# LIPOPEROXIDE IN DOG THORACIC DUCT LYMPH

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## ABSTRACT

Lipoperoxide levels were examined in thoracic duct lymph (TDL) of 11 dogs. In four dogs with sodium citrate added in vitro to prevent coagulation, TDL had notably higher levels than in serum. After centrifugation, however, lymph supernatant levels of lipoperoxide closely approximated that in serum suggesting that the bulk of lipoperoxide remained in the sediment and derived from circulating cells. Interstitial accumulation of lipoperoxide, a breakdown product of cell membranes, may be a potent "toxic factor" responsible for trophic changes associated with chronic lymphedema.

Besides prostaglandins and thromboxane, a major metabolite of arachidonic acid metabolism is lipoperoxide which has been implicated in harmful processes such as bullae formation and aging of the skin (pigmentation and wrinkling), as well as hemolysis, atherosclerosis, and carcinogenesis. Moreover, lipoperoxide levels in the dermis of patients with chronic lymphedema are higher than in serum (1). To pursue the potential importance of lipoperoxide as a causative factor in trophic changes associated with peripheral lymphedema, lipoperoxide levels were now examined in canine thoracic duct lymph (TDL).

#### MATERIALS AND METHODS

Under general anesthesia (sodium pentobarbital 30mg/Kg BW), lymph was

obtained from the cervical thoracic duct in 11 dogs and the specimens divided into three groups and quantitatively analyzed for lipoperoxide using a fluorometric assay (1). Group I consisted of three dogs in which no anticoagulant was added and the lymph (1ml) was simply allowed to "clot." Group II consisted of four dogs in which 0.1ml of sodium citrate (3.8%) was added to 0.9ml of thoracic duct lymph (TDL). Group III consisted of four dogs in which sodium citrate was added as in Group II but thereafter lymph was centrifuged for 10 minutes at 1500rpm and the supernatant extracted and reassayed for lipoperoxide. Simultaneous with taking of TDL lymph samples, blood was obtained from the abdominal aorta, and the serum also assayed for lipoperoxide content (2).

### RESULTS

As shown in Fig. 1, thoracic duct lymph with anticoagulant added contained a much higher concentration of lipoperoxide than blood serum, but that after centrifugation lymph supernatant content now approximated serum levels. In other words, the bulk of lipoperoxide after centrifugation was confined to the lymph sediment rich in lymphocytes and red blood cells. Some microfilaria were also seen in smears of the sediment.

## **COMMENT**

Lipoperoxide derives from cell membranes rich in arachidonic acid which pro-

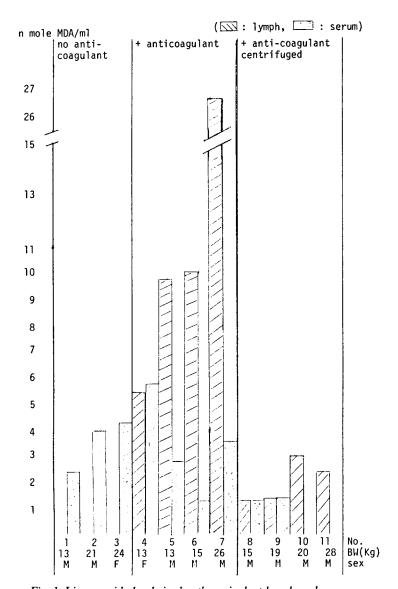


Fig. 1. Lipoperoxide levels in dog thoracic duct lymph and serum.

bably accounts for the high levels in thoracic duct lymph emanating from circulating cells. Indeed, its "disappearance" from the supernatant after centrifugation lends support to an origin from cells which are now localized in the sediment. (It should be noted that filarial infection in dogs in Japan is exceedingly common and indeed microfilaria were seen in the centrifuged sediment. Accordingly, it is possible but not likely that this parasitic infestation contributed to the high lipo-

peroxide levels in TDL.)

In an earlier study, Okada, et al (3) found that lipoperoxide content in heart muscle increased after experimental lymphatic obstruction. Because of its known "toxic" effects on skin and subcutaneous tissue, the deposition and accumulation of lipoperoxide in soft tissues with long-standing lymphatic obstruction may contribute to some of the deleterious trophic changes in the extremities associated with primary and secondary lymphedema.

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