polysulphate is primarily due to its high sulphation, although there might be the other factors; and (2) the sulphate groups would be linked, at least, to the C(4) position in the galactosamine moiety by the sulphation, in addition to the C(6) position in the galactosamine moiety of chondroitin sulphate from shark cartilage.

Further work on physiological properties of this material will appear elsewhere. I thank Prof. Y. Oshima and Prof. S. Suzuki for their valuable advice and criticism, Dr. T. Furuhashi for providing chondroitin polysulphate, and Dr. T. Abe for assistance in the determination of anticoagulant activities.

KATSUMI MURATA

Department of Physical Therapy
and Internal Medicine,
Faculty of Medicine, University of Tokyo.

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Identification of Formic Acid as the *p*-Bromophenacyl Ester

IN the course of work on the microbial metabolism of glycine and glyoxylic acid we have had occasion to isolate formic acid as the *p*-bromophenacyl ester which is widely quoted[1,2] as having m.p. 140°. It was observed that the derivative, m.p. 140–42°, isolated from authentic sodium ^{14}C-formate (95,200 counts/min./μmol.) was inactive and showed no depression in melting point when mixed with *p*-bromophenacyl alcohol, m.p. 142°. A search of the literature revealed that Neish and Lemieux[3] have made a similar observation and have described a preparation of authentic *p*-bromophenacylformate, m.p. 92°. In my hands the methods of Neish and Lemieux yields a derivative, m.p. 98° (92,200 counts/min./μmol.), when the above-labelled formate is used in the preparation (cf. Heilbron and Bunbury[4], who quote m.p. 99°).

In view of the apparent widespread use of '*p*-bromophenacylformate', m.p. 140°, in the identification of formic acid, I have thought it desirable to direct attention to the paper of Neish and Lemieux[3].

In view also of the present interest in the microbial metabolism of C(2) compounds it may be noted that the puzzling degradation of labelled glycine and glyoxylic acid by a strain of *Pseudomonas* to formic acid (from the carboxyl group) and carbon dioxide (from the α-carbon) requires reinvestigation as the evidence adduced by the author[5,6] involved the characterization of the isolated formic acid as the '*p*-bromophenacyl ester', m.p. 140°.

G. W. CROSBIE

Department of Biochemistry,
University of Glasgow.

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Production and Utilization of Free Acetate in Man

THE concentration of acetate in the blood determined enzymatically[1] in a number of experimental subjects was found to be about 2 μgm./ml., independent of the nutritional régime (see Table 1). Attempts to raise the level of acetate in the blood by oral ingestion of acetic acid or acetates were unsuccessful, because the experimental subjects were not able to consume a sufficient quantity of those substances.

Table 1. CONCENTRATION OF ACETATE IN HUMAN SERUM. RESULT OF DUPLICATE ANALYSES IN ONE EXPERIMENTAL SUBJECT

	(μgm./ml.)
Fasting for 17 hr.	1·5–1·5
2 hr. after a heavy mixed meal	2·1–2·5
2 hr. after the intake of 90 gm. fat	2·5–2·7

It was, however, observed[2] that during the metabolism of ethanol the level of blood acetate increased to 19–57 μgm./ml. in 20 subjects with concentrations of ethanol in the blood ranging from 0·2 to 2·6 mgm./ml. The acetate concentration showed no correlation to the ethanol concentration.

The formation of free acetate from ethanol was shown to take place in the liver and to be the major, if not the only, pathway of alcohol metabolism[3].

This constant and continuous production of acetate makes it possible in a simple way to study the utilization of acetate in the human organism. The rate of disappearance of ethanol from the blood, provided absorption from the intestine is completed, equals the rate of production of acetate. If no acetate is lost from the organism and a steady-state concentration in the blood has been reached, formation of acetate will equal the utilization. The loss of acetate through the urine is negligible, as the concentration of acetate in urine was less than that in blood in all cases examined.

Acetate is utilized mainly in the peripheral tissues[3], as may be inferred also from the work of Forsander, Räihä and Suomalainen[4], who showed that while very little radioactive carbon dioxide is formed by rat liver perfused with blood containing carbon-14 labelled ethanol, marked utilization occurred when the blood was allowed to perfuse also the hind-quarters of the animal.

Ethanol metabolism and therefore the rate of production of acetate[5] may be changed within certain limits. Thus fasting reduces the metabolism of alcohol somewhat, while administration of large quantities of fructose may increase it by a factor of 2 (ref. 6), as illustrated in Fig. 1.

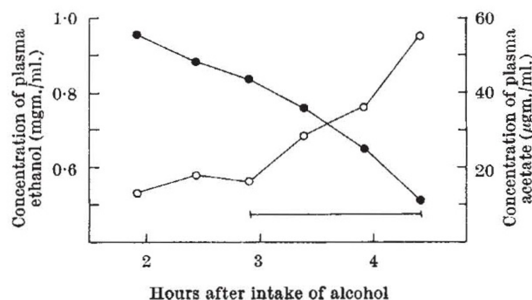

Fig. 1. Influence of fructose on the rate of disappearance of plasma ethanol ●—● and the concentration of plasma acetate ○—○. The experimental subject had fasted for 16 hr. before the ingestion of ethanol, which was finished at 0 hr. Fructose (175 gm.) was given in small portions throughout the period indicated by the horizontal line

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