

**RESPONSES OF ATLANTIC SALMON
AND BIVALVE MOLLUSCS TO
PARALYTIC SHELLFISH**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Paralytic shellfish poisoning (PSP) toxins are a group of potent neurotoxins produced by certain strains of marine dinoflagellates. Blooms of these algal species can result in the passage of PSPs through marine food webs, with detrimental effects on the marine environment and human health. These toxins have been implicated as the causative agent in some of the many fish kills that have occurred during blooms of PSP producing dinoflagellates. As such, PSPs represent a potential threat to fisheries resources and aquaculture. Little is known of the fate of these compounds in fish, but they have been shown to accumulate in the liver of mackerel sampled after bloom events. Analysis of fish tissues also suggests that transformation of these toxins does occur following absorption since the PSP analogues differ from those to which the fish were exposed. The induction of xenobiotic metabolising enzyme activities has also been noted in Atlantic salmon exposed to PSPs and may represent a detoxification mechanism.

Better understood is the fate of PSPs in shellfish. Bivalve molluscs accumulate significant of PSPs during toxic blooms and pose a serious health risk to vertebrate consumers. Many species of marine invertebrates are reported to be resistant to the effects of neurotoxins, a trait that is innate in some species and acquired in others. There are reports of PSP biotransformations in shellfish,

namely epimerisations, decarbamoylations and reductive elimination's that alter the profile of the toxins as they are transferred from causative dinoflagellate to shellfish tissue. There are also reports of proteins, inducible on exposure to PSPs that can bind to saxitoxin. Such a mechanism may be responsible for inactivating PSPs and could confer resistance to invertebrates that express these proteins.

Little is known about the sub-lethal effects of exposure from this group of toxins on marine organisms. Laboratory based exposure experiments on Atlantic salmon (*Salmo salar*) indicate that intra-peritoneal exposure to low doses (2-4 µg/kg) of saxitoxin causes an induction of hepatic glutathione S-transferase (GST) activity within four days. Doses approximating the LD50 for this compound (4 µg/kg) had little effect on blood plasma ionic concentration of surviving fish.

Mussels (*Mytilus edulis*), like other invertebrates, appear insensitive to the paralytic effects of PSTs. Exposure to high doses (intra-muscular, >100 µg/100g soft tissue) of saxitoxin, however, causes an induction of digestive gland GST activity. This is in contrast to scallops (*Pecten maximus*) which showed no induction of GST activity after acquiring high digestive gland toxicities from feeding on cultures of toxic dinoflagellates. After toxic events, scallops retain PSTs considerably longer than mussels. It is suggested that the induction response of GST in mussels may be partly responsible for this discrepancy in toxicokinetics between the two species.

In conclusion further work is required to define the metabolic pathways leading to the detoxification and excretion of saxitoxins and related compounds.

References

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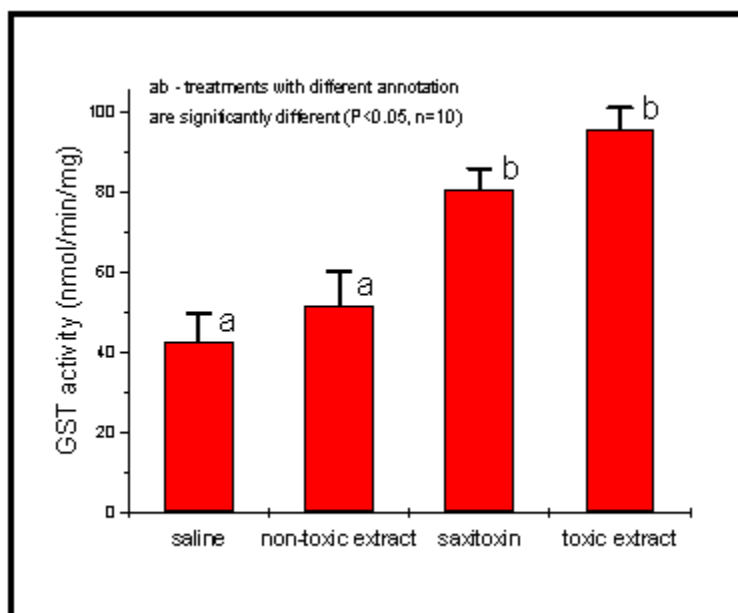


Fig 1. Hepatic glutathione S-transferase (GST) activities of Atlantic salmon administered intra-peritoneal injections of physiological saline, saxitoxin and extracts of a toxic (*Alexandrium fundyense*) and a non-toxic (*Scrippsiella trochoidea*) strain of dinoflagellate over 21 days. Error bars represent + or - SEM, $n = 10$ (except for saxitoxin group where $n = 9$). Ab, groups with different notation are significantly different ($P < 0.05$)

