Effects of perfluorooctane sulfonate (PFOS) on swimming behavior and membrane potential of paramecium caudatum. (ゾウリムシ尾状核の遊泳行動および膜電位に関するペルフルオロオクタタン sulfonate (PFOS)の影響)


Persistent perfluorinated organic compounds such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) were distributed widely in the global. PFOS (15 microM or higher) caused backward swimming of paramecia. The Triton-extracted paramecia, where the membrane was disrupted and the externally applied chemicals are freely accessible to the ciliary apparatus, showed forward swimming up to 0.1 microM Ca2+ in the medium and backward swimming at about 0.2 microM and higher. PFOS (0.1 mM) did not change the relationship between the swimming directions and free Ca2+ concentrations. Effects of various surfactants including PFOS and PFOA on the swimming direction of paramecia were compared with the hemolysis of mouse erythrocytes as an indicator of surfactant activities. The hemolysis did not correlate with their swimming behavior. PFOS caused triphasic membrane potential changes both in the wild-type paramecia and caudatum non-reversal (CNR) mutants, the latter is defective in voltage-gated Ca2+ channels. An action potential of the wild-type specimen was induced at lower current intensity when PFOS was present in the medium. Voltage-clamp study indicated that PFOS had no effect on the depolarization-induced Ca2+ influx responsible for the action potential. The membrane potential responses obtained were similar to those obtained by the application of some bitter substances such as quinine that activate chemoreceptors of paramecia. Since the CNR specimens did not exhibit PFOS-induced backward swimming at concentrations examined, the backward swimming is attributable to the influx of Ca2+ into the cilia through voltage-gated Ca2+ channels. The Ca2+ channels are most probably activated by the depolarizing receptor potentials resulted from the PFOS-induced activation of chemoreceptors.

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