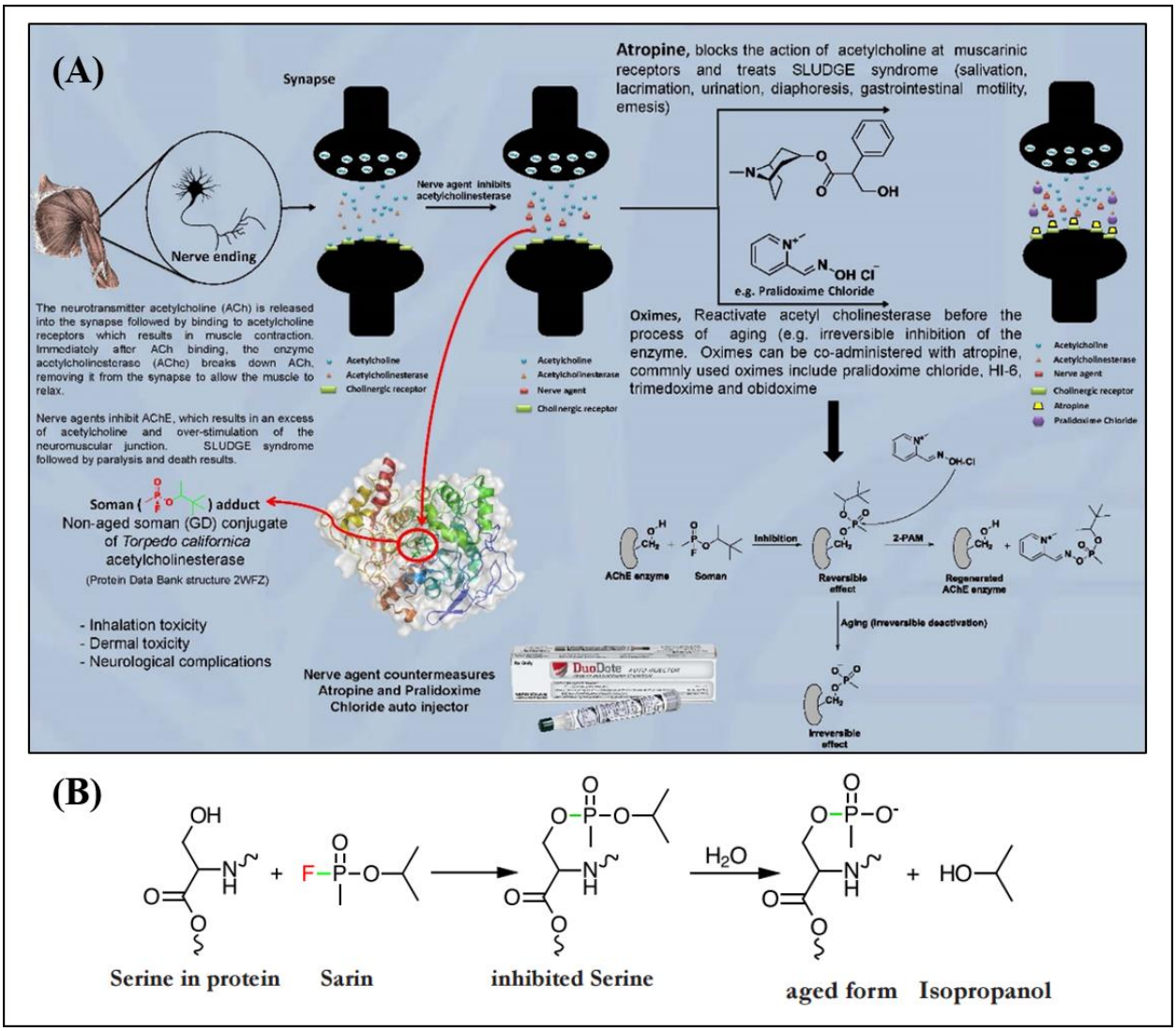
(A)
Synapse
Nerve ending
Nerve agent inhibits acetylcholinesterase
Atropine, blocks the action of acetylcholine at muscarinic receptors and treats SLUDGE syndrome (salivation, lacrimation, urination, diaphoresis, gastrointestinal motility, emesis)
e.g. Pralidoxime Chloride
Oximes, Reactivate acetyl cholinesterase before the process of aging (e.g. irreversible inhibition of the enzyme. Oximes can be co-administered with atropine, commnly used oximes include pralidoxime chloride, HI-6, trimedoxime and obidoxime
The neurotransmitter acetylcholine (ACh) is released into the synapse followed by binding to acetylcholine receptors which results in muscle contraction. Immediately after ACh binding, the enzyme acetylcholinesterase (AChe) breaks down ACh, removing it from the synapse to allow the muscle to relax.
Nerve agents inhibit AChE, which results in an excess of acetylcholine and over-stimulation of the neuromuscular junction. SLUDGE syndrome followed by paralysis and death results.
Acetylcholine
Acetylcholinesterase
Cholinergic receptor
Nerve agent
Atropine
Pralidoxime Chloride
Soman ( ) adduct
Non-aged soman (GD) conjugate of Torpedo californica acetylcholinesterase
(Protein Data Bank structure 2WFZ)
- Inhalation toxicity
- Dermal toxicity
- Neurological complications
Nerve agent countermeasures Atropine and Pralidoxime Chloride auto injector
DuoDote
AChE enzyme
Soman
Inhibition
Reversible effect
2-PAM
Regenerated AChE enzyme
Aging (irreversible deactivation)
Irreversible effect
(B)
Serine in protein
Sarin
inhibited Serine
aged form
Isopropanol