that it was offered, with much evidence, as a cause of schizophrenia.[23] During attempts to remove some of the less accessible foci, many patients died. At least the theory of autoimmunity is unlikely to lead to such therapeutic disasters.

Progress with Air Pollution

Since the winter of 1962 the National Survey on Air Pollution has been collecting data throughout the year on the concentration of smoke and sulphur dioxide in a representative sample of British towns. The data are processed by computer and are available to all interested workers through the Warren Spring Laboratory, Stevenage. In its 32nd Report,[1] covering a period of eight years since the establishment of the first smoke control areas, the Warren Spring Laboratory discusses the trends in the pattern of pollution as a consequence of the Clean Air Act of 1956[2] and of the changing fuel requirements of modern technology.

There has been a decrease in the consumption of coal with an increase in that of fuel oil, most of which has a high sulphur content. As a result the emission of smoke into the atmosphere has fallen while that of sulphur dioxide has risen. The decrease in smoke concentration in the ambient atmosphere was to be expected, but there has also been a welcome decrease in the sulphur dioxide concentration over the country as a whole, with a smaller decrease in London. This has been attributed to the care which is now being given to the location of new industrial sites and such factors as the height and design of industrial chimneys.[3] The level of pollution depends on the amount of material emitted and on the weather conditions determining how much pollution is blown away, so that the weather is a major factor in setting the pattern of pollution from year to year. Since the Warren Spring studies show considerable fluctuations from one year to the next any trend so far must be interpreted with caution.

While the Clean Air Act is helping to clear the air of smoke, the removal of sulphur dioxide from it presents a greater problem. The report points out the high cost of the process for removing sulphur dioxide at the Battersea and Bankside power stations and the lack of success in developing a more convenient system. At Battersea 20–30 tons of Thames water are used for every ton of coal burnt, and extension of the process is limited by the capacity of the river to take the effluent. The most practicable way at present of limiting pollution from sulphur dioxide is by sending it aloft by means of suitable chimneys and with a high speed of emission, so that the gas is diluted before coming down again.

Studies at Stevenage and Hornsea on the distribution and dispersion of pollution in streets carrying heavy motor traffic have shown that though pollution can be high it is also extremely local in character. The black smoke from diesel engines arouses public indignation, but petrol engines produce more carbon monoxide and higher concentrations of unburnt hydrocarbons, which are more likely to be injurious to health.[4] Devices for removing smoke from diesel exhausts are uneconomical. The solution lies in the correct maintenance and operation of the vehicle, and this should be controllable by adequate legislation. However, according to a report[6] issued by the National Society for Clean Air last week, if all manufacturers set their engines to the level permitted by a forthcoming British Standard, smoke on the road would increase. In the U.S.A. stringent regulations will apply to all 1968 petrol engines, limiting the discharge of carbon monoxide and hydrocarbons. Britain is free of the irritant photochemical smog produced by the action of strong sunlight on unburnt hydrocarbons which is so characteristic of Los Angeles,[7] but the dangerous effects of a build-up of carbon monoxide are equally applicable here, and this problem has not yet been given sufficient attention.

That air pollution worsens the plight of a patient with chronic lung disease is undoubted, and it is likely to contribute to the production of chronic bronchitis. But there is no proof yet that either smoke or sulphur dioxide in the concentrations encountered in polluted air are the substances causing the harm, though they may be. However, since the concentrations of other harmful substances are likely to vary in the same way as those of smoke and sulphur dioxide, these two are likely to remain at least useful indices of air pollution.

[1] *The Investigation of Atmospheric Pollution 1958–1966*, 32nd report, 1967. H.M.S.O. London.
[2] *Clean Air Act*, 1956, Chapter 52, 1957. H.M.S.O. London.
[3] *Memorandum on Chimney Heights*. 1963. H.M.S.O. London.
[4] Reed, L. E., *Roy. Soc. Hlth. J.*, 1966, **86**, 227.
[5] Lawther, P. J., ibid.
[6] *Air Pollution from Road Vehicles*, National Society for Clean Air, 1967. London.
[7] Hamming, W. J., MacBeth, W. G., and Chass, R. L., *Arch. environm. Hlth.*, 1967, **14**, 137.

S.T.P.

By the standards of " pop " culture S.T.P. is an elderly drug ; it has been publicly discussed for nearly two months. On 16 June *International Times*, the fortnightly newspaper of the hippie movement, predicted its arrival in Great Britain,[1] by mid-July a tablet had been passed to the Home Office for analysis,[2] and by the end of July *International Times* had reported that the drug was in wider use in Britain[3] and warned that it could be dangerous.

The American Food and Drug Administration has identified S.T.P. as methyl dimethoxy methyl amphetamine,[4] a substance chemically related to both amphetamine and mescaline, but a good deal of doubt remains. *Time*,[5] which gives the credit for the distribution if not the synthesis of this latest hallucinogen to a San Francisco chemist, reports that the drug may be 5-methoxy-NN-dimethyltryptamine. This substance is related to the known hallucinogens bufotenine, dimethyltryptamine, and psilocybin, and also to a principle in reed canary grass which causes staggers in sheep.[6] It has also been suggested that S.T.P. is related to a group of hallucinogens, the piperidyl glycolates, studied in the 1950s by L. G. Abood and his collaborators[7] or to a chemical warfare agent called BZ.[5] These suggestions are based on the cholinergic effects of S.T.P. Alternatively, S.T.P. may well be related to a group of benzilic acid derivatives, of which the tranquillizer benactyzine, the hallucinatory anti-Parkinsonian compound benzhexol, the arousing drug pipradol, and the enzyme potentiator S.K.F. 525 are other examples. S.K.F. 525 proved to be so powerful and so non-specific a substance that it was useless for therapy: the reported dangerous potentiation of S.T.P. by the usual

[1] *International Times*, 16 June, 1967.
[2] *The Times*, 8 August, 1967.
[3] *International Times*, 28 July, 1967.
[4] *F.D.A. Press Release Nos.* 67-78, 11 July, 1967.
[5] *Time*, International Edition, 7 July, 1967.
[6] Wilkinson, S., *J. chem. Soc.*, 1958, p. 2079.
[7] Abood, L. G., Ostfeld, A., and Biel, J. H., *Arch. int. Pharmacodyn.*, 1959, **120**, 186.

antagonists of lysergic acid diethylamide (L.S.D.)[2 3] suggest that it may be just such another substance.

Not surprisingly, no controlled experiments have yet been done with S.T.P., but there seems little doubt that it is an active, dangerous, and powerful substance. A single dose (however much that may be) is said to give a 72-hour "trip,"[5] and the risk of suicide or psychotic breakdown seems likely to be correspondingly greater than with L.S.D.

Whatever its identity, the emergence of S.T.P. has raised several questions of interest. Whose is the responsibility to collect and sift information on drugs introduced in this way, to promote research, and to advise society on their control and appropriate uses? Perhaps the L.S.D. and Cannabis Subcommittee of the Home Office's Standing Advisory Committee on Drug Dependence should extend its terms of reference to include drugs of this kind as they are developed.

L.S.D. was not the first hallucinogen: S.T.P. is certainly not the last. New compounds may be developed with totally new actions for which the appropriate scientific descriptive language does not yet exist—a fact that may itself contribute to anxiety about such drugs. The definitive chemical identification of S.T.P. has probably been delayed as a result of deliberate obfuscation by those responsible for its synthesis and distribution—several different substances were apparently sold or given to the Federal Drugs Administration as S.T.P. by its informants.[4]

The iconoclasm of hippiedom and the ill-considered advocacy of people who should know better, including, unfortunately, a few members of the medical profession, are acting together to create an atmosphere in which firm action to control the spread of drug-taking is unpopular. Nevertheless, such action is needed. The statutory measures for the control of drugs need to be made more flexible, so that as soon as compounds such as S.T.P. enter circulation their unauthorized possession and distribution can be made an offence. Society has a responsibility to protect its members from drugs that can make them frankly insane or indeed kill them, as it has to stop the spread of communicable diseases.

Healing Processes in Wounds

Reactions to injury tend to be studied intensively in time of war and to be neglected in the subsequent peace. But fortunately for medicine the healing of wounds presents enough basic problems of deep interest to biologists to ensure that it remains a field of active research.[1] Though clinicians demand practical information about factors influencing healing, facts can come only from careful experimental work, and such experiments have already exposed some widely held fallacies.

The healing of a wound starts as an inflammatory reaction with infiltration by polymorphs and mononuclear phagocytes, and removal of debris by these cells. The reaction seems to be of importance to the subsequent healing. The ingrowth of new blood vessels, lymphatics, and nerves by budding from existing structures is relatively well established[2] and has fairly recently been confirmed by electron-microscopy.[3] A practical point is that new blood vessels are abnormally permeable to particulate matter,[4] including bacteria, and this may possibly facilitate infection of the wound from the blood stream.

The commonly held view of the origin of fibroblasts is that they migrate from the borders of the wound after division of existing fibroblasts. Alternatively it has been suggested that fibroblasts may be derived from haematogenous mononuclear cells.[5] However, all spindle-shaped cells in granulation tissue are not fibroblasts. The fibroblast can be identified with certainty only by electron-microscopy. It is then seen to have a large nucleus and nucleolus, extensive rough-surfaced endoplasmic reticulum, dense cytoplasmic bodies, peripheral cytoplasmic vesicles, dispersed Golgi network, numerous large mitochondria, and aggregates of fine filaments at the periphery of the cell.[6] The possibility that blood cells turn into fibroblasts may perhaps be proved only by use of blood cells labelled with a stable marker in conjunction with electron-microscopy.

Experimental studies show that collagen is synthesized by hydroxylation of proline and then biosynthesis of the protein in the endoplasmic reticulum of the fibroblasts. From here the protein passes into the extracellular space, where it acquires the characteristic banding (700 Å) of collagen. The mitochondria are not significantly involved in this process but the Golgi complex and peripheral vesicles are, especially if the collagen is destined to enter into a complex with polysaccharide. The clinical relevance of these observations is apparent from study of scorbutic animals, vitamin-C deficiency being one cause of defective healing of wounds. In scurvy wounds are normal except for the absence of collagen fibrils. Electron-microscopy shows the fibroblasts have an abnormal endoplasmic reticulum, with plenty of ribosomes but lack of regular orientation. Filamentous extracellular material (presumably fibrillar protein) is present, but this lacks the regular banding of collagen. It is not yet clear whether failure of wound healing in vitamin-C deficiency is due to changes in the properties or the structure of fibroblastic ribosomes and endoplastic reticulum or whether these are secondary to a failure to hydroxylate proline.[7]

In addition to the synthesis of collagen, contraction of the wound area is a vital part of healing. It is now realized that this phenomenon is not due to contraction of collagen fibres or to loss of extracellular or intracellular water. Nor is it due to ingrowth of epithelium from the wound margins. The contraction is in fact a complex and poorly understood process, yet it is of great importance in living tissues. It seems to be due to tensions within the granulation tissue filling the wound, orientated towards lines of stress, and associated with removal of cells and extracellular material, including collagen.[8-10] Likewise the cessation of wound contraction is also of clinical importance. M. Abercrombie[10] believes that it may occur when the advancing edges of the

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[3] Cliff, W. J., *Phil. Trans. B*, 1963, **246**, 305.
[4] Schoefl, G. I., *Ann. N.Y. Acad. Sci.*, 1964, **116**, 789.
[5] Gillman, T., and Wright, L. J., *Nature (Lond.)*, 1966, **209**, 263.
[6] Ross, R., and Benditt, E. P., *J. Cell. Biol.*, 1965, **27**, 83.
[7] —— in *Wound Healing*, ed. S. M. Levenson, J. M. Stein, and N. Grossblatt, 1966. Washington, D.C.
[8] Abercrombie, M., James, D. W., and Newcombe, J. F., *J. Anat. (Lond.)*, 1960, **94**, 170.
[9] Billingham, R. E., and Russell, P. S., *Ann. Surg.*, 1956, **144**, 961.
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