Cytotoxicity and apoptosis inducing effects of phenylpropanoids.

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This study was carried out to determine cytotoxicity and apoptosis inducing effects of the seven pheylpropanoids. We have determined cytotoxocity by MTT(3-(4,5-dimethyl-thiazol-2-yl)-2,5-dipheryl-2H-tetrazo-liumbromide)assay and investigated the extendency of apoptosis by DAPI assay. The IC50s (ug/ml) of each phenylpropanoids by MTT(HL-60 cell) are 10.3(KYS50046), 15.9(KYS50047), >100 (KYS50049), >100 (KYS50050), 50.9(KYS50051), 52.3(KYS50153) and 48.36(KYS50154). Based on this result KYS50046 and KYS50047 showed significant cytotoxicity. When we observe Structure-activity relationship(SAR) of these compounds, KYS50049-51 which are unefficient in the cytotoxicity have methoxy group instead of hydroxy group on the phenol ring (ortho-form) compared to KYS50046-7. There is no sugar ring in KYS50153-4 which are also unefficient in cytotoxicity, whereas KYS50046-7 have sugar ring on the ring. So we suppose that the existences of hydroxy group on the phenol ring and sugar ring are *essential moiety* for cytotoxicity and apoptosis inducing effects.

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Hydroquinone from Coffee Modulates Reactivity of Peroxynitrite and Nitric Oxide Production

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Peroxynitrite (ONOO-), a potent cytotoxic oxidant formed by the reaction of nitric oxide (.NO) and superoxide radical (.O2-), may be rapidly lethal in a cellular mileu if left unchecked due to oxidization and nitration process. In the present study, we investigated ONOO- scavenging effect of hydroquinone from coffee, consisting of both beneficial and hazardous biochemical substances, and its biological effect on NO metabolism in the murine macrophage RAW 264.7.
Hydroquinone strongly scavenged ONOO--induced dihydrorhodamine 123 (DHR123) oxidation not by electron donation but by nitration of the compound itself in a dose-dependent manner. The compound also decreased ONOO--induced nitrotyrosine of GSH reductase, suggesting that hydroquinone directly neutralizes ONOO- and may prevent ONOO--induced damage of GSH reductase. Furthermore, hydroquinone also suppressed NO production, which is one of upstream sources via inhibition of inducible NO synthase (iNOS) expression in lipopolysaccharide (LPS)-activated RAW 264.7 cells. The inhibitory effect of hydroquinone was mediated through blocking LPS-induced signaling pathway, since hydroquinone potently inhibited nuclear factor-kB (NF-kB), and phosphorylation of extracellular signal related kinases 1 and 2 (ERK 1/2), a member of the MAPK family. Our results suggest that hydroquinone may be regarded as a potent regulator of ONOO--mediated diseases via both directly scavenging and indirectly blocking ONOO- production pathways, such as NO synthesis.

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Long-term Evaluation of the Mouse Model for Validity of Studying the Effects of Helicobacter pylori Infection on Gastric Carcinogenesis

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This study was aimed at evaluating the effect of *H. pylori* infection on gastric carcinogenesis. Four-week-old pathogen free C57BL/6 mice (n=115) were infected with SS1, the mouse-adapted *H. pylori* strain. *H.*