New Mexico. These experiences provided the leadership experience required to implement the modern systems of evacuation, echeloned treatment, centralized medical supply, preventative medicine, field medical records and tented field hospitals. Similarly, Walter Reed (1851–1902), a namesake for Army Medicine, had fifteen different assignments in his first eighteen years. It was during these assignments that he developed his passion for the research of infectious diseases, which would later serve him (and the Army) well. (Marble, 2011) In both of these cases highly trained medical officers looked beyond what some might call a mundane assignment and created opportunities for the advancement of military medicine. Thus, underutilization appears to be a frame of mind versus a reality. It would be interesting to hear what impact Dr. Conway's exposure to the 3rd ID had on his deployment experience and if he would bring these experiences back to military GME to better train the next generation of military physicians.

Currently, the Accreditation Council for Graduate Medical Education (ACGME) determines what is required in GME. Military GME might add military requirements to ACGME standards, as the Uniformed Services University of the Health Sciences (USUHS) has done in medical education by adding "700 additional hours of military education" to the Liaison Committee on Medical Education (LCME) requirements. Conversely, since it is a professional's responsibility to meet the standards of their profession, military residents might accomplish this without a potentially limiting proscriptive set of requirements. Sources for independent military professional education already exist in several forms. All the military services offer leadership courses appropriate for officers during residency (e.g., Captains Career Course, Squadron Officer School, etc.). The Borden Institute offers a wide variety of free textbooks on military medicine that should be on the reading list of any physician concerned about being qualified to care for those in harm's way.

It certainly would be an exciting chapter in military GME if military professional education standards were defined and implemented. Until then, the duty to uphold the position of officer lies on the shoulders of residents.

Capt Eric G. Meyer, USAF MC
Lackland AFB, TX

Disclaimers: The opinions expressed in this document are solely those of the author and do not represent an endorsement by or the views of the University of Texas Health Science Center at San Antonio, the Uniformed Services University of the Health Sciences, the United States Air Force, the Department of Defense, or the United States Government.

Dear Editor:

In Response to: "Case Reports: Death of Active Duty Soldiers Following Ingestion of Dietary Supplements Containing 1,3-Dimethylamylamine (DMAA)" (**Mil Med 2012; 177(12): 1455–59**).

As medical examiners, toxicologists, and staff physicians at the Armed Forces Medical Examiner System (AFMES), our mission is to determine the cause and manner of death for service members and deaths under federal jurisdiction. The AFMES is assigned as the repository for DOD mortality data by Department of Defense Directive 5154.30. As such, our organization tracks and monitors all active duty deaths and has knowledge of the circumstances of the deaths that are described by Eliason et al (2012).

The recent article by Eliason et al (2012) describes two cases of military personnel who were taking commercially available dietary supplements containing DMAA who collapsed during physical exercise and died. DMAA is a small molecule that shares structural similarities to amphetamine and has sympathomimetic properties. DMAA has been used as a nasal decongestant, a weight loss supplement, an athletic performance enhancer and a party drug. The authors make several valid points, including the importance of supplement use in the patient history. However, we would like to address several data and interpretation concerns.

The description of Case 1 history fails to mention that this soldier was new to the area, was engaged in physical training for the first time without prior acclimatization, and the cause of death was determined to be hyperthermia by the AFMES. Case 2 was 31 years-old, not 32, and her DMAA level was not "performed 4 days after admission," but was received by the AFMES Forensic Toxicology Laboratory five days after the incident. The date the sample was obtained is unknown to us, but was drawn while the patient was alive in the intensive care unit, likely upon admission. The DMAA level for Case 2 was incorrectly cited as 0.02 mg/L in the discussion section when the actual value was 0.04 mg/L. The Case 2 autopsy was performed by a civilian medical examiner. In both cases, the cause of death was recorded as "hyperthermia" with the manner of death classified as "accident" with a note in the opinion of both autopsies that the presence of supplements is of unknown significance. The DMAA level quantitation was performed by the Forensic Toxicology Laboratory at the AFMES in both cases.

We also were concerned about some aspects of the interpretation offered by the authors. The authors mention that Case 2 had sickle cell trait and that sickle cell trait is known to be associated with hyperthermia and rhabdomyolysis during strenuous exercise. The authors' statement, "the ambient temperature at time of collapse was approximately 23°C/73°F and thus an unlikely explanation for cardiac arrest," is incorrect because heat injury can occur at low environmental temperatures (71°F) in unacclimated individuals. While ephedra and DMAA share some similar properties, suggesting that "this entire scenario is reminiscent of ephedra" is an overstatement in the sense that, unlike ephedra, no military or civilian fatalities have occurred in which DMAA was believed to have caused the death. The AFMES recently collaborated with the Army's Office of the Surgeon General to review the circumstances of all fatalities involving exercise in which DMAA possibly could have contributed to death for all active duty fatalities and found very low (0.026 – 0.22 mg/L) detectable DMAA levels in only four cases; these levels were too low to credibly attribute DMAA as a cause of death.

It should be noted that the countries that are regulating DMAA do so primarily because of widespread recreational use and abuse involving much larger doses of DMAA, resulting in blood concentrations that are four to 50 times greater than levels in these two cases (Gee et al., 2012. Ann Emerg Med 60: 431–434). The DMAA levels in the cases presented were much lower than those seen in cases in which DMAA more convincingly contributed to an adverse effect. Eliason et al. (2012) listed caffeine and DMAA and their synergetic effect, but only attributed or related death to DMAA. We believe that further research on the effects of DMAA, particularly synergistic effects of DMAA in combination with other substances, is warranted, but the association of DMAA with sudden death in these two cases is questionable cases is premature.

Edward Reedy, PhD, MD
Timothy Lyons, PhD, MS
Peter Seguin, MD, MPH
Abubakr Marzouk, MD
Dori Franco, DO
Christine Erdman, PhD, MPH
Armed Forces Medical Examiner System
Dover AFB, DE

Dear Editor:

In Response to Letter to the Editor Re: "Case Reports: Death of Active Duty Soldiers Following Ingestion of Dietary Supplements Containing 1,3-Dimethylamylamine (DMAA)" (**Mil Med 2012; 177(12): 1455–59**).

We thank the authors for their detailed letter and we wish to take this opportunity to again highlight and acknowledge the superb work that the professionals of the Armed Forces Medical Examiner System performed in these two cases and for their overall contributions to the safety of our armed forces personnel. However, we would like to address some of the concerns raised in their letter regarding our presentation of two patients who died following ingestion of DMAA.

Regarding the presentation of Case 1, we were informed that the patient was acclimated as he had just transferred from the rigorous physical environment of advanced infantry training school in Fort Benning Georgia, a site at least as challenging as the mountains of West Texas.

Furthermore, while Dr Reedy and team are correct that per AFMES the ultimate cause of death was hyperthermia, the statement that "heat injury can occur at any temperature" is too simplistic. Certainly they agree that the probability of morbidity and mortality due to heat injury is directly correlated to ambient temperature and humidity.[2] Therefore a mild dry El Paso morning is less likely to be causative of heat stroke than would a humid hot afternoon; consequently additional factors (including toxicological) should be searched for when a young Soldier collapses. It is well documented that caffeine and combinations of stimulants (thermogenic agents) can significantly increase metabolic rate, which could have contributed to an additional heat load.

Regarding the presentation of Case 2, we appreciate their pointing out that the second patient was indeed 32 years old at the time of her initial collapse and not 31 years old, 6 months as we reported. However, we believe that we disclosed her sickle cell trait and its potential implications for heat injury in both the case report and discussion sections and cited no less than 9 published references linking sickle cell trait and heat injury.

Drs. Reedy and colleagues are concerned with our statement that these patients' deaths are reminiscent of those previously associated with ephedra and indicate that our "association of DMAA with sudden death in these two cases is premature." However, as its foundation this was a simple case series of two young Soldiers who used DMAA and who suffered hyperthermic cardiac arrest. The entire purpose of case reports is to generate hypotheses, not to test them; to alert other clinicians to be on the lookout for similar situations; and to stimulate careful prospective scientific inquiry into an important research topic. More than 30 years ago, clinicians described a few clustered cases of association between the recreational supplement amyl nitrite and the development of a rare cutaneous cancer known as Kaposi's Sarcoma in homosexual men. Although the proposed causation of amyl nitrite to Acquired immunodeficiency syndrome was subsequently disproven in favor of a retroviral etiology, these published reports were useful for encouraging further inquiry into what later became a landmark medical discovery. The case report format remains an important scientific forum for reporting just such early associations (and not causations) as we did in our paper. If we had made bolder statements of "X caused Y", we would indeed have been guilty of the classic logical fallacy *cum hoc ergo propter hoc* ("Correlation does not imply causation").

We appreciate the collaborative efforts of AFMES and the Army's Office of the Surgeon General to review the potential for adverse effects, if any, that DMAA may have on our active, young military population. Although the DMAA serum levels in the reported military deaths "were too low to credibly attribute DMAA as the cause of death," the toxicity level as a concern is still unclear. We had made that point in our report.[1] It is possible that DMAA acts synergistically with other sympathomimetics like caffeine, but the extent of that potentially adverse synergy is still unclear. As a result of the missing links in the general understanding of this substance, and the announcement by the Food and Drug Administration on April 11, 2013 that "dietary supplements containing DMAA are illegal", DMAA containing products can no longer be sold on any military base or post. We thank you for the opportunity to respond to your letter.

References are available from the author upon request.

Michael Eliason
Patricia Deuster, PhD
Bruce Adams, MD
Anthony Cancio
Lori Bestervelt, PhD
Amy Eichner, PhD
Uniformed Services University
Bethesda, MD