Establishing the Cholinesterase Levels in Jordan: An Investigation and Monitoring Model for Pesticides Outbreak

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Abstract

Background Aims: To establish and validate the method used to analyze cholinesterase in blood and to establish a baseline level among Jordanians living in a heavy agricultural activity area and as well as among those living in an urban non-agricultural area.

Materials and Methods: Modified Ellman procedure was used to analyze 851 and 1033 blood samples from heavy agricultural activity and urban area for cholinesterase activity in erythrocytes and Plasma.

Results: sample collected from heavy agricultural activity showed low cholinesterase level average 1037U/L ± 279 and 23 U/gHb ± 7.9 compared with urban area average 1616 U/L ± 662 and 31 U/gHb ± 13 for plasma and erythrocyte respectively. The levels in workers living in heavy agriculture activity showed 52% inhibition in plasma and 41.1% in erythrocyte when compared to the established normal levels that were observed in urban area.

Conclusions: Clinicians using cholinesterase for clinical diagnosis and management should be aware that baseline levels are different according to the residence of their patients.

Keywords: Cholinesterase, Pesticides, Toxicity, Outbreak, Monitoring, Programs.

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Introduction

In the Environment Protection Agency (EPA) 2011 report on pesticides industry sales and usage; 5.2 billion pounds is the amount of pesticides which was used in 2006-2007,[1] and on a yearly basis three million pesticide poisoning cases occur with a consequential 220,000 deaths according to the world Health organization (WHO).[2,3] The overall deaths and the increase in consumption indicate how widely pesticides are being used in agriculture.

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The concomitant poisoning would be due to the malpractice in handling of these pesticides, accidents and suicide attempts.[3] In Jordan the quantities of pesticide used in 1999 were about 1347 tons,[4] however, the number of fatal pesticides poisoning cases has increased over a 20 year period from 23.5 to 35 cases per year, while in other study OP was responsible for 308 deaths over 13 years.[5,6]

Cholinesterase (CHE) belongs to the group of hydrolases splitting the ester bond and they hydrolyze choline esters more rapidly than any other group of enzymes, where they have a critical role in regulating nerve transmissions in the central and the peripheral nervous system.[7,8] Inhibition of CHE in turn leads to the accumulation of acetylcholine (ACH); the neurotransmitter at all ganglia in the autonomic nervous system and at many synapses in the brain, skeletal muscular junctions at some post ganglionic nerve endings of the sympathetic nervous system and the adrenal medulla.[9] There are two types of the cholinesterase depending on their affinity to the natural substrates – choline esters; the erythrocyte/ true cholinesterase or Acetylcholinesterase (ACHE) and plasma/ Pseudocholinesterase or Butyrylcholinesterase (BCHE). ACHE has a higher affinity to acetylcholine than BCHE and shows higher activity in erythrocytes. As it happens, muscular and neuronal ACHE is not accessible for measurement; instead the surrogate biomarker Erythrocytes-ACHE can be easily obtained because of the structural and functional similarities. BCHE on the other hand is synthesized in the liver and is present in plasma and the pancreas.[7,10,11]

Evidently biological monitoring of toxicity biomarkers like cholinesterase is crucial and a trusted module for any comprehensive assessment of exposure. Such monitoring programs are used to test ACHE and BCHE activity at an annual baseline before OP/CB spray season, and follow up tests are conducted throughout the spray season to evaluate CHE inhibition relative to baseline levels.[12]

To our knowledge there is no biological monitoring or baseline data available in the Middle East region, including Jordan for such exposure levels of OP or CB that might be useful for assessment of the relevance to human toxicity, especially those related to nerve gases outbreaks.

With this research we aimed to establish and compare baseline levels for the Jordanians in an agricultural area (Alghor) and non-agricultural urban area (Alsalt). A vulnerable group of workers who handled pesticides in spraying also tested to identify if significant inhibition in their CHE levels have occurred and compared them with other residents of Alghor area in which their CHE baseline levels were determined earlier in the first stage.

MATERIALS AND METHODS

Subject recruitment Following ethical clearance, a cohort study was conducted. The sample of the study was a group of healthy adult volunteers from both genders who were selected by simple random sampling.

Study area and population Blood specimens were collected in EDTA tubes from two different areas for the measurement, 12 fields from each area were incorporated, a total of 851 samples were collected from an agricultural area known as Alghor area (Jordan Valley) and 1033 sample collected from Alsalt

area (Al-Balqa'a Governorate). Tubes were transferred to the University of Jordan toxicology laboratory in an Ice box. For baseline establishment samples were taken From Alsalt fields from 841 adult males and 181 adult females, while 542 male's samples and 309 female's samples were drawn from Alghor area.

Reagents Acetylthiocholine iodide and 5, 5'-Dithio-2-nitrobenzoic acid (DTNB) were obtained from sigma (St. Louis, MO, USA). All other reagents were obtained from Merck (Darmstadt, Germany). DTNB Stock solution, Buffers (pH 7 and 8, 0.13M) and Zijlstra solution (containing 200 mg potassium ferricyanide, 50 mg potassium cyanide, and 1000 mg sodium bicarbonate in 1000 ml of distilled water, pH 7.4) were prepared and used at the toxicology laboratory.

Apparatus and Method After collection and transfer to the toxicology laboratory, samples were centrifuged and plasma separated and kept at -20C until analysis. Total CHE activity in erythrocytes and plasma was determined following the method described in previous studies with some modifications;[13,14] Ellman procedure measures cholinesterase in erythrocytes(ACHE) and whole blood and doesn't centrifuge, instead quinidine sulphate is used to inhibit plasma cholinesterase. Whereas in our study, samples were centrifuged to separate erythrocytes from plasma and cholinesterase was measured in both erythrocytes (ACHE) and plasma (BCHE).

Statistical analysis Statistical analysis using SPSS version 11.5 was performed to analyze data collected from both geographical areas.

Table 1. ACHE and plasma BCHE activity baseline levels In Alsalt (non-contaminated area)

Alsalt Field	No. samples	Mean ACHE ± SD	Range ACHE	Mean BCHE ± SD	Range BCHE
All fields	1033	31±13	18 – 44	1616±662	954 – 2278
Males	841	31±13.7	18 – 45	1585±678	900 – 2260
Females	181	29±10.6	18 – 40	1570±583	980 – 2157

S: Alsalt area (AlBalqa'a Governorate) non contaminated area. F: Field

Table 2. ACHE and BCHE activity baseline levels In Alghor (contaminated area)

Alghor Field	No. samples	Mean ACHE ± SD	Range ACHE	Mean BCHE ± SD	Range BCHE
All fields	851	23.8±7.9	16 – 32	1037±279	758 – 1316
Males	542	23.7±7.7	16 – 31	1061±29	725 – 1306
Females	309	23.89±8.3	16 – 32	1075±254	820 – 1330

G: Alghor (Jordan valley district) contaminated area. F: Field

RESULTS

Baseline results Blood samples from the first stage were analyzed for BCHE and ACHE activity in 12 fields for both areas of study; Alghor area, a heavily contaminated area with pesticides, and Alsalt which is a non-agricultural area. Results showed a significant difference in BCHE and ACHE activities from both areas, the average of BCHE from all Alsalt fields was 1616 U/L±662 and Alghor BCHE was 1037 U/L±279 (35.8% lower than that of Alsalt fields), alternatively the average ACHE was found to be 31 U/gHb±13 from the fields of Alsalt and 23 U/gHb±7.9 from the fields of Alghor (25.8% lower than that of Alsalt fields). Results are shown in tables 1 and 2.

These results supported the fact that the contaminated area would have higher rates of CHE inhibition due to higher exposure levels to pesticides compared with other non agricultural areas that would have minimal exposure to that cholinesterase inhibitor pesticides.

Table 3. ACHE and plasma BCHE levels in 35 male pesticide workers in Alghor area

No. of exposed samples to pesticide	Age	ACHE level	BCHE level
35 sample	21-41	9-25	351-813
Average	28	14	496.6
SD	-	3.57	106.3
CV%	-	25.5	21.4

Vulnerable group results Samples were drawn from the most vulnerable workers with the worst agricultural practices; the workers known for handling and spraying pesticides in their daily work in Alghor area. They were tested for ACHE levels and BCHE. Their average ACHE level was 14 U/gHb±3.57 and BCHE level was 496 U/L±106.3 (table 3). Approximately, 52% inhibition in BCHE levels and 41.1% inhibition in ACHE have occurred in these individuals compared with baseline average values collected from all fields in Alghor area.

DISCUSSION

Cholinesterase contains in its active site a serine residue with a free hydroxyl group that covalently reacts with ACH, acetylating the serine while releasing the choline group. This serine residue, and in a matter of microseconds, is deacetylated by hydrolysis and is liberated to degrade a further ACH molecule. The same scenario is repeated when OPs in the oxon form (i.e. phosphate form) act in response with the serine residue, nonetheless the de-phosphorylation process is a great deal slower by hours and days than that of the deacetylation, therefore, the serine residue on the phosphorylated ACHE is not accessible to break down ACH, which is simply demonstrated by OP toxicity that results in ACHE inhibition and accumulation of ACH in the nervous system. It is according to the type of localization, peripheral and central, muscarinic or nicotinic symptoms are observed.[7,15] That's why therapeutic strategies are directed to antagonize overstimulation of

muscarinic receptors with atropine and to reactivate inhibited ACHE with oximes.[3,16]

The correlation involving depression in ACHE and BCHE activity and toxic symptoms have been described as being complex; depending on OP structure, the route of exposure and the nature of exposure,[17] and in common sense multiple exposures which cause a more gradual reduction in CHE activity are more endured than a single acute exposure.[18]

Both ACHE and BCHE inhibition are considered to be markers of early biologic effects related to OP/CB exposure,[19] and according to previous studies ACHE inhibition by OP causes clinical features due to the overstimulation of cholinergic synapses where it may reflect the levels of these anticholinergic toxicities in the parasympathetic system, neuromuscular junction and the central nervous system and in the early stages of exposure and recovery.[8,16,18] BCHE by contrast appears to not result in clinical features.

In populations based studies, farm workers showed inhibited levels of CHE activity compared with those not employed in agriculture,[8,12,18,19,20,21] while workers who mix and load or spray would face the highest risks because they handle OP/CB in their concentrated forms.[19] Chronic exposure to OP in these individuals may be monitored by measuring the activity of BCHE as an index of exposure but not ACHE, and may be used as a potential biomarker of exposure to OP.[20,22] If any of the biomarkers is used, fluctuations in CHE levels necessitate the need to establish a baseline CHE to determine if suppression has occurred.

Similarly our results showed a good connection with the level of agricultural activities and provided a setting for the times with the highest exposure levels; since these measurements were taken during the peak level of pesticides use in these areas. Alghor area, the northern part of Jordan Valley, has been described as unique in nature; being a terrain lower than the sea level (400 meters below the sea level) with elevated temperatures all around the year when compared with the temperate climate of the surrounding areas, it is also relatively narrow in width, and farms and residential areas are very close to each other, which makes it the key agricultural land in Jordan.[23] Contamination in Alghor area is partly due to the high temperatures leading to higher rates of pesticides evaporation. The lack of precautions taken during spraying and the malpractice with pesticides are two anticipated factors that explicate the higher exposure of farmers to pesticides as described in a previous study.[24] These two factors are also anticipating in the high pesticide exposure characterized by the levels of BCHE inhibited (average 1037 U/L±279) and ACHE (average 23.8 U/gHb±7.9) in the population occupying Alghor area when compared with the other terrain; Alsalt area which is a province in the highlands of AlBalqa'a Governorate, about 790 – 1100 meter above sea level and located northwest to Amman, the capital,[25] with minimal to non agricultural activities. Alsalt has shown clearly higher CHE levels with an average of 1616 U/L±662 in plasma and 31 U/gHb±13 in erythrocytes in contrast to Alghor area.

In the cross sectional comparison we have made for pesticide handlers in Alghor area, especially when testing workers who spray

pesticides, we have found that they had (496 U/L±106.3) or 52% inhibition in their BCHE levels and (14U/gHb±3.57) or 41.1% inhibition in their ACHE levels when compared to the average levels in the baseline of Alghor area.

It is apparent though; that the interpretation of ACHE and BCHE monitoring results is problematic and confounded by numerous factors such as the interindividual and intraindividual variations which sequentially interfere with the overall enzymatic activity leading to a complicated results interpretation.[26,27] However, in our study we aimed to establish baseline levels but not to detect intoxication levels where significant difference may be falsely interpreted.

Furthermore, reduced activity of ACHE and BCHE that are not related to OP exposure can be genetically originated not related to sex, race or age where it accounts, for 23% of the variation in ACHE among humans,[28,29] physiologically related like age, gender, pregnancy and given that ACHE has a wide range of normal values in human population and is affected by the number of red blood cells (RBCs) in any particular sample. That's why and in general the coefficients obtained from BCHE measurements range between 15-25%, while for ACHE the range is rather lower at 10-15%. These findings are compounded by the observation that consecutive monthly or consecutive daily measurements in healthy individuals reached a variation of 20% and in some occasions exceeding 40%.[9] In our results in the contaminated area, coefficients obtained were 8% to 13.3% for BCHE and ACHE respectively, and in the non contaminated area 18% for BCHE measurements and 15.5% for ACHE.

BCHE activity invariably shows relatively greater depression than ACHE from baseline values in samples taken soon after sub acute or multiple exposures to OP, in other means the degree of BCHE inhibition after exposure to dimethoxy OPs for instance will be reduced 50% after 12 days post exposure and recovery is essentially completed after 50 days, yet 15% of the ACHE will be inhibited in 12 days post exposure where recovery to normal was completed in 82 days.[18]

Despite the fact that all OPs share the general structure and mode of toxicity,[27,30] BCHE activity measurement on admission was found to be inconvenient and fluctuated,[9] it could be utilized suitably when OP is identified and when its sensitivity and specificity is known to that OP. Added to this, BCHE has limited efficacy because of its limited 11 day half-life in plasma and conversely, measuring ACHE will not identify the specific OP inhibitor.[11]

Currently CHE assessment and monitoring remains the only useful mean in providing a measure of the physiological response after OP exposure, and in spite of the limitations and the doubts which spanned for nearly 4 decades on whether it can be a tool of reliance or not, the most important consideration for the reluctance to accept cholinesterase estimations as a reliable measurement in OP exposure is the evidential inability to attribute all ill health aspects following such exposure to CHE inhibition.[9]

Well-beings health surveillance programs utilize biomarkers to identify disease presence and progression, monitor drug delivery or metabolism and monitor chemical exposure. Since exposure varies from low level chronic

exposure during pesticide application to high dose acute exposure including the release of nerve agents or toxic industrial OP; rapid and exact assessment of these agents and the extent or time period of exposure will help to direct the therapy to the victim and to evaluate level of threat prone onto others, furthermore combining approaches which are based on measuring the individuals pre exposure activity levels would in turn improve precision, but this is practical only in certain situations for agricultural workers.[11] Paradoxically, if baselines are not established and pesticides causing poisoning are not identified clinicians usually base treatment on clinical signs and symptoms.[29]

CONCLUSION

Determination of ACHE activity is vital in monitoring and studying the exposure to pesticides and chemical warfare agents, therapeutic monitoring of OP poisoning and in anticholinesterase dosage for alzehimer patients,[16] when there is a marked decreases in ACHE activity it can be used as prognostic factor in OP poisoning, coma, respiratory failure and hemodynamic disturbances.[31] Some clinicians use BCHE for differential diagnosis of either CB or OP poisoning and the efficacy of antidote treatment can be achieved and can be evaluated by utilizing serial measurement of BCHE activities; CB requires fewer number of BCHE serial measurement to reach diagnosis and to follow up the patient compared with the OP poisoning.[32]

Inhibition of CHE reflects exposure to OP rather than toxicity, and in spite of its faults, inhibition of ACHE mostly reflects inhibition at synapses thus representing a useful biomarker for OP/CB toxicity. In case of exposure to nerve agents which are subgroup of organophosphates of high toxicity, early determination of ACHE is mandatory to confirm clinical diagnosis without sophisticated verification of the nerve agent.[3]

CHE is subject to a large degree of variation induced by physiological and pathological factors which can cause confusion in cases when baseline CHE levels are unknown. BCHE is a less than perfect biomarker for OP poisoning if baseline levels are unknown in an individual, consequently ACHE measurement in the same individual would yield. More consistent and clinically reliable results with better association in terms of clinical presentation.[33] It is more scientific or pragmatic to study all exposure levels in-depth; CHE inhibition and the diagnostic health effects from OP/CB/ nerve agents, and all fluctuant variables, and to include them in effective health surveillance programs, taking in consideration the risk assessment of combined exposure to multiple OP pesticides in most exposed groups and identifying subpopulations including children, elderly, and the genetically susceptible individuals, that may be at higher risk, so as to help clinicians into better response to OP/CB/nerve agents poisoning in the future as reported in a previous study.[30]

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Abbreviations

ACHE	Acetylcholinesterase
BCHE	Butyrylcholinestersae
OP	Organophsphates
CB	Carbamates
DTNB	5, 5'-Dithio-2-nitrobenzoic acid
EDTA	Ethylene diamine tetra acetic acid
TNB	5-thio-2-nitrobenzoic acid

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تحديد مستوى الكولين استريز في الأردن: نموذج تقصي ورصد لحدوث انتشار التسمم بالمبيدات

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الملخص

الخلفية والأهداف: تقصي ورصد الطريقة المستخدمة لمعرفة مستوى الكولين استريز في الدم وتحديد المستوى الطبيعي بين الأردنيين الذين يعيشون في مناطق زراعية كثيفة، وكذلك بين أولئك الذين يعيشون في مناطق حضرية وغير زراعية.

طرق البحث: تم استخدام طريقة Ellman المعدلة لتحليل 851 عينة دم من الأشخاص الذين يعيشون في المناطق الزراعية الكثيفة، و1033 عينة دم من الأشخاص الذين يعيشون في المناطق الحضرية وغير الزراعية، وذلك لمعرفة مستوى الكولين استريز في كريات الدم الحمراء والبلازما.

النتائج: ان العينات التي جمعت من المناطق الزراعية الكثيفة أظهرت انخفاض معدل مستوى الكولين استريز 1037 U/L و23 U/gHbمقارنة مع المناطق الحضرية، وغير الزراعية 1616 U/L و31U/gHb في البلازما وكريات الدم على التوالي. كما أظهرت النتائج انخفاض معدل مستوى الكولين استريز بنسبة 52% و41.1% في البلازما وكريات الدم الحمراء على التوالي عند العمال الذين يعيشون في المناطق الزراعية الكثيفة مقارنة مع المستويات الطبيعية التي سجلت في المناطق الحضرية.

الخاتمة: ينبغي على الأطباء الذين يستخدمون مستوى الكولين استريز للتشخيص السريري والعلاج أن يكونوا على علم بأن المستوى الطبيعي يختلف وفقا لمكان إقامة مرضاهم.

الكلمات الدالة: الكولين استريز، التسمم بالمبيدات، الأردن.