

may also be unhydrolysed nucleotide. Extraction of the spots and identification by ultra-violet absorption measurements has not been successful, since the amyl alcohol used in the chromatography is contaminated by a substance with absorption in this region. Purification of the amyl alcohol has been unsuccessful, and it has not yet been possible to get a synthesized amyl alcohol.

These investigations show that the prosthetic group is a dinucleotide. Kornberg³ has shown that diphosphopyridine nucleotide (co-enzyme, co-enzyme I) can undergo a direct phosphorylation to triphosphopyridine nucleotide (co-enzyme II) by adenosine triphosphate and a yeast enzyme. Since the dinucleotide described here has the same characteristic, one may suppose an analogous structure. However, it does not seem to contain nicotinic amide but another as yet unidentified substance with basic properties.

Further investigations are going on, and a more detailed description will appear elsewhere.

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BERTIL GELOTTE

¹ Gelotte, B., *Biochim. Biophys. Acta* (in the press).

² Snellman, O., and Gelotte, B. (see preceding communications).

³ Kornberg, A., *J. Biol. Chem.*, **182**, 805 (1950).

The Prosthetic Group of Actin

A STUDY of the prosthetic group of *G*-actin and *F*-actin has been made with paper chromatography, using as solvent an aqueous solution of 5 per cent dibasic sodium phosphate and a solution of 0.5 per cent lauryl amine in amyl alcohol as described elsewhere¹. We could detect only one spot when investigating the prosthetic group of *F*-actin, the R_F value of which was 0.67. From *G*-actin also only one spot was visible, with the R_F value 0.64. These R_F values correspond to the values found for the phosphate-absorbing protein derived from the actomyosin complex reported in another communication². Acid hydrolysis of the prosthetic groups from the two kinds of actin showed, upon paper chromatography, identical spots with the R_F values 0.40 and 0.85 respectively. The prosthetic group of actin is a dinucleotide containing adenosine and another hitherto unidentified base. This dinucleotide can occur in two forms, one richer in phosphorus (in *G*-actin) and the other poorer in phosphorus (in *F*-actin). It may be mentioned that in no circumstances have any spots derived from adenosine tri- or di-phosphate been detected. The R_F values for these substances under the same conditions are 0.83 and 0.76, so they must occur in insignificant amounts.

Earlier, Straub *et al.*³ have found that actin contains a prosthetic group which they consider to be adenosine triphosphate in *G*-actin, and which during the polymerization of *G*-actin to *F*-actin splits off one phosphoric acid residue and is transformed into adenosine diphosphate.

The absorption spectrum of the isolated prosthetic group in a Beckman spectrophotometer gives a curve which resembles that of an adenosine derivative. The relation of the absorption curve to pH, however, is different from that of an adenosine phosphoric acid. It seems also that the prosthetic group can in certain circumstances be split, giving off adenosine-5-phosphate, which can be deaminated by Schmidt's deaminase. These results have supported the in-

correct assumption that the prosthetic group is adenosine triphosphate instead of the dinucleotide.

G-actin contains the dinucleotide which is rich in phosphorus, and *F*-actin contains the dinucleotide poorer in phosphorus. During the transformation of *G*-actin into *F*-actin, the phosphorus-rich and therefore more labile dinucleotide gives off one phosphoric acid residue and is transformed into the dinucleotide of *F*-actin.

Upon contraction *in vitro* of actomyosin with adenosine triphosphate, the myosin splits off a phosphoric acid residue of the triphosphate. It is possible for the energy-poor dinucleotide in *F*-actin to take up such a phosphoric acid residue in the nascent state. *F*-actin is thus transformed to *G*-actin during the contraction.

Straub *et al.* have found that when *F*-actin is dialysed against a solution of adenosine triphosphate for a longer time, the *F*-actin is converted into *G*-actin. At the same time adenosine triphosphate disappears and adenosine diphosphate appears. He assumes that the diphosphate in *F*-actin takes the place of triphosphate. From our results the process is better explained by assuming that the dinucleotide takes up a phosphoric acid residue from the adenosine triphosphate on account of its instability; it always loses phosphorus gradually with time. We have also found that the dinucleotide can take up phosphoric acid residues given off by the adenosine triphosphate in such a manner.

It seems thus that adenosine triphosphate does not play the dominant part previously assigned to it in the contraction process. This role may be taken by a dinucleotide (one group of which is adenosine, the other is not yet identified) which can take up and give off a phosphoric acid residue. The adenosine triphosphate acts as a phosphorus donor.

In many respects this dinucleotide resembles coenzyme, which also occurs both as a phosphorus-rich and a phosphorus-poor compound (co-enzyme II and I). A comparison of the dinucleotide with coenzyme has shown that they are not identical.

Further details of these investigations, which were supported financially by the Swedish Natural Science Research Council, will be given elsewhere.

OLLE SNELLMAN
BERTIL GELOTTE

Institute of Biochemistry,
Uppsala.
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¹ Snellman, O., and Gelotte, B. (see preceding communication).

² Gelotte, B. (see preceding communication).

³ Straub, F. B., and Feuer, G., *Biochim. Biophys. Acta*, **4**, 455 (1950).

CHEMISTRY OF CANCER

A MEETING, under the joint auspices of the Chemical Society, the Royal Institute of Chemistry, the Society of Chemical Industry, and the Institute of Petroleum, was held in Manchester on March 15, with the general title "The Chemistry of Cell Division". The proceedings were sadly interrupted by the sudden death of Prof. G. A. R. Kon while contributing to the discussion at the end of the morning session, and the meeting was abandoned. Three papers were, however, read.

In his introductory survey, Prof. A. Haddow dealt with the biological effects (tumour-inhibitory, cytological and carcinogenic) of cytotoxic substances, with special reference to the nitrogen mustards. It is