Gestational Diabetes is one of the most common complications of pregnancy, affecting more than 200,000 pregnant women per year in the United States. Previous pharmacological management of gestational diabetes was limited to insulin injections as it was suspected that oral hypoglycemic medications could cross the placenta and induce hypoglycemia in the fetus. However, recent research has shown that the breast cancer resistance protein (BCRP/ABCG2) efflux transporter in the placenta restricts fetal accumulation of the hypoglycemic drug glyburide by actively extruding the drug back into the maternal circulation. As a result, there has been a significant increase in the prescribing of glyburide to treat gestational diabetes. Investigation of risk factors that may decrease the ability of BCRP to efflux glyburide and prevent fetal exposure is therefore important for optimizing obstetrical care. For example, a genetic polymorphism at nucleotide position 421 (C/A) can decrease the ability of BCRP to transport drugs. Similarly, the dietary soy isoflavone, genistein can inhibit the activity and reduce the expression of BCRP in human placental choriocarcinoma cells. This presentation will highlight our research efforts which aim to determine whether dietary concentrations of genistein impair the ability of BCRP to efflux glyburide from the placenta by directly inhibiting transporter function and reducing its expression in placental trophoblasts. Importantly, this research will provide much needed data regarding genetic (C421A BCRP) and dietary (genistein) risk factors for fetal glyburide exposure and potentially neonatal hypoglycemia. These findings may lead to future clinical studies that improve the individualized prescribing of glyburide for women with gestational diabetes.