



Medline ® Abstracts for References 19,51,52 of 'Acute toxic-metabolic encephalopathy in children'

19 [PubMed](#)

TI Reye's and Reye's-like syndromes.

AU Pugliese A, Beltramo T, Torre D

SO Cell Biochem Funct. 2008;26(7):741.

The review reports various questions about Reye's syndrome and Reye's-like syndromes. Although there is a significant decrease in the classic Reye's syndrome cases, because of the reduced employment of salicylates in children (salicylate seems to be the most important inducing factor of the syndrome in paediatric subjects affected by viral infection), the problem is still of interest considering the presence of different Reye's-like forms. All these pathological situations are associated with various aetiologic or predisposing causes that are examined in the text. Particular attention is placed on metabolic disorders, especially of fatty acid metabolism, and also of one amino acid. In fact, a latent form can also be the basis of possible biochemical disturbances induced by various exogenous factors such as viral infections, particularly of the respiratory tract (more rarely of bacterial aetiology), or produced by microbial toxins, or by chemical substances, including some therapeutic drugs. A full discussion of biochemical mechanisms of salicylate-induced Reye's syndrome is reported. Finally a possible diagnostic differentiation from classic Reye's syndrome and Reye's-like syndromes plus therapeutic prospects are briefly examined.

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51 [PubMed](#)

TI Reye's syndrome in the United States from 1981 through 1997.

AU Belay ED, Bresee JS, Holman RC, Khan AS, Shahriari A, Schonberger LB

SO N Engl J Med. 1999;340(18):1377.

BACKGROUND: Reye's syndrome is characterized by encephalopathy and fatty degeneration of the liver, usually after influenza or varicella. Beginning in 1980, warnings were issued about the use of salicylates in children with those viral infections because of the risk of Reye's syndrome.

METHODS: To describe the pattern of Reye's syndrome in the United States, characteristics of the patients, and risk factors for poor outcomes, we analyzed national surveillance data collected from December 1980 through November 1997. The surveillance system is based on voluntary reporting with the use of a standard case-report form.

RESULTS: From December 1980 through November 1997 (surveillance years 1981 through 1997), 1207 cases of Reye's syndrome were reported in patients less than 18 years of age. Among those for whom data on race and sex were available, 93 percent were white and 52 percent were girls. The number of reported cases of Reye's syndrome declined sharply after the association of Reye's syndrome with aspirin was reported. After a peak of 555 cases in children reported in 1980, there have been no more than 36 cases per year since 1987. Antecedent illnesses were reported in 93 percent of the children, and detectable blood salicylate levels in 82 percent. The overall case fatality rate was 31 percent. The case fatality rate was highest in children under five years of age (relative risk, 1.8; 95 percent confidence interval, 1.5 to 2.1) and in those with a serum ammonia level above 45 microg per deciliter (26 micromol per liter) (relative risk, 3.4; 95 percent confidence interval, 1.9 to 6.2).