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Purification and Characterization of Pig Lung Carbonyl Reductase.

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A tetrameric pyrazole-sensitive carbonyl reductase was purified to homogeneity from pig lung. The enzyme reduced aliphatic and aromatic carbonyl compounds with NADPH as a preferable cofactor to NADH, and catalyzed the oxidation of secondary alcohols and the aldehyde dismutation in the presence of NAD (P). The pig enzyme exhibited negative cooperativity with respect to the carbonyl substrates. The hydrogen ion acted as an allosteric effector abolishing the negative interaction. Immunohistochemical study with the antibodies against the enzyme revealed that the enzyme was localized in the ciliated cells, nonciliated bronchiolar cells, Type II alveolar pneumocytes, and the epithelial cells of the ducts of the bronchial glands in the pig lung.

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Activation of Carbonyl Reductase from Pig Lung by Fatty Acids.

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The NADPH-linked reductase activity of pig lung carbonyl reductase was activated by fatty acids, especially by *cis*-unsaturated fatty acids. Arachidonic acid gave the highest activation. The binding of arachidonic acid to the enzyme was instantaneous and reversible, shifted the pH optimum of the activity from 5.8 to 6.5. In addition, arachidonic acid acted as an allosteric effector abolishing the negative interaction of the enzyme with carbonyl substrates which was seen without the fatty acid, but the activation increased both V_{max} and $[S]_{0.5}$ values for the substrates. These results suggest that *cis*-unsaturated fatty acids may be potential modulators of pulmonary carbonyl reductase.

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Structural and Functional Comparison of Two Human Liver Dihydrodiol Dehydrogenases Associated with 3α-Hydroxysteroid Dehydrogenase Activity.

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Human hepatic two monomeric dihydrodiol dehydrogenases were co-purified to homogeneity with androsterone dehydrogenase activity. The affinity of the two enzymes for the steroidal substrates was higher than that for the xenobiotic substrates. The two enzymes differed from each other on peptide mapping and in their heat-stabilities, and showed different inhibitor sensitivities to anti-inflammatory drugs and stereospecificities for xenobiotic alcohol substrates. These differences suggest that the two enzymes are 3α-hydroxysteroid dehydrogenase isoenzymes.