terial generic names used in conjunction with a specific epithet. To ascertain whether this desire was general, I addressed a letter to the editors of 77 English-language journals and asked each to complete a questionnaire and to amplify his answers when necessary. The editors of *Nature* kindly published a note inviting others interested in the problem to write to me. Replies have been received from the editors of 65 journals. The questions asked them and their answers follow.

1) Do you insist that the generic name be written in full when it is first used? Yes, 57; no, 6.
2) May generic names subsequently be abbreviated? Yes, 62.
3) When abbreviations are allowed, which form is used? Single letter, 29; author's choice, 15; set of approved abbreviations, 18.
4) Do you issue a set of instructions to authors? Yes, 33.
5) Do you encourage the use of common names where these are unambiguous? Yes, 28; no, 16.
6) Are you in favor of attempting to standardize abbreviations? Yes, 52; no, 4; doubtful, 3. In favor of standardizing at the international level, 34; first in the English language, 27.

Analysis of the replies failed to show any significant difference in the views of editors in different countries; nor was there much difference between journals devoted to medical or nonmedical sciences. Generally, medical journals give more freedom of choice about abbreviations to authors, but about half of them issue instructions or suggestions that authors are supposed to follow. Several American medical journals use generic abbreviations given in Dorland's *Medical Dictionary*.

Possible solutions. Many editors sent helpful letters with their replies to the questionnaire, and several stressed the fact that clarity is more important than space-saving. Four solutions are presented for consideration, three of these being in current use:

1) The generic name is written out in full the first time that it is used in each paragraph. The initial letter (without a second letter) is used as an abbreviation on the second occasion that the genus is mentioned in the same paragraph, provided that no other genus with the same inital letter has been mentioned between the first and second naming of the genus in question, in which case the name is again given in full (*Biological Reviews*).

2) The same principle as 1 is applied to each page instead of to each paragraph. But, wherever it would not be obvious which genus is indicated, the generic name is spelled out. (*Annals of the New York Academy of Sciences.*)

3) The same principle applied to each paper; that is, the full name when first mentioned (i) in the title, (ii) in the paper, and (iii) in the summary, with subsequent use of a single (initial) letter for the generic name. Where this would be ambiguous, the name would be written out. (This is the current practice of the editors of 29 journals who replied to the questionnaire.)

4) Do not use abbreviations of generic names, except when a list of species of the same genus is involved, in which case an initial letter abbreviation would be used. This suggestion takes as an analogy the practice of spelling out the long names of organic chemicals, any one of which may occur more than once in a sentence.

S. T. COWAN, *Joint Permanent Secretary International Committee on Bacteriological Nomenclature, National Collection of Type Cultures, London, N.W.9*

18 November 1954.

Conversion of Kynurenine into 3-Hydroxykynurenine in Man

Our finding that hydroxykynurenine is responsible for the diazo-reaction has been confirmed by Dalgliesh and Tekman (*1*), but the route of the formation of 3-hydroxykynurenine from tryptophan in man is yet unknown.

Dalgliesh and Tekman (*1*) suggested that an alternative pathway from tryptophan to hydroxykynurenine might have been involved, possibly via 7-hydroxytryptophan. However, 7-hydroxytryptophan has been synthesized by Ek and Witkop (*2*) and shown not to be attacked by the enzyme system converting tryptophan into kynurenine. This result was confirmed by us by adding liver or kidney homogenate of mice to 7-hydroxytryptophan, which was kindly supplied by B. Witkop.

We have now made the following experiments. When 0.5 to 2.0 g of tryptophan was administered to the urochromogen-reaction-positive but diazo-negative patients suffering from tuberculosis, their urine turned strongly diazo-positive and, on paper chromatogram, the spots of kynurenine and 3-hydroxykynurenine dectectable with diazo reagents became larger and stronger. This fact seemed to indicate that tryptophan is converted into 3-hydroxykynurenine via kynurenine. So we administered kynurenine to the urochromogen-positive but diazo-negative patient suffering from pulmonary tuberculosis and saw that the diazo-reaction of the urine turned strongly positive. On detection by paper chromatogram, the spot of 3-hydroxykynurenine enlarged and became stronger.

The preparation of urine for paper chromatographic detection was done by the procedure already described (*3*). The urochrom fraction thus prepared was precipitated with Hopkins-Cole's reagent. The precipitate was treated with hydrogen sulfide, and the filtrate was concentrated to a sirup under diminished pressure with exclusion of oxygen. This fraction containing 3-hydroxykynurenine was purified by paper chromatography, and identification was effected by comparing with the pure sample of 3-hydroxykynurenine on paper chromatogram using several solvent systems as developer. The results are summarized in Table 1.

Table 1. Rf values of 3-hydroxykynurenine obtained with various solvent systems as developer.

Solvent systems, samples	Butanol, acetic acid, water (4: 1: 5)	Butanol saturated with 1-percent NH_4OH	70-percent isopropanol	Methanol, butanol, benzene, water (4: 2: 2: 2)
3-Hydroxykynurenine fraction obtained from the urine	0.38	0.02	0.30	0.36
Pure 3-hydroxykynurenine	.37	.06	.29	.36

The ultraviolet absorption spectrum of this purified sample was the same as that of pure 3-hydroxykynurenine, showing $\lambda_{max.}$ at 370 mμ at *p*H 7.3, which shifted to 310 mμ at *p*H 1.0. The injection of pyridoxine to the diazo-positive patients did not influence the excretion of 3-hydroxykynurenine in urine (*4*).

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References and Notes

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26 July 1954.

Enzymatic Conversion of δ-Amino Levulinic Acid to Porphobilinogen

Porphobilinogen, a compound isolated from the urine of patients with acute porphyria (*1*) has been shown by Bogorad and Granick (*2*) to be converted to protoporphyrin by colorless extracts of *Chlorella* cells. This experiment signifies that enzymes carrying out the conversion of porphobilinogen to protoporphyrin are water-soluble and, thus, that the individual enzymes ought to be amenable to the customary procedures of isolation. Porphobilinogen can also be converted to protoporphyrin by hemolyzates of chicken and red blood cells, as was shown by Falk, Dresel, and Rimington (*3*), and we have confirmed this finding. On the basis of these experiments, porphobilinogen may be considered to be the natural monopyrrole precursor of protoporphyrin.

The precursor of porphobilinogen itself appears to be δ-amino levulinic acid, a compound shown by Shemin and Russell (*4*) to be converted to protoporphyrin by duck erythrocytes. We have found that δ-amino levulinic acid is converted to porphyrins by extracts of *Chlorella* cells, by extracts of spinach, by a strain of *Tetrahymena geleii,* and by extracts of chicken erythrocytes.

Dresel and Falk (*5*) have recently observed the conversion of δ-amino levulinic acid to porphobilinogen by hemolyzed chicken erythrocytes. Recently we have obtained an extract from chicken erythrocytes that converts δ-amino levulinic acid to prophobilinogen. From washed erythrocytes, which have been hemolyzed with water and the hemoglobin denatured with $CHCl_3$-ethanol, a filtrate has been obtained that contains the active enzyme. This aqueous extract is almost colorless. Furthermore, alkaline extracts of the residue contain enzymes that convert porphobilinogen to porphyrins.

The enzyme in the aqueous extract sediments in the ultracentrifuge somewhat more rapidly than does hemoglobin. It may be separated from the accompanying ferritin by starch electrophoresis and appears to be colorless. No appreciable loss of activity accompanies dialysis, so that no loosely bound coenzyme appears to be involved in its activity. Its maximum activity is at *p*H 6.5. The enzyme preparation is somewhat unstable, half of its activity being lost in a week at icebox temperature. Activity is likewise lost during starch electrophoresis in Veronal buffer. In one preparation, δ-amino levulinic acid was observed to be converted to porphobilinogen to the extent of 90 percent of the theoretical as determined with the Ehrlich reagent. The porphobilinogen thus formed has an R_f value in a butanol-acetic-water mixture identical with crystalline porphobilinogen.

The condensation of two molecules of δ-amino levulinic acid to form porphobilinogen requires that two bonds be formed (Fig. 1), a carbon-carbon bond at *A* and a carbon-nitrogen bond at *B,* with the simultaneous removal of two water molecules. One might therefore consider that two enzymes may be involved in this condensation. This does not appear likely since the activity of the enzyme was found to be directly proportional to its concentration through a tenfold dilution of the enzyme preparation. Starch electrophoresis indicated one peak of activity, likewise suggesting that only one enzyme was involved. Whether one enzyme simultaneously brings about the aldol condensation at *A* and the ketimine condensation at *B* remains to be determined. However, since enzymes in general appear to have only one specific activity, it

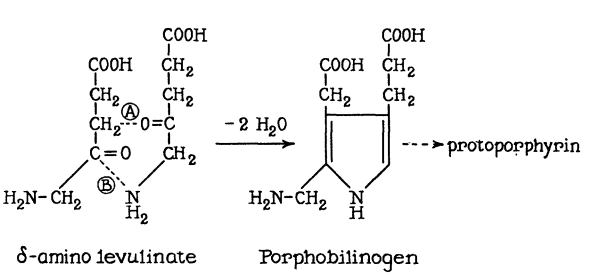

Fig. 1. Structure of porphobilinogen.