Caveolin-1-mediated post-traditional regulation of inducible nitric oxide synthase in human colon carcinoma cells

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Reactive oxygen species are now widely recognized as important players contributing both to cell homeostasis and the development of disease. In this respect nitric oxide (NO) is no exception. The discussion here will center on regulation of the inducible form of nitric oxide synthase (iNOS) for two reasons. First, only iNOS produces micromolar NO concentrations, amounts that are high by comparison with the picomolar to nanomolar concentrations resulting from Ca2+-controlled NO production by endothelial eNOS or neuronal nNOS. Second, iNOS is not constitutively expressed in cells and regulation of this isoenzyme, in contrast to endothelial eNOS or neuronal nNOS, is widely considered to occur at the transcriptional level only. In particular, we were interested in the possibility that caveolin-1, a protein that functions as a tumor suppressor in colon carcinoma cells (Bender et al., 2002; this issue), might regulate iNOS activity. Our results provide evidence for the existence of a post-tr