

## Hostile Intent Attribution and Aggressive Behavior in Children Revisited: A Meta-Analysis

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To test specific hypotheses about the relation between hostile intent attribution (HIA) and children's aggressive behavior, a multilevel meta-analysis was conducted on 111 studies with 219 effect sizes and 29,272 participants. A positive association between HIA and aggression was found, but effect sizes varied widely between studies. Results suggested that HIA is a general disposition guiding behavior across a broad variety of contexts, whereas the strength of the relation between HIA and aggression depends on the level of emotional engagement. The relation is stronger for more reliable HIA measures, but is not stronger for reactive aggression or co-morbid attention-deficit hyperactivity disorder than for aggression in general. The importance of understanding specific moderators of effect size for theory development is discussed.

Hostile intent attribution (HIA) is defined as the tendency to attribute hostile intent to others in social situations with a negative outcome for the individual, where the intention of the other person is ambiguous. In a typical study, HIA is measured by presenting children social situations with a negative outcome caused by a peer, who's intentions are ambiguous, and subsequently asking about the intentions of the peer in the presented social situation. Social-cognitive models propose that children who frequently interpret the intentions of others as hostile in ambiguous situations will be more prone to respond aggressively, as a way to retaliate or defend themselves, than children who attribute non-hostile intent after being hindered (Crick & Dodge, 1994; Dodge, 1980). Moreover, social-cognitive theory states that HIA not only causes aggressive behaviors but also maintains aggressive behavior patterns. The latter follows from the assumption that aggressive children, as a result of their aggressive behavior, will more frequently be confronted with problematic social interactions. These problematic social interactions prohibit aggressive children to challenge their hostile beliefs about the intentions of others and limit the opportunity to acquire prosocial behavioral strategies. The crucial role of HIA in the development and maintenance of aggression has

been supported in experimental (e.g., Dodge, 1980; Lochman & Dodge, 1998), longitudinal (e.g., Dodge et al., 2003; Lansford, Malone, Dodge, Pettit, & Bates, 2010), and longitudinal-experimental studies (e.g., Lochman & Wells, 2002), making HIA a plausible target for effective cognitive-behavioral interventions (CBT) to reduce aggressive behavior in children (e.g., Hudley & Graham, 1993; Lochman & Wells, 2002).

The construct HIA has much potential to further our understanding of the development of aggressive behavior problems and to improve clinical practice. HIA may mediate links between aggression, distal riskfactors in children (such as executive functioning deficits or difficult temperament), and their environments (such as early harsh life experiences, rejection by peers, and coercive family interactions [e.g., Dodge, 2006]). More specifically, social-cognitive theory states that the tendency to attribute hostile intentions to others derives from transactions between early aversive child experiences such as harsh parenting and peer rejection on the one hand and child susceptibility to such experiences on the other hand (Dodge, 1980; Dodge et al., 2003; Dodge, Pettit, Bates, & Valente, 1995; Lansford, Malone, Dodge, Pettit, & Bates, 2010; Weiss, Dodge, Bates, & Pettit, 1992). Thus, children who experienced harsh parenting and

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This research was supported by a grant from The Netherlands Organization for Scientific Research (grant number 453-15-004/511) to B.O. De Castro.

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Child Development published by Wiley Periodicals, Inc. on behalf of Society for Research in Child Development

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0009-3920/2019/9005-0026

DOI: 10.1111/cdev.13255

peer rejection, and exhibit underlying vulnerabilities, such as executive functioning deficits or difficult temperament, could be particularly prone to develop hostile attribution styles and subsequent aggressive behavior patterns.

However, further progress in our understanding of HIA in aggressive behavior seems to be thwarted by unexplained variation in the strength of the relation between HIA and aggression. The last meta-analysis on the relation between aggressive behaviors in children and HIA demonstrated a modest robust relation ( $d = 0.35$ , fail-safe number of studies: 3.411) that did, however, vary widely between studies (De Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). This meta-analysis was conducted in 2002 and showed that the relation between HIA and aggression was stronger for children exhibiting more severe aggressive behavior (clinically referred aggressive children vs. nonreferred children), children between 8–12 years, children low on sociometric status and in studies that did not control for children's intelligence. Moreover, the use of staged interactions (standardized real-time interactions with a peer) and hypothetical stories read to or by children yielded higher effect sizes than the use of hypothetical stories presented through video-clips and pictures. Effect sizes were not related to aggression function (e.g., reactive aggression, general aggression), type of social context (e.g., provocation, nonprovocation), setting (e.g., individual, group), response format (e.g., open responses, rating scales, or multiple choice), and type of HIA scoring (e.g., hostile responses, hostile minus benign attributions).

Despite identifying several moderators of effect, this meta-analysis could not explain the significant variation in effect sizes between studies properly. In addition, this meta-analysis did not formulate specific hypotheses about moderators of the relation between aggressive behavior in children and HIA. Fortunately, since 2002 a number of important reviews and theoretical articles have suggested adaptations to social information processing (SIP) theory that may help to explain the divergent findings between studies. For example, De Castro (2004) suggested how HIA may be most evident in emotionally engaging situations, Peets, Hodges, Kikas, and Salmivalli (2007) suggested that HIA may be unique to interactions with specific familiar peers (i.e., disliked peers), whereas both Dodge (2006) and Schultz, Grodack, and Izard (2010) suggested that HIA may be specific to particular developmental stages. As far as we know, it has not yet been tested whether these hypotheses are supported

by actually explaining variance in findings between studies. To test specific hypotheses about moderators of the relation between HIA and aggression in children, we conducted a new meta-analysis. Advances in theory suggest five specific hypotheses about moderators of the relation between HIA and aggression in children:

First, the relation between HIA and aggression may be stronger in emotionally engaging situations. Social-cognitive theories postulate that for many children the actual processes leading up to aggression only occur when they are emotionally and personally involved (Anderson & Bushman, 2002; Lemerise & Arsenio, 2000). Moreover, empirical research suggests that aggression is often associated with excessive anger or anxiety (Granic, 2014; Hubbard et al., 2002) and that the induction of negative emotions results in more severe HIA and aggression (De Castro, Slot, Bosch, Koops, & Veerman, 2003; Dodge & Somberg, 1987; Reijntjes et al., 2011). An explanation might be that strong emotions (e.g., excessive anger) derail cognitive resources and thereby inhibit deliberate reflective processing. Strong emotions may force individuals to mainly rely on automatic SIP driven by hostile beliefs about the intentions of others established through early aversive child experiences. Since HIA and aggression are associated with aversive social experiences such as peer rejection (e.g., Dodge et al., 2003; Lansford et al., 2010), it seems that strong emotions in aggressive children steer the automatic interpretation of the intention of others in future social situations congruent with hostile memories of previous social interactions. This line of reasoning suggests that particularly social situations that are emotionally involving elicit the automatic and emotional processes that activate HIA. Thus, based on social-cognitive theories we hypothesized that the strength of the relation between HIA and aggression increases with the level of emotional involvement the social situation elicits. This would have direct implications for clinical practice since it implies that CBT should target HIA using emotionally engaging- and personally involving situations.

Second, the relation between HIA and aggression may be stronger in social situations with familiar others, encountered in previous problematic social situations (i.e., disliked others), than toward unfamiliar others. Social-cognitive theory proposes that the tendency to attribute hostile intent to others is a general cognitive disposition toward both familiar and unfamiliar others. This is based on the assumption that HIA steers SIP across a broad variety of contexts. However, several empirical studies suggest that HIA may only be present in social

situations with others who were encountered in previous problematic encounters (Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001; Peets, Hodges, Kikas, & Salmivalli, 2007; Peets et al., 2008). If HIA would be limited to interactions with specific familiar peers, this would have serious implications for social-cognitive theory and clinical practice. It would suggest that HIA is context-specific and only guides SIP in social situations with disliked others known from previous problematic encounters. Importantly, all current evidence-based CBTs are based on the assumption that a general cognitive disposition needs to be targeted to establish significant and prolonged changes in SIP and subsequent behaviors across a wide range of contexts. If HIA were person-specific, such broad generalization would not take place, which would question our expectations of CBT treatment potential. In line with the SIP model, we hypothesized that the relation between HIA and aggression is present in social situations with both unfamiliar and familiar others (e.g., Dodge, 2006). In addition, we expected this relation to be stronger in situations with familiar others encountered in previous problematic social encounters (i.e., disliked others) than with unfamiliar others.

Third, the relation between HIA and aggression is expected to be present irrespective of the sociometric status of participants. Social-cognitive theory postulates that HIA is a general cognitive disposition that guides SIP across contexts. Thus, social cognitive models propose that the tendency to attribute hostile intent to peers is not uniquely related to specific past experiences of peer rejection but could also be a result of other aversive social experiences (e.g., harsh parenting). Therefore it could be expected that both aggressive nonrejected children and aggressive-rejected children make hostile attributions about peers' intentions. Nonetheless, the previous meta-analysis suggested that the relation between HIA and aggression was stronger for aggressive-rejected samples than for generally aggressive samples. This finding suggests that the relation between aggression and HIA might be stronger when the social situation matches specific memories of being rejected by peers. We therefore hypothesized that the relation between HIA and aggression would be present in both aggressive and aggressive-rejected samples, yet would be particularly pronounced in aggressive-rejected samples.

Fourth, the relation between HIA and aggression may be stronger when aggression is operationalized as reactive aggression. Reactive and proactive aggression are proposed to have distinct etiologies

(Dodge & Coie, 1987; Frick, Cornell, Barry, Bodin, & Dane, 2003; Polman, De Castro, Koops, Van Boxtel, & Merk, 2007; Poulin & Boivin, 2000; Raine et al., 2006, but see Bushman & Anderson, 2001, for a critique). Reactive aggression is defined as an emotional, impulsive aggressive response to a perceived threat, provocation, or frustration aimed at defending oneself or retaliation. In contrast, proactive aggression is defined as coldblooded, planned aggressive behavior aimed at instrumental, material, or social personal gain (Dodge, 1991). It can be assumed that children who frequently attribute hostile intent to others will be more likely to perceive threats or provocations in other's behaviors and thereby engage in reactive aggressive behaviors. In addition, based on the same theory no relation between HIA and proactive aggression would be expected. The previous meta-analysis (De Castro et al., 2002) did not find an effect of function of aggression. However, this finding was based on only four studies. As suggested by the authors, a lack of power may explain this null-finding. Based on theory, we therefore hypothesized that the relation between HIA and aggression is stronger for reactive aggression and weaker for aggression measured as a general construct (with no differentiation between reactive- and proactive aggression).

Fifth, the strength of the relation between HIA and aggression may be positively associated with the proportion of children meeting criteria for attention-deficit hyperactivity disorder (ADHD). Social-cognitive theories state that aggression driven by HIA is partly due to limited cognitive capacities (e.g., Dodge & Pettit, 2003) and this seems to be supported by empirical research (e.g., Ellis, Weiss, & Lochman, 2009). Moreover, research demonstrated that ADHD is positively associated with both aggression and executive functioning deficits (Doyle, 2006; Hummer et al., 2010; King & Waschbusch, 2010; Waschbusch, 2002). Given the important role of executive functioning deficits in SIP (e.g., Ellis et al., 2009; Van Nieuwenhuijzen, et al., 2006), it is expected that particularly aggressive children with executive functioning deficits may find it difficult to accurately process information from the social environment, making them more susceptible to attribute hostile intent to others in social situations. Therefore we hypothesized that the strength of the relation between HIA and aggression increases with the proportion of ADHD diagnoses in the aggressive sample.

Methodologically, the previous meta-analysis included too few studies to analyze important combinations of moderators, such as studies combining

a clinical sample with in vivo provocation. Fortunately, while the 2002 meta-analysis only contained studies up to January 1998, many excellent studies into the relation between childhood HIA and aggression have been carried out since. The present extension of this meta-analysis allowed to include all eligible studies within a timeframe over 40 years (instead of 25 years in the previous meta-analysis). Moreover, due to statistical limitations at the time (e.g., inability to model dependency in effect sizes), the previous meta-analysis was only able to derive a single effect size from each study. As a result of statistical developments (e.g., multilevel meta-analysis), our extension of this meta-analysis could accommodate dependency in effect sizes and therefore allowed to derive multiple effect sizes from each study.

To test specific hypotheses about moderators of the relation between HIA and aggression in children, we conducted a new meta-analysis to test specific hypotheses, including more than double the number of studies, more variance and more precise assessment of moderators than the 2002 meta-analysis, and using statistical innovations to model effects. As explained above, methodological characteristics that were hypothesized to influence effect sizes included the type of stimulus presentation and provocateur's status in the presented social situation. Child characteristics that were hypothesized to influence effect sizes included sociometric status, function of aggression and proportion of ADHD diagnoses in the sample. In addition, we coded all variables included in the previous meta-analysis (De Castro et al., 2002) and exploratively tested whether the moderator effects were replicated.

## Methods

### *Study Selection*

Child aggression was operationalized as all behaviors leading to psychological, physical, or material harm of others. Thus, this operationalization covered a broad range of behaviors including categorizations on a syndrome-level (e.g., diagnoses of disruptive behavioral disorders), categorizations on a symptom-level (e.g., starting fights), and behavioral outcomes measured on a continuum (e.g., externalizing behaviors). HIA was operationalized as the attribution of hostile intent to peer's behaviors in social situations where the peer's intentions are ambiguous or differ systematically across situations (e.g., partly ambiguous, partly hostile, and partly benign).

All empirical studies into the relation between childhood aggression and the attribution of hostile intent to peer's behavior conducted between January 1998 and October 2017 were searched in the following databases: PsycINFO, Web of Science, PubMed and Google Scholar. Within all search databases the following strings were searched: "aggress\*" OR "violence" OR "violent behavior\*" OR "behavior problem\*" OR "conduct disorder\*" OR "conduct problem\*" OR "antisocial behavior\*" OR "behavior disorder\*" OR "oppositional defiant disorder\*" OR "disruptive behavior\*" in combination with "attribution\*" OR "hostil\*" OR "social cognit\*" OR "social perception" OR "interpretation bias" OR "social information processing" OR "cognitive style" OR "cognitive bias" OR "Kenneth. A. Dodge." The search was limited to human participants, childhood (0–12 years) or adolescence (13–17 years), and English language. It is important to note that the literature search of this extension started where the literature search from the previous meta-analysis ended (De Castro et al., 2002). This search resulted in 6,834 studies. In addition, all studies that cited the original meta-analysis were also searched in the Web of Science database. This search retrieved 329 additional studies resulting in 7,163 studies total. After removal of duplicates, 4,973 potential studies remained for further evaluation of eligibility. The authors acknowledge that although the search process was extensive and thorough, the possibility that specific studies were not identified can not be ruled out.

The strategy to evaluate study eligibility consisted of two steps. First, all retrieved studies were scanned on title and abstract for exclusion. Second, for all remaining articles full-texts were evaluated for eligibility. A flow diagram for the search and identification of studies is depicted in Figure 1. Thus, 4,973 studies were scanned on title and abstract, which resulted in the exclusion of 4,653 studies. Subsequently, the 320 remaining articles full-texts were evaluated for eligibility. The current meta-analysis applied identical inclusion and exclusion criteria as the 2002 meta-analysis. The inclusion and exclusion criteria were the following:

1. HIA and aggression were empirically assessed using standardized instruments.
  - a. When studies distinguished between reactive- and proactive aggression, effect sizes were derived from the reactive aggression data only, since based on theory no relation between HIA and proactive aggression was expected.

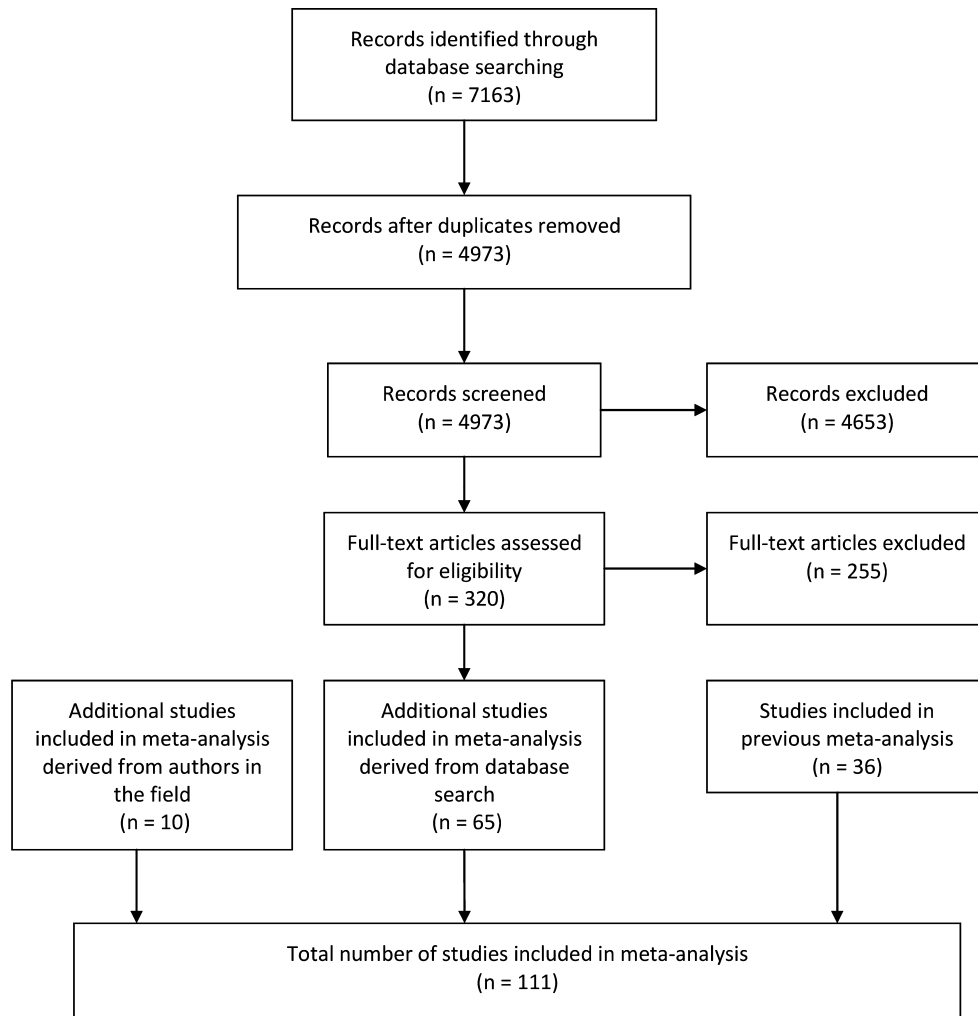


Figure 1. Flow diagram of search and identification of studies.

- b. Studies that compared clinically aggressive children to other clinical groups, but not to nonaggressive controls were excluded since no reliable comparison could be made between clinical groups.
- c. Studies that used rejection as the only selection criterion were excluded. Studies that used both aggression and rejection as selection criterion were included.
- d. Studies that used social competence instead of aggression as a selection criterion were excluded. Low social competence and aggression are not opposite poles on a continuum and therefore low social competence was not considered as an indicator of aggression.
- e. Studies that used ADHD as the main selection criterion were only included when the

ADHD group demonstrated high aggression scores as well.

2. HIA and aggression were measured on the same time point. Studies that measured HIA and aggression on different time points were excluded since it is impossible to determine whether this relation would have been identical on the same time point (e.g., Fontaine et al., 2010; Godleski & Ostrov, 2010).
3. HIA was operationalized as specific cognitions about a presented social situation. Thus, studies that assessed hostility as a general pattern of cognitions or personality trait were excluded (e.g., Rubio-Garay, Carrasco, & Amor, 2016).
4. HIA was not measured following experimental manipulation. It is impossible to determine the effect of the experimental manipulation on

the relation between HIA and aggression. Thus, with regard to studies that used experimental manipulations such as the induction of emotions (e.g., De Castro, Slot, Bosch, Kooops, & Veerman, 2003; Reijntjes et al., 2011) or treatment (e.g., Stoltz, Deković, van Londen, De Castro, & Prinzie, 2013) effect sizes were derived from premanipulation data only.

5. The presented social situations were standardized social interactions with peers. Studies that presented social situations concerning social interactions with solely adults were excluded. In studies that used social interactions with peers and adults and reported a composite score, effect sizes were based on this composite score. We decided to focus on interactions with peers only because of the presumed role of peer rejection as a cause for hostile attributions (Dodge, 2006) and the fact that almost every study on HIA and childhood aggression used social situations with peers to measure HIA. Studies that used unstandardized stimulus materials were not included since unstandardized stimulus materials prohibit to make between study comparisons.
6. Part of the stimulus materials were required to be ambiguous. Studies that solely presented nonambiguous social situations were excluded. Regarding studies that used a mixture of ambiguous- and nonambiguous social situations and reported a composite score of HIA, effect sizes were based on this composite score.

To derive reliable estimates of true effect sizes and to minimize the possibility of publication bias, multiple authors in the field were contacted for unpublished data. In addition, for studies that measured HIA and aggression but did not report sufficient information to calculate effect sizes, authors were contacted for additional information. The previous meta-analysis of De Castro et al. (2002) included 41 studies, however, one study (Dodge & Price, 1994) needed to be excluded from the present meta-analysis since it used a measure of behavioral competence instead of aggression. In addition, the previous meta-analysis treated different samples tested in the same study (Crick & Dodge, 1996; Lochman & Dodge, 1994) as independent studies, however, these were treated as from the same study in the present meta-analysis. From the 36 independent studies included in the previous meta-analysis, 51 effect sizes were derived, and the new

search resulted in an additional 75 studies (68%) and 168 effect sizes (77%). Thus, the present meta-analysis included 111 studies and 219 effect sizes in total. An overview of the included studies and effect sizes in this meta-analysis is provided in Supporting Information (see Table S24).

### *Coding*

To examine whether specific variables influenced the relation between HIA and aggression child characteristics and methodological characteristics were coded for each effect size.

### *Methodological Characteristics*

Methodological characteristics that were hypothesized to influence the relation between HIA and aggression were operationalized in following manner:

*Type of stimulus presentation.* Type of stimulus presentation was used as an indicator of the level of emotional engagement and coded categorically. Categories consisted of hypothetical stories read by the participant, hypothetical stories read to the participant (e.g., read by experimenter, played from audiotape), video-taped hypothetical stories, hypothetical stories presented through pictures, cartoons or illustrations, hypothetical stories presented through both audio and pictures, cartoons or illustrations, hypothetical stories presented through doll-play, real-time computerized interactions between the participant and a presumed peer or real-time interactions between the participant and a real peer.

*Provocateur's status.* Provocateur's status was coded categorically. Categories consisted of the provocateur in the presented social situation being an unknown peer, a boy or girl from the neighborhood or school, a classmate, a friend, or an enemy of the participant.

### *Child Characteristics*

Child characteristics that were hypothesized to influence the relation between HIA and aggression were operationalized in following manner:

*Sociometric status.* Sociometric status was coded categorically. Categories consisted of effect size was based on an aggressive-rejected sample (samples consisting of aggressive-rejected children) or an aggressive sample (samples where only aggression was measured).

*Function of aggressive behaviors.* Function of aggressive behaviors was coded categorically. Categories consisted of aggression was measured as reactive aggression or aggression measured as a general construct.

*Proportion of ADHD in the sample.* Proportion of ADHD in the sample was coded as a continuous variable representing the proportion of ADHD diagnoses in the sample.

#### *Additional Moderators*

The additional moderators were coded as in the 2002 meta-analysis. Details are provided in Supporting Information.

#### *Inter-Rater Agreement*

To make sure all studies were coded consistently, the studies included in the original meta-analysis were recoded for the present analysis.

To determine inter-rater agreement, 41 randomly selected studies (of 111 studies; 37%) were coded by a second rater. In case of rater disagreement, the two raters discussed the discrepancy and tried to solve this by consensus. In rare cases where no consensus could be achieved, a third rater was asked to solve the discrepancy. Cohens kappa's for categorical variables were calculated and satisfying, ranging from 0.74 to 1.00 ( $M = .83$  and median = .80). Inter-rater reliability of the coding of continuous variables was examined with a two-way random-effect model, absolute agreement, average-measures intra-class correlations (ICCs). ICCs were good ranging from 0.66 to 0.90 ( $M = .79$ , median = .84 and  $SD = .11$ ). Frequency distributions of child- and methodological characteristics are reported in Table 1.

#### *Statistical Analysis*

All study outcomes were transformed into Fisher  $Z$ . Fisher  $Z$  is similar to a correlation coefficient, but corrects for nonlinearity of extreme correlation coefficients. Fisher  $Z$  calculations were derived from reported test statistics and if required test statistics were derived from reported means and standard deviations. Subsequently, Fisher  $Z$  scores were re-transformed into Cohen's  $d$  to facilitate interpretation. According to Cohen (1988), a Cohen's  $d$  of 0.3, 0.5, and 0.8 represents, respectively, a small, medium, and large effect size.

We applied a multilevel modeling approach using the "metafor" package (Viechtbauer, 2010) of the R Statistical Software version 3.0.2. A multilevel

modeling approach allows to derive multiple effect sizes from each study by modeling dependency in effect sizes (Van den Noortgate, López-López, Marín-Martínez, & Sánchez-Meca, 2013). To account for dependency in effect sizes, a three-level meta-analytic model was estimated. A three-level meta-analytic model estimates sample variance for each effect size on Level 1, variance in effect sizes within studies on Level 2, and variance in effect sizes between studies on Level 3 (Hox, 2002; Wibbelink & Assink, 2015). The standard errors of the coefficients in the three-level meta-analytic models were estimated with the Knapp and Hartung (2003) method. Parameters were estimated using Restricted Maximum Likelihood estimation (Wibbelink & Assink, 2015). Analyses were conducted in four steps.

1. We first tested whether the overall mean effect size significantly deviated from zero.
2. Two log-likelihood ratio tests were used to evaluate whether estimating within-study variability (Level 2) and between-study variability (Level 3) in effect sizes significantly improved model fit. Subsequently, the Higgins and Thompson (2002) method was used to demonstrate how much variance in effect sizes was due to sampling variability (Level 1), within-study variability (Level 2), and between-study variability (Level 3).
3. The influence of multiple moderators on the relation between HIA and aggression was analyzed using a multilevel mixed-effect model. Since including multiple moderators in one model inflates the Type II error rate, separate three-level mixed-effect models were fitted for each moderator separately. Subsequently, significant moderators were fitted in a three-level mixed-effect model to address possible confounding among moderators. A multi-model inference approach was used to fit each possible model including none, one, and up to all of the selected moderators to the data and compare the goodness of fit of each model using Akaike information criterion values (see Burnham & Anderson, 1998). This method allows to examine the relative importance of each predictor when taking all possible models into consideration. Dependence in study characteristics prohibited to examine higher order interaction effects, as several combinations of child- and methodological characteristics often occurred and others rarely or never occurred.
4. Fourth, since the previous meta-analysis showed a significant effect of aggression

Table 1  
Moderators of Effect Size (ES) by Severity Classification

		Aggression severity														
All		Nonreferred general				Nonreferred extremes				Clinically referred						
Characteristic and level	No. of studies	ES	N	d	No. of studies	ES	N	d	No. of studies	ES	N	d	No. of studies	ES	N	d
<b>Child characteristics</b>																
<b>Sociometric status</b>																
Aggressive	97	202	28,002	0.30	58	124	22,269	0.28	28	50	4,285	0.31	14	28	1,448	0.43
Aggressive-rejected	15	17	1,270	0.61	1	2	80	0.51	14	15	1,190	0.62	—	—	—	—
<b>Aggression function</b>																
General	98	193	22,502	0.33	50	111	17,259	0.26	38	55	3,877	0.41	13	27	1,366	0.42
Reactive	18	26	6,770	0.36	11	15	5,147	0.40	7	10	1,541	0.27	1	1	82	0.62
% ADHD	7	22	919	$\beta_0 = .39\beta_1 = .00SE = .00t = -0.05p = .95$	—	—	—	—	2	7	249	$\beta_0 = -.62\beta_1 = .01SE = .00t = 1.86p = .122$	5	15	670	$\beta_0 = .53\beta_1 = .00SE = .00t = 0.14p = .890$
<b>Methodological characteristics</b>																
<b>Type of stimulus presentation</b>																
Self-reading	17	30	6,363	0.44	12	22	5,787	0.38	5	8	605	0.64	—	—	—	—
Pictures	6	7	1,340	0.25	3	3	501	0.28	2	3	775	0.11	1	1	64	0.37
Audio	42	82	10,152	0.36	19	50	7,809	0.24	17	21	1,795	0.45	7	11	548	0.58
AudioPictures	14	32	2,756	0.27	8	15	2,027	0.27	6	15	689	0.26	1	2	40	0.17
Video	19	35	3,674	0.23	11	16	2,447	0.20	5	7	595	0.23	4	12	632	0.39
Real-time (physical)	2	3	57	1.33	—	—	—	—	2	3	57	1.33	—	—	—	—
Doll-play	1	3	98	0.27	1	3	98	0.27	—	—	—	—	—	—	—	—
Real-time (computerized)	1	2	75	0.36	—	—	—	—	1	2	75	0.36	—	—	—	—
Unclear	10	25	—	—	5	17	—	—	3	6	—	—	2	2	—	—
<b>Provocateur's status</b>																
Don't know eachother	31	54	6,303	0.29	15	24	4,009	0.25	13	18	1,803	0.33	4	12	491	0.39
From neighborhood/school	37	76	8,369	0.35	18	46	6,427	0.29	17	25	1,722	0.41	3	5	220	0.51
<b>Classmate</b>																
Friend	19	31	4,214	0.41	9	13	2,982	0.26	7	11	984	0.58	3	7	248	0.62
Enemy	7	10	1,396	0.25	5	8	1,143	0.26	1	1	112	-0.13	1	1	141	0.35
Unclear	3	4	724	0.26	3	4	724	0.23	—	—	—	—	—	—	—	—
Total	22	44	—	—	15	31	—	—	4	10	—	—	3	3	—	—
Total	111	219	29,272	0.33	59	126	22,349	0.27	41	65	5,475	0.39	14	28	1,448	0.48

Note. K = number of studies; — = not applicable or not tested; d = Cohen's d; ADHD = attention-deficit hyperactivity disorder.



severity on the relation between HIA and aggression, and to avoid confounding between aggression severity and other moderators, subset analyses were run for each of the three aggression severity groups separately (i.e., nonreferred children with normal aggression scores, nonreferred children with extreme aggression scores, clinically referred aggressive children). Findings from these subset-analyses corresponded to the main study findings and are therefore only reported in Supporting Information.

#### *Publication Bias*

The fail-safe N method is frequently used in meta-analyses (e.g., in the 2002 HIA meta-analysis), but has been criticized for not providing a valid assessment of publication bias and its statistical weakness (e.g., Becker, 2005; McDaniel, Rothstein, & Whetzel, 2006). It remains unclear whether a funnel plot, weighted Egger's test, and the trim and fill method are informative indicators of publication bias in heterogeneous data sets (e.g., Coburn & Vevea, 2015; Van Assen, Van Aert, Nuijten, & Wicherts, 2014).

To handle publication bias we therefore tried to include as many effect sizes derived from unpublished data as possible. This effort resulted in 66 effect sizes derived from unpublished data of 219 effect sizes total (30.1%). Unpublished data were not only operationalized as each effect size derived from unpublished studies, but also as each effect size derived from published studies where additional information needed to be provided by the authors. If publication bias was present it would be expected that effect sizes derived from unpublished data were smaller than effect sizes based on published data. However, the results showed that effect sizes derived from unpublished data were actually larger than effect sizes derived from published data ( $d = 0.40$  vs.  $d = 0.31$ ,  $p = .128$ ) and thereby indicated no effect of publication bias toward null-findings. In addition, using a strict criterion where unpublished data were operationalized as each effect size derived from unpublished studies (e.g., dissertations) showed no indication of publication bias toward null-findings. This strict criterion resulted in 16 effect sizes derived from unpublished studies (7.3%) and results demonstrated that effect sizes derived from unpublished data were significantly larger than effect sizes derived from published data ( $d = 0.54$  vs.  $d = 0.31$ ,  $p = .014$ ).

#### *Funnel Plot*

Figure 2 shows a funnel plot of the effects. Although this was not used as an indicator of publication bias, it allows to evaluate whether there is a pattern in the data. A weighted Egger test demonstrated that effect sizes were not distributed in symmetrical manner across the funnel ( $r_\tau = .16$ ,  $p < .001$ ). Larger studies were mainly distributed around the overall mean effect size, whereas smaller studies were more spread across the funnel. Moreover, the funnel plot demonstrated multiple datapoints fall outside of the funnel, indicating these datapoints show significant heterogeneity in effect size relative to its standard error. However, examining the leverage values and Cook's distance of the datapoints demonstrated none should be considered as outliers or indicate excessive influence on the results. In addition, the funnel plot showed a gap on the bottom left, indicating that relatively large positive effect sizes combined with a large standard error were more often observed than negative effect sizes with a large standard error. A plausible explanation might be that larger positive effect sizes were derived from clinically referred aggressive samples which in general showed larger effects ( $d = 0.48$ ) and consisted of a smaller sample (mean  $N = 103$ ) than studies with nonreferred aggressive samples (respectively,  $d = 0.27$  and mean  $N = 379$ ).

## **Results**

### *Overall Effect Size*

Two hundred and nineteen effect sizes from 111 studies with 29,272 participants were included in this meta-analysis. Figure 3 shows the distribution of effect sizes. One hundred and eighty-six of 219 effects were in the hypothesized direction. The overall weighted mean effect size was  $d = 0.33$ , which significantly deviated from zero,  $SE = .03$ ,  $t(218) = 12.16$ ,  $p < .001$ , 95% CI [0.28, 0.39]. Thus, overall results demonstrated a robustly significant, modest positive association between childhood aggression and HIA.

However, this mean effect size should be interpreted with care, because effect sizes varied significantly between studies. The test for residual heterogeneity of the main-effect model showed there was significant heterogeneity in effect sizes not explained by the model,  $Q(218) = 748.57$ ,  $p < .001$ . In addition, two likelihood ratio tests demonstrated that effect sizes differed significantly within,  $\chi^2(1) = 7.68$ ,  $p = .006$ , and between studies,  $\chi^2(1) = 48.57$ ,  $p < .001$ .

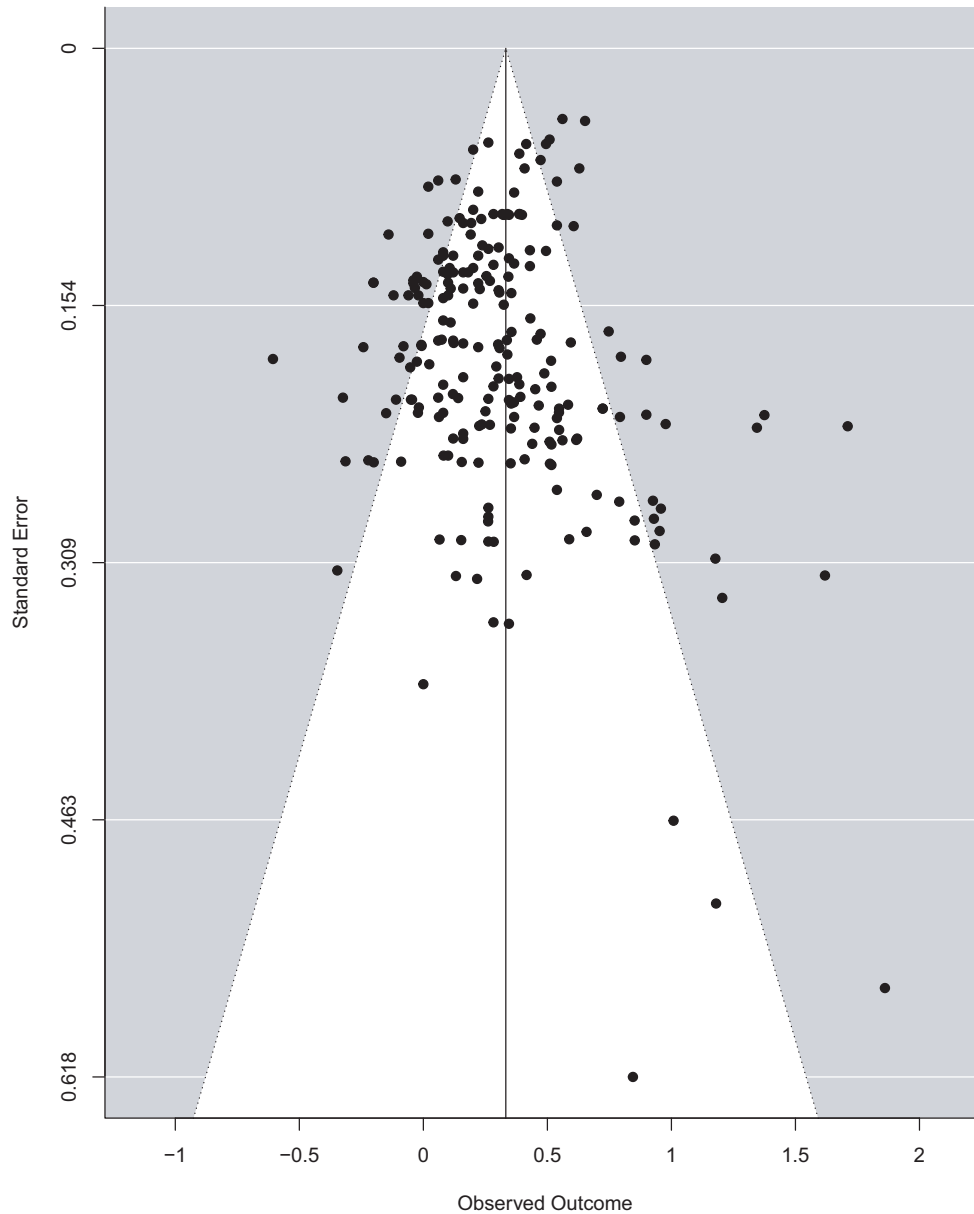


Figure 2. Funnel plot with Fisher's  $Z$  transformed Cohen's  $d$ . On the  $y$ -axis are the standard errors of the effect sizes, with smaller standard errors representing larger sample sizes. On the  $x$ -axis are the associations between childhood hostile intent attribution and aggression.

Subsequently, the distribution of the total variance in effect sizes across the three levels was examined. The percentage of the variance in effect sizes explained by sampling variability was 23.68%. The percentage of the variance in effect sizes explained by differences within studies (within-study variability) was 7.42%. The percentage of the variance in effect sizes explained by differences between studies (between study variability) was 68.90%. The two likelihood ratio tests and test for (residual) heterogeneity indicated that specific child- and methodological

characteristics could possibly explain the variability in effect sizes. Therefore planned univariate moderator analyses were conducted.

#### *Moderator analyses*

The statistics for the test of the moderators ( $Q_M$ ) and statistics for the test of residual heterogeneity ( $Q_E$ ) are reported in Supporting Information (see Table S2). For all the moderators the test of residual heterogeneity was significant, demonstrating there

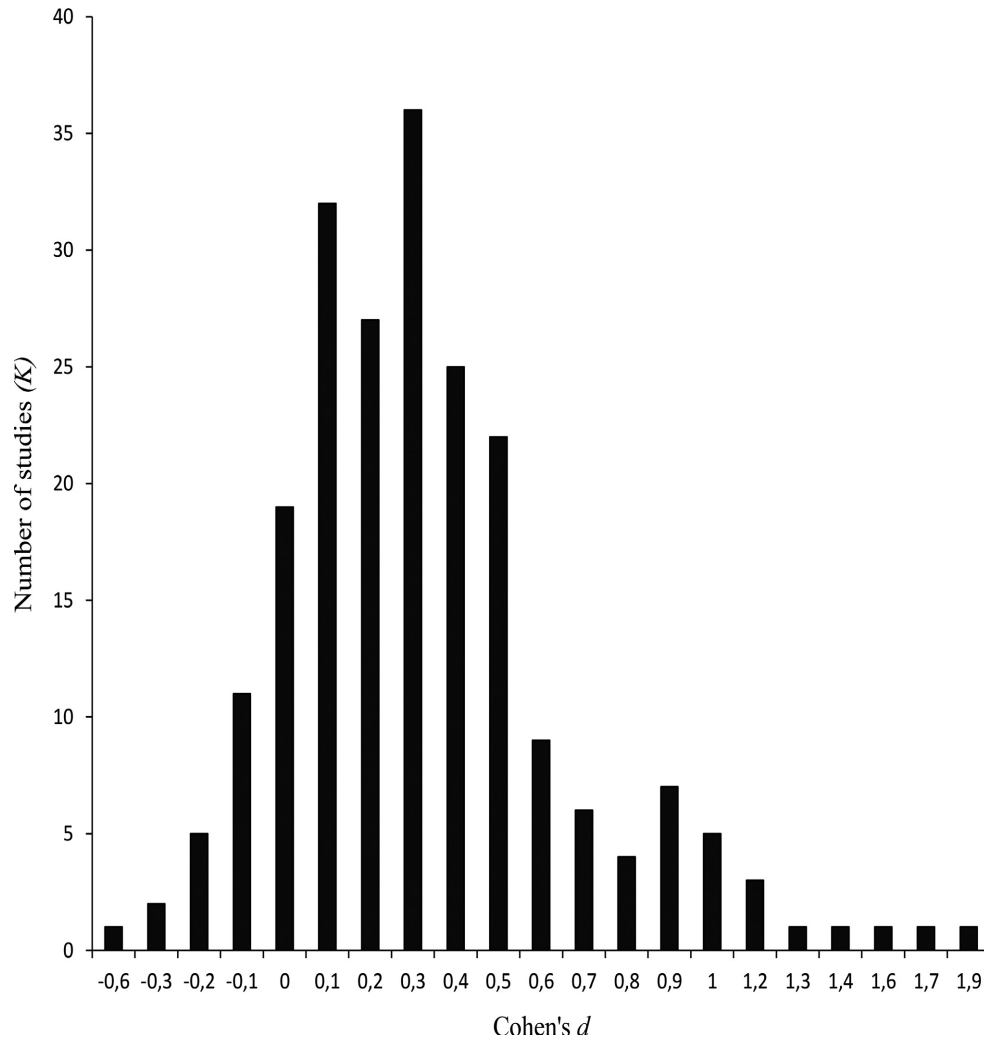


Figure 3. Distribution of effect sizes.

was still unexplained variance in effect sizes beyond each moderator.

#### *Emotional Involvement*

To examine whether effect sizes were dependent on emotional involvement, moderation by type of stimulus presentation was tested. Mean effect sizes derived from self-read ( $d = 0.44$ ), auditorial ( $d = 0.36$ ), pictorial ( $d = 0.25$ ), auditorial and pictorial ( $d = 0.27$ ), videotaped hypothetical stories ( $d = 0.23$ ), and real-time interactions with a real peer ( $d = 1.33$ ) significantly deviated from zero. The mean effect sizes derived from real-time computerized interactions with a presumed peer ( $d = 0.36$ ) and hypothetical stories presented through doll-play ( $d = 0.27$ ) did not deviate from zero, indicating there was no relation between

HIA measured through these types of stimulus presentation and aggression. The mean effect size of HIA measured through real-time interactions with a real peer was significantly larger than the mean effect sizes of all other types of stimulus presentation (vs. self-read,  $p = .013$ ; vs. auditorial,  $p = .006$ ; vs. pictorial,  $p = .004$ ; vs. auditorial and pictorial,  $p = .003$ , vs. videotaped,  $p = .002$ ; vs. real-time computerized interactions with a presumed peer,  $p = .033$ ; vs. doll-play,  $p = .016$ ). The mean effect size of HIA measured through self-read hypothetical stories was significantly larger than the mean effect size derived from videotaped hypothetical stories ( $p = .024$ ). The coefficients for the type of stimulus presentation are reported in Supporting Information (see Table S3). Thus, in line with our hypothesis, results on the type of stimulus presentation indicate that the strength of

the relation between HIA and aggression increased with the level of emotional involvement the social situations elicited.

#### *HIA Toward Familiar Versus Unfamiliar Others*

To examine whether the relation between HIA and aggression is present in situations with both familiar and unfamiliar others, but stronger in situations with disliked others encountered in previous problematic social encounters, moderation by provocateur's status was tested. Results showed that the relation between HIA and aggression significantly deviated from zero for all types of provocateur's status ( $d = 0.25-0.41$ ). However, no differences between types of provocateur's status were found ( $p = .539$ ). Thus, contrary to our hypothesis, results on the provocateur's status indicate that the strength of the relation between HIA and aggression was not dependent on the familiarity of peers.

#### *HIA in Aggressive-Rejected and Aggressive Samples*

To examine whether the relation between HIA and aggression is present in aggressive-rejected and aggressive samples, moderation by sociometric status was tested. The mean effect sizes of aggressive-rejected samples ( $d = 0.61$ ) and aggressive samples ( $d = 0.30$ ) both significantly deviated from zero. Results showed that in both kinds of samples there was a small to moderate positive association between HIA and aggression. In addition, the mean effect size of aggressive-rejected samples was significantly larger than the mean effect size of aggressive samples ( $p < .001$ ). The coefficients for sociometric status are reported in Supporting Information (see Table S4). Thus, in line with our hypothesis, results indicate that the relation between HIA to peers and aggression existed irrespective of the sociometric status of participants, and was stronger for children who are both aggressive and rejected.

#### *HIA and Reactive Aggression*

To examine whether the relation between HIA and aggression is stronger for reactive aggression, moderation by function of aggression was tested. Results showed that the relation between HIA and aggression significantly deviated from zero for both reactive aggression ( $d = 0.36$ ) and aggression measured as a general construct ( $d = 0.33$ ). However, no differences between the types of aggression

function were found ( $p = .602$ ). Thus, contrary to our hypothesis, results indicate that the relation between HIA and aggression was not stronger for reactive aggression than for aggression in general.

#### *HIA and Proportion of ADHD Diagnosis in the Sample*

To examine whether the strength of the relation between HIA and aggression increased with the proportion of ADHD diagnoses in the aggressive sample, moderation by ADHD comorbidity was tested. The association between HIA and aggression was not dependent on the percentage of ADHD diagnoses in the sample ( $p = .958$ ). Thus, contrary to our hypothesis, results indicate that the strength of the relation between HIA and aggression did not increase with the proportion of ADHD comorbidity in the aggressive sample.

#### *Exploratory Analyses of Moderators*

Consistent with the findings in the meta-analysis of De Castro et al. (2002), effect sizes in the current meta-analysis were larger in samples with more severe behavioral problems. Moreover, aggression assessed by a staff-member was associated with higher effect sizes than all other types of informants, except for aggression assessed by an observer. In addition, results demonstrated that effect sizes were larger when more reliable HIA measures were used. For the other exploratory moderators no effects were found. For details see Supporting Information.

#### *Multi-Model Inference: Selection of Moderators*

To examine whether moderators explained significant variance in effects size over and above the effects of other moderators, we used a multi-model inference approach. This procedure resulted in 74 effect sizes (of 219) used for estimating all possible models. Results demonstrated that moderators were too confounded to distinguish unique effects of moderators when multiple models were taken into account (see Supporting Information for details).

## **Discussion**

Social-cognitive theories propose a relation between HIA and aggression in children and specific moderators of this relation. This meta-analysis found an overall modest positive association between

childhood HIA and aggression (mean effect size  $d = 0.33$ ). However, this mean effect size should be interpreted with care, because effect sizes varied significantly between studies. As expected, the relation between HIA and aggressive behavior was found to be stronger in emotionally engaging situations, and not to be limited to interactions with known peers, nor to rejected-aggressive children, nor to reactive aggression, nor to a comorbid ADHD diagnosis. In line with the previous meta-analysis (De Castro et al., 2002), results showed that the association between childhood HIA and aggression is stronger in more severely aggressive samples. In addition, the exploratory moderator analyses demonstrated that the strength of the association between HIA and aggression was dependent on the reliability of the HIA measures and the type of informant to assess aggression.

We tested specific hypotheses about moderators of the relation between HIA and aggression in children. The first hypothesis stated that the relation between HIA and aggression is stronger in emotionally engaging situations. In line with our hypothesis, effect sizes derived from real-time interactions with a real peer were very large ( $d = 1.33$ ), and significantly larger than for other types of stimulus presentation. However, it should be mentioned that only three effect sizes derived from two different studies concerned real-time interactions with a real peer. Almost 98% of the effect sizes were derived from studies using hypothetical stories to measure HIA. Although hypothetical stories were presented in different formats (e.g., audiotaped, videotaped), their effect sizes were relatively small ( $d = 0.23$ – $0.44$ ). The findings seem to be in line with SIP models that postulate that HIA in aggressive children is particularly present in personally involving and emotionally engaging situations (Dodge, 1991).

Methodologically, it is important to note that results only showed a large effect for real-time interactions with a real peer and not computerized real-time interactions with a presumed peer. A plausible explanation could be the lack of observations for computerized real time interactions (two effect sizes from one study), which could have resulted in an unreliable estimate of the true effect size. Another explanation could be that this study assessed computerized real-time interactions with a presumed peer through a race-car game (Yaros, Lochman, Rosenbaum, & Jimenex-Camargo, 2014). This type of stimulus presentation might not have elicited sufficient levels of emotional engagement to evoke strong HIA, because the peer's behavior may

have been considered legitimate in the gaming context. In sum, the findings on the type of stimulus presentation suggest that particularly social interactions that evoke sufficient emotional engagement elicit the automatic and emotional processes that activate HIA. This finding has implications for clinical practice, since it implies that CBT should assess and target HIA in emotionally engaging situations.

The second hypothesis stated that the relation between HIA and aggression is present in social situations with both unfamiliar and familiar others. In addition, we expected this relation to be stronger in situations with disliked others encountered in previous problematic social situations. Results demonstrated that the relation between HIA and aggression was present irrespective of the provocateur's familiarity. Results did not show that the relation between HIA and aggression was stronger in social situations with disliked others who children had encountered in previous problematic social situations. This finding might suggest that HIA is not context-specific. However, another explanation could be the lack of observations (four effect sizes from three studies) on HIA toward disliked others encountered in previous problematic social interactions, which could have resulted in unreliable estimates. Nonetheless, the findings seem to be in line with social-cognitive theory that proposes that the tendency to attribute hostile intent toward others derives from a general cognitive disposition toward both known and unknown others. For clinical practice this implies that CBT interventions could target a general cognitive disposition to establish significant and prolonged changes in SIP and subsequent behaviors across a wide range of contexts.

The third hypothesis stated that the relation between HIA and aggression is present irrespective of the sociometric status of participants, yet would be particularly pronounced in aggressive-rejected samples. Results showed support for this hypothesis and demonstrated that the relation between HIA and aggression was present in both aggressive-rejected and generally aggressive samples, however, was stronger in aggressive-rejected samples. This finding supports the assumption that HIA derives from a general cognitive disposition that guides information processing across a broad range of contexts. In addition, since our meta-analysis only included studies that used social situations with peers to measure HIA, the finding that the relation between HIA and aggression was stronger in aggressive-rejected samples might indicate that the relation between HIA and aggression is stronger

in situations that match specific memories of rejection by peers. For clinical practice this implies that CBT could possibly be more effective when HIA is targeted in contexts similar to specific memories of aversive social experiences.

The fourth hypothesis stated that the relation between HIA and aggression is stronger when aggression is operationalized as reactive aggression. Results did not support this hypothesis and demonstrated no difference in effect sizes based on aggression measured as reactive aggression or as a general construct. An explanation could be the method used for the coding of this variable. Since empirical research suggests that the majority of aggressive children (Dodge, Lochman, Harnish, Bates, & Pettit, 1997) to some extent engage in reactive aggressive behaviors, it may well be true that a substantial part of the samples where aggression was measured as a general construct, were primarily reactive- or reactive-proactive samples. This could have caused the null-result for this hypothesis. Another explanation could be that the relation between reactive HIA and aggression was based on 26 effect sizes and only one of these effect sizes was derived from clinically referred aggressive samples. Since aggression severity seems to contribute to the strength of the relation between HIA and aggression it would be expected that the relation between HIA and reactive aggression is particularly strong in clinically referred aggressive samples. Although the one effect size derived from clinically referred aggressive samples was relatively large ( $d = 0.62$ ), a lack of observations prohibits from drawing firm conclusions.

The fifth hypothesis stated that the relation between HIA and aggression is stronger in aggressive samples consisting of children with ADHD. Results did not support this hypothesis and demonstrated no effect of ADHD on the relation between HIA and aggression. However, only 22 effect sizes (10%) were based on samples where the presence of a ADHD diagnosis was measured and the majority of these samples were not full-ADHD samples. The lack of observations on ADHD comorbidity could have caused a lack of power to detect true effects and thereby the null-findings for this moderator. Another explanation could be that deficits in cognitive capacities in ADHD children are similar to deficits in cognitive capacities in aggressive children.

Exploratory analyses showed that the strength of the association between HIA and aggression significantly increased with higher Cronbach's  $\alpha$  reliability. Cronbach's  $\alpha$ 's were reported for only 97 of 219 effect sizes and ranged from 0.37 to 0.94, with a

mean of .73. In addition, since more than half of all effect sizes were derived from studies that did not report a Cronbach's  $\alpha$  for the HIA measure, it remains unclear how the reliability of the HIA measure influenced effect sizes in these studies. It could be that at least several studies that did not report a Cronbach's  $\alpha$  for the HIA measure, used an unreliable instrument to measure HIA and thereby reduced effect sizes. Thus, despite emphasis put on the importance of reliability of HIA measures in the previous HIA meta-analysis, still less than half of the studies included reported a Cronbach's  $\alpha$ . This seems cause for worry, as clinical decision making should not depend on unreliable measures or idiosyncracies of particular vignettes chosen to assess HIA. The finding that larger effect sizes were associated with a higher Cronbach's  $\alpha$ , emphasizes the importance for clinicians and researchers to only use reliable instruments to adequately measure HIA.

Exploratory analyses also demonstrated that the type of informant to assess aggression in children moderated the association between aggression and HIA. Results showed that aggression assessed by a staff-member yielded larger effect sizes than aggression assessed by all other type informants, except for aggression assessed by an observer. The latter might be due to a lack of observations ( $k = 2$ ). A plausible explanation for the fact that effect sizes were larger in studies where aggression was assessed by a staff-member might be that all these studies ( $k = 5$ ) were performed in clinically referred aggressive samples. Since results demonstrated that the severity of aggressive behavioral problems contributes to the strength of the association between childhood aggression and HIA, the larger effect sizes for aggression assessed by a staff-member might be explained by the severity of aggressive behavioral problems for this subgroup.

Although the univariate moderator analyses demonstrated that several moderators influenced the relation between childhood HIA and aggression, a multi-model inference approach to combine these moderators was not feasible. An explanation might be that there was a strong interdependence between child- and methodological characteristics, where specific combinations of child- and methodological characteristics frequently, rarely, or never occurred (e.g., real-time interactions for clinically referred aggressive samples). As a result, moderators were too confounded to distinguish unique effects of moderators when taking multiple models into account. Moreover, results demonstrated that the predictors that yielded the largest effect sizes

consisted of relatively few observations. For example, only 28 effect sizes (12.8%) were derived from clinically referred aggressive samples, 17 effect sizes (7.8%) from aggressive-rejected samples and only three effect sizes (1.4%) from real-time interactions with a real peer. The lack of observations on the strongest predictors could also be an explanation for the fact that a model without moderators included best fitted the data.

The large amount of residual heterogeneity seems to suggest that we did not capture important moderators of effect yet. Perhaps surprisingly, SIP theory is more specific about moderators of HIA performance than current research methods capture. For example, this meta-analysis did not examine the effect of several demand characteristics of HIA tasks that are implied by SIP theory. Cognitive capacities are considered key moderators of SIP (e.g., Dodge & Pettit, 2003) and tasks to measure HIA may inadvertently differ in the cognitive capacities they require for children. For example, to understand the task and to indicate that they do not interpret intentions as hostile (e.g., by requiring complex words like “accidental” or “unintended”) or the amount of working memory understanding a task requires (e.g., remembering that you were the actual target child in the vignette while watching a video). In the current meta-analysis too few studies assessed executive functioning (e.g., working memory) and this prohibited from adequately testing the effect of this moderator. Therefore this meta-analysis used IQ as an indicator of cognitive abilities. However, this moderator did not show an effect. Nonetheless, given that children differ greatly in cognitive abilities, the presumed role of cognitive abilities in SIP, and the methods used to measure HIA varied considerably between studies, it could be that this influenced the results. Systematically studying (and varying) such test characteristics would be highly informative in understanding the roles of cognitive functioning in HIA.

Another moderator that was not measured in this meta-analysis was social desirability. Since 98% of the effect sizes were based on paper-pencil hypothetical stories to measure HIA, it could be that social desirability influenced participants' responses in studies using hypothetical stories. More specifically, it could be that using a paper-pencil format in an individual or group-based setting reminds children of an exam or test and therefore children may feel more reluctant to give socially undesirable answers. Another moderator that was not measured and could have influenced results is socioeconomic status (SES). Research indicates that low SES is

associated with chronic stressors such as parental psychopathology, deprived neighborhoods, and social isolation (Baum, Garofalo, & Yali, 1999; Pinderhughes, Nix, Foster, & Jones, 2001). From a schema-theory perspective it can be assumed that these chronic stressors contribute to the development and maintenance of hostile schemata and thereby HIA (Nas, De Castro, & Koops, 2005). In this meta-analysis, 137 effect sizes (63%) from 69 studies (62%) were based on samples from the United States, a nation with large socioeconomic inequalities (e.g., gini index; Central Intelligence Agency, 2009). It could be that effect sizes depend on the magnitude of variance in SES both within and between samples. Unfortunately, an insufficient number of studies ( $k = 5$ ) included in the current meta-analysis measured SES and this prohibited from adequately testing the effect of this moderator.

#### *Strengths and Limitations*

An important strength of this meta-analysis is that it included studies from over 40 years of research on the relation between childhood HIA and aggression, and applied a multilevel modeling approach to analyze results. Multilevel model analyses allow to correct for dependency in effect sizes within studies and thereby allows to derive multiple effect sizes per study (Van den Noortgate et al., 2013). This resulted in 219 effect sizes based on the relation between aggression in children and HIA. In addition, this meta-analysis not only examined the overall relation between childhood HIA and aggression, but also examined specific theory-driven moderators of this relation. Thus, we obtained findings that inform our understanding of when and how HIA is related to aggression, with clear implications for the nature of HIA.

An important limitation of this meta-analysis is the strong interdependence between study characteristics. In other words, many studies used similar methodologies to measure HIA and aggression. As a consequence, specific combinations of child and methodological characteristics frequently, rarely, or never occurred. The lack of observations for various specific combinations of child and methodological characteristics might have contributed to confounding of moderators when included in one model. This made it impossible to disentangle specific effects of certain child and methodological characteristics. A second limitation is that publication bias was only addressed through one method. This method yielded no indication for publication bias toward null-findings, and the fact that effect sizes from unpublished

data were larger than effect sizes from published data could suggest true effect sizes in this meta-analysis were actually underestimated. More certainty about publication biases could be attained when multiple methods for testing publication bias become available for multilevel meta-analyses.

#### *Future Recommendations and Implications*

The significant amount of residual heterogeneity emphasizes the need for theory development and research on the effects of specific combinations of child- and methodological characteristics on the relation between childhood HIA and aggression. Therefore, future research may focus on testing a variety of child and methodological characteristics that are not frequently measured to date. To examine the effect of emotional engagement, researchers could manipulate the level of emotional engagement across presented social situations and directly compare HIA in real-time interactions and HIA as assessed through hypothetical stories using a within-subjects design.

In addition, context specificity of HIA seems to deserve more attention because of its relevance to intervention. To further examine the effect of social experiences on SIP in different contexts, future studies may link experiences in specific contexts (e.g., harsh parenting and peer rejection) prospectively to HIA in the same and differing contexts (e.g., with peers or adults) and manipulate the provocateur's status (e.g., unknown, friend, enemy) and type of context (e.g., provocation, peer entry, expectation, failure, unjust punishments). This would allow to evaluate whether the relation between HIA and aggression is stronger when the current social situation matches specific memories of previous aversive social experiences.

Last but not least, the current analysis did not address malleability of HIA and its effects on aggressive behavior. Experimental research on moderators of the relation between HIA and aggression may go hand in hand with experimental micro trials testing specific ways to reduce HIA. Recent studies suggest that HIA may be reduced with relatively simple means, such as implicit cognitive bias modification (Penton-Voak et al., 2013) or parental instructed story reading (Van Dijk, Poorthuis, Thomaes, & De Castro, 2018). Such experimental manipulation of HIA may help understand the dynamics of HIA and simultaneously inform effective intervention.

#### *Conclusion*

In sum, the meta-analytical findings indicate that HIA is a general cognitive disposition that guides information processing across a broad variety of contexts, including interactions with unknown peers. The relation between HIA and aggression is stronger in social situations that elicit sufficient emotional engagement and for more severely aggressive children. In addition, the relation between HIA and aggression depends on the reliability of HIA measures, but is not stronger for reactive aggression or proportion of ADHD diagnoses in the samples. Future research will further our understanding of this key variable in the development of aggressive behavior.

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