

SUBLETHAL EFFECTS OF CADMIUM ON OVARIAN MORPHOGENESIS IN *TILAPIA NILOTICA*

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Onset of ovarian differentiation is at least one week delayed in Cd-treated juvenile *Tilapia*. Ultrastructurally, oogonia, primary oocytes and follicle cells show damaged membranes and cell organelles. While normally vitellogenesis starts at day fifty-three, most of the treated cells do not have yolk vesicles by this time. Ultrastructural features of steroidogenesis in theca cells are not evident by day forty-five. The ovary of treated fish appears smaller and contains less gametogenic cells than that of untreated fish.

All these observations indicate reproductive failure due to cadmium toxicity in *T. nilotica*.

Introduction

Cadmium is largely accumulated in the kidney, pancreas, liver, spleen and intestines in mammals. Accumulated metal causes disorders, such as degeneration or malfunction of these organs. (Eisler and Gardner, 1973).

In teleosts, sites of major accumulation vary with species (Tokumaru *et al.*, 1980). However, reports dealing with histological responses to cadmium are restricted to very short-term experiments on mature fish (Gardner & Yevich, 1970; Schweiger, 1957).

The purpose of this work is to analyze the ultrastructural effects of sublethal dose of cadmium on the histogenesis of the ovary of *Tilapia nilotica* from the embryo to maturity.

Materials and Methods

Tilapia nilotica four-day posthatch larvae were exposed to 0.5 ppm cadmium chloride for eight weeks. Fish were harvested after four- and eight-week treatments and ovaries were processed for electron microscopy.

Specimens were fixed in 2.5% glutaraldehyde, washed in phosphate buffer (pH 7.2), postfixed in OSO_4 , dehydrated in acetone and propylene oxide and embedded in araldite resin. Ultrathin sections were stained with uranyl acetate and lead citrate.

Results and Discussion

By the second week of treatment, primordial germ cells reach the gonad blastema. Treated and untreated fish have the same ridge appearance and the PGCs have similar ultrastructural features (Herrera, 1987).

While the elongated primordium of the ovarian cavity appears on the sixteenth day in untreated fish, there is about one week delay in the treated larvae. By the fourth week, several pathological changes are observed (Figs. 1 and 2) in the germ cells and somatic tissues. (Herrera, 1988 a, b).

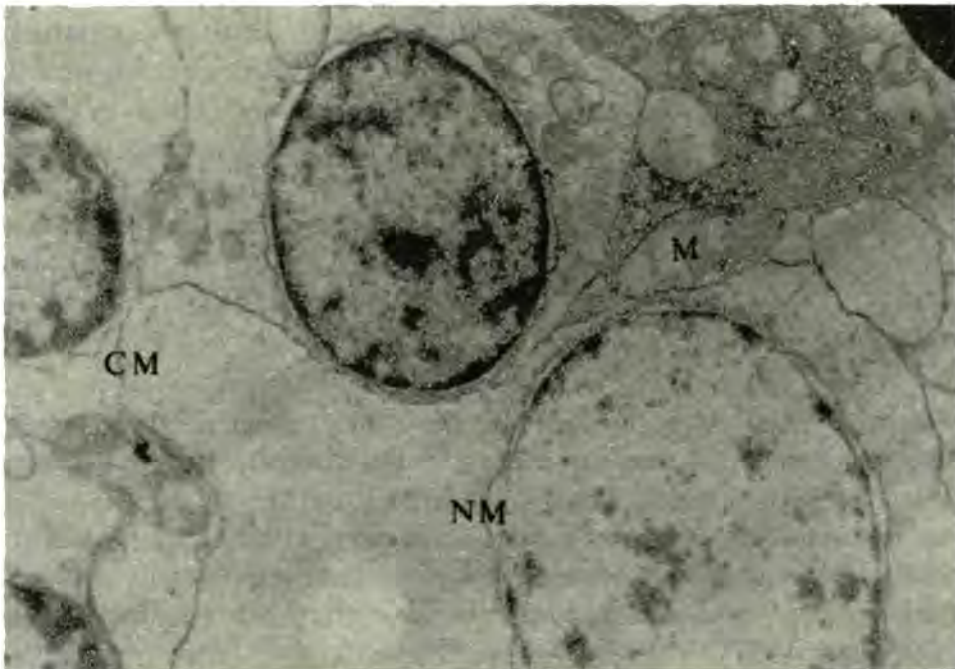


Figure 1. Swollen oocytes with dilated nuclear membrane and enlarged mitochondria and ER. 7,000x.

By seven to eight weeks, ultrastructural changes observed in the oocytes are dilated mitochondria, enlarged ER and formation of vacuoles and electron dense bodies. (Fig. 3)

Somatic cells closely show damage (Fig. 4) which probably explains delayed vitellogenesis.

In the gonads, the deleterious effects in the gametogenic cells are reportedly due to injurious effects of Cd on the vasculature and on steroid synthesis (Sanga-

lang and O' Halloran, 1972). This was observed in the capillaries of the ovary (Figs. 5, 6).

This same condition of blood vessel destruction had been observed in the testis (Herrera, 1988c). This lead to aspermatogenesis, orchitis and epididymitis (White *et al.*, 1978).

Templeton and Cherian (1985) have offered an explanation to Cd-induced injury. Free Cd adversely affects numerous enzyme systems leading to pathological observations.

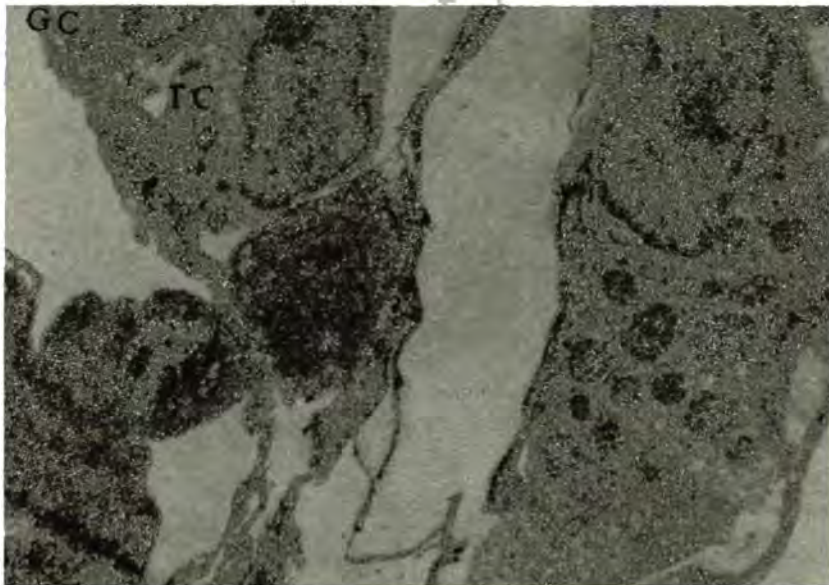


Figure 2. Granulosa cells (GS) and theca cells (TC) scattered in the ovarian cavity. 7,000x.



Figure 3. An egg cell with discontinuous cell membrane (CM), ruptured nuclear membrane (NM) and disorganized cell organelles. 5,000x.



Figure 4. An atretic steroidogenic theca cell beside a destroyed blood vessel (BV). A blood cell (BC) with dilated nuclear membrane. 3,000x.

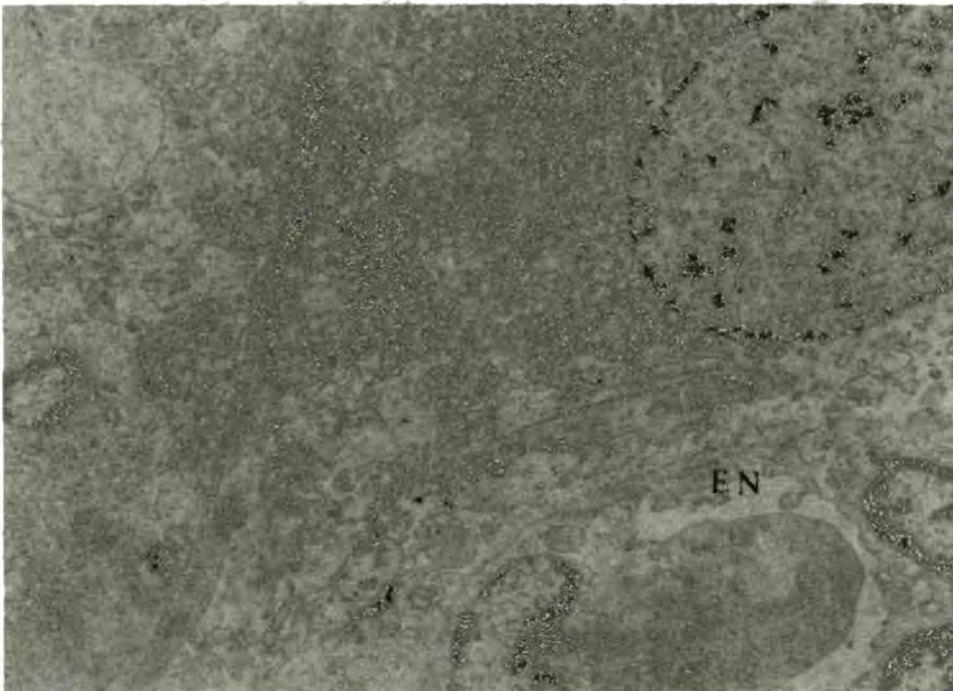


Figure 5. An intact capillary with healthy endothelial cells (EN). 7,000x.

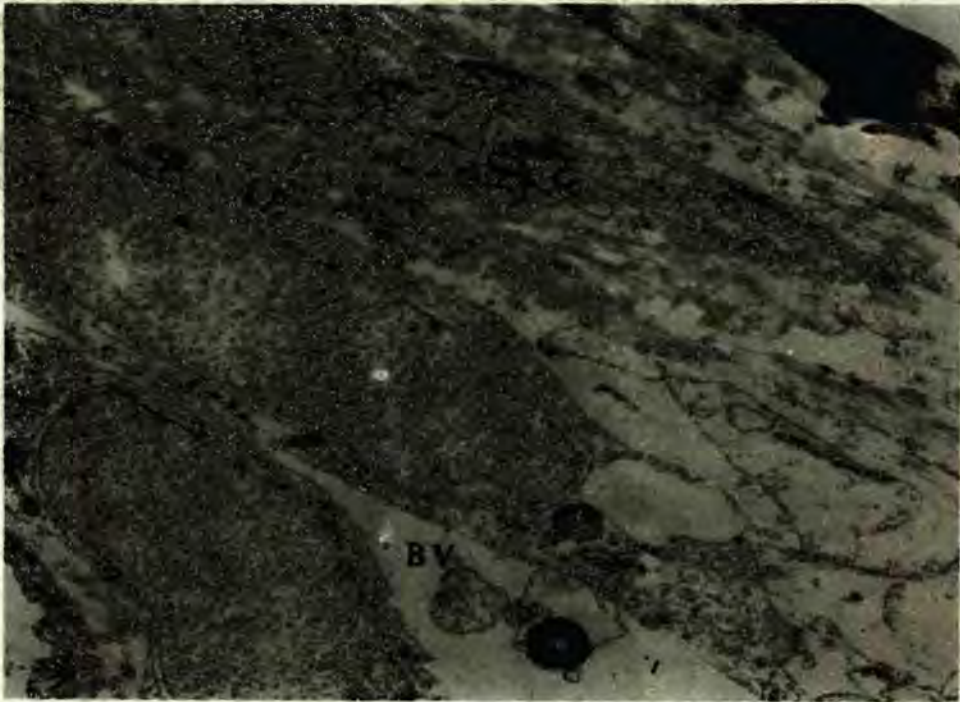


Figure 6. Damaged vascular and other somatic tissues of the ovary. 5,000x.

Summary

Ovarian histogenesis is adversely affected by long-term exposure of fry to sublethal cadmium from the larval to the maturation stage. Onset of differentiation and vitellogenesis are delayed. Gametogenic cells and somatic tissues show damage starting on the fourth week. The treated ovary is smaller and has less gametogenic cells than the normal. All these pathological changes indicate a high probability of reproductive failure.

Acknowledgment

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