Whether Lack of Measurement of Erythrocyte Cholinesterase or Acetyl Cholinesterase Activity Providing us Misleading Information in Organophosphorus Exposure?

Sheemona Chowdhary¹ · Dibyajyoti Banerjee¹

Received: 2 December 2017 / Accepted: 24 January 2018 / Published online: 6 February 2018
© Association of Clinical Biochemists of India 2018

It is an established fact that erythrocytes contain acetylcholinesterase and it is considered as gold standard biomarker for organophosphorous poisoning [1]. Plasma contains high concentration of Butyrylcholinesterase that cleaves acetylthiocholine significantly [2]. However, many researchers use acetylthiocholine in plasma or serum and claim for the estimation of acetylcholinesterase activity. Some authors address the specificity issue by performing the experiments in presence and absence of a specific butyrylcholinesterase inhibitor and others publish results even without taking any care to report for specific acetylcholinesterase activity [3, 4]. It appears that due to misconception of facts plasma cholinesterase activity and plasma acetylcholinesterase activity is used synonymous by authors even at recent times. We feel that such misconception should be corrected because of the following reasons.

Firstly, plasma contains less acetylcholinesterase activity (or true cholinesterase activity) compared to pseudocholinesterase [5]. Secondly, erythrocyte cholinesterase which is mostly acetylcholinesterase and not pseudocholinesterase is the gold standard biomarker in organophosphorus pesticide poisoning [1]. Further, pseudocholinesterase has a high extent of inter as well as intra individual variations [6]. So, understanding the effect of

organophosphorus pesticide exposure by estimation of plasma cholinesterase is not advisable. Perhaps for such reasons some researchers have not seen cholinesterase inhibition in plasma (claimed as plasma acetylcholinesterase) even after high dose (10 times the maximal theoretical daily intake) of chronic pesticide exposure [7]. There is an earlier report that in chronic organophosphorus pesticide exposure erythrocyte acetylcholinesterase significantly decreases [8]. In this type of study if erythrocyte cholinesterase activity is measured we feel that inhibition will be detected. In spite of this well known facts people are not refraining from reporting plasma acetylcholinesterase activity in the context of organophosphorous exposure. So, we feel to demonstrate erythrocyte cholinesterase and plasma cholinesterase to point out the difference.

We have done a staining for cholinesterase activity in hemolysate and plasma using acetylthiocholine by procedure given in detail in supplementary. We have observed that even with dilute hemolysate acetylthiocholine produces better enzyme activity bands compared to undiluted plasma (Fig. 1). Therefore, we request the researchers to estimate hemolysate cholinesterase activity using acetylthiocholine to report for acetylcholinesterase activity.

Electronic supplementary material The online version of this article (<https://doi.org/10.1007/s12291-018-0737-y>) contains supplementary material, which is available to authorized users.

✉ Dibyajyoti Banerjee
dibyajyoti5200@yahoo.co.in

¹ Department of Experimental Medicine and Biotechnology,
Postgraduate Institute of Medical Education and Research,
Chandigarh 160012, India

Fig. 1 Bar diagram representation of band densities [mean $\pm$ SD ($n = 6$)] obtained after Native PAGE of hemolysate (1:11) and plasma using acetylthiocholine as a substrate. The staining for enzyme activity was performed using Karnovsky and Roots staining solution [9]. Corresponding representative gel pictures are shown below. * $p < 0.0001$

References

1. Chowdhary S, Bhattacharyya R, Banerjee D. Acute organophosphorus poisoning. *Clin Chim Acta*. 2014;431:66–76.

2. Masson P, Lockridge O. Butyrylcholinesterase for protection from organophosphorus poisons; catalytic complexities and hysteretic behavior. *Arch Biochem Biophys*. 2010;494:107–20.
3. Manu MS, Prashant V, Akila P, Suma MN, Basavanagowdappa H. A retrospective analysis of serial measurement of serum cholinesterase in acute poisoning with organophosphate compounds. *Toxicol Int*. 2012;19:255–9.
4. Alkalay A, Rabinovici G, Zimmerman G, Agarwal N, Kaufer D, Miller BL, et al. Plasma acetylcholinesterase activity correlates with intracerebral β -amyloid load. *Curr Alzheimer Res*. 2013;10:48–56.
5. Mendel B, Mundell DB, Rudney H. Studies on cholinesterase. Specific tests for true cholinesterase and pseudo-cholinesterase. *Biochem J*. 1943;37:473–6.
6. Brock A, Brock V. Factors affecting inter-individual variation in human plasma cholinesterase activity: body weight, height, sex, genetic polymorphism and age. *Arch Environ Contam Toxicol*. 1993;24:93–9.
7. Velmurugan G, Ramprasath T, Swaminathan K, Mithieux G, Rajendhran J, Dhivakar M, et al. Gut microbial degradation of organophosphate insecticides induces glucose intolerance via gluconeogenesis. *Genome Biol*. 2017;18:8.
8. Ranjbar A, Pasalar P, Abdollahi M. Induction of oxidative stress and acetylcholinesterase inhibition in organophosphorus pesticide manufacturing workers. *Hum Exp Toxicol*. 2002;21:179–82.
9. Karnovsky MJ, Roots L. A “direct coloring” thiocholine method for cholinesterases. *J Histochem Cytochem*. 1964;12:219–21.