Maternal ingestion of cocoa constricts fetal ductus arteriosus: an experimental study

Objective: Cocoa has antioxidant and antiinflammatory effects by downregulating COX2 receptors expression in the endothelium and enhancing nitric oxide bioavailability. In this study, we investigated whether cocoa causes ductus arteriosus constriction (DAC) in fetal Wistar rats and if it has antioxidant effects on fetal tissues.

Methods: Wistar pregnant rats at the 21st day of gestation were submitted to administration of cocoa (7.2, 72 or 720mg/kg) 12 h before cesarean section, indomethacin (10 mg/kg) or water 8h before cesarean section. Thorax and livers were stored. By digital microscopy, images were digitalized and the diameters were measured. Fetal and maternal liver catalase and superoxide dismutase activities and TBARS levels were analyzed. Statistical analysis used one way ANOVA and differences were compared by Tukey’s tests.

Results: Maternal ingestion of cocoa 720mg/kg and indomethacin 10mg/kg decreased fetal internal DA diameter when compared to water, (mean: 135 ± 53 µm x 92 ± 59 µm x 263±81µm, P<0.0001, n=7-33) respectively. Cocoa alone increased DA wall thickness, when compared to indomethacin and control (200 ± 56µm x 160 ± 36µm x 136 ± 34µm, P<0.0001, n=7-33) respectively. Similar to indomethacin, cocoa decreased the ratio between ductal internal diameter and pulmonary artery internal diameter compared to control group (0.4 ± 0.1 x 0.2 ± 0.09 x 0.72 ± 0.3, n=7-33), respectively. Sagittal cross sections of the fetal heart showed DAC after administration of both cocoa and indomethacin compared with the control group. Cocoa did not alter oxidative stress markers in fetal nor maternal livers, a similar effect to indomethacin.

Conclusion: These results constitute pharmacological evidence supporting the role of cocoa as a potential cause of fetal ductal constriction, which prompts a note of caution for maternal consumption of chocolate in late pregnancy.