



INSTITUTE FOR DEFENSE ANALYSES

Analysis of the Startle Response to Flashbang Grenades

Poornima Madhavan
Ruhi Srinivasan

June 2018

Approved for public release;
distribution is unlimited.

IDA Document D-8945

Log: H 18-000043



The Institute for Defense Analyses is a non-profit corporation that operates three federally funded research and development centers to provide objective analyses of national security issues, particularly those requiring scientific and technical expertise, and conduct related research on other national challenges.

About This Publication

This work was conducted by the Institute for Defense Analyses (IDA) under contract HQ0034-14-D-0001, Project DU-2-4106, "Independent Research Assessments," for the Joint Non-Lethal Weapons Directorate (JNLWD). The views, opinions, and findings should not be construed as representing the official position of either the Department of Defense or the sponsoring organization.

For More Information

Poornima Madhavan, Project Leader
pmadhava@ida.org, 703-578-2822

Leonard J. Buckley, Director, Science and Technology Division
lbuckley@ida.org, 703-578-2800

Copyright Notice

© 2018 Institute for Defense Analyses
4850 Mark Center Drive, Alexandria, Virginia 22311-1882 • (703) 845-2000.

This material may be reproduced by or for the U.S. Government pursuant to the copyright license under the clause at DFARS 252.227-7013 (a)(16) [Jun 2013].

INSTITUTE FOR DEFENSE ANALYSES

IDA Document D-8945

**Analysis of the Startle Response to
Flashbang Grenades**

Poornima Madhavan
Ruhi Srinivasan

Executive Summary

Background

The surprise or “startle” element associated with flashbang grenades may trigger varying degrees of psychological and physiological distress that could have implications for the design of effective flashbang grenades. The startle response is the fastest known generalized motor reaction of humans and animals to unexpected or surprising stimuli. The Institute for Defense Analyses (IDA) was tasked with examining the neural, physiological and psychological components of the startle response associated with flashbang grenades to fully understand the processes driving human responses to flashbangs.

Analysis

IDA analyzed what happens physiologically and psychologically between the generation of a startling stimulus (such as a sudden flash or sound) and subsequent behavior change in the organism being subjected to the stimulus. Although flashbang grenades comprise both auditory and visual components, the predominant focus of this report is on the auditory component (the “bang”) since research on the startle effect focuses largely on the effects of auditory stimuli on startle. Research also revealed evidence for asymmetric muscle activation in response to startle, with faster head rotation toward the dominant side of the body and greater flexion of the dominant arm to protect or defend against the startling stimulus. This provides important insights into how humans may physically orient themselves immediately following the experience of a startling stimulus.

Analysis of cardiovascular and circadian systems revealed a clear connection between fear and the magnitude of the startle response, indicating that the mere expectation of an aversive stimulus (known as fear-potentiated startle) may exert as powerful an effect on the startle response as the physical experience of the flashbang itself. Fear-potentiated startle caused by a stimulus that is perceived to be threatening can trigger a full stress response, leading to severe levels of cognitive impairment. On the other hand, startle responses elicited by stimuli that are perceived to be nonthreatening or even pleasant subside quickly and return the organism rapidly to its original state of homeostasis. That is, the startle effect wears off rapidly when the startle-eliciting stimulus is not explicitly associated with a significant negative outcome, whether physical or psychological.

Gender and age are important determinants of susceptibility to startle. Women tend to demonstrate a greater degree of susceptibility to startle, especially when the stimulus is acoustic in nature; some of this is associated with women’s enhanced hearing ability

compared with men. Advancing age has been associated with a decrease in speed of responding to a startling stimulus but a potential increase in the overall likelihood of being startled.

Recommendations

Implications of this research include ways to enhance the human effectiveness of flashbang grenades without increasing the physical intensity of the flashbang or raising the risk of significant injury. Startle-modification techniques are cognitive methods to systematically magnify or inhibit the startle effect by physically pairing pre-selected secondary stimuli with the original startle-eliciting stimulus. IDA recommends that future efforts at improving the effectiveness of flashbang grenades be focused on startle modification methodology; this will require further research into startle inhibition and facilitation and the development of startle-modification metrics relevant to flashbangs, as discussed in this report.

Contents

1.	The Startle Response	1
2.	Physiology of the Startle Response	5
	A. Neural Pathway	5
	B. Motor Pathway	8
	C. Cardiovascular System	9
	D. Circadian System and Effects of Light-Dark Cycles	11
3.	Psychological Variables Associated with the Startle Response	13
	A. Affect (Emotion)	13
	B. Individual Differences	16
	1. Gender	16
	2. Age	19
	3. Personality	20
4.	Startle Modification	23
	A. Startle Habituation and Sensitization	23
	B. Startle Inhibition and Facilitation	24
5.	Findings and Recommendations	29
	A. Findings	29
	B. Recommendations	30
	References	A-1
	Abbreviations	B-1

1. The Startle Response

Flashbang grenades are popular nonlethal weapons used by domestic law enforcement and military forces. Although the U.S. military has used grenade simulators for more than 60 years, the Operations Research Unit of the British 22 Special Air Service (SAS) Regiment is credited with developing the first modern flashbang grenade in the early 1970s. The SAS also developed and refined the tactics for the employment of these devices. The first documented operational use of a flashbang was by Israeli commandos during Operation Thunderbolt to rescue passengers of a hijacked Air France jetliner at Entebbe, Uganda, in July 1976.

The typical flashbang grenade's explosive energy generates a deafening boom accompanied by a brilliant flash of light that can be temporarily blinding. Today, flashbang grenades have become a common component of tactical police teams anywhere in the country. Examples of police-use cases include barricaded gunmen, where the grenades provide enough of a distraction to allow the police to storm the building when the gunmen are temporarily disoriented by the flashbang. Realistic flashbang training has never been more affordable, and flashbang training options allow teams to train more often, in more places, and with much greater safety than has been the case in the past.

Flashbangs have proven to be of low lethality over many years of tactical use, but they can be either a lifesaver or a liability, depending on the manner in which they are deployed. According to law enforcement officials, flashbang grenades, when appropriately deployed, can prevent needless injuries and protect arresting officers from bodily harm because a disoriented suspect is more likely to be compliant and less likely to require force during an arrest. When improperly deployed, however, they have the potential to cause serious bodily injuries and property damage. In addition, the surprise or "startle" element associated with flashbangs can trigger varying degrees of psychological and physiological distress. Arguably, the startle effect alone may result in more serious consequences (such as stress to vital organs such as the heart) than the physical effects of the flashbang per se. Therefore, in addition to the physical effects of flashbang exposure, the startle associated with flashbang grenades should be studied in order to fully understand the processes underlying human responses to flashbangs and consequently improve the design of flashbangs.

The startle response (SR), typically a response to unexpected stimuli, is the consequence of involuntary activation of the motor tracts that is generated in the brainstem and the fastest known generalized motor reaction of humans and animals. In rodents, the

SR is characterized by immediate activation of the facial and skeletal muscles, leading to a whole-body flinch within a few milliseconds. In humans, the SR typically consists of an early brief and generalized muscle contraction lasting a few milliseconds, followed by a more elaborate activity, known as the orienting response (OR), resulting from the central integration of all sensory information conveyed by the stimulus. In humans, the SR is most commonly elicited by auditory stimuli, although visual (McManis et al. 2001), somatosensory (Gokin and Karpukhina 1985) and vestibular stimuli (Bisdorff, Bronstein, and Gresty 1994) have also been known to produce the effect. The SR is physiologically and psychologically altered in some disease states, generating dysfunctions that manifest as either excessive or defective SRs.

As mentioned above, the SR comprises two components: (1) an involuntary startle reflex and (2) a voluntary OR. The first component is a fast involuntary reflex contraction of the face and limb muscles that follows a rostral-caudal (“head-to-toe”) progression. This involuntary reflex is also characterized by a combination of eye closure, facial grimacing, neck flexion, and arm abduction or flexion (Wilkins, Hallett, and Wess 1986; Brown et al. 1991a, b), that are more-or-less symmetrically distributed across the human body. In some people, the effects are only partial. Following this early involuntary, rapid reflex, the second component of the SR is embodied in some kind of voluntary muscle activity (organized in a component referred to above as the OR). The OR is a vaguely defined behavioral reaction that is hypothesized to result from a combination of motor actions and emotional reactions such as curiosity, fear, or annoyance (Gogan 1970). The motor component of the OR consists of maneuvers involving the whole body that are strongly influenced by context and individual differences. In most humans, the OR is generally limited to slow movements of the head or upper limbs, laughter, or guttural vocal expressions. If the startle-eliciting stimulus is located nearby and within the visual field of the subject, some humans may exhibit more complex reactions, such as quickly rising from a seated position and heading toward the nearest exit before attempting to look for any reasonable explanation for the stimulus. The OR is also associated with physiological changes in galvanic skin resistance, transient increases in blood pressure, and acceleration of heartbeat frequency (Gautier and Cook 1997; Holand et al. 1999). The responses elicited by a startling stimulus therefore include rapid and immediate involuntary changes in excitability at the cortical and subcortical levels (the startle “reflex”) that are followed by somewhat slower shifts in voluntary motor functions (the OR). Generally, the larger the startle, the larger the corresponding OR (Gogan 1970). While the initial reflex reaction serves a basic protective function, the OR is the final expression of the behavioral change in preparation for defense or attack.

For psychophysicologists, the SR has a number of advantageous characteristics (Haerich 1997). First is the ubiquity of the SR; analogous startle-like rapid escape responses are found across a range of organisms from fish and crustaceans to humans,

making the phenomenon relatively easy to observe in an experimental setting (Eaton, 1984; Landis and Hunt 1939). Second, specific neural circuitry underlying the SR has been identified in rats, again facilitating laboratory research on this topic. Finally, there exists a readily quantifiable response in the human, that is, the electromyographic measurement of the eyeblink reflex component of an SR. Although questions have been raised about the degree of similarity between the neural circuitry of the SR in rats and humans, the similarities nevertheless provide the opportunity for more in-depth investigation of an important behavioral phenomenon observed in humans using rodent models.

Descriptions from the physiological and psychological literature of reactions induced by startling stimuli contain a multitude of contradictory terminology. In addition to the broader “startle response” (that we refer to as SR), there are two types of SR specific to acoustic stimuli with some physiological differences—the acoustic startle response (ASR) and the acoustic (eye)blink reaction (ABR). The ABR is a rapid involuntary blinking of the eye in response to a startle probe that lasts for a few milliseconds; it is induced more frequently, is more consistent, and is less prone to habituation with repeated presentation of the stimuli than the ASR. The ASR is similar to a general SR in that it comprises both an involuntary physiological reaction and a voluntary OR. The peculiarities of the ABR are due to a different patterned organization of the orbicularis oculi (OOc; see Figure 2) muscle response to startling stimuli in comparison to other muscles (Bisdorff, Bronstein and Gresty 1994); the response is typically patterned in proportion to the type and strength of sensory inputs and the subject’s degree of muscle preparation to react. ASR testing has revealed increased reflex responses in neurologic conditions such as hyperkplexia (i.e., very pronounced SR), multiple system atrophy, and blepharospasm (restless legs syndrome), and decreased reflex responses in dementia and cervical dystonia.

In the ensuing chapters, we examine the various dimensions of the ASR, the ABR, and the OR in an attempt to better understand what happens physiologically and psychologically between the eliciting of a startling stimulus and the organism’s response. Specifically, this research offers the potential to develop a better understanding of the interaction of information- and emotional-processing systems involved in the SR and the role of individual differences in response patterns to startle-inducing stimuli. Study of the SR will provide several answers about human information processing in situations where humans come in contact with intense visual, auditory, and vibrotactile stimuli for very brief durations. In the case of nonlethal weapons, the ability to understand and subsequently modify the SR is relevant to the effective deployment of flashbang grenades because this ability provides an opportunity to enhance the effectiveness of the flashbang without necessarily increasing the intensity of the flashbang (and consequently increasing risk of significant injury).

2. Physiology of the Startle Response

Research has documented that unanticipated events can provoke a universal SR within the central nervous system, which results in brief, but widespread changes in neuromuscular activity (DeAngelis 2012; Koch 1999). When an SR is provoked, neuromuscular changes to preparatory (feed-forward) and reactive (feedback) muscle contractions in the extremities can be observed, potentially altering the stiffness regulation necessary for energy absorption and dynamic body stabilization (Freeman and Wyke 1966; Lacroix 1981). Although the SR itself is brief, typically lasting no longer than a few milliseconds to a few seconds, the physiological consequences of startle may be potentially far-reaching and have long-lasting implications for the functions of vital organs such as the heart and the brain, as well as the limbs. In this chapter, we discuss the neural pathway of the SR and its relationship with resulting motor and cardiovascular responses. We conclude this chapter with a discussion of how the SR is modulated by the organism's circadian rhythm and the effects of light (versus darkness) on the SR.

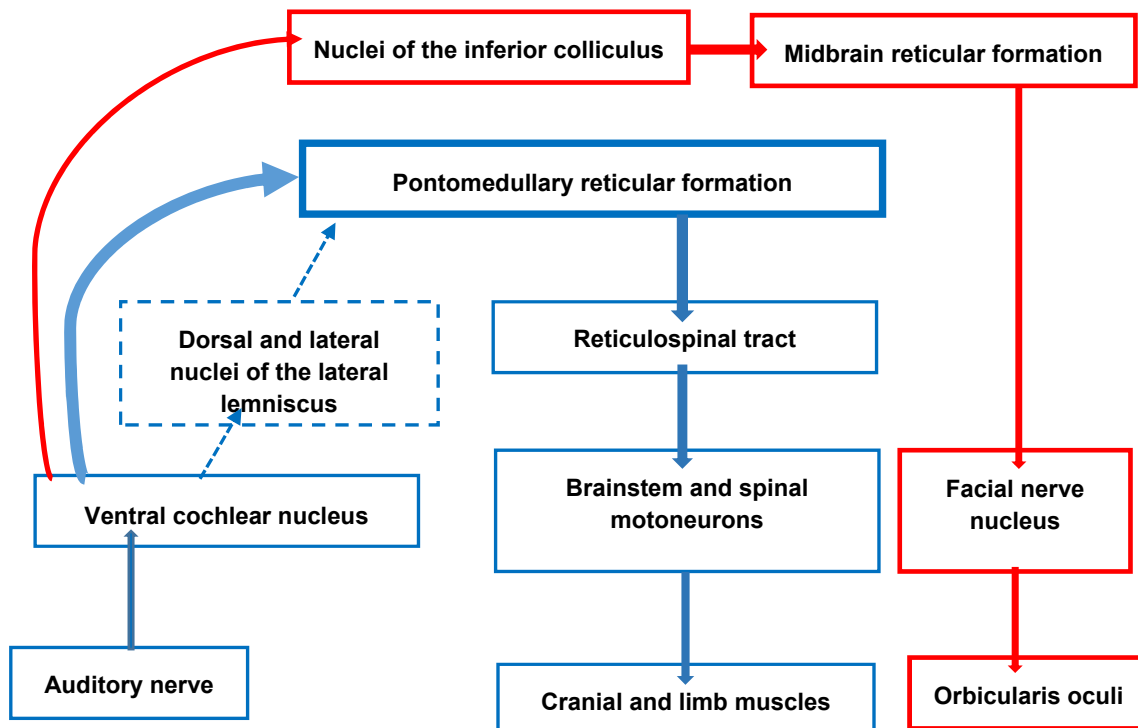
A. Neural Pathway

Studies on animals have contributed essential information about the neural circuits involved in the SR (Davis et al. 1982; Koch 1999; Yeomans and Frankland 1995). Various lines of evidence suggest that the nuclei of the amygdala figure prominently in the potentiation of the SR in the rat. Impulses are transmitted to the amygdala via an intricate pathway involving the sensory nerves, neurons, the thalamus, and the amygdala. First, there are direct, monosynaptic projections from the amygdala nuclei to the reticular site, an important component of the ASR pathway. Second, electrical stimulation of the amygdala directly enhances the SR amplitude (Davis 1997). Last, lesions of either the central or lateral and basolateral nuclei of the amygdala lead to a reduction of the SR. From these data, Davis and colleagues (1982) have concluded that amygdaloid nuclei play essential roles in the generation and development of the SR. Similarly, LeDoux (1990) has also argued that the amygdala is primarily involved in the process by which the SR is potentiated, particularly in response to aversive stimuli. His data suggest that in situations involving fear-inducing startle probes, the lateral and then the central nuclei of the amygdala are activated by the sensory thalamus in response to simple fear-inducing acoustic stimuli; in the rat, separate pathways from the amygdala appear to mediate autonomic (blood pressure and heart rate via the lateral region of the hypothalamus) and somatic ("freezing" response) components of the aversive SR.

In line with LeDoux's (1990) suggestion of dual-SR pathways from the amygdala, Davis and colleagues (1982) have proposed that two neural circuits affect the magnitude of a rat's SR: a primary, obligatory circuit (from the cochlear nucleus through the nucleus reticularis pontis caudalis to the spinal cord) and a secondary, modulatory circuit based on projections to the reticular site from the central nucleus of the amygdala. The primary circuit is engaged by a reflex-eliciting stimulus that is intense and has a rapid rise time. The modulatory circuit has been proposed to account for the observation that the SR in rats is augmented by a nonreflex-eliciting stimulus (e.g., steady-state light) that, through conditioning, has become associated with an aversive outcome (e.g., electric shock) (Bradley et al. 2001; Sierra-Mercado et al. 2006). For human subjects, startle probe reflexes show significant modulation as a function of the aversiveness of a pictorial stimulus (Bradley et al. 2001): as rated unpleasantness of a stimulus increases, corrugator muscle responses increase, heart rate decelerates, and the SR is augmented.

The general idea derived from animal models and experiments with human subjects is that voluntary motor acts (such as a defensive head movement to avoid a sudden blinding flash of light) can be executed to counteract the startle effect. This voluntary counteraction of the startle effect is only possible upon activation of the cerebral neuronal circuits. Specifically, in situations where rapid movement execution is needed, the neural circuits must transmit information in such a way that it is ready for the release of action at the appropriate movement. The exact neural mechanisms underlying motor preparation are not known, but in broad terms, they involve a build-up of excitability in subcortical structures in accordance with the program to be executed. This enables an unexpected and abrupt sensory input to trigger the appropriate motor response by direct activation of the prepared subcortical structures (Valls-Solé, Kumru, and Kofler 2008).

Although there have been several theories about the neural circuits involved in the SR, some of the strongest evidence of a "startle pathway" has been presented in the context of the ASR. This pathway is depicted in Figure 1. Based on experiments with rats, Davis and colleagues proposed that the circuit of the ASR that allows for the immediate activation of subcortical structures for motor response involves the cochlear nucleus, the nucleus of the lateral lemniscus, and the motoneurons of the brainstem and the spinae through the medial reticulospinal tract (represented by blue arrows and blue boxes in Figure 1). At present, the ASR is considered to be conveyed through a very simple circuit (Yeomans and Frankland 1995; Davis 1996; Koch 1999), with activation of neurons in the pontomedullary reticular formation after direct synaptic activation from the cochlear nucleus. Neurons of the pontomedullary reticular formation play a crucial role in maintaining behavioral arousal and consciousness; the cochlear nucleus is where the processing of any kind of acoustic information begins and outputs from the cochlear nuclei are received in higher regions of the auditory brainstem.



Source: Adapted from Vals-Solé et al. 2008.

Figure 1. The Circuit of the SR to Auditory Stimuli. In blue are the structures mediating the ASR as described by Davis et al. (1982). In red are the structures mediating the ABR, as described by Hori et al. (1986).

An alternative pathway comprising neurons of inferior colliculus and the midbrain has been suggested (Hori, Yasuhara, Naito, et al. 1986) to account for the peculiarities of the response recorded from the OOc muscle to sound stimuli, the ABR. The ABR follows a different neural pathway than the ASR and is represented by red arrows and red boxes in Figure 1. The difference in neural pathways is primarily because of the differences in the sensory inputs and degree of muscle preparation needed to react in the case of the eyeblink (in ABR) versus other gross bodily movements (in ASR) (Hori et al. 1986). Specifically, in the case of the ABR, responses would need to be faster and more consistent than other muscles (such as those controlling the extremities) to protect a delicate and vital organ like the eyes from potential damage. This is accomplished by activation of the OOc.

Apart from direct motor responses, a startling auditory stimulus also causes changes in the excitability of the structures along the motor pathway (Furubayashi et al. 2000). The motor effects of the SR are discussed further in the next section on motor responses to startle. In the context of voluntary movements (such as when performing a simple reaction time task), an unexpected startling stimulus in conjunction with the original stimulus speeds up reaction time relative to the original stimulus alone (Valls-Solé et al. 2005).

Controlled simple reaction time experiments in laboratory settings have demonstrated that the simple motor task of performing a ballistic wrist movement is executed much faster, but is otherwise unchanged, in the presence of a secondary startle-eliciting stimulus; similarly, saccadic eye movements reach the target faster but with the same degree of precision in the presence (versus absence) of a secondary startling stimulus (Castellote et al. 2007). This enhancing effect of a startle probe on motor responses has been labeled the StartReact effect (Vals-Solé, Kumru, and Kofler 2008).

B. Motor Pathway

In parallel with the activation of neural circuits, a startling auditory stimulus triggers changes in the excitability of structures along the motor pathway. Specifically, the pattern of the ASR is modified according to posture (Brown et al. 1991b), with the ASR of leg muscles being shorter in latency and twice as frequent when the subject is standing than when seated. The ASR is understood to be a generalized, bilaterally synchronous response to sudden loud noise that is symmetrically distributed across both sides of the human body (Brown et al. 1991a ,b; Grosse and Brown 2003). However, studies on clinical samples have revealed that some special populations (such as patients of dementia) tend to display asymmetrical distribution of symptoms, which suggests interesting questions about gross body responses to startle-eliciting stimuli. Some studies have shown that the ASR is modified by body laterality (Kofler et al. 2008), with larger ASR observed on the side of the dominant hand.

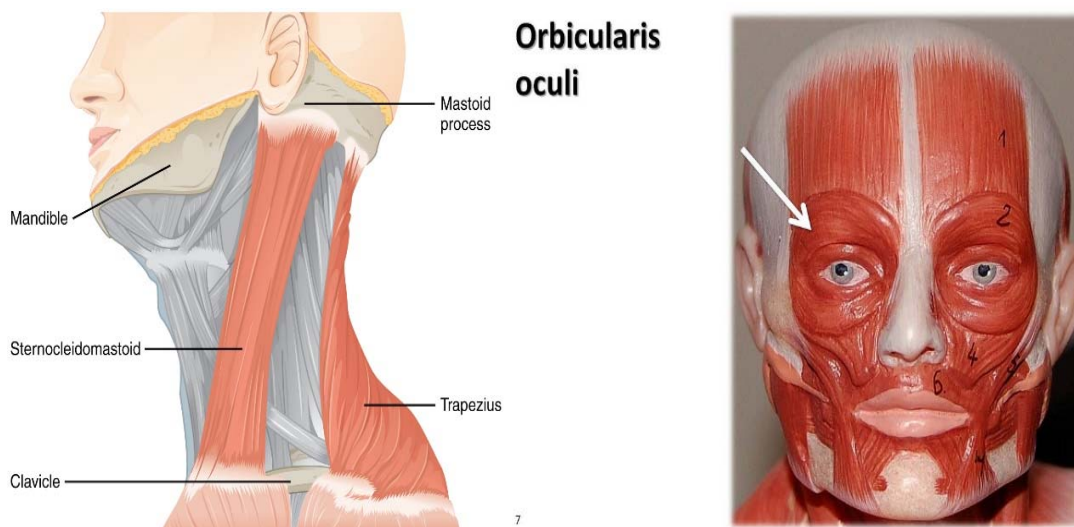


Figure 2. Sternocleidomastoid and Orbicularis Oculi

Kofler and colleagues (2008) studied potential ASR side-to-side differences in normal subjects, related them to handedness, and provided a basis for comparison with potential ASR side-to-side differences observed in patients with various pathological conditions.

Results revealed that the ASR is not completely symmetric even in a normal nonclinical population; significant response size differences were found in the sternocleidomastoid muscles, which are pairs of muscles that connect the sternum, clavicle, and mastoid process of the temporal bone that serve to turn and nod the head (see left panel of Figure 2) and the facial muscle controlling the OOc that arises from the nasal part of the frontal bone (see right panel of Figure 2). The latter serves to close the eyelids and is used in blinking, winking, and squinting. Also, there is a trend toward size asymmetry in the biceps brachii, which are muscles of the upper arm between the shoulder and elbow. Although the effect for biceps brachii was small for the entire sample in the Kofler et al. (2008) study, analyses of subgroups revealed significantly larger biceps brachii response asymmetry in favor of the dominant (versus nondominant) side in left-handers and significantly larger biceps brachii ASRs in right-handed than in left-handed subjects. Observed asymmetrical muscle-activation patterns may serve a functional role in generating motor responses to startle: larger responses in sternocleidomastoid contralateral to the dominant hand would rotate the head toward the dominant side; larger responses in biceps brachii ipsilateral to the dominant hand would induce more flexion movement in the dominant arm, possibly to withdraw the arm or prepare for defense (Cadenhead et al. 2000). The ability to predict directional movement of the head, limbs, and torso in response to startle is a useful tool for analyzing the motor consequences of exposure to a startle probe.

C. Cardiovascular System

Various physiological adjustments have evolved to support rapid responses to perceived danger (Gautier and Cook 1997). Much of the research on physiological responses to suddenly occurring external threats (such as those posed by flashbang grenades) can be aggregated into three frameworks or models involving the cardiovascular system.

The first framework focuses primarily on the effect of sudden startling stimuli on sensory processing. Nearly four decades ago, Graham (1979) proposed that heart rate could be used to distinguish among OR, SR, and defense reactions: ORs are characterized by initial cardiac deceleration, SRs by short-latency (quick) cardiac acceleration, and defense reactions by long-latency (slow) cardiac acceleration. Both physical and emotional variables have been demonstrated to drive whether the exhibited reaction is typically defensive versus orienting versus startling. Orienting and defensive responses have been related to the affective content of the startle-eliciting stimulus (e.g., Cook et al. 1986) whereas SR has been shown to be potentiated by aversive emotional states of the subject that may have existed independently of the conditioning stimulus (e.g., Cook et al. 1992; Lang, Bradley, and Cuthbert 1990; Vrana, Spence, and Lang 1988).

The second framework treats SR as a special case of the hemodynamic (biochemical) regulation underlying the “fight or flight” response (e.g., Abrahams, Hilton, and Zbrożyna

1960). The SR thus defined is considered to involve intense emotional and physiological adjustments necessary to support reactions to imminent danger; similar constellations of physiological adjustments have been observed when people perform physically undemanding but cognitively challenging tasks (Brod et al. 1962).

The third conceptual framework is derived from investigations of relationships between psychological stress and cardiovascular disease (Gautier and Cook 1997). Contrary to the first two frameworks, this framework does not emphasize emotional coping as an integral component of immediate responses to threatening stimuli. Instead, this framework dichotomizes typical human threat responses into active and passive coping mechanisms, with each determining the degree of susceptibility to the SR. During active coping (characteristic of the SR and defense reactions), cardiac rate is typically under sympathetic influence (i.e., the component of the autonomic nervous system that readies a person for the fight-or-flight response), whereas during orienting and passive tasks, heart rate is primarily under parasympathetic control (i.e., the component of the autonomic nervous system that prepares the body for rest, digestion, and recovery mode). Across all three frameworks, there is agreement that repeated evocations of reactions that involve active coping contribute to the long-term development of cardiovascular disease.

Overall, cardiovascular reactivity is expected to be larger in individuals showing greater affective SR. To test this hypothesis, Gautier and Cook (1997) studied the performance of experimental subjects that varied on affective characteristics that correlate with startle modulation (mainly, fearfulness) and cardiovascular reactivity (parental history of cardiovascular disease) on a cognitively challenging task with intermittent loud background noises. The primary research question was whether the SR, cardiovascular reactivity, and performance on cognitive tests (when exposed to sudden loud noises) shared affective modulatory influences (i.e., potentiation by aversive versus pleasant imagery). Short-latency heart rate acceleration (which the authors refer to as “cardiac startle”) was observed in response to sudden intense noise, but there was no subsequent long-latency fight-or-flight response that is characteristic of defense reactions, thereby eliminating the possibility of any subsequent motor response.

There is evidence in the literature that predictability inhibits the cardiac response to physically intense stimuli (Fernández and Vila 1989), and it is possible that Gautier and Cook (1997) reduced the likelihood of observing the late acceleratory defense response by warning participants about an imminent loud noise before the start of the experiment. Nevertheless, one of the major findings of this study was that startle modulation and cardiovascular reactions are related through negative affect engagement. The principal new finding of this study was a significant association between the SR and blood pressure reactivity during cognitively challenging tasks. Specifically, in addition to rapid cardiovascular reactions to loud noise, greater affective modulation of startle was associated with higher systolic blood pressure for male participants. Final analyses of the

data from Gautier and Cook's study also revealed that fearful individuals showed larger cardiac acceleratory responses to intense noise, suggesting that cardiovascular reactions to startle-potentiating stimuli are difficult to disengage from affective reactions and that the two must be studied in conjunction to understand the comprehensive effects of startle-inducing stimuli on human responses.

D. Circadian System and Effects of Light-Dark Cycles

As circadian rhythms have been found to influence a number of phenomena, including sleep/wake cycles, motor activity, and hormone concentration levels, researchers have hypothesized that they may also affect the SR (Horlington 1970). Research with nonhuman subjects has demonstrated that the ASR exhibits circadian modulation, with as much as a twofold increase in potentiated startle seen during the dark conditions of a light-dark cycle (Chabot and Taylor 1992a). Miller and Gronfier (2006) examined whether changes to startle reactivity could occur in human subjects studied at different intervals during the 24-hour circadian cycle. The researchers examined whether ABR would differ when tested just after waking (in "light" conditions) or shortly before bedtime (in "dark" conditions), hypothesizing increased startle in the morning than in the evening due to higher concentrations of cortisol, which is related to the circadian rhythm. The results, however, demonstrated higher SR during the evening (after sunset) than in the morning, despite higher levels of cortisol in the morning. These results are in line with previous research using rats that demonstrated higher SR amplitudes during the dark period than during the light period of a 24-hour light-dark cycle (Chabot and Taylor 1992a; Frankland and Ralph 1995). This pattern of circadian-rhythm-associated SR is persistent, with research demonstrating that amplitude differences can be found after multiple, repeated measurements (Chabot and Taylor 1992a, b). Therefore, it appears that this modulation of startle is under endogenous control.

While light-dark cycles can influence endogenous circadian rhythms in mammals, light may also have different, and possibly contradictory, effects on startle that are unrelated to the circadian rhythm due to properties of the stimulus itself. Studies have demonstrated that light may have anxiogenic, or anxiety-provoking, effects in rats and mice, who are nocturnal animals (Walker and Davis 1997). Rats and mice tend to prefer the darkened areas of experimental set-ups and even in open environments prefer to operate under conditions where there is low illumination. Therefore, light may be an aversive stimulus and provoke fear, with the potential to increase SR magnitude when placed in a set-up with high illumination levels. Walker and Davis (1997) compared the ASR of rats that were tested under low-illumination and under high-illumination conditions and discovered that higher levels of illumination resulted in an increase in SR amplitude. Providing the rats with anxiolytic (anxiety-reducing) drugs such as buspirone was found to

decrease this effect, suggesting that the reason for the initial effect reflects the anxiogenic properties of light for rodents.

Unlike nocturnal animals such as rats and mice, humans are diurnal, and some evidence has shown that they tend to be more anxious in dark environments due to fears of the dark and the associated loss of visual information (Grillon et al. 1997). Therefore, it is possible that humans may experience magnified SRs in dark environments (as opposed to illuminated environments) in the same way that nocturnal animals are more fearful and startle more easily in brightly illuminated spaces. In a study with human participants, Grillon and colleagues (1997) examined startle eye-blink reflex responses in light and in dark environments. Darkness was found to increase SR magnitude in humans; this effect was attributed to affective as opposed to attentional explanations. The researchers reasoned that this increased SR was due to humans' fear of the dark that typically has its origins in childhood; these findings led to the conclusion that deeply entrenched fears might be meaningfully correlated with increased SR magnitudes in humans.

3. Psychological Variables Associated with the Startle Response

The magnitude and latency of the SR has been known to vary widely between and within individuals. Specific to humans, research has revealed influences of gender and age on the ASR (Kofler, Müller, Reggiani and Valls-Solé 2001; Kofler, Müller, Wenning et al. 2001): some studies have reported that women have higher ASR probabilities and larger responses than men, and older subjects have been shown to demonstrate larger SR magnitudes in the extremities compared with younger subjects. The excitability of SR circuits is also modulated by mood, attention, fear, and other emotional states (Ho et al. 1987; Lang, Bradley, and Cuthbert 1990). Research has revealed a relationship between the size of the SR and degree of emotional arousal derived from the stimulus and the engagement of the whole body in preparation for reaction to it (Lang and Davis 2006). In this chapter, we report in detail on the role of cognition and emotion (affect) in shaping the SR and the manner in which individual differences influence human startle reactivity.

A. Affect (Emotion)

A substantial body of research has shown that the SR is potentiated by situations that involve the processing of aversive information. Specifically, it has been shown that the reflex blink magnitude (elicited by an acoustic stimulus) increases when a subject perceives or remembers unpleasant events (Cook et al. 1991). Conversely, tasks and environments involving pleasant stimuli foster increased attention and diminish the magnitude of the SR (Lang, Bradley, and Cuthbert 1990). The “aversive stimulus → negative affect (fear) → startle” relationship, which has been termed “fear potentiated startle,” has been consistently demonstrated in experiments with animal subjects. The converse of fear-potentiated startle is “joy attenuated startle” and refers to the diluting effect of pleasant stimuli (and resulting positive affect) on the magnitude of the SR.

Specific to acoustic stimuli, the ASR comprises fast contractions of skeletal and facial muscles, as well as closing of the eyes and acceleration of the heart rate in response to sudden high-intensity noise bursts (Koch 1999). In laboratory settings, the stimuli that are associated with the ASR are aversive and have been suggested to induce a state of fear or anxiety (Leaton and Cranney 1990). Therefore, the ASR itself might be considered an indicator of innate fear (Armbruster et al. 2014), with the ASR being modulated by cognitive processes such as attention, as well as emotional states triggered by the presentation of pleasant or unpleasant stimuli (Lang et al. 1990; Bradley and Lang 2000). As discussed in Chapter 2, Gautier and Cook (1997) studied the performance of

experimental subjects varying on characteristics that correlate with affective startle modulation (mainly, fearfulness) and cardiovascular reactivity (parental history of cardiovascular disease) on a cognitively challenging task with intermittent loud noises. In contrast to earlier findings (Cook et al. 1991; Stevenson and Cook 1994), fearful individuals did not show enhanced affective modulation of the ABR. Further analyses revealed that although unpleasant imagery did not potentiate startle in low-fear individuals, arousing imagery did.

In contrast to the findings of Gautier and Cook (1997), the majority of studies with aversive stimuli demonstrate increased levels of attention, particularly in anxious subjects (Mercado et al. 2006), and affective modulation has long been considered an established precondition for the ASR (Bradley, Cuthbert and Lang 1999). Grillon and Davis (1995) demonstrated that larger ASRs were potentiated by the impending threat of electric shock; that is, ASRs are associated with a startle-eliciting stimulus. In general, these researchers have found that affective modulation of ASRs in the context of anticipation (of negative outcomes) exerts a different and potentially stronger pattern of effects than those obtained from perception (of the startling stimulus) alone. Referred to as fear-potentiated startle, this magnified SR in the presence of actual or perceived threat can have consequences for cognition, including degradations in working memory or problem-solving ability (Martin et al. 2015). During the SR, the amygdala conducts an almost instantaneous appraisal of incoming stimuli in determining threat. Stimuli that are not determined to be a threat allow for a faster return to homeostasis. On the other hand, startle caused by a threat that either is or is perceived to be life-threatening can cause a full stress response, thereby leading to cognitive impairment that may affect the organism's actions or behavior in the situation (Martin et al. 2015).

In laboratory studies of animals, threatening cues have been shown to activate a neural circuit that is initiated when relevant sensory input activates the basolateral nuclei of the amygdala. Projections from this structure to other brain sites modulate a series of reflex behaviors that prepare the organism for overt defensive behavior (Bradley et al. 2001). The composite of responses initiated by this defense-motive circuit include freezing and active flight, fear bradycardia, increase in blood pressure, and potentiation of the SR. On the basis of physiological reactions measured during perception of unpleasant visual stimuli in the laboratory, researchers have proposed that human responses to sudden unpleasant stimuli can be ordered according to the degree to which they evoke defense system activation (Lang, Bradley, and Cuthbert 1997). The laboratory participant who is reacting to an unpleasant visual stimulus is in a state analogous to that of an animal freezing in response to a sudden predatory threat; that is, the individual is exhibiting a patterned cascade of reflex responses observed in animals at different stages of predator imminence.

Figure 3 shows a schematic depiction of the Defense Cascade Model (Bradley and Lang 2000; Lang 1995; Lang, Bradley, and Cuthbert 1997), which proposes that different

pronounced activation, oriented attention starts to give way to metabolic mobilization for active defense signaled by a change in the SR. That is, the SR is now potentiated (Vrana et al. 1988). Studies with human subjects have revealed that potentiation of the SR is augmented when phobic subjects come in contact with previously feared stimuli; conversely, research has demonstrated that the SR is inhibited when people view pleasant stimuli and that the greatest startle inhibition occurs for stimuli that are rated as the most positively arousing (Bradley, Cuthbert, and Lang 1999). In one particular study, a pleasant odorant (vanillin) was found to reduce SR amplitude compared with neutral air, and an unpleasant odorant (hydrogen sulfide) was found to enhance the SR amplitude relative to neutral air (Miltner et al. 1994). These differences seem to be directly related to the emotional valence of these odorants—the unpleasant odorant induced negative affect, whereas the pleasant odorant induced positive affect.

B. Individual Differences

1. Gender

Studies have reported conflicting results on the effects of gender on the SR. Some research suggests that women may have different reactions to unanticipated events than men (Hausmann et al. 2000; McCormick and Teillon 2001), with women reportedly demonstrating faster responses and higher lower limb activation in response to auditory stimuli (Kofler et al. 2001). One of the earliest efforts to examine gender differences in startle (Gautier and Cook 1997) revealed that startle potentiation by negative affect (fear and anger compared with pleasant relaxation) predicted pressor responses during a cognitively challenging task among men but not women. Post hoc analyses, however, suggested that this gender difference was primarily due to responses during anger-inducing imagery; SR during fear-inducing imagery predicted responses equally across genders.

Many studies have reported stronger fear-potentiated startle in women than in men (Armbruster et al. 2014; Bradley et al. 2001; Gard et al. 2007), but other studies have contradicted these results, reporting no demonstrated gender differences in SR (Dichter, Tomarken, and Baucom 2002; Hillman Rosengren, and Smith 2004). Investigations on the physiology of ASR parameters (e.g., latency, startle probability, muscular area affected by ASR) have revealed stronger overall ASR amplitude in women than in men but no differences in startle probability and latency as measured over the OOc, although the area over the muscle affected was larger for women (Kofler et al. 2001). More recently, Quevedo et al. (2010) reported higher response probabilities of ASR in women in a sample of healthy individuals; similarly, women showed an overall greater startle amplitude, as well as greater fear-potentiated startle (relative to men), in an emotional startle paradigm. Interpretation of gender differences in SR is complicated by several factors, one of which is gender differences in rated arousal occasioned by situational factors. Very few consistent

gender differences have emerged in the startle-modulation literature, although men have demonstrated greater potential for startle inhibition (Swerdlow et al. 1993).

These conflicting results for gender in humans are mirrored in animal studies using different strains of rats. Note, however, that unlike in humans, the SR in rodents is usually assessed by whole-body ballistic ground reaction forces, which have been found to be dependent on body weight, which in turn is usually higher in male rats (Blaszczyk and Tajchert 1996). One possible reason for the inconsistencies regarding gender differences in SR for humans might be lack of control for menstrual cycle phase or for the use of hormonal contraceptives. A small number of studies on the effects of menstrual cycle on the SR have revealed robust gender differences in magnitude of the SR as a function of luteal phase. Specifically, it has been demonstrated that compared to young men, premenopausal women show smaller SR (Kumari, Aasen, Papadopoulos, Bojang, Poon, Halari and Cleare 2008); there are no demonstrated differences in SR between postmenopausal women and men of similar age.

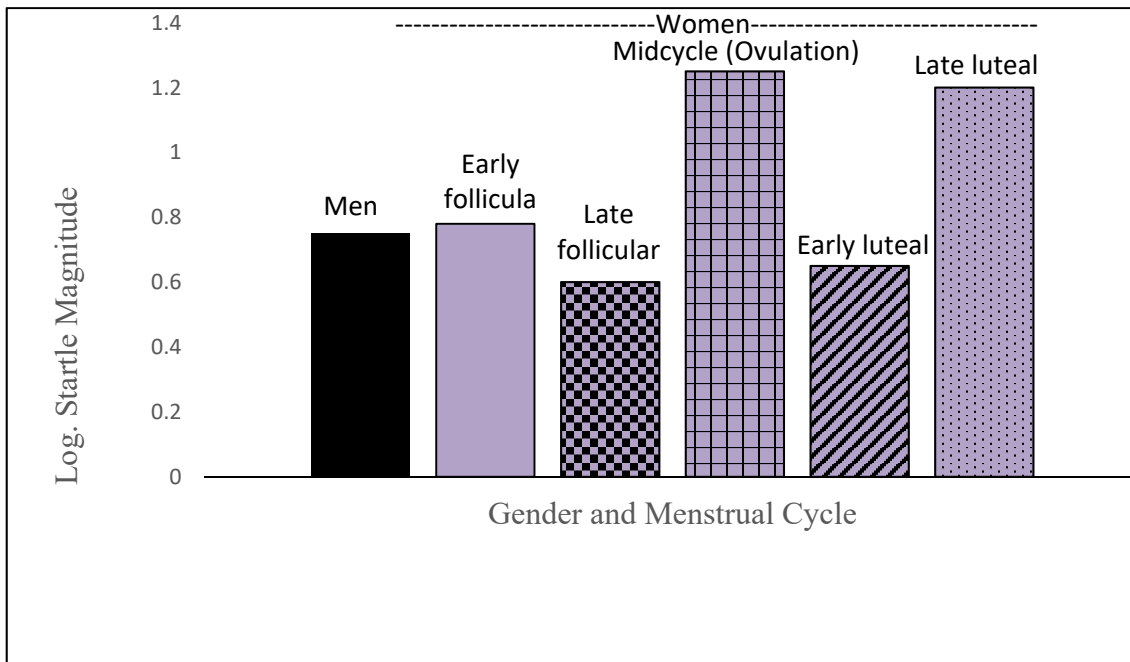
In one of the largest studies conducted to date on the effects of menstrual cycle on the SR, Armbruster and colleagues (2014) investigated (1) whether men ($n = 109$) and women ($n = 114$) differ in the SR and its affective modulation, (2) the size of such a potential effect, and (3) the possible influence of menstrual cycle phase on ASR. Calculations of the menstrual cycle phase were based on information given by the participant regarding (1) the first day of the last period, (2) the length of the menstrual cycle, and (3) its regularity. Menstrual cycle phases were determined as follows:

- Midcycle (ovulation) included days 13–15 before the next menstruation.
- Early luteal phase was defined as the 7 days following midcycle (days 16–22 for a 28-day cycle).
- Late luteal phase comprised the 6 days immediately preceding the estimated onset of the next menstrual cycle (days 23–28 for a 28-days cycle).
- Follicular phase consisted of the days before the estimated midcycle (days 1–12 for a 28-day cycle), with the first half designated as the early follicular phase and the second half as the late follicular phase.

Figure 4 presents the results of the study.

As can be seen in Figure 4, startle magnitudes differed significantly within the female sample over the course of the menstrual cycle, with larger startle magnitudes during ovulation and the late luteal phases. These were the only times when startle magnitudes in women were significantly larger than in men; there were no observed gender differences during the early and late follicular and early luteal phases. In general, hormonal changes over the course of the menstrual cycle affect nearly all systems of the female body, including physical, cognitive, and affective functions. The extensive distribution of steroid

receptors throughout different tissues results in widespread and varied effects of steroids on physiological and motor functions, many of which are context-dependent (Armbruster et al. 2014). In addition to regulating mood in the brain, changing progesterone levels over the course of the menstrual cycle have been found to result in elevated ASR, especially in animal models (Gulinello, Orman, and Smith 2003). In addition to progesterone, other modulating factors include changes in serotonin, norepinephrine, and dopamine levels during the premenstrual phase and changes in the concentration of gonadal hormones and their metabolites.



Source: Adapted from Ambruster et al. (2014).

Figure 4. Effects of Menstrual Cycle on the SR in Human Female Subjects

In the study by Ambruster and colleagues (2014), self-report data showed a general pattern of reduced anxiety and neuroticism during midcycle compared with other cycle phases. There were significant differences between the late luteal phase—marked by higher anxiety scores—and ovulation, but there were no significant differences between late luteal and early follicular phases. Thus, while higher anxiety scores during the late luteal phase matched larger startle magnitudes during that part of the cycle, this does not appear to apply to the ovulation phase with equally high startle magnitudes. Most important, different mechanisms underlying larger SRs during ovulation and the late luteal phase should be taken into consideration when drawing conclusions regarding the effect of gender on the SR. While the late luteal phase has been characterized by higher anxiety, depression and irritability, the midcycle phase has been associated with heightened well-being and self-esteem (Farage, Osborn, and MacLean, 2008). The ovulation phase is characterized by a

surge in estrogen that has been associated not only with less negative emotionality but also with positive effects on hearing (Hultcrantz, Simonska, and Stenberg 2006). Women have not only been found to have better hearing than men in general but also to demonstrate increased hearing sensitivity during ovulation. This might be a contributing factor to larger startle magnitudes to *acoustic* stimuli during the ovulation phase. In addition, positive effects of estrogen on general arousal of the central nervous system might also have potentially contributed to startle magnitudes.

Note that the size of the observed effect on startle magnitude in the study by Ambruster and colleagues (2014) during the ovulation and late luteal phases was only small to moderate (effect size = 0.105). Nevertheless, this finding points to the necessity of considering information about menstrual cycle phase when evaluating the SR in women and for drawing conclusions about observed differences between men and women in the SR. Although physiological parameters are not always consistent with accompanying self-reports, it can be summarized that a general shift toward heightened negative emotionality at the end of the menstrual cycle makes women more susceptible to startle relative to men during specific stages in the menstrual cycle. Further studies on the SR should also investigate the potential effects of hormonal contraceptives on startle magnitudes and latency.

2. Age

The ASR is considered a brainstem reflex in response to unexpected loud stimuli. One of the few studies to date that has examined age differences in SR (Kofler, Müller, Reggiani, et al. 2001) investigated the responses of 54 healthy nonmedicated adult volunteers (with no complaints of excessive startle) in three age groups: under 30, 30–50, and above 50. After ascertaining normal bilateral hearing thresholds, ASRs were elicited by binaurally presented tone bursts that differed randomly in tonal frequency and intensity. Electromyographic recordings were simultaneously obtained following each stimulus from the masseter, OOc, sternocleidomastoid, biceps brachii, abductor, rectus femoris, tibialis anterior, and soleus muscles. All subjects displayed similar ASR patterns with subtle but consistent age differences. ASRs were most prevalent in facial and neck muscles and more frequent in upper than in lower extremities. Subjects below age 30 had significantly lower ASR probability than any of the older subjects. Separate analysis of individual muscles and muscle groups revealed that significant age-related differences were limited to extremity muscles. ASR area did not differ significantly in all the muscles combined. There was, however, a tendency toward greater response latencies (that is, slower onset of the SR) with advancing age in all individual muscles, reaching statistical significance in the masseter and abductor muscles.

In the above study, the researchers concluded that subclinical age-dependent slowing of peripheral nerve conduction may have contributed to increased ASR latencies in older

subjects; median ASR latency differences between age groups amount to 94 milliseconds in extremity muscles and 18 milliseconds in facial and neck muscles. Interestingly, electromyography responses to sudden free fall have been found to resemble ASRs, with responses being delayed in older subjects in a manner similar to ASR latencies. This suggests that age-dependent slowing of central reticular processing rather than peripheral nerve slowing might be an alternative explanation for age-related latencies in ASRs. These findings were not attributable to differences in perceptual ability, because the diminished ASR was still found among elderly adults even with sounds that were easier for them to perceive (Ford et al. 1995).

Another study comparing ABRs of participants varying in age from 20 to 60 revealed that the youngest participants (within the 20–29 age bracket) had significantly higher startle magnitudes than the oldest participants (within the 50–60 age bracket) (Ludewig et al. 2003). The results suggested a trend toward a decrease in startle magnitudes with advancing age, with the most notable decline in adults aged 50 and older. In contrast to increasing ASR latencies and decrease in ASR magnitudes, the results of the study by Kofler and colleagues (2008) suggest a trend toward ASR disinhibition in older subjects. That is, an increase in ASR probability was observed with advancing age, likely because older subjects may exert less cortical inhibitory influence on complex brainstem reflexes than younger subjects, despite any clinical signs of cortical dysfunction (Jacobs and Grossman 1980). In summary, advancing age has been associated with an increase in latencies (or slowing down) of the SR and a reduction in SR magnitudes, but also with a potential increase in the probability of the SR occurring.

3. Personality

Research has also investigated individual differences in the SR related to personality factors. One notable study compared the magnitude of ABR for participants who differed along the dimensions of harm avoidance, reward dependence, novelty seeking, extraversion, neuroticism, and psychoticism (Corr et al. 1995). ABR was measured during affective picture viewing, in which participants viewed pleasant, unpleasant, or neutral stimuli. A potentiated SR was found for participants scoring high in harm avoidance, a measure characterized by the tendency to avoid punishment and novelty and exhibit intense responses to aversive stimuli. Other research has suggested relationships between the SR and extraversion, with extraversion being correlated with greater startle magnitudes; typically, differences in SR reactivity between individuals classified as extraverted or introverted tend to be influenced by attention (Blumenthal 2001). Certain personality characteristics such as harm avoidance, anxiety, and fearfulness have been linked to differences in SR reactivity, with individuals scoring higher on these dimensions generally exhibiting increased startle and individuals scoring lower on these dimensions showing decreased startle (Justus and Finn 2007).

The relation between psychopathic tendencies, such as fearless dominance, and SR reactivity has also been established, with the results suggesting that individuals exhibiting certain psychopathic tendencies show deficient fear-potentiated SR (Benning, Patrick, and Iacono 2005; Ebner-Priemer et al. 2005) and almost no propensity to startle. Fear is directly relevant to the SR, because the SR can be conceptualized as a defensive mechanism in response to a potential threat. Therefore, individual differences in fearfulness may be associated with startle reactivity. Vaidyanathan, Patrick, and Bernat (2009) proposed that fear and fearlessness may be ends of a spectrum that could be related to the SR. In their study, participants filled out various measures of fear and fearlessness, including the Emotionality-Activity-Sociability-Fear scale, the Fear Survey Schedule, the Harm Avoidance subscale of the Tridimensional Personality Questionnaire, the Thrill and Adventure Seeking scale of the Sensation Seeking Scale, and the Psychopathic Personality Inventory. Participants viewed positive, negative, or neutral images while data on ABR were collected. The results indicated that participants scoring higher in measures of fearfulness exhibited ABR that were highest in magnitude for pictures of negative, threatening images, whereas participants scoring higher in measures of fearlessness exhibited attenuated ABR for the same negative images. These findings contribute to the literature suggesting that startle potentiation-related deficiencies in individuals with psychopathic tendencies is associated with reduced amygdala reactivity.

4. Startle Modification

The SR is a protective response elicited by a sudden and intense stimulus. Although the SR is primarily a reflexive response that can be reliably elicited, the SR is not stereotypic in that it can be modulated by emotions such as fear (fear potentiated startle) and joy (joy attenuated startle), by non-associative learning processes such as habituation and sensitization, and by other sensory stimuli through sensory gating processes. Interestingly, the ability to modulate the SR makes the SR an excellent tool for assessing emotions, learning, and sensory gating in humans. More important, the potential to modulate or “change” the occurrence, magnitude, and duration of the SR presents opportunities as well as challenges to the study and application of the SR to specific real-world scenarios. In the sections below, we discuss four ways that the SR is altered as a function of the unique characteristics of the environment in which the response is elicited.

A. Startle Habituation and Sensitization

The primary pathway mediating the SR is very short and well described, qualifying startle also as an excellent model for studying the underlying mechanisms for behavioral plasticity on a cellular/molecular level (Valsamis and Schmid 2011). Review of the literature reveals two distinct and independent processes that govern behavioral responses to repetitive stimulation from startle-eliciting stimuli (Haerich 1997): (1) an incremental process called sensitization (i.e., becoming more prone to startle) and (2) a decremental process called habituation (i.e., becoming less prone to startle). These processes are discussed below.

The dual-process theory of habituation (Groves and Thompson 1970) proposes that habituation occurs within specific stimulus-response pathways producing a decrement in responding, whereas sensitization acts more generally, incrementing responding by producing a state change in the organism. As both mechanisms are proposed to be simultaneously active, the resultant change in behavior in response to a repeated startle probe (either incremental or decremental) represents a summation of their respective influences (Haerich 1997). Sensitization, which generally occurs during early repeated exposures to a stimulus, is responsible for a transitory increase in SR amplitude. The process of sensitization involves stimulus-induced changes in the level of arousal and is dependent on the aversiveness of the stimulus. Typically, sensitization occurs when the stimuli are noxious or fearful and the subject is exposed to it in continuous stretches (as opposed to short, discrete bursts). However, sensitization gradually wanes because the salience of the stimulus decreases with repeated presentation, eventually giving way to habituation.

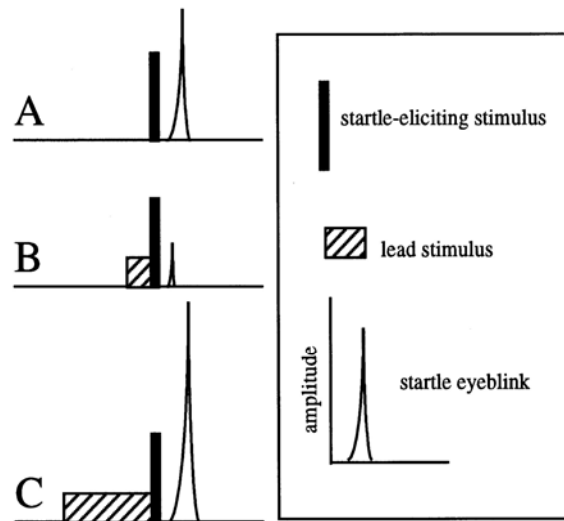
Habituation is the result of several repeated exposures of the subject to a startle-inducing stimulus; specifically, repeated exposures over time decreases SR amplitudes. Like most polysynaptic reflexes, stimulus repetition leads to rapid habituation of the SR. In the case of non-aversive stimuli, habituation can occur as quickly as the subject's second exposure to the stimulus. Habituation is a form of non-associative learning; it can also be viewed as a form of sensory filtering, since it reduces the organism's response to a nonthreatening stimulus. This frequently poses challenges to the neurophysiological evaluation of these responses, which may be overcome at least partly by applying stimuli separated by a very long time or by using auditory stimuli of different tones and frequencies. Habituation within a short time frame (such as within a testing session in an experimental setting) is called short-term habituation and is reversible within a period of several minutes without stimulation. Habituation between testing sessions is called long-term habituation and may last for several days, months, or longer, depending on the strength of habituation. Habituation is stimulus specific; that is, habituation to one stimulus does not (in the majority of cases) transfer to other stimuli even if there are similarities in modality and other characteristics. In their classic definition of habituation, Thompson and Spencer (1966) provide nine parametric characteristics, of which three specify aspects of stimulus timing and presentation that can modify the magnitude or the rate of habituation. Specifically, habituation may be reduced by: (1) high-intensity stimuli, (2) limiting previous exposure to the stimulus, and (3) decreasing the frequency with which the stimulus is presented.

B. Startle Inhibition and Facilitation

Over the last few decades, researchers have developed techniques external to the organism that can be applied to successfully modify (facilitate or inhibit) the SR. These techniques, which are known as inhibition and facilitation, must not be confused with habituation and sensitization, which are naturally occurring responses of an organism to repeated contact with a startle probe. Among the many physiological responses to startling stimuli, the eyeblink is reportedly one of the most effective indicators of an innate neural response to the startle probe. To examine the eyeblink response in a typical experimental setting, the startle-eyeblink-modification paradigm involves presenting a series of trials in which a startle-eliciting stimulus (e.g., sudden loud noise) occurs in the absence of any other stimulus (Figure 5, panel A), intermixed with trials in which the startle-eliciting stimulus closely follows a non-startling stimulus called a "lead stimulus" or "prepulse" (Figure 5, panels B and C) (Filion, Dawson, and Schell 1998). In Figure 5, the timing of the stimulus (in milliseconds) is represented on the *x*-axis, and the amplitude of the SR is represented on the *y*-axis.

The interval between the onset of the lead stimulus and the startle-eliciting stimulus is called the "lead interval." The dependent measure most commonly reported in human

startle research is a change or percent-change score reflecting the difference in size of the ABR elicited under these conditions. Startle amplitude inhibition refers to cases in which the ABR is smaller in the lead stimulation condition than in the baseline condition (Figure 5, panel B vs. A); startle amplitude facilitation refers to cases in which the ABR is larger in the lead stimulus condition than in the baseline condition (Figure 5, panel C vs. A), and startle latency facilitation refers to cases in which the latency of the startle reflex is shorter in the lead stimulus condition than in the baseline condition (Figure 5, panel C vs. B).



Source: Adapted from Filion, Dawson, and Shell 1998.

Figure 5. Illustration of Startle Inhibition and Facilitation.

There are three main types of lead-stimulation-startle-modification-effects observed at short lead intervals such as described above. The first effect is inhibition of startle amplitude (or suppression of the magnitude of startle), an effect also referred to in the literature as prepulse inhibition (PPI). Amplitude inhibition is produced by a wide range of lead stimuli and occurs within a lead interval range of approximately 30–500 milliseconds. The startle amplitude inhibition effect is quite robust, typically in the range of 50%–80% inhibition, and is quite reliable, occurring in 90%–100% of normal adult participants who show reliable SRs. Startle inhibition can be produced by visual, acoustic, olfactory, and vibrotactile lead stimuli, and their inhibitory effect is seen even when the modalities of the lead and startle stimuli differ. Moreover, an increase or a decrease in stimulus energy can also serve as an effective lead stimulus.

The second lead-stimulation-startle-modification effect is an amplitude facilitation effect or magnification of the startle effect (panels C vs. A in Figure 5), which has been shown to occur across sensory modalities for: (1) acoustic startle with vibrotactile lead stimuli at 25-, 50-, and 100-millisecond lead intervals (e.g., Flaten and Blumenthal 1999); (2) for electrically elicited startle with acoustic lead stimuli at a lead interval of 10

milliseconds (e.g., Boelhouwer, Teurlings, and Bruina 1991); and (3) for acoustic startle with visual and electrocutaneous lead stimuli at lead intervals of 30 and 60 milliseconds (Graham 1980). To date, the main focus of research on this amplitude-facilitation effect has been on its physiological significance; a smaller amount of research has focused on its psychological significance.

The third startle modification effect is a latency-facilitation effect, or, decrease in the speed of the SR. Latency facilitation is observed at lead intervals of approximately 100 milliseconds or less, regardless of the modalities of the lead and startle stimuli (e.g., Graham 1975; Graham and Murray 1977; Braff et al. 1978; Blumenthal and Gescheider 1987; Blumenthal and Tolomeo 1989). The psychological significance of this latency-facilitation effect has also received relatively little attention; the effect has been reported inconsistently (many reports do not include the latency measure at all) and is often reported as merely co-occurring with amplitude-facilitation effects. Both amplitude facilitation and latency facilitation have been grouped very broadly under the umbrella of prepulse facilitation (PPF), although in most research PPF has been treated as being synonymous with amplitude facilitation alone.

A number of variables affect PPI and PPF, with certain factors determining whether inhibition or facilitation is more likely to occur. The time course is one consideration, with the onset and duration of the prepulse stimulus influencing the type of startle attenuation or intensification that may occur. Certain characteristics of the stimulus itself, including stimulus intensity and the modality of the stimulus, are also influential. The degree to which the prepulse or lead stimulus and the actual startle-inducing stimulus are related may also have an effect on whether PPI or PPF occur, based on the match or mismatch between these modalities.

Two temporal variables that can affect PPI and PPF are the lead time and stimulus duration. Lead time, which refers to the amount of time by which the prepulse or lead stimulus precedes the startle-inducing stimulus, can influence the magnitude of the startle response in either direction. Research suggests that certain lead times can induce optimal PPF or PPI, with the results indicating that generally, PPF occurs for lead times that are shorter than 20 milliseconds or longer than 500 milliseconds (Hsieh, Swerdlow, and Braff 2005). For lead times in between, PPI is likely to occur, generally for lead times ranging between 30 and 500 milliseconds (Hsieh, Swerdlow, and Braff 2005; Plappert, Pilz, and Schnitzler 2004). Maximal PPI may also be influenced by prepulse modalities, with acoustic lead stimuli demonstrating maximal PPI at 100–150 milliseconds, tactile lead stimuli causing maximal PPI at 150–250 milliseconds, and visual lead stimuli causing maximal PPI at around 240 milliseconds (Neumann, Lipp, and Pretorius 2004). In summary, PPF can be maximized by introducing a lead stimulus before the startle stimulus at very short lead times of less than 20 milliseconds or at very long lead times of more than 500 milliseconds.

The duration and characteristics of the prepulse can also have an influence on the response type (Hoffman and Fleshler 1963). Long-duration prepulses have been associated with PPI—as the stimulus duration of the prepulse increases, the likelihood of prepulse inhibition increases as well (Schmajuk and Larrauri 2005). Background noise can also affect the SR; increasing the background noise tends to result in an increase in the amplitude of the SR (Hoffman and Fleshler 1963; Schmajuk and Larrauri 2005). However, breaking up the background noise from a continuous sound into a series of pulses can lead to suppression of the SR, as can an overall reduction in the background noise. In terms of stimulus intensity, maximal PPI has been found for stronger prepulses; as prepulse intensity decreases, the amplitude and magnitude of SR has been shown to decrease as well (Blumenthal 1996; Ison et al. 1997). Conversely, maximal PPF has been found for relatively weak prepulses (Ison et al. 1997). Prepulse facilitation has been found to occur more reliably when the prepulse stimulus and the startle-inducing stimulus are from different sensory modalities, although studies suggest this is more likely at very short lead intervals (Neumann, Lipp, and Pretorius 2004). The quality of the prepulse stimulus can also influence the SR, with auditory white noise prepulses being demonstrated as more effective at magnifying the amplitude of startle compared with pure auditory tones (Stoddart, Noonan, and Martin-Iverson 2008). Future research should continue examining all the various factors that influence PPI or PPF in greater detail to determine the variables and the context within which the SR can be magnified or inhibited.

5. Findings and Recommendations

The SR, which is typically an organism's first response when exposed to an unexpected stimulus, is the fastest generalized motor reaction of humans and animals caused by the involuntary activation of the motor tracts originating in the brainstem. The SR is an extensively studied phenomenon in neuropsychology and cognitive psychology, with particular focus on ways to modulate or alter the SR in different situations. The SR is relevant to the design of nonlethal weapons such as flashbang grenades because a large part of the effectiveness of flashbangs may stem from the suddenness or unexpectedness of the stimulus, which potentiates an immediate and reflex aversive reaction on the part of the human. This report describes the psychological, psychological, and other components of the SR and discusses ways to modify it. This discussion is relevant to the design of flashbangs since it can suggest enhancements to their effectiveness using cognitive techniques that do not raise the risk of significant injury.

A. Findings

First, the SR, which is commonly believed to be an entirely involuntary reflex reaction, comprises both an involuntary and a voluntary component. The first component of the SR is the well-known rapid involuntary reflex contraction of the face and limb muscles accompanied by a combination of eye closure, facial grimacing, neck flexion, and arm abduction or flexion. Immediately following the early, involuntary, rapid reflex, however, is a less studied second component of the SR that is embodied in voluntary muscular and motor activity. In the context of flashbangs, protective actions (such as lifting arms to cover one's face or attempts to remove oneself from a situation) are voluntary actions that receive cues from and immediately follow involuntary reflex actions such as the eyeblink. The voluntary motor component is significant because research has demonstrated the possibility of asymmetric muscle activation in response to startle; that is, humans tend to exhibit faster voluntary head rotation toward the dominant side of the body and greater flexion of the dominant arm to protect or defend against the startling stimulus. This provides important insights into how humans may physically orient themselves immediately following the experience of a startle probe; consequently, broad generalizations regarding the effects of a startling stimulus on human responses may be made given the preponderance of right-handedness in any given population. Therefore, efforts to improve the design and physical orientation of flashbangs must focus on both the voluntary, as well as the involuntary, components of the SR to achieve maximum effectiveness.

Second, affect or emotion modulates the SR in a substantial way across a variety of situations. Empirical research has repeatedly demonstrated that anticipation of negative outcomes exerts a potentially stronger pattern of effects on the SR than the effects obtained from mere perception of the startling stimulus alone. During the SR, the amygdala conducts an almost instantaneous appraisal of incoming stimuli in determining threat. Startle caused by a threat that either is or is perceived to be threatening to life or limb can cause a full-stress response, leading to cognitive impairment that may significantly affect the organism's actions or behavior in the situation. Referred to as fear-potentiated startle, this magnified SR in the presence of actual or perceived threat can have serious consequences for cognition, including degradations in working memory or problem-solving ability. Fear-potentiated startle has strong effects on motoric activities associated with the fight/flight complex—and even on physiological systems that could in some instances cause the organism to “freeze” or have a cardiac arrest. This impact of fear-potentiated startle should be considered when designing and deploying flashbangs for two reasons: (1) the risk of significant injury from perceived threat alone can have severe consequences due to its ability to generate damaging negative affect, and (2) it might be possible to enhance the effectiveness of flashbangs by manipulating affect alone without increasing the physical intensity of flashbangs.

Third, research has documented widespread individual differences in human vulnerability to startle-inducing stimuli. Although the literature on the role of gender on the startle effect abounds in conflicting results, one interesting finding is the relationship between hearing and startle potentiation. Research has revealed that women on average tend to have superior hearing ability compared with men, which also tends to be enhanced during some phases of the menstrual cycle; this makes women more likely to demonstrate larger startle magnitudes in response to acoustic stimuli relative to men. Similarly, younger adults (below age 30) demonstrate greater startle magnitudes and shorter latencies (quicker response times) than older adults, although the probability of being startled tends to be higher in older adults. These findings have implications for situations where flashbangs are deployed for specific segments of the population that might be segregated by gender or age; consideration of specific age- or gender-related reactions to startle-inducing stimuli would be beneficial in enhancing the effectiveness of the flashbang in such circumstances.

B. Recommendations

In this report, we discussed the neural, sensorimotor, affective, cognitive and individual difference variables that characterize human responses to flashbangs. To use this information to develop more effective flashbangs, there is the need for an integrative model that captures how the effectiveness of flashbangs can be improved by incorporating these variables into startle-modification techniques. The literature on startle modification has demonstrated that there are methods to systematically magnify or inhibit the startle effect

via cognitive techniques that facilitate (or improve) the amplitude (size) and the latency (speed) of the SR and minimize the probability of startle inhibition (suppression). These techniques, known as PPI and PPF, typically involve pairing secondary stimuli (auditory, visual, tactile) with the primary stimulus (in this case, the flashbang) at predetermined intervals to elicit specific cognitive effects. The ability to modify the SR is relevant to the effective deployment of flashbangs because it allows for the opportunity to enhance the human effectiveness of the flashbang without necessarily increasing the intensity of the flashbang (and consequently raising the risk of significant injury). Therefore, we recommend that future efforts be focused on examining ways to enhance humans' neurological, physiological, and cognitive responses to flashbangs through research on startle-modification techniques. Future efforts must also focus on the interplay between the visual (the "flash") and auditory (the "bang") components of the flashbang to fully understand the relative effects of the flash and the bang as well their timing on human performance.

References

- Abrahams, V. C., S. M. Hilton, and A. Zbrożyna. 1960. "Active Muscle Vasodilatation Produced by Stimulation of the Brain Stem: Its Significance in the Defense Reaction." *The Journal of Physiology* 154:491–513.
- Armbruster, D., A. Strobel, C. Kirschbaum, and B. Brocke. 2014. "The Impact of Sex and Menstrual Cycle on the Acoustic Startle Response." *Behavioural Brain Research* 274:326–33.
- Benning, S. D., C. J. Patrick, and W. G. Iacono. 2005. "Psychopathy, Startle Blink Modulation, and Electrodermal Reactivity in Twin Men." *Psychophysiology* 42:753–62.
- Bisdorff, A. R., A. M. Bronstein, and M. A. Gresty. 1994. "Responses in Neck and Facial Muscles to Sudden Free Fall and a Startling Auditory Stimulus." *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section* 93:409–16.
- Blaszczyk, J., and K. Tajchert. 1996. "Sex and Strain Differences of Acoustic Startle Reaction Development in Adolescent Albino Wistar and Hooded Rats." *Acta Neurobiologiae Experimentalis* 56:919–26.
- Blumenthal, T. D. 1996. "Inhibition of the Human Startle Response is Affected by Both Prepulse Intensity and Eliciting Stimulus Intensity." *Biological Psychology* 44:85–104.
- Blumenthal, T. D. 2001. "Extraversion, Attention, and Startle Response Reactivity." *Personality and Individual Differences* 31:495–503.
- Blumenthal, T. D., and G. A. Gescheider. 1987. "Modification of the Acoustic Startle Reflex by a Tactile Prepulse: The Effects of Stimulus Onset Asynchrony and Prepulse Intensity." *Psychophysiology* 24:320–27.
- Blumenthal, T. D., and E. A. Tolomeo. 1989. "Bidirectional Influences of Vibrotactile Stimuli on Modification of the Human Acoustic Startle Reflex." *Psychobiology* 17:315–22.
- Boelhouwer, A. J. W., R. J. M. A. Teurlings, and C. H. M. Brunia. 1991. "The Effect of an Acoustic Warning Stimulus upon the Electrically Elicited Blink Reflex in Humans." *Psychophysiology* 28:133–39.
- Bradley, M. M., and P. J. Lang. 2000. "Affective Reactions to Acoustic Stimuli." *Psychophysiology* 37:204–15.
- Bradley, M. M., M. Codispoti, B. N. Cuthbert, and P. J. Lang. 2001. "Emotion and Motivation I: Defensive and Appetitive Reactions in Picture Processing." *Emotion* 1 (3): 276–98.

- Bradley, M. M., B. N. Cuthbert, and P. J. Lang. 1999. *Startle Modification: Implications for Neuroscience, Cognitive Science, and Clinical Science*. Cambridge, U.K: Cambridge University Press.
- Braff, D., C. Stone, E. Callaway, M. Geyer, I. Glick, and L. Bali. 1978. "Prestimulus Effects on Human Startle Reflex in Normals and Schizophrenics." *Psychophysiology* 15:339–43.
- Brod, J., V. Fenci, Z. Hejl, J. Jirka, and M. Ulrych. 1962. "General and Regional Hemodynamic Pattern Underlying Essential Hypertension." *Clinical Science* 23:339–49.
- Brown, P., J. C. Rothwell, P. D. Thompson, T. C. Britton, B. L. Day, and C. D. Marsden. 1991a. New Observations on the Normal Auditory Startle Reflex in Man. *Brain* 114:1891–1902.
- Brown, P., J. C. Rothwell, P. D. Thompson, T. C. Britton, B. L. Day, and C. D. Marsden. 1991b. "The Hyperekplexias and Their Relationship to the Normal Startle Reflex." *Brain* 114:1903–28.
- Cacioppo, J. T., and G. G. Berntson. 1994. "Relationship between Attitudes and Evaluative Space: A Critical Review, with Emphasis on the Separability of Positive and Negative Substrates." *Psychological Bulletin* 115:401–23.
- Cadenhead, K. S., N. R. Swerdlow, K. M. Shafer, M. Diaz, and D. L. Braff. 2000. "Modulation of the Startle Response and Startle Laterality in Relatives of Schizophrenic Patients and in Subjects with Schizotypal Personality Disorder: Evidence of Inhibitory Deficits." *American Journal of Psychiatry* 157:1660–68.
- Carlsen, A. N., R. Chua, J. T. Inglis, D. J. Sanderson, and I. M. Franks. 2004a. "Can Prepared Responses be Stored Subcortically?" *Experimental Brain Research* 159:301–9.
- Carlsen, A. N., R. Chua, J. T. Inglis, D. J. Sanderson, and I. M. Franks. 2004b. "Prepared Movements Are Elicited Early by Startle." *Journal of Motor Behavior* 36:253–64.
- Castellote, J. M., H. Kumru, A. Queralt, and J. Valls-Solé. 2007. "A Startle Speeds up the Execution of Externally Guided Saccades." *Experimental Brain Research* 177:129–36.
- Chabot, C. C., and D. H. Taylor. 1992a. "Circadian Modulation of the Rat Acoustic Startle Response." *Behavioral Neuroscience* 106:846–52.
- Chabot, C. C., and D. H. Taylor. 1992b. "Daily Rhythmicity of the Rat Acoustic Startle Response." *Physiology & Behavior* 51:885–89.
- Cook, E. W. III, T. L. Davis, L. W. Hawk, E. L. Spence, and C. H. Gautier. 1992. "Fearfulness and Startle Potentiation during Aversive Visual Stimuli." *Psychophysiology* 29:633–45.
- Cook, E. W. III, L. W. Hawk, T. L. Davis, and V. E. Stevenson. 1991. "Affective Individual Differences and Startle Reflex Modulation." *Journal of Abnormal Psychology* 100:5–13.

- Cook, E.W., III, R. L. Hodes, and P. J. Lang, et al. 1986. "Preparedness and Phobia: Effects of Stimulus Content on Human Visceral Conditioning." *Journal of Abnormal Psychology* 95:195–207.
- Corr, P. J., G. D. Wilson, M. Fotiadou, V. Kumari, N. S. Gray, S. Checkley, and J. A. Gray. 1995. "Personality and Affective Modulation of the Startle Reflex." *Personality and Individual Differences* 19:543–53.
- Davis, M. 1996. "Differential Roles of the Amygdala and Bed Nucleus of the Stria Terminalis in Conditioned Fear and Startle Enhanced by Corticotropin-Releasing Hormone." In *Perception, Memory and Emotion*, edited by T. Ono, B. L. McNaughton, S. Molotchnikoff, E. T. Rolls, and H. Nishijo, 525–48. Oxford, U.K.: Elsevier Ltd.
- Davis, M. 1997. "The Neurophysiological Basis of Acoustic Startle Modulation: Research on Fear Motivation and Sensory Gating." In *Attention and Orienting: Sensory and Motivational Processes*, edited by P. J. Lang, R. F. Simons, and M. T. Balaban, 69–96. Mahwah, NJ: Lawrence Erlbaum Associates.
- Davis, M., D. S. Gendelman, M. D. Tischler, and P. M. Gendelman. 1982. "A Primary Acoustic Startle Circuit: Lesion and Stimulation Studies." *Journal of Neuroscience* 2:791–805.
- DeAngelis, A. 2012. "Interaction Effects of the Startle Response and Hormonal Changes on Knee Stiffness." Thesis. University of Delaware.
- Dichter, G. S., A. J. Tomarken, and B. R. Baucom. 2002. "Startle Modulation before, during and after Exposure to Emotional Stimuli." *International Journal of Psychophysiology* 43:191–6.
- Eaton, R.C, ed. 1984. *Neural Mechanisms of Startle Behavior*. New York: Plenum Press.
- Ebner-Priemer, U. W., S. Badeck, C. Beckmann, A. Wagner, B. Feige, I. Weiss, K. Lieb, and M. Bohus. 2005. "Affective Dysregulation and Dissociative Experience in Female Patients with Borderline Personality Disorder: A Startle Response Study." *Journal of Psychiatric Research* 39:85–92.
- Farage, M. A., T. W. Osborn, and A. B. MacLean. 2008. "Cognitive, Sensory, and Emotional Changes Associated with the Menstrual Cycle: A Review." *Archives of Gynecology and Obstetrics* 278:299–308.
- Fernández, M. C., and J. Vila. 1989. "Sympathetic-Parasympathetic Mediation of the Cardiac Defense Response in Humans." *Biological Psychology* 28:123–33.
- Filion, D. L., M. E. Dawson, and A. M. Schell. 1998. "The Psychological Significance of Human Startle Eyeblink Modification: A Review." *Biological Psychology* 47:1–43.
- Flaten, M. A., and T. D. Blumenthal. 1999. "Caffeine-Associated Stimuli Elicit Conditioned Responses: An Experimental Model of the Placebo Effect." *Psychopharmacology* 145:105–12.
- Ford, J. M., W. T. Roth, B. G. Isaacks, P. M. White, S. H. Hood, and A. Pfefferbaum. 1995. "Elderly Men and Women Are Less Responsive to Startling Noises: N1, P3 and Blink Evidence." *Biological Psychology* 39:57–80.

- Frankland, P. W., and M. R. Ralph. 1995. "Circadian Modulation in the Rat Acoustic Startle Circuit." *Behavioral Neuroscience* 109:43–48.
- Freeman, M. R., and B. Wyke. 1966. "Articular Contributions to Limb Reflexes: The Effects of Partial Neurectomy of the Knee-Joint Postural Reflexes." *British Journal of Surgery* 53:61–68.
- Furubayashi, T., Y. Ugawa, Y. Terao, R. Hanajima, K. Sakai, K. Machii, H. Mochizuki, Y. Shiio, H. Uesugi, H. Enomoto, and I. Kanazawa. 2000. "The Human Hand Motor Area Is Transiently Suppressed by an Unexpected Auditory Stimulus." *Clinical Neurophysiology* 111:178–83.
- Gard, D. E., M. G. Gard, N. Mehta, A. M. Kring, and C. J. Patrick. 2007. "Impact of Motivational Salience on Affect Modulated Startle at Early and Late Probe Times." *International Journal of Psychophysiology* 66:266–70.
- Gautier, C. H., and E. W. Cook. 1997. "Relationships between Startle and Cardiovascular Reactivity." *Psychophysiology* 34:87–96.
- Gogan, P. 1970. "The Startle and Orienting Reactions in Man. A Study of Their Characteristics and Habituation." *Brain Research* 18:117–35.
- Gokin, A. P., and M. V. Karpukhina. 1985. "Reticular Structures of the Cat Brain Participating in Startle Reflexes in Response to Somatic Stimuli of Different Modalities." *Neurophysiology* 17:380–90.
- Graham, F. K. 1975. "The More or Less Startling Effects of Weak Prestimulation." *Psychophysiology* 12:238–48.
- Graham, F. K. 1979. "Distinguishing among Orienting, Defense, and Startle Reflexes." In *The Orienting Reflex in Humans*, edited by H. D. Kimmel, E. H. van Olst, and J. F. Orlebeke, 137–67. Hillsdale, NJ: Erlbaum.
- Graham, F. K. 1980. "Control of Reflex Blink Excitability." In *Neural Mechanisms of Goal-directed Behavior and Learning*, edited by R. F. Thompson, L. H. Hicks, and V. B. Shuyrkov, 511–19. New York, NY: Academic Press.
- Graham, F. K., and G. M. Murray. 1977. "Discordant Effects of Weak Prestimulation on Magnitude and Latency of the Reflex Blink." *Physiological Psychology* 5:108–14.
- Grillon, C., and M. Davis. 1995. "Acoustic Startle and Anticipatory Anxiety in Humans: Effects of Monaural Right and Left Ear Stimulation." *Psychophysiology* 32:155–61.
- Grillon, C., M. Pellowski, K. R. Merikangas, and M. Davis. 1997. "Darkness Facilitates the Acoustic Startle Reflex in Humans." *Biological Psychiatry* 42:453–60.
- Grosse, P., and P. Brown. 2003. "Acoustic Startle Evokes Bilaterally Synchronous Oscillatory EMG Activity in the Healthy Human." *Journal of Neurophysiology* 90:1654–61.
- Groves, P. M., and R. F. Thompson. 1970. "Habituation: A Dual-Process Theory." *Psychological Review* 77 (5): 571–81.
- Gulinello, M., R. Orman, and S. S. Smith. 2003. "Sex Differences in Anxiety, Sensorimotor Gating and Expression of the $\alpha 4$ Subunit of the GABAA Receptor in

- the Amygdala after Progesterone Withdrawal.” *European Journal of Neuroscience* 17:641–48.
- Haerich, P. 1997. “Long Term Habituation and Sensitization of the Human Acoustic Startle Response.” *Journal of Psychophysiology* 11:103–14.
- Hausmann, M., D. Slabbekoorn, S. H. Van Goozen, P. T. Cohen-Kettenis, and O. Gunturkun. 2000. “Sex Hormones Affect Spatial Abilities during the Menstrual Cycle.” *Behavioral Neuroscience* 114 (6): 1245–50.
- Hillman, C. H., K. S. Rosengren, and D. P. Smith. 2004. “Emotion and Motivated Behavior: Postural Adjustments to Affective Picture Viewing.” *Biological Psychology* 66:51–62.
- Ho, K. J., P. Kileny, D. Paccioletti, and D. R. McLean. 1987. “Neurologic, Audiologic, and Electrophysiologic Sequelae of Bilateral Temporal Lobe Lesions.” *Archives of Neurology* 44:982–87.
- Hoffman, H. S., and M. Fleshler. 1963. “Startle Reaction: Modification by Background Acoustic Stimulation.” *Science* 141:928–30.
- Holand, S., A. Girard, D. Laude, C. Meyer-Bisch, and J. L. Elghozi. 1999. “Effects of an Auditory Startle Stimulus on Blood Pressure and Heart Rate in Humans.” *Journal of Hypertension* 17:1893–97.
- Hori, A., A. Yasuhara, H. Naito, and M. Yasuhara. 1986. “Blink Reflex Elicited by Auditory Stimulation in the Rabbit.” *Journal of the Neurological Sciences* 76:49–59.
- Horlington, M. 1970. “Startle Response Circadian Rhythm in Rats: Lack of Correlation with Motor Activity.” *Physiology & Behavior* 5:49–53.
- Hsieh, M. H., N. R. Swerdlow, and D. L. Braff. 2005. “Effects of Background and Prepulse Characteristics on Prepulse Inhibition and Facilitation: Implications for Neuropsychiatric Research.” *Biological Psychiatry* 59:555–59.
- Hulcrantz, M., R. Simonoska, and A. E. Stenberg. 2006. “Estrogen and Hearing: A Summary of Recent Investigations.” *Acta Oto-Laryngologica* 126:10–14.
- Ison, J. R., M. K. Taylor, G. P. Bowen, and S. B. Schwarzkopf. 1997. “Facilitation and Inhibition of the Acoustic Startle Reflex in the Rat after a Momentary Increase in Background Noise Level.” *Behavioral Neuroscience* 111:1335–52.
- Jacobs, L., and M. D. Grossman. 1980. “Three Primitive Reflexes in Normal Adults.” *Neurology* 30:184–88.
- Justus, A. N., and P. R. Finn. 2007. “Startle Modulation in Non-incarcerated Men and Women with Psychopathic Traits.” *Personality and Individual Differences* 43:2057–71.
- Koch, M. 1999. “The Neurobiology of Startle.” *Progress in Neurobiology* 59:107–28.
- Kofler, M., J. Müller, L. Reggiani, and J. Valls-Solé. 2001. “Influence of Gender on Auditory Startle Responses.” *Brain Research* 921:206–10.

- Kofler, M., J. Müller, M. Rinnerthaler-Weichbold, and J. Valls-Solé. 2008. "Laterality of Auditory Startle Responses in Humans." *Clinical Neurophysiology* 119:309–14.
- Kofler, M., J. Müller, G. K. Wenning, L. Reggiani, P. Hollosi, S. Bösch, G. Ransmayr, J. Valls-Solé, and W. Poewe. 2001. "The Auditory Startle Reaction in Parkinsonian Disorders." *Movement Disorders* 16:62–71.
- Kumari, V., I. Aasen, A. Papadopoulos, F. Bojang, L. Poon, R. Halari, and A.J. Cleare. 2008. "A Comparison of Prepulse Inhibition in Pre- and Postmenopausal Women and Age-Matched Men." *Neuropsychopharmacology* 33:2610–18.
- Lacroix, J. M. 1981. "The Acquisition of Autonomic Control through Biofeedback: The Case against an Afferent Process and a Two-Process Alternative." *Psychophysiology* 18 (5): 573–87.
- Landis, C., and W. Hunt. 1939. *The Startle Pattern*. New York: Farrar and Rinehart.
- Lang, P. J. 1995. "The Emotion Probe: Studies of Motivation and Attention." *American Psychologist* 50:372–85.
- Lang, P. J., and M. Davis. 2006. "Emotion, Motivation, and the Brain: Reflex Foundations in Animal and Human Research." *Progress in Brain Research* 156:3–29.
- Lang, P. J., M. M. Bradley, and B. N. Cuthbert. 1990. "Emotion, Attention, and the Startle Reflex." *Psychological Review* 97:377–95.
- Lang, P. J., M. M. Bradley, and B. N. Cuthbert. 1997. "Motivated Attention: Affect, Activation, and Action. In *Attention and Orienting: Sensory and Motivational Processes*, edited by P. J. Lang, R. F. Simons, and M. T. Balaban, 97–135. Mahwah, NJ: Lawrence Erlbaum Associates.
- Leaton, R. N., and J. Cranney. 1990. "Potentiation of the Acoustic Startle Response by a conditioned Stimulus Paired with Acoustic Startle Stimulus in Rats." *Journal of Experimental Psychology: Animal Behavior Processes* 16:279–87.
- LeDoux, J. E. 1990. "Information Flow from Sensation to Emotion Plasticity in the Neural Computation of Stimulus Values." In *Learning and Computational Neuroscience: Foundations of Adaptive Networks*, edited by M. Gabriel and J. Moore, 3–52. Cambridge, MA: Bradford Books/MIT Press.
- Ludewig, K., S. Ludewig, A. Seitz, M. Obrist, M. A. Geyer, and F. X. Vollenweider. 2003. "The Acoustic Startle Reflex and its Modulation: Effects of Age and Gender in Humans." *Biological Psychology* 63:311–23.
- Martin, W. L., P. S. Murray, P. R. Bates, and P. S. Lee. 2015. "Fear-Potentiated Startle: A Review from an Aviation Perspective." *The International Journal of Aviation Psychology* 25:97–107.
- McCormick, C. M., and S. M. Teillon. 2001. "Menstrual Cycle Variation in Spatial Ability: Relation to Salivary Cortisol Levels." *Hormones and Behavior* 39 (1): 29–38.

- McManis, M. H., M. M. Bradley, W. K. Berg, B. N. Cuthbert, and P. J. Lang. 2001. "Emotional Reactions in Children: Verbal, Physiological, and Behavioral Responses to Affective Pictures." *Psychophysiology* 38:222–31.
- Mercado, F., L. Carretie, M. Tapia and G. Gomez-Jarabo. 2006. "The Influence of Emotional Context on Attention in Anxious Subjects: Neurophysiological Correlates." *Journal of Anxiety Disorders* 20:72–84.
- Miller, M. W., and C. Gronfier. 2006. "Diurnal Variation of the Startle Reflex in Relation to HPA-Axis Activity in Humans." *Psychophysiology* 43:297–301.
- Miltner, W., M. Matjak, C. Braun, H. Diekmann, and S. Brody. 1994. "Emotional Qualities of Odors and Their Influence on the Startle Reflex in Humans." *Psychophysiology* 31:107–10.
- Neumann, D. L., O. V. Lipp, and N. R. Pretorius. 2004. "The Effects of Lead Stimulus and Reflex Stimulus Modality on Modulation of the Blink Reflex at Very Short, Short, and Long Lead Intervals." *Attention, Perception, & Psychophysics* 66:141–51.
- Plappert, C. F., P. K. Pilz, and H. U. Schnitzler. 2004. "Factors Governing Prepulse Inhibition and Prepulse Facilitation of the Acoustic Startle Response in Mice." *Behavioural Brain Research* 152:403–12.
- Quevedo, K., T. Smith, B. Donzella, E. Schunk, and M. Gunnar. 2010. "The Startle Response: Developmental Effects and a Paradigm for Children and Adults." *Developmental Psychobiology* 52:78–89.
- Schmajuk, N. A., and J. A. Larrauri. 2005. "Neural Network Model of Prepulse Inhibition." *Behavioral Neuroscience* 119:1546–62.
- Sierra-Mercado, D., K. A. Corcoran, K. Lebrón-Milad, and G. J. Quirk. 2006. "Inactivation of the Ventromedial Prefrontal Cortex Reduces Expression of Conditioned Fear and Impairs Subsequent Recall of Extinction." *European Journal of Neuroscience* 24:1751–58.
- Stevenson, V. E., and E. W. Cook III. 1994. "Affective Modulation of Startle in Fearful and Schizotypal College Students." Paper presented at the Meeting of the Society for Research in Psychopathology, Miami, FL.
- Stoddart, C. W., J. Noonan, and M. T. Martin-Iverson. 2008. "Stimulus Quality Affects Expression of the Acoustic Startle Response and Prepulse Inhibition in Mice." *Behavioral Neuroscience* 122:516–26.
- Swerdlow, N. R., P. Auerbach, S. M. Monroe, H. Hartston, M. A. Geyer, and D. L. Braff. 1993. "Men Are More Inhibited than Women by Weak Prepulses." *Biological Psychiatry* 34:253–60.
- Thompson, R. F., and W. A. Spencer. 1966. "Habituation: A Model Phenomenon for the Study of Neuronal Substrates of Behavior." *Psychological Review* 73:16–43.
- Vaidyanathan, U., C. J. Patrick, and E. M. Bernat. 2009. "Startle Reflex Potentiation During Aversive Picture Viewing as an Indicator of Trait Fear." *Psychophysiology* 46:75–85.

- Valls-Solé, J., H. Kumru, and M. Kofler. 2008. "Interaction Between Startle and Voluntary Reactions in Humans." *Experimental Brain Research* 187:497–507.
- Valls-Solé, J., M. Kofler, H. Kumru, J.M. Castellote, and M.T. Sanegre, 2005. "Startle-Induced Reaction Time Shortening Is Not Modified By Prepulse Inhibition." *Experimental Brain Research* 165:541–48.
- Valls-Solé, J., A. Solé, F. Valdeoriola, E. Munoz, L. E. Gonzalez, and E. S. Tolosa. 1995. "Reaction Time and Acoustic Startle in Normal Human Subjects." *Neuroscience Letters* 195:97–100.
- Valsamis, B., and S. Schmid. 2011. "Habituation and Prepulse Inhibition of Acoustic Startle in Rodents." *Journal of Visualized Experiments* 55:1–10.
- Vrana, S. R., E. L. Spence, and P. J. Lang. 1988. "The Startle Probe Response: A New Measure of Emotion?" *Journal of Abnormal Psychology* 97:487–91.
- Walker, D. L., and M. Davis. 1997. "Anxiogenic Effects of High Illumination Levels Assessed with the Acoustic Startle Response in Rats." *Biological Psychiatry* 42:461–71.
- Wilkins, D. E., M. Hallett, and M. M. Wess. 1986. "Audiogenic Startle Reflex of Man and its Relationship to Startle Syndromes: A Review." *Brain* 109:561–73.
- Yeomans, J. S., and P. W. Frankland. 1995. "The Acoustic Startle Reflex: Neurons and Connections." *Brain Research Reviews* 21:301–14.

Abbreviations

ABR	acoustic blink reaction
ASR	acoustic startle response
OOC	orbicularis oculi
OR	orienting response
PPF	prepulse facilitation
PPI	prepulse inhibition
SAS	Special Air Service
SR	startle response

REPORT DOCUMENTATION PAGE*Form Approved*
OMB No. 0704-0188

The public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing the burden, to Department of Defense, Washington Headquarters Services, Directorate for Information Operations and Reports (0704-0188), 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to any penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number.

PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ADDRESS.

1. REPORT DATE June 2018		2. REPORT TYPE Final		3. DATES COVERED (From-To) Jan 2018 – Feb 2018	
4. TITLE AND SUBTITLE Analysis of the Startle Response to Flashbang Grenades				5a. CONTRACT NUMBER HQ0034-14-D-0001	
				5b. GRANT NUMBER	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S) Madhavan, Poornima Srinivasan, Ruhi				5d. PROJECT NUMBER DU-2-4106	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) Institute for Defense Analyses 4850 Mark Center Drive Alexandria, VA 22311-1882				8. PERFORMING ORGANIZATION REPORT NUMBER IDA Document D-8945	
9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES) Joint Non-Lethal Weapons Directorate 3097 Range Road Quantico, VA 22134-5100				10. SPONSOR/MONITOR'S ACRONYM(S) JNLWD	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION/AVAILABILITY STATEMENT Approved for public release; distribution is unlimited (12 June 2018).					
13. SUPPLEMENTARY NOTES					
14. ABSTRACT The surprise or "startle" element associated with flashbang grenades may trigger varying degrees of psychological and physiological distress that could have implications for the design of effective flashbang grenades. The startle response is the fastest known generalized motor reaction of humans and animals to unexpected or surprising stimuli. IDA was tasked with examining the neural, physiological and psychological components of the startle response associated with flashbang grenades in order to fully understand the processes driving human responses to flashbangs. This report provides a detailed review and analysis of the startle response in humans and its implications for the design of flashbang grenades.					
15. SUBJECT TERMS flashbang grenades; human behavior; non-lethal weapons; physiology; psychology; startle effect					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT Uncl.	b. ABSTRACT Uncl.	c. THIS PAGE Uncl.			Dr. Shannon Foley
			SAR	45	703-432-0916