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Executive Summary

Background

Flashbang grenades (FBGs) are a type of intermediate force capability used in operational contexts to create diversions, confuse or disorient those exposed, or control crowds. These target behaviors are achieved through the activation of psychological and physiological responses to FBG components (flash of brilliant light, auditory bang, overpressure) while minimizing significant injury to its human targets. Knowledge of the psychological and physiological effects of FBG exposures is critical for technology developers and operators to understand weapon-effect thresholds. IDA has identified the stress response as a strategic core area in FBG effectiveness. The objective of this report is to analyze stress as a potential significant mediator of FBG target behavior. Our analysis is concerned with how the stress response is triggered, but more important, how the stress response motivates a behavioral change so that the military goal can be achieved. We focused on the DoD context for FBG use and behavioral outcomes or targeted behavior of use. In addition, we provide research opportunities to further advance JIFCO's FBG portfolio in characterizing FBG human effectiveness.

Foundational Research on the Stress Response

Stress, the body's response to a challenging physical or psychological stimulus, is triggered when an individual's homeostatic balance has been disturbed (Lazarus R. S., From psychological stress to the emotions: A history of changing outlooks, 1993). As conventional FBGs are typically deployed, target audiences are exposed to short-duration detonations of limited explosive (sound, light, pressure) energy, inducing acute stress effects that trigger a complex cascade of biological processes within the central nervous system and peripheral tissues. This cascade is a defensive or protective mechanism to counter the stressor and/or survive the threat. Although some of these processes occur as part of the startle reflex, this reflex should not be confused with the stress response because they are in fact two distinct physiological reactions; the startle reflex can be thought to occur during the very beginning of the acute stress response. Although the human effects of FBG exposure are complicated by a variety of factors (e.g., duration, intensity, predictability of stressor; how stressor is perceived by subject), to better understand these effects we recommend a multimodal approach that continuously and unobtrusively measures the physiological parameters of the stress response.

Psychological Effects of Flashbang Grenades

FBGs have *psychological effects* that fall in three broad capabilities, mediated via the stress response: (1) create diversions via a short-lived startle reflex; (2) disorient and confuse individuals, impairing memory and cognitive processes; or (3) control or disperse (move) crowds via flight/fight/freeze actions. Several research opportunities exist in each of these areas.

Create Diversions

1. Include time as an independent variable—There are few data on how long an FBG acts as a diverting stimulus. The startle reflex lasts only a fraction of a second and has little effect on ongoing actions, while the defensive reflex (fear and stress) can last seconds, even a few minutes.
2. Incorporate individuals with differing experiences—The probability and amplitude of the defense response depends on the existing fear state of performers (e.g., PTSD), as well as the intensity and valence of the emotional stimulus.
3. Employ FBGs in combination with other stressors—Although there is scant research on this topic, FBGs used in combination with other internal and external stressors could increase, or in some cases decrease, FBG effects. Internal stressor could include the physiological state of the individual; external stressors could include other intermediate force capabilities, such as the Active Denial System, or multiple-impulse flashbang devices.
4. Employ more realistic tasks and test conditions—FBGs are deployed on groups of people engaged in heterogeneous activities. FBGs or similar stimuli should be tested in group settings, and tasks should resemble behaviors similar to those that FBG deployers would want to disrupt. Researchers should measure the speed and accuracy of task performance over time—before, during, and after FBG detonation.

Disorient and Confuse

1. Factorially combine stress intensity and timing—To achieve the desired effect on a particular cognitive capability requires a specific mix of stress intensity and timing. Researchers could factorially combine these variables to demonstrate the effects of stress intensity and timing within and between tasks. Results could be used to provide guidance on how best to deploy FBGs for specific effects.
2. Assess effects of acute stress on different types of tasks—Incorporate more realistic and relevant tasks that capture aspects of performance that could be controlled by FBGs, such as spatial navigation or attentional vigilance.

Controlling Crowds

1. Test movement patterns—If a crowd perceives FBG explosions as threats to their physical safety, the detonation can evoke or stop movement, depending on conditions. Relevant to the tactical deployment of FBGs is assessing how targets move if provided an escape route or freeze if no escape route exists.
2. Adopt an observational research strategy—One approach to overcome some of the shortcomings of human research on stress and movement is to develop a database of results from FBG deployments that have been recorded in video repositories, such as YouTube. Researchers could start by coding crowd reactions (e.g., freezing), as well as relevant conditions (e.g., day vs. night conditions). Examining their intercorrelations would help us better understand these FBG effects in actual tactical deployment situations.

Vision Effects and the Stress Response

The primary objective of the "flash" component of FBGs is to elicit a temporary response through the targeted individual's visual system via ocular pain and bleached photoreceptors (leading to the development of flash blindness, where the only thing visible is an afterimage of the flash) to achieve the target behaviors. Flash-blindness effects depend on the task and prevailing light conditions, making the effect of flash blindness difficult to predict (Kosnik, 1994). In addition, perception and attention may play a role in the duration of afterimage duration. If flash blindness occurs, it can temporarily disturb vision and concentration, which can affect performance. In response to stress, pupils will dilate, while in response to light, a stronger constricting action will occur, resulting in a reduction of light entering the retina. This constriction of the retina may serve as a protective mechanism. It may be that the flash component is the least stress-inducing component of the FBG and has a different function in weapon design (e.g., overstimulation).

Research Opportunities

1. Timing of flash-blindness effects—If flash blindness is a short-lived deficit, the effects of the flash on the stress response might also be short-lived. In other words, it might be the case that the flash plays a role independent of the stress response, perhaps through stimulus overload.
2. Psychological effects of temporary blindness or partial blindness—One open question regarding the flash component is its connection to the stress response. While the physiological effect of flash blindness might be short-lived, the lasting psychological effects of being temporarily blind or partly blind might be extremely stressful and therefore have a significant effect on behavior and task performance.

3. Physiological effects—The pupillary light reflex might serve to protect the eyes from flash blindness, thus reducing the flash effectiveness; however, the research is inconclusive. In addition, stress priming might moderate the protective effects of the pupillary light reflex.

Auditory Effects and the Stress Response

The bang component of FBGs is a type of acute noise stressor using decibel levels typically between 170 dB and 180 dB to trigger a psychophysiological response in a target. The stress response generated from acute noise has significant psychological and behavioral effects on the affected person. A number of different stress hormones affect auditory function (e.g., cortisol, adrenaline), and they are secreted differently, depending on the intensity of the noise exposure. In the context of FBGs, a sudden increase in noise is especially stressful and can disrupt ongoing activity due to unpredictability and a person's lack of control. The cognitive and behavioral effects of sudden noise exposure are inextricably tied to the stress response—a sudden acoustic stressor can change a person's behavior by lowering performance or affecting interactions toward others because of overall psychological and cognitive stress (Kjellberg, 1990).

Research Opportunities

1. Stress and priming—Anticipatory effects of sound can affect one's behavior and stress response. For example, individuals previously exposed to a loud bang might modify their behavior in anticipation of a sound in similar situations. In addition, it is important to understand the effects of impulse or sudden noise when there is considerable background noise.

2. Performance outcome variation—Outcome variables for FBG include motor movements (e.g., target accuracy), as well as cognitive (e.g., target identification and acquisition) and communication outcomes. Varying the outcome to understand more acutely which behaviors are most affected by noise exposure and the stress response can provide a wider understanding of FBG effectiveness. Auditory functioning tasks van be measured via cortisol, heart rate, speech intelligibility, cognitive flexibility (e.g., remembering GPS coordinates), and spatial awareness (moving/escaping)

3. Combined effects—While previous research has investigated FBG components individually, combining the bang component with vision effects or overpressure will shed light on the specific role of the bang in generating the stress response and effect on subsequent behavioral outcomes.

Overpressure and the Stress Response

Blast overpressure is a form of a shock wave at pressure levels above normal atmospheric pressure resulting from a sonic boom, an explosion, or firing of a weapon (Sherman, 2014). This overpressure can damage the vestibular system and lead to dizziness, which can activate the stress response. In addition, the overpressure stimulates the autonomic nervous system (via heart-related effects due to the activation of the vasovagal reflex), which also results in the activation of the stress response. Because there is scant research on overpressure and stress, for human effectiveness of FBG exposure, a critical research effort is needed to disentangle the mechanisms and time course for the differential contributions of the blast from the auditory trauma components since they are closely related. This work is needed not only to better understand the critical components of the FBG that lead to desired actions in the targets but also to better characterize the risk of significant injury caused by FBG exposure.

Research Opportunities

1. Establish connection between physiology and performance decrements—Due to limited research on overpressure and performance, the first step would be to establish which physiological component of overpressure leads to distraction effects and performance decrements. There is some anecdotal evidence that the overpressure component from FBG is potentially the most physiologically significant component. Part of this effort could include disentangling the mechanisms and time course for the differential contributions of the blast from the auditory trauma components.
2. First-order effects of overpressure and the stress response—After understanding which overpressure components contribute to performance decrements, establishing the connection between the stress response and overpressure would greatly improve understanding of FBG human characterization. This could include cognitive and behavioral effects due to vestibular/thoracic effects or psychological response to stress.

Contents

1. Introduction

A. Background

The Department of Defense (DoD) states that non-lethal weapons (NLWs) are weapons, devices, and munitions explicitly designed and primarily employed to immediately incapacitate targeted personnel or materiel, while minimizing fatalities, permanent injury to personnel, and undesired damage to property (DOD, 2013, DOD, 2015). NLWs are intended to have reversible effects on personnel and materiel. Counter-personnel NLWs can potentially deny access to and move, disable, and suppress the targeted personnel (DOD 2013). Examples of counter-personnel IFCs are sting-ball grenades, human electro-muscle incapacitation (HEMI) devices (TASERS), dazzling lasers, and flashbang grenades (Joint Non-Lethal Weapons Program (JNLWD) 2013).[1] As part of the DoD Directive 3000.03e, DoD Executive Agent for Non-Lethal Weapons and Non-Lethal Weapons policy, it is DoD policy that:

> Developers of NLW will conduct a thorough human effects characterization in accordance with DoD Instruction (DoDI) 3200.19 to help understand the full range of effects and limitations prior to operational employment of NLW.

Flashbang grenades (FBGs) are a type of NLW, also known as an intermediate force capability (IFC), used to induce psychological and physiological responses while simultaneously minimizing significant injury in human targets. The explosive energy from FBGs generates a loud boom and a flash of bright light intended to temporarily deafen and blind. FBGs have three main components: the flash (or light), the bang (or sound), and the blast overpressure (i.e., sudden increase in air pressure). FBGs cause a range of human effects, owing to a combination of these three components. The effectiveness of the device depends on the appropriate design for the context of use (e.g., the user could disable a person or move a crowd, depending on desired behavior effect from the weapon), extensive laboratory and field testing, and user training. The importance of IFC effectiveness research has been discussed by a number of researchers, all noting that IFC effectiveness is not fully understood, especially regarding cognitive and psychological effects on behavioral outcomes due to IFC exposure. Information about how IFCs affect people psychologically and physiologically is critical if technology developers and operators are to understand weapon-effect thresholds. A full review of IFC effectiveness is outside the

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[1] In 2019, JNLWD formally changed its name to the Joint Intermediate Force Capabilities Office (JIFCO).

scope of the current work, but we point the reader to the following references for more detailed discussions of weapon effectiveness: (Burgei, Foley, & McKim, 2015; Cazares, Belanich, Snyder, Picucci, & Holzer, 2015; Mezzacappa, 2014; Mezzacappa, et al., 2017; National Research Council, 2003; North Atlantic Treaty Organization (NATO), 2009; Rappert, 2004; Silver, 2005).

Prior work conducted by IDA on the human effects of FBGs includes Madhavan and Srinivasan (2018), who provide a comprehensive review of the physiological startle response to FBGs. The startle response, an involuntary activation of the motor tracts generated in the brainstem, is the fastest known generalized motor reaction in humans and animals. It is characterized by an immediate activation of the facial and skeletal muscles leading to a whole-body flinch within a few milliseconds. In humans, the startle response is commonly elicited by auditory stimuli, but can also be generated via visual, somatosensory, and vestibular stimuli. Further details about the startle response will be discussed later in this current paper as it relates to the present topic—the stress response.

A second effort conducted by IDA characterizing the human effects of FBG is Madhavan and Dobbins (2018), who developed a theoretically driven causal analysis model (formally "path analysis") that isolated the physiological and psychological effects of FBGs. The causal model identified five immediate effects of flashbangs:

- Overpressure effects—the physiological effects of sudden increases in air pressure following the detonation of a flashbang.
- Acoustic effects—the consequences of sudden loud bursts of sound characteristic of the "bang" component on the human neural and motor systems.
- Vision effects—the consequences of sudden temporary blinding bursts of light characteristic of the "flash" component on the human oculomotor and other systems.
- Startle effects—the shock or "startle" element associated with flashbangs and psychological and physiological distress it might trigger.
- StartReact—a potential improvement in the performance of planned motor movements.

Based on these five immediate effects, Madhavan and Dobbins (2018) developed a detailed causal diagram that illustrates the sequential steps in human responses to FBGs that can used to develop robust, theoretically motivated human-subject experiments.

IDA made key, evidence-based modifications to the causal model in an effort to understand which paths in the model are worthy of experimentation, can be assumed not to contribute to effectiveness, or are knowledge gaps regarding human effectiveness. Figure 1 shows the most recent causal model (see Madhavan & Dobbins 2018 for the original). Overall, the causal model update provided a systematic outline of research that can provide greater granularity in our understanding of the effectiveness of FBGs. The proposed research consists primarily of human or animal subject experiments progressing from testing first-order sensory effects to testing cumulative effects of FBG components. In addition to identifying behavioral human or animal experiments, IDA also identified key areas that need further exploration in the form of a gap analysis. One strategic core area is the role of the *stress response* in FBG effectiveness (not to be conflated with the startle reflex; this important distinction will be discussed in subsequent sections).

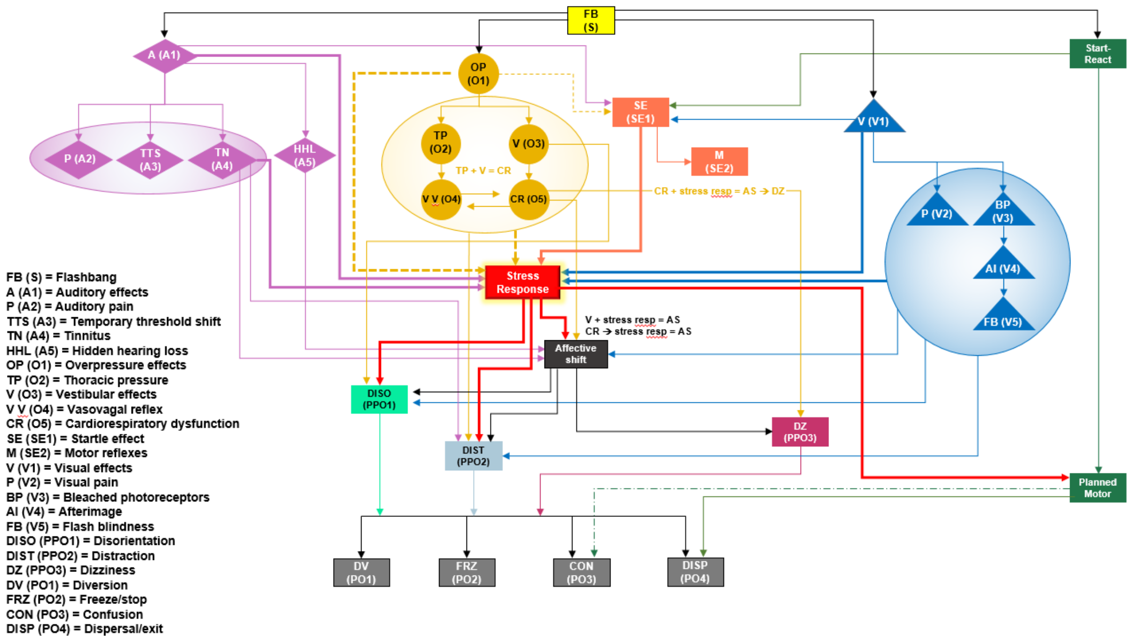

Figure 1. Causal Diagram Representing Integrated Effects of Overpressure, Startle, StartReact, and Disrupted Hearing and Vision on Performance Outcomes

Stress, the body's physiological, psychological, and behavioral response to a stimulus or event (Kavanagh, 2005), develops when the demands of the environment exceed the physiological and psychological resources of the individual (Delahaij & Gaillard, 2008; Lazarus & Folkman, 1984; Taylor, 2007). Stress has been shown to motivate a wide range of outcomes, from the facilitation/inhibition of overt behavioral responses to the enhancement/debilitation of cognitive processes. The stress response, one of the most robust and reliable responses, has been researched for centuries in both animals and humans (our understanding of the stress response comes from animals). Because of its central role in psychological and physiological changes, it is potentially a significant driver of FBG human effectiveness.

B. Objectives

As the casual model shows, a number of potential factors may influence goal behavior (e.g., moving, disabling). From Figure 1, it's clear that many of the physiological effects lead to the stress response, suggesting that this response is a potential mediator for FBG effectiveness behavioral outcomes of interest. In addition to the physiological effects, FBGs have additional *psychological effects* that are mediated through the stress response. Most claims for psychological effects fall into three broad categories of capabilities: that FBGs are able to (1) create diversions; (2) disorient and confuse individuals; or (3) control or disperse (move) crowds.

The relationship between FBG components (flash, bang, and overpressure), a target's stress, and behavioral outcomes can be visualized in the simplified schematic in Figure 2. The relationship between FBGs and goal behavior for use has been established (path A). For example, if the goal is to temporarily blind or deafen a target, path A is involved. However, if the goal is to create diversions, disorient and confuse, or control/move an individual or crowd (i.e., affect task performance and behavior), path B is involved. The objective of this report is to analyze path B as a potential significant mediator of behavior. Our analysis is concerned with how the stress response is triggered, but more important, how the stress response motivates a behavioral change so that the military goal can be accomplished. Our focus is on the DoD context of FBG and behavioral/targeted outcomes for FBG use. The next chapter details how many of the behavioral and cognitive effects of FBGs are mediated through the body's response to stress.

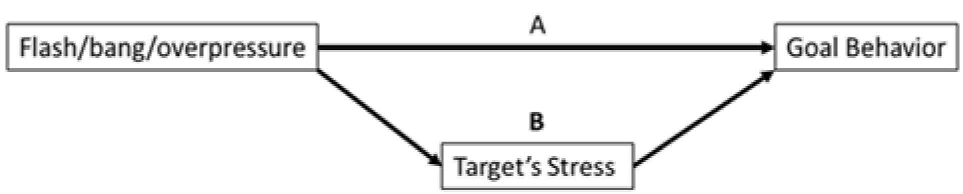

Figure 2. Simplified Schematic of FBG Behavioral Paths. The stress response is a potential significant mediating factor of one's behavioral response to FBGs.

With that said, our goal in this report is to analyze what makes FBGs effective; we strategically focus on the stress response induced by an FBG as one of the possible reasons for behavioral change by the target. We present empirical evidence that inform the current understanding of the stress response as it relates to FBGs, and we identify key priority related areas (gaps) for research. The research presented in this report draws from the literature on both human and animal stress response. These key gaps will be presented, along with a discussion of methodological considerations (e.g., metrics to consider) to implement in future efforts. While the focus is on human effectiveness, the stress literature spans both human and animal models, and therefore we do reference animal models from an existing literature and potential research standpoint. In addition, although this work focuses on outcomes for targeted individuals, we also consider effects of FBGs on the warfighter and address this whenever possible.

This report is organized as follows. First, we define the stress response and discuss the neurochemical and biological processes that follow once someone is exposed to a stressor. Second, we present the second side of the stress response—the psychological and behavioral effects that occur that ultimately would affect someone's performance and behavior after being exposed to an FBG. Next, we discuss each of the key FBG components—visual, auditory, and overpressure—providing key physiological detail where appropriate to understand the stress response; in addition, we detail the psychological and behavioral outcomes connected to each component. Through each chapter, we identify gaps and priority areas for further human-effects-characterization work central to furthering technological development.

2. Foundational Research on the Stress Response

Stress is the body's response to a challenging physical or psychological stimulus. Often, this stimulus induces fear, which is a normal reaction to threatening situations and is a common occurrence in the daily lives of both humans and animals. From an evolutionary perspective, fear is not the subjective experience of being afraid, but instead the ability to detect and respond to danger. As a, result the ability to detect and respond to danger is a function that fear responses evolved to perform. The subjective feelings of fear that occur when this system is activated in the human brain is a result of not only these unconscious fear responses but also a separate system for conscious awareness (e.g., Kahneman, 2011). Because each of these processes occur involuntarily in the presence of danger, the neural systems underlying the response to a fear-inducing stimulus are relatively similar in animals and humans (LeDoux J. , 2000).

The body's response to such challenging stimuli is the stress response, which is triggered when an individual's homeostatic balance has been disturbed (Lazarus R. S., From psychological stress to the emotions: A history of changing outlooks, 1993).[2] The exposure duration to the stressor and the subsequent stress response can be classified as either acute (short-term) or chronic (long-term), each having different effects on the health, well-being, and performance of the subject that is stressed (Arza, et al., 2019; Epel, et al., 2018; Hellhammer, Stone, Hellhammer, & Broderick, 2010). As conventional FBGs are typically deployed, target audiences are exposed to short-duration detonations of limited explosive energy; thus, FBGs are considered to be capable of inducing only acute stress effects. However, it is theoretically possible to evoke chronic stress if targets are subjected to repeated FBG detonations or to extremely high energy (and physically dangerous) explosions.

The body's response to acute stress provides the necessary alertness, energy, physiological regulation, and immunological activation to counter the stressor to survive.[3] The stress response activates a complex cascade of biological processes within the central nervous system and peripheral tissues such as in the autonomic nervous system (e.g.,

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2 Homeostasis is the body's optimal steady internal, physical, and chemical conditions maintained by living systems.

3 In fact, animal research has shown that because environmental conditions can change quickly, engaging and being able to quickly switch between defensive behaviors increases the probability of survival (Hellhammer et al., 2010).

increased heart rate and blood pressure), endocrine system (e.g., hormone release), skeletal system (e.g., conditioned immobility), immune system, modulations of pain sensitivity (analgesia), and somatic reflexes (e.g., startle, eye-blink responses) (Smith & Vale, 2006). The changes in or activation of these processes are dependent on time and proximity to threat in terms of their onset; some engage in a rapid first-wave response while others are delayed (second-wave response) (Rodrigues, LeDoux, & Sapolsky, 2009; Sapolsky, Romero, & Munck, 2000; Stockhorst & Antov, 2016). This chapter provides background to understanding the body's response to stress. The next section describes a prevailing model of the stress response that informs both biological and behavioral research.

A. The Defense Cascade

Several decades ago researchers began to describe the physiological and behavioral reactions to stress as not a single response, but rather as "a cascade of different response events that change in different ways and at different levels as activation increases…" (Bradley, 2000, p. 630). This stream of multiple responses has been labeled the "defense cascade." One of the more influential models of this cascade was that developed by Schauer and Elbert (2010). This particular model is highlighted because it describes the full biphasic nature of the defense cascade from the initial uproar of the nervous system to its potential shutdown at extreme exposure to stress.

As illustrated in Figure , the Schauer-Elbert model includes six progressive stages of fear and distress as the organism endures mounting distress and experiences increasing dissociation, a defensive disposition, "which includes alterations in perception of time, place, and self during and immediately after trauma exposure" (Schauer & Elbert, 2010, p. 111). Starting with the organism in an attentive immobile state, the arousal pattern increases up to Stage 2/3 (Flight/Fight) due to activation of the sympathetic nervous system (SNS). However, at the point of maximum threat imminence, Stage 4 (Fright), the SNS is co-activated with the parasympathetic nervous system (PNS), and the organism becomes tonically immobile. Beyond this point, the PNS becomes dominant and shuts down the organism, resulting in flaccid immobility (Stage 5, Flag) and even loss of consciousness (Stage 6, Faint). Table 1 describes each of the relevant stages for FBGs (i.e., stages 1–3).

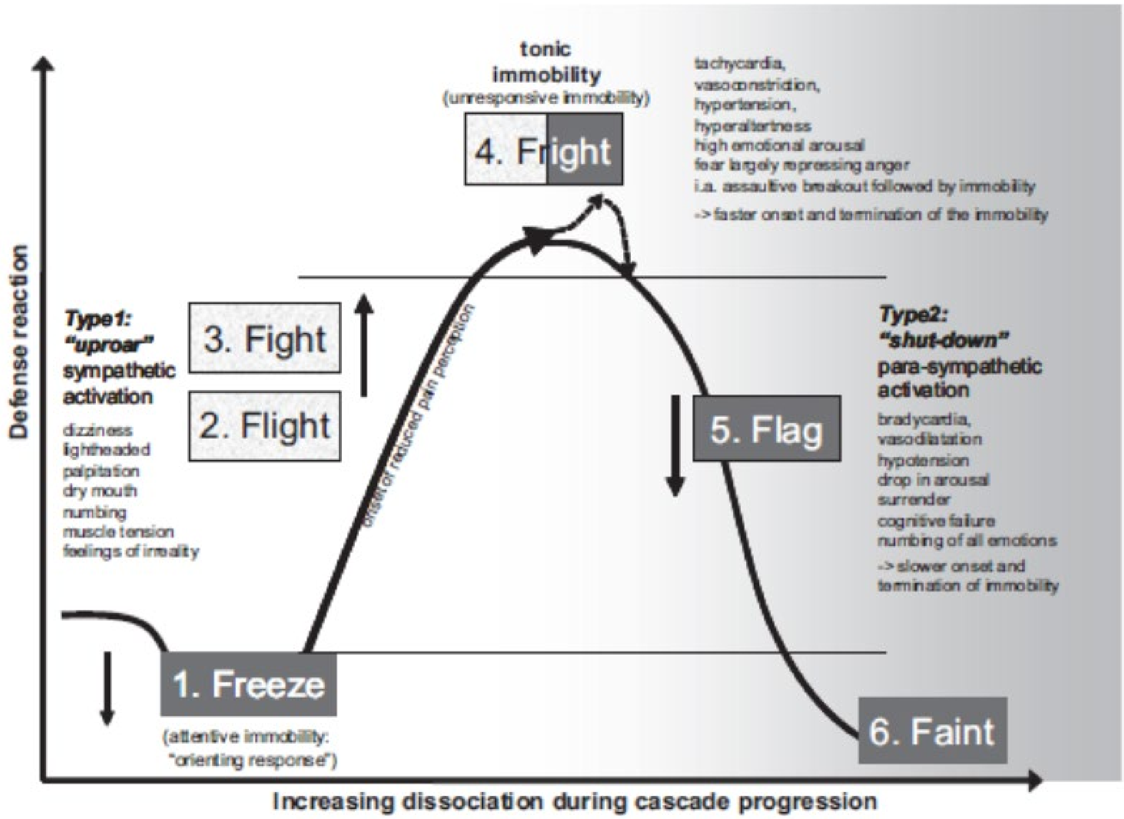

Figure 3. Schematic Illustration of the Defense Cascade as It Progresses along the 6-F Course of Action. From Schauer and Elbert (2010).

Table 1. Three Stages in the Schauer-Elbert Model of the Defense Cascade Relevant for FBGs

Stage	Biological Responses	Behavioral Responses	Cognitive Effects
1. Freeze	**Attend to threat** • Decreased heart rate • Decreased startle response	• Decreased motor activity	• Focused attention on threat
	Prepare to respond • Increased heart rate • Increased startle response	• Continued freeze but prepare for movement	• Heightened sensory perception • Increased processing of contextual details
2. Flight *3. Fight*	**Characterized by the "uproar" due to activation of the SNS** • Discharge of SNS- aka "alarm response" • Prolonged activation of locus coeruleus • Activation of adrenals for counterstrike: heart rate acceleration, blood pressure elevation, vasoconstriction • Release of endorphins to reduce pain	• If escape allowed, flight response is more likely • If escape blocked, fight response or immobility if fighting is untenable	• Endorphins reduce somatosensory perception and awareness

Stage	Biological Responses	Behavioral Responses	Cognitive Effects
	• Reorganization of blood supply: vasoconstriction of peripheral vessels to decrease blood loss from potential injury and increased blood flow through heart and muscles • Increased respiration rate to oxygenate organs and muscles • Increased perspiration to cool body • Reduction in bodily activity (e.g., digestion) not needed		

Using the Schauer-Elbert model to interpret the stress response to FBGs, we suspect that conventional devices are capable of reliably eliciting responses only up through the first four stages (i.e., an acute stress response). However, Stage 4 (Fright) may be achievable only for high-energy FBGs deployed on unsuspecting and naive targets. Also note that stress levels at or beyond Stage 4 could lead to permanent cognitive impairments. Thus, it would be technically—and ethically—difficult to induce Stress Stages 4–6 with conventional FBGs.

Because of ethical concerns, human research on these higher stress stages is very limited. However, there is some research on higher stress stages using fear-inducing stimuli consistent with previous traumatic experiences. For example, images of a firearm pointed at the viewer were presented to victims of gun violence, and audio scripts of personal violence-related trauma were read to victims of violence. Such stimuli can produce increased heart rate and reduced body sway in individuals with related past trauma, which are indicative of Stage 4 tonic immobility (Volchan, et al., 2017).

B. Startle Reflex and the Stress Response

The startle reflex is often confused with the stress response, but they are in fact two distinct physiological reactions. Startle is a reflex that manifests as a rapid, generalized motor response to a sudden, surprise stimulus. It is an oligiosynaptic reflex[4] that is mediated by the brainstem and leads to bilateral blink and activation (almost always) of the craniocervical muscles; however, limb movement in response to startle is variable (i.e., does not always occur) (Hallett, 2012). Madhavan and Srinivasan (2018) described the startle reflex as "…the fastest known generalized motor response [sic] of humans and animals to unexpected or surprising stimuli" (p. iv). Behaviorally, startle temporarily disorients and distracts the affected individual(s), however the next chapter discusses in more detail how the startle reflex is unlikely to be the driving force for mediating behavioral responses due to how short lived it is. As soon as the targeted individuals

[4] Oligosynaptic refers to neural conduction pathways made up of only a few nerve cells and are thus interrupted only by a few synaptic junctions.

perceive the stimulus as non-life-threatening, the startle reflex quickly subsides and the organism returns to its original state of homeostasis. However, when the stimulus effects are intensely aversive or continue to be perceived as a threat, they can trigger a full stress response (Madhavan & Srinivasan, 2018).

Given the timing of the startle reflex, it can be thought to occur during the very beginning of the acute stress response when the threatening and/or unexpected stimulus is detected. This stimulus causes the brain to engage in parallel processing to deal with the threat. Specifically, the defensive response system is activated, leading to the motor effects of the startle reflex. Simultaneously, subcortical pathways engage in "quick-and-dirty" processing of the threatening stimulus. In doing so, the amygdala quickly detects the stimulus, priming it for additional information received along the cortical pathway (LeDoux J. E., 1996; Li, Stutzmann, & LeDoux, 1996). This parallel activation is followed by an orienting response, which relies on the activation of the amygdala to centrally integrate all sensory information triggered by the stimulus (Inman, et al., 2018). It is hypothesized that the orienting response is a result of both motor actions and emotional reactions elicited by the stimulus (e.g., curiosity, fear) (Gogan, 1970). The motor component of the orienting response is highly dependent on context and individual differences. In summary, exposure to a startling stimulus causes quick and immediate activation of cortical and subcortical pathways, leading to the initial startle reflex and orienting response. As the brain continues to process the stimulus, overt or voluntary motor actions are engaged in preparation for defense or attack (Inman, et al., 2018; Madhavan & Srinivasan, 2018).

C. Neuroendocrine Activation in Response to Threat or Stress

During the earliest phases of the stress response, the amygdala quickly and repeatedly fires, sending signals to the hypothalamus, which activates the neurotransmitters and hormones of the sympathetic nervous system. Table 2 shows these secretions (released as part of the first-wave stress response) (Joëls & Baram, 2009; Sapolsky, Romero, & Munck, 2000; Stockhorst & Antov, 2016).

Table 2. Neurotransmitters and Hormones of the Stress Response

Neurotransmitters	Hormones
• monoamine neurotransmitters (noradrenaline [NA], dopamine [DA], and serotonin [5-HT]), • adrenaline and NA from the adrenal medulla	• prolactin • glucagon • growth-hormone • arginine-vasopressin • renin • corticotropin-releasing hormone (CRH) from the hypothalamus,

Neurotransmitters	Hormones
	• adrenocorticotropic hormone (ACTH) from the pituitary • endogenous opiates

The second wave of the response, which takes place several minutes after exposure to the stress-inducing stimulus, includes the peripheral release of glucocorticoid (GC) resulting from activation of the hypothalamus-pituitary-adrenal (HPA) axis.[5] Figure 3 provides details on the timing and stress-response mediators of both waves.

Stress response	First wave	Second wave	
Onset after stressor	Rapid	Delayed	
Onset effects in periphery	Rapid	Delayed	
Duration in the brain Duration in the periphery	Short lived Short lived	Longer, genomic actions Longer, genomic actions	
	Within the brain: Timing of mostly ↑ (increases) and few ↓ (decreases) of single mediators		
Immediately / within sec	↑ Brain monoamines with synaptic actions Noradrenaline (NA) Dopamin (DA) Serotonin (5-HT)	Peak in brain~ 20-30 min Genomic actions lasting hours (h), days (d), months (m)	↑ GCs long-lasting genomic actions
< 1 min	↑ Corticotropin releasing hormone (CRH) Adrenocorticotropic hormone (ACTH)		
< 1 min	↓ Gonadotropin releasing hormon (GnRH) Gonadotropins		
< 1 min	↑ Opioids, Endocannabinoids		
Within min	↑ Non-genomic glucocorticoid (GC)-actions* via transmembrane receptors *: similar for other steroid hormones		
	Periphery: Timing of selected effects		
Immediately / within sec	↑ Sympathetic activity ↑ Release of NA and adrenaline (A) from adrenal medulla	Peak in blood ~20-30 min Genomic actions: h, d, m Tissue: > 1h Genomic actions: h, d, m	↑ GCs from adrenal cortex ↓ Sexual steroids from ovaries, testes
	Feedback to the brain		
	NA, A feedback via vagus nerve leading to ↑ NA within the brain		Peripheral GCs feedback to brain mineralcorticoid receptors (MRs), and GC receptors (GRs)
	Actions on brain fear circuits and microcircuits		
	Transmitters, neuropeptides, hormones also act within fear extinction circuitry		

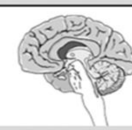

Figure 3. Timing of First- and Second-Wave Release of Stress-Response Mediators. From Stockhorst and Antov (2016).

5 The HPA-axis coordinates responses to real or perceived threats and is intimately involved in adaptation to physical or psychological stress. In response to stress, the paraventricular hypothalamic nucleus (PVN) is activated causing the release of CRH and other hormones. These travel to the anterior pituitary where they elicit the secretion of ACTH, which travels via the circulatory system to the adrenal cortex, leading to the synthesis and release of glucocorticoids (Herman 2010).

D. Measuring the Stress Response

Although a large corpus of literature exists regarding the neurobiological, physical, and endocrine reactions to and outcomes from the stress response, the research is limited on other critical aspects relevant to the use of FBGs. For example, given the potential for long-term, stress-related health implications, a vast (and growing) body of research focuses on the effects of exposure to chronic stressors (both physical and psychological), such as increased risk of cardiovascular disease, stroke, diabetes, post-traumatic stress disorder (Mariotti, 2015). However, there is limited research on the effects of acute stressors such as FBG explosions. That said, acute stress response can be measured experimentally in a number of ways, both invasively and noninvasively. Psychometric questionnaires measure stress through the subjective assessment of behavioral and cognitive change. Such assessments, however, have limited utility for examining the acute stress response due to the rapid onset and habituation of the stress response (i.e., the effects of the stress response likely wears off before a participant can fill our a psychometric questionnaire at the end of an experiment). In experimental settings, such a questionnaire might be the only tool available. If this is the case, the questionnaire needs to be carefully created to retrospectively capture psychological data from stimulus exposure. Likewise, although biochemical markers to measure the hormonal response to stress have been developed, they may not be suitable for a continuous stress-monitoring method. Further, there is a significant gap in the research regarding the complex relationship between the activation of biochemical markers and the intensity of the perceived stress (Arza, et al., 2019). For FBG research, these significant gaps and individual differences that drive behavior make the stress response relatively complex to measure experimentally, although not impossible with appropriately designed experiments. Further, the physiological and psychological responses to stress may differ. For instance, people may not exhibit certain physiological activations, but still have a psychological response to a stressful trigger.

The use of non-hormonal biosignals to measure the psychophysiological responses to stress, on the other hand, has considerable support. A majority of these methods rely on changes in the autonomic nervous system, specifically its sympathetic and parasympathetic components, in response to acute stress. The most commonly used biosignal-based measures are electrocardiography (ECG) (from which heart rate [HR] and heart rate variation [HVR] can be derived) and skin conductance (SC) (Chen, Zhao, Ye, Zhang, & Zou, 2017; Gjoreski, Luštrek, Gams, & Gjoreski, 2017; Han, et al., 2017). Other measures have also been used extensively to study the physical response to acute stress, such as electroencephalograms (EEG), skin temperature, pulse photoplethysmography, respiration, pupil diameter, electromyography, and blood pressure (Mahmoud, Shanableh, Bodala, Thakor, & Al-Nashash, 2017; Vinkers, et al., 2013). Examining the stress response is complicated because the reaction to the stress is dependent on the exposure time to the stressor (acute vs. chronic), how the stressor is perceived by the subject, the intensity of the stressor, the subject's ability (or belief) that he or she can control or cope with the stressor or response, and the predictability of the stressor (Rabasa, et al., 2015). Because of the complicated nature of the stress response, some researchers (Arza, et al., 2019; Oken, Chamine, & Wakeland, 2015) conclude that a single stress marker cannot provide a global assessment of the stress response and instead recommend using a multimodal approach to unobtrusively and continuously measure the physiological parameters of the acute stress response.

3. Psychological Effects of Flashbang Grenades

FBGs are specifically designed to temporarily blind and deafen individuals, but there ae also claims that these devices have additional *psychological effects* that are mediated through the stress response. Most claims for psychological effects fall into three broad categories of FBG capabilities: (1) create diversions; (2) disorient and confuse individuals; or (3) control or disperse (move) crowds. We examine what is meant by these claims and the implications for tactical deployment of these IFCs. We also provide suggested research opportunities for each of these FBG capability areas that aim to further characterize psychological states of an FBG target that lead to weapon effectiveness.

A. Create Diversions

FBGs create diversions by directing attention away from some activity or objects that the blue force does not want the targeted audience to see. For instance, an FBG could be used to divert someone's attention while blue forces enter a room. Startle and defense reflexes and emotion and attention can be exploited to this end. We discuss each in turn.

1. Startle and Defense Reflexes

It is often claimed that FBGs create diversions through their capability to evoke a startle reflex. Indeed, even low-energy FBGs should be able to elicit a reliable startle reflex if a target is sufficiently close to the detonation. Human research indicates that startle reflexes are evoked by most sensory modalities (acoustic, visual, tactile), although acoustic stimuli are studied most often in the laboratory. The stimulus should be moderately intense and relatively brief. The reflex habituates with repeated presentations, but rapidly dishabituates within a matter of seconds or minutes (Lang, Bradley, & Cuthbert, 1990). The most important characteristic of a startle-eliciting stimulus is its nearly instantaneous onset latency. The most common stimulus for eliciting a startle response in the laboratory is a 50 ms burst of white noise at about 95 dBs (Bradley, 2000). In light of these data and based on their sound-producing capabilities, FBGs can be expected to be able to evoke a startle reflex to divert attention. The problem is that the effects of startle are weak and extremely short-lived. The reflex itself lasts only a fraction of a second. In laboratory tasks using acoustic stimuli, the startle reflex has little or no effect on any ongoing tasks (Lang, Davis, & Ohman, 2000). *This suggests that the startle reflex may only be one piece of the FBG effectiveness puzzle, but not the central or sole driving force behind FBG effectiveness.*

To capture and hold attention, the diverting stimulus must be more enduring and potentially threatening. According to the defense-cascade models, more intense and fearsome stimuli induce organisms to freeze and attend to the stimulus in preparation for response. Some researchers have labeled this response as the attentive-immobility response (Kozlowska, Walker, McLean, & Carrive, 2015; Schauer & Elbert, 2010); others refer to this as the defense response (Cook & Turpin, 1997; Graham, 1992; Turpin, 1986). Compared with the startle reflex, the stimuli that elicit the defense response are more intense and sustained with longer onset latencies. The duration of the defense response depends on the intensity and time course of the stimulus and the response modality, but regardless of measurement methods, the duration of the defense response is an order of magnitude longer than the startle reflex. For instance, the cardiac defense response has two components: a brief period of fast heart deceleration followed by sustained acceleration—the whole pattern lasting up to 80 s. Also, the defense response is slower to habituate than the startle reflex.

In summary, these studies suggest that the sound of an FBG may be sufficient to evoke a startle reflex, which could divert attention for a few seconds. But to capture and hold attention, the FBG would need to be more intense to elicit a defense response. We suppose that a defense response could be elicited by accompanying the sound with the visual flash as is usually done, or by detonating multiple FBGs. However, there are currently no empirical studies that identify the conditions under which FBGs or their components reliably evoke defense responses.

2. Emotion and Attention

In addition to having physiological effects on vision and audition, FBGs have emotional effects in that they induce fear in their targets that can divert attention (Madhavan & Dobbins, 2018). In that regard, there is much research indicating that emotionally arousing images help shape information gathering in that motivationally relevant objects receive heightened attention (e.g., Pessoa, Pereira, & Oliveira, 2010). In this section, we selectively review research to show methods and results of how emotional images can direct attention toward an object or divert attention away from another. We connect then these findings to FBG effectiveness.

Much of the published research in this area has employed imagery from the International Affective Picture System (IAPS). The IAPS is a database designed to provide a standardized set of pictures for studying emotion and attention (Lang, Bradley, & Cuthbert, 2008). The images are scaled on affective valence (pleasant vs. unpleasant), arousal (calm vs. excited), and dominance (in control vs. dominated). IAPS pictures have been used to study a variety of psychophysiological measures, such as functional magnetic resonance imaging, electroencephalography (EEG), and galvanic skin response (GSR). The stimuli have also been used to study phenomena, such as attentional capture and attentional blindness. The IAPS could potentially be informative for experimental work for FBGs to explore emotional responses.

a. Attentional Capture

The *dot probe task,* a typical method for assessing attentional bias toward or against emotional stimuli, could be employed in experimental studies. In this task, each trial begins with the presentation of a central fixation cross for 500 ms. The cross is replaced by two stimuli, one threatening and one neutral. After 1000 ms or so, the pictures are removed and replaced by a dot that appears in the location of one or the other stimulus. The dot is removed, and the subject responds by indicating, as quickly as possible, where the dot was located. "Congruent" trials are those in which the dot replaced the threat-neutral stimulus; "incongruent" trials are those where the dot replaces the threat-related stimulus. Research participants are assumed to respond faster to the stimulus to which they are attending. Faster responses to the threat-based stimulus suggest a bias toward the emotional stimulus (vigilance), whereas slower responses indicate a bias against the emotional stimulus (avoidance).

Findings from research using the *dot probe task* indicate that emotional stimuli are more effective at capturing and holding attention, particularly those that convey some sort of threat to the observer (e.g., Bodenhausen & Hugenberg, 2009; Kalat & Shiota, 2007). The findings are not always consistent, however, and appear to depend on a number of moderating variables such as individual trait-anxiety (i.e., high-trait anxiety vs. low-trait anxiety), and threats that pose potential danger or actual harm. Studies show that those with high-trait anxiety are initially more attracted to harm-related stimuli, but then quickly divert their attention (Calvo & Avero, 2005). These temporal effects were not as strong for low-anxiety subjects. Thus, for anxious subjects, harm-related images capture attention initially, but later those same threatening images were avoided.

These findings suggest that FBGs may effectively capture attention to the extent that they arouse emotions or are perceived as a threat to the observer. Conceivably, this could distract attention away from some object or activity that the FBG deployers do not want their targets to see or hear. However, the effects of emotional stimuli vary over time in that observers are first attracted to emotional stimuli and later avoid the stimulus, and differ between individual observers (i.e., less anxious people are less affected by emotional stimuli).

b. Attentional Blindness

Emotion-induced blindness has been studied by variants of the attentional blink paradigm. This paradigm requires subjects to search visually through a stream of sequential images presented rapidly (one every 100 ms, a task called rapid serial visual presentation). For the attentional blink task, subjects are instructed to look for two targets, T1 and T2.

For instance, in a stream of horizontal pictures of various landscapes, the subjects may be instructed to identify targets (landscapes or architectural photos) that are rotated 90 degrees. If T2 occurs too soon after T1 (≤200 ms), detection of T2 is degraded. However, if T2 occurs later in the sequence (≥800 ms), there is no effect on detection of T2. These results have been interpreted as the first target causing a brief blink in attention that impairs detection of the second target. Emotional stimuli are particularly effective in inducing the attentional blink. Research shows that when participants are presented with distracting images of human or animal violence (i.e., an emotional distractor), attentional blink is induced, preventing them from recognizing a target image; these effects can vary by individual (e.g., high or low in hard avoidance) (Most, Chun, Widders, & Zald, 2005).

The attentional blindness findings suggest that an FBG detonation may not have to hold attention to effectively distract attention; rather, an emotionally arousing explosion could effectively block attention to any subsequent stimulus. Again, there are individual differences, observers scoring low in harm avoidance being less susceptible to these effects. Also, like attentional capture, the attentional blindness effect lasts only seconds, but potentially enough time for military operators to perform.

c. Summary

Compared with neutral stimuli, emotion-inducing images are effective in capturing attention and distracting from perception of other stimuli. Research suggests that emotional images capture attention quickly and unconsciously. *Thus, we predict that the effectiveness of an FBG in capturing and diverting attention is positively related to the fear induced by the explosion, which is in part determined by the intensity of the detonation*. These effects these effects may be short lived, however. Typical findings for laboratory tasks indicate that the effects of emotional images last less than 1 s.

3. Research Opportunities

Most research on emotion and attention focuses on individual subjects performing laboratory tasks; ethical considerations restrain the intensity of the fear-inducing stimuli employed in this research. To better understand the capability of an FBG to create diversions via the stress response, r*esearch should address to what extent the existing literature on emotion and attention pertains to operationally relevant settings in which FBGs are typically deployed.* In that regard, methodological approaches used to research the effects of magic tricks on stage audiences (e.g., Macknik et al., 2008) could be adapted to determine the extent to which FBGs control the attention of a group of individuals. Given that context, there are three sets of variables that present opportunities for research: time, individuals with different experiences, and FBGs in combination with other stressors. Each is discussed in turn.

a. Include Time as an Independent Variable

There are few data on how long an FBG acts as a diverting stimulus. The startle reflex lasts only a fraction of a second and has little effect on ongoing actions. On the other hand, the defensive response can last seconds, even a few minutes. But is the diversion of attention longer or shorter than the defense response itself? Thus, the diversion must be measured at different points in time from the onset of the FBG detonation.

b. Incorporate Individuals with Differing Experiences

Cook and Turpin (1997) asserted that there is much laboratory research indicating that probability and amplitude of the defense response depends on the existing fear state of performers as well as the intensity and valence of the emotional stimulus The effect of experience has been demonstrated in multiple recent studies (e.g., Volchan et al., 2011). In the referenced research, trauma-exposed subjects who were diagnosed as either having or not having PTSD listened to audio scripts of their autobiographical traumatic experiences. Subjects with post-traumatic stress disorder (PTSD) showed more signs of tonic immobility, including reduced body sway (as recorded on stabilometer platforms), accelerated HR, and diminished HR variability, than those without PTSD*. In general, the degree to which FBGs distract individuals depend on their previous experience with FBGs or with stimuli and situations similar to FBG detonations.*

c. Employ FBGs in Combination with Other Stressors

Although there is scant research on this topic, it appears reasonable that FBGs used in combination with other internal and external stressors could increase, or in some cases decrease, FBG effects. Internal stressor could include the physiological state of the individual (e.g., sleep-deprived, exhausted, dehydrated, under the influence of alcohol or stimulants). External stressors could include other IFC technologies, such as the Active Denial System, or multiple-impulse flashbang devices (i.e., something to provide additional emotional arousal to capture attention).

d. Employ More Realistic Tasks and Test Conditions

Whereas stress effects are tested individually on laboratory tasks, FBGs are deployed on groups of people engaged in heterogeneous activities. FBGs or similar stimuli should be tested in group settings. The tests should be designed to assess two aspects of attention. First, subjects should be queried to determine the accuracy of their perceptions (or memories) of the situation—especially, of the object of the diversion. Second, to assess ability of the FGB to disrupt ongoing responses, researchers should measure the speed and accuracy of task performance over time—before, during, and after FBG detonation. Furthermore, the tasks should at least resemble behaviors that FBG deployers would wish to disrupt (e.g., group chants, movements toward troops, destructive behavior).

B. Disorient and Confuse Individuals

Research shows that chronic stress can act to profoundly disorient or confuse its victims (Schauer & Elbert, 2010). But as we have discussed, conventional FBGs are not capable of inducing chronic stress. On the other hand, FBGs can evoke acute stress, which has been shown to impair memory and cognitive processes. Although less severe, these cognitive impairments potentially contribute to disorientation and confusion of targeted individuals.

1. Effects of Acute Stress

The effect that acute stress has on cognition depends on at least two variables. One is the intensity of the stressor. Calvo and Guitierrez-Garcia (2016) maintain that mild stress may actually improve performance on simple tasks. But as intensity increases, higher order processes suffer. "In general, under high stress, the more flexible higher-order 'cognitive' functions tend to be replaced by more rigid 'habit' memory functions in the control of learning and response" (p. 142).

Another variable is the timing of the stressor event. In reviewing the effects of stress on memory performance in both human and animal subjects, Schwabe and Wolf (2013) argued that stress can have either a positive or negative effect that is dependent on its temporal relation with the learning and retrieval events. Impairment of episodic memory could sow confusion in targets about their tactical plans. As illustrated in Figure 4, stress administered shortly before, during, or after learning improves memory, whereas stressors that are more remote from the learning event, either before or after, impair memory. The researchers interpret this pattern as an adaptive tendency to form lasting memories of the stressful event:

> …strong memories are formed for information that is present around the time of the stress experience and directly related to the stressor. This memory enhancement for stress-related information, however, may come at the cost of impaired memory for events unrelated to the stressor. (Schwabe & Wolf, 2013, p. 61)

Schwabe and Wolf's (2013) findings suggest that targets tend to forget events that occur well before or after an FBG detonation, but memory is enhanced for events occuring immediately before and after a detonation and during the detonation. *These results suggest conditions where FBGs can actually reduce confusion and produce results counter to the FBG deployers' intentions.*

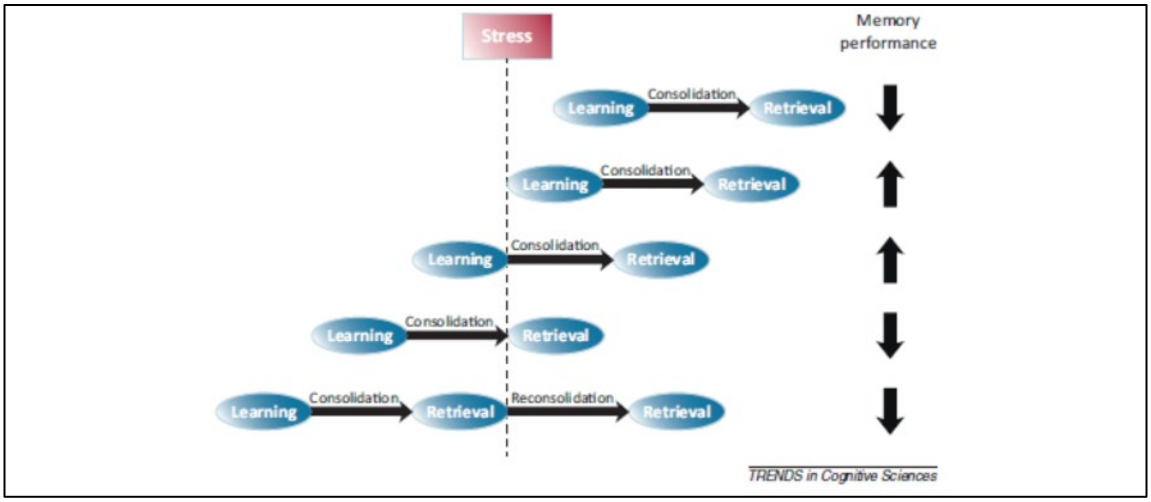

Figure 4. Time-Dependent Impact of Stress on Episodic Memory Performance. From Schwabe and Wolf (2013).

2. Research Opportunities

a. Factorially Combine Stress Intensity and Timing

The research suggests that to achieve the desired effect on a particular cognitive capability requires a specific mix of stress intensity and timing. To test this hypothesis, researchers could factorially combine these variables to demonstrate the effects of stress intensity and timing within and between tasks. Results could be used to provide guidance on how best to deploy FBGs for specific effects.

b. Assess Effects of Acute Stress on Different Types of Tasks

There is accumulating evidence from animal research that whereas moderate stress disrupts cognitive task performance, conditioned habit learning is enhanced; however, there are much fewer data on humans (Schwabe &Wolf 2013), leaving open questions about how stress modulates cognitive and memory system. For human research on FBG's ability to disorient or confuse, research needs to incorporate more realistic and relevant tasks that capture aspects of performance that could be controlled by FBGs, such as spatial navigation or attentional vigilance.

C. Controlling Crowds

1. Flight, Fight, or Freeze

A recent spate of research on the freeze response reveals that certain types of movement or non-movement can be attained, depending on perceptions of threat to the human (e.g., Roelofs, 2017; Volchan et al., 2017). According to this research, the target(s) of a potential attack by a predator (1) freeze (take no action), (2) take flight from the situation, (3) fight their attackers, or (4) simply ignore the attack and continue what they were doing. These movement options depend on the victim's perceptions of (1) the imminence of the attack, (2) perceived available escape route(s) (from the attack), and (3) the expected success of a counterattack. The routes to those alternative responses are depicted as a flowchart in Figure 5. Note that the figure distinguishes between two types of freezes: If the threat is perceived as a potentially dangerous, but not necessarily imminent, the target adopts *attentive immobility,* whereby a target maintains orientation on the threat in preparation for fight or flight. On the other hand, if the threat is perceived as a clear and present danger from which there is no escape, targets become *tonically immobile*, where they shut down their systems' responses to stress in preparation for inevitable injury or even death.

Figure 5 suggests the different "routes" a crowd might take in a stressful situation. However, while Figure 5 depicts a static situation wherein a crowd picks one route based on the situation, a more realistic depiction of this process is more fluid. In other words, in stress-inducing situations a crowd can reappraise as the situation unfolds (i.e., Figure 5 is more "fluid"). For example, in the case of FBGs, an initiated flight response may be interrupted by a reappraisal, wherein an escape route is not unavailable, perhaps leading to a fight response, only to be followed by a reappraisal that the threat is not vulnerable to a counterattack, leading to freeze, and finally another reappraisal once an escape route becomes available, and flight is the "final" response.

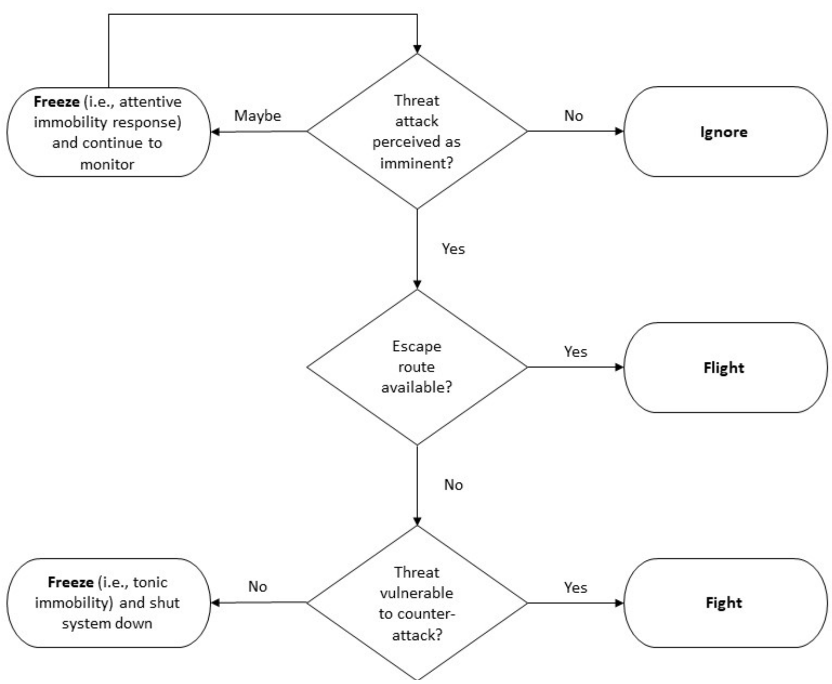

Figure 5. Routes of Defensive Behaviors. Derived and adapted from Volchan, et al. (2017) and Roelofs (2017).

2. Research Opportunities

a. Test Movement Patterns

If a crowd perceives FBG explosions as threats to their physical safety, the stress model presented in Figure 2 indicates the detonation can evoke or stop movement, depending on contextual conditions. Two movement patterns to experimentally assess emerge from the model that are relevant to the tactical deployment of FBGs:

1. If the FBG deploying forces want their targets to move away from a specific location, they need to provide or make targets aware of an escape route to another site. Blocking escape routes could lead to fight or freeze responses.
2. On the other hand, if FBG deployers want their targets to literally freeze in their tracks, the FBG detonation must be fearsome with no way to escape. It is also important that the deployer appear invulnerable to preclude the target crowd from mounting a counterattack.

b. Adopt an Observational Research Strategy

One approach to overcoming some of the shortcomings of human research on stress and movement is to develop a database of results from FBG deployments that have been recorded in video repositories, such as YouTube. Researchers could start by coding crowd reactions (e.g., movement toward vs. away from deploying force, freezing, vocalizations), as well as relevant conditions (e.g., FBG type, crowd size, day vs. night conditions). Examining their intercorrelations would provide productive opportunities to understand these FBG effects in actual tactical deployment situations. Note that such correlational research does not provide definitive proof of cause-effect relations; nevertheless, it may provide evidence for or against in the applicability of laboratory results to field settings. Also, we assume that most existing videos depict FBG use in police actions, and not military operations. Nevertheless, we maintain that police actions more closely resemble military operations than do laboratory experiments.

4. Vision Effects and the Stress Response

The primary objective of the "flash" component of an FBG is to elicit a temporary response through the targeted individual's visual system via ocular pain and bleached photoreceptors (which can lead to the development of flash blindness, where the only thing visible is an afterimage of the flash; see Figure 1). The more intense the flash, the longer the perception of an afterimage (or duration of the flash blindness) (Brindley, 1962). Unlike the single-impulse noise effects of an FBG, which are currently produced at just below the threshold that may cause permanent hearing injury, the flash intensity sits at almost a magnitude lower than the level causing vision damage. This decreased intensity provides a workable safety margin in military and non-military applications and where countermeasures to vision effects can play a role. That said, flash-blindness effects depend on the task and prevailing light conditions, making the effect of flash blindness difficult to predict (Kosnik, 1994); empirically measuring performance is usually necessary to fully assess the effects of FBG flash blindness and other flash effects. Finally, another complicating factor to the effectiveness of the flash component of FBGs is the individual variability to the persistence of the afterimage or flash blindness (Atkinson & Crawford, 1992).

The need for adequate vision in a situation perceived as potentially life-threatening is vital to a soldier or combatant. If some form of light or flash is noticed in darkness, the eyes automatically try to focus the source, making the flash component of an FBG fairly effective (North Atlantic Treaty Organization (NATO), 2006). However, the effect of intense light delivery is not ensured with FBGs because the response of the target significantly interacts with the component's effectiveness. For example, if someone is wearing protective eyewear, is not looking in the exact direction of the weapon, or is keeping an eye closed, the effect of the light component is neutralized. The subsequent sections describe such factors to FBG flash effectiveness and present details regarding the physiological, stress, cognitive, and behavioral effects of flash exposure. The section ends with a summary of research opportunities to better understand the human effectiveness of the flash component of an FBG as it relates to the stress response.

A. Physiological Effects of Flash Exposure

The retina of the eye is lined with approximately 5.5 million photoreceptors, each classified as either rods or cones (see Figure 6). Rods contain rhodopsin (a visual pigment) and are involved in peripheral, low-light vision, while cones (which contain a variety of photopsin pigments to absorb different light wavelengths) are involved in detailed color vision (Schwiegerling, 2004). When the retina is hit by a photon of light (Figure 6B), the light is converted from radiant energy into a neural signal, which indicates the start of visual perception (Youssef, Sheibani, & Albert, 2011). The visual cortex assembles the image, and other cortical areas identify the image. Although cones respond to bright light, the rods are the primary photoreceptor involved in the physiological response to flash exposure. As described by Madhavan and Dobbins (2018), exposure to a sudden brilliant light causes rhodopsin in the rods to absorb the light energy. This light absorption leads to bleaching of the photoreceptor and actually causes the rhodopsin to become almost transparent and unresponsive. Rods are quite sensitive to bleaching; their response saturates when as little as 6% of the rhodopsin molecules absorb light. It is this bleaching of retinal photoreceptors that leads to the perception of a visual afterimage. In the case of exposure to a bright flash of light (e.g., from an FBG), the perception of this afterimage is called flash blindness, where almost nothing is visible except for the afterimage of the flash.

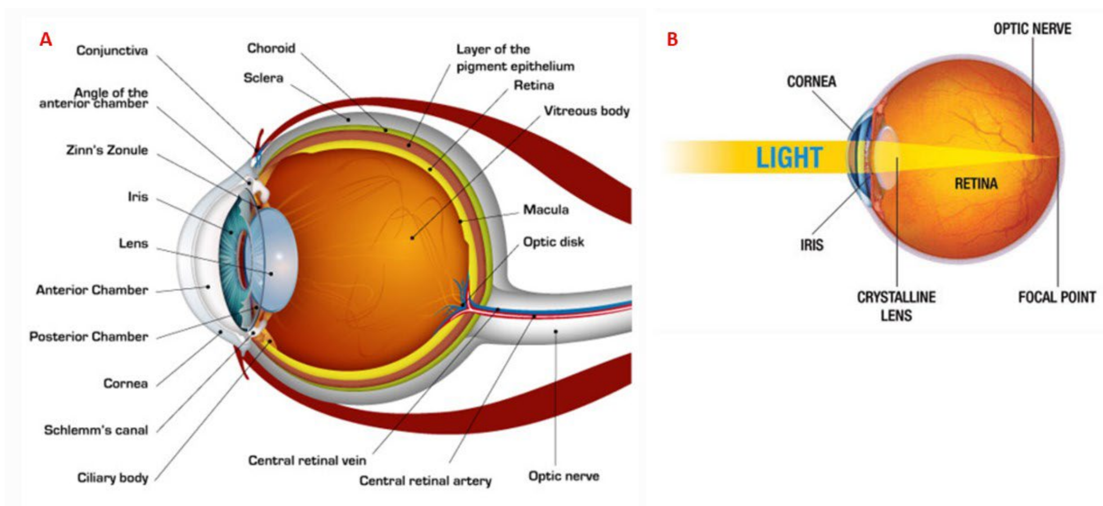

Figure 6. Anatomy of the Human Eye (A) and Diagram of Light Entering the Eye (B). Adapted from the National Keratoconus Foundation (National Keratoconus Foundation, 2020). For a full description of the psychophysical process of sight, see Belliveau, Somani, & Dossani (2020).

The degree to which the photoreceptors are bleached has a direct relationship to how long the afterimage will be perceived (or flash blindness will persist), such that in cases of total bleaching, it takes approximately 25 minutes for the photoreceptors to regenerate; thus, the afterimage should persist for the same length of time (Brindley, 1962).[6] Although complete bleaching of the photoreceptors is possible, this rarely occurs. As a result, most

[6] See Madhavan and Dobbins (2018) for additional information on the physiological effects of the flash component of FBGs.

flash-caused afterimages persist for several seconds to several minutes (Smith & Wallace, 1982); the disappearance of these afterimages is related to dark adaptation. In addition, age plays a role in photoreceptor restoration such that the younger an individual is, the quicker the photoreceptors will regenerate (Messenio, Marano, Gerosa, Iannelli, & Biganzoli, 2013). Any experimental work needs to consider that there will be individual variation in response to the flash component and subsequent behavior. The photochemical process of receptor bleaching alone does not account for the appearance of afterimages; postreceptoral neural adaptation[7] plays a role as well (Suzuki & Grabowecky, 2003). Evidence for this comes from the findings regarding the role that adaptation plays in the perception of illusory contours and the filling in of color, luminance, size perception, and attention in afterimages. This neural adaptation occurs in the cortical visual areas (well beyond the level of the retina) (Shimojo, Kamitani, & Nishida, 2001; Dong, Holm, & Bao, 2017).

Research has identified a number of mediating factors regarding the effects of flash exposure on afterimage formation and duration. In terms of the human eye, pupil size, adaptation state, age of the individual, and location and number of photoreceptors engaged can influence the afterimage. Likewise, features of the flash itself, such as energy incident on the retina, duration of the flash, light wavelength, and number and frequency of the flashes, directly impact afterimages (Madhavan & Dobbins, 2018; VanMeenen, et al., 2006). Further, Hall and Wilsoncroft (1964) showed a significant increase in afterimage duration when subjects were exposed to a flash while the lights flickered on and off.

B. Effects of Flash Exposure on Stress, Cognition, and Behavior

The autonomic nervous system controls the continuous tuning of pupil size by directing the muscles of the iris (see Figure 6) to regulate the amount of light that enters the eye. The sphincter muscles of the iris cause the pupils to constrict, reducing the amount of light entering the eye (known as the pupillary light reflex), while the dilator muscles cause the pupils to expand, increasing the amount of light entering the eye, particularly in low-light conditions.

Interestingly, however, research has shown that the pupillary light reflex is actually much more than a reflex—it is a cognitively mediated response that depends on the brightness of the stimulus, awareness of the stimulus, interpretation of the stimulus, and thoughts occurring simultaneously with the perception of the stimulus (Mathôt & Van der Stigchel, 2015). For example, Naber, Frassle, and Einhauser (2011) have shown that when images of different brightness are presented to each individual eye simultaneously, the pupil constricts when the brighter stimulus dominates attention and the pupils dilate if the

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[7] Neural adaptation in the visual system refers to a brief and temporary change in sensitivity or perception when exposed to a new stimulus or the lingering aftereffects when the stimulus is removed (such as an afterimage) (Webster, 2011).

darker stimulus dominates attention. In addition, the pupils constrict or dilate based on the perceived brightness (which might not actually reflect the actual brightness of the image of an image), even if that image is simply imagined and not actually seen (Laeng & Sulutvedt, 2014; Binda, Pereverzeva, & Murray, 2013). Together, these studies suggest that the pupils adjust to stimuli that are attended to, even if they are not directly looked at. If it is known that the stimulus is bright (e.g., the flash from an FBG), the pupils will begin to constrict as the eyes move toward the bright stimulus and well before the stimulus comes into sight (Mathôt, van der Linden, Grainger, & Vitu, 2015). Based on this literature, the pupils of individuals exposed to an FBG may begin to constrict (which reduces the amount of light entering the eyes) from the flash before it appears in their visual fields because (1) they expect the flash to appear, and (2) they are aware of how bright the flash will be. In addition, recall that the stress response activates a complex cascade of biological processes, including somatic reflexes, that may also lead to the eye-blink response, which may lead individuals to close their eyes in response to a sudden flash or bang from an FBG. The constriction of the pupils and the eye-blink response both serve to reduce the effects of the flash in terms of the duration, intensity, and potential impairments of flash afterimages.

There also seems to be some interaction between cognition (specifically perception and attention) and duration of afterimage perception, though the literature focuses broadly on afterimages and not just those created by a flash of light. Research also indicates that the size of the afterimage depends on the perceived size of the inducing stimulus and that increasing the visibility of the stimulus increases the duration of the afterimage (Sperandio, Lak, & Goodale, 2012; van Boxtel, Tsuchiya, & Koch, 2010).

Further, although not focused on bright light exposure, Smith and Wallace (1982) investigated the role of stimulus recognizability (i.e., ability to verbally label objects) on afterimage persistence. They found that the most highly recognizable stimuli produced the most enduring afterimages (i.e., it is possible that a flash of light in a particular shape will lead to a more persistent afterimage than just a shapeless bright light will). In another study, researchers found that individuals high in visuospatial skills (ability to attend to, manipulate, and evaluate spatial inputs in a mental model of the physical space) experience longer afterimage durations such as those caused by bright, short-lived flashes. These results are thought to be due to the longer visual persistence (or iconic memory) in these individuals (Atkinson & Crawford, 1992).

Existing literature looking at vision effects and FBGs has quantitatively measured distance, threshold, and time effects of the flash component via test events (Beier & Simonds, 2014; Beier, Fleming, & Ashworth, 2017). Human effectiveness is typically discussed in terms of thresholds of response; for example, an FBG meets the required 85% visual obscuration level for 10 s or greater, or the pressure output is the appropriate A-weighted decibels. But what's typically missing from these discussions are the subsequent psychological, cognitive, and behavioral effects that ultimately affect a target's performance. For example, having one's vision obscured at 85% for 10 s will result in what kind of altered task performance? This behavioral understanding is a key component to characterizing human effects of FBGs since the purpose of FBGs is not only to cause physiological effects (e.g., bleached photoreceptors) but also to change behavior.

The flash effect impairs vision and potentially disturbs concentration, which translates to disrupted behavior (e.g., disorientation and confusion). For example, the dazzling effect of light is known to increase reaction times in automobile drivers and also heavily disturb pilot behavior (Santos, Pinto Coelho, Mendonça, & Ferreira, 2019). In fact, Nakagawara, Montgomery, and Wood (2007) identified 58 airline mishaps from the National Transportation Safety Board and Federal Aviation Administration Incident Data Systems that were attributed to pilot vision issues from exposure to bright light after partial or full dark adaptation. These pilots said that they had become distracted or disoriented after experiencing glare or flash blindness from the bright runway lights. The authors noted that exposure to light that is several orders of magnitude more luminescent than what the eyes have adapted to in the dark can reduce or eliminate a pilot's ability to correctively perceive depth or see obstacles and the terrain. In other words, the experience of glare or flash blindness in the absence of an afterimage can be sufficient to cause disorientation or impair performance though negative effects on attention.

Other research supports the finding that human subjects experiencing dazzling effects from lights report discomfort and visual impairment that contribute to impaired concentration on performance tasks (e.g., increase reaction time), suggesting that dazzling lights can impair brain functioning (Santos et al., 2019). Researchers note that the psychological effects of light exposure (e.g., laser) can significantly affect performance; in fact, simply the threat of light exposure can alter target-engagement strategies and reduce shooting accuracy, even when no exposure is actually delivered (Mastroianni, Zwick, & Stuck, 1989; Mastroianni, King, Zwick, & Stuck, 1989; Kosnik, 1994). In experimental work, it's important for participants to be naïve to a light stimulus (i.e., participants are unaware they will be exposed to light) so they are not inoculated against any adverse psychological effects that could be of interest. But in general, the psychological effects of light exposure in an operational context remains undetermined (Kosnik, 1994).

In terms of the stress response, the hypothalamus activates the sympathetic nervous system in response to a stressful stimulus, causing the pupils to dilate. Alternatively, when the body returns to homeostasis, the hypothalamus activates the parasympathetic nervous system,[8] leading to pupil constriction. Recall that the hypothalamus is also connected to the amygdala, which is involved in the stress response. Thus, pupil dilation and constriction are closely tied to the stress response. Although the pupils dilate in response to cognitive

[8] The parasympathetic nervous system is also activated when bright light hits the retina, leading to pupillary constriction.

or emotional events, we note that the pupillary response to light (i.e., constriction) is significantly larger (Fong, 2012). This means that although the FBG deployment context may heighten the overall levels of stress experiences and thus lead to pupil dilation, the pupillary response in anticipation of, or in response to, the flash may lead to pupillary constriction. As noted above, this constriction will reduce the amount of light entering the retina and bleaching photoreceptors, thereby reducing the duration and intensity of the flash afterimage. The degree to which this anticipatory pupillary light response protects individuals from flash exposure, particularly in stressful situations, is unknown. Likewise, little research exists connecting acute vision effects and behavior change via the stress response. Chronic stress and its effects on vision have been extensively studied and documented. For example, continuous stress and elevated cortisol levels negatively affect the eye and brain due to autonomous nervous system imbalance and vascular dysregulation and can lead to the development or progression of certain visual system disorders (Sabel, Wang, Cárdenas-Morales, Faiq, & Heim, 2018).[9]

Some acute vision research focuses on the effects of dazzling light and glare from car headlights at night on professional drivers (e.g., truck drivers, bus drivers). While professional drivers are not the same population of interest as those who might be subject to FBGs, these drivers are essentially engaged in a threat-avoidance task with an already heightened degree of stress making their psychological state somewhat similar to a target population for FBGs. Research shows that professional drivers consistently face visual signals, including cognitive-relevant and aversive signals like car headlights. When empirically tested via exposure to a glare stimulus that simulates a bright headlight, this population experiences telltale signs of the stress response, including increased blood pressure, hyper alertness, and facial clenching (Belkić, et al., 1994; Emdad, et al., 1998). These studies suggest that blinding light stimuli are indeed capable of inducing a stress response.

As mentioned previously, there is effectively no existing research, empirical or otherwise, that discusses the connection between the stress response and acute vision loss. Due to the highly variable effectiveness of an FBG's flash component, it's still worthwhile to identify how the stress response can affect vision for individuals exposed to an FBG but not affected by the flash (e.g., not looking toward the detonation or eyes closed), a likely scenario. The introduction of stressors has been shown to interfere with peripheral target acquisition due to the activation of the sympathetic nervous system; this is often called "tunnel vision" (defined as a loss of peripheral vision with relative preservation of central

[9] Some research suggests that those with PTSD have atypical visual processing that structurally manifests in reduced gray matter in the visual cortex, providing evidence for long-lasting macrostructural changes in regions specialized for visual processing (e.g., Chao, Lenoci, & Neylan, 2012; Mueller-Pfeiffer, et al., 2013). Although this report is not focused on chronic stress, it is possible that FBG targets of interest could include those with PTSD, including military personal who also experience FBG effects.

vision, resulting in a constructed circular field of view (e.g., Verhage, Noppe, Feys, & Ledegen, 2018; Williams, 1988). Tunnel vision narrows one's visual field and may cause a distorted or altered understanding of one's environment, thereby affecting performance (e.g., target accuracy). Tunnel vision is affected by situational factors like task demands and cognitive load (Williams, 1988). Similarly, the stress of exposure to FBGs could cause one's visual acuity to decrease, even if not exposed to the flash per se (e.g., the target is wearing goggles or isn't looking at the location of detonation).

C. Mitigating Factors to Flash Exposure

The effectiveness of the flash component is highly variable, depending on a number of environmental, situational, and individual factors. Each of these mitigating factors potentially ameliorate the negative effects of an FBG flash:

1. Environmental Factors

Ambient light significantly affects the effectiveness of the flash component. The flash is most effective in nighttime conditions or dimly light rooms, due both the extreme luminosity contrast between the flash and the background and the dark adaptation of the human eye to the dark background. From a neurophysiological standpoint, at low levels of light, the pupils dilate to maximize the perception of information, while at high light levels, the pupils constrict to reduce the level of light adaptation of the photoreceptors. This constriction reduces the amount of light that enters the eye. During daytime, the constricted pupils therefore also restrict the amount of light from the flash.

2. Situational Factors

Eye protection and target location are two situational factors that can contribute to the effectiveness of the flash component. If someone is wearing light-blocking eye protection, the intensity of the light will be decreased and not as effective in changing one's behavior. Similarly, if someone is simply looking in the opposite direction or not directly at the detonation point, the effect of the flash will be reduced.

3. Individual Factors

As with all FBG components, the effectiveness of the weapon system is subject to individual-level variation and behavioral differences (i.e., certain people are more affected by noise or by light than others, age differences affect recovery). In the case of afterimages (and flash blindness), individual differences exist in both the perception and duration of these visual effects (Atkinson & Crawford, 1992). Behaviorally, research shows that saccades (rapid eye movement between two points) can reduce the duration of weak but not strong afterimages, such that increased frequency of the saccades has a greater effect on weak afterimage duration. Note that blinking or pursuit movements do not decrease the duration of low- or high-intensity afterimages; however, they do lead to a strong afterimage duration in lighted conditions. It is believed that saccades reduce weak afterimage duration because they cue the visual system that the afterimage is not a real object (Powell, Sumner, & Bompass, 2015). Blinking, on the other hand, causes a change in luminance between blinks, which counteracts the perceptual fading mechanism of the cortex, thus extending the duration of strong afterimages (Brindley, 1962).

D. Research Opportunities

There are significant gaps when it comes to the visual component of FBGs. The flash component has been reported to both be a significant factor (Brence, et al., 2002; Paulissen & Huisjes, 2001) and an insignificant one, making its role in characterizing weapon effectiveness unclear. However, since the flash intensity sits at almost a magnitude lower than the level causing vision damage, and because conscious and subconscious (e.g., pupillary light reflex) mitigating actions are simple and effective, *it may be that the flash component is the least stress-inducing component of the FBG and has a different function in weapon design* (*e.g., overstimulation, multiplicative effects*). IDA nevertheless offers the following recommendations for fruitful research avenues to understand the role of stress and FBG effectiveness:

1. Timing of Flash-Blindness Effects

If flash blindness is a short-lived deficit, the effects of the flash on the stress response might also be short-lived. In other words, it might be the case that the flash plays a role independent of the stress response, perhaps through stimulus overload.

2. Psychological Effects of Temporary or Partial Blindness

One open question regarding the flash component is its connection to the stress response. While the physiological effect of flash blindness might be short-lived, the lasting psychological effects of being temporarily blind or partly blind might be extremely stressful and therefore have a significant effect on behavior and task performance. Although the blindness is temporary, it is difficult for an individual to estimate or predict how long the blindness will last. Therefore, the stress induced from the flash component might stem from being unable to make decisions, appraisals, or engage in the best action in the face of some other danger. In this sense, it is possible that the uncertainty regarding when vision will return to a level where perception can occur contributes to or exacerbates the stress response. The impact of light and blindness on the individual could be explored by looking into the impact on target acquisition by varying length, brightness, or color of the flash, and color of the uniform of red team targets. The impact of secondary flashes is could also be studied to explore increasing effectiveness of FBGs, especially if the blue team knows the precise interval of the flash.

3. Physiological Effects

The pupillary light reflex might serve to protect from flash blindness, thus reducing the flash effectiveness, but the research is inconclusive. In addition, stress priming might moderate the protective effects of the pupillary light reflex.

5. Auditory Effects and the Stress Response

Acute noise is a common workplace stressor, especially in the military, where Service members are exposed to acute and chronic noise on a regular basis. FBGs expose military personnel and operational targets to sound at decibel levels typically between 170 dB and 180 dB, triggering a psychophysiological response. The physiological response includes aural pain, tinnitus, and temporary threshold shift (TTS) that generate a stress response and affective shift (see Figure 1). The stress response generated from acute noise has significant psychological and behavioral effects on the affected person. This section discusses the basics of audition, exposure to acute noise, and the stress response as it relates to the acoustic component of FBGs, with attention paid to the behavioral outcomes caused by FBGs—stopping approaching combatants, clearing an area of people, and moving people from one area to another. In this section, we discuss current research on acute noise stress as it is relevant to FBGs. First, we identify the physiological mechanisms related to auditory function and the stress response. Then, we discuss the stress response and tinnitus and TTS. Next, we present relevant behavioral and cognitive outcomes connected to acute noise stress. Finally, we describe specific actionable gaps in knowledge that are prime areas of further research for the sponsor.

A. Physiological Effects of Acute Sound Exposure

Hearing is a complex process where sound waves in the air are changed into electro-chemical signals and carried along the auditory nerve to the cortex where the signal is decoded. The process begins when sound waves enter the outer ear and travel down the ear canal to the eardrum (see Figure 7). The vibrations of the eardrum are transmitted to the three bones of the middle ear, which amplify the sound vibrations, before sending them to the cochlea in the inner ear. The vibrations cause the fluid in the cochlea to form a traveling wave along the basilar membrane, where hair cells located at the wide end of the cochlea detect high-pitched sounds or those close to the center of the cochlea detect lower pitched sounds. As the hair cells move, their microscopic projections bend, causing the electrophysiological cascade (depolarization) that creates the electrical signal carried by the auditory nerve to the cortex, where the signal is deciphered (National Institute on Deafness and Other Communication, 2015).

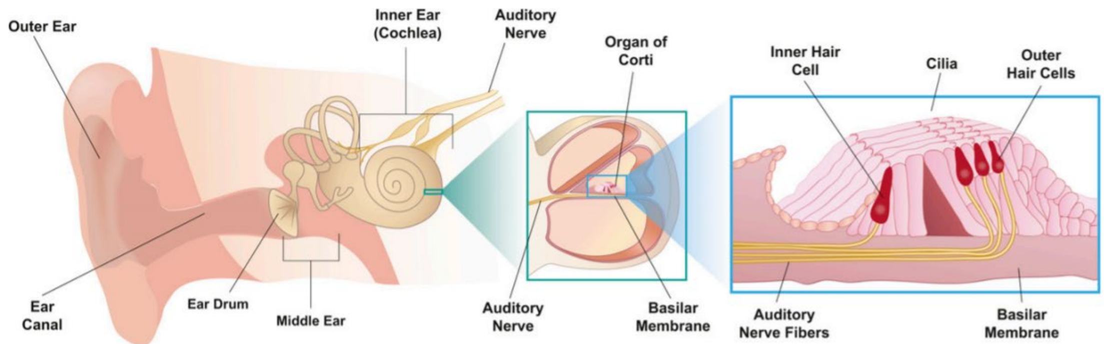

Figure 7. Parts of the Ear. Adapted from Tepe et al. (2017). The figure depicts the anatomy of the ear (left), cross section of the cochlea (middle), and the organ of Corti magnified (right) (Tepe, Smalt, Nelson, Quatieri, & Pitts, 2017)

Although hearing loss due to excessive exposure to noise has been recognized in humans for centuries, research on animal models of noise-induced hearing loss (NIHL) took off in the 20th century. NIHL can result from long-term, continuous-noise exposure or from a single or repeated sudden-noise exposure (i.e., acoustic trauma) (Le, Straatman, Lea, & Westerberg, 2017). Research suggests that exposure to sudden-impulse noise[10] leads to worse outcomes than exposure to steady-state noise (Suvorov, et al., 2001). The magnitude of and recovery from NIHL is dependent on the sound level, type (e.g., impact sound or continuous noise), duration, and frequency of exposure, as well as individual differences (e.g., age, gender, prior history of noise exposure, smoking, diet) (Ryan, Kujawa, Hammill, Le Prell, & Kil, 2016). Not all excessive noise exposure results in hearing loss; it can also result in tinnitus, hyperacusis, or more commonly, shifts in hearing thresholds.[11] Impulse noise from assault rifles and airbag deployment has been strongly associated with the development of TTS and tinnitus. These shifts lead to an increase in the hearing threshold and can be temporary (TTS) or permanent (PTS). Because FBG exposure is associated with TTS, we will focus on TTS in this report, but note that repeated TTS can result in PTS, which would be considered a significant injury from a less-lethal weapons perspective. We also note that both the duration of the sound exposure and the psychophysiological, cognitive, and behavioral effects can be considered acute or chronic. In this section, we are focused on acute sound exposure (e.g., single or multiple blasts) and the acute (or temporary) effects of the exposure on hearing, cognition, and behavior.

[10] Note that there are different types of noise (steady, intermittent, fluctuating, irregular, or impulse type), which can be defined by many parameters. The level of the sound along these parameters is what determines the likelihood of developing NIHL, but due to individual differences, the level of the parameters that cause NIHL can vary considerably from person to person. See Pawlaczyk-Łuszczyńska et al. (2004) and Flamme, Liebe, & Wong (2009) for descriptions of these parameters.

[11] Tinnitus is the acoustic perception of a specific frequency sound in the absence of an external sound source; hyperacusis is the oversensitivity to sound (Heeringa & van Dijk, 2014).

In TTS, decreased sound sensitivity occurs suddenly and immediately after acute sound exposure and returns to normal levels within minutes to weeks after sound exposure. Although individual variation exists, models predict that TTS sets in when individuals are exposed to noise exceeding 60–80 dB, and the degree of TTS is dependent on the length of time of noise exposure (Miller, 1974). Early research by Davis et al. (1946) examined the development of TTS in subjects exposed to intervals of intense tones (at specific frequencies, decibel levels, and exposure times) across several days. The research team examined impairments in speech comprehension and auditory sensitivity and also measured time to recovery from TTS. The team found that although TTS had been induced in many of the study trials, there did not seem to be evidence of cumulative injuries across trials. The greatest hearing loss occurred at frequencies one-half an octave higher than the exposure tone, although the frequency of the tone is an important moderator to the development of TTS. The team discovered that, in general, both TTS and recovery from TTS occur quickly during the first minute of exposure and then more slowly thereafter. That said, individual differences rendered some subjects more susceptible to TTS. For example, there were differences in the sound frequency levels that produced TTS as well as in the rate of recovery for a given level of TTS.

In terms of operational relevance, Price, Kalb and Garinther (1989) conducted a study for the Army and found that although the distance over which voices can be normally heard is relatively large, TTS reduces that distance to one-sixth that of normal hearing. When examining the detection of non-speech sounds such as those from enemy personnel, they found that TTS led to a twentyfold decrease in the detection distance of a rifle bolt closing and the sound of a magazine being inserted into a rifle compared with normal hearing. Likewise, when they examined the ability to detect the sound of rustling leaves (i.e., enemy personnel walking nearby), those with normal hearing were able to detect the sound early enough to allow for a 2-minute warning of approach, but those with TTS could not hear the approach at all.

Animal models indicate that TTS does not result in hair cell damage; however, the exact molecular and neurobiological causes of TTS are still being investigated. What is known is that excessive noise causes mechanical damage to the cochlea and basilar membrane. There are also inflammation-supporting processes (related to the stress response), as well as processes that support an increase in programmed cell death (pro-apoptotic) that occur with TTS. These molecular and biochemical changes affect normal auditory processes, including the potential driving hair cell depolarization, cellular mechanisms supporting hair cell activity, and innervation of inner hair cells that send impulses to the brain (Ryan, Kujawa, Hammill, Le Prell, & Kil, 2016; Yan, et al., 2013). The loss of synaptic connections between inner hair cells and afferent neurons after acute noise-induced cochlear trauma is a result of glutamate excitotoxicity that damages the post-synaptic terminals (Kujawa & Liberman, 2009). It appears that nerve fibers with low spontaneous firing rates are most susceptible to such damage (Furman, Kujawa, & Liberman, 2013). Although these fibers do not contribute to neural responses (thus, standard pure-tone audiograms do not detect hearing loss caused by this damage), these fibers are critical for proper hearing of speech sounds and result in what is termed “hidden hearing loss” (Lobarinas, Salvi, & Ding, 2013; Schaette & McAlpine, 2011). In addition to the clinically recognized deficits experienced by Service members who routinely suffer noise exposure, they also suffer from changes in hearing threshold due to this synaptic damage; however, it is often undiagnosed due to the current lack of testing for such hidden hearing loss (Bressler, Goldberg, & Shinn-Cunningham, 2017). Note that a single traumatic sound exposure can lead to different types of damage within the cochlea, resulting in differing outcomes (e.g., PTS in the midfrequency ranges while TTS in the lower frequency ranges).

In addition to TTS, tinnitus is frequently an auditory outcome of FBG exposure. Tinnitus, like TTS, is a symptom of an underlying condition and in the FBG context, can be caused by exposure to loud noises and blast-wave injury. (Note: there are a number of other causes of tinnitus, as well). Acute tinnitus onset begins immediately following noise exposure and might be perceived on the same or opposite side of the exposed ear. The pitch of acute tinnitus is usually in the higher frequency range, most often above the frequency of the acoustic stimulus (Atherley, Hempstock, & Noble, 1968; Loeb & Smith, 1967). Tinnitus is considered to have three components: auditory, attentional, and emotional. In the case of acute tinnitus, the auditory component may be the only symptom of the deficit. However, to be a clinical concern, the auditory perception of the sound has to attract a great deal of attention from the sufferer such that the hyperattentiveness to the sound leads to negative emotional reactions (Kaltenbach & Manz, 2012; Jastreboff & Hazell, 2004). Although the neurobiology of tinnitus is still under investigation and not well understood, animal studies indicate changes in auditory-related neural activity within a few hours after intense noise exposure (Eggermont, 2015; Heffner & Koay, 2005). It appears that noise-induced tinnitus is a result of damage to, or overstimulation of, the ear, which triggers an increase in excitation and a decrease of inhibition (through the loss of inhibitory synapse) in neurons in the central auditory system. This excitation makes the neurons behave as though they were responding to sound, even when there is no physical sound to be heard (Kaltenbach & Manz, 2012). It is possible that the auditory damage associated with TTS leads to acute tinnitus as well. For example, Schaette, Turtle, and Munro (2012) were able to induce reversible tinnitus by having volunteers insert an earplug (simulating mild frequency hearing loss) in one ear for 7 days. The tinnitus frequency in these subjects matched the frequency of the hearing loss. It is believed that the sudden decrease in cochlear output to the auditory nerve might trigger a compensatory mechanism that triggers an increase in spontaneous neural activity that mimics the frequency at or near the frequency of the hearing loss; it is proposed that this compensatory mechanism is the cause of the tinnitus (Hertanzo, Lipford, & Depireux, 2020).

There are a few challenges with, and limitations of, the human effectiveness assessments regarding the bang component of FBGs. A challenge to the human effects of acoustic-energy exposure is the fact that there could be an asymmetrical effect on hearing. For example, in the head shadow effect, if the sound is to one side of the person's head the ear on that side may receive a louder sound impulse because the sound travels directly to the ear while the ear on the other side is shielded by the head (i.e., sound waves have to travel around the head to enter the opposite ear), leading to asymmetrical hearing effects (McFadden, 1993). In addition, a significant asymmetry will occur if one ear is more proximal to the source of the sound than the other ear, which is often seen in military personnel with weapon noise exposure (Nageris, Raveh, Zilberberg, & Joseph, 2007). There is also some evidence to suggest that individual differences in ear anatomy and physiology leads to the left ear being "weaker" and thus more susceptible to NIHL, which can lead to asymmetric effects, depending on the location of the sound source. For example, (Axelsson & Ringdahl, 1989) have found that the effects of tinnitus are magnified in the left ear compared with the right, suggesting that the left ear is indeed the weaker one.[12]

Another potential complication to understanding the human effects of FBG exposure is factoring in the timing effects of the flash and the bang because activation of defensive reflexes can mitigate the human effects of the flash or the bang. For example, Firth (1981) demonstrated that exposure to a sudden, loud sound (95 dB) increased pupil size and inhibited the pupillary light reflex. Therefore, if the bang of an FBG is experienced before the flash is seen, it is possible that the subsequent pupil dilation and inhibition of pupillary light reflex allow more light to enter the retina, leading to a greater magnitude of photoreceptor bleaching and therefore an increase in the intensity and duration of the flash afterimage. Subsequent exposure to a second auditory bang resulted in no change to initial pupil size (indicative of habituation) but with continued inhibition of the pupillary light reflex. Firth (1981) concluded that the pupillary light reflex indicated arousal level determined by the sensory stimulation associated with the loud noise, but pupil size indicated actual attentional arousal of the individual.

Crucially for FBG effectiveness, the contributions of extrinsic (*e.g., characteristics of the sound*) *or intrinsic factors* (*e.g., cortisol levels, hormones*) *to the development of tinnitus or TTS* (*or PTS*) *are still unknown, making it difficult to predict who will suffer from which auditory deficit.* Specifically, the reliable prediction of noise-induced TTS and/or tinnitus will depend on an equation that considers the intensity, spectrum, duration,

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[12] The IDA team offers another hypothesis regarding ear asymmetry—the differences may be due to cortical dominance, similar to that of handedness (or dominance/preferred foot). Although there does not seem to be a relationship between handedness and ear dominance/preference (Nageris, Raveh, Zilberberg, & Joseph, 2007), the role of laterality on the asymmetric effects of noise exposure is unknown.

and other characteristics of the auditory stimulus and the moderating effects of gender and other intrinsic factors (Hertanzo, Lipford, & Depireux, 2020).

One major limitation to the research on TTS and tinnitus is that the focus tends to be on workplace safety and thus chronic sound exposure. *Little human-effects research exists regarding the effects of acute or impulse noise* (*across its various parameters*) *and task performance specific to operational contexts. Likewise, little work considers what the consequences of impaired task performance due to acute noise does to human effectiveness of FBGs in operational contexts.* In terms of animal models for auditory deficits, noise exposure tends to be well defined and controlled (experimentally); however, the experimental conditions vary considerably and may not adequately characterize the appropriate context or human behavior in relevant settings. For example, animal studies utilize a wide range of sound levels, peak intensities, frequencies, and frequency ranges; they may or may not use anesthesia; and they may involve a single ear or both ears (Hertanzo, Lipford, & Depireux, 2020). In general, however, the studies focus on examining the auditory deficits themselves and not the consequences of those deficits. Given the findings regarding asymmetrical hearing effects, it is safe to say that both animal and human studies need to keep in mind the exposure and subsequent hearing effects to both ears and not assume that the impact and outcome both ears will be equivalent for both ears.

B. Effects of Sound Exposure on Stress, Cognition, and Behavior

As is discussed in other areas of this report, the stress response is primarily physiological with psychological and behavioral outcomes. Specific to the auditory system, exposure to an acute stressor like a loud bang activates neural functioning at many levels of the central nervous system, thereby affecting auditory performance (Banis & Lorist, 2012; Joëls & Baram, 2009; Mazurek, Haupt, Olze, & Szczepek, 2012). The auditory system is modulated by different intrinsic and extrinsic physiological mechanisms, including the cardiovascular system, drugs, neurotransmitters, and extra-auditory structures (i.e., other structures of the central nervous system with direct or indirect inputs to the auditory system). Extra-auditory structures of interest to FBG human effectiveness include the limbic system, which regulates instinctive behavior and emotions; the limbic system is thought to attach emotional significance (e.g., affective shifts) to acoustic stimuli (Al-Mana, Ceranic, Djahanbakhch, & Luxon, 2008; LeDoux, Sakaguchi, & Reis, 1983). (Also see Madhavan and Srinivasan, 2018, for more discussion about the specific motor and neural pathways related to the acoustic startle reflex.) The limbic system also has hormone receptors for stress-related hormones (Gray & Bingman, 1996). Another extra-auditory system involved with acoustic function and behavior is the reticular system, a system concerned with the behavioral state or arousal and alertness that is involved in the stress response (Jennes & Langub, 2000). The ascending reticular system reacts more strongly to “important” than to “unimportant” stimuli; these reactions are related to hearing in noise and selective attention.

A number of different hormones affect auditory function (e.g., reproductive steroids, melatonin), but for current purposes, the hormones of interest are stress-response hormones. Cortisol is the main hormone secreted in response to stress, and cortisol receptors (called glucocorticoid receptors) have been found in the inner ear of animals and humans. In the cochlea, cortisol receptors are present in sensory and non-sensory tissues, suggesting that this stress-response hormone plays a role in homeostasis of inner ear fluids and signal transduction. Adrenaline and endorphin hormones are also activated during stress and regulate auditory function; since these hormones regulate auditory function and changes in stress, they also contribute to changes in auditory function. These stress-response hormones are secreted differently, depending on the intensity of the noise exposure. Empirical studies show that acute noise exposure near the threshold of pain (i.e., extreme, intense noise) can cause an increased release of cortisol, but acute noise exposure between 90 and 100 dB can cause an increase of catecholamines. Further, non-habituated noise primarily affects the release of adrenaline (Ising & Braun, 2000; Ising, et al., 1990). These stress hormones, especially cortisol, which is often measured via saliva, are frequently measured in empirical studies (Mazurek, Haupt, Olze, & Szczepek, 2012), providing one approach to measuring the stress response in those exposed to acute noise stress. Note, however, that research has found that task demands and prior noise exposure levels can affect whether hormone levels increase or decrease, making them a potentially difficult stress marker to measure (Frankenhaeuser & Lundberg, 1977). Also note that depending on the sound exposure level, the specific hormone to measure is of concern.

Noise exposure has a range of negative effects, ranging from interference of cognitive processes to detriments to mental and physical health (Stansfeld & Matheson, 2003). For example, noise can stimulate the sympathetic nervous system or the pituitary-adrenal-cortical system, which in turn activates a variety of processes related to the stress response (Lusk, Gillespie, Hagerty, & Ziemba, 2010). One of the easiest ways to measure the acute effects of noise on the stress response is through the continuous monitoring of noise, blood pressure (BP), and HR simultaneously. Using these measures, Lusardi et al. (1996) showed that acute exposure to loud music significantly increased systolic and diastolic BP as well as HR. This finding persisted for the first hour of exposure (with BP and HR measured every 15 min) but returned to normal thereafter.

A great deal of literature focuses on the effects of noise on attention, information processing, strategic responding, reaction time, intelligence and concentration, cardiovascular health, sleep, depression, and neurodegenerative disorders; however, much of the findings are the result of chronic noise exposure and/or noise pollution (Jafari, Khosrowabadi, Khodakarim, & Mohammadian, 2019). For example, studies show that moderate to high noise-level exposure increases fast erroneous responses on reaction-time tests; these error rates increase with increased time on the task and are likely due to impaired control (Rabbitt, 1979). On the other hand, noise only interrupts vigilance task performance when a subject is required to monitor several signal sources. In other words, noise seems to increase attentional selectivity but have less of an impact on vigilance performance on complicated tasks (Hockey, 1970). In terms of the military, Service members face a higher risk of hearing loss due to combat deployment (where they are exposed to weapon fire), proximity to blasts, and combat-related head trauma. As will be discussed in the next section, blast overpressure has wide-ranging effects on the human body, but the auditory system, in particular, is extremely vulnerable to blast damage. Given the various modes of auditory insults possible in military operations, hearing impairments and/or dysfunction in auditory perception can reduce situational awareness by impeding sound-detection thresholds, sound-localization thresholds, and speech intelligibility (Tepe, Smalt, Nelson, Quatieri, & Pitts, 2017).

In the context of an FBG, a sudden increase in noise is especially stressful and can disrupt ongoing activity (Kjellberg, 1990; Banis & Lorist, 2012); if the activity is auditory in nature (e.g., communication), performance will be especially deteriorated. Brier et al. (1997) exposed participants to loud, pure discontinuous noise under both controllable and uncontrollable conditions. Under uncontrollable conditions, they found enhanced stress responses, anxiety, and tension (via self-report and hormone levels) relative to that found under the controllable conditions. Banis and Lorist (2012) also exposed participants to continuous (85 dB(A), 0–10 kHz) or discontinuous (75–95 dB(A), 0–10 kHz) white noise (2–7 s in length) with random inter-pulse-intervals (also 2–7 s in length) during a gambling task and found that acute noise stress did affect feedback processing (i.e., decision-making or higher order cognitive control function) in the noise condition; however, unpredictability of the noise stressor did not seem to affect behavior, but participants could have habituated to the noise condition. Habituation to noise occurs easily, provided that the noise is found to be of no importance for the individual, though noise at least 90 dB or above elicits the defensive reflex and this habituates very slowly. *The relationship between the duration of noise exposure* (*i.e., the length of time someone is exposed to acute stress*) *and the inter-impulse interval* (*i.e., the length of time between noise exposure*) *is largely an open question for FBGs, especially for multi-bang flashbang scenarios, where exposure, dose-response curves, and time to baseline are significant gaps in characterizing the effects of acute noise. In other words, cognitive and behavioral flexibility with aperiodic, unpredictable noise stress is a significant gap area with regard to characterizing FBG effectiveness.*

The cognitive and behavioral effects of sudden noise exposure are inextricably tied to the stress response—acute noise, the stress response, and psychological and behavioral performance depend on several situational factors, including the individual's task, operational (or experimental) setting, and the individual factors, such as age, gender, genetic make-up (Mazurek, Haupt, Olze, & Szczepek, 2012). In fact, individual differences are a significant component that influences one's response to noise—the same noise can elicit wildly different responses from different people (Mazurek, Haupt, Olze, & Szczepek, 2012; Kjellberg, 1990). Anecdotal evidence suggests that one's background (e.g., prior FBG exposure, PTSD) could dramatically affect a person's reaction to an FBG. Research suggests important two individual differences predict behavior in response to acute noise stress: coping strategy (style, efficacy, and behavior) (Delahaij & Gaillard, 2008) and skill level (Sheffield, Brungart, & Blank, 2016).

A sudden acoustic stressor (e.g., FBG) can change a person's behavior by lowering performance or affecting behavior toward others via overall psychological and cognitive stress (Kjellberg, 1990). Lupien et al. (2007) note that the two most important psychological determinants of the stressfulness of a situation are an individual's lack of control and unpredictability. In addition, prior exposure to noise also makes subsequent noise exposure stressful. For example, Frankenhaeuser & Lundberg (1977) exposed participants to two noise-exposure sessions on successive days and found that the participants' initial noise exposure (56 dB, 72 dB, or 85 dB) influenced their subsequent task performance the next day, even if they were exposed to lower noise or no noise. That is, initial noise exposure has carryover effects, louder noise exposures leading to greater performance decrements. In this regard, FBGs are particularly stressful due to their nature of being sudden, unpredictable, difficult to habituate to (in a multi-bang context), and not under the control of the targeted individuals.

The stress response can also narrow auditory attention and affect cognitive flexibility, ultimately degrading working memory (Szalma & Hancock, 2011). These researchers contend that any communication or speech held in the articulatory loop component of memory (such as GPS coordinates or directions one would rehearse) is susceptible to a noise-specific working memory decrement (Szalma & Hancock, 2011; Baddeley & Hitch, 1974), which is particularly relevant for FBGs. Generally speaking, noise makes cognitive tasks more difficult, indicating some sort of psychological stress, while also narrowing attention. Further, task strategy is also affected, with noise increasing speed but reducing accuracy, thus leading to more errors, an effect sometimes attributed to an increase in arousal level (Hillier, Alexander, & Beversdorf, 2006). That is, motor tasks might increase in speed, but communication tasks might break down altogether.

Research on the effects of auditory stressors typically focuses on chronic sound exposure (e.g., work environments with constant noise, like construction sites). There is some research on acute auditory stressors, which focuses on emergency responders (e.g., firefighters, paramedics) and their reactions to unpredictable and sudden noise exposure, which could be applicable to FBG detonations. Not only do emergency responders work under stressful conditions, but they are often exposed to acute noise in the form of alarms. These occupational stressors are accompanied by a behavioral stressor, in that responders need to mobilize after being exposed to the acute alarm. This type of environment resembles that where FBGs are used, in that those being exposed to the device are in a stressful situation, and there is an intended behavioral stressor (stopping, moving, clearing).

In addition, research has noted that sudden exposure to an acute sound like an emergency alarm or FBG evokes a physiological stress response with behavioral consequences (Hall, et al., 2016). These investigators researched the stress response (via heart-rate monitoring and salivary cortisol measure) and behavioral reactions resulting from acute emergency alarm (1558 Hz and 105 dB) exposure used to immediately mobilize participants (e.g., put on shoes and protective gear) during the day or night. While the researchers were focused on the stress response and performance between day and night conditions (something not unrelated to FBG use), the crucial outcome relevant to acute noise and the stress response was the detection of significant cortisol levels at night. The low predictability of the alarm exposed participants to a stressful situation. Cortisol levels are affected by circumstances that involve low predictability; thus, a detectable increase was found.

1. Temporary Threshold Shift and Stress

As with the literature on human effects, research on tinnitus and TTS is predominantly focused on the stress response to chronic auditory conditions, and there is a long, and rich history of this research in military populations. Chronic tinnitus and PTS[13] have been consistently observed to cause significant psychological stress, leading to changes in working memory, cognitive load, and situational awareness (e.g., Rossiter, Stevens, & Walker, 2006). On the other hand, there is a dearth of research regarding *acute* hearing acuity effects (i.e., *acute* tinnitus and TTS), operational performance, and the role of the stress response in an FBG context. Some research suggests that higher levels of hearing loss (TTS), similar to that experienced from FBG exposure, likely encourage people in combat positions to switch combat effectiveness strategies to a more passive and defensive position (i.e., TTS effects disorient, confuse, and control behavior, Sheffield et al. 2016). Likewise, speech intelligibility decreases, which can force soldiers to dramatically alter behavior to adjust for auditory deficits (Keller, et al., 2017). Along these lines, Sheffield et al. (2016) showed in a realistic combat training experiment that higher performing, more experienced teams with simulated significant acute simulated hearing loss (i.e., TTS) performed *worse,* as measured by simulated kill ratios and hitting waypoints, than lower performing, less experienced teams with significant acute hearing loss. In other words, experienced soldiers more effectively use information perceived via hearing to accomplish a number of combat tasks; when hearing is impaired, it is associated with significant losses

[13] Hearing loss that recovers to baseline levels in hours, days, or weeks following exposure is considered TTS, while any permanent hearing loss is considered PTS (Ryan et al. 2016). TTS recovery is highly individual and depends on physiological factors as well as prior and subsequent noise exposure.

in combat effectiveness. At the individual level, even one affected person with hearing loss can disrupt communication across a team and significantly decrease situational awareness, leading to behavioral changes like ceasing movement, stopping communication, or changing task strategy (Sheffield, Ziriax, Keller, Barns, & Brungart, 2017).

While Sheffield et al. (2016; 2017) did not look specifically at the stress response, one could argue that significantly impeding hearing acuity, and thus removing an effective communication tool for experienced soldiers or combatants, triggers a stress response in team members that results in worse performance and significantly altered behavior. In general, the stress response from acute hearing loss results in changes to one's behavior in an attempt to adapt (Keller, et al., 2017; Wickens & Hollands, 2000). That said, research shows that there is a negative relationship between the stress response and TTS, meaning the more stressed someone is, the less TTS they will experience. This negative relationship is related to blood flow to the cochlear increasing during times of stress, thus decreasing cochlear fatigue and decreasing TTS (Thompson, Dengerink, & George, 1987; Melnick, 1978; Kryter & Poza, 1980; Bohne, 1976; Lim, 1980). *Hearing decrement, the stress response, and performance in operational contexts should be tested empirically within a specific flashbang context* (*focusing on individuals and/or teams*) *to tease apart the specific contributing components to FBG effectiveness.*

2. Tinnitus and Stress

Research on acute tinnitus effects is scant and mostly focused on behavioral and physiological animal models using rats and hamsters (Kaltenbach, Tinnitus: Models and mechanisms, 2011). The bulk of evidence regarding tinnitus effects show that chronic tinnitus induces a stress response; however, it is unclear if acute tinnitus effects induce a stress response in a similar way. Scientists agree that tinnitus can be triggered by injury to the inner ear, causing decreased activity of the auditory system (Kaltenbach, 2011); however, the stress response itself can also induce tinnitus (Mazurek, Haupt, Olze, & Szczepek, 2012), thereby making the FBG context particularly complex. Tinnitus is usually accompanied by some psycho-social distress *before* the onset and progression of tinnitus; exposure to high levels of stress *and* a significant noise further contributes to the probability of developing tinnitus (Mazurek, Haupt, Olze, & Szczepek, 2012). For chronic tinnitus, research has noted that salivary cortisol is chronically elevated in those with tinnitus; it's unclear if cortisol is also elevated in those with acute tinnitus effects.

C. Mitigating Factors to Bang Exposure

The effectiveness of the bang component is not as variable as the flash component, making mitigating factors less effective. That said, the effects of the bang can still be mitigated by environmental, situational, and individual factors.

1. Environmental

The environment can have an impact on the effectiveness of the bang component. A flashbang's sound waves will travel differently to a potential target if the flashbang is deployed in an outdoor field (i.e., no wave obstruction), outdoor urban environment (i.e., wave obstruction from buildings, cars etc.), or indoor environment (i.e., wave obstruction from walls, waves bouncing off walls, hallway configuration).

2. Situational

Ear protection is one situational factor that can contribute to the effectiveness of the bang component. If someone is wearing ear protection that dampens the intensity of the bang, the effectiveness of the bang component could potentially decrease. In addition, individuals may manually shield their ears from noise, reducing the effects of acoustic energy. It is not clear, however, if wearing ear protection or manually shielding one's ears would eliminate or decrease the stress response. Momentary losses of hearing may only impair performance in activities for which an individual is used to relying on hearing; not being able to hear in a situation when hearing is not critical may generate little stress (other than, perhaps, discomfort from tinnitus or TTS). One operational context to consider is close-quarter battle, where exposure to acute sound is such that special earplugs are now common in elite units and law enforcement. In close-quarter battle, one does not rely on hearing (i.e., communication is non-verbal), making earplugs quite effective. In the case of a crowd-control situation, this doesn't apply.

3. Individual

As noted previously, with all FBG components the effectiveness of the weapon system is subject to individual-level variation and behavioral differences, such as sensitivity to extreme noises or extreme noise differences, PTSD, and prior extreme noise exposure. For example, research has shown that individuals with identical noise exposure will have vastly different levels of TTS (Melnick, 1978; Thompson, Dengerink, & George, 1987).

D. Research Opportunities

To summarize the relevant auditory research, acute noise stressors like that of an FBG induce a variety of physiological, psychological, and behavioral effects on the person exposed. FBG-specific behaviors of interest include stopping approaching combatants, clearing an area of people, and moving people from one area to another, in addition to disrupting communication. Acute auditory exposure that is sudden and unpredictable can drive these behaviors by inducing a stress response that affects the auditory system (physiologically) and subsequent performance (e.g., slower to mobilize, restricted situational awareness, increase tension, and decreased cognitive flexibility). Overall, noise increases levels of general alertness and attentional selectivity, while also influencing strategic effects like becoming more passive, stopping behavior, or reducing performance accuracy. IDA offers the following recommendations for fruitful research venues to understand the role of stress and FBG effectiveness.

1. Stress and Priming

Anticipatory effects of sound can affect one's behavior and stress response. For example, individuals who have previously been exposed to a loud bang might modify their behavior in anticipation of a sound in s similar situations. How this relates to individual behavior when exposed to FBGs is currently unanswered. It is also important to understand the effects of impulse or sudden noise when there is considerable background noise. In other words, how does being primed by significant background noise affect the cognitive, behavioral, and stress responses to a sudden burst of sound?

2. Performance Outcome Variation

Outcome variables for FBG include motor movements (e.g., target accuracy), but also cognitive outcomes (e.g., target identification and acquisition) and communication outcomes. Varying the outcome to understand more acutely which behaviors are most affected by noise exposure and the stress response is worthy of investigation and can provide a wider understanding of FBG effectiveness. Auditory functioning tasks van be measured via cortisol, heart rate, speech intelligibility, cognitive flexibility (e.g., remembering GPS coordinates), and spatial awareness (e.g., moving or escaping).

3. Combined Effects

While previous research has investigated FBG components individually, combining the bang component with vision or overpressure effects will shed light on the specific role of the bang in generating the stress response and its effect on subsequent behavioral outcomes.

4. Miscellaneous Efforts

There are additional areas that don't necessarily fall into the above categories:

- Effects of TTS and/or tinnitus on the stress response and on task attention.
- Contributions of extrinsic and/or intrinsic factors to the development of tinnitus or TTS (or PTS) are still unknown, making it difficult to predict who will suffer from which auditory deficit.

6. Overpressure and the Stress Response

Blast overpressure (BOP) is a form of a shock wave at pressure levels above normal atmospheric pressure resulting from a sonic boom, an explosion, or the firing of a weapon (Sherman, 2014). The overpressure component of an FBG leads to thoracic pressure and vestibular effects in humans and animals, as well as a number of cognitive and behavioral outcomes as a result of blast exposure. With regard to overpressure and the stress response, literature suggests that dizziness can result from vestibular system damage, and this dizziness can activate the stress response. Cardiorespiratory dysfunction and triggering of the stress response can also lead to an affective shift that causes dizziness. This section begins with an overview of BOP wave physics and the physiological effects of BOP. Next, we describe the effects of BOP on stress, cognition, and behavior. Finally, we discuss some potential research opportunities related to human effectiveness of FBG-induced BOP. (Note: unlike the auditory and vision components, there are no factors that could mitigate BOP to dampen effects.)

A. Physiological Effects of Overpressure

An explosion creates a blast wave that progresses from the site of the explosion (or exploded device) as a sphere of compressed and rapidly expanding gasses, which replaces an equal volume of air at high velocity (blast waves travel faster than sound). Immediately following the propagation of the positive blast wave, negative pressure or suction of that blast wave, known as blast wind, is generated (Institute of Medicine, 2009; Owen-Smith, 1981; Rossle, 1950). In general, an explosion will cause an individual to experience both the blast wave itself and the subsequent blast wind. This wave and wind exposure can lead to damage to pressure-sensitive organs (e.g., ears, lungs). That said, the duration and human effects of the blast wave and wind depend on the type of explosive, the distance from detonation, and the number and types of items in the path of the wave. For example, in an open field, the energy of the blast waves decreases exponentially with distance from the origin of the blast, but because these blast waves still reflect off the ground, the reflected waves interact with primary wave, altering the characteristics of the original blast wave. On the other hand, blast waves inside building rebound (reflect) off walls and rigid objects, resulting in complex pressure waves that may enhance the effects of the original blast wave (Ben-Dor, Igra, & Elperin, 2001; DePalma, Burris, Champion, & Hodgson, 2005). In fact, Rice and Heck (2000) have reported that explosions near or within hard, solid surfaces are amplified two to nine times due to shock-wave reflection; individuals standing between a blast and a building experience two to three times the degree of injury as an individual exposed to a blast in an open space.

In terms of human effects (specifically injuries), explosive blasts have five acute effects on the body:

1. Primary blast injuries that are a consequence of the shock-wave body interaction.
2. Penetrative injuries (secondary blast mechanisms) from explosion debris.
3. Acceleration/deceleration injuries where the body part suddenly accelerates due to the pressure wave and then suddenly decelerates when it contacts a solid, stationary object (tertiary mechanism).
4. Flash burns from the explosion (quaternary mechanism).
5. Injury from post-explosion environmental contaminants (Institute of Medicine, 2014).

Although all five injuries can occur from an FBG detonation, the primary blast mechanism results in the traumatic loading of the chest wall by the blast, causing a shock wave that propagates into the lung, and the pressure difference across the alveolar-capillary interface causes disruption, hemorrhage, pulmonary contusion, and subcutaneous emphysema. Pulmonary injuries may be life-threatening if extensive (see Institute of Medicine (2014) for additional details regarding blast mechanics and injury).

When transmitted through the body, BOP increases pressure in the organs. This overpressure wave causes the lungs to suddenly hyperinflate, which in turn stimulates the vasovagal reflex. The vasovagal reflex initially leads to apnea but then quickly turns to rapid breathing, bradycardia, and hypotension (due to dilation of peripheral blood vessels). The heart-related effects of vasovagal reflex activation are due to an increase in parasympathetic nervous system activation of the heart (Zucker, 1986); in other words, BOP exposure can be considered an activator of the autonomic nervous system.

B. The Effects of Overpressure on Stress, Cognition, and Behavior

Some literature exists on short-term/immediate cognitive effects of blast events, but the findings are inconclusive. LaValle et al. (2019) investigated which blast components can be reliably measured during military operations and can be associated with negative consequences in a study of 202 students in an Urban Mobility Breachers Course.[14] Students

[14] Military “breachers” are Service members who use explosives to gain entry into buildings and compounds. Breachers are repeatedly exposed to blast overpressure during training and operations (Kamimori, et al., 2018). For example, some U.S. Marine Corps instructors are exposed to more than 240 blasts per year over a 2–3 year assignment (Kubli, Pinto, Burrows, Littlefield, & Brungart, 2017).

were exposed to a maximum of 5 psi and to a number of different blast events (as few as two events and as many as four; note that FBG events are around 5 psi). Neurocognitive performance was assessed using the Defense Automated Neurobehavioral Assessment (DANA) Rapid, a mobile application that includes three visual subtasks of cognitive performance: reaction time, procedural reaction time (i.e., decision-making), and go/no go trial. Results show neurocognitive impairment immediately after blast exposure (<5 minutes), where decision-making was impaired (part of procedural reaction time) and was the most sensitive to performance change. In addition, peak overpressure was associated with degradations in neurocognitive performance, high peak overpressure being associated with greater degradations.

On the other hand, Kamimori et al. (2018) conducted a longitudinal study of military breachers to identify any measurable changes in blood-serum-based neurotrauma biomarkers, neurocognitive performance, symptoms, or neuroimaging findings in a population repeatedly exposed to low-level blast pressure. The participants were exposed to repeated blasts due to operational training and field exposure (maximum blast exposure was less than 4 psi), and the time between blast exposure and data collection ranged from 4 days to 2.2 years. The researchers found no changes in neurotrauma biomarker concentrations (UCH-L1 and GFAP), neurocognitive performance (on the Automated Neurocognitive Assessment Metric), symptom reporting, or neuroimaging results. The findings suggest that low-level explosive blast exposure during the first 5 years of breaching training and practice does not result in any identifiable neurocognitive deficit or diagnosable medical injury, as long as the overpressure exposure is below 4 psi. (Kamimori, et al., 2018).

Along the same lines, animal models show that primary blast effects can cause significant behavioral impairments and cognitive deficits (e.g., Rigby & Chan 2007) in a dose-response relationship (i.e., the greater the BOP, the greater the deficits). These deficits are kicked off through an initial alternation of glucose metabolism that leads to a decline in the energy reserve of the brain. Over time, the cascade in metabolic, genetic, and inflammatory events leads to neurodegeneration. Interestingly, there is evidence that some brain structures, such as the cerebellum, brainstem, corticospinal system, frontal cortex, medulla, and optic tract, are more sensitive to blast effects due to their anatomic features and localization or because of the functional properties of its neural pathways and cells (Koliatsos, et al., 2004).

Traumatic brain injury (TBI), mild traumatic brain injury (mTBI), and chronic neurodegeneration resulting from military blast exposures (e.g., IEDs) have gained significant attention in recent years due to their incidences in Operation Iraqi Freedom and Operation Enduring Freedom. But the connection between the stress response and blast exposure has not yet been widely studied. In fact, of all the FBG components, overpressure has the least amount of literature on the stress response. Although literature does exist on chronic neurocognitive deficits, little is found for immediate or acute effects. Recent research on BOP exposure has partly focused on better characterizing mTBI (Ahlers, et al., 2012).

What is known about blast exposure and the stress response is that exposure to BOP can lead to an altered psychological health status, eventually leading to the development of PTSD. Animal studies show that rats exposed to a single low-level blast on three consecutive days develop anxiety and PTSD-related behavioral traits that persist for at least 9 months beyond blast exposure. Interestingly, the animals were exposed to the blast under anesthesia; therefore, the PTSD-related behavioral changes developed in the absence of a psychological stressor (Perez-Garcia, et al., 2019). The pathophysiology of blast-injury exposure involves complex cascades of chronic psychological stress, autonomic dysfunction, and neuro/systemic inflammation (Kobeissy et al., 2013). In animal models, Tümer et al. (2013) showed an increased expression of tyrosine hydroxylase and dopamine hydroxylase (both of which are catecholiamine-biosynthesizing enzymes) in the rat medulla and an increase in plasma norephinepherine concentrations 6 hours after blast injury. These results suggest that blast-wave exposure (or overpressure) triggers the endocrine mechanisms of the stress response through activation of the sympathetic nervous system.

C. Blast Exposure and Auditory Deficits

In terms of human effectiveness of FBG exposure, a critical research effort is needed to disentangle the mechanisms and time course for the differential contributions of the blast from the auditory trauma components. This work is needed not only to better understand both the critical components of the FBG that lead to desired actions in the targets but also better characterize the risk of significant injury caused by FBG exposure. At the moment, it is unclear which component, the BOP or the bang exposure, most contributes to cognitive deficits commonly observed after FBG exposure, or if a combination of the two contributes to more than the sum of each individual effect. As a practical example, one study suggests that 44% of Service members exposed to blasts have abnormal performance on two or more tests of central auditory functioning. In addition, 40% of Service members exposed to blasts during combat performed abnormally on tests utilizing complex sounds, even when their hearing fell within normal ranges or the blast did not result in mTBI (Gallun, et al., 2012).[15] These deficits remained for at least a year after blast exposure (and may have persisted beyond the study period). This is a particular concern for Service members because understanding speech in environments with competing sounds requires selective attention,

[15] The control group in this study, Service Members not exposed to blast, had completely normal performance on tests of central auditory functioning and 3% performed abnormally on tests of complex sound recognition.

memory, and other cognitive abilities; the areas of the brain involved in these higher-level functions are known to be vulnerable to damage in high-explosive blasts (Ling, Bandak, Armonda, & Gerald, 2009).

Sajja et al. (2019) have observed that the current literature on the symptomology of very-low-level blast overpressure (vLLB) with simultaneous high sound pressure (high sound pressure often associated with blast-wave exposure) is lacking. For instance, much of the blast exposure research characterizes the exposures by peak amplitude of the overpressure or number of total impulses experienced by the subject, focusing only on the overpressure, without assessing the associated sound exposure even though some of the sub-concussive symptomology (e.g., tinnitus, headache, hearing issues) also occurs as a response to intense sound exposure. In response to this, Sajja and his team exposed subjects, who wore double ear protection, at two sites on the Fort Benning grenade course range to vLLB and measured the corresponding sound meter data.[16] The subjects reported transient headaches, slower reaction time, lightheadedness, tinnitus, restlessness, frustration, and irritability after blast exposure. The findings suggest that a significant acoustic exposure occurring simultaneously as a low-level overpressure exposure may contribute to Breacher's brain-like symptomology. In other words, Breacher's brain symptoms may not be due to the effects of overpressure on the brain but instead may be caused by the effects of intense sound exposure on the brain (Sajja, et al., 2019).

On the other hand, Kubli et al. (2017) conducted a study to examine the existence of acute or long-term auditory changes due to repeated low-level blast exposures Marine breachers are subject to during training and conclude that the current blast-exposure levels in the military training environment do not have an obvious negative effect on hearing. In their study, participants were excluded if they had a history of severe TBI or profound hearing loss at or below 2 KHz. Each subject wore ear and body protection and was exposed to repeated blast exposures during training (no blast exceeded 4 psi). The subjects received a shortened auditory assessment within an hour of the breaching course and completed a battery of tests every 6 months for a period of 17 months. The results of this study showed no immediate (within an hour after exposure) or longitudinal effects of blast exposure on hearing (Kubli, Pinto, Burrows, Littlefield, & Brungart, 2017).

In terms of animal models and the potential deficits when FBG components are combined, Race and his colleagues (2017) examined differences in post-injury auditory system pathophysiology in rats exposed to mild blast plus acoustic impulse versus those exposed to the acoustic impulse alone. Their results indicate that blast exposure plus acoustic impulse resulted in abnormal auditory functioning across all levels of the auditory

[16] The Sajja et al. (2019) study exposed subjects to vLLB ranging from 0.14 to 0.42 psi at site #1 and 0.22 to 0.30 psi at site #2, which corresponded to sound exposure ranging from 153.72 to 163.22 dBP (dB peak pressure).

system relative to the impulse exposure alone. Interestingly, the dysfunction was observed in the processing of temporally modulated sounds, meaning that these central auditory processing deficits become more pronounced with increased complexity of auditory processing tasks such as sound localization, speech and non-speech sound recognition in noise, or language processing (Race, Lai, Shi, & Bartlett, 2017).

Based on these the results, we conclude that findings from human and animal models examining the effects of blast or intense sound exposure show the detrimental physiological effects and cognitive-behavioral impacts of such exposure. That said, there is a need to better understand contributions of the blast, the bang, or the combination of blast and bang on the deficits observed.

D. Research Opportunities

We recommend that any research looking into BOP effects use animal studies of the blast overpressure effects of FBGs. In fact, over the past several decades, experimental animal models for blast injury have been developed using rats, mice, ferrets, rabbits, and larger animals such as sheep and swine (Kobeissy et al. 2013). While Kobeissy et al. (2013) investigated blast exposures of higher magnitude, for example roadside IEDs, Figure 8 details BOP variables that can be examined in animal studies for FBGs. One particular metric in human studies that could be exploited in research is time as a variable to regain balance (this serves as a proxy for vestibular function). IDA offers the following recommendations for fruitful research deficits as they relate to the stress response and performance impairment related to FBG exposure:

1. Establish Connection between Physiology and Performance Decrements

Due to there being such limited research on BOP and performance, the first step in any research paradigm investigating BOP would be to establish which physiological component of BOP leads to distraction effects and performance decrements. There is some anecdotal evidence that the BOP component from FBG is potentially the most physiologically significant component and the hardest component to "ignore" (i.e., even when you are mentally aware and prepared for an FBG blast, you cannot ignore the physiological effects of BOP).

2. First-Order Effects of Overpressure and the Stress Response

After understanding which BOP components contribute to performance decrements, establishing the connection between the stress response and overpressure would greatly improve understanding of FBG human characterization. This could include cognitive and behavioral effects due to vestibular/thoracic effects or psychological response to stress.

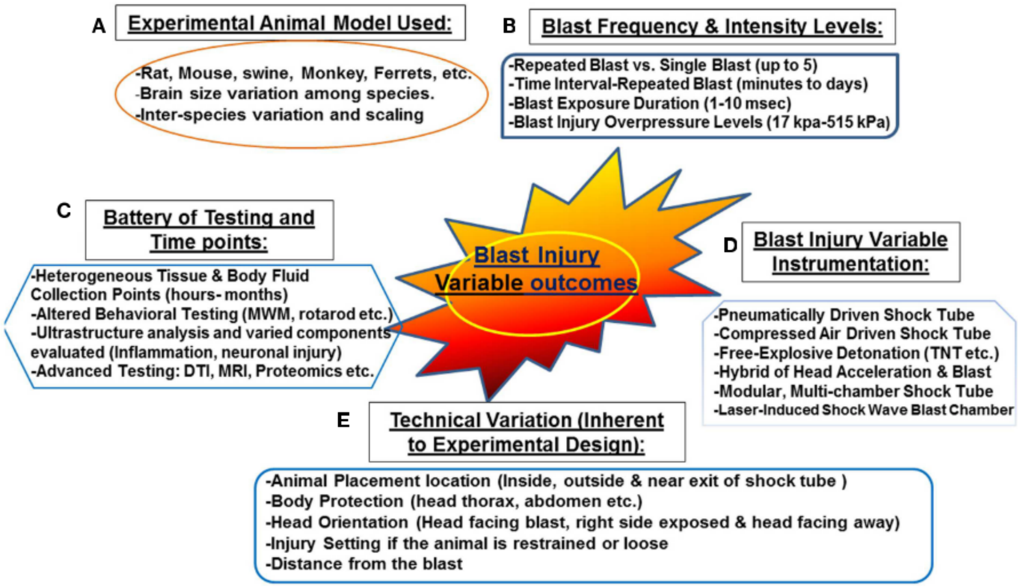

Figure 8. Blast Overpressure Variables That Can Be Examined in Animal Studies for FBGs. From Kobeissy et al. (2013, 13).

7. Conclusions and Recommendations

In this report, we identify the critical importance of the stress response as a mediating variable in understanding the effectiveness of FBGs, specifically as it drives relevant IFC behavior. Prior research efforts on FBGs have provided scientific principles behind observed weapon effects and defined the weapon effects on the human body (e.g., the startle reflex and modeling efforts for risk of significant injury). IDA's work analyzing the role of the stress response as a mediating variable in effectiveness is a key strategic effort to further our understanding of *why* the FBG device has the effects that we observe on humans—a question mostly ignored until now. The objective of IFC deployment is to change behavior in targeted groups or individuals in a predictable way (so as to maximize success in certain military interventions). The connection between FBG physiological effects and the cognitive, psychological, and behavioral changes in targets remains mostly unexplored. IDA's stress analysis aims to fill these critical gaps in our understanding of the effectiveness of FBGs by laying the foundational work that may inform device developers, testers, and individuals who determine concepts of operation, as well as future research explorations.

Throughout this analysis we have discussed important elements of operational contexts; metrics and measurements in experimental, laboratory, and observational settings; and relevant performance outcomes for FBGs that should be examined with regards to the stress response. Of critical importance, this report examines how each FBG component is connected to the stress response—physiologically and psychologically, and behaviorally—and provides the JIFCO and the IFC community at large with research opportunities that would significantly advance our understanding of the effects of FBGs. Furthermore, although the focus of this report is on FBGs, the stress response carries over to other IFC devices; a similar exploration of how stress affects the sensory components of other IFC devices may therefore offer a significant contribution to characterizing human effectiveness and IFCs. The recommendations for future research efforts are briefly summarized in Table 3; please refer to each individual section for details about each recommendation.

Table 3. Summary of Research Opportunities for the Stress Response and FBG Components

Psychological (Chapter 3)	Visual (Chapter 4)	Auditory (Chapter 5)	Overpressure (Chapter 6)
• Incorporating time and intensity as factors • Individual differences • Realistic test conditions • Crowd movements • Video recording analysis of movement patterns and relevant FBG conditions	• Timing of flash blindness effects • Psychological effects of temporary and partial blindness • Physiological effects of pupillary reflex	• Stress and priming • Performance outcome variation • Combined effects	• Established connection between physiology and performance decrements • Experiment with first-order effects of overpressure and stress response

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Abbreviations

ACTH	adrenocorticotropic hormone
BP	blood pressure
CRH	corticotropin releasing hormone
DANA	Defense Automated Neurobehavioral Assessment
dB	decibels (unweighted sound pressure levels)
dB-A	A-weighted decibels (weighted to human hearing)
DoDI	Department of Defense Instruction
EEG	electroencephalograph(y)
FBG	flashbang grenade
GC	glucocorticoid
GFAP	glial fibrillary acid protein
GPS	Global Positioning System
HPA	hypothalamus-pituitary-adrenal (axis)
HR	heart rate
IAPS	International Affective Picture System
IDA	Institute for Defense Analyses
IED	improvised explosive device
IFC	intermediate force capability
JIFCO	Joint Intermediate Force Capabilities Office
mTBI	mild traumatic brain injury
NA	noradrenaline
NIHL	noise-induced hearing loss
NLW	non-lethal weapon
PNS	parasympathetic nervous system
PTS	permanent threshold shift
PTSD	post-traumatic stress disorder
SNS	sympathetic nervous system
5-HT	serotonin
TBI	traumatic brain injury
TTS	temporary threshold shift
UCH-L1	ubiquitin carboxy-terminal hydrolase L1

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14. ABSTRACT

Flashbang grenades (FBGs) are a type of intermediate force capability used in operational contexts to create diversions, confuse or disorient those exposed, or control crowds, while simultaneously minimizing significant injury to its human targets. These target behaviors are achieved when the psychological and physiological responses to FBG components (flash of brilliant light, auditory bang, overpressure) are activated. IDA has identified the stress response as a strategic core area in FBG effectiveness. This report analyzes stress as a potential significant mediator of FBG target behavior. Our analysis is concerned with how the stress response is triggered and, more important, how the stress response motivates a behavioral change so that the military goal can be accomplished. We focus on the DoD context for FBG use, behavioral outcomes, and targeted behavior of use. In addition, we provide research opportunities to further advance JIFCO's FBG portfolio and efforts to characterize FBG human effectiveness.

15. SUBJECT TERMS

flashbang; human effectiveness; JIFCO; joint intermediate force capability; Non-Lethal Weapon (NLW); stress response

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