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Uptake and antiproliferative effect of molecular iodine in the MCF-7 breast cancer cell line

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Abstract

This study analyzes the uptake and antiproliferative effect of two different chemical forms of iodine, iodide (I-) and molecular iodine (I2), in MCF-7 cells, which are inducible for the Na+/I- symporter (NIS) and positive for pendrin (PDS). The mouse fibroblast cell line NIH3T3 was used as control. Our results show that in MCF-7 cells, I- uptake is sustained and dependent on NIS, whereas I2 uptake is transient with a maximal peak at 10 min and a final retention of 10% of total uptake. In contrast, no I- was taken up by NIH3T3 cells, and although I2 was captured with the same time pattern as in MCF-7 cells, its uptake was significantly lower, and it was not retained within the cell. The uptake of I2 is independent of NIS, PDS, Na+, and energy, but it is saturable and dependent on protein synthesis, suggesting a facilitated diffusion system. Radioiodine was incorporated into protein and lipid fractions only with I2 treatment. The administration of non-radiolabeled I2 and 6-iodo-5-hydroxy-8,11,14-eicosatrienoic acid (6-iodolactone, an iodinated arachidonic acid), but not KI, significantly inhibited proliferation of MCF-7 cells. Proliferation of NIH3T3 cells was not inhibited by 20 microM I2. In conclusion, these results demonstrate that I2 uptake does not depend on NIS or PDS; they suggest that in mammary cancer cells, I2 is taken up by a facilitated diffusion system and then covalently bound to lipids or proteins that, in turn, inhibit proliferation.

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