

The present study was conducted to survey functional biomarkers for evaluation of niacin nutritional status. Over 500 enzymes require niacin as a coenzyme. Of these, we chose the tryptophan degradation pathway. To create niacin-deficient animals, quinolinic acid phosphoribosyltransferase-knock out mice were used in the present study because wild type mice can synthesize nicotinamide from tryptophan. When the mice were made niacin-deficient, the urinary excretion of xanthurenic acid (XA) was extremely low compared with control mice; however, it increased according to the recovery of niacin nutritional status. The urinary excretion of kynurenic acid (KA) was the reverse of XA. Kynurenine 3-monooxygenase, which needs NADPH, was thought to be suppressed by niacin deficiency. Thus, we calculated the urinary excretion ratio of XA:KA as a functional biomarker of niacin nutrition. The ratio increased according to recovering niacin nutritional status. Low values equate with low niacin nutritional status.

Effects of recovery from niacin deficiency on the urinary excretion ratio of XA/KA in mice. Low values equate to low niacin nutritional status.