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1. Introduction

Behaving in a goal-directed manner requires monitoring our environment to flexibly adapt our responses according to a given feedback from our own action. As such, the achievement of directed actions is linked to efficient monitoring processes. Action monitoring can be described as awareness from current behavior performance. Therefore, monitoring is an important component of self-regulation (Benn et al., 2014; Hacker et al., 1998). A central aspect of monitoring refers to the ability to identify differences between intended and executed responses (Taylor et al., 2007; Yeung & Summerfield, 2012). This capacity to detect and use information relative to error detection is critical to implement adaptive behavior or self-regulation (Ridderinkhof et al., 2009).

Psychopathy is characterized by a set of affective, relational, and behavioral symptoms (including egocentricity, impulsivity, irresponsibility, shallow emotions, pathological lying, manipulation, persistent violation of social norms and expectations, lack of empathy, guilt and remorse) leading to a specific pattern of difficulties to successfully adapt behavior with respect to social rules and norms (Brazil et al., 2009). Consequently, while the prevalence of psychopathy reaches approximately 1% in the general population, it is getting close to 7% - for the conservative estimation - in the inmate population (Boduszek et al., 2019). In addition, the lifelong abnormalities to adapt and learn behavior guided by reward and punishment observed in psychopathic criminals lead to high rates of recidivism with a four times greater likelihood of violent recidivism compared to non-psychopathic criminals (DeLisi, 2018; Rice & Harris, 1997).

The incapacity of psychopathic individuals to adapt behavior and learn from punishment has been largely investigated in laboratory settings using response reversal tasks, probabilistic learning task or passive avoidance learning (Brazil et al., 2013; Budhani et al., 2006; Finger et al., 2008; Vitale et al., 2005; Von Borries et al., 2010). On the one hand, results related to reversal learning suggest that the maladaptive behavior is driven by abnormal processing of negative reinforce during

reversal modalities of the task but not by the impossibility to adapt behavior from the error-commission. On the other hand, psychopathic individuals fail to inhibit punished responses during passive avoidance suggesting that initial learning rules are degraded (Blair et al., 2004; Newman et al., 1990). Nevertheless, the incapacity to reversal behavior to meet the demands of the task suggests that psychopathic individuals display abnormal processing of error-related information and are less sensitive to the negative feedback following an erroneous response. Psychopathic individuals can adequately process negative feedback at a neural level but cannot use this feedback to adapt behavior. The abnormalities in feedback and error-related information processing to signal non-adapted behavior in psychopathic individuals are also highlighted by neuroimaging studies that revealed atypical activities in the posterior cingulate cortex and anterior insula in response to punished errors during the reversal task (Gregory et al., 2015). These results suggest that the processing of error-related information plays a central role in the reported learning deficits in psychopathic individuals and leads to inoperative learning behavior or at least atypical choices to respect the demands of the environment.

The processing of error-related information in humans can be measured by response-locked components (Bernstein et al., 1994; Gehring & Fencsik, 2001; Holroyd & Coles, 2000; Scheffers et al., 1996; Yeung et al., 2004). According to such electrophysiological studies, the error-related negativity (ERN) and the error-positivity (Pe) are two components associated with action monitoring and the processing of error-related information. In different tasks, error commissions elicited these components even in absence of or prior to explicit feedback. The ERN and Pe component reflect a neural activity that detects and processes error commissions, respectively (Simon, 2010; Wiswede et al., 2011). The ERN component is a negative deflection with fronto-central distribution peaking around 20ms to 80ms following an erroneous response, whereas the Pe component is a positive deflection with a broad midline scalp distribution peaking between 200ms and 500ms following error commission. Despite their association with regard to the processing of

error-related information, these components are highly dissimilar. According to the error-awareness hypothesis (Overbeek et al., 2005), the ERN component appears during aware or unaware error commissions, while the Pe component appears only when the subject is conscious of the error commission. Indeed, contrary to the ERN, the Pe seems to vary according to the degree of awareness of the error (Godefroid et al., 2016; Nieuwenhuis et al., 2001). Regarding the functional significance of the Pe component, it is important to note that the time course of this component is similar to the P3 component. The P3 is a positive component, which follows a motivational stimulus with a peak in the range of the Pe component with similar scalp distribution. As noted by Overbeek *et alii* (2005), the Pe reflect a P3 related to the motivational significance of the error.

The observation of the ERN component is also traditionally associated with incorrect responses. A common view is that ERN reflects error monitoring which processes the signal for the detection of a mismatch or conflict between the planned response and the executed one (Yeung et al., 2004). The respective functional implication of both components is therefore dissimilar but complementary for adaptive and efficient error and post-error processing.

Over the last decades, a growing panel of studies has reported atypical response-locked components of error monitoring among psychopathic individuals. Regarding psychopathy, the modern conception of this disorder examines it with a separate dimensional construct or, at least, as integrating a higher-order dimensional construct (for example, the 4-factor model (Hare, 2003) or the hierarchical 3-factor model (Cooke & Michie, 2001)).

Thus, most studies consider that the severity of dimensions related to the disorder could be the major source of variability in response-locked components of error monitoring. The higher-order dimensional structure of psychopathy, as assessed by the Psychopathy Checklist-Revised (PCL-R), comprises two higher-order dimensions, namely an Interpersonal-Affective dimension (Factor-1) and an Impulsive-Antisocial dimension (Factor-2). This checklist pools 4 sub-factors related to interpersonal, affective, lifestyle and antisocial traits. Despite the high internal

consistency of the measure of psychopathy between these two higher-order dimensions (Hare et al., 1990; Harpur et al., 1989), which points in the direction of a possible unitary entity of psychopathy, some evidence challenges the unitary hypothesis. Different subtypes, distinguishable in terms of personality structure and reflecting different etiologies, can be isolated among psychopathic individuals.

Before the dimensional conception of psychopathy, typological models had already introduced an alternative conception of psychopathy by highlighting the heterogeneity among psychopathic individuals. The pioneer models using typological classification described the existence of primary and secondary psychopathy as separable subtypes (Blackburn, 1975; Levenson et al., 1995; Karpman, 1941). Across this classification, primary psychopathy is most commonly reported as a subtype including emotional hypo-reactivity features, a lack of anxiety, and typical anomalies in attentional functioning, whereas secondary psychopathy groups together emotional hyper-reactivity features and impulsivity. In many ways, this anterior classification mirrors current clinical and theoretical characterizations of psychopathy that use a dimensional structure.

Indeed, based on differences in personality structure, a classification of psychopathic individuals reported two subgroups with different profiles (Hicks et al., 2004). One subgroup is emotionally stable and characterized by low stress reactions and high agency; and the second subgroup is characterized by aggressiveness, highly negative emotionality, low constraint, and low communion. This dissociable etiological pathway is a limitation regarding the unity of the entity of psychopathy. Previous studies reported a deficit in aversive startle potentiation specifically related to the interpersonal-affective dimension, suggesting an abnormal defensive reactivity (Patrick, 1994). The "two-process theory of psychopathy" (Patrick & Bernat, 2009) and the "dual-pathway model of psychopathy" (Fowles & Dindo, 2009) are major models that emphasize the leading involvement of two distinctive etiological processes in the same psychopathic individuals: abnormal defensive reactivity / low-fear temperament and impulsivity /regulatory discontrol.

The TriPM scale (Patrick et al., 2009) is the standard tool assessing psychopathy as a non-unitary entity composed of separate dimensions. The main principle of this model is that psychopathy can be described using three distinct phenotypic constructs: Disinhibition, Boldness, and Meanness. These constructs reflect separate brain processes depicted by the two-process theory of psychopathy. Indeed, Patrick (2012) suggested that Boldness and Meanness reflect the underactivity of the brain's defensive motivational system, which appears more relevant to affective-interpersonal features in psychopathy disorder. Disinhibition and externalizing-propensity rather seem to be linked to a dysfunction in the fronto-cortical regulatory circuitry that is important to regulate emotion and guide decision making and action (Fowles & Dindo, 2009; Patrick & Bernat, 2009; Skeem et al., 2011). Finally, the theoretical core of Patrick's conceptualization is that the three dimensions should be measured as three separate constructs with intersecting components (Patrick et al., 2012). Note that these approaches toward psychopathic disorder using models positing separable etiologic processes fit with the higher-order dimensions observed in the instruments used to assess psychopathic disorder such as the PCL-R.

A further indication for a possible non-unitary construct of psychopathy disorder comes from studies exploring comorbidity. Specific features of psychopathic individuals integrate an externalizing spectrum. According to the *Diagnostic and Statistical Manual of Mental Disorders (DSM-V, 2013),* the externalizing spectrum is linked to antisocial personality disorder and underlying disinhibited / impulsive / disruptive patterns of behavior, and alcohol or drugs abuse. These features are highly related to disruption of error processing. Previous studies reported that individuals with a high externalizing tendency show a reduced ERN component (for review see Pasion & Barbosa, 2019), but a normal Pe component (McDonald et al., 2019). Considering the distinctions in cognitive functioning between individuals with externalizing and psychopathy disorder (Baskin-Sommers, & Newman, 2014), the similarity in the disinhibited behavior and ERN responses doesn't necessarily indicate that abnormalities of error-processing observed in

psychopathic individuals are driven by externalizing features among psychopathic individuals. However, this close relation may indicate that impulsive-antisocial traits in psychopathy could be more involved in disruption of error processing. It remains important to point that individuals with high level of psychopathy are more likely to engage in externalizing behaviors (Marcus et al., 2019) and that compared to interpersonal-affective traits, impulsive-antisocial seems to be particularly related to the externalizing tendency (Patrick et al., 2005). Regarding the limitation of heterogeneity in the cognitive functioning pointed out by Baskin-Sommers and Newman (2014), it remains important to report that the characterization of psychopathic individuals, either through separate dimensional features or through typological classification, remains contested. Indeed, one of the most prominent models in the field of psychopathy that considers the unity of the etiology of psychopathic disorder is the response modulation hypothesis (RMH: Newman, 1998; Newman & Baskin-Sommers, 2011). The RMH refers to the automatic shift of attention from the implementation of ongoing goal-directed behavior to the self-evaluation of the behavior or response set (Wallace et al., 1999).The RMH suggests that psychopathy consists mainly of atypical abnormalities of attention reflecting an impairment in brain processes dedicated to the stream of stimulation (Newman & Baskin-Sommers, 2016). This putative deficiency leads psychopathic individuals to the incapacity to process peripheral stimuli, signaling the need to adapt behavior.

In line with the RHM, previous behavioral studies using Flanker's task or the Stroop task have reported the difficulty of psychopathic individuals to adapt their behavior in accordance with the requests of environment (Hiatt et al., 2004; Vitale et al., 2007; Zeier et al., 2009). As previously mentioned, the deficit in response modulation among psychopathic individuals may also be captured in passive avoidance learning or probabilistic learning tasks (Brazil et al., 2013; Budhani et al., 2006; Finger et al., 2008; Vitale et al., 2005; Von Borries et al., 2010). In these tasks, psychopathy induces an inability to adapt performances in accordance with the feedback established by peripheral sources. According to the conception of Newman and Baskin-Sommers (2011), in

psychopathic individuals, an early attentional bottleneck blocks the processing of peripheral cues, leading to inoperative capacity to change behavior from contextual information. Starting from this consideration, the RMH should only predict deficits in post-error response modulation and a disruptive Pe component; it predicts no deficit for the early process of error detection reflected by ERN, which appears during aware or unaware error commissions.

Despite the behavioral adaptation deficits observed in psychopathic individuals, electrophysiological studies on ERN and Pe components evoked by erroneous responses remain unclear for this population. The majority of studies exploring error-monitoring in psychopathic individuals reported a slight or no significant modulation of ERN and a significant reduction of Pe component compared to control group. However, when regarding separate dimensions or higher order dimensions, studies reported mixed results. The degree of severity of interpersonal-affective traits and impulsive-antisocial traits may well be the main confounding factor, as well as representing separate dimensions as assessed by the TriPM scale. In the same way, studies conducting in offenders with psychopathy are more likely to point the absence of abnormalities in the ERN component (Steele et al., 2016) whereas almost all studies conducted in individuals from a community sample describe reduced ERN amplitude (Pasion et al., 2016).

In a seminal review, Schulreich (2016) pointed out the specific modulation of ERN amplitude related to interpersonal-affective (PCL-R Factor-1) and impulsive-antisocial (PCL-R Factor-2) and the likely overlap with externalizing behaviors. The author concluded that inconsistencies among studies exploring ERN in psychopathy could be resolved by considering this heterogeneity through the dimensional construct of psychopathy. In a recent review, Clark and collaborators (2019) indicated that no studies exploring error monitoring in psychopathic individuals reported significant modulation of ERN and when a modulation is reported, it seems to be dependent on the dimension of psychopathy construct. New articles have been published since these reviews (Maurer et al., 2018; Paiva et al., 2020; Ribes-Guardiola et al., 2020; Van Heck et al., 2017; Zijlmans et al., 2019)

and the results of ERN regarding psychopathy are still too heterogeneous to conclude without a meta-analysis. With regards to psychopathy dimensions and modulation of the Pe component, literature reports have been even less homogeneous than for the ERN component. Previous studies using the PCL-R reported reduction of the Pe component associated with interpersonal-affective traits in criminal sample (Factor-1; Maurer, 2016b). In community sample, one study using TriPM scale reported significant reduction of the Pe amplitude (Ribes-Guardiola et al., 2020) associated with the Disinhibition dimension. This dimension is generally not considered to be related to Factor-1 of the PCL-R (Wall et al., 2015). **Finally, one last study screening participants from community sample according to the PPI scale and triarchic construct did not find a significant association between the Disinhibition dimension and the amplitude of the Pe component (Venables et al., 2018). However, in the results reported by Venable *et alii* (2018), a factorial analysis revealed a convergence of scales assessing Disinhibition with reduced Pe amplitude. The association with the Pe component was stronger with the disinhibition scale of the externalization spectrum inventory (ESI-DIS; Patrick et al., 2013) than with the PPI, the disinhibition scale of the multidimensional personality questionnaire (MPQ - D: Patrick et al., 2002) and the Gough socialization scale (SO scale; Gough, 1960).** To note, in opposition one study rather indicates an absence of significant Pe modulation in individuals with externalizing behavior (McDonals et al., 2019).

Considering the psychopathy construct, the proposition of Schulreich (2016) and Clark and collaborators (2019) remains to be clearly demonstrated with a meta-analysis grouping results across error-related components in psychopathy. These key points in the understanding of psychopathy disorder are precisely the aim of the current study. Beyond the discrepancies between studies, the absence of consistent results highlights the need for a systematic review and meta-analysis of the existing literature. Therefore, the main goal of the current study was to assess if psychopathy affects ERN and Pe components. In the first model, the psychopathy disorder

integrates the meta-analytic model as a unitary construct and implies that individuals with a high score on the PCL-R constitute a homogeneous group. However, as reported previously, the unitary conceptualization of psychopathy is associated with several limitations that consider the etiological heterogeneity among psychopathic individuals. Consequently, to assess the relevance of the dissociation according to distinctive etiological processes, the second model integrated psychopathy following separate dimensional structures and higher-order dimensions. The objective was to evaluate if the dimension of psychopathy could explain the particular modulation of ERN and Pe components during the process of error-related information. To achieve these goals, we conducted a systematic review of the literature and a meta-analysis. Given the close relationship between impulsive-antisocial traits and externalizing tendency, we hypothesized that only the impulsive-antisocial dimension of psychopathy will be associated with reduced ERN. Regarding the Pe component, despite heterogeneous results reported in the literature, we also hypothesized a link between Pe reduction and impulsive-antisocial dimension. Considering total score of psychopathy, we hypothesized that the total score of psychopathy will be associated with reduced Pe during post-error response modulation.

We also explored the response-locked component through the dimensional construct and the task-specific effect. Based on a recent review focusing on ERN and externalizing disorder (Pasion & Barbosa, 2019), we classified tasks according to “performance monitoring” (pooling Flanker, Simons and Stroop tasks) and “inhibitory control” (pooling Stop-signal and Go/Nogo tasks). We predicted a strong implication of the Impulsive-Antisocial dimension in the reduction of the ERN amplitude in tasks involving inhibitory control. Finally, regarding the possible dissociation between institutionalized and non-institutionalized individuals, we performed subsequent analyses on the sample.

The essential question addressed with this meta-analysis is the following one: to what extent are psychopathic individuals able to use or not their knowledge about error-commission to

adapt behavior and correct these errors? Psychopathic individuals meet in most cases the psychiatric standards for mental sanity. For experimental consideration, however, they failed to maintain efficient cognitive functioning (i.e., error-processing, learning) and cannot regulate behavior accordingly.

2. Materials and methods

2.1 Search

Systematic research was conducted following the recommendations of the Cochrane collaboration (Chandler et al., 2013) and the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines (Moher et al., 2009).

2.2 Identification

We conducted a systematic search in the PubMed and Web of Science databases using a term list, including words relative to *Psychopathy, Aggressor, Offender, Antisocial Personality Disorder, Event-related potential, Error related negativity, Error related positivity and Feedback related negativit*y. We used words with combination of thesaurus [MeSH Terms] related to medical subject headings description, [Other Term] related to keywords used in studies, and [Title/Abstract] related to words in title and abstract of studies. We conducted until March 2020 a search for full-length original articles published in English and peer-reviewed journals. No other search filters were applied.

2.3 Screening and Eligibility

Two investigators (WV, CN) independently screened the results according to the eligibility criteria, first on titles and abstracts and then on full-text articles. Studies were retained if they included patients or participants with psychopathy and if task design allowed a measure of the ERN or Pe components.

Thus, eligibility criteria used for inclusion were: (1) Original Data (2) Fully published (3) Peer reviewed (4) Inclusion of psychopathic individuals or psychopathy traits according to a clinical scale (5) Inclusion of an original measure of the ERN amplitude (6) Inclusion of an original measure of the Pe amplitude.

2.4 Quality assessment

The *Standard Quality Assessment Criteria for Evaluating Primary Research Papers* (Kmet, Cook, & Lee, 2004) was used to evaluate the quality of included studies. Two investigators (WV, CN) assessed the quality of included studies. The checklist was used in its original form, each study was assessed against the 14 items using a 3-point scale with 2 representing fully met, 1 representing partially met and 0 meaning a study did not meet the criterion. A summary score for each paper was calculated following the equation: (actual score/potential maximum score) *100. The potential maximum score considers items not applicable for some studies. The scores obtained were calculated as a linear score from 0 to 100 and divided into three categories: low (≤ 49), moderate (50–74), or high (≥ 75) quality studies.

Discrepancies between investigators were resolved by discussion with a third investigator (SG). For the total score of each study, a pre-consensus inter-rated agreement was calculated using the Cohen's weighted kappa score (Landis & Koch, 1977). Then, the inter-rated agreement obtained for the two investigators was qualitatively interpreted using standardized recommendations (Byrt, 1996). Note that the quality assessment was not used to determine study eligibility in the meta-analysis but is reported to inform the interpretation of findings.

2.5 Screening of participants

Regarding the clinical group, psychopathic individuals were screened using the Psychopathy Check List – Revised (PCL-R: Hare, 2003), the Psychopathy Check List – Screening Version (PCL-SV: Hart, Cox, & Hare, 1995), the Psychopathy Check List – Youth Version (PCL-YV:

Forth, Kosson, & Hare, 2003), the Self-Report Psychopathy Short-Form (SRP-SF: Paulhus et al., 2009), the Young Psychopathy Inventory (YPI: van Baardewijk et al., 2010) and the Multidimensional Personality Questionnaire - Brief Form (MPQ – BF: Patrick et al., 2002) and the Triarchic Psychopathy Measure (TriPM: Patrick et al., 2009)

The development of tools for assessing psychopathy is traditionally based on dimensional models (Benning et al., 1993; Hare, Harpur et al., 1990; Lilienfeld & Widows, 2005). As indicated earlier, the model comprises interpersonal-affective (Factor-1), impulsive-antisocial (Factor-2) and 4 facets related to interpersonal, affective, lifestyle and antisocial traits (Hare & Neumann, 2006). The PCL-R is a standard tool to assess psychopathy according higher order dimensional construct.

The PCL-R is a semi-structured interview scale dedicated to identifying personality traits and behavior related and higher-order dimensions. The PCL-R is composed of 20 items grouped into Factor-1 (items 1-8, 16) and Factor-2 (items 3, 9-15, 18, 19). Factors are composed with four facets (Interpersonal and Affective for Factor 1 and Lifestyle and Antisocial related to Factor 2). Response format is a 3-point Likert scale, and total score can range from 0 to 40.

The PCL-R and other versions as PCL-YV or PCL-SV are similar to PCL-R in terms of factor structure (Forth et al., 2003). In the same way, the SRP-SF also fits with the PCL-R factor structure (Cooke et al., 1999; Neumann & Pardini, 2014). However, measures between YPI and PCL-YV designed for youth population, seem to be more distinctive (Chauhan et al., 2014; see limitation section).

A second model used for assessing psychopathy refers to the separate dimensional constructs of the triarchic model (Patrick et al., 2009). This model integrates three distinct phenotypic constructs: Disinhibition, Boldness, and Meanness. The PCL-R (Factor-1) is associated with boldness and meanness phenotypic constructs, but not with the disinhibition construct, whereas the Disinhibition construct is similar to the PCL-R (Factor-2; Wall et al., 2015). The Boldness subscale is also related to the fearless dominance (FD) construct indexed by the scores on

the PPI-R. These scores are considered as the operationalization of a similar construct (Drislane et al., 2014). To note, relations between the Meanness dimension, Disinhibition dimension, and higher-order dimension of PCL-R (Factor-2) are related to the externalization features of psychopathy (Patrick et al., 2005).

For the diagnosis of psychopathy with PCL-R, an often-used cut-off is a total score of 30 in United-State and 25 in Europe. In the current meta-analysis, 6 studies using the PCL-R in offenders or incarcerated individuals reported a cut-off between 25 and 30 to diagnose psychopathic individuals (Brazil et al., 2009; 2011; Maurer et al., 2016a, 2016b; Munro et al., 2007; Von Borries et al., 2010). For the PCL-SV a classical cut-off is a total score of 18 to diagnose psychopathy. In current analysis, the study from Bresin and collaborators (2014) included 14 individuals with PCL-SV score above 18. The remaining sample of this study was screened with the PCL-SV without cut-off (score range from 1 to 23 - authors also indicated that 51% had a diagnosis of antisocial personality disorder). Regarding the YPI scale, the cut-off should be based on standard deviation or derived from the receiver operating characteristic (ROC) analysis. Both studies using YPI and included in current analysis reported only a descriptive analysis of the scoring (Maurer et al., 2018; Zijlmans et al., 2019). The remaining 6 studies screened individuals from community sample with triarchic contruct scales (Pasion & Barbosa, 2016; Ribes-Guardiola et al., 2020; Paiva et al., 2020; Venables et al., 2018), the MPQ – BF (Heritage, & Benning, (2013) and the SRP-SF (van Heck et al., 2017). Considering the total score of psychopathy and dimensional construct, we have chosen to include all studies in the analysis. We mediated the analysis by adding moderators, according to the type of scale. To do so, when analyzing the higher-order dimensional construct and the separate dimensional constructs, we performed supplementary analyses: in one, the mixed model included only the PCL-R + SRP-SF dimensions and in the other one, the mixed model included only the TriPM + PPI dimensions. For the analysis including TriPM and PPI, we pooled the boldness dimension with fearless dominance, and the disinhibition dimension was associated with impulsive-

antisocial. For the PCL-R + SRP-SF analysis, the interpersonal-affective dimension was associated with affective-callousness, and impulsive-antisocial with lifestyle-antisociality.

Finally, in an analysis presented in the Supplementary material, we explored age as a covariate, considering that previous studies reported modulation across age in the response-locked component of error monitoring (Mathewson et al., 2006).

2.6 Data collection process and data items

We extracted ERN and Pe measures from the included studies. Time window for amplitude measurement and electrodes cluster site were also reported. We collected the number of participants, socio-demographic and clinical characteristics of participants (age, sex, clinical diagnoses, incarceration status, co-morbidity), as well as information on experimental designs (tasks, stimuli). When data were missing or not fully reported, we contacted the corresponding author for further information. We contacted 8 authors in order to retrieve data and 3 have sent us additional results (Bresin et al., 2014; Pasion & Barbosa, 2016; Paiva et al., 2020).

2.7 Data extraction and methods of meta-analysis model

We conducted quantitative analyses using R project with Metafor package, version 3.2.1 (Viechtbauer, 2010). Firstly, we computed a random effect model on ERN and Pe effect size. This random effect model provided a pooled Cohen's *d* effect size over total score of psychopathy among studies.

To explore the psychopathy dimensions regarding factors of construct, we used a mixed-model effect corresponding to a random effect model with a moderator variable. To account for the distribution of these dimensions across the psychopathic population, we performed analyses based on correlational coefficients after Z – Fisher transformations. This strategy avoids the artificial creation of a subgroup inherent to Cohen's d calculation. We performed subsequent analyses with a

moderator variable regarding the sample (Clinical (PCL-R>25) vs. Community) or regarding scale (PCL-R vs. TriPM).

We based our primary outcome on the ERN and Pe components and pathological scoring. We extracted the ERN and Pe results for each group or the r coefficient or the F value resulting from the ANOVA. The F values, with only one degree of freedom for the numerator, were the only ones that could be used for conversion to an effect size estimate. Raw data and r coefficient values were chosen in priority to calculate the effect size (see supplementary material for data).

When raw data were available (mean and SD), Cohen's d effect size was calculated between the control group and test group. Z-Fisher transformations for mixed models were computed mainly from the original r coefficient between the psychopathic dimension and the amplitude of the components.

In most cases, we extracted data at midline frontal or central sites (included Fz or Fcz or Cz electrodes according to the 10-20 international system).

Following the data extraction, we computed a random effect model based on Cohen's *d* with Maximum Likelihood estimator on ERN and Pe effect size. We used random effect model in order to take into account between-study variability and therefore provided a more conservative estimate of composite effect size. Results of model were reported according to $I^{\wedge 2}$ for residual heterogeneity or unaccounted variability, relative risk [95% CI], Q-test for residual heterogeneity, and *p* value for significance. Finally, Egger's test (weighted regression models with multiplicative dispersion) was used to test funnel plot asymmetry and potential publication bias.

For exploring psychopathy dimension, we used mixed effect model. The Models reported the results according to $I^{\wedge 2}$ for residual heterogeneity or unaccounted variability, relative risk [95% CI], QE-test for residual heterogeneity, QM-test for the omnibus test of coefficients, and *p* value for significance. Egger's test was also performed to test funnel plot asymmetry and potential publication bias.

In the analysis presented in the Supplementary material, we reported one model, with aging as a variable in a mixed effect model. The moderator variable included 2 dimensions (Adults vs. Youths). We also reported equivalent analyses based on Cohen's *d* regarding analyses based on the dimensional construct of the psychopathic disorder.

Table.1: *Included studies*

Effect sizes included in the analysis were calculated from the mean and standard deviation (SD), *F*-test value, and sample size by entering values into effect size calculator (Lipsey & Wilson, 2001) and collecting effect size and variance of *d* (Vd). We also calculated effect size on the correlation coefficient (*r*) by converting them in Cohen's *d* and collecting effect size and Vd (Cohen, 1988; Rosenthal et al., 1994).

Effect sizes and Z fisher transformation were reported according to the polarity of the effect and deflection of the component: Negative Cohen's *d* or Z-fisher for ERN = Increase amplitude of the component; Positive Cohen's *d* or Z-fisher for ERN = Decrease amplitude; Negative Cohen's *d* or Z-fisher for for PE = Decrease amplitude; Positive Cohen's *d* or Z-fisher for PE = Increase amplitude. When different kinds of stimuli were used within a task (Munro et al., 2007), we selected letter stimuli in order to maintain homogeneity between studies.

3. Results

3.1 Selection of studies

The primary search yielded 206 results. The fig.1 depicts the flow chart diagram related to the systematic research. Among the 206 abstracts assessed for screening, exclusion criteria removed 19 duplicates and 162 abstracts. Manual search from the reference lists of included studies have completed the systematic research and finally added 1 study. The remaining 26 studies were then

assessed for eligibility based on full-length articles. Finally, 10 articles were excluded. Sixteen articles were then included in the meta-analysis. These 16 articles reported 36 data-sets for ERN component and Pe component analysis**.**

Figure.1: *Flow Chart of the included studies.*

3.2 Characteristics of included studies

Experimental paradigms used to measure ERN and Pe amplitude included Eriksen flanker task, Go/NoGo task, Simon's task, Learning task and Empathic task.

The meta-analysis on ERN/Pe included 940 participants in the test group. Among participants in the test group, 104 were diagnosed with psychopathy (PCL-R>25 or PCL-R>30), 418 offenders were screened with psychopathy traits. The remaining sample is composed of 418 participants from community sample and screened with psychopathy traits. The sample test was composed of 634 males and 331 females. The mean age for the test sample was M = 30.66 SD = 8.45 (Adult group: M = 33.53 SD = 6.65; Youth group: M = 19, SD = 2.9). The control group included 112 participants (M = 35.17 SD = 9.58).

Participants have different status (incarcerated, institutionalized or free). Included studies reported comorbidity and excluded participants with historic of brain injury or other psychiatric condition. The ERN was generally measured at midline central electrodes sites in time windows ranging from 0ms to 250ms post-response. The Pe component was extracted from a centro-parital cluster in time windows ranging from 75 to 500 ms post-response.

3.3 Quality assessment of the included studies

The overall quality of the included studies obtained after consensus between the two assessors (WV & CN) was high (mean score = 84.66 %, SD = 8.11, ranging from 72.73 to 95.45 %).

Interpreter pre-consensus agreement for the global score was ‘strong’ according to the weighted Cohen’s kappa score interpretation (mean k = 0.88, SD = 0.09, ranging from 0.67 to 1). The score and Cohen's weighted kappa related to checklist for assessing the quality of quantitative studies are available in supplementary material.

3.4 Influence of psychopathy on the ERN and Pe amplitudes – analyses based on Cohen’s d estimate.

The analysis of the ERN amplitude regarding the total score of psychopathy included 14 data sets. The meta-analysis reported a slight but significant reduction of the ERN amplitude for the group test (Cohen's d = 0.1802; 95 % CI 0.06 to 0.29; $p < 0.05$; I^2 = 41% see Fig.2A). Q-test reported weak heterogeneity (Q (df = 13) = 24.87, $p < 0.05$). Regarding TriPM vs. PCL-R and associated scales, with the addition of a moderator, the analysis remains significant (QM (df = 1) = 8.81, $p < 0.05$; I^2 = 0%) with homogeneity (QE (df = 12) = 16.08, $p > 0.05$). The model reported a reduction of the ERN amplitude for both scales (TriPM Cohen's d = 0.25; 95% CI 0.08 to 0.42; $p <$ 0.05; PCL-R Cohen's d = 0.10; 95% CI 0.01 to 0.19). There was no significant effect on Egger’s test, suggesting a symmetrical forest plot and no significant publication bias [t = 0.63; p = 0.54; see Fig.4A]. Analysis of the Clinical vs. Community sample revealed no effect of the moderator (QM (df = 1) = 1.6251, p = 0.20; I^2 = 28%). For the Pe amplitude, the RE model included 8 data sets. The model reported a significant reduction of the Pe amplitude for the group test (Cohen's d = -0.21; 95% CI -0.40 to -0.01; $p < 0.001$, I^2 = 79%; see Fig-2B) but homogeneity was not found (Q (df = 7) = 40.0620, $p < .01$). Egger’s test was non-significant [t = -0.73; p = 0.49].

Figure.2: *Forest plot for the ERN and Pe component.*

3.5 Influence of interpersonal-affective and impulsive-antisocial dimensions on Pe amplitude: Analysis based on Z-Fisher estimate.

Analysis including studies exploring the Pe modulation through psychopathy dimensions included 9 data sets. The meta-analytic model was homogenous (QE (df = 6) = 13.07, $p = 0.054$), but effect of moderator was not significant (QM (df = 1) = 0.0439, $p = 0.83$). At least, no difference was found between dimensions (Interpersonal-Affective: Z-Fisher Estimate = -0.0613; 95% CI -0.17 to 0.05; $p = 0.31$ / Impulsive-Antisocial: Z-Fisher Estimate = 0.0163; 95% CI -0.16 to 0.13; $p = 0.83$). There was no significant effect on Egger's test [$t = 1.56$; $p = 0.16$].

3.6 Influence of interpersonal-affective and impulsive-antisocial dimensions on ERN amplitude – analyses based on Z-Fisher estimate.

The analysis of the ERN amplitude and dimensions of psychopathy included 18 data sets. The omnibus test indicated a significant effect of the moderator (QM (df = 1) = 5.40, $p < 0.01$; $I^{\wedge 2} = 2.67\%$ see Fig.3A) with homogeneity (QE (df = 16) = 21.22, $p = 0.17$). The mixed effect model indicated a significant reduction of the ERN amplitude for the Impulsive-Antisocial dimension (Z-Fisher Estimate = 0.13; 95% CI 0.02 to 0.23; $p < 0.01$). The Interpersonal-Affective dimension was not significant (Z-Fisher Estimate = -0.01; 95% CI -0.09 to 0.06; $p = 0.7$). There was no significant effect on Egger's test [$t = -0.15$; $p = 0.88$; see Fig.4B].

Regarding individuals from the community sample and individuals with a clinical diagnostic of psychopathy, with the addition of a moderator, the model remains significant (QM (df = 1) = 13.01, $p < 0.01$; $I^{\wedge 2} = 0.06\%$ see Fig.3B) with homogeneity between studies (QE (df = 14) = 13.67, $p = 0.47$). Reduction of the ERN amplitude for Impulsive-Antisocial dimension was significant in the community sample (Z-Fisher Estimate = 0.2543; 95% CI 0.10 to 0.40; $p < 0.01$) but not in the clinical sample (Z-Fisher Estimate = 0.0448; 95% CI -0.10 to 0.19; $p = 0.12$). Regarding the Interpersonal-Affective dimension, a non-significant modulation of the ERN amplitude was reported in the clinical sample (Z-Fisher Estimate = -0.0296; 95% CI -0.13 to -0.07; $p = 0.57$) and

in the community sample (Z-Fisher Estimate = 0.0334; 95% CI -0.11 to 0.18; p = 0.66). No significant publication bias was reported [t = -0.81; p = 0.43; see Fig.4C].

Figure.3: *Forest plot for the ERN related to interpersonal-affective (Factor-1) vs impulsive-antisocial (Factor-2) and Forest plot for the ERN related to Factors and Diagnostic*

3.7 Influence of interpersonal-affective (Factor-1) and impulsive-antisocial (Factor-2) higher-order dimensions on the ERN amplitude – Analyses include the PCL-R + SRP-F score.

The analyses that included only studies using the PCL-R+SRP-SF score revealed no significant effect of the moderator (QM (df = 1) = 0.42, p = 0.51) and homogeneity between studies (QE (df = 16) = 5.30, p = 0.72). Neither the Impulsive-Antisocial (*Z-Fisher estimate* = 0.0453; 95% CI -0.09 to 0.18; p = 0.51) nor the Interpersonal-Affective dimension (*Z-Fisher estimate* = -0.0246; 95% CI -0.19 to 0.07; p = 0.61) were significant. Egger's test reported no significant publication bias [t = -0.68; p = 0.51].

3.8 Influence of the Disinhibition and Boldness dimension on ERN amplitude – Analyses include the TriPM + PPI score.

The analyses that included studies using the TriPM+PPI score revealed a significant effect of the moderator (QM (df = 1) = 8.78, p < 0.01; $I^{\wedge 2}$ = 0.03%) with homogeneity (QE (df = 6) = 6.75, p = 0.36). The analyses revealed a significant modulation effect for the Disinhibition dimension (Z-Fisher estimate = 0.2591; 95% CI 0.08 to 0.43; p < 0.01) and no effect regarding the Boldness dimension (Z-Fisher estimate = 0.0041; 95% CI -0.11 to 0.12; p = 0.94). Egger's test reported no significant publication bias [t = -0.27; p = 0.79; see Fig.4D].

3.9 Influence of the Interpersonal-Affective and Impulsive-Antisocial dimensions on the ERN amplitude regarding task effects– Analyses based on Z-Fisher estimate.

The current model included a moderator with factors related to the tasks effects (Monitoring - Interference (Flanker + Simon tasks) vs. Inhibitory control (Go/NoGo + Stop-signal task). The analyses revealed a non-significant effect of moderator (QM (df = 1) = 2.99, $p = 0.08$; $I^{\wedge 2} = 8.25\%$) and no difference between task groups (Monitoring - Interference tasks: Z-Fisher Estimate = -0.0374; 95% CI -0.13 to 0.06; $p = 0.46$ and Inhibitory control tasks: Z-Fisher Estimate = 0.1041; 95% CI -0.01 to 0.22; $p = 0.08$). Regarding the psychopathy dimensions from all scales, with addition of moderator, the effect remains non-significant (QM (df = 1) = 5.67, $p = 0.12$; $I^{\wedge 2} = 12\%$) with heterogeneity (QE(df = 14) = 23.78, $p = 0.04$). Nevertheless, there is a significant reduction of the ERN amplitude for the Impulsive-Antisocial dimension in Inhibitory control tasks (Z-Fisher Estimate = 0.2013; 95% CI 0.02 to 0.37; $p < 0.05$). No other effect was found (Impulsive-Antisocial x Monitoring - Interference tasks: Z-Fisher Estimate = 0.1044; 95% CI -0.10 to 0.31; $p = 0.32$ / Interpersonal-Affective x Monitoring - Interference tasks: Z-Fisher Estimate = -0.0895; 95% CI -0.23 to 0.05; $p = 0.22$ / Interpersonal-Affective x Inhibitory control tasks: Z-Fisher Estimate = 0.1111; 95% CI -0.06 to 0.28; $p = 0.20$).

Figure.4: *Funnel plots for meta-analytic models.*

4. Discussion

We have specifically designed current analysis for clarifying empirical finding from the existing literature regarding the error-processing component in psychopathic individuals. It should first be noted that the assessment of the quality of studies indicated that, overall, they are of high quality; therefore, the findings of the current meta-analysis are of interest.

In the following section, we reported explanations in sub-sections corresponding to results section. In order to simplify the expression and interpretation of effects for each analysis, we provided an explanation of effects size according to Cohen (2008): regarding the significant Z-Fisher effects reported in mixed models, the distribution varied from 0.15 to 0.25. The desired Z-Fisher effects are between 0.2 and 0.45. More precisely, an interpretation of Cohen (2008) considered Z-Fisher effects between 0.1 and 0.2 as small effects, 0.24 and 0.33 as medium effects, and larger than 0.37 as large effects. For Cohen's *d,* in a random effect model, the distribution varied from 0.18 to 0.25. According to Cohen (2008), these effects are small. In summary, mixed models showed strong and robust effects with homogeneity between studies regarding $I^{\wedge 2}$ and QE-test, whereas results reported with the random effect model seem to be weaker.

4.1 Total score of psychopathy ~ ERN:

The main results of the random effect model integrating a total score of psychopathy indicated that psychopathic individuals displayed lower ERN amplitudes following error commission than controls. These results should be considered with caution given the small effect size (Cohen's $d = 0.18$) and the significant heterogeneity found between studies ($I^{\wedge 2} = 48\%$). Despite the limitations, this result remains possibly in contradiction with the RMH, which is predicting an efficient early process of error detection reflected by ERN (Steele et al., 2016). It cannot be excluded that this reduction should be led by externalizing traits in psychopathy disorder. However, it is worth mentioning that the limitation of the unitary construct resulting from the putative implication of externalizing traits in psychopathic individuals does not necessarily lead to the overall rejection of the response modulation hypothesis. The RMH predicts a general abnormality in selective attention. By evaluating psychopathy according to the interaction of externalizing traits, several studies reported homogeneity among psychopathic individuals and reported that externalizing features may mediate the phenotype of psychopathy disorder. For example, in individuals with externalizing features (for review see Pasion et al., 2019), an atypical

modulation of the N2 component was related to attentional selectivity but, interestingly, was unrelated to the level of psychopathy (Munro et al., 2007). Indeed, several studies reported a response modulation deficit specific to psychopathic individuals and low externalizing traits (Zeier & Newman, 2013) or specific neurobiological dissociation between externalizing traits and psychopathy regarding attentional control (Rodman et al., 2016).

These results point to a specific response modulation process underlying an atypical functioning in psychopathic individuals without externalizing traits. In the current analysis, the impossibility to mediate models with the addition of externalizing traits evaluated in the sample is a limitation. This limitation constitutes one of the main take-home messages of the present work; the systematic screening of externalizing / internalizing disorder in combination with psychopathy ratings is one of the improvements for an advanced comprehension of the atypical error-monitoring process in psychopathic individuals.

4.2 Dimension of psychopathy ~ ERN:

Regarding dimensions of psychopathy, the mixed-model indicated that the reduction of ERN amplitudes was led by impulsive-antisocial traits, whereas interpersonal-affective traits do not report significant modulations of ERN amplitudes. This result depicts a moderate effect (Z-Fisher Estimate = 0.13) with good homogeneity between studies included in the analysis ($I^{\wedge 2}$ = 2.67%). In agreement with the starting hypothesis, this result is another indication in favor of the implication of the externalizing traits in atypical error-processing in psychopathy.

This result is also in line with evidence for an etiological heterogeneity among psychopathic individuals. Several studies describe, relative to higher order dimensions, the etiological heterogeneity of psychopathy construct for emotional processing (Hicks & Patrick, 2006; Schienle et al., 2017; Venables et al., 2015), attentional processing (Baskin-Sommers et al., 2012; Verona et al., 2012) and conditioning (Veit et al., 2013).Despite limitation regarding externalizing traits, the higher order dimensions remain useful for studying error monitoring in psychopathy. However, all

studies did not report systematically the ERN responses relative to these dimensions of psychopathy. Among studies with dimensional analysis, Bresin and collaborators (2014), characterized individuals on PCL-R Factor-1 are by a superior monitoring of errors. It is suggested that these characteristics could be practical to pursue some specific behavior related to psychopathy disorder (e.g., manipulate someone or premeditate action (Verona, 2016)). Pasion and collaborators (2016) also describe this ERN modulation in individuals scoring on interpersonal-affective as individual with high social efficiency. Results of the current analysis are in line with previous studies and suggest that error monitoring seems to be efficient in individuals with high scores on affective traits of psychopathy.

In sum, it seems that psychopathic individuals with impulsive-antisocial traits displayed a general deficit in executive function involving monitoring and error processing whereas psychopathic individuals with interpersonal-affective traits tend to maintain efficient cognitive functioning through efficient attentional control and error processing (Baskin-Sommers et al., 2009).

To conclude, it is important to notice that the normal functioning of error processing through the ERN component cannot be reduced to the width of the negative deflection. A large panel of psychopathology disorders displayed negative or positive modulations of the ERN component (Olvet & Hajcak, 2008). Larger amplitude of the ERN component can also be linked to abnormal behaviors (Holmes & Pizzagalli, 2008). Indeed, patients with depression or obsessive-compulsive disorders show larger ERN amplitudes associated with abnormal action monitoring or increased sensitivity to mistakes or negative feedback (Gehring et al., 2000; Riesel, 2019; Steffens et al., 2001).

4.3 Scales and dimension of psychopathy ~ ERN:

By adding scales as moderators (TriPM vs. PCL-R and associated scales) in the random-effect model analyzing the ERN modulation, homogeneity was found between studies ($I^{\wedge 2} = 0\%$)

and a strong effect size was reported regarding TriPM scale (Cohen's $d = 0.25$) and relative small effect size regarding the PCL-R (Cohen's $d = 0.10$).

The addition of a moderator focusing on scales in the mixed-model reported interesting results. The mixed-model integrating psychopathy dimension through the TriPM+PPI scale depicted a strong effect (Z-Fisher estimate = 0.26) with homogeneity ($I^{\wedge 2} = 0.03\%$) related to the Disinhibition dimension. Whereas mixed-model grouping studies according to scales revealed that studies assessing psychopathy with unitary or higher-order dimensions (PCL-R+SRP-SF) report no significant modulation of the ERN across dimensions. This result points in the direction of a fundamental limitation regarding screening of psychopathy traits in community and incarcerated samples. While all studies exploring psychopathy traits in criminal sample used the PCL-R, all studies using TriPM explored psychopathy traits in community sample. The combination of different scales for screening psychopathy over the same sample seems to be a further possibility pointed out by the current analysis. The combination of the PCL-R assessments with measures such as the TriPM in incarcerated samples in future studies could help to clarify if such measures exhibit similar or different associations with the ERN. Combining both measures might also be useful to distinguish effects specific to clinical ratings of psychopathy vs. externalizing/disinhibition. The use of the TriPM scale in combination with other scales from studies exploring psychopathy among incarcerated or criminal samples can be particularly relevant. The TriPM was developed as an integrative framework to help integrate findings across research studies and reconcile differing conceptions of psychopathy (Drislane et al., 2014). Thus, its use in combination with the PCL-R seems to be a good recommendation for future studies exploring psychopathy among incarcerated or criminal samples.

4.4 Community and Criminal sample ~ ERN:

As previously mentioned, the majority of studies reporting ERN modulation included individuals from community samples. The current model confirms this fact with a significant

reduction of the ERN amplitude related to the Impulsive-Antisocial dimension in community samples, but not in the clinical samples. The meta-analytic model reported a strong effect (Z-Fisher Estimate = 0.25) with high homogeneity ($I^{\wedge 2} = 0.06\%$). This central result of the current analysis raises a theoretical issue: whereas individuals from criminal samples with high levels of psychopathy are more likely to engage in externalizing behavior, it is reasonable to think that a putative implication of externalizing features among psychopathic individuals should lead to a reduction in ERN amplitude, which is not the case. Furthermore, regarding the impulsive-antisocial dimension, which seems to be particularly related to the externalizing tendency (Patrick et al., 2005), this reduction should have been reinforced. As mentioned above, we expected that the modulation of the ERN amplitude among psychopathic individuals would be partly related to externalizing features of psychopathy through the close relation between externalizing disorder and the Impulsive-Antisocial Factor-2 of the PCL-R. Also, the PCL-R total score, PCL-R Factor 1 and PCL-R Factor-2 scores usually correlate positively with each other. Despite the significant correlations between interpersonal-affective (PCL-R Factor-1), impulsive-antisocial (PCL-R Factor-2) and the total score of the PCL-R, the externalizing disorder related to PCL-R Factor-2 seems to have no relation with psychopathy, as evaluated by the total score of the PCL-R. Additionally, no relation was found with the Interpersonal-Affective dimension. This may explain why the ERN amplitudes varied as a function of psychopathy dimensions, but in opposite directions.

This fact leads us to believe that a higher-order dimension in individuals with diagnosed psychopathy is not relevant to explain error processing due to externalizing disorder comorbidity. Indeed, an atypical comorbidity pattern between psychopathy and externalizing disorder is described in the literature. Contrary to the classic pattern of behavior disorder that shows externalizing symptomatology in conjunction with internalizing symptomatology (Vollebergh et al., 2001), psychopathic individuals are exceptions to this rule. In psychopathic disorder, externalizing tendencies are manifest without internalizing symptomatology. From a clinical perspective, the

heterogeneity of the psychopathy construct could point towards an explanation related to the uncoupled externalizing and internalizing dimensions. Indeed, high level of psychopathy and impulsive-antisocial features are more likely to lead to externalizing behaviors (Marcus et al., 2019) whereas interpersonal-affective features prevent from engaging in internalizing behavior (Blonigen et al., 2005) with a negative association to specific symptoms of internalizing psychopathology (Willemsen & Verhaeghe, 2012). The interpersonal-affective traits in psychopathic individuals seem to protect them against the possible emergence of internalizing symptomatology. This possibility is in line with the non-unitary construct of psychopathy. Individuals with a high level of externalizing features of psychopathy overlap with the classic pattern of behavior disorder, while individuals with a high level of interpersonal-affective features draw the outline of a clinical picture *sui generis*. From an etiological perspective, the reasons explaining the uncoupled externalizing and internalizing dimensions in individuals with psychopathic disorder are also reflected and developed through the hypothesis regarding the alternative processes in the emergence of psychopathy (the "two-process theory of psychopathy" of Patrick & Bernat, 2009) and the "dual-pathway model of psychopathy" of Fowles & Dindo, 2009).

4.5 Task effect and aging ~ ERN:

Regarding tasks (Monitoring - Interference (Flanker + Simon tasks) vs. Inhibitory control (Go/Nogo + Stop Signal task), the model reports no significant effect of moderator, and homogeneity was not found, even after the addition of a moderator regarding dimensions of psychopathy. Nevertheless, a significant reduction of the ERN amplitude was reported for the Impulsive-Antisocial subgroup for Inhibitory control tasks (Z-Fisher Estimate = 0.20). However, this result should be taken with caution given the absence of homogeneity and the moderator effect in the main analysis.

Overall, studies included in current meta-analysis used in the same proportion the Inhibitory control and the Monitoring – Interference tasks. The homogeneity between studies and the use of

Inhibitory control task in seven studies (Go/Nogo + Stop Signal task) can explain the strong effects observed for the Impulsive-Antisocial subgroup in the current meta-analysis. These observations are in strait line with previous studies reporting specific links between the Impulsive-Antisocial traits and the ERN as measured in Inhibitory control task (Pasion et al., 2019; Ribes-Guardiola et al., 2020). By the way, this relative homogeneity is also a limitation regarding the possible dissociation between cognitive control tasks and emotional tasks observed in psychopathy (Munro et al., 2007). Indeed, previous studies have reported that deficits in attentional processing during emotional perception are linked to the severity of interpersonal affective traits (Sadeh & Verona, 2012; 2008). This point figures another potential questioning regarding task effects. The question should be clarified with studies including affective stimuli during the Inhibitory control task as in Munro *et alii* (2007). In this way, it will possible to identify if inhibitory deficit could be limited to Impulsive-Antisocial trait or if it taps also more closely onto the other psychopathy dimension (Affective-Interpersonal).

Considering the supplementary analyses, the aging moderator revealed no significant impact on ERN and Pe modulations. However, a tendency for reduced ERN amplitude was observed in the adult group. These results are in line with previous findings that report a reduction of the ERN amplitude in older adults compared to the youth population (Mathewson et al., 2005; Themanson, Hillman, & Curtin, 2006).These observations support previous reviews in the field related to psychopathy dimension and error-processing (Schulreich, 2016).

4.6 Total score of psychopathy ~ Pe:

As reported by the random effect model, the reduction of the Pe amplitude seems to be more significant in psychopathic individuals compare to control group. Unfortunately, insufficient data were available to compute a random effect model with homogeneity between studies. Considering that the Pe component reflects error-awareness (Overbeek et al., 2005), a more pronounced deficit

observed in psychopathic individuals could indicate a close relation with attentional and motivational processing deficit.

Previous research characterized psychopathy disorder by fundamental disturbances in attentional processes (Hiatt & Newman, 2006; Kosson & Newman, 1986) and monitoring (Budhani et al., 2006). The RMH argues that psychopathic individuals are thus unable to process a peripheral target related to the primary focus. Once their attention is engaged in goal behavior, they fail to relocate attention for capturing other relevant information (Patterson & Newman, 1993). This reduced ability to shift attention leads to a subsequent failure to adapt the dominant behavior.

Traditionally, the response modulation theory of psychopathy (Newman & Baskin-Sommers, 2011) suggests that psychopathy consists mainly of attentional deficits. Abnormalities in attention processing and monitoring could also be implicated in deficits observed in psychopathy. With deficient attentional processing, an individual is going to ignore the relevant information needed to correctly accomplish and evaluate behavior. Consequently, the incapacity to process peripheral stimuli signalling inappropriate responses seems to be associated with disturbance in monitoring, when current behavior requires evaluation to be correctly accomplished. As previously mentioned, the response modulation theory predicts disturbance in post-error processing (Pe component; Steele et al., 2016). Results obtained in the current meta-analysis are closed to the model prediction. The model of response modulation fit with current analysis regarding the Pe component. Indeed, psychopathic individuals displayed a significant reduction of Pe component reflecting disturbance in post-error processing while ERN effect seems to be more slightly. However, results from models that include a total score of psychopathy remain weak in terms of effect size, and heterogeneity was reported between studies.

4.7 Dimension of psychopathy ~ Pe:

As previously mentioned, the results for psychopathy dimension and Pe amplitude are opposite to some extent. Consequently, moderator effect in the mixed model was not found despite

the homogeneity of studies. However, in the present analysis, two studies did not find a specific association the amplitude of the Pe component and either the impulsive-antisocial or the interpersonal-affective traits (Maurer et al., 2018; Zijlmans et al., 2019), and one study reported a specific association with interpersonal-affective traits of the PCL-R (Maurer, 2016b). Regarding the triarchic construct, one study using TriPM reported an association with the Pe amplitude and the Disinhibition dimension (Ribes-Guardiola et al., 2020), and one study using the PPI-Disinhibition facet did not reveal this association (Venables et al., 2018). **As previously mentioned, in the exploratory factor analysis grouping Disinhibition scales (ESI-DIS, MPQ-DIS, PPI-DIS), Venables *et alii* (2018) reported a significant association between the Disinhibition scale factor and the reduced Pe amplitude.**

These results state the difficulties to study post-error processing among psychopathic individuals and the divergence across scales for assessing psychopathic disorder as a unitary/higher-order or as a separate dimensional construct. Indeed, whereas PCL-R seems to indicate a specific association between Pe amplitude and interpersonal-affective traits (Factor-1), the TriPM scale indicates associations with the disinhibition dimension, which is generally considered as similar to the PCL-R (Factor-2), and with the impulsive-behavioral style facet (Wall et al., 2015; Venables et al., 2014). **In line with the results from Venables *et alii* (2018) revealing an association between Disinhibition scales and Pe amplitude, Ribes-Guardiola *et alii* (2020) suggested that the pattern of the post-error processing reflected by the Pe amplitude could be related to the disinhibition dimension, in accordance with the pattern of the P3 component.** This statement highlights a potential implication of the Pe component in the processing of motivational events, such as errors in performance, and suggests a putative explanation regarding dysfunction in post-error processing in individuals with externalizing tendency.

However, the results from the current meta-analytic model do not allow to established that the pattern of post-error processing reflected by the Pe component could be described exactly as a

P3 component related to the motivational significance of the error. Pasion *et alii* (2018) have previously reported the reduced amplitude of P3 component as a potential neurobiological marker of externalizing disorder. In the current meta-analysis, we did not find a significant association between psychopathy dimension related to externalizing disorder and modulation of the Pe component. The current result appears to be rather consistent with previous work reporting the absence of significant modulation of the Pe component in individuals with externalizing traits (McDonals et al., 2019). To conclude, these results emphasize the need for further studies exploring the functional implication of the Pe component through the dimensions of psychopathy.

4.8 Psychopathy and others attentional markers:

Attentional processes and monitoring are closely linked. When attention is engaged, monitoring can allow the relocation of the attention focused on the goal-directed behavior, if potentially relevant information is available in the environment. Combined with attentional processes, monitoring plays a role in decision-making (Taylor et al., 2007), affective modulation (Keil et al., 2005; Schupp et al., 2003) and error monitoring (Xiao et al., 2015). Attentional processing has been largely explored in psychopathy disorder. For example, a meta-analysis comparing patients with psychopathy and healthy controls was already realized on the P3 component for examining attentional impairments in psychopathy. The P3 component has traditionally been associated with attentional processing disturbance in psychopathy. Gao and Raine (2009) have shown that there are selection attention processing discrepancies between patients and controls, as revealed by a reduced P3 amplitude and by a longer P3 latency (Gao & Raine, 2009).

In the same way, several evoked responses are related to cognitive disturbance in psychopathy (for review, see Clark et al., 2019). For example, the late positive component (LPP) is known to be modulated by arousal and attentional processing and reflects a facilitated processing of emotional stimuli (Schupp et al., 2004a). For instance, threatening faces elicited larger LPP than other facial emotions in controls (Schupp et al., 2004b). Controls with higher psychopathic traits, as

compared to those with lower psychopathic traits, displayed smaller LPP amplitude when presented with arousal stimuli (for review, see Vallet et al., 2019). This characteristic of LPP could reflect the deficit in attentional resources toward arousal stimuli in psychopathic individuals. For example, it has been reported that negative stimuli (compared to neutral and positive stimuli) block attentional disengaging and inhibit motor responses following stimulus presentation (Chen et al., 2014).

Interestingly, ERN components appear to be also depended on emotional context. Previous studies reported that the ability to process internal states can modulate behaviors involving error detection with an increase of ERN amplitude in emotional context. Indeed, individual with alexithymia do not display this enhancement in emotional context (Maier et al., 2016). Considering that disturbances in emotional processing are heralded as the core features of psychopathy, it seems consistent to hypothesize that psychopathic individuals displayed impairments in error monitoring. However, in psychopathy, it remains difficult to know the extent to which the lack of insight into the internal affective state is impaired. Psychopathic individuals may have many interests to dissimulate or lies about their internal states (Miller et al., 2000).

4.9 Brain correlates ~ Component of error-processing:

The most probable hypothesis concerning the source generation of the ERN and Pe component is the anterior cingulate cortex (ACC). Source localization studies (Herrmann et al., 2004; Hinault et al., 2019; Keil et al., 2010; Miltner et al., 2003) and lesions studies (Stemmer et al., 2004) reported a relationship between the ACC and the ERN and Pe components. While the ERN component seems to be more related to the caudal region of the ACC, the Pe component is rather related to the caudal and rostral regions of the ACC (Edwards et al., 2012).

Regarding neural substrates that may underpin error monitoring and attention processing, previous works reported the implication of the ACC to detect events indicating a need to shift the focus of attention (Botvinick et al., 2004; Bush et al., 2000; Kerns et al., 2004). Close relation between the anterior insular cortex (AIC) and the Pe component has also been described (Ullsperge

et al., 2010). The AIC seems to be implicated in the detection of salient events like error commission, which is eliciting an attentional and orienting response. Indeed, the AIC might support the salient-network and be involved in error-awareness through functional connections with frontal and parietal cortices (Klein et al., 2007).

Using functional magnetic resonance imaging (fMRI), neuroimaging studies revealed several abnormalities in psychopathic individuals and antisocial personality across brain structures implicated in error monitoring and attentional processing. Among them, aberrant functional network connectivity was reported in paralimbic system (Espinoza et al., 2018). Reduction of hemodynamic activity was also observed in the ACC, the OFC, the dorsolateral PFC (DLPFC), the amygdala, the ventro-medial PFC and the superior temporal gyrus ((STS); Blair, 2008; Cope et al., 2014; Ermer et al., 2013; Kiehl et al., 2001; Lockwood et al., 2013). A meta-analysis of fMRI studies in psychopathic individuals and antisocial personality (Yang & Raine, 2009) confirmed the presence of functional and structural abnormalities in paralimbic area and particularly in the ACC.

5. Limitations

Several limitations should be acknowledged. First, as commonly observed in the field of psychopathy, there was heterogeneity in the clinical samples regarding the definition of psychopathy used in the studies and included in the model. Our analyzed clinical samples consisted of individuals with different levels of psychopathy, as measured by clinical scales. Several clinical scales were used to diagnose psychopathy and antisocial traits: the PCL-R, the PCL-SV, the PCL-YV, and the TriPm. These scales assessed different factors of psychopathy, and relative correlations are reported between scales. The most important discrepancy between scales is related to YPI and PCL-SV. Chauhan *et alii* (2014) explored relations between these scales using a multi-measure longitudinal research design in a sample of 122 offending girls. Results reported moderate correlation between YPI and PCL-YV. The main limitation regarding scales refers to the conceptualization of psychopathy. As mentioned previously, PCL-R and associated scales involve

higher-order dimensions of psychopathy, whereas the TriPM scale considers psychopathy as a separate dimensional construct. A second limitation concerning scale, which has already been pointed out by Schulreich (2016), is the core conceptualization. Whereas the PCL-R is specifically designed for assessing behavior in criminal samples, the TriPM focuses on personality aspects linked to three distinct phenotypic constructs.

In addition, regarding screening, the lack of externalizing measures across the samples precludes the possibility to consider externalizing disorder as a covariate in the models. This limitation prevents the possibility to dissociate the effect of externalizing behavior from psychopathy.

Medication could also constitute a confounding factor. For instance, it has been reported that antidepressant medication can enhance performance of participants in cognition tasks (Amado-Boccara et al., 1993; Harmer et al., 2013). Benzodiazepines and psychotropic medications are known to affect EEG activity (Aiyer et al., 2016). Medication of psychopathic individuals was not systematically reported in the included studies and this factor may have influenced their performance and modified their EEG activity.

6. Conclusion

Overall, the current meta-analysis revealed that psychopathic individuals displayed abnormal performance monitoring that could be evaluate objectively by ERN and Pe amplitude. The main results revealed that individuals with interpersonal-affective traits displayed non-significant modulation of ERN amplitude reflecting normal performance monitoring whereas individuals on impulsive-antisocial displayed reduced ERN amplitude. Regarding the later stage involved in conscious error processing, while the Pe component depicted a significant reduced amplitude considering the total score of psychopathy, neither the antisocial-Impulsivity dimension nor the Affective-Interpersonal dimension led to a significant association with the Pe component. In addition, the central results of the analyses highlight the heterogeneity between the samples

(community vs. incarcerated) and the major importance to combine screening tools in future studies exploring psychopathy among clinical samples (dimensional and high order conception for example).

These results address *in fine* a fundamental questioning regarding the capacity of psychopathic individuals to use error detection to adapt behavior and correct errors in real life. Almost all psychopathic individuals meeting the standard for mental sanity, however, from experimental consideration, they fail to maintain efficient cognitive functioning and cannot regulate behavior in accordance with the task. The question may legitimately be asked whether psychopathic individuals are deficient or not in the cognitive processing needed to monitoring behavior? Neuroimaging technique such as EEG and functional magnetic resonance imaging provide objective measured related to abnormal cognitive functioning as error detection. For now, these elements remain as unique interest for clinical research but this possible transferrin in forensic psychiatry is a major concern (Mouchet-Mages, 2013) and must be discussed further considering the development of neuroimaging techniques.

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Disclosure of interest

The authors report no conflict of interest.

Figure captions:

Figure.1: PRISMA flow chart of the search process; Error-related negativity (ERN); Positivity potential (Pe)

Figure.2: Forest plot for the meta-analysis on the ERN and Pe components shows the results (sample size for group test and group control, effect size and relative risk) of individual studies grouped according to measure components. RE, Random effect model; ML, Maximum Likelihood.

Figure.3: Forest plot for the meta-analysis based on psychopathy factors (A) and sample (B) regarding ERN modulation. Forest plot shows the Fisher's Z Transformed Correlation Coefficient (sample size, effect size and Fisher's Z score) of individual studies grouped according to psychopathy dimension (Interpersonal-Affective, Impulsive-Antisocial) and sample (Community, clinical). ML, Maximum Likelihood.

Figure.4A, .4B, .4C and .4D: Funnel plot for the meta-analysis on the ERN components. Point represents the observed effect sizes against standard error. In current meta-analysis, majority of point falling on the pseudo-confidence interval region and Eggers test for funnel plot asymmetry reported no publication bias.

Table.1: *: Ribes-Guardiola et al., 2020: averaging the ERN's on channel numbers: 5, 6, 7, 8, 9, 14, 15, 16, 17, 21, 22, 23, 24, 29, 30, 186, 198, 207, 215, 257 and Pe on channel numbers: 45, 53, 60, 79, 80, 81,88, 89, 90, 100, 101, 129, 130, 131, 132, 142, 143, 144, 155, 257; Maurer et al., 2016a - ERN/Ne and Pe (AF3, AFz, AF4, F3, Fz, F4), Maurer et al., 2016b - ERN (F3, Fz, F4, FC3, FCz, FC4, C3, Cz and C4) Pe (C3, Cz, C4, P3, Pz, P4, FO3, FOz and FO4), Steele et al., 2016 – ERN (F3, Fz, F4, FC3, Fcz, FC4, C3, Cz, & C4) Pe (C3, Cz, C4, CP3, CPz, CP4, P3, Pz, & P4); N: sample size; M: Mean; SD: Standard deviation; ERN: Error related negativity; Pe: Error-positivity; Nr: not reported. Note that for Pasion & Barbosa (2016), only the total score of TriPM was included in the meta-analysis.

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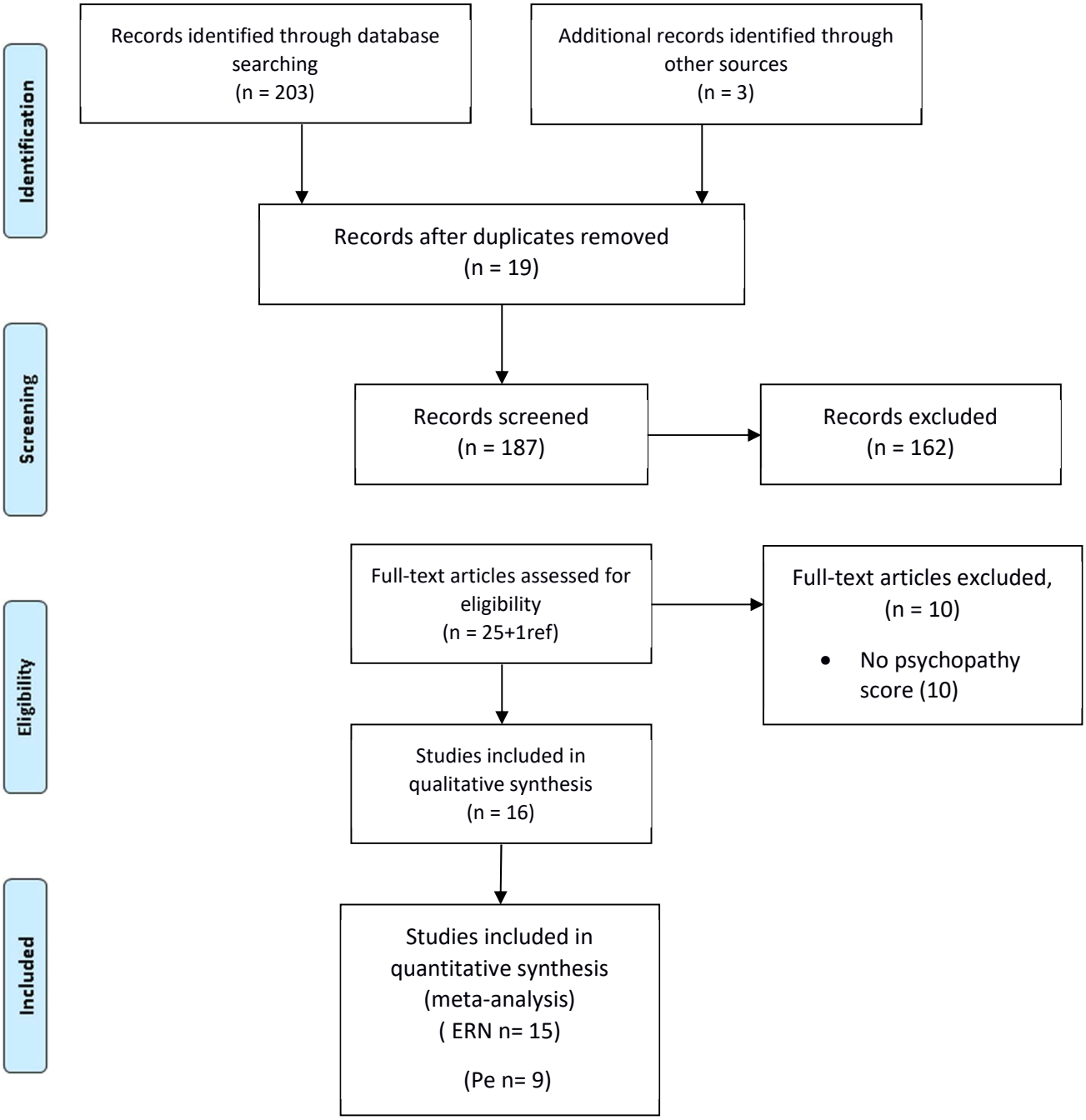
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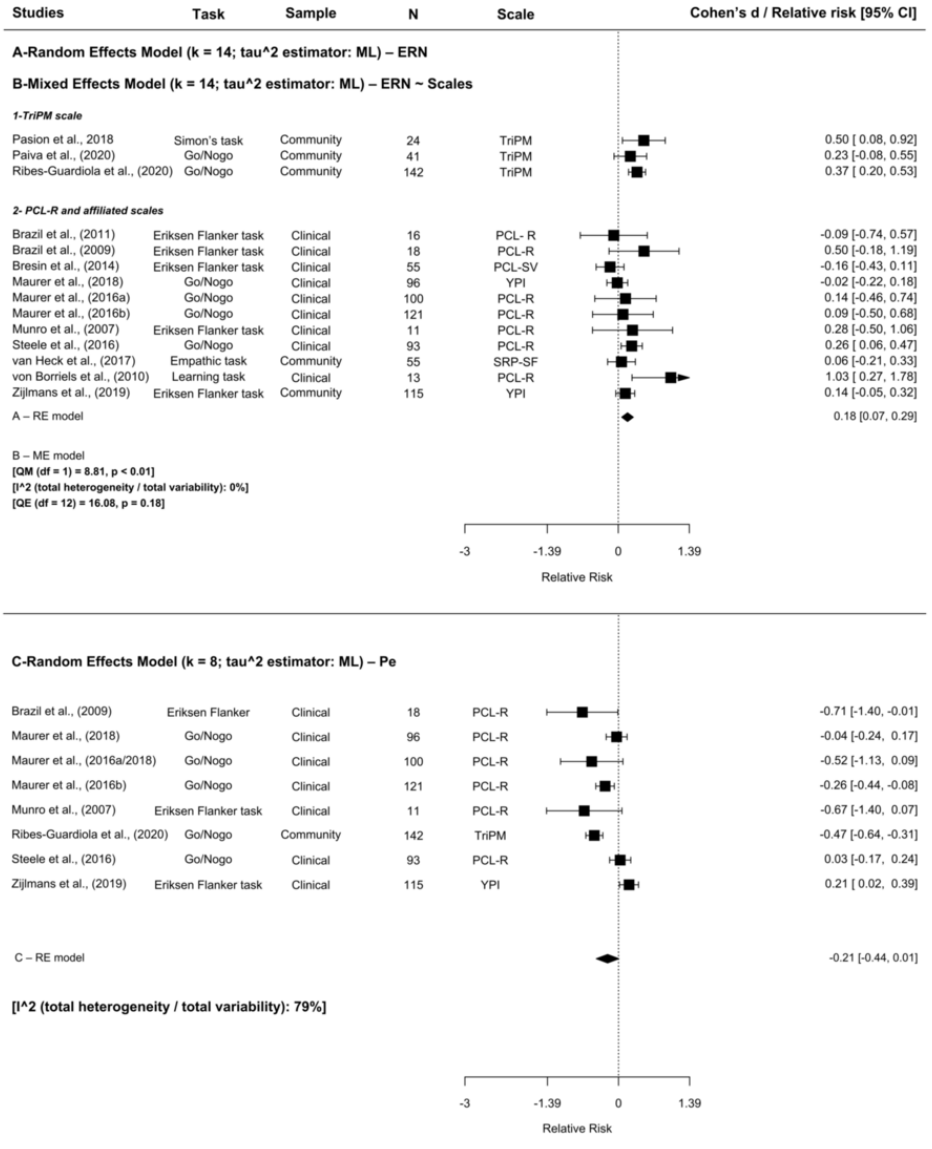
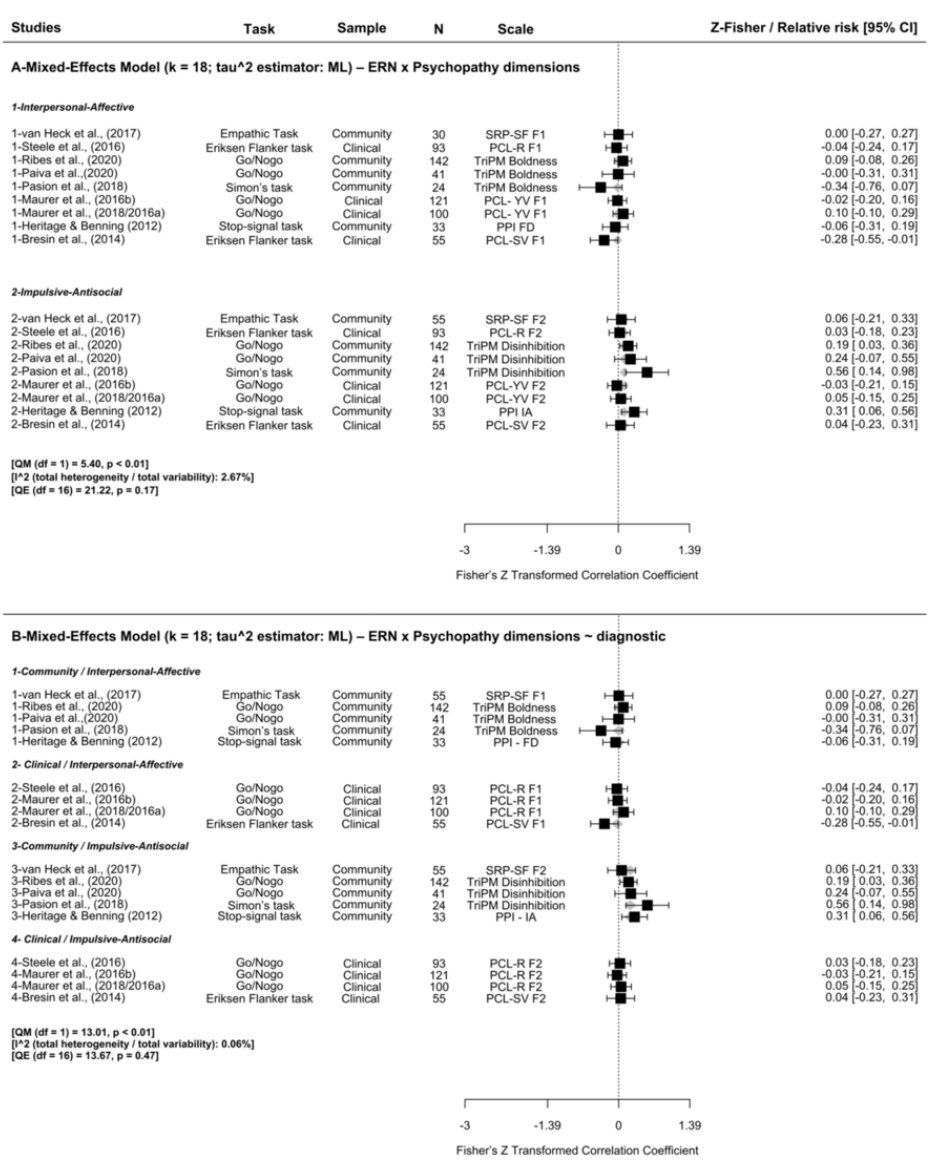
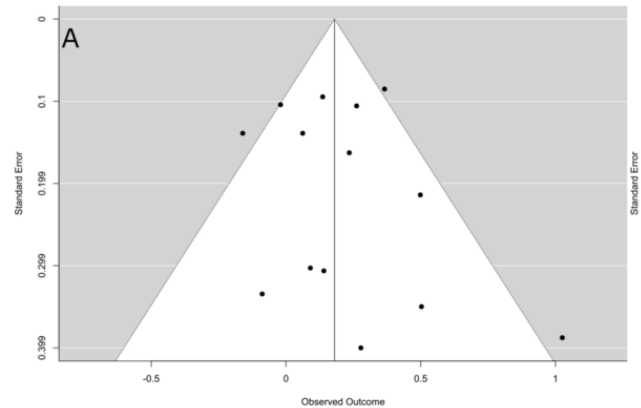
Records identified through database searching
(n = 203)
Additional records identified through other sources
(n = 3)
Records after duplicates removed
(n = 19)
Screening
Records screened
(n = 187)
Records excluded
(n = 162)
Eligibility
Full-text articles assessed for eligibility
(n = 25+1ref)
Full-text articles excluded,
(n = 10)
No psychopathy score (10)
Studies included in qualitative synthesis
(n = 16)
Included
Studies included in quantitative synthesis
(meta-analysis)
(ERN n= 15)
(Pe n= 9)


Studies
Task
Sample
N
Scale
Cohen's d / Relative risk [95% CI]
A-Random Effects Model (k = 14; tau^2 estimator: ML) – ERN
B-Mixed Effects Model (k = 14; tau^2 estimator: ML) – ERN ~ Scales
1-TriPM scale
Pasion et al., 2018 Simon's task Community 24 TriPM 0.50 [0.08, 0.92]
Paiva et al., (2020) Go/Nogo Community 41 TriPM 0.23 [-0.08, 0.55]
Ribes-Guardiola et al., (2020) Go/Nogo Community 142 TriPM 0.37 [0.20, 0.53]
2- PCL-R and affiliated scales
Brazil et al., (2011) Eriksen Flanker task Clinical 16 PCL- R -0.09 [-0.74, 0.57]
Brazil et al., (2009) Eriksen Flanker task Clinical 18 PCL-R 0.50 [-0.18, 1.19]
Bresin et al., (2014) Eriksen Flanker task Clinical 55 PCL-SV -0.16 [-0.43, 0.11]
Maurer et al., (2018) Go/Nogo Clinical 96 YPI -0.02 [-0.22, 0.18]
Maurer et al., (2016a) Go/Nogo Clinical 100 PCL-R 0.14 [-0.46, 0.74]
Maurer et al., (2016b) Go/Nogo Clinical 121 PCL-R 0.09 [-0.50, 0.68]
Munro et al., (2007) Eriksen Flanker task Clinical 11 PCL-R 0.28 [-0.50, 1.06]
Steele et al., (2016) Go/Nogo Clinical 93 PCL-R 0.26 [0.06, 0.47]
van Heck et al., (2017) Empathic task Community 55 SRP-SF 0.06 [-0.21, 0.33]
von Borriels et al., (2010) Learning task Clinical 13 PCL-R 1.03 [0.27, 1.78]
Zijlmans et al., (2019) Eriksen Flanker task Community 115 YPI 0.14 [-0.05, 0.32]
A – RE model 0.18 [0.07, 0.29]
B – ME model
[QM (df = 1) = 8.81, p < 0.01]
[I^2 (total heterogeneity / total variability): 0%]
[QE (df = 12) = 16.08, p = 0.18]
-3
-1.39
0
1.39
Relative Risk
C-Random Effects Model (k = 8; tau^2 estimator: ML) – Pe
Brazil et al., (2009) Eriksen Flanker Clinical 18 PCL-R -0.71 [-1.40, -0.01]
Maurer et al., (2018) Go/Nogo Clinical 96 PCL-R -0.04 [-0.24, 0.17]
Maurer et al., (2016a/2018) Go/Nogo Clinical 100 PCL-R -0.52 [-1.13, 0.09]
Maurer et al., (2016b) Go/Nogo Clinical 121 PCL-R -0.26 [-0.44, -0.08]
Munro et al., (2007) Eriksen Flanker task Clinical 11 PCL-R -0.67 [-1.40, 0.07]
Ribes-Guardiola et al., (2020) Go/Nogo Community 142 TriPM -0.47 [-0.64, -0.31]
Steele et al., (2016) Go/Nogo Clinical 93 PCL-R 0.03 [-0.17, 0.24]
Zijlmans et al., (2019) Eriksen Flanker task Clinical 115 YPI 0.21 [0.02, 0.39]
C – RE model -0.21 [-0.44, 0.01]
[I^2 (total heterogeneity / total variability): 79%]
-3
-1.39
0
1.39
Relative Risk


Studies
Task
Sample
N
Scale
Z-Fisher / Relative risk [95% CI]
A-Mixed-Effects Model (k = 18; tau^2 estimator: ML) – ERN x Psychopathy dimensions
1-Interpersonal-Affective
1-van Heck et al., (2017) Empathic Task Community 30 SRP-SF F1 0.00 [-0.27, 0.27]
1-Steele et al., (2016) Eriksen Flanker task Clinical 93 PCL-R F1 -0.04 [-0.24, 0.17]
1-Ribes et al., (2020) Go/Nogo Community 142 TriPM Boldness 0.09 [-0.08, 0.26]
1-Paiva et al.,(2020) Go/Nogo Community 41 TriPM Boldness -0.00 [-0.31, 0.31]
1-Pasion et al., (2018) Simon's task Community 24 TriPM Boldness -0.34 [-0.76, 0.07]
1-Maurer et al., (2016b) Go/Nogo Clinical 121 PCL- YV F1 -0.02 [-0.20, 0.16]
1-Maurer et al., (2018/2016a) Go/Nogo Clinical 100 PCL- YV F1 0.10 [-0.10, 0.29]
1-Heritage & Benning (2012) Stop-signal task Community 33 PPI FD -0.06 [-0.31, 0.19]
1-Bresin et al., (2014) Eriksen Flanker task Clinical 55 PCL-SV F1 -0.28 [-0.55, -0.01]
2-Impulsive-Antisocial
2-van Heck et al., (2017) Empathic Task Community 55 SRP-SF F2 0.06 [-0.21, 0.33]
2-Steele et al., (2016) Eriksen Flanker task Clinical 93 PCL-R F2 0.03 [-0.18, 0.23]
2-Ribes et al., (2020) Go/Nogo Community 142 TriPM Disinhibition 0.19 [0.03, 0.36]
2-Paiva et al., (2020) Go/Nogo Community 41 TriPM Disinhibition 0.24 [-0.07, 0.55]
2-Pasion et al., (2018) Simon's task Community 24 TriPM Disinhibition 0.56 [0.14, 0.98]
2-Maurer et al., (2016b) Go/Nogo Clinical 121 PCL-YV F2 -0.03 [-0.21, 0.15]
2-Maurer et al., (2018/2016a) Go/Nogo Clinical 100 PCL-YV F2 0.05 [-0.15, 0.25]
2-Heritage & Benning (2012) Stop-signal task Community 33 PPI IA 0.31 [0.06, 0.56]
2-Bresin et al., (2014) Eriksen Flanker task Clinical 55 PCL-SV F2 0.04 [-0.23, 0.31]
[QM (df = 1) = 5.40, p < 0.01]
[I^2 (total heterogeneity / total variability): 2.67%]
[QE (df = 16) = 21.22, p = 0.17]
-3
-1.39
0
1.39
Fisher's Z Transformed Correlation Coefficient
B-Mixed-Effects Model (k = 18; tau^2 estimator: ML) – ERN x Psychopathy dimensions ~ diagnostic
1-Community / Interpersonal-Affective
1-van Heck et al., (2017) Empathic Task Community 55 SRP-SF F1 0.00 [-0.27, 0.27]
1-Ribes et al., (2020) Go/Nogo Community 142 TriPM Boldness 0.09 [-0.08, 0.26]
1-Paiva et al.,(2020) Go/Nogo Community 41 TriPM Boldness -0.00 [-0.31, 0.31]
1-Pasion et al., (2018) Simon's task Community 24 TriPM Boldness -0.34 [-0.76, 0.07]
1-Heritage & Benning (2012) Stop-signal task Community 33 PPI - FD -0.06 [-0.31, 0.19]
2- Clinical / Interpersonal-Affective
2-Steele et al., (2016) Go/Nogo Clinical 93 PCL-R F1 -0.04 [-0.24, 0.17]
2-Maurer et al., (2016b) Go/Nogo Clinical 121 PCL-R F1 -0.02 [-0.20, 0.16]
2-Maurer et al., (2018/2016a) Go/Nogo Clinical 100 PCL-R F1 0.10 [-0.10, 0.29]
2-Bresin et al., (2014) Eriksen Flanker task Clinical 55 PCL-SV F1 -0.28 [-0.55, -0.01]
3-Community / Impulsive-Antisocial
3-van Heck et al., (2017) Empathic Task Community 55 SRP-SF F2 0.06 [-0.21, 0.33]
3-Ribes et al., (2020) Go/Nogo Community 142 TriPM Disinhibition 0.19 [0.03, 0.36]
3-Paiva et al., (2020) Go/Nogo Community 41 TriPM Disinhibition 0.24 [-0.07, 0.55]
3-Pasion et al., (2018) Simon's task Community 24 TriPM Disinhibition 0.56 [0.14, 0.98]
3-Heritage & Benning (2012) Stop-signal task Community 33 PPI - IA 0.31 [0.06, 0.56]
4- Clinical / Impulsive-Antisocial
4-Steele et al., (2016) Go/Nogo Clinical 93 PCL-R F2 0.03 [-0.18, 0.23]
4-Maurer et al., (2016b) Go/Nogo Clinical 121 PCL-R F2 -0.03 [-0.21, 0.15]
4-Maurer et al., (2018/2016a) Go/Nogo Clinical 100 PCL-R F2 0.05 [-0.15, 0.25]
4-Bresin et al., (2014) Eriksen Flanker task Clinical 55 PCL-SV F2 0.04 [-0.23, 0.31]
[QM (df = 1) = 13.01, p < 0.01]
[I^2 (total heterogeneity / total variability): 0.06%]
[QE (df = 16) = 13.67, p = 0.47]
-3
-1.39
0
1.39
Fisher's Z Transformed Correlation Coefficient


A
Standard Error
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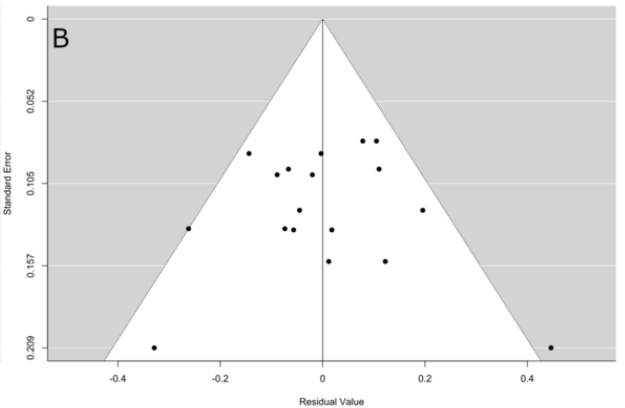
0.199
0.299
0.399
-0.5
0
0.5
1
Observed Outcome


B
Standard Error
0
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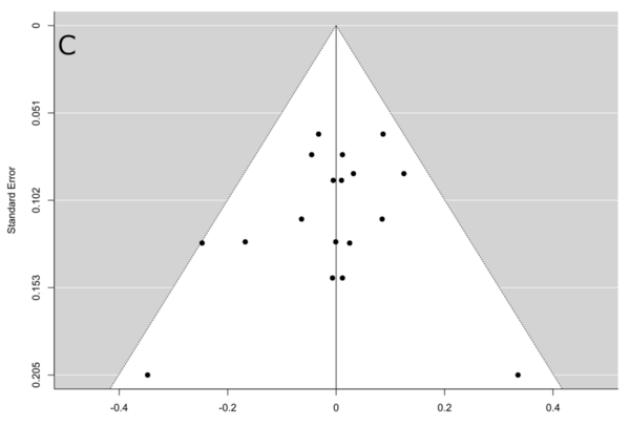
0.105
0.157
0.209
-0.4
-0.2
0
0.2
0.4
Residual Value


C
Standard Error
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0.102
0.153
0.205
-0.4
-0.2
0
0.2
0.4
Residual Value

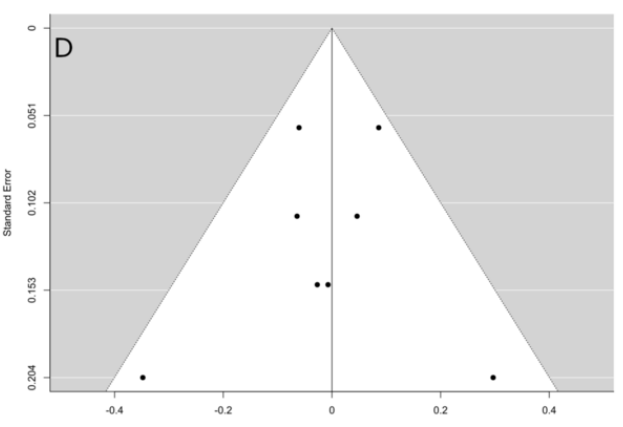
D
Standard Error
0
0.051
0.102
0.153
0.204
-0.4
-0.2
0
0.2
0.4
Residual Value

***Table.1**: Characteristics of the studies included in the meta-analysis*

Reference	Subject	Scale	Age	Sex	Electrode	Reference	Time	Task	Stimuli	Quality score
Ribes-Guardiola et al. (2020)	Healthy with psychopathy traits	TriPM	M = 20.58 SD = 4.69	41 males – 101 Females	Average*	Cz	ERN 0-100ms Pe 150-400ms	Go/NoGo task and Eriksen flanker task	Letters	90.91%
Paiva et al. (2020)	Healthy with psychopathy traits	TriPM	M = 26.6 SD = 6.12	16 Males – 25 Females	Fcz	Cz	ERN 50-150ms	Go/NoGo task	Letters	90.91
Zijlmans et al. (2019)	Youth criminals	YPI	M = 22.46 SD = 2.32	115 Males	ERN – Fcz Pe - Cz	Mastoids	ERN 25-150ms Pe 250-400ms	Eriksen flanker task	Letters	81,82%
Maurer et al. (2018)	Youth criminals	YPI	M = 17.38 SD = 0.86	96 Males	Average*	Nose	ERN 0-100ms Pe 94-500ms	Go/NoGo task	Letters	86,36%
Venables et al. (2018)	Healthy with psychopathy traits	PPI-D	M = 20.5 SD = 3.8	85 Males – 64 Females	Pe - Fcz	Cz	Pe 93-273ms	Eriksen flanker task	Arrows	86,36%

van Heck et al. (2017)	Healthy with psychopathy traits	SRP-SF	M = 31.8, SD = 8	26 Males – 29 Females	Fz, Cz, Pz	Mastoids	ERN 20-70ms	Empathic task	Pictures	72.72%
Maurer et al. (2016(a))	Youth criminals	PCL-YV	M = 17.62, SD = .81	21 Males	Fronto-central cluster*	Nose	ERN 0-100ms Pe 94-500	Go/NoGo task	Letters	95.45%
Maurer et al. (2016(b))	Adults criminals	PCL-R	M = 33.94, SD = 9.55	121 Females	Fronto-central cluster* Centro-parietal cluster*	Nose	Nr	Go/NoGo task	Letters	72,73%
Pasion & Barbosa (2016)	Healthy with psychopathy traits	TriPM	M = 28.2, SD = 9.96	24 Males	Fz	Average	ERN 80-160ms	Simon's task	Arrows	81,82%
Steele et al. (2016)	Adults criminals	PCL-R	M = 34.53, SD = 9.81	93 Males	Fronto-central cluster* Centro-parietal cluster*	Nose	ERN -50-100ms Pe 75-500ms	Go/NoGo task	Letters	72,73%
Bresin et al. (2014)	Adults criminals	PCL-SV	M = 33, SD = 9,8	55 Males	Cz	Mastoids	ERN 0-250ms	Eriksen flanker task	Letters	72,73%
Heritage & Benning (2013)	Healthy with psychopathy traits	MPQ-BF	M = 36, SD = 12	29 Males – 37 Females	Fz	Nr	ERN 0-100ms	Stop-signal task	Letters	72.72%

Brazil et al. (2011)	Adults criminals	PCL-R	M = 36, SD = 8	18 Males	Fz, Cz	Earlobes	ERN 50-150ms	Eriksen flanker task	Arrowheads	86,36%
Von Borries et al. (2010)	Adults criminals	PCL-R	M = 37, SD = 9.5	13 Males	Cz	Earlobes	ERN 0-200ms	Learning task	Pictures	90,91%
Brazil et al, (2009)	Adults criminals	PCL-R	M = 39, SD = 9.5	16 Males	ERN - Fz, Cz Pe - Cz	Earlobes	ERN 0-150ms Pe 250-400ms	Eriksen flanker task	Letters	81,82%
Munro et al. (2007)	Adults criminals	PCL-R	M = 45.9, SD = 3.5	11 Males	Fz, FCz,Cz, and Pz	Mastoids	ERN 0-150ms Pe 150-350ms	Eriksen flanker task	Letters	95,45%