N-Acetylcysteine, a glutathione precursor, reverts vascular dysfunction and endothelial epigenetic programming in intrauterine growth restricted guinea pigs

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Key points:
Intrauterine growth restriction (IUGR) is associated with vascular dysfunction, oxidative stress and signs of endothelial epigenetic programming of the umbilical vessels. There is no evidence that this epigenetic programming is occurring on systemic fetal arteries. In IUGR guinea pigs we studied the functional and epigenetic programming of endothelial nitric oxide synthase (eNOS) (Nos3 gene) in umbilical and systemic fetal arteries, addressing the role of oxidative stress in this process by maternal treatment with N-acetylcysteine (NAC) during the second half of gestation. The present study suggests that IUGR endothelial cells have common molecular markers of programming in umbilical and systemic arteries. Notably, maternal treatment with NAC restores fetal growth by increasing placental efficiency and reverting the functional and epigenetic programming of eNOS in arterial endothelium in IUGR guin