

## Acoustic Startle in Maltreated Children

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We investigated the eyeblink component of acoustic startle reactions in maltreated children. Previous research indicates that acoustic startle is enhanced in adult males with posttraumatic stress disorders (PTSD) whereas findings on women with PTSD have been inconsistent. In accord with the only previous report for children with PTSD, we found that maltreated boys, particularly those who had been physically abused, responded to increases in startle probe loudness with smaller increments in amplitude of startle eyeblink and smaller reductions in blink latency than did comparison boys. Results for girls were inconsistent: younger maltreated girls had smaller startle amplitude and slower onset latency than controls, whereas older maltreated girls exhibited the opposite pattern.

**KEY WORDS:** maltreatment; physical abuse; acoustic startle.

The present study is an initial exploration of the effects of maltreatment on the strength of the acoustic reflex in children. The acoustic startle reflex is an obligatory response to a sudden and unexpected stimulus that is marked by the cessation of ongoing behaviors and by a particular series of protective behaviors (Davis, 1984; Landis & Hunt, 1939). The complete response in humans consists of a patterned sequence of rostral to caudal flexor movements in which the first feature is a brief closing of the eyes (i.e., an eyeblink) and a facial grimace, followed by neck flexion and then, shoulder and back contraction and flexion of the legs as the body assumes a defensive shrunken posture (Landis & Hunt, 1939, chap. 3). Of this group of movements the eyeblink is the most sensitive and consistent across individuals, and this is the response that is most often measured in studies of this reflex. Humans' startle eyeblink is measured from electromyographic activity detected by electrodes overlying the orbicularis oculi muscle, located below each eye. As shown in Fig. 1, the startle eyeblink can be quantified with respect to amplitude and

latency of onset of the electromyographic changes constituting this response.

The simple reflex pathway for acoustic startle proceeds from the ear to the ventral cochlear nucleus to the caudal pontine reticular nucleus and, from there, to the spinal cord and to muscles, including orbicularis oculi. Startle expression in humans and in laboratory animals is affected by learning (habituation) and coincident perceptual and emotional events. Insignificant neutral stimuli (e.g., soft sounds) that occur at brief intervals just prior to reflex elicitation inhibit startle, whereas stimuli presented at longer intervals before a startle probe may facilitate the reflex (Graham, 1975; Hoffman & Ison, 1980).

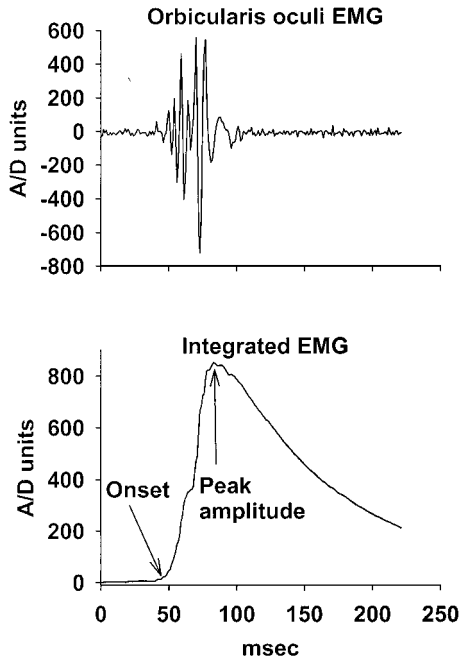
Startle expression in humans and in laboratory animals is affected by emotional factors, a connection that may be grounded in the evolutionary value of startle for immediate protection and its enablement of a basic posture from which other defensive or aggressive responses might quickly emerge. Nociceptive, anxiogenic, or unpleasant stimuli that briefly precede startle stimuli strongly potentiate this response (Brown, Kalish, & Farber, 1951; Davis & Astrachan, 1978; Lang, 1995). The effects of unpleasant stimulation or content are presumed to enhance startle through evocation of learned or innate responses in the amygdala, which then potentiate the startle reflex at the level of the caudal pons through a direct monosynaptic pathway (Davis, 1990; Frankland & Yeomans, 1995; Koch, 1999; Lang, 1995).

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**Fig. 1.** Illustrative record of a child's acoustic startle reflex evoked by a 115-dB probe. The top trace depicts the EMG response from orbicularis oculi and the bottom record the coincident rectified-integrated record from a contour follower. The Y-axis of both traces is in arbitrary, analog-to-digital (A/D) units that form scales that are not comparable for the two graphs. The X-axis represents time from probe onset in milliseconds. Starting at approximately 40 ms postprobe the EMG trace displays spikes of increasing amplitude that peak at around 80 ms poststimulus. For technical reasons, the integrated record from the contour follower lags slightly behind the EMG response.

Of particular relevance to the present research are reports of startle abnormalities in anxiety disorders. Importantly, the previously mentioned potentiation of startle eyeblink amplitude by unpleasant visual slides was absent in criminal psychopaths (Patrick, Bradley, & Lang, 1993), whereas posttraumatic stress disorder (PTSD) in adult men was linked to exaggerated acoustic startle blinks (Butler et al., 1990; Morgan et al., 1995, 1996; Orr, Lasko, Shalev, & Pitman, 1995). In contrast, results for women with PTSD have been inconsistent. Analogous to findings for traumatized men, women with PTSD resulting from sexual assault had *exaggerated* startle blinks to loud sounds (albeit from the left and not the right orbicularis muscle; Morgan, Grillon, Lubin, & Southwick, 1997), whereas women with PTSD emanating from childhood sexual abuse had *normal* startle blink amplitude (Metzger et al., 1999). Finally, the only study involving pediatric anxiety (Ornitz & Pynoos, 1989) detected *smaller* than normal amplitude of startle blinks in boys and girls with PTSD.

The rationale for the present study is that anomalies of startle in PTSD suggest that similar abnormalities might be found in maltreated children whose experience in traumatization would be expected to induce high levels of anxiety and PTSD. Thus, individual differences in startle might serve as a marker for anxiety disturbances in childhood maltreatment and provide clues to the effects of traumatization on these children.

Child maltreatment, a major societal problem with distressingly high prevalence, may represent the greatest failure of the caregiving environment to provide many of the expectable experiences necessary to facilitate normal developmental processes (Cicchetti & Lynch, 1995). Maltreatment sets in motion a probabilistic path of epigenesis for children characterized by an increased likelihood of failure and disruption in the successful resolution of major stage-salient issues of development and having grave implications for functioning across the life span. These developmental disruptions include maladaptive affective communication (Gaensbauer, Mrazek, & Harmon, 1981), disruptions of attachment (Cicchetti & Barnett, 1991), deviations in the development of an autonomous self (Cicchetti, 1991), sensitization to displays of anger (Maughan & Cicchetti, 2002; Pollak, Klorman, Thatcher, & Cicchetti, 2001), maladaptive social information processing (Dodge, Pettit, Bates, & Valente, 1995), problematic peer relationships (Bolger, Patterson, & Kupersmidt, 1998; Cicchetti, Lynch, Shonk, & Manly, 1992), linguistic delays (Coster & Cicchetti, 1993), and difficulties adapting to the academic and social challenges of school (Eckenrode, Laird, & Doris, 1993; Shonk & Cicchetti, 2001).

The study of child maltreatment, an "experiment of nature," can be utilized to illustrate how social experience can alter brain structure, function, and organization. Given that the mechanisms of neural plasticity cause the brain's anatomical differentiation to be dependent on stimulation from the environment (Cicchetti & Tucker, 1994; Greenough, Black, & Wallace, 1987), it is not surprising that maltreatment also exerts negative impacts on biological development and is a major risk factor for the emergence of psychopathology. For example, neuroendocrine (Cicchetti & Rogosch, 2001; DeBellis, Baum, et al., 1999), catecholaminergic (Kaufman & Charney, 2001), and brain structural anomalies (DeBellis, Keshavan, et al., 1999) have been found in maltreated children.

Notably, both pediatric and adult PTSD are associated with structural brain changes (DeBellis, Keshavan, et al., 1999; Kaufman & Charney, 2001). Moreover, maltreatment is linked with an excess of externalizing and internalizing disorders, including PTSD (Cicchetti & Toth, 1995; DeBellis, Baum, et al., 1999). Thus, disturbances

of anxiety and traumatization in childhood maltreatment and the sensitivity of the startle reflex to these conditions suggested the utility of examining startle patterns in maltreated children for ultimately developing objective physiological markers of the severity of traumatization. As a first approach to these issues, we studied acoustic startle in maltreated and nonmaltreated comparison children to a range of auditory intensities in order to describe any abnormalities in response magnitude, onset latency, and habituation. In addition, we examined differences among subtypes of maltreated children. To our knowledge, ours is the first prospective investigation of startle abnormalities in maltreated children, so that the research was necessarily exploratory in nature. In addition to the lack of previous research on startle in maltreated children, inconsistencies in the literature made it difficult to propose specific hypotheses. The single study on pediatric PTSD (Ornitz & Pynoos, 1989) led to predicting smaller than normal startle amplitude in maltreated children whereas the literature on adult PTSD reviewed earlier suggested the opposite.

## METHOD

### Participants

#### *Maltreated Children*

The sample was composed of 109 maltreated and 103 nonmaltreated low-income demographically comparable children.

The maltreated sample was recruited from the Monroe County Department of Social Services (DSS) records, and community comparison participants were solicited through fliers posted in the same impoverished neighborhoods in which the maltreated families resided. The maltreated sample was representative of families receiving services at the Monroe County Department of Social Services (DSS). Determination of maltreatment status was based on detailed examination of family records at DSS. Assessment of maltreatment history was based on multiple informants such as mothers, child protective services workers, neighbors, and other community members (e.g., teachers and daycare providers). Prior to enrolling in the study, mothers of both maltreated and nonmaltreated children provided written consent to allow project staff to examine any existing DSS records.

Specific maltreatment experiences were coded from DSS records using the objective operational criteria delineated in the Barnett, Manly, and Cicchetti (1993) Maltreatment Classification System. Prior investigations using this system have shown it to be reliable and valid in differen-

tiating among maltreatment subtypes (e.g., Bolger et al., 1998; Manly, Kim, Rogosch, & Cicchetti, 2001; Smith & Thornberry, 1995). Coding was conducted by trained doctoral students and PhD clinical psychologists, and adequate reliability was obtained (weighted Kappas = .86–1.00). All maltreated children had documented reports of sexual abuse, physical abuse, neglect, and/or emotional abuse, and comparison children lacked any such documented maltreatment experiences.

#### *Classification of Maltreatment*

*Sexual abuse* involved any attempted or actual sexual contact between a child and caregiver for purposes of the caregiver's sexual satisfaction or financial benefit. Acts included in this category varied from exposure to pornographic material or adult sexual activity to sexual touching and fondling, attempted penetration, forced intercourse, and prostitution of the child. *Physical abuse* involved the infliction of physical injury on a child other than by accidental means (e.g., beating the child causing bruises, welts, broken bones, burns, choking). *Neglect* involved failure to provide for a child's basic physical needs (i.e., for adequate food, clothing, shelter, medical treatment), lack of supervision (e.g., leaving child in the care of dangerous caregivers, leaving child without adult supervision), or moral-legal neglect (e.g., exposing the child to criminal activity, failure to send child to school). Finally, *emotional abuse* involved extreme thwarting of children's basic emotional needs for psychological safety and security, acceptance and self-esteem, and age-appropriate autonomy. Examples of emotional abuse in terms of increasing severity included belittling or ridiculing the child, using fear and intimidation, blaming the child inappropriately, extreme negativity and hostility, exposure to severe domestic violence, abandoning the child, confining the child in an enclosed space, and suicidal and homicidal threats.

Consistent with the literature and as detailed below (Barnett et al., 1993), the majority of the 109 maltreated children in this study ( $n = 64$ , 62.1%) experienced multiple forms of maltreatment. A designation for a child's primary type of maltreatment was made based on the degree to which the form of maltreatment present violated cultural standards. Any child who had been sexually abused was given a primary subtype classification of sexual abuse ( $n = 5$ ), regardless of whether the child had experienced other subtypes of maltreatment. Several sexually abused children had also experienced emotional abuse ( $n = 3$ ), neglect ( $n = 5$ ), and physical abuse ( $n = 2$ ). Children who had been physically abused but not sexually abused were given a primary classification of physical abuse ( $n = 27$ ),

irrespective of the presence of any other existing subtypes of maltreatment. Of these physically abused youngsters, there were 19 who had also been victims of emotional abuse and 19 who had experienced neglect. Children were classified as neglected ( $n = 55$ ) if they had undergone neglect but not physical or sexual abuse. The neglected children included 37 who had also experienced emotional abuse. Finally, children were classified as emotionally abused ( $n = 17$ ) if they had experienced no additional type of maltreatment.

In addition, 80% of the maltreated children experienced their onset of maltreatment during the first 2 years of life; however, continued concerns related to maltreatment were present, as all of the maltreated families were being monitored by authorities.

In analyses focused on comparing maltreatment subtypes, we omitted 5 maltreated children with insufficient information for subtyping and the 5 sexually abused children because their low number precluded meaningful analyses. However, these 10 participants were included in all analyses that contrasted comparison and aggregated maltreated children.

#### *Demographic Characteristics*

Demographic characteristics of the sample are presented in Table I. Maltreated and comparison samples were comparable in racial composition,  $\chi^2(1, N = 212) < 1$ , *ns*; gender makeup,  $\chi^2(1, N = 212) = 1.46$ , *ns*; Hollingshead socioeconomic level,  $\chi^2(1, N = 212) = 3.12$ , *ns*; and age,  $F(1, 208) < 1$ , *ns*. Similarly, the comparison and three subtype groups considered in our analyses did not differ in their ethnic composition,  $\chi^2(3, N = 202) = 4.05$ , *ns*; gender makeup,  $\chi^2(3, N = 202) = 4.28$ , *ns*; or socioeconomic status,  $\chi^2(3, N = 202) = 5.48$ , *ns*. However, these four samples differed in age,  $F(3, 194) = 3.04$ ,  $p < .04$ ; specifically, physically abused children were older ( $p < .05$ ) than children in all other groups.

At the time of testing, all children were reported to be in good health and not using any medications.

#### **Procedure**

Before testing, children's auditory thresholds were checked with an AudioMedtric Technology Maico 27 audiometer to insure adequate hearing. We presented 1-, 2-, and 4-kHz tones to each ear in descending order of intensity: 40, 30, 25, and 20 dB SPL. All study participants detected each tone at 20 dB SPL with at least one ear and all but one of the tones at 20 dB SPL with the other ear.

Children were tested individually in an IAC sound-attenuated chamber. An experimenter sat next to the child throughout the testing. Excerpts from animated movies were shown without sound on a monitor. Starting 1 min after the onset of the video presentation, children received white noise probes (48 + 2-ms rise/decay time) generated by Coulbourn Instruments solid state logic and delivered to TDH 39 headphones calibrated by AudioMedtric Technology. Six probes at each of four auditory intensities (70, 85, 100, and 115 dB SPL) were presented in random order and at intertrial intervals ranging from 24 to 32 s. The rationale for our choice of loudness levels was as follows. Probes of 100 dB or higher would be expected to evoke startle whereas the lower levels of intensity were needed to characterize any anomalies in the emergence of acoustic startle, including unusually low thresholds. It should be noted that, because of their extreme brevity, the 115-dB probes are perceived as having the loudness of a vigorous hand clap at a distance of about 20 cm from the ear.

The electromyogram (EMG) was monitored from a pair of In Vivo Metric electrodes taped over the right orbicularis oculi, amplified by means of Grass Model 12 recorders (0.1 and 1 kHz nominal lower and upper cutoff frequencies), and processed by a contour follower (100-ms time constant). As described earlier, it is the contraction of orbicularis oculi that is detected by electromyographic activity from this site and eventuates in an eyeblink. The contour follower rectifies (changes the positive and negative spikes making up the EMG signal to the same polarity) and smooths the EMG signal, approximating integration of the voltage spikes, in order to facilitate visualizing and scoring these reactions. Figure 1 displays an illustration of startle depicting a child's EMG response and coincident smoothed record to a single presentation of a 115-dB probe.

A Northgate 386 computer equipped with a Labmaster DMA analog-to-digital (A/D) converter digitized raw and rectified-integrated EMG at a rate of 1 kHz from 150 ms before through 375 ms following each probe's onset. A mid-forehead electrode served as ground.

#### **Scoring**

Two authors (RK, DC) inspected raw and concomitant rectified-integrated EMG records for each trial and determined by consensus which epochs should be eliminated because of excessive background noise; another author (JRI) resolved any disagreements or questions. The percentage of responses eliminated by this screening averaged 4.41 ( $SD = 6.52$ ) for comparison children and 5.05 ( $SD = 6.73$ ) for maltreated children. The remaining

**Table I.** Demographic Characteristics of Samples

Sample	Caucasian <sup>a</sup>	Minority <sup>a</sup>	Male <sup>a</sup>	Female <sup>a</sup>	Low SES <sup>b</sup>	Age <sup>c</sup>
<i>Aggregated groups</i>						
Comparison	32	71	51	52	73	6.48 (2.36)
Maltreated	33	76	63	46	84	6.81 (2.37)
<i>Maltreatment subtypes</i>						
Sexual abuse	1	4	3	2	50	7.05 (2.30)
Physical abuse	10	17	17	10	88	7.70 (2.20)
Neglect	20	35	32	23	85	6.61 (2.30)
Emotional abuse	2	15	6	11	82	5.64 (2.29)
Not classified	0	5	5	0	80	7.33 (3.17)

<sup>a</sup>Number of subjects.<sup>b</sup>Percent of subjects in Hollingshead levels IV and V.<sup>c</sup>Mean (*SD*) in years.

rectified-integrated EMG records were submitted to a computer program (Balaban, Losito, Simons, & Graham, 1986) that automatically scored on a trial-by-trial basis the two main dependent variables of startle eyeblinks: response amplitude (maximum deflection) in A/D units and onset latency in millisecond. (By convention, amplitude of startle responses is measured in arbitrary units that vary with amplifier calibration, contour follower characteristics, and A/D converter.) Valid responses for all trials were defined as those rectified-integrated EMG records that did not contain any activity in the first 20 ms following the probe, included a deflection (eyeblink) that began within 120-ms poststimulus, peaked by 150-ms poststimulus, lasted at most 95 ms, and showed a clear return to baseline. These commonly used criteria were intended to insure that the eyeblinks measured were elicited by the acoustic probe and ended sufficiently close to the occurrence of the probe so that they were not confounded with subsequent activity.

Table II shows the frequency for each sample and loudness level of responses with and without zero amplitude classified as valid (deemed not to have excessive background noise) and the frequency of responses rejected for excessive background noise or failure to meet scoring criteria. By far, valid responses with and without zero amplitude were most frequent. Maltreated and comparison subjects did not differ in the number of zero-amplitude responses,  $F(1, 196) = 1.67$ , *ns*, or nonzero responses,  $F(1, 196) < 1$ , *ns*. Predictably, however, increasing probe loudness elicited more nonzero responses,  $F(3, 588) = 231.61$ ,  $p < .0001$ , and fewer zero-amplitude responses,  $F(3, 588) = 227.43$ ,  $p < .0001$ .

Startle amplitude and latency were averaged for valid responses for each probe intensity. In the event of no detectable responses, we followed a dual strategy. One approach involved assigning these trials a conservative es-

timate of amplitude (0 A/D units) and latency (120 ms). A second strategy consisted of computing mean amplitudes and latencies exclusively for nonzero responses. However, using this method presented the difficulty that some subjects had only zero-amplitude responses, especially for low probe intensities. Therefore, analyses of nonzero responses were restricted to reactions to 100- and 115-dB intensities, for which, as detailed earlier, there were fewer zero-amplitude reactions. These data were available for 44 comparison boys, 47 comparison girls, 48 maltreated boys, and 38 maltreated girls

## RESULTS

Each set of analyses starts with results for measures that included zero-amplitude responses. Next,

**Table II.** Mean Number of Trials Per Response Category for Comparison and Maltreated Children

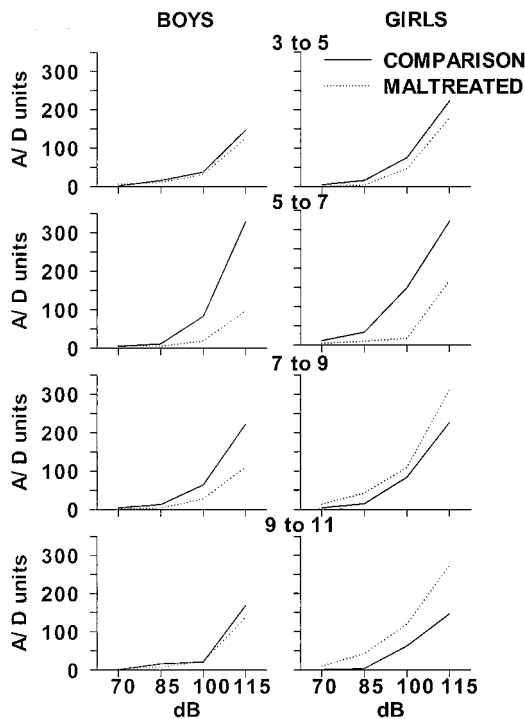
Group	Probe loudness			
	70	85	100	115
<i>Nonzero amplitude</i>				
Comparison	0.20	0.77	1.74	3.42
Maltreated	0.25	0.62	1.53	2.90
<i>Zero amplitude</i>				
Comparison	5.26	4.59	3.53	1.92
Maltreated	4.88	4.50	3.59	2.07
<i>Not meeting scoring criteria</i>				
Comparison	0.37	0.33	0.41	0.41
Maltreated	0.65	0.51	0.54	0.74
<i>Excessive noise</i>				
Comparison	0.16	0.31	0.32	0.26
Maltreated	0.22	0.37	0.34	0.28

*Note.* There were six trials per probe loudness, but totals may not add up to six because of rounding.

supplementary findings from analyses excluding zero-amplitude are presented. Both sets of data were entered into univariate analyses of variance with maltreatment/comparison status, gender, and age (<5, 5–6.99, 7–8.99, >9) as between-subject factors, probe intensity as a within-subject variable, and Greenhouse–Geisser correction for nonsphericity. Trend analyses of results including zero-amplitude reactions were conducted to evaluate whether dependent variables changed according to linear or quadratic functions as a function of age.

### Response Amplitude

As depicted in Fig. 2, averaging across maltreatment groups and gender, startle amplitude predictably increased with probe loudness, Intensity,  $F(3, 585) = 138.91, p < .0001$ ; Intensity linear,  $F(1, 195) = 145.98, p < .0001$ . Girls had larger blinks overall than boys,  $F(1, 195) = 6.25, p < .02, SEM = 3.6$  A/D units, and exhibited a greater impact of probe loudness, Intensity  $\times$  Gender,



**Fig. 2.** Startle amplitude (A/D units) for comparison and maltreated children across four noise intensities. Results are displayed separately for boys and girls of four age groups. The results generally indicate greater startle blink amplitude for comparison boys than maltreated boys. In contrast, the results for girls depend on age. Younger maltreated girls had larger startle amplitude than comparison girls, and older maltreated girls displayed the opposite trend.

$F(3, 585) = 3.60, p < .05$ ; Intensity linear  $\times$  Gender,  $F(1, 195) = 4.39, p < .04$ . Although the interaction of Intensity  $\times$  Maltreatment  $\times$  Gender fell short of significance,  $F(3, 585) = 2.49, ns$ , the overall pattern of interactions with gender prompted separate analyses by gender.

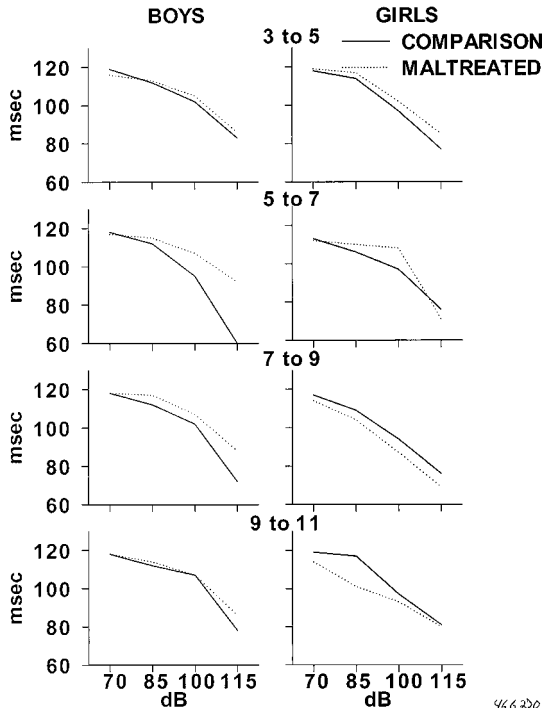
Compared with nonmaltreated boys, maltreated boys displayed smaller response amplitudes overall,  $F(1, 106) = 7.72, p < .01$ , and lesser increases of eyeblink amplitude with probe loudness, Intensity  $\times$  Maltreatment,  $F(3, 318) = 7.69, p < .005, SEM = 7.8$  A/D units, Intensity linear  $\times$  Maltreatment,  $F(1, 106) = 8.45, p < .005$ . As seen in Fig. 2, these differences were more pronounced in the middle age groups (5–9), Age quadratic  $\times$  Maltreatment,  $F(1, 106) = 4.47, p < .04$ , Age quadratic  $\times$  Intensity  $\times$  Maltreatment,  $F(3, 318) = 4.28, p < .04$ ; Age quadratic  $\times$  Intensity linear  $\times$  Maltreatment,  $F(1, 106) = 4.66, p < .04$ . Notably, the analysis restricted to nonzero responses to the louder two probes also disclosed less enlargement of blinks by increasing loudness among maltreated boys, Intensity  $\times$  Maltreatment,  $F(1, 84) = 5.84, p < .02, SEM = 9.5$  A/D units.

The effects of maltreatment among girls were less clearcut. As depicted in Fig. 2, across probe intensities, younger maltreated girls tended to have *smaller* responses than comparison girls whereas older maltreated girls tended to emit *larger* startles than their peers, Age linear  $\times$  Maltreatment,  $F(1, 89) = 3.61, p < .07$ . This pattern was more pronounced for nonzero startles to the louder probes, Age  $\times$  Maltreatment,  $F(1, 77) = 4.29, p < .05, SEM = 20.5$  A/D units.

### Onset Latency

Results on latency of startle onset are illustrated in Fig. 3. Overall, as expected, increasing probe loudness shortened startle onset latency, Intensity,  $F(3, 585) = 271.19, p < .0001$ , Intensity linear,  $F(1, 195) = 376.51, p < .001$ .

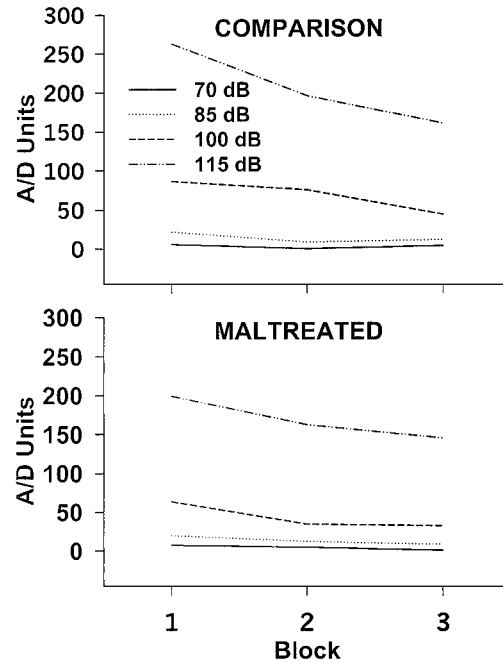
As seen in Fig. 3, girls displayed shorter blink onset latency overall than boys, Gender,  $F(1, 195) = 3.97, p < .05, SEM = 1.4$  ms. In addition, the genders differed in the combined effects of age, intensity, and maltreatment, Intensity quadratic  $\times$  Age linear  $\times$  Gender,  $F(1, 195) = 5.49, p < .03$ ; Intensity quadratic  $\times$  Age quadratic  $\times$  Maltreatment  $\times$  Gender,  $F(1, 195) = 6.02, p < .02$ . The latter interaction reflects differences among maltreatment groups as a function of age and gender in the unevenness of the reduction of startle latency for louder probes. Therefore, the results were again analyzed separately for gender.



**Fig. 3.** Startle onset latency (milliseconds) for comparison and maltreated across for four noise intensities. The results are displayed separately for four age classifications. Maltreated boys displayed slower onset of acoustic startle reflex and smaller reductions of startle latency by louder probes than did comparison boys. For girls, the decrease in onset latency with probe loudness was more curvilinear for younger maltreated participants than for comparison girls whereas the reverse was found for older girls.

Maltreated boys differed from comparison boys in having slower latencies overall, Maltreatment,  $F(1, 106) = 4.80, p < .04, SEM = 0.9$  ms, as well as less pronounced speeding of blink latency with louder probes, Intensity  $\times$  Maltreatment,  $F(3, 318) = 6.55, p < .003, SEM = 1.4$  ms; Intensity linear  $\times$  Maltreatment,  $F(1, 106) = 8.45, p < .004$ . When only nonzero responses to the higher two intensities were considered, maltreated boys again had slower onset latencies,  $F(1, 84) = 5.31, p < .03, SEM = 1.6$  ms.

As depicted in Fig. 3, among girls the decrease in onset latency with probe loudness was more curvilinear for younger maltreated participants than for comparison girls whereas the reverse was found for older girls, Maltreatment  $\times$  Age quadratic  $\times$  Intensity quadratic,  $F(1, 89) = 4.78, p < .04$ . In contrast, girls did not differ by maltreatment status for nonzero responses, Maltreatment,  $F(1, 77) < 1, ns$ ; Intensity  $\times$  Maltreatment,  $F(1, 77) < 1, ns, SEM = 1.3$  ms. This discrepancy may reflect the undue contribution of imputed latencies in the former anal-



**Fig. 4.** Startle amplitude (A/D units) for comparison and maltreated children for four noise intensities over trial blocks. The graph illustrates the absence of differences between maltreated and comparison children in startle amplitude change with repeated presentations.

ysis or the exclusion of subjects with zero-amplitude responses in the latter analysis.

**Response Decrement Over Trials**

To examine change in response amplitude over trials, results including zero-amplitude reactions were averaged for valid responses over pairs of successive presentations of each probe loudness. Predictably, as shown in Fig. 4, the decrement in response amplitude over trials was less pronounced for louder probes, Intensity linear  $\times$  Blocks linear,  $F(1, 162) = 20.43, p < .0001$ . However, this pattern was comparable for maltreated and comparison samples, Intensity linear  $\times$  Blocks linear  $\times$  Maltreatment,  $F(1, 162) = 2.38, ns$ .

**Maltreatment Subtypes: Startle Amplitude**

Separate analyses evaluated maltreatment subtype (none [comparison], physical abuse, neglect, emotional abuse), age, gender, and probe intensity. Statistical adjustments for age differences among subtypes were not applied, because neither startle amplitude nor onset

latency was linearly related to age, both regression,  $F_s(1, 192) = 0.00, ns$ .

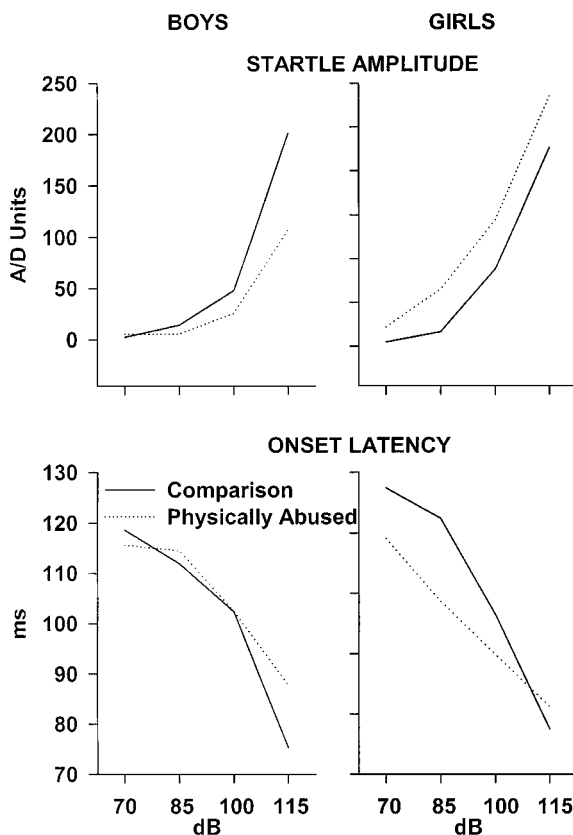
Planned pairwise comparisons were performed among comparison and maltreatment subtypes. Because all significant findings involved differences between physically abused and comparison children, we will omit mention of other contrasts. An interaction of Gender  $\times$  Physically abused versus Comparison,  $F(1, 193) = 4.07, p < .05$ , indicated discrepant findings for boys and girls regarding differences in blink amplitude between physically abused and comparison children. As depicted in Fig. 5, averaging across probe intensities, physically abused boys had *smaller* blinks than comparison boys ( $M \pm SEM = 36.1 \pm 9.8$  vs.  $66.4 \pm 9.5$  A/D units, respectively) whereas physically abused girls had *larger* average blinks than did comparison girls ( $M \pm SEM = 129.3 \pm 32.9$  vs.  $84.0 \pm 12.2$  A/D units, respectively). Separate analyses

for boys disclosed that increases in startle amplitude with probe loudness were marginally lower among physically abused than control boys, Intensity  $\times$  Physically abused versus Comparison,  $F(3, 306) = 3.60, p < .06$ ; Intensity linear  $\times$  Physically abused versus Comparison,  $F(1, 102) = 3.82, p < .06$ . This discrepancy was more pronounced for nonzero startles to the louder two probes, Intensity  $\times$  Physically abused versus Comparison,  $F(1, 83) = 4.08, p < .05, SEM = 9.1$  A/D units.

Physically abused and comparison girls did not differ significantly in startle amplitude, probably because only 10 physically abused girls were available.

### Maltreatment Subtypes: Startle Onset Latency

As also depicted in Fig. 5, physically abused and comparison children also differed on startle latency. Specifically, onset latency decreased less with increasing probe loudness among physically abused children, *independent of gender*, Intensity  $\times$  Physically abused versus Comparison,  $F(3, 579) = 4.46, p < .02$ , Intensity linear  $\times$  Physically abused versus Comparison,  $F(1, 193) = 5.11, p < .03$ , Intensity quadratic  $\times$  Physically abused versus Comparison,  $F(1, 193) = 5.65, p < .02$ , Gender  $\times$  Intensity  $\times$  Physically abused versus Comparison,  $F(3, 579) < 1, ns, SEM = 1.9$  ms. Notably, a related pattern of slower onset latency among physically abused children for the higher two probe intensities was found in an analysis of nonzero responses, Physically abused versus Comparison,  $F(1, 162) = 4.40, p < .04, SEM = 1.0$  ms.



**Fig. 5.** Startle amplitude (A/D units) and onset latency (milliseconds) for comparison and physically abused boys and girls across four noise intensities. Physically abused boys displayed smaller startle amplitude and smaller increases in amplitude with increasing loudness than comparison boys. Physically abused children, irrespective of gender, exhibited smaller decreases of startle onset latency for louder probes.

## DISCUSSION

### General Findings

The present results are in general agreement with developmental studies of acoustic startle. Consistent with reports by Ornit and colleagues (Ornit, 1999; Ornit, Russell, Yuan, & Liu 1996) that mature levels of habituation are obtained by ages 3–5, no age differences emerged for amplitude decrement over trial blocks. Also in accord with Ornit and collaborators (Ornit, Guthrie, Kaplan, Lane, & Normal, 1986; Ornit, Guthrie, Sadehpour, & Sugiyama, 1991), startle amplitude and latencies decreased, albeit not significantly, with age and stabilized by age 8. However, in contrast to Ornit et al.'s finding of comparable startle amplitude and latency for boys and girls 4–8 years old (Ornit et al., 1991), we observed larger startle amplitude and longer latency for

girls than boys, particularly for louder probes. It is difficult to attribute these gender differences to the few minor discrepancies between our protocol and Ornitz et al.'s procedures (Ornitz et al., 1991), namely that we presented no trials with lead stimuli and that we used four intensity levels for startle stimuli rather than one.

As reported by other investigators (Hawk, personal communication, January 15, 2002; McManis, personal communication, January 15, 2002), we detected a greater frequency of zero-amplitude responses than in studies of adults. Ornitz et al. (1991) reported success in minimizing zero-amplitude responses by blanking out background videotapes before probe presentation and withholding probes during episodes of low arousal or excessive activity. On the other hand, findings of elevated rates of zero-amplitude responses in children prompt continued attention to this issue. We stress, however, that our results are essentially unchanged for measures including or excluding zero-amplitude responses.

### Differences Between Maltreated and Comparison Boys

Turning to the main focus of the study, maltreated boys' startle blinks had smaller amplitude and slower onset latency and were less affected by increasing probe loudness than were those of comparison boys. Among maltreatment subtypes, this pattern was most salient for boys who were physically abused, the type of maltreatment in the subtypes considered in our analysis that most violates cultural standards. These findings are particularly noteworthy because maltreated and comparison children were closely matched on demographic factors. The interpretation of the reduction of startle among maltreated boys, particularly those exposed to physical abuse, is necessarily speculative. However, it is tempting to explain the results in terms of a generalized experience of defensive reactions by these boys leading to reduced responsiveness to noxious or abrupt stimulation. Ornitz and Pynoos (1989, p. 869) proposed that the reduced acoustic startle responsiveness found in children with PTSD might reflect cortically mediated attentional dysfunction that controls brainstem-mediated startle responses.

Of relevance to the present results on startle is a line of research involving cortisol secretion in traumatized and healthy individuals. Notably, Cicchetti and Rogosch (2001) found that, compared to children with other maltreatment subtypes and to nonmaltreated comparisons, physically abused children exhibited a suppression of cortisol and significantly less diurnal variation of the hypothalamic-pituitary-adrenal axis. These findings are consistent with reports of depressed cortisol secretion

in the *acute* reaction to stress by adults who subsequently developed PTSD (Aardal-Eriksson, Eriksson, & Thorell, 2001; Delahanty, Raimonde, & Spoonster, 2000; Yehuda, McFarlane, & Shalev, 1998). Of related interest is the finding that elevation of cortisol by exogenous administration of yohimbine, an  $\alpha_2$  adrenergic antagonist, also enhanced amplitude of acoustic startle in healthy volunteers (Stine et al., 2001). On the other hand, a 20-mg dose of hydrocortisone (an ester of cortisol) inhibited startle amplitude in normal adults (Buchanan, Brechtel, Sollers, & Lovallo, 2001); a 5-mg dose of hydrocortisone had the opposite effect. In combination, this series of studies involving *adults* suggest that stress leading to PTSD may be linked to acute hypocortisolism and that startle amplitude is modulated by alterations in cortisol levels.

Insofar as our physically abused male participants were tested when maltreatment concerns were still ongoing, their pattern of reduced startle responsiveness is consistent with reports of diminished cortisol levels in acutely traumatized adults with PTSD and a separate cohort of physically abused children previously evaluated in our laboratory. In addition, our results are in accord with findings of the depression of startle by exogenous cortisol. Although startle responsiveness and cortisol regulation are linked to separate but interconnected neurobiological systems, in both the present research on startle and in Cicchetti and Rogosch's study on cortisol, physically abused children manifested diminished responsiveness. Physically abused children are often exposed to danger and their suppression of cortisol and smaller responses to startle may reflect allostatic load (McEwen & Stellar, 1993), the long-term effect of physiologic responses to stress. Repeated social challenges such as those encountered by physically abused children can disrupt basic homeostatic regulatory processes (Cicchetti & Rogosch, 2001; Repetti, Taylor, & Seeman, 2002). Accumulated allostatic load may eventuate in a number of deleterious biological consequences, including impairments in sympathetic and parasympathetic nervous system functioning, hyper- or hypocortisolism, and dysregulation of serotonergic functioning (McEwen, 1998; Repetti et al., 2002).

Our results for maltreated boys also are consistent with those of Ornitz and Pynoos (1989) for diminished startle responses among children with PTSD. Interestingly, both studies contrast with reports that adult men with PTSD exhibit *larger* amplitude of acoustic startle than controls. There is no apparent explanation for this difference, but the consistency in the emerging literature on acoustic startle in traumatized children is encouraging. The discrepancy between pediatric and adult samples of traumatized individuals might be clarified by longitudinal studies. This strategy could help in establishing whether

the timing of traumatization or age at testing determines an outcome of under- or overresponding to startle probes. Because 80% of the maltreated children in the present study experienced their onset of maltreatment within the first 2 years of life and also had current active DSS monitoring, it is impossible to disentangle whether age of onset versus chronicity of the trauma drive our findings on startle responsiveness. It would also be useful for future research to include paradigms involving startle modification by inhibitory and facilitatory prestimulation (Hoffman & Ison, 1980). Ornitz and Pynoos (1989) also found that, besides diminished unmodulated startle reactions, children with PTSD were deficient in prepulse inhibition of startle responses. Thus, it would be interesting to determine whether maltreated children also resemble children with PTSD in this respect.

### Findings for Girls

A notable, and unexpected, gender difference concerned smaller startle amplitudes and generally slower onset latencies for younger maltreated than comparison girls in contrast to larger responses and shorter latencies among older maltreated girls. Curiously, though, differences in onset latencies between maltreated and control girls were *less* salient for the loudest probes. Unfortunately, the small size of our sample of physically abused girls prevented sensitive comparisons with other maltreatment subtypes. At this point, we cannot explain this developmental difference in results for maltreated girls.

Though we cannot provide a convincing explanation for the sex differences that we observed in the maltreated sample, it is instructive to relate our findings on maltreated girls to the limited literature on startle in traumatized adult women. Notably, Metzger et al. (1999) obtained comparable startle amplitude among women with PTSD and comparison women; however, the women with PTSD were all victims of childhood sexual abuse and differed with respect to whether they had a history of current or past PTSD. Thus, it is not possible to separate the effects of gender, age, type of abuse, history of traumatization, and the delay between the trauma and startle assessment. Interestingly, Morgan et al. (1997) reported larger than normal startle amplitude in prospectively identified women with PTSD related to sexual assault. Though these findings are consistent with our results for older maltreated girls, clearcut conclusions are difficult because of the dearth of research on startle in traumatized women. Another interpretive difficulty stems from differences in type of trauma in samples of men and women with PTSD (war trauma vs. sexual abuse). Some of these discrepant results might be clarified by a longitudinal study of startle in sexually abused girls.

Clearly, additional research will be needed to understand developmental and sex differences relevant to the effects of traumatizing experiences on acoustic startle.

### Limitations

This study has some limitations that merit mention. Because of the paucity of previous research on traumatized children and the contradictory findings for adult males and females, it was not possible to advance strong hypotheses. In addition, the results for maltreated girls follow the pattern of discrepant results for women with PTSD. To date, no satisfactory explanation has been proposed for these gender differences in adults with PTSD and it is equally difficult to do so for the first study examining these issues in maltreated children. However, the present findings provide the basis for designing future research that can address these issues.

It may be noted that the findings that we obtained for physically abused children are dependent on our hierarchical classification of maltreatment. It might seem desirable to evaluate the separate influence of maltreatment experiences (emotional abuse, neglect, physical abuse, and sexual abuse) that overlap across these classifications. As noted and documented earlier, this overlap is a common phenomenon and by no means an idiosyncratic finding. It might have been desirable to perform sequential multiple regression analyses to evaluate the unique contribution of each type of maltreatment experience to our results on startle. Unfortunately, the relatively small size of the sample did not afford sufficient statistical power to perform this analysis. Thus, although the present results demonstrated convincingly the attenuation of acoustic startle responsiveness in physically abused boys, it remains for future work to delineate the extent of startle anomalies in children who have experienced other subtypes of maltreatment.

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