Paul Pencharz, MD, PhD, FRCPC: Pathogenesis of Obesity Caused By Disease
Obesity is a serious complication of diseases and their treatment. Obesity is caused by ingestion of more energy that is expended. Neuromuscular handicap often reduce energy expenditure. Cancer therapy, organ transplantation and psycho-tropic treatment involve the use of drugs which alter energy metabolism and increase the risk of obesity.

John J. Reilly, BSc, PhD: Energy Metabolism in Childhood Obesity: Recent Advances
Obesity is a disorder of chronic energy imbalance. The ‘rate’ of daily energy imbalance in modern children is much larger than expected. Low total energy expenditure (TEE), combined with the modern food environment, predispose to obesity, but conclusive empirical evidence of low TEE as a cause of obesity remains elusive. The presentation will discuss the complexities of energy expenditure in obesity etiology. Promising approaches for understanding the role of energy expenditure in the etiology of obesity – and in informing obesity prevention strategies- will be highlighted.

Jill Hamilton, MD & Greg Wells, PhD: Hypothalamic obesity
Hypothalamic obesity, a syndrome of intractable weight gain and hyperphagia, occurs commonly following treatment for pituitary or hypothalamic tumors. Postoperative obesity occurs in 35-60% of children, with increased cardiovascular disease risk and mortality in adulthood. We have been evaluating metabolic risk, including mitochondrial function using non-invasive magnetic resonance spectroscopy and imaging techniques in these youth during exercise, and before and after a pilot treatment trial of diazoxide and metformin.

Sonia Caprio, MD: Insulin Resistance and Ectopic Fat Distribution in Obese Adolescents
Although obesity is the most common cause of insulin resistance in children and adolescents, some obese youth may be very insulin sensitive and thus be at reduced risk for the development of the adverse cardiovascular and metabolic outcomes driven by insulin resistance. Studies from our group demonstrated that obese youth with impaired glucose tolerance (IGT) are significantly more insulin resistant than those with normal glucose tolerance (NGT), despite having an overall equal degree of adiposity. The difference in insulin sensitivity was attributed to different patterns of lipid partitioning – where those with severe insulin resistance were characterized by increased deposition of lipid in the visceral and intramyocellular (IMCL) compartments... Recent studies from our group found that some obese youngsters present with a much reduced amount of the superficial abdominal depot, increased visceral fat, hepatic steatosis and increased IMCL. In addition, they have low adiponectin, low leptin levels, marked insulin resistance and hypertryglyceridemia.

Tom Clandinin, PhD: The Role of LCPUFA in Obesity