Supplementary figure 1. Cytoprotective effect of nucleosides and nucleotides in hypoxia-reoxygenation injury. LLC-PK1 cells were subjected to 20 hour-long OGD in presence of 300 μM adenosine (Ado), cytidine (Cyd), guanosine (Guo), thymidine (TdR), uridine (Urd), adenine (Ade), cytosine (Cyt), guanine (Gua), thymine (Thy) and uracil (Ura) then re-supplemented with glucose and oxygen for 24 hours. The viability was measured by the MTT assay and expressed as percent values of controls (Data are shown as mean ± SD values, *$p<0.05$ compared to OGD).

Table 1. **The LOPAC molecules showing the highest protective effect in the hypoxia screens**. Hypoxia-reoxygenation injury was induced in LLC-PK1 cells and the LOPAC library was screened for protective effect in various set-ups. From each screen the ten highest-ranking cytoprotective compounds are shown with the respective activity in the other assays and the known biological function. (I.) In the pre-treatment screen (PRE) the compounds (50 μM) were applied prior to the OGD, (II.) in the post-treatment screen (POST) the cells received the compounds after the OGD. Adenosine pretreatment (30 μM) was combined (III.) with compounds pre-treatment (ADE PRE) and (IV.) with the application of the test compounds following the hypoxia (ADE POST). The shown cytoprotective effect represents the increase in viability compared to the OGD in PRE and POST screens or compared to the adenosine-treated cells subjected to OGD (ADE) in ADE PRE and ADE POST screens. (The viability values of CTL, OGD and ADE groups were 100%, 40% and 70%, respectively.)

Abbreviations: SERCA: sarco/endoplasmic reticulum Ca^{2+}-ATPase, DAMP: 4-Diphenylacetoxy-N-methylpiperidine methiodide, AEBSF: 4-(2-Aminoethyl) benzenesulfonyl fluoride hydrochloride, R(+)-6-Bromo-APB hydrobromide: (+)-6-Chloro-7,8-dihydroxy-3-allyl-1-phenyl-2,3,4,5-tetrahydro-1H-3-benzazepine hydrobromide

Supplementary table 1. **Protective effect of PARP inhibitors and compounds interfering with adenosine uptake or metabolism in the hypoxia screens**. Hypoxia-reoxygenation injury was induced in LLC-PK1 cells and the LOPAC library was screened for protective effect in various set-ups. From each screen PARP inhibitors and adenosine uptake compounds are shown with the respective activity in the other assays and the known biological function. (I.) In the pre-treatment screen (PRE) the compounds (50 μM) were applied prior to the OGD, (II.) in the post-treatment screen (POST) the cells received the compounds after the OGD. Adenosine pretreatment (30 μM) was combined (III.) with compounds pre-treatment (ADE PRE) and (IV.) with the application of the test compounds following the hypoxia (ADE POST). The shown cytoprotective effect represents the viability increase compared to the OGD in PRE and POST screens or compared to the adenosine-treated cells subjected to OGD (ADE) in ADE PRE and ADE POST screens. (The viability values of CTL, OGD and ADE groups were 100%, 40% and 70%, respectively.)