Table 9. Effects of combined nutrients on the metabolism and distribution of MeHg.

Nutrient	MeHg exposure	Nutrient dose	Animal model	Duration of experiment	Effects	Ref.
Protective effects						
Cysteine and methionine	0.05 mM MeHgCl, 0.5 mL intracarotid injection	0.1 mM L-Cys, 0.1 mmol L-Met, 0.5 mL, intracarotid injection	Rat	15 s	Inhibited brain Hg uptake compared to cys alone	(*82*)
Cystine and selenium	15–25 ppm MeHgCl, in diet	0.4% L-cystine, 0.6 ppm selenite in diet, diet *ad libitum*	Rat	6–10 wk	Growth rate was similar to control diet; slightly decreased kidney Hg	(*70*)
Cystine and selenium	10 ppm MeHgCl, in diet	6 ppm selenite and 15 ppm cystine in diet	Japanese quail	16 wk	Prolonged survival time, improved egg production, improved fertility	(*71*)
Cystine and selenium	25 ppm MeHg, in diet	0.6 ppm selenite, 0.4% cystine of diet	Rat	10 wk	Prevented increase in SGPT and SGOT levels; effect was less than Se alone	(*177*)
Selenium and glutathione	0.3 µM MeHgCl	25 µM selenite and 5 mM GSH	*In vitro*	96 hr	Se and GSH decreased benzene-extractable Hg over time via cleavage of the Hg–C bond, but separately they did not; suggests reduction of selenite is needed for the degradation of MeHg	(272)
Selenium and methionine	10 ppm MeHgCl in diet	6 ppm selenite and 15 ppm Met in diet	Japanese quail	16 wk	Prolonged survival time, improved egg production, improved fertility	(*71*)
Selenium and vitamin E	1.4×10^{-5} M MeHgCl	1×10^{-5} M selenite; 1.0×10^{-5} M DL-α-tocopherol acetate	*In vitro* rat cerebrallar tissues	4 d	Protective effects of Se and vitamin E are additive	(*202*)
Selenium in tuna fish	0.05–20 ppm MeHgOH	0.49 ppm Se from tuna	Japanese quail	6 wk	Decreased MeHg toxicity and prolonged survival compared to corn, soya diet; decreased incoordination, mortality, growth inhibition	(*183*)
Selenium in tuna fish	20 ppm MeHgCl in diet	Tuna meal vs casein diet; natural Se in tuna or 0.5–1.5 ppm selenite	Rat	70 d	Se in tuna and selenite had same protective effect on growth; Se in tuna was half as effective as selenite in prevention of neurologic symptoms	(*273*)
Vitamin B complex	1 mg MeHgCl/kg bw/d, d 0–7, s.c. injection	20 mg vitamin B complex/kg bw/d, s.c. injection, d 8–14	Mouse	15 d	α-gal and β-gal activities recovered toward normal in brain and spinal cord; maximum recovery of α-gal was in spinal cord; α-gal recovered in kidney and testes; β-gal recovered in kidney but not liver and testes	(*180*)
Vitamin B complex	1 mg MeHg/kg bw/d, injection type NA, 7 d	20 mg vitamin B/kg bw/d complex, 7 days after Hg exposure, s.c. injection	Mouse	14 d	Mobilized Hg from all tissues; decrease in Na, K, Mg, Mn, Cu, Zn, Cr, and Ni for most organs was restored toward normal, but recovery was not complete; recovery of decreased Fe in brain and spinal cord but not kidney	(*127*)
Vitamin B complex	1 mg MeHgCl/kg bw/d, 7 d, s.c. injection	20 mg/kg bw, s.c. injection for 7 d after the 7-d MeHg treatment	Mouse	14 d	α- and β-Glycosidases activities recovered in brain, spinal cord; B-complex showed maximum recovery of β-glycosidase in the spinal cord; inhibition of liver and kidney enzyme activities was enhanced	(*103*)
Vitamin E and selenium	30 ppm MeHgCl in diet	0.05–0.6 ppm selenite and 10–500 IU α-tocopherol, in diet	Japanese quail	28–34 d	Vitamin E added to the protective effect of Se on mortality and clinical symptoms; vitamin E had a greater protective effect at low levels of Se; improved growth rate with larger effect attributed to vitamin E	(*274*)
Vitamin E and selenium	10–30 ppm MeHgCl in drinking water	50–500 ppm vitamin E diet and 0.1 ppm Se in diet for 8 wk	Rat	17–48 wk	Both Se and vitamin E protected signs of toxicity, growth and survival	(*252*)
Vitamin E, selenium	30 ppm MeHgCl in diet	500–1,000 IU α-tocopherol with 0.05–0.6 ppm selenite, in diet	Japanese quail	28–34 d	Protected toxicity (altered hematocrit, decreased bond calcification, survival rate); vitamin E provided little added protection at high levels Se	(*204*)
Vitamin E, selenium	10 ppm MeHg in diet	100–500 mg D-α-tocopherol acetate; 0.6 ppm selenite,in diet	Japanese quail	18 wk	Combined treatment offered more protection than either alone	(*208*)
Vitamins E and A	10–15 ppm MeHgCl in drinking water	50, 500 ppm vitamin E, 2,000–10,000 IU vitamin A/kg, in diet	Rats	NA	Protected toxicity at high vitamin E concentrations but not at low vitamin E concentrations (growth, morbidity, mortality)	(*148*)
Enhanced toxicity						
Methionine and protein	20 µmol Hg as MeHg/kg bw, oral administration	1% Met in diet to 7.5% or24.8% protein diet	Rat	NA	Met increased Hg in brain with low protein but not high protein diet; Met increased Hg in liver, plasma and decreased Hg in kidney	(*257*)

(*Continued*)

Table 9. *Continued.*

Nutrient	MeHg exposure	Nutrient dose	Animal model	Duration of experiment	Effects	Ref.
Vitamin C and copper	100 nM MeHg	10 µM copper sulfate with 100 µM L-ascorbate	*In vitro* (rat brain cells)	10 d	Increased oxygen reactive substances, and decreased activities of antioxidant enzymes, when effects were not observed with Hg alone	(275)
Other effects						
Selenium-L-methionine	1 nmol MeHgCl/mL, 5 wk, during and after pregnancy, in drinking water	3 µg Se–Met/mL in drinking water (diet already contains 0.9 ppm Se)	Mouse	9–10 wk	Percent Hg deposited in offspring *in utero* and during lactation was not influenced by Se–Met	(*276*)