Observations on the United Nation Report released 16 September 2013

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Numerous people have asked me for my comment on the recently released report by the United Nations Mission to Syria. Since my current schedule does not allow me to speak or correspond with everyone who wants my comments, I have assembled some observations and comments in this paper. This paper is based on information available to me at the time of writing, and I reserve the right to revise my opinions as further information becomes available. An additional paper, to be published next week, will contain some additional analysis of various theories that I am working on.

General:

Overall, I am generally satisfied with the methodology of the inspection team as described in the report. Appendix 7 contains the most interesting information. There was a practical limit to the amount and type of samples that the team was able to take, due to their size and access. I believe that this report is conclusive evidence that Sarin was used, both because of the environmental evidence and the medical evidence.

Comparison to US and UK "intelligence dossiers" :

The embarrassing truth is that the level of detail in this report is high when compared to the earlier documents released by intelligence agencies in the US and UK. By comparison, the US and UK documents are unencumbered by hard facts and seem quite parsimonious in their level of detail. This raises the inevitable question of "why?' – it would seem to be in the best interests of the US and UK to give the type and level of detail that the UN report gave.

Sample blanks:

The report shows excellent use of blanks and control samples to ensure the integrity of the evidence collection process. The purpose of such samples is to ensure that there is a safeguard in place to verify that containers, wipes, and solvents were not contaminated or cross-contaminated with anything that would give a false positive. For a brief discussion of blanks and their importance in CBRN forensics, please refer to my book[1].

Environmental Samples:

The environmental samples, detailed in Appendix 7 are fairly conclusive and damning evidence of use of Sarin. My previous skepticism on the use of Sarin was based on video evidence and interpretation of signs and symptoms of exposure in the lack of actual physical evidence. The physical evidence is compelling and I have no choice but to believe that Sarin was used. The presence of actual Sarin was detected, as was a variety of degradation products, such as isopropyl methylphosphonic acid (IMPA or IPMPA as it is referred to in the report). IMPA in particular is damning evidence. In addition, the positive samples were from places where I would have looked for samples, such as metal fragments, soil samples, rubber gaskets, and window seals. These are locations where small amounts of Sarin could persist. The presence of DIMP is interesting, as it is a by-product of Sarin manufacture and also a decomposition product. At this stage, I must do more research into the implications of the presence of DIMP. It could be an impurity in the Sarin, an indicator of Sarin having decomposed by exposure to heat, or both.

Blood sampling:

The blood samples are interesting, but I wish that the report was more clear as to their methodology. As I have stated in previous papers and as is firmly stated in the scientific literature, direct testing for GB/Sarin in blood plasma is not a reliable diagnostic technique:

> Analyzing for parent nerve agents from biomedical matrices, such as blood or urine, is not a viable diagnostic technique for retrospective detection of exposure[2]

Since a direct test for Sarin is unlikely to have been useful, I really wish that the report would specify how the blood and urine samples were actually tested. Presumably, the OPCW labs tested for biomarkers such as IMPA and methylphosphonic acid (MPA). In the academic literature, this is the accepted manner for analysis of blood samples. These chemicals are degradation products of Sarin. If IMPA was detected in the blood and/or urine samples, it is a very clear indication of Sarin exposure. MPA would be a general indicator of exposure to nerve agents, but would not narrow it down specifically to Sarin. Soman (GD) and Cyclosarin (GF)[3], and possibly some novel agent in the same family would also lead to MPA being present. But it would still be a strong indicator of use of a chemical nerve agent. Or perhaps they tested for acetylcholinesterase (AChe) inhibition. AChe levels would be an indicator of exposure to toxic organophosphorous compounds (which include, but are not limited to the nerve agents) or carbamates, a chemical family which also includes some medicines and some pesticides. Since the environmental samples were analyzed quite closely for MPA, IMPA, and related compounds, I presume that the blood testing did so as well. But I feel that the report should have been specific in this regard. If IMPA was detected in blood samples, that is as equally compelling as evidence as the environmental samples.

Medical Case Histories:

My initial confusion about the decidedly mixed bag of signs and symptoms continues. The signs and symptoms reported are only partially consistent with Sarin exposure. The UN team was able to collect information on 36 survivors, who showed the following breakdown of signs and symptoms:

Signs and Symptoms from UN Report

Sign / Symptom	% Reported (N=36)
Dyspnea / Difficulty breathing	81%
Eye irritation	22%
Excessive tearing	8%
Blurred vision	42%
Excessive salivation	22%
Coughing	11%
Nausea	3%
Vomiting	22%
Convulsions	19%
Loss of consciousness	78%
Disorientation	39%
Miosis (pinpointed pupils	14%

Source: UN Report, page 13

It should be noted that all of these signs/symptoms are possible in cases of nerve agent exposure. I am very intrigued by the distribution of the symptoms, as this causes much confusion in my mind. Clearly, due to the physical findings, Sarin was employed. But the exact presentation of signs and symptoms seems skewed from our conventional understanding of nerve agent exposure. I would eagerly devour a detailed explanation from a toxicologist (which I am not) or other competent medical expert.

Comparison with Tokyo Incident

The largest basis for comparison in the available medical literature are the various studies published after the 1995 Tokyo subway incident. One study[4] shows the breakdown of signs and symptoms of all 111 inpatients admitted to St. Luke's International Hospital on 20 March 1995 after the terrorist dispersal of Sarin. The breakdown is shown below on the next page.

Signs and Symptoms from Tokyo

Sign / Symptom	% Reported (N=111)
Miosis	99.0
Headache	74.8
Dyspnea	63.1
Nausea	60.4
Eye Pain	45.0
Visual Darkness	39.6
Vomiting	36.9
Fatigue	36.9
Cough	34.2
Agitation	33.3
Fasciculations (localized twitching)	23.4
Convulsion	2.7

Source: Ohbu et al., 1997, p. 590

These statistics from Tokyo omit those who were considered to be suffering only mild symptoms who were not admitted to the hospital, but merely treated and released. It is not possible to make a direct comparison, as the Tokyo signs/symptoms were upon admission, not upon examination days later as was the case with the UN report. The Tokyo statistics broadly match what is expected of Sarin in the military medical literature. Needless to say, there are vast differences between the UN data and the Tokyo data. Incidentally, the Tokyo data is reinforced by earlier data from first responders to an earlier Sarin incident in Matsumoto, wherein eye troubles such as miosis were nearly universally encountered by emergency responders to the incident.[5]

The "Textbook" understanding of Sarin Exposure:

It is easy to note that the Tokyo syndrome more closely represents the classically understood nerve agent exposure syndrome. For purposes of comparison, the table on the following page shows the officially understood progression of symptoms, according to the canonical US military medical textbook:

TABLE 5-4

EFFECTS OF EXPOSURE TO NERVE AGENT VAPOR

Amount of Exposure	Effects*
Small (local effects)	Miosis, rhinorrhea, slight bronchoconstriction, secretions (slight dyspnea)
Moderate (local effects)	Miosis, rhinorrhea, slight bronchoconstriction, secretions (moderate to marked dyspnea)
Large	Miosis, rhinorrhea, slight bronchoconstriction, secretions (moderate to marked dyspnea), loss of consciousness, convulsions (seizures), generalized fasciculations, flaccid paralysis, apnea, involuntary micturition/defecation possible with seizures

*Onset of effects occurs within seconds to several minutes after exposure onset.

Source: Medical Aspects of Chemical Warfare (2008), page 169.

Signs/Symptoms out of Proportion:

The difference between the pre-Ghouta understanding of Sarin exposure syndrome and the UN report is striking. In particular, I would like to highlight a number of paradoxes in the UN figures. Others may find other points of interest as well. I find the following points interesting:

- Relative lack of miosis: Miosis is broadly considered the threshold symptom for nerve agent exposure. Many patients without miosis have shown positive blood tests for Sarin exposure and the presence of signs/symptoms indicative of far greater level of exposure. Miosis after nerve agent exposure can last for a number of weeks[6] and the Tokyo experience confirms that miosis is resistant to atropine injections[7]. The military textbook says categorically: "Miosis and respiratory involvement are almost invariant with inhalational exposure."[8]

- Convulsions without mild/intermediate symptoms: The presence of convulsions without miosis or gastrointestinal symptoms is puzzling. Convulsions are a relatively advanced symptom showing a high level cholinergic crisis brought on by nerve agent intoxication. It is strange that there are patients (such as patients SN 24 and 27) that show positive exposure to Sarin by blood test, convulsions, but no excess salivation, excess tearing, or miosis. That is very strange to me.

- Loss of consciousness: Generally, loss of consciousness is considered to be a very grave sign in nerve agent poisoning, happening shortly before death. How is it 78% of the patients had lost consciousness? The sample victims include at least two patients (patients SN 32 and 34) with positive blood results, but no other distinctive nerve signs or symptoms.

Is it possible that we are looking at exposure to multiple causes of injury? Were some of the examined victims exposed to other things in addition to Sarin? I am not stating that Sarin was not used. It clearly was. My point is that it is either not behaving as we have understood it in the past or that other factors were at work in addition to Sarin.

Understandable limitations of medical case histories.

The medical case history information could be limited in its applicability for the following reasons:

- A sample of population 36 survivors interviewed by the inspection team cannot conceivably be considered a scientifically or statistically accurate sample of the population of affected victims. It would be considered scientifically unsound to draw widespread conclusions based simply on this sample.

- By definition, dead people cannot be interviewed. Since Sarin is highly lethal, the people with the most severe signs and symptoms are unavailable for interview.

- A person who was exposed to Sarin but is available to consent to give a blood and/or urine sample days later has either been exposed to a mild level of the agent or was exposed to a more serious level but was the recipient of aggressive treatment.

- No attempt was made to control for any differential or multiple etiologies. In other words, it is possible, indeed likely in a wartime environment, that at least some of the 36 people interviewed about their signs and symptoms have been made ill by something else or by more than one cause. For example, a combination of conventional smoke exposure (common in the urban wartime environment) and Sarin exposure.

- Much of the world's knowledge of Sarin exposure is based on a handful of incidents and studies on animals which have been extrapolated to humans. It is certainly possible that there is more to know about Sarin than what is captured in the existing literature.

About the author: Dan Kaszeta is the author of "CBRN and Hazmat Incidents at Major Public Events: Planning and Response" (Wiley, 2012) as well as a number of magazine articles and conference papers. He has 22 years of experience in CBRN, having served as an officer in the US Army Chemical Corps, as

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References:

[1] Kaszeta, D. *CBRN and Hazmat Incidents at Major Public Events: Planning and Response*. Wiley, 2012, pp 293-295.
[2] US Army Office of the Surgeon General. *Textbook of Military Medicine: Medical Aspects of Chemical Warfare*. 2008. p. 694. Freely available in public domain online.
[3] Ibid, page 696.
[4] Ohbu S, et al. Sarin Poisoning on Tokyo Subway, *Southern Medical Journal*, Vol 90, Issue 6, June 1997.
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[6] Sidell FR. Soman and sarin: clinical manifestations and treatment of accidental poisoning by organophosphates. *Clinical Toxicology*. 1974;7:11
[7] Ohbu, op. cit., p. 30.
[8] Medical Aspects of Chemical Warfare, op. cit. p. 169.

