ferritin appeared to have some protein sub-units missing. Since incubation with apoferritin produced normal ferritin, the authors suggest that in ferritin synthesis the iron polymer core is formed first from low molecular weight iron chelates, and the protein sub-units then aggregate round it to form the stable ferritin molecule.

In a second paper the same group show that the iron can be removed from ferritin by chelating agents under very mild conditions (*Biochemistry*, **7**, 613; 1968). Ferritin was dialysed against solutions of the chelating agents at *p*H 7·4 and 25° C. 0·1 M nitrilotriacetate removed 44 per cent of the iron, 0·1 M ethylenediamine tetraacetic acid 3 per cent and 0·1 M sodium citrate 1 per cent. No iron was removed by buffer alone after 14 days dialysis. During the first 48 hours, iron was removed from non-crystalline ferritin more rapidly than from crystalline ferritin. Thus iron can be removed from ferritin as a chelate, but there are obviously large differences in the rate and extent of the reaction with different chelating agents.

The glycoprotein which carries iron in the blood, transferrin, was already known to react with suitable chelating agents (Bates, Billup and Saltman, *J. Biol. Chem.*, **242**, 2810; 1967). Kronfeld (*Biochemistry*, **7**, 945; 1968) has now studied the effect of chemical modification of the human transferrin molecule on its capacity to bind ferric ions, to bind to reticulocytes, and the incorporation of ^{59}Fe from transferrin into haem in reticulocytes. Substitution of the amino groups increased the binding of transferrin to the reticulocytes, but only if the substitution changed the net charge on the molecule. Since there was little effect on incorporation into haem, the increased binding seems to be to sites where the iron cannot enter the cell. Ferric ion binding decreased sharply when 85–90 per cent of the free amino groups had been trinitrophenylated, suggesting that the amino groups involved in iron binding are relatively inaccessible. Almost all the carbohydrate was removed by enzymatic digestion without producing any measurable effect on the activity of transferrin, so the function of this part of the molecule remains unknown.

The Origins of 5S RNA

from our Molecular Biology Correspondent

EVER since its discovery in *E. coli* ribosomes, the low molecular weight RNA component (5*S* RNA) has provoked argument about possible functions and origins. In the latter regard, the careful genetic mapping experiments of Smith *et al.* (*J. Mol. Biol.*, **33**, 123; 1968) on *B. subtilis* produced the result that the cistrons on the DNA coding for 16*S*, 23*S* and 5*S* RNA are not clearly separated. On the other hand, hybridization of the ribosomal RNA species with the DNA gave the result that 9–10 genes for 16*S* and 23*S* RNA were present, but only 3–4 for 5*S*. This was taken as an indication, though not yet a proof, that the 5*S* RNA was an entity synthesized under independent genetic control.

A contrary viewpoint has now been put by Hecht *et al.* (*Proc. US Nat. Acad. Sci.*, **59**, 1278; 1968). The argument rests mainly on the kinetics of appearance of the three ribosomal RNA components and their precursors. For most of the evidence concerning the latter, one is directed to results "reported previously", for which the reference turns out to be a publication "in press". It is alleged, however, that these precursors, which have a higher molecular weight, are synthesized according to an apparent first-order rate law. This condition is formally required for *ab initio* transcription, whereas a deviation from first-order is taken to imply formation by conversion of some other primary transcription product. The order of the kinetics of appearance of both 16*S* and 5*S* RNA is found to be about 3 (although the rather generous scatter of the points on the double-logarithmic plot must be admitted to offer a fairly wide choice). Nevertheless, a difference appears to exist, compared with the precursor of the 16*S* RNA. Secondly, chloramphenicol, which leads to the accretion of uncompleted ribosomes ("CM-particles") but does not interfere with transcription, arrests the formation of 5*S* RNA. (One might mention here that the absence of this species in CM-particles was reported last year by Galibert *et al.* Of course, the appearance of mature 16*S* and 23*S* RNA is by the same token also prevented.) On introduction of actinomycin, which blocks transcription, some formation of the ribosomal RNAs continues. Hecht *et al.* state that they have observed no precursor RNA of low molecular weight, and that therefore the 5*S* RNA arises by way of a high molecular weight species, and they favour the 23*S* precursor, which is said to have a molecular weight exceeding that of mature 23*S* RNA by the correct amount. It will be easier to evaluate the cogency of these arguments, and the evident conflict with the results of Smith *et al.*, when the promised details are published.

Meanwhile, a further study from Marmur's laboratory (Morell and Marmur, *Biochemistry*, **7**, 1141; 1968) has appeared which contains a number of important results. Working again with *B. subtilis* ribosomes they have shown that, as in *E. coli*, one 5*S* RNA molecule is present per 70*S* ribosome, and that this resides exclusively in the 50*S* sub-unit. Moreover, it is tenaciously held—it is known that some 20 per cent of the protein can be stripped from the ribosomes by treatment with caesium chloride without disruption of the structure. This treatment is found not to remove the 5*S* RNA. Morell and Marmur have found that yet more protein can be removed without dislodging the RNA. Treatment with EDTA to extract all the magnesium ions causes unfolding without loss of protein, and under these circumstances the 5*S* RNA is liberated. It is unfortunate that it has not yet proved possible to regain native ribosomes after this treatment, and so test the requirement for 5*S* RNA.

RNA synthesis has also been studied: pulse labelling experiments show that the label enters the 23*S* RNA much sooner than the 5*S*, and the presence of a pool of 5*S* precursor is therefore indicated. The 40*S* ribosomal precursor particles have also been examined and 5*S* RNA is shown to be still substantially absent at this stage. Its appearance, accompanying maturation of the 50*S* ribosomes, can be explained either by its assimilation from the pool or in terms of a common precursor for it and the 23*S* species. Enzymatic cleavage of the 5*S* from the 23*S* RNA in its final stages of maturation would then explain the observed lag in labelling of the 5*S* RNA. This would agree with the surmise of Hecht *et al.*, but would leave the hybridization results unexplained. The last word is clearly yet to come.