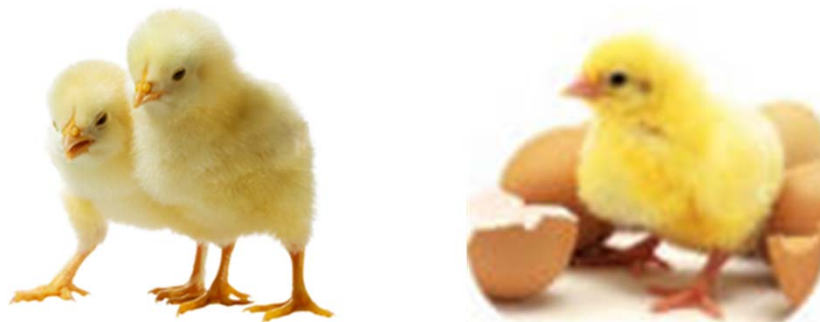


Prenatal hypoxia programs cardiac dysfunction through changes in β -adrenergic signaling

Dr Jordi Altimiras
Linköpings universitet, Sweden



Prenatal hypoxia restricts fetal growth and increases risk of adult cardiovascular disease. Programming of β -adrenergic receptor mediated inotropic effects are likely involved since they are desensitized in fast-growing chickens. To understand the mechanisms underlying receptor desensitization, we incubated eggs in normoxia or hypoxia and determined cardiac contractility before and after β -adrenergic stimulation when the chickens were 5 weeks old. Prenatal hypoxia caused contractile dysfunction. cAMP levels were lower and so was the β 1AR/ β 2AR ratio in hypoxic birds, G_i expression was unchanged, while G_s was increased. These changes resemble those reported in early human heart failure. We conclude that prenatal hypoxia has a programming effect on adult β -adrenergic function and speculate that prenatal hypoxia leads to heart failure.



Friday January 25th at 10.15
Room 127 (zoofys kaffestue), building 1131