Maternal hypothyroxinemia and brain development: II. Biochemical, metabolic and behavioural correlates.


Source
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Abstract
Using a rat model, we have investigated the influence of maternal hypothyroxinemia throughout pregnancy on brain development in young and adult progeny. Although no consistent change was observed in whole brain total protein concentration, the subcellular distribution of protein was adversely affected. Isolation of glycoprotein from developing brain by concanavalin A-affinity chromatography and subsequent resolution by gel electrophoresis revealed the selective compromise of particular glycoprotein species. Furthermore, both control and experimental progeny expressed unique glycoprotein species which either persisted over the period studied or were transient. Calcineurin, a regulator of neurite elongation, was compromised in young progeny, as were a number of lysosomal enzymes (beta-D-glucosidase and aryl sulphatase). In adult progeny, the content of cerebroside sulphate (a major myelin galactolipid) was reduced in midbrain and paleocortex, and brain region-specific compromise was observed for acetylcholine metabolic enzymes. These changes were associated with alterations in behavioural output. We conclude that the availability of maternal thyroxine to the fetus may be a critical determinant for normal brain development and function.

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