Original

Accumulation and Elimination Profiles of Paralytic Shellfish Poison in the Short-necked Clam *Tapes japonica* Fed with the Toxic Dinoflagellate *Gymnodinium catenatum*

(Received August 15, 2006)

Mohamad SAMSUR[1], Tomohiro TAKATANI[2], Yasunaga YAMAGUCHI[3], Takefumi SAGARA[4], Tamao NOGUCHI[5] and Osamu ARAKAWA[2,*]

[1] Graduate School of Science and Technology, Nagasaki University: 1–14, Bunkyo-machi, Nagasaki-shi, Nagasaki 852–8521, Japan;
[2] Faculty of Fisheries, Nagasaki University: 1–14, Bunkyo-machi, Nagasaki-shi, Nagasaki 852–8521, Japan;
[3] Maruzen Pharmaceuticals Corporation Ltd.: 14703–10, Mukaihigashi-machi, Onomichi-shi, Hiroshima 722–0062, Japan;
[4] Faculty of Junior College, Shikoku University: 123–1, Ebisuno, Furukawa, Ohjin-cho, Tokushima-shi, Tokushima 771–1192, Japan;
[5] Tokyo Health Care University: 3–11–3, Setagaya, Setagaya-ku, Tokyo 154–8568, Japan;
* Corresponding author

The paralytic shellfish poison (PSP)-producing dinoflagellate *Gymnodinium catenatum* (*Gc*) was fed to the short-necked clam *Tapes japonica*, and the accumulation, transformation and elimination profiles of PSP were investigated by means of high-performance liquid chromatography with postcolumn fluorescence derivatization (HPLC-FLD). The short-necked clams ingested most of the *Gc* cells (4×10^6 cells) supplied as a bolus at the beginning of the experiment, and accumulated a maximal amount of toxin (181 nmol/10 clams) after 12 hr. The rate of toxin accumulation at that time was 16%, which rapidly decreased thereafter. During the rearing period, a variation in toxin composition, derived presumably from the transformation of toxin analogues in the clams, was observed, including a reversal of the ratio of C2 to C1, and the appearance of carbamate (gonyautoxin (GTX) 2, 3) and decarbamoyl (dc) derivatives (decarbamoylsaxitoxin (dcSTX) and dcGTX2, 3), which were undetectable in *Gc* cells. The total amount of toxin contained in clams and residue (remaining *Gc* cells and/or excrement in the rearing tank) gradually declined, and only about 1% of the supplied toxin was detected at the end of the experiment.

Key words: paralytic shellfish poison; short-necked clam; *Tapes japonica*; dinoflagellate; *Gymnodinium catenatum*

Introduction

Along the coastal waters off Kyushu in Japan, paralytic shellfish poison (PSP)-producing dinoflagellates, *Alexandrium catenella* (designated *Ac* below) and *Gymnodinium catenatum* (designated *Gc* below), occur every year, and intoxicate many edible bivalves such as the short-necked clam *Tapes japonica*, the mussel *Mytilus galloprovincialis* (formerly classified as *M. edulis*), the oyster *Crassostrea gigas*, and the scallop *Clamys nobilis*, presenting a serious problem for the fisheries industriy[1)–4)].

Accumulation and elimination profiles of PSP in bivalves differ greatly depending on the bivalve species[3), 5), 6)]. The mussel rapidly reaches a high level of toxicity, which declines shortly (usually within several weeks) after disappearance of the causative dinoflagellate. Scallops, such as *Patinopecten yessoensis* and *Chlamys nipponensis*, are known to become much more toxic than the mussel under the same environmental conditions, and require several months until the toxicity drops below the regulation level. On the other hand, levels of intoxication of the short-necked clam and the oyster are generally low, and the toxicity quickly declines, as in the mussel.

PSP compositions of bivalves are not necessarily the same as those of the causative dinoflagellates, and interspecies differences are also observed among bivalves collected from the same area at the same time. For example, the mussel and the oyster better reflect the composition of the causative plankton[1), 7), 8)], whereas clams such as *Meretrix lamarckii*, *Pseudocardium sachalinensis* and *Protothaca staminea* often show very different compositions[9), 10)]. The difference of toxin profiles between the bivalves and the causative plankton is considered to arise mainly from the enzymatic and/or chemical transformation of toxins in bivalve tissues[9), 11)–14)], and possible participation of selective