Conclusions: A high GI diet during gestation exacerbated a poorer maternal metabolic profile with obesity and although gene ex- pression appeared predominantly stable, further investigations in any potential morphometric and related placental modifications a required.

III-4 Oxidized components of frying oil ingested during pregnancy disturb vitamin A metabolism and are po tially teratogenic in mice

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We previously observed a higher incidence of congenital malformations in the fetuses of dams fed an oxidized frying oil (OFO)-con diet during pregnancy. Since OFO is known to activate PPARα and modulate CYP450 enzyme activity and since some CYP450 enzy are known to be involved in retinoid metabolism, we hypothesized that, during pregnancy, maternal ingestion of OFO, specifically oxidized components (i.e. the polar fraction), modulates PPARα or aryl hydrocarbon receptor (AhR) transactivity, altering the metab of retinoic acid (RA). Pregnant C57BL/6J mice were divided into four groups which, from d1 (conception) to d18, were fed a diet co ing 10 g/100 g of fresh soybean oil (SO, control), OFO or the non-polar (NP) or polar (PO) fraction of OFO. Reporter assays testing t transactivity of PPARα and AhR showed that free fatty acids from OFO, specifically the PO fraction, upregulated PPARα and downre lated AhR transactivity. Our results showed that the incidence of abnormalities in terms of gross morphology and skeletal ossificatio the fetus was greatest in the PO fraction group, followed by the OFO group, both values being significantly higher than in the othe groups. Hepatic expression of genes encoding enzymes associated with RA synthesis and catabolism in dams and fetuses was diffe tially affected by PO fraction assault. We conclude that OFO-mediated teratogenesis is associated with disturbed RA metabolism in dams and fetuses caused, at least in part, by modulation of PPARα and AhR transactivity by the oxidized components in OFO.