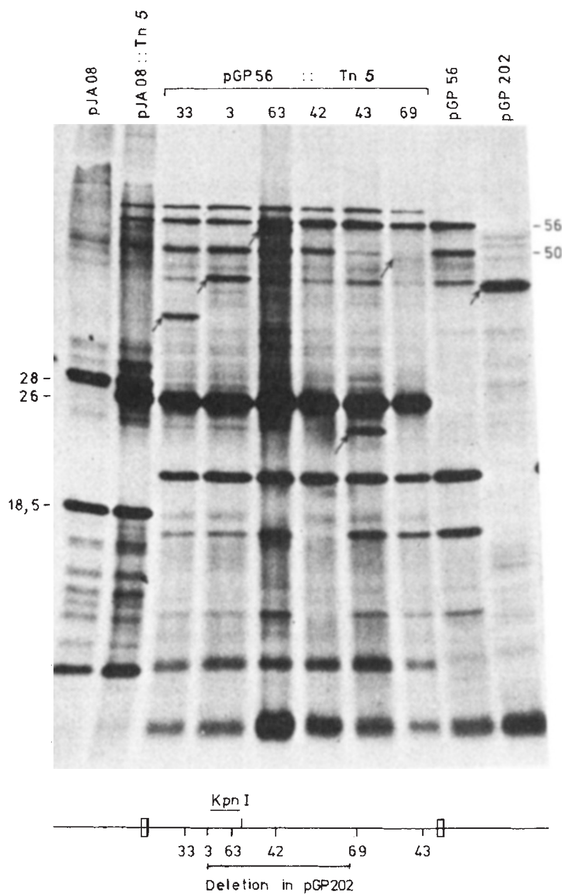

Fig. 4 Autoradiograph showing polypeptides synthesized in minicells isolated from cells containing pGP56, derivatives of pGP56 and plasmids with Tn*5* insertions. Minicells were labelled with ^{35}S-L-methionine and the polypeptides separated on a 12.5% polyacrylamide gel[13]. The autoradiograph shows the polypeptide pattern of minicells containing the following plasmids: control plasmid pJA08 (provided by R. Brandsma) (lane 1), pJA08::Tn*5* (lane 2), pGP56::Tn*5* no. 33 (lane 3), pGP56::Tn*5* no. 3 (lane 4), pGP56::Tn*5* no. 63 (lane 5), pGP56::Tn*5* no. 42 (lane 6), pGP56::Tn*5* no. 43 (lane 7), pGP56::Tn*5* no. 69 (lane 8), pGP56 (lane 9), pGP202 (lane 10). The *S* gene product (56K), *S'* gene product (50K), β-lactamase (28K)[13], neomycin phosphotransferase II (26K)[8] and the product of SSB (18.5K)[17] are indicated. Truncated proteins are indicated by →.

splicing within the prokaryotic cell. The recombination of a common region to different regions to form variable functional genes is a well known phenomenon in eukaryotes in the assembly of immunoglobulin genes[10,11]. In the case of G inversion, the variability is in the Sv or the Sv' region. The common region of the S and S' proteins is probably the part which becomes attached to the phage tail, whereas the variable part recognizes the receptor sites of the different hosts. This is now being tested by isolating and mapping mutants of Mu with other host ranges. Although we have shown that *S* and *S'* share a common region of DNA in α, this does not directly imply that their respective products are identical at the N-terminus. The calculated N-terminus of genes *S* and *S'* differed by ~75–100 bp. This may be due to errors in restriction mapping and/or molecular weight estimation. Moreover, we do not know exactly at which position translation has stopped in the various Tn*5* insertions. However, the observed difference may reflect a real difference in length of the Sc part of the two proteins. Therefore, definite proof for the presence of a common part in *S* and *S'* at the protein level must await determination of their amino acid sequences.

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Erratum

In the letter by P. M. Conn *et al.*, *Nature* **296**, 653–655 (1982), the title should read 'Conversion of a gonadotropin-releasing hormone antagonist to an agonist: implication for a receptor microaggregate as the functional unit for signal transduction'. In Fig. 2 the lines labelled *a*, *b* and *c* should be termed (1), (2) and (3) to correspond to the text. In ref. 4 the page numbers should read 264–265, and the journal in ref. 6 is *Endocrinology* **109**, 2040–2045 (1981). In addition the following 'Note added in proof' should have appeared: Additional evidence for the significance of microaggregation comes from the observation of potency enhancement of a GnRH agonist in conditions which favour receptor microaggregation[20]. In addition, a hormone antagonist has been converted to an agonist by antibody cross-linking[21].

20. Conn, P. M., Rogers, D. C. & McNeil, R. *Endocrinology* (in the press).
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Corrigendum

In the letter 'Conversion of the chemical energy of methylmalonyl-CoA decarboxylation into a Na^+ gradient' by W. Hilpert and P. Dimroth, *Nature* **296**, 584–585 (1982), in Fig. 1 the total incubation volume should be 0.67 ml, not 10.67 ml as shown.

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