CORRIGENDUM

doi:10.1038/nature14624

Corrigendum: Fatty acid carbon is essential for dNTP synthesis in endothelial cells

Sandra Schoors, Ulrike Bruning, Rindert Missiaen, Karla C. S. Queiroz, Gitta Borgers, Ilaria Elia, Annalisa Zecchin, Anna Rita Cantelmo, Stefan Christen, Jermaine Goveia, Ward Heggermont, Lucica Goddë, Stefan Vinckier, Paul P. Van Veldhoven, Guy Eelen, Luc Schoonjans, Holger Gerhardt, Mieke Dewerchin, Myriam Baes, Katrien De Bock, Bart Ghesquière, Sophia Y. Lunt, Sarah-Maria Fendt & Peter Carmeliet

Nature **520**, 192–197 (2015); doi:10.1038/nature14362

We thank our colleagues from the metabolism community (Emile Van Schaftingen and Guido Bommer, University of Louvain, Belgium, and Frans Schuit, University of Leuven, Belgium), who alerted us to a possible confusion arising from our Article. In particular, the statement in the abstract that “Isotope labelling studies in control endothelial cells showed that fatty acid carbons substantially replenished the Krebs cycle” and similar phrases later in the text could be misunderstood as implying anaplerosis. By no means did we intend to suggest that the Krebs cycle is replenished by the net contribution of fatty acid carbons in the traditional sense of anaplerosis. Indeed, even though labelled acetyl-CoA from fatty acids enters the Krebs cycle and labels oxaloacetate, we did not want to imply net formation of oxaloacetate from acetyl-CoA, derived from fatty acids. Rather, our data suggest that under conditions of adequate availability of anaplerotic substrates (glucose, glutamine), fatty acid oxidation is important to produce sufficient amounts of dNTPs. Fatty acids provide acetyl-CoA, which helps to sustain the TCA cycle and dNTP synthesis for proliferation in conjunction with an anaplerotic substrate. Why the anaplerotic nutrients glucose and glutamine do not sustain sufficient aspartate and nucleotide synthesis in the CPT1A-silenced endothelial cells is an intriguing but outstanding question.