is trying to form a classification based on character similarities and correlations or trying to understand the phylogeny of a group of organisms by referring to characteristics observed only in the living representatives.

Another point discussed with some animation was whether all characters of living organisms are adaptive, even though many of them may appear not to be. Possibly the obvious phenotypic effect of a pleiotropic gene whose other, less obvious, feature is influenced by selection pressures may lead us astray at times. It was generally accepted that all variants might be adaptive, but perhaps what did not emerge too clearly was how much of this adaptive variation was based on present selection pressures, and how much was caused by pressures acting in the remote past, so setting a pattern for a particular group from which it cannot now escape.

Although one must admit that the Systematics Association had taxonomists primarily in mind when it arranged this symposium, there were few "pure" taxonomists present; and indeed most of the audience were either biochemists, microbiologists or taxonomists who had already used biochemical techniques in some way or other. No doubt, however, taxonomists and others will read the papers when they are published by the Systematics Association. The combination of papers and discussions from workers who are actively using a wide range of biochemical and serological techniques led to lively discussions and a feeling that the symposium had served a useful purpose. To me it seems that the strong links being forged between descriptive and experimental disciplines, and the wide and excitingly different biochemical and biophysical techniques now being introduced into taxonomic studies, are some of the most exciting developments in contemporary biology.

Symmetry Conserved

from our Molecular Biology Correspondent

Two articles about the sub-units of aldolase illustrate the scope for error in the evaluation of quaternary structure in enzymes. Aldolase is by no means the only case which has been the subject of this kind of ambiguity, and there are others which are still unresolved.

In this column last June two papers were briefly discussed which gave apparently conclusive evidence that mammalian aldolase consists not of four sub-units, as had initially been suggested, but of three, two of them identical. This was based on the one hand on molecular weight determinations on the intact and dissociated enzyme, and on the other on measurements of the number of catalytic sites and on hybridization studies. It now turns out that the conclusion is wrong, and that the enzyme has after all four sub-units —a result altogether more compatible with current concepts of enzyme structure.

Morse, Chan and Horecker (*Proc. US Nat. Acad. Sci.*, **58**, 628; 1967) have renounced their earlier conclusion; they find four C-terminal tyrosyl groups per molecule, which indicates the presence of four chains (whereas three C-terminal and N-terminal groups have been reported at various times by several workers in the past). But there still appear to be only three active sites in the intact enzyme. Whether one site is blocked in the enzyme as isolated, or whether the attachment of substrates at three sites inhibits the fourth, is not yet known.

The most comprehensive new evidence on the quaternary structure of aldolase comes, however, from Penhoet, Kochman, Valentine and Rutter (*Biochemistry*, **6**, 2940; 1967). In the first place they have prepared hybrids of aldolases from different tissues by acid-dissociation and reaggregation. A mixture of the two native aldolases, A and C, yields a set of five components on hybridization, which all possess the molecular weight of intact aldolases. Whereas the parent molecules alone produce no new species on dissociation, each hybrid gives rise to a complete set of five components, made up of combinations of the two types of sub-unit. When these are made from equimolar mixtures of radioactively labelled A and unlabelled C, their specific activities are in the ratio 1 : 0·75 : 0·5 : 0·25 : 0, the first and last corresponding to reconstituted A and C. It follows that the combining unit is the quarter-molecule—α in aldolase A, and γ in C—and the species formed on hybridization are α_4 (intact A), $\alpha_3\gamma$, $\alpha_2\gamma_2$, $\alpha\gamma_3$ and γ_4 (intact C). These elegant experiments are supported by electron microscopy of intact aldolases, which shows clearly the presence of four globular sub-units in intact molecules, arranged tetrahedrally.

It is interesting to examine with hindsight the published molecular weight determinations on aldolase and its sub-units. For intact aldolase, two leading laboratories produced values of 142,000 and 160,000. Whereas this discrepancy might still be regarded as marginally tolerable, the corresponding values for dissociated sub-units were 51,000 and 37,000, and this is obviously sufficient to cause an irreconcilable difference in numerology. This demonstrates that alarming differences in molecular weights are still possible, even in the most expert hands. The conditions of dissociation were different in the two studies, however, and it must be supposed that the trouble arose from incomplete dissociation, perhaps obscured by non-ideality. (Penhoet *et al.* have also redetermined the molecular weight of the intact enzyme by high-speed sedimentation equilibrium, and obtain values in the range 150,000–157,000.)

The results may be seen as a vindication of Kawahara and Tanford, who had the correct answer for the number of sub-units. It suggests also the advantages of dissociation by 6*M* guanidine hydrochloride, which Tanford and his associates have advocated, and have shown in a series of painstaking investigations (the most recent of which have just appeared in *J. Amer. Chem. Soc.*, **89**, 5023, 5030; 1967) to eliminate all measurable non-covalent interactions in all of the large number of protein chains which have been examined. Further reflexions on the experimental discrepancies concerning aldolase are to be found in the paper by Penhoet *et al.*, and make salutary reading.

T_4 Messenger RNA

from our Cell Biology Correspondent

It is well known that when *T* even phage infect *E. coli*, some phage specific proteins are made early in the infective cycle, and as the cycle progresses the synthesis of these 'early' proteins ceases and other 'late' proteins are made. Is the mechanism regulating the cessation of 'early' protein synthesis and initiation of 'late' protein