General Causality Orientations in Self-Determination Theory: Meta-Analysis and Test of a Process Model

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Author Contributions

MSH and KH were responsible for study conceptualization, data collection, and manuscript preparation and editing. MSH was responsible for data preparation and data analysis.

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Abstract

Causality orientations theory, a key sub-theory of self-determination theory, identifies three distinct causality orientations: autonomy, control, and impersonal orientation. The theory proposes generalized effects of the orientations on motivation and behavior. We meta-analyzed studies (*k*=83) testing relations between causality orientations, forms of motivation from self-determination theory, and behavior. Pooled data were used to test a process model in which autonomous and controlled forms of motivation mediated relations between causality orientations and behavior. Results revealed that autonomy and control orientations were positively correlated with autonomous and controlled forms of motivation, respectively. Impersonal orientation was correlated negatively with autonomy orientation and autonomous forms of motivation, and positively with control orientation and controlled forms of motivation. Process model tests revealed total effects of autonomy orientation on behavior, comprising direct and indirect effects through autonomous motivation, and a positive direct effect of control orientation on behavior and a negative indirect effect through controlled motivation, resulting in a zero total effect. Analysis of age, gender, behavior type, study design, and study quality revealed few moderator effects on model relations. Findings support effects of autonomy orientation on motivation and behavior, and the processes involved, and identifies constructs that could be targeted, or circumvented, in behavioral interventions.

Keywords: Causality orientations theory; Autonomy orientation; Control orientation; Impersonal orientation; Autonomous and controlled motivation

Introduction

A multitude of psychological theories has been applied to predict human motivation and behavior (e.g., Bandura, 1977; Conner, 2015; Deci & Ryan, 1985b; Kruglanski et al., 2012; Maslow, 1943; Weiner, 1986). These theories provide valuable knowledge on the motivational determinants of behavior, and the mechanisms involved, and identify potentially modifiable targets for behavior change interventions (Hagger, Cameron, et al., 2020; Hagger, Moyers, et al., 2020). Prominent among these theories is self-determination theory (Deci & Ryan, 1985b; Ryan & Deci, 2017), a needs-based theory that focuses on the *qualities* or content of motivation, rather than quantity, as the key determinant of behavior. Research applying self-determination theory has tended to focus on the forms of motivation individuals experience when acting (e.g., autonomous and controlled), and contingencies in the environment that give rise to those forms of motivation. Less attention, by comparison, has been paid to individual differences in the types of motivation specified in *causality orientations theory*, a sub-theory of self-determination theory (Deci & Ryan, 1985a).

The central premise of causality orientations theory is that individuals differ in the extent to which they interpret the cause of their behavior as either emanating from the self, or emanating from others or external events. Three causality orientation dimensions are proposed (Deci & Ryan, 1985a): *autonomy* orientation, which reflects individuals being oriented toward events in the environment (e.g., optimal challenges, informational feedback) that support their autonomous motivation; *control orientation*, which reflects the tendency to be oriented toward being controlled by external events (e.g., rewards, deadlines, punishments) that undermine autonomous motivation; and *impersonal* orientation, which describes individuals' tendencies to interpret their actions as beyond their intentional control.

According to the theory, individuals endorsing an autonomy orientation tend to interpret their own actions as originating from their self and view situational contingencies on which their behavior depends (e.g., incentives, behavior of leaders) as supportive of their autonomy, while those with

control orientation tend to interpret their actions as emanating from others and view situational contingencies as controlling their behavior. Individuals with an impersonal orientation do not see reasons behind their actions and do not view situational contingencies as supportive of their motivation. Individuals reporting an autonomy orientation are more likely to experience tasks and actions as autonomously motivated and are, therefore, more likely to persist with tasks and adaptive outcomes (e.g., positive affect, well-being). Individuals reporting a control orientation, in contrast, tend to experience tasks as controlled by others or external events (e.g., deadlines), and are more likely to desist on tasks and experience maladaptive outcomes (e.g., negative affect, frustration, ill-being). Impersonal orientation is linked to a lack of motivation, and also to desistence with behaviors (Deci & Ryan, 1985a; Ryan & Deci, 2017).

Although research has generally tended to support these theory-specified patterns of effects of causality orientations on motivational and behavioral outcomes (e.g., Deci & Ryan, 1985a; Duriez, 2011; Jerković et al., 2017; Knee & Zuckerman, 1998), there are also studies that have demonstrated very small or null effects (e.g., Hodgins et al., 1996; Jerković et al., 2017; Zuckerman et al., 1988). Currently, it is unclear whether the observed inconsistencies in the effects of causality orientations could be attributable to genuine variability across studies or is an artifact of sample size, a key source of error in observed effects across studies, or whether there are systematic within-person or external moderators responsible for the variability. However, to date, there has been no synthesis of research on causality orientations and their effects on motivation and behavior. Such a synthesis will make an important contribution to resolving observed inconsistencies by outlining the size and true variability of effects of causality orientations on motivation and behavior across studies after accounting for sampling error. In the event of non-trivial variability remaining in effects across studies, the analysis also enables tests for the effects of salient moderators (e.g., interpersonal conditions, study design, and measurement) on relations between the orientations, motivation, and behavior. The synthesis also affords the opportunity to conduct a large-scale test of the key process by which causality orientations

are proposed to affect behavior; through mediation by motivation types experienced toward the behavior. Estimating the averaged effects and variability of causality orientations, and testing the model and moderators, provides self-determination theorists and those interested in individual differences with cumulative evidence for the relevance of causality orientations in determining motivational styles and promoting behavior. The research has value to those interested in motivating individuals and changing their behavior (e.g., educators, managers, health professionals) by providing information on salient intrapersonal conditions which may enhance or undermine autonomous motivation and behavioral adoption and persistence. These are conditions that could be accounted for, or circumvented, when designing interventions to promote motivation and change behavior, and may signal the kinds of strategies or techniques that might be applied in interventions .

Considering this knowledge gap, the current analysis aimed to synthesize studies examining relations between general causality orientation dimensions, forms of motivation from self-determination theory, and behavior. The research also aimed to use the pooled data across studies to test predictions of a process model linking causality orientations to behavior through the mediation of forms of motivation from self-determination theory. In addition, the study aimed to test whether proposed effects within the process model tested in the pooled data from multiple studies varied according to key moderator variables. Results are expected to provide important data on the size and variability of effects of causality orientations on motivation and behavior across studies, provide insight into the potential mechanisms by which causality orientations relate to behavior, and provide data on the factors that moderate model effects.

Self-Determination Theory

Self-determination theory is a leading theory of motivation which outlines the determinants of human motivation, and the processes by which the determinants lead to motivation and behavior (Deci & Ryan, 1985b, 2000; Ryan & Deci, 2017). Broadly, self-determination theory explains how different forms of motivation experienced by individuals determine the uptake of, and persistence on,

tasks and behaviors, and the effects of the forms of motivation on intrapersonal outcomes related to well-being. The extent to which social-structural factors in the interpersonal environment (e.g., the actions of leaders and significant others) support motivation, and the extent to which individuals' experience that their basic psychological needs are supported and not frustrated, are further theoretical processes that determine motivation, persistence, and adaptive outcomes. The theory is described as a 'meta-theory' comprising six sub-theories, termed 'mini-theories' by Ryan and Deci (2017), each describing specific motivational phenomena. Two sub-theories from self-determination theory are directly germane to the current research: organismic integration theory and causality orientation theory[1]. Next we describe the premises and predictions of these two theories and how they inform our proposed process model which describes the processes by which causality orientations relate to behavior through forms of motivation.

Organismic Integration Theory

Organismic integration theory (Deci et al., 1994; Deci & Ryan, 2000) outlines how the forms of motivation experienced by individuals when performing tasks and actions impact future motivation and behavioral persistence, and how social-structural factors can lead to shifts in the forms of motivation individuals experience when performing tasks and behaviors[2]. A core distinction in the theory is that between *autonomous* and *controlled* forms of motivation. Autonomous forms of motivation reflect performing tasks and behaviors for self-determined or self-endorsed reasons, while controlled forms of motivation reflect performance of tasks and behaviors as other-determined or externally-referenced reasons. The theory predicts that individuals engaging in tasks or behaviors for autonomous reasons are likely to persist with the behavior and experience adaptive outcomes, such as well-being and interest. This is because the individual perceives their actions as consistent with their

[1]A full discussion of the major predictions of the other 'mini-theories' that comprise self-determination theory (cognitive evaluation theory, basic psychological needs theory, goal contents theory, and relationship motivation theory) is beyond the scope of this article. Readers interested in greater detail are directed to Ryan and Deci's (2017) comprehensive and lucid treatment of these sub-theories.

[2]In the context of the current study, *behavior* represents a behavioral response relevant to the context of interest, and is expected to correspond with the behavior referred to in the measures of motivation adopted.

genuine sense of self and is independent of any external controlling contingency. Controlled reasons for engaging in tasks or behaviors may still lead to persistence, but persistence is entirely dependent on the presence of external contingencies and, if absent, likely lead to desistence. Several forms of autonomous and controlled forms of motivation are specified, represented on a continuum of motivation that reflects reasons or perceived causes of action, known as the perceived locus of causality (Ryan & Connell, 1989). The theory proposes that individuals are motivated to take in or *internalize* actions and behaviors that are controlled motivated so that they eventually become part of their repertoire of actions that are experienced as autonomous and need satisfying (Deci et al., 1994; Ryan & Connell, 1989).

Intrinsic motivation is the prototypical form of autonomous motivation, located at one extreme of the continuum. Intrinsic motivation reflects behavioral performance with the absence of external contingencies and for reasons that are fully endorsed by the individual and their genuine sense of self. Adjacent to intrinsic motivation on the continuum lies *identified regulation*, another form of autonomous motivation which reflects engaging in activities to attain self-endorsed outcomes. In contrast, *external regulation* is the prototypical form of controlled motivation, located at the opposite extreme of the continuum. This reflects performance of behaviors that is strictly determined by others (e.g., commands, orders) or controlling events in the environment (e.g., rewards, punishments, deadlines). Adjacent to external regulation on the continuum lies *introjected regulation*, which reflects engaging in activities for externally-referenced reasons, but originating from the self. Studies indicate that autonomous behavioral regulations are consistently related to behavioral persistence and adaptive outcomes (e.g., interest, enjoyment, and well-being) in education (Deci et al., 1991), health (Ng et al., 2012), industry (Gagné & Deci, 2005), social (Knee et al., 2002), and exercise (Hagger & Chatzisarantis, 2007) domains. In contrast, controlled behavioral regulations are related to engagement, but not long term persistence or adaptive outcomes, and are often associated with

maladaptive outcomes (e.g., negative affect and ill-being; Hein et al., 2015; Koestner & Losier, 2002).

The internalization process results in individuals shifting their perceived locus of causality for the behavior from controlled forms of motivation to autonomous forms. This process is adaptive given that autonomous forms of motivation are associated with behavioral persistence, well-being, and functional outcomes. When social agents in the position to influence the motivational environment in which individuals act display autonomy-supportive behaviors (e.g., providing choice, meaningful rationale, and informational feedback, and avoiding controlling language) they promote perceived autonomy support and autonomous motivation among those acting in the environment. Such behaviors have been the focus of behavioral techniques and interventions based on the theory aimed at promoting internalization and autonomous motivation (e.g., Chatzisarantis et al., 2009; Hagger, Hankonen, et al., 2020; Jang et al., 2016; Reeve & Cheon, 2020; Teixeira et al., 2020). In summary, organismic integration theory elucidates two key overarching premises of self-determination theory: (1) autonomous forms of motivation are most likely to lead to effective behavioral engagement and persistence, and optimal functioning and adaptive outcomes, while controlled forms of motivation are only likely to lead to behavioral persistence as long as the controlling contingencies are present, are less likely to lead to optimal functioning, and may be linked to maladaptive outcomes; and (2) the interpersonal environment fostered by social agents can determine the types of motivation experienced for tasks and behaviors in particular contexts, and promote internalization of tasks so that they are experienced as autonomous.

Causality Orientations Theory

Causality orientations theory deals with individual differences in motivation in the context of self-determination theory (Deci & Ryan, 1985a; Ryan & Deci, 2017). According to the theory, individuals differ in the extent to which they interpret their actions as autonomous and originating from the self, or controlled and determined by events perceived as external to the self (Deci & Ryan,

1985a). These *causality orientations* are presented as generalized traits that bias actions and behavior contexts. An *autonomy* causality orientation reflects a tendency for individuals to orient themselves toward environmental events that support autonomous motivation and psychological need satisfaction. Autonomy-oriented individuals are more likely seek to engage in behaviors out of volition, exhibit autonomous forms of motivation, perceive their actions as originating from their genuine sense of self, and interpret external contingencies like rewards as informational and supporting psychological needs (Hagger & Chatzisarantis, 2011; Ryan & Deci, 2017). Such individuals will be more likely to adopt autonomous motivational styles: "…when people are high in autonomy orientation, they tend to use identified and integrated styles of regulation and to have a high level of intrinsic motivation" (Ryan & Deci, 2017, p. 217). In contrast, a *control orientation* reflects a tendency for individuals to be oriented toward external events and contingencies. Controlled oriented individuals tend to experience social contexts in terms of reinforcements such as rewards and punishments with which they must comply or resist, and therefore tend to experience actions as regulated by events that originate outside the self. They are, therefore, more likely to adopt "the external or introjected styles of regulation and to have a low level of intrinsic motivation" (Ryan & Deci, 2017, p. 217). As a consequence, they likely have few opportunities to experience intrinsic motivation and need satisfaction. An *impersonal* causality orientation reflects a generalized tendency to experience behaviors as beyond a personal sphere of personal control; actions tend to be viewed as outside personal volition or intention and are likely accompanied by feelings of incompetence and low mastery. An impersonal orientation, therefore, may lead to avoidance of new or novel actions in fear of being shown to be incompetent.

Causality orientations have typically been measured using the general causality orientations scale (GCOS; Deci & Ryan, 1985a). Research indicates a theoretically-consistent pattern of correlations among GCOS dimensions: small, often negative, correlations between the autonomy and control orientations, positive correlations between the control and impersonal orientations, and

negative correlations between the autonomy and impersonal orientations (Deci & Ryan, 1985a; Vallerand et al., 1987). This correlation pattern suggests that, in keeping with many individual difference and personality constructs, the causality orientation dimensions are not orthogonal. This is consistent with the theoretical premise that individuals' endorse each orientation to some extent and likely exhibit 'profiles' of scores on the three dimensions (Anderson, 1982; Assadi & Hassanein, 2014; Deci & Ryan, 1985a; Tobe et al., 2016). Individuals are, therefore, expected to vary in the level of endorsement of each dimension. Studies have also shown that autonomy causality orientations are associated with indices of adaptive functioning such as autonomous forms of motivation like intrinsic motivation and identified regulation (Ng et al., 2012), perceived autonomy support (Deci & Ryan, 1985a), ego-development (Deci & Ryan, 1985a), non-contingent self-esteem (Deci & Ryan, 1985a), attitude-behavior consistency (Koestner et al., 1992), and relationship-maintaining behaviors (Knee et al., 2002). In contrast, a control causality orientation has been shown to be related to maladaptive traits and outcomes such as Type A personality (Deci & Ryan, 1985a), self-serving attributions (Knee & Zuckerman, 1996), and self-handicapping (Knee & Zuckerman, 1998). The impersonal orientation tends to be positively correlated with control-related constructs like external locus of control, self-related constructs such as public self-consciousness and social anxiety, and emotional outcomes such as depression, and negatively correlated with self-esteem and ego development (Deci & Ryan, 1985a).

Within self-determination theory, causality orientations can be viewed as generalized orientations that have small but wide-ranging effects across contexts and behaviors (Deci & Ryan, 1985a). Like many individual difference constructs, causality orientations are not unequivocally deterministic of behavior. Causality orientations effects on behavior may be mitigated by contextual factors, such as the interpersonal conditions or social environment in which the behavior takes place, which may support or undermine autonomous motivation. For example, causality orientations may interact with situational or contextual factors in determining motivation and behavior (Hagger &

Chatzisarantis, 2011), or may simply have additive effects alongside situational factors (Hagger et al., 2015). Causality orientations are, therefore, expected to act as 'distal' determinants of behavior, and contribute to predicting behavior alongside, or in conjunction with, contextual factors.

A Process Model

Although causality orientations are conceptualized as constructs that have distal effects on behavior, there is relatively little theoretical and empirical work on the processes involved. Based on the basic premises of causality orientations theory, and theory and research on other personality and individual difference constructs that are conceptualized as having distal effects on motivation and behavior (e.g., Bogg, 2008; Conner & Abraham, 2001; Hagger et al., 2006; Phillips et al., 2003; Rhodes & Courneya, 2003), we propose that relations between causality orientations and behavior should be mediated by the forms of motivation from self-determination theory. The forms of motivation are represented by constructs from the perceived locus of causality from organismic integration theory. The model outlines how causality orientations relate to behavior by serving as a distal influence or source of information that contributes to the type of behavioral regulation individuals experience with respect to their behavior (Adams et al., 2017; Neighbors et al., 2004).

Specifically, autonomy causality orientation is predicted to be positively related to autonomous forms of motivation. In contrast, positive relations are hypothesized between control causality orientation and controlled forms of motivation[3]. In addition, impersonal causality orientation is predicted to be negatively related to autonomous forms of motivation, and positively related to controlled forms of motivation. Autonomous forms of motivation are also expected to positively predict behavior, while controlled forms of motivation are expected to negatively predict behavior (Chatzisarantis et al., 2003; Ng et al., 2012). Autonomy causality orientation is expected to positively predict behavior mediated by autonomous forms of motivation, while control orientation is expected

[3]We refer to the motivational mediators in their aggregated autonomous and controlled forms when specifying predictions of the process model. However, these aggregate forms could be substituted for their more specific autonomous (autonomous motivation, identified regulation) and controlled (introjected regulation, external regulation) forms.

to negatively predict behavior mediated by controlled forms of motivation. Finally, impersonal causality orientation is expected to negatively predict behavior through autonomous forms of motivation, and positively predict behavior through controlled forms of motivation.

The value of this model is that it provides a mechanistic description of how generalized causality orientations relate to particular behaviors by determining the form of motivation individuals adopt with respect the behavior. As with many theories of personality and individual difference, contextual factors will also be highly salient influences on motivation in specific contexts and for specific behaviors. From the perspective of self-determination theory, such influences are likely to be the social-structural factors that likely affect the type of motivation adopted, such as autonomy supportive or controlling behaviors displayed by leaders or significant others in the interpersonal environment. Such influences are expected to act in parallel with the causality orientation in determining behavior, the valence of which may depend on the relative strength or salience of the social-structural factors. Nevertheless, the model provides a basic understanding of the processes by which causality orientations relate to behavior, and may provide theorists and researchers with a framework for understanding the relevance of causality orientations to determining individuals' motivation and behavior.

The Value of a Meta-Analysis of Causality Orientations Theory and the Process Model

Although the research literature has generally provided support for the theoretically predicted patterns of relations among the causality orientations dimensions, and their effects on forms of motivation from self-determination theory and behavior, the research does not provide unequivocal support. Some studies have identified very small or null effects of these constructs on behavior (e.g., Hodgins et al., 1996; Jerković et al., 2017; Patterson, 2017; Zuckerman et al., 1988). For example, Jerković and colleagues examined relations between autonomy and control orientation and cannabis consumption. Results indicated that correlations were in the predicted direction, but were small and not statistically significant. The observed variability in the effects of causality orientation on

motivation and behavior may be entirely, or in part, attributable to sampling error, a major source of error in observed effects across studies. However, it is also possible that the variation in the effects may be attributable to key moderator variables. Resolution may lie in a meta-analytic synthesis of the extant research on causality orientation effects, which would enable an evaluation of the extent to which the variability in the effects is due to sampling error, and provide true variability estimates for the effects across studies. Furthermore, assuming substantive variability remains, the analysis would permit tests of the effects of key moderator variables on effects in groups of studies characterized by the levels of the moderators pending sufficient data.

To date, there has been no attempt to synthesize effects of causality orientations on motivational and behavioral outcomes across the extent literature. Such an endeavor will have value to theorists, researchers, and practitioners alike. For theorists and researchers, a synthesis will provide an apt test of a key premise of causality orientations theory, which states that the orientation dimensions have small but broad effects on motivation and behavior. It will also afford resolution of the potential conditions that may exacerbate or attenuate effects of causality orientations on motivation and behavior through analysis of candidate moderators. This will provide critical information for theorists interested in accounting for the possible conditions that may assist in providing better prediction of behavior. It may also indicate whether researchers designing experiments and interventions to change motivation and behavior based on self-determination theory should consider controlling for the unique effects of causality orientations. For practitioners, the analysis may assist in identifying the strategies or techniques that could be adopted in interventions aimed at promoting motivation and behavior. For example, the analysis may highlight whether there is value in targeting change in causality orientations in interventions aimed at changing motivation and behavior, consistent with research suggesting that even relatively stable traits, such as personality dimensions, are changeable through intervention (Roberts et al., 2017).

In addition, there is very little research examining the role that forms of motivation from self-determination theory play in explaining links between causality orientations and behavior. Our proposed process model outlines a potential mechanism predicting that forms of motivation as set out in organismic integration theory serve to mediate relations between causality orientations and behavior, with specific, characteristic patterns of effect. A meta-analytic test of research on relations between causality orientations, motivation, and behavior affords an opportunity to provide a test of this unique model using pooled data on these relations across multiple studies. Specifically, we propose to use synthesized data from existing studies to test process model predictions using meta-analytic structural equation modeling. Testing model predictions will provide formative evidence of a potential mechanism by which causality orientations relate to behavior, and may serve as a basis for future research on how causality orientations relate to behavior. It may also inform development of interventions focusing on changing behavior based on causality orientations theory. For example, the research could potentially provide information on the value of targeting causality orientation dimensions, and whether such change could be transmitted to motivation and behavior.

The Present Study

In the present pre-registered study (https://osf.io/7nz6d), we aimed to conduct a meta-analytic synthesis of studies on general causality orientations from self-determination theory, and relations between causality orientations and forms of motivation from self-determination theory. Specifically, we aimed to estimate the size and variability of (1) intercorrelations among the general causality orientations dimensions; (2) relations between the general causality orientations dimensions and autonomous and controlled forms of motivation from self-determination theory; (3) relations between general causality orientations and behavior; (4) effects of causality orientations on behavior mediated by autonomous and controlled forms of motivation based on our proposed process model (Figure 1); and (5) effects of key moderators on relations between causality orientations and forms of motivation from self-determination theory and behavior.

Our approach involved identification of all studies reporting relations between measures of general causality orientation dimensions, forms of motivation from self-determination theory including aggregate measures of autonomous or controlled motivation and individual behavioral regulations from the perceived locus of causality (intrinsic motivation, identified regulation, introjected regulation, and external regulation), and measures of behavior. To achieve our first two aims, we extracted relevant effect size data from included studies and subjected them to meta-analysis to estimate the size and variability of relations among the causality orientation dimensions and motivational and behavioral outcomes. To achieve our third aim, we computed a pooled correlation matrix and associated matrix of variances/covariances using random effects meta-analysis and tested our proposed process model by fitting a structural equation model specifying model effects to the matrix using meta-analytic structural equation modeling.

We addressed our final aim by estimating effects in our proposed model in groups of studies determined by levels of salient moderator variables: gender distribution, behavior type, sample type, study design, and study quality. In terms of predictions for the moderator variables, we expected no differences in relations between causality orientation dimensions, forms of motivation from self-determination theory, and behavior by sample and behavior type as causality orientations theory is proposed to map motivational processes that are universal across populations and behaviors. Given limited research suggesting that females tend to endorse autonomy orientation and autonomous motivation more than males (Stevens et al., 2015), we expected studies on samples with a high proportion of females to report larger effects of autonomy orientation on motivation and behavior. In addition, the preponderance of research in psychology, including self-determination theory, conducted on student samples that tend to be homogenous, educated, and affluent, may lead to biases compared to studies on non-student samples (Henrich et al., 2010), so our sample type moderator analysis aimed to explore this question. Furthermore, we tested whether study design and quality moderated relations among causality orientation dimensions, forms of motivation, and behavior

across studies. Cross-sectional designs may inflate relations between constructs due to artifacts such as common-method variance and proximity in measurement relative to designs including a time lag between measures or experimental designs, and studies with lower study quality may exhibit higher error variance in effect size tests, which can both attenuate and inflate effect sizes (Podsakoff et al., 2003).

Our specific pre-registered hypotheses for relations among causality orientations, motivation, and behavior in the current meta-analysis follow. Specifically, we predicted:

H1: A positive non-zero effect of autonomy causality orientation on autonomous motivation;

H2: A negative non-zero effect of autonomy causality orientation on controlled motivation;

H3: A positive non-zero effect of control causality orientation on controlled motivation;

H4: A negative non-zero effect of control causality orientation on autonomous motivation;

H5: A positive non-zero effect of impersonal causality orientation on controlled motivation;

H6: A negative non-zero effect of impersonal causality orientation on autonomous motivation;

H7: A positive non-zero effect of autonomous motivation on behavior.

H8: A negative non-zero effect of controlled motivation on behavior.

H9: A positive non-zero indirect effect of autonomy causality orientation on behavior mediated by autonomous motivation.

H10: A negative non-zero indirect effect of control causality orientation on behavior mediated by controlled motivation.

H11: A negative non-zero indirect effect of impersonal causality orientations on behavior mediated by autonomous and controlled motivation.

Hypotheses relating to intercorrelations among the causality orientation dimensions and effects of moderators on relations among the proposed process model were not pre-registered.

Method

Search Strategy

The meta-analysis was pre-registered on the Open Science Framework: https://osf.io/7nz6d. A systematic keyword search was used to search the following electronic bibliographic databases: PubMed, PsychINFO, Web of Science, and Scopus. Databases were searched up to and including May 4, 2018 with no lower limit. Additional studies were located from a manual search of the reference lists of published research on self-determination theory. Unpublished data sets were located by emailing key authors in the field identified from the studies in the literature search.

Characteristics of Included Studies

Studies were included if they reported at least one correlation between two of the general causality orientation dimensions, or between at least one causality orientation dimension and a measure of forms of motivation from self-determination theory (e.g., autonomous motivation, intrinsic motivation, identified regulation, introjected regulation, external regulation, controlled motivation), or a measure of behavior. Most studies were expected to be correlational and cross-sectional or prospective in design. Experimental and intervention studies were also included, but data were only included where they were not affected by an experimental manipulation, prime, or intervention (e.g., an autonomy supportive intervention) aimed at changing the variables of interest. To ensure this was the case, data for relations between causality orientation, motivation, or behavior were taken from baseline measures or from the control group in experimental or intervention studies. For example, we used baseline data from the GCOS administered to participants before the introduction of the experimental manipulation (an ego-involved/non-ego involved manipulation) from Bober and Grolnick's (1995) experimental study. In another example, we used data from participants assigned to the control group who did not receive the experimental manipulation (introduction of a reward for performing a target task) in Hagger and Chatzisarantis' (2011) experimental study. None of data from the included studies, therefore, were affected by a manipulation or intervention aimed at changing the causality orientations dimensions or a self-determination theory construct. Given that

most data were expected to be correlational, the zero-order correlation coefficient was selected as the effect size metric.

Articles identified in the initial search after removal of duplicates (k = 1,033) were subjected to a title, keyword, and abstract screen for eligibility by two members of the research team. The resulting list of eligible studies was then subjected to full-text review against inclusion criteria to produce a final set of included studies (k = 69). A flow diagram of the study search, screening, and selection process is presented in Appendix A (supplemental materials). In addition, nine studies included multiple samples, so each was treated as a separate study in the analysis resulting in a final sample of 83 studies (see Appendix B, supplemental materials). A full list of included studies is available in Appendix C (supplemental materials).

Classification of Measures

Data for relations between causality orientation dimensions, forms of motivation, and behavior were extracted from studies meeting inclusion criteria. Studies used a relatively narrow range of measures of the causality orientation dimensions and forms of motivation. With respect to causality orientations, the majority of studies used the General Causality Orientations Scale (Deci & Ryan, 1985a). A few studies used context-specific versions of the scale with identical item content to those in the general version with the exception of a direct reference to a target behavior (e.g., the Exercise Causality Orientations Scale; Rose et al., 2001). These scales were treated as equivalent. Measures of behavioral regulations from organismic integration theory were derived from standardized questionnaires based on Ryan and Connell's (1989) perceived locus of causality measure, or derivative versions (e.g., Levesque et al., 2007; Mullan et al., 1997). Studies usually reported at least one form of motivation from the perceived locus of causality: *intrinsic motivation*, *identified regulation*, *introjected regulation*, or *external regulation*. In some cases, the autonomous (intrinsic motivation and identified regulation) and controlled (introjected and external regulation) forms of motivation were aggregated to form global measures of autonomous or controlled motivation. Three

studies reported measures of *amotivation,* a form of motivation separate from the perceived locus of causality reflecting a lack of intentionality or motivation toward tasks. As very few effect sizes between amotivation and causality orientation dimensions were available, this construct was excluded from the analysis. Behavioral engagement was usually measured as frequency of participation in the target behavior referred to in the motivational measures adopted in the study, usually by self-report. There was considerable diversity in the behaviors adopted including health (e.g., physical activity, meditation, sport injury rehabilitation, smoking cannabis, type 'A' behavior, self-care behaviors), educational (e.g., self-regulation of learning, teaching behaviors), occupational (e.g., career search, creative performance), and social (e.g., aggression, self-presentation, social interaction) behaviors. Some studies used non-self-report measures from which behavior was inferred, such as experimental measures of behavioral persistence (e.g., Hagger & Chatzisarantis, 2011; Øverup et al., 2017, Study 3).

Effect Size Data Extraction

Relevant effect size data for relations among measures of causality orientation dimensions, forms of motivation or behavioral regulation, and behavioral measures were extracted. The majority of studies were correlational in design with a few intervention or experimental studies. None of the latter studies reported manipulations of causality orientations and examined their effects on motivation or behavior, so data for baseline measures or the control group were extracted for these studies. In addition to effect size data, sample characteristics (mean sample age, standard deviation, and range; gender distribution), target behavior definition and operationalization, study design, measures used to tap causality orientations, forms of motivational or behavioral regulations, and type of behavioral measure were also extracted. These data were summarized in Appendix D (supplemental materials). Full characteristics of studies and data extracted are available in a spreadsheet available online: https://osf.io/gjs5v/.

Moderator Coding

We aimed to estimate our proposed process model in groups of studies defined by levels of five moderator variables: sample age, sample gender distribution, sample type (student vs. non-student), study design, and study quality. Moderator coding is summarized in the study characteristics table in Appendix D (supplemental materials). With respect to the age moderator, we aimed to distinguish between studies on younger and older samples, based on sample average age and distribution. Many studies were on younger samples, defined as having a mean age of 40 or younger with low variability (SD ≤ 15). However, defining an older samples category presented difficulties given the high variability and range in samples with older average age. We therefore compared model effects in sets of studies on younger samples and samples comprising older samples and samples of 'mixed' age with high variability. Similarly, a substantive number of studies were on female only or predominantly female (≥75% female) samples, but studies on male only or predominantly male samples numbered very few. We therefore compared model effects in sets of studies on predominantly female samples, and studies on mixed gender samples (between 25% and 74% female). Given the preponderance of student samples in psychological research (Henrich et al., 2010), we also compared model effects in groups of studies on student and non-student samples. We thought it might be useful to examine effects on studies using cross-sectional designs, that is, designs where all constructs were measured simultaneously, and studies where measures were separated by a time lag or used an experimental or intervention design, even though data extracted in the studies did not represent effects of experimental or intervention manipulations. We therefore compared model effects on groups of studies using cross-sectional and non-cross-sectional (experimental, intervention, and longitudinal designs). We had intended to conduct moderator analyses by behavior type on the basis that the strength of effects of orientation and motivational constructs may vary due to the type of behavior targeted in the analysis. However, the highly disparate types of behaviors adopted precluded the formation of meaningful groups of studies based on behavior type.

Study quality was assessed using the 20-item quality of survey studies in psychology (Q-SSP) checklist (Protogerou & Hagger, 2020). Studies meeting stipulated quality standards on each Q-SSP item were assigned a score of 1 and those not meeting standards, or provided insufficient information for evaluation, were assigned a score of 0. Two raters with previous experience in assessing study quality analysis scored the studies. Inter-rater reliability was tested on a set of double-coded studies (k = 10). Level of agreement on study scores across raters was evaluated using Gwet's (2008) AC1/AC2 coefficient, which is an agreement statistic similar to Cohen's Kappa statistic, but adjusts for noted concerns such as low observed Kappa values when agreement is high due to imbalances in the marginal totals of agreement tables (Cicchetti & Feinstein, 1990; Di Eugenio & Glass, 2004). Results revealed good agreement (median agreement = 90%, range = 70% to 100%) between raters and good agreement statistics (median AC1/AC2 coefficient = .842, range = .406 to 1.000, median p = .001, range .000 to .196). Studies attaining 1 score for 75% or more on the Q-SSP items were classified as 'acceptable' in quality, while studies attaining scores for fewer than 75% of the items were classified as 'questionable' in quality. This dichotomous study quality variable was used in the moderator analyses. The checklist criteria and item descriptions are presented in Appendix E (supplemental materials). Quality scores for each study and inter-rater reliability analyses are presented in the data spreadsheet available online: https://osf.io/gjs5v/.

Data Dependency

Some studies provided multiple measures of behavior or causality orientations and, therefore, multiple effect sizes. However, inclusion of multiple effect sizes from the same study as separate effects in a meta-analysis violates the assumption of independence. As a consequence, we aggregated these effect sizes using Hunter and Schmidt's (2004) formula to deal with within-study dependency. The imputed correlation between the within-study effect sizes was set at r = .50 as recommended by Wampold et al. (1997). Details of aggregated studies and the behavioral dependent variables are

provided in Table B1 (Appendix B, supplemental materials). Data on the aggregation analysis are provided in the data spreadsheet available online: https://osf.io/gjs5v/.

Data Analysis

Conventional meta-analysis. Averaged correlations corrected for sampling error among causality orientation dimensions, motivational styles from the perceived locus of causality, and behavioral measures were estimated using conventional meta-analyses adopting a random effects model with a maximum likelihood estimator using the metafor (Viechtbauer, 2010) function in R. Fixed effects estimates are also provided for comparison. Variability and heterogeneity estimates were also computed, including Cochran's (1952) *Q* statistic, the τ^2 statistic, and the I^2 statistic and its 95% confidence interval. The *Q* statistic assesses whether the observed variance in the effect size of interest is due to true variation across studies rather than variation within each study (i.e., due to sampling error), the τ^2 statistic represents the true variability in the effect size across studies after accounting for sampling error, and the I^2 statistic represents the percentage of variance in the effect size that is due to true heterogeneity rather than variability due to chance alone. Statistically significant *Q* and τ^2 values with I^2 values exceeding 25% with wide confidence intervals are considered indicative of substantive heterogeneity in the effect size estimate after correcting for sampling error, and suggest that other variables (moderators) may account for the observed variance across studies. Forest plots were also produced to provide visual comparisons of the correlations in each study and the averaged correlations across studies for each effect. We also tested the discriminant validity of the causality orientation dimensions. Discriminant validity was considered supported if the confidence intervals about the averaged correlation coefficient between two constructs did not include the value of one (1.00) (Bagozzi & Kimmel, 1995). We used Cohen's (1988) conventions for evaluating small (.10), medium (.30), and large (.50) effect sizes for the averaged correlations.

Assessment of bias. The potential effect of selective reporting bias on each correlation from the conventional meta-analysis was evaluated using regression analyses based on 'funnel' plots. In the analysis, effect sizes from each study are regressed on its precision estimate based on the standard error (Egger et al., 1997). The analysis yields an ostensibly unbiased estimate of the effect size by effectively accounting for dependency for the effect size on study precision (standard error estimate). Two methods are used: the precision effect test (PET) and the precision effect estimate with standard error (PEESE). The PET regresses study effect size on the inverse of its variance estimate with the intercept serving as an unbiased estimate of the true mean effect size. However, the PET may underestimate the true mean effect size when there is evidence of a non-zero effect (Stanley & Doucouliagos, 2014). The intercept derived from regressing study effect size on the variance estimate, the PEESE, has been shown to provide a more precise estimate of the true mean effect in cases where there is evidence of a non-zero effect. Stanley and Doucouliagos, therefore, propose the PET-PEESE approach with decision rules based on the statistical significance of the PET bias-corrected estimate. In cases where the PET estimate is statistically significant, implying a non-zero effect, the PEESE estimate is taken, while in the absence of a statistically significant PET estimate, the PET estimate is used. We computed PET and PEESE estimates, with *t*-test for bias, and statistical significance of the corrected effect from zero to provide an indication of selective bias in each estimate using the PETPEESE function in R (Carter et al., 2019).

Meta-analytic structural equation modeling. Few studies included effect size estimates for relations between causality orientation dimensions and separate perceived locus of causality constructs: intrinsic motivation, identified regulation, introjected regulation, and external regulation. This precluded estimation of a full model that included all behavioral regulations as mediators of the effects of causality orientations on behavior. We therefore collapsed effect sizes that comprised autonomous (intrinsic motivation, identified regulation and, where measured, integrated regulation) and controlled (introjected regulation, external regulation) behavioral regulations into effect sizes

representing aggregated autonomous and controlled motivation constructs. These constructs were used in subsequent model tests of proposed models.

Relations among constructs in proposed models were estimated using meta-analytic structural equation modeling using the MetaSEM package (Cheung, 2015; Cheung & Hong, 2017) in R. Multiple relations among sets of constructs from social cognitive models are typically tested using a univariate approach, which involves initial correction of correlations among variables in the model for bias across studies using conventional meta-analytic techniques. The resulting matrix of bias-corrected correlations is then used as input for a multiple regression analysis or path analysis to test model predictions. Although this method has been used in many previous studies (e.g., Hagger et al., 2016; Ng et al., 2012), it has been subject to criticism because it requires the use of a common sample size to estimate standard errors of model parameters, such as the harmonic mean of the sample size across studies, and assumes that the correlation matrix is a covariance matrix, which likely leads to bias in the standard errors, confidence intervals, and chi-square values of the model (Cheung, 2015; Cheung & Hong, 2017).

Meta-analytic structural equation modeling offers a two-stage alternative method that addresses the problems inherent in the univariate approach. In the first stage, correlation matrices among constructs of the proposed model from each study included in the analysis are transformed to account for study-specific random effects using meta-analysis, enabling them to be analyzed as covariance matrices, the typical ‘input’ for a regular structural equation model. Parameter estimates (intercepts) produced in the first stage represent the zero-order correlations among constructs corrected for sampling error in the meta-analysis with 95% confidence intervals. As with conventional meta-analysis, the *Q* statistic provides an overall test of the homogeneity of model estimates, with a statistically significant value indicative of substantive heterogeneity. Statistics to evaluate homogeneity in each of the model parameters are also provided: the τ^2 statistic and the I^2 statistic and its 95% confidence interval.

In the second stage of the analysis, a model representing predicted relations among study variables is fitted to the covariance matrix from the first stage. Missing data are imputed using the full information maximum likelihood method. We tested our pre-registered hypotheses in a mediational model (Model 1) in which general causality orientations predicted behavior mediated by autonomous and controlled motivational styles from self-determination theory (Figure 1). Estimating this model in the full sample was eminently feasible given the available data and provided the opportunity to estimate indirect effects. Fit of the proposed models with the data from the first stage meta-analysis was evaluated using multiple criteria for goodness-of-fit: the model goodness-of-fit chi-square and associated significance test, the comparative fit index (CFI), the Tucker-Lewis index (TLI), the standardized root mean square of the residuals, and the root mean error of approximation (RMSEA). A non-significant chi-square value, CFI and TLI values that approach or exceed .95, a SRMSR value of less than .008, and a RMSEA value of .005 or less indicate good fit of the model with the data (Hu & Bentler, 1999). The analysis yields standardized beta coefficients (β) and Wald confidence intervals for direct effects, making evaluation of effect sizes relatively straightforward. Evaluating effect sizes for indirect effects is more of a challenge because the coefficients for indirect effects are products of multiple standardized coefficients, so will likely be much smaller than those for direct effects. As a consequence, coefficients of .075 or larger were considered medium-to-large in size while coefficients below this value were regarded small by comparison (Seaton et al., 2010).

The paucity of available data for some cells in the pooled correlation matrices across included studies for most moderator groups meant that we could not estimate the full mediation model in the moderator groups. As a consequence, we estimated two further models (Figure 2): A model representing the effects of the causality orientations on motivational styles from self-determination theory (Model 2) and a model representing effects of causality orientations on behavior (Model 3). Models 2 and 3 were estimated for the full sample, as well as separately in groups of studies defined

by levels of the coded moderator variables: sample age, sample gender distribution, sample type, study design, and study quality.

Model goodness-of-fit statistics were not computed as all proposed models were fully saturated. Effects among model constructs were estimated along with Wald confidence intervals based on the standard errors. Differences in parameter estimates of proposed effects in Models 2 and 3 estimated in moderator groups was evaluated using 95% confidence intervals of the difference in the parameter estimates across the models (Schenker & Gentleman, 2001). To the extent that the interval does not include zero, a statistically significant difference in the parameter estimates across models is confirmed. A formal test of difference is also provided using Welch's *t*-test.

Results

Conventional Meta-Analysis

Zero-order correlations. Results of the conventional random-effects meta-analysis of zero-order correlations among the causality orientation dimensions, motivational constructs from the perceived locus of causality, and behavior are presented in Table 1 with variability and heterogeneity estimates. Forest plots for the correlations among the autonomy, control, and impersonal causality orientation dimensions in each study, with the meta-analytic summary effect, are presented in Figures 3, 4, and 5. Forest plots for the remaining correlations are presented in Appendix F (supplemental materials). Correlations among the three causality orientation dimensions revealed a similar pattern of relations to those found in primary research (e.g., Deci & Ryan, 1985a; Ng et al., 2012). A small positive non-zero correlation was found between autonomy and control orientations (Figure 3), a small negative non-zero correlation was found between autonomy and impersonal orientations (Figure 4), and a larger positive non-zero correlation was found between control and impersonal orientations (Figure 5). Application of Bagozzi and Kimmel's (1995) criteria indicated support for the discriminant validity of the causality orientation dimensions.

Correlations among the causality orientation dimensions and constructs from the perceived locus of causality continuum also revealed a predictable pattern of effects, consistent with those reported in previous primary studies (Deci & Ryan, 1985a; Vallerand et al., 1987). Specifically, we found positive non-zero small-to-medium sized correlations between autonomy causality orientation and autonomous motivation, identified regulation, and intrinsic motivation. The correlation of autonomy orientation with introjected regulation, a more controlling form of motivation, was small in size. Correlations of autonomy orientation with controlled motivation and external regulation were small in size no different from zero. In addition, the analysis revealed positive non-zero small-to-medium sized correlations between control orientation and controlled motivation and external regulation, and positive, small-sized correlations between control orientation and identified and introjected regulation. Correlations between control orientation and autonomous and intrinsic motivation were small in size and no different from zero. We found a non-zero small-to-medium sized negative correlation between impersonal causality orientation and autonomous motivation, and non-zero small-to-medium sized positive correlation between impersonal orientation and external regulation. Correlations between impersonal orientation and the other behavioral regulations were no different from zero. Finally, we found a non-zero small-sized positive correlation between autonomy orientation and behavior. Correlations of control and impersonal orientations and behavior were small in size and no different from zero.

Bias estimates. Results from the PET-PEESE analyses revealed substantive non-zero bias in a few of the correlations (see Table 1). However, the corrected estimates for the correlations from the analysis were not appreciably different from the fixed effect estimates on which they were based. Our bias analyses did not lead us to substantially alter our conclusions with respect to the size of the correlations in the analysis and whether or not they differed from zero[4]. These findings suggest that

[4]In some cases, correlations were computed when there were fewer than five effect sizes available. Such estimates should be interpreted with caution given the small sample size. Similarly, where there were fewer than five studies bias statistics were not computed as they are likely to be highly imprecise.

the majority of the correlations among causality orientations, forms of motivation from self-determination theory, and behavior were not adversely affected by small-study bias, that is, a tendency for larger correlations to be observed in smaller studies. The latter is said to be an indicator of 'publication bias' in which publication outlets tend to favor smaller studies reporting larger, statistically significant effects. However, findings should be interpreted with the caveat that substantive heterogeneity in effect sizes may lead to imprecision in PET-PEESE results.

Meta-Analytic Structural Equation Modeling

Stage 1: Correlations. Zero-order averaged correlations corrected for sampling error from the first stage of the MASEM analysis for each of the three models estimated are presented in Table G1 (Appendix G, supplemental materials). Correlations followed an identical pattern to those found in the conventional meta-analysis. Heterogeneity statistics revealed moderate-to-high heterogeneity in each correlation according to the I^2 statistic, and values for the *Q*-statistic across studies also indicated substantial heterogeneity in the models.

Stage 2: Structural equation model. Standardized parameter estimates with variability statistics, confidence intervals, and *z*-tests for difference from zero for all three models are presented in Table 2. Focusing on Model 1, consistent with our pre-registered hypotheses, we found non-zero direct medium-sized positive effects of autonomy causality orientation on autonomous motivation (H1), and control causality orientation on controlled motivation (H3). We also found a non-zero direct small-to-medium-sized positive effect of control causality orientation on behavior, which was not consistent with our hypotheses. We also found non-zero direct small-sized negative effects of impersonal causality orientation on autonomous motivation (H6), and controlled motivation on behavior (H8). However, effects of autonomy orientation on controlled motivation (H2), control orientation on autonomous motivation (H4), impersonal orientation on controlled motivation (H5), and autonomous motivation on behavior (H7) were small and no different from zero, so current data did not support these pre-registered hypotheses.

Focusing on indirect effects, we found a non-zero indirect small-sized negative effect of control orientation on behavior mediated by controlled motivation, consistent with our pre-registered hypothesis (H10). However, indirect effects of autonomy orientation on behavior mediated by autonomous motivation (H9) and impersonal orientation on behavior mediated by autonomous and controlled motivation (H11) were no different from zero. Two effects of note emerged from the analysis of model total effects. First, we found a non-zero large-sized positive total effect of autonomy orientation on behavior. This effect is determined by the sum of the indirect effect of autonomy orientation on behavior mediated by autonomous motivation, and the direct effect of autonomy orientation on behavior. Although the individual direct and indirect effects of autonomy orientation on behavior were small and no different from zero, these effects combined produce the non-zero total effect. Second, the total effect of control orientation on behavior was no different from zero. Examination of the constituent effects of the total effect revealed an indirect effect of the control orientation on behavior mediated by controlled motivation that was negative in sign, and a direct effect that was positive in sign, both of which were non-zero. However, when combined, these two effects resulted in a small net total effect that was no different from zero. The analysis of direct and indirect effects, therefore, provides insight into the processes by which causality orientations relate to behavior, which would not otherwise be identified in models that did not include motivational styles as mediators of effects of causality orientations on behavior. Examination of effects from Models 2 and 3 corroborated the latter conclusion. Results of Model 2 identified a non-zero small-sized positive effect of autonomy orientation on behavior, but effects of control and impersonal orientations were small and no different from zero. Model 3 corroborated the pattern of non-zero direct medium-sized positive effects of autonomy causality orientation on autonomous motivation and control causality orientation on controlled motivation. It also confirmed the non-zero small-sized negative effect of impersonal orientation on autonomous motivation.

Moderator analyses. Small numbers of studies reporting relations among the causality orientations and motivational orientation and behavior resulted in empty cells in the input correlation matrices for the analyses of many of the moderator groups for the mediation model (Model 1). As a consequence, we tested moderator effects in models specifying effects of causality orientations on behavior (Model 2) and causality orientations on motivational styles (Model 3). Effects of moderators were tested by estimating each model in groups of studies defined by levels of the sample age, sample gender distribution, sample type, study design, and study quality moderator variables. The only exception were the analyses in the age and gender moderators for Model 3. Small numbers of studies in the predominately female samples, and older and mixed age samples, moderator groups resulted in empty cells for the input correlation matrices for these groups, precluding model estimation. We therefore conducted moderator analyses comparing estimates in the full sample with model estimates in the balanced gender samples and younger samples moderator groups. These analyses amounted to sensitivity analyses examining whether model effects differed as a result of omitting studies with predominately female samples and with older and mixed age samples. Standardized parameter estimates and comparisons across moderator groups are presented in Tables H1 and H2, (Appendix H, supplemental materials).

For Model 2, observed effects of autonomy orientation on behavior were larger in studies with a balanced gender profile, studies adopting non-cross-sectional designs, studies on non-student samples, and studies of questionable quality compared to studies on predominantly female samples, studies using cross-sectional designs, students on student samples, and studies of acceptable quality, respectively. However, high variability in effect sizes within moderator groups meant that the only moderator effect that was different from zero was that for study design. In addition, the negative effect of impersonal causality orientation on behavior was larger (more negative) in studies on older and mixed age samples, studies adopting non-cross-sectional designs, and studies on non-student samples compared to studies on younger samples, studies using cross-sectional designs, and studies

on student samples, respectively. However, none of these differences was different from zero, due to the high variability in the effects across moderator groups. For Model 3, observed effect sizes of causality orientations on motivational styles were not appreciably different across moderator groups and patterns of effects were unchanged. Only two effects were close to exhibiting a non-zero difference; the effect of autonomy orientation on autonomous motivation was larger for studies with predominantly female participants and studies using non-cross-sectional designs compared to the overall sample and studies adopting cross-sectional designs. However, effect sizes in both cases were still small-to-medium and the mean differences fell short of conventional levels for statistical significance by a trivial margin.

Discussion

The aim of the present study was to estimate relations among general causality orientations, forms of motivation from self-determination theory, and behavior across multiple studies using meta-analysis. Meta-analyses of correlations revealed theoretically-consistent pattern of intercorrelations among the autonomy, control, and impersonal causality orientation dimensions, and with forms of motivation from self-determination theory. Tests of the proposed process model using aggregated data from the meta-analysis supported some, but not all, of our pre-registered hypotheses. Autonomy orientation predicted autonomous motivation consistent with predictions. However, the direct effect of autonomy orientation on behavior, and the indirect effect of autonomy orientation on behavior mediated by autonomous motivation, were small and no different from zero. Together these small effects contributed to a non-zero total effect of autonomous motivation on behavior. Controlled motivation predicted behavior, but the effect was positive, contrary to predictions. We also observed a non-zero negative indirect effect of control orientation on behavior mediated by controlled motivation, consistent with predictions. These effects resulted in a net zero total effect of control orientation on behavior, which, while not consistent with our predictions, coincides with research demonstrating small positive or null effects of control orientation on behavior (e.g., Øverup et al.,

2017; Sadabadi et al., 2011; Van den Berghe et al., 2013). Moderator analyses identified few effects for the selected moderators on model effects.

Correlations

Intercorrelations among the causality orientation dimensions from the current meta-analysis revealed a pattern congruent with those observed in previous research (Deci & Ryan, 1985a; Olesen, 2011; Ryan & Deci, 2017; Vallerand et al., 1987), and also supported discriminant validity of the dimensions. Correlations of the autonomy orientation dimension with the control and impersonal dimensions indicated that these dimensions are not orthogonal, consistent with the theoretical premise that they should not be considered polar opposites but rather individual differences that relate to motivation and behavior independently. The larger positive correlation between impersonal and control orientation dimensions suggest a higher level of commonality of these dimensions. The association likely represents a key shared aspect of these orientations: both represent a generalized perspective on activities as lacking in personal endorsement and support for autonomous needs.

Current findings also indicated that the causality orientation dimensions had a theoretically-consistent pattern of correlations with motivational and behavioral outcomes (Deci & Ryan, 1985a; Ryan & Deci, 2017). Autonomy orientation was correlated with autonomous motivation and separate autonomous forms of motivation from the perceived locus of causality, while control orientation was correlated with controlled motivation and controlled forms of motivation. In fact, the pattern of relations indicated larger correlations of autonomy orientation with more autonomous forms of motivation, intrinsic, and identified regulations, and weaker associations with more controlled forms of motivation, introjected, and external regulations. A similar graduated pattern was observed for control orientation, which exhibited larger correlations with the external and introjected regulations and smaller associations with identified regulation and intrinsic motivation. Interestingly, the impersonal orientation was correlated only with external regulation and negatively related to autonomous motivation, consistent with the interpretation that this orientation reflects the generalized

perceptions of a lack of personal endorsement of actions. These patterns of effects were also replicated in the structural equation models in which orientations were set as simultaneous predictors of autonomous and controlled motivation. These effects reflect the perspective from general causality orientations theory that the orientations reflect a source of information on which individuals base the types of regulation that determine their subsequent behavior, as well as the level of internalization individuals experience toward those behaviors, consistent with organismic integration theory (Deci & Ryan, 1985a; Ryan & Deci, 2017). It is also important to note that effect sizes for the correlations between causality orientations and forms of motivation were small-to-medium in size, this leaves a substantive proportion of the variance in motivation unexplained. To speculate, this unexplained variance may be attributed to situational influences on motivation such as contingencies in the environment that support, thwart, or frustrate motivation, such as social agents' display of autonomy supportive behaviors or use of rewards or punishments (Deci & Ryan, 1985b, 2000).

With respect to behavior, autonomy orientation alone exhibited a unique non-zero correlation with behavior with a small effect size while correlations of control and impersonal orientations were no different from zero. These findings highlight the relevance of autonomy orientation to the prediction of behavior, and suggest that while the other orientations may have motivational relevance, their effects are not translated to behavior. These findings also align with the theoretical perspective that causality orientations have modest effects on behavior, but their effects are likely pervasive across multiple behaviors, contexts, and populations (Adams et al., 2017; Deci & Ryan, 1985a; Ryan & Deci, 2017). Situational determinants, such as the effects of environmental contingencies that support forms of motivation and satisfy psychological needs for the particular behavior and context, are likely to have a stronger influence. This is a perspective shared more broadly with theory on personality, that effects of generalized orientations are likely to be modest relative to situational influences (John et al., 2010). Consistent with this perspective, and current findings, causality orientations therefore serve as intrapersonal biases that may affect behavior when environmental

influences are muted or neutral, but their effects may be swamped or overridden entirely by strong situational influences (Deci & Ryan, 1985a; Ryan & Deci, 2017).

Process Model

An important goal of the current meta-analysis was to estimate a unique process model in which causality orientations related to behavior mediated by autonomous and controlled forms of motivation. Although this model has not been previously tested, it is based on predictions derived from the causality orientations theory and organismic integration theory. Findings broadly supported pre-registered predictions for the effects of causality orientation dimensions on behavior mediated by forms of motivation. A key finding was the positive non-zero total effect of autonomy causality orientation on behavior, which corroborated the overall correlation between these constructs. Interestingly, the direct and indirect effects of which the total effect was comprised were no different from zero, but together they produced the non-zero total effect. This finding suggests that although autonomous motivation may be implicated in the process by which generalized autonomy orientations relate to behavior, a small residual effect is present. We provide three speculative interpretations of these effects. First, imprecision in the measurement of autonomous motivation may mean that it is not efficient in mediating the effect of autonomy orientation on behavior. Second, the residual effect of autonomy orientation may be mediated by other unmeasured constructs, such as implicit motives that reflect automated non-conscious motivational processes that are not captured by measures of autonomous motivation (Burton et al., 2006; Keatley et al., 2012; Levesque & Pelletier, 2003). Third, potential moderators of the indirect effects may exist which determine whether the effect of autonomy orientation is mediated by autonomous motivation, such as the presence of environmental contingencies that support, thwart, or frustrate needs. Such moderators my determine the strength of situational forms of motivation on behavior and, therefore, determine the strength of the mediated pathway, a moderated mediation effect (e.g., Hayes, 2018; Wiedemann et al., 2009; Zhou et al., 2015).

A candidate moderator of this mediation path may be behavior type. High heterogeneity in behavior type in studies included in the current study precluded a behavior type moderator analysis. However, previous meta-analyses have indicated that effects of autonomous motivation on behavior vary considerably by behavior type. For example, Ng et al. (2012) revealed that autonomous motivation-behavior relations ranged from a small effect size for medication adherence to a medium-sized effect for healthy eating. In the current analysis, the averaged correlation between autonomous motivation and behavior was close to the lowest of these estimates (r = .099), and it is noteworthy that this effect was based on very few studies from the current sample (k = 4). Given that the autonomous motivation-behavior relation is an important component of the indirect effect of autonomy orientation on behavior, we reasoned that variation in the size of this effect in the process model would alter the size of the indirect effect. As an illustration, we re-estimated the current process model using the current data but substituted the correlation and variability estimates for the autonomous motivation-behavior relationship with the smallest (r = .11) and largest (r = .41) values from Ng et al.'s meta-analysis. As expected, when the autonomous motivation-behavior correlation was substituted for the smallest effect, the indirect effect of autonomy orientation on behavior was small, β = .028, 95% CI [-.001, .062], and comparable to the effect found in the current set of studies, β = .023, 95% CI [-.014, .061]. However, the effect was substantively larger and non-zero, β = .141, 95% CI [.075, .206] when the largest effect was substituted[5]. These ancillary analyses effectively demonstrate how varying the effect of autonomous motivation on behavior influences the indirect effect of autonomy orientation on behavior, consistent with the process model. Situational factors are, therefore, likely to play an important role in determining the proposed indirect effects of autonomy orientation on behavior.

A priority avenue for future research arising from these analyses is to test the process model in the presence of candidate moderators likely to affect the autonomous motivation-behavior relation and, by extension, the indirect effect of autonomy orientation on behavior through autonomous

[5]Full results of these ancillary analyses are available online: https://osf.io/gjs5v/

motivation. One potential moderator indicated by the variability in the effect size of the autonomous motivation-behavior relationship in Ng et al.'s (2012) meta-analysis is behavior type. It seems some behaviors may be more likely to be experienced as autonomously motivated than others. This is likely to be due to factors typically present in the context in which the behavior is routinely performed that determine the extent to which they are perceived to be autonomously motivated and need satisfying. Such factors are likely to include the behaviors displayed, and contingencies utilized, by social agents in that context that support (e.g., availability informational feedback) or thwart (e.g., rewards, deadlines) psychological need satisfaction. Such tests will provide further insight into the extent to which autonomy orientation influences behavior due to differing contextual factors.

A further important and unique finding of the present analysis was that the net zero total effect of control orientation observed in the process model comprised a positive direct effect and a negative indirect effect mediated by controlled motivation. These findings illustrate the value of the process model as it identifies two distinct pathways by which control orientation relates to behavior, which effectively cancel each other out. Such pathways would not be detected if analyses were confined solely to analysing the effect of control orientation on behavior without considering mediation effects (c.f., Ng et al., 2012). A similar pattern of direct and indirect effects of opposing signs have been observed in other meta-analytic tests of process models (e.g., Hagger et al., 2017). The negative indirect effect of control orientation mediated by controlled motivation is consistent with our pre-registered hypothesis, and with self-determination theory, and suggests that control orientation may serve as a source of information on which individuals base their motivation toward specific behaviors, and this tends to lead to desistence or avoidance of behaviors. A likely reason for this is that controlled motivation is associated with maladaptive outcomes such as psychological need frustration, negative affect, and behavioral avoidance. So, when faced with the prospect of performing a behavior that is perceived as controlled by external forces (e.g., punishments, deadlines, controlling language from social agents), individuals may opt not to engage in the behavior and avoid it altogether. This is

consistent with research that has demonstrated negative relations between external regulation, the most extreme form of controlled motivation on the perceived locus of causality, and behavior (Ng et al., 2012). On the other hand, the positive direct effect may reflect engagement in the behavior attributable to conditioned processes brought about by rewarding contingencies or other externally-referenced motivated actions that may be automatic or non-conscious in nature. Such effects likely reflect repeated past experiences with the behavior that covary with the controlling contingencies.

While both effects together result in a net zero effect in the current analysis, it is unlikely that both effects occur simultaneously. Instead, the extent to which each pathway determines behavior likely depends on the presence of moderator variables that determine the relative strength of each, or both, paths. For example, the direct effect of control orientation on behavior may be determined by the extent to which controlling contingencies like rewards or punishments lead to spontaneous engagement in behavior independent of controlled motivation. As before, such an effect may be a function of non-conscious motives generated out of habitual or routine experience of the behavior in the presence of the reinforcing factors. Research has suggested that such action patterns reflect habits, and likely coincide with implicit evaluations of the behavior (for reviews, see Hagger, 2019; Hagger, 2020). Similarly, the indirect effect may be a function of contingencies that affect the extent to which the behavior is perceived as controlled motivated. Variation in the relationship between controlled motivation and behavior will, ultimately, affect the strength of the indirect effect of control orientation on behavior, similar to the way the strength of the autonomous motivation-behavior relationship affects the indirect effect of autonomy orientation on behavior. Such contingencies may include contextual factors that determine whether a behavior is experienced as controlled motivated, such as factors that thwart psychological needs (e.g., use of controlling language) or signal external control over the behavior (e.g., use of rewards or punishments). Future research should examine the indirect effect of control orientation on behavior by varying behavior type and these contextual

contingencies. Such research may shed light on the extent to which control orientation directly predicts behavior or whether the effect is directed through controlled motivation.

Analysis of Moderators

While we were unable to examine effects of moderator variables in the full process model due to small numbers of studies, moderator analyses of two truncated models revealed a few moderator effects. That the effect of autonomous motivation on behavior was larger among studies adopting cross-sectional designs and non-student samples was not surprising. Studies adopting cross-sectional designs not only tend to have strong correspondence between measure of behavior and psychological constructs, but the measures will also have been taken concurrently, which likely exacerbates common method variance (Podsakoff et al., 2003). Both methodological artifacts are known to inflate relations. Similarly, student samples typically comprise participants from privileged, educated, and ethnically homogenous groups, which is likely to affect relations among constructs (Henrich et al., 2010). In addition, studies with lower quality may lead to imprecision in effect size estimates. Although lower quality studies are expected to attenuate true effects, additional error variance associated with imprecisions in study design may also inflate relations (Johnson et al., 2015). That balanced gender samples demonstrated larger autonomy orientation-behavior relations is perhaps less easily explained. For example, research has demonstrated larger mean values for autonomy orientation among females, although it does not necessarily follow that mean differences on constructs are translated into relations with other constructs (Deci & Ryan, 1985a). Further, our comparison moderator group comprising balanced gender samples included substantive absolute numbers of female participants, so concluding that females' behavior is less likely associated with autonomy orientation based on the current data may be premature. Future research formally testing gender differences in model effects in representative samples would be necessary to elucidate current findings.

Moderator analyses also indicated that the negative effect of impersonal orientation on behavior was larger in older and mixed age samples, studies adopting non-cross-sectional designs, and studies on non-student samples, and the negative effect of this orientation on autonomous motivation was larger in studies of questionable quality. The study design and sample type moderation effects can similarly be attributable to the potential for these design features to introduce additional error variance to tests of the effect. However, the age-related effect may be due to the greater experience of older samples with behaviors experienced as lacking in intentionality or motivational rationale, consistent with an impersonal orientation. Chronic experience of such behaviors, such as in the workplace or interpersonal relationships, may increase the likelihood of some people developing an impersonal orientation which subsequently affects behavioral engagement and the type of motivation adopted in those contexts. This might also be exacerbated by a lack of perceived contextual factors that may mitigate effects of impersonal orientation, such as information in the environment that signal the lack of a clear rationale or reason for acting which, over time, lead to a lack of motivation (Bartholomew et al., 2011; Deci et al., 1994). Younger samples may not have had as many opportunities for these perceptions to develop. Knowledge of the effects of impersonal orientations on autonomous motivation and behavior may have important implications for practice. For example, social agents in leadership positions may considered providing clarity in the rationales provided for performing tasks for individuals operating in the environment, which may not only foster autonomous motivation in particular contexts, but also serve to stymie the impact of impersonal orientation on motivation and behavior. Research is needed to examine whether interventions that provide such rationales moderate effects of impersonal orientation on behavior in given contexts, particularly in older individuals.

Finally, it is also important to note that the majority of the moderator analyses should be interpreted with an important caveat; despite large observed differences in the observed averaged effect sizes across moderator groups, large variability and wide confidence intervals meant that differences should not be considered robust. Only the effect of study design moderator for the

autonomy orientation-behavior relationship and the effect of the study quality moderator on the impersonal orientation on autonomous motivation achieved non-zero coefficients in formal difference tests. These findings reflect an important general observation in the current research; the substantive heterogeneity in effect sizes across studies not attributable to sampling error. Although such observations are commonplace in meta-analyses of correlational research in psychology, it creates considerable difficulties in estimating true effects among constructs from psychological theories, and isolating effects that reflect theoretical predictions from external factors that moderate those effects. One solution is the conduct of multiple replication studies testing model effects in which specific conditions are kept constant. Such studies might adopt experimental designs in which randomization to conditions may negate variation in sample characteristics, or correlational designs in randomly selected samples that include measures of environmental and contextual characteristics to control for potential moderators. This may be an important direction in which to take future tests of effects of causality orientations on motivational and behavioral outcomes in the current process model.

Strengths, Limitations, and Future Research Directions

The current research has numerous strengths: (1) Use of meta-analytic data from multiple studies to test relations among causality orientation dimensions, forms of motivation, and behavioral outcomes; (2) Use of meta-analytic structural equation modeling to test pre-registered effects of a unique process model in which causality orientations relate to behavior mediated by autonomous and controlled forms of motivation; and (3) Testing effects of key moderator variables of relations among causality orientation dimensions and motivational and behavioral outcomes. Overall, current findings are expected to guide future research by identifying current gaps in evidence, as well as guide practice by identifying potential means to facilitate autonomous motivation and minimize effects of individual difference factors that may undermine autonomous motivation.

However, the current analysis has a number of limitations, many of which signpost potential avenues for future research. A prominent limitation was the relatively small numbers of studies

estimating relations between the causality orientation dimensions and the behavioral regulations from the perceived locus of causality. As a result, only very small numbers of tests of some effects or, in some cases, a solitary study, were available. This meant available data were sufficient to estimate the process model using autonomous and controlled motivation as mediators rather than separate behavioral regulations. More research testing relations between causality orientations separate regulation styles is warranted. The relatively few numbers of studies also precluded an analysis in which the autonomous and controlled motivation constructs were conceptualized as latent variables indicated by separate locus of causality components (e.g., autonomous motivation indicated by intrinsic motivation and identified regulation measures, and controlled motivation measured by introjected and external regulation components). As research in this area expands this may be a viable avenue for future syntheses. In addition, the small numbers of studies also placed limits on our moderator analyses. For example, small numbers of studies on predominantly male samples or older samples meant that our moderator analyses were restricted to comparisons between a specific moderator group (e.g., predominantly female samples, younger samples) and a broader aggregate category (e.g., mixed gender samples, samples with a wide age range). This suggests that researchers should prioritize testing effects of these moderators on relations between process model constructs. Also, there was no critical mass of studies testing effects of process model constructs on specific behaviors, which precluded analysis with behavior type as a moderator. Given previous meta-analyses have demonstrated that some of the component effects of the process model vary by behavior type (Ng et al., 2012), and our ancillary analysis illustrating how effects within the model change when substituting for these effects, future studies should consider testing behavior type as a moderator of model effects. Furthermore, as the literature expands, future meta-analytic tests of the process model with age, gender, and behavior type moderator analyses may be possible.

Another limitation of the current study was the large variability in effect sizes across studies in both the zero-order correlations among causality orientation dimensions, forms of motivation, and

behavior as well as effect sizes from the process model. High variability is expected in correlational studies derived from multiple contexts, populations, and behaviors, and has been observed in previous meta-analyses of correlations and tests of process models (e.g., Cheung & Hong, 2017; Hagger et al., 2017; Ng et al., 2012; Zhang et al., 2019). High variability should catalyze a search for pertinent moderators of model effects. However, current moderator analyses did little to resolve heterogeneity in model effects, and were also limited by small numbers of studies in some moderator groups. The extent of the variability places limits on capacity to draw definitive conclusions on the true size of the effects specified in the current process model. Resolution may lie in the systematic conduct of replications of model effects in primary studies on representative samples and with strict controls on potential moderating variables. Such an endeavor would be time consuming and expensive, but the payoff large given it may yield greater precision in effects and allow for conclusions to be drawn on a narrower range of possible values for the true size of model effects. It may also serve as a platform for future systematic evaluation of the effects of candidate moderators.

A further limitation relates to the dearth of studies examining the role of causality orientations as moderators of effects of forms of motivation on behavior. Theory (e.g., Deci & Ryan, 1985a; Ryan & Deci, 2017) and previous research (e.g., Ewing, 2010; Hagger & Chatzisarantis, 2011; Knee & Zuckerman, 1998) suggest that causality orientations should serve to moderate effects of motivation-related phenomena on behavior and related outcomes. However, much of the research has been confined to moderation of effects of other constructs such as personality (Jerković et al., 2017), coping (Knee & Zuckerman, 1998), and cultural ideology (Duriez, 2011), and few have examined the role of causality orientations on processes within self-determination theory (Hagger & Chatzisarantis, 2011). Specifically, there is hardly any research that has systematically evaluated effects of causality orientations on motivation-behavior relations, particularly the potential role that causality orientations may play in exacerbating or undermining effects of the types of motivation experienced for particular behaviors and in particular contexts on behavior. This meant that testing moderator effects of

causality orientations on motivation-behavior relations in the current meta-analyses was not feasible. Testing such moderator effects remains an important avenue for future research.

It is also important to acknowledge limits to the generalizability of the current findings. The current analysis was conducted with due diligence paid to locating all available studies and datasets testing relations among causality orientations, autonomous and controlled forms of motivation, and behavior. The current model has, therefore, been tested on research conducted across multiple populations, contexts, and behaviors, and, therefore, represents averaged effects among the constructs of the proposed model synthesized using random effects meta-analysis from the currently available evidence. We also tested whether the proposed pattern of effects was conditional on specific features of the studies involved through moderator analyses. Given that relatively few moderator effects were identified, the averaged effects among constructs identified in the current research can be considered broadly generalizable. However, generalizability of findings should be interpreted with the caveat that model effects represent averaged effects across sample, and does not rule out the potential for moderators unaccounted for in the current analysis. We have identified other potential moderators in our discussion of limitations and these should be considered priorities for future research, which may provide further evidence on the extent to which these findings can be generalized.

Finally, causality orientations theory proposes that the autonomy, control, and impersonal orientations are not orthogonal – individuals likely endorse each orientation to some degree rather than endorsing one dimension and not others (Deci & Ryan, 1985a). This interdependence highlights the imperative of examining the unique effects of each dimension on motivational and behavioral outcomes while simultaneously accounting the effects all dimensions, as we have in our proposed process model. Researchers have taken this a step further by exploring how characteristic profiles of causality orientation dimensions may relate to outcomes. Such an approach takes the relative salience assigned to each dimension into account when exploring relations of the dimensions outcomes. For example, Anderson et al. (1989) identified characteristic profiles of causality orientation dimensions

and coping strategies used by nurses to cope with work-related stress. Nurses with profiles representing high levels of problem-focused coping and autonomy orientation reported lowest levels of work-related stress. The profile approach has only received relatively limited attention in the literature on causality orientations theory, and the dearth of research precluded a synthesis of profiles across studies in the current analysis. As the research literature testing profiles of causality orientations expands, future syntheses of studies may consider examining the contribution of profiles of causality orientations to predicting motivation and behavior, consistent with similar recommendations for constructs from the perceived locus of causality (Howard et al., 2017).

Conclusion

Based on self-determination theory, the current study meta-analyzed studies testing relations among causality orientation dimensions, motivational styles, and behavior. Specifically, the study tested the pattern of intercorrelations among the causality orientation dimensions, correlations among the dimensions with forms of autonomous and controlled motivation from the perceived locus of causality, and tested a series of pre-registered hypotheses of a unique process model based on causality orientations theory and organismic integration theory, in which orientation dimensions predicted behavior mediated by autonomous and controlled forms of motivation. Results supported theoretically-predictable pattern of intercorrelations among the causality orientations, and their relations with forms of motivation and behavior. Test of the process model revealed an indirect total effect of autonomy orientation on behavior comprising direct and indirect effects through autonomous motivation, and a net zero effect of control orientation on behavior comprising a positive direct effect and a negative indirect effect through controlled motivation. Moderator analyses revealed relatively few non-zero moderator effects, but identified trends in effects for sample gender, study quality, study design, and sample type. Consistent with effects across personality and individual research, current findings suggest that individual differences in causality orientations have pervasive but small effects on the types of motivation adopted by individuals in multiple contexts, behaviors, and populations,

and on behavioral engagement. The presence of indirect and total effects in the process model provide initial evidence for the suggested process involved, although effects were small and highly variable. Results also identify some key evidence gaps, particularly in the need for systematic replication of relations between causality orientations, behavioral regulations from self-determination theory, and behavior, and the need for systematic evaluation of moderator effects among process model constructs. Consistent with the tenets of self-determination theory, particularly cognitive evaluation theory and organismic integration theory, current findings also suggest that contextual factors, such as need-supportive behaviors and contextual contingencies presented by social agents in leadership positions, may be critical in determining the type of motivation experienced by individuals and behavioral persistence beyond effects of individual differences in causality orientations.

Statements

Preregistration statement: All major hypotheses in the current study were preregistered, the registration document is accessible online: https://osf.io/7nz6d. Exploratory hypotheses that were not preregistered have been labelled as such.

Sampling statement: We report results from a meta-analyses, we have clearly described how studies were located and data extracted. Sample sizes including number of studies and total sample sizes associated with each tested effect are reported.

Open materials statement: We provide information regarding all procedures applied and all measures assessed in this study in the manuscript itself and in the supplemental materials accessible online: https://osf.io/gjs5v/.

Open data statement: The data needed to reproduce the results are open and data are openly accessible via the following link: https://osf.io/gjs5v/

Reproducible script statement: Data analysis scripts that allow reproduction of all reported results are accessible via the following link: https://osf.io/gjs5v/

Effects statement: We report basic descriptive statistics, effect sizes, exact *p*-values, and 95% confidence (credible) intervals where appropriate.

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Table 1

Zero-Order Parameter Estimates from Conventional Fixed and Random Effects Model Meta-Analysis for Relations Among General Causality Orientations, Motivational Styles from Self-Determination Theory, and Behavior with Heterogeneity and Bias Statistics

Effect	Meta-analytic models										Bias statistics[a]		
	Random effects							Fixed effects		Q	r^{+}_{PET}	r^{+}_{PEESE}	p-BIAS
	k	r^{+}_{RE}	SE	CI_{95} LL	CI_{95} UL	I^2	τ^2	r^{+}_{FE}	SE				
Aut.–Aut. Mot.	24	.337***	.050	.239	.435	94.938	.054	.342***	.013	302.537***	.262***	.320***	.080
Aut.–Beh.	21	.147**	.048	.053	.242	89.517	.041	.126***	.016	148.786***	.071	.085***	.158
Aut.–Con.	66	.109***	.029	.052	.165	92.923	.049	.124***	.008	782.186***	.139***	.137***	.379
Aut.–Con. Mot.	12	.060	.044	-.027	.015	77.999	.016	.032	.017	37.094***	-.007	.022	.120
Aut.–Ex. Reg.	7	-.027	.049	-.123	.069	75.631	.012	-.041*	.018	15.732*	-.057	.054*	.555
Aut.–Id. Reg.	8	.340***	.042	.258	.424	78.230	.010	.264***	.018	47.568***	.117***	.191***	<.001
Aut.–Ij. Reg.	8	.172**	.064	.047	.297	89.122	.027	.097***	.018	57.515***	-.019	.037*	<.001
Aut.–In. Mot.	8	.376***	.059	.261	.492	90.897	.024	.299***	.018	88.078***	.133***	.221***	<.001
Aut.–Imp.	48	-.103***	.029	-.159	-.047	89.482	.033	-.083***	.009	384.159***	-.027	-.050***	.018
Con.–Aut. Mot.	19	-.011	.047	-.104	.081	88.333	.034	-.003	.015	98.223***	-.014	.006	.479
Con.–Beh.	20	.073	.039	-.002	.149	79.254	.022	.099***	.017	79.307***	.214***	.150***	.010
Con.–Con. Mot.	12	.292***	.051	.193	.392	88.892	.025	.341***	.016	52.012***	.390***	.367***	.028
Con.–Ex. Reg.	6	.294***	.045	.206	.381	73.387	.008	.300***	.019	13.486*	.301***	.303***	.853
Con.–Id. Reg.	7	.083**	.018	.047	.118	0.025	.000	.082***	.018	9.664	.089**	.090***	.619
Con.–Ij. Reg.	7	.176***	.030	.118	.233	40.267	.002	.207***	.018	11.010	.285***	.251***	.003
Con.–In. Mot.	6	.018	.048	-.077	.113	72.703	.009	-.014	.019	15.782**	-.060	-.032	.106
Con.–Imp.	47	.273***	.029	.216	.330	90.750	.034	.295***	.010	415.503***	.329***	.316***	.226
Imp.–Aut. Mot.	9	-.203**	.067	-.333	-.072	86.811	.034	-.237***	.026	48.427***	-.550***	-.373***	.008
Imp.–Beh.	13	-.057	.042	-.139	.025	75.717	.017	-.061	.020	54.351***	-.097	-.064*	.926
Imp.–Con. Mot.	6	.073	.113	-.149	.295	92.188	.070	.042	.033	64.995***	-.169	-.087	.119
Imp.–Ex. Reg.	5	.278***	.031	.217	.339	0.000	.000	.284***	.034	0.450	.286	.288***	.994
Imp.–Id. Reg.	5	.081	.055	-.026	.189	60.626	.009	.091	.034	9.483	.287	.196*	.228
Imp.–Ij. Reg.	5	.042	.088	-.130	.213	85.092	.032	.039	.034	27.697***	-.009	.017	.770
Imp.–In. Mot.	5	-.168	.102	-.368	.032	89.939	.046	-.183***	.034	34.970***	-.328*	-.242***	.445
Aut. Mot.–Beh.	4	.099***	.033	.034	.163	00.001	.000	.098**	.033	5.108	–	–	–
Aut. Mot.–Con. Mot.	7	.178	.132	-.081	.437	97.570	.116	.354***	.019	122.689***	.493***	.418***	<.001
Con. Mot.–Beh.	3	-.146	.075	-.292	.001	57.473	.010	-.143**	.049	4.433	–	–	–
Id. Reg.–Beh.	2	.089	.110	-.207	.224	69.920	.017	-.018	.057	3.26	–	–	–
Id. Reg.–Ex. Reg.	4	.267	.143	-.013	.547	96.428	.077	.350***	.020	47.414***	–	–	–

Id. Reg.–Ij. Reg.	5	.428***	.076	.277	.578	91.355	.026	.541***	.020	42.634***	.637***	.579***	.001
Ij. Reg.–Beh.	2	.070	.068	-.063	.202	24.122	.002	.075	.057	1.319	–	–	–
Ij. Reg.–Ex. Reg.	4	.505***	.113	.285	.726	95.184	.047	.857***	.020	113.847	–	–	–
In. Mot.–Ex. Reg.	4	.191	.148	-.099	.480	96.478	.083	.183***	.020	40.917	–	–	–
In. Mot.–Id. Reg.	4	.653***	.059	.538	.769	91.586	.013	.631***	.020	29.468***	.489***	.566***	<.001
In. Mot.–Ij. Reg.	4	.380***	.099	.186	.573	93.438	.035	.321***	.020	27.832	–	–	–

Note. [a]Bias statistics for effects based on small numbers of studies are unlikely to provide reliable estimates, so these statistics have not been computed for effects based on fewer than 5 studies. r^{+}_{RE} = Corrected effect size estimate from conventional random effects meta-analysis model; r^{+}_{FE} = Corrected effect size estimate from conventional fixed effects meta-analysis model; SE = Standard error; CI_{95} = 95% confidence interval; LL = Lower limit of CI_{95}; UL = Upper limit of CI_{95}; I^2 = Higgins and Thompson's (2002) I^2 statistic for parameter estimate; τ^2 = Estimated variance in population; Q = Cochran's Q statistic from conventional analyses; r^{+}_{PET} = Effect size estimate corrected for bias using the precision-effect estimate; r^{+}_{PEESE} = Effect size estimate corrected for bias using the precision-effect estimate with standard errors; p-$_{BIAS}$ = Probability value for the precision estimate using the PET-PEESE procedure; Aut. = Autonomy causality orientation; Con. = Control causality orientation; Imp. = Impersonal causality orientation; Aut. Mot. = Autonomous motivation; Con. Mot. = Control motivation; Beh. = Behavior; In. Mot. = Intrinsic motivation; Id. Reg. = Identified regulation; Ij. Reg. = Introjected regulation; Ex. Reg. = External regulation.
* $p < .05$ ** $p < .01$ *** $p < .001$

Table 2

Standardized Path Coefficients for Direct and Indirect Effects for the Meta-Analytic Structural Equation Model (Stage 2) of the Causality Orientation Dimensions on Behavior Mediated by Motivational Styles (Model 1), on Behavior Only (Model 2), and on Motivational Styles Only (Model 3)

Effect	β	*SE*	Wald CI_{95}		*z*	*p*
			LL	UL		
Model 1						
Direct effects						
Aut.→Aut. Mot.	.316	.050	.219	.413	6.377	<.001
Con.→Aut. Mot.	.003	.053	-.101	.107	0.054	.957
Imp.→Aut. Mot.	-.169	.067	-.301	-.037	-2.507	.012
Aut.→Beh.	.101	.054	-.005	.206	1.867	.062
Aut. Mot.→Beh.	.092	.055	-.017	.200	1.658	.097
Con.→Beh.	.131	.051	.030	.232	2.554	.011
Con. Mot.→Beh.	-.198	.068	-.332	-.064	-2.892	.004
Imp.→Beh.	-.044	.050	-.142	.055	-0.863	.388
Aut.→Con. Mot.	.028	.044	-.059	.114	0.629	.529
Con.→Con. Mot.	.283	.059	.167	.400	4.759	<.001
Imp.→Con. Mot.	.001	.114	-.223	.225	0.005	.996
Indirect effects						
Aut.→Aut. Mot.→Beh.	.029	.018	-.006	.064	1.625	.104
Aut.→Con. Mot.→Beh.	-.005	.009	-.023	.012	-0.613	.540
Con.→Aut. Mot.→Beh.	.000	.005	-.009	.010	0.054	.957
Con.→Con. Mot.→Beh.	-.056	.024	-.103	-.009	-2.326	.020
Imp.→Aut. Mot.→Beh.	-.015	.011	-.037	.006	-1.419	.156
Imp.→Con. Mot.→Beh.	.000	.023	-.044	.044	-0.005	.996
Sum of indirect effects[a]						
Aut.→Beh.	.023	.019	-.014	.061	1.212	.226
Con.→Beh.	-.056	.025	-.104	-.008	-2.266	.023
Imp.→Beh.	-.016	.025	-.065	.034	-0.611	.541
Total effects[b]						
Aut.→Beh.	.124	.046	.034	.214	2.711	.007
Con.→Beh.	.075	.042	-.007	.158	1.784	.074
Imp.→Beh.	-.059	.043	-.143	.025	-1.384	.166
Correlations						
Aut.↔Con.	.106	.027	.054	.159	3.946	<.001
Aut. Mot.↔Con. Mot.	.175	.118	-.056	.405	1.483	.138
Aut.↔Imp.	-.098	.027	-.150	-.046	-3.706	<.001
Imp.↔Con.	.268	.028	.213	.324	9.523	<.001
Model 2						
Direct effects						
Aut.→Beh.	.124	.046	.034	.213	2.703	.007
Con.→Beh.	.076	.042	-.007	.158	1.805	.071
Imp.→Beh.	-.059	.043	-.143	.024	-1.393	.164
Correlations						
Aut.↔Con.	.106	.027	.053	.159	3.935	<.001
Aut.↔Imp.	-.098	.027	-.150	-.046	-3.705	<.001
Imp.↔Con.	.268	.028	.213	.324	9.524	<.001
Model 3						
Direct effects						

Aut.→Aut. Mot.	.316	.050	.218	.413	6.364	<.001
Con.→Aut. Mot.	.003	.053	-.101	.106	0.053	.957
Imp.→Aut. Mot.	-.169	.067	-.301	-.037	-2.506	.012
Aut.→Con. Mot.	.028	.044	-.058	.114	0.632	.528
Con.→Con. Mot.	.283	.059	.167	.400	4.765	<.001
Imp.→Con. Mot.	.000	.114	-.224	.224	0.004	.997
Correlations						
Aut.↔Con.	.106	.027	.053	.159	3.943	<.001
Aut. Mot.↔Con. Mot.	.175	.118	-.056	.405	1.483	.138
Aut.↔Imp.	-.098	.027	-.151	-.046	-3.705	<.001
Imp.↔Con.	.268	.028	.213	.324	9.513	<.001

Note. [a]Sum of indirect effects of causality orientation dimensions on behavior; [b]Total effect of causality orientation dimensions on behavior. β = Standardized path coefficient; Wald CI_{95} = Wald 95% confidence interval; LL = Lower limit of CI_{95}; UL = Upper limit of CI_{95}; CI_{95} = Conventional 95% confidence interval; β_{diff} = Difference in standardized path coefficient; Aut. = Autonomy causality orientation; Con. = Control causality orientation; Imp. = Impersonal causality orientation; Aut. Mot. = Autonomous motivation; Con. Mot. = Control motivation; Beh = Behavior.

Figure 1. Proposed model illustrating effects of general causality orientations on behavior mediated by forms of motivation from self-determination theory (Model 1). Hypothesized signs for effects of causality orientations and forms of motivation are depicted using positive (+) and negative (–) signs.

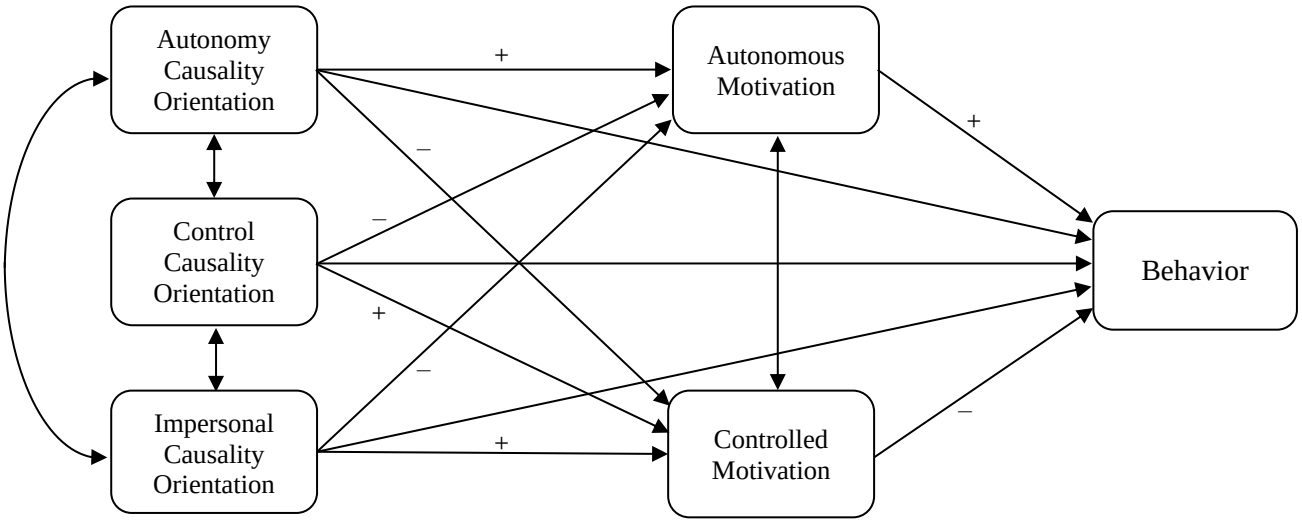
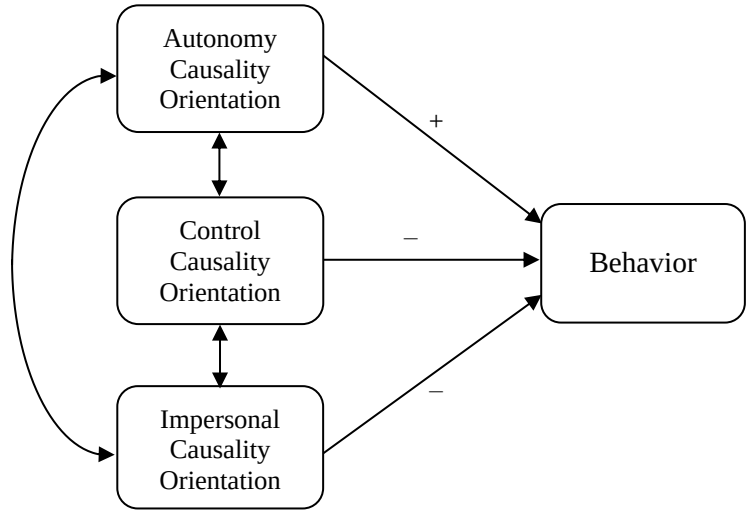
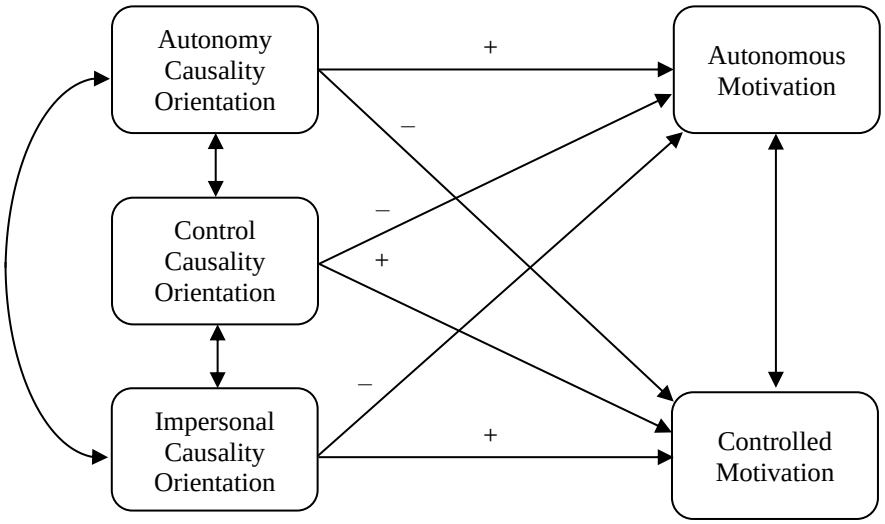


Figure 2. Proposed structural equation models of effects of general causality orientations on (a) behavior only (Model 2) and (b) motivational orientations from self-determination theory only (Model 3). Hypothesized signs for effects are depicted using positive (+) and negative (–) signs.



(a)



(b)

Figure 3. Forest plot of relations between autonomy and control causality orientations for studies included in the meta-analysis with summary effect.

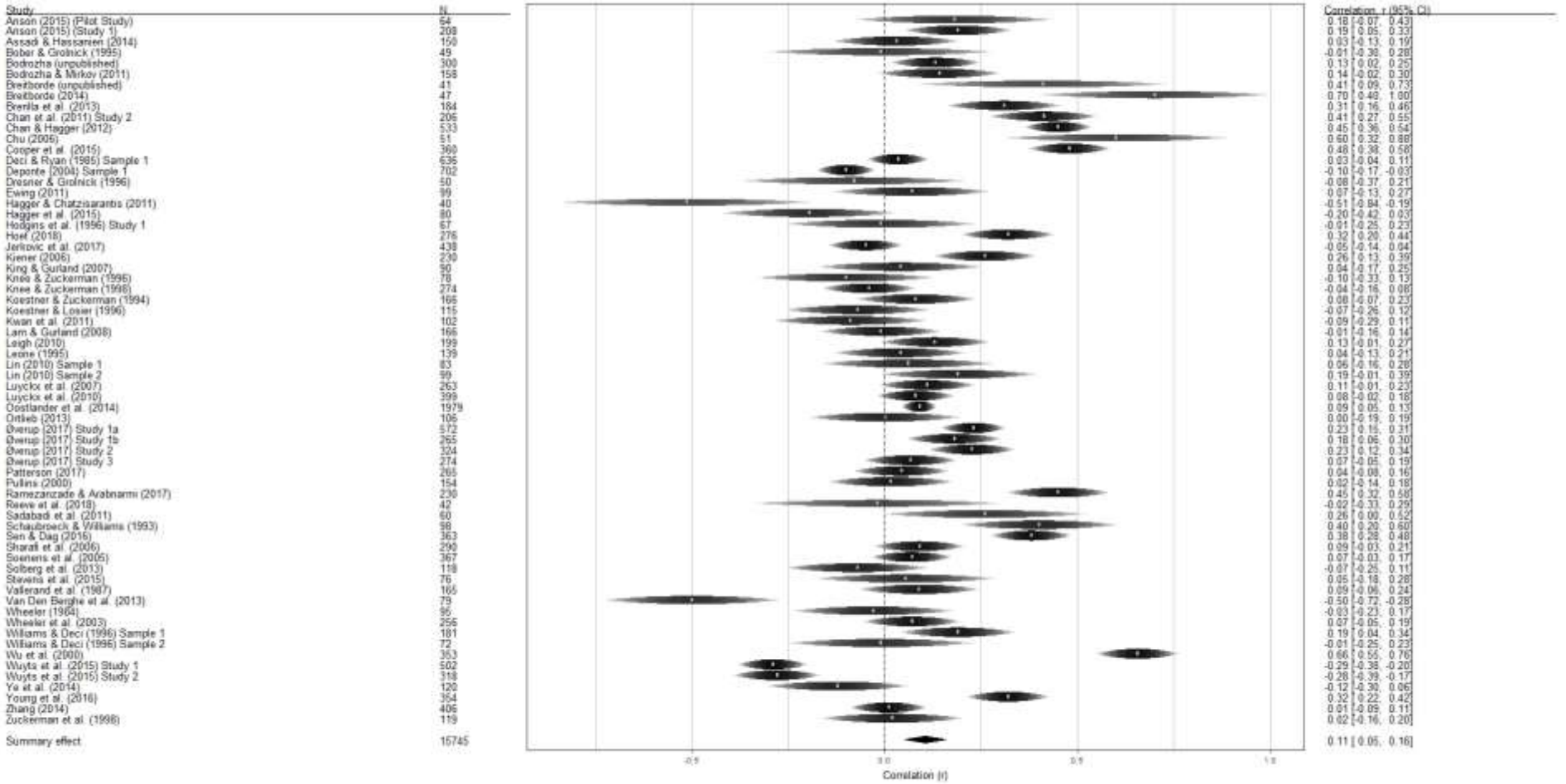


Figure 4. Forest plot of relations between autonomy and impersonal causality orientations for studies included in the meta-analysis with summary effect.

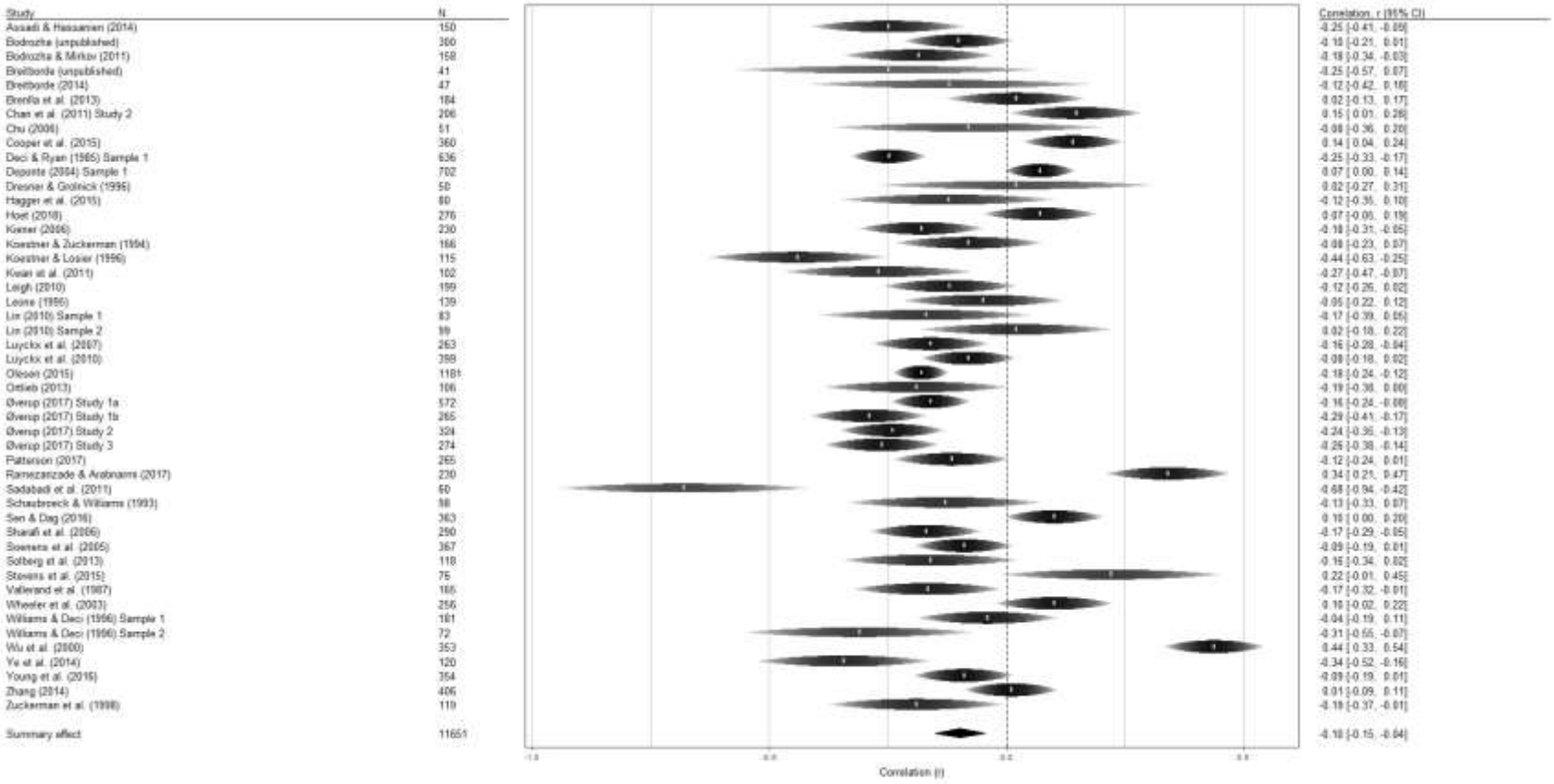
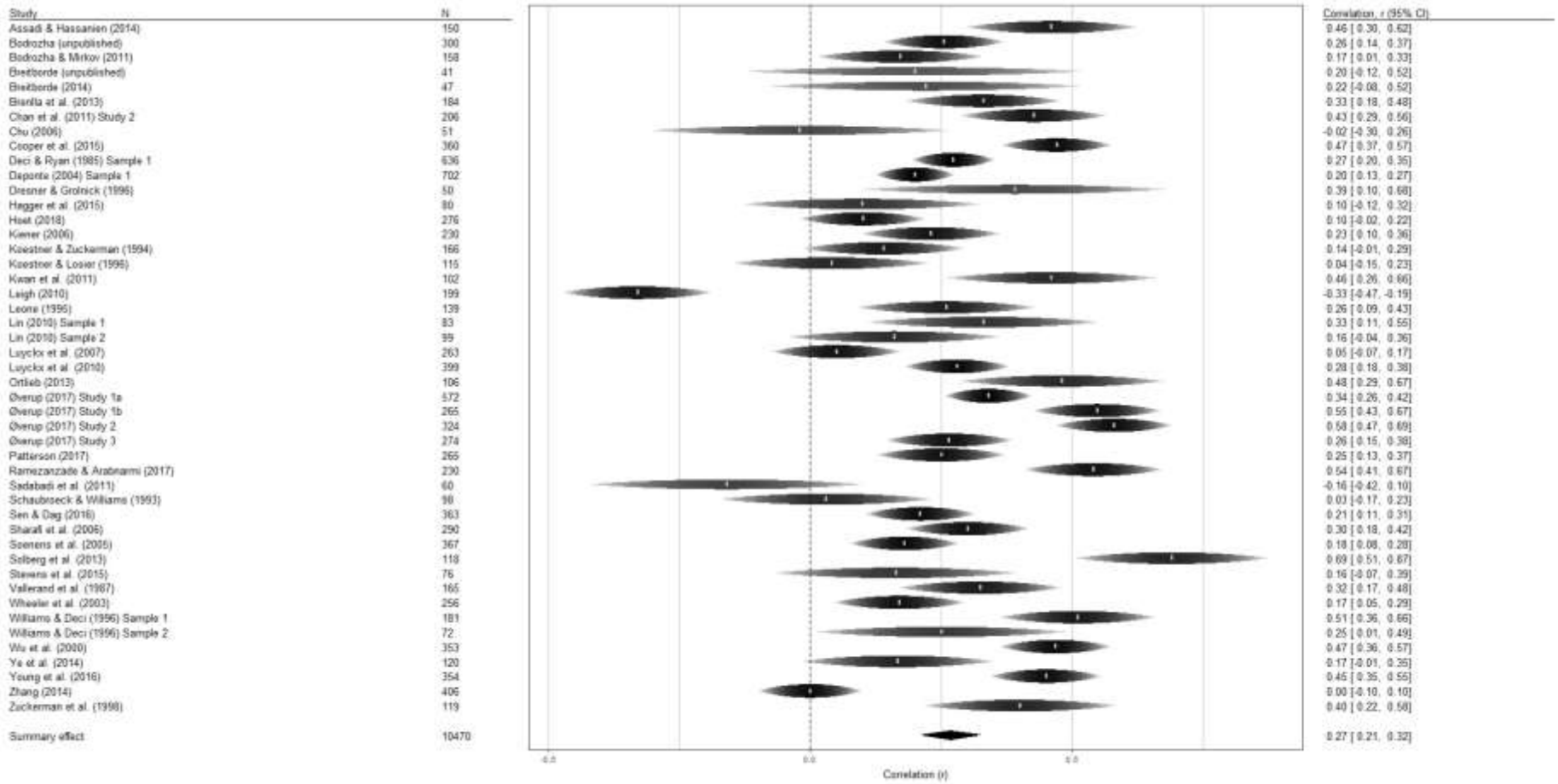
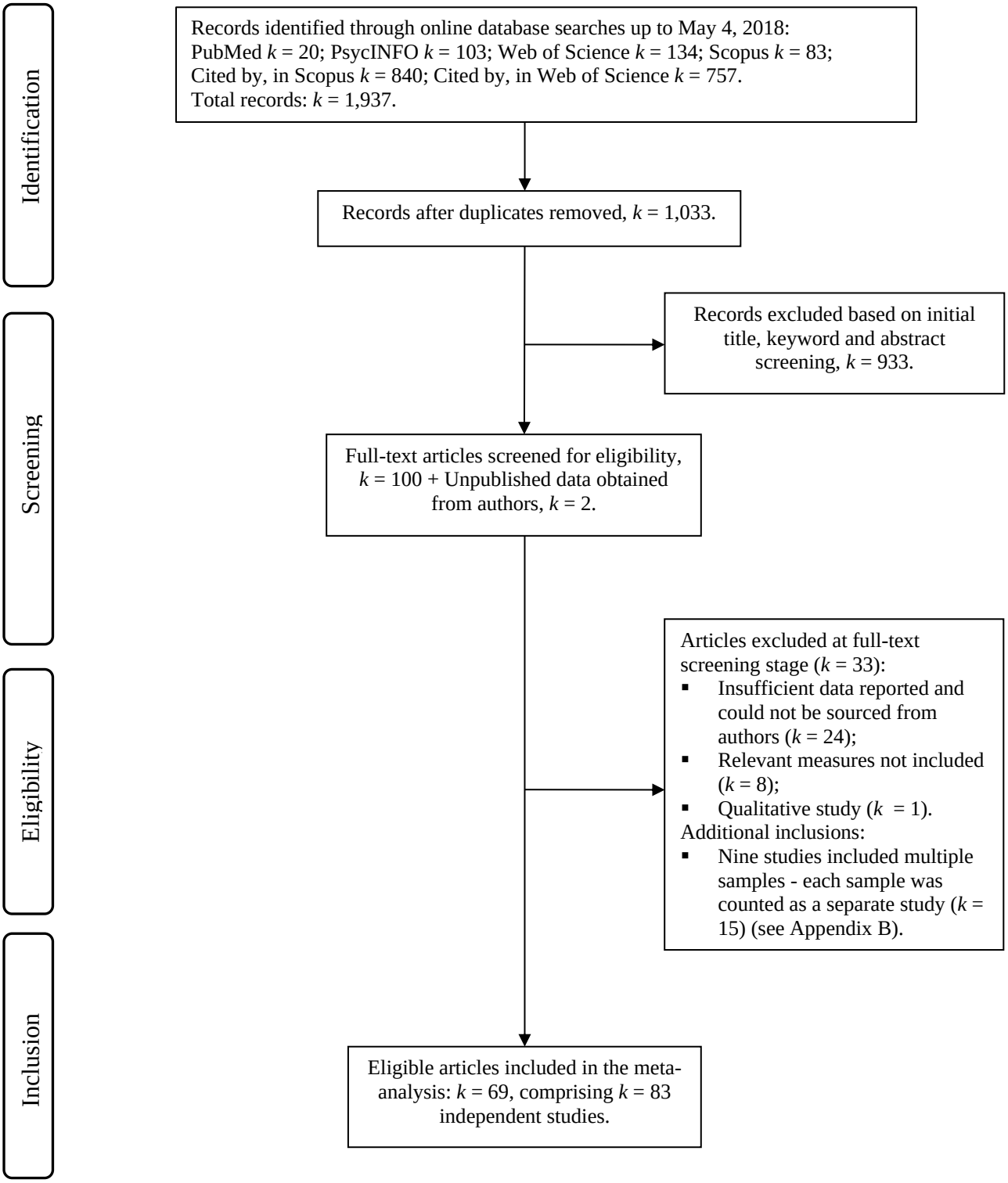


Figure 5. Forest plot of relations between control and impersonal causality orientations for studies included in the meta-analysis with summary effect.



Appendix A: Flow Diagram of Study Search and Inclusion Procedure.



Appendix B: Multiple and Overlapping Studies

Table B1
Studies Included in Meta-Analysis with Multiple Samples or Measures of Constructs or Behaviors

Study	Samples[a]	Multiple measures of behavior/constructs[b]	Treatment[c]
1. Anson (2015)	2	2 samples, 1 behavior	Separate studies
2. Chan, Hagger, & Spray (2011)	1	1 behavior, 4 constructs (4 separate measures of autonomous/controlled motivation)	Aggregated[d]
3. Deci & Ryan (1985)	3	3 samples, 3 behaviors	Separate studies
4. Deponte (2004)	3	3 samples, 3 behaviors	Separate studies
5. Hodgins et al. (1996)	2	2 samples, 1 behavior	Separate studies
6. Jones, (2002)	1	1 behavior, 4 constructs (4 separate measures of autonomous/controlled motivation)	Aggregated[d]
7. Kiener (2006)	1	2 behaviors (2 separate meansures of environmental exploration related to career; self-exploration related to career), 1 construct	Aggregated[d]
8. Kwan et al. (2011)	1	1 behavior, 4 constructs (4 separate measures of autonomous/controlled motivation)	Aggregated[d]
9. Lin (2010)	2	1 behavior	Separate studies
10. Ooostlander et al. (2014a,b)	1	1 behavior, 4 constructs (4 separate measures of autonomous/controlled motivation)	Aggregated[d]
11. Øverup (2017) Study 1b[e]	1	2 constructs (2 separate measures of GCOS scales)	Multiple measures aggregated[d]; sample treated separately
12. Øverup (2017) Study 2[e]	1	2 behaviors (2 separate measures of physical aggression, 2 separate measures of psychological aggression)	Multiple measures aggregated[d]; sample treated separately
13. Øverup (2017) Study 3[e]	1	3 behaviors (3 separate measures of behavior physical aggression, psychological aggression, proxy aggression)	Multiple measures aggregated[d]; sample treated separately

14. Rose et al. (2001)	3	1 behavior, 3 constructs (3 separate measures of autonomous/controlled motivation)	Multiple measures aggregated[d], samples treated separately
15. Rose et al. (2005)	1	1 behavior, 3 constructs (3 separate measures of autonomous/controlled motivation)	Aggregated[d]
16. Sutton (2014)	1	1 behavior, 3 constructs (3 separate measures of autonomous/controlled motivation)	Aggregated[d]
17. Van Den Berghe et al. (2013)	1	1 behavior, 9 constructs (9 separate measures of autonomous/controlled motivation)	Aggregated[d]
18. Williams & Deci (1996)	2	1 behavior	Separate studies
19. Wyuts et al. (2015)	2	2 samples, 1 behavior	Separate studies

Note. [a]Number of independent samples reported in study; [b]Number separate behaviors or measures of constructs reported in each sample; [c]How samples were treated in the meta-analysis; [d]Effect sizes aggregated across constructs or behaviors in each sample using Hunter and Schmidt's (2004) formula for aggregating dependent correlations with the correlation for the within-study effect sizes set at .50 (Wampold et al., 1997); [e]Including Øverup (2017) Study 1a, these studies represented four samples and were also treated as separate studies.

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Appendix D: Summary Characteristics

Table D1
Summary Characteristics and Moderator Coding of Studies Included in Meta-Analysis

Study	Constructs available for inclusion	N	Sample age[a]	Sample gender[b]	Moderator coding				
					Gender[c]	Sample type[d]	Sample age[e]	Study design[f]	Study quality
Anson (2013) (Pilot Study)	AUT, CON, AUTMOT	64	20.32 (*SD* = 2.16)	76.56% female, female = 49, male = 12	FEM	STU	YNG	CS	ACC
Anson (2013) (Study 1)	AUT, CON, ID, IJ, BEH	208	37.27 (*SD* = 14.12), range = 18-70	52.88%, female = 110, male = 72	BAL	NST	YNG	CS	ACC
Assadi & Hassanien (2014)	AUT, CON, IMP, AUTMOT, CONMOT, BEH	150	48.11 (*SD* = 16.06)	54% female, female = 81, male = 69	BAL	NST	NA	CS	ACC
Bober & Grolnick (1995)	AUT, CON	49	–	81.63% female, female = 40, male = 9	FEM	STU	NA	NCS	QUE
Bodroža (unpublished)	AUT, CON, IMP	300	20.19 (*SD* = 2.05)	–	NA	STU	YNG	CS	NA
Bodroža & Mirkov (2011)	AUT, CON, IMP	158	22.18 (Range = 19-40)	85.4% female, female = 135, male = 23	FEM	STU	YNG	CS	QUE
Breitborde (unpublished)	AUT, CON, IMP	41	–	–	NA	NST	NA	CS	NA
Breitborde et al. (2014)	AUT, CON, IMP	47	21.91	10.64%, female = 5, male = 42	BAL	NST	YNG	CS	QUE
Brenlla et al. (2013)	AUT, CON, IMP	184	31.78 (*SD*=12.64)	57.61% female, female = 106, male = 78	BAL	NST	YNG	CS	QUE
Chan et al. (2011) Study 2	AUT, CON, IMP, AUTMOT, CONMOT, IM, ID, IJ, EX, BEH	206	24.75 (*SD* = 4.13)	52.43% female, females = 108, males = 98	BAL	NST	YNG	CS	QUE

Chan & Hagger (2012)	AUT, CON, AUTMOT, CONMOT, BEH	533	16.79 (*SD* = 2.80)	49.70% female, female = 265, male = 268	BAL	NST	YNG	NCS	QUE
Chu (2006)	AUT, CON, IMP	51	26.70 (*SD* = 5.4)	45.1% female, female = 22, male = 28	BAL	STU	YNG	CS	ACC
Cooper et al. (2015)	AUT, CON, IMP	360	21.76 (*SD* = 6.55), Range = 18-60	83.16% female, female = 277, male = 83 (23.1%)	BAL	STU	YNG	CS	QUE
Deci & Ryan (1985) Sample 1	AUT, CON, IMP	636	–	–	NA	NA	NA	CS	QUE
Deci & Ryan (1985) Sample 2	AUT, CON, IMP, BEH	73	–	–	NA	NA	NA	CS	QUE
Deci & Ryan (1985) Sample 3	AUT, CON, IMP, BEH	51	–	–	NA	NA	NA	CS	QUE
Deponte (2004) Sample 1	AUT, CON, IMP	702	20.8 (*SD* = 3.9)	69.9% female, 30.1% male	BAL	STU	YNG	CS	QUE
Deponte (2004) Sample 2	AUT, CON, IMP, BEH	108	–	–	NA	STU	NA	CS	QUE
Deponte (2004) Sample 3	AUT, CON, AUTMOT, CONMOT	66	–	–	NA	STU	NA	CS	QUE
Dresner & Grolnick (1996)	AUT, CON, IMP, AUTMOT, CONMOT	50	19.58	100% female	FEM	STU	YNG	CS	QUE
Duriez (2011)	CON, CONMOT	440	18.34 (*SD*=1.68)	85% female, female = 374, male = 66	FEM	STU	YNG	CS	QUE
Ewing (2011)	AUT, CON	99	–	–	NA	NST	NA	CS	ACC
Hagger & Chatzisarantis (2011)	AUT, CON, BEH	40	21.2 (*SD* = 3.47)	70% female, female = 28, male = 12	BAL	STU	YNG	NCS	QUE
Hagger et al. (2015)	AUT, CON, IMP, AUTMOT, CONMOT, BEH	80	22.96 (*SD* = 8.38)	57.50% female, female = 46, male = 34	BAL	STU	YNG	NCS	QUE
Hodgins et al. (1996) Study 1	AUT, CON, AUTMOT, BEH	67	18.3, Range = 17-21	82.09% female, 55 = female, 12 = male	FEM	STU	YNG	NCS	QUE
Hodgins et al. (1996) Study 2	AUT, CON, BEH	86	22.4, Range = 8-44	60.47% female, female = 52, male = 34	BAL	STU	MIX	NCS	QUE
Hoet (2018)	AUT, CON, IMP	276	18.70 (*SD* = 2.89), Range = 17-53	78.62% female, females = 217,	FEM	STU	MIX	NCS	ACC

				male = 58, unreported = 1					
Jerkovic et al. (2017)	AUT, CON, BEH	438	19.62 (*SD* = 0.83)	62.1% female, female = 272, male = 166	BAL	STU	YNG	CS	QUE
Jones (2002)	AUT, IM, ID, IJ, EX	117	Range = 19-65	–	NA	NST	MIX	NCS	ACC
Kiener (2006)	AUT, CON, IMP, BEH	230	Range = 18-26	48.26% female, female = 104, male = 119	BAL	STU	YNG	CS	QUE
King & Gurland (2007)	AUT, CON	90	–	63.33% female, female = 57, male = 33	BAL	STU	NA	NCS	QUE
Knee & Zuckerman (1996)	AUT, CON	78	–	62.80% female, female = 49, male = 29	BAL	STU	NA	NCS	QUE
Knee & Zuckerman (1998)	AUT, CON	274	–	66.1% female, female = 181, male = 81, not reported = 12	BAL	STU	NA	NCS	QUE
Koestner & Zuckerman (1994)	AUT, CON, IMP	166	–	58.43%, female = 97, male = 69	BAL	STU	NA	NCS	QUE
Koestner & Losier (1996)	AUT, CON, IMP, AUTMOT	115	–	74.78% female, female = 86, male = 29	FEM	STU	NA	NCS	ACC
Kwan et al. (2011)	AUT, CON, IMP, IM, ID, IJ, EX, BEH	102	18.23 (Range = 18-27)	58.82% female, female = 60, male = 44	BAL	STU	YNG	NCS	QUE
Lam & Gurland (2008)	AUT, CON, AUTMOT	166	20-39 (35.0%), 40-49 (26.3%), 50-59 (30.0%), 60+ (9%)	79.4% female, female = 132, male = 34	FEM	NST	MIX	CS	QUE
Leigh (2010)	AUT, CON, IMP, BEH	199	22.21 (*SD* = 6.12)	50.25% female, female = 100, male = 99	BAL	STU	YNG	CS	ACC
Leone (1995)	AUT, CON, IMP	139	Under 30 (37.9%), 31-50 (50.4%), 50+ (11.7%)	81.29% female, female = 113, male = 26	FEM	NST	MIX	CS	ACC

Lin (2010) Sample 1	AUT, CON, IMP	83	22.94 (*SD* = 7.4)	87.95% female, female = 73, male = 10	FEM	STU	YNG	NCS	ACC
Lin (2010) Sample 2	AUT, CON, IMP	99	36.55 (*SD* = 2.39)	67.68% female, female = 67, male = 22	BAL	NST	YNG	NCS	ACC
Luyckx et al. (2007)	AUT, CON, IMP	263	19.14 (*SD* = 0.95)	72.62% female, female = 191, male = 72	BAL	STU	YNG	CS	ACC
Luyckx et al. (2010)	AUT, CON, IMP	399	18.67 (*SD* = 0.63)	78.95% female, female = 315, male = 84	FEM	STU	YNG	CS	ACC
Mekler et al. (2017)	AUT, AUTMOT	237	32.80 (*SD* = 12.21)	75.11% female, female = 178, male = 84, not reported = 11	FEM	NST	YNG	NCS	QUE
Olesen (2011)	AUT, IMP	1181	21.80 (*SD*=4.36)	59.01% female, female = 697, male = 484	BAL	STU	YNG	CS	QUE
Oostlander et al. (2014a,b)	AUT, CON, IM, ID, IJ, EX	1979	64.08 (*SD* = 11.76)	59.53% female, female = 1178, male = 736, not reported = 65	BAL	NST	OLD	CS	QUE
Ortlieb (2013)	AUT, CON, IMP	106	18-30 (30.1%), 31-48 (54.4%), 48+ (15.5%)	4.72% female, female = 5, male = 101	BAL	NST	MIX	CS	ACC
Øverup (2017) Study 1a	AUT, CON, IMP	572	22.85 (*SD* = 5.13)	88.29% female, female = 505, male = 67	FEM	STU	YNG	CS	ACC
Øverup (2017) Study 1b	AUT, CON, IMP	265	23.04 (*SD* = 6.09)	91.19% female, female = 239, male = 26	FEM	STU	YNG	CS	ACC
Øverup (2017) Study 2	AUT, CON, IMP, BEH	324	23.40 (*SD* = 5.89)	83.64% female, female = 271, male = 44	FEM	STU	YNG	CS	ACC
Øverup (2017) Study 3	AUT, CON, IMP, BEH	274	23.62 (*SD* = 5.06)	85.40% female, female = 234, male = 39	FEM	STU	YNG	CS	ACC

Patterson (2017)	AUT, CON, IMP, BEH	265	38, Range = 24-67	57.74% female, female = 153, male = 109	BAL	NST	MIX	CS	ACC
Pullins (2000)	AUT, CON	154	–	–	NA	STU	NA	NCS	QUE
Ramezanzade & Arabnarmi (2017)	AUT, CON, IMP	230	–	47.83% female, female = 110, male = 120	BAL	STU	NA	CS	QUE
Reeve (1998)	AUT, AUTMOT	142	–	77.46% female, female = 110, male = 32	FEM	STU	NA	CS	QUE
Reeve et al. (2018)	AUT, CON, AUTMOT, CONMOT	42	33.7, Range = 25-52	59.52% female, female = 25, male = 17	BAL	NST	MIX	NCS	QUE
Rose et al. (2001) Sample 1	AUT, IM, ID, IJ, EX	294	34.9 (*SD* = 11.4)	60.20% female, female = 177, male = 117, not reported = 11	BAL	NST	YNG	NCS	QUE
Rose et al. (2001) Sample 2	AUT, CON, IMP, AUTMOT	289	37.3 (*SD* = 11.2)	57.79% female, female = 167, male = 121, not reported = 1	BAL	NST	YNG	CS	QUE
Rose et al. (2001) Sample 3	AUT, IMP, BEH	592	35.8 (*SD* = 11.3), Range = 16-66)	55.57% female, female = 329, male = 222, not reported = 12	BAL	NST	YNG	NCS	QUE
Rose et al. (2005)	AUT, CON, IMP, IM, ID, IJ, EX	184	Males: 33.99 (*SD* = 13.86); Females: 28.85 (*SD* = 11.21)	54.89% female, female = 101, male = 83	BAL	NST	YNG	CS	QUE
Sadabadi et al. (2011)	AUT, CON, IMP, BEH	60	30 and older	50.00% female, female = 30, male = 30	BAL	NST	YNG	CS	QUE
Schaubroeck & Williams (1993)	AUT, CON, IMP	98	Male median = 21; Female median = 18	30.61% female, female = 30, male = 68	BAL	STU	YNG	NCS	QUE
Sen & Dag (2016)	AUT, CON, IMP	363	21.39 (*SD* = 3.63)	65.84% female, female = 239 male = 124	BAL	STU	YNG	CS	QUE

Sharafi et al. (2006)	AUT, CON, IMP	290	29.2	57.93% female, female = 168, male = 122	BAL	NA	YNG	CS	QUE
Soenens et al. (2005)	AUT, CON, IMP	367	18, Range = 17-25	80.11% female, female = 294, male = 73 male	FEM	STU	YNG	CS	QUE
Solberg et al. (2013)	AUT, CON, IMP	118	74.2 (*SD* = 4.5)	67.80% females, female = 80, male = 38	BAL	NST	OLD	NCS	ACC
Stevens et al. (2015)	AUT, CON, IMP	76	25.09 (*SD* = 5.22)	76.32% female, female = 58, male = 18	FEM	STU	YNG	CS	QUE
Sutton (2014)	AUT, CON, IM, ID, IJ, EX	57	36.9 (*SD* = 8.8)	30.10% female, female = 31, male = 72	BAL	NST	YNG	CS	ACC
Taylor et al. (2008)	AUT, AUTMOT	204	34.34 (*SD* = 11.09), Range = 22–60	46.57% female, female = 95, male = 100, not reported = 9	BAL	NST	YNG	CS	QUE
Thill et al. (1998)	AUT, AUTMOT	182	–	100% male	BAL	STU	NA	NCS	QUE
Vallerand et al. (1987)	AUT, CON, IMP	165	18.55 (*SD* = 1.63)	–	NA	STU	YNG	CS	QUE
Van Den Berghe et al. (2013)	AUT, CON, BEH	79	36.1 (*SD* = 11.0), Range = 21-61	48.10% female, female = 38, male = 41	BAL	NST	YNG	CS	QUE
Wheeler (1984)	AUT, CON	95	20.7 (*SD* = 0.25)	74.74% female, female = 71, male = 24	FEM	STU	YNG	NCS	QUE
Wheeler et al. (2003)	AUT, CON, IMP	256	19.4 (*SD* = 1.8)	100% female	FEM	STU	YNG	CS	QUE
Williams & Deci (1996) Sample 1	AUT, CON, IMP, AUTMOT, CONMOT	181	–	–	NA	STU	NA	NCS	QUE
Williams & Deci (1996) Sample 2	AUT, CON, IMP, AUTMOT, CONMOT	72	–	–	NA	STU	NA	NCS	QUE
Wu & Hwang (2000)	AUT, CON, IMP	353	33.6 (*SD* = 9.05)	46.5% female, female = 164, male = 189	BAL	NST	YNG	CS	ACC
Wuyts et al. (2015) Study 1	AUT, CON	502	Females: 42 (*SD* = 4.61), Range = 27–56; Males:	50.60% female, female = 254, male = 248	BAL	NST	OLD	CS	QUE

Wuyts et al. (2015) Study 2	AUT, CON	318	45 (*SD* = 5.26), Range = 27–59 40 (*SD* = 4.57), Range = 25–60	52.52% female, female = 167, male = 151	BAL	NST	MIX	NCS	QUE
Ye et al. (2014)	AUT, CON, IMP	120	32.69 (*SD* = 6.803)	36.67% female, female = 44, male = 76	BAL	NST	YNG	CS	QUE
Young et al. (2016)	AUT, CON, IMP	354	23.9	88.98% female, female = 315, male = 39	FEM	STU	YNG	CS	QUE
Zhang (2014)	AUT, CON, IMP	406	25-44 (60%); under 44 (85.4%)	51.97% female, female = 211, male = 195	BAL	NST	YNG	NCS	QUE
Zuckerman et al. (1998)	AUT, CON, IMP, BEH	119	–	48.74% female, female = 58, male = 61	BAL	STU	NA	CS	QUE

Note. [a]Mean age of sample expressed in years and variability statistics (where reported); [b]Gender distribution of sample expressed as percentage of females with exact frequency of female and male participants; [c]Sample gender moderator coding coded as female only or predominantly female samples (≥75% female) and studies on mixed gender samples (comprised between 25% and 74% female); [d]Sample type moderator coded as student samples and non-student samples; [e]Sample age moderator coded as younger samples (aged 40 years or younger with low variability), samples comprising older individuals (aged >40 years with low variability) and samples of 'mixed' age with high variability; [f]Study design moderator coded as cross-sectional and non-cross-sectional (experimental, intervention, and longitudinal) studies. AUT = Autonomy causality orientation; CON = Control causality orientation; IMP = Impersonal causality orientation; IM = Intrinsic motivation; ID = Identified behavioral regulation; IJ = Introjected behavioral regulation; EX = External behavioral regulation; AUTMOT = Autonomous motivation; CONMOT = Controlled motivation; BEH = Behavioral measure; *SD* = Standard deviation; FEM = Predominantly female samples; BAL = Samples of 'balanced' gender distribution; STU = Studies on student samples; NST = Studies on non-student samples; YNG = Studies on younger samples with low variability; OLD = Studies on older samples with low variability; MIX = Samples with high variability in participant age; CS = Studies adopting cross-sectional studies; NCS = Studies adopting non-cross-sectional designs; ACC = Studies of 'acceptable' quality; QUE = Studies of 'questionable' quality; NA = Study not assigned to a moderator category.

Appendix E: Study Quality Checklist

Quality Assessment Checklist for Survey Studies in Psychology (Q-SSP)

Study:

Research domain	Quality item	Yes	No	Not stated clearly	N/A
Introduction (Rationale)	1. Was the problem or phenomenon under investigation defined, described, and justified?				
Introduction (Rationale)	2. Was the population under investigation defined, described, and justified?				
Introduction (Rationale)	3. Was there a connection between the hypotheses or aims or research questions, and the background research?				
Introduction (Variables)	4. Were operational definitions of all study variables provided?				
Participants (Sampling)	5. Were participant inclusion criteria stated?				
Participants (Sampling)	6. Was the participant recruitment strategy described?				
Participants (Sampling)	7. Was a justification/ rationale for the sample size provided?				
Data (Collection)	8. Was the attrition rate provided? (applies to cross-sectional and prospective studies)				
Data (Analyses)	9. Was a method of treating attrition provided? (applies to cross-sectional and prospective studies)				
Data (Analyses)	10. Were the data analysis techniques justified (i.e., was the link between hypotheses/ aims / research questions and data analyses explained)?				
Data (Measures)	11. Were the measures provided in the report (or in a supplement) in full?				
Data (Measures)	12. Was evidence provided for the validity of the measures (or instrument) used?				
Data (Collection)	13. Was information provided about the person(s) who collected the data (e.g., training, expertise, other demographic characteristics)?				
Data (Collection)	14. Was information provided about the context (e.g., place) of data collection?				
Data (Collection)	15. Was information provided about the duration (or start and end date) of data collection?				
Data (Results)	16. Was the study sample described in terms of key demographic characteristics?				
Data (Discussion)	17. Was discussion of findings confined to the population from which the sample was drawn?				
Ethics	18. Were participants asked to provide (informed) consent or assent?				

Ethics	19. Were participants debriefed at the end of data collection?				
Ethics	20. Were funding sources or conflicts of interest disclosed?				

Note. Checklist items taken from Protogerou, C., & Hagger, M. S. (2020). A checklist to assess the quality of survey studies in psychology methods in psychology. *Methods in Psychology*, *3*, 100031. https://doi.org/10.1016/j.metip.2020.100031

Appendix F: Forest Plots of Studies Included in Meta-Analysis for Effects Among Causality Orientations, Forms of Motivation from Self-Determination Theory, and Behavior

Figure F1. Forest plot of relations between autonomy causality orientation and autonomous motivation for studies included in the meta-analysis with summary effect.

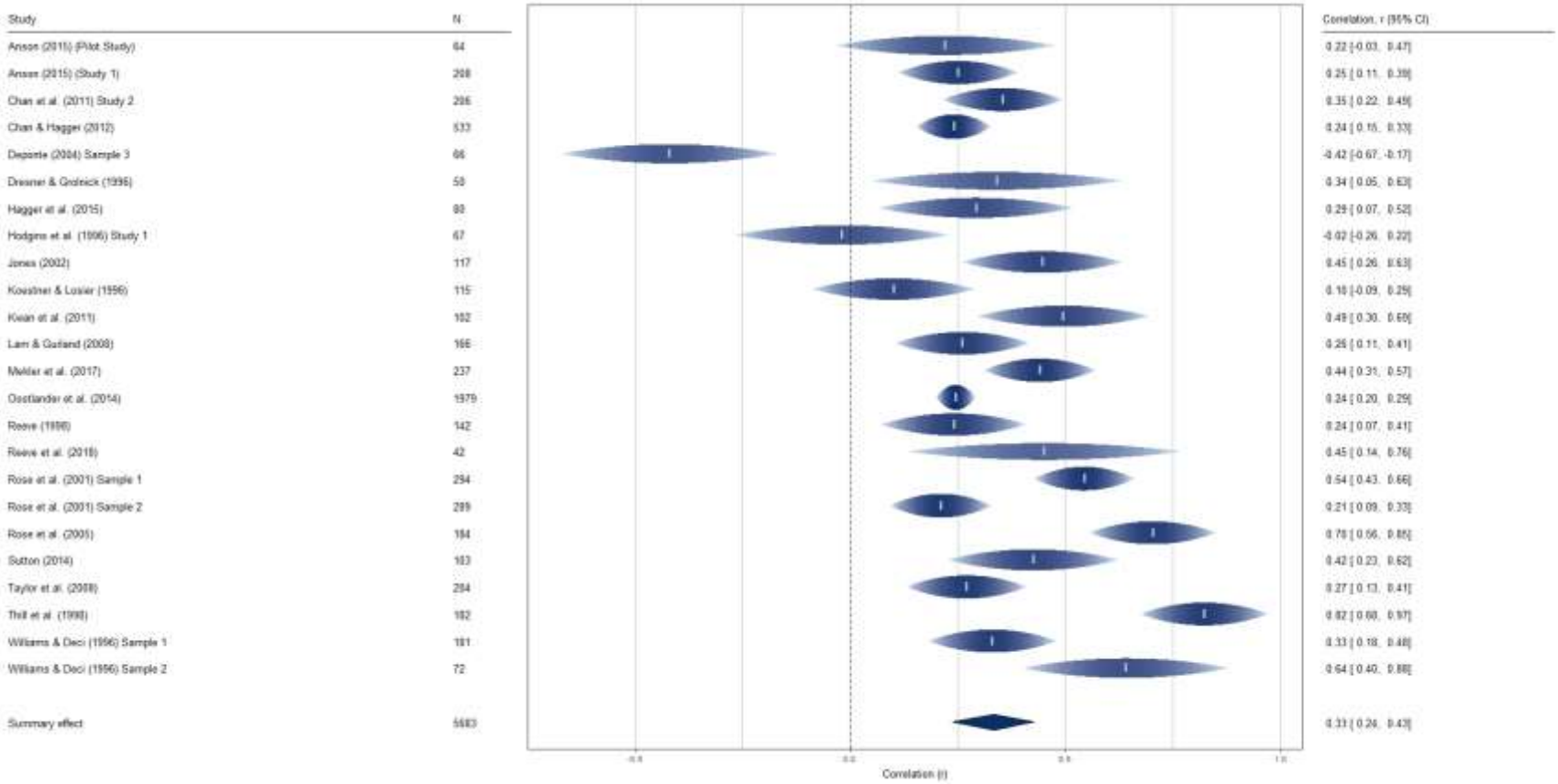


Figure F2. Forest plot of relations between autonomy causality orientation and behavior for studies included in the meta-analysis with summary effect.

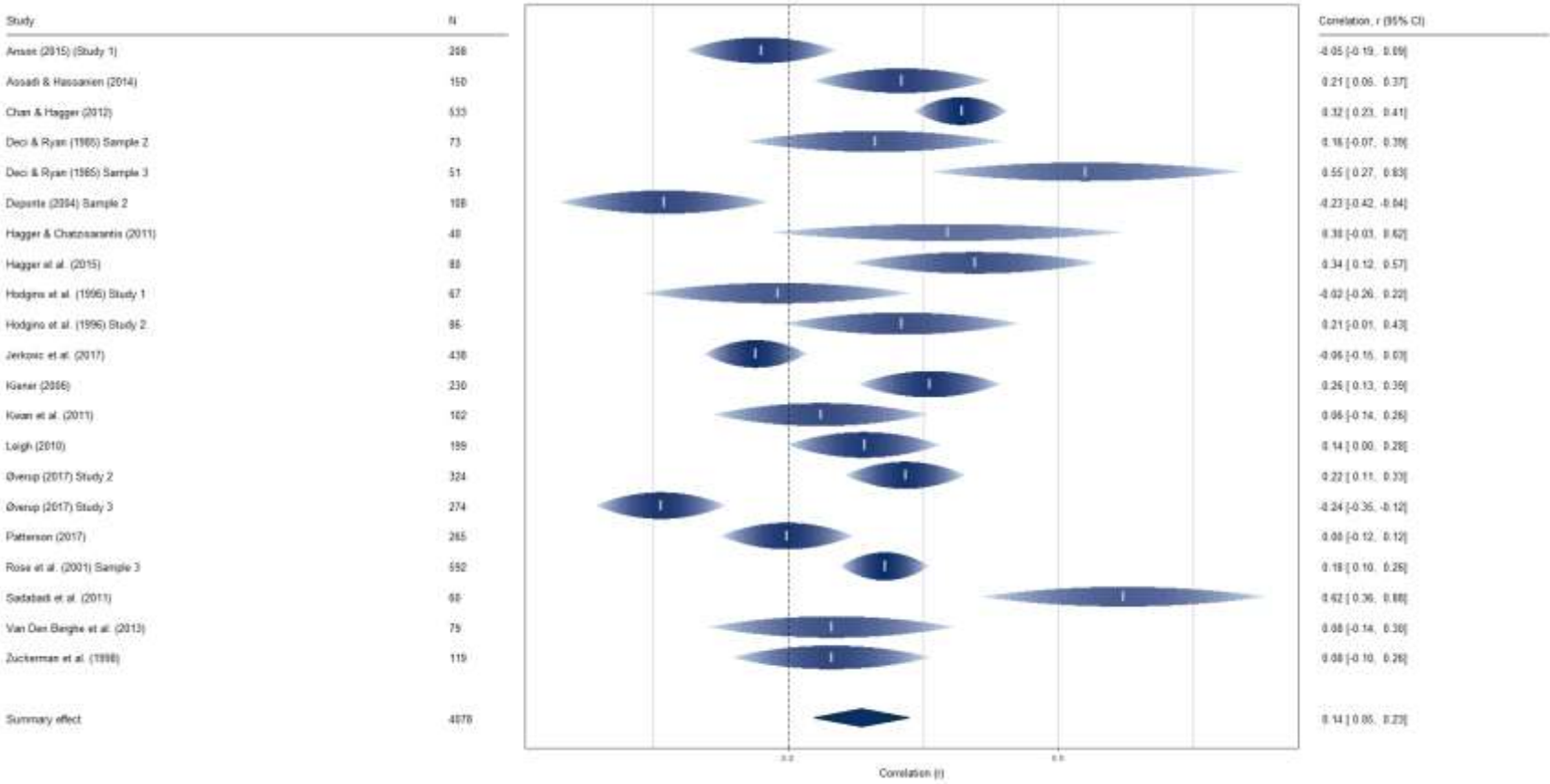


Figure F3. Forest plot of relations between autonomy causality orientation and controlled motivation for studies included in the meta-analysis with summary effect.

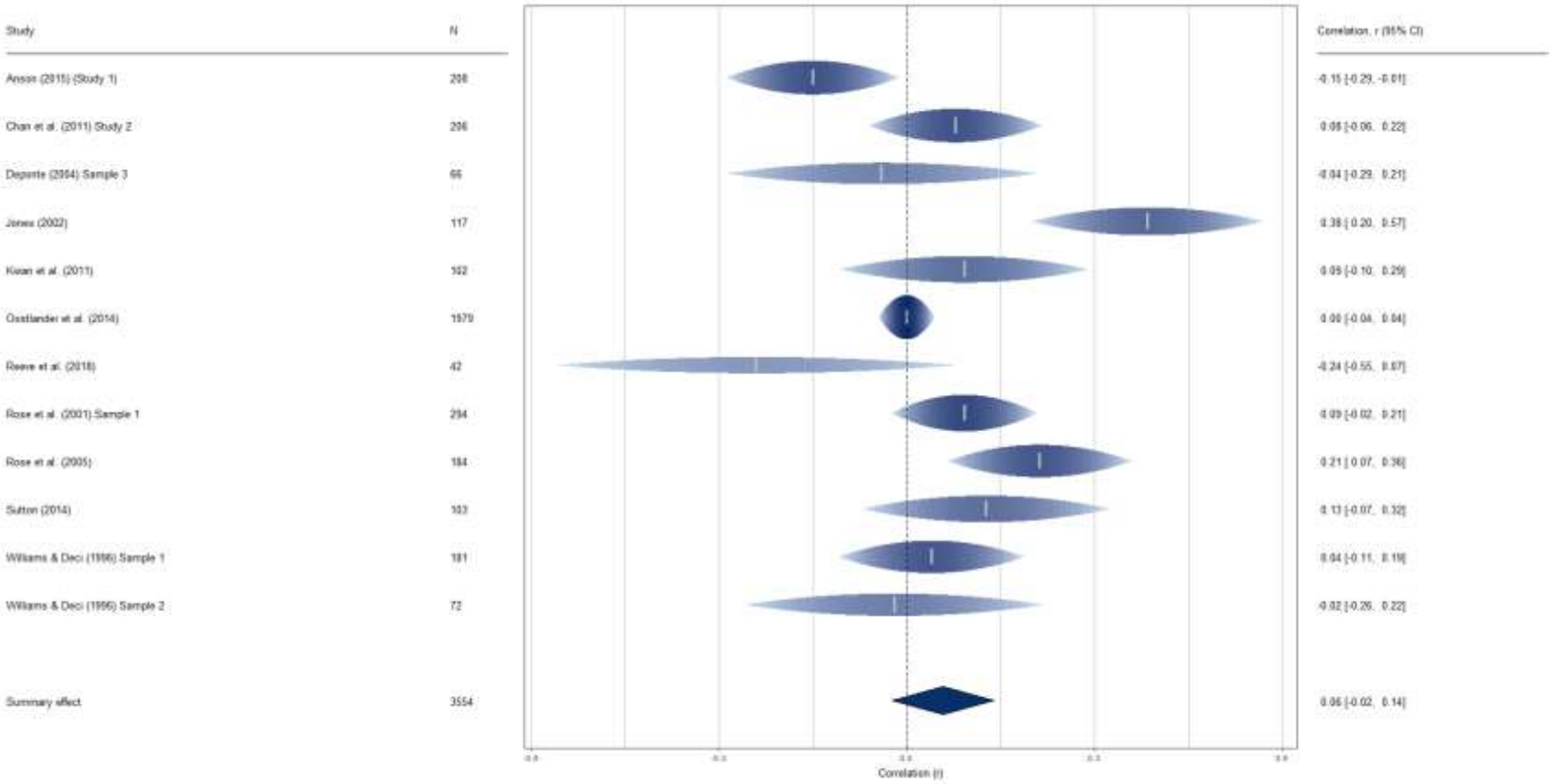


Figure F4. Forest plot of relations between autonomy causality orientation and external regulation for studies included in the meta-analysis with summary effect.

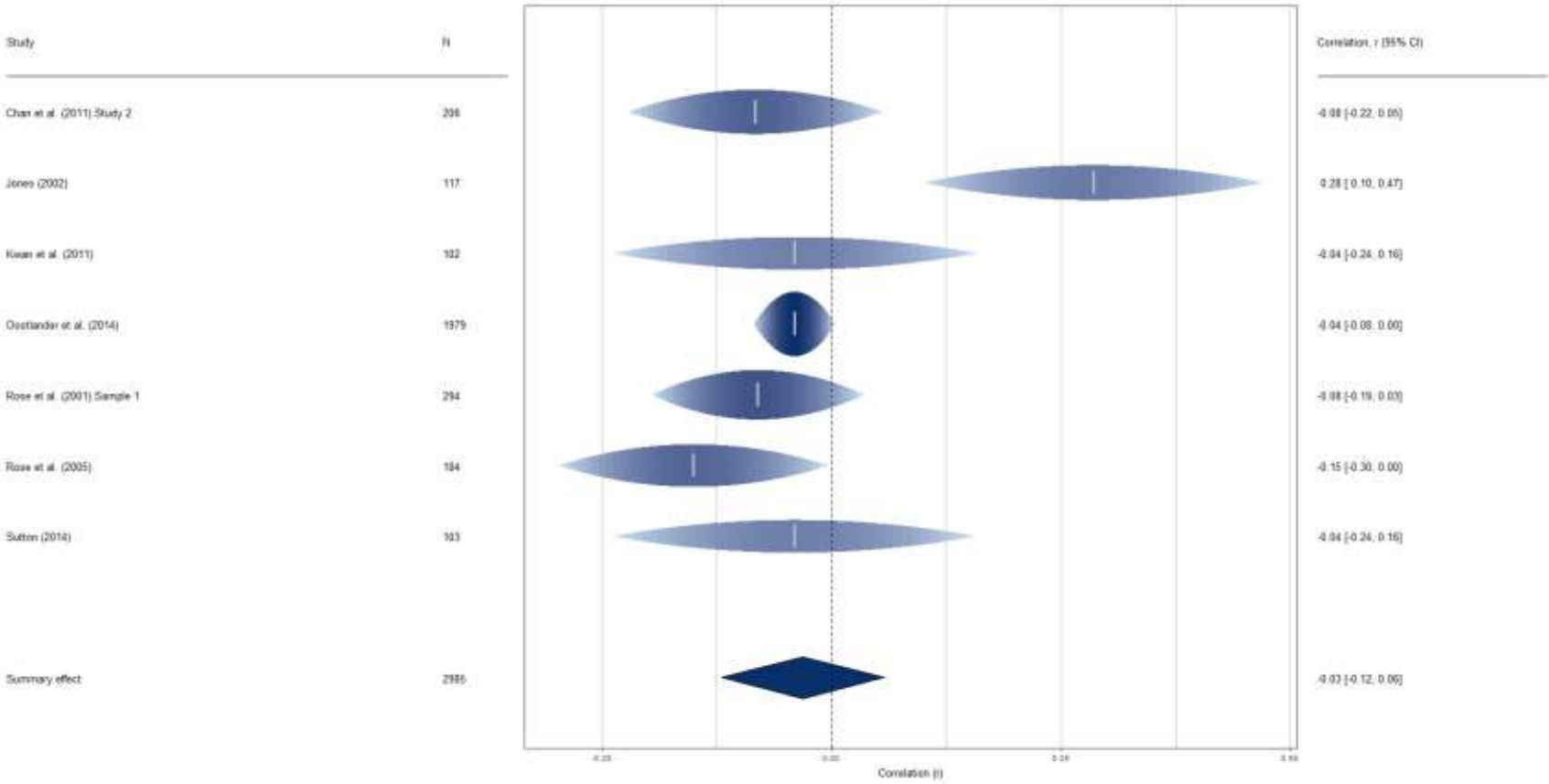


Figure F5. Forest plot of relations between autonomy causality orientation and identified regulation for studies included in the meta-analysis with summary effect.

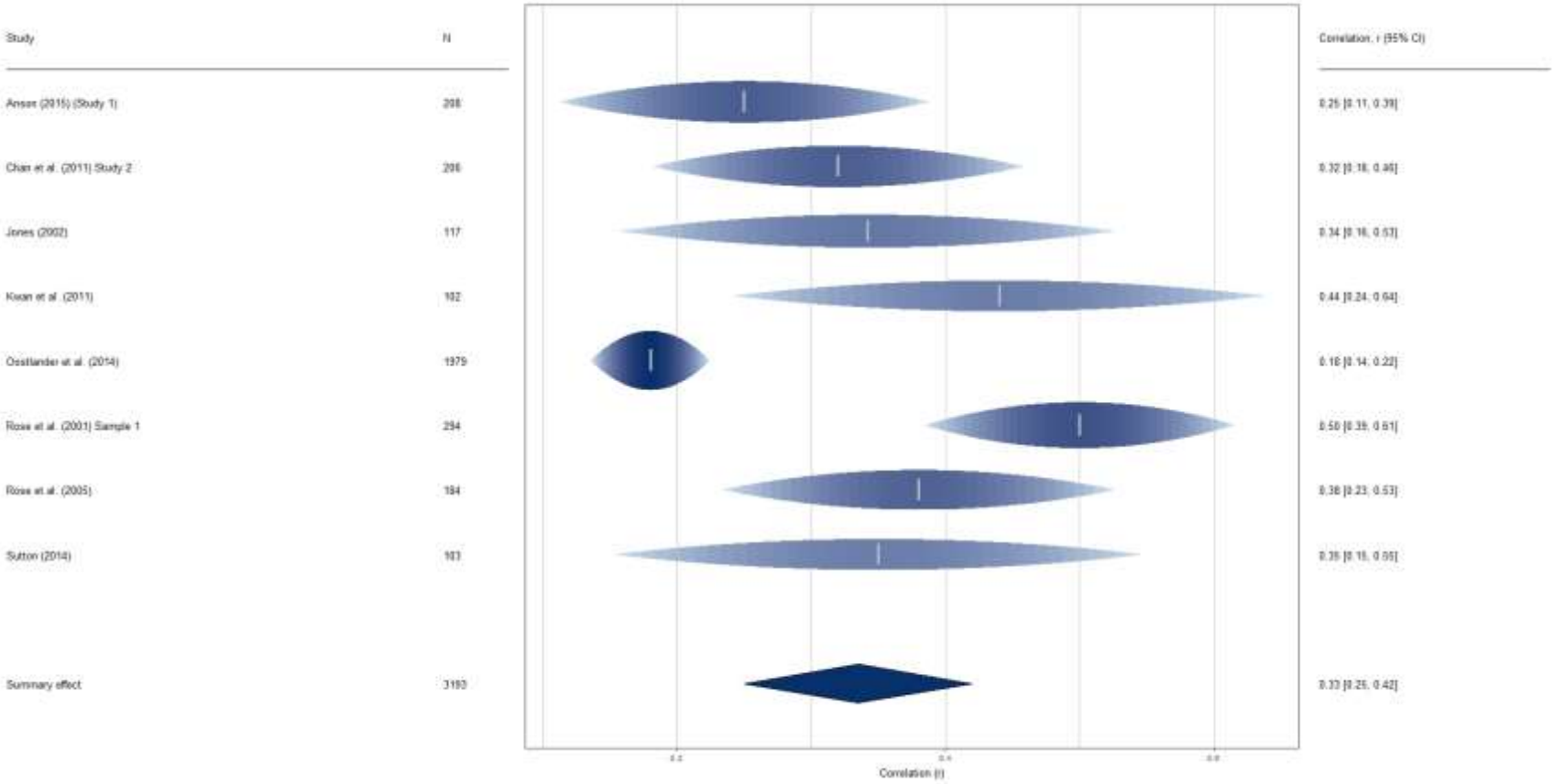


Figure F6. Forest plot of relations between autonomy causality orientation and introjected regulation for studies included in the meta-analysis with summary effect.

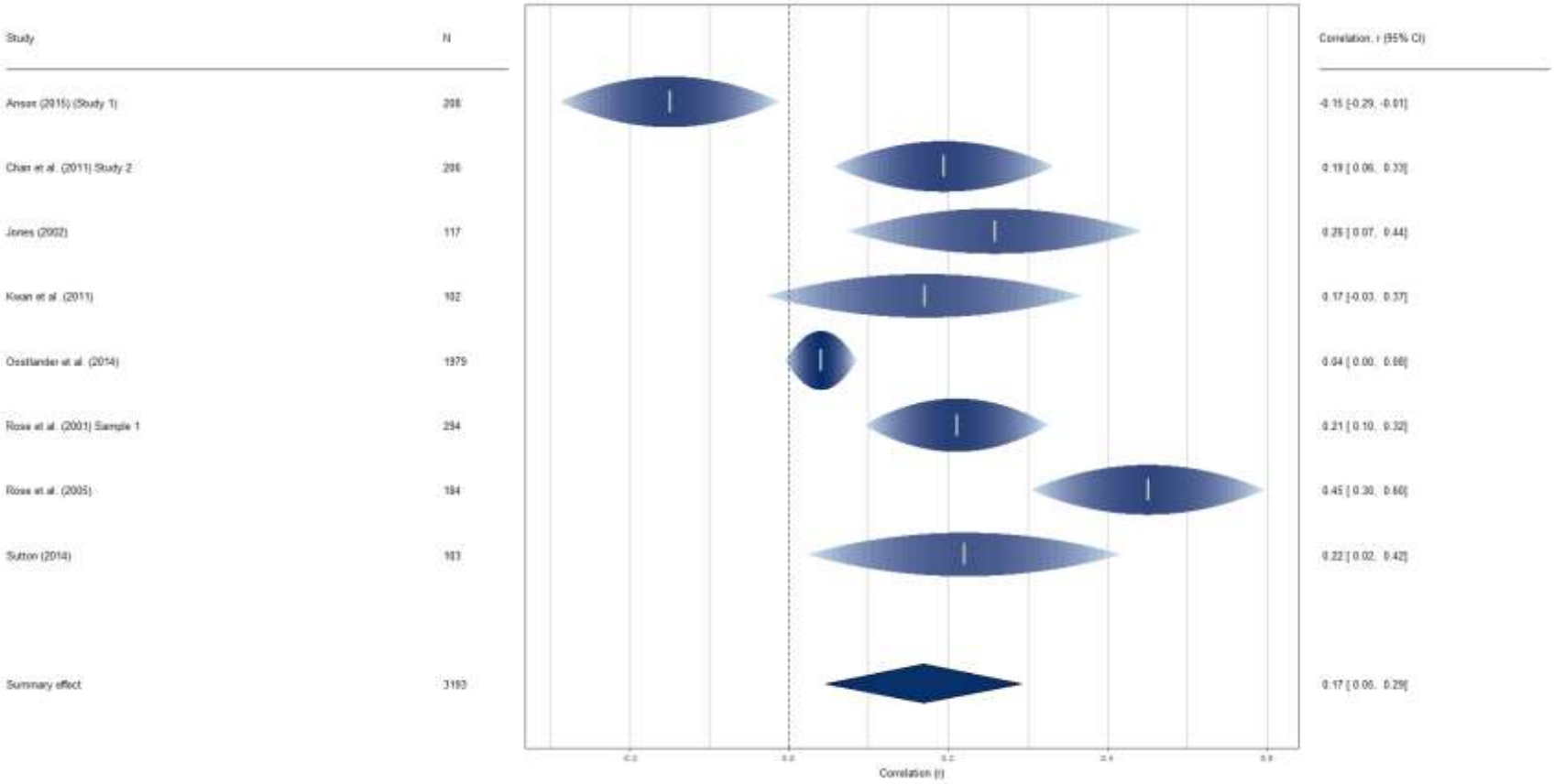


Figure F7. Forest plot of relations between autonomy causality orientation and intrinsic motivation for studies included in the meta-analysis with summary effect.

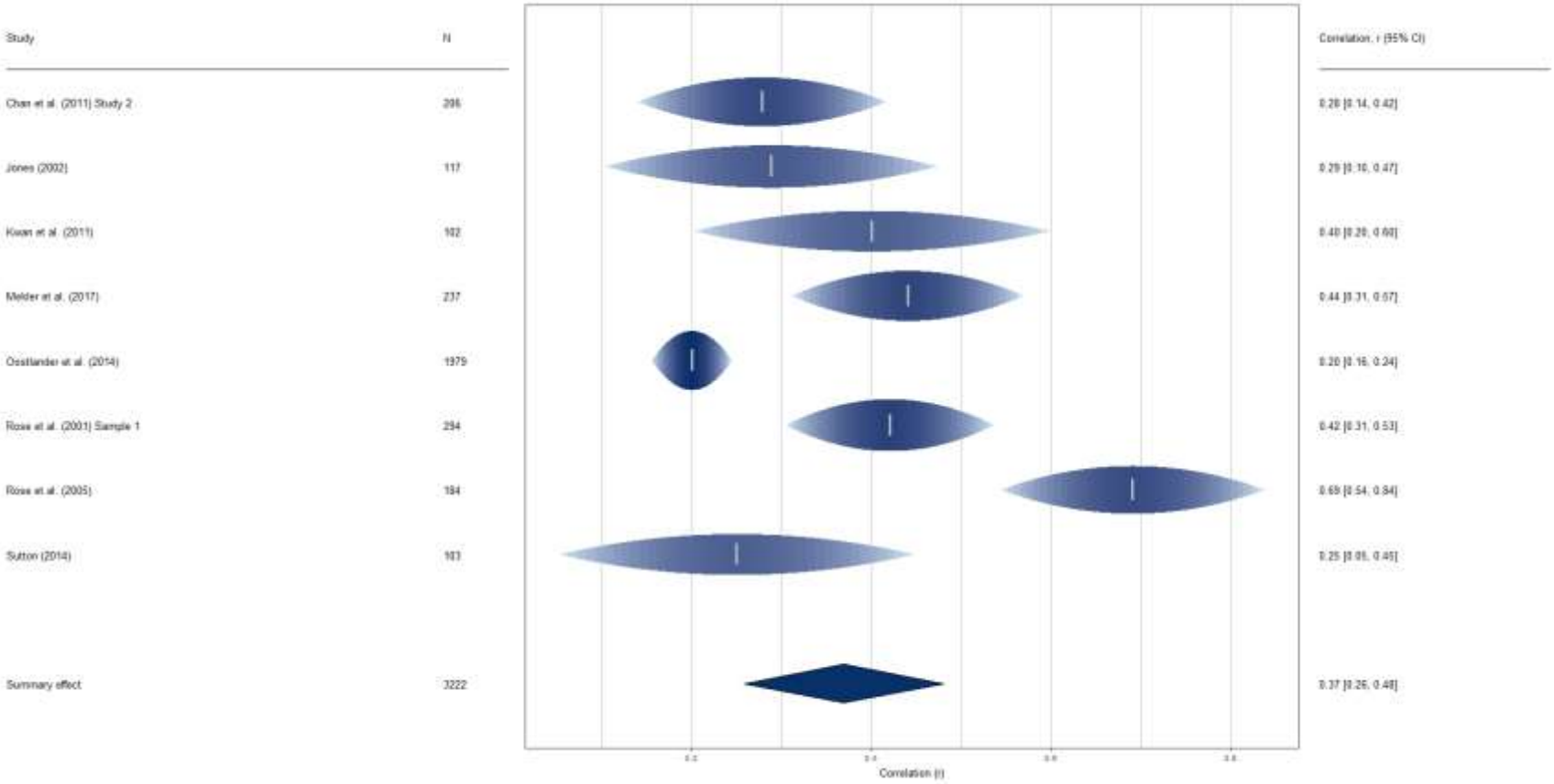


Figure F8. Forest plot of relations between autonomous motivation and behavior for studies included in the meta-analysis with summary effect.

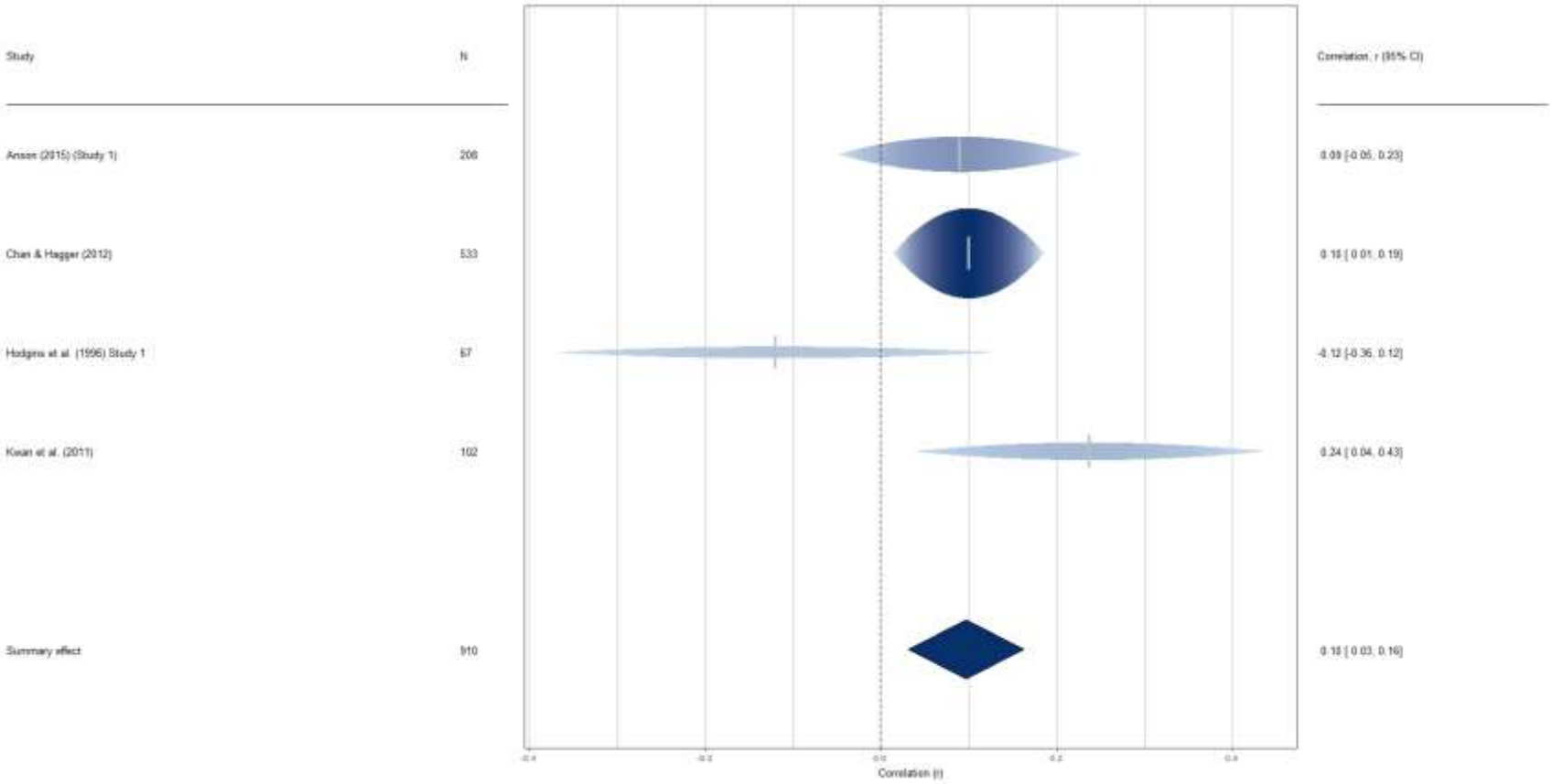


Figure F9. Forest plot of relations between autonomous motivation and controlled motivation for studies included in the meta-analysis with summary effect.

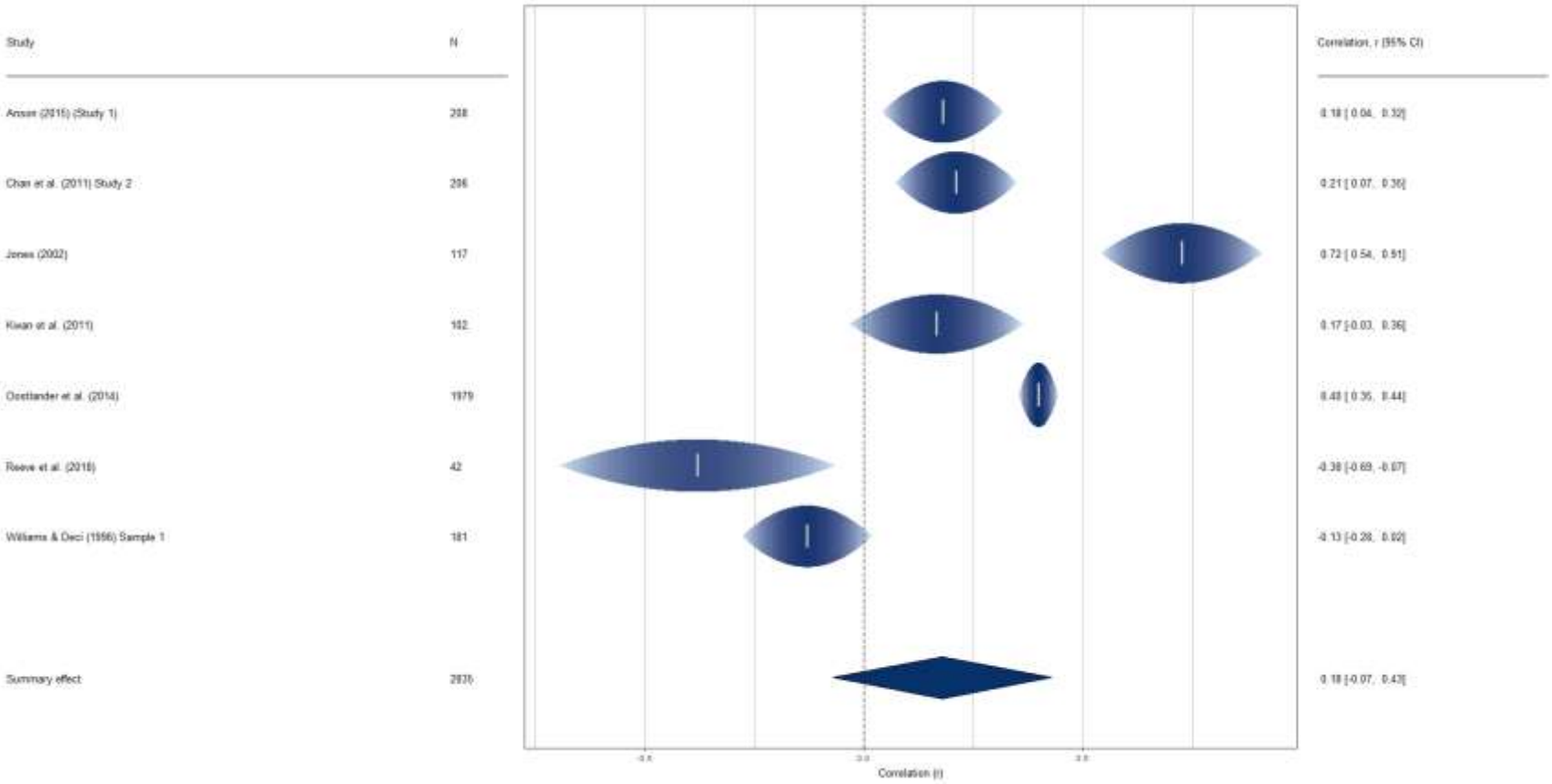


Figure F10. Forest plot of relations between control causality orientation and autonomous motivation for studies included in the meta-analysis with summary effect.

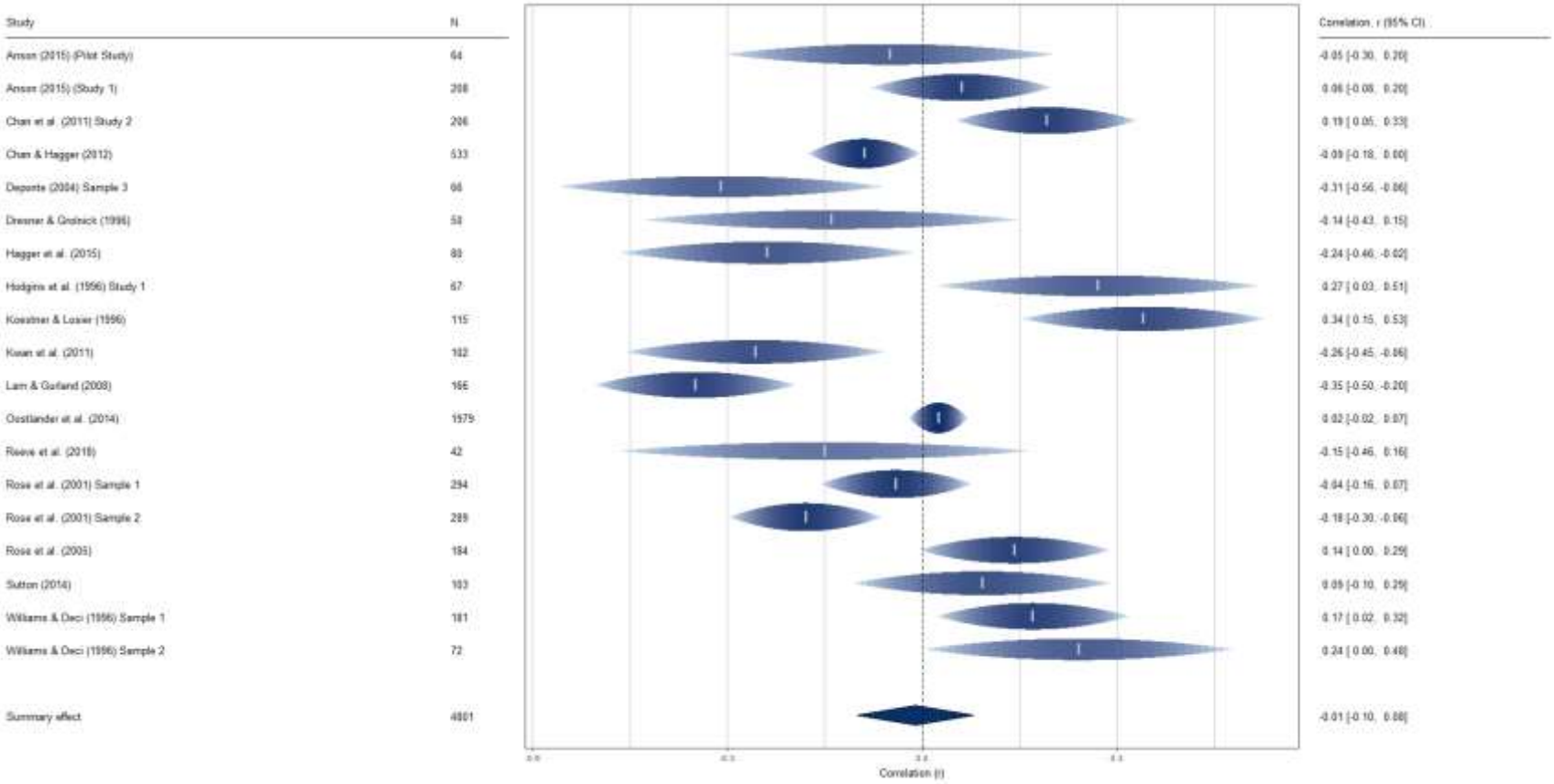


Figure F11. Forest plot of relations between control causality orientation and behavior for studies included in the meta-analysis with summary effect.

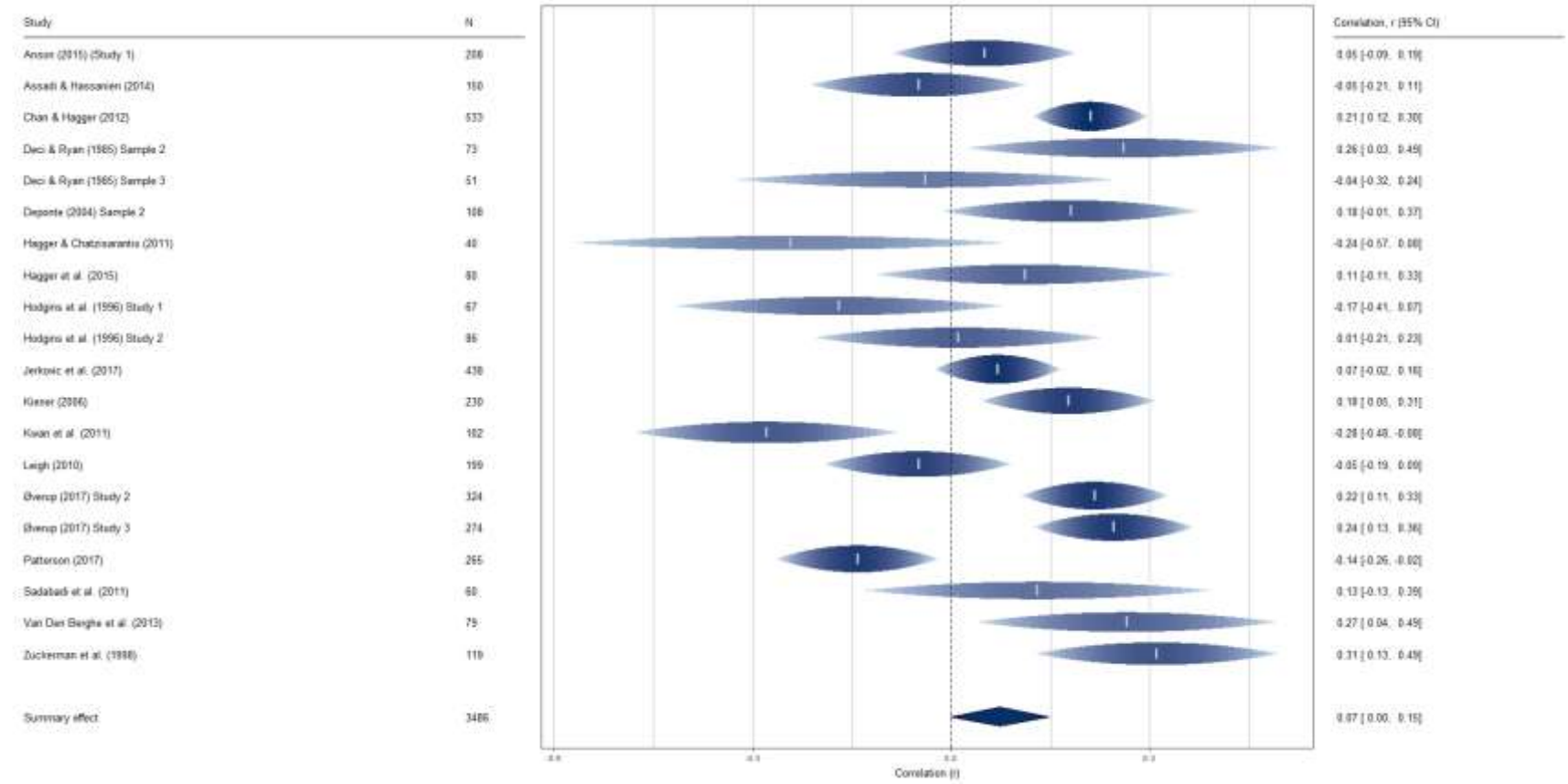


Figure F12. Forest plot of relations between control causality orientation and controlled motivation for studies included in the meta-analysis with summary effect.

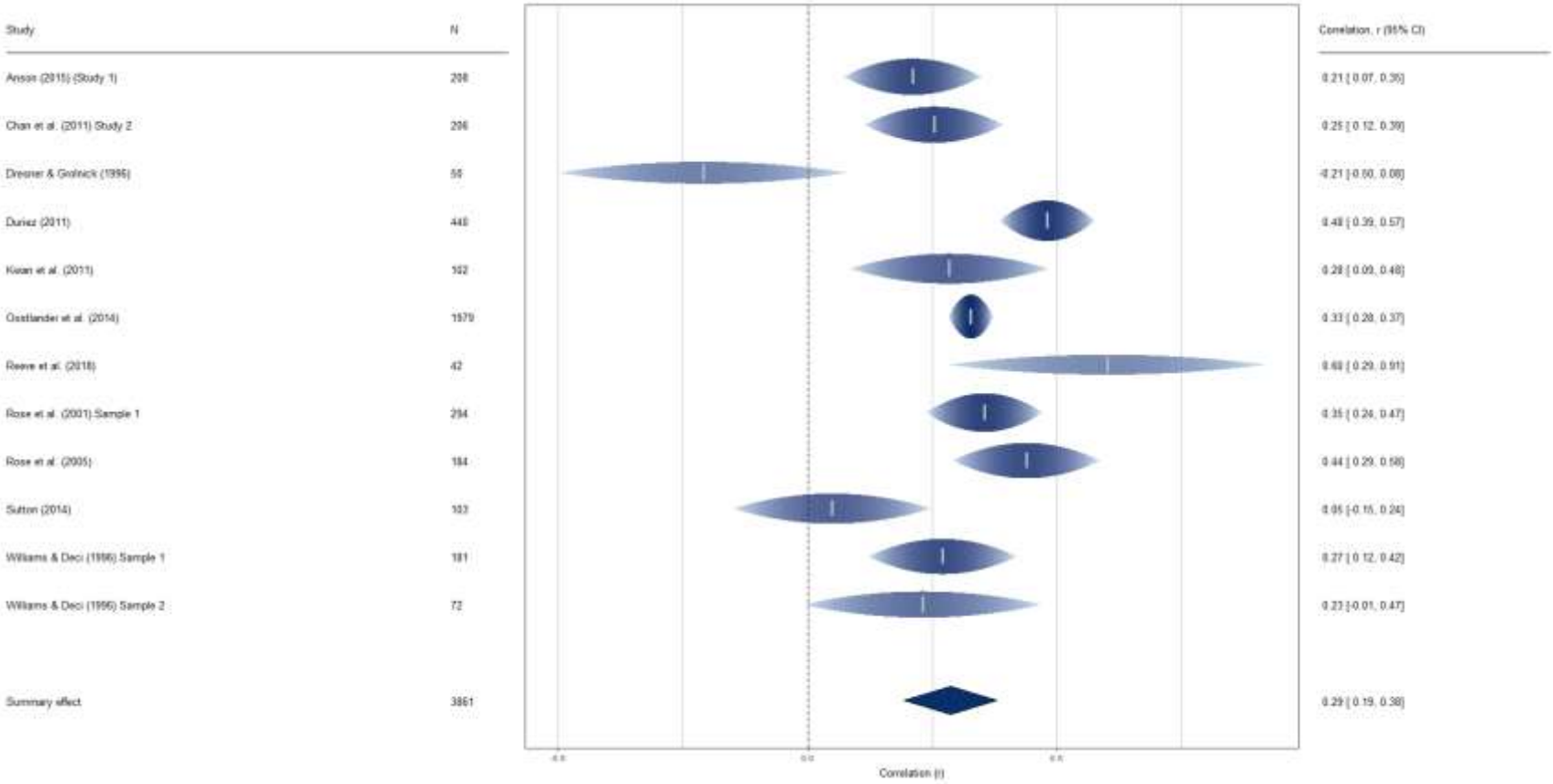


Figure F13. Forest plot of relations between control causality orientation and external regulation for studies included in the meta-analysis with summary effect.

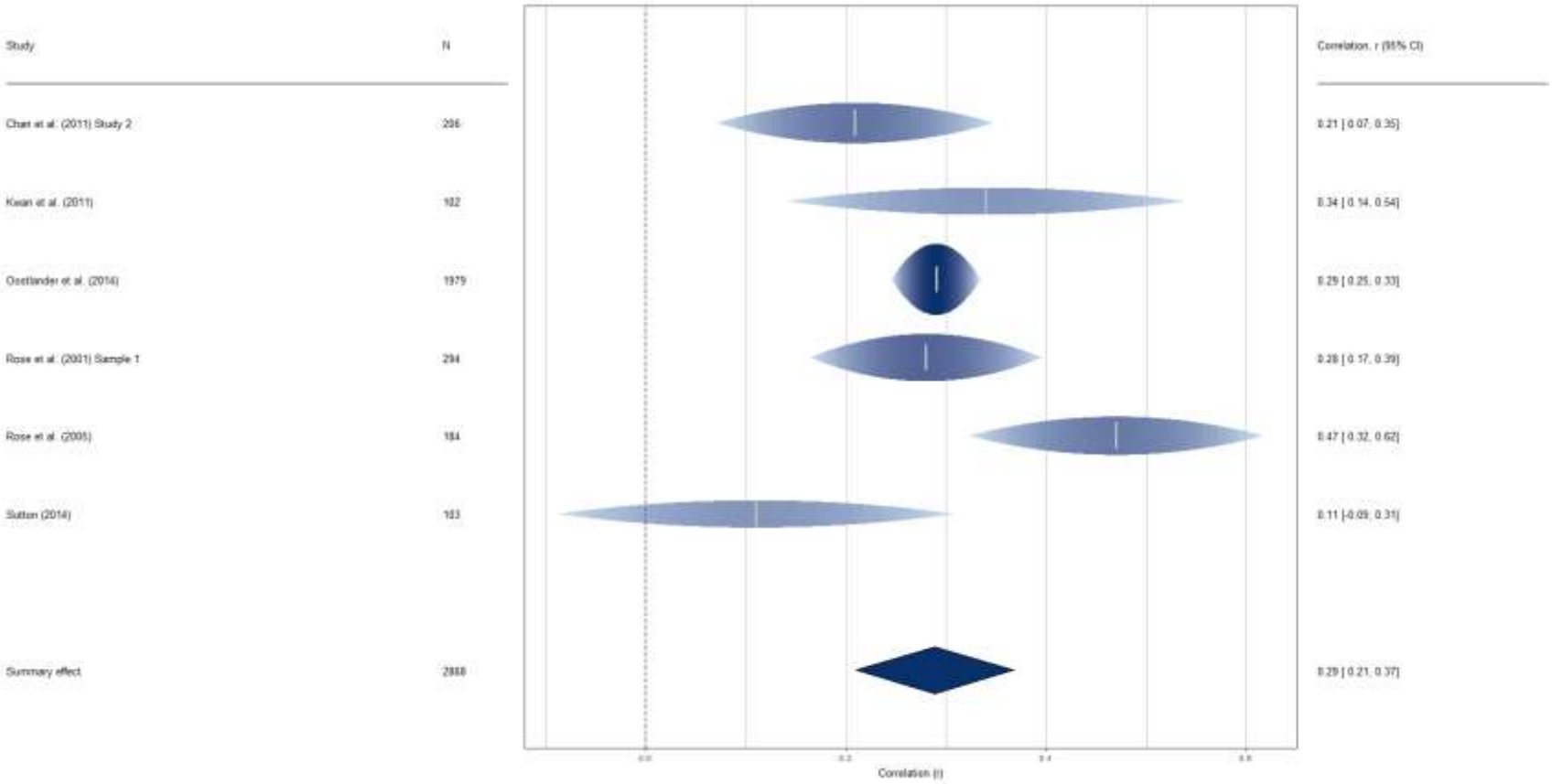


Figure F14. Forest plot of relations between control causality orientation and identified regulation for studies included in the meta-analysis with summary effect.

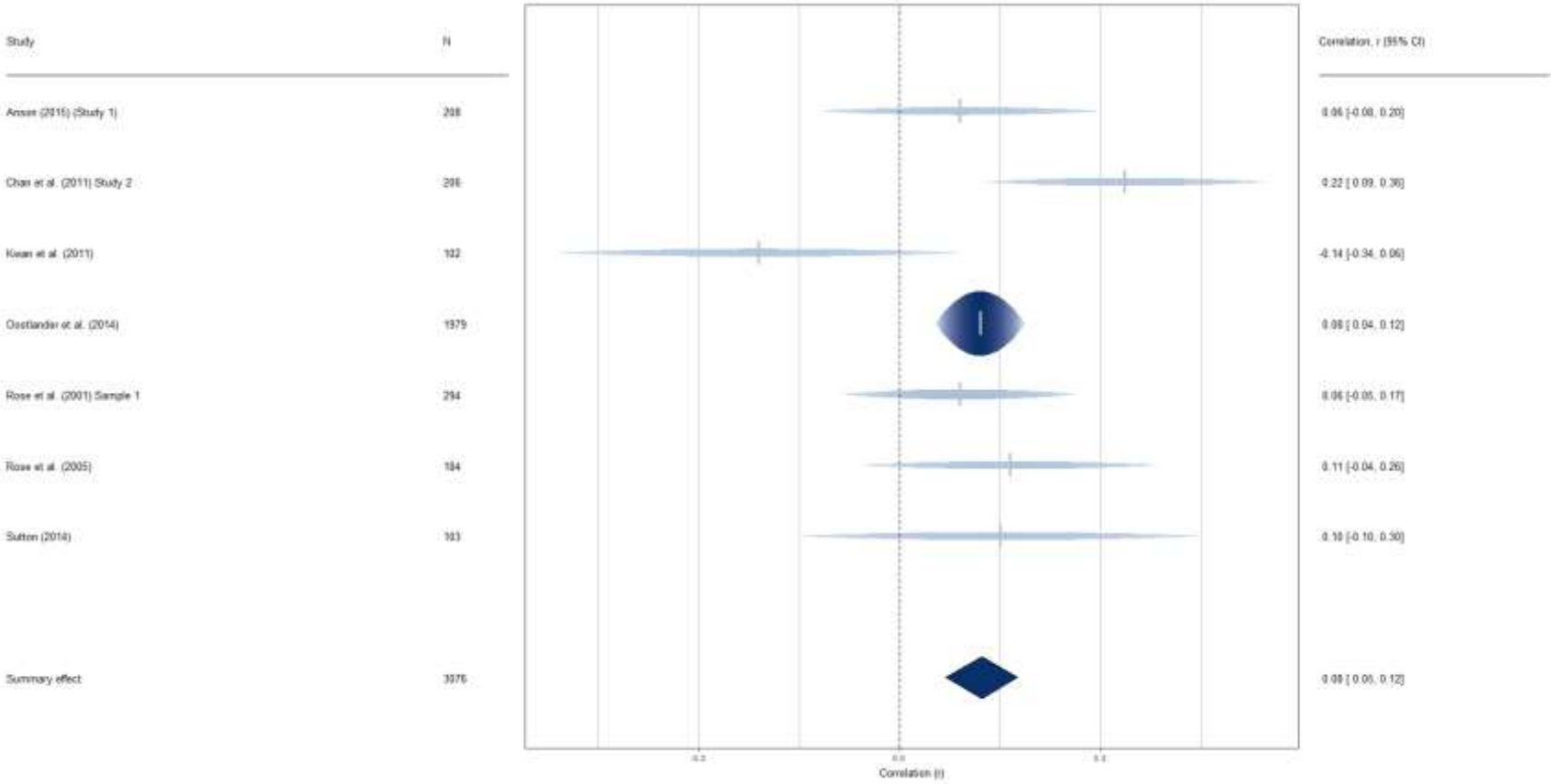


Figure F15. Forest plot of relations between control causality orientation and introjected regulation for studies included in the meta-analysis with summary effect.

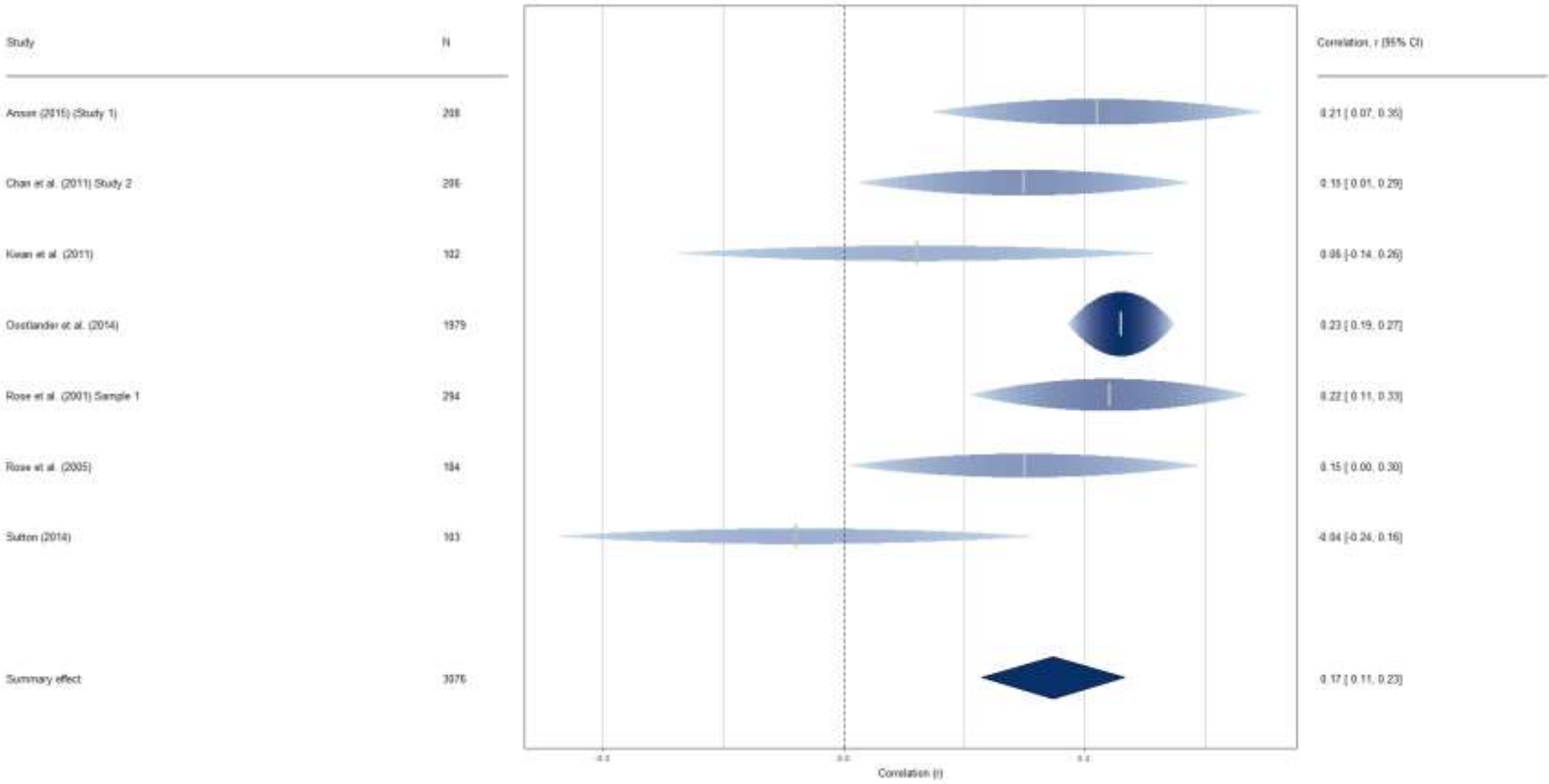


Figure F16. Forest plot of relations between control causality orientation and intrinsic motivation for studies included in the meta-analysis with summary effect.

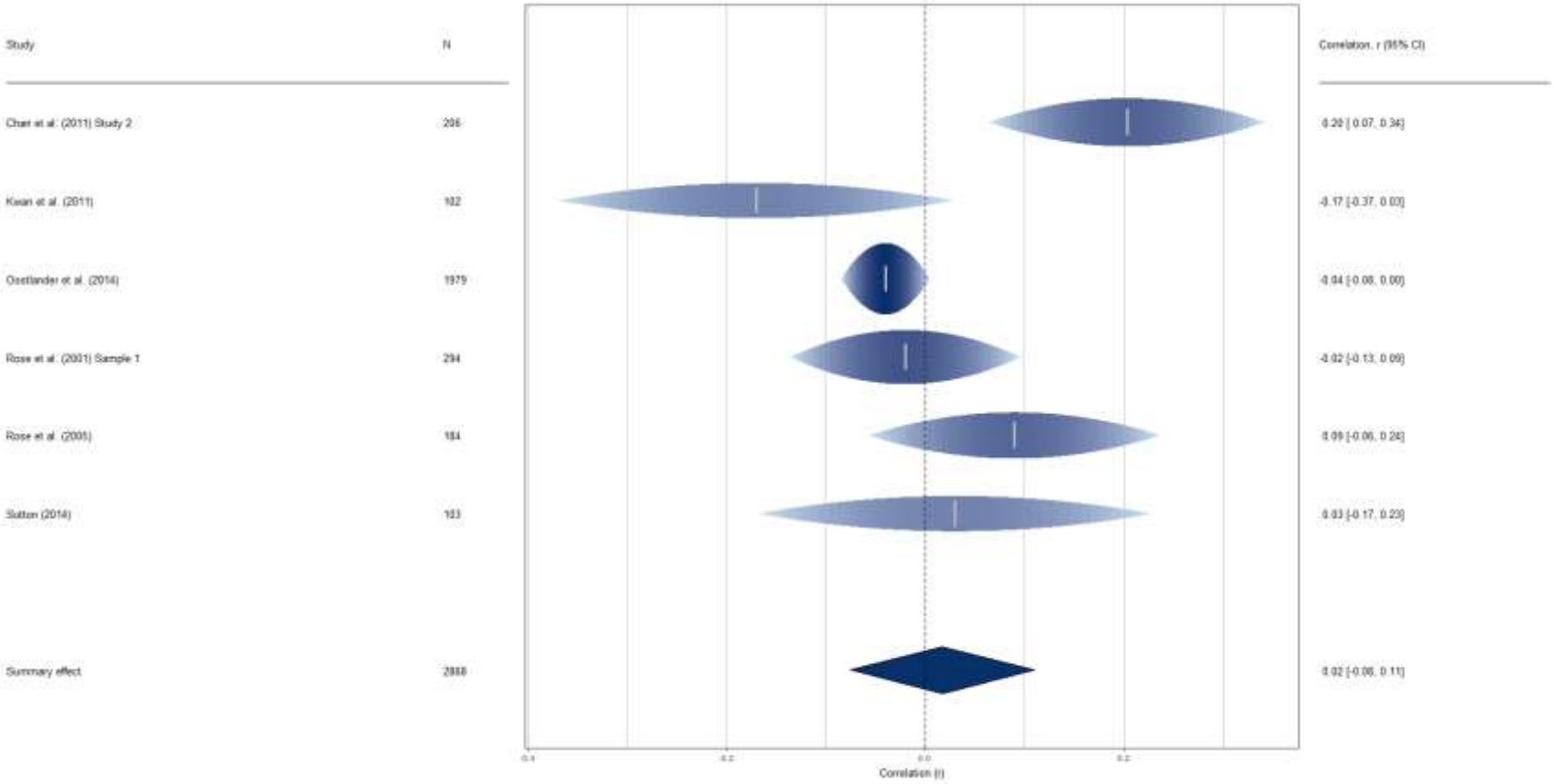


Figure F17. Forest plot of relations between controlled motivation and behavior for studies included in the meta-analysis with summary effect.

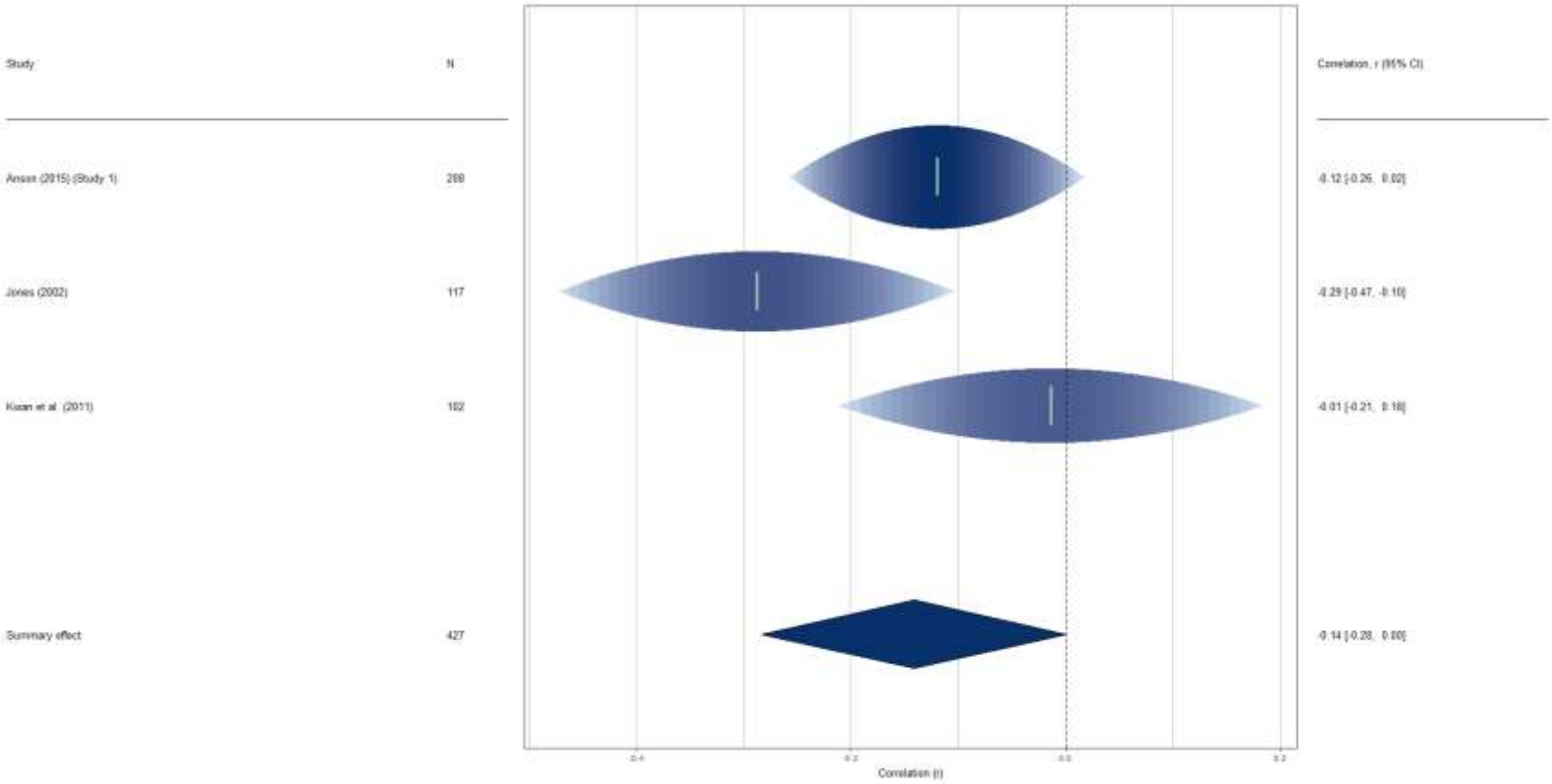


Figure F18. Forest plot of relations between identified regulation and behavior for studies included in the meta-analysis with summary effect.

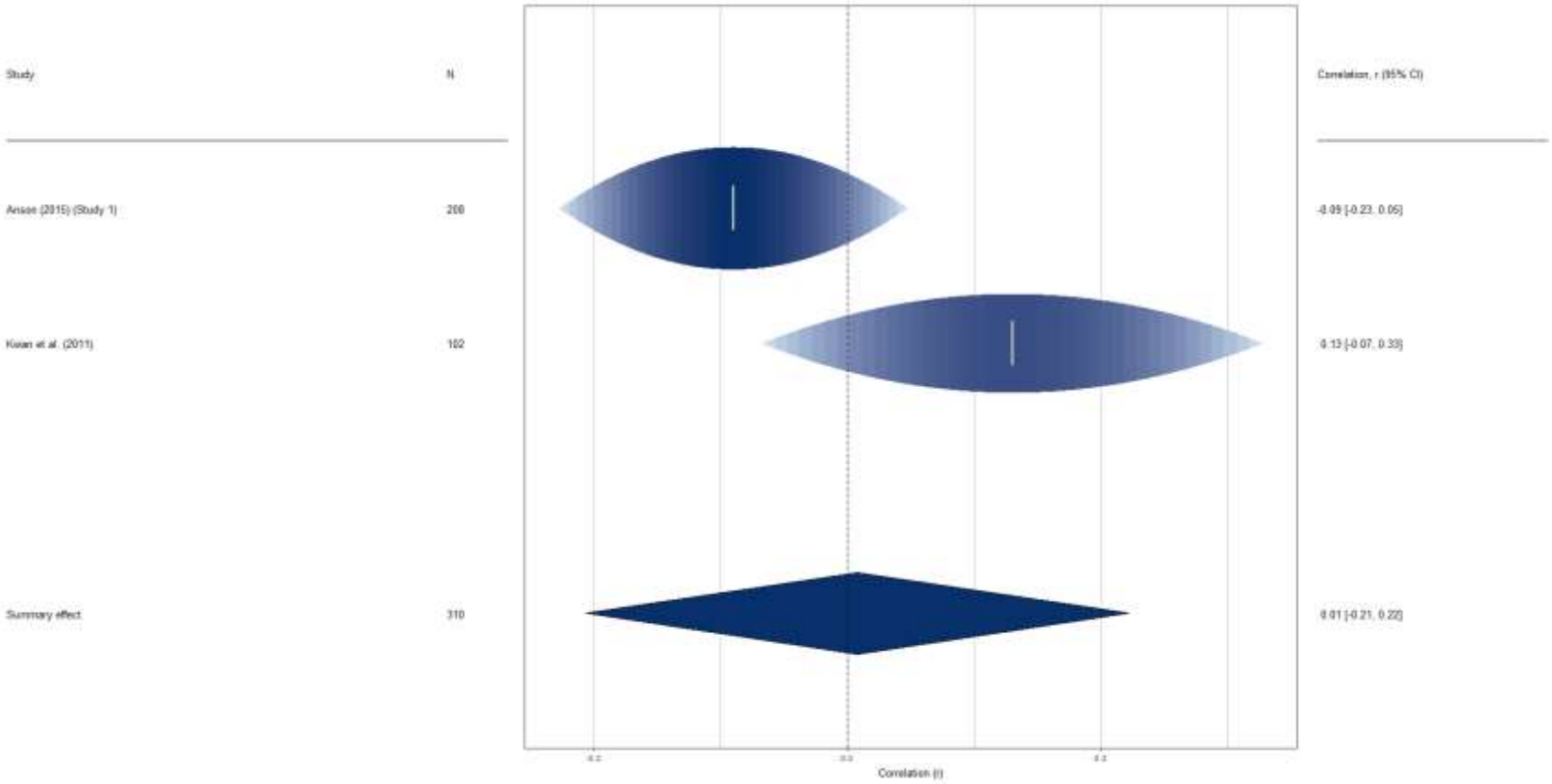


Figure F19. Forest plot of relations between identified regulation and external regulation for studies included in the meta-analysis with summary effect.

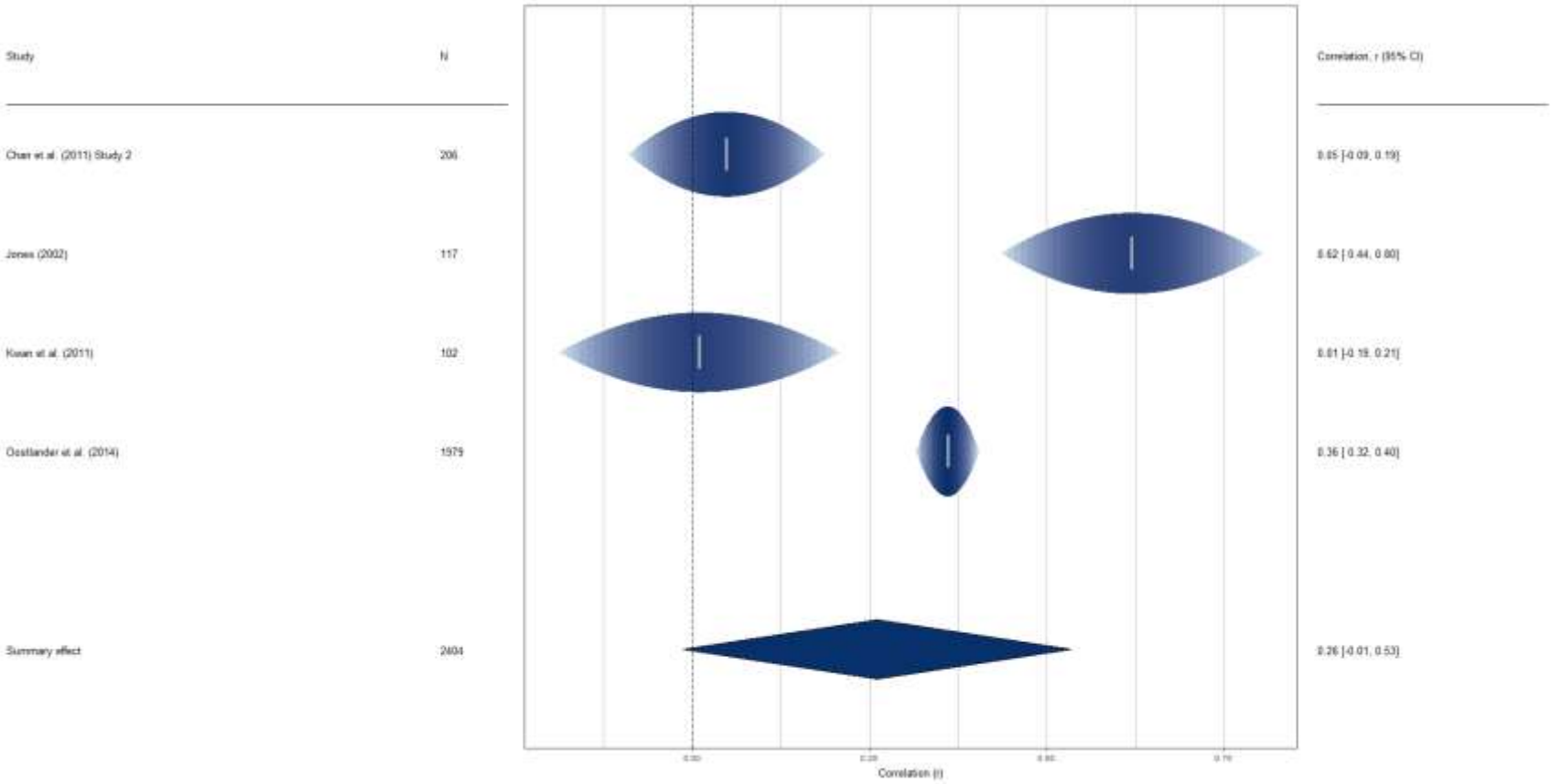


Figure F20. Forest plot of relations between identified regulation and introjected regulation for studies included in the meta-analysis with summary effect.

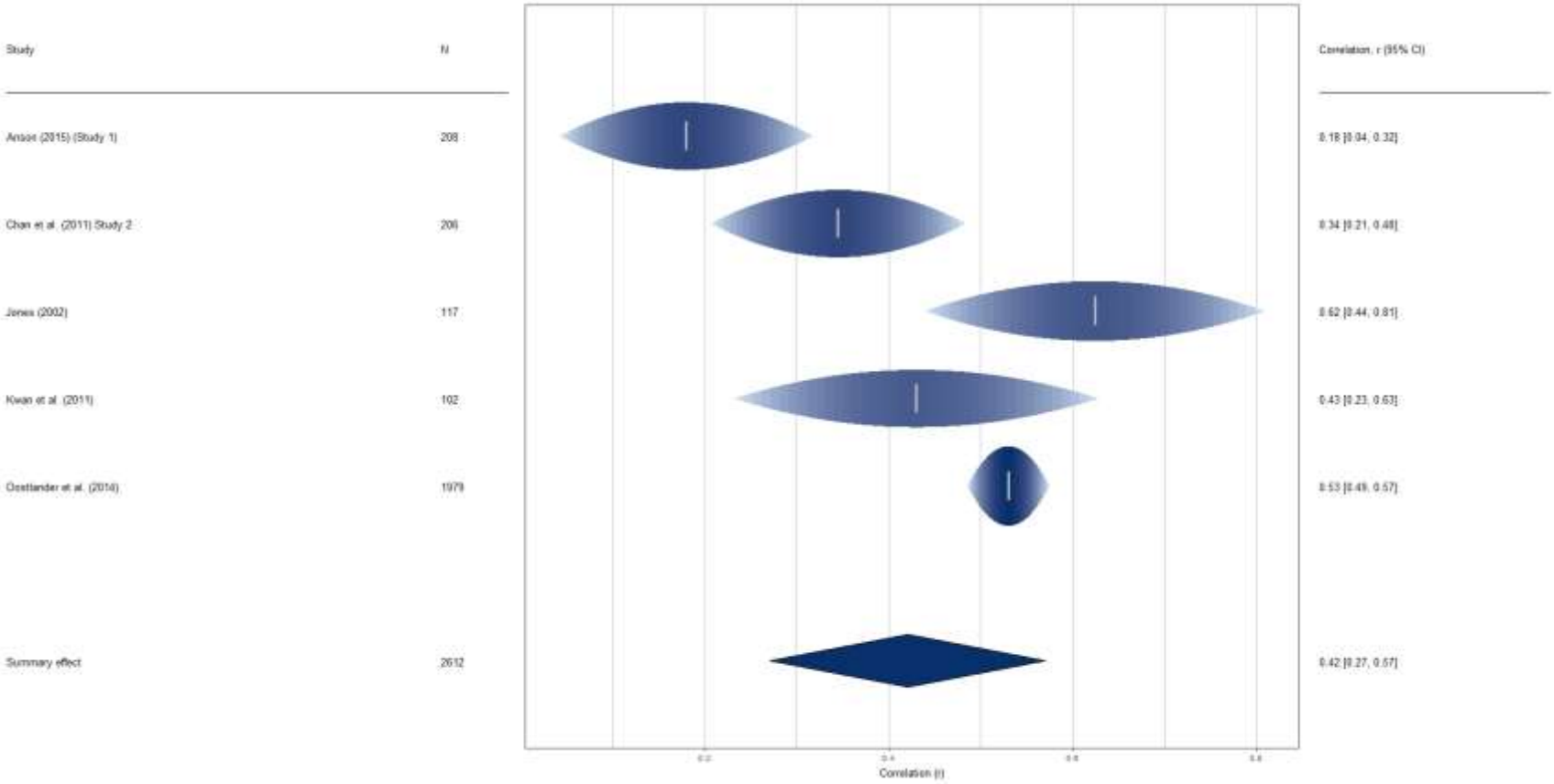


Figure F21. Forest plot of relations between introjected regulation and behavior for studies included in the meta-analysis with summary effect.

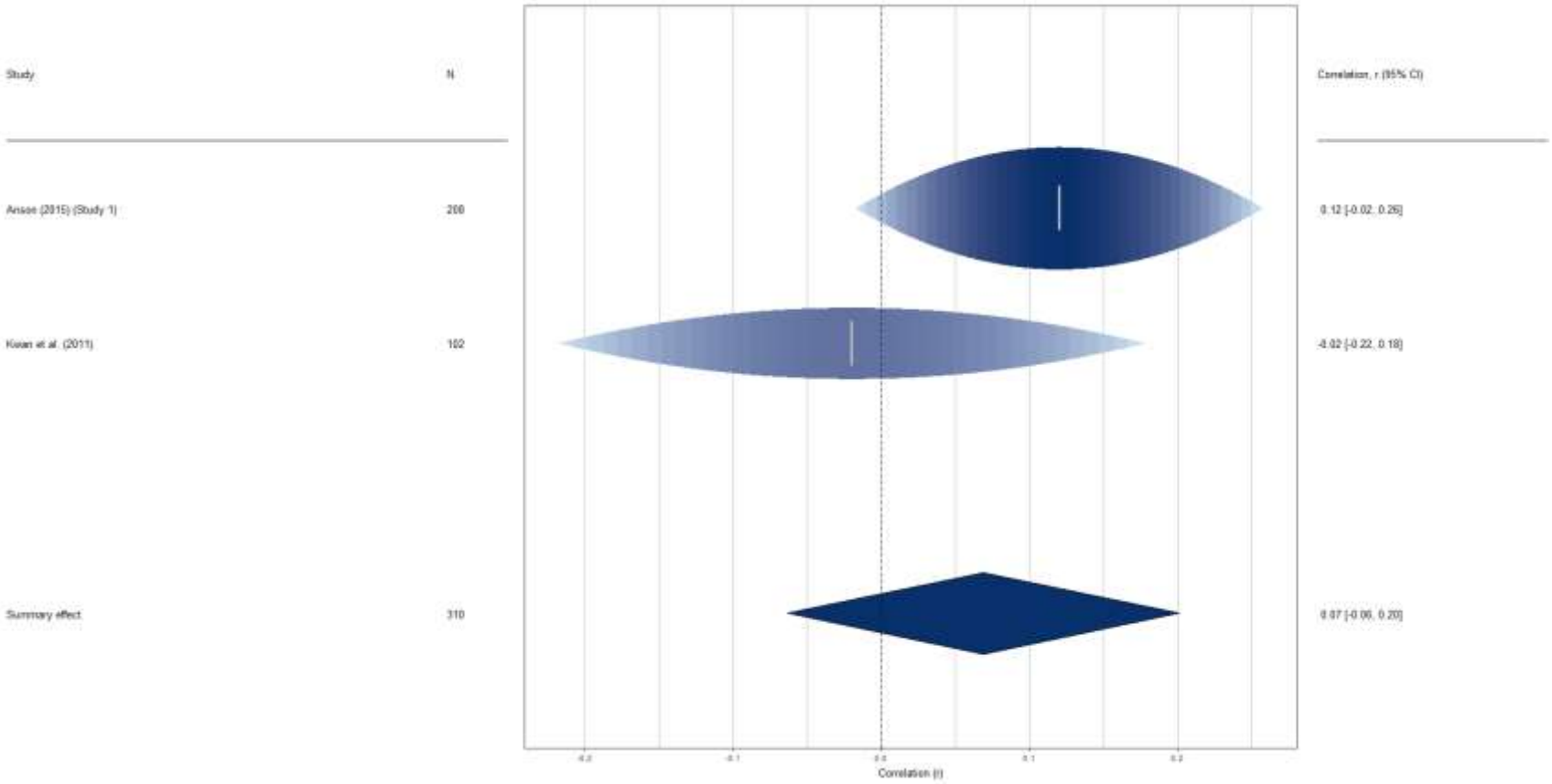


Figure F22. Forest plot of relations between introjected regulation and external regulation for studies included in the meta-analysis with summary effect.

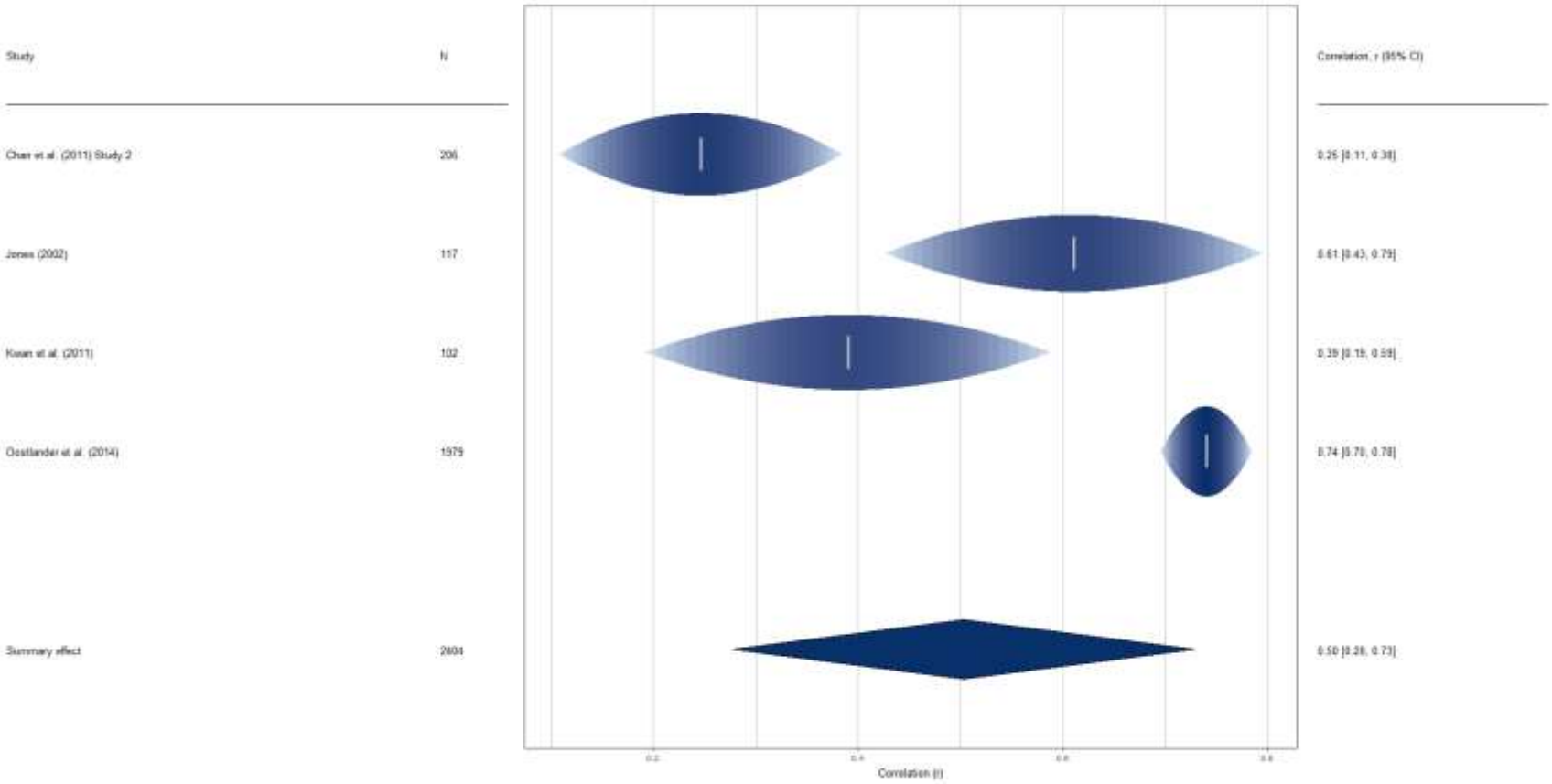


Figure F23. Forest plot of relations between intrinsic motivation and external regulation for studies included in the meta-analysis with summary effect.

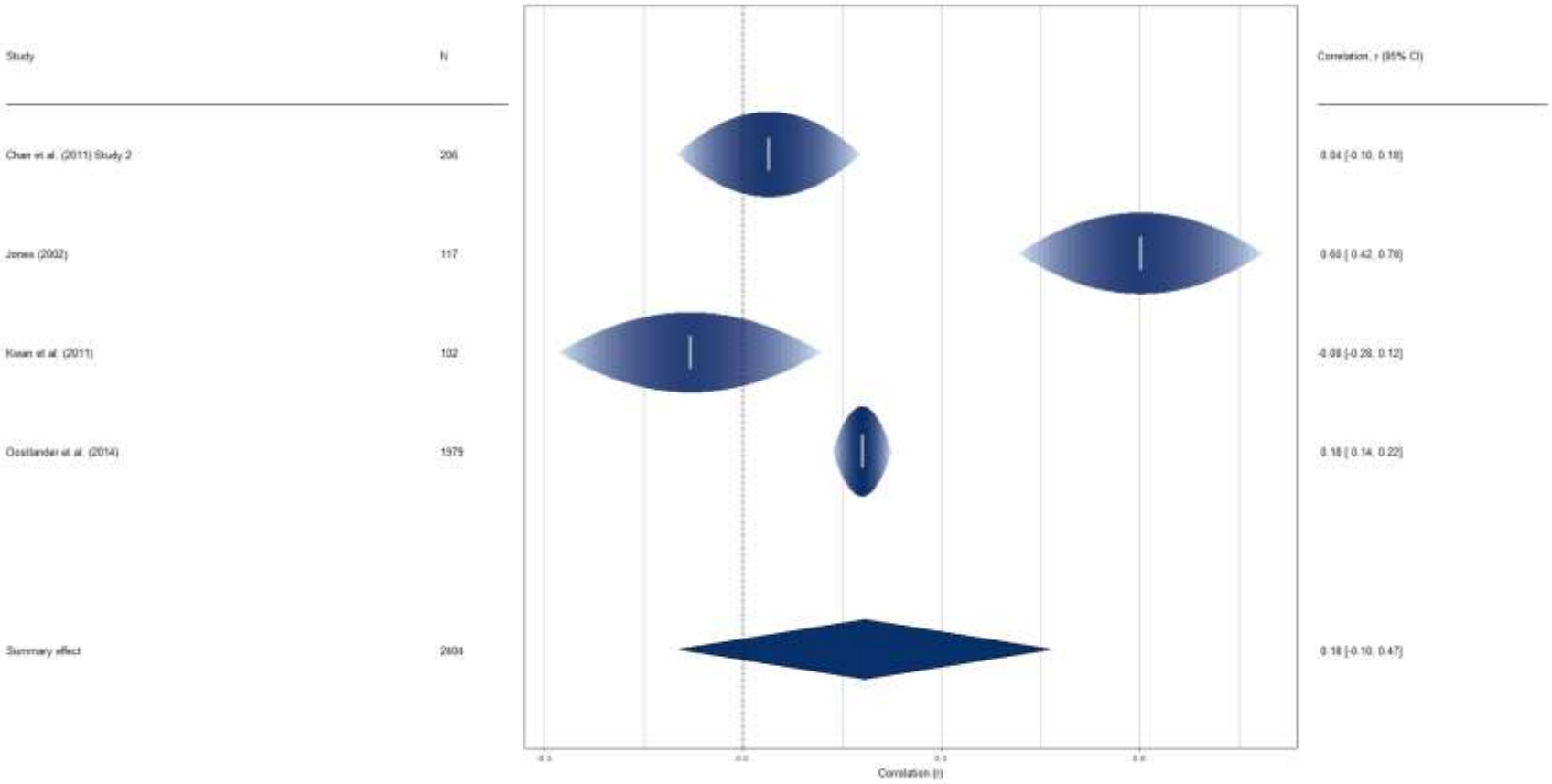


Figure F24. Forest plot of relations between intrinsic motivation and identified regulation for studies included in the meta-analysis with summary effect.

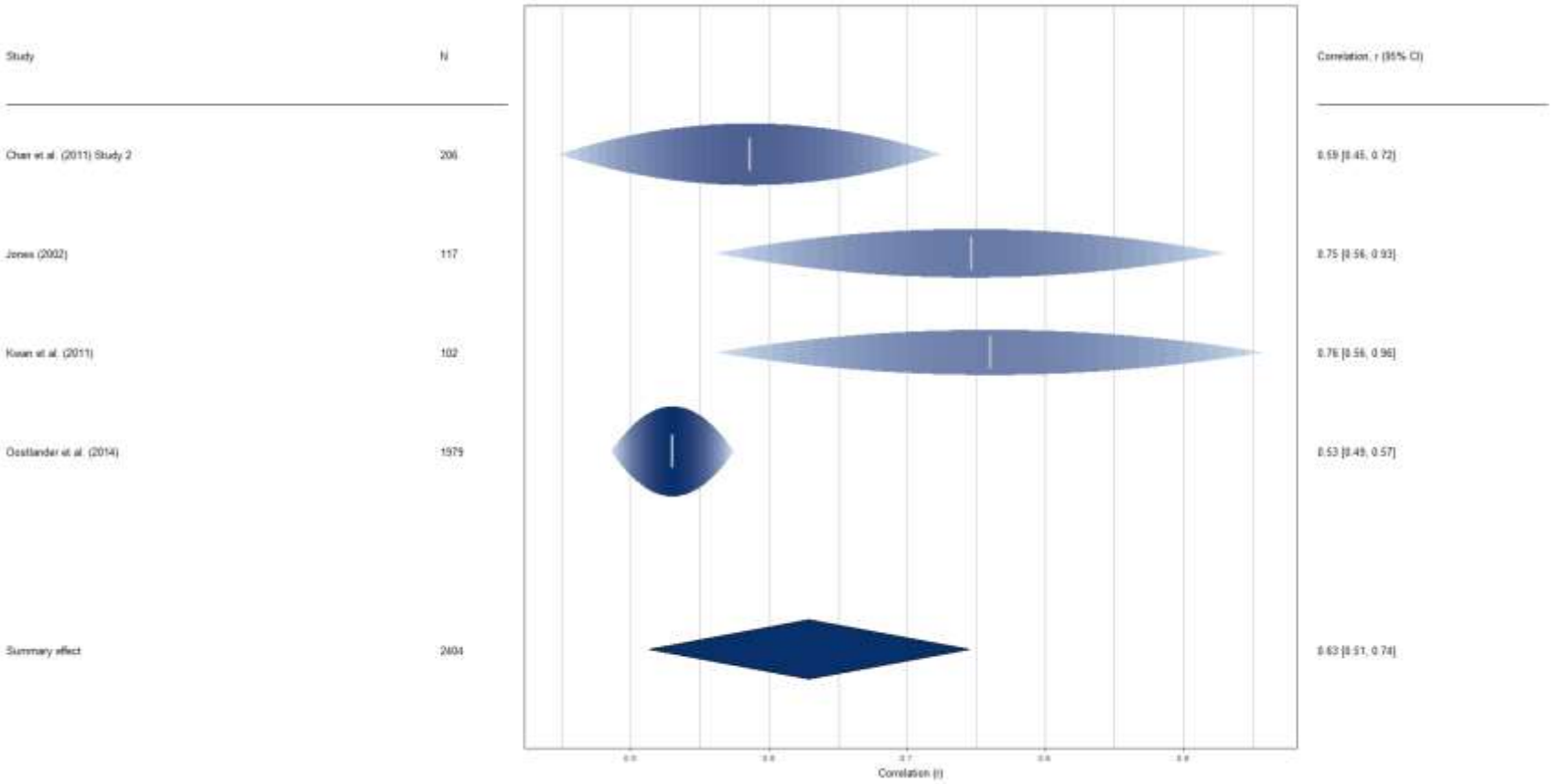


Figure F25. Forest plot of relations between intrinsic motivation and introjected regulation for studies included in the meta-analysis with summary effect.

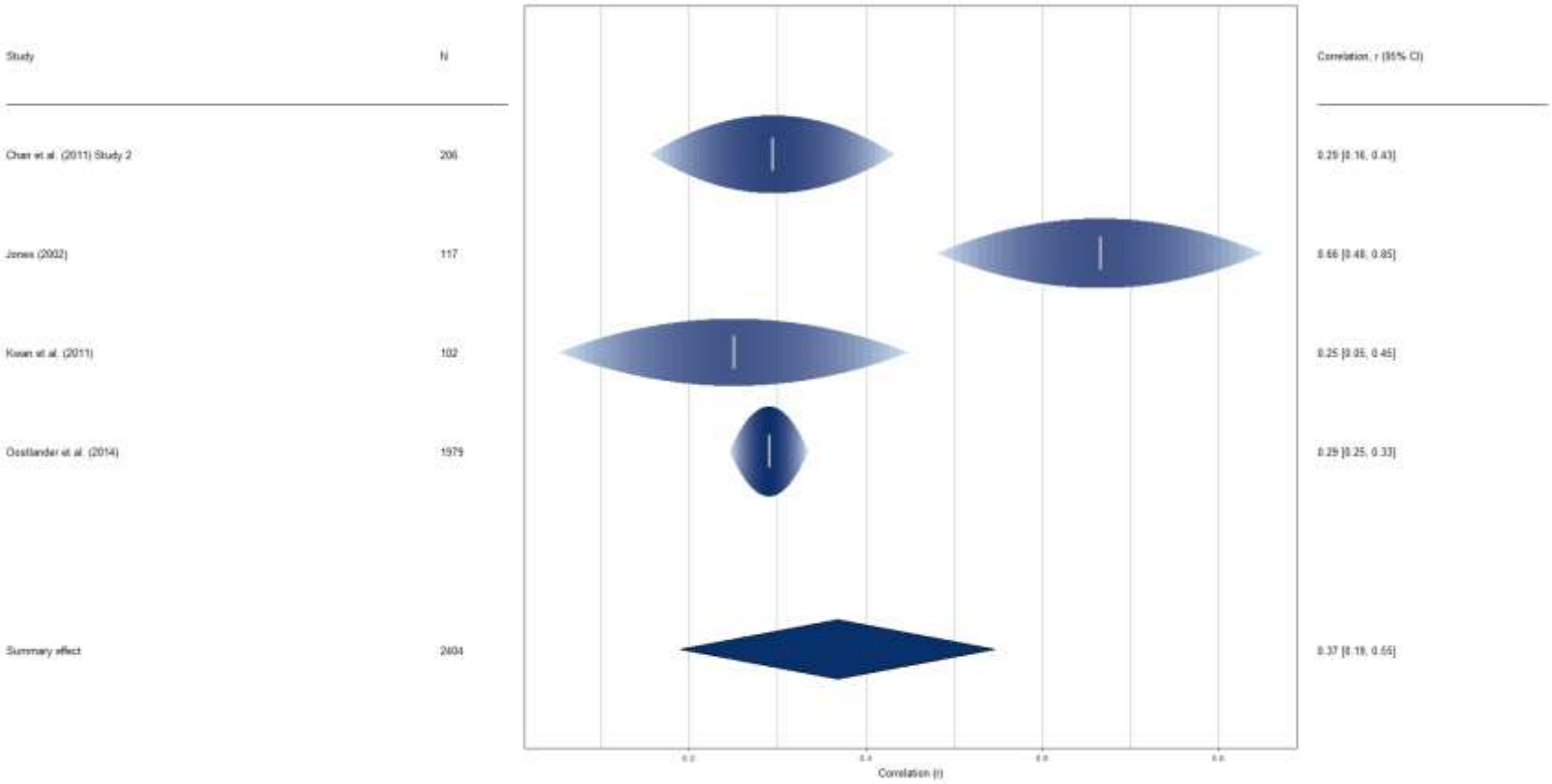


Figure F26. Forest plot of relations between impersonal orientation and autonomous motivation for studies included in the meta-analysis with summary effect.

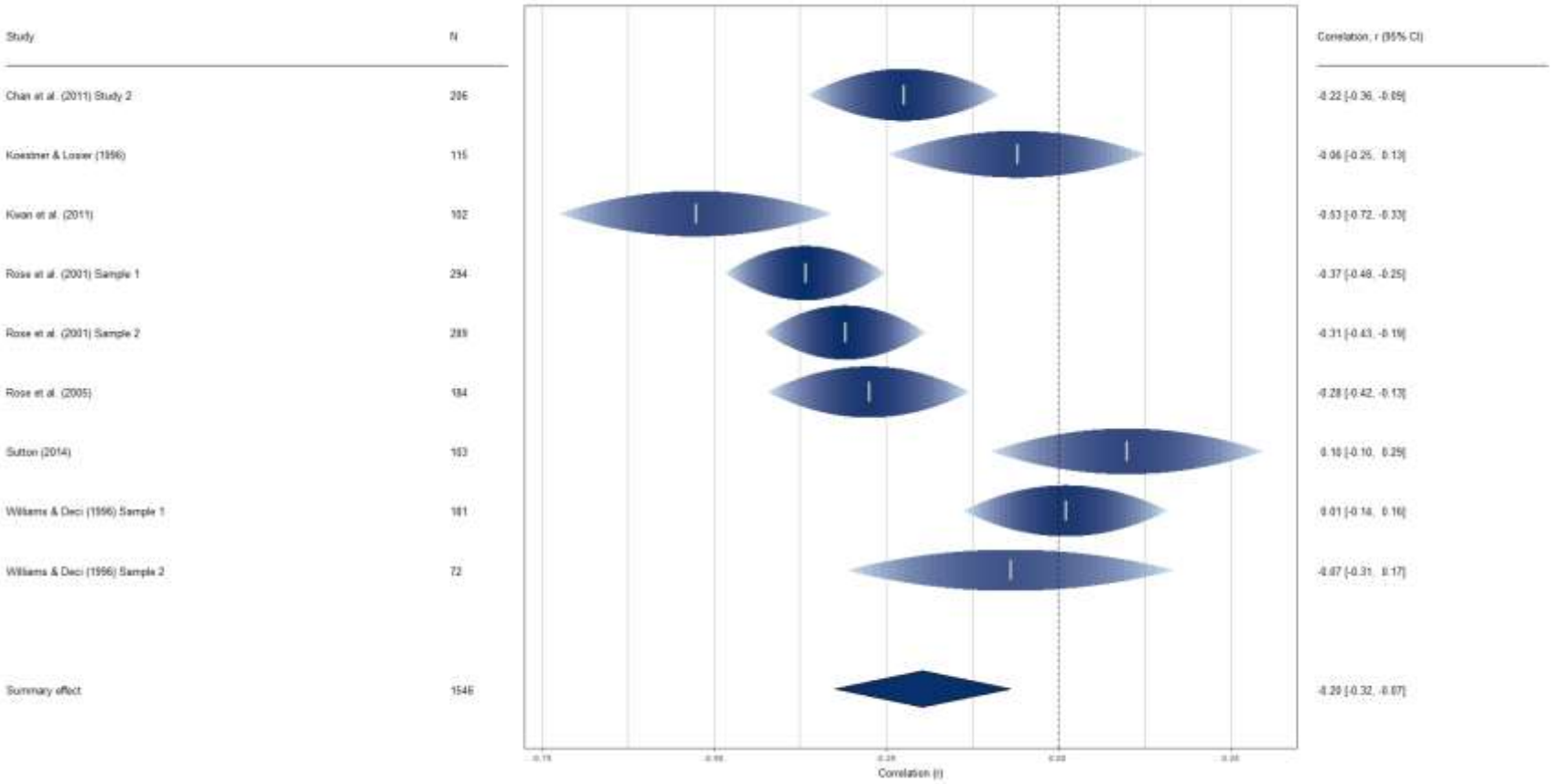


Figure F27. Forest plot of relations between impersonal orientation and behavior for studies included in the meta-analysis with summary effect.

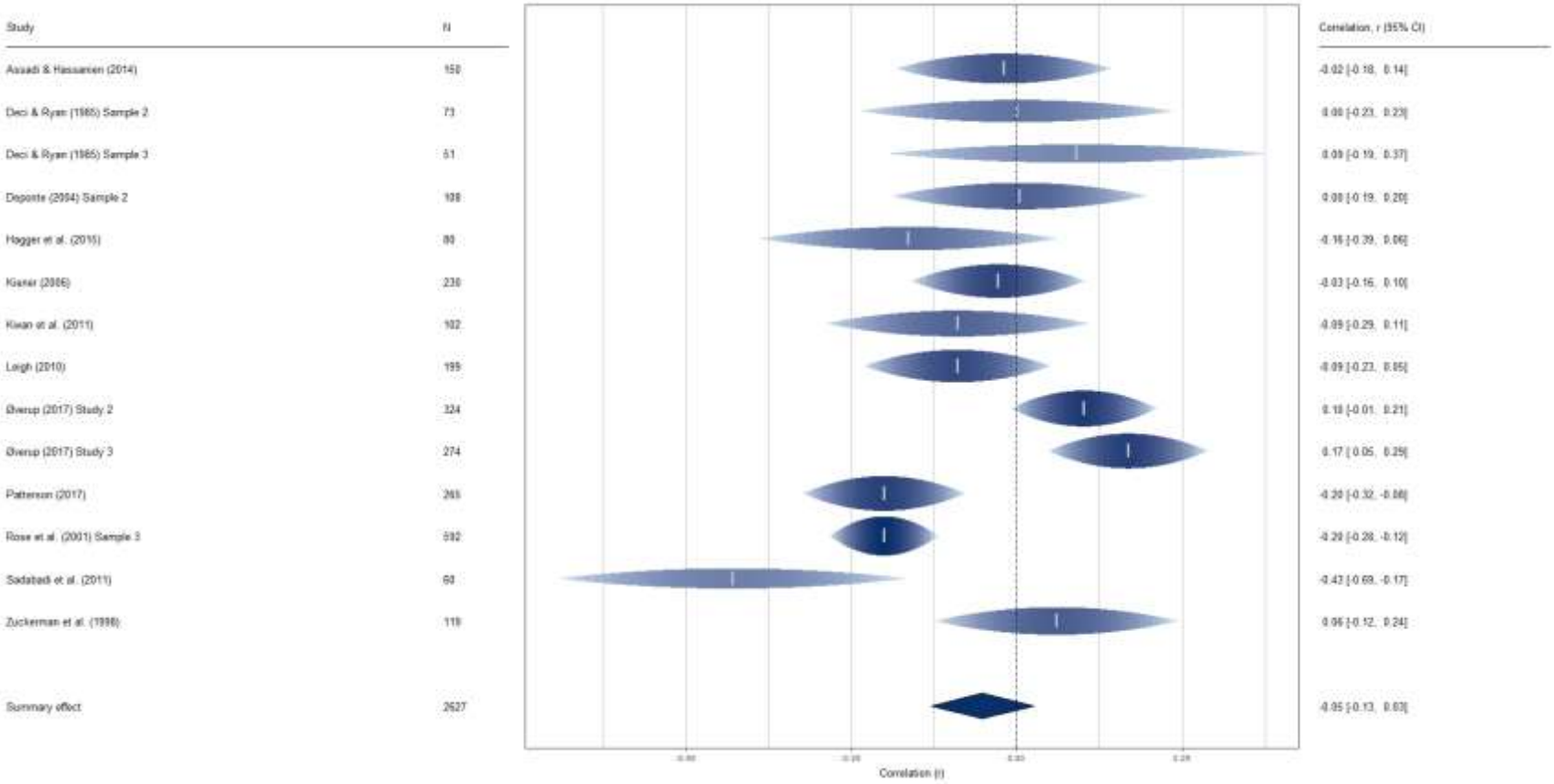


Figure F28. Forest plot of relations between impersonal orientation and controlled motivation for studies included in the meta-analysis with summary effect.

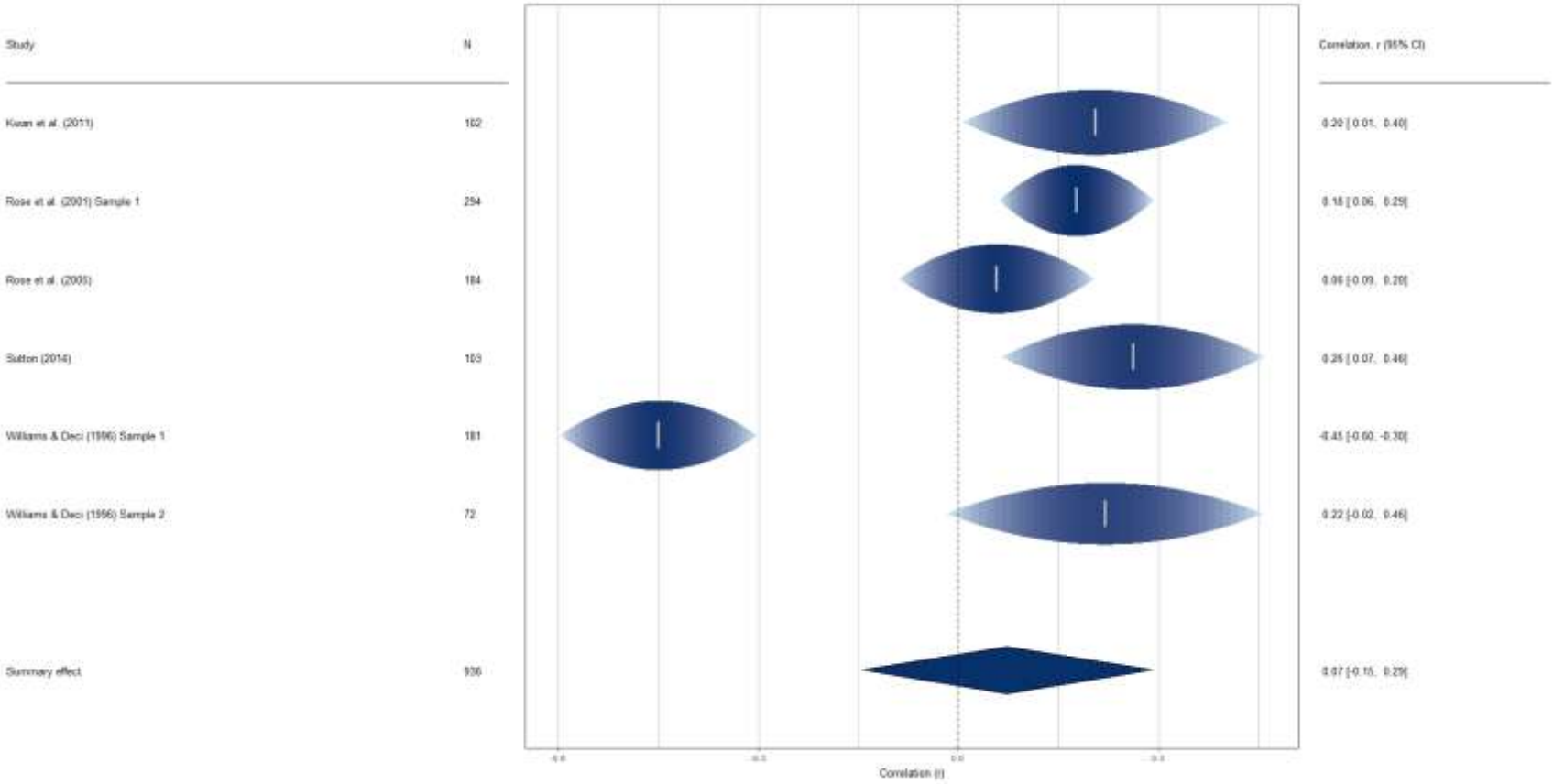


Figure F29. Forest plot of relations between impersonal orientation and external regulation for studies included in the meta-analysis with summary effect.

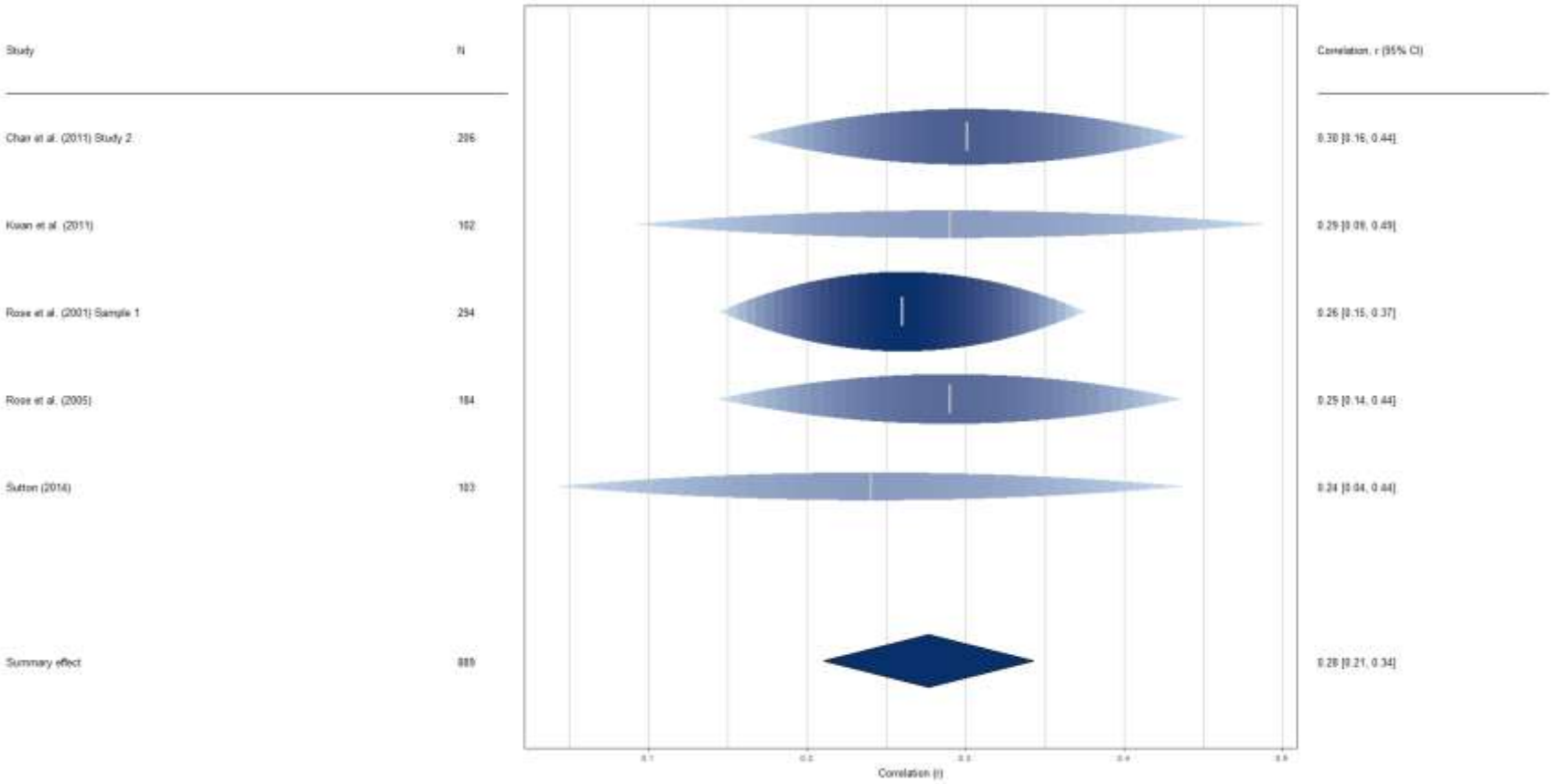


Figure F30. Forest plot of relations between impersonal orientation and identified regulation for studies included in the meta-analysis with summary effect.

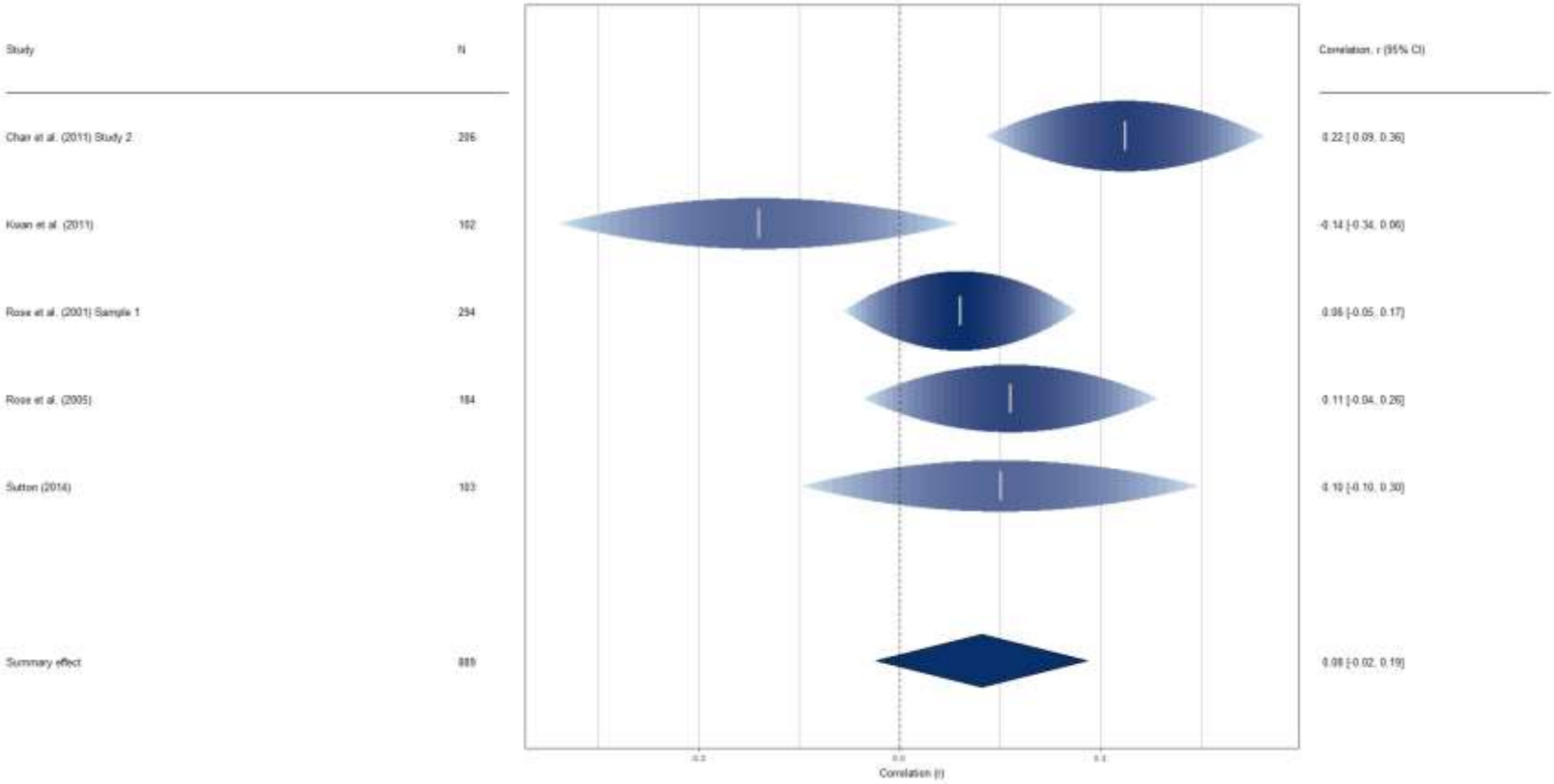


Figure F31. Forest plot of relations between impersonal orientation and introjected regulation for studies included in the meta-analysis with summary effect.

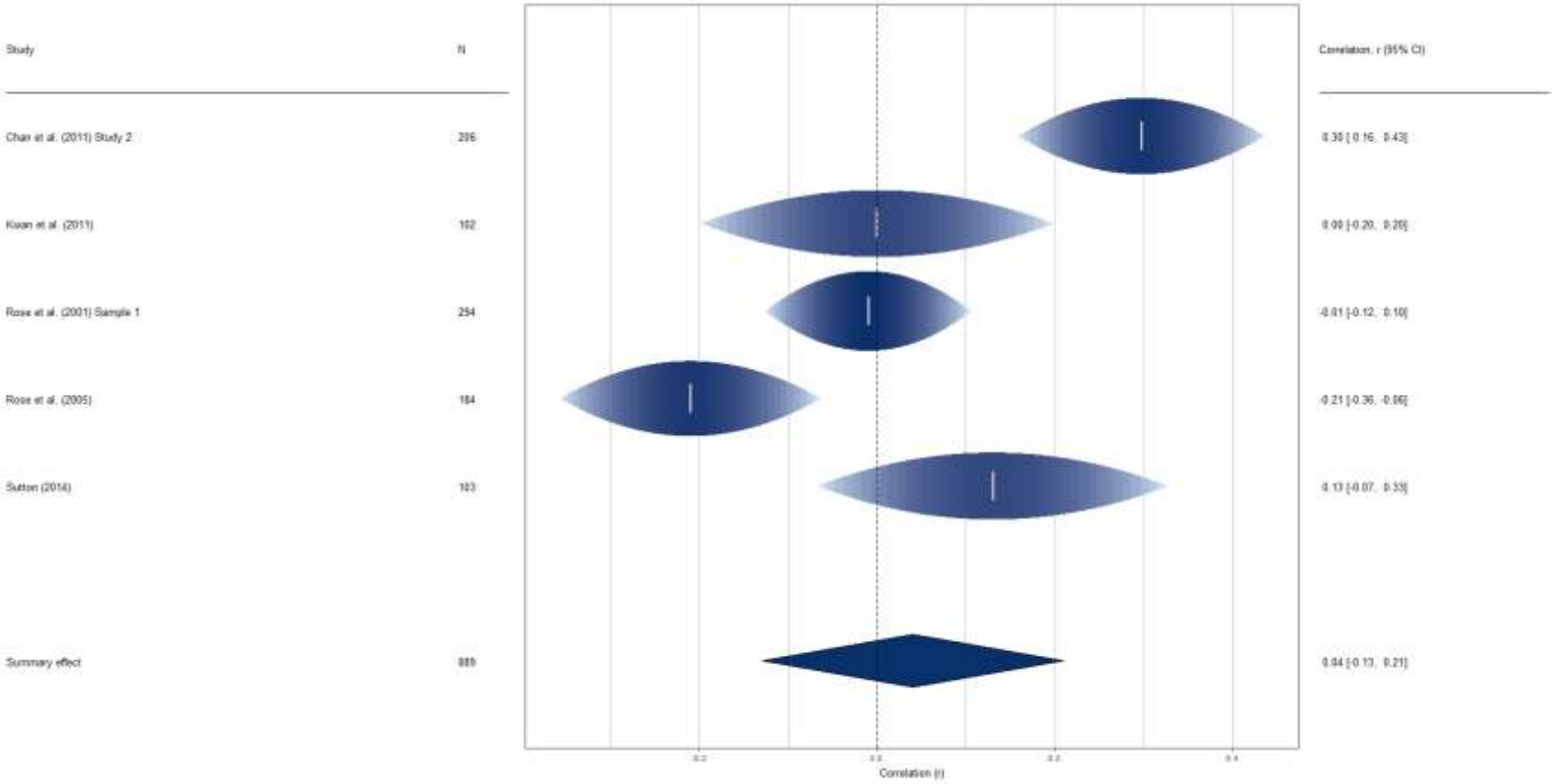
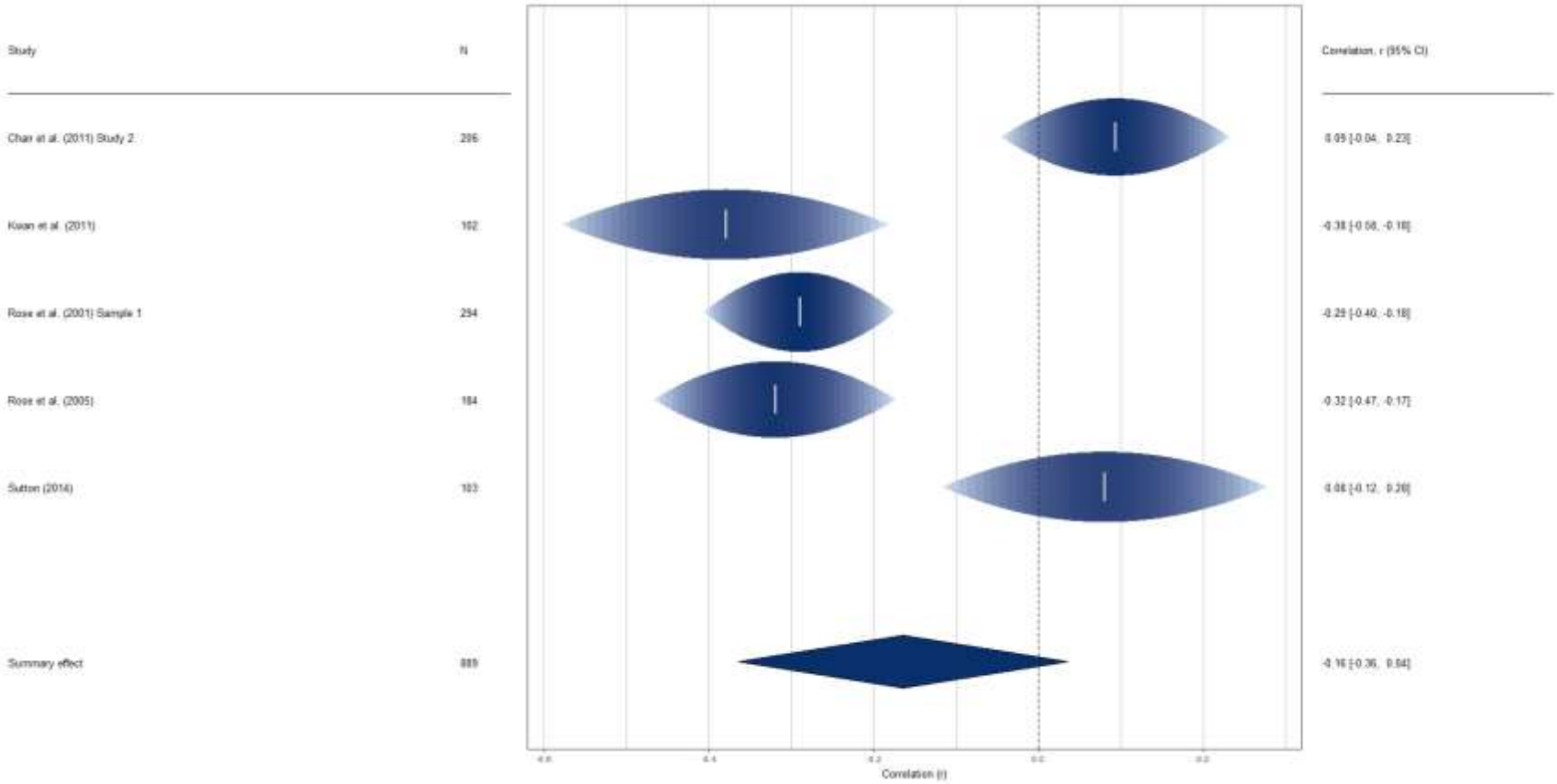


Figure F32. Forest plot of relations between impersonal orientation and intrinsic motivation for studies included in the meta-analysis with summary effect.



Appendix G: Meta-Analytic Structural Equation Modeling (Stage 1) Results

Table G1

Zero-Order Parameter Estimates from Meta-Analytic Structural Equation Models (Stage 1) for Relations Among General Causality Orientations, Motivational Styles from Self-Determination Theory, and Behavior with Heterogeneity and Bias Statistics

Effect	r^+	SE	CI_{95}		p	I^2	τ^2
			LL	UL			
Model 1[a]							
Aut.–Con.	.106	.027	.054	.159	<.001	9.79	.041
Aut.–Imp.	-.098	.027	-.150	-.046	<.001	86.76	.027
Aut.–Aut. Mot.	.333	.047	.240	.425	<.001	91.58	.045
Aut.–Con. Mot.	.058	.040	-.020	.135	.144	73.29	.012
Aut.–Beh.	.138	.044	.052	.224	.002	88.46	.032
Con.–Imp.	.268	.028	.213	.324	<.001	88.5	.031
Con.–Aut. Mot.	-.009	.044	-.095	.078	.841	87.19	.029
Con.–Con. Mot.	.286	.046	.196	.376	<.001	81.09	.018
Con.–Beh.	.073	.037	.001	.145	.048	81.7	.019
Imp.–Aut. Mot.	-.199	.060	-.316	-.082	.001	85.62	.025
Imp.–Con. Mot.	.074	.103	-.129	.276	.475	93.06	.056
Imp.–Beh.	-.051	.037	-.124	.022	.172	74.57	.012
Aut. Mot.–Con. Mot	.181	.115	-.045	.407	.116	95.32	.085
Aut. Mot.–Beh.	.097	.033	.032	.162	.004	0.00	–
Con. Mot.–Beh.	-.141	.056	-.251	-.031	.012	35.41	.002
Model 2[b]							
Aut.–Con.	.106	.027	.053	.159	<.001	9.79	.041
Aut.–Imp.	-.098	.027	-.150	-.046	<.001	86.72	.027
Aut.–Beh.	.138	.044	.051	.224	.002	88.46	.032
Con.–Imp.	.268	.028	.213	.324	<.001	88.47	.031
Con.–Beh.	.073	.037	.001	.145	.046	81.47	.018
Imp.–Beh.	-.051	.037	-.124	.022	.170	74.51	.012
Model 3[c]							
Aut.–Con.	.106	.027	.053	.159	<.001	9.80	.041
Aut.–Imp.	-.098	.027	-.151	-.046	<.001	86.79	.027
Aut.–Aut. Mot.	.332	.047	.240	.425	<.001	91.61	.045
Aut.–Con. Mot.	.058	.039	-.019	.135	.142	73.01	.011
Con.–Imp.	.268	.028	.213	.324	<.001	88.52	.031
Con.–Aut. Mot.	-.009	.044	-.095	.077	.839	87.14	.028
Con.–Con. Mot.	.286	.046	.196	.377	<.001	81.08	.018
Imp.–Aut. Mot.	-.199	.060	-.316	-.082	.001	85.65	.025
Imp.–Con. Mot.	.074	.103	-.129	.276	.475	93.06	.056
Aut. Mot.–Con. Mot	.181	.115	-.045	.407	.116	95.31	.085

Note. [a]Cochran's Q statistic for the model was 2170.147 (df = 297, $p < .001$); [b]Cochran's Q statistic for the model was 1611. 414 (df = 210, $p < .001$); [c]Cochran's Q statistic for the model was 1904.381 (df = 240, $p < .001$). r^+ = Corrected effect size estimate from random effects meta-analytic structural equation modeling analysis; SE = Standard error; CI_{95} = 95% confidence interval; LL = Lower limit of CI_{95}; UL = Upper limit of CI_{95}; I^2 = Higgins and Thompson's (2002) I^2 statistic for parameter estimate; τ^2 = Estimated variance in population; Aut. = Autonomy causality orientation; Con. = Control causality orientation; Imp. = Impersonal causality orientation; Aut. Mot. = Autonomous motivation; Con. Mot. = Control motivation; Beh. = Behavior.

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Appendix H: Moderator Analyses for Meta-Analytic Structural Equation Models

Table H1

Parameter Estimates and Confidence Intervals from Meta-Analytic Structural Equation Models of Causality Orientations on Behavior (Model 2) for Moderator Groups with Comparisons

Moderator and Parameter	Model A			Model B			Model comparisons[a]		
	β	Wald CI_{95}		β	Wald CI_{95}		β_{diff}	CI_{95}	
		LL	UL		LL	UL		LL	UL
Gender[b]									
Direct effects									
Aut.→Beh.	-.008	-.242	.226	.148	.064	.232	-.156	-.404	.092
Con.→Beh.	.104	-.107	.316	.062	-.033	.157	.043	-.189	.275
Imp.→Beh.	.097	-.015	.209	-.124	-.203	-.046	.221	.085	.358
Correlations									
Aut.↔Con.	.112	.057	.166	.108	.021	.196	.003	-.099	.106
Aut.↔Imp.	-.114	-.191	-.037	-.074	-.151	.004	-.040	-.150	.069
Imp.↔Con.	.286	.206	.367	.247	.160	.333	.040	-.078	.158
Age[c]									
Direct effects									
Aut.→Beh.	.126	.016	.236	.043	-.122	.208	.083	-.115	.281
Con.→Beh.	.077	-.025	.179	-.041	-.171	.089	.118	-.048	.283
Imp.→Beh.	-.077	-.198	.044	-.182	-.324	-.040	.104	-.082	.291
Correlations									
Aut.↔Con.	.148	.076	.220	-.018	-.131	.095	.166	.032	.300
Aut.↔Imp.	-.086	-.147	-.025	-.075	-.164	.014	-.011	-.119	.097
Imp.↔Con.	.243	.177	.308	.347	.167	.527	-.104	-.296	.088
Study design[d]									
Direct effects									
Aut.→Beh.	.089	-.034	.212	.192	.104	.279	-.102	-.253	.049
Con.→Beh.	.110	.022	.198	-.005	-.173	.164	.115	-.076	.305
Imp.→Beh.	-.044	-.137	.050	-.157	-.245	-.070	.114	-.014	.242
Correlations									
Aut.↔Con.	.147	.082	.212	.027	-.057	.110	.120	.014	.226
Aut.↔Imp.	-.090	-.153	-.027	-.122	-.206	-.039	.032	-.072	.137
Imp.↔Con.	.281	.220	.342	.232	.111	.353	.048	-.087	.184
Sample type[e]									
Direct effects									
Aut.→Beh.	.075	-.035	.185	.148	.004	.293	-.074	-.255	.108
Con.→Beh.	.060	-.048	.168	.111	-.029	.251	-.051	-.228	.126
Imp.→Beh.	.004	-.079	.088	-.205	-.289	-.121	.209	.091	.328
Correlations									
Aut.↔Con.	.111	.055	.166	.109	-.006	.223	.002	-.126	.129
Aut.↔Imp.	-.094	-.149	-.039	-.096	-.223	.032	.002	-.137	.140
Imp.↔Con.	.257	.188	.326	.291	.184	.398	-.034	-.161	.093
Study quality[f]									
Direct effects									
Aut.→Beh.	.036	-.105	.177	.165	.058	.271	-.129	-.306	.048
Con.→Beh.	.047	-.086	.181	.100	-.002	.202	-.052	-.220	.116
Imp.→Beh.	-.013	-.144	.119	-.100	-.196	-.005	.088	-.075	.250
Correlations									

Aut.↔Con.	.156	.083	.229	.075	.004	.146	.081	-.021	.183
Aut.↔Imp.	-.121	-.205	-.037	-.079	-.147	-.011	-.043	-.151	.066
Imp.↔Con.	.278	.167	.389	.264	.205	.324	.014	-.112	.140

Note. [a]Model comparisons made using Schenker and Gentleman's (2001) 'standard method' using confidence intervals about the mean difference; [b]Moderator analysis comparing effects in studies with predominantly female samples (Model A) and effects in studies with approximately balanced gender distributions (Model B); [c]Moderator analysis comparing effects in studies on younger samples (Model A) and effects in studies on older and mixed age group samples (Model B); [d]Moderator analysis comparing effects in studies using cross-sectional designs (Model A) and effects in studies using non-cross-sectional designs (Model B); [e]Moderator analysis comparing effects in studies on student samples (Model A) and effects in studies on non-student samples (Model B); [f]Moderator analysis comparing effects in studies of acceptable quality (Model A) and effects in studies of questionable quality (Model B). β = Standardized path coefficient; Wald CI_{95} = Wald 95% confidence interval; LL = Lower limit of CI_{95}; UL = Upper limit of CI_{95}; CI_{95} = Conventional 95% confidence interval; β_{diff} = Difference in standardized path coefficient; Aut. = Autonomy causality orientation; Con. = Control causality orientation; Imp. = Impersonal causality orientation; Beh = Behavior.

Table H2

Parameter Estimates and Confidence Intervals from Meta-Analytic Structural Equation Models of Causality Orientations on Motivational Styles (Model 3) for Moderator Groups with Comparisons

Moderator and Parameter	Model A			Model B			Model comparisons[a]		
	β	Wald CI_{95}		β	Wald CI_{95}		β_{diff}	CI_{95}	
		LL	UL		LL	UL		LL	UL
Gender[b]									
Direct effects									
Aut.→Aut. Mot.	.316	.218	.413	.387	.278	.495	-.071	-.217	.074
Con.→Aut. Mot.	.003	-.101	.106	-.010	-.113	.093	.013	-.133	.159
Imp.→Aut. Mot.	-.169	-.301	-.037	-.242	-.386	-.097	.073	-.123	.269
Aut.→Con. Mot.	.028	-.058	.114	.017	-.066	.099	.011	-.108	.130
Con.→Con. Mot.	.283	.167	.400	.277	.193	.360	.007	-.137	.150
Imp.→Con. Mot.	.000	-.224	.224	.095	.009	.181	-.095	-.335	.145
Correlations									
Aut.↔Con.	.106	.053	.159	.109	.022	.196	-.003	-.105	.099
Aut. Mot.↔Con. Mot.	.175	-.056	.405	.186	-.009	.380	-.011	-.313	.291
Aut.↔Imp.	-.098	-.151	-.046	-.074	-.151	.004	-.025	-.119	.069
Imp.↔Con.	.268	.213	.324	.246	.160	.333	.022	-.081	.125
Age[c]									
Direct effects									
Aut.→Aut. Mot.	.316	.218	.413	.328	.233	.422	-.012	-.148	.123
Con.→Aut. Mot.	.003	-.101	.106	-.007	-.113	.099	.010	-.138	.158
Imp.→Aut. Mot.	-.169	-.301	-.037	-.241	-.384	-.098	.072	-.122	.267
Aut.→Con. Mot.	.028	-.058	.114	.048	-.052	.148	-.020	-.152	.112
Con.→Con. Mot.	.283	.167	.400	.220	.075	.365	.063	-.123	.249
Imp.→Con. Mot.	.000	-.224	.224	.113	.024	.202	-.112	-.353	.129
Correlations									
Aut.↔Con.	.106	.053	.159	.148	.076	.220	-.042	-.131	.048
Aut. Mot.↔Con. Mot.	.175	-.056	.405	.209	.113	.304	-.034	-.284	.216
Aut.↔Imp.	-.098	-.151	-.046	-.086	-.147	-.025	-.013	-.093	.068
Imp.↔Con.	.268	.213	.324	.242	.177	.308	.026	-.060	.112
Study design[d]									
Direct effects									
Aut.→Aut. Mot.	.256	.123	.389	.379	.250	.508	-.123	-.308	.063
Con.→Aut. Mot.	-.032	-.165	.101	.058	-.098	.214	-.090	-.295	.115
Imp.→Aut. Mot.	-.166	-.326	-.006	-.166	-.365	.034	.000	-.256	.255
Aut.→Con. Mot.	.010	-.091	.111	.069	-.068	.206	-.059	-.229	.111
Con.→Con. Mot.	.220	.049	.391	.331	.224	.437	-.111	-.312	.091
Imp.→Con. Mot.	.080	-.087	.246	-.034	-.341	.273	.114	-.235	.464
Correlations									
Aut.↔Con.	.147	.082	.212	.027	-.057	.110	.120	.015	.226
Aut. Mot.↔Con. Mot.	.304	.173	.434	.065	-.327	.457	.239	-.174	.652
Aut.↔Imp.	-.090	-.153	-.027	-.122	-.205	-.039	.032	-.072	.137
Imp.↔Con.	.280	.219	.342	.232	.111	.353	.048	-.087	.184
Sample type[e]									
Direct effects									
Aut.→Aut. Mot.	.267	.074	.461	.345	.258	.431	-.078	-.289	.134
Con.→Aut. Mot.	.017	-.164	.198	-.007	-.130	.117	.024	-.196	.243
Imp.→Aut. Mot.	-.138	-.364	.087	-.200	-.348	-.052	.062	-.208	.332

Aut.→Con. Mot.	-.007	-.119	.105	.046	-.071	.163	-.053	-.215	.109
Con.→Con. Mot.	.260	.033	.487	.276	.174	.379	-.016	-.265	.233
Imp.→Con. Mot.	-.086	-.482	.310	.077	-.023	.178	-.163	-.572	.245
Correlations									
Aut.↔Con.	.111	.055	.166	.110	-.004	.225	.000	-.127	.127
Aut. Mot.↔Con. Mot.	-.013	-.228	.203	.257	-.024	.538	-.270	-.624	.085
Aut.↔Imp.	-.094	-.149	-.039	-.096	-.224	.032	.002	-.137	.142
Imp.↔Con.	.257	.188	.326	.291	.184	.398	-.034	-.162	.093
Study quality[f]									
Direct effects									
Aut.→Aut. Mot.	.280	.149	.411	.325	.210	.441	-.045	-.220	.129
Con.→Aut. Mot.	.072	-.103	.246	-.006	-.119	.108	.077	-.131	.286
Imp.→Aut. Mot.	.020	-.135	.174	-.228	-.357	-.100	.248	.047	.449
Aut.→Con. Mot.	.141	-.131	.413	.015	-.051	.081	.126	-.153	.406
Con.→Con. Mot.	.057	-.088	.202	.330	.211	.448	-.273	-.460	-.086
Imp.→Con. Mot.	.290	.078	.503	-.050	-.299	.199	.340	.013	.668
Correlations									
Aut.↔Con.	.156	.082	.229	.075	.004	.146	.081	-.021	.183
Aut. Mot.↔Con. Mot.	.396	.010	.781	.078	-.154	.310	.318	-.132	.767
Aut.↔Imp.	-.121	-.205	-.037	-.079	-.147	-.011	-.042	-.150	.066
Imp.↔Con.	.278	.167	.389	.264	.205	.324	.014	-.112	.140

Note. [a]Model comparisons made using Schenker and Gentleman's (2001) 'standard method' using confidence intervals about the mean difference; [b]Sensitivity analysis comparing effects in full sample (Model A) and effects in studies with approximately balanced gender distributions (omitting studies with predominantly female samples; Model B); [c]Sensitivity analysis comparing effects in full sample (Model A) and effects in studies with younger samples (omitting studies with older and mixed age samples; Model B); [d]Moderator analysis comparing effects in studies using cross-sectional designs (Model A) and effects in studies using non-cross-sectional designs (Model B); [e]Moderator analysis comparing effects in studies on student samples (Model A) and effects in studies on non-student samples (Model B); [f]Moderator analysis comparing effects in studies of acceptable quality (Model A) and effects in studies of questionable quality (Model B). β = Standardized path coefficient; Wald CI_{95} = Wald 95% confidence interval; LL = Lower limit of CI_{95}; UL = Upper limit of CI_{95}; CI_{95} = Conventional 95% confidence interval; β_{diff} = Difference in standardized path coefficient; Aut. = Autonomy causality orientation; Con. = Control causality orientation; Imp. = Impersonal causality orientation; Aut. Mot. = Autonomous motivation; Con. Mot. = Control motivation; Beh = Behavior.

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