A paper by Smith, Barnett, Brenner and Russell in the current issue of the *Journal of Molecular Biology* (**54**, 1; 1970) must surely fall into the first category. These workers have combined techniques of genetic selection with sequence analysis of radioactive RNA, to examine the effects of specific mutations on the functional capabilities of the su^{+}_{III} tRNA from *Escherichia coli*. This tRNA, which accepts tyrosine, can suppress amber mutations because of a mutation in its anticodon which enables it to recognize the UAG codon.

Smith and his colleagues have previously reported isolating three mutant su_{III} tRNAs which are functionally partially defective as the result of replacement of G by A residues at specific points. They have now obtained su_{III} mutants which are temperature-sensitive suppressors, and which revert to wild type by mutations at second sites. They selected the first site mutations (either spontaneous or produced by treatment with N-methyl-N-nitroso-N′-nitroguanidine) by constructing a strain of *E. coli* in which amber suppression by su^{+}_{III} tRNA is lethal, in this case by permitting the accumulation of UDP galactose within the cells. Mutants of the su_{III} locus, which can no longer suppress the amber stop, do not allow the formation of UDP galactose and so are saved. In this way, temperature-sensitive mutants can be derived which are lethal at 32° C when they suppress, and are saved at 42° C when they no longer work. N-methyl-N-nitroso-N′-nitroguanidine is unidirectional in its mutagenic activity, and is therefore suitable for obtaining second site revertants. These mutants were selected by using an amber stop in the tryptophan operon, which is suppressed by the revertants.

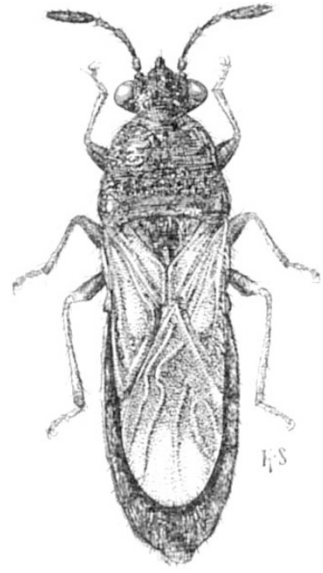
The su_{III} tRNA synthesized by these mutants was isolated by transducing the su_{III} gene, with the aid of the bacteriophage φ80, into host cells (usually containing the same su_{III} mutation), thus obtaining an enhanced yield of the su_{III} tRNA. ^{32}P-orthophosphate was added to the culture in the post-infection period, to obtain highly radioactive tRNA, and the su_{III} tRNA was purified by chromatography on benzoylated DEAE-cellulose, followed by electrophoresis through polyacrylamide gels. Finally, the mutant tRNAs were analysed by Sanger's techniques to find out exactly what went wrong.

The results were bounteous. Two temperature-sensitive suppressor mutants formed in the amino-acid acceptor arm of the tRNA through the alteration of G–C pairs to pairs which no longer form hydrogen bonds (A,C and G,A). Second site revertants of these mutants, containing A–U and G–U pairs respectively, are indistinguishable from the wild type in their suppressor activities *in vivo*. A similar G–C→A C→A–U mutation and reversion were encountered in the dihydrouracil-containing stem. In all of these cases, the K_m value for the aminoacylation of the revertant is the same as for the su^{+}_{III} tRNA, revealing that in these positions the nucleotide sequences as such are not specifically recognized by the aminoacyl tRNA synthetase. Smith *et al.* describe three further striking second site revertants, where suppressor activity is only partially restored, in the dihydrouracil-containing loop and stem. Here G–A mutations resulted in su^{+}_{III} tRNA, and subsequent C→U changes nearby were sufficient to distort the structure of this area in such a way that the tRNA became partially functional again. In two of these mutants, the U was modified to dihydroU. This experimental system will certainly be valuable in future studies of the specificity and function of tRNA modification.

A final, important and strange finding was that very much less of the suppression-defective mutant tRNAs were synthesized in the phage-infected cells than the amount of wild type su^{+}_{III} tRNA present. But second site revertants with wild type activity yielded roughly the same amounts of tRNA as the wild type. Some novel and subtle control mechanism relating the transcription of these tRNA genes to the functional activity of the gene products must therefore exist. In this connexion, one double mutant isolated by Smith *et al.* is particularly interesting, for it displays stronger suppressor activity *in vivo* than its parent single mutant, but nevertheless yields an su_{III} tRNA of identical structure. In fact, the second mutation lies outside the tRNA gene (although it must be close to it, because it co-transduces with it), and Smith *et al.* suggest that it lies within the promoter region, controlling the rate of transcription of the tRNA, and thus exerting its effect *in vivo*.

ENTOMOLOGY

South African Plant Bugs



In a revision of the plant bug genus *Ischnodemus* (Hemiptera: Lygaeidae, Blissinae) in the Ethiopian region, J. E. Slater and J. E. Harrington describe thirteen new species from localities chiefly in the savanna of eastern and southern Africa (*Ann. S. Afr. Mus.*, **26**, 211; 1970). This illustration shows the male holotype of one of the new species, *I. tenebrosus*, from Middelburg in South Africa. Its total length is 4·67 mm.

WHALES

Conservation can Succeed

from our Marine Vertebrate Correspondent

NEARLY everywhere that whaling has flourished its progress has followed the same tragic sequence—an initial period of profitable and increasing exploitation after the recognition of its potential, followed by an