

NIH Public Access

Author Manuscript

Published in final edited form as:

Curr Dir Psychol Sci. 2008 April; 17(2): 153-158. doi:10.1111/j.1467-8721.2008.00566.x.

Cognitive Emotion Regulation: Insights from Social Cognitive and Affective Neuroscience

Kevin N. Ochsner^{*} and Columbia University

James J. Gross Stanford University

Abstract

Recent developments in the study of cognitive emotion regulation illustrate how functional imaging is extending behavioral analyses. Imaging studies have contributed to the development of a multi-level model of emotion regulation that describes the interactions between neural systems implicated in emotion generation and those implicated in emotional control. In this article, we review imaging studies of one type of cognitive emotion regulation, namely reappraisal. We show how imaging studies have contributed to the construction of this model, illustrate the interplay of psychological theory and neuroscience data in its development, and describe how this model can be used as the basis for future basic and translational research.

Keywords

emotion; emotion regulation; cognitive control; amygdala; prefrontal cortex

Homer's Illiad – like many of our greatest literary works – is the story of failed emotion regulation. The age and ubiquity of such stories highlights the importance of effective emotion regulation. Only recently, however, have significant strides been made in the development of brain-based models of this ability. This progress has been spurred by the emergence of social cognitive and affective neuroscience (SCAN), which use neuroscience techniques to address questions about the mechanisms underlying emotion-cognition interactions. In this article, we demonstrate how such research has advanced our understanding of cognitive emotion regulation.

Multi-level Models

One tenet of SCAN research is that behavior and mental processes should be explained using multi-level models that link (a) measures of behavioral, experiential, and physiological responses to (b) descriptions of information processing mechanisms and (c) their neural substrates. The goal is to provide a richer and deeper account of a phenomenon of interest by drawing upon all three levels levels of analysis at once, rather than relying on a single level.

corresponding author: Kevin Ochsner, Department of Psychology, Columbia University, 369 Schermerhorn hall, 1190 Amsterdam Ave., New York, NY 10027, ochsner@psych.columbia.edu.

Developing such multi-level models requires an interplay among data across levels. For example, behavioral data constrain the inferences we can draw about brain function. Indeed, we can only draw inferences about the neural bases of psychological processes our behavioral manipulations and measures are designed to address. At the same time, neuroscience data provide insights into underlying information processing mechanisms not possible using behavioral methods alone. For example, imaging data may provide information about when and to what extent neural systems are engaged during a task. Although both sides of this two-way street deserve attention, due to space limitations, we focus here on how neuroscience data powerfully supplement behavioral data in the context of cognitive emotion regulation.

Behavioral Studies of Cognitive Emotion Regulation

Empirical work on emotion regulation began with descriptive psychodynamic studies of defense mechanisms, which in the 1960's spawned empirical work on the factors influencing an individual's ability to cope with stressful situations, and today continue to inspire developmental studies of a child's ability to self-regulate. Building upon these studies, contemporary models conceive of emotions as arising from brain systems that appraise the significance of stimuli with respect to our goals and needs. Appraisals may involve multiple stages and kinds of processing that govern attention to, evaluation of, and response to a stimulus, and emotion regulatory strategies are thought to work by impacting them in different ways (Gross, 1998).

Behavioral studies have tested one prediction of these models, namely that different behavioral consequences should be observed depending upon what stage or kind of emotion generative process a strategy influences. For example, asking participants to cognitively reappraise upsetting images in neutral terms can lessen negative emotion, as indexed by startle responses (Jackson et al, 2000). By contrast, asking participants to suppress only the behavioral expression of disgust elicited by a video may limit behavior while boosting autonomic responding and leaving experience unchanged (Gross, 1998).

Findings such as these have important implications for understanding the costs and benefits of regulating emotion in different ways. Importantly, however, they only indirectly inform models of the underlying information processing mechanisms. As described below, neuroimaging studies are beginning to provide new insights into underlying mechanisms.

Neuroimaging Studies of Cognitive Emotion Regulation

Neuroimaging studies of emotion regulation build on a foundation of prior animal and human neuroscience findings that have identified structures critical for triggering affective responses or effectively controlling "cold" cognitive abilities such as attention and memory. Although various aspects of emotion regulation have been examined, some of the most theoretically informative work has been done on cognitive reappraisal, which involves rethinking the meaning of affectively charged stimuli or events in terms that alter their emotional impact. In the context of the psychological approach to emotion regulation outlined above, imaging studies of reappraisal can be seen as addressing four questions about underlying mechanisms.

What is the Nature of Cognition-Emotion Dynamics?

The first, and perhaps most fundamental, question is what kind of cognition-emotion dynamics underlie effective attempts to reappraise. As shown in Table 1, studies published to date indicate that reappraisal depends upon interactions between prefrontal and cingulate regions implicated in cognitive control and systems like the amygdala and insula that have been implicated in emotional responding. These findings dovetail with behavioral work by demonstrating different modulatory effects depending upon the intended effect of reappraisal: having the goal to think about stimuli in ways that maintain or increase emotion may boost amygdala activity whereas having the opposite goal may diminish it. Furthermore, changes in emotional experience and autonomic responding may correlate with the concomitant rise or fall of prefrontal and/or amygdala activity (see 2,5,8,11,13 in Table 1).

What are the Subcomponents of Reappraisal?

A second question is whether reappraisal is a unitary ability or fractionates into subcomponents. Psychological theory would suggest fractionation, given that reappraisal is cognitively complex and should require processes necessary for generating, maintaining and implementing a cognitive reframe as well as processes that track changes in one's emotional states. As Table 1 indicates, imaging findings bear out this view. During reappraisal, activated regions include dorsal portions of PFC implicated in working memory and selective attention, ventral portions of PFC that have been implicated in language or response inhibition, dorsal portions of the anterior cingulate cortex (dACC) implicated in monitoring control processes, and dorsal portions of medial PFC implicated in reflecting upon one's affective states. In addition, it appears that reappraisal may modulate systems involved in different aspects of emotional appraisal, including the amygdala, which has been implicated in the detection and encoding of affectively arousing stimuli, and the insula, which receives viscerosensory inputs and may play a general role in affective experience.

Although Table 1 highlights the finding that PFC/ACC are consistently activated by reappraisal, the specific regions activated varies across studies. Differences in how reappraisal is operationalized may be important here. Consider, for example, that studies have asked participants to reappraise by either a) reinterpreting situational or contextual aspects of stimuli (e.g. imagining an image is faked, or that an apparently sick person in the hospital will get well soon), or b) *distancing* oneself from stimuli by adopting a detached 3rd person perspective. This is interesting, because behavioral work indicates that both can be effective for regulating emotion, but doesn't tell us whether they depend upon similar or different mechanisms – a question imaging data is well suited to address. Although only a single study has directly compared these strategies within subjects (4 in Table), comparing across studies in Table 1 suggests one hypothesis that could be tested in future work. Whereas reinterpretation may differentially depend upon dorsal PFC systems for selective attention (as one encodes contextual as compared to central aspects of stimuli) as well as left lateralized systems for language and verbal working memory (as one constructs a 'new story' about the meaning of a stimulus), *distancing* may depend more upon medial systems for evaluating the self-relevance of images and right PFC systems generally involved in attentional control.

What is the Relation Between Reappraisal and Other Forms of Emotion Regulation?

The third question is how reappraisal relates to other forms of emotion regulation. We have theorized that reappraisal (which has its primary impact relatively early in the emotion-generative process) should differ importantly from other forms of emotion regulation such as expressive suppression (which has its primary impact relatively late in the emotion generative process). Imaging data now support this prediction by showing that the two strategies engaged different kinds of cognition-emotion interactions over the course of viewing emotionally evocative film clips (11 in Table): for reappraisal, early frontal engagement produced decreased amygdala/insula activity over time, whereas for suppression, late frontal engagement produced increasing amygdala/insula activity over time. These data are intriguing because they suggest why reappraisal and suppression have divergent effects on behavior and experience, and also show that they may depend upon similar control systems, albeit at different times.

More generally, imaging data may be used to make comparisons between the mechanisms supporting reappraisal and more distant forms of regulation, including those that involve learning to update affective associations as they change over time during extinction of a conditioned affective response or reversals of stimulus-reinforcer associations. Such comparisons can reveal that high-level cognitive forms of regulation like reappraisal may depend more upon dorsal frontal systems involved in working memory, language and goal representation. By contrast, forms of regulation that depend upon learning that the affective outcomes associated with stimuli or responses are changing over time may differentially depend upon ventral frontal systems directly connected with the subcortical systems essential for learning these associations in the first place.

How Does Reappraisal Relate to Non-Affective Forms of Control?

Finally, imaging data can inform our understanding of the relationship between reappraisal and other non-affective forms of cognitive control. Indeed, one of the most striking aspects of recent work on reappraisal is its demonstration that some forms of emotion regulation can depend upon lunguistic and cognitive processes not typically thought of as having emotion-related functions. Whether the specific systems recruited are merely similar or are truly the same can not yet be discerned, however, because comparisons of reappraisal, or other forms of emotion regulation, to non-affective forms of control have not yet been made in a single study.

Conclusions and Future Directions

Our review of behavioral and neuroimaging findings regarding cognitive emotion regulation illustrates how a SCAN approach can extend behavioral research by (a) clarifying the temporal dynamics of relevant processes, (b) helping to decompose complex processes into simpler ones, (c) relating processes in a given family of strategies to one another, and (d) distinguishing one group of processes from others not in that group.

The data and theory reviewed above support an emerging multi-level model of a functional architecture supporting cognitive emotion regulation. On this model, cognitive strategies vary in their reliance on prefrontal and cingulate systems for attention, response selection,

Ochsner and Gross

working memory, language, mental state attribution, and autonomic control. The regulatory effects of any given strategy – such as reappraisal – can be understood in terms of its reliance upon specific component control processes and the regulatory effects they exert on systems involved in various aspects of emotional responding, such as the amygdala and insula.

This way of modeling emotion regulation provides a framework for guiding basic and translational research. For basic research, the model provides a means of understanding how a given strategy, such as reappraisal, is not a singular function but rather is comprised of a family of related ways of reinterpreting the meaning of stimuli, which in turn depend upon related but distinct constellations of brain regions. Research has only just begun to examine these issues, however, and future work is needed to determine how different elements of these control networks are recruited and functionally connected with one another during different forms of reappraisal and related forms of regulation. Indeed, future work could use imaging to distinguish the mechanisms underlying the many ways that one can use controlled cognition to regulate via distraction or the suppression not of expressive behavior but of unwanted thoughts or feelings (cf. Ohira et al, 2006). Given that the majority of work to date has examined only these deliberate forms of regulation, their relationship to automatic forms of regulation will be important to address (e.g. Jackson et al, 2003). It also will be important to clarify how the neural dynamics of regulation vary with the valence, duration, discreteness, and interpersonal nature of the emotions to be regulated, all of which could influence the emotion and control systems. As Table 1 indicates, some variability in results already may be attributable to differences in stimuli and the emotions they elicit.

Another important direction for basic research is suggested by the observation that much of the work to date has been motivated by the logic of 'reverse' inference, in that the meaning of reappraisal-related activity is interpreted based on other work that suggests functions for the activated regions. This is a very sensible approach when tackling a new topic of study about which little is initially known about neural mechanisms. As the field matures, however, and theories of the functional architecture of reappraisal become more refined, studies increasingly will be able to test specific hypotheses about the functional roles played by discrete brain systems. In fact, this already has begun to happen. In our first reappraisal study (Ochsner et al, 2002), for example, we expected and interpreted the meaning of lateral and medial PFC activity during reappraisal in light of prior work on cognitive control. For our second study (Ochsner et al, 2004), we formulated and tested hypotheses about the expected dependence of two different types of reappraisal (reinterpretation vs. distancing, noted above) on lateral as opposed to medial PFC. These hypotheses were based on a both a psychological theory of the processes involved and a neurobiological theory of the brain regions upon which they depend. When studies are designed in this way, their results can inform both theories of the psychological and neural bases of emotion regulatory mechanisms. In so doing, research will help clarify the functional roles played by the brain systems involved in emotion regulation. This is already happening as well. As noted above, reappraisal has been shown to recruit prefrontal and cingulate regions similar to those involved in 'cold' forms of cognitive control. Findings like these expand our knowledge of what specific brain regions do, and may alter our sense of what domain-general computations they perform. Neuroscience theories of prefrontal function will be informed

by future work clarifying the computations carried out by regions that are uniquely or commonly involved in emotional and non-emotional control.

As basic science studies address these and related issues, an increasingly stable foundation will be available for translational work seeking to understand how normal and abnormal differences in emotional responding and regulation may be expressed in terms of the development, tuning, integrity and recruitment of component emotion and control processes. It already has been shown that ruminators show greater amygdala modulation during reappraisal (Ray et al, 2006). Future work could examine, for example, how disorders such as depression and anxiety can be explained in terms of abnormal responsivity in systems that trigger emotion responses, failures to recruit systems used to down or up-regulate them effectively, or both.

As we look to the future, it is useful to consider how the SCAN approach to emotion regulation might transform our theoretical and empirical agenda. Much work in this area is motivated by simple two factor models in which cognitive and affective processes engage in a tug-of-war for control of behavior. The SCAN approach suggests that ultimately these models will prove overly simplistic, and that a more fruitful tack will entail developing an integrated framework for specifying what combinations of interacting subsystems are involved in emotional responding as individuals exert varying degrees and kinds of regulatory control over them. With any luck, this work may offer a rejoinder to ancient cautionary tales of regulatory failures by informing modern scientific knowledge about when and how emotion regulation is effective.

Acknowledgments

This work was supported by NIH Grants MH58147 and MH076137 and NIDA grant DA022541.

References

- Beauregard M, Levesque J, Bourgouin P. Neural correlates of conscious self-regulation of emotion. Journal of Neuroscience. 2001; 21(18):RC165. [PubMed: 11549754]
- Eippert F, Veit R, Weiskopf N, Erb M, Birbaumer N, Anders S. Regulation of emotional responses elicited by threat-related stimuli. Hum Brain Mapp. 2006; 28(5):409–423. [PubMed: 17133391]
- Goldin PR, McRae K, Ramel W, Gross JJ. The neural bases of emotion regulation: Reappraisal and Supression of Negative Emotion. Biological Psychiatry. (in press).
- Gross JJ. The emerging field of emotion regulation: An integrative review. Review of General Psychological. 1998; 2:271–299.
- Harenski CL, Hamann S. Neural correlates of regulating negative emotions related to moral violations. Neuroimage. 2006; 30(1):313–324. [PubMed: 16249098]
- Jackson DC, Malmstadt JR, Larson CL, Davidson RJ. Suppression and enhancement of emotional responses to unpleasant pictures. Psychophysiology. 2000; 37(4):515–522. [PubMed: 10934910]
- Jackson DC, Mueller CJ, Dolski I, Dalton KM, Nitschke JB, Urry HL, et al. Now you feel it, now you don't: frontal brain electrical asymmetry and individual differences in emotion regulation. Psychol Sci. 2003; 14(6):612–617. [PubMed: 14629694]
- Kalisch R, Wiech K, Critchley HD, Seymour B, O'Doherty JP, Oakley DA, et al. Anxiety reduction through detachment: Subjective, physiological, and neural effects. Journal of Cognitive Neuroscience. 2005; 17(6):874–883. [PubMed: 15969906]

- Kalisch R, Wiech K, Herrmann K, Dolan RJ. Neural correlates of self-distraction from anxiety and a process model of cognitive emotion regulation. J Cogn Neurosci. 2006; 18(8):1266–1276. [PubMed: 16859413]
- Kim SH, Hamann S. Neural correlates of positive and negative emotion regulation. Journal of Cognitive Neuroscience. 2007; 19(5):1–23. [PubMed: 17214558]
- Levesque J, Eugene F, Joanette Y, Paquette V, Mensour B, Beaudoin G, et al. Neural circuitry underlying voluntary suppression of sadness. Biol Psychiatry. 2003; 53(6):502–510. [PubMed: 12644355]
- Levesque J, Joanette Y, Mensour B, Beaudoin G, Leroux JM, Bourgouin P, et al. Neural basis of emotional self-regulation in childhood. Neuroscience. 2004; 129(2):361–369. [PubMed: 15501593]
- Ochsner KN, Bunge SA, Gross JJ, Gabrieli JD. Rethinking feelings: an FMRI study of the cognitive regulation of emotion. Journal of Cognitive Neuroscience. 2002; 14(8):1215–1229. [PubMed: 12495527]
- Ochsner KN, Ray RD, Cooper JC, Robertson ER, Chopra S, Gabrieli JDE, et al. For Better or for Worse: Neural Systems Supporting the Cognitive Down- and Up-regulation of Negative Emotion. Neuroimage. 2004; 23(2):483–499. [PubMed: 15488398]
- Ohira H, Nomura M, Ichikawa N, Isowa T, Iidaka T, Sato A, et al. Association of neural and physiological responses during voluntary emotion suppression. Neuroimage. 2006; 29(3):721–733. [PubMed: 16249100]
- Phan KL, Fitzgerald DA, Nathan PJ, Moore GJ, Uhde TW, Tancer ME. Neural substrates for voluntary suppression of negative affect: A functional magnetic resonance imaging study. Biol Psychiatry. 2005; 57(3):210–219. [PubMed: 15691521]
- Ray RD, Ochsner KN, Cooper JC, Robertson ER, Gabrieli JDE, Gross JJ. Individual differences in trait rumination modulate neural systems supporting the cognitive regulation of emotion. Cognitive, Affective & Behavioral Neuroscience. 2005; 5:156–168.
- Schaefer SM, Jackson DC, Davidson RJ, Aguirre GK, Kimberg DY, Thompson-Schill SL. Modulation of amygdalar activity by the conscious regulation of negative emotion. J Cogn Neurosci. 2002; 14(6):913–921. [PubMed: 12191458]
- Urry HL, van Reekum CM, Johnstone T, Kalin NH, Thurow ME, Schaefer HS, et al. Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. J Neurosci. 2006; 26(16): 4415–4425. [PubMed: 16624961]
- van Reekum CM, Johnstone T, Urry HL, Thurow ME, Schaefer HS, Alexander AL, Davidson RJ. Gaze fixation repdicts brain activation during the voluntary regulation of picture-induced negative affect. Neuroimage. 2007; 36(3):1041–55. [PubMed: 17493834]

Recommended Reading

- Kosslyn SM. If neuroimaging is the answer, what is the question? Philosophical Transactions of the Royal Society of London B: Biological Sciences. 1999; 354(1387):1283–1294.
- Gross JJ. The emerging field of emotion regulation: An integrative review. Review of General Psychological. 1998; 2:271–299.
- Gross, JJ., editor. The handbook of emotion regulation. New York: Guilford Press; 2007.
- Ochsner, K. Social cognitive neuroscience: Historical development, core principles, and future promise. In: Kruglanksi, A.; Higgins, ET., editors. Social Psychology: A Handbook of Basic Principles. 2007.
- Ochsner KN, Gross JJ. The cognitive control of emotion. Trends in Cognitive Sciences. 2005; 9(5): 242–249. [PubMed: 15866151]

_
_
<u> </u>
_
0
-
-
D
-
_
_
_
_
\mathbf{n}
_
_
- C
5
\geq
a
Aar
/an
Jani
/lanu
/lanu:
Janus
Janus
Aanus
/ anusc
Janusci
/anuscri
/anuscri
/anuscrip
/anuscrip
/anuscript

Ochsner and Gross

Table 1

Design and Results for Functional Imaging Studies of Cognitive Reappraisal

									Re	sults		
			Ď	sign		Con	trol Systen	IS			Emotion systemetry	ems
#	Study	Stimulus	Emotion	Strategy	Goal	lat PFC	ACC	med PFC	Amyg	Insula	Other	Behavior
-	Beauregard et al, 2001	Films	Sexual Arousal	More Dist	Dec	Ld/Rd	Rd	Rd	ы	ı	Hyp	Less arousal
7	Ochsner et al, 2002	Images	Neg	Reint	Dec	Ldv	Ld		Г	Г		Less affect
ю	Schaeffer et al, 2002	Images	Neg	Reint	Maintain	'n	nr	nr	$\Gamma/R^{\Omega\Omega}$	nr	nr	Sustained affect
4	Levesque et al, 2003	Films	Sadness	More Dist	Dec	Rdv	ı	ī	L	Г	ı	Less sad
2	Ochsner et al, 2004	Images	Neg	Reint+More Dist	Dec	Ld/Rd	Lv/Rv	ï	L/R	L/R	ı	Less affect
		Images	Neg	Reint+Less Dist	Inc	Ld	Lv	Ld	Г	ı	ı	More affect
		Images	Neg	Reint > More Dist	Dec	Ld	ı	,	ı	ı	ı	No difference
		Images	Neg	More Dist > Reint	Dec		Ls		ı	ı	ı	No difference
9	Levesque et al, $2004^{arOmega}$	Films	Sadness	Reint	Dec	Ldv/Rdv	Rd	Ld/Rd	ı	·	ı	Less sadness
٢	Kalisch et al, 2005	Anticip Shock	Anxiety	Dist	Dec	Rd	ı	Rd	ı	ı	R dmPFC	Less Anxiety, HR
×	Phan et al, 2005	Images	Neg	Reint	Dec	Ld/Rdv	Ld/Rd	Rd	L	Ra	ı	Less affect
6	Harenski et al, 2006	Images	Neg Nonmoral	Reint	Dec		,		ı			Less affect
		Images	Moral Violation	Reint	Dec	Ldv/Rv	ı	·	L/R	ı	ı	Less affect
		Images	Moral > Neg	Reint	Dec	Ldv/Rd	Rd	Ld	ı	ı	ı	Less affect
10	Kalisch et al, 2006	Anticip Shock	Anxiety	More Dist	Dec	Ld	Ls	ī	ı	ı	ı	NC anxiety
11	Ohira et al, 2006	Images	Neg + Pos	Supp**	Dec	Lv		Lv/Rv	Г	- R Tr	np pole	NC affect; SCL up
12	Urry et al, 2006	Images	Neg	Reint	Dec	ı	ı	Ld/Rv*	$L^{*}R^{*}$	ı	ı	Less affect, pupils dilate
		Images	Neg	Reint	Inc	Ldv	Ld	Ld	L/R	I	ı	More affect, pupils dilate
13	Eippert et al, 2006	Images	Neg	More Dist	Dec	Ldv	Rd/Ld		L	ı	I	Less SCL

		Jesign		C011	ITOI SYSTEE	SI			Emouon sysu	ems
timulu	Emotion	Strategy	Goal	lat PFC	ACC	med PFC	Amyg	Insula	Other	Behavior
Images	Neg	Less Dist	Inc	Ld/Rdv	Ld/Rd	Rd	L/R	·	·	More SCL/Startle
Images	Neg	Reint	Dec	Ldv/Rdv	Ld/Rd	ı	,		,	Less arousal
	Pos	Reint	Dec	Ldv/Rdv	Ld/Rd		Я	,	ı	More arousal
	$\operatorname{Pos} > \operatorname{Neg}$	Reint	Dec			ı	ı	ı	ı	Less arousal
	Neg > Pos	Reint	Dec	Ldv/Rdv	Ld/Rd	ı	ı	ı	ı	Less arousal
	Neg	Reint	Inc	Ldv	Rdv	Ld/Rd	Г	,		More arousal
	Pos	Reint	Inc	Ldv/Rv		Rd	L/R	ı	ı	More arousal
	$\operatorname{Pos} > \operatorname{Neg}$	Reint	Inc	Ldv/Rd		Ld	Г	ı	ı	More arousal
	Neg > Pos	Reint	Inc		ı		Rd	·	·	More arousal
Films	Disgust	Reint	Dec	Ldv/Rd (ely)	ı	Ld (ely)	R (lt)	L (lt)	,	Less affect
Films	Disgust	Suppress face	Dec	Lv/Rdv (lt)	ı	d (lt)	R(lt) up	La (lt) up	·	Less affect + face
Films	Disgust	Reint > Supp	Dec	Ldv (ely)	ı	Ld (ely)	ı	La (ely)	ı	Less affect
Films	Disgust	Supp > Reint	Dec	Lv/Rv (lt)	d (lt)	Lv (lt)	ı	ı	ı	Less face
Images	Neg	Reint	Inc>Dec>Base	Ldv/Rdv		Rd	,			Inc/Dec more/less affect
Images	Neg	Reint	Inc>Dec=Base		Lv	ı	ı	ı	ı	as above
Images	Neg	Reint	Inc=Base>Dec	,	·	ı	L/R	ı	ı	as above
Images	Neg	Reint	Inc>Base>Dec		ı	·	,		R Put	as above

Curr Dir Psychol Sci. Author manuscript; available in PMC 2014 November 23.

Amyg = amygdala, Hyp = hypothalamus, ely = activity in early phase of stimulus presentation, lt = activity in late phase of stimulus presentation; For Behavior: HR = heart rate, pupils dilate = greater pupil condition in which emotional responses are allowed to flow naturally, and a reappraisal condition in which responses are regulated cognitively. Column labels: # = identifier for referring to study in text; reinterpreting the meaning of the stimulus); Goal = increase or decrease response; Control Systems = systems associated with control processes; Emotion Systems = systems associated with emotional modulated in accord with reaptraisal goals; nr = not reported, L = left, R = right, d = dorsal, v = ventral, m = medial, l = lateral, a = anterior, PFC = prefrontal cortex, ACC = anterior cingulate cortex, Note: Studies are organized chronologically. Unless otherwise specified, activations are for contrasts in which the same stimulus type (e.g. negative image) is presented in two conditions: A baseline psychologically distant, face = facial expression, supp = suppress facial behavior; For Control and Emotion Systems: Unless otherwise noted, control systems are activated and emotion systems are Study = study listed in references; Stimulus = type of stimulus employed; Emotion = type of emotional/affective response elicited; Strategy = type of strategy (varying psychological distance or responses. Abbreviations: For Stimulus: Anticip = anticipate; For Emotion: Pos = positive affect, Neg = negative affect; For Strategy: reint = cognitively reinterpret, dist = become more or less dilation, which is an indicator of effort or arousal, face = facial expression, NC = no change.

For Study 9,

means that regions of vmPFC correlate inversely with the amygdala, but neither region showed significant change in the overall group contrast to identify regions activated or modulated by reappraisal.

Results

Ochsner and Gross

** Ohira et al instructed participants to, "suppress emotional response" but didn't make clear if that meant expressive behavior or experience. We assume the former because they observed no changes in experience and compared their paper to Gross's expressive suppression work.

 $\mathcal{N}_{\text{Used}}$ only children as participants.

 $\Omega\Omega^{}_{}$ Did ROI analyses collapsing across both amygdalae.