Eur J Vasc Endovasc Surg (2015) 49, 753–753

CORRESPONDENCE

Re: 'Protective Effect of Focal Adhesion Kinase against Skeletal Muscle Reperfusion Injury after Acute Limb Ischemia'

This interesting article demonstrates that glycolytic muscles are more prone to deleterious effects induced by ischemia reperfusion (IR) than oxidative muscles.[1]

We agree that the higher mitochondrial content in slow oxidative fibers may buffer IR related calcium overload and fuel enough energy to protect muscle cells.

Further, we propose that another protective mechanism might be in play. Indeed, oxidative stress has been shown to be a key factor involved in IR related mitochondrial dysfunction,[2] and oxidative muscles are characterized by enhanced antioxidant defenses, compared with fast glycolytic type muscle fibers.[3] Thus, reduced antioxidant defenses probably explain glycolytic muscle sensitivity to IR. Accordingly, enhancing glycolytic muscle antioxidant defenses with an antioxidant, totally protected the glycolytic muscle against IR.[3] Thus, besides higher mitochondrial content, enhanced oxidative muscle protection against IR relies on the increased antioxidant pool.

These data are important since they not only support the fact that susceptibility to IR induced injury differs between organs depending on their metabolic phenotype, but also suggest the need to adapt therapeutic strategies to the specific antioxidant power of the target organ to be protected.

REFERENCES

1 Flück M, von Allmen RS, Ferrié C, Tevaearai H, Dick F. Protective effect of focal adhesion kinase against skeletal muscle reperfusion injury after acute limb ischemia. *Eur J Vasc Endovasc Surg* 2015;**49**:306–13.

2 Lejay A, Choquet P, Thaveau F, Singh F, Schlagowski A, Charles AL, et al. A new murine model of sustainable and durable chronic critical limb ischemia fairly mimicking human pathology. *Eur J Vasc Endovasc Surg* 2015;**49**(2):205–12.

3 Guilbert A-S, Charles AL, Guillot M, Kindo M, Lejay A, Meyer A, et al. Muscles susceptibility to ischemia reperfusion injuries depends on metabolic phenotype. *Acta Physiologica* 2015 [abstract, in press].

A.-L. Charles
Fédération de Médecine Translationelle, Equipe d'Accueil 3072, Mitochondrie, Stress oxydant et Protection Musculaire, Université de Strasbourg, Strasbourg, France

A. Lejay
Fédération de Médecine Translationelle, Equipe d'Accueil 3072, Mitochondrie, Stress oxydant et Protection Musculaire, Université de Strasbourg, Strasbourg, France
Department of Vascular Surgery and Kidney Transplantation, University Hospital, Strasbourg, France

J. Zoll
Fédération de Médecine Translationelle, Equipe d'Accueil 3072, Mitochondrie, Stress oxydant et Protection Musculaire, Université de Strasbourg, Strasbourg, France
Department of Physiology and Functional Explorations, University Hospital, Strasbourg, France

N. Chakfe
Fédération de Médecine Translationelle, Equipe d'Accueil 3072, Mitochondrie, Stress oxydant et Protection Musculaire, Université de Strasbourg, Strasbourg, France
Department of Vascular Surgery and Kidney Transplantation, University Hospital, Strasbourg, France

B. Geny*
Fédération de Médecine Translationelle, Equipe d'Accueil 3072, Mitochondrie, Stress oxydant et Protection Musculaire, Université de Strasbourg, Strasbourg, France
Department of Physiology and Functional Explorations, University Hospital, Strasbourg, France

*Corresponding author. Department of Physiology and Functional Explorations, University Hospital, B.P. 426, 67091, Strasbourg Cedex, France.
Email-address: bernard.geny@chru-strasbourg.fr (B. Geny)

Available online 30 March 2015

http://dx.doi.org/10.1016/j.ejvs.2015.02.016