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Sugammadex for reducing the cerebral injury during reperfusion after ischemia

(after Ozbilgin *et al.*, 2016)

The insufficient blood supply resulting in hypoxia in patients with ischemia is caused by atherosclerosis (accumulation of cholesterol-rich plaques in the arteries) and is restored by reperfusion causing tissue damages called reperfusion injury. Cerebral ischemia/reperfusion (I/R) may cause permanent brain damage and behavioral dysfunction. The study of Ozbilgin *et al.* provided experimental evidence that Sugammadex has neuroprotective effect in transient global cerebrial ischemia [1].

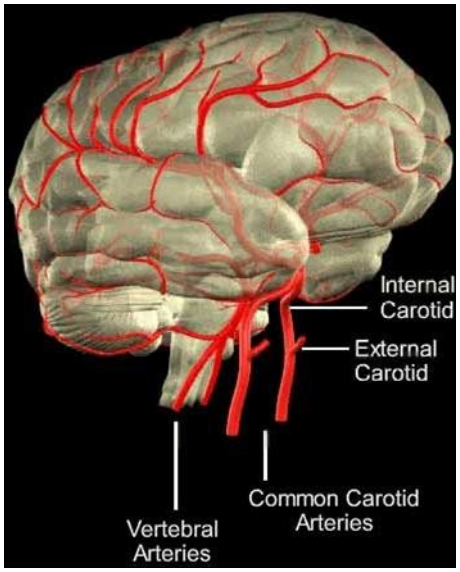

Fig. 1 Arteries of the brain (www.strokecenter.org)

Formerly the neuroprotective effects of cholesterol-extracting cyclodextrins was described [2,3]. Methyl-BCD was found to protect the hippocampal areas from the effect of anoxia [4]. Also, HPBCD showed neuroprotective features [2,5] explained by changing membrane cholesterol concentration, which has an influence on several cell trafficking processes, for instance, on transporter-mediated glutamate release from nerve terminals [3].

On the other hand, the neuroprotective and neurotoxic effects of several anesthetics used in surgery have been studied in global cerebral I/R models [6]. The effects of Sugammadex, a

recently introduced agent for the reversal of neuromuscular blockage caused by rocuronium-type anesthetics have not been explored as yet. Normally, Sugammadex cannot get through the blood–brain barrier (BBB) because of the large size and hydrophilicity of its molecules. In neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease, traumatic brain or spinal cord injury, however, the permeability of BBB can be changed and Sugammadex may pass. In cell cultures, Sugammadex (75 μg/mL for 24 h) was found to cause apoptosis/necrosis and neuron death attributed to changing cholesterol homeostasis [7]. Sugammadex decreased membrane-associated, cytosolic and mitochondrial cholesterol levels in these *in vitro* experiments [7] although it is well known that gamma-CD and its derivatives show only very slight interaction with cholesterol compared to the beta-CD derivatives [8]. The neuronal damage by Sugammadex could be prevented by preloading the neurons with soluble cholesterol (it is not clear how cholesterol is solublilized, most probably by random methylated BCD, which makes the situation more complicated). The elevated cellular cholesterol levels decrease the neuronal death caused by prion peptides [9] or oxidative stress [10].

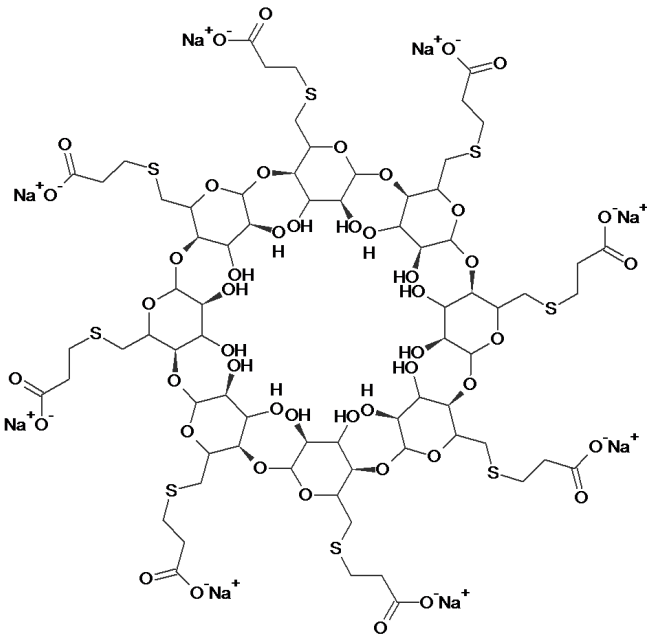

Fig.2 Structural formula of Sugammadex

The pioneering studies of Ozbilgin *et al.* on the effect of Sugammadex were performed *in vivo*, in a global cerebral hypoxia model in rats. The injury was induced by bilateral common carotid occlusion. Complete interruption of blood flow for 10 min was followed by reperfusion for 24 h. After the ischemic period the rats were treated with Sugammadex (16 mg/kg and 100 mg/kg, respectively) intravenously in the 5th minute of reperfusion. The same surgical procedure was performed in the control group without Sugammadex treatment. After 24 hours, the neurological condition of the rats was assessed with a scoring system including the evaluation of spontaneous activity, symmetrical movement, forepaw outstretching, limp placement, climbing, etc. To detect DNA fragmentation in cell nuclei, the TUNEL (terminal deoxynucleotidyl transferase-mediated dUTP nick end-labeling) reaction was applied. Immunohistochemistry was also performed for active caspase-3. In the treatment groups, in both the hippocampus

and the parietal cortex, lower results of the TUNEL and caspase assays were obtained than in the control group. The high dose of Sugammadex was found to be more neuroprotective. This result is in accordance with the total score obtained from behavioral neurological evaluation tests in rats.

Contrary to the *in vitro* results the *in vivo* studies of Ozbilgin *et al.* showed that Sugammadex had a protective effect against ischemia/reperfusion injury of brain opening new therapeutic strategies in carotid artery surgery.

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Éva Fenyvesi

CycloLab Cyclodextrin R&D Laboratory, Ltd.

Budapest, HUNGARY

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Depression, Oral bioavailability, Molecular modeling studies

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Carboxymethyl cellulose, Cross-linkage, Epichlorohydrin, High swelling, Sodium salicylate

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RAMEB, Complexation, Sepiolite

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Hp-β-CD, Sucrose, Mannitol, Polymorph composition, FTIR-ATR, Calorimetry (DSC), X-ray diffraction (XRD), Protein formulation

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Rhamnolipid, Bovine serum albumin, Alginate sodium, Models of lipopolysaccharide, proteins and polysaccharides, Accelerated solvent extraction, Earthworm accumulation

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cyclodextrin, Heptakis(2,3-di-O-methyl-6-sulfato)-β-cyclodextrin, Heptakis(2,3-di-O-acetyl-6-sulfato)-β-cyclodextrin, Fluoroquinolones: ofloxacin, gatifloxacin and lomefloxacin, Enantioselectivity, ^{1}H NMR, ROESY

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Silica based monoliths, Organic polymer based monoliths, Organic-silica based hybrid monoliths, Preparation strategy

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Glassy carbon, Cyclic voltammetry, Electro-oxidation, Electrochemical nano-sensor

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Capillary electrophoresis, Sodium dodecyl sulphate, γ-Cyclodextrin, Chiral separation, Impurities

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e-mail: cyclolab@cyclolab.hu