Turner N, Hariharan K, TidAng J, Frangioudakis G, Beale SM, Wright LE, Zeng XY, Leslie SJ, Li J-Y, Kraegen EW, Cooney GJ, Ye J-M. Enhancement of muscle mitochondrial oxidative capacity and alterations in insulin action are lipid species dependent: potent tissue-specific effects of medium-chain fatty acids. *Diabetes* 2009;58:2547–2554

In the print version of the article listed above, in Table 2, the units for the muscle and liver triglycerides are incorrect. The correct units are μmol/g. The online version reflects these changes.

Basu R, Basu A, Grudzien M, Jung P, Jacobson P, Johnson M, Singh R, Sarr M, Rizza RA. Liver is the site of splanchnic cortisol production in obese nondiabetic humans. *Diabetes* 2009;58:39–45

The authors of the article listed above have erroneously used the intravenous ICG infusion rate (in μg/min) and the arterial-portal ICG concentration difference to calculate portal venous blood flow. Because the liver is the sole source of clearance of ICG, the ICG infusion rate is appropriate for calculation of splanchnic blood flow but not for determination of portal blood flow. The authors confirm that this error does not influence the main conclusion of the article that the viscera do not produce cortisol. Based on previous data, one can assume portal blood flow to be ~80–90% of splanchnic blood flow, allowing extrahepatic splanchnic exchange to be estimated using assumed rather than measured portal blood flow. Such an estimate gives a result similar to that reported in the study. Moreover, the authors' conclusion is in fact independent of portal blood flow because it depends solely on the lack of dilution of D4 cortisol (i.e., lack of change in molar enrichment) between arterial and portal venous blood for either total cortisol or D3 cortisol.