Identical kinetics of human erythrocyte
and muscle acetylcholinesterase with
respect to carbamate pre-treatment,
residual activity upon soman
challenge and spontaneous
reactivation after withdrawal of the
inhibitors.

The efficacy of oxime treatment in
soman poisoning is limited due to
rapid aging of inhibited
acetylcholinesterase (AChE).
Pre-treatment with carbamates was
shown to improve antidotal treatment
substantially. Recently, by using a
dynamically working in vitro model
with real-time determination of
membrane-bound AChE activity, we
were able to demonstrate that
pre-inhibition of human erythrocyte
AChE with pyridostigmine or
physostigmine resulted in a markedly
higher residual AChE activity after
inhibition by soman or paraoxon than
in the absence of reversible inhibitors.
The purpose of the present study was
to compare the effect of carbamate
pre-treatment and soman challenge
with human erythrocyte and muscle
homogenate AChE. Both enzyme
sources were immobilized on particle
filters which were perfused with
acetylthiocholine, Ellman's reagent
and phosphate buffer. AChE activity
was continuously analyzed in a
flow-through detector. Pre-inhibition of
AChE with pyridostigmine or
physostigmine resulted in a
concentration-dependent increase in
carbamylation, residual activity after
soman inhibition and fraction of
decarbamylation AChE after
discontinuation of the inhibitors
without differences between human
erythrocyte and muscle AChE. This data support the view that human erythrocyte AChE is an adequate surrogate marker for synaptic AChE in OP poisoning.

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