Nutritional effects on the regulation of prenatal and postnatal growth
(Nahrungabhängige Beeinflussung der Regulation von pränatalem und postnatalen Wachstum)

Prenatal growth. Prenatal growth is dependent on the ability of the mother to provide adequate nutritional support to the placenta and the fetus and the ability of the fetus to manage the maternal resources appropriately. The term ‘maternal constraint’ describes the limitation of fetal growth by the maternal environment and the mother’s capacity to supply nutrients to the fetus. Prenatal growth is therefore regulated by endocrine factors which influence the partitioning of nutrients between mother, placenta and fetus and regulate the utilisation of available substrate by the fetus.

Transition from prenatal to postnatal growth regulation. Prenatal growth and development are adapted to conditions of maternal and placental substrate limitation. After birth the neonatal animal does not need this acute link to the maternal system. The major rise in fetal glucocorticoids during the immediate pre-partum period initiates a switch from a fetal to a postnatal state of growth regulation. The essential role of this rise in fetal glucocorticoids on lung maturation is well known. However, perinatal glucocorticoids have a far more general role in switching fetal growth and metabolism to a postnatal state. For example, perinatal glucocorticoids play a major role in cardiac development, induction of hepatic gluconeogenic enzymes and activation of a variety of endocrine systems. The developmental switch to postnatal growth regulation allows growth to be directly linked to environmental factors and nutrition.

Influence of early life nutrition on postnatal growth and metabolism. The biological phenomenon of lifetime consequences of nutrition (and other environmental factors) during prenatal life on postnatal growth and metabolism is increasingly recognised. This concept has been termed the ‘fetal origins hypothesis’ and the process which underlies this concept has been termed ‘programming’. One general thesis is that the fetus adapts to adverse environmental cues in utero with permanent readjustments of homeostatic systems to maximise its chances for survival. These adaptations may include resetting of metabolic and endocrine systems and a change of growth trajectory. Recent studies have raised the possibility that changes in maternal nutrition and consequently altered materno-placental supply of nutrients may alter fetal metabolism and endocrine status with major postnatal health consequences compatible with the ‘fetal origins hypothesis’. In addition, there is increasing interest in the lifetime consequences of nutrition during infancy and its role on growth and metabolic health in later life. Scientific progress in these areas will be discussed to identify further opportunities for research in farm animals.