Z301 **IGF-II and activin A mitigate the cytotoxicity of zinc in Xenopus presumptive ectoderm**

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Exess zinc cytotoxicity was estimated by Xenopus animal cap assay, and the results showed that 200 uM or more zinc caused disassociation of cells in animal cap explants. To mitigate excess zinc stress in the disassociated cell mass of presumptive ectoderm, three growth factor candidates, activin A, IGF-I, and IGF-II were added at the lethal zinc level. The 10 ng/ml level of activin A rescued 100% of the explants to a maximum of 500 uM of zinc, and 13% of the explants were rescued at 800 uM of zinc. IGF-II did not have any antagonizing effect on zinc toxicity, but IGF-II displayed a stress-mitigating effect. The 1-100 ng/ml concentrations of IGF-II rescued 100% of the explants in 200 uM of zinc, and 1 ng/ml of IGF-II rescued 13% of explants in 400 uM of zinc. Thus, activin A has a more potent mitigating effect on zinc toxicity in vitro. Activin A is well known as a potent mesodermal inducer in vitro, and the mesodermal induction effect of activin A was inhibited at the combined dose of 1-50 ng/ml of activin A and 0.1-100 uM of zinc in the animal cap assay. Zinc cytotoxicity of zinc causes mitochondrial degeneration, and inhibition of cell adhesion, and these effects may reduce the competence of presumptive ectoderm and result in inhibition of organ induction.