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Thyroiditis in chronic obstructive pulmonary disease: a case of overlap syndrome. A 56-year-old female patient with end-stage chronic obstructive pulmonary disease (COPD) developed a thyroiditis of the hyperthyroid type in the absence of respiratory infections. Case reports and case series have established a higher prevalence of autoimmune thyroid disease in patients with COPD than in the general population. Literature data suggest that the lung inflammation may trigger the autoimmune process that involves the thyroid in the "overlap syndrome". In particular, a critical role of the proinflammatory cytokine TNF-alpha and Fas-Fas ligand interaction in the pathogenesis of autoimmune thyroiditis has been suggested. In the presented patient, the functional capacity of the thyroactive cells was markedly reduced whereas the typical features of the activated T-cell cytotoxicity such as CD8-positive lymphocytes were not demonstrable. A diagnostic challenge in the context of COPD lies in the fact that the clinical presentation of a thyroiditis is quite similar to the common COPD symptoms of cough and dyspnea. Thus, the association between the 2 diseases is often overlooked and the patient has to be treated for COPD before thyroid dysfunction can be treated. In the absence of other causes of thyroid dysfunction, a thyroiditis should be suspected in patients with COPD. We propose that the chronic inflammatory lung disease may play a role in the pathogenesis of this syndrome.

Peculiarities of storage proteins: molecular modeling of alpha-lactalbumin mutants. A rational design of mutants of alpha-lactalbumin was performed. The necessary modifications were estimated from a homology model based on the crystal structure of bovine beta-lactoglobulin and from the analysis of a large number of point mutations. This approach was employed to produce (1) a mutant of the bovine alpha-lactalbumin, with a marked increase in its thermal stability and (2) a mutant, that exhibited higher stability than the wild-type protein towards the oxidation of sulphydryl groups. The mutations did not alter the secondary structure and tertiary structure of the proteins. The mutant that had higher stability was constructed by substituting four residues

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