Ischemia-reperfusion syndromes of the heart and brain are the leading cause of death and long-term disability worldwide. Development of effective treatments for myocardial infarction, stroke, cardiac arrest and their sequelae requires preclinical models that replicate specific features of ischemia-reperfusion. The complexities of intact animals, including the integrated function of organ systems, autonomic innervation and endocrine factors, often preclude detailed study of specific components of ischemia-reperfusion injury cascades. Ischemia represents the interruption of metabolic fuel and oxygen delivery to support cellular oxidative metabolism; reintroduction of oxygen upon reperfusion of ischemic tissue triggers oxidative stress which initiates the reperfusion injury cascade culminating in injury and death of cells and tissues. Thus, cultured cells subjected to hypoxia, fuel deprivation and reoxygenation replicate the cardinal features of ischemia-reperfusion, while accommodating interventions such as siRNA suppression of specific genes and pharmacological activation or inhibition of signaling cascades that are not feasible in more complex preparations, especially intact animals. This chapter describes an in vitro OGD-reoxygenation cell culture model, an excellent preparation to examine the cellular mechanisms mediating ischemia-reperfusion injury and/or cytoprotection [1)].

1)

Ryou MG, Mallet RT. An In Vitro Oxygen-Glucose Deprivation Model for Studying Ischemia-Reperfusion Injury of Neuronal Cells. Methods Mol Biol. 2018;1717:229-235. doi: 10.1007/978-1-4939-7526-6_18. Review. PubMed PMID: 29468596.