



# **Pediatric Neurology Part III: Chapter 170. Disorders of fatty acid oxidation (Handbook of Clinical Neurology)**

*Ingrid Tein*

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# Pediatric Neurology Part III: Chapter 170. Disorders of fatty acid oxidation (Handbook of Clinical Neurology)

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## **Pediatric Neurology Part III: Chapter 170. Disorders of fatty acid oxidation (Handbook of Clinical Neurology) Ingrid Tein**

Recognition of fatty acid oxidation (FAO) disorders is important for the pediatric neurologist as they present with a spectrum of clinical disorders, including progressive lipid storage myopathy, recurrent myoglobinuria, neuropathy, progressive cardiomyopathy, recurrent hypoglycemic hypoketotic encephalopathy or Reye-like syndrome, seizures, and mental retardation. They constitute a critical group of diseases because they are potentially rapidly fatal and a source of major morbidity. There is frequently a family history of sudden infant death syndrome in siblings. Early recognition and prompt institution of therapy and appropriate preventive measures, and in certain cases specific therapy, may be life-saving and may significantly decrease long-term morbidity, particularly with respect to CNS sequelae. All currently known conditions are inherited as autosomal recessive traits. There are now at least 25 enzymes and specific transport proteins in the  $\beta$ -oxidation pathway and 18 have been associated with human disease. The most common defect is medium-chain acyl-CoA dehydrogenase (MCAD) deficiency, which had an incidence of 1 in 8930 live births in one series. The identification of serum acylcarnitines by electrospray ionization-tandem mass spectrometry of dried blood spots on filter paper in newborn screening programs has significantly enhanced the early recognition of these disorders.

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