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Prevention of Carcinogen and Inflammation-Induced Dermal Cancer by Oral Rapamycin Includes Reducing Genetic Damage
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Hepatitis B Virus X Protein Stabilizes Cyclin D1 and Increases Cyclin D1 Nuclear Accumulation through ERK-Mediated Inactivation of GSK-3β
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Sarsaparilla (Smilax Glabra Rhizome) Extract Inhibits Cancer Cell Growth by S Phase Arrest, Apoptosis, and Autophagy via Redox-Dependent ERK1/2 Pathway
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ABOUT THE COVER
Colorectal cancer is the third most common form of cancer in the United States and accounts for approximately 50,000 deaths annually. Although colonoscopic screening programs have significantly reduced cancer rates, colorectal cancer continues to inflict a significant health burden on the population. Identifying dietary agents and supplements that reduce the risk of colorectal cancer development could offer a powerful accompaniment to present screening efforts. It is, however, important to understand the context and limitations with which specific cancer prevention agents function in order to apply them effectively to responsive patient populations. Vitamin D has been implicated in colon cancer prevention, but its activity has proven difficult to establish in human intervention trials. Evidence has been obtained that colonic lesions may lose their responsiveness to vitamin D at early stages of development. The cover illustration depicts a colon tumor formed in the Apc^min/+ mouse analyzed for expression of the high affinity vitamin D receptor, VDR. VDR expression is lost in tumors formed in this preclinical model, which makes them less responsive to the gene regulatory actions of vitamin D. For more information on the potential mechanisms underlying VDR suppression, see the article by Giardina et al. (beginning on page 387).