<table>
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<td>Nejad, A; Lee, SS; Vanhoutte, PM; Leung, SWS</td>
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ANTIRETROVIRAL TREATMENT CAN AFFECT THE RELEASE OF NO AND EDCF,
BUT NOT EDH IN RAT ARTERIES

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Despite improving clinical outcomes, highly active antiretroviral therapy (HAART) is an
independent potential risk factor for cardiovascular diseases. Currently the recommended
HAART regimen commonly comprises a protease inhibitor (PI) with ritonavir (RTV)-boosting or
a non-nucleoside reverse transcriptase inhibitor (NNRTI), and two nucleoside reverse
transcriptase inhibitors. The present study examined whether or not boosted lopinavir (LPVr), a
PI, and/or efavirenz (EFV), an NNRTI, affect the regulation of vascular tone in ways leading to
cardiovascular complications. Male Sprague Dawley rats were treated with LPVr (80/20 mg/kg),
RTV (20 mg/kg), EFV (160 mg/kg) or the vehicle methylcellulose (0.5%) once daily by oral
gavage two weeks after they were fed normal or high fat diet. After eight weeks of antiretroviral
treatments, superior mesenteric arteries were isolated and suspended in organ chamber for the
study of vascular reactivity. Endothelium-dependent relaxations to acetylcholine were not
different between rats fed with normal and high fat diet. None of the antiretroviral treatments
affected acetylcholine-induced relaxation in rats fed with normal diet. However, RTV
significantly reduced acetylcholine-induced relaxation in rats fed with high fat diet. This
reduction was not observed in arteries incubated with indomethacin. By contrast, LPVr enhanced
acetylcholine-induced nitric oxide (NO)-mediated relaxation in the high fat group. Endothelium-
dependent hyperpolarization-mediated relaxations were not affected by these antiretroviral
treatments. As a result, chronic treatment with RTV may cause cyclooxygenase-dependent
contractions in rats with high fat. While RTV impairs vascular relaxation, its combination with
lopinavir does not cause vascular dysfunction, probably because lopinavir causes activation of
NO signalling pathway.