tive of the P_{T7} strain (P_{T7}-ΔRBS) lacking a ribosome binding site in front of *lacZ*. Though untranslated, the *lacZ* gene is still efficiently transcribed in this strain because the T7 RNA polymerase is insensitive to polarity[14]. The transcript pattern was faint in this case, showing that the *lacZ* mRNA is unstable in the absence of translation; however, it was strongly enhanced in the presence of overproduced DEAD-box proteins, indicating a marked stabilization (Fig. 4*b*). We conclude that DEAD-box proteins stimulate the β-galactosidase expression from P_{T7} by stabilizing the corresponding transcripts.

The T7 RNA polymerase runs ~eightfold faster than the *E. coli* polymerase or ribosomes over the *lacZ* gene, creating a large ribosome-free mRNA region behind itself[16]. In particular, by the time of the first ribosome binding, a much longer stretch of naked RNA has been synthesized by the T7 than by the *E. coli* RNA polymerase. Now, mRNAs that are partially devoid of ribosomes are often unstable in bacteria[4], especially if their 5′ region is ribosome free[17,18]. We hypothesized that P_{T7} transcripts are particularly unstable because of this unique mode of synthesis, and that DEAD-box proteins stabilize them by protecting their naked regions. Were this view correct, then those *E. coli* RNA polymerase transcripts that are unstable because of an inefficient translation should also be stabilized by DEAD-box proteins. The following observations support this view.

The P_{lac} transcript pattern, and hence stability, are unaffected by DEAD-box proteins (Fig. 4*c*). Two point mutations were used to depress the translation of this transcript. One (*713*) hits the Shine–Dalgarno element (SD), whereas the other (*701*) creates a secondary structure sequestering this element. We reported previously[15] that, within the genuine *lacZ* gene, these mutations depress translation without affecting transcription; moreover, as a consequence of the translation–stability relationship, they destabilize the transcripts, most of which now decay before reaching full length. Consistently, the mutations weakened the P_{lac} transcript pattern, especially in the higher molecular mass range. The overproduction of the DeaD product reversed this effect, restoring a pattern more like that of the wild-type P_{lac} (Fig. 4*c*). Hence, DEAD-box proteins stabilize P_{lac} transcripts lacking a functional SD. Genes naturally lacking such an element are not uncommon in *E. coli*, and for one of them (*malT*[19]) we checked that overexpressed DEAD-box proteins strongly stabilize the corresponding mRNA (not shown).

Like many components of the protein-synthesizing machinery, the product of the *thrS* gene, threonyl-tRNA synthetase, binds its own mRNA and represses its translation[20,21]. As for most similar cases, including ribosomal protein mRNAs[4], the repression destabilizes the *thrS* transcript (M. Springer, personal communication). Compared with control, cells overproducing the DeaD or SrmB products accumulated the *thrS* transcript (Fig. 4*d*). By monitoring the transcript decay on northern blots after rifampicin addition, we showed that this accumulation stems from a marked stabilization (Fig. 4*d*). Interestingly, mutations have been characterized that prevent repressor binding, resulting in a constitutively high *thrS* translation ('O^c' mutations[21]). In a strain carrying such mutation, the level of the *thrS* transcript, and hence its stability, were not increased by DEAD-box proteins (not shown).

The decay of most *E. coli* mRNAs, including the *lacZ*[15] and *thrS* (O. Yarchuk and M.D., unpublished results) transcripts, is controlled, presumably directly[22], by RNase E. Conceivably, overexpressed DEAD-box proteins hinder RNase E action by binding directly to its cleavage sites or by unwinding the surrounding mRNA structure[23]. Our results also show that the stability of certain mRNAs requires either a tight synchronization between transcription and translation, or the overproduction in the same cell of RNA-binding proteins such as DEAD-box helicases. In several expression systems, the gene of interest, borne on a multicopy plasmid, is transcribed in *E. coli* by the T7 RNA polymerase[24,25]. We speculate that for some target genes the effectiveness of these systems depends on the presence of cellular DEAD-box proteins, and can in turn be improved by their overproduction. □

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ERRATUM

Reverse occlusion leads to a precise restoration of orientation preference maps in visual cortex

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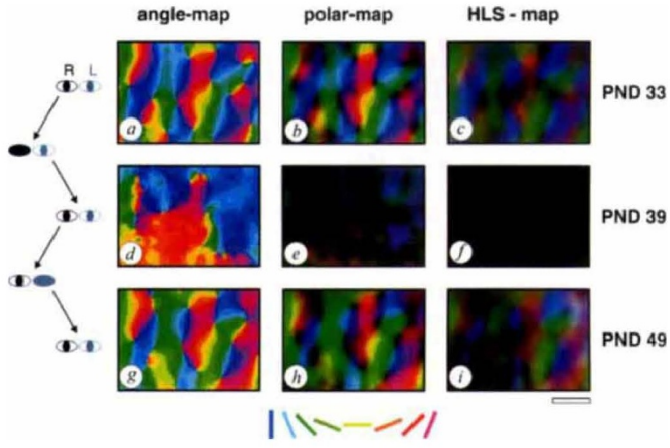

As a result of a printing error, the colour code for the angle maps, polar maps and HLS maps in Fig. 2 was omitted. The correct figure is shown here, with the coloured bars at the bottom indicating how orientation preference is translated into colour. □