Red wine polyphenol compounds favor neovascularisation through estrogen receptor α-independent mechanism in mice.

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Résumé en anglais	Red wine polyphenol compounds (RWPC) exert paradoxical effects depending on the dose on post-ischemic neovascularisation. Low dose RWPC (0.2 mg/kg/day) is pro-angiogenic, whereas high dose (20 mg/kg/day) is anti-angiogenic. We recently reported that the endothelial effect of RWPC is mediated through the activation of a redox-sensitive pathway, mitochondrial biogenesis and the activation of α isoform of the estrogen receptor (ERα). Here, we investigated the implication of ERα on angiogenic properties of RWPC. Using ovariectomized mice lacking ERα treated with high dose of RWPC after hindlimb ischemia, we examined blood flow reperfusion, vascular density, nitric oxide (NO) production, expression and activation of proteins involved in angiogenic process and muscle energy sensing network. As expected, high dose of RWPC treatment reduced both blood flow and vascular density in muscles of mice expressing ERα. These effects were associated with reduced NO production resulting from diminished activity of eNOS. In the absence of RWPC, ERα deficient mice showed a reduced neo-vascularisation associated with a decreased NO production. Surprisingly in mice lacking ERα, high dose of RWPC increased blood flow and capillary density in conjunction with increased NO pathway and production as well as VEGF expression. Of particular interest is the activation of Sirt-1, AMPKα and PGC-1α/β axis in ischemic hindlimb from both strains. Altogether, the results highlight a pro-angiogenic property of RWPC via an ERα-independent mechanism that is associated with an up-regulation of energy sensing network. This study brings a corner stone of a novel pathway for RWPC to correct cardiovascular diseases associated with failed neovascularisation.
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Liens
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