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An EMILIN1-Negative Microenvironment Promotes Tumor Cell Proliferation and Lymph Node Invasion
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Phase IB Randomized, Double-Blinded, Placebo-Controlled, Dose Escalation Study of Polyphenon E in Women with Hormone Receptor–Negative Breast Cancer

Deregulation of XPC and CypA by Cyclosporin A: An Immunosuppression-Independent Mechanism of Skin Carcinogenesis
Weinong Han, Keyoumars Soltani, Mei Ming, and Yu-Ying He
Gut-derived LPS promotes inflammatory hepatic injury and apoptosis by activating Toll-like receptor 4 (TLR4). Reduction of endotoxin using antibiotics regimen or TLR4 ablation in mice greatly attenuates hepatocyte apoptosis in a Con A–induced hepatitis model. Wild-type (wt) and TLR4 knockout (TLR4−/−) mice were injected with Con A intravenously and sacrificed 0, 6, and 20 hours thereafter. The micropictogram featured on the cover (magnification ×200) shows the apoptotic cells (brown) of liver sections from Con A-injected wt mice with antibiotic treatment using TUNEL assay in contrast to normal hepatocytes (green). Quantification of the apoptotic cells induced by Con A was significantly suppressed in both antibiotic-treated (P < 0.01) and TLR4−/− mice (P < 0.001; not shown) compared with control mice; this was further confirmed by the activation of caspase-3 and PARP demonstrating that the activation of TLR4-signaling pathway is important in Con A-induced hepatic injury in mice. See article by Lin et al. (beginning on page 1090) for more information.