In Loeb's colloid studies it was a transfer from NaCl to more dilute NaCl or water which produced a marked increase in swelling. In the case of the viability of the bacteria it is a transfer from $CaCl_2$ to a very dilute solution which causes a profound effect. Whether the death of the bacteria in the latter case is due to the fact that compounds are formed in the cell wall or protoplasm which hinder the elimination of toxic waste products of cell metabolism or whether such compounds favor the ingress of water which causes deadly hydrolyses (which Phelps has suggested as the lethal factor in similar cases), or whether such compounds favor the loss of necessary constituents of the cell to the water outside, we are not prepared to say. Experiments are now being conducted to throw light upon this point. It seems clear in any case from the conditions of the experiment that the effect is not due to any direct toxicity of the salt but to some change which it produces in the rate of exchange between the inside of the cell and its environment.

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Experimental tri-nitro-toluene poisoning.

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In an attempt to produce experimentally in dogs a state of poisoning by tri-nitro-toluene analogous to the condition recently observed among munition workers in England and America, the following methods of administering the poison have been employed: (1) Feeding by mouth (TNT in butter); (2) skin inunction (TNT in lard); (3) subcutaneous injections (TNT in olive oil); (4) intravenous injections (TNT in acetone); (5) intraperitoneal injections (TNT in albolene). Only the first three methods have proven satisfactory. Intravenous injection of any considerable quantity of an acetone solution causes immediate death, probably from a precipitation of the TNT in the blood stream, and consequent pulmonary embolism. The toxic action of the acetone, too, may be a factor. Negative results with intraperitoneal injections of an albolene solution were probably due to faulty absorption.

Feeding, inunction, and subcutaneous injections have resulted regularly in a more or less chronic state of poisoning ending in death. The rapidity with which toxic symptoms appear and the duration of life depend apparently on both the quantity of poison given and the frequency and method of administration. Skin inunction has so far given most constant results.

Symptoms observed: (1) Vomiting; seen only in feeding cases; apparently due to direct irritation of the stomach by the poison; (2) diarrhea; frequently present, its occurrence is not related to any particular method of administration; (3) depression, surliness, weakness, and emaciation, very marked in later stages; (4) leucocytosis. In one case a slight relative increase in lymphocytes, polychromasia, and nucleated R. B. C. (megaloblasts) was noted.

Autopsy findings: Heart, lungs, and gastro-intestinal tract negative. Liver showed grossly either no change or the picture of a moderate chronic passive congestion with lobules dark red in center and pale in periphery. Isolated yellowish, opaque nodules were sometimes seen scattered throughout the organ. Microscopically, a moderate central degeneration of the liver cells with congestion of the capillaries about the efferent veins was found. Fatty changes were demonstrable here and there, but the lesion was not uniformly distributed. In one case there were nodules of liver cells comprising several lobules, showing extreme fat accumulation. In the bone marrow, lymph nodes, and spleen there was an increased amount of blood pigment lying free in the tissues and in large phagocytic cells.

The outstanding feature of the autopsy findings is the absence of lesions which would explain the death of the animal. The changes in the liver, while definite and perhaps significant, are not comparable to those found in cases of toxic jaundice in human beings, where the destruction of liver tissue is as extreme as in acute yellow atrophy.