at a final concentration of 15 per cent, a condition not likely to precipitate or denature most proteins. Compounds I and II are also much more intensely coloured, especially in acid or base respectively, than DDPM or Nosoh's water-soluble azomercurial[7] (see extinction coefficients in Table 1) and hence should provide a much more sensitive indicator of any labelled peptides which may be isolated from derivatized proteins. The colour of peptides labelled with I or II separated by paper chromatography or electrophoresis could be considerably intensified by exposure to the fumes of concentrated hydrochloric acid or ammonium hydroxide.

Work is now in progress to test the applicability of compounds I and II for the labelling of SH proteins for the purpose of isolating cysteine-containing peptides.

This investigation was supported in part by research grant *GM* 04616 from the National Institute of General Medical Services, U.S. Public Health Service, and grant *G*13965 from the National Science Foundation.

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[1] Bennett, H. S., and Yphantis, D. A., *J. Amer. Chem. Soc.*, **70**, 3522 (1948).
[2] Bennett, R. J., and Seligman, A. M., *Science*, **116**, 323 (1952).
[3] Horowitz, M. G., and Klotz, I. M., *Arch. Biochem. Biophys.*, **63**, 77 (1956).
[4] Akerfeldt, S., *Acta Chem. Scand.*, **13**, 627 (1959).
[5] Witter, A., and Tuppy, H., *Biochim. Biophys. Acta*, **45**, 429 (1960).
[6] Clark-Walker, G. P., and Robinson, H. C., *J. Chem. Soc.*, 2810 (1961).
[7] Nosoh, Y., *J. Biochem.* (Japan), **50**, 450 (1961).
[8] Whitmore, F. C., and Middleton, E. B., *J. Amer. Chem. Soc.*, **43**, 621 (1921).
[9] Liener, I. E., *Biochim. Biophys. Acta*, **53**, 332 (1961).
[10] Bernhard, S. A., and Gutfreund, H., *Biochem. J.*, **63**, 61 (1956).
[11] King, T. P., Yphantis, D. A., and Craig, L. C., *J. Amer. Chem. Soc.*, **82**, 3350 (1960).

L-Cysteine as Inhibitor of Potassium Retention by Brain Slices

L-CYSTEINE, in common with several other amino-acids, is able to inhibit the uptake of L-histidine by brain slices, but the inhibition which it produces, unlike that produced by the other amino-acids, is non-competitive[1]. It therefore appeared possible that cysteine might also have an inhibitory action on other transport phenomena. Its effect on the transport system for potassium, as indicated by alteration in the level of potassium in brain slices (normally maintained well above that of a suitable environment[2]), has therefore been investigated, and compared with the effect of other amino-acids.

The technique used was essentially similar to that described elsewhere, with similar precautions taken[1]. Slices of rat brain were incubated with agitation for 1 h at 37° C in Krebs's bicarbonate saline which contained the amino-acid under investigation. The tissue was then treated as described elsewhere[3] and the potassium estimated in tissue fluid by means of a flame photometer. There was no detectable alteration in the amount of any of the amino-acids in the suspending medium during the period of incubation, as judged by visual inspection of paper chromatograms[1]. (Reduced glutathione, which might be expected from its structure to have effects somewhat similar to those of cysteine, did show evidence of alteration in its nature and so has not been included in the series.) Cysteine did not interfere with the estimation of potassium when present at ten times the concentration of the latter. The *p*H of the suspending medium was measured at the end of all experiments and lay within the range 7·3–7·5.

The concentration of potassium in isolated tissue suspended in a suitable medium may be altered by the presence of amino-acids[2,4,5], but, as shown in Table 1, cysteine produced a greater reduction in the concentration of potassium in brain slices than did any of the other amino-acids investigated.

Table 1. EFFECT OF AMINO-ACIDS ON POTASSIUM IN BRAIN SLICES

Amino-acid added to suspending medium	No. of tissue samples for each amino-acid	Potassium concentration in tissue m.equiv./l.
None	24	46 ± 4
Glutamic acid	8	50 ± 3*
Aspartic acid	8	50 ± 4*
Taurine	8	49 ± 2
Tyrosine	8	48 ± 2
Alanine, glycine, proline, valine	6	47 ± 2 to ± 3
S-Ethyl-cysteine, histidine hydrochloride, methionine, phenylalanine	6 or 8	46 ± 1 to ± 5
S-Methyl-cysteine	10	44 ± 4
β-Alanine, leucine, 2-thiolhistidine, ornithine hydrochloride	8	44 ± 3
γ-Aminobutyric acid, cystine, tryptophan	8	44 ± 2
Lysine hydrochloride	10	43 ± 4
Arginine hydrochloride	8	43 ± 3
Cysteic acid	10	43 ± 3*
Cysteine hydrochloride	16	38 ± 3‡†

Amino-acids were of the L-form where applicable. Initial concentration of each amino-acid in suspending medium 2 mM. Figures shown for potassium concentration are mean and standard deviation. Incubation 1 h 37° C.
*$P < 0.05$, †$P < 0.001$: significance of difference from concentration of potassium with no added amino-acid. ‡$P < 0.01$: significance of difference from concentration of potassium in the presence of any of the other amino-acids used.

The effect of cysteine appears to be dependent on the presence of a free thiol group not attached to a resonating system. Those amino-acids containing a blocked thiol group (*S*-ethyl-cysteine, *S*-methyl-cysteine, cysteic acid, cystine and methionine) or in which the thiol group was attached to a resonating system (2-thiolhistidine) altered the concentration of potassium in the tissue relatively slightly or not at all.

Cysteine has been found to have a toxic effect on the intact animal[6] and on cultured tissue[7] and also to alter the activity of certain intracellular enzymes *in vivo*[8]. It is therefore likely, particularly in view of the non-competitive nature of its effect on L-histidine uptake, that the inhibitory effect of cysteine on the transport of both L-histidine and potassium is the result of interference with intracellular processes on which both types of transport are dependent.

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[1] Neame, K. D., *J. Neurochem.*, **11**, 67 (1964).
[2] Pappius, H. M., and Elliott, K. A. C., *Canad. J. Biochem. Physiol.*, **34**, 1053 (1956).
[3] Neame, K. D., *J. Neurochem.*, **6**, 358 (1961).
[4] Terner, C., Eggleston, L. V., and Krebs, H. A., *Biochem. J.*, **47**, 139 (1950).
[5] Riggs, T. R., Walker, L. M., and Christensen, H. N., *J. Biol. Chem.*, **233**, 1479 (1958).
[6] Dent, C. E. (personal communication).
[7] Eaton, M. D., and Scala, A. R., *Exp. Cell Res.*, **33**, 481 (1964).
[8] Emmelot, P., Mizrahi, I. J., Naccarato, R., and Benedetti, E. L., *J. Cell Biol.*, **12**, 177 (1962).

Changes in the Fatty Acid Composition of Sebum associated with High Carbohydrate Diets

EXCESS of carbohydrate in the diet has been considered to be of significance in the ætiology of certain types of skin disorder[1]. With this in mind the opportunity was taken to examine the fatty acid composition of the sebum in subjects who were on experimental diets containing a relative excess of carbohydrate.

The subjects, seven adult males, were given an experimental diet consisting of 500 g of either sucrose or raw maize starch daily with *ad libitum* quantities of lean meat and green vegetables with vitamin supplements. The experimental diet was given for 25 days followed by 25 days on a normal free-choice diet, with a final 25 days on a diet which contained the alternative carbohydrate. Full details of the dietary intake are given elsewhere[2].