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# An evolutionary model for aggression in youth: Rethinking aggression in terms of the Catalyst Model

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ABSTRACT

Like many areas of psychology, aggression research has been impacted by psychology's replication crisis. Until recently, aggression was understood largely from the perspective of social cognitive models, wherein aggression was conceptualized as learned scripts. However, more recent rigorous research has suggested that cognitive scripts may play only a small role in aggression. By contrast, genetic and evolutionary inputs are powerful and combine with both developmental and situational life stress to increase the risk for serious aggression. This article examines aggression from the Catalyst Model and how diathesis-stress approaches to understanding aggression can help reduce error in predicting clinically significant aggression. This article also addresses related issues of measurement error and smallest effect size of interest (SESOI) as they relate to aggression research.

Scholars have been interested in the topic of aggression for decades. Yet aggression research, like so much else in psychology, has found itself embroiled in the replication crisis and aggression as we are coming to understand it today looks quite different from how we conceptualized it ten years ago. This is in part because of the decline of some theories, the survival of some others, and even the resurgence of some. We can now synthesize available information as part of an overarching framework that includes data from genetics and evolutionary science along with our understanding of how trauma and stress can exacerbate underlying proclivities. We're also beginning to more fully understand the foibles of our field, how easily discussions of aggression can slip into moralizations and even authoritarian, censorious policy endorsements, as well as problems of measurement, conceptualization, and a tendency to ignore the importance of effect sizes. This article is designed to be a part of moving this field forward, both in understanding the past, but also in guiding us toward the future wherein aggression is more clearly understood as an evolutionary, motivational process rather than a mechanistic one based in priming and social cognition. I begin by discussing some of the systematic issues that have caused misunderstanding of aggression before turning to an evolutionary motivational model of aggression.

## 1. The limits of past science and theory

### 1.1. What do we mean by aggression?

First, we must fully grapple with the concept we intend to deal with. It's worth noting the typical definition of aggression as behavior produced to cause physical harm or humiliation to another person who wishes to avoid it (Baron & Richardson, 1994). Overall, this definition is not a bad one, despite it has some limitations. However, the *intent* part of this definition is critical, as it must be if we're to fully understand aggression.[1] This acknowledgement of the definitional importance of *intent* has been controversial for some concepts such as *microaggressions.* Some scholars have asserted the microaggression concept is misnamed given it incorporates both behaviors with aggressive intent as well as those without such intent (Lilienfeld, 2017). Nonetheless, as a motivational concept, intent is fundamental to our concept of aggression.

Some may disagree with this focus on intent. For instance, in some circumstances aggressors may be able to convince themselves of good motives. As an extreme example, the owner of a slave, anywhere across the world, may physically mistreat their slave. Doing so might be rationalized as helping the slave to "learn" or civilizing them in this sense,

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[1] I'm aware this runs counter to much of the social narrative on the political left which has begun to eschew intent as critical in understanding the impact of behavior. However, I argue this has been largely a mistake, one likely to do more harm than good (and itself possibly aggressively motivated). For a nuanced view of the current social narrative see Bloom, 2021.

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convincing oneself that the aggression is a benefit to the one receiving it. This certainly points to the complexity of human motivations, and the power of rationalization to reduce guilt. However, the evident nature of the injuries (physical or social) imposed and the obvious benefit to the aggressor I believe mitigates this to a nuance rather than a counterexample. This differs from the microaggression of saying "America is a melting pot" where the harmfulness of such a statement is not in clear evidence.

Although the Baron and Richardson definition provides a useful starting point, as I have argued before (Ferguson & Beaver, 2009) it does reflect a potential bias in assuming that aggression is inherently bad. My concern is that, from such a definition, it may be assumed that aggression has no adaptive function and is always pathological and undesirable. In reality, aggression likely exists on a continuum, from its complete absence (likely a bad outcome) through the euphemistically named *assertiveness* (related to moderate levels of aggression), through aggression that is clinically significant and pathological, doing unwarranted harm to others. In moderate doses, aggression may very well be adaptive, guiding individuals toward many behaviors approved of by society including standing up for one's beliefs, assertiveness, defending others in need, careers in law enforcement, the military, business, legal affairs, etc., sporting activities, political involvement, debate and discourse indeed including scientific debate (Hawley & Vaughn, 2003; Smith, 2007). Thus, it may be helpful to employ a slightly less moralistic definition of aggression, namely: "Behavior which is intended to increase the social dominance of the organism relative to the dominance position of other organisms**, or** which is defensively necessary to increase survivability." This takes an evolutionary perspective suggesting that aggression is, in proper doses, related to social status and reproductive success (Jokela & Keltikangas-Järvinen, 2009). From this understanding, and stripped of moralistic language, we can see that when we most often speak of aggression as a social or personal ill, it is *pathological* aggression we intend to reference, even when it is not always clear in many studies that this is what we have measured. Almost every article on aggression contains the implicit implication it is *bad* even if they make no empirical effort to demonstrate this, so let us acknowledge that much about aggression is probably *good* for both individuals and societies, but it's the *bad* stuff we're mostly interested in.

The line between aggression that is adaptive and pathological is ultimately likely to be gray. In this sense, I use pathological to imply aggression that carries a high risk to the individual (physical or social), or does unwarranted harm to another (harm that is not necessary to increase survivability or status). That social sanction is one potential risk ultimately makes the concept of pathological aggression dependent upon social context, creating inevitable fluidity on the margins. Consider, for example, the behavior of rape. Rape can be considered an adaptation, particularly for men who have difficulty achieving consenting partners (Thornhill & Palmer, 2000). Rape is morally repugnant and, at least to some degree (albeit too often out of consideration for family honor and property rights than women's trauma), historically prohibited within most cultures, definitions of what constitutes rape shift between cultures and over time. Rape of women during times of war, for instance, often carry little risk, going unpunished (in some cases, societies may have allowed men to keep war captive women). Is rape not pathological, then, in circumstances where there is little risk to the male and his society may even sanction the behavior in question?

Indeed, it is worth noting there is no diagnosis for rape behavior in the Diagnostic and Statistical Manual, though presumably such behavior would fall under Antisocial Personality Disorder. Nonetheless, though recognizing this is a fair point for debate, I would argue most rape scenarios fall within the *unwarranted* harm clause as they involve a wanton disregard and cruelty toward an individual whose suffering is immeasurably higher than any gain acquired by the rapist.

Bullying man be another complex example. Such behavior may be adaptive to the degree it enhances social status among peers. However, it may also come at a reputation cost among some members of the community who resent the aggression. Such behaviors naturally remain a concern to educators and parents, given the impact on victims, even if outcomes for perpetrators are largely positive. Thus, some acts of aggression may include both adaptive and pathological qualities.

This sense that social response to aggression is important to the distinction of pathological aggression can be noted in the development of aggression in children (e.g., Hawley, 1999, 2014). Prosocial and aggressive behavior may often be perceived as opposites, both pathologically and morally, though aggression may be employed on behalf of the community. Aggression may be employed to take resources away from the community for oneself or to take resources from others for the community. Aggression which benefits the community, in particular, may increase the social standing of the individual and be adaptive, whatever its cruelty toward others. Thus, complexity in this distinction is certainly recognized.

### 1.2. The mismeasurement of aggression

Perhaps one of the most enduring issues for aggression research has been the difficulty in knowing how to properly measure it. This is particularly true in laboratory studies understandably limited legally and ethically in how much "harm" people can be asked to apply to others . As such, laboratory studies have been particularly hampered by both standardization and validity issues, though survey studies are by no means immune to similar concerns.

Laboratory studies typically rely on individuals giving out bursts of white noise (or more extreme, electric shocks), putting hot sauce in people's food or putting people's hands in buckets of ice water. Though such instruments can be interesting, there are certainly gulfs between these behaviors and the assaults, homicides, and rapes of particular interest to society. These problems have been known for decades (e.g., Tedeschi & Quigley, 1996) and yet little effective progress has been made regarding a "better mousetrap" of a well-standardized, clinically validated measure of laboratory aggression which predicts serious real-world aggressive behavior. The best we might say is that these measures might relate to some variant of prank-level aggression such as people might play on one another on April Fool's Day.

Such measures also have problems with lack of standardization, meaning that, in particular, aggression might be extracted from them multiple ways to the advantage of a researcher's hypothesis. This gives researchers undue flexibility to choose outcomes that best fit their hypotheses and ignore those which do not. Perhaps most notorious on this level is the Competitive Reaction Time Test (CRTT), a game-like scenario in which participants believe they are playing a reaction-time game against a (fictional) opponent. They are told they can give bursts of white noise (or electroshocks in more extreme variants) as punishment for their opponent losing. The white noise can vary in terms of loudness or duration. This sets up two outcomes (loudness and duration), but then it becomes unclear if only the first trial (of 25) should be considered, or all 25, or only trials after the opponent wins or opponent loses. Should the whole 10-point scale for loudness and duration be considered or should a cut-off of, say 7 and above be considered "aggressive"? One could even do strange things such as take the square root of the duration score and multiplied this by the intensity score (e.g., Carnagey & Anderson, 2005). On and on we might go. One analysis of 130 aggression papers found that the CRTT had been used in 157 separate ways to measure aggression, with numerous scholars using it differently between papers *in their own lab* with little justification (Elson, 2016). Using the same sample, these disparate outcomes from the CRTT could be used to either support or challenge a hypothesis depending upon the researcher's whim or prior expectations (Elson et al., 2014).

Evident demand characteristics also limit the validity of many aggression studies. Participants who are able to guess study hypotheses

may alter their behavior, causing false positives. These concerns have now called into question the validity of findings of famous experiments such as the Stanford Prison Experiment (Resnick, 2018) or the Milgram studies of obedience (Perry et al., 2023). In effect, results were driven by participant hypothesis guessing (or outright experimenter coaching in the case of the Stanford Prison Experiment), not real behaviors. I submit that even Bandura's bobo doll studies are likely to be the result of the kids interpreting the videos as instructions, not actual aggressive motivation and should cease being taught as if they were meaningful aggression studies.[2] Distractor tasks can help reduce hypothesis guessing though, unfortunately, they are used in few studies.

Survey-based studies may also sometimes use ad-hoc surveys which may be unstandardized or have unknown clinical validity. However, measures such as the Child Behavior Checklist (CBCL) or Aggression Questionnaire (AQ) are at least available. Yet, here too, it's not always known when a change in aggression is clinically meaningful. Let's say, for instance, a prime for aggression is administered to a group of college students, generally a low aggression population. They are then administered a survey measure of aggressive intentions, with a 5-point Likert scale, 5 being most aggressive. A mean item score of 4 out of 5 would indicate clinically significant aggression. In a sample for this experiment, the prime increases aggression from a mean score of 1.4 among these college students to 1.8, with a sample large enough to make this "statistically significant." But is it of clinical significance if all scores remain beneath the clinical cut-off of 4.0? Too often, aggression studies are guilty of implying clinical significance when they have established no such thing (Markey et al., 2015). Indeed, for a group low in aggression, an increase might actually be *beneficial* (i.e., more assertive), but we don't know without careful validity testing.

### 1.3. Overreliance on social cognitive models

There have been multiple models advanced, from frustration-aggression, to catharsis, to explain aggression. Yet, since at least the 1960s, social cognitive models have held sway through much of psychology. Here, I argue, this has been a critical mistake.

Social cognitive models of aggression have their roots in the old bobo doll studies by Bandura, who advanced the notion that aggression could be learned by watching others (e.g., Bandura et al., 1989). Yet, this error may have begun in thinking that what was witnessed in the bobo doll studies was the implanting of aggressive motivation rather than simply young children attempting to follow instructions. This original error, the failure to see the obvious demand characteristics in the bobo doll studies for what they were (see Tedeschi & Quigley, 1996 for in depth criticisms of the bobo paradigm), has likely done much to misinform our understanding of aggression ever since. Put bluntly: Bandura's bobo dolls should probably no longer be taught as if they inform us much of anything about aggression.

The most current iteration of social cognitive models of aggression are the General Aggression Model (DeWall & Anderson, 2011) and its cousin, the General Learning Model (Prot et al., 2015). Advocates of such theories hardly restrain them to aggression, but eagerly apply them to "violent evil" and heroism (Anderson & Carnagey, 2004), terrorism and torture (DeWall & Anderson, 2011), suicide and even climate change (Dewall, Anderson & Bushman, 2011). Briefly, these models suggest that aggression (and other behaviors) are primarily learned through modeling and repetition which, in turn, increase the frequency of such behaviors in the future. Though such models may mention affective, personality or biological inputs to aggression, they rarely elucidate the same and typically portray aggression as a largely cognitive, mechanistic learning process. Such theories are primarily, though not exclusively, applied to media violence studies (by contrast, and perhaps tellingly, they are virtually absent from criminal justice research).

To date, evidence has increasingly failed to support social cognitive models of aggression (e.g., Ballard et al., 2012; Kuhn et al., 2019; Sauer et al., 2015). This appears to be particularly true in preregistered and more rigorous studies (e.g., Drummond et al., 2020). Further, such models remain largely naïve to evolutionary and biological inputs to aggression. Coupled with the critical failure to truly perceive the causal mechanisms of compliance with instructions in the original bobo doll studies, it is probably time to cease the use of social cognitive models of aggression as theoretical rationale for empirical studies.

### 1.4. Smallest effect size of interest (SESOI)

The last issue I wish to discuss before moving more clearly into theoretical and motivational issues is that of SESOI. This has been a struggle for psychology, one which I submit is on par with the replication crisis insofar as it relates to validity of findings and conclusions in psychology (though unlike the replication crisis there is no sense of improper scientific behavior.) Mainly, this is the issue of what effect sizes are capable of being distinguished as "true" effects versus the noise of messy social science that comes from response unreliability, demand characteristics, single-responder bias, survey priming, mischievous responding and a host of other issues that are likely to cause false positive statistically significant results that are actually noise, particularly in large samples with low sampling error.

This can cause great confusion about whether a small effect size is sufficient to support a given hypothesis. For instance, in one recent meta-analysis on video game violence and aggression (Prescott et al., 2018), found a very small longitudinal relationship with other factors controlled ($r = 0.078$) which was statistically significant. The authors interpreted this as hypothesis supportive.[3] However, a subsequent reanalysis of much of the same data (Drummond et al., 2020) concluded that most of this weak effect could be attributed to the noise from poor study designs and in studies with best practices, the effect was negligible ($r = 0.012$). This can be contrasted against another meta-analysis on aggression and empathy (Vachon et al., 2014), wherein the relationship between aggression and empathy was calculated at $r = .11$, larger than either of the two video game metas, yet interpreted as largely non-hypothesis supportive.

These conflicts indicate clear confusion about what effect size is meaningful. How could an $r = 0.078$ be meaningful if $r = 0.11$ is not? One might argue that the context of the research question matter, but this feels like a pat dismissal of a serious (and self-serving) problem in psychology. In a recent analysis (Ferguson & Heene, 2021), the issue of "noise" in aggression research was examined in two large datasets, by looking at the effect sizes of nonsense relationships (correlations between aggression and other variables with no theoretical relationship with aggression). It was found that "statistically significant", but "noise" effects were quite common below effect size $r = .10$. This means it may be virtually impossible to distinguish true effects from noise below this value. Some noise effects were larger than $r = 0.10$, though very few far exceeded 0.20. From this, it is probably the case that effect sizes below $r = 0.10$ should not be interpreted as hypothesis supportive even if statistically significant, and those between 0.10 and 0.20 interpreted with caution. This is probably true throughout psychology, but we can say pretty clearly that for aggression research, the methods are not precise enough to distinguish true effects from noise below $r = 0.10$ and may struggle to do so for effects below $r = 0.20$. Measurement precision, naturally, is a factor in these concerns, but

[2] I once asked Dr. Bandura and his personal assistant for any data, notes, or records of the original experiments, but received no reply.

[3] It is worth noting that, due to their massive power, meta-analyses are almost always "statistically significant" and, for meta-analyses, p-values should probably not be used to indicate hypothesis support.

these recommendations are likely to be generous given the precision common in *good* social science research.

## 2. A diathesis stress model of aggression

From the first part of this article, to summarize, I suggest that social science has relied too heavily on mechanistic social cognitive learning models of aggression. These models are weakened by the limited data to support them as well by their amotivational approach, implying that aggression acts as a mechanistic cognitive script. Some theorists may object to this characterization, but the simple truth is most such theories make scant mention of motivation and their leading proponents treat aggression quite literally as if it worked like the impact of cigarettes on lung cancer (see Markey et al., 2015 for quotes). My intent is not to say learning plays *no* role in aggression, but rather that psychology has treated it like a hammer hypothesis with everything beginning to look like a nail.

In the second part of this article, I offer an alternative, one I feel is better situated in the data, one which is more applicable not only to mild aggression but to criminal aggression and one which attempts to speak to motivational structures. By implying that a diathesis-stress (dual inputs from genetics and biology with environmental strain and stressors) approach may be more helpful for aggression than the historically predominant social cognitive model, I make no claim to original thought. Indeed, diathesis-stress models have existed for decades and that which I present here for aggression, the Catalyst Model, is but a variation on this theme. In this section, I briefly discuss evolutionary and genetic inputs for aggression and how these fit into a motivational structure that can help us to better understand and predict future aggression.

### 2.1. The evolution of aggression

The world of aggression research is littered with declarative moralistic statements that have failed withstand the test of time and perhaps few are as infamous as the Seville Statement on Violence (Adams et al., 1990). This statement, produced in the 1980s and published, as such things inevitably are, in the flagship journal of the American Psychological Association, suggested that violence had no link to human nature. Predictably, in the nearly 30+ years since this statement was constructed, a wealth of evidence emerged to link biology and genetics to aggression and violent behavior (Barnes et al., 2014).

For decades, evidence from behavioral genetics studies have clarified that approximately half or slightly more of the variance in serious aggression and antisocial behaviors is due to genetics (e.g., Rhee & Waldman, 2002). Yet, it is important to note that the impact of any *single* gene polymorphism on serious aggression is quite weak (Vassos et al., 2014). Thus, the genetic contribution is due to complex interactions between hundreds or thousands of genes with the environment. There is no single gene "for" aggression. By contrast the influence of shared environmental factors such as parenting on aggression are surprisingly weak, typically less than 10%. The remaining 30–40% or so of unshared variance is difficult to attribute. Some might argue these are learning factors, but that is unknown. Instead, though learning elements may be one part of this pie piece, so too are idiosyncratic biological inputs such as infections and head injuries, or potentially even issues such as self-regulation, agency and, more metaphysically, free choice.

If such a substantial portion of aggressive behavior is genetic it must have evolved thusly which means that, to some degree at least, aggressive behavior in moderate doses must be adaptive. Thus, evolutionary theory suggests that aggressive behaviors evolved, particularly among males, as a function of status and reproductive success, linked also to risk-taking (Klasios, 2019). Such an approach describes individual differences in aggression, gender differences in aggression as well as the universality of male aggression across cultures, an observation which stretches back to archaeological data from pre-modern indigenous societies (McCall & Shields, 2008) and, for that matter, in other ape species such as chimpanzees (Goodall, 1979). This observation also fits well with the updated definition for aggression suggested earlier in the article, focused on status rather than exclusively on causing harm.

In this sense evolution can be thought of as creating a species wide predilection for aggression which is adaptive, yet individual differences in the level of that predilection will also occur as is typical under genetic variance. Thus, some individuals may be more and less prone to aggressive responses. These propensities can be exacerbated by harsh conditions during development, creating particular gene × environment interactions which can strengthen or weaken the original genetic propensities (Holz et al., 2018). Conflicts in the attachment with parental figures appears particularly salient in such interactions (Davies et al., 2019) though other factors such as general community violence exposure may also be important (Musci et al., 2019). Put generally, genetic risk combined with exposure to early harsh environment appears to put youth on a trajectory toward pathological aggression more than is the case for other youth without the genetic risk, without the environmental risk, or without either. Such aggression may actually be adaptive, serving a deterrence function, within the harsh environment of origin, but become pathological once applied outside that environment where it no longer serves a deterrence function and social costs are high.

### 2.2. Motivation

This understanding of gene × environment interactions can help us understand *who* is more prone to serious aggression yet does not inform us much as to *why* they behave aggressively when they do. To do so, we must turn to and incorporate empirically supported motivational models.

From a motivational model, we'd expect that most aggression is not random,[4] nor is it a mechanistic activation of cognitive scripts but, rather, is activated to motivational structures. One way to examine this is in terms of *Self-Determination Theory* which, briefly, states that motivations of competence, autonomy, and relatedness (social needs), tend to be primary. The disconnect between aggression research and motivational research can be witnessed in the field of video game violence where in such games were typically portrayed as mechanistic learning boxes or, more extreme, "murder simulators", with little regard for why people chose to play such games in the first place.

Basic motivations … to survive and gain reproductive status, are evolutionarily and, thus, genetically derived. Status needs inevitably involve social interactions and the desire to see ourselves and be seen as socially worthy. Self-determination theory translates this into subgroupings for social contact, competence and autonomy, the latter two arguably being indicative of higher status individuals. Genetics can determine our basic proclivities regarding how to meet these needs (using aggression versus cooperation, say), with early childhood environment further shaping our social behavior (abuse or neglect increasing the frequency of aggressive behaviors). Further, the current environment can be seen as either aiding or frustrating our motivations.

It is worth noting that the effect sizes for gene × environment interactions for individual genes are very small (e.g., Bleys et al., 2018). Thus, one must be cautious to deemphasize language regarding a "gene for" aggression and such. Though overall evidence suggests a large genetic component for aggression (Slawinski et al., 2019), this evidence suggests an additive, not genetically monocausal process.

Aggression, then, can be seen as a response to the frustration of such motivations. Once again, some research has documented this in video games, wherein frustration of motives (competence in this case), causes

[4] Though it may be so in situations of chronic mental illness or other atypical circumstances.

aggression whereas violent content does not (Przybylski et al., 2014) though other research has indicated violent games have little impact of any kind (Květon & Jelínek, 2020). However, perceptions of frustration of motivation, appears to be associated with aggressiveness in real-life contexts (e.g., Choe & Read, 2019; Neighbors et al., 2002).

This approach bears a non-trivial resemblance to the *Frustration-Aggression Hypothesis* (Dollard et al., 1939), which has existed for the better part of a century. Berkowitz (1989) later revised the model to note the importance of negative affect in the creation of aggression. Thus, we can say that much of aggression is the result of the thwarting of motivations which produce negative affect and, from that, aggression. This, itself, differs between individuals based on the combination of genetic risk and early life experiences as explained earlier.

### 2.3. Catharsis

Of course, some might note that in some cases, aggression may occur under situations of positive affect. I include here situations in which two or more individuals may have consented to an aggressive-appearing activity such as in sports. The difference I highlight can be illustrated by hockey. Much of ice hockey is physical, though most of that is in-bounds and part of the game. All players effectively consent to this activity and, as such, this differs from the famous fistfights which are not part of the game and tend to arise out of frustration and negative emotionality. By contrast the *in bounds* and socially approved physical aspects of the game may often be felt as exhilarating and "fun" for the participants, even if, given it elevates one's status (i.e., scoring) at the expense of others, and may still be considered aggressive, if mildly so.

Thus, though it is certainly the case that much of aggression and particularly the most serious and pathological kind, may occur in the context of negative emotionality, some aggression must be understood as adaptive and even pleasant, at least for the individual engaged in it. One way to think of this is as aggression as a more general vent for stress.

The main idea of this *catharsis hypothesis* was that engaging in aggression in safe ways (such as through sports or fantasy play), would reduce engagement in inappropriate aggression (bullying, criminal violence, etc.) This theory had roots in the frustration-aggression hypothesis and attained some early support (e.g., Feshbach, 1955). However, the catharsis hypothesis fell out of favor with advocates of social cognitive models who argued, perhaps prematurely, that it amounted to a kind of myth (Bushman et al., 2001). Yet this dismissal of catharsis may have been premature and self-serving given its conflicting predictions vis a vis social cognitive models, which have proven deficient in turn.

More recent research has suggested that the implications of catharsis are complex and nuanced. Part of the problem in some studies appears to have been in forcing people to engage in behaviors the *experimenters* thought would have been cathartic but were useless if participants didn't share the same belief. Some evidence has suggested that cathartic release of aggression may improve mood, although this pattern may then be repeated (Bresin & Gordon, 2013). Other evidence has suggested that activities such as playing violent video games may reduce negative emotions, although results can vary depending upon the statistical model used (Lee et al., 2020).

One recent study (Ferguson et al., 2018) sought to examine this in a sample of individuals primed to feel frustrated. Compared to individuals given no opportunity to reduce their frustration, those allowed to engage in a cathartic activity (i.e., hit a bobo doll) were less aggressive. However, least aggressive were those who were allowed to choose between several calming activities (including playing video games, violent or non-violent.) These results suggest that, contrary to the claims of social cognitive theorists, catharsis can indeed work, although best outcomes are seen for those who engage in *mood management.* That is to say, giving vent to anger in a safe way can reduce anger and prevent aggression, but distracting oneself with pleasant experiences is more efficient still.

It is recognized that it is helpful to consider aggression from a multidimensional perspective. The essence of the Catharsis Approach is that individuals may be motivated toward aggression to reduce negative emotional statements such as anger. Some evidence suggests that, unlike fear, anger is associated with approach behaviors (Carver & Harmon-Jones, 2009). This would appear to fit with Catharsis insofar as a need to reduce frustration and anger could draw people toward a stimulus to release their emotional state rather than to avoid it.

It's also arguable that some cathartic-appearing activities may simply be adaptive. For instance, watching aggressive sports or war movies may be vicariously reinforcing of dominance motivations, rather than specifically cathartic. In this sense, many aggressive displays may relate to audience satisfaction rather than expressions of frustration or anger.

### 2.4. The Catalyst Model

In 2009 Ferguson and Beaver presented the Catalyst Model, which is a diathesis-stress evolutionary model of serious aggression. This model is built upon considerable evidence suggesting evolutionary and genetic roots of serious aggression, but also acknowledging that harsh early environment can play a substantial role. This model is presented in Fig. 1.

According to this model, genetic risk combines with a harsh early environment (family violence, neglect, etc.) to produce personalities which differ in their proclivity toward serious aggression. This helps us to understand the range of individual differences in aggressiveness, even for individuals presented with similar environmental events which may trigger aggressiveness. Each individual has a behavioral response

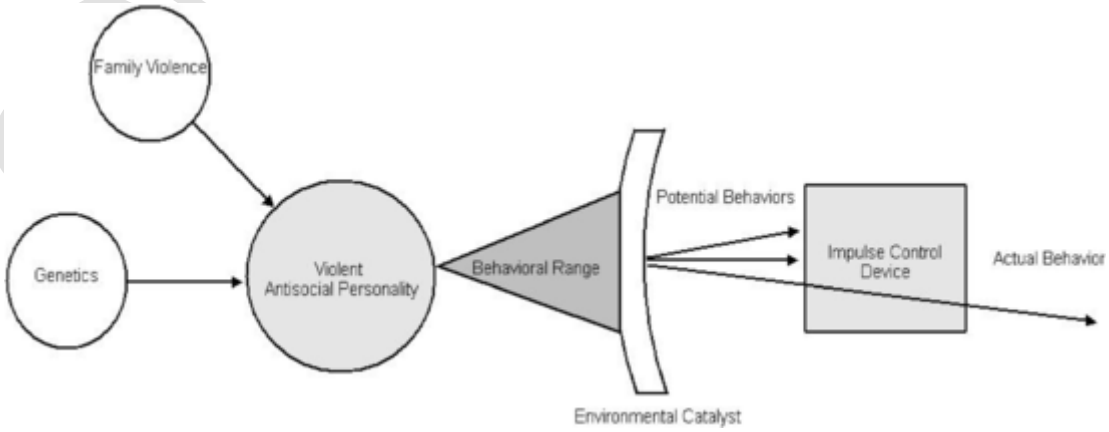


**Fig. 1.** A catalyst model for serious aggressive behavior.

range or a natural proclivity to engage or not engage in serious aggression.

Aggression, however, is triggered by environmental stimuli in ways which are consistent with Self-Determination Theory or the Frustration-Aggression Hypothesis. Motivational needs are thwarted and negative affect rises. This increases the probability of aggressive behavior. As such, the overall probability of aggressive behavior can be considered as a function of an individual's preexisting propensity or trait aggression as indicated by their personality, with the specific motivational qualities of a specific event occurring in real time.

This creates the motivation to engage in aggressive behavior. The individual still may or may not respond aggressively. Serious aggressive behavior is typically not socially sanctioned and may come with non-trivial long-term costs. As such, an individual's impulse control may still lead them to weigh between behavioral options and choose a less aggressive response. Indeed, considerable evidence notes that damage to impulse-control areas of the brain, particularly in the frontal lobes, is associated with increased aggression (Bannon et al., 2015; Cristofori et al., 2016). Likewise, evidence suggests that reduced impulse control related to alcohol consumption is related to aggressive behavior (e.g., Cunradi et al., 2011; Giancola, 2013).

The Catalyst Model specifically does not involve other elements once thought to increase aggression. Specifically, media violence is not considered a cause of aggression, given the overall underwhelming evidence for this belief (Savage & Yancey, 2008) particularly in preregistered studies (Drummond et al., 2020). However, it is recognized that perpetrators of aggression may sometimes model *stylistic* (e.g., stylistic catalysts) elements of a fictional event, but not the motivation to commit aggression itself. So, for instance, a criminal may decide to commit a homicide. They may have learned from a crime drama to use bleach to try to remove evidence from a scene. That is a stylistic catalyst. The homicide would have occurred with or without the crime drama, but perhaps the perpetrator wouldn't have used bleach if not for the show.

Under this model, aggression need not necessarily be reactive to stress. Development of more aggressive personalities through gene × environment interactions can also drive proactive aggression, in which aggression is used to achieve resources or status in a calculated way. Reactive aggression may accelerate during times of stress, though proactive aggression may occur at any time.

The Catalyst Model has proven an effective modeling approach for understanding serious aggression and violent crime (e.g., Miles et al., 2017; Schwab-Reese et al., 2020; Surette, 2013; Surette & Maze, 2015). It is important to note that it is very much a *clinical* model geared toward serious aggression and as such may not be as effective in predicting prank-level aggressive behaviors or milder aggressive behaviors which are socially sanctioned (debating, sports aggression, defending oneself, etc.) Nonetheless, given the particular attention focused on serious aggression as a matter of social concern, particular as related to activities ranging from bullying to violent crime, the Catalyst Model appears to be an empirically validated approach to understanding these behaviors.

At its core, the model helps us to understand not just who may be at higher risk for aggression but also motivationally, why some individuals may feel the need to behave aggressively. By understanding these motivational issues, it may be more possible to target clinical interventions and prevention efforts at serious aggressive behavior. If individuals have needs that are thwarted, how might we assist them in addressing those needs without feeling motivated to engage in serious aggression. Such efforts appear to be more likely to bear fruit than typical moral advocacy agendas designed around regulating media content which, aside from being unconstitutional in the United States, show little empirical promise as an effective prevention strategy.

The difficulty for clinicians may come for individuals who have developed aggressive strategies under some circumstances which prove to be adaptive in those circumstances (e.g., abusive or neglectful homes), but are finding those strategies riskier outside those contexts. Helping patients to understand how more prosocial strategies may get their needs met with fewer risks may bear fruit, but may also come up against ingrained patterns developed during earlier years when heightened aggression was a viable strategy and prosocial strategies were less effective. Having empathy for those developmental circumstances may aid the clinician with their own feelings of frustration when patients don't immediately and intuitively grasp prosocial strategies.

It is worth noting too that some forms of serious aggression may be socially sanctioned. For instance, social majorities may sanction aggressive wars if these increase resources, territorial gains, or national pride. Although the Catalyst Model was designed to examine for variance between individuals within societies, future research could examine how it may apply to how individuals rally larger societies towards more aggressive versus more pacific causes. This could help us to understand who is most likely to become a demagogue and under what circumstances are they most likely to achieve social status and influence.

The Catalyst Model certainly could benefit from more research, particularly in longitudinal format (Miles et al., 2017). It is also worth highlighting that, frankly, the Catalyst Model is hardly a creation of brilliant innovation, largely structured on pre-existing diathesis-stress models of psychopathology. Yet it does appear efficient in both understanding the complex etiology of serious aggression, as well as highlighting some factors which appear to be less relevant such as violence in media. It also returns motivation as a key element to our understanding of aggression.

### 2.5. Concluding thoughts

Scholars have been interested in the study of aggression for nearly a century dating back to the Frustration-Aggression Hypothesis. Some important, early work was done on this topic, though it is concluded that the Social Cognitive era largely represented a period of stagnation in our understanding of aggression. This was due, in part, to widespread methodological problems and methodological flexibility (and potential p-hacking) in much of the research, as well as a theoretical model which placed too much emphasis on mechanistic learning and not enough on biology, stress, affect, and motivation. It is hoped that the Catalyst Model will provide a road forward to greater research productivity in aggression, particularly that coming from preregistered research.

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## Uncited reference

Przybylski et al., 2010.

## Declaration of competing interest

The author has no conflicts of interest to declare.

## Data availability

No data was used for the research described in the article.

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