in a number of ways. Many children voice frustration because their exacerbations interfere with their daily activities and social lives. Children need education on their condition and reassurance from the health team that they can learn to control and cope with their asthma and live a normal life.

Children in disruptive family situations (divorce, separation, violence, custodial battles) may disregard their daily asthma medication regimen or may be at higher risk as a result of neglect by adults who are in charge of their care. Adolescents struggling with a sense of identity and body image often regard asthma as a condition that will "go away," especially if there is a time lapse between symptoms, and may abandon the therapeutic regimen. Referral for counseling and guidance is appropriate where the child's or adolescent's life is potentially in harm's way and the therapeutic regimen for asthma is abandoned due to personal or family crises.

Cystic Fibrosis

CF is a life-shortening disease, inherited as an autosomal recessive trait. The affected child inherits the defective gene from both parents, with an overall risk of one in four if both parents carry the gene. The condition has a frequency of 1 in 3500 live births among Caucasians (Egan, Green, and Voynow, 2016). The mutated gene responsible for CF is located on the long arm of chromosome 7. This gene codes a protein of 1480 amino acids called the **cystic fibrosis transmembrane conductance regulator (CFTR)**. The CFTR protein is related to a family of membrane-bound glycoproteins. The glycoproteins constitute a cAMP-activated chloride channel and regulate other chloride and sodium channels at the surfaces of the epithelial cells.

Pathophysiology

CF is characterized by several clinical features, which are increased viscosity of mucous gland secretions, a striking elevation of sweat electrolytes, an increase in several organic and enzymatic constituents of saliva, and abnormalities in autonomic nervous system function. Although both sodium and chloride are affected, the defect appears to be primarily a result of abnormal chloride