against which they are being employed, both in the air (*6, 11*) and *in vitro* (*24*). Finally, direct evidence of the lethal action of glycol vapor was obtained by testing the viability of microorganisms isolated by sedimentation from propylene glycol saturated atmospheres, and in no instances were any viable bacteria found (*4*).

Since it has been found that the rate of bactericidal action of glycol vapors depends directly upon the concentration of glycol in the air (Puck [*10*]), we could infer that the observed lack of lethal effect of propylene and triethylene glycol vapor for *E. coli* reported by Nagy and Mouromseff was due to insufficient glycol in their experiments. They report no determinations of glycol in the air. As Robertson (*23*), Lester *et al.* (*25*), and Bourdillon (*13*) have shown, considerable quantities of glycol vapor are lost by condensation on walls and other surfaces of the room, so that evaporation of a quantity of glycol calculated to produce saturation of a given space may result in only an insignificant fraction of such concentration. Furthermore, as was shown by Puck and Chaney (*26*), the presence of a visible fog is never in itself sufficient to prove the existence of a saturated atmosphere of glycol vapor. Hence no valid conclusions about the effectiveness of the glycols or any other bactericidal vapors can be drawn without knowledge of the concentration employed. In our experiments, conducted under optimum conditions of temperature and humidity, and employing atmospheres known to be saturated with propylene glycol, the killing of airborne *E. coli* was essentially instantaneous. Triethylene glycol acted more slowly.

Triethylene glycol vapor under similar conditions was found to be highly bactericidal against airborne *E. coli*. However, the rate of kill was demonstrably less than that observed for propylene glycol. This difference conforms to the results of *in vitro* studies (*24*) which demonstrated that, of the two agents, propylene glycol is the more rapidly acting germicide.

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Comments and Communications

On Leukotaxine

As pointed out in numerous publications, inflammation is a manifestation of severe cellular injury in vertebrates (*1*). It has also been pointed out that any irritant injures the cell, and injury alters the biochemistry of the cell, with consequent release of various common denominators (*1*, p. 135, footnote). These common denominators include leukotaxine, the leukocytosis-promoting factor (thermolabile and thermostable), necrosin, pyrexin, and leukopenic factor, leukopenin, and the more recently described exudin (*2*). These factors, or common denominators, are responsible for the fundamental, stereotyped reaction of inflammation. It is incorrect to state that one of them alone, and not all of them or some of them, initiate acute inflammation, as Moon asserts that the writer claims (*3*).

It has often been pointed out that the cells, when injured, liberate leukotaxine, and only when injured do they form this substance. Even a diagram of this process appears in a recent monograph (*1*, p. 123). It would be difficult, therefore, to envisage that normal tissue cells as such would contain leukotaxine. In recent studies Moon has questioned the hypothesis on the ground that leukotaxine has not been shown to exist in normal tissue (*3*). One would not expect to find it in perfectly normal cells. Moon presents absolutely no evidence supporting the view that leukotaxine does not exist in normal tissue (*3*). His own studies indicate that a chemotactic substance is possibly present in damaged tissue; and this substance does not necessarily come from exudate where leukotaxine had originally been recovered. Moon obtained a quantity of muscle from a freshly killed rabbit and ground the material in a meat chopper. A saline extract was then made. Similar extracts were made from kidney, liver, skin, and lung. These extracts were then injected intradermally into the ears of rabbits, and

the lesions were studied (*3*). As pointed out in earlier studies, macerating tissue as Moon has done is the best way to injure cells and thus liberate leukotaxine. This has been shown by the writer to occur with macerated testicular extract, from which a permeability factor has been obtained (*4*). Rigdon has shown the presence of a similar permeability factor, which is one of the properties of leukotaxine, with saline tissue extract of skin, muscle, and testicle (*5*). The writer has recently shown that any cells from invertebrates and also from dogs, when crushed, will yield necrosin, as obtained in exudate (*6*). One must recall that an exudate represents the products of cell injury admixed with elements from the blood. The severely injured cells, however, as obtained from extracts by crushing or mashing previously normal cells, yield the same factors as obtained from exudate.

Lewis termed the substance from injured tissue the "H-substance," presumably because it was either histamine or was closely allied to it (*7*). Leukotaxine has no common property with histamine either biologically or chemically (*8*). This fact accounts for the term leukotaxine. No one knows where in the cell, or in which type of injured cell, leukotaxine is formed. In spite of Moon's statement, the writer has never stated that leukotaxine is derived from leukocytes (*9*). Therefore, the term proposed by Moon of "cytotaxine" is unwarranted by the known facts (*3*). Finally, as pointed out in a recent discussion (*9*), instead of being reluctant to accept the term leukotaxine, it would greatly facilitate the discussion if Moon would chemically prove whether leukotaxine is present in the experimental types of inflammation he has studied. He states: "It is our impression that the tissue substance which produced the effects maintained is not a specific chemical entity, although this belief is not based on chemical evidence" (*3*). It is the writer's firm belief that Moon's studies merely indicate that he has substantiated earlier workers who postulated the presence of a substance which is liberated by damaged cells and which is in turn chemotactic. The concrete chemical studies of the writer have demonstrated the presence of such a chemical substance in the product of cell injury, and this substance he has termed leukotaxine (*10*). The presence of leukotaxine has been confirmed separately by Duthie and Chain (*11*), Cullumbine (*12*), Minami and Inugami (*13*), Pasquali (*14*), and by Yeshuri (*15*).

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I APPRECIATE the opportunity to comment on Dr. Menkin's communication. It is not unusual for workers to disagree in their evidence and interpretations. Often this is desirable, for it emphasizes the fact that the problem is not yet solved, and that further evidence should be sought and published. Failure to agree should not arouse controversial antagonism. If the questioned interpretation is correct, it will be reinforced by further investigation; if incorrect, then it is fortunate that the question was raised. I am content to allow our colleagues to judge the merits of both viewpoints.

Many of our critic's assertions are not factual, and most of his objections are invalid. A few instances will be cited.

Our statement regarding the origin of leukotaxine is misquoted. We said (*1*): "He [Menkin] subjected inflammatory exudates obtained from suppurative pleuritis to chemical cleavage. By fractionation and purification, he obtained a crystalline substance which he termed leukotaxine."

Without substantial evidence, Menkin asserts that leukotaxine is present in all normal tissues, that it is liberated from them when injured, and that maceration is the best way to do this. Therefore, he reasons that the extracts used in our experiments owe their effects to leukotaxine. His position would be fortified by isolating leukotaxine from muscle, skin, lung, liver, kidney, and others. Why has he not done this? If these tissues present an easily available source for leukotaxine, why was it necessary laboriously to produce suppurative exudates as a source of material for his studies?

Dr. Menkin protests against our allusion to leukotaxine as the one factor claimed by him to be responsible for inflammation. Yet in the recapitulation of his book *The Dynamics of Inflammation* (pp. 204–206), he attributes the major phenomena of inflammation, including capillary permeability and migration of leukocytes, to leukotaxine—"*which appears* [italics mine] as if it *may* belong to the group of relatively simple polypeptides." No other factor is mentioned here.

Subsequently, he postulated sundry additional factors: necrosin, pyrexin, leukopenin, exudatin, and two leukocytosis-promoting factors. Numerous substances of animal origin will cause fever, necrosis, and leukopenia. One wonders by what criteria these can be distinguished from Menkin's pyrexin, necrosin, and leukopenin. Many pathologists have found this multiplicity of hypothetical factors to be confusing rather than clarifying. The chemical structure is undetermined, yet our experiments are criticized for

lack of chemical identifications. We should be told the chemical nature of these substances and criteria for distinguishing them before identification is required.

Rigdon (*2*) merely concluded: "A substance has been obtained from the skin, muscle and testicles of normal rabbits which produces a local increase in capillary permeability." By what logic does Dr. Menkin assume that this substance is leukotaxine? With equal logic he might assume that histamine—which increases capillary permeability—is leukotaxine.

The statement "Leukotaxine has no common property with histamine, either chemically or biologically" is not factual. Histamine causes both dilatation and permeability of capillaries. Menkin claims that leukotaxine has these same properties.

Duthie and Chain (*3*) found in extracts of liver, kidney, and muscle substances that increased capillary permeability. They devised a method whereby a polypeptide was obtained from fibrin by peptic digestion. This product was strongly chemotactic; it caused increased capillary permeability and leukocytic infiltration in the skin of animals. A polypeptide having similar properties was obtained from Witte's peptone and from many proteins. These workers were convinced that they were dealing with a general physiologic action of protein breakdown products. They were unable to establish a chemical relationship between these products and leukotaxine, and doubted that the claim that leukotaxine is a polypeptide has been established. Yet Menkin cited these authors, Rigdon, and others as having corroborated his findings.

The most valid criticism leveled at our report is that it substantiates the conclusions of earlier workers. This, we humbly admit, *it does.*

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