

Curcumin, a major constituent of turmeric, corrects cystic fibrosis defects.

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Cystic fibrosis is caused by mutations in the gene encoding the cystic fibrosis transmembrane conductance regulator (CFTR). The most common mutation, DeltaF508, results in the production of a misfolded CFTR protein that is retained in the endoplasmic reticulum and targeted for degradation. Curcumin is a nontoxic Ca-adenosine triphosphatase pump inhibitor that can be administered to humans safely. Oral administration of curcumin to homozygous DeltaF508 CFTR mice in doses comparable, on a weight-per-weight basis, to those well tolerated by humans corrected these animals' characteristic nasal potential difference defect. These effects were not observed in mice homozygous for a complete knockout of the CFTR gene. Curcumin also induced the functional appearance of DeltaF508 CFTR protein in the plasma membranes of transfected baby hamster kidney cells. Thus, curcumin treatment may be able to correct defects associated with the homozygous expression of DeltaF508 CFTR.

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