remarkably constant at 650° C., and rises about 50° C. during suction.[2] The term "tar" is often employed in discussing smoking as if this material were something different from and separable from "smoke." The truth of the matter is that the whole of the particulate matter in tobacco smoke can be condensed to a "tar" and that, because of the very small size of the initially formed droplets, it is of remarkably uniform composition.[3] Because of confusion in the use of the term "tar" it is recommended that the term "smoke condensate" be employed to replace it.

Many ideas have been proposed for reducing "harmfulness" and "injurious products" in tobacco smoke. Several hundred compounds are known to be present[4] and some of these are known to have definite biological effects.[5] Whatever proposals are made, it is evident from the nature of smoke that successful reduction of the effect of smoking can be achieved only by taking in less smoke. The use of pinholes in the cigarette is one method of doing this.

REFERENCES

1 Smyth, C. N., *Brit. med. J.*, 1959, **1**, 506.
2 Lindsey, A. J., ibid., 1954, **2**, 1352.
3 —— *Brit. J. Cancer*, in press.
4 Kennaway, E., and Lindsey, A. J., *Brit. med. Bull.*, 1958, **14**, 124.
5 Wynder, E. L., *The Biologic Effects of Tobacco*, 1955. Little, Brown and Company, Boston.

French Cigarettes and Lung Cancer

Q.—*French cigarettes do not cause yellow tobacco-stains on the hands. Is this due to absence of tarry substances and nicotine from French cigarettes? Is the incidence of lung cancer among French cigarette-smokers lower than among British smokers?*

A.—This question cannot be answered simply. The amount of staining of skin by smoke is affected by the type of smoke and by the habit of the smoker in handling his cigarette. The suggestion that there are no "tarry substances" and nicotine in the French brand of cigarettes is definitely wrong. The amounts may be greater or less than in other brands.

The incidence of lung cancer in France is lower than in this country in the ratio 140/461, but so also is the consumption of cigarettes in the ratio 585/1,378.[1] It has been demonstrated that the incidence of this disease is approximately proportional to the amount smoked, so French cigarettes smoked in France may be somewhat less carcinogenic than British cigarettes smoked in Britain, but many other factors are also likely to contribute to the difference. These may be related to other differences in smoking habits—for example, the amount of the cigarette smoked—or they may be entirely independent—e.g., atmospheric pollution and occupational exposure.

REFERENCE

1 Doll, R., Hill, A. B., Gray, P. G., and Parr, E. A., *Brit. med. J.*, 1959, **1**, 322.

Inheritance of Porphyria

Q.—*Three members of a family have porphyria. Which of many relatives should be warned against taking sulphonamides or barbiturates in order to prevent precipitating the complaint?*

A.—Acute intermittent porphyria is essentially due to a dominant gene,[1] or perhaps one of two dominant genes if, as seems probable, the South African form is distinct.[2] The chance of a relative being susceptible decreases as the relationship becomes more remote. Excluding fresh mutation, it is 1 in 2 for first degree relatives of the index cases (first degree relatives include children and brothers and sisters), 1 in 4 for second degree relatives (such as grandchildren and nephews and nieces), and 1 in 8 for third degree relatives. But a full family study would help to indicate the part of the family at risk.

It is claimed that after childhood all those susceptible to the South African variant of the illness may be detected by the presence of uroporphyrin in the faeces or in the urine. In the form reported from Sweden and commonly seen in this country,[3] many of those susceptible, but not all, may be detected by the presence of porphobilinogen in the urine. There is no doubt that barbiturates may precipitate attacks, and the sulphones have also been implicated, but this is not the case so far as I know for sulphonamides.

REFERENCES

1 Waldenström, J., *Amer. J. Med.*, 1957, **22**, 758.
2 Dean, C., and Barnes, H. D., *Brit. med. J.*, 1958, **1**, 298
3 Goldberg, A., *Quart. J. Med.*, 1959, **28** n.s., 183.

Congenital Osteogenesis Imperfecta

Q.—*A young, healthy woman was recently delivered of a full-term baby suffering from fragilitas ossium. There is no history of it in either her or her husband's family. What are the chances of this couple producing healthy offspring?*

A.—Osteogenesis imperfecta tarda is due to a dominant mutant gene, but the genetic status of severe congenital osteogenesis imperfecta is not fully known. Assuming that this case is the first clinical appearance of a dominant mutation, the chances of this couple producing healthy offspring are excellent. Only one consecutive series is available on which to base an empirical risk.[1] In this series 6 patients had 19 brothers and sisters, all of whom were unaffected.

REFERENCE

1 Seedorf, K. S., *Osteogenesis Imperfecta: a Study of the Clinical Features and Heredity Based on 55 Danish Families Comprising 180 Affected Members*, 1949. Copenhagen.

Total Absence of Hair

Q.—*Two paternal aunts and two paternal uncles and the paternal grandfather of an expectant mother were all born with no hair on the head, no eyebrows, and no eyelashes. What chance has her baby of being born with the same condition? Is it due to a recessive gene?*

A.—Total absence of hair, without other evidence of ectodermal abnormality, is a rare condition. But there are several reports of more than one member of a family affected.[1 2] There is probably more than one clinical type, and more than one mutant gene may be responsible. In some families the pattern of inheritance is that of a dominant gene and in others that of a recessive gene. In this family it is probable that a dominant gene is responsible. Assuming that the woman herself shows no signs of the condition the chance that her baby will be affected is not great.

REFERENCES

1 Cockayne, E. A., *Inherited Abnormalities of the Skin and its Appendages*. 1933. London.
2 Touraine, A., *L'Hérédité en Médecine*, 1955. Paris.

Correction.—In the paper on chronic bronchitis by Dr. J. McC. Murdoch and his colleagues (December 12, p. 1277) the word "inactive" should have appeared instead of "active" in the second line under Table VII (p. 1281).

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