

LAY ABSTRACT

In 2004, California reported 36,483 infants out of 545,758 births (6.7%) had low birth weights (<2500 g). Since 1994 this amounts to an 8% increase in infants with low birth weight due to the increase in the number of pregnancies with multiples, however there are still ~5% of singleton pregnancies that result in low birth weight. There are lasting negative consequences of poor fetal growth that manifest in adulthood in the form of hypertension, obesity, diabetes and heart disease. The frequency of these diseases has substantially increased in our population and they represent a significant financial burden, as well as personal tragedy, to the state of California. A better understanding of the role of maternal nutrition in fetal growth will make a significant contribution to our ability to understand fetal growth, and reduce the risk for low birth weight. Our group is interested in the mechanisms by which select micronutrients in the maternal diet regulate fetal growth and affect infant birth weight. In this proposal we suggest that several biochemical pathways involved in the regulation of the growth of the fetus and its placenta are impacted by maternal zinc metabolism, and the amount of dietary zinc consumed during gestation. U.S. surveys report that 55% of women do not meet the recommended dietary allowance for zinc, an essential nutrient that is known to cause birth defects and low birth weight in humans who are deficient. Using a model for gestational zinc deficiency in the rat, we will investigate the mechanisms by which zinc can impact fetal growth, with a particular emphasis on placental function. These studies will provide new insights into appropriate dietary recommendations for the optimization of pregnancy outcome.