CORRECTIONS

Modulation of *Mycobacterium tuberculosis* DnaA protein–adenine nucleotide interactions by acidic phospholipids

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Figures 5 and 6 in the above paper contained errors. The correct Figures and their legends appear below:

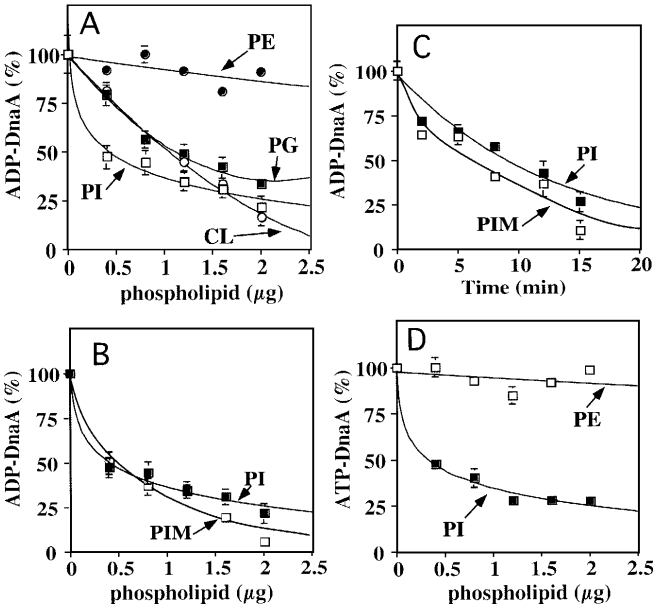

Figure 5 Dissociation of adenine nucleotides by phospholipids

M. tuberculosis DnaA protein was incubated either with [^{14}C]ADP (732 pmol) or with [α-^{32}P]ATP (100 pmol) at 0 °C for 15 min in 100 μl of buffer E. At the end of incubation, different concentrations of phospholipid vesicles were added, reactions were continued at 37 °C and incubation proceeded for 15 min. Samples were collected on membranes and the amount of radioactivity retained was determined. (**A**) Effect of commercial phospholipids on ADP dissociation (□, PI; ■, PG; ○, CL; ●, PE). (**B**) Effect of *M. tuberculosis* PIM on ADP dissociation (□, PIM; ■, PI). (**C**) Time course of ADP dissociation. The DnaA–ADP complexes prepared as described above were incubated with either PI or PIM (2.0 μg) for different periods of time. Samples were processed as described above (□, PIM; ■, PI). (**D**) Effect of phospholipids on ATP dissociation (□, PE; ■, PI).

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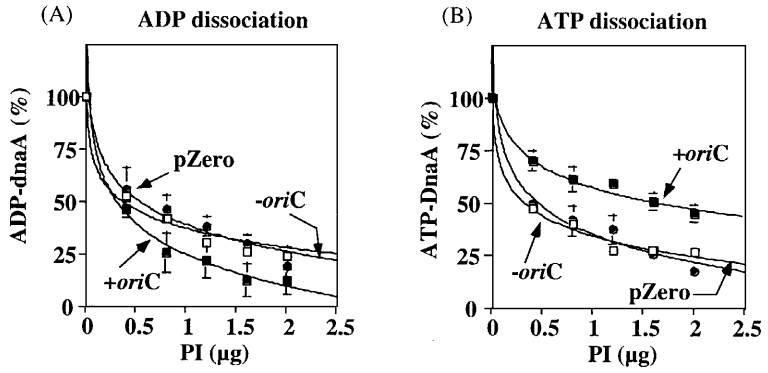

Figure 6 Effect of *oriC* on ADP or ATP dissociation

(**A**) ADP dissociation. DnaA (10 pmol) was incubated with [^{14}C]ADP (732 pmol) at 0 °C for 15 min in a reaction volume of 100 μl. At the end of incubation, either *oriC* (0.05 μg), pZErO (0.05 μg) or nothing (control) and indicated concentrations of PI were added. Samples were then transferred to 37 °C and incubation continued for 15 min before processing as described above. (**B**) ATP dissociation. DnaA (1 pmol) and [α-^{32}P]ATP (100 pmol) were incubated in a reaction volume of 100 μl before the addition of DNA and PI or PIM. Samples were processed as described above. Essentially similar results were obtained with other ratios of ATP–DnaA protein.

Crystal structure of human dehydroepiandrosterone sulphotransferase in complex with substrate

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There were several errors in the first paragraph in the right-hand column on p. 169 of the above paper. Accordingly

for:

In the DHEA-ST structure, O-17 of DHEA has one very weak hydrogen bond to the carbonyl oxygen of residue Tyr-238 (3.4 Å). Similar binding of O-17 to that seen with EST would be impossible, due to loop displacements. O-17 in the EST structure has a single weak hydrogen bond (3.0 Å) to the side chain of Asn-86; the most closely corresponding residue in the DHEA-ST structure is Met-16, whose side chain cannot form a hydrogen bond. Although the hydrogen bonding is weak, it is clear that the orientation of DHEA at the O-17 end is governed by the orientation of the surrounding loop structures.

read:

The O-17 in the EST structure has a single weak hydrogen bond (3.0 Å) to the side chain of Asn-86, whose closest corresponding residue in the DHEA-ST structure is Met-16, whose side chain cannot form a hydrogen bond. It is clear that the orientation of DHEA at the O-17 end is governed by the orientation of the surrounding loop structures, via Van der Waals interactions.