
The aryl hydrocarbon receptor (AhR) is involved in regulation of mechanisms for detoxification of xenobiotics, as well as vitamin A metabolism. Vitamin E is a fat-soluble nutrient whose metabolism is initialized via the cytochrome P450 system. Thus, AhR absence could alter hepatic regulation of α-tocopherol metabolism. To test this hypothesis, we assessed vitamin E status in adult (2-5 m) and old (21-22 m), wild-type and AhR-null mice. Plasma α-tocopherol concentrations in AhR-null mice (2.3±1.2 μmol/L, n=19) were lower than those of wild-type mice (3.2±1.2, n=17, P=.0131); those in old mice (3.2±1.2, n=20) were higher than those of adults (2.2±1.0, n=16, P=.0075). Hepatic α-tocopherol concentrations were not different between genotypes, but were nearly double in old (32±8 nmol/g, n=20) as compared with adult mice (17±2, n=16, P< .0001). Hepatic Cyp3a concentrations in AhR-null mice were greater than those in wild-type mice (P=.0011). Genotype (P=.0047), sex (P<.0001) and age (P<.0001) were significant modifiers of liver α-tocopherol metabolite (α-CEHC) concentrations. In general, Cyp3a concentrations correlated with hepatic α-tocopherol (r=0.3957, P<.05) and α-CEHC (r=0.4260, P<.05) concentrations. Since there were no significant genotype differences in the hepatic α- or γ-tocopherol concentrations, AhR-null mice did not have dramatically altered vitamin E metabolism. Since they did have higher hepatic α-CEHC concentrations, these data suggest metabolism was up-regulated in the AhR-null mice in order to maintain the hepatic tocopherol concentrations similar to those of wild-type mice.