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A systematic review and meta-analysis of the effect of emotion regulation on cortisol☆

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ABSTRACT

It is generally acknowledged that hormones are implicated in socioemotional behavior, yet little is known about the role of hormones in the context of emotion regulation. The aims of the present review and meta-analysis were to review and synthesize the available evidence pertaining to the effect of emotion regulation instructions on hormones, and to investigate whether this effect varies according to: type of hormone, context (e.g., emotion-induction procedure), emotion regulation characteristics (e.g., emotion regulation strategy), and presence and type of psychiatric disorder. PubMed, PsycINFO, and CINAHL were searched for experimental studies assessing the effect of instructed emotion regulation on levels of hormones (i.e., testosterone, cortisol, oxytocin, estradiol, and vasopressin) in physically healthy adults. The literature search yielded 17 relevant studies, 16 investigating cortisol and one investigating testosterone. Of these, 12 cortisol studies had eligible data for the meta-analysis. The results of the meta-analysis indicated no statistically significant effect of receiving an emotion regulation instruction compared with receiving no instruction on the cortisol response to subsequent emotion induction ($g = -0.05$, $p = .48$). However, within-person comparisons of change from an unregulated response to a regulated response indicated a significant change in cortisol levels ($g = 0.18$, $p = .03$) consistent with the specified regulation goal (i.e., either up- or downregulation). No statistically significant effects were found in subgroup meta-analyses conducted according to context, emotion regulation characteristics or psychiatric disorders. Taken together, the findings indicate that emotion-induction procedures are associated with increases in cortisol that may subsequently return to equilibrium regardless of emotion-regulation instructions. Based on the large gaps in research (e.g., few studies investigated other hormones than cortisol, few studies included self-report measures of emotions) identified in the present review, we conclude that the effect of emotion regulation on hormones remains poorly understood.

Prospero registration: CRD42020157336.

1. Introduction

Recent decades have seen a surge of interest in the link between the body and the mind, originating from the assumption that to understand the one, you need to understand the other. In 2009, the National Institute of Mental Health launched the Research Domain Criteria (RDoC) initiative, emphasizing multimodal approaches to studying psychological phenomena, with special attention to biological and physical assessments [62,86]. In the years following this initiative, a growing body of empirical literature has investigated biological and physiological components of psychological and behavioral phenomena such as emotions and mental health disorders. This focus has also extended to endocrinology [1,47], where hormones have been found to play a central role in regulating psychological processes and human behavior [6]. In the present article,

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we focus on hormones in the context of emotion regulation.

It is generally acknowledged that human socioemotional behaviors (e.g., attention, motivation, and trust) are influenced by a number of hormones (i.e., chemical messengers originating from the endocrine glands; [6,106]. Hormones function to coordinate and regulate activities of cells in the body, ultimately leading to changes in the probability that overt behaviors or physiological reactions occur [83,97,106]. There is considerable evidence from both non-human and human studies that endogenous hormone levels are associated with socioemotional behaviors [7,19,20], and that exogenous manipulation of hormone levels (i.e., drug administration to increase hormone levels or to block the effect of hormones) may lead to behavioral changes in humans such as changes in stress responses, aggressive behavior, and decision making [6,20,82,83,93]. For example, research indicates that administration of testosterone leads to increased vigilance and motivation to act, while administration of oxytocin promotes search for proximity to others [6,20]. As such, the existing evidence provides support for the notion that acute changes in hormone levels affect subsequent behavior. Furthermore, the association between behavior and hormones appears to be bidirectional as behavioral changes may also affect subsequent hormone levels, with several studies demonstrating that psychological states (e.g., feeling powerful, feeling stressed) and physical behaviors (e.g., aggressive behavior) alter hormone levels in humans [12,22,46]. For example, studies have shown cortisol increases in response to psychosocial stress [34] and testosterone increases in response to success experiences [78,91]. These findings suggest the possibility that hormone levels and the associated behavioral inclinations may be influenced through manipulations of both physical behaviors and psychological states.

Emotion regulation (i.e., the processes by which individuals influence which emotions they have, when they have them, and how they experience and express them [38]; p. 275) represents one way of altering one's psychological state [53,73]. Over the last three decades, a considerable amount of research has been devoted to evaluating the effect of emotion regulation, linking emotion regulation abilities and habitual use of specific emotion regulation strategies to mental and physical health outcomes [41,79]. However, only little is known about how hormones are implicated in emotion regulation. It has been suggested that hormones may mediate the effect of emotion regulation on mental and physical outcomes [72]. Consistent with the idea that there may be a link between hormones and emotion regulation, correlational research indicates an association between self-reported habitual use of specific emotion regulation strategies and daily hormone levels [84], and between self-reported habitual use of specific emotion regulation strategies and hormonal changes in response to stressors [63,66,75,94]. For instance, research suggests that self-reported habitual use of suppression is associated with steeper cortisol awakening responses [84] and greater cortisol reactivity to stressors [66,94]. However, given that these results are correlational, they do not provide sufficient evidence of a causal link between emotion regulation and hormonal changes. It may be that greater cortisol reactivity to stressors incites greater use of suppression or that habitual use of suppression is associated with other factors (e.g., poor regulation of activity in the autonomic nervous system) that may affect hormone levels. In order to establish if, and the extent to which, emotion regulation affects hormone levels, there is a need for experimental research through which causal inferences can be drawn. Studies demonstrating the impact of experimentally instructed emotion regulation on hormones do exist [24,122], but such possible effects have yet to be systematically evaluated.

When investigating the effect of emotion regulation on hormone levels, it is important to consider potential moderating factors. In the sections below, we elaborate on the following potentially moderating factors: 1) type of hormone and the context, 2) emotion regulation characteristics (i.e., emotion regulation goal and type of emotion regulation strategy), and 3) type of population (i.e., healthy vs. psychiatric). Based on previous research of hormones in the context of human emotional behavior [6], we focus on the following hormones: cortisol, testosterone, oxytocin, vasopressin and estradiol.

1.1. Type of hormone and context

Different hormones appear to be exerting their influence differently dependent on the context. Hence, two potential moderating factors of the effect of emotion regulation on hormone levels are the type of hormone under investigation and the context within which the hormones are investigated.

Although experimental studies linking specific discrete emotions (e.g., sadness, happiness) with specific changes in hormone levels are sparse and show inconsistent results, there is fairly robust evidence of differences in hormonal responses to different emotional *contexts* [92]. The strongest evidence of the impact of emotional contexts on hormone levels comes from experimental studies of hormonal responses to *stressful social-evaluative contexts* (e.g., the Trier Social Stress Test, TSST; [56]). Such studies provide clear evidence of increased levels of cortisol in response to stressful social-evaluative contexts [27,34,44], while the impact of such contexts on testosterone, oxytocin, estradiol and vasopressin is less clear [89,99]. Hormonal changes have also been investigated in other emotional contexts, including social challenges, competitions, and mate-seeking [32,121]. Results from these studies indicate increases in testosterone in response to social challenges (e.g., insults; [18,59]), competition against others (especially when winning; [3]), and cues and interactions relevant to reproduction (e.g., exposure to pornographic movies; [121]). Within warm and friendly emotional contexts, experimental studies suggest an increase in oxytocin both when interacting with close social partners [36] and with strangers [54]. Cortisol, testosterone, and oxytocin responses to emotional contexts have received the bulk of empirical attention, while the impact of emotional contexts on hormones such as estradiol and vasopressin remains relatively unexplored.

1.2. Emotion regulation characteristics

A second potential moderating factor of the effect of emotion regulation on hormone levels pertains to emotion regulation characteristics, including the type of emotion regulation strategy applied and the emotion regulation goal.

Historically, researchers have distinguished between putatively adaptive and maladaptive emotion regulation strategies [4,8], implying that some strategies (e.g., suppression of expression) are less effective at regulating an emotional response compared to others (e.g., reappraisal; [8]). However, researchers now recognize that no emotion regulation strategy is inherently effective or ineffective in and of itself [4,60]. Instead, the effectiveness of emotion regulation strategies is believed to vary according to the context within which they are applied [8,60]. For example, it has been proposed that distraction may be more effective in the context of intense negative emotions than reappraisal, which may in turn be more effective than distraction in the context of less intense negative emotions [102,103]. Although there is a relative dearth of experimental research directly investigating the effectiveness of emotion regulation strategies in different contexts, a few studies appear to support the idea that emotion regulation effectiveness vary according to context (e.g., reappraisal is more effective in the context of controllable stressors vs. uncontrollable stressors; [60,112]). It follows that the hormonal effect of emotion regulation may also vary according to the type of strategy applied, considering the emotional context.

Concerning goals, researchers investigating emotion regulation have predominantly focused on pro-hedonic goals (i.e., downregulating negative emotions and upregulating positive emotions; [85,111]). However, emotion regulation strategies may also be applied in the service of contra-hedonic goals (i.e., downregulating positive emotions and upregulating negative emotions; [111]). For example, one may suppress the urge to laugh after witnessing a co-worker trip over their feet or one may ruminate about how one was wronged to increase anger in

preparation for a fight. In everyday life, pro-hedonic goals are far more common than contra-hedonic goals [31,40]. Given a greater focus on, and experience with, pro-hedonic goals in everyday life, people may be better able to regulate their responses – including their hormonal responses – in the context of such goals as opposed to contra-hedonic goals.

1.3. Type of population

Researchers suggest that abnormal responses to emotional situations and difficulties in regulating emotions may represent transdiagnostic constructs underlying a broad range of mental health disorders [10,107]. A third potential moderating factor of the effect of emotion regulation on hormone levels may thus concern the psychiatric characteristics of the sampled population.

Within the context of clinical research, studies suggest that self-reported emotional reactions to emotional situations and emotion-regulation abilities may differ between people with mental health disorders (e.g., depression, anxiety, bipolar disorder) and healthy controls without mental health disorders [14,5,23,71,115]. For example, research has linked depression with abnormal self-reported reactivity to emotional situations [13,5], and with difficulties regulating emotions [71,115]. Furthermore, research suggests that hormonal reactivity to emotional situations differs between people with mental health disorders and healthy people [16,123]. For example, a recent meta-analysis suggests blunted cortisol reactivity to stressors in women with affective disorders (i.e., anxiety and depression) and increased cortisol reactivity to stressors in men with affective disorders [123]. Given such differences in both emotional reactivity (both self-reported and hormonal) and emotion regulation abilities between people with mental health disorders and healthy individuals without mental health disorders, it is possible that hormonal responses to emotion regulation differ as well.

1.4. Aims

The aims of the present systematic review and meta-analysis were to review and synthesize the available experimental evidence pertaining to the impact of emotion regulation on hormone levels, and to investigate whether the impact of emotion regulation on hormones varies according to type of hormone (i.e., testosterone, cortisol, oxytocin, estradiol and vasopressin), context (i.e., emotion-induction procedure), emotion regulation characteristics (i.e., emotion regulation goal and type of emotion regulation strategy), and the presence and type of psychiatric disorders.

2. Materials and methods

The present review and meta-analysis was preregistered at PROSPERO (CRD42020157336). Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; [81]) guidelines were followed for reporting its results.

2.1. Search strategy

The electronic databases PubMed, PsycINFO, and CINAHL were searched without time restrictions (until December 3rd, 2019). The search terms were established using the PICO model (see Appendix A). Search filters were employed to limit the search to English language, peer-reviewed, journal research articles on quantitative studies with human adults (18 years or older). Additional records were identified through "backward searching" in reference lists of identified relevant records and "forward searching" in newer publications citing identified relevant records.

2.2. Inclusion criteria

Only peer-reviewed, English-language, experimental studies assessing the effect of instructed emotion regulation on levels of prominent hormones (i.e., testosterone, cortisol, oxytocin, estradiol and vasopressin) in physically healthy adults (18 years and above) were included. To be eligible, studies had to assess differences in hormone levels between two or more experimental conditions (i.e., between different types of emotion regulation conditions or between emotion regulation and control conditions) or between two or more different populations (e.g., clinical and non-clinical) within the same experimental condition. Records on case studies, qualitative studies, and grey literature (i.e., abstracts, dissertations, and literature published outside traditional academic publishing channels) were excluded.

The search and selection process consisted of two steps: first, two independent researchers screened titles and abstracts of the retrieved articles, applying the inclusion and exclusion criteria to identify relevant records. Second, the two researchers screened full texts of eligible records to determine whether they should be included. Disagreements were resolved through negotiation and consultation with the last author.

2.3. Risk of bias

A pre-specified checklist proposed by Laufer and colleagues [69] was used to assess the presence of potentially confounding influences during measurement of hormones (see Appendix B). The first author rated all articles according to the checklist and the second author checked all ratings for accuracy. The assessments of consideration of confounders yielded aggregated scores between 0 and 16. Studies were categorized based on tertiles of the aggregated scores for relevant items as having poor consideration of confounders (<33% of achievable score), fair consideration of confounders (33.1–66% of achievable score) or excellent consideration of confounders (>66.1% of achievable score).

2.4. Data extraction

Descriptive data were extracted by two independent researchers using a pre-specified data-extraction sheet (see Appendix C). Effect sizes were extracted or computed by the first author and checked for accuracy by the last author. When sufficient data or key details were not reported, a request for data was made to study author(s). When authors failed to respond or were unable to supply the requested data (i.e., means and standard deviations to enable calculation of effect sizes), effect sizes were estimated from other statistics including *N*, *t*-values, *F*-values, Beta-values, and *p*-values.

2.5. Statistical analysis

In addition to a narrative synthesis of the findings, we conducted meta-analyses synthesizing effects from identified studies. The primary outcome in the meta-analyses was the effect of emotion regulation instructions on hormone levels operationalized as the difference in hormone levels 1) *between* conditions (i.e., between an active experimental group who were instructed to regulate and a passive control group who were not instructed to regulate) and 2) *within* experimental conditions (i.e., from an unregulated response to a regulated response calculated only for participants who received an emotion regulation instruction). Hence, between-group effects indicate the extent to which emotion regulation is associated with goal-consistent changes in hormone levels (e.g., larger decrease in cortisol for reappraisal vs. control group). Within-group effects index changes in hormones only for participants who received an instruction to regulate. Hence, they do not speak to the effect of emotion regulation on hormone levels on their own as there is no way of knowing whether such changes would also occur in the absence of emotion regulation instructions. Rather, within-group effects were included to nuance the findings of the between-group analyses. If between-group effects are significant, separate analyses of within-group effects only for participants who received an instruction to regulate may be used to further elaborate on the strength of effects for the various

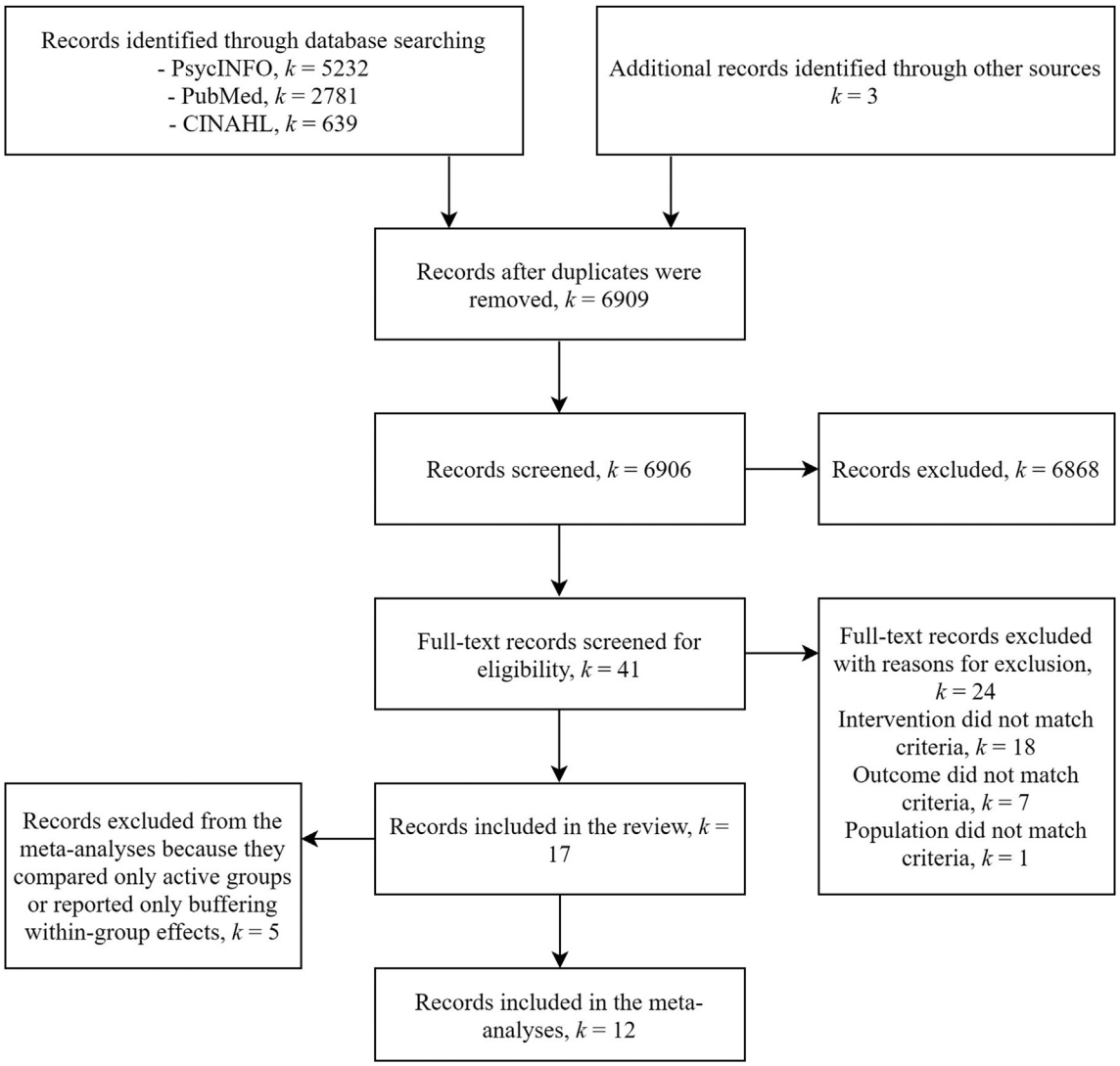

Fig. 1. PRISMA flow chart of study selection process. After PRISMA flow diagram [80].

strategies. If the between-group effects are not significant, separate analyses of within-group effects may be applied to illustrate the natural trajectory of hormonal changes *regardless* of emotion regulation instructions.

Due to varying study designs (i.e., variation in timing of emotion regulation instructions and measurement of hormone levels), we distinguish between *buffering effects* and *reactivity effects* as these could not be averaged in a meaningful way. Buffering effects refer to effects derived from studies where participants received an instruction to regulate their emotions *before* they were exposed to emotion induction, and where hormone levels were assessed at baseline and during emotion regulation. That is, these effects refer to the change from baseline (i.e., before participants received instructions) to regulated emotional response (i.e., when participants applied the instructed strategy). For instance, within a stress context, one would expect an increase in cortisol for all participants, although smaller for those who received instructions to down-regulate their emotions. Reactivity effects, on the other hand, refer to effects derived from studies where participants received an instruction to regulate their emotions *after* they were exposed to emotion induction. That is, these effects refer to change from an unregulated emotional response (i.e., reactivity when participants were exposed to emotion induction with no instruction to regulate) to a regulated emotional response (i.e., reactivity when participants were exposed to emotion induction after receiving an instruction to regulate). As such, within a stress context, one would expect a decrease in cortisol for participants who received instructions to down-regulate their emotions.

Effect sizes were calculated as the standardized mean differences adjusted for small samples (Hedges' *g* [42], with 95% confidence intervals between groups and/or between conditions. Hedges' *g* is a variation of Cohen's *d* that is more reliable when analyses are based on studies with small samples [9]. To ensure independence among observations [9], effect sizes were averaged across and within outcomes such that each study was represented with only one effect size in each analysis. Each effect size was weighted by its precision (inverse variance), so that studies with larger samples contributed more to the estimated overall effect size [9].

To determine whether emotion regulation affects hormone levels, we conducted meta-analyses for each type of hormone investigating 1) between-group effects, comparing change in hormone levels between active experimental groups and passive control groups. A significant positive effect indicates that the active group experienced a larger goal-consistent change in hormones than the control group (e.g., the experimental group was better at down-regulating cortisol in response to a stressor compared to the control group) and 2) within-group effects, comparing hormonal responses to emotion-inducing stimuli before and after receiving an instruction within the same experimental group. A significant positive effect indicates a significant goal-consistent change in hormones (e.g., there was a significant decrease in cortisol from

Table 1
Overview of study characteristics of included studies.

Author	N	Effects; Buffering or reactivity effects	Effects; Between or within comparisons	Hormonal outcome	Emotion regulation strategy	Emotion regulation goal	Induction procedure	Emotion induced	Population type; healthy or psychiatric
Akinola et al. (2016) [2]	97	Buffering	Between (control), within	Salivary cortisol	Reappraisal	Downregulate neg	Social evaluative	Negative	Healthy
Cruess et al. (2015) [21]	120	Buffering	Between (active, control), within	Salivary cortisol	Mindfulness, somatic relaxation	Downregulate neg	Social evaluative	Negative	Healthy
Denson et al. (2009) [25]	48	Buffering, reactivity	Between (active), within	Salivary cortisol	Rumination (provocation-focused), rumination (self-focused), distraction	Downregulate neg	Provocation	Negative (anger)	Healthy
Denson et al. (2014), experiment 1 [24]	85	Buffering	Between (control), within	Salivary cortisol	Reappraisal	Downregulate neg	Social evaluative	Negative	Healthy
Denson et al. (2014), experiment 2 [24]	88	Buffering	Between (control), within	Salivary cortisol	Reappraisal	Downregulate neg	Pain	Negative	Healthy
Kane et al. (2018) [52]	66	Buffering	Between (control), within	Salivary cortisol	Expression	Downregulate neg	Social evaluative	Negative	Healthy
Kinner et al. (2014) [55]	72	Buffering	Between (active), within	Salivary cortisol	Reappraisal, distraction	Downregulate pos and neg, upregulate pos and neg	Visual	Positive, negative	Healthy
Kogler et al. (2015) [61]	40	Buffering, reactivity	Within	Salivary cortisol	Combination (suppression and reappraisal)	Downregulate neg	Performance	Negative	Healthy
Kuehner et al. (2009) [65]	58	Buffering, reactivity	Between (active), within	Salivary cortisol	Mindfulness, distraction, rumination	Downregulate neg	Combination (visual and auditory	Negative (sadness)	Healthy
Lemoult et al. (2014) [70]	97	Reactivity	Between (active), within	Salivary cortisol	Distraction, rumination	Downregulate neg	Social evaluative	Negative (sadness)	Both; a healthy group and a group with major depressive disorder
Mauersberger et al. (2018) [75]	145	Buffering,	Between (control & active), within	Salivary cortisol	Reappraial, suppression	Downregulate neg	Social evaluative	Negative	Healthy
Peters et al. (2016a) [88]	88	Buffering	Between (active), within	Salivary cortisol	Expression, suppression	Downregulate neg, upregulate neg	Visual	Negative	Healthy
Peters et al. (2016b) [87]	88	Buffering	Between (active), within	Salivary testosterone	Expression, suppression	Downregulate neg, upregulate neg	Visual	Negative	Healthy
Rozek et al. (2018) [96]	100	Buffering	Between (control), within	Salivary cortisol	Attention to current thoughts	Downregulate neg	Social evaluative	Negative	Healthy
Salzmann et al. (2018) [98]	71	Buffering	Between (active), within	Salivary cortisol	Gratitude, distraction, self-efficacy enhancement	Downregulate neg	Combination (social evaluative and pain)	Negative	Healthy
Shull et al. (2016) [104]	71	Buffering	Between (active), within	Salivary cortisol	Distraction. rumination	Downregulate neg	Social evaluative	Negative	Healthy
Zhan et al. (2017) [120]	60	Buffering; reactivity	Within	Salivary cortisol	Reappraisal	Downregulate neg	Social evaluative	Negative (anger)	Healthy
Zoccola et al. (2014) [122]	32	Buffering; reactivity	Between (active), within	Salivary cortisol	Distraction. rumination	Downregulate neg	Social evaluative	Negative	Healthy

Note: total N is based on the total sample for which hormonal outcomes were available.

Table 2
Pooled effect sizes for between-group effects across outcomes and moderator variables.

	Sample size		Heterogeneity				Global Effect Sizes			
Outcome	*K*	*N*	Q^a	df	*p*	I^2	Hedges' g^b	95% CI	95% PI	*p*
Buffering effect (baseline to regulated reactivity)										
Combined effects	6	581	4.96	5	.42	0.00	−0.05	−0.20; 0.10	-	.48
Type of strategy										
- Reappraisal	4	415	2.82	3	.42	0.00	−0.04	−0.25; 0.16	-	.70
Context										
- Social evaluative	5	493	3.13	4	.54	0.00	−0.01	−0.17; 0.14	-	.86
Type of goal										
- Down-regulate neg.	6	581	4.96	5	.42	0.00	−0.05	−0.20; 0.10	-	.48
Population										
- Healthy	6	581	4.96	5	.42	0.00	−0.05	−0.20; 0.10	-	.48
Timing of measurement										
- 0–30 minutes after induction	5	514	6.61	4	.16	39.52	−0.13−	−0.35; 0.09−	-	.24
- 30+ minutes after induction	4	339	1.41	3	.70	0.00	0.07	0.26; 0.12	-	.48

Note: CI = confidence intervals, PI = prediction intervals. [a]For the *Q*-statistic, *p*-values of < .10 are considered indicative of heterogeneity. [b]Effect sizes are reported as Hedges' *g* (standardized mean differences, adjusted for small sample bias) and can be interpreted with reference to the guidelines: <0.3 = small, 0.5 = medium and >0.8 = large [17]. A positive effect size indicates that the active group (i.e., participants instructed to regulate) experienced a larger goal-consistent change in hormones than the control group (i.e., participants not instructed to regulate), while a negative effect size indicates the opposite. Adhering to the principle of independency between effects, effect sizes were combined when studies reported results for more than one measure. Thus, only one effect size per study was used in the analyses.

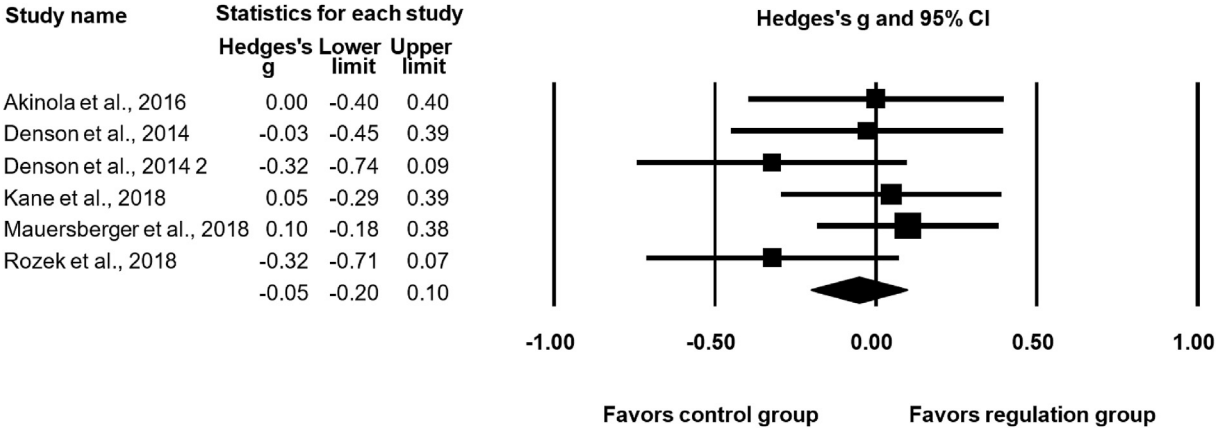

Fig. 2. Forest plot for pooled effect sizes for between-group effects.

Table 3
Pooled effect sizes for within-group effects across outcomes and moderator variables.

	Sample size		Heterogeneity				Global Effect Sizes			
Outcome	*K*	*N*	Q^a	df	*p*	I^2	Hedges' g^b	95% CI	95% PI	*p*
Regulating effect (reactivity to regulated reactivity)										
Combined effect	6	335	7.31	5	.20	31.64	0.18	0.02; 0.35	-	**.03**
Type of strategy										
- Distraction	4	235	1.31	3	.73	0.00	0.39	0.16; 0.62	-	**<.01**
- Rumination	4	235	0.44	3	.93	0.00	0.23	0.03; 0.43	-	**.02**
Context										
- Social evaluative	3	189	7.02	2	.03	71.51	0.18	−0.26; 0.63	−4.96; 5.32	.42
Type of goal	6	335	7.31	5	.20	31.64	0.18	0.02; 0.35	-	**.03**
- Down-regulate neg.										
Type of population										
- Healthy	6	289	8.61	5	.13	41.90	0.20	0.02; 0.38	-	**.03**
Timing of measurement										
- 0–30 minutes after induction	4	206	3.39	3	.34	11.40	0.05	−0.11; 0.20	-	.55
- 30+ minutes after induction	3	138	0.49	2	.78	0.00	0.35	0.17; 0.52	-	**<.01**

Note: CI = confidence intervals, PI = prediction intervals. [a]For the *Q*-statistic, *p*-values of < .10 are considered indicative of heterogeneity. [b]Effect sizes are reported as Hedges' *g* (standardized mean differences, adjusted for small sample bias) and can be interpreted with reference to the guidelines: <0.3 = small, 0.5 = medium and >0.8 = large [17]. A positive effect size indicates a goal-consistent change in hormones from unregulated emotional response to regulated emotional response, while a negative effect size indicates the opposite. Adhering to the principle of independency between effects, effect sizes were combined when studies reported results for more than one measure. Thus, only one effect size per study was used in the analyses.

unregulated emotional responses to regulated emotional responses). Note that since it would be difficult both to interpret between-group effects between two active groups and within-group buffering effects (i.e., change from baseline to regulated response), because it would be unclear whether effects indicate successful or non-successful regulation, the between-group meta-analysis was only conducted with between-group effects between a passive control group and an active group, while the within-group meta-analysis was only conducted with within-group reactivity effects (i.e., change from unregulated response to regulated response). To address the second research question of whether the effects vary according to the proposed categorical moderators: type of hormone, context, emotion regulation characteristics (emotion regulation strategy and emotion regulation goal), and presence and type of psychiatric disorder, planned moderator analyses (i.e., meta-ANOVAs or meta-regressions) and separate subgroup meta-analyses were conducted. Specifically, when more than $K = 3$ studies were available for two or more levels of a categorical moderator, meta-ANOVAs or meta-regressions were conducted. When more than $K = 3$ studies were available for only one level of a categorical moderator, a separate meta-analysis was conducted for that level of the moderator. Lastly, other sources of heterogeneity were explored, including gender distribution (percentage), mean age, years of education, income, race/ethnicity, timing of measurement, and consideration of confounders. These moderators were included because they have previously been shown to affect hormone levels and hormonal reactivity [26,67,95,113]. Gender distribution and mean age were specified as moderators a priori, while years of education, income, race/ethnicity, timing of measurement, and consideration of confounders were specified as moderators post hoc. The possible moderating effects of the moderators gender distribution, mean age, years of education, income, and race/ethnicity were explored using meta-regressions, while separate moderator analyses and subgroup meta-analyses were conducted to explore timing of measurement and consideration of confounders.

The presence and extent of heterogeneity between studies (i.e., the degree to which the dispersion of effect sizes may be due to variation beyond that of sampling error) were estimated using the Q- and the I^2-statistics. The Q-statistic provides an estimate of whether the heterogeneity is statistically significant (i.e., $p = .10$; [90], and the I^2-statistic indicates the extent of the heterogeneity in percentages [45]). When the results suggested heterogeneity, i.e., $Q > 0.10$, the expected dispersion of effect sizes across various populations was explored by calculating the 95% prediction interval.

Publication bias was explored visually by use of funnel plots and statistically using Egger's tests for funnel plot asymmetry [29]. In accordance with best-practice recommendations [33], hypotheses about potential moderating factors were tested regardless of the results of the heterogeneity tests.

All meta-analyses were computed using random-effects models as these models are pertinent when the goals is to estimate the mean of a distribution of true effect sizes and because they permit heterogeneity in the analyses of overall effects [9]. The meta-analyses were conducted using Comprehensive Meta-Analysis software (version 3.3.070, Biostat, Englewood, NJ, USA).

2.5.1. Supplementary Bayesian analyses

To aid the interpretation of the results, we conducted Bayesian Model-Averaged meta-analyses [37] of the overall between-group and within-group effects. The procedure examines results of four models: a) fixed-effect null hypothesis. b) fixed-effect alternative hypothesis, c) random-effects null hypothesis, and d) random-effects alternative hypothesis. Bayesian Model-Averaged analyses address two questions in light of the observed data: What is the plausibility that the overall effect is non-zero and is there between-study variability of the true effect sizes? We chose an uninformed prior probability, i.e., 25%, of the four models and 2000 iterations. We used the previously recommended default of a zero-centered Cauchy prior with a scale of 0.707 for the effect size [37]. For the between-study variation, we used an empirically informed prior distribution of non-zero between-study deviation estimates based on standardized mean difference effect sizes from 705 meta-analyses [116]. This distribution has been approximated by an Inverse-Gamma (1, 0.15) prior on the standard deviation (Tau) [37]. The Bayesian analyses were conducted with the computer software JASP (Version 0.12.2) [48].

3. Results

3.1. Results from search and data extraction

Fig. 1 shows a PRISMA flowchart delineating the search and selection process. A total of 6906 records were identified through the literature search, while three records were identified through other sources. The search and selection process yielded 17 relevant records to be included in the present review (see Table 1). Out of the 17 studies, five studies did not provide relevant data for the meta-analysis, because they either 1) provided between-group effects for comparisons of only *active experimental groups* (e.g., comparing a group instructed to reappraise with a group instructed to suppress) or 2) provided within-group *buffering* effects (i.e., change in hormones from baseline to regulated response). Agreement about inclusion and exclusion was 99% in both the preliminary screening and the full-text screening. Of the included records, one record reported results from two experiments [24], while two records described results from the same experiment [87,88]. The final sample of studies (marked with an asterisk in the reference list) comprised 17 experiments, 1338 participants in total, and were published in scientific journals between 2009 and 2018.

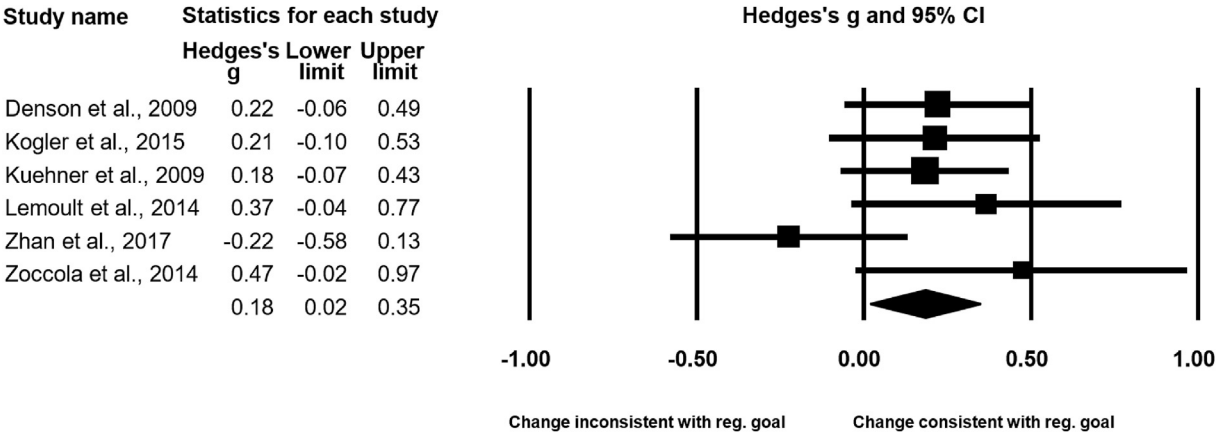

Fig. 3. Forest plot for pooled effect sizes for within-group effects.

For the 12 studies included in the meta-analysis, a request to study authors for data needed (means and standard deviations at the various timepoints) to calculate effect sizes was made, to which all 12 authors replied. An overview of study characteristics can be found in Table 1. The majority of the included studies investigated salivary cortisol ($K = 17$). Only one study assessed salivary testosterone, while no studies assessed vasopressin, oxytocin and estradiol.

3.2. Results from meta-analyses

All but one of the studies included in the present review investigated cortisol. Meta-analyses were conducted combining effects for studies of cortisol. For between-group effects, none of the included studies employed a reactivity design (i.e., change from unregulated response to regulated response). Hence, the between-group meta-analysis was conducted with studies investigating buffering effects (i.e., change from baseline to regulated response) comparing groups who received an emotion regulation instruction with control groups who did not. Here, the overall combined effect was non-significant indicating no significant differences in regulation of hormonal responses between groups who were instructed to regulate their emotions and control groups. Results are presented in Table 2. Fig. 2 shows a forest plot for effects. The number of studies was insufficient to conduct formal moderator analyses. Non-significant effects were identified for the subgroup meta-analyses conducted for the following categories of the proposed moderators: reappraisal strategy instructions, social evaluative emotion induction, goals aimed at downregulating negative emotions, and healthy populations. Concerning other sources of heterogeneity, meta-regressions indicated no significant moderating effects of gender distribution ($B = 0.01$; $SE = 0.01$; $p = .26$; $K = 6$) or mean age ($B = 0.02$; $SE = 0.01$; $p = .14$; $K = 6$). In order to assess the moderating effect of timing of measurement, a dichotomous variable indexing measurements conducted at 0–30 minutes after emotion induction and measurements conducted more than 30 minutes after emotion induction was created. As cortisol reaches peak concentrations in saliva 15–20 minutes after activation of the adrenal glands [35,57], the variable was created so as to capture both cortisol *during* emotion induction (assessed 0–30 minutes after emotion induction) and cortisol *after* emotion induction (assessed more than 30 minutes after emotion induction). The results suggested no significant effects according to timing of measurement (see Table 2). Too few studies reported income, education level or race/ethnicity to conduct moderator analyses for these variables.

A meta-analysis was then conducted to explore within-group reactivity effects. Similar to the between-group meta-analysis, the within-group meta-analysis was conducted only for studies of cortisol. Results are presented in Table 3. Fig. 3 shows a forest plot for effects. The overall combined effect was significant, indicating a significant within-group, goal-consistent change in hormone levels from unregulated emotion response to regulated emotion response. The number of studies was insufficient to conduct formal moderator analyses. Significant, goal-consistent changes were identified in subgroup meta-analyses only including studies applying a distraction or rumination instruction, studies investigating goals aimed at downregulating negative emotions, and studies only including healthy participants. Results from subgroup meta-analyses for studies applying social evaluative emotion induction were not significant. Regarding other sources of heterogeneity, results suggested significant positive effects when measurements of cortisol were conducted *after* 30 minutes, but not *before* (see Table 3). Meta-regressions indicated no significant moderating effects of gender distribution ($B = <0.01$; $SE = 0.01$; $p = .44$; $K = 6$) or mean age ($B = <0.01$; $SE = 0.01$; $p = .29$; $K = 6$). Too few studies reported income, education level or race/ethnicity to conduct moderator analyses for these variables.

Of the studies included in the meta-analyses, three studies included emotion induction characterized by specific features or additional manipulations [52,75,120]. Excluding these studies from the meta-analyses did not significantly alter the results.

3.3. Risk of bias

Funnel plots and Egger's tests indicated no evidence of publication bias for the overall between-group analysis, $p > .12$, for the overall within-group analysis, $p > .66$, nor for any of the subgroup meta-analyses, $ps > .12$.

Consideration of confounders was categorized as poor in two studies, fair in six studies and excellent in seven studies (see Appendix D). It is worth noting that 10 studies considered the confounding effect of somatic disease, while only four studies considered the confounding effect of psychological comorbidities and none considered the confounding effect of lifetime trauma. There were insufficient studies to compare effects according to categories for consideration of confounders. Subgroup meta-analyses conducted separately according to categories for consideration of confounders are reported in Appendix D. Note that the effects included in the meta-analyses were *not* adjusted for confounders.

3.4. Heterogeneity of effects

The *Q*-statistics suggested no significant systematic variation in effects overall, nor for the separate subgroup meta-analyses conducted except for the findings from the within-group subgroup meta-analysis for studies investigating social evaluative emotion induction. For the between-group effects, the I^2 largely confirmed the absence of heterogeneity. For the within-group effects, the I^2 indicated some heterogeneity both overall and for the subgroup meta-analysis investigating effects within the context of social evaluative emotion induction, healthy population and effects derived from cortisol measurement between 0 and 30 minutes after emotion induction. These finding should be viewed in the light of the small number of studies included in the meta-analyses, which may have biased the results of the heterogeneity tests (cf. [117]).

3.5. Post-hoc power analyses

A post-hoc random effects statistical power-analysis [43] revealed that to detect an effect similar in magnitude to the observed between-group effect ($g = -0.05$), with an alpha of 5% and a statistical power of 80%, would require 220 trials with an average of 96 participants per trial. To detect a combined effect of 0.18, similar to that found in the within-group meta-analysis, with an alpha of 5% and a statistical power of 80%, would require 29 trials with an average of 56 participants per trial.

3.6. Supplementary Bayesian results

For the combined between-group effect of emotion regulation on cortisol, the results of the Bayesian Model-Averaged meta-analysis revealed moderate evidence for the null hypothesis, i.e., that there is no effect of emotion regulation on cortisol, corresponding to a Bayes Factor (BF), i.e., the likelihood ratio of the marginal likelihood of the two competing hypotheses, of 8.80. Given the prior distributions and the observed data, the null-hypothesis is 8.8 times more likely than the alternative hypothesis. In contrast, the BF for homogeneity was only 2.40, indicating that the probability that the effect sizes are homogenous is only twice the probability that they are heterogeneous, a probability classified as anecdotal evidence [50]. The posterior probabilities of each of the four hypotheses were as follows: the fixed-effect null hypothesis (63.8%), the random-effects null hypothesis (26.0%), the fixed-effect alternative hypothesis (6.8%), and the random-effects alternative hypothesis (3.4%).

The Bayesian Model-averaged meta-analysis of the within-group effect showed that the alternative hypothesis was slightly more likely (58.5% probability) than the null hypothesis (41.75%), yielding a BF of 1.41, corresponding to anecdotal evidence [50]. Likewise, given the observed data, the probability of heterogeneous effect sizes (52.1%) only slightly exceeded the probability of homogeneous effect sizes (47.9%)

(BF of 1.1).

3.7. Narrative review

To further contextualize the lack of effect of emotion regulation on hormones, we provide a narrative review of studies including participants with psychiatric disorders, studies comparing at least two active groups (i.e., groups that receive an emotion regulation instructions), studies that investigated measures of trait emotion regulation, and studies that investigate self-reported emotions.

3.7.1. Presence and type of psychiatric disorder

Only one study included a psychiatric population and it was therefore not possible to conduct a subgroup meta-analysis exploring effects within the context of psychiatric populations. In addition to a group of healthy individuals, LeMoult and Joormann [70], included participants with major depressive disorder (MDD). Healthy participants and participants with MDD were randomly assigned to receive a distraction instruction or a rumination instruction. The results revealed that regardless of instruction, the healthy population experienced a significant decline in cortisol levels from unregulated emotional response to regulated emotional response. However, participants with MDD only experienced a significant decline in cortisol levels when receiving the distraction instruction. Group comparisons revealed that the change in cortisol levels for participants with MDD who were instructed to ruminate significantly differed from the change in cortisol level experienced by healthy participants and by participants with MDD who were instructed to distract themselves.

3.7.2. Comparison of active groups

Out of the included studies assessing cortisol in healthy participants, 10 studies compared two or more active groups. Five of these 10 studies only compared active groups and had a design that prohibited them from being included in the within-group meta-analysis. Out of the 10 studies, four compared rumination and distraction. Of these four studies, three studies found no significant between-group differences in changes in cortisol [65,70,104], while one study found that distraction was associated with more goal-consistent changes in cortisol levels than rumination [122]. It should be noted that in the study by Kuehner and colleagues [65], the distraction group was combined with a mindfulness group and subsequently compared to a rumination group. Six other studies reported between-group differences for active groups. Comparing mindfulness and somatic relaxation, Cruess and colleagues [21] found that somatic relaxation was associated with more goal-consistent changes in cortisol levels than mindfulness. Comparing self-focused rumination, provocation-focused rumination and distraction, Denson and colleagues [25] found that provocation-focused rumination and distraction were associated with more goal-consistent changes in cortisol levels than self-focused rumination, while there were no significant differences between provocation-focused rumination and distraction. Comparing expression and suppression, Peters and colleagues [87,88] found that expression was associated with more goal-consistent changes in cortisol levels than suppression, while no between group-differences in changes in testosterone were reported. Comparing gratitude, distraction and self-efficacy enhancement, Salzmann and colleagues [98] found that self-efficacy enhancement and distraction led to more goal-consistent changes in cortisol levels than gratitude, while there were no significant differences in effects between self-efficacy enhancement and distraction. Absence of group differences were reported in a study comparing reappraisal and distraction [55] and a study comparing reappraisal and suppression [75].

3.7.3. Trait emotion regulation

To provide further context to the findings from the meta-analyses, we reviewed studies assessing trait emotion regulation. Five of the included studies assessed trait emotion regulation at strategy level in addition to the experimental investigation of the effect of emotion regulation instructions on hormones [61,70,75,98,104]. Of these five studies, three studies provide results from analyses of interaction effects between trait emotion regulation and the state emotion regulation manipulation. Mauersberger and colleagues [75] reported that trait reappraisal was associated with more effective regulation of cortisol in the context of a state reappraisal manipulation. Shull and colleagues [104] found that trait rumination was associated with less effective regulation of cortisol in the context of a state rumination manipulation. Lastly, Salzmann and colleagues [98] found no interaction effects between trait gratitude and a state gratitude manipulation on cortisol.

3.7.4. Self-report measures of emotions

As emotion regulation hinges on the very presence of an emotion, it is important to explore self-reported emotional experiences when assessing emotion regulation. Of the included studies in the present meta-analyses, 12 studies assessed participants' self-reported emotional responses to the emotion-induction procedures. Nine studies included baseline self-report measures of emotions in addition to post-induction and/or post-regulation measures allowing for assessments of change in the self-reported experience of emotions following emotion induction and emotion regulation instruction. In the remaining three studies that included self-report measures of emotions, emotional responses were assessed retrospectively upon completion of the emotion-induction procedure. Hence, out of the studies included in the present review and meta-analysis, around half included self-report measures of emotions allowing them to assess whether the emotion-induction procedure did indeed elicit an emotional response that the participants could then regulate, and to assess whether participants did indeed succeed in regulating the emotion. None of the studies, however, investigated the association between (change in) self-reported emotional responses and change in cortisol levels, and there were not enough studies providing the necessary data for analyses of the moderating effect of change in self-report measures of emotion on change in hormone levels in the present review and meta-analysis.

4. Discussion

The present systematic review and meta-analysis aimed to review and synthesize the available experimental evidence pertaining to the impact of emotion regulation on hormone levels, and to assess whether the impact of emotion regulation on hormone levels varies according to four pre-specified moderators: type of hormone, context, emotion regulation characteristics, and the presence and type of psychiatric disorders. Overall, the review and meta-analysis revealed major gaps in the current state of research, with one of the most distinct shortcomings being the lack of research pertaining to the effect of emotion regulation on other hormones than cortisol. Only one of the 17 identified studies investigated a different type of hormone than cortisol (i.e., testosterone), and the meta-analysis was therefore only conducted with studies assessing cortisol. Concerning between-group buffering effects (i.e., comparing change in hormone levels between active experimental groups and passive control groups), no significant effects were detected, indicating that emotion regulation instructions were not more or less effective than no instruction in terms of regulating cortisol. This conclusion based on the frequentist meta-analysis was corroborated by the Bayesian analysis, showing that, based on the observed data, the null-finding is almost nine times more likely than the alternative hypothesis. The overall within-group results showed significant, goal-consistent changes from unregulated response to regulated response, but the Bayesian analysis indicated that this finding was only slightly more likely than a null-finding. Taken together, these results tentatively suggest that emotion-induction procedures are associated with increases in cortisol that may subsequently return to equilibrium regardless of emotion regulation instructions. However, more research is needed to confirm the findings.

So why may emotion regulation instructions *not* lead to a significantly

greater goal-consistent change in hormonal responses to an emotional situation than no instruction? Below, we review three possible answers pertaining to: 1) the correlation between self-reported experiences and hormonal changes, 2) participants' regulatory efforts and regulatory effectiveness, and 3) sensitivity of the method of measurement.

First, one explanation for the findings may be that the correlation between self-reported experiences of emotions and changes in cortisol is negligible. As such, the participant may succeed in regulating their self-reported emotional response upon exposure to an emotional stressor, but this has little to no effect on the hormonal response to this stressor. This interpretation is consistent with research reporting that most studies examining covariation between self-reported emotional responses to psychosocial stressors and salivary cortisol find little to no covariation between them [15,27]. Hence, it is possible that the two response systems (i.e., subjective and endocrine) are only loosely coupled and the interaction between them during emotional situations may vary. This is similar to the conclusion reached by researchers studying covariation between the subjective response system and the physiological response system (e.g., measures of activity of the autonomous nervous system such as skin conductance or heart rate) during emotions [68,76,77,105]. This is not as to say that cortisol, or hormones in general, are irrelevant to the study of emotion regulation. Instead, hormonal measures may be viewed as an important addition to a multimodal measurement approach, where measures tapping different response systems are cross-referenced in order to obtain a complete picture of the emotion regulation process.

Second, the absence of an effect of emotion regulation on hormones may be due to participants' regulatory efforts or (lack of) regulatory effectiveness. One possibility is that the cognitive exertion associated with applying an emotion regulation strategy may paradoxically have led to an increase in cortisol matching that of unregulated emotional responses. Indeed, several studies have shown that cognitive exertion is associated with an increase in cortisol [13,27] and it is generally acknowledged that all emotion regulation strategies require some level of cognitive effort [39,49]. Hence, the absence of an effect of emotion regulation on cortisol may be due to continued activation of the endocrine system in response to the cognitive demands of emotion regulation. Another possible explanation for the present findings may be that participants did not succeed in regulating their emotional response. Research generally indicates substantial individual differences in emotion regulation effectiveness (i.e., successful goal-consistent regulation of emotions; [8,28,51,60]). However, given that only around half of the included studies investigated change in self-reported emotional responses in addition to cortisol changes and none of the studies investigated the association between change in self-reported emotional responses and changes in cortisol levels, it is difficult to assess the validity of this explanation. Yet another possible explanation for the findings may be that different emotion regulation strategies have opposing effects on cortisol (e.g., rumination may be associated with an increase in cortisol while distraction may be associated with a decrease in cortisol), and when these effects are pooled in the meta-analyses, they cancel each other out leading to non-significant results. Given the small number of studies included in the meta-analyses, it was not possible to provide a comprehensive assessment of direction of effects at the strategy level.

Third, the findings may be explained with reference to the sensitivity of the applied hormone measures. All studies assessed salivary hormone levels. Saliva assessments of cortisol can be considered indirect measures as cortisol is released from the adrenal glands into the general circulation and subsequently slowly diffuse into saliva, reaching peak concentrations 15–20 minutes after activation of the adrenal glands [35,57]. The slow diffusion of cortisol into saliva may explain the finding from the within-group meta-analysis showing no effects when cortisol measurement was conducted between 0 and 30 minutes after emotion induction, but significant, goal-consistent effects when cortisol measurement was conducted after 30 minutes. Saliva assessments of cortisol may be considered less sensitive than blood samples for two reasons: first, cortisol hormones that are bound by proteins are too large to pass through the salivary gland, leaving only the "unbound" hormone fraction available for saliva assessments [35,57]. Second, saliva contains a relatively high concentration of cortisol-metabolizing enzymes that convert cortisol to cortisone [64,108]. For these reasons, cortisol levels found in saliva are significantly lower than the absolute cortisol levels found in blood, although the two types of measures are correlated [114,58]. As such, saliva cortisol assessments may not have been sensitive enough to capture between-group differences in changes in cortisol during emotion regulation in the studies included in the present meta-analysis. An additional consideration concerning sensitivity of the hormone measures pertains to sampling frequency. There was substantial variation in the number of times hormone levels were sampled in the included studies ranging from two times to 10 times. It is possible that more frequent sampling is needed to detect continuous changes in cortisol. Hence, lack of measurement sensitivity may explain why no effect of emotion regulation on cortisol was identified.

Concerning the pre-specified moderators, it was not possible to assess them using formal moderator analyses given the small number of studies in each category. The small number of studies can be considered a noteworthy finding in and of itself, speaking to the lack of research on the topic. Results from the subgroup meta-analyses largely mirrored the overall findings with non-significant results for between-group effects and significant results for within-group effects.

It should be noted that the overall findings of the meta-analyses pooled the effects of *different* types of emotion regulation strategies on cortisol, possibly obscuring any strategy-specific effects. Given the scarce number of studies investigating the same emotion regulation strategies, it was only possible to assess strategy-specific between-group effects for reappraisal and strategy-specific within-group effects for rumination and distraction. As different strategies may require different resources (e.g., reappraisal may require more self-control resources than distraction; [100,101,110]); and vary in effectiveness depending on the context (e.g., reappraisal may be more effective in the context of less intense negative emotions than distraction; [102,103]); and therefore impact hormones differently, a promising avenue for future research is to tease out such potential strategy-specific effects.

Furthermore, it is important to reiterate that the association between hormones and behavior is likely bidirectional. In the context of the present review and meta-analysis, we have investigated the effect of emotion regulation instructions on hormone levels, however, hormones may also affect emotion regulation. For example, an increase in cortisol following stress-induction may impede subsequent efforts to regulate emotions according to an emotion regulation instruction. In support of this idea, research has shown that exogenous administration of hormones (e.g., testosterone, cortisol) influences neural circuits associated with emotion regulation [74,118,119]. However, direct evidence linking exogenous administration of hormones to emotion regulation success or failure is lacking. Hence, an important area for future research is to clarify the bidirectional relationship between emotion regulation and hormones.

Concerning considerations of confounders, the majority of the studies could be categorized as fair or excellent in terms of their consideration of confounders. This finding shows that researchers are generally aware of the confounders that can potentially influence hormone assessments. However, there was a noteworthy pattern evident in the confounders considered. Specifically, while most studies considered physical confounders (i.e., somatic disease), psychological confounders (i.e., psychological comorbidities, lifetime trauma) were only considered in four out of 15 studies. This lack of attention to psychological confounders is also evident in the small number of studies assessing the confounding effect of trait emotion regulation and the small number of studies assessing changes in self-reported emotions during emotion regulation. The discrepancy between consideration of physical and psychological confounders may reflect the greater attention paid to physical confounders in research on hormones in general [109]. However, psychological confounders should not be overlooked, as a growing body of

evidence indicates that psychological factors such as the presence of mental health disorders, chronic stress and exposure to life stressors may alter hormonal responses to acute stressors [11,13,64]. Furthermore, the results reported by LeMoult and Joormann [70] and by Zhan and colleagues [120], indicate that the presence of mental health disorders or stress may alter hormonal responses to emotion induction and emotion regulation. However, more research is of course needed to determine the validity of this finding.

Aligning with the RDoC initiative, we believe that it is important to apply a multimodal assessment of psychological phenomena such as emotion regulation to obtain a more complete understanding of them. As the present review and meta-analysis have revealed, there is so much left to learn about the role of hormones in emotion regulation. One of the most noteworthy gaps in the current state of research is the lack of studies pertaining to other hormones than cortisol. Another prominent gap in the current state of research concerns self-report measures of emotions. To gain a better understanding of hormones in the context of emotion regulation, it is important to ascertain the subjective change in emotions from baseline to regulated response and/or from reactivity to regulated response in order to assess whether 1) an emotion was indeed induced, 2) the participant managed to regulate their subjective emotional response in a goal-consistent way, and 3) the subjective response system and the endocrine response system are correlated during emotion regulation or only loosely coupled. However, only around half of the reviewed studies included self-report measures of emotions allowing for assessments of self-reported change in emotions and there were not enough studies providing the necessary data for analyses of the moderating effect of changes in self-report measures of emotion on changes in hormone levels in the present meta-analysis. Lastly, we would like to advocate for the consideration of confounders in future research on hormones and emotion regulation, and for research addressing and teasing out the bidirectional relationships between hormones, emotions, and emotion regulation.

5. Limitations

First, the results from the frequentist power analyses and the Bayesian analyses indicate that the overall findings should be considered preliminary at best. Given the small number of available studies, we were not able to properly assess the moderating effect of all of the proposed moderators (e.g., type of hormone, level of education), leaving it unclear whether they had an effect on the results or not. Notably, as all but one of the available studies investigated cortisol, the conclusions drawn based on the findings are limited to cortisol. In addition, all studies relied on salivary measures and it is therefore unclear whether the conclusions extent to other types of measures (e.g., blood measures).

Second, the review and meta-analysis only included experimental studies conducted with adults in the laboratory, and it is therefore unclear whether the results generalize to everyday life or to children and young adults.

Third, around half of the included studies assessed self-reported emotions. As such, it is unclear whether participants experienced an emotion following emotion induction, whether they were able to regulate this emotion and whether changes in the self-reported experience of emotions was associated with hormonal changes.

Fourth, the review and meta-analysis assessed the effect of emotion regulation *instructions* on hormone levels. It is possible that participants who were instructed to regulate their emotions did not use the instructed emotion regulation strategy or that participants in the control groups regulated their emotions despite not being instructed to do so. Hence, ultimately the results only speak to the effect of receiving an instruction to regulate emotions, not to the effect of actual emotion regulation. With use of manipulation checks to confirm (the absence of) emotion regulation, future studies may tease out the effect of actual emotion regulation on hormone levels.

6. Conclusion

The present review and meta-analysis provide an overview of the existing experimental evidence pertaining to the effect of emotion regulation on cortisol. The results indicate that emotion-induction is associated with an increase in cortisol levels, while emotion regulation does not affect cortisol levels. However, these findings should be viewed as preliminary in light of the small number of studies available. Large gaps in this body of research were identified, suggesting that there is so much more to learn about the role of hormones in emotion regulation.

CRediT authorship contribution statement

Mai B. Mikkelsen: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing - original draft, Writing - review & editing. **Gitte Tramm:** Data curation, Investigation, Writing - review & editing. **Robert Zachariae:** Conceptualization, Formal analysis, Methodology, Writing - review & editing. **Claus H. Gravholt:** Conceptualization, Methodology, Writing - review & editing. **Mia S. O'Toole:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing - review & editing.

Declaration of competing interest

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Supplementary data

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