Vitamin E status and metabolism in adult and aged aryl hydrocarbon receptor null mice. M. G. Traber, D. J. Mustacich, L. C. Sullivan, S. W. Leonard, A. Ahern-Rindell, N. Kerkvliet, *J. Nutr. Biochem.*, **21**, 1193-1199 (2010).

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 $(2.3\pm1.2 \,\mu\text{mol/L}, n=19)$ were lower than those of wild-type mice $(3.2\pm1.2, n=17, P=.0131)$; those in old mice $(3.2\pm1.2, n=20)$ were higher than those of adults $(2.2\pm1.0, n=16, P=.0075)$. Hepatic α -tocopherol concentrations were not different between genotypes, but were nearly double in old $(32\pm8 \text{ nmol/g}, n=20)$ as compared with adult mice $(17\pm2, n=20)$ 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 y-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.