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Nuclear Targeting and the Nuclear Employment Augmentation Team (NEAT)

ABCA-Optimizing CBRN Interoperability

Fukishima Daiichi Operating and Design Characteristics with Relevance to the Army Reactor Program

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Toxin Weapons: What's the Worry?

Regulating CBRN Survivability

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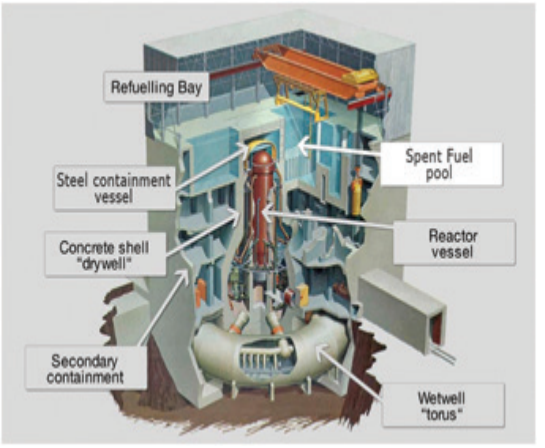

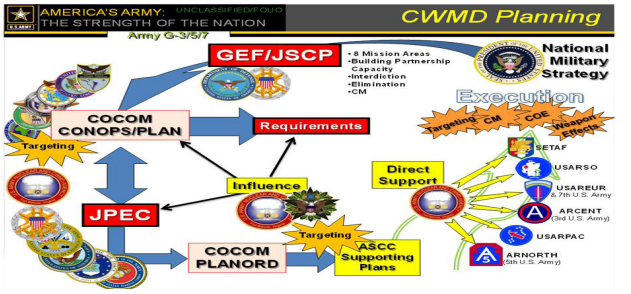

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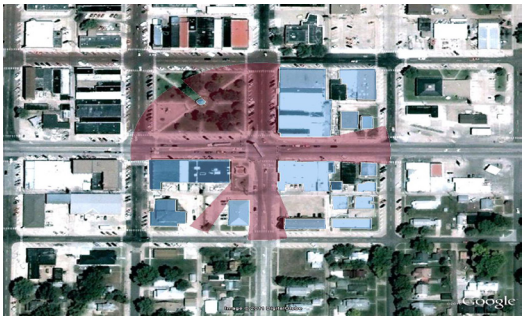

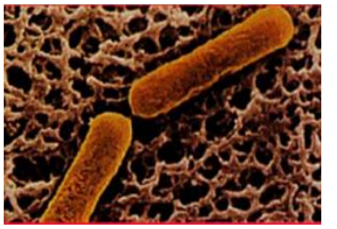

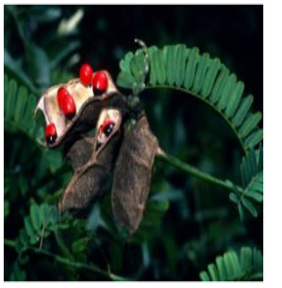

BIOGRAPHY
COLONEL JUAN ARIEL CUADRADO

Colonel Cuadrado commissioned in the US Army Field Artillery in 1984 and joined the Chemical Corps in 1987. Early career assignments include the 2-162nd Field Artillery Battalion (Puerto Rico); the1-39th Field Artillery Airborne Battalion (Fort Bragg); and the 18th Field Artillery Airborne Brigade in Fort Bragg, and Operations Desert Shield and Desert Storm. As an Army Functional Area 52 (Nuclear and Counterproliferation Officer), he served in a variety of technical and operational assignments including the Department of Chemistry and the Photonics Research Center in the United States Military Academy in West Point; Los Alamos National Laboratories; the Defense Threat Reduction Agency; United States Forces Korea; the National Nuclear Security Administration, Department of Energy; and the Office of Nuclear Deterrence Policy, Office of the Undersecretary of Defense for Policy in the Pentagon. Colonel Cuadrado holds a B.S. in Industrial Chemistry (University of Puerto Rico), a Masters in Analytical Chemistry (Florida State University), and a Masters in National Resource Strategy (Industrial College of the Armed Forces, NDU). Colonel Cuadrado currently serves as Deputy Director of the U.S. Army Nuclear and Combating WMD Agency (USANCA) in Fort Belvoir, Virginia.

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Mailing Address:
Director, U.S. Army Nuclear and CWMD Agency (USANCA), 5915 16th Street Bldg 238 Fort Belvoir, VA 22060-0529.

Telephone: 703-806-7848, DSN 656-7848, Fax 703-806-7900

Electronic Mail: usarmy.belvoir.hqda-dcs-G-3-5-7.mail.usanca-mailbox@mail.mil

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USANCA's Senior Leaders Transition, the 7th Annual Combating WMD Conference, and Recent Army Documents on Capabilities for Countering WMD

COL JUAN CUADRADO
Acting Director
U.S. Army Nuclear and CWMD Agency

Shortly after the publication of the previous issue of the Combating Weapons of Mass Destruction (WMD) Journal, the USANCA Director, Mr. Peter Bechtel, accepted a new assignment as Director of the Capabilities Integration Directorate (DAMO-CI) in HQDA G-3/5/7. He appointed me Acting Director until a new SES was selected to serve in this challenging dual-hatted position as Deputy G-3/5 and USANCA Director. After a very exciting and rewarding six months I am pleased to announce that Mr. Daniel M. Klippstein has been appointed as Deputy Director for Plans and Policy, Office of the Deputy Chief of Staff, G-3/5/7 and will soon start his duty as the new SES-Director for USANCA.

In the last issue of the Combating WMD Journal, Mr. Peter Bechtel discussed the implications of recent strategic guidance and CBRN capabilities required for the Joint Force in the 21st Century. As the Acting Director of USANCA, I see daily how this strategic guidance is influencing the Army to refocus from a COIN Centric position to a broader mission spectrum requiring new capabilities. In particular, as a result of recent strategic guidance, the Army is taking a new look at the Combating Weapons of Mass Destruction (CWMD) mission area and determining what new capabilities are required across DOTMLPF. I thought it might be useful to update you on the critical Army documents that have recently been published from our Army Leadership, which describe Army capabilities required for the future, to include countering WMD.

Additionally, as USANCA recently hosted its 7th Annual Combating WMD Conference here at Fort Belvoir, I wanted to highlight the key take-aways from the conference and describe some of the common themes and challenges we gathered from both senior Army leaders and CWMD Action Officers from across the Army, especially from the Army Service Component Commands (ASCCs). I trust that you, at the conclusion of this discussion, will see how the CWMD Community will continue to play a critical role in facilitating CWMD capability development and executing CWMD missions in both Army and Joint formations.

A tiered strategic plan framework headlined by The Army Plan informs the US Army's CWMD strategy. The Army Plan consists of four sections; the Army Strategic Planning Guidance; the Army Planning Priorities Guidance; Army Programming Guidance Memorandum and the Army Campaign Plan. These documents describe and define the Army's long range vision, set the priorities, level of effort, resource levels and synchronize the details.

Section I of the Army Plan is the Army Strategic Planning Guidance (ASPG). The ASPG outlines the Army's vision and role as part of the Joint Force. The document also nests Army guidance within National, OSD and Joint Directives and provides Army leaders a vision, direction, and strategic objectives over the near, mid and long-term.

Last year, USANCA in collaboration with HQDA G-3/5/7's CWMD and Proliferation Policy Division (SSD) authored the first ever CWMD Appendix to the Army Campaign Support Plan. This appendix was designed to complement the US Government's CWMD efforts and compliments the Guidance for Employment of Force (GEF), Joint Strategic Capabilities Plan (JSCP), the National Military Strategy for CWMD, and the DoD Global Campaign Plan for CWMD. The Appendix is designed to bridge the gap between National Strategy and Army Strategy and provide senior army leaders a framework for CWMD.

As an Army Staff Officer involved in CWMD issues you must understand how the Army is using these documents to develop capabilities for the Joint Force Commander to effectively counter WMD in their Area of Responsibility. Most importantly, you are the subject matter expert on your staff that must articulate how to integrate these capabilities with our General Purpose Forces (GPF). Countering WMD is not a specialist function only performed by CBRN forces or special operations forces. Rather, it is a military mission that requires maneuver formations, primarily BCTs, to understand how to plan/integrate CWMD Enablers into their formation in order to support CWMD mission areas. The topic of CWMD while conducting Unified Land Operations was the theme of our conference this year, and I would like to share with you some of the highlights resulting from our conference.

This year we were fortunate to have key senior leaders from across the DoD CWMD Community share their insights with the conference attendees. The keynote speaker was MG Tucker, ADCS G-3/5/7, who recently commanded the 2 ID in Korea and brought his insights as a Division Commander who had to plan on conducting Elimination Operations on the Korean Peninsula. MG Tucker voiced his concern that we must do much more to prepare and train GPFs to conduct WMD Elimination (WMD-E) and Interdiction. He asked "How does a BCT prepare and train to conduct CWMD mission sets?" MG Tucker stated that CWMD is a growth industry and with the establishment of Regionally Aligned Forces the Army needs to prepare for an increase in GCCs requirements for Army CWMD capabilities in the future.

Maj Gen Crabtree, Commander for SJFHQ-E at that time, discussed his organization's mission, which is to plan and train to enable command and control for WMD-E, but cautioned that they have no forces assigned to do the WMD-E mission. This highlights again the critical role the Army plays in CWMD and especially WMD-E.

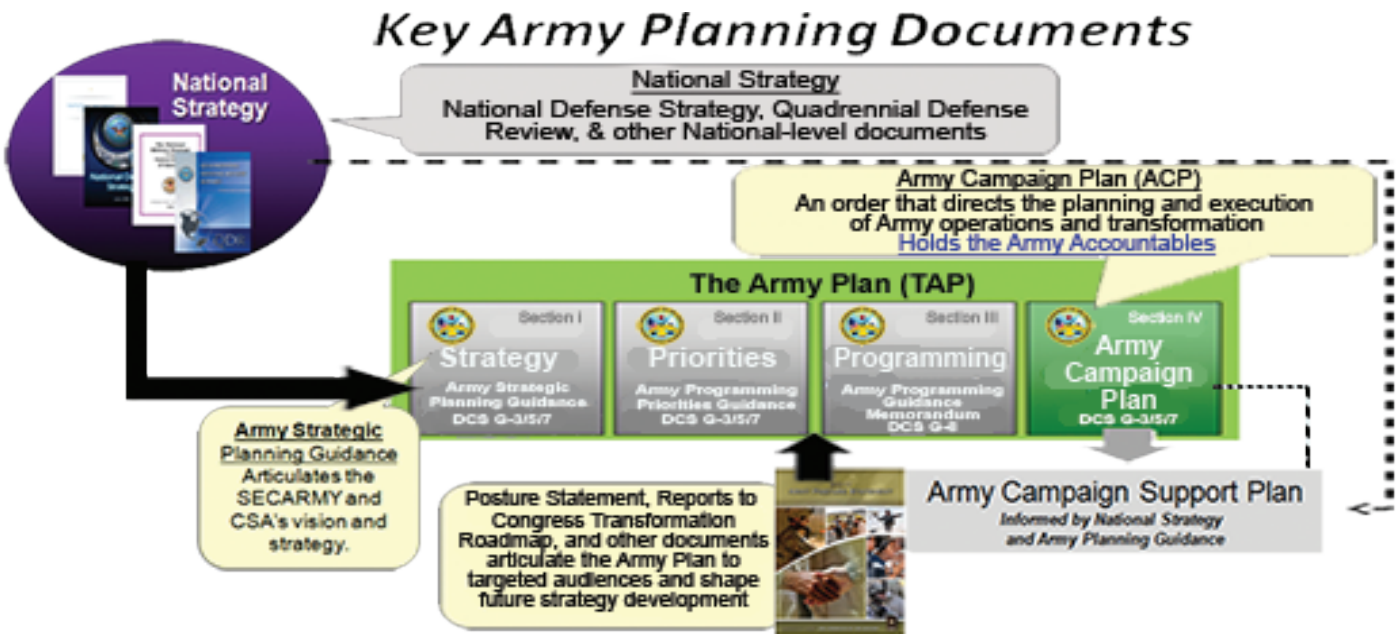
MG Smith, Commander 20th SUPCOM, presented an overview of the 20th capabilities and recent activities. It was an impressive statement of how far the 20th has developed its capabilities and reach globally in a relatively short timeframe. Increasingly, the 20th is getting more involved with ASCCs to support opportunities to develop partner nation capacity to support CWMD, leveraging the expertise found only in the 20th SUPCOM. Of course the 20th is very engaged supporting planning and exercises on the Korean Peninsula.

The other speakers at our conference came from across the Army and the Intelligence Community. Of particular interest was hearing from all the ASCCs, who spoke to CWMD activities and challenges in their respective areas. It was a good opportunity to synchronize and coordinate activities across ASCCs, as well as exchange best practices and lessons learned. Consistent in all the briefings and discussion was recognition of an increasing Army role in nonproliferation and counterproliferation activities.

With an anticipated increase in WMD proliferation, the Army must enhance its WMD detection, identification, interdiction, exploitation, and forensics capabilities. Army CBRN formations will play a major role in future CWMD operations, but their capacity remains limited. As a result, GPF will be required to execute complimentary CBRN tasks. However, GPF currently lack the mission essential tasks, training and equipment to effectively conduct full-spectrum CWMD operations. The Army will need to incorporate sufficient training of its GPFs on WMD-related tasks to successfully execute all possible aspects of CWMD Unified Land Operations.

Where nations are less capable, it is critical to focus U.S. Army assistance on building indigenous capability to combat WMD threats. Building partner capacity (BPC) is essential to increasing barriers to WMD proliferation and use; improving the ability to identify, mitigate and defeat emerging WMD threats by developing an active layered defense. The National Military Strategy to Combat WMD (NMS-CWMD) highlights cooperation with security partners as a critical enabler, and it includes, as one of the eight key mission areas, "security cooperation and partner activities (SCPA)." These sentiments are reinforced in the Quadrennial Defense Review (QDR) BPC Roadmap and the Office of the Secretary of Defense (OSD) Guidance for Employment of the Force (GEF). The Joint Strategic Capabilities Plan requires Theater and CWMD Campaign Plans enunciating further guidance toward CWMD SCPA. Together, the aforementioned strategic guidance documents provide a strong impetus for the Army to identify and assess current partner capacity-building efforts, and to identify and address the most critical gaps.

We are living in interesting times today in DoD as the Army refocuses after almost 12 years of combat operations. With the increasing awareness of the WMD threat, the Army will continue to develop and transform relevant capabilities to counter the WMD Threat world-wide. The Army's cohort of FA52 Officers across the Army and Joint Force, especially at the strategic and upper operational levels, provide a unique nuclear and CWMD expertise to serve as a key player and advisor in these areas, complementing the equally relevant role of the CBRN Defense Officers. I ask you all to stay relevant by maintaining your technical skills required as a FA52 Officer and by staying current on strategic and operational guidance, such as The Army Plan discussed here. You are uniquely positioned to mesh strategic and operational guidance and tasks with technical nuclear and CWMD capabilities and policy, ensuring the Army is always ready for future threats and challenges.



The Decision to Expand Biological Threat Reduction Efforts into Africa

Ralph F. Kerr, PhD

After many years of success reducing the threat of weapons of mass destruction (WMD) primarily in the former Soviet Union (FSU) via the Cooperative Threat Reduction (CTR) program, the United States Government (USG) expanded defense department measures to reduce biological threats in Africa. This expansion of the Biological Threat Reduction (BTR) program comes at a critical time as national security challenges appear to be shifting from Al Qaeda, Iraq and Afghanistan to global economics, cyber threats, China, and the Arab Spring.[1] The expansion of BTR was sponsored by Senator Richard Lugar (R-Indiana) and this legislation appears to have glided through the legislative process. As one of the original sponsor's of the CTR program, not to mention being the most senior Republican in the Senate and the Republican leader of the Senate Foreign Relations Committee at the time[2], Senator Lugar appears to have welded much power which greatly assisted the passage of this legislation. Understanding the context of this issue and the process involved in approving the expansion of BTR into Africa will shed light on this important national security decision.

A Brief History of the CTR and BTR Programs

Congress initiated threat reduction efforts with the FSU in November 1991 and in December 2011 the CTR program celebrated twenty years of success[3]. The program's accomplishments to date are impressive and include the elimination of more than seven thousand nuclear warheads; almost eight hundred intercontinental ballistic missiles (ICBMs); nearly five hundred ICBM silos; close to two hundred ICBM mobile launchers; 33 nuclear carrying submarines; and many other FSU WMDs[4]. Through this program thousands of chemical weapons were neutralized; fissile materials were secured and safeguarded; and weapons facilities were converted to peaceful purposes. Additionally, the work of many former weapons scientists and engineers was redirected to other useful purposes besides weapons research and development. The brainchild of former Senator Sam Nunn (D-Georgia) and Senator Lugar, this program was originally created to thwart "the nuclear security and proliferation risks emanating from the disintegrating Soviet state." The CTR program initially focused on those former Soviet states (Ukraine, Kazakhstan and Belarus) that were left with nuclear weapons after the fall of the Soviet Union as well as Russia itself[5]. Ukraine, Kazakhstan and Belarus are now nuclear free countries and Russia has many less WMDs, WMD capabilities and WMD infrastructure than it did prior to the dissolution of the Soviet Union[6].

On the biological threat side, engagement with Russian officials proved to be more difficult at first than working on nuclear or chemical threat reduction issues[7]. In the late 1990s, as part of the CTR program, the US endeavored to reduce the threat of the FSU's massive biological weapons (BW) complex[8] by assisting Russian officials in what became known as the BTR program. These efforts primarily sought to improve safety and security at some of Russia's BW sites and employ scientists in other-than-BW or "nonthreatening" research (e.g., biological sciences, public health and agriculture protection). To date, the US has helped Russia eliminate the infrastructure and equipment at numerous former BW research and production centers; enhance safe and secure storage and handling of biological pathogens; develop modern surveillance, warning, and response networks; and conduct cooperative research programs to increase transparency and thus discourage transfer of BW knowledge and expertise to other countries[9]. But not all has gone as desired by US officials with the BTR program in Russia as several sites suspected of conducting BW research have remained off-limits (e.g., laboratories in Sergiev Posad, Yekaterinburg, and Kirov)[10]. So the question arises that if there is still work to be done in Russia to reduce biological threats, then why move on to someplace else, especially Africa?

Evolving Threat

The simple answer may be that the biological threat has evolved over time and therefore, expanding threat reduction efforts makes perfect sense. If that is the case, it also does not mean that the US has 'cut and run' from our efforts to curve biological threats in Russia. The Defense Threat Reduction Agency (DTRA) is the executing agency of the Department of Defense's CTR work and a quick scan of the their Fiscal Years 2011 and 2012 Budget Estimates for CTR reflect continued resourcing requests for BTR efforts in Russia[11]. Mr. Kenneth Myers, the Director of DTRA, also confirmed this point in testimony he provided on June 24, 2010 before the Senate's Committee on Foreign Relations. "As these important efforts with our partners in the Former Soviet Union continue, we also are taking the knowledge and capabilities acquired through the Nunn-Lugar program implementation to new partners across the globe."[12]

Some of these newest partners are countries in east Africa. This is because "major infectious diseases known to have potential in biological warfare are endemic in eastern Africa"[13] and senior USG officials are concerned that some of these disease samples may easily be obtained by terrorists. These officials believe pathogens could be stolen due to lax security at some laboratories where disease-causing bacteria and viruses are studied and samples are stored.[14] Mr. Andrew Weber, the Assistant Secretary of Defense for Nuclear, Chemical and Biological Defense, who

accompanied Senator Lugar on trip to eastern Africa in November 2010 to look at some of these laboratories, described this dilemma as a "nexus between active terrorist groups, ungoverned spaces, and human and animal health laboratories working on endemic diseases, some of which are rare and exotic." Mr. Weber explained that, "we want to make sure that the pathogens that could be used by terrorists are better secured and that there's an enhanced capability to monitor infectious disease outbreaks."[15] Two of the best examples of endemic disease causing pathogens that could be obtained in Africa and used against Americans may be anthrax and Ebola. These two diseases are considered by many experts in the biological counter-proliferation field as significant threats because of their potential for causing death, the fear that their use may cause, and the economic losses that would result due to clean-up costs in the case of anthrax dissemination across a large area. In 1993, sixteen Aum Shinrikyo cult members went to Zaire (present day Democratic Republic of Congo) "to learn as much as possible about and, ideally, to bring back samples of Ebola virus."[16] This is the same cult that attacked the Tokyo subway with sarin gas in 1995 and that had also experimented with anthrax - cult members sprayed anthrax spores from the top of an eight-story building in eastern Tokyo attempting to cause a major biological emergency.[17] Although the Aum cult's attempts to develop and effectively use biological agents failed, the threat of others trying to use Ebola and/or anthrax from samples collected in Africa, or other diseases for that matter, still poses a serious biological threat risk.

The rise of al Shabab, a powerful Islamist insurgent group that claimed responsibility for the deadly suicide bombings in Kampala, Uganda during the World Cup Finals in July 2010, has alarmed officials and refocused U.S. security interest and attention in the area.[18] Al Qaeda, which sought to develop biological weapons in Afghanistan and considered by some experts to still be pursuing such efforts, has established cells in East Africa.[19] These Al Qaeda linked cells, when combined with radicalization among sectors of the Muslim population, are the most serious threats in East Africa to the US and our allies according to Senator Lugar.[20] Mr. Myers (Director, DTRA) explained the evolution of the biological threat emanating from Africa in terms that may sound almost like a simple math equation. First is the fact that there are many disease pathogens being legitimately researched for their effects on humans, animals and plants; then there is the poor quality of security at many of these research laboratories; and this is happening in a region where terrorist cells appear to be growing. More simply stated, the combination of these factors: bioagents + quality (of security) + quantity (of laboratories) + (terrorist) organizations = dangerous situation.[21] This all adds up to a biological threat that should not be ignored. Adding weight to the dilemma this equation is the fact that history shows us that the Aum cult tried to collect samples of a bioagent (Ebola) from Africa. This is further compounded because many experts believe that the FSU obtained many of their samples for their biological warfare program from Africa as well.[22]

National Security Concerns

There has been a consistent concern about biological terrorism within the executive and legislative levels of the USG for some time; in fact one may even speculate such concern goes back to when biological agents were first considered for use as military weapons.[23] Against the backdrop of recent terrorist attacks including the World Trade Center bombing in 1993, the Oklahoma City bombing of the Alfred P. Murrah Federal Building on April 19, 1995, and the sarin gas attack in the Tokyo subway by Aum Shinryko on March 20, 1995, President Bill Clinton released Presidential Decision Directive-39 (PDD-39), *U.S. Policy on Counterterrorism*, in June 1995. Regarding WMD in general, this policy stated:

The United States shall give the highest priority to developing effective capabilities to detect, prevent, defeat and manage the consequences of nuclear, biological or chemical (NBC) materials or weapons use by terrorists. The acquisition of weapons of mass destruction by a terrorist group, through theft or manufacture, is unacceptable. *There is no higher priority than preventing the acquisition of this capability or removing this capability from terrorist groups potentially opposed to the U.S.*[24]

During the Clinton administration, Congress also passed the Nunn-Lugar-Domenici Defense against WMD Act of 1996 that provided for the training of first responders to effectively handle WMD terrorist incidents. Funding was provided for the Department of Defense to train 120 cities' RAID (Rapid Assessment and Initial Detection) Teams to respond to chemical, biological, radiological and nuclear hazards. There were over two dozen findings in this act of which several specifically addressed concern of the WMD threat, to include bioterrorism:

- The problems of organized crime and corruption in the states of the former Soviet Union increase the potential for proliferation of nuclear, radiological, biological, and chemical weapons and related materials.

- The conditions described in [the previous] paragraph have *substantially increased the ability of potentially hostile nations, terrorist groups, and individuals to acquire WMD and related materials and technologies* from within the states of the former Soviet Union and from unemployed scientists who worked on those programs.

- The *acquisition or the development and use of WMD [are] well within the capability of many extremist and terrorist movements,* acting independently from or as proxies for foreign states.

- *The potential for the national security of the United States to be threatened by nuclear, radiological, chemical, or biological terrorism must be taken seriously.*[25]

Although this last finding mentioned "national security," the Act did not detail the national security interests threatened by WMD (or bioterrorism specifically). The only specific mention of a national security interest is found in finding 13 where it characterized the WMD threat posed a "significant and growing" to US citizens.[26]

These actions during the Clinton administration - the establishment of official policy on WMD counterterrorism, funding to counter WMD, and working with Congress on the Defense against

WMD Act were the beginning of a trend where USG officials sought to take prudent, actionable measures to counter the potential use of WMD that included biological threats. At the beginning of President George W. Bush's administration and in the immediate aftermath of the Amerithrax[27] attacks, additional measures were put in place by the White House and Congress to confront terrorism and the biological threat. This was due in a large part to speculation "that the next act that would be carried out by the Al Queda group in the United States would involve chemical and biological terrorism."[28] These concerns resulted with the promulgation of numerous national level strategies and directives[29] to address the WMD threat, the consolidation of twenty some odd organizations in to the new Department of Homeland Security (DHS), and increased funding for more extensive biodefense measures. To address the biological threat directly, the Bush administration released Homeland Security Presidential Decision-10/ National Security Presidential Directive-33 (HSPD-10/NSPD-33), *National Biodefense Strategy* (often referred to as *Biodefense for the 21st Century*), in April 2004. This strategy specifically addressed the threat of biological terrorism to national security stating that attacks with biological weapons could:

- Cause catastrophic numbers of acute casualties, long-term disease and disability, psychological trauma, and mass panic;

- Disrupt critical sectors of our economy and the day-to-day lives of Americans; and

- Create cascading international effects by disrupting and damaging international trade relationships, potentially globalizing the impacts of an attack on United States soil.[30]

Concerned that terrorists would aerosolize a biological agent that could result in thousands of casualties, the Bush administration established the BioWatch program. This program detects certain biological agents in the air through a comprehensive protocol of monitoring and laboratory analysis to provide warning of a potential bioterror event. BioWatch 'sensors' are located in approximately thirty metropolitan areas throughout the United States and provide continuous air monitoring. Where emplaced, this program can detect a biological agent within 36 hours of release thereby allowing time for federal, state and local officials to place in to effect response and recovery efforts.[31]

Supporting the previous administration's biodefense strategy and seeking to "encourage the alignment of global attitudes against the intentional misuse of the life sciences… to harm people, agriculture, or other critical resources," President Barack Obama released the *National Security Strategy for Countering Biological Threats in November 2009*.[32] This latest strategy also clearly articulates the threat to national security posed by biological agents. Paragraph four of the introduction states:

The effective dissemination of a lethal biological agent within an unprotected population could place at risk the lives of hundreds of thousands of people. The unmitigated consequences of such an event could overwhelm our public health capabilities, potentially causing an untold number of deaths. The economic cost could exceed one trillion dollars for each such incident. In addition, there could be significant societal and political consequences that would derive from the incident's direct impact on our way of life and the public's trust in government.[33]

Based on the clear threat to national security as articulated above in the Bush and Obama biodefense strategies; the fact that there are many endemic, deadly pathogens stored in numerous, poorly secured laboratories throughout Africa; and the possibility that samples of pathogens could end up in the hands of terrorists, then it only seemed prudent to try and take some measures to prevent this from happening. In looking at Mr. Myers equation of this situation: bioagents + quality (of security) + quantity (of laboratories) + (terrorist) organizations = dangerous situation, it seemed to make perfect sense to national leaders that if the quality (of security) could be improved at laboratories where pathogens are researched and stored, then samples of such pathogens ending up in the hands of terrorists might be thwarted. According to Mr. Myers, taking this approach was getting 'the biggest bang for the buck.' Paying to improve security at laboratories and thus preventing the access to dangerous pathogens was a lot cheaper in the long run than trying to go after it and take pathogens away from terrorists after they already have them, or even worse, facing the consequences of a biological attack - i.e., treating casualties and paying for clean up and recovery operations.[34] There has been a precedent from both the executive and legislative branches over several administrations to fund prudent, actionable measures to counter WMD threats. Examples included the RAID teams during the Clinton administration and the BioWatch program during the Bush administration, thus addressing this particular biothreat by expanding BTR into Africa was also considered prudent. Deciding to do this did not happen overnight.

Dance and Decision

During the Bush administration, funding for the BTR program increased to incorporate expansion of efforts in Russia. The funding for BTR efforts in Fiscal Year (FY) 2004 was $54.2 million (M) requested and approved, but this met with some push back due to concern from House members wanting to restrict cooperative research at any site until it could be certified that no prohibited biological weapons (BW) research was taking place. House members were also concerned about the vulnerability of pathogens to theft or loss and they therefore permitted the use of up to 25% of the appropriated funds to address these issues (i.e., looking into cooperative research to ensure no BW research was taking place and conducting assessments of security at research laboratories). In FY 2005, of $55M authorized for the BTR, over half of the funds were shifted from cooperative research efforts to biosafety and biosecurity enforcement because of the Congressional concerns about possible biological weapons programs. Additionally, funding was approved for expanding BTR efforts into Kazakhstan, Uzbekistan and Georgia at this time also for biosecurity and biosafety efforts.[35] Congress' actions to fund biosafety and biosecurity measures via BTR during this time established a precedent that would continue. During the remainder of the Bush administration, funding for BTR efforts continued to grow with a significant expansion

in FY 2008 appropriations ($144.5M) being approved by Congress due to growing concerns about the threat of biological weapons proliferation. Senator Lugar sought to add an additional $100 million in Fiscal Year 2008 appropriations for additional expansion of BTR but the Senate reduced this amount to $50M.[36] According to Mr. Myers, it was during this period (2006 - 2007) that Senator Lugar was seeking support to expand BTR efforts into other countries outside the FSU. When I asked him point blank if the BTR expansion received support because of the Senator's influence, Mr. Myers said that Senator Lugar charted a path and worked as necessary with whomever he thought he needed to bring on the team to try and get this done. He characterized Senator Lugar as being patient, diplomatic, methodical, knowledgeable and not concerned about receiving credit.[37] Not only were additional resources (i.e., funding) approved for the BTR program because of Senator Lugar's efforts, the National Defense Authorization Acts of 2007 and 2008 also directed the National Academy of Sciences to conduct studies on the proliferation of biological weapons. The first report released in 2007 primarily looked at BTR efforts within the FSU but included an astounding "concern" that received attention from nonproliferation experts and national leaders alike:

While this study did not address in depth facilities outside the FSU, committee members are aware of *poor security conditions at some facilities in other regions of the world that handle dangerous pathogens. Their impressions concerning the lack of adequate security at facilities throughout* South Asia, the Middle East, *Africa*, and Latin America are consistent with reports to the committee by U.S. government specialists who have visited a number of the facilities…*Some vulnerable facilities are located in developing countries in Africa and Southeast Asia where reports suggest that there are safe havens for terrorist cells.*[38]

The second study report by the National Academies of Science was released in early 2009. Study members were asked to assess capabilities of developing countries to control dual-use technologies; assess approaches used in the FSU that could be employed in other countries; review international programs contributing to nonproliferation; and recommend USG integrated actions for programs outside the FSU.[39] The first recommendation of the study was that "DoD should, within the U.S. government's evolving global biological engagement strategy, promptly expand BTRP into selected developing countries beyond the FSU."[40] As the process of funding BTR continued to increase through the end of the Bush administration ($184.5M requested and 174.3M appropriated for FY 2009) there was an initial dip with the new Obama administration for FY 2010 in which $152M was requested and approved. For FY 2011, $209M was requested and approved, and for FY2012, $259.5M has been requested.[41] In concert with the general increase of funding for BTR over that past few years, support for expanding to other countries also gained traction. This support was prominent amongst Congressional members and within the DoD where senior members from both Policy and Acquisition, Technology and Logistics (AT&L) offices, as well as DTRA, pulled together and aligned their efforts supporting BTR expansion. The combination of national strategies highlighting biological threat concerns, sustained resourcing over the life of the BTR program, studies by expert panels recommending expansion of BTR into Africa, and national leaders advocating for and supporting BTR expansion might be described as a dance of sorts that led to the decision to expand BTR efforts into Africa.

The above measures alone did not seal the deal, so to speak, and the dance needed additional synchronization and action. This synchronization and action included steps taken by senior leaders to bring this effort together. Although not every effort or 'dance step' of all the senior leaders involved in supporting and gaining the decision to expand BTR efforts into Africa is presented, the following dance steps helped tremendously in gaining this final decision and show how this decision came about. In support of Senator Lugar's efforts seeking to expand the BTR program into other countries outside the FSU during the 2006 - 2007 timeframe, the defense department (who is overall in charge of executing this program) not only looked hard at this issue, but readily took the challenge on for action. Mr. Joseph Benkert's testimony to the Senate Armed Services Subcommittee on Emerging Threats and Capabilities on April 2, 2008 may have been the first formal dance step that attests to this. His statement for the record reads in part:

- CTR advocates have been asking when CTR will "go global." It is a good question, and we are looking at opportunities... I would offer the following thoughts in this regard.

- CTR will always be ready to address stocks of WMD if they are found, and if applicable governments ask for our assistance to eliminate them. However, the WMD threat is no longer only about addressing WMD at its source. As we think about CTR in a global context, it must be in the way CTR has already been moving – increasing foreign institutional capacity to address WMD threats. The bio-security case is a good example. CTR's biological threat reduction program was originally conceived to address the threat posed by the legacy of the Soviet Biopreparat – a complex of especially dangerous pathogens, infrastructure and scientific expertise. Biopreparat doesn't exist outside the states of the former Soviet Union, although a bio-terrorism threat does exist. Our challenge is to make the original CTR bio-security model applicable to the global threat. This is going to focus much more on building foreign capacity than the infrastructure heavy work that was necessary to address the legacy of Biopreparat...

- With the forgoing in mind, I am happy to report that we are ready to move forward with CTR to address global threats.[42]

Following Mr. Benkert's testimony, action officers within the Office of the Secretary Defense for Policy began exploring options for expansion in concert with the recommendations from the two previously mentioned National Academies of Science study reports recommendations. 2010 was a busy year with regard to the efforts to expand BTR into Africa. On January 28, Senator Lugar gave a speech in Washington, D.C. on Nunn-Lugar and Arms Control declaring that, "The Nunn-Lugar program is well

positioned to enter a new phase of global security engagement."[43] With specific regard to the BTR program, he said: Biological threat reduction is an area ripe for the Global Security Cooperation I envision, particularly in view of what the President has proposed. *[Note: I believe this is referring to the National Strategy for Countering Biological Threats – RFK.]* The work of securing dangerous pathogens, building central reference laboratories and establishing disease surveillance and monitoring are all critically needed in many parts of the world. Nunn-Lugar biological engagement directly serves key U.S. interests, including safeguarding the welfare of our troops and detecting emerging infectious diseases and pandemics before they threaten the American people.[44]

The next month after this speech (February 2010), DTRA submitted its budget request asking for funding to expand BTR efforts into select areas of Africa.[45] And the Quadrennial Defense Review (QDR) was released highlighting this issue by directing: Expand the biological threat reduction program. Countries that have the infrastructure and capability to report and track the spread of an outbreak of disease are able to save more lives. Detecting, diagnosing, and determining the origin of a pathogen will enable U.S. authorities to better respond to future disease outbreaks and identify whether they are natural or man-made. Accordingly, we are expanding the biological threat reduction program to countries outside the former Soviet Union in order to create a global network for surveillance and response.[46]

This direction in the QDR to expand BTR codified the move for DoD to 'go global' and thus when Mr. Benkert's successor testified before the same committee later that spring, he spoke directly about Africa. Dr. Michael Nacht told the Senate Armed Services Subcommittee on Emerging Threats and Capabilities on April 21, 2010, "The department is also considering expanding the [BTR] program into Sub-Saharan Africa…" [47] And the dance continued into the summer with Mr. Myers testifying before the Senate's Committee on Foreign Relations on June 24 in which he addressed expanding the BTR program.[48] All these measures and the final congressional appropriations for DTRA's expansion of BTR efforts into Africa sealed the deal.

Conclusion

The decision to expand the BTR program into Africa was achieved over the course of several years (2007 – 2010) and included the convergence of advocacy and gentle prodding from an influential Senator, strategic vision from the White House, resourcing support from Congress, and willingness from senior DoD leaders to take this mission on and DTRA to execute. Expanding BTR into Africa did not happen out of the blue, but rather grew from the successes of the CTR program and precedents of other measures enacted by national leaders (e.g., RAID teams and BioWatch). The expansion of BTR into Africa to provide biosafety, biosecurity, and biosurveillance at laboratories where legitimate life sciences research is conducted is clearly in the interest of national security. These biosafety, biosecurity, and biosurveillance measures will go a long way toward thwarting the chances of dangerous disease pathogens ending up in the hands of those who wish to cause harm to us, our friends and allies.

BIOGRAPHY

Dr. Kerr is a Senior Chemical, Biological, Radiological and Nuclear (CBRN) Analyst at the Joint Requirements Office for CBRN Defense in the J8 Directorate of the Joint Staff. He holds a M.S. and Ph.D. in Biodefense from George Mason University, Virginia; a M.S. in National Security Strategy from the National War College, Fort McNair, Washington, D.C.; and a M.A. in Political Science from Jacksonville State University, Alabama. Dr. Kerr served as an Army Chemical Officer from 1982 - 2003.

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Fukishima Daiichi Operating and Design Characteristics with Relevance to the Army Reactor Program

Phil Shubert
Army Reactor Program Manager, U.S. Army Nuclear and CWMD Agency

Basics of commercial nuclear power designs and how they applied to Fukishima

- Boiling Water Reactor (BWR) vs. Pressurized Water Reactor (PWR)
- Spent Fuel storage designs
- Emergency Core Cooling
- Emergency Power Supply configurations
- Design Basis Accidents

Figure 1. Fukushima Daiichi Nuclear Station

The March 2011 event in Japan has renewed international interest in nuclear events. The importance of nuclear events in our past and in our future cannot be minimized. Because of the enormous potential for both its benefits and the potential for disaster, nuclear has always generated great debate. Nuclear provides the promise of an abundant supply of "non greenhouse gas producing" energy while simultaneously posing severe dangers. For these reasons discussion on the future of nuclear uses is rarely unemotional. The sensational nature of nuclear events coupled with its technical complexity has news reporting focused on the perilous and at times heroic circumstances surrounding these occurrences. This article discusses design features of currently operating power plants focusing on Fukishima Daiichi characteristics. The Fukushima Plants were Boiling Water Reactors (BWR). For water moderated plants in the U.S. there are two basic designs, Boiling Water Reactors and Pressurized Water Reactors (PWR). Basic diagrams of these two types of Nuclear power plants are depicted below. (Figures 2 and 3 respectively) The basic difference between these two types is that a pressurized water reactor has an extra set of major components, steam generators and a pressurizer.

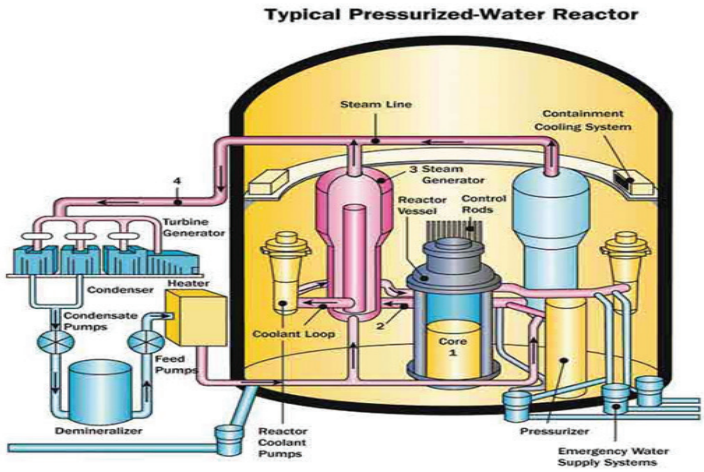


Figure 2. Pressurized Water Reactor

The pressurizer maintains pressure in the reactor side cooling water sufficient to prevent boiling. The steam generator is utilized to allow heat to be transferred from water that has been exposed to the nuclear core to circulating water in the steam cycle. By using a steam generator to transfer heat from the reactor side cooling system, the steam passing

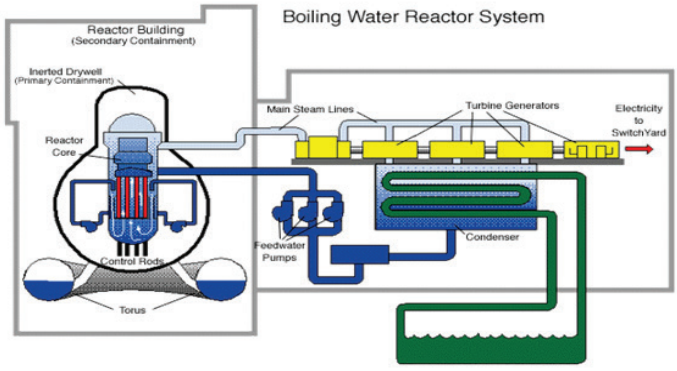


Figure 3. Boiling Water Reactor

through a turbine in a PWR does not get exposed to the Reactor fuel. A PWR design typically provides an environment outside of its primary containment walls free of radiological contamination, while a BWR typically has areas of contamination outside its primary containment.

The Boiling Water design does not have a pressurizer or steam generators. The core itself transfers enough heat to turn cooling water into steam. This is the type of design utilized in all of the plants at Fukishima Daiichi Nuclear station. Since there is steam exposed to the core in a BWR design, by its nature there is one less barrier to contamination when comparing a BWRS to a PWRS design.

Six BWR units at the Fukushima Nuclear Station:

Unit 1: 439 MWe BWR, 1971 (unit was in operation prior to event)

Unit 2: 760 MWe BWR, 1974 (unit was in operation prior to event)

Unit 3: 760 MWe BWR, 1976 (unit was in operation prior to event)

Unit 4: 760 MWe BWR, 1978 (unit was in outage prior to event)

Unit 5: 760 MWe BWR, 1978 (unit was in outage prior to event)

Unit 6: 1067 MWe BWR, 1979 (unit was in outage prior to event)

In addition to the core heat transfer designs, one of the additional design features to consider is the configuration of the pools that store spent fuel. Commercial power plants typically are designed to replace 1/3 of its core at each refueling with new fuel and to store 1/3 of its exposed fuel in a storage pool. Plants store the fuel in a pool with sufficient water to act as a barrier to radiation until the spent fuel has decayed to a level where it can be transferred to a cask for dry storage. The pool is also designed to have spacing the proper fuel and water chemistry to prevent inadvertent criticality.

One difference to note is the elevation difference between the refueling floor at Fukishima and other plants where the refueling floor is at the same or lower level from the reactor core. Many designs recognize that keeping a sufficient level over the spent fuel requires a greater head pressure to fill when the pool is elevated. Designs have to consider many ways of preventing the inadvertent draining or loss of level such as loop seal vents to prevent inadvertent siphoning. Plants also have to consider pool refill and residual heat removal to prevent evaporative losses from dropping pool levels. Typically 8-10 feet over the fuel top is required but most plants keep 20 plus feet as a safety margin. According to The Nation, "Spent fuel pools at Fukushima were not equipped with backup water-circulation systems or backup generators for the water-circulation system they do have."

It is important to note that in both PWR and BWR designs the fuel pools are normally outside the primary containments. Secondary containments can vary significantly in their construction. They range from sheet metal walls to reinforced concrete. They typically are not designed for increased pressures or ability to withstand explosions. The primary containments are much stronger and designed to specific pressure and missile damage requirements.

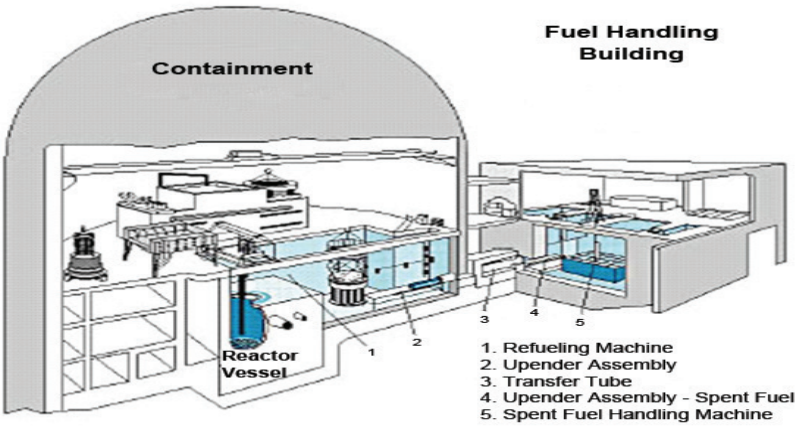


Figure 4. Typical PWR arrangement releative to Spent Fuel pool

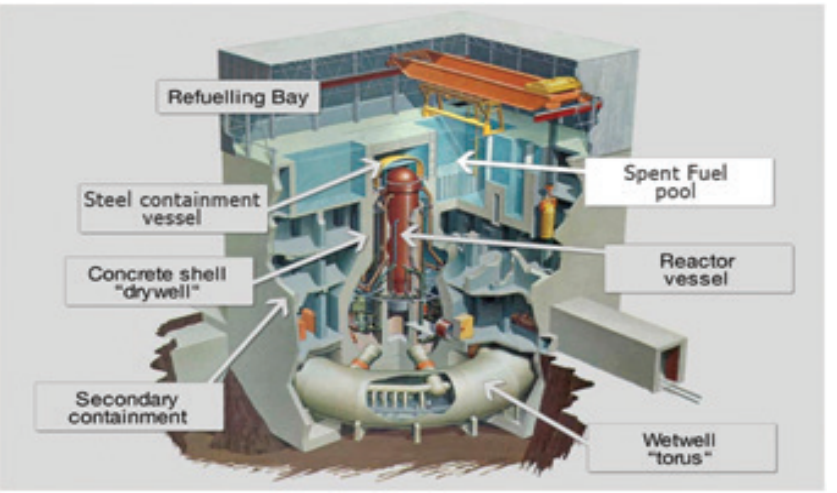


Figure 5. BWR Layout similar to Fukishima Daiichi

Reactor Control System Designs

In both PWR and BWR designs the control rods are designed to insert within 4 seconds from a trip or "scram" signal. They are failsafe in that a loss of power in either system causes the full insertion of rods. In a PWR their weight and springs act to drop the Control rods. In a BWR hydraulic pressure is applied to the rods, to insert from the bottom up, on loss of power.

Emergency cooling systems

A PWR typically has high head injection pumps and old leg accumulators that are designed to automatically inject borated water into the core in case of an emergency. The high head injection pumps require electrical power, the cold leg accumulators are tanks at elevations higher than the core that can empty automatically if the reactor cooling system's pressure drops below the head pressure of the accumulators. PWRs have a residual heat removal capability for long term heat removal from the core, but this system relies on electrical power for pump and valve operation. There are normally piping points where spool pieces can be installed to utilize other water sources to provide emergency water. These typically use water from other storage tanks or via pumps like high pressure fire pumps etc. Many times these are either gravity flow (require reactor system pressures to be low) or diesel powered pumps. For a complete loss of power, with no instrumentation or controls, offsite support would be required.

The BWR is specifically designed to respond to pressure transients, having a "pressure suppression" type of design which vents overpressure using safety relief valves to below the surface of a pool of liquid water within the containment, known as the "wetwell" or "torus". This is the large ring depicted in the illustration. BWRs, like the PWRs, would need electrical power to provide continued injection and residual heat removal should water inventory be lost. Like PWRs, there are normally piping points where spool pieces can be installed so that other water sources can provide emergency water. These typically use water from other storage tanks, or via pumps like high pressure fire pumps. Many times these are either gravity flow (requireing reactor system pressures to be low) or diesel powered pumps. In a complete loss of power situation with no instrumentation or controls, offsite support would be required.

What happened at Fukishima

On 11 March 2011, a magnitude 9 earthquake generated a series of large tsunami waves that struck the east coast of Japan. The earthquake and tsunami created widespread devastation to the northeastern portions of Japan. Over 14,000 lives were lost and 1000s more were missing or displaced.

The earthquake did affect several nuclear power plants: Tokai, Higashi Dori, Onaqawa, and TEPCO's Fukushima Dai-ichi and Dai-ni. This article will discuss the most severe of these events at the Fukishima Dai-ichi Station.

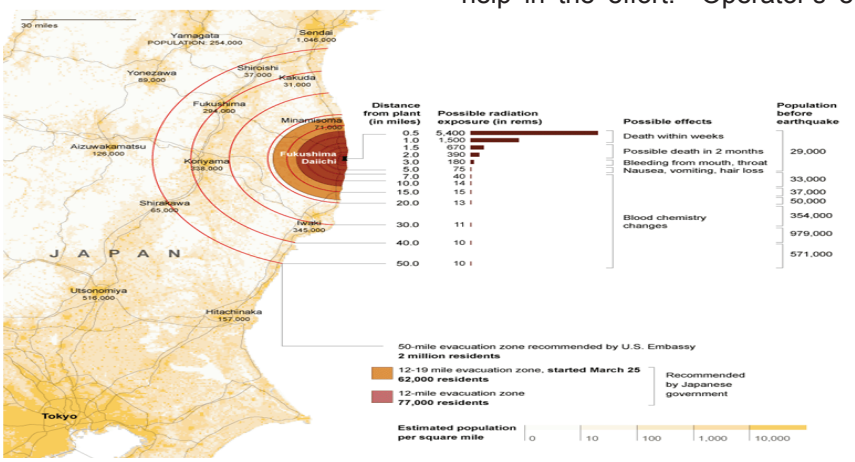


Figure 6. Overviews map showing evacuation and other zone progression and selected radiation levels.

The earthquake initiated a loss of offsite power at Dai-ichi and the automatic systems automatically inserted rods at its three operational reactors. Initiation of these automatic functions was based on detection of seismic activity. Diesel Generators onsite were in operation supplying shutdown power as designed. 46 minutes after the earthquake the first in a series of tsunami waves begin to strike the east coast of Japan. The Dai-ichi plant's design basis accident was for a maximum 5.7 meter wave. Waves that struck the Dai-ichi plant were estimated at greater than 14 meters. Only 1 D/G at unit 6 (B train) remained on power. A/C power and batteries were not available, a long recognized event that is extremely challenging. US procedures have recognized for years that normal emergency procedures are inadequate to handle this situation and have required US plants to develop emergency contingency actions (ECAs) to assist operators in this situation. Much of the guidance in these ECAs direct the site staffs to pursue support from offsite sources while working to restore power. In the wake of severe emergency affecting so many lives outside of the station proper, the Japanese faced an emergency that taxed responders far beyond their planned resource levels. The magnitude of the impact to communication, transportation and emergency personnel for response to the general populace's immediate needs compounded the difficulty in obtaining offsite support. The station operators have been lauded by many of the organizations that subsequently were marshaled to help in the effort. Operator's efforts, heroic at time to restore ability to mitigate the event have been documented. **Following is a summary of the emergency response at the station as described by the Institute of Nuclear Power Operations.**

With no core cooling to remove decay heat, core damage may have begun on Unit 1 on the day of the event. Steam-driven injection pumps were used to provide cooling water to the reactors on units 2 and 3, but these pumps eventually stopped working; and all cooling water to the reactors was lost until fire engines were used to restore water injection. As a result of inadequate core cooling, fuel damage also occurred in units 2 and 3. Challenges in venting containments contributed to containment pressures exceeding design pressure, which may have caused containment damage and leakage.

Hydrogen generated from the damaged fuel in the reactors accumulated in the reactor buildings either during venting operations or from other leaks and ignited, producing explosions in the Unit 1 and Unit 3 reactor buildings and significantly complicating the response. The hydrogen generated in Unit 3 may

have migrated into the Unit 4 reactor building, resulting in a subsequent explosion and damage. The loss of primary and secondary containment integrity resulted in ground-level releases of radioactive material. Following the explosion in Unit 4 and the abnormal indications on Unit 2 on the fourth day of the event, the site superintendent directed that all nonessential personnel temporarily evacuate, leaving approximately 70 people on site to manage the event.

During releases, dose rates as high as 1,193 millirem per hour (mrem/hr) (11.93 mSv/hr) were measured at the site boundary, approximately 0.6 miles (1 km) from units 1&4. Windows for the emergency response center had to be covered with lead shielding to reduce dose rates in the center. Organized offsite radiation surveys began on March 16. Radiation levels off site at that time ranged from 0.1 mrem/hr (1 µSv/hr) to 20 mrem/hr (200 µSv/hr). Thirty-seven miles (60 km) northwest of the station, the dose rate was 0.8 mrem/hr (8 µSv/hr). Water and soil samples indicated the presence of strontium, iodine, and cesium. Food and water restrictions were implemented in some areas as a result of radioactivity. People within the 12.4 miles (20 km) surrounding the station were evacuated, and those living up to 18.6 miles (30 km) away were directed to shelter inside their homes as the releases of radioactive gases and materials increased as the event progressed and more fuel damage occurred. Potassium iodide tablets and powder were distributed to local governments beginning March 21. Because the evacuations had already been completed, the potassium iodide was not issued to the population to reduce radioactive iodine intake.

Radiation surveys of the on-site areas surrounding units 1and 3 showed dose rates as high as 13 rem/hr (0.13 Sv/hr) in areas around units 2 and 3. More detailed surveys performed over the following weeks discovered localized dose rates greater than 1,000 rem/hr (10 Sv/hr) around equipment and debris outside units 1 and 3.

Some personnel who responded to the event received high doses of radiation. Two control room operators received the highest doses a calculated internal and external dose of 67.8 rem (0.678 Sv) and 64.3 rem (0.643 Sv). The majority of dose received by these workers was internal (85-87 percent). Potassium iodide was provided to some station personnel on March 13. As of the end of March, approximately 100 workers had received doses of greater than 10 rem (0.1 Sv).

The Fukushima event was rated as a level 7 event on the International Nuclear and Radiological Event (INES) scale. The Nuclear Safety Commission of Japan estimated approximately 17 million curies (6.3 E17 Bq) of iodine-131 equivalent radioactive material was released into the air and 0.127 million curies (4.7 E15 Bq) into the sea between March 11 and April 5. The 1986 accident at Unit 4 of the Chernobyl nuclear power plant was the only other nuclear accident to have a level 7 INES rating.

Relevance to Army Reactor Program

The US Army's decommissioned reactors and its operating Fast Burst Reactor do not have the catastrophic potential of the magnitudes experienced at the Japanese reactors. There are, however, underlying safety considerations that should be examined by all personnel with roles of responsibility related to sites under the Army Reactor Program's umbrella. When developing safety analysis and evaluations, one should consider the potential for traditional events to exceed design basis. The principles that are ingrained in the Army's nuclear culture include a conservative approach to operations, maintenance and engineering that seeks safety margins beyond just meeting design basis. This safety culture embodies a "Defense in Depth" approach that looks to ensure redundant safety features are second party verifications, be a part of the operating and maintenance procedures, safety analysis and design. This event shows how a common mode failure can create a situation where design basis conditions can be exceeded. One example of a common mode event is the typical design of US plants to consider a 100 year flood level as design basis. Floods or Tsunamis like the Fukishima case can effect redundant trains of equipment and disrupt procedures that operating crews are trained to respond with. One can postulate some other common mode events that even though remote in possibility, could come to bear at sites under Army permits. Fire and security threats are examples where the "Defense in Depth" concepts we use would challenge redundant systems. The continuing professionalism by all in the Army Reactor Program to learn as much as possible about the challenges the Fukishima plant experienced in handling "Beyond Design Basis" events is crucial. The final chapter of the Fukishima saga has yet to be written. We will learn much from this event as we have in previous nuclear accidents. Introspection by those of us in the nuclear field has served us well and will serve us in this regard. Even though the same magnitudes of catastrophic potential do not exist for the Army Reactors, we still have the same professional requirements as the Fukishima nuclear workforce. We have the duty to uphold the public trust that has been placed upon us. The lessons learned from this event will be a valuable tool to address future considerations on handling events that exceed "design basis", stay tuned.

BIOGRAPHY

Mr. Phil Shubert currently heads the Army's Reactor Office. He is a Mechanical Engineering graduate from the University of Tennessee and served in the Navy as a Naval Flight Officer, with over 200 carrier landings on the USS Forrestal. He later served as a Senior Reactor Operator for the Tennessee Valley Authority (TVA), where he supervised the loading of the first fuel assembly at Watts Bar and was Unit Operating Supervisor for the initial critical for Watts Bar in 1996. From 1997-2007 he served with the Joint Warfare Analysis Center (JWAC), a joint command located at Dahlgren Naval Base. From 2007-2011, he was a US manager for Alstom, which is a French multinational conglomerate. Phil returned to US government service in 2011 as the Army Reactor Program manager under the US Army Nuclear and Combating WMD Agency (USANCA).

ABCA-Optimizing CBRN Interoperability

LTC Michael S. Quinn
CBRN Officer, U.S. Army Nuclear and CWMD Agency

"Optimizing Coalition Interoperability"
www.abca-armies.org

The mission of the American, British, Canadian, Australian and New Zealand Armies' Program (ABCA) is to optimize ABCA Armies' interoperability in order to deliver successfullly coalition operations. Initiated in 1947 by America, Great Britain, and Canada, the program was a means to continue fostering the close cooperation that the countries had established during World War II. Australia jointed the program in 1964 and New Zealand in 2006, effectively forming today's ABCA Armies Program which many may know as the "5 Eyes Community".

Strategically guided by an executive council and overseen and directed by national directors, capability and support groups execute the daily activities of the Program. The Program consists of five capability groups: Command, Sense, Shield, Sustain and Act. Capability Group Shield is one of the two groups led by a US colonel. Over the last several years Capability Group Shield, similar to the US protect function, has focused on the interoperability of Counter IED and CBRN Combating /Countering WMD Operations.

Figure 1. ABCA Project Team conducting a critical analysis of national data gathered and submitted before the meeting.

The Shield CBRN sub group recently focused efforts on two interoperability topics: mass casuality decontamination operations during a WMD event and national CBRN equipment capabilities. An ABCA Best Practices Guideline for Military Mass Casality Decontamination Operations During a Domestic Hazmat/ Weapons of Mass Destruction Incident

Figure 2. UK soldiers and airmen, wearing the new GSR, rehearsing CBRN survey techniques at Winterbourne Gunner, UK.

was published as ABCA Publication 370 in Feb 2012 and is available on the ABCA web page. Driven by changes to technology, tactics, techniques, and procedures over the last 10 years, the most recent project, national CBRN equipment capabilities, analyzed four key functional areas: individual protective equipment; detection, identification and monitoring; decontamination; and medical countermeasures in order to reassess interoperability.

Following a four month information gathering period, the United Kingdom (UK) hosted the CBRN sub group project team at the UK's Defence CBRN Center at Winterbourne Gunner, Salisbury, UK for a report writing and training observation week. (Figure 1) The report revealed several key points: first, both the US and UK are moving away from the NATO standard threaded protective mask canister filter with the fielding of the M50 series mask (US) and the General Service Respirator (UK) while Canada, Australia and New Zealand are maintaining the C-4 and S-10 protective masks respectively. Second, the US is introducing two new pieces of equipment: a low volatile hazard detection capability to the M256A1 kit which is being fielded as the M256A2 and an additional flag marker, "Toxic", to the CBRN marking kit.

On the surface, these two new pieces of equipment may appear minor, however the significance of highlighting both the M256A2 kit and flag marker are twofold: ABCA partners are made aware that these capabilities exist and are available through the national stock number system (NSN) and second, ABCA Standards require modification in order to define the new equipment for situational awareness and interoperability during coalition operations.

Understanding how each country equips, trains, and mans their CBRN force is critical function of the ABCA Shield team in order to enhance interoperability. The Royal Air Force (RAF) is the CBRN proponent for the UK military and as of April 2012, the RAF assumed total responsibility for the UK's CBRN Regiment at RAF Honington. The UK's former Joint CBRN Regiment was commanded by 1st Royal Tank Regiment with two army troops of Light Role (similar to Tech Escort CRT) and Fox Recon teams and two RAF squadrons of bio detection systems. The RAF now provides all CBRN specialist capability to the UK Army in support of operations. While at the Defence CBRN Center, the Project Team toured the instruction facilities, mask confidence exercise facility as well as observed soldiers and airmen conducting CBRN enhanced specialist training. (Figure 2) The visit provided valuable insight to the UK's CBRN transformation and training concepts, providing each ABCA nation better situational awareness when they address future interoperability issues.

For access to ABCA Publication 370, ABCA Best Practices Guideline for Military Mass Causality Decontamination Operations During a Domestic Hazmat/ Weapons of Mass Destruction Incident, and additional ABCA interoperability products, log on to www.abca-armies.org. Once at the site, select "register" to submit a request for a password in order to view all ABCA documents.

BIOGRAPHY

LTC Michael S. Quinn is a CBRN Officer assigned to the US Army Nuclear and Combating WMD Agency's Analysis Division. He has a BS in Chemistry from Auburn University and a MS in Health Sciences from Touro University. He was previously assigned as the Corps CBRN Officer, Allied Rapid Reaction Corps (NATO) and BN XO & S3, 22nd Chemical BN (TE). His email address is michael.s.quinn@us.army.mil

Figure 3. Shield Project Team. (l-r) LTC M. Quinn (US), Mr. J. Pool (US), Mr. B. Parker (US), CSM P. Taylor (UK), WO3 J. Stinnett (USMC), MAJ M. Pettersen (NZ), MAJ M. Robertson (CA), LTC M. Boggs (NZ)

Using Google Earth and Primary Ray-Tracing to Model Thermal Effects of a Surface Burst

Mr. Matthew Jackson
Nuclear Engineer, U.S. Army Nuclear and CWMD Agency

Introduction and Limitations of Models

Most software tools that perform prompt calculations do not take terrain or line-of-sight effects into account. In the case of thermal effects for a nuclear explosion, this can prove to be a major limitation in an urban environment. Since the thermal radiation outpaces the blast wave, it will become incident on the surface of the structures/equipment before the blast wave can translate or collapse them. The more robust manner to address this would be to create 3D grid, trace the rays from the point source, compute transmission coefficients for the ambient air and the materials forming the various structures, and compute primary and secondary rays. However to get a reasonably accurate solution, we could choose to only trace the primary ray, assume 100% absorption by the wall, which will give a rough picture of the effect of structures on thermal effects. For this purpose we can use a simplistic, circular, grid. Rays will be sent out from 0 to 360 degrees, any buildings intersecting these rays will be flagged, and their intersection point computed. In Google Earth it is relatively easy to draw in polygons to define a structure, with the limitation that all 3D structures will be modeled as prisms.

Some of the primary limitations of the model are listed below. The model does not currently take terrain elevation into account; however this could readily be achieved by modeling key features as polygons, or by incorporating Digital Terrain Elevation Data. It is also assumed that all structures modeled as polygons are provided as a list of points in ordered pairs (as they would be if originating from Google Earth). The explosion is currently modeled as a point source and is implemented as a 2D solution. Rise and change in diameter of fireball is ignored as well.

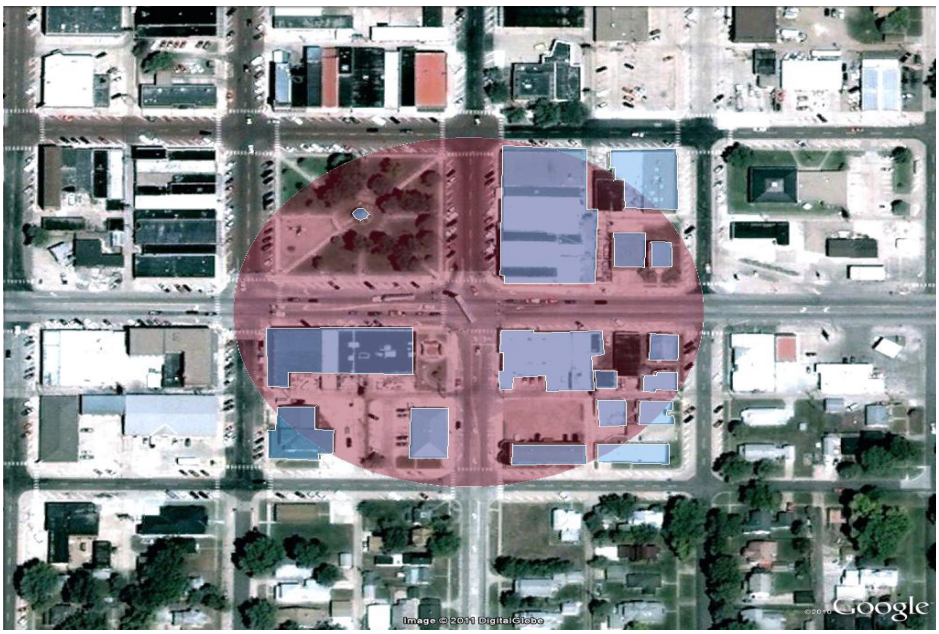


Figure 1. Thermal Effects Plot without Ray Tracing (©2011 Google, ©2011 DigitalGlobe)

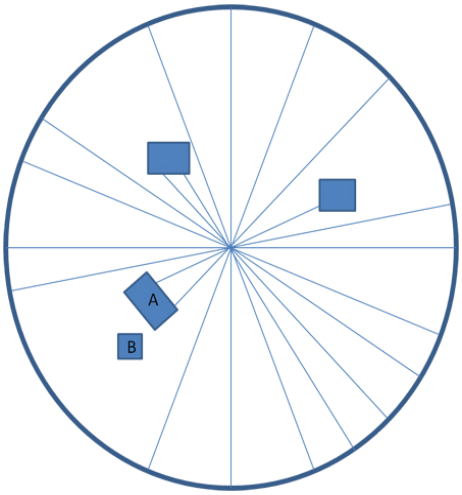


Figure 2. Example Grid

Background Relations and Definitions

Let R be defined as the radius of effect, a radius defined by the user based on calculations gleaned from equations provided by EM-1 or similar resources. Let V1 represent the vector formed from the source to a node on the circumference of the effect radius and let V2 represent the vector formed from two

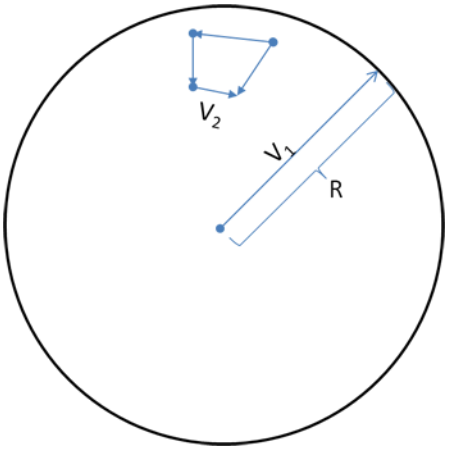


Figure 3. Definition of Vectors $V1_1$ $V2_2$ and R

points forming a segment of a structure.

Before delving into the details of the algorithm, several mathematic functions must be defined. As mentioned earlier, one of the assumptions of the code is that all polygons defined must be ordered pairs. In most grids, outer boundaries are oriented counterclockwise (CCW). Consider figure 3: the square block represents a structure, and the circle represents the desired thermal effect. The outer boundary must remain counterclockwise, yet upon removing the section of the circle blocked by the structure, the segment highlighted in red, while CCW relative to square polygon, is clockwise relative to the outer boundary. This means that when reading in the boundary points which define the building, they should be tested for orientation (counterclockwise or clockwise). The easiest way to do this is to take the cross product of the two vectors forming the points; if the cross product is less than zero, the points are ordered clockwise, otherwise they are CCW and their ordering must be reversed, or in mathematical

$$\overrightarrow{V_1} \times \overrightarrow{V_2} > 0 \qquad (1)$$

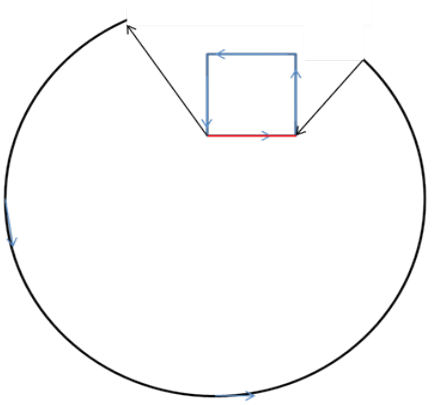
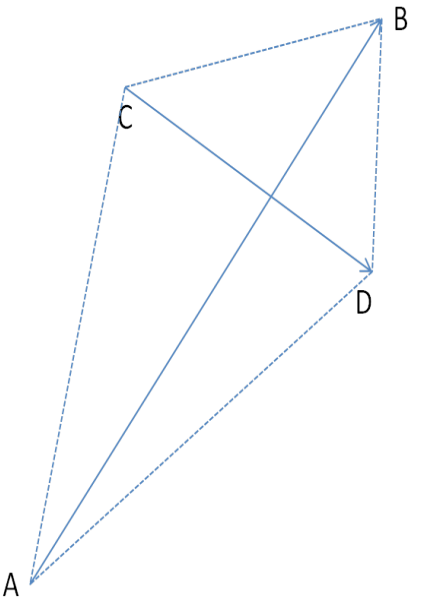


Figure 4. Structure Blocking Thermal Rays

terms segments are ordered CCW if: For the purpose of ray-tracing, it is also necessary to determine whether one line intersects another, and if so, where that intersection point lies. There are a few ways to determine whether two lines intersect, but the easiest way is to simply take advantage of the CCW algorithm defined above. Following the methodology outlined by Dr. Jeff Erickson1, two line segments (Figure 4) intersect only when:

$$CCW(A,C,D) \neq CCW(B,C,D) \;\&\; CCW(A,B,C) \neq CCW(A,B,D), \qquad (2)$$



where CCW is defined in equation (1).

Equation 2 will be used to flag edges that intersect, but the intersection point also needs to be computed. Let two lines exist for the 4 coordinates (P_1 -> P_2, P_3->P_4):

$$L_1 = aV_1 + P_1$$
$$L_2 = bV_2 + P_3 \qquad (3)$$

The intersection point occurs where L1 = L2:

$$aV_1 + P_1 = bV_2 + P_3$$
$$a\,V_1 = bV_2 + (P_3 - P_1)$$
$$a(V_1 \times V_2) = b\,(V_2 \times V_2) + (P_3 - P_1) \times V_2$$
$$a(V_1 \times V_2) = (P_3 - P_1) \times V_2$$
$$a((V_1 \times V_2) \cdot (V_1 \times V_2) = ((P_3 - P_1) \times V_2) \cdot$$
$$a = \frac{(((P_3 - P_1) \times V_2) \cdot (V_1 \times V_2))}{((V_1 \times V_2) \cdot (V_1 \times V_2))} \qquad (4)$$

$(V_1 \times V_2)$

Substituting (3) into equation (4) yields the intersection point P:

$$P = aV_1 + P_1 \qquad (5)$$

In the scenario defined in figure 2, P1 would represent the coordinate defining the location of the source, and P3 the first coordinate of a line segment along a structure boundary.

When using latitude and longitude for coordinate pairs, it is necessary to take into account the coordinate system when computing the distance. Google Earth uses the WGS84 coordinate system, which defines the following variables (all lat/lon should be converted to radians first)2:

a=6378137.0

$$E = \sqrt{\frac{1-b^2}{a^2}} \qquad (6)$$

$$R_{earth} = \frac{a(1-E^2)}{(1-E^2\sin^2(latitude))^{1.5}} \; [meters]$$

b=6356752.3142

Using the spherical law of cosines, the distance between two latitude/longitude pairs { $[lat_1,lon_1],[lat_2,lon_2]$ } can be calculated:

$$d = \arcsin(\sin(lat_1)*\sin(lat_2)+\cos(lat_1)*\cos(lat_2)*\cos(lon_2-lon_1))*R \qquad (7)$$

For this model, the outer boundary is defined by a circle (in theory it could be defined differently if a geometry for the outer boundary is more appropriate). To compute the coordinate at the outer radius of a circle at a given angle θ (assumed in radians), the following

$$lat_{circ} = lat_{src} + \frac{180*R\sin\theta}{R_{earth}*\pi} \qquad (8)$$

$$lon_{circ} = lon_{src} + \frac{180*R\cos\theta}{R_{earth}*\pi*\cos(lat_{src}*\frac{\pi}{180})}$$

relations may be used:

Determining Location of Structures

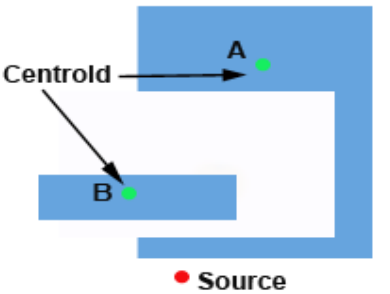


Figure 6. Centroid of Unique Geometry

Relative to Source

Buildings are positioned in a plethora of orientations relative to the source. Ray tracing can be used to determine which buildings intersect a given ray, but has no inherent way of determining if one structure is shielded by another (see Building B in Figure 6). Fortunately, this can be accomplished by ranking the buildings by their distance from the source. There are at least two ways to do this: the most accurate would be to compute distances along the edges till the minimum length was found. A quicker, and for most cases, sufficiently accurate method, is to compute the distance from the centroid of structure to ground zero. For the purpose of simplicity, the latter was chosen. It is possible for unique geometries (figure 5) that a building with a more distant centroid (Building A) could be shielding a building with a less distant centroid (Building B). A computationally expensive way to correct this would be to loop through all structures, determine the closest point of intersection f_k for each building for a given angle θ:

for θ=0 to 2π

$\vec{u}=\{lon_{src}+R_{eff}\cos(\theta), lat_{src}+R_{eff}\sin(\theta), 0\}$

for j=0 *to Number of Buildings*
for i=0 *to Number of Edges in Building j*

if $\vec{v_i}$>*intersection(edge i,* $\vec{u}$*)*

$\vec{v_i}$>*intersection(edge i,* $\vec{u}$*)*

end if

end loop i,j

$f_k(\theta)=\min(\vec{v_i})$

end loop θ

[A.1]

To gain some additional utility out of the process identified above, the edges on each building could also be reordered by distance from source, eliminating the need to loop through all edges for each angle where an edge is found to intersectV_1.

Intersection Identification and Constructing the Boundaries of the New Polygon

In prior sections the methodology for identifying segments that intersect and their intersection point was discussed. Additionally, algorithms for computing the distance for lat/long pairs was discussed. The generic algorithm for drawing the thermal effects polygon would be:

1. Read in building data (from kml or other source).
   a. Check whether elements are oriented clockwise or counterclockwise
      i. If counterclockwise, reverse directions of ordered pairs
   b. Compute centroid of building (or skip if using algorithm A.1)

2. Rank buildings by distance, either using algorithm A.1 or by computing distance from source to centroid.

3. Determine coarseness of mesh and construct array to hold angle, an integer value to flag angles that intersect a structure, as well as the {*x,y,z*} coordinates for the intersection of the structure and V1.

4. Loop over all angles, compute V1.
   a. Loop over all buildings
      i. Loop over all building edges
         - Compute V_2

         - If angle has not been already flagged as intersected by a building, check for intersection of V_1, V_2 (Eq. 2), and if true, compute intersection point (Eq.3-5).

         - Store intersection point with smallest distance from source in array constructed in step 3 (Eq. 6-7).

5. Create polygon array to store lat, lon coordinates of effect polygon.

6. Construct polygon by looping over all angles

   a. If angle has been flagged as intersecting a building, store {x,y,z} intersection coordinate stored in array of step 3 in the array created in step 5.

   b. If not, compute the lat, lon at circumference using Eq. 8 and store in the array of step 5.

7. Write file to kml (note kml expects coordinate pairs to be in the order {lon, lat, z}.

Results

A program was written using the algorithm outlined in the previous section. The program takes the source coordinates, HOB, radius of effect, and lat/lon as inputs. As mentioned earlier, this initial implementation is only 2D, thus it would be invalid for HOB >> 0. At HOB above the average building heights the projection of shadowing onto the ground must be computed, as opposed to simply using the intersection point of the thermal ray with the building edge.

For the plot in figure 8, a hypothetical thermal effect with a radius of 100m was modeled. Rough outlines of buildings in the area were drawn in Google Earth and saved as a kml file, which was read by the algorithm outlined in the previous section, and used to compute the boundary of the effect polygon. At the beginning of this article, a thermal effect circle was drawn, ignoring all terrain and building interaction; but as seen in figure 7, a large area that was originally predicted to be within the thermal effect, was in fact a gross overestimate (excepting the upper right quadrant), while in other quadrants the presence of structures rendered the radius of effect largely a meaningless quantity. Thus by implementing this simple algorithm, it was possible to re-shape the area expected to be impacted by a given thermal effect

Future Work

The logical first step would be to extend the algorithm to three dimensions, and modify the code to support airbursts. This would primarily involve modifying the intersection code to project the results from the intersection point to the surface (or a fixed height above the ground). It would also be useful to determine a data source for altitude and use that to model the terrain as well as the buildings. Third, adding in support for translucent structures could potentially be used to model trees and other foliage which serve as a partial screen to incident rays. Finally, it would prove useful to utilize the delphic rise model to account for fireball shape and rise, which in turn would provide a more comprehensive model for the thermal footprint on a given landscape.

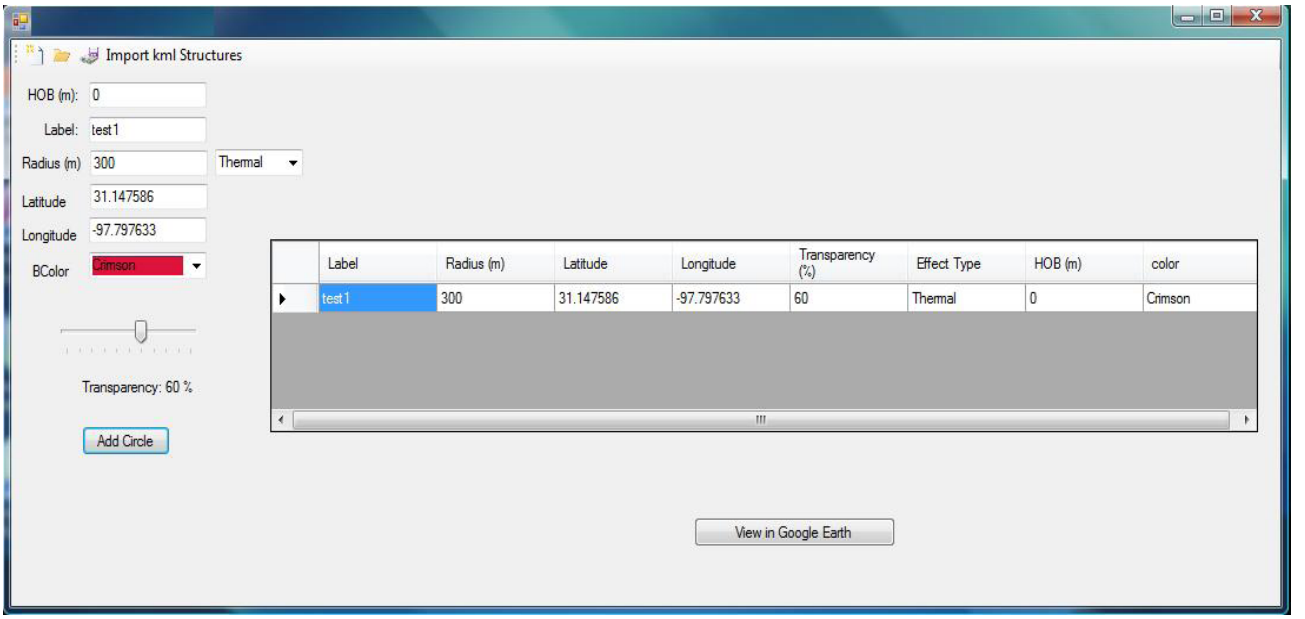


Figure 7. Thermal Effects Program Input

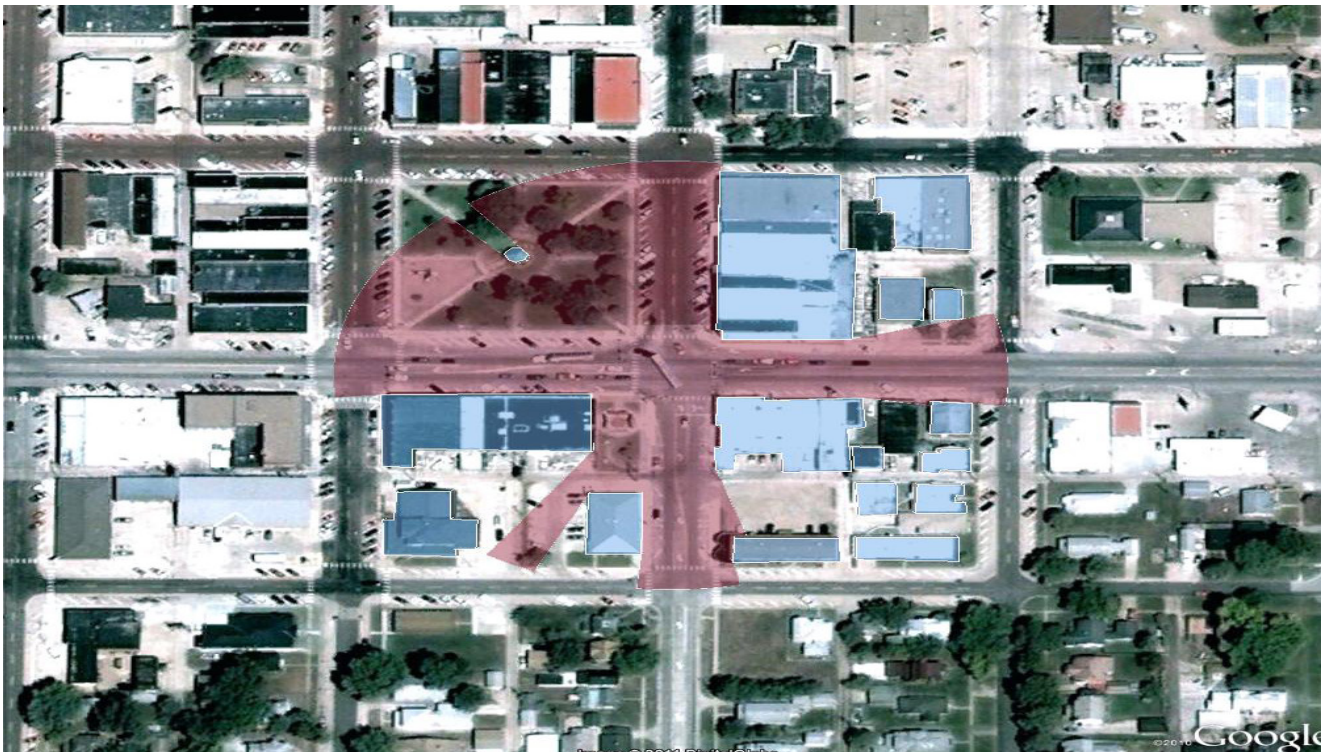


Figure 8. 100m Thermal Effect with Ray Tracing (©2010 Google, ©2011 DigitalGlobe)

BIOGRAPHY

Matthew Jackson is a nuclear engineer at the U.S. Army Nuclear and CWMD Agency, and can be reached at matthew.r.jackson3@us.army.mil or 703-806-7867 should any questions arise from this article.

Toxin Weapons: What's the Worry?

Mitchell Wise, PhD
U.S. Army Nuclear and CWMD Agency

Toxins are poisonous metabolites produced by living organisms. Although classified as biological warfare agents by the 1972 Biological Warfare Convention they lack the ability to reproduce, as is the case with traditional biological agents like Bacillus anthracis (the causative agent of the disease anthrax). Thus, toxins are frequently claimed as chemical warfare agents as well as biological agents. However, the DoD classifies all toxins, including those produced by microorganisms or plants, as chemical agents. Owing to their complex structures, chemical synthesis of these agents is either not possible or impractical for production on an industrial scale. Many, if not most, toxins are formed as proteins or peptides (short chain length proteins) and are produced by representatives of all the major kingdoms of life. Mycotoxins are one exception to this general rule. These are structurally complex organic compounds produced by fungi.

Advances in biotechnology have further blurred the distinction between biology and chemistry. Genentech, a San Francisco area biotech firm created in 1976, is a pioneer in the biotech industry. Scientists at Genentech successfully engineered a recombinant version of the human protein insulin into bacteria, an invention that has not only changed the life of millions of diabetics worldwide but has also heralded the industrial-scale manufacture of protein pharmaceuticals. It is now possible to produce thousands of pounds of insulin annually in a single plant. This technology is adaptable to virtually any protein and, similarly, to production of other complex biochemicals.

Small-scale toxin production does not require the sophisticated materials and engineering efforts necessary for chemical weapons production; indeed, crude preparations of small quantities of toxins are not beyond the capabilities of a Master of Science level biochemist or microbiologist or even lay persons. There have been several instances in recent years where ricin, a toxin that can be extracted from beans of the castor oil plant, has been used or its use attempted by fringe terrorist groups in the US. However, on an industrial scale, biological toxins require a substantial input of technology. Converting these toxins into weapons capable of widespread dissemination to produce mass casualties requires financial support unlikely to be found at less than a State level.

This article will examine the potential development of toxins as weapons of mass destruction (WMD) and explore the rationales for such development. Although there are literally thousands of biological toxins known, the focus here will be on four that many experts believe have the greatest potential for weaponization: 1) botulinum (neuro) toxin (BoNT); 2) the mycotoxin T2; 3) ricin; and 4) staphylococcal enterotoxin B (SEB).

In examining the potential for toxins as WMD it might be worthwhile to first enumerate the quantities of these toxins required for battlefield employment. According to Franz [1] it would take about 8 kg (kg = kilogram or 1,000 grams) toxin per 100 m^2 battlefield area for BoNT, the most potent toxin known to man, and about 800,000 kg per 100 m^2 for T2 toxin, the least potent of the toxins considered here, to effectively contaminate a battlefield. Even considering the advances in bioengineering, the production of these quantities is a daunting technological challenge.

Botulinum Toxin. BoNT is reputed to be the most toxic substance known (in theory a single gram could kill a million people). Intoxication by BoNT can occur by ingestion, inhalation or direct entry into the blood stream (through injection or an open wound). The estimated LD50 for humans (lethal dose for 50% mortality rate) is about 1 ng toxin per kg body weight by intravenous (or intraperitoneal) injection and only slightly less, 3 ng toxin per kg, by inhalation[2] (ng = nanogram or 1/billionth of a gram)[3]. The time period after exposure is highly variable and depends on route of entry. Foodborne cases may present symptoms anywhere from 2 hours to 8 days but typically in 12 to 72 hours after ingestion. Inhalation cases occur in as little as 12 hours in test monkeys administered high doses. The few known human cases of inhalation poisoning resulted in symptoms after 3 days [4]. But, as these were laboratory accidents, they undoubtedly resulted from very low doses.

Interestingly, BoNT was the first toxin to be registered with the FDA as a therapeutic agent; originally used as a treatment for strabismus (crossed eyes), it has subsequently found application for additional neuromuscular disorders and, more famously, for cosmetic purposes. Thus, commercial production of the toxin is accomplished by fermentation of the bacterium Clostridium botulinum (Hall strain); improved efficiencies in large-scale production for this commercial product are inevitable.

Fortunately, in terms of biodefense, the physicochemical properties of BoNTs render the toxins susceptible to chemical and environmental degradation. BoNTs are inactivated by disinfectants such as sodium hypochlorite (Clorox®) and they are denatured at elevated temperatures. The Centers for Disease Control (CDC) recommended protocol for inactivation of BoTN contamination of surfaces is 30 minutes exposure to sodium hypochlorite (> 0.1%) or sodium hydroxide (> 0.25 N). The CDC also reported that ozone treatment (> 2 mg per L) effectively inactivates BoTN in solution. The toxin is also destroyed by heating at 80oC for 30 minutes or 100oC for 10 minutes.

Although BoTN can be effectively inactivated by chemical or heat treatment, the thermal stability and bioavailability of BoNTs are also influenced by its purity and by associated (non-toxic) "neurotoxin-associated proteins" (NAPs)[5]. Production of BoTN by C. botulinum typically involves concomitant production of these proteins which provide some protection against degradation by gastric acid and enzymes[5]. Thus, a terrorist attack might well involve use of crude preparations of NAP containing BoTNs that would likely increase the potency of the toxin. Association with matrix proteins also affects the thermal stability of BoNTs and may be critical in detoxifying commercial food products or preventing terrorist attacks on the food system.

BoNT (as well as spores of C. botulinum) can be decontaminated with gaseous formaldehyde at 0.3 to 0.6 g per ft^3 for 2 to 48 hours. Aqueous (seawater) solutions of formaldehyde were used for large-scale decontamination of Bacillus anthracis from areas of Gruinard Island, the location of biological warfare tests during World War II[6]. Decontamination of BoNT with ethylene oxide is not effective. It would be of interest to determine if ozone is an effective decontaminate.

The extreme toxicity and relative ease to aerosolize are characteristics of BoNT that make it attractive as a bioweapon. Efforts to weaponize this toxin, however, were abandoned by the US prior to termination of the biological weapons program in 1969 due to its instability. There is documented evidence that the Iraqis likewise pursued efforts to weaponize BoNT with similar difficulties[7]. The prospect of terrorist employment of BoNT was recently modeled for contamination of the supply chain of milk and the efficacy of standard pasteurization for its decontamination. These investigators determined that 1 g of toxin could poison 100,000 individuals and that 10 g could affect 500,000 people, most of whom would be children [8]. This analysis has, however, been disputed[9]. Highly sensitive and rapid detection methods for botulinum are currently available, such as the M1R electrochemiluminescence assay produced by BioVeris and deployed by the DoD[10].

Efforts to produce an effective vaccine for BoNT were initiated as early as 1924. These early efforts were oriented around development of an immunogenic toxoid, i.e. a variant of the BoNT protein rendered non-toxic by treatment with formaldehyde. During World War II the Allies re-intensified efforts to produce a BoNT toxoid vaccine, succeeding in producing a reasonably effective bivalent vaccine (i.e. effective against two sero types (A and B) of the BoNT) shortly after the conclusion of WW II. In the 1970s, under contract with the DoD, the Michigan Department of Public Health (MDPH) developed a pentavalent vaccine providing immunity for up to a year against serotypes A through E. In recent years vaccines have been produced using recombinant protein fragments, i.e., a portion of the coding region of the BoNT gene that has been "cloned" into another microbe (either bacterium or yeast) to artificially produce high levels of the specific protein that retains the immunogenicity but not the toxicity of the BoNT. These cloned fragments can then be used, in the proper formulation, as vaccines[11]. Dynport announced in January 2012 successful completion of phase II clinical trials for their recombinant vaccine. This was timely because the CDC and Prevention terminated the use of the MDPH pentavalent vaccine in November 2011 for concerns about its loss in effectiveness due to storage for more than 30 years.

T2 Mycotoxins. At the other extreme of toxicity are the trichothecene (pronounced tri-kó-thee-seen) mycotoxins. In considering mycotoxins as potential weapons the history of their discovery is of some note. After several thousand citizens of the Soviet Union died as a result of consuming fungal contaminated wheat during WW II, Soviet scientists discovered mycotoxins as the likely cause. In the late 1970s and early 1980s the Soviet Union was accused of employing weaponized T2, termed "Yellow Rain", against isolated tribal peoples in South East Asia and Afghanistan. Although there is some evidence that T2 was employed, these allegations are hotly disputed by several reputable investigators. See J.B. Tucker[12] for an excellent review of this controversy. Practical employment of T2 as a battlefield WMD is, nevertheless, questionable[13].

Although several related mycotoxins are known, T2 is one of the most toxic and, hence, most likely to be developed as a weapon. These metabolites are low molecular weight (250 to 500 Daltons), essentially non-volatile compounds produced by certain filamentous fungal species. While T2 is typically produced by species of Fusarium, trichothecenes are also produced by species of Cephalosporium, Myrothecium, Trichoderma and Stachybotrys. Unlike the other three toxins discussed here, trichothecenes can be absorbed through the skin and they are particularly stable to heat. Temperatures in excess of 200 oC may be required for their complete inactivation. The trichothecenes are derived from a group of natural compounds known as sesquiterpenes and are characterized by an epoxide ring at the C13-C14 position as well as an olefinic double bond at the C8 position (Fig 3). The highly reactive epoxide group is critical to their toxicity. The various trichothecenes are differentiated by substitutions around the trichothecin ring; these substitutions all contribute to one degree or another to the toxicity of the various forms.

The trichothecenes are highly effective inhibitors of protein biosynthesis. In laboratory cellular assays T2 exerts its effect within 5 minutes. T2 also inhibits DNA synthesis in mice and rats, although to a lesser degree than protein synthesis[2]. The pathology in humans depends on both route and magnitude of exposure. Acute dermal exposure results in formation of severe irritation and blisters on the skin that may last for weeks. Respiratory exposure results in extreme irritation of the throat with coughing and deep chest pain.

As a condition that has been linked to Fusarium contaminated grain, alimentary toxic aleukia (ATA) results in necrotic lesions in the mouth, esophagus and stomach with accompanying leucopoenia (reduced white blood cell count) and frequently death. Putative victims of "yellow rain" also suffered severe gastrointestinal bleeding, diarrhea and death.

The estimated LD_{50} for T2 in humans is approximately 1,200 µg toxin per kg body weight by inhalation and approximately 25 to 50 µg per kg by ingestion (1µg = 1/millionthof a gram). In mice the LD50 for intravenous injection of T2 is

4.2 to 7.3 µg per kg, which approximates the LD50 (6.6 µg per kg) for dermal application. For monkeys the LD50 for dermal application of T2 in DMSO is >8.0 µg per kg 2. Thus, as a vesicant T2 is approximately 400 times more toxic than the chemical agent mustard[2].

As indicated previously T2 is particularly heat stable, thus the principle means of decontamination is through chemical treatment. Hypochlorite (household bleach), particularly in an alkaline solution, is regarded as an effective decontaminant. Although some reports have indicated that hypochlorite does not alter the epoxide ring, the combination of alkaline hydrolysis of side group bonds and oxidation by the hypochlorite appears to substantially reduce the toxicity of any resulting by products. One particularly innovative study demonstrated that aqueous ozone was an effective decontaminant. Ozone is highly reactive with carbon double bonds and appears to further react with the trichothecene degradation products to form smaller molecules not detected by the usual analytical methods (UV spectroscopy or mass spectrometry). The trichothecene compound deoxynivalenol, which is a common contaminant of wheat, barley, and maize, is also sensitive to aqueous sodium bisulfate. There are additional reports on enzymatic degradation of trichothecene mycotoxins. However, none of these enzymatic processes is currently practical for military applications. Physical methods such as gamma or microwave irradiation have only shown limited success.

Effective decontamination of T2 or other trichothecenes should be easily accomplished with the current inventory of military decontaminates (DF-200, STB, DS-2), collateral material damage not withstanding. Direct experimental demonstration of these processes on representative surfaces such as chemical agent reactive coating (CARC) or the fabric in all purpose combat uniform (ACU) etc. is needed.

Ricin. Ricin is a toxin derived from beans of the castor plant (Ricinus communis L.) and has been the subject of scientific study since the mid 19th century. Indian farmers have, for centuries, fed small amounts of seeds of the abrus plant (which makes abrin, a toxin similar to ricin) to calves as a crude means of immunizing them from toxic beans that could be ingested during grazing. Paul Ehrlich, a renowned immunologist of the early 20th century, used this same toxin in pioneering research on the mechanisms of humoral immunity and antibody action.

Ricin is a dimeric glycoprotein (a protein chain with attached sugar residues) composed of A and B subunits with a reported molecular weight of approximately 64,000 kDa. The protein is translated from a single gene as a single protein which is subsequently cleaved within the plant cell into two separate proteins held together by a disulphide bond. The DNA coding sequence for ricin has been determined and both the A and the B subunits of ricin have been cloned and heterologously expressed in bacteria. The A subunit is responsible for the toxicity of ricin through its activity as a RNA glycosidase that inactivates ribosomes (molecular structures required for protein synthesis). The B subunit is a lectin which binds to carbohydrate residues on the exterior of target cells and facilitates entry of the A subunit into the interior of the cell.

Ricin can be absorbed into the body by inhalation, ingestion or injection into the circulatory system. Inhalation and injection are the most lethal routes, with an estimated LD50 for humans of 3 to 5 µg per kg (based on animal studies). Death by inhalation involves hypoxia from pulmonary edema and alveolar (lung) flooding[2]. Intravenous administration results in intravascular coagulation and lesions of the kidneys and liver, with the exact cause of death not clearly understood. Rat toxicological response to ricin exposure by inhalation is delayed, with the onset of clinical symptoms appearing 8 to 12 hours after exposure. A similar delayed response was observed with primates[2].

While ingestion of ricin can also induce toxicity, the toxic dose is approximately 100-fold greater than the dose for inhalation or injection toxicity. The toxin may be poorly adsorbed by or degraded in the digestive track. Nevertheless, it poses a potential threat from deliberate contamination of food sources.

Castor beans are abundant and used for the industrial production of castor oil which is used as a laxative and as an ingredient in numerous industrial products including lubrication oils, glues, detergents and dyes. The annual production of castor oil world-wide is in excess of 1.2 million metric tons and, depending on the extraction process, the mash waste from castor oil production can also yield significant quantities of ricin. Interest in the use of ricin as a chemical agent by the US and other nations began during WW I. During WW II the US produced a weapon system capable of delivering aerosolized ricin, but it was never employed and subsequent efforts to improve the system were abandoned. Because the raw material for ricin is readily available and extraction of the toxin is relatively simple, there is concern that it can be employed as a terror weapon. There is evidence that Iraq attempted to weaponize ricin. Indeed there are numerous media reports of sundry radical groups attempting to use ricin in terrorist attacks. The August 12, 2011 New York Times, for example, published an article describing efforts by the Yemeni branch of al-Qaida to produce significant quantities of ricin. According to the New York Times report these Islamic terrorists have been importing large quantities of castor beans and appear to be attempting to pack the extracted ricin with small explosives to produce an improvised toxin devise for employment in a confined space such as a shopping mall, airport or subway station.

Ricin, like most proteins, is thermolabile. Although somewhat more stable than botulinum toxin, it is nevertheless denatured at temperatures above 80ºC[14]. Decontamination of ricin on surfaces of militarily relevant materials (polycarbonate, butyl rubber, steel and Tyvek®) was demonstrated with hypochlorite (Chlorox®) and the commercial decontaminate Peridox®, a mixture of hydrogen peroxide, peroxyacetic acid and acetic acid[15].

Animal studies demonstrated that ricin strongly induces an immune response and that both passive and active immunity can mediate the toxicity of ricin. In fact, Soligenix, a development stage pharmaceutical company, has recently licensed an experimental vaccine,

RiVax, that was developed at the University of Texas Southwest Medical Center. RiVax contains the ricin A subunit that is genetically engineered to retain its immunogenicity thus providing protection while not possessing any toxic properties[16]. Phase I clinical trials proved successful and the company is currently seeking Food and Drug Adminstration (FDA) approval for status as an "orphan drug," a classification designated for treatments of rare diseases allowing for various incentives from the FDA.

Because ricin is essentially untreatable and the course of its pathology irreversible by about 8 to 24 hours after inhalation and because vaccines for the general public are unlikely in the near term, post exposure treatments are being sought. Two approaches appear viable: (1) intervention with small molecule inhibitors of the A chain enzyme activity and (2) passive immunization with anti-ricin antibodies. Both these approaches are showing promise but are far from the clinical trial stage.

Like BoNT, ricin has found a place in the pharmacology arena. The notion of a "magic bullet" drug to specifically target and kill aberrant cells or tissues such as cancer was posited by Ehrlich at the turn of the 20th century. Over the last 40 years these magic bullets have been developed in the form of immunotoxins (IT). ITs are agents that combine an antibody or recombinant cell binding portion of an antibody to a toxic compound. Bacterial and plant toxins such as ricin have been extensively employed in ITs. Advances in understanding the molecular structure and function of these toxins as well as the biotechnology required to manipulate and produce large quantities of this protein have resulted in therapeutic application of ricin. From a bioweapons standpoint, research and development of ITs using ricin might be exploited to mask weapon development. Detection of ricin can be accomplished fairly rapidly (< 15 min) by immunosorbent assays or electrochemiluminescence methods[10].

Staphylococcal Enterotoxin B. Staphylococcal entertoxin B (SEB) is one of more than 20 serologically distinct proteins produced by strains of Staphylococcus aureus. These also include SEA, SEB, SEC1-3, SED-E, SEG-K, and SEU. Although the toxin is excreted from the producing organism and might rightly be termed an exotoxin, the "entero" term results from the fact that they are adsorbed through the gastrointestinal tract[17]. Based on the cDNA sequence of a cloned SEB, it is a 239 amino acid protein, with a molecular weight of 28,300. Like botulinum toxin, SEB is expressed as a single peptide (without post-translational modification) that is highly susceptible to proteolytic cleavage by trypsin. The cleaved proteins remain associated through an internal disulphide bond and retain all the immunological and toxic properties of the parent protein. The enterotoxins, as well as other related exotoxins, are termed superantigens due to their ability to tightly bind major histocompatibility complex (MHC-II) receptors on antigen presenting white blood cells (e.g. macrophages,T-cells, etc). They also avidly bind T-cell antigen binding receptors. By executing both these activities simultaneously they circumvent the normal antigen recognition mechanism.

Up to one in five T-cells may thus be activated (compared to one in 10,000 in a normal immune response) resulting in massive up-regulation of various immunological regulatory factors (cytokines) such as: interferon gamma, interleukin-6 and tumor necrosis factor-alpha[2]. Symptoms of SEB intoxication can be induced with as little as 30 ng, but the LD50 for inhalation exposure is estimated to be 0.02 µg/kg, however an incapacitating dose is estimated at 0.0004 µg/kg[2]. These estimates are highly variable between individuals, likely due to genetic variation in the MHC-II receptors.

Exposure results in fever, headache, myalgia, coughing, and nausea with vomiting and diarrhea through oral exposure. Symptoms, depending on dose, may occur in 2 to 12 hours after exposure. Although generally considered an incapacitating toxin, at high enough dose SEB can be lethal, typically as a result of pulmonary edema. Clinical diagnosis of SEB is likely to be difficult due to the generalized symptoms.

Of the three protein toxins described in this report, SEB is considered the most stable to thermal denaturation. Because SEB is a serious threat in the food industry, numerous studies have been conducted on the thermal stability of the toxin in food products. Early investigations on the thermal stability of SEB resulted in some confusion. The protein seemed to actually be more resistant to denaturation at higher temperature (100ºC) than at lower temperatures (80ºC). However, it was eventually demonstrated that SEB has a tendency to reactivate after initial heat treatment and that this reactivation occurred more readily with more rapid initial inactivation[18]. Aeration or mechanical agitation (stirring) circumvented this process; thus, it was determined that heat treatment at 100ºC was, in fact, an effective decontamination process.

A 1986 study by Natick Laboratories evaluated the stability of botulinum toxin and SEA (a variant of SEB) on several surfaces. Immunological assays (ELISA and radioimmunoassay) were used to establish toxicity. On MRE (Meal, Ready to Eat) foil and BDU (Battle Dress Uniform) fabric, SEA was completely stable for up to two weeks at 30ºC at a relative humidity ranging from 0 to 90 %. At higher temperatures (37ºC) on MRE foil SEB showed no loss of activity after two days but was degraded by 84% in 14 days. At 45ºC the toxin was 80% degraded in two days and 94% degraded after 14 days. On glass under these conditions it was somewhat more stable. SEB was completely denatured with 5.25% Clorox® in 10 minutes.

Efforts in the 1960s and 1970s to develop a vaccine against SEB focused on the creation of toxoids using dilute formaldehyde (as previously described with BoNT). Although unsuccessful, these efforts did demonstrate that SEB is susceptible to inactivation by formaldehyde.

One major concern that cannot be ignored by the modern warfighter is the threat of contaminating limited volume water sources. Two recent studies have addressed this contingency. A 2009 study by investigators at Edgewood Chemical and Biological Center and Temple University evaluated the stability of SEB and ricin in chlorinated water using polyacrylamide gel electrophoresis (PAGE), circular dichroism (CD) and liquid chromatography-mass spectrom-

etry (LC-MS) to analyze the structural integrity of the toxins as well as a T-cell proliferation assay (a manifestation of its superantigen effect) for SEB and a murine 3T3 fibroblast cell toxicity assay for ricin. They found that after 30 days at 25ºC in a buffered solution neither toxin showed any adulteration in mass (PAGE, LC-MS), 3-D structure (CD) or toxicity (cellular assay). SEB was irreversibly denatured by heating to 95ºC. After 5 days treatment at total free chlorine (TFC) to toxin molar ratios of 10:1 and 25:1 SEB retained 55% and 16% activity respectively. At molar ratios above 50:1 no activity was detectable. (Ricin was completely inactivated at a TFC molar ratio of 100:1 in 24 hours.)

Another study to determine the efficacy of hand held assays (HHAs), of the type used by the DoD to analyze BWA threats, in detecting SEB and ricin in water matrices it was found that the limit of detection increased from as low as 0.9-12.5 ng/mL (depending on model tested) to values over 2,500 ng/mL in water treated with 1 or 2 mg/L cholorine (public drinking water generally ranges from 0.3-1.0 mg/L). Examination of the toxin in treated vs. non-treated by PAGE and CD indicated that the toxin underwent substantial structural changes in the chlorine treated water. Thus, although toxicity was not directly evaluated, substantial denaturation of these toxins by chemical treatment likely occurred.

Conclusions. Advances in biotechnology over the past three and a half decades have considerably altered the potential for large scale production of toxins. Pharmaceutical grade proteins, such as insulin, are now produced on a massive scale. Although the mycotoxins, like T2, cannot, at present, be biosynthesized through genetic engineering, this limitation is only short term. The ability to produce paclitaxel, an anti-tumor drug derived from a similar biosynthetic pathway, through heterologous gene expression of the biosynthetic enzymes in bacteria, portents a similar capability for mycotoxins. But, even now, large scale production of T2 in a manner analogous to penicillin production is entirely possible.

Toxins present several desirable characteristics as weapons. They are extremely toxic and in most cases curative treatment is not possible. However, with the exception of the mycotoxins, they are quite susceptible to denaturation. Ricin certainly represents a potential weapon. It was successfully weaponized by the US and has been deployed on numerous occasions by terrorists. Nevertheless, battlespace employment of toxin weapons against a sophisticated military is highly questionable. Protection can be afforded by current individual protective equipment and toxins are relatively easy to decontaminate. Because they are non-volatile, aerosolization is also difficult to achieve. And, even with current advances in biotechnology, their production remains quite expensive.

On the other hand, like other bioweapons, it is relatively easy to present toxin weapon development under the aegis of biomedical research. Production facilities would be, for the most part, identical to pharmaceutical plants. Thus, state actors, desiring to avoid international scrutiny, could easily disguise research and development of more toxic forms and higher production levels as medical or pharmaceutical research.

Another desirable feature of toxin weapons is the element of plausible deniability. Toxins, because they are naturally occurring, can go unrecognized as the cause of mass casualties or can be attributed to natural causes. The Yellow Rain controversy clearly illustrates this potential.

Utilization of toxin weapons by terrorist groups is of greater concern, although production of mass casualties on the scale of the attacks on the World Trade Center is unlikely. The use of toxins to contaminate food or drinking supplies could be psychologically devastating, especially if employed against children. The principle source of terrorism today is from Islamic inspired groups, these organizations have, so far, shown a far greater penchant for conventional high explosives. However, the pernicious creativity shown by these groups suggests that when it comes to inventing horror they have no limits. Collusion of oil rich states such as Iran, which possesses at least a modicum of technological sophistication, with terrorist organizations like Hezb'allah should not be ignored.

BIOGRAPHY

LTC Mitchell L. Wise is a Chemical Officer (IMA) with the US Army Nuclear and Combating Weapons of Mass Destruction Agency, Ft Belvoir, VA. He has a B.S. in Zoology from the University of Georgia, an M.S. in Fish Pathology from Auburn University, and a Ph.D. in Biochemistry and Biophysics from Oregon State University. He was previously assigned as the Chemical Officer for Headquarters, Multi-National Forces, Iraq. His email address is mitchell.wise@us.army.mil

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Nuclear Targeting and the Nuclear Employment Augmentation Team (NEAT)

CW5 Bruce D. Brandes and CW5 Stephen A. Gomes
Nuclear Targeting Officers
U.S. Army Nuclear and CWMD Agency

Over the years the Army has seen a rapid decrease in not only the number of nuclear munitions available to the inventory, but the ability of the Army to fight both offensively and defensively in a nuclear environment. As we start the transition from counter insurgency (COIN) operations and into full spectrum operations, our Soldiers require more training in basic defensive operations, while the Staff requires training on offensive nuclear weapons employment to include the integration and synchronization of these weapons into the Joint Force Land Component Commander's scheme of maneuver.

Currently, there is a misunderstanding that precision airstrikes using conventional munitions can achieve desired effects on "deep targets" and thus nuclear weapons are not necessary. This presumes a number of planning factors favorable to the U.S. and its allies such as: air superiority, target access, and ability to destroy hard and deeply buried targets. The illustration depicted in Conventional versus Nuclear Figure 1., demonstrates the numerous conventional bombs needed to achieve the same destructive force as a nuclear weapon, and you can imagine the number of sorties required to deliver the conventional munitions. Offensive operations with nuclear weapons can significantly reduce combat losses of aircrew and aircraft.

The Army's premiere targeteers are its field artillery warrant officers. Trained as targeteers for life, the transition from a conventional targeting officer to a nuclear targeting officer can be a dramatic leap of faith. This is further compounded by the last 12 years of war, where the Army has focused in a COIN operating environment, targeting single high value individuals with conventional kinetic strikes. The U.S. Army has not trained to target large troop formations with nuclear weapons or plan nuclear strikes for terrain denial since the early 90s.

As we continue into an era of not only fiscal austerity, but nuclear austerity, we must look again at the use of nuclear weapons in a tactical and even "precision strike" role. "Mutually assured destruction" and "missiles over the pole" are no longer likely scenarios. As we experience globalization of technology and resources, the ability of those who would wish to do harm to the United States and our allies are indeed high. Never before have political and economic sensitivities created such an impact on the capability to employ a weapon long considered taboo. As some nations seek to proliferate nuclear weapons, and other nations seek to increase the number of warheads in their current nuclear weapons stockpile, the U.S. Army must improve its ability to fight and survive in a nuclear environment. By understanding nuclear weapons effects, the Land Component Commander can synchronize targets and integrate nuclear operations into the scheme of maneuver.

The Army left the nuclear weapons delivery business nearly 22 years ago. With the last of the 8 inch artillery units being pulled from Germany in 1990, the decline of the Army's nuclear mission began. Yet the Army and Marine forces are the most likely of the Services to operate in a nuclear environment if a conflict should ever escalate to this point

In 1992, the Chief of Staff of the Army, GEN James Peay III, wanted to maintain nuclear expertise in the Army following President's Bush's Presidential Nuclear Initiative in 1991. GEN Peay III mandated that "Upon request by the supported Combatant Commander, the Department of the Army provide a U.S. Nuclear and CWMD Agency (USANCA) Nuclear Employment Augmentation Team (NEAT).

As directed by Army Regulation 10-16, U.S. Army Nuclear and Combating Weapons of Mass Destruction Agency, the NEAT provides nuclear targeting expertise and assistance in analyzing the impact of offensive nuclear weapons on the friendly scheme of maneuver. This is further codified in the Joint Strategic Capabilities Plan (JSCP). To that end, we have always maintained offensive nuclear training, but certainly not at the levels of the Cold War. The Defense Threat Reduction Agency's Theater Nuclear Operations Course (TNOC), a basic primer on theater nuclear operations, is conducted at the Defense Nuclear Weapons School at Kirtland AFB. While this course provides a familiarization with Nuclear Operations at the COCOM level, it does not provide the level of targeting proficiency to support adaptive planning in support of theater nuclear operations.

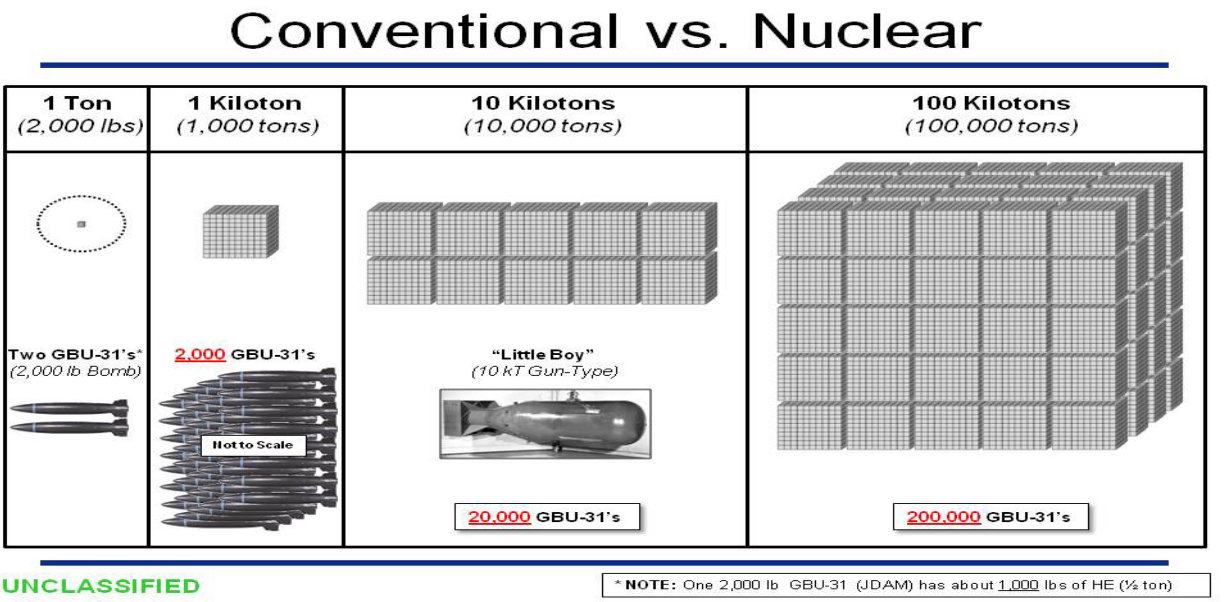


Figure 1. Conventional versus Nuclear

The NEAT derives its guidance from military strategy, down to the Guidance for Employment of Forces, and the JSCP (Figure 2.). This guidance then directs the COCOM to develop numbered OPLANS. These plans incorporate the nuclear planning process for which COCOM staffs are neither trained nor resourced to conduct. One of the key components of the NEAT mission is planning support to OPLANS and exercises. In the direct support role, the NEAT deploys to supported commands to assist, advise and plan for crisis action and adaptive nuclear scenarios. In support of its NEAT mission, USANCA maintains 2-12 officer/warrant officers/civilians to deploy in support of operations to include week-long training events to full duration combat deployments. In this capacity, the NEAT becomes part of the COCOM staff in an OPCON relationship.

The NEAT composition is based upon the mission set. It is an "adhoc" team composed of officers and DA civilians tailored to the mission. The range and scope of the NEAT includes the following:

- Formal "on the-ground" theater nuclear support
- The NEAT augments Joint Forces Land Component in theater

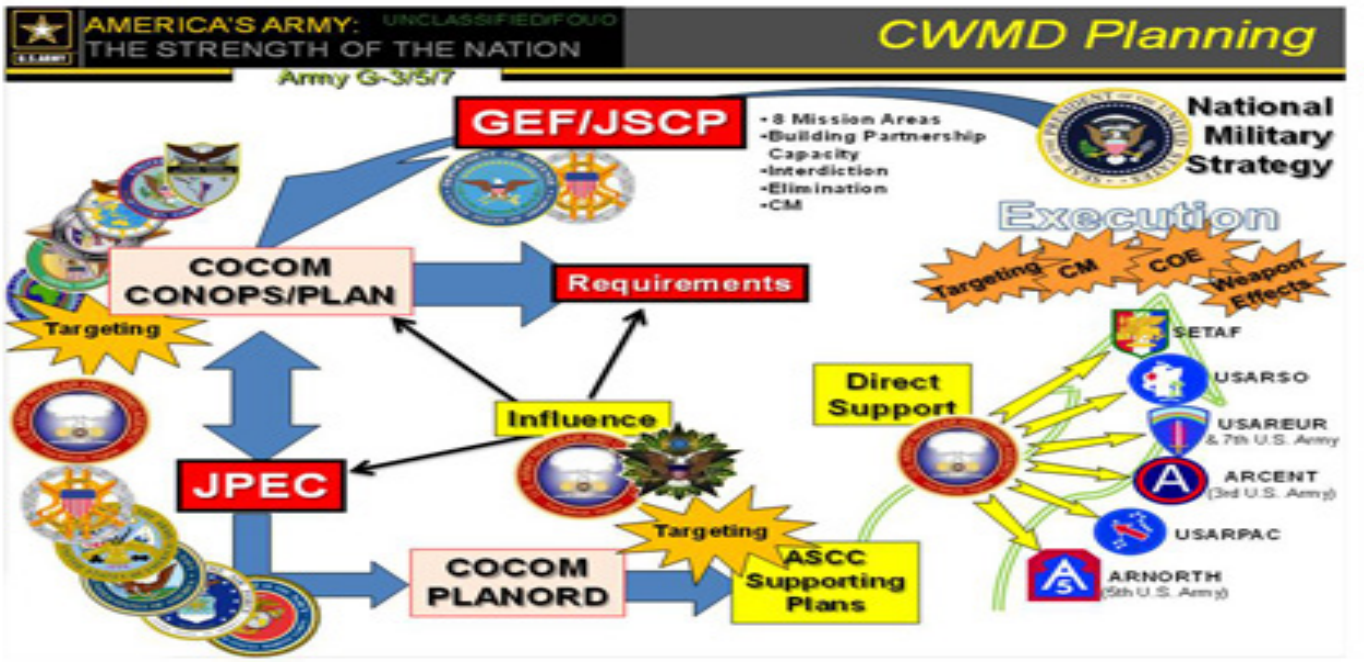


Figure 2. Guidance for Employment of Forces, and the JSCP

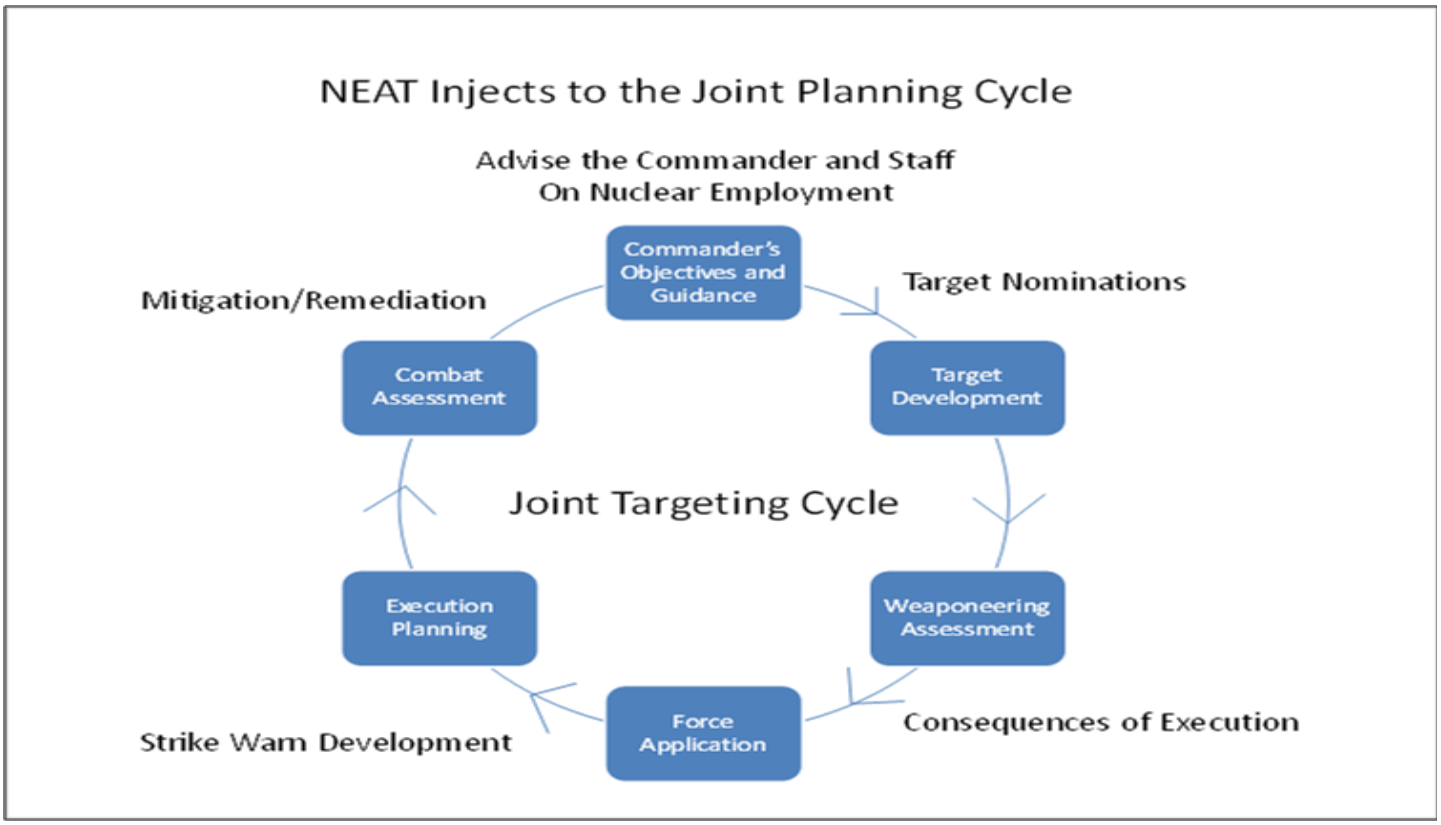


Figure 3. NEAT injects to the Joint Planning Cycle

- Supports the Army's capability to plan for joint employment of nuclear weapons, conventional attacks on nuclear facilities, and friendly force vulnerability to nuclear weapons.
- Provide Pre-Strike Theater nuclear planning, prepare STRIKWARN messages, recommend movement of friendly forces and provide Post-Strike recommendations regarding operations/movement.
- Support Army Planning efforts to counter WMD.
- Understand and communicate the impact of WMD effects on military operations – educate the force.
- Enhance force survivability in CBRN environments—manage the Army's CBRN survivability program.
- Enhance interoperability of multinational forces in a nuclear environment

NEAT Reach back/Resources/Expertise:
-Nuclear reactor operations
-High Altitude Electromagnetic Pulse (HEMP) effects
-Nuclear medical science

Integrating NEAT operations into the COCOM should be treated as any other War Fighting Function (WFF). Participation in the 6 step joint targeting process is the key to a successful integration of Nuclear and WMD operations. Keeping CWMD as a standalone entity will invariably de-synch nuclear operations with the scheme of maneuver.

The NEAT participates in real world deployments and exercises, and continues to augment COCOM staffs for nuclear support. Until such time as a nuclear free world, USANCA's NEATs remain prepared to deploy and assist any command in planning nuclear operations.

BIOGRAPHY

CW5 Bruce Brandes is a Nuclear Targeting Officer at USANCA, he has served as a targeting officer at all levels throughout his 30 years of active duty, and serves as an instructor for the Theater Nuclear Operations Course. His email address is bruce.d.brandes.mil@mail.mil

CW5 Stephen A. Gomes is a Nuclear Targeting Officer assigned at USANCA. He recently returned from a deployment in Kuwait as the ARCENT Command Electronic Warfare Officer. He is a graduate of the Theater Nuclear Operations Course (TNOC) and the Joint Targeting School, Dam Neck, VA. His email address is stephen.a.gomes.mil@mail.mil

Biotoxins Used As Warfare Agents – Part 2

John S. Nordin PhD
AristaTek, Inc.

This article is modified from a series of papers written by the same author for "The First Responder", a newsletter of AristaTek, Inc., available at http://www.aristatek.com. Part 1, which presented an overview of Biotoxins and discussed Ricin as an example, was presented in the CMWD Journal.

Review: What kind of Biotoxins Might Be Used as Warfare Agents?

A 'biotoxin" is a poison produced by living organisms including certain bacteria, plants, algae, fungi, protozoa, reptiles, fish, mollusks, and insects. Over 400 biotoxins have been identified and many more exist in nature. Roughly about 15 or 20 of these usually appear on various lists published by governmental agencies as having potential for use as warfare agents. Two natural biotoxins are classified as "Schedule 1 Chemical Warfare Agents" under the United Nations agreement on biological weapons, e.g. the Chemical Weapons Convention in 1993 and earlier agreements. These are saxitoxin and ricin.

On June 12, 2002, President George W. Bush signed into law the Public Health and Safety Act of 2002 (PL 107-188), which requires that the Department of Health and Human Services maintain a list of biological agents and toxins, which pose a severe treat to public safety. The list of biotoxins, as it appears in the August 23, 2002 Federal Resister, (see also 42 CFR Part 72, Appendix A) is as follows:

- Abrin
- Botulinum neurotoxins
- Clostridium perfringens epsilon toxin
- Conotoxins
- Diacetoxyscirpenol
- Ricin
- Saxitoxin
- Shigatoxin and Shiga-like toxins
- Staphylococcal enterotoxins
- Tetrodotoxin
- T-2 toxin

In addition, The Centers for Disease Control and Prevention website (http://www.bt.cdc.gov/agent/biotoxins/) also lists the following biotoxins which do not appear in the Public Health and Safety Act of 2002:

Brevetoxin
Colchicne
Digitalis
Nicotine
Strychnine
Trichothecene

We will look at Saxitoxin, Brevetoxin, Arbin, Botulinum neurotoxins, and Clostridium perfrigens epsilon toxin in this article.

Saxitoxin and Brevetoxin

Saxitoxin and brevetoxin are both potent toxins produced by algae known as dinoflagellates. Saxitoxin is a potent toxin which if ingested (or injected) works as a selective sodium channel blocker in the nervous system resulting in rapid, incurable paralysis and death. Brevetoxins (there are more than one kind of Brevetoxin) bind to different sites than saxitoxin allowing an unchecked flow of sodium ions in and out of nerve cells. A dose of 0.2 milligram of saxitoxin is fatal to an average-weight human being. Brevetoxins are rarely fatal and patients usually recover, but the person may suffer permanent nerve damage and dementia. Saxitoxin is produced by "red-tide" algae, one of the algae species being"*Alexandrium tamarense*". This algae species is found in marine waters throughout the world. The usual mode in which humans ingest this toxin is from eating contaminated shellfish (clams, mussels, oysters), which in turn have ingested ("filtered") the algae at some previous time in their life, even though the "red tide" may have past and the waters look clean. Cooking the shellfish does not destroy the toxin.

In the 1950's the CIA began experimenting with the biotoxin. Saxitoxin was produced and stockpiled in the United States as part of a chemical weapons program at Fort Detrick, MD using the code name Agent TZ. It is not clear what the CIA involvement was, but Internet sources1 suggest development of dart guns and manufacture of suicide tables for their agents if captured. Agent TZ is soluble in water and can be ingested or inhaled. If an intended victim is jabbed

Figure 1. Red Tide Algae, a Source of Saxitoxin Consumed by Shellfish which in Turn Is Eaten by Humans

Red tide off California coast, Noctiluca sp., photo by PJS Franks, from Woods Hole Oceanographic Institution photo gallery

Figure 2. Red tide off New Zealand coast, photo by M. Godfrey, from NIWA Science website

with a dart tip, death occurs quickly. The method used by the CIA at the chemical weapons program at Fort Detrick, MD involved harvesting a certain species of clam grown in "red tide" waters, and extracting the saxitoxin from the clams.

President Nixon in 1969 ordered the CIA to destroy its entire stock collected over the years and not engage in addi-

tional covert research with saxitoxin. In 1975, the CIA director revealed to Congress that they still possessed 10 grams of saxitoxin, in violation of the 1969 presidential order, which was then evenly distributed to scientists and medical researchers under the auspices of the National Institute of Health. Saxitoxin is useful in the study of nerve disorders because it selectively blocks only the sodium channels but does not affect potassium or calcium channels or the chloride ion count or acetylcholine response.

In 1977, a paper was published in the open literature[2] on the chemical synthesis of saxitoxin. The synthesis was modified by Jacobi[3] published in 1984, and with another update in 2006[4] yielding an optically pure product mimicking what is produced in nature. The Jacobi synthesis was carried at Wesleyan University in Connecticut. The three methods were reviewed in the open Internet by J. Baird[5] in 2006.

Saxitoxin is said to be 1000 times more toxic than the nerve gas Sarin. The 0.2 milligram fatal dose for an average weight human is based on LD50 mice studies (the lethal dose resulting in the death of 50% of the test animals in 24 hours). The LD50 for mice is 8 microgram/kg, but humans are 4 times more sensitive to than mice to oral doses of saxitoxin because the human digestive track is longer and more saxitoxin is absorbed. The lethal oral dose for humans is 1 to 4 mg depending upon age and physical condition. Children are apparently more sensitive than adults.

Alexandrium tamarense is not the only algae species that produce biotoxins. The causative algal species mostly belong in the general classification of dinoflagellates, which include Alexandrium tamarense, Alexandrium circinalis (a fresh water species), A. minutum, A. ostenfeldi, A. catenella (Pacific coast), A. fundyense (northeastern U.S. and Canadian coast), Gymnodinium catenatum, Karenia brevis (eastern Gulf of Mexico), various Dinophysis sp. (Europe, Asia, Japan), and Pyrodinium bahamense (Philippines and elsewhere). The affected waters may be red, grey, brown or other colors, e.g. "brown tide", or the waters may not be noticeably colored. At least 12 different biotoxins are produced from various dinoflagellate species and have also been studied. The biotoxins fall under the general chemical classification of tetrahydropurines, of which saxitoxin is the first studied and best known. There are also other biotoxins besides tetrahydropurines produced from algae blooms.

No human deaths have been directly attributed to direct contact to "red tide" algae, but people may experience respiratory irritation when winds blow aerosol onshore from waters containing "red tide" algae. Skin irritation and burning is possible when swimming in areas affected by "red tide" algae. Deaths of people and animals (including birds, fish, turtles, etc.) occur because of consumption of mollusks and other creatures, which feed on the algae; the mollusks concentrate the biotoxin in their flesh as the result of their filter feeding. The biotoxins have resulted in deaths of fish, marine animals, sea turtles, and birds. Generally, fish and shrimp caught in "red tide" waters are safe to eat at least in smaller quantities (check with the local health department/authorities to be sure). However, the 1987 deaths of 14 humpback whales off Massachusetts in Cape Cod Bay were traced to the whales eating mackerel, which in turn had eaten smaller fish and zooplankton, which had consumed large amounts of Alexandrium tamarense. The saxitoxin was concentrated in the food chain. The 1987 death of 700 bluenose dolphins was also similarly traced to bioaccumulation of neurotoxins in their fish diet.

Four types of human shellfish poisoning have been identified:

- Paralytic shellfish poisoning (PSP)
- Amnesic shellfish poisoning (ASP)
- Neurotoxic shellfish poisoning (NSP)
- Diarrhetic shellfish poisoning (DSP)

PSP description:

The onset of symptoms occurs within 5 to 30 minutes after ingestion of contaminated shellfish. Initially there is a slight tingling progressing to numbness around the mouth, neck, and face. In severe cases, these symptoms spread to the extremities in coordination and respiratory difficulty. There may be difficulty swallowing, sense of throat constriction, speech incoherence, headache, dizziness, nausea, possible vomiting, and reduced eye pupil size.

Biotoxin	Algae species	Poisoning Type	Fatal dose
Saxitoxin	Alexandrium sp. and certain other dinoflagellates	PSP	0.2 mg for average weight human. 100% recovery from non-fatal doses without brain damage
Brevetoxin (There are several toxins)	Gymnodinium breve (Gulf of Mexico and Caribbean), Karenia sp. and other dinoflagellites	NSP	Deaths rare, but patients may suffer dementia. Rat and human studies show brain damage. non-fatal doses without brain damage
Domoic acid (CAS 14277-97-5)	Nitzchi pungens (a diatom) implemented in 1987 Canadian outbreak. Also Pseudonitzschia sp. (east and west coast of U.S. and Gulf of Mexico)	ASP	Rat LD50 (injected subcutaneously) 0.33 mg/kg. In a 1987 Canadian outbreak, of 145 patients studied, the three patients who died were elderly and showed severe damage to the hippocampus and other parts of the brain. For 10 cases, from mussel analysis, a 4.2 mg/kg oral dose of toxin resulted in severe neurological effects.
Okadaic acid (CAS 78111-18-8)	Dinophysis sp.; also Prorocentrum lima	DSP	Deaths rare, but elderly patients experience memory loss. Large dose of okadaic acid from contaminated mussels results in permanent neurological sequelae. LC50 (mice, injected subcutaneously) 0.192 mg/kg

Table 1. Example Biotoxins from Eating Shellfish

In severe cases, within 2 to 12 hours, there is complete paralysis and death from respiratory failure in the absence of ventilatory support. Without artificial respiration, up to 75% of severely affected patients, die within 12 hours. Gastric lavage and administration of activated charcoal or dilute bicarbonate solution is also recommended. Benzedrine is effective in aiding artificial respiration. The PSP symptoms mimic acute organophosphorous pesticide or nerve gas Sarin poisoning, but the use of anticholinesterase agents is not recommended for PSP patients and may do more harm than good. After about 12 hours, if death has not occurred, patients start to recover gradually and are without residual symptoms after a few days. Cooking the shellfish does not destroy the biotoxin.

It is imperative to obtain samples of the shellfish tissue and their source so that diagnosis can be made. The mouse bioassay of the food extract is the usual diagnostic method. Radioimmunoassay and indirect enzyme-linked immunoabsorbent assay have been developed for saxitoxin, but not all of the biotoxins, which cause PSP. HPLC analysis methods have been developed for all of the PSP toxins.

NSP description:

There are several different kinds of brevetoxin (given names such as brevetoxin A, brevetoxin B, and other names). Brevetoxins act by disrupting the flow of sodium ions in nerve cells. They bind to the sites near the nerve cells allowing an unchecked flow of sodium ions in and out of the cells (in contrast to saxitoxin which binds different sites and blocks sodium ions from passing through the sodium channel). Brevetoxin poisoning rarely results in death, and patients recover within a few days, but permanent nerve damage and dementia can occur. Symptoms include false temperature sensations, muscular aches, dizziness, and anxiety. These are usually accompanied by vomiting, diarrhea, and abdominal pain. Cooking the shellfish does not destroy the biotoxin.

ASP description:

Much of what is known is the result of investigation of 153 cases of acute intoxication, which were reported in 1987 as the result of individuals eating mussels harvested from Prince Edward Island, Canada. The onset of symptoms varied between 15 minutes to 38 hours after eating. Many of the patients were elderly and suffered gastrointestinal distress and also neurological effects that included memory loss and dementia. Younger patients seemed to have more digestive problems. Twenty-three (23) patients required intensive care because of seizures, coma, profuse respiratory secretions, or unstable blood pressure. Three patients died. The cause of death was coma, encephalopathy, convulsions, and cardiovascular collapse. In the mouse bioassay, mice were given intraperitioneal injections of extracts from the mussels. The mice soon exhibited an uncontrolled scratching of both shoulders with their hind legs, and most of the mice died within 3.5 hours after injection. Further research demonstrated that the toxin was domoic acid, an amino acid with a molecular weight of 311. The involved mussels contained between 31 and 128 mg of domoic acid per 100 grams of mussel tissue. The toxin was produced by an algae (diatom) called Nitzschi pungen, which in turn was ingested by the mussels during their normal filter feeding. In ten patients studied, there was a clear dose response between amount of domoic acid in mussels consumed and neurological effects. Further tests using monkeys using mussel extracts gave similar dose response to the ten patients studied. Later research demonstrated the presence of domoic acid in anchovies in California, and razor clams and crabs off British Columbia. Domoic acid caused the death of large numbers of Brown pelicans and cormorants in 1991 and over 400 sea lions in 1998, both incidents off the California coast. The anchovies in California had been eating the diatom, Pseudonitzschia australis that produced the domoic acid; the seabirds that died in 1991 in turn ate the anchovies.

In the brain, domoic acid damages the hippocampus and amygdaloid nucleus. It damages neurons by activating AMPAS and kainatic receptor causing an influx of calcium. The uncontrolled increase of calcium causes the nerve cells to degenerate. When the hippocampus is damaged, long-term memory loss occurs. There is no antidote for domoic acid. Cooking the mussels does not destroy domoic acid.

DSP description:

Acute high-dose exposure to okadaic acid, which accumulates in certain clams and some crabs, is the underlying cause of human DSP. Symptoms occur between 30 minutes to 12 hours after eating contaminated shellfish. Symptoms include diarrhea, nausea, vomiting, abdominal cramps, and chills. Gastrointestinal bleeding and hiccups have occurred. Full recovery is usually experienced within a few days, but death can occur especially in elderly patients due to coma, seizures, and/or pulmonary edema. DSP mostly occurs in Europe, Japan, and South America. Over 5000 people experienced DSP in Spain in 1981. Okadaic acid (and/or its esters) primarily affects the cells lining the intestinal gut; the exact mechanism is not clear, but the chemical is thought to stimulate phosphorylation that controls sodium secretion by intestinal cells and also affects calcium ion transport in general across cell membranes in the body. Patients over 50 years old may also experience neurological effects including memory loss, severe anterograde amnesia, and motor or sensorimotor neuropathy. Cooking the shellfish does not destroy the biotoxin.

Abrin

Abrin is a potent toxin extracted from the seeds of the Jequirity Pea (Abrus precatorius) plant. The adult fatal dose for abrin is about 0.0026 mg if ingested, or about 0.04 micrograms per kilogram (µg/kg) of body weight. The fatal dose can vary between individuals. Its toxicity is roughly 75 times that of ricin. It is also fatal by inhalation or by absorption through sensitive skin areas as in the eyes.

Like ricin, the toxin acts by inhabitation of body protein synthesis. Initial symptoms by ingestion include watery diarrhea at first, later nausea, vomiting, abdominal cramps, and chills. The vomiting and diarrhea becomes bloody. Severe dehydration may result followed by low blood pressure. Other symptoms may include hallucinations, seizures, and blood in the urine. Within days, the person's liver, spleen, and kidneys may

stop working. Death could take place within 36 to 72 hours of exposure. If death has not occurred within 5 days, the person usually recovers but may suffer long-term organ damage. There is no specific antidote. If abrin powder is inhaled, pulmonary edema and hemorrhaging can result. Abrin dust in air can result in blindness or at minimum severe eye irritation. There have also been reports[6] of people who have ingested the seeds and slipped into a coma, a condition called "acute demyelinating encephalitis" , resulting in death.

The plant itself goes by many names, including: Rosary Pea, Jequirity Pea, Jequirity Bean, Crab's Eye, Deadly Crab's Eye, Precatory pea, Precatory bean, Roseary Pea, Abrus seed, Jumble beans, Ratti seeds, Prayer beads, Tentos de America, Tentos dos mundos, Jequirite, Aivoeiro, Buddist rosary bead, Ruti, Indian bead, and Wild liquorice. The plant itself is a vine which grows up to 10 feet tall and has clusters of rose pink or purplish flowers about 0.5 to 0.8 inches long. The flowers produce brown seedpods with shiny red and black seeds about 5 to 8 mm long. The plant itself is grown in tropical and subtropical areas and has been planted in warmer parts of the United States. The red and black seeds are sometimes used in necklaces and other decorative items.

One seed if chewed can kill, especially a child. All parts of the plant are toxic to some degree, but the seed, which contains abrin, is especially toxic. If the dried seed is swallowed whole, it is less toxic but some of the toxic elements can still leach out by the digestive enzymes. Sucking on the seeds can release some of the toxic contents. Immature seeds are poisonous if ingested even whole. If holes are drilled in the seed as in a necklace and the seed is ingested, the toxic contents will leach out by intestinal secretions.

Five glycoproteins have been purified from the seeds, four of which (abrin a, b, c, and d) are extremely toxic. The other glycoprotein is "abrus agglutinin" which is a powerful hemagglutinator but is relatively non-toxic to animal cells; its molecular weight is approximately 134,900.

Arbin (a, b, c, and d) are the toxic parts. The molecular weight is between 63,000 and 67,000. Abrin has been given CAS# 1393-62-0. Purified arbin is a yellowish-white powder. Most of its toxicity is destroyed if heated to 80°C (176°F) for 30 minutes.

Figure 2. Illustrations of Jequirity Pea

The U.S. Department of Health and Human Services – Centers for Disease Control (CDC) Website provides a summary table (Table 2).

As far as we have been able to determine from an Internet search, Abrin is not known (to date) to have been used in any wars or terrorist attacks. Abrin also has potential medical uses such as in treatment to kill cancer cells.

Botulinum neurotoxins One of the most toxic biotoxins known is botulinum neurotoxin, with an estimated human lethal dose of 0.001 micrograms per kilogram of body weight (µg/kg) by inhalation, or 1 µg/kg if ingested. The poisoning is called botulism. There are actually seven types of botulinum neurotoxins recognized, labeled serotypes A through G, with type A being the most toxic and types B and E causing less severe toxicity to humans. The botulinum neurotoxins are produced primarily by a spore-forming bacterium known as Clostridium botulinum and to a lesser extent by spore-forming bacteria Clostridium baratii and Clostridium butyricum. Clostridium sporogenes does not produce the neurotoxin. These bacteria and its spores are found worldwide in soil, ponds, coastal waters, sediments, the intestinal tracks of fish and mammals, and in shellfish.

Human botulism is primarily caused by types A, B, E, and F neurotoxin. Types C and D most commonly affect fowl, birds, cattle, and horses and do not affect humans. Type E typically occurs from eating contaminated fish. Type G, isolated from a soil in Argentina, has not yet been linked to any outbreak. All of them are single polypeptide chains (molecular weight about 150,000) that work by blocking the release of acetylcholine from peripheral cholinergic nerve endings. What this means is that the nerve terminals are blocked, and the muscles do not work, usually starting at the eyes and face, then the throat, chest, and extremities. When the diaphragm and chest muscles become fully involved, respiration is in hibited and death occurs. There are differences on how the different types interact with nerve endings and block

Exposure	Symptoms	Personal Protective Equipment	First Responder Response
Inhalation	Irritation (see ingestion for other symptoms)	Pressure demand, self-contained breathing apparatus (SCBA) (SCBA CBRN, if available) is recommended in response to non-routine emergency situations. Breath Response (pressure demand) HEPA PAPR.	Fresh air, rest, half-upright position. Perform CPR if necessary. Seek medical attention immediately.
Skin	Potential for allergic skin reaction; redness, blisters, pain. May be absorbed	Tychem® BR or Responder® CSM protective clothing. Eyes should be protected when possible.	Remove contaminated clothes. Rinse skin with plenty of water or shower (and soap if available). Seek medical attention immediately.
Eyes	Tearing, swelling of the eyelids, pain, redness, corneal injury.	Goggles with respiratory protection or full face-piece respirator.	Immediately flush with large amounts of tepid water for at least 15 minutes. Seek medical attention immediately.
Ingestion	Cardiovascular (heart and blood circulation) shock from severe dehydration, life-threatening low blood pressure, fast heart rate (tachycardia) and irregular heart rhythms (arrhythmias). Central Nervous System (brain) - drowsiness, disorientation, hallucinations, seizures, coma. Gastrointestinal (stomach and intestines) - burning pain in the mouth, abdominal pain, nausea, vomiting, diarrhea, bleeding and swelling of the lining of the GI tract, liver cell damage and death. Genitourinary (kidneys and urine) - blood in urine, low or no urinary output, kidney cell damage and death. Musculoskeletal - muscle weakness, tremors, and muscle spasm (tetany) Eyes - dilated pupils and bleeding in the back of the eye (retinal hemorrhage) Skin - blue skin (cyanosis) and redness (flushing) Symptoms delayed 1-3 days. May be fatal.	Do not eat, drink, or smoke during work. Wash hands before eating	Rinse mouth. Do not induce vomiting. Use slurry of activated charcoal. In the event of vomiting, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Early and aggressive IV fluid and electrolyte replacement. Seek medical attention immediately.

Table 2. CDC Emergency Preparedness and Response for Abrin7

the release of acetylcholine, which is the reason why some types are more toxic than others or are more toxic to bird or animal species or to humans.

The neurotoxin itself is destroyed if heated to 80°C (176°F) for 10 minutes or longer. If heated to 100°C (212°F), destruction occurs quicker. However, cooking food containing spores does not destroy the spores at 100°C (212°F). When canning food, the spores must be destroyed by moist heat at temperatures of at least 121°C (250°F). The spores if ingested normally do not prorogate and produce neurotoxin in adult humans because gut conditions are too acidic, but the spores can colonize and produce neurotoxin in the intestinal tracks of infants under 12 months of age.

Four different kinds of botulism poisoning are recognized:

1. Inhalation botulism. This kind does not occur naturally and could mean that a terrorist has released the toxin as an aerosol.

2. Food borne botulism. This is usually the result of ingestion of food that has been inadequately processed or inadequately cooked before being eaten. If food is contaminated with Clostridium botulinum spores, and the bacteria allowed to grow under anaerobic conditions, the neurotoxin will be produced. If not cooked properly to destroy the toxin, botulism food poisoning will occur. The most common source is home-canned foods. There have been incidences of poisoning in commercial products. Neurological symptoms usually appear within 6 hours to 8 days of ingestion of the food, the shorter time associated with more severe poisoning.

3. Infant (intestinal) botulism. This results from ingestion of bacteria spores which germinate in the intestinal gut and produce the toxin. This can happen as the result of food, soil, or dust contaminated with the spores. The infant gut is most susceptible to spore germination, but adult infections can occur if acidic conditions are not maintained in the gut. This is the most common incidence of botulism poisoning reported to the CDC in the United States. Pediatricians do not recommend feeding honey to infants as the honey could contain the spores.

4. Wound botulism. This happens when wounds become contaminated with dirt containing botulism spores. This is especially a problem with chronic injection drug users. The incubation period is between 4 and 14 days since contamination, the shorter time corresponding to the more severe infection. Neither the spores nor the neurotoxins are able to penetrate intact skin. About 110 cases of U.S. botulism poisoning are reported to the CDC annually.

Confirmation of botulism poisoning is done by either detection of botulinum toxin or by isolation of Clostridium botulinum in a clinical specimen and the patient also displays clinical symptoms of botulism poisoning. The specimen could be a person's stool sample in the case of infant or food borne botulism or

a serum sample, or a food sample. The standard test for botulinum toxin testing is the mouse neutralization test. A more sensitive test8 published in 2006 is an amplified enzyme-linked immunosorbent assay (ELISA) test applicable for botulinum neurotoxin in foods.

Antitoxin therapy is available and is administrated to adult patients with food borne or wound or inhalation botulism. The antitoxin must be administrated early. It is not administrated to infants. The CDC must release and approve its use. The antitoxin is a horse serum product and may cause serum sickness in approximately 20% of treated persons. A human-derived antitoxin product for infants is or has been under evaluation9 in California. Also, the decision to administer the antitoxin often can't wait for confirmation by testing for the toxin or bacteria, and must be made on the basis of display of symptoms especially if a suspect food is identified or there is evidence of a wound which may have been contaminated. The patient's serum should be collected before administration of the antitoxin.

The specific antitoxins available from the CDC are
• Licensed Bivalent Anti-AB equine antitoxin, Aventis-Pasteur
• IND Univalent Anti-E equine antitoxin, Aventis-Pasteur
• IND Heptavalent despeciated (Fab'2) equine Anti-ABCDEFG antitoxin, U.S. Army California Dept. of Health Services, Infant Botulism Treatment and Prevention Program
• Licensed Anti-AB human antitoxin for infant botulism "Baby-BIG"

The classic symptoms of botulism poisoning include double vision, blurred vision, drooping eyelids, slurred speech, difficulty swallowing, dry mouth, and muscle weakness. Infants appear lethargic, feed poorly, are constipated, have a weak cry, and have a poor muscle tone. If untreated, these symptoms may progress to cause paralysis of the arms, legs, trunk, and respiratory muscles. (according to the CDC website). Because of respiratory failure, the patient may need to be on a breathing machine (ventilator) for weeks. If severe, the person may require intensive medical and nursing care for months. The person affected by botulism poisoning is not infectious.

Several countries are reported[10] to have produced botulinum neurotoxins as weapons. The United States once produced botulinum neurotoxin under military code name X. The entire stockpile of botulinum neurotoxin was ordered destroyed by President Nixon (1969) along with other biological agents. The former Soviet Union continued their biological weapons research program. In April 1992, President Boris Yeltsin declared that his country had continued a massive offensive biological warfare buildup which included botulinum warfare buildup. In 1995, Iraq admitted to the United Nations Special Commission inspection team that it had produced 19,000 liters of botulinum neurotoxin concentrate for use in specially designed missiles and sprayers, including approximately 10,000 liters loaded in military weapons. In 1990, Iraq deployed 13 missiles with a 600 km range containing botulinum toxin.

Fortunately, botulinum neurotoxins are unstable in the environment. Therefore the range of an aerosol attack out in the open is limited. The neurotoxin would be expected to degrade in the environment within one or two days. Contaminated surfaces can be cleaned with a 0.1% chlorine bleach solution.

Clostridium perfrigens epsilon toxin

Clostridium perfrigens is the name of the microorganism and epsilon toxin is one of several biotoxins that it produces. Gas gangrene results from wound contamination by this microorganism, which grows in the wound and produces biotoxins. In addition, Clostridium perfringens can cause food poisoning. Clinical symptoms resulting from food poisoning include intense abdominal cramps and diarrhea which begins between 8 to 24 hours after eating food containing these bacteria. The toxins are a particular problem with farm animals, in particular, goats, sheep, young caves, and pigs. Theoretically, a person could also become poisoned by inhalation of dust containing the microorganism or its toxin. The toxins can be transmitted in contaminated food, water, or as an aerosol. Specific information on human poisoning by the epsilon toxin is minimal, but farm animals are affected.

There are 5 different strains of *Clostridium perfringens* (labeled Types A, B, C, D, and E) and perhaps at least 20 toxins (labeled alpha, beta, beta2, etc, epsilon, iota, etc.; some sources cite 12 toxins). The toxins vary in toxicity and in their effect on animals and people. The epsilon toxin (which is produced by strains B and D) is singled out as being the most toxic, based primarily on intervenous injection of the toxin to test animals. The LD_{50} value (lethal dose resulting in death of 50% of the test animals) for the epsilon toxin using a mouse as the test animal is 0.78 nanograms (0.1 µg/kg of body weight), (ref: Iowa State University, Center for Food Security and Public Health website). Goats and lambs injected with the

Toxin	LD50 (µg/kg)
Botulinum toxin A	0.0012
Botulinum toxin B	0.0012
Botulinum toxin C1	0.0011
Botulinum toxin C2	0.0012
Botulinum toxin D	0.0004
Botulinum toxin E	0.0011
Botulinum toxin F	0.0025

Table 3. LD_{50} values for Botulinum Toxins[11]

epsilon toxin develop severe pulmonary edema (fluid in their lungs) and show neurological symptoms. Rats injected (intraperitionally) develop cerebral edema. Onset of neurological symptoms by intervenous injection may occur anywhere from 2 minutes to one hour in test calves to 0.5 hours to 3 hours for goats and lambs. The epsilon toxin works by causing potassium and fluid leakage from body cells. Neurological symptoms include stupor (mild poisoning) to coma and death (more severe poisoning).

About one-third of the incidences of food poisoning in the United States is caused by *Clostridium perfringens*, mostly from the type A strain produc-

ing the alpha toxin, or enterotoxin. A more serious form is the type C strain producing the beta2 toxin. Other strains besides type A produce the alpha toxin. Symptoms (from the alpha toxin) of food poisoning include diarrhea, nausea, severe abdominal cramps, and bloating but rarely vomiting or fever. The symptoms appear 8 to 24 hours after ingestion of contaminated food. Patients generally recover within a day or two, although the elderly can take longer. Deaths are rare. However, the type C strain producing the beta2 toxin can cause sloughing of the mucosa off the intestines and intestinal perforation, and severe infection, often resulting in death. Symptoms include a bloody stool and probably vomiting and fever in addition to type A symptoms. Intervention including removal of part of the intestine with prolonged antibiotic treatment and intravenous fluid replacement may be necessary. This type of food poisoning may be called "necrotizing enteritis" or "pig-bel disease". The beta2 toxin is usually associated with enteritis in pigs, therefore the name "pig-bel disease."

Information on human food poisoning from the epsilon toxin is sketchy and is complicated by proper identification of the strain and maybe more than one toxin can be produced. Fluid replacement including electrolyte monitoring is part of treatment as potassium loss is a feature of epsilon toxin poisoning. Limited information available on the Internet seems to suggest that the symptoms are similar to that produced by the alpha toxin.

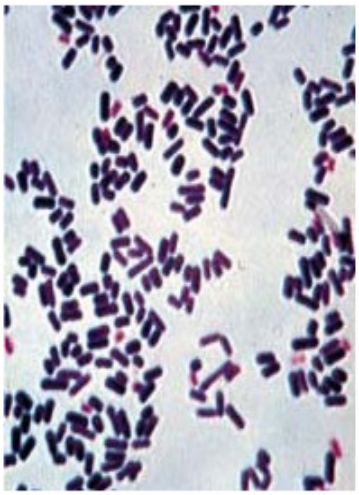
The purified alpha toxin can be lethal by inhalation as an aerosol, causing acute pulmonary disease, vascular leak, hemolysis, thrombocytopenia, and liver damage.

The toxins including the epsilon toxin can be destroyed by heat. Thorough cooking of food is required. The vegetative form of the bacteria can be destroyed at 70°C (158°F), but much higher temperatures and prolonged heating is required to kill the spores. The high temperatures required to kill all of the spores also compromise the nutritional value of the product, and sometimes food processors use a combination of gamma irradiation treatment and heat, or add salt or other preservatives. When cooking meat, it is also important to cool the meat product down quickly and refrigerate if consumed at a later time. More details are in a paper published by the University of Wisconsin, Food Research Institute[12].

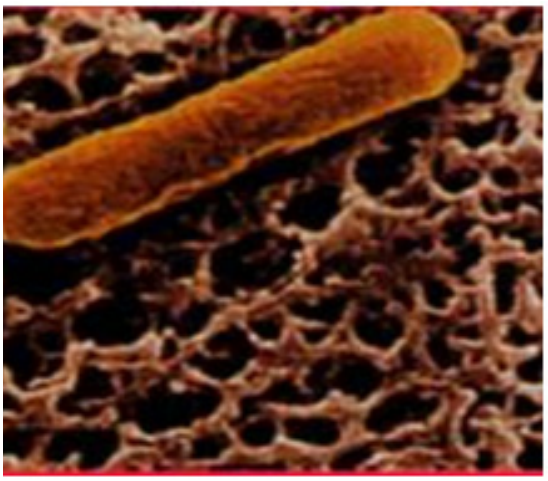
Toxin identification in humans and animals can be done using the mouse neutralization test (MNT) or enzyme-linked immunosorbent assay test (ELISA) on clinical samples (e.g. feces, intestinal fluids). Another test is the polymerase chain reaction assay test (PCR).

Toxin	LD50 (µg/kg)
Clostridium perfringens epsilon toxin	0.1
Clostridium perfringens beta toxin	0.4
Clostridium perfringens lechithinase toxin	3
Clostridium perfringens delta toxin	5
Clostridium perfringens perfringolysis O	13 to 16
Clostridium perfringens enterotoxin	81
Clostridium perfringens kappa toxin	1500

Table 4. LD50 Values for Clostridium perfringens Toxins11



Clostridium perfringens gram positive bacterial stain, anaerobic, rod-shaped



Tissue smear showing Clostridium perfringens

Figure 3. Photos of Clostridium botulinum bacteria

An Internet search failed to turn up evidence that Clostridium perfringens epsilon toxin has been weaponized. The South African biological weapons program between 1981 and 1984 developed the bacteria as a potentially lethal agent to resemble food poisoning. In 1991, Iraq representatives informed the United Nations Special Commission inspection team that Iraq had researched the offensive use of Clostridium perfringens as a potentially lethal agent, and had produced 90 gallons of the bacteria.

Presumably the toxin can be manufactured by fermentation of Clostridium perfringens or more likely by another microorganism expressing the cloned gene for the toxin. The microorganism. E. coli has already been cloned to produce the epsilon toxin for the production of a vaccine for inoculation of farm animals. The purified toxin or even the crude material could be released as an aerosol. A person inhaling the material would be expected to suffer severe pulmonary edema (fluid buildup in the lungs) in addition to vascular leak, organ damage, and neurological problems including cerebral edema, as the result of potassium leakage from body cells. The alpha toxin is also a potentially lethal agent when dispersed as an aerosol.

BIOGRAPHY

Dr. Nordin received his PhD degree in biochemical engineering from the University of Minnesota and has worked with several engineering firms and with the not-for-profit Western Research Institute on environmental and public safety problems. He is currently a co-owner of AristaTek Inc

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Additional information on biotoxins may be found at the website, http://www.globalsecurity.org under Weapons of Mass Destruction.

Regulating CBRN Survivability

Nicholas P. Haugen
U.S. Army Nuclear and CWMD Agency

Program Managers (PMs) responsible for developing military systems work to implement requirements defined by Capabilities Development Documents (CDDs) and Capabilities Production Documents (CPDs). These documents are approved by the Joint Requirements Oversight Council (JROC), a four-star level panel established by law. The JROC, as its name makes clear, is a "joint." It is chaired by the Vice Chairman of the Joint Chiefs of Staff and has representatives from each of the Services. If it finds a capability being developed by a Service to be of joint interest, it can assume responsibility for the CDDs and CPDs that define the system's requirements. The JROC is also a military organization. Its members are military officers, and its work is managed by the Joint Staff. As the joint nature of the JROC prevents Services from working on redundant efforts or at cross purposes, the military nature of the JROC helps to ensure that systems are being developed to satisfy user needs.

One of the key ways that the JROC manages requirements is to define critical requirements as Key Performance Parameters (KPPs). If a PM cannot get his system to meet a KPP, then his program will fail unless the JROC reduces the KPP requirement. Below the level of a KPP is a Key Systems Attribute (KSA). According to Chairman of the Joint Chiefs of Staff guidance, "Attributes or characteristics considered essential to achieving a balanced solution/approach to a system, but not critical enough to be designated a KPP" should be designated KSAs.[1] A KPP is often a grouping of multiple KSAs. A KSA can also stand alone independent of an overarching KPP.

There are six mandatory KPPs for all CDDs and CPDs. These are mandatory in the sense that the CDD or CPD writer must either include each one or justify its exclusion. They are Force Protection, Survivability, Sustainment, Net-Ready, Training, and Energy. This article argues that for relevant Army systems, CBRN survivability should be a mandatory KSA under the Survivability KPP.[2]

What are you talking about?

The official DoD definition of CBRN survivability is "The capability of a system to avoid, withstand, or operate during and/or after exposure to a CBR environment (and relevant decontamination) or a nuclear environment, without losing the ability to accomplish the assigned mission."[3] Meanwhile, "The intent of the Survivability KPP includes reducing a system's likelihood of being engaged by hostile fire…reducing system's vulnerability if hit by hostile fire…and allowing the system to survive and continue to operate in a CBRN environment, if required."[4] Of note, survivability refers to a system's ability to survive, not its ability to protect its occupants. In the case of the mandatory KPPs, a system's ability to protects its occupants is addressed by the Force Protection KPP. In the world of CBRN environments, a large system will protect its occupants with a collective protection system or other passive defense measures.

The logical link between the Survivability KPP and CBRN survivability is specifically authorized by DoD guidance. DODI 3150.09, the CBRN Survivability Policy, states "For CBRN mission-critical systems, the CBRN survivability performance attribute(s) will be evaluated to determine KPP or KS0 designation. They may be combined with the force protection, survivability, or net-ready KPP, if appropriate."[5]

De-conflicting Current Regulation

The most important reason why the Army should implement a requirement for a mandatory KSA for CBRN survivability is that doing so would de-conflict and clarify existing regulatory requirements. There is currently a disconnect between DoD guidance, which tells a Program Manager (PM) he doesn't need to make his system survivable, and Army guidance, which tells him he must.

Department of Defense acquisition guidance tells PMs that if something is not listed as part of a KPP, then that requirement is in the PM's trade space. The exact language reads, "Performance requirements that do not support the achievement of KPP thresholds shall be limited and considered part of the engineering trade space during development."[6] Most Army CDDs and CPDs list CBRN survivability requirements in the additional performance attributes section of a CDD or CPD. So an Army PM would reasonably conclude that he has the authority to trade away the CBRN survivability requirements defined in his CDD or CPD. However, Army regulation tells him otherwise. Army Regulation 70-75, Survivability of Army Personnel and Materiel, states:

"If an item is designated as mission critical/mission essential or is a critical component of one or more mission critical end items, it will be nuclear and [Chemical Biological, and Radiological (CBR)] contamination survivable. If this critical item or component is electronic equipment, at a minimum, it will be survivable to high-altitude electromagnetic pulse (HEMP). If this critical item is a weapon system…it will also survive the initial nuclear weapons effects of blast, thermal radiation, initial nuclear radiation, and source region electromagnetic pulse (REMP)….waiver processes ex-

ist for nuclear survivability criteria and (CBR) contamination criteria…This waiver process does not change the need to meet the survivability requirement."[7]

One might argue that requirements like this exist elsewhere in regulation and that does not automatically elevate the requirement to a KSA. However, the fact that the regulation establishes a waiver process for systems that need relief, and that even that waiver process only allows for alternative methods of achieving CBRN survivability, makes clear that the intent of the Army is to have this requirement met in all cases.

One might also argue that, while AR 70-75 creates a unique threshold somewhere above additional performance attribute, it is not appropriate to raise it to a KSA. Joint guidance tells the developer that he should not make an attribute a KSA unless he is "willing to consider restructuring the program if the attribute is not met?"[8] In other words, because a system fails to meet its CBRN survivability requirements does not necessarily mean that the Army should end the program.

This argument is based on a shortsighted understanding of the world's threats. If one compares a list of states with nuclear weapon development programs, or of states that have not signed the Chemical Weapons Convention, one is likely to see a list of potential adversaries. Indeed, some of these states are potential adversaries precisely because of their possession or development of weapons of mass destruction. If we create a situation where our weapons systems cannot survive in a CBRN environment, we are presenting an easily exploitable vulnerability to our most likely adversaries.

Enforcement Mechanisms

Currently, if an Army system fails to meet a Key Systems Attribute, the first place the program will go is the Army Requirements Oversight Council (AROC). This is the General Officer-level body that manages Army requirements. Documents and requirements validated by the AROC are passed to the JROC. The responsibilities of the AROC include making resource decisions based on military need and risk and program affordability. Its principal members include the Vice Chief of Staff of the Army, the Deputy Chief of Staff (DCS)/G-3/5/7, the DCS G-1, the DCS G-2, the DCS G-4, the CIO/G-6, the DCS G-8, the principal military deputy to the ASA(ALT), and a representative from U.S. Army Training and Doctrine command (TRADOC).[9] If a future system were to fail to meet its CBRN survivability KSA, this is the body that would approve a reduction in its requirements.

Now consider the Nuclear and Chemical Survivability Committee (NCSC). This is the body that currently approves waivers of CBRN survivability criteria. In doing so, it considers military need and risk and program affordability. Its membership includes representatives of the ASA(ALT) the DCS G-2, DCS G-3, the DCS G-4, the DCS G-8, Army Materiel Command, TRADOC, Army Test and Evaluation Command, and U.S. Army Research, Development, and Engineering Command.[10] Making CBRN survivability a mandatory KSA for appropriate systems would transfer the responsibility for reducing requirements from the NCSC to the AROC. With the redundancy of membership and responsibility, this would be a rational consolidation of functions.

Some might argue that the AROC should not be burdened with evaluating requests in this specialized area. They might be surprised to learn that the AROC has already done so. In 2011 the AROC met and considered the reduction of CBRN survivability requirements as part of a scope reduction of M109 Family of Vehicles requirements reduction. CRBN survivability was considered alongside KPP and KSA requirements on the grounds that Army regulation took it out of the PM's trade space. The AROC, meeting at the GO level, decided to reduce nuclear survivability requirements from a threshold to an objective requirement, and to retain HEMP and CBR contamination survivability requirements as threshold. While this was a disappointing outcome from the perspective of an advocate for nuclear survivability, it was a decision made along the lines one would expect of a serious decision: senior leaders representing all relevant perspectives within the Army, taking into account relevant information on cost, military risk, and impacts on the development timelines.

What Systems Are Relevant?

Whether or not a mandatory KSA for CBRN survivability is an appropriate policy response depends to a great deal on which systems it is applied to. According to the AR 70-75 quotation above, current CBRN survivability policy is to be applied to all "mission critical systems." This term is defined broadly in both DoD and Army policy. Indeed, its definition is so loose that it can be applied almost arbitrarily. Naturally, this is not a good situation. For one thing, it allows different combat developers to come to different conclusions about what systems are Mission Critical, even within the same Center of Excellence. For another, It turns the question of "is my system mission critical" into "do I want my system to have CBRN survivability requirements."

A wiser policy should attach more specific guidance to whether or not a system requires CBRN survivability requirements. It should also account for the fact that HEMP, other nuclear weapons effects such as blast, and CBR contamination are three distinct threats. The only distinctions made in the current policy is that mission critical systems with electronics should be HEMP survivable (in addition to being CBR contamination survivable), and a mission critical system that is a weapon should also be survivable to nuclear weapons effects besides HEMP.

Taking each CBRN environment in turn, one can make much more specific determinations about what systems need to be survivable. In the case of CBR contamination, the threat that can create this is a state actor with chemical weapons. A terrorist or insurgent may acquire or produce enough agent to destroy the occasional platform, but would be just as able to do so with a conventional munition. To destroy an operationally significant number of vehicles through CBR contaminiation, an enemy would need to have a very large quantity of weapons and military forces equipped and trained to employ them on the battlefield. The case of Iraq before

the first Gulf War is a good case. What sorts of formations are required to face enemies of this sort? Those designed for high intensity conflict. Besides vehicle of the Heavy Brigade Combat Team, we also need to make equipment that supports the operations of ports and airfields survivable, as these operational chokepoints would be natural targets for an enemy with chemical weapons.

In the case of nuclear weapons effects besides HEMP, these are generated by tactical nuclear weapons. Like chemical weapons, these are the weapons of the same states capable of fighting us in a high-intensity conflict. If a terrorist were to acquire a nuclear weapon, he would most likely have only one or a very few, and would not want to "waste" it on a deployed U.S. Army formation. Why take out ten tanks when you can kill tens of thousands of people? In the case of these weapons, we can also choose to skip the hardening of equipment that supports the operations of ports and airfields. If a nuclear weapons were to hit a port or airfield, the port or airfield itself is likely to be unusable.

Finally, in the case of HEMP, the requirement must be applied broadly. HEMP is an attractive option for an adversary, and a HEMP can impact a broad geographic area. HEMP is an attractive option because, unlike making a significant impact with tactical nuclear weapons, which requires many weapons, generating a HEMP requires only one weapon plus a ballistic missile capability. Generally speaking, the larger the weapon, all other factors being equal, and the higher up it can be detonated, the broader the area that can be impacted. Within that area, systems with electronics that are powered on are susceptible to upset and destruction. Also, due to its wide ranging impact, one must assume that resupply and repair systems will not be up and running for a significant period of time. To ensure that the proper equipment is made survivable to HEMP, the requirement should be applied to any piece of equipment, the instantaneous and permanent loss of which throughout a theater of war, would prevent an the Army from completing its mission within that theater. This would prevent systems like trucks from being developed without HEMP hardening as happens today.

Next Steps

The proper place to codify these requirements is Army Regulation 71-9, Warfighting Capabilities Determination. That document serves as the Army's supplement to the JCIDS guidance provided by the Joint Staff, so any Army-unique requirements would best be captured there. It establishes the AROC and lays out its responsibilities and functions, so the proposal that the AROC consider KSAs for CBRN survivability would be codified in it.

In closing, it is worth noting that this proposal is not a radical departure from what the Army does today. The Army already takes CBRN survivability out of the PMs trade space, it just does so in a way that is confusing. The Army already calls together GO-level representatives to consider failures to meet CBRN survivability standards, it just does so in a body that is redundant to the AROC. The Army already imposes this requirement on "mission critical systems," but does not provide meaningful guidance about what mission critical means. This proposal to impose a mandatory KSA for CBRN survivability is a call to rationalize the way the Army does business

BIOGRAPHY

Mr. Haugen is the CBRN Survivability Manager at USANCA. Previously, he worked for DTRA, SAIC, Inc., and Pacific Northwest National Laboratory, and served on active duty in the U.S. Army. He is a graduate of the U.S. Military Academy, Georgetown University, and the U.S. Naval War College. He is currently enrolled as a master's candidate with University of Maryland University College, where he is studying Cybersecurity Policy.

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Highlighted Courses available at the Defense Nuclear Weapons School (DNWS) and Defense Threat Reduction University (DTRU) Theater Nuclear Operations Course (TNOC)

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Correction:
2010 CWMD Journal article by Dr. Timothy Joseph, The Mission that Drove World History - The Manhattan Project, was incorrectly labeled as a nuclear detonation occurring over Hiroshima vice Nagasaki."

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