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Sommario/riassunto	There is extensive evidence from animal models that gonadal steroids, produced in fetal and neonatal life, act on the developing organism to produce sex differences far beyond the reproductive system. That early gonadal steroid exposure also plays an important role in human development is supported by studies of individuals with disorders of sex determination and differentiation. It is much less clear whether normal variation in gonadal steroid exposure predicts sexually dimorphic health outcomes or within-sex variation. This is largely due to challenges related to the assessment of gonadal steroid exposure in the developing fetus and neonate. Regarding the prenatal period, serial measurements of serum hormone levels in the fetus, for use in studies of later development, are not possible for ethical reasons. Researchers have measured hormones in maternal blood, umbilical cord blood, and amniotic fluid; used putative anthropometric indices such as the relative lengths of the 2nd and 4th digits (2D:4D); evaluated common variants in genes related to hormone production, transport, and metabolism; and examined development in opposite sex twins and the offspring of mothers with hyperandrogeny. Each of these approaches

has particular strengths and notable weaknesses. Regarding the neonatal period, serial measurements in serum are often impractical for studies of typical development. Salivary hormone assays, frequently used in studies of older children and adults, have not been extensively investigated in neonates. The most appropriate timing for testing is also open to debate. Early work suggested that testosterone levels in males begin to rise after the first postnatal week, peak around the 3rd to 4th months of life, and then drop back to very low levels by 1 year. However a more recent study of 138 infants did not demonstrate this pattern. Testosterone was highest on the day of birth and gradually dropped over the first 6 months. Even less is known about patterns of early estrogen exposure, though highly sensitive bioassays indicated that sex differences are present in early childhood. In addition, the design and interpretation of studies may be impacted by widespread acceptance of conceptual frameworks that are not well-supported empirically. For example, many researchers presume that the free hormone hypothesis, which states that unbound hormone is more readily diffusible into tissues and thus a better measure of actual exposure, is true. However this hypothesis has been challenged on multiple grounds. A second example: it is generally accepted that masculinization of the human brain is primarily mediated by the androgen receptor (in contrast to rodents where the estrogen receptor plays a major role), in part because chromosomal males with complete androgen insensitivity generally espouse a female gender identity. However this is not always the case, and other sexually dimorphic outcomes have not been carefully assessed in CAIS. The aim of this research topic is to gather together experimental and review papers which address the diverse challenges in assessing prenatal and neonatal gonadal steroid exposure for studies of human development with the expectation that this will allow more critical appraisal of existing studies, identify critical research gaps, and improve the design of future studies.