Table 8. Effects on the metabolism and distribution of methyl mercury: vitamins and phytochemicals.

Nutrient	MeHg exposure	Nutrient dose	Animal model	Duration of experiment	Effects	Ref.
Protective effects						
Pantothine	80 ng Hg/mL as MeHgCl in tank water	10 µg pantothine/mL water in tank	Goldfish	24 hr	Decreased MeHg uptake by fish	(*250*)
Coenzyme A	80 ng Hg/mL as MeHgCl in tank water	0.6 µg coenzyme-A/mL water	Goldfish	24 hr	Protected fish against MeHg uptake	(*250*)
Vitamin B_{12}	1 mg MeHgCl/kg bw/d, 7 d, s.c. injection	2 mg/kg bw, s.c. injection for 7 d after 7-d MeHg treatment	Mouse	14 d	α- and β-Glycosidase activities recovered in brain, spinal cord; inhibtion of liver and kidney enzyme activities enhanced	(*103*)
Vitamin B_{12}	1 mg MeHgCl/kg bw/d, d 0–7, s.c. injection	2 mg vitamin B_{12}/kg bw/d, d 7–14, s.c. injection	Mouse	15 d	α-gal and β-gal activities recovered toward normal in brain and spinal cord; spinal cord had maximum recovery of β-gal; β-gal recovered in kidney and testes; α-gal recovered in kidney	(*180*)
Vitamin C	1 mg MeHgCl/kg bw/d, 7 d, s.c. injection	5 mg vitamin C/kg bw, s.c. injection for 7 d after 7-d MeHg treatment	Mouse	14 d	α- and β-Glycosidases activities recovered in brain, spinal cord; vitamin C showed maximum α-gal activity compared to other vitamins; inhibition of liver and kidney enzyme activies was enhanced	(*103*)
Vitamin C	1 mg MeHgCl/kg bw/d, d 0–7, s.c. injection	5 mg vitamin C/kg bw/d, d 7–14, s.c. injection	Mouse	15 d	α-gal and β-gal activities recovered toward normal in brain and spinal cord; β-gal recovered in kidney and testes; α-gal recovered in kidney but not in liver and testes	(*180*)
Vitamin E	10^{-5} M MeHgCl in buffer	$0.4–2.0 \times 10^{-5}$ M DL-α-tocopherol acetate	*In vitro* rat (cerebrallar tissues)	4 d	Inhibited toxic effect of MeHg on development of nerve fibers, glial cells, and fibroblasts	(*144*)
Vitamin E	10–40 mg MeHgCl/L in drinking water	10, 100 or 1,000 mg α-tocopherol/kg in diet	Mouse	2 wk	High tocopherol in diet protected against MeHg-induced lipid peroxidation in liver; deficient diet enhanced MeHg-induced lipid peroxidation; protected GSH–peroxidase activity	(*146*)
Vitamin E	4 µM MeHgCl	5, 7, and 10 µM DL-α-tocopherol acetate	*In vitro* embryonic neural retinal cells	24 hr	Provided protective effect on cell aggregation compared to MeHgCl alone; less effect than Se	(*200*)
Vitamin E	15 ppm MeHgCl in diet	0.05% all rac-α-tocopherol acetate in diet	Japanese quail	22 d –29 d	Protected against Hg-induced mortality	(*142*)
Vitamin E	30 ppm MeHgCl, in diet	500–1,000 IU vitamin E, in diet	Japanese quail	28–34 d	Protective effect was eventually overcome by toxic effect of Hg	(204)
Vitamin E	10–30 ppm MeHgCl, in drinking water	50–500 ppm vitamin E, in diet	Rat	6–13 wk	Fewer signs of toxicity and greater growth and survival over Hg alone	(*252*)
Vitamin E	20 mg MeHgCl/kg bw	2.0 ppm DL-α-tocopherol acetate/d, s.c. injection, 4 wk	Golden hamster	4 wk	Prevented signs of ill health: ataxia and paralysis of hind limbs as well as extensive lesions in cerebellum, loss of granule celles, glial fibers, necrosis, and active phagocytosis of debris	(*143*)
Vitamin E	2 ppm MeHg/d, injection type NA	2 ppm vitamin E/d, injection type NA	Hamster	4 wk	Prevented neurologic disturbances (degenerative changes in granule cells, morphologic changes)	(*253*)
Vitamin E	25 ppm MeHgCl in diet	50–500 mg/kg vitamin E in diet	Japanese quail	15 d	Protected mortality; high level had no additional protection compared to low level	(*84*)
Vitamin E	10 ppm MeHgCl in diet	700 mg/kg all rac-α-tocopheryl acetate in diet	Japanese quail	21 d	Did not affect GSH–peroxidase activity; Hg did not alter lipid peroxidation	(*141*)
Vitamin E	32 ppm MeHgCl in diet	Vitamin E (not given), dose NA	Japanese quail	24 d	Prevented decrease in SGOT levels in severe Hg toxicity; did not affect SGPT levels	(*207*)
Vitamin E	1 mg MeHg/kg bw/d, 7 d, injection	60 mg vitamin E/kg/d s.c. injection 7 d, after Hg exposure	Mouse	14 d	Restored decrease in Na, K, Mg, Mn, Cu, Zn, Cr, and Ni for most organs toward normal, but recovery was not complete; Fe decrease in brain and spinal cord did not recover; kidney Fe decreased	(*127*)
Vitamin E	1 mg MeHgCl/kg bw/d, 7 d, s.c. injection	60 mg vitamin E/kg bw, s.c. injection for 7 d after 7-d MeHg treatment	Mouse	14 d	α- and β-Glycosidase activities recovered in brain, spinal cord; vitamin E showed maximum recovery of β-glycosidases in the brain compared to other vitamins; liver and kidney enzyme activities inhibition was enhanced	(*103*)
Vitamin E	0.4 µM MeHgCl	10^{-5} to 10^{-2} M vitamin E	*In vitro* mouse neuroblastoma cells	3 d	Protected toxicity	(*251*)
Vitamin E	1 mg MeHgCl/kg bw/d, d 0–7, s.c. injection	60 mg vitamin E/kg bw/d, d 7–14, s.c. injection	Mouse	15 d	α-gal and β-gal activities recovered toward normal in brain and spinal cord; maximum recovery was in the brain; β-gal was further inhibited in kidney and testes; α-gal recovered in kidney but not in liver and testes	(*180*)

(*Continued*)

Table 8. *Continued.*

Nutrient	MeHg exposure	Nutrient dose	Animal model	Duration of experiment	Effects	Ref.
Vitamin E	10 ppm in drinking water	500 ppm vitamin E in diet with 0.1 ppm Se	Rat	Not given	Protected against toxicity (growth, neurologic symptoms, survival); suggests role of vitamin E is not just to spare Se	(*254*)
Vitamin E	10 ppm MeHg, in diet	100–500 mg D-α-tocopherol acetate, in diet	Japanese quail	18 wk	Protected survival; effect was dose dependent; protection extended from parents to offspring	(*208*)
Vitamin E	2 mg MeHg/kg bw, injection type NA	2 ppm α-tocopherol acetate in diet, injection type NA	Golden hamster	4 wk	Prevented toxic symptoms (neural damage, necrosis in cerebellum and calcarine cortex, ataxia, paralysis of hind limbs)	(*143*)
Vitamin E (γ-tocopherol)	15 ppm MeHgCl in diet	0.05% γ-tocopherol acetate in diet	Japanese quail	NA	Protected against Hg-induced mortality	(*142*)
Enhanced toxicity						
β-Carotene	10–40 mg MeHgCl/L in drinking water, 2 wk	1,000, 10,000, or 100,000 IU β-carotene/kg bw, in diet	Mouse	4 wk	Dietary excess of β-carotene enhanced MeHg-induced lipid peroxidation in brain, liver, and kidney	(*146*)
Vitamin A	10–15 ppm MeHgCl in drinking water	2,000–10,000 IU vitamin A/kg bw in diet	Rat	NA	Enhanced toxicity (growth, morbidity, mortality)	(*148*)
Vitamin C	20 µM MeHgOH, aqueous	0.4 mM ascorbate, aqueous	*In vitro* rat liver	10 min	Released Hg^0 and Hg^{2+}; Hg^{2+} release was proportional to ascorbate concentration	(*193*)
Vitamin C	0.4 µM MeHgCl	20–120 µg L-ascorbate/mL	*In vitro* mouse neuroblastoma cells	3 d	Enhanced toxicity	(*251*)
Other Effects						
Pantothenate	80 ng Hg/mL as MeHgCl in tank water	10 µg calcium pantothenate/mL water	Goldfish	24 hr	No effect on MeHg uptake by fish	(*250*)
Vitamin A	MeHg, dose NA	Vitamin A, dose NA	*In vitro* tissue culture	NA	NA	(*255*)
Vitamin C	0.4 µM MeHgCl	20–120 µg L-ascorbate/mL	*In vitro* rat glioma cells	3 d	No effect	(*251*)
Vitamin C	Nonoccupational environmental exposure	500 or 1,000 mg L-ascorbic acid/d for 3 months	Human	3 mo	No effect of vitamin C on Hg body burden as measured by hair and blood Hg	(*44*)
Vitamin C	80 ng Hg/mL as MeHgCl in water	10–1,000 times higher than Hg on a molar basis	Gold fish	24 hr	Reduction of MeHg toxicity was not consistent; possibly a role for vitamin C in the degradation of MeHg to Hg^{2+}; increased degradation increased mortality of fish	(*256*)
Vitamin E	0.4 µM MeHgCl	10^{-5} to 10^{-2} M vitamin E	*In vitro* mouse neuroblastoma cells	3 d	No effect	(*251*)
Vitamin E	13 ppm MeHgCl in drinking water	50 ppm vitamin E in diet compared to 275 ppm synthetic antioxidant DPPD	Rat	2 mo	DPPD decreased liver and brain Hg but increased kidney Hg compared to vitamin E	(*252*)
Vitamin E	20 ppm MeHgCl in drinking water	50 ppm vitamin E in diet compared to 275 ppm synthetic antioxidant DPPD	Rat (Se deficient)	7–9 wk	Vitamin E was not able to protect toxicity, but synthetic antioxidant DPPD did	(*252*)

DPPD, *N,N´*-Diphenyl-*p*-phenylenediamine.