Prenatal administration of rosiglitazone, a peroxisome proliferator-activated receptor γ agonist, can attenuate hyperoxic lung injury in a preterm rabbit model

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Objective

Rosiglitazone, a peroxisome proliferator-activated receptor gamma (pparγ) agonist, can attenuate hyperoxic lung injury in a rodent model by accelerating lung maturity. Our aim was to examine the effect of prenatal administrated rosiglitazone in the preterm hyperoxic rabbit model.

Methods

Pregnant rabbits were injected with saline or rosiglitazone (3mg/kg) 48 and 24 hours prior to preterm delivery at 28 days (term: 31 d). The newborns were nursed in normoxia (21% O2) or hyperoxia (>95% O2) in an incubator. Sacrifice took place either immediately after birth, 24 hours later or at seven days of life, when lungs were harvested.

Results

Immuno-histochemical stainings showed a significant increase in VEGF, FLK-1 and SP-b in rosiglitazone-treated animals examined immediately after delivery and a persisting increase in SP-b at day seven. Morphometry revealed a significant reduction after 7 days of hyperoxic exposure of both mean wall transection length (lmw) and mean terminal bronchiolar density (mtbd) in the rosiglitazone-group coinciding with thinner alveolar septa and a higher amount of alveoli per terminal bronchiole. Functional assessment demonstrated a significant decrease in tissue damping (resistance, g) in treated animals compared to hyperoxic controls at day 7.

Conclusion

In a preterm rabbit model prenatal maternal administration of rosiglitazone accelerates lung maturity and can improve pulmonary outcome after hyperoxic exposure.