chromium salts could induce tumours at sites of cell damage, where release of hydrolytic enzymes and subsequent formation of glycidal can occur.

MUTAGENICITY OF HAIR COLOURANTS IN BACTERIA: POSSIBLE LINK WITH CARCINOGENICITY. S. VENITT and C. T. BUSHELL, Pollards Wood Research Station, Bucks.

Preliminary results (C. E. Searle, unpublished data) obtained in mice suggest a possible carcinogenic hazard associated with certain hair colourants. Eleven such products were tested for mutagenicity in bacteria, since there is a reasonable correlation between carcinogenicity and mutagenicity. Six colourants were strongly mutagenic in *Salmonella typhimurium* TA 1538 (frameshift): of these, one required metabolic activation with liver supernatant (LS), 5 did not. Two of these were also mutagenic in another frameshift mutant, TA 1537. We obtained negative results in all strains which revert by base-pair substitution (*S. typhimurium* TA 1535 and *E. coli* WP2). 2-nitro-p-phenylenediamine (2NP) and 4-nitro-o-phenylenediamine (4NO), both dyes known to be present in one of the colourants tested, were mutagenic (without LS) in TA 1537 and TA 1538. A combination of TLC and spot testing showed that 5 of the 6 mutagenic colourants contained one or both of these dyes, together with other unidentified mutagenic components. It is suggested that products containing these dyes should be withdrawn from use pending further studies of their biological activity.

TESTS OF TWO HAIR COLOURANTS FOR CARCINOGENICITY BY REPEATED APPLICATION TO MOUSE SKIN. C. E. SEARLE and D. G. HARNDEN, Department of Cancer Studies, University of Birmingham Medical School and O.H.B. GYDE, Department of Haematology, East Birmingham Hospital.

Routine questioning of a patient with neutropenia revealed that she frequently used 2 "semi-permanent" hair colourants. When she subsequently developed myeloproliferative disease it was decided to initiate preliminary tests on these materials by topical application to mice since no information was available on their long-term effects.

One preparation contains 2-nitrophenylenediamine, the other an azo-dye metal derivative and an aminonitrophenol, in addition to detergent, perfume and a clay base. They are being applied twice weekly in aqueous acetone to the clipped skin of A and DBA mice. Some of each preparation is probably absorbed orally through licking.

So far, malignant lymphomata have developed after 26–57 weeks in 6–9% of treated mice and 0–3% of controls. Only low concentrations of dyes themselves are present in the solutions applied and tests using single constituents are now needed.

2-Nitro-p-phenylenediamine and 4-nitro-o-phenylenediamine are present in a number of proprietary hair colourants. When added to cultures of human blood lymphocytes, a small increase in chromatid aberrations was observed at 48 h.

THE NITROSATION OF FOOD AMINES UNDER STOMACH CONDITIONS. C. S. DYKE and C. L. WALTERS, B.F.M.I.R.A., Leatherhead, Surrey.

Nitrosamines can be formed by the action of nitrous acid on secondary and tertiary amines and quarternary compounds. Nitrosation is catalysed by thiocyanate which is secreted in the saliva and particularly that of smokers (Boyland, *Nature, Lond.*, 1974, **248**, 601). Nitrosation of food amines at high levels of nitrate (0·14 mol/l) atypical of the stomach of the consumer has led to the formation of up to 80 μg/kg N-nitrosopiperidine (N Pip). At 0·145 mmol/l nitrite, a level considered to be the maximum likely to occur normally in the stomach, nitrosation occurred but to a much reduced extent with the formation of volatile nitrosamines, particularly in the presence of thiocyanate. Studies in which volunteers were given a meal including cured meat containing nitrite within the legal limit have so far been negative, following recovery of the meal after 30 min, whilst corresponding *in vitro* studies have revealed 1·7 μg/kg N Pip after 1 h and 3·4 μg/kg after 3 h. The possibility therefore exists that nitrosamines could be formed in the stomach after a longer residence time, unless this is precluded by an inhibitory physiological factor such as ascorbate.