Modulation of eukaryotic release factors activity by eIF3j

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Translation termination has long been considered the shortest stage of protein biosynthesis, in which only two protein factors, eRF1 and eRF3, are involved. It turned out that in addition to the two main proteins, additional proteins can take part in translation termination. One such protein is human eIF3j. eIF3j is known as the labile subunit of eukaryotic translation initiation factor eIF3. The yeast homolog of this protein, HCR1, has been reported to participate in controlling the translation termination and stop codon readthrough. Also, it was shown that HCR1 functions in the recycling of the 60S ribosomal subunit in vivo. We revealed role of human eIF3j in translation termination using a reconstituted mammalian in vitro translation system. We showed that eIF3j stimulates peptidyl-tRNA hydrolysis induced by a complex of release factors eRF1-eRF3. Using toe-printing assay, we determined that eIF3j improves the efficiency of stop codon recognition by release factors. Experiments on the binding of eIF3j with eRF1, eRF3 and ribosomal complexes confirmed its direct involvement in translation termination. Moreover, we found that eIF3j could interact with eRF3 in solution. Thus, we have shown that the human translation initiation factor eIF3j, like its yeast homologue, is involved in the regulation of translation termination.

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