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A Teratological Study Of Faulty Neural Tube Closure And Neuro- Protection In Gallus gallus domestics

Abstract
The most severe congenital abnormalities are known as neural tube defects (NTDs), which are characterized by defective neural tube closure during early development. The present study investigates the embryo-toxic effect of serotonin and cadmium chloride and the ability of folic acid and inositol to exert a neuroprotective effect in an in vivo model based on fertilized chick embryos (Gallus gallus domesticus). Fourteen eggs were assigned to graded serotonin exposure of 10, 25, and 50 µL, fixed cadmium chloride of 10 µL, and also co-treated with folic acid at 50 µL or inositol at 100 µL. Embryos were incubated for 48–72 hours, harvested, and examined for survival and gross structural changes. The outcome indicated that serotonin and cadmium chloride induced dose-dependent developmental abnormalities, comprising notches in the neural tube, notochord-brain separation, and spina bifida-like development. Combined exposure was embryonic lethal. Folic acid was able to prevent damage in lower dose groups but not with high serotonin. Inositol was not protective and all the inositol-treated embryos failed to develop. The results illustrate the dose sensitivity of embryonic tissues to teratogens and show the preeminent importance of folic acid. Inositol might need co-factors or combination therapy to be effective.
This study provides experimental evidence for prenatal screening, medically controlled use of drugs, and nutritional supplementations during pregnancy.
Key words: Neural tube defects (NTDs), Teratology, Chick embryo model (Gallus gallus domesticus), Serotonin, Cadmium chloride (heavy metal), Folic acid, Inositol, Neurodevelopment, Embryo-toxicity, Neuroprotection, Anencephaly, Spina Bifida Cystica, Meningocele, Encephalocele, Myelocele, and Meningomyelocele.

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