Magnesium Stearate (Stearic Acid) Research

Ingested Stearic Acid shown to cause inhibition of T -cell response leading to suppression of immune function

Comment

The mechanism by which ingested stearic acid [18:0] selectively kills T-cells was examined. The protocol adopted used highly enriched population ofB and T lymphocytes (.95%) and albumin complex fatty acids.

In T-cells, the accumulation of destructive stearic acid (18:0) containing phosphatidyl choline coincided directly with a rapid collapse of the cell membrane integrity. No such depolarization was observed in B-cells.

The ultimate effect of the observed changes in the membrane composition of the stearic acid (18:0) treated T-cells lead to the T-cell membrane collapse and cell death.

The selective toxicity of stearic acid (18:0) to the T -cells and its rapid mechanism of action should be a clinical consideration.

It is estimated that 90% of the vitamin/mineral products consumed today contain magnesium stearate. Immune compromised patients who are chronically ill, seeking to deal with a primary cause, nutrional deficiency, often take handfuls of capsules to get vitamin, mineral and other key nutrients from capsules that contain stearic acid or magnesium stearate and get a powerful immuno suppressive treatment.

Most retailers and distributors are not aware of this threat and mistakenly claim 100% purity for their products. Ask your suppliers to provide a written statement that guarantees their supplements are free of stearates and other potentially harmful manufacturing additives. In fact, ask for full label disclosure listing of every component used to produce each product.

Abstract

Studies were performed to determine the mechanism by which stearic acid (18:0) selectively inhibits T-dependent immune responses in vitro. Incubation of mitogen-activated B and T cells with 18:0 resulted in dissimilar patterns of incorporation of the saturated fatty acid into their membranes. High-performance liquid chromatography (HPLC) analyses of T cells showed an accumulation of disaturated [corrected] 18:0-containing phosphatidylcholine (PC) that replaced normal cellular PC. Less significant quantities of the same PC species were seen to accumulate in B-cell membranes; rather, they increased their proportion of oleic acid (18: 1)-containing PC. The different lipid compositions of the lymphocyte cell membranes after exposure to 18:0 were correlated with their plasma membrane potentials. In T cells, the accumulation of disaturated [corrected], 18 :O-containing PC coincided with a rapid (within 8hr) collapse of membrane integrity, as determined by flow cytometry. The collapse of membrane integrity was found to be time and dose dependent. No such depolarization was observed in B cells which, by virtue of their desaturating ability, were able to avoid incorporating large amounts of disaturated [corrected] 18:0-containing phospholipids into their membranes. It is proposed that a lack of stearoyl-CoA desaturase in T cells precludes them from desaturating exogenously derived 18:0, thus leading to increase proportions of 18:0-containing disaturated [corrected] PC in their cell membranes. The increased abundance of this PC species may enhance membrane rigidity to an extent that plasma membrane integrity is significantly impaired, leading to a loss of membrane potential and ultimately cell function and viability.

Citation: Tebbey PW, Buttke TM. Molecular basis for the immunosuppressive action of stearic acid [magnesium stearate] on T cells. *Immunology,* 1990 Jul; 70(3):379-86.
Int J Immunopham1990; 12(7):799-807

Identification of T -helper cells as the target of stearic acid Inhibition in primary antibody responses in vitro. -- Pourbohloul SC, Buttke TM.

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We have previously shown that albumin-complexed stearic acid (18 :0) inhibited in vitro primary anti-TNP plaque-fonning cell (PFC) responses to trinitrophenyl keyhole limpet hemocyanin (TNP-KLH), but did not affect primary PFC responses to trinitrophenyl lipopolysaccharide (TNP-LPS). The present studies were done to identify the cellular target of fatty acid inhibition. The addition of 18:0 at the initiation of antibody cultures exerted a dose-dependent inhibitory effect on subsequent PFC responses to TNP-KLH, and removal of the fatty acid after 20 h did not reverse its inhibitory effect. Preincubation of isolated T -cells with TNP-KLH and 18:0 resulted in a similarinh1Dition of subsequent PFC responses, but a preincubation of isolated B-cells had no effect. The addition of 18:0 to the culture system in vitro led to a marked reduction in the level ofIL-2 detectable in culture supernatants, and PFC responses could be restored by providing exogenous mouse recombinant IL-2. The addition of antigen-primed T -helper cells to antibody cultures partially abrogated the inhibition by 150 microM 18:0, apparently due to their greater production ofIL-2. Lastly, following overnigh tincubation of unfractionated splenic lymphocytes in the presence ofTNP-KLH and [1-14C]- 18:0, B-cells were shown to contain nearly 5-fold more radiolabeled oleic acid (18:1) than T-cells. Collectively, these findings implicate T-helper cells as the priinciple target of 18:0-inhibition of primary antibody response in vitro, possobly as a result of the inability of T-helper cells to avoid an over accumulation of searic acid in their membrane phospholipids.

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Inhibition of lymphocyte proliferation by free fatty acids. ll. Toxicity of stearic acid towards phytohaemagglutinin-activated T cells. -- Buttke TM, Cuchens MA.

Studies were performed to further characterize the effects of saturated fatty acids on murine T lymphocyte proliferation. Flow cytometry was used to show that their inhibitory effects of stearic acid (18:0) on [3H] thymidine uptake can be correlated with changes in cellular DNA content. Additional studies using flow cytometry and fluorescein diacetate as a viability stain showed that exogenous 18:0 was toxic for phytohemagglutinin (PHA)-stimulated T cells, whereas the viability of unstimulated T cells was less affected by 18:0. The inhibitory effects of 18:0 on T cell proliferation were evident as early as 4 hr after fatty acid addition and after a 10-hr exposure, the effects of 18:0 could not be reversed by washing the cells or by adding oleic acid (18:1). It is proposed that the inhibitory effects of 18:0 are dependent upon PHA- induced changes in T cell lipid metabolism.

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