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LETTERS TO THE EDITOR

Increased Subcutaneous Adipose Tissue Blood Flow in UVB-inflamed Human Skin

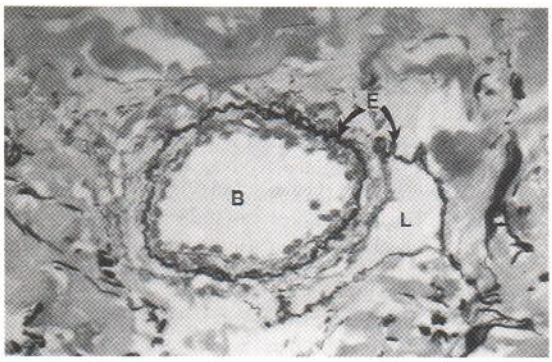

Fig. 1.

Sir,

The authors of the paper "Increased subcutaneous adipose tissue blood flow in UVB-inflamed human skin" (1) pose interesting questions but they also make certain assumptions, which may be incorrect. The release of mediators of inflammation in the upper dermis is indeed a consequence of UVB injury. However, it is not true that the only way such mediators can spread to subcutaneous tissue is by diffusion. If one inoculates an agent, such as adrenalin, into the upper dermis, one finds that it quickly travels through the lymphatics into the subcutaneous tissues and along lymphatic trunks. One can trace the effect of these agents on subcutaneous blood flow by the white line of vasoconstriction which appears in the upper dermis overlying the lymphatic, extending many centimetres proximally. The adrenalin can diffuse out of lymphatic trunks and affect adjacent arteries and one may assume that vasodilators could do the same. The close anatomical relationship of lymphatic (L) with blood vessel (B) is illustrated in Fig. 1, taken from the deep dermis (2).

The second assumption which may be incorrect is that the only significant absorption of Xenon is in subcutaneous adipose tissue. There is, however, around each hair follicle and around each sweat gland a quite substantial amount of dermal adipose tissue (Fig. 2). Is it known what contribution this dermal adipose tissue makes to the clearance of Xenon?

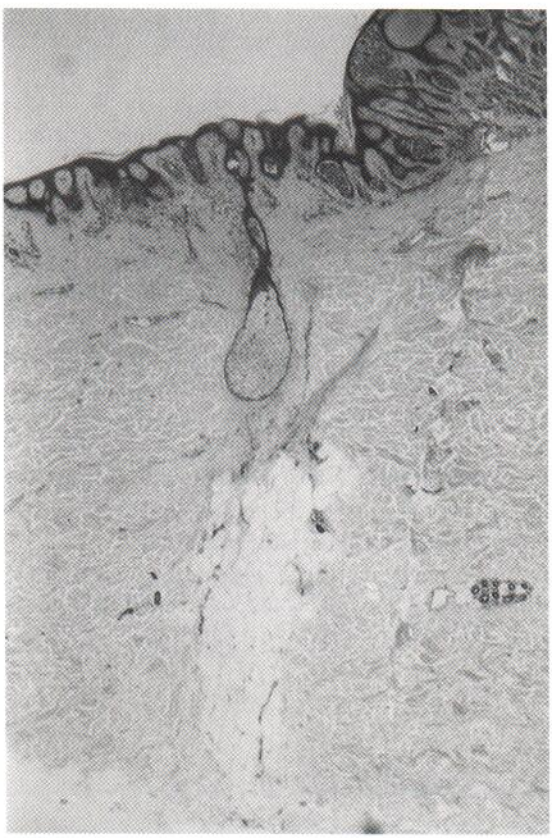

Fig. 2.

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Response to Dr Ryan's Letter

We are pleased to get this opportunity to reply to the comments of Dr Ryan. The following questions are raised: 1) Do inflammatory mediators influence subcutaneous adipose tissue blood flow (SBF) via lymphatic drainage from the skin to the adipose tissue? An example concerning adrenaline is presented. 2) Do fatty tissue around hair follicles influence 133 xenon washout?

1) The blanching observed along the lymphatic vessels does not necessarily indicate decreased dermal blood flow and is certainly not evidence for decreased SBF. In several situations, pale skin do not reflect diminished skin blood flow, e.g. in corticosteroid-treated skin and in the Woronoff ring (1,2). Communications between the blood and lymphatic system have previously been discussed by Dr Ryan (3). Drainage from venous vessels to the lymphatics has been described, but the reverse flow direction has not been reported. Additionally, the lymphatic vessels transport fluid proximally, not vertically into the depth of the tissues. Consequently, it is most likely that any vasoactive metabolite contained in the lymph will have no influence on SBF.

2) Experiment using autoradiography on frozen tissue and dissections of cutaneous tissue from cutaneous tissue, in the frozen state, suggested that following one hour after labelling the 133 xenon was cleared from the skin (4). Secondly, monoexponential washout from selected cutaneous tissue arrangements, implied washout from homogenously perfused tissue compartments (1,4). Consequently, a "sink" caused by fatty tissue around hair-follicles constitutes no pitfall.

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