“A TERATOLOGICAL STUDY OF FAULTY NEURAL TUBE CLOSURE AND NEURO-PROTECTION IN *Gallus gallus domesticus*”

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Introduction

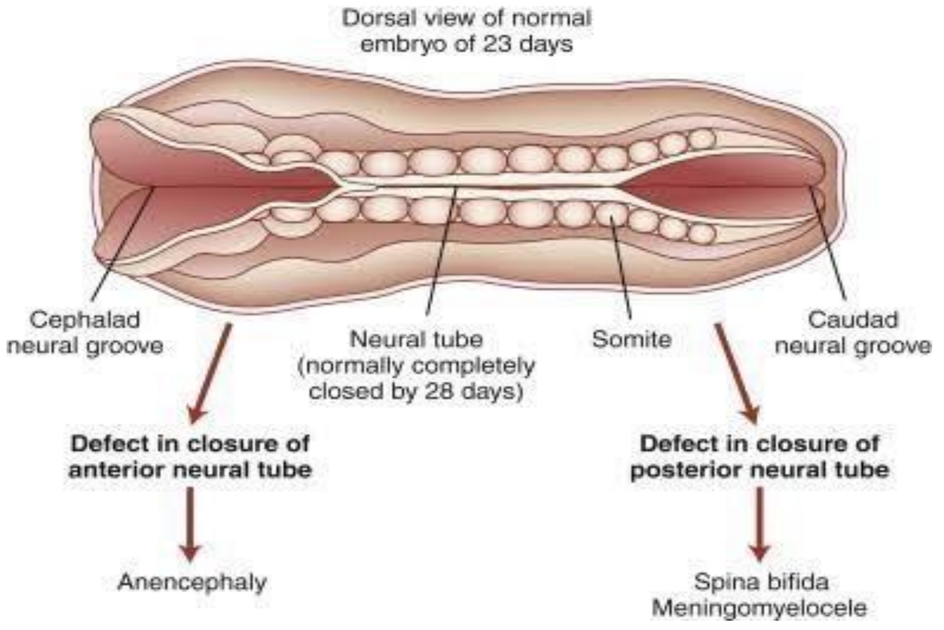
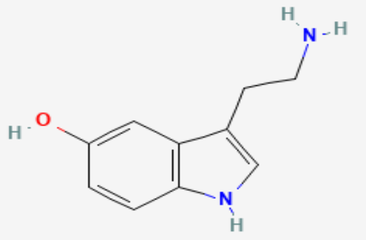


Fig 1.1 . Neural tube defects resulting from defects in closure. Defect in closure of the anterior neural tube causes anencephaly. Defective closure of the posterior neural tube produces a variety of clinical findings, depending on the location and severity of the lesion. These posterior defects are generally referred to as spina bifida.

Teratogenicity refers to the ability of a substance or agent to cause structural or functional abnormalities in a developing fetus or embryo, which can manifest as congenital malformations, growth retardation, or functional impairments after birth.

Serotonin



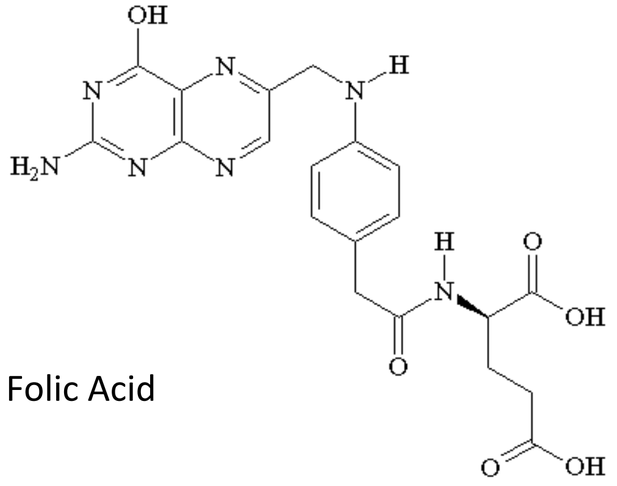
PubChem- CID 5202

Cadmium Chloride

Pubchem- CID 24947

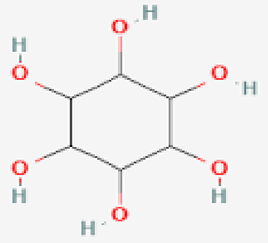
Introduction

A "neuroprotectant for NTD" most likely refers to agents that protect against the neuronal damage associated with neurodegenerative disorders (NDs)



Folic Acid

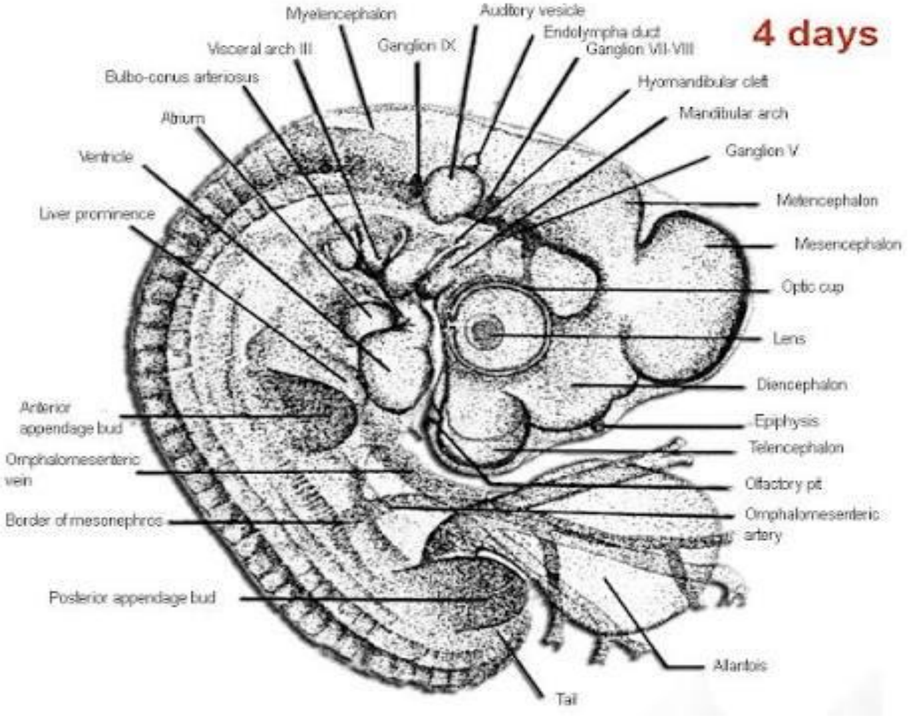
Dominic Taylor and Paul May., 2008



Inositol

PubChem- CID 892

Why Chick embryo



Objectives

To assess the impact of serotonin in an early embryonic development, particularly neutral tube formation, in a dose-dependent manner.

To draw correlation between observed embryonic malformations and human congenital disorders such as neural tube defect, spontaneous abortion or developmental delays.

To evaluate the teratogenic effects of cadmium chloride, a known environmental toxin on the neural an structural development of chick embryo.

To investigate the embryological influence of inositol as a potential protective agent and compare its effectiveness with that of folic acid

To determine the combined effect of serotonin and cadmium chloride and observe whether they produce additive or synergetic toxicity.

To analyse the neuroprotective role of folic acid, when co-administered with serotonin and cadmium chloride, and assess its ability to prevent or reduce neural tube defects.

Methodology

01 Egg preparation

02 Candling

03 Windowing

04 Injection

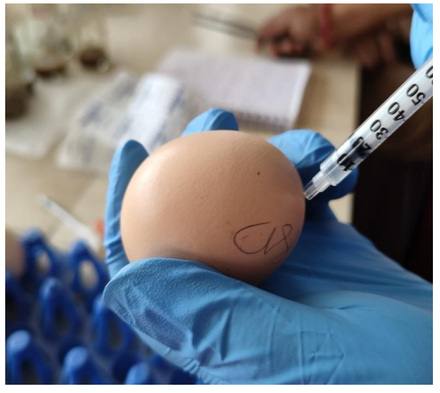
05 Incubation

06 Embryo retrival

07 Staining

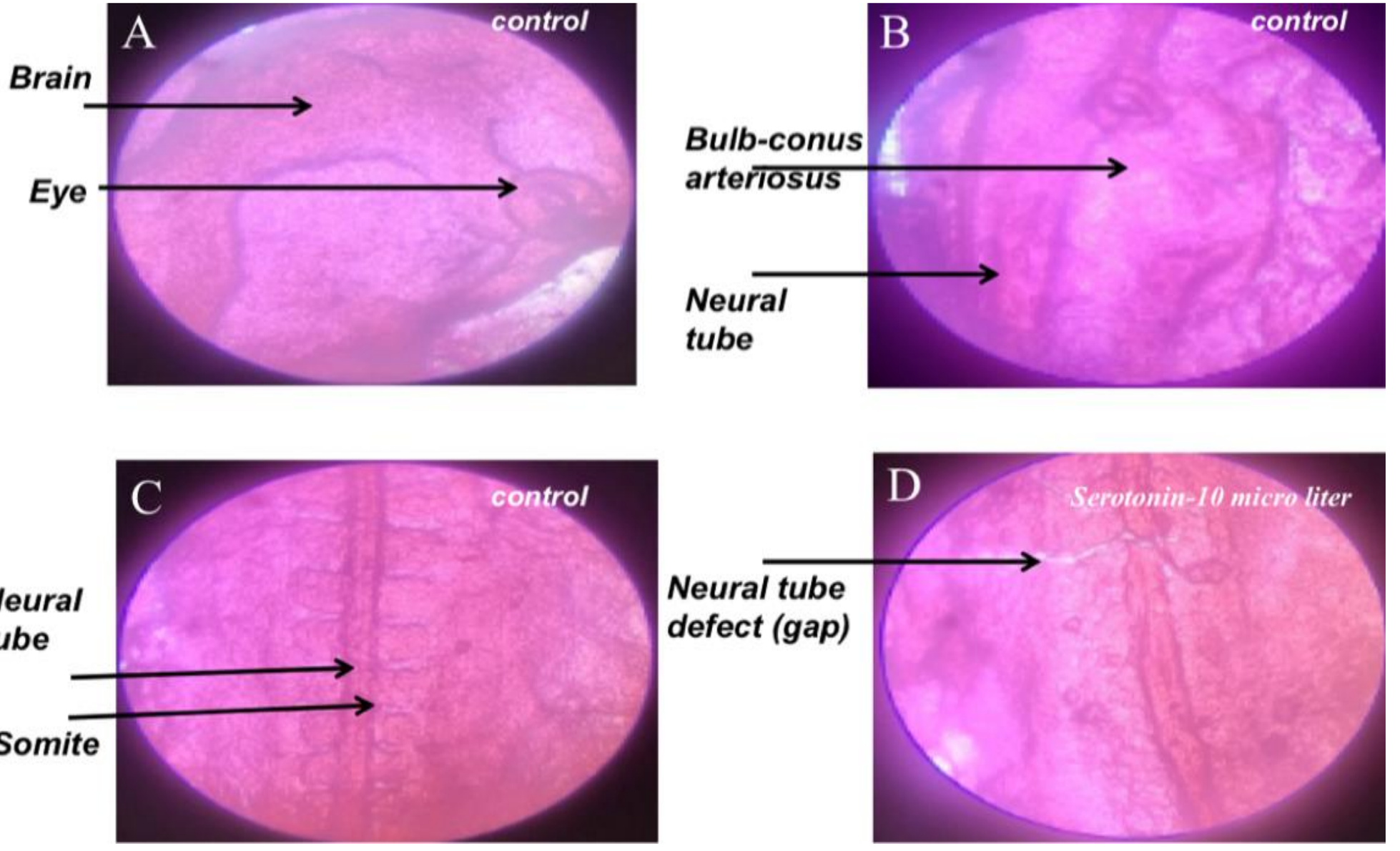
08 Microscopic observation

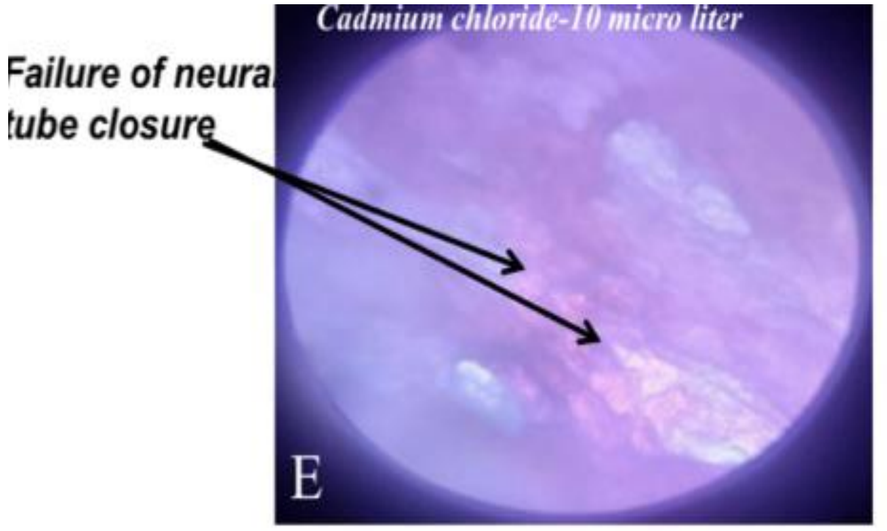
09

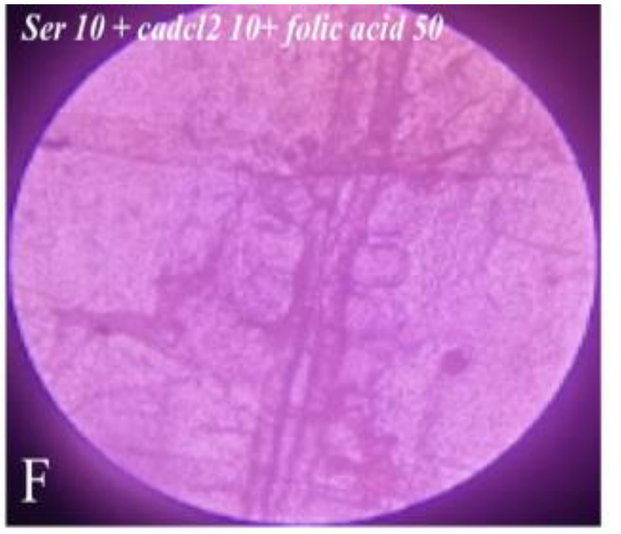


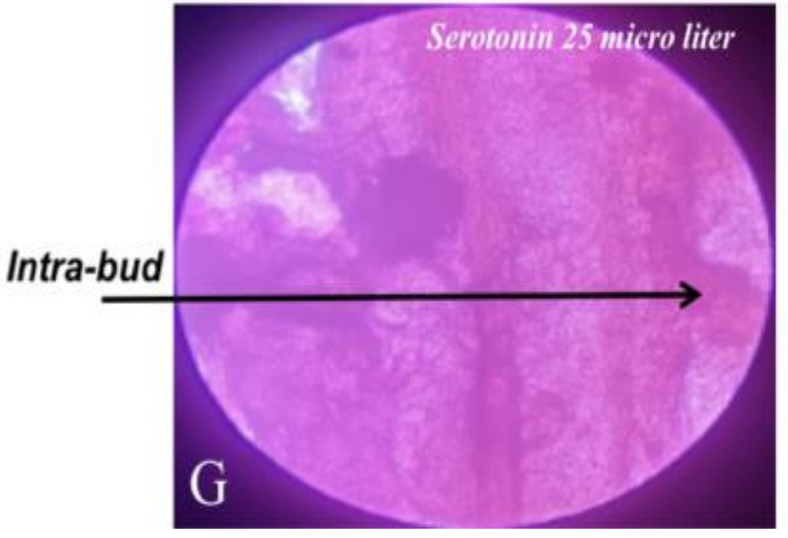
Results

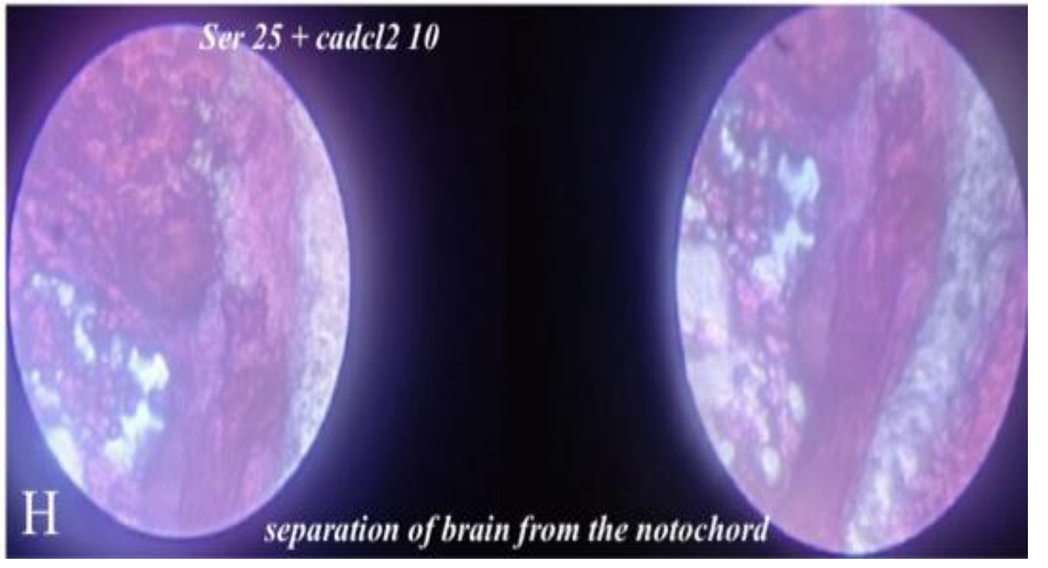
Group	Treatment			Results
1	Control			Normal development, no defects
	Teratogen	Heavy Metal	Supplements	
2	serotonin 10 µL	-	-	Neural tube gap
3	-	cadmium chloride 10 µL	-	Neural tube closure failure
4	serotonin 10 µL	cadmium chloride 10 µL	-	Embryo did not survive
5	serotonin 10 µL	cadmium chloride 10 µL	Folic acid 50 µL	partial rescue, minor defects
6	serotonin 25 µL	-	-	Intra-bud formation, disrupted vasculature
7	serotonin 25 µL	cadmium chloride 10 µL	-	Detachment between brain and notochord
8	serotonin 25 µL	cadmium chloride 10 µL	Folic acid 50 µL	Notochord preserved, mesodermal development inadequate
9	serotonin 50 µL	-	-	No development, rotten egg
10	serotonin 50 µL	cadmium chloride 10 µL	-	No development, rotten egg
11	serotonin 50 µL	cadmium chloride 10 µL	Folic acid 50 µL	No development, folic acid inefficient at high dose
12	serotonin 10 µL	cadmium chloride 10 µL	Inositol 100 µL	No development
13	serotonin 25 µL	cadmium chloride 10 µL	Inositol 100 µL	No development
14	serotonin 50 µL	cadmium chloride 10 µL	Inositol 100 µL	No development


A
control
Brain
Eye
B
control
Bulb-conus arteriosus
Neural tube
C
control
Neural tube
Somite
Neural tube defect (gap)
D
Serotonin-10 micro liter


Failure of neura
tube closure
Cadmium chloride-10 micro liter
E


Ser 10 + cadcl2 10+ folic acid 50
F


Intra-bud
Serotonin 25 micro liter
G


Ser 25 + cadcl2 10
H
separation of brain from the notochord

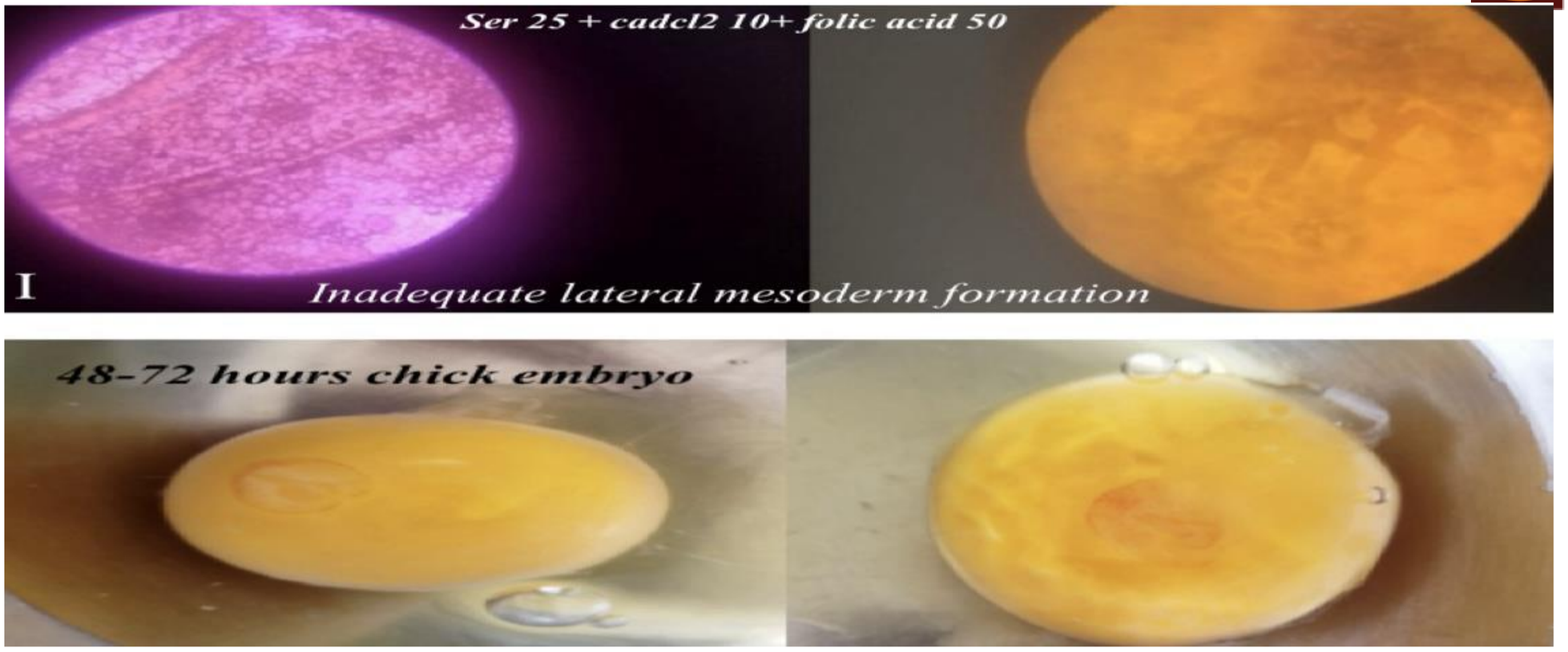


Fig 3.1: Morphological changes in chick embryos (48–72 h incubation) after serotonin, cadmium chloride, and folic acid treatment. Control embryos (A–C) developed normally with normally formed brain, eye, and intact neural tube, and with well-segmented somites and bulb-conus arteriosus visible. (D) Serotonin treatment (10 µL), and not (A–C), produced a focal disruption in the neural tube, which signifies failure of closure at this location. (E) Cadmium chloride (10 µL) produced gross neurulation abnormalities, including failure of neural tube closure. (F) Folic acid treatment (50 µL) with a combination of serotonin (10 µL) and cadmium chloride (10 µL) partially restored the midline continuity, indicating folic acid's protective role against toxin-induced malformation. (G) Increased concentration of serotonin (25 µL) perturbed axial organization, resulting in abnormally developed intra-bud. (H) Simultaneous treatment with high-dose serotonin (25 µL) and cadmium chloride (10 µL) caused over-separation of brain from notochord, which is a sign of general disruption in neural and axial patterning. (I) Supplementing the serotonin (25 µL) + cadmium chloride (10 µL) treatment with folic acid (50 µL) resulted in better neural alignment and fewer gross malformations.

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Conclusion

This study demonstrates that serotonin and cadmium chloride significantly disrupt neural tube closure in chick embryos, with dose-dependent increases in abnormalities and embryonic mortality. Even low cadmium exposure was sufficient to cause developmental interference. While folic acid provided partial protection, it was less effective at higher teratogenic doses, and inositol showed no clear neuroprotective role. These findings underline the vulnerability of early pregnancy to toxic exposures, emphasize the importance of maternal nutrition and pharmaceutical safety, and point to the need for further research on co-therapies, dose thresholds, and neuroprotective mechanisms.


THANK YOU