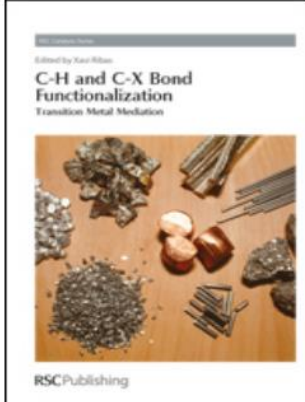
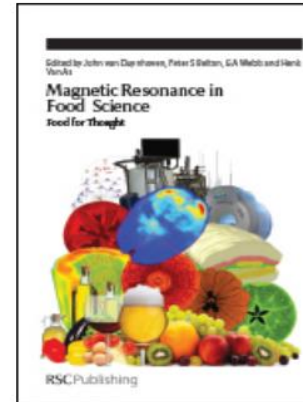
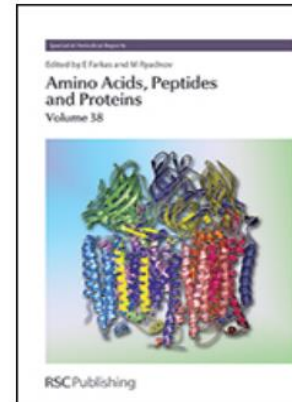
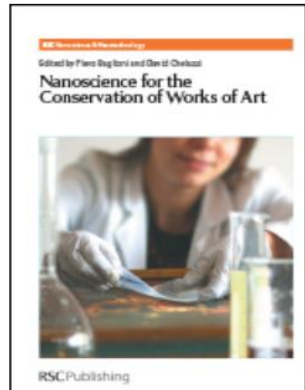
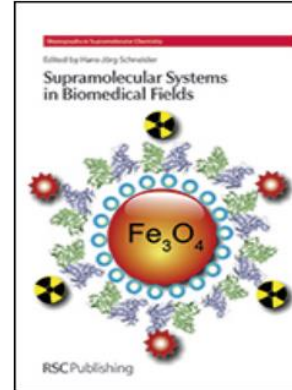
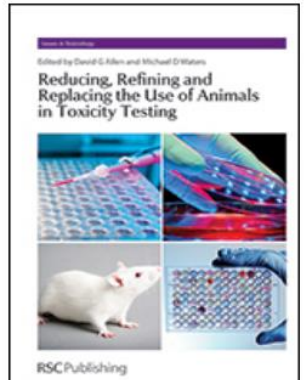
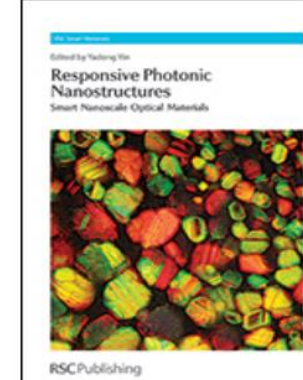
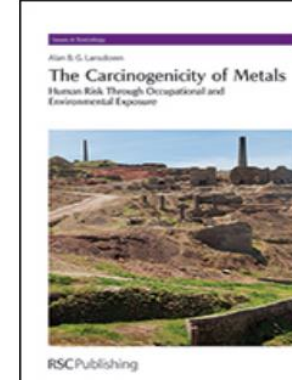
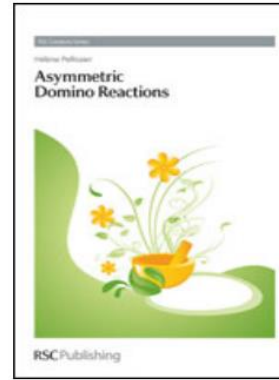


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About this book

Advances in Dermatological Sciences is a series of books that covers the latest achievements over the last 10 years in dermatological research. This book is the first in the series and is an internationally-recognized reference work explaining how new research is changing the way we think about the skin and what we can do to improve it.

1 INTRODUCTION

Carboxylesterases (CES) are present in a number of skin cells and have known physiological and exogenous substrates including drugs, chemicals, pro-drugs, carbamate and pyrethroid insecticides, cosmetic chemicals and environmental toxicants. A typical reaction scheme for CES is presented in Figure 1. During drug discovery and design, an ester linkage is frequently included to selectively target a pro-drug to a tissue or to improve dermal absorption and deliver a novel compound. Skin is an important tissue regulating uptake of environmental chemicals as well as a delivery site for drugs targeted locally to the skin and systemically.

Saitoh and Hasekawa [1] classified five groups of CES enzymes (CES 1-5) on the basis of amino acid homology and substrate specificity, with the majority of identified CES enzymes belonging to either the CES-1 or CES-2 sub-families. Amino acid sequence homology between human carboxylesterase 1 (hCE-1; a member of CES-1 family) and human carboxylesterase 2 (hCE-2; CES-2 family) is 40%. However, the substrate selectivity of these two enzymes is different. The hCE-1 enzyme readily hydrolyses substrates with low molecular weight alcohol groups and higher molecular weight acyl groups such as cocaine (methyl ester), ropivacaine, and diazepam. In contrast to hCE-1, the hCE-2 enzyme efficiently hydrolyses compounds with high molecular weight alcohol groups and relatively smaller carboxylate groups such as 4-methylumbelliferyl acetate, benzoic acid, and 4-acetylumbelliferyl CPT-11 or SN-38 [2]. The hCE-1 and hCE-2 genes encode the two major forms of human liver microsomal carboxylesterase enzymes.

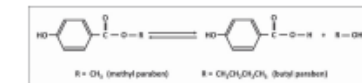


Figure 1 General reaction scheme for paraben hydrolysis.

Cutaneous Biology

Carboxylesterase activity has been found both in the microsomal and cytosolic fractions of the skin [3].

There are differences in the distribution of CES isoforms between extra-hepatic tissues (such as the small intestine, lung, skin) and the liver which results in differing hydrolysis profiles [4]. Shetage et al. [5] compared the differences in distribution between CES-1 and CES-2, but did not refer to skin. Hydrolysis profiles have been defined using isozyme specific substrates such as CPT-11 isozyme as pro-drug for CES-2 and methyl phenylacetate for CES-1. Several nuclear receptor (NR) family members regulate drug-inducible expression and activity of CES-1 and 2 in mammals liver and intestine but these have not been investigated in the skin [5].

Parabens (4-hydroxy benzoic acid esters) are substrates for CES enzymes. Parabens are widely used in typically applied cosmetic products and are known to penetrate the skin and have some oestrogenic effects in vivo. Parabens have been used as model substrates to identify carboxylesterase activity in human liver and skin [5]. Previous studies have shown that parabens are rapidly hydrolysed after oral administration by the liver [6]. The capacity for hydrolysis of the parabens in skin varies with the side-chain of the substrate. Lohmeyer et al. showed that in human skin and subcutaneous fat tissue, parabens could be hydrolysed by 4 different carboxylesterase enzymes classified by their isoelectric points [7]. Parabens are hydrolysed to 4-OH benzoic acid by carboxylesterases in both skin microsomes and in cytosol by CES-1 and CES-2 differentially relating to the leaving group. Inhibition by isoprenoids specific for CES-2

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