Transfer of a cyanobacterial neurotoxin within a tempe pathways for human exposure

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Citation Report

#	Article	IF	CITATIONS
1	β-N-Methylamino-L-Alanine Induces Neurological Deficits and Shortened Life Span in Drosophila. Toxins, 2010, 2, 2663-2679.	1.5	25
2	The Cyanobacteria Derived Toxin Beta-N-Methylamino-L-Alanine and Amyotrophic Lateral Sclerosis. Toxins, 2010, 2, 2837-2850.	1.5	89
3	Distinguishing the cyanobacterial neurotoxin β-N-methylamino-l-alanine (BMAA) from its structural isomer 2,4-diaminobutyric acid (2,4-DAB). Toxicon, 2010, 56, 868-879.	0.8	63
4	Distinguishing the cyanobacterial neurotoxin β-N-methylamino-l-alanine (BMAA) from other diamino acids. Toxicon, 2011, 57, 730-738.	0.8	59
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6	Determination of the non protein amino acid β-N-methylamino-l-alanine in estuarine cyanobacteria by capillary electrophoresis. Toxicon, 2011, 58, 410-414.	0.8	27
7	Contribution of geolocalisation to neuroepidemiological studies: Incidence of ALS and environmental factors in Limousin, France. Journal of the Neurological Sciences, 2011, 309, 115-122.	0.3	29
8	Early hippocampal cell death, and late learning and memory deficits in rats exposed to the environmental toxin BMAA (l²-N-methylamino-l-alanine) during the neonatal period. Behavioural Brain Research, 2011, 219, 310-320.	1.2	76
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15	Solid phase extraction of β-N-methylamino-L-alanine (BMAA) from South African water supplies. Water S A, 2011, 37, .	0.2	3
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