DEMONSTRATIONS

The polymorph transfer reaction: a model system for the study of acute inflammation

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An important feature of acute inflammation is an influx of polymorphonuclear leucocytes (PMN-leucocytes). Animal models of acute inflammation (excluding anaphylactic reactions) generally employ local injections of agents such as carrageenin, kaolin or turpentine. In other experiments, immunological responses such as the Arthus reaction, are utilized. These reactions consist of a complex series of events involving mast cells, platelets, complement activation, and leucocyte chemotaxis, etc. As an alternative approach, we have attempted to produce a simplified model system to analyse some aspects of the pharmacology of PMN-leucocytes *in vivo*. The objective of this work was to try to correlate the production of prostaglandins by PMN-leucocytes *in vitro* (Higgs & Youlten, 1972), with the finding that exogenous prostaglandins can potentiate inflammatory exudation *in vivo* (Williams & Morley, 1973).

New Zealand White rabbit PMN-leucocytes were prepared from glycogen-induced peritoneal exudate (Hirsch & Church, 1960), and injected intradermally into up to 36 sites on the back of a recipient rabbit (5×10^6 PMN-leucocytes in 0.1 ml Hanks solution/site). Control sites received Hanks solution alone. After a fixed interval (60 min in the present experiments), the rabbit was given intravenous ^{131}I-labelled albumin, and each skin site received a supra-injection of bradykinin mixed with ^{133}Xe, or ^{133}Xe in saline alone. Exudation and increased blood flow were measured over the next 30 min as previously described (Williams, 1976a).

Using this technique (which we have termed the 'polymorph transfer reaction'), we have observed clear potentiation of bradykinin-induced exudation in sites injected with PMN-leucocytes. No potentiation of bradykinin responses was observed in sites injected with cells mixed with indomethacin (1 μg/site). The concomitant changes in blood flow were consistent with the results of experiments using exogenous prostaglandins as previously reported (Williams, 1976b).

These results suggest that responses to exogenous bradykinin can be potentiated by endogenous prostaglandin.

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Table 1

Treatments (doses/0.1 ml) (n=6)		*Plasma Exudation ± s.e. mean (μl total–14.2 μl control)*	*Increased Blood flow ± s.e. mean (% control)*
1st injection	*2nd injection*		
PMNs (5×10^6)	Saline	10.3 ± 2.3	35.6 ± 8.4
Hanks	Bradykinin (0.5 μg)	63.5 ± 10.3	38.3 ± 11.0
PMNs (5×10^6)	Bradykinin (0.5 μg)	123.1 ± 15.9	76.4 ± 9.0
PMNs = indomethacin (1 μg)	Bradykinin (0.5 μg)	64.9 ± 12.4	25.1 ± 10.8
Hanks (control)	Saline	0.0 ± 1.9	0.0 ± 8.5

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