molecule in the protein synthetic process. 4.5S RNA is under stringent control, as are the ribosomal and transfer RNAs (tRNAs), and also like tRNAs, is processed by RNase P. It has a long lifetime, as do other RNAs involved in protein synthesis¹³. More direct is the observation that the earliest event following 4.5S RNA depletion is the appearance of initiation-incompetent ribosomes. Three hours after the removal of IPTG, which is roughly three generations, initiation activity is 65% that of control. The longevity of the molecule (longer than that of 5S RNA, ref. 13) suggests that this number of cell divisions must occur before dilution and turnover cause the cellular level to drop below that required for proper function. Also, there is no detectable alteration in the protein makeup of cells, as judged by two dimensional electrophoresis, until after initiation becomes impaired (D.B.B., T. A. Phillips, F. C. Neidhardt & M.J.F., in preparation). Patterns of labelled proteins remain constant until initiation activity drops below the 65% level. Decreases below this point are accompanied by an increase in expression of several proteins. There is no apparent accumulation of incomplete peptides. Taken together, these correlations support the view that impairment of translational initiation occurs as a direct consequence of the loss of 4.5S RNA.

The data suggest that the 4.5S RNA participates in creating or maintaining the viability of a ribosomal subunit. That ribosomal particles appear normal, as judged by the somewhat insensitive sedimentation assay, suggests that a late stage of assembly or maturation is affected. It is unlikely that ribosomal RNA maturation is affected by a lack of 4.5S RNA; if assembled, ribosomes containing unprocessed RNA would show altered sedimentation. Evidence that the 4.5S RNA is not a structural component comes from the fact that active ribosomal particles can be assembled *in vitro* from individual RNAs and proteins without 4.5S RNA being present^{14,15}. Of course, this finding does not preclude the possibility that 4.5S RNA might have a catalytic role in assembly *in vivo*. Thus, 4.5S RNA may not be required for initial assembly or maturation, but for the development or maintenance of a functional form as subunits recycle into each initiation phase. Interestingly, the 4.5S RNA contains exposed sequences similar to the Shine-Dalgarno sequence of mRNA. Two such overlapping domains occur in the apex region of the predicted hairpin-like secondary structure and this region is particularly susceptible to nuclease (ref. 10 and B. Sneath, M.J.F. & J. N. Vournakis, in preparation). Perhaps the 4.5S RNA binds to the 3' end of the 16S RNA and assists in mRNA binding or prevents the degradation of the 3' end of the 16S RNA. In the absence of 4.5S RNA, the 3' end of the 16S RNA could be degraded, resulting in an inability to participate in initiation.

Experiments are under way to determine which subunit contains the defect responsible for translational inactivation, which event in the initiation process is affected by this defect, and whether conditions can be found in which 4.5S RNA will restore activity to depleted extracts. Results from these experiments will allow the models discussed above to be tested.

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Erratum

A cell surface molecule distributed in a dorsoventral gradient in the perinatal rat retina

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In this letter the wrong Fig. 4 was used. This is the correct Fig. 4 and its legend, which is as published.

Fig. 4 Biochemical characterization of the JONES antigen using TLC/immunoblot analysis. *a*, The JONES antigen partitions into the aqueous phase of a Folch extract and is detected as a single band on immunoblots of lipids resolved by TLC. This band migrates to a position between that of G_{M2} and G_{M1} ganglioside standards run at the same time. (1) Whole lipid extract; (2) organic phase; (3) aqueous phase; (4) G_{D1a} ; (5) G_{M1} ; (6) G_{M2} . 1-3, Immunostaining of the nitrocellulose blot of a TLC plate; 4-6, resorcinol staining of purified standards run at the same time. *b*, The epitope recognized by JONES antibody contains sialic acid as it is removed by treatment with neuraminidase (+) but not buffer (-).

Methods. P4 rat retinas were homogenized in chloroform/methanol (2:1) to give a crude lipid extract³⁶. This was partitioned into organic and aqueous phases by 0.1 M KCl⁹. Gangliosides and other highly polar lipids (including some phospholipids) partition into the aqueous phase, whereas most neutral glycolipids and some less polar gangliosides partition into the organic phase. The sialic acid content of the fractions was determined by the resorcinol method³⁷. Samples containing 0.3-0.4 μ g sialic acid were applied to a precoated HPTLC plate (Merck, silica gel 60, 0.2 mm thick) and developed with chloroform/methanol/0.25% aqueous $CaCl_2$ (60:40:10). Chromatograms were dried, wetted with isopropanol/water (2:1) and blotted onto nitrocellulose³⁸. The nitrocellulose blot was blocked in 5% normal goat serum and incubated with JONES antibody for 4 h. After washing, the blot was incubated in peroxidase-conjugated goat anti-mouse IgG (H + L) for 2-4 h and washed. Bound antibody was visualized by incubation with 3,3'-diaminobenzidine. Ganglioside standards were run on parallel lanes as controls and were visualized by resorcinol staining³⁷. For neuraminidase digestion the aqueous phase retinal lipids were dried under N_2 and resuspended in 0.1 M sodium acetate, pH 5.5, with or without neuraminidase (Sigma type X from *Clostridium perfringens*) at 8 mU per μ g sialic acid. Incubations were carried out for 15 h at 37°C. Digested lipids were extracted into chloroform:methanol, 2:1, dried under N_2 and resuspended in a small volume of chloroform:methanol, 2:1. Samples were run and analysed by TLC/immunoblot as described above.