Raising the blockade

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At crucial points in the metabolism of all organisms, a protein with the unwieldy name of Translation Elongation Factor P (EF-P, for short) takes center stage. What it actually does during protein synthesis has only now been elucidated – by researchers at Ludwig-Maximilians-Universität in Munich.

The research group led by Kirsten Jung, Professor of Microbiology at LMU, actually focused on how bacteria cope with stress, for example how the receptor meolecule CadC monitors the acidity in the environment and alerts the cell to take countermeasures to protect itself. However, one day Kirsten Jung found herself asking questions about protein synthesis, the core biosynthetic process that makes all metabolism possible. This arose because she discovered that, in the absence of Elongation Factor P, the cell doesn't make enough CadC to carry out its job effectively.

Recovering from a stall

How then does EF-P regulate protein synthesis in general, and, in particular, the synthesis of CadC? In collaboration with Daniel Wilson's group at LMU's Gene Center, Jung's team has now teased out the mechanism. Molecular machines called ribosomes translate the genetic blueprints for proteins into the correct sequence of amino acids as they move along a messenger RNA molecule. However, when the blueprint calls for the addition of several successive proline amino acids onto the growing protein chain, the ribosomes grind to a halt. It turns out that EF-P is required to get these stalled ribosomes going again. Moreover, the

factor not only fulfills this function in bacteria and in archaea, but also in the cells of eukaryotic organisms, which have their own versions of EF-P.

Jung and Wilson, who also cooperate within the context of the "Center for integrated Protein Science Munich", an Excellence Cluster, believe that the translation stop imposed by a short run of prolines provides a means of adjusting protein copy numbers in response to changing conditions. In bacteria, a functionally diverse set of around 100 proteins is known to contain such proline-rich motifs. This suggests that the stalling phenomenon indeed has a more general regulatory role, and may even provide a target for new antibiotics. Indeed, Daniel Wilson's group recently discovered the last enzyme in the EF-P modification pathway: For its rescue activity, the factor must be modified by other enzymes – which are not found in humans.

Provided by Ludwig Maximilian University of Munich

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