CORRIGENDUM

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Insights into the pathogenesis of *Mycoplasma pneumoniae* (Review)

JUN HE, MIHUA LIU, ZHUFENG YE, TIANPING TAN, XINGHUI LIU, XIAOXING YOU, YANHUA ZENG and YIMOU WU

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Following the publication of this review, an interested reader alerted us to the fact that a couple of figures had been reproduced from a pair of previous publications without proper acknowledgement of the original source/authors. Figs. 2 and 3, as featured in our review, had originally appeared (with only minor modifications) as Figs. 2 and 4, respectively, in the following articles: Rottem S: Interaction of mycoplasmas with host cells. Physiol Rev 83: 417-32, 2003; and Pilo P, Vilei EM, Peterhans E, Bonvin-Klotz L, Stoffel MH, Dobbelaere D and Frey J: A metabolic enzyme as a primary virulence factor of *Mycoplasma mycoides* subsp. *mycoides* small colony. J Bacteriol 187: 6824-6831, 2005.

Permission to publish these figures was sought retrospectively from the publishers [The American Physiological Society (Fig. 2) and The American Society of Microbiology (Fig 3)]. Subsequently, Figs. 2 and 3 are reprinted in this Corrigendum, together with strap-lines that properly acknowledge the source articles.

In addition, we omitted to explain that the glycerol metabolism causing injury in host cells refers to *Mycoplasma mycoides* subsp. *mycoides*. Consequently, this information has also been inserted into the corrected legend for Fig. 3 (opposite), with a pair of supporting references.

We profusely apologize to the authors of the previous publications (Dr Joachim Frey and colleagues) for our having failed to include a proper acknowledgement of their figure, or to have credited their work appropriately.

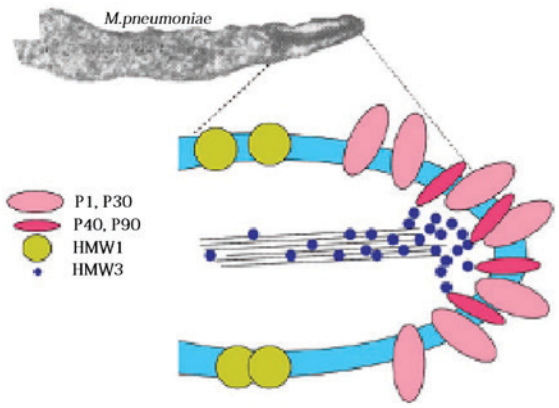

Figure 2. Structure of the *Microplasma pneumoniae* adhesion protein. The adhesion protein of *M. pneumoniae* includes key proteins P1 and P30, adhesion factor-associated proteins, P40, P90, HMW 1 and HMW 3. These components jointly constitute a characteristic high-electron-density 'adhesion protein complex'. This complex stabilizes the integrity of the *M. pneumoniae* apical organ structure by forming a cytoskeleton, anchoring the protein P1 to the cytoskeleton of the adhesive organs, and allowing the P1 proteins accumulating in the adhesion cell organs to adhere. **The Figure was taken from Rottem S, 2003 (1)**.

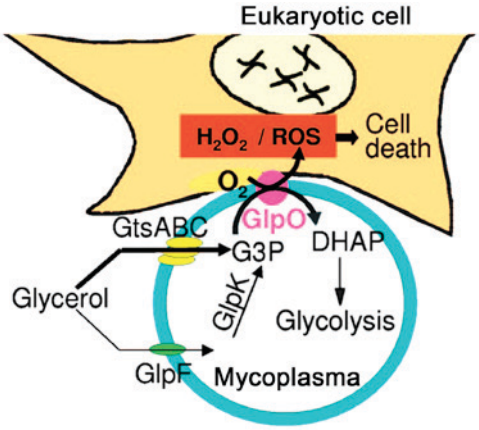

Figure 3. Toxic injury of *Mycoplasma pneumoniae* in glycerol. Following adherence of *M. pneumoniae* onto the surface of eukaryotic cells, cytoskeletal rearrangement occurs, and H_2O_2 and ROS are synthesized and released by *M. pneumoniae* in glycerol, which lead to oxidative stress and subsequent cell death. **Note that glycerol metabolism, attributed as a virulence factor in *Mycoplasma* species, was initially discovered in *Mycoplasma mycoides* subsp. *mycoides*, and subsequently found to be present in *M. pneumoniae* (2,3). The Figure was taken from Pilo *et al*, 2005 (2).** ROS, reactive oxygen species.

References

1. Rottem S: Interaction of mycoplasmas with host cells. Physiol Rev 83: 417-32, 2003.
2. Pilo P, Vilei EM, Peterhans E, Bonvin-Klotz L, Stoffel MH, Dobbelaere D and Frey J: A metabolic enzyme as a primary virulence factor of *Mycoplasma mycoides* subsp. *mycoides* small colony. J Bacteriol 187: 6824-6831, 2005.
3. Hames C, Halbedel S, Hoppert M, Frey J and Stükle J: Glycerol metabolism is essential for cytotoxicity of *Mycoplasma pneumoniae*. J Bacteriol 191: 747-753, 2009.