In the prenatal human, corticotropin-releasing hormone (CRH) derived from maternal placenta could influence the fetal hippocampus. Sustained stress during pregnancy activates the maternal neuroendocrine stress axis, resulting in increased production and release of placental CRH into the bloodstream. In contrast to hypothalamic CRH production, which is suppressed by stress-induced glucocorticoids (GCs), CRH-gene expression in placenta is enhanced by GCs, so that maternal stress leads to progressively higher fetal plasma CRH levels. This maternal-origin CRH reaches the fetal brain (red curved arrow) \[54\], influencing fetal learning and/or memory functions \[12,53\], presumably by activating hippocampal CRH receptors. Arrows indicate facilitatory pathways but do not imply monosynaptic connections. Blunt-ended lines denote inhibitory feedback.