Sugammadex, causes neuronal apoptosis in primary cultures, with a less action with rocuronium or vecuronium are in the medium of culture

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Sugammadex, a γ-cyclodextrin that encapsulates selectively steroidal neuromuscular blocking agents, such as rocuronium or vecuronium, has changed the face of clinical neuromuscular pharmacology. Sugammadex allows a rapid reversal of muscle paralysis. Sugammadex appears to be safe and well tolerated. Its blood-brain barrier penetration is poor (<3% in rats), and thus no relevant central nervous toxicity is expected. However the blood brain barrier permeability can be altered under different conditions (i.e., neurodegenerative diseases, trauma, ischemia, infections, or immature nervous system). Here we show that clinically relevant sugammadex concentrations cause apoptotic/necrotic neuron death in primary cultures. Studies on the underlying mechanism revealed that sugammadex-induced activation of mitochondria-dependent apoptosis associates with depletion of neuronal cholesterol levels. Furthermore SUG increase CytC, AIF, Smac/Diablo and CASP-3 protein expression in neurons in culture, related with mitochondrial activation pathway and increase in oxidative stress. On the other hand rocuronium or vecuronium produced less damage caused by sugammadex alone.

Biography
Alma Casasempere is an MD, doing her PhD in the department of the Physiology. She is in the 3rd year and next will defense her thesis in the Hospital of La Fe.

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