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A short review on VX agent

The modern-day assassinator

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Abstract: VX nerve agent is a modern-day chemical warfare agent which falls under the category of Nerve agent. It is derived from organophosphorus (OPC). These OPC are used to produce insecticides and from them nerve agents are made. VX is one of the most lethal agents because of its chemical properties that allow VX to stay in the environment for a long time. It is considered to be more poisonous that the nerve gas which was used in Wolrd war -II by Germany. VX agent inhibits acetylcholine hydrolysis which leads to muscular spasms and causes death.

IndexTerms – AchE,Ach ,VX, OPC

I. INTRODUCTION

VX is a human made chemical warfare agent. It falls under the category of nerve agent. Nerve agent are most toxic and chemical modern warfare agents. Just like insecticide kills an insect within seconds the same way nerve agent kills people within seconds of use. VX is an Acetylcholinesterase enzyme inhibitor which blocks Acetylcholine hydrolysis in the PNS (Peripheral nervous system) and CNS (Central nervous system). This leads to muscle contraction and leads to death

History -

VX Nerve agent made headlines when half -brother of North Korean leader Kim Jong-un, Kim Jong -Nam was assassinated at Kula Lumpur airport in Malayasia.[1] VX is a human made classified chemical warfare weapon which was used during Iran-Iraq war in 1980. VX is a type of Organophosphate. These organophosphorus compounds are considerably used worldwide as pesticides which have potent health hazards in humans. In,1936 German scientist Dr. Gerhard Schrader who was working on pesticide while mixing some chemicals discovered the effects of organophosphorus on humans.[2] After this the era of lethal Nerve gas production began. VX is the most abundant chemical warfare agent in the chemical arsenal of Soviet Union, USA and Great Britian

Organophosphorus compounds and it's derivatives-

Organophosphorus compounds (OPCs) are derived from phosphoric acids and its derivatives. OPC have a peculiar property that they contain at least one carbon-phosphorus bond. This pentavalent phosphorus containing organic compounds are used as insecticides and pesticides the substituents attached to the phosphorus of these esters of phosphoric acids play a vital role in toxicity.[3,4] Organophosphorus pesticides are thiols, amides, or esters of phosphonic, phosphinic, phosphoric, or thiophosphoric acids which bear two extra side chains of cyanide, phenoxy or thiocyanate. Neurotoxic CWA, also known as nerve gases or nerve agents, are OPCs with high toxicity towards mammals, particularly topically or by inhalation. Their toxicity is higher than that of OPCs used as insecticides. While most OPC's used as insecticides are phosphonothioates with the P=S bond, nerve gases are usually phosphonofluoridates and S-substituted phosphonothioates
[3,4]

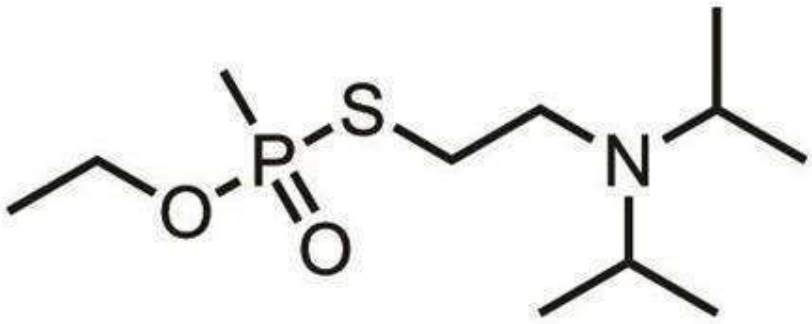
The nerve agents which are derived from OPC are classified into four types – [3]

1. G-series agents
2. V-series agent
3. GV- series agent
4. Novichok series agent

VX agent

VX agent was discovered in Great Britain in 1950. VX is a type of OPC and is one of the most lethal modern chemical warfare agents Several properties of VX make it a special. VX is a well-known irreversible cholinesterase inhibitor. Nerve agents usually absorbed through the nasal route or topical route and effectively act as inhibitor of enzyme acetylcholinesterase (AchE). Acetylcholinesterase is an enzyme that catalyses breakdown of acetylcholine and some of choline esterase that act as

neurotransmitter[5,6]. AchE is found at neurotransmitter junction and cholinergic brain synapses. The main activity of AchE is it serves to terminate synaptic transmission and breakdown of acetylcholine. The inhibition of AchE leads to accumulation of acetylcholine in the synaptic cleft and causes paralysis. [6]



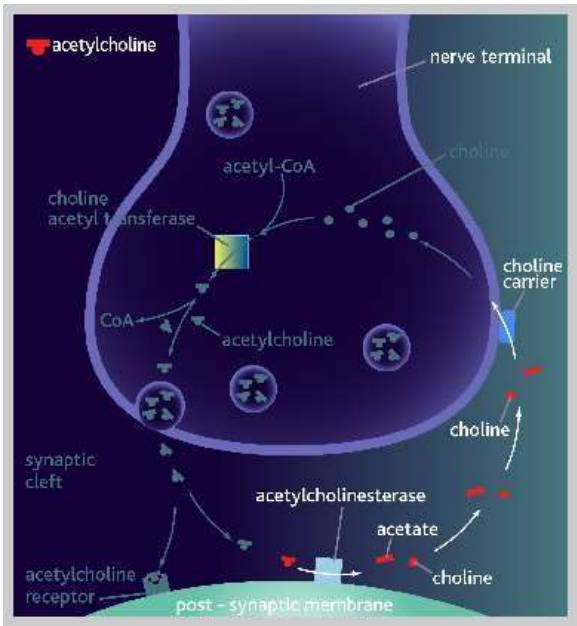
Chemical structure of VX nerve agent
(http://www.chm.bris.ac.uk/webprojects2006/Macgee/Web%20Project/nerve_gas.htm)

Chemical and Physical properties of VX agent –
Molecular weight – 267.3 g mol^{-}
Odor – None (odorless)
Boiling point – 298 ℃
Melting point - -51℃
Liquid density – 1.009 g ml^{-}
Vapor density – 9.3
Volatility – 10.5 mg m^{-3}

Nomenclature-
It was discovered by Ranaji Ghoshn in the 1950's . The"V" in the VX stands for Venomous Nerve agent X

Mode of action of VX agent –
AchE catalyses the breakdown of acetylcholine. AchE is found in synapse between nerve cell and muscle cells. It breaks acetylcholine into two components acetic acid and Choline. Ach initiates secretion of different body fluids and contraction of skeletal muscles Following transmission of an impulse across the synapse by the release of acetylcholine, acetylcholinesterase is released into the synaptic cleft.[7,8] Inhibitors like VX agent function by binding to the substrate binding site of the enzyme. Inhibition leads to involuntary muscle contraction and excessive secretion of fluids as the acetylcholine accumulates in the PNS and CNS, this leads to respiratory collapse which in turn is the cause of death. The binding of VX to AchE is irreversible, this is the main reason behind VX being dangerous. Once the VX agent is absorbed via eyes, skin or inhaled the phosphorous atom of VX covalently binds to serine hydroxyl group in the active site of AchE, where VX agent acts as a competitive inhibitor and thus inactivates acetylcholine hydrolysis.[9,10] This results into muscle spasms and unvoluntary muscle contractions. Ultimately constant stimulated contractions lead to respiratory failure. VX agent has low volatility and this being the reason it stays into the environment for a longer time and causes a lethal effect on the overall environment.[10]

Ach is a potent neurotransmitter, which is released from the neurons. After the release they diffuse across the synaptic cleft and combine with nicotinic acetylcholine receptor molecules in the membrane of the postsynaptic cell. The interaction of acetylcholine with nicotinic acetylcholine receptor produces large transient increase in the permeability of the membrane to specialized ions resulting in signal transduction for nerve impulse.



Mechanism of action of AchE. If the AchE is inhibited, then spasms occur.

Symptoms of VX agent exposure –[10]

1. Blurred Vision
2. Difficulty while breathing, along with vomiting , drooling , urinating , defecating
3. Muscles Spasm
4. Obnoxious feeling
5. Slurred Speech
6. Areflexia

In long term or chronic effects, the victim might go into coma or die Antidote-
Atropine which itself is a potent toxin, acts as an anti-dote against Nerve agent VX by removing it from the enzyme. [10] Conclusion –

VX was developed for use in warfare, and studies focused on its acute toxic effects. The current review also focuses on its history, mode of action, lethality, antidote in brief.Like the structurally related OP pesticides, VX is highly toxic to other animals

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