Tin Compounds Inhibit the Plasma Cell Response to Metallic Tin

Transfer of Inhibition by Parabiosis

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ABSTRACT

Injection of metallic tin powder causes intense proliferation of plasma cells in draining lymph nodes of Lewis rats. Pretreatment orally with soluble tin salts prevents this response to subsequently injected metallic tin. In the present work, pretreatment with tin salts by parenteral injection was just as effective as addition to the drinking water. This new approach made the following experiments possible. Poorly soluble tin compounds were found to be inhibitory when injected parenterally. Tin salts injected parenterally into one of two rats joined in parabiotic union prevented the plasma cell response to metallic tin in both parabionts. The transfer of the inhibitory effect via the cross-circulating blood represents significant progress toward understanding the mechanisms involved. The evidence suggests the possibility that tin salts elicit an intermediary substance or process that is responsible for inhibition of the plasma cell response to metallic tin.

Index Entries: Lymph nodes; parabiosis; plasma cells; stannous chloride; tin.

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INTRODUCTION

Injection of metallic tin powder causes a striking proliferation of plasma cells in the draining lymph nodes of Lewis rats (1,2). Pretreatment with stannic sulfate or stannous chloride in the drinking water prevents the response to metallic tin, although sodium, zinc, silver, and copper salts are ineffective (3). These observations might be interpreted as an immune response to metallic tin and as the production of immunologic tolerance by ionic forms of tin. However, there is no previous evidence that tin can be antigenic or tolerogenic, even though it is known that tin can interact with proteins (4). Therefore, we have continued to study these interesting relations between metallic tin and tin compounds in the production and prevention of plasmacellular lymphadenopathy.

METHODS

Metallic tin powder, type TF-1 from Amax Corp. (Greenwich, CT) or type 115 from Alcan Corp. (Elizabeth, NJ), was stirred in 3% hydrogen peroxide for 1 or 2 h, centrifuged, washed in saline, and suspended in saline at 200 mg/mL. Lewis rats of either sex, 2–3 mo old, were maintained in hanging wiremesh cages on Purina Rodent Chow 5001 and tap water. They were fasted overnight and then injected intraperitoneally (ip) with 1 mL of the tin suspension. Necropsies were performed 2 wk later to evaluate the effects of the tin powder on the lymph nodes that drain the peritoneal cavity. These mediastinal lymph nodes were weighed fresh, then fixed in Bouin’s fluid and embedded in paraffin. Sections were cut and stained with hematoxylin and eosin. The slides were arranged randomly and read without knowledge of the treatment. Plasma cell hyperplasia was scored 4+ when sheets of plasma cells and plasmablasts occupied at least one-third of the lymph node sections. Nodes with the usual content of plasma cells in medullary cords were scored zero. Intermediate degrees of hyperplasia were scored 1+, 2+, or 3+. The abdominal nodes, which were only slightly affected, and the normal nondraining nodes were not studied.

For prevention of plasmacellular lymphadenopathy, stannous chloride (SnCl₂·2H₂O) was prepared in two ways, usually by dissolving in a small volume of sterile water, centrifuging out insoluble hydroxychlorides, and diluting with HCl and sodium citrate to avoid precipitation (3). This preparation had a final concentration of 0.0075M by atomic absorption spectrophotometry. Alternatively, stannous chloride was suspended and partly dissolved in water at 10 mg/mL, and used as a cloudy suspension or separated by centrifugation into soluble and insoluble (probably hydroxychloride) components before injection. Other relatively insoluble tin compounds were suspended in water. The various tin compounds were injected subcutaneously (sc) in the flanks or in the hindpaws (the latter with the aid of ether anesthesia). In addition, the