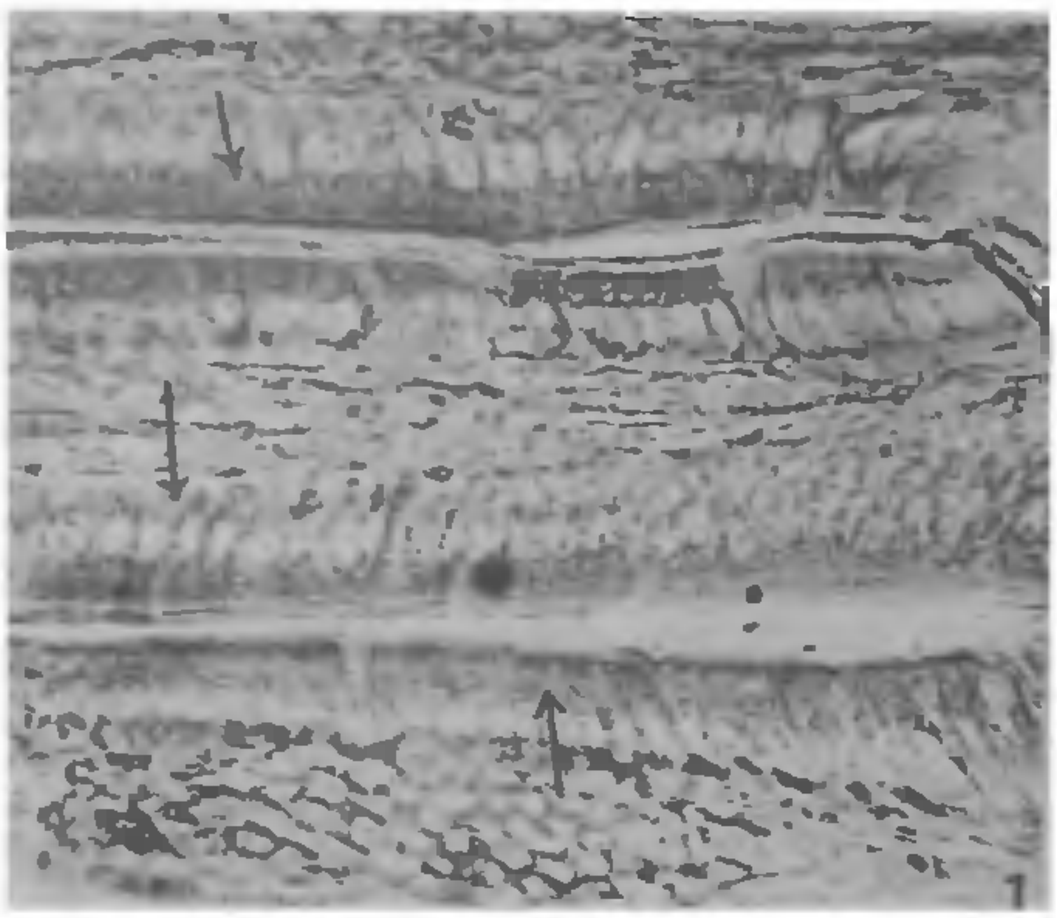
The symptoms of Zn-deficiency characterized by low food intake, spiny hair coat, skin lesions, growth retardation in terms of weight gain, started appearing in mice of ZD diet group after 2 weeks of the treatment and the severity of symptoms increased as the duration of feeding on low Zn diet was prolonged to 3 weeks.

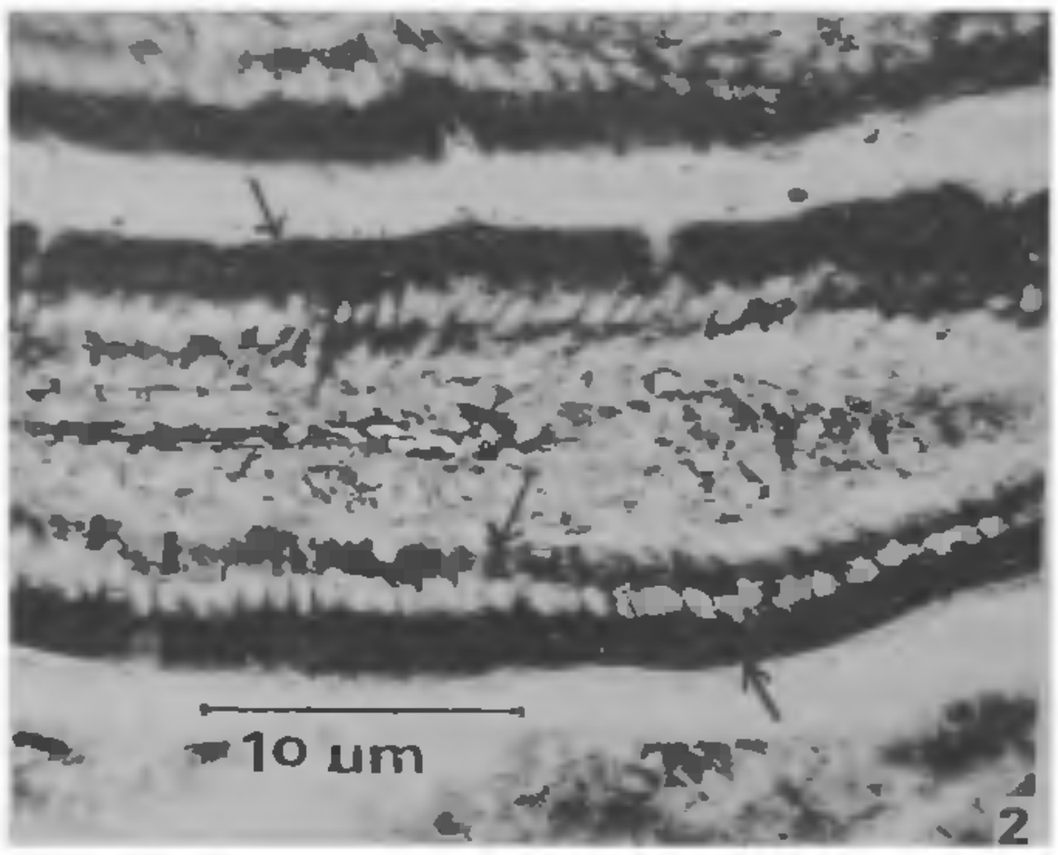
The average food intake during the first 2 weeks was statistically insignificant between the two groups. Thereafter, the ZD group started consuming less food. At the end of the third week, they consumed 28% less ration than ZS control and lost at an average of 23.04 g/kg body weight. The feeding was then suspended for 3 h and 8 h and the animals of both groups were dissected feeding for 3 h and 8 h. The duodenum and jejunum were cut into small pieces and fixed in formalin-calcium and neutral formalin fluids. The cold gelatin sections were cut at 10 μ and stained in Sudan black B (SBB), acid haematin (AH) and Nile blue sulfate (NBS) following Pearse[7].

The cytoplasm of columnar, long and narrow absorptive epithelial cells of duodenum and jejunum that line the luminar surface of the villi stained intensely and homogeneously with SBB and NBS in ZD animals in contrast to a moderate reaction limited to a few granules concentrated more apically than basally in ZS animals sacrificed after 3 h of their meals (figures 1 and 2). The sudanophilia in ZD mice was so intense that the cytoplasm appeared pitch black in SBB preparation. A slight reduction in SBB and NBS reaction was noticed at 8 h stage in ZD mice. However, the reaction was far more intense than ZS animals which practically lacked sudanophilic granules at this stage.

These cytochemical results suggest a massive lipid accumulation, predominantly triglycerides, far more in excess in mucosal epithelial cells of ZD mice than ZS control at 3 h and 8 h stages despite the equal amounts of corn oil in their diets. This envisages a slower rate of lipid transport to lacteal. These results are in conformity with those of Koo and Turk[3] who through electronmicroscopic and chromatographic studies concluded that the exit block to the movement of lipid droplets out of mucosal cells occurs due to the failure of mucosal synthesis of proteins required for the formation of chylomicrons in Zn-deficient rats. Almost similar results have been obtained following treatment of rats with protein synthesis inhibitors[8,9]. The essentiality of Zn in protein synthesis is well-established[10,11].

The persistence of lipids for a longer duration in mucosal epithelial cells in ZD mice than ZS control perhaps is responsible for imposing a prolonged inhibitory effect on stomach emptying process through feedback mechanism which may be one of





Figures 1 and 2. T. S. jejunum of **1.** Zn-supplement, and **2.** Zn-deficient mice after 3 h of starvation (formal-calcium/SBB).

the causes of low dietary intake in ZD animals — one of the clinical manifestations of Zn deficiency in the animals examined so far. Such an inhibitory mechanism due to the presence of fats in mucosal epithelial cells of jejunum has been well-documented[4,5].

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NEUROSECRETORY CELLS AND VITELLOGENIN SYNTHESIS IN *THIACIDAS POSTICA* (INSECTA: LEPIDOPTERA)

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VITELLOGENINS (VG) are specific female proteins in insects which are synthesized in the fat body, released into the haemolymph and taken up selectively by the maturing oocytes against a concentration gradient[1,2]. Hormonal control of synthesis of VG has been reported in many insects. It is controlled in some insects by juvenile hormone (JH)[1–6], and in others by neurosecretory cells (NSC)[7,8]. VG synthesis can also be stimulated *in vitro*, by methoprene[9,10]. In the mosquito, *Aedes aegypti* an egg development neurohormone (EDNH) produced by the brain may have an indirect role in stimulating the yolk protein synthesis[11,12], while the function of corpus allatum (CA) is to allow the development of previtellogenic oocytes up to the resting stage[13]. The situation in Lepidoptera is not clear. In some lepidopteran insects the CA have been reported non-essential for VG synthesis[14,15], while in others it was essential[16,17]. In the former, the role of NSC has not been examined. The present study was undertaken to investigate the role of CA as well as NSC in the control of VG synthesis in *Thiacidas postica*.

Caterpillars collected from the field were reared in the laboratory at 27 ± 1°C, 70–75% RH and 16 h photoperiod. Larvae were fed on fresh