Biochimica et Biophysica Acta 1827 (2013) 60

Contents lists available at SciVerse ScienceDirect

Biochimica et Biophysica Acta

journal homepage: www.elsevier.com/locate/bbabio

Corrigendum

Corrigendum to "The ζ subunit of the α-proteobacterial F_1F_O-ATP synthase in *Paracoccus denitrificans*: A novel control mechanism of the central rotor" [Biochim. Biophys. Acta 1817S (2012) S27–S28]

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The abstract which originally appeared should be replaced with the following:

The ζ subunit [1] is a novel inhibitory element of the *Paracoccus denitrificans* F_1 (PdF_1)-ATPase and related α-proteobacteria, which works as a noncompetitive inhibitor of the sulfite-activated PdF_1-ATPase, with an IC_{50} of 220 nM when reconstituted into a PdF_1 construct lacking the endogenous ζ and ε subunits. In contrast, the ε subunit of the PdF_1-ATPase was unable to inhibit the sulfite-activated PdF_1-ATPase, thus confirming that ζ rather than ε plays a central role in the inhibition of the α-proteobacterial F_1-ATPase. The ζ subunit inhibitory region was determined by limited proteolysis and functional inhibitory reconstitution experiments. Further analyses showed that during PdF_1-ATPase activation by sulfite, the ζ subunit exposes its N- and C-termini. This contrasts with the ε subunit, which buries its C-terminal domain in the F_1 domain. The ζ subunit can also be reversibly cross-linked with rotor (γ, ε) and stator (α, β) subunits of the PdF_1-ATPase, as was previously also found for the IF_1 inhibitor of the mitochondrial F_1-ATPase [2]. The present data thus suggest that the mechanism of regulation by the ζ subunit involves the control of the intrinsic gyration of the central stalk of the α-proteobacterial F_1F_O-ATPase, similar to the inhibitory mechanisms by the ε and IF_1 subunits in eubacterial and mitochondrial adenosine triphosphate (ATP) synthases. The three-dimensional structure of the *P. denitrificans* ζ subunit determined by nuclear magnetic resonance (NMR) spectroscopy has a four-helix bundle architecture (PDB ID: 2LL0), which is different from the structures of the aforementioned ε and IF_1 subunits. The present data thus indicate that a novel mechanism, with a new structural basis, contributes to regulation of the intrinsic gyration of the bacterial F_1F_O nanomotor.

References

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[2] F. Minauro-Sanmiguel, C. Bravo, J.J. Garcia, J. Bioenerg. Biomembr. 34 (2002) 433–443.

PII of original article: S0005-2728(12)00214-9.
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0005-2728/$ – see front matter
http://dx.doi.org/10.1016/j.bbabio.2012.09.010