# Cyclooxygenase-2 preserves flow-mediated remodelling in old obese Zucker rat mesenteric arteries



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| Titre | Cyclooxygenase-2 preserves flow-mediated remodelling in old obese Zucker rat mesenteric arteries |
| Type de publication | Article de revue |
| Auteur | Belin de Chantemèle, E.-J. [1], Vessieres, Emilie [2], Guihot, Anne-Laure [3], Toutain, Bertrand [4], Loufrani, Laurent [5], Henrion, Daniel [6] |
| Editeur | Oxford University Press |
| Type | Article scientifique dans une revue à comité de lecture |
| Année | 2010 |
| Langue | Anglais |
| Date | 2010 |
| Numéro | 3 |
| Pagination | 516 - 25 |
| Volume | 86 |
| Titre de la revue | Cardiovascular Research |
| ISSN | 0008-6363 |
| Mots-clés | Acetylcholine [7], Age Factors [8], Animals [9], Blood Pressure [10], Cyclooxygenase 2 [11], Cyclooxygenase Inhibitors [12], Enzyme Inhibitors [13], Hypertrophy [14], Indomethacin [15] |

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| Résumé en anglais | AIMS: Resistance arteries have a key role in the control of local blood flow and pressure, and chronic increases in blood flow induce endothelium-dependent outward hypertrophic remodelling. The incidence of metabolic syndrome increases with age, and the combination of these two risk factors impairs endothelium integrity, in part through an inflammatory process. We hypothesized that cyclooxygenase-2 (COX2) would affect remodelling in 12-month-old obese rats compared with young rats.<br>METHODS AND RESULTS: Mesenteric arteries of obese and lean Zucker rats were alternatively ligated to generate high flow (HF) in the median artery. After 21 days, arteries were isolated for in vitro analysis. After 21 days, outward hypertrophic remodelling occurred in HF arteries in obese (498 +/- 20 vs. 443 +/- 18 mum intraluminal diameter in normal flow (NF) arteries, $P < 0.01$), but not in lean rats (454 +/- 17 vs. 432 +/- 14, NS; n = 12 per group). Endothelium-dependent (acetylcholine)-mediated relaxation (AMR) was lower in obese than in lean rats. AMR was reduced by NO-synthase blockade in all groups, and eNOS expression was higher in HF than in NF arteries without difference between lean and obese rats. Indomethacin further reduced AMR in HF arteries from obese rats only. Obesity increased COX2 immunostaining in mesenteric arteries. Acute COX2 inhibition (NS398) significantly reduced AMR in HF arteries from obese rats only, suggesting production of vasodilator prostanoid(s). In obese rats chronically treated with the COX2 inhibitor celecoxib, outward remodelling did not occur in HF arteries and AMR was improved without reaching the level found in lean rats.<br>CONCLUSION: COX2 preserved in part flow-mediated arterial remodelling in old obese rats. Nevertheless, this effect was not sufficient to keep endothelium-dependent relaxation to the level obtained in lean rats. |
| URL de la notice | http://okina.univ-angers.fr/publications/ua8209 [16] |
| DOI | 10.1093/cvr/cvp411 [17] |
| Lien vers le document | http://dx.doi.org/10.1093/cvr/cvp411 [17] |
| Titre abrégé | Cardiovasc Res |

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[17] http://dx.doi.org/10.1093/cvr/cvp411