

Modulation of mitochondrial capacity and angiogenesis by red wine polyphenols via estrogen receptor, NADPH oxidase and nitric oxide synthase pathways.

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Résumé en anglais	Red wine polyphenolic compounds (RWPC) are reported to exert vasculoprotective properties on endothelial cells, involving nitric oxide (NO) release via a redox-sensitive pathway. This NO release involves the activation of the estrogen receptor- α (ER α). Paradoxical effects of a RWPC treatment occur in a rat model of post-ischemic neovascularization, where a low-dose is pro-angiogenic while a higher dose is anti-angiogenic. NO and ER α are key regulators of mitochondrial capacity, and angiogenesis is a highly energetic process associated with mitochondrial biogenesis. However, whether RWPC induces changes in mitochondrial capacity has never been addressed. We investigated the effects of RWPC at low (10(-4)g/l, LCP) and high concentration (10(-2)g/l, HCP) in human endothelial cells. Mitochondrial respiration, expression of mitochondrial biogenesis factors and mitochondrial DNA content were assessed using oxygraphy and quantitative PCR respectively. In vitro capillary formation using ECM gel(®) was also performed. Treatment with LCP increased mitochondrial respiration, with a maximal effect achieved at 48h. LCP also increased expression of several mitochondrial biogenesis factors and mitochondrial DNA content. In contrast, HCP did not affect these parameters. Furthermore, LCP modulated both mitochondrial capacity and angiogenesis through mechanisms sensitive to ER, NADPH oxidase and NO-synthase inhibitors. Finally, the inhibition of mitochondrial protein synthesis abolished the pro-angiogenic capacity of LCP. These results suggest a possible association between the modulation of mitochondrial capacity by LCP and its pro-angiogenic activity. These data provide evidence for a role of mitochondria in the regulation of angiogenesis by RWPC.
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