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Risk factors for persistent delinquent behavior among juveniles: A meta-analytic review

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HIGHLIGHTS

- Multiple risk domains need to be addressed when preventing/treating LCP offending.
- The mean effect of social relationship risk factors was largest during childhood.
- Siblings may be more influential in developing LCP offending than mothers.
- The potential of interventions solely focused on the family may be relatively low.
- A true categorical distinction between LCP and AL offenders may not be very likely.

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ABSTRACT

Multiple risk domains have been identified for life-course persistent (LCP) offending, but a quantitative review of the effect of different risk domains was not yet available. Therefore, we performed a series of multilevel meta-analyses to examine the effect of several risk domains for LCP offending relative to adolescence-limited (AL) offending. We included 55 studies reporting on 1014 effects of risk factors, and classified each factor into one of 14 risk domains. The results revealed a significant effect for 11 domains ranging from $d = 0.200$ to $d = 0.758$. Relatively large effects were found for the criminal history, aggressive behavior, and alcohol/drug abuse domains, whereas relatively small effects were found for the family, neurocognitive, and attitude domains. The physical health, background, and neighborhood domains yielded no effect. Moderator analyses showed that effects of sibling-related risk factors were larger than effects of mother-related risk factors, and that the effect of the relationship domain was largest during childhood. We conclude that most risk domains contribute to the development of LCP offending and that differences between AL and LCP offenders may be quantitative rather than qualitative. Implications of the present results for risk assessment and the prevention/treatment of LCP offending are discussed.

Contents

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1. Introduction

Criminological research on juvenile delinquency showed that a relatively small group of life-course persistent offenders is responsible for a disproportionate amount of offenses (Farrington & West, 1993; Moffitt, 1993, 2006). This chronic delinquent behavior does not only have serious negative consequences for both victims and offenders (McGuw & Iacono, 2005; Piquero, Daigle, Gibson, Leeper Piquero, & Tibbetts, 2007), it also imposes substantial monetary costs to the criminal justice system and the larger society (Cohen, Piquero, & Jennings, 2010). Because of the high negative impact of life-course persistent offending, it is important to prevent juveniles from developing a chronic delinquent career by timely delivering effective treatment or prevention. Consequently, it is not only relevant to determine which juveniles are at risk for chronic delinquency and should be subjected to treatment or prevention programs, clinicians must also decide on what exactly should be addressed in these programs. For both purposes, research into risk factors for persistent delinquent behavior is crucial. Until now, a systematic review in which the strength of risk factors for life-course persistent offending is statistically summarized, is not available. Therefore, the aim of the present study was to statistically summarize the effects of risk factors for life-course persistent (LCP) offending relative to adolescence limited (AL) offending by conducting a series of meta-analyses on several domains of risk factors.

In the influential developmental taxonomic theory developed by Moffitt (1993), she stated that two qualitatively distinct offender profiles can be distinguished based on the timing and duration of antisocial involvement of juvenile offenders. The first profile, labeled life-course persistent, comprises a small group of individuals (around 5–8% of the general population), who exhibit changing manifestations of persistent and serious antisocial behavior across the life course. These individuals start with antisocial behavior in childhood and continue to show this behavior in adolescence and adulthood. According to Moffitt, the origins of this stable disruptive behavior must be found in an interaction between children's neuropsychological impairments and a disadvantaged environment. The second profile of antisocial involvement is referred to as adolescence-limited, and comprises a rather common group of individuals whose antisocial behavior is restricted to the adolescent phase of life. This adolescence-limited antisocial behavior is primarily nonaggressive and finds its origins not in neuropsychological impairments, but in the fact that adolescents wish to be treated as adults and therefore mimic their life-course persistent antisocial peers in an attempt to obtain the status and privileges of adulthood.

Although the developmental taxonomic theory of Moffitt (1993) had a major and valuable influence on different fields of research since its publication, there is also empirical evidence suggesting the need for evaluating and perhaps reformulating the taxonomic theory. Recently, Fairchild, Van Goozen, Calder, and Goodyer (2013) conducted a comprehensive systematic review of 61 empirical studies in which they compared the two profiles of antisocial involvement. These researchers found several results that do not concur with Moffitt's theory. First, although a substantial proportion of individuals exhibit severe antisocial behavior in early childhood, these children desist from this behavior during adolescence. Second, when severe antisocial behavior emerges in the adolescent period, it is seldom limited to adolescence. Third, both the life-course persistent and adolescence-limited profiles resemble each other in terms of childhood risk factors, personality traits, neuropsychological vulnerabilities, and changes in brain structure and function. Based on these findings the researchers concluded that the distinction in developmental antecedents between adolescence-limited and life-course persistent offenders is rather quantitative (i.e., pertaining to the amount of antecedents) than qualitative (i.e., pertaining to the nature of antecedents), which is not in line with Moffitt's theory.

The aim of the current study builds specifically on the finding of Fairchild et al. (2013) that adolescent-onset delinquent behavior does not necessarily need to stop in early adulthood. If adolescent-onset offenders comprise both adolescence-limited offenders and potential life-course persistent offenders, then the question arises which risk factors have the greatest impact on life-course persistent offending relative to adolescence-limited offending. Answering this question is not only relevant for gaining more general knowledge of antecedents of chronic delinquency, but also for improving both risk assessment procedures as well as strategies for prevention and intervention.

The great diversity of risk factors that have been subject of research throughout the years, emphasizes the need to classify risk factors in order to determine how groups of (more or less) similar risk factors are associated with persistent delinquent behavior. These groups of risk factors are often referred to as “domains”. An example of such a classification can be found in the cumulative developmental model of serious delinquency, in which Loeber, Slot, and Stouthamer-Loeber (2008) distinguished the following five domains of risk factors: individual, family, peers, school, and neighborhood. A somewhat different but influential categorization of risk factors was presented by Andrews and Bonta (2010) as part of their risk-need-responsivity (RNR) model for offender assessment and treatment. In this model, risk factors for recidivism are classified into eight major domains. Four of these domains are referred to as “the big four” because of their strong predictive power

of criminal behavior (i.e., history of antisocial behavior; antisocial personality pattern; antisocial cognition; and antisocial peer affiliations), while the other domains are designated by "the moderate four" because of their moderate association with criminal behavior (i.e., family/marital circumstances; school/work; leisure/recreation; and substance abuse). Classifications of risk factors for juvenile delinquency are often based on the RNR model, such as the risk classifications used in both the Washington State Juvenile Court Assessment (WSJCA; Barnoski, 2004) and the Youth Level of Service and Case Management Inventory (YLS/CMI; Hoge, 2001). Since the RNR model is one of the most influential and important models for offender assessment and treatment, we decided to use the RNR model as a guidance for our classification of risk factors.

Risk factors that were examined by Fairchild et al. (2013) related to personality, genetic, and neurobiological factors. An improvement of the present study is that other risk factors, such as emotional and behavioral problems, alcohol and/or drug abuse, and family characteristics, will also be examined. Moreover, although Fairchild et al. conducted an important and informative systematic literature review, it was qualitative in nature. The present study advances research on the effects of risk factors for life-course persistent delinquency by examining these effects meta-analytically (i.e., quantitatively).

The present study is relevant for several reasons. First, examining the effects of different risk factors advances the fundamental knowledge of the types of risk factors that contribute most to life-course persistent offending behavior. Second, more insight into the effects of risk factors can help improve current risk and needs assessment practices. Both types of assessment pertain to two important *what works principles*: the risk principle and the needs principle (Andrews & Bonta, 2010; Andrews, Bonta, & Hoge, 1990; Andrews, Bonta, & Wormith, 2006). The risk principle states that the intensity of an intervention should be proportional to the risk of reoffending, implying that high risk offenders require intensive treatment, whereas low risk offenders require minimal or no treatment. According to the needs principle, interventions aimed at reducing delinquent behavior should address the criminogenic needs of offenders, which are changeable offender characteristics that are associated with reoffending (i.e., dynamic risk factors). For both risk and needs assessment, proper instruments need to be available that measure all relevant static and dynamic risk factors. The more relevant risk factors are measured, the more accurate a risk and/or needs assessment will be. Third, the results of the present study can support the development and improvement of programs aimed at the treatment or prevention of persistent delinquency. In order to effectively prevent future delinquency, these treatment and prevention programs should target the right set of dynamic risk factors (i.e., criminogenic needs) among at-risk juveniles. Knowledge of the effect of dynamic risk factors is essential for determining which risk factors can best be addressed in these programs.

In sum, the present review meta-analytically summarized the literature on the effects of risk factors for life-course persistent offending relative to adolescence-limited offending. The first aim of the present study was to determine the effect of a number of different domains of risk factors for persistent delinquent behavior. In addition, we examined whether the effects of these risk domains statistically differed from each other. A second aim was to investigate how the effect of each risk domain was moderated by risk factor characteristics, sample descriptors, and/or research design descriptors.

2. Methods

2.1. Sample of studies

For the selection of studies, several criteria were formulated. First, we selected studies that examined the development of delinquent behavior among juveniles. However, we did not include studies that solely focused on juvenile sexual offending, since this behavior can be explained by specific factors that are different from other types of offending behavior (e.g., Becker, 1990, 1998; Worling & Långström, 2006). Second, studies that were solely based on samples of adult offenders (disregarding their younger years) and studies in which the sample consisted exclusively of psychiatric inpatients who had severe mental health problems, such as personality disorders, schizophrenia or other related disorders, were excluded. Third, we selected studies that distinguished between juveniles following a life-course persistent offending trajectory and juveniles following an adolescence-limited offending trajectory, so that effects of risk factors for life-course persistent offending relative to adolescence-limited offending could be examined. We included studies in which the life-course persistent and adolescence-limited offending trajectories were operationalized in different ways, including life-course persistent versus adolescence-limited, persisters versus desisters, early-onset versus late-onset, and chronic versus late-onset. Studies that examined only one of these offending trajectories or exclusively other offending trajectories were not included. Finally, studies had to report on results of bivariate analyses of the association between risk factors and trajectory membership or provide enough details to calculate a bivariate test statistic.

Until spring 2014, we searched for articles, book chapters, dissertations, and reports in the following six electronic databases: PsycINFO, ERIC, Criminal Justice Abstracts, Sociological Abstracts, Pubmed, and Google Scholar. The following combination of three components were used in this search strategy: ("early onset" OR "childhood onset" OR "life course" OR "lifelong" OR "persist*" OR "chronic") AND ("desist*" OR "time limited" OR "adolescen* limited" OR "adolescen* onset" OR "late onset") AND ("delinquen*" OR "antisocial" OR "offend*" OR "crim*" OR "serious behavio*"). In addition, an expanded search was performed using the additional syntax AND ("trajector*") AND ("juvenile*" OR "youth"), to ensure that manuscripts reporting on developmental trajectories of delinquency would be retrieved. To determine whether the retrieved studies could be included in our meta-analysis, we read titles, abstracts and, if necessary, full article texts.

Second, reference lists from several primary studies and review articles (e.g., Fairchild et al., 2013; Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009; Jennings & Reingle, 2012; Moffitt, 2006; Piquero, 2008) were also checked to find additional studies. Third, we contacted several authors in the field to ask whether they had suggestions for relevant studies that would be suitable for inclusion in our review. In total, our literature search strategy yielded 149 studies. After thoroughly screening these studies, we finally found 48 studies reporting on results of 55 different samples that met our inclusion criteria (see Appendix A for a flow chart of the search results and Appendix B for an overview of included studies and their characteristics). In cases where manuscripts reported on effects of risk factors in more than one sample of participants, each sample was entered as a separate study in the dataset.

2.2. Missing data

When conducting a meta-analysis, it can be very difficult to identify, retrieve, and include every study that is relevant regarding the research question in the meta-analysis. Furthermore, articles reporting non-significant results are less likely to be published than articles reporting significant results, and this problem was referred to by Rosenthal (1979) as the "file drawer problem". To examine the possibility of missing data due to publication bias, limitations in the search strategy, or any other cause, we conducted the funnel-plot-based trim and fill method as described by Duval and Tweedie (2000a, 2000b) by using the function "trimfill" of the metafor package (Viechtbauer, 2010) in the R environment (Version 3.2.0; R Core Team, 2015). Among the available techniques for assessing the possible existence of missing data and its implications for the results in meta-analysis, the trim and fill method is a conceptually easy method to adjust for the impact of missing effect sizes (Nakagawa & Santos, 2012). Moreover, Nik Idris (2012) indicated that the trim and fill method is superior in detecting the existence of

missing data as compared to both Egger's linear regression test (Egger, Davey-Smith, Schneider, & Minder, 1997) and Begg and Mazumdar's Rank Correlation test (Begg & Mazumdar, 1994). In short, the trim and fill method restores the symmetry of an asymmetric funnel plot by imputing missing effect sizes that are calculated on the basis of existing effect sizes.

2.3. Coding of studies

In developing a coding form, guidelines proposed by Lipsey and Wilson (2001) were followed. The variable of most interest in the present meta-analysis is the domain (i.e., group) in which a risk factor for life-course persistent offending can be classified. Coding this variable was needed for examining the effects of different risk domains for life-course persistent offending relative to adolescence-limited offending. In order to classify risk factors into domains, we made a classification scheme that was based on (1) a combination of the central eight criminogenic risk and need factors as described by Andrews and Bonta (2010), (2) the classification of risk factors that is used in risk assessment instruments, such as the Washington State Juvenile Court Assessment (WSJCA; Barnoski, 2004) and the Youth Level of Service and Case Management Inventory (Hoge, 2001), and (3) a screening of risk factors described in the primary studies that were included in the present review.

The following 13 risk domains were distinguished in our classification of risk factors: Criminal history (factors relating to the history of delinquency, such as having ever been incarcerated, being young at first arrest, high level of versatility in criminal offending, having ever used a weapon, and having ever been convicted for a violent crime); Aggression (factors relating to aggressive behavior, such as being physically and/or non-physically aggressive and having fought with parents and/or teachers); Alcohol/drug abuse (mainly factors relating to alcohol and drug abuse, such as being a heavy drinker, use of hashish prior to 17 years of age, being a drug user, registered alcohol abuse prior to 18 years of age, but also factors such as high daily cigarette use and being a gambler); Relationship (factors relating to the nature and quality of relationships with primarily peers, such as having delinquent peers, experiencing peer rejection, being a gang member, having poor relationship with peers, and deviant peer associations); Emotional and behavioral problems (factors relating to internalizing and externalizing problems, such as being depressed, having emotional problems, showing symptoms of conduct disorder, having a disruptive behavior disorder, and having an ADHD diagnosis); Sexual behavior (factors relating to sexual behavior, such as having a high number of sex partners, having had sex prior to 19 years of age, and having had promiscuous and/or irresponsible sex); School/Employment (factors relating to education and employment, such as poor academic achievement, being a frequent truant, having a lack of interest in school, having an unstable job record, and not being employed); Family (factors relating to familial problems, such as having criminal family members, low positive parenting, large family size, having a poor relation with parents, and parental conflict); Static background factors (gender and ethnicity related factors, such as being non-Caucasian and being male); Neurocognition/physiology (factors relating to (neuro)cognitive functioning, intelligence, and heart rate, such as low verbal ability, low nonverbal IQ, reading problems, having a neurological abnormality, and low resting heart rate); Attitude (factors relating to the attitude towards delinquency, anti-social behavior, and official authorities, such as having a positive attitude towards delinquency, being hostile to the police, being anti-establishment, and low perceived likelihood of getting caught); Neighborhood (factors relating to the quality of the living environment such as neighborhood problems, high community crime, and having a negative impression of the neighborhood), and Physical health (factors relating to physical health such as having a history of health problems, having ever had a head injury, and having ever been hospitalized for an illness). Risk factors that could not be classified into one of these 13 risk domains were classified into an additional rest category (see Table C2 in Appendix C for an overview of risk factors in this category), making the total number of risk domains 14. The minimum amount of risk factors for creating a separate domain was set to five.

Besides classifying risk factors into risk domains, we coded several sample characteristics, research design descriptors and risk factor characteristics that may moderate the effect of risk domains. First, the percentage of males in the sample was coded since previous research suggests differences in risk factors for offending trajectories between males and females (e.g., Fergusson, Horwood, & Nagin, 2000; Hipwell et al., 2002; Keenan & Shaw, 1997; Silverthorn & Frick, 1999). Second, we coded the percentage of cultural minorities (i.e., non-Caucasians), since several researchers found ethnic differences in risk factors for delinquency (e.g., Baier & Pfeiffer, 2008; Van der Put, Stams, Deković, Hoeve, & Van der Laan, 2013; Veen et al., 2011). Third, the type of measurement used to assess the history of antisocial behavior may influence the effect of the criminal history and aggression risk domains due to under- or over-reporting problems (e.g., Auty, Farrington, & Coid, 2015; Huizinga & Elliott, 1986). Therefore, type of measurement (i.e., self-report, parent report, teacher report, peer report, official record, or other source) was also coded.

As for risk factor characteristics, we coded type of risk factor (static or dynamic) since research on risk factors for delinquency suggest that static risk factors (e.g., a history of antisocial behavior) are more strongly associated with delinquency than dynamic risk factors (e.g., a poor school performance) (e.g., Andrews & Bonta, 2010; Cottle, Lee, & Heilbrun, 2001; Van der Put et al., 2011). Further, as children grow older, there are changes in both the extent to which children are exposed to risk factors (Loeber, Farrington, Stouthamer-Loeber, & White, 2008; Loeber, Slot, et al., 2008) and the impact of risk factors (Farrington, Loeber, Jolliffe, & Pardini, 2008; Loeber, Slot, & Stouthamer-Loeber, 2006). Therefore, the age period in which risk factors are present (childhood, adolescence, or life-course) was also coded. In addition, where possible, we grouped risk factors within primary risk domains into subgroups of (more or less) similar risk factors, in order to determine the effect of subdomains of risk factors.

Lastly, for descriptive purposes, we coded publication year, mean age of participants at start of the study, the country in which the research was performed, and the labels of the two offending trajectories that we found to best represent the life-course persistent and adolescence-limited offender profiles (see Appendix B for a full overview of trajectory pairs that we compared within each primary study). Table C1 of Appendix C presents a detailed overview of all variables coded for each primary study and Table C2 of Appendix C presents all subdomains that were created within seven primary risk domains.

To assess interrater agreement, 10 studies (reporting on 137 risk factors) were randomly selected and coded by the first and fourth author. Percentages of agreement were calculated to assess inter-rater reliability. The percentual agreement ranged from good (at least 80% agreement) for the variables percentage of males in the sample (90%), percentage of cultural minorities in the sample (90%), and mean age of the sampled subjects at start of the study (80%), to perfect (100% agreement) for the variables publication year, sample size, type of measurement, country in which the research was performed, and number of extracted risk factors per study. The percentual agreement for the variables domain of risk factor, type of factor (static or dynamic), age period in which a risk factor was present, and calculated effect size was, respectively, 96.4%, 90.5%, 88.0%, and 89.9%.

2.4. Statistical analyses

For each risk factor in each study, we calculated Cohen's *d* to express the effect of a risk factor for life-course persistent offending relative to adolescence-limited offending. The formulas of Ferguson (1966), Rosenthal (1994), and Lipsey and Wilson (2001) were used to transform test statistics into Cohen's *d*. Most *d* values were calculated based

on reported means and standard deviations or proportions. If a risk factor was more present in the life-course persistent offending group than in the adolescence-limited offending group, a positive *d* value was assigned to the effect of the risk factor, whereas a negative *d* value was assigned to effects of risk factors that were more present in the adolescence-limited offending group. If *d* values could not be calculated from the information provided in the article text, we contacted the authors for additional information. For a total of 56 non-significant risk factors in all primary studies, we could not calculate an effect size since the necessary information to calculate Cohen's *d* was not available. For these cases, we assigned the value of zero to Cohen's *d* (Mullen, 1989). This is a conservative estimate of the true effect size and seemed to us a more preferable method than excluding primary studies from the dataset because of missing data of non-significant risk factors.

Since extreme effect sizes may have a disproportionate influence on conclusions drawn from statistical analyses, we checked for outliers by searching for effect sizes with standardized scores larger than 3.29 or smaller than −3.29 (Tabachnik & Fidell, 2013). Five effect sizes in four domains of risk factors were identified with a *z* value exceeding 3.29, and one effect size was identified with a *z* value below −3.29: thus in total, six effect sizes were identified as an outlier. To reduce the impact of these outliers, the raw *d* values of the outliers were substituted by a new *d* value that equaled the highest (or lowest) effect that fell within the normal range. In this way a disproportionate influence of the outlying cases on the results of the statistical analyses was reduced.

Most primary studies that we included based on the formulated in- and exclusion criteria (see the section Sample of studies) report on multiple risk factors, and therefore more than one effect size can be extracted from these studies. However, a key assumption in traditional meta-analytic approaches is that the included subject samples are independent, so including multiple effect sizes based on the same sample violates this assumption (Lipsey & Wilson, 2001). Following scholars reporting on recent meta-analyses (e.g., Houben, Van den Noortgate, & Kuppens, 2015; Kuppens, Laurent, Heyvaert, & Onghena, 2013; Rapp, Van den Noortgate, Broekaert, & Vanderplasschen, 2014; Weisz et al., 2013), a multilevel random effects model was used for the calculation of combined effect sizes and for the moderator analyses in order to deal with dependency of study results (Hox, 2002; Van den Noortgate & Onghena, 2003). Van den Noortgate and Onghena (2003) compared this multilevel approach to traditional meta-analytical methods, and concluded that the maximum likelihood multilevel approach is in general superior to the fixed-effects approaches employed in traditional meta-analysis, and that for models without moderators, the results of the multilevel approach are not substantially different from results of the traditional random-effects approaches.

In the present study, we used a three-level meta-analytic model to analyze the data, modeling three sources of variance: sampling variance of the observed effect sizes (Level 1), variance between effect sizes from the same study (Level 2), and variance between studies (Level 3) (see also Cheung, 2014; Houben et al., 2015; Van den Noortgate, López-López, Marin-Martinez, & Sánchez-Meca, 2013, 2014). This model was used to obtain an overall estimate of the effect of each risk domain and in case of significant variation between effect sizes from the same study and/or between studies, it was subsequently extended by including moderator variables to determine whether this variation can be explained by characteristics of studies or effect sizes. An important advantage of this three-level approach to meta-analysis is that effect sizes extracted from the same study (i.e., dependent effect sizes) can be modeled. In this way, analyses are conducted using all available effect sizes, so that all information can be preserved and maximum statistical power can be achieved. Since each risk domain comprised qualitatively different risk factors for life-course persistent offending relative to adolescence-limited offending, we performed a separate meta-analysis for each of the 14 risk domains.

For the statistical analyses we used the function "rma.mv" of the metafor package (Viechtbauer, 2010) in the R environment (version 3.2.0; R Core Team, 2015). The R syntax we used was written so that the three sources of variance as described by for instance Van den Noortgate et al. (2013, 2014) were modeled (Wibbelink & Assink, in preparation). The *t*-distribution was used for testing individual regression coefficients of the meta-analytic models and for calculating the corresponding confidence intervals (Knapp & Hartung, 2003). When models were extended with categorical moderators consisting of three or more categories, the omnibus test of the null hypothesis that all group mean effect sizes are equal, followed an *F*-distribution. To determine whether the variance between effect sizes from the same study (Level 2), and the variance between studies (Level 3) were significant, two separate one-tailed log-likelihood-ratio-tests were performed in which the deviance of the full model was compared to the deviance of a model excluding one of the variance parameters. The sampling variance of observed effect sizes (Level 1) was estimated by using the formula of Cheung (2014). All model parameters were estimated using the restricted maximum likelihood estimation method and before moderator analyses were conducted, each continuous variable was centered around its mean and dichotomous dummy variables were created for all categorical variables. The log-likelihood-ratio-tests were performed one-tailed and all other tests were performed two-tailed. We considered *p*-values $< .05$ as statistically significant.

3. Results

3.1. Descriptives, central tendency and variability, and assessment of missing data

The present study included 48 manuscripts, describing 55 studies (k) from 1995 to 2014. The total sample of $N = 13872$ juveniles consisted of $n = 4596$ life-course persistent offenders and $n = 9276$ adolescence-limited offenders and the size of the samples described in the included studies (at start of the study) ranged from 98 to 4164 participants. The mean age of the participants at start of the study was 11.80 years ($SD = 6.51$). Studies were conducted in the USA ($k = 29$), Canada ($k = 7$), Europe ($k = 13$), Australia or New Zealand ($k = 5$), and in one non-Western country ($k = 1$). In total, the coded studies produced 1014 separate effect sizes, each reflecting the effect of a risk factor for life-course persistent offending relative to adolescence-limited offending.

An overview of the overall effects of the 14 domains of risk factors is presented in Table 1. Each overall effect represents the effect of a risk domain for life-course persistent offending relative to adolescence-limited offending. The overall effect of eleven domains was significant, and the magnitude ranged from small ($d = 0.200$ for the attitude domain) to large ($d = 0.758$ for the criminal history domain), based on criteria for the interpretation of effect sizes as formulated by Cohen (1988). The overall effect of three domains (i.e., the background, physical health, and neighborhood domains) was not significant, meaning that these effects did not significantly deviate from zero. The results of the likelihood-ratio tests showed that there was significant variance between effect sizes from the same study (i.e., level 2 variance) in eleven risk domains and that there was significant variance between studies (i.e., level 3 variance) in nine risk domains (see Table 1). We conducted moderator analyses within 13 risk domains in order to determine characteristics of effect sizes or studies that can explain level 2 or level 3 variance. There was no significant level 2 or level 3 variance in the sexual behavior domain, and we therefore did not perform moderator analyses within this risk domain.

The trim and fill analyses suggested that bias was present in 10 of the 14 domains of risk factors, as indicated by an asymmetrical distribution of effect sizes, and therefore "corrected" overall effects were calculated for these risk domains (see Table 2). After these trim and fill analyses, the overall effects of ten domains were significant and ranged from a small effect ($d = 0.243$ for the neurocognition/physiology domain) to a large effect ($d = 1.105$ for the criminal history domain), according

Table 1
Results for the overall mean effect sizes of the 14 risk domains.

Domain of risk factors	# Studies	# ES	Mean *d* (SE)	95% CI	Sig. mean *d* (*p*)	% Var. at level 1	Level 2 variance	% Var. at level 2	Level 3 variance	% Var. at level 3	Statistical difference between domains[a] ($p < .05$)
(1) Criminal history	29	148	0.758 (0.104)	0.553, 0.964	<.001***	3.3	.315***	57.1	.218***	39.6	3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14
(2) Aggression	15	25	0.561 (0.118)	0.317, 0.805	<.001***	15.9	.017	7.7	.166**	76.4	6, 7, 8, 9, 10, 11, 13
(3) Alcohol/drug abuse	19	57	0.432 (0.082)	0.269, 0.596	<.001***	16.9	.023*	16.9	.090***	66.2	1, 9, 10, 13
(4) Sexual behavior	3	7	0.408 (0.096)	0.174, 0.643	.005**	52.3	.030	47.7	.000	0.0	13
(5) Relationship	19	51	0.384 (0.080)	0.223, 0.545	<.001***	10.3	.132***	65.4	.049	24.4	1, 9, 10, 13
(6) Emotional and behavioral problems	32	150	0.373 (0.057)	0.260, 0.486	<.001***	17.8	.060***	37.8	.071***	44.4	1, 2, 10, 13
(7) School/employment	21	63	0.295 (0.052)	0.192, 0.399	<.001***	21.7	.052***	54.5	.023	23.8	1, 2, 10, 13
(8) Other	17	27	0.265 (0.075)	0.110, 0.419	.002**	16.5	.015*	16.8	.059*	66.7	1, 2
(9) Family	38	273	0.250 (0.032)	0.186, 0.313	<.001***	25.1	.070***	59.5	.018***	15.5	1, 2, 3, 5
(10) Neurocognition/ physiology (static)	27	132	0.243 (0.049)	0.157, 0.349	<.001***	23.0	.011***	15.2	.044***	61.8	1, 2, 3, 5, 6, 7
(11) Background (static)	24	32	0.207 (0.108)	−0.013, 0.426	.064	9.5	.062**	21.8	.195***	68.7	1, 2
(12) Attitude	6	19	0.200 (0.092)	0.006, 0.394	.044*	16.1	.046***	52.0	.028	32.0	1
(13) Physical health	6	14	0.083 (0.250)	−0.457, 0.623	.745	9.4	.015	3.8	.340*	86.8	1, 2, 3, 4, 5, 6, 7
(14) Neighborhood	10	16	−0.087 (0.180)	−0.470, 0.297	.637	4.6	.100***	28.0	.240	67.3	1

Note. # studies = number of studies; # ES = number of effect sizes; SE = standard error; CI = confidence interval; Sig = significance; Mean *d* = mean effect size (*d*); % Var = percentage of variance explained; Level 2 variance = variance between effect sizes from the same study; Level 3 variance = variance between studies.
[a] The numbers in this column refer to other domains of risk factors from which one domain statistically differs at $p < .05$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

to the criteria of Cohen (1988) for interpreting the magnitude of effect sizes. The overall effects of the background, physical health, and neighborhood domains were still not significant, and the overall effect of the attitude domain was reduced to non-significant. For each risk domain, the funnel plot of effect sizes against the standard error is presented in Appendix E.

3.2. Moderator analyses

The results of all moderator analyses are presented in Table 3, where moderators are classified into sample descriptors, research design descriptors, and risk factor characteristics. Below, the moderating variables tested are described by domain of risk factors in which effect sizes proved to be heterogeneous (i.e., significant level 2 and/or level 3 variance; see Table 1).

3.2.1. Criminal history

First, smaller effects of criminal history risk factors for persistent offending were found when the percentage of cultural minorities in samples increased. Second, we found that effects of criminal history factors that were measured using official records were larger than effects of factors that were measured using self-reports. We found no significant moderating effect of percentage of males.

3.2.2. Aggression

For aggression, we only found that effects of risk factors for persistent offending were smaller when the percentage of males increased. No significant moderating effect was found of percentage of minorities, type of measurement, type of factor, and age period in which risk factors are present.

3.2.3. Alcohol/drug abuse

We only found a moderating effect of the age period in which risk factors are present. Risk factors for persistent offending present during adolescence yielded larger effects than risk factors present during childhood. No significant moderating effect was found of percentage of males, percentage of minorities, type of factor, age period in which risk factors are present, and type of abuse.

3.2.4. Relationship

We found that effects of relationship risk factors decreased when the percentage of minorities in samples increased. Further, we found a

Table 2
Results for the overall mean effect sizes of the 14 risk domains after conducting trim and fill analyses.

Domain of risk factors	# Studies	# ES	Mean *d* (SE)	95% CI	Sig. mean *d* (*p*)
(1) Criminal history	41	178	1.105 (0.119)	0.871, 1.340	<.001***
(2) Aggression	16	26	0.606 (0.119)	0.360, 0.852	<.001***
(3) Alcohol/drug abuse	21	59	0.471 (0.088)	0.295, 0.647	<.001***
(4) Sexual behavior	–	–	–	–	–
(5) Relationship	22	54	0.459 (0.089)	0.281, 0.637	<.001***
(6) Emotional and behavioral problems	42	168	0.499 (0.060)	0.381, 0.617	<.001***
(7) School/employment	–	–	–	–	–
(8) Other	20	30	0.353 (0.080)	0.190, 0.516	<.001***
(9) Family	–	–	–	–	–
(10) Neurocognition/physiology (static)	–	–	–	–	–
(11) Background (static)	29	38	−0.018 (0.133)	−0.288, 0.252	.894
(12) Attitude	9	23	0.082 (0.096)	−0.117, 0.282	.401
(13) Physical health	10	19	0.325 (0.179)	−0.052, 0.702	.087
(14) Neighborhood	11	17	−0.013 (0.181)	−0.396, 0.371	.946

Note. # studies = number of studies; # ES = number of effect sizes; Mean d = mean effect size (*d*); SE = standard error; CI = confidence interval; Sig = significance.
Dashes indicate a symmetrical distribution of effect sizes in a risk domain, meaning that trimming and filling of effect sizes was not necessary.
*** $p < .001$.

Table 3
Results for continuous and categorical moderators (bivariate models).

Moderator variables	# Studies	# ES	Intercept (95% CI)/mean *d* (95% CI)	β (95% CI)	*F* (df1, df2)[a]	*p*[b]	Level 2 variance	Level 3 variance
(1) Criminal history								
Sample descriptors								
Percentage of males	29	148	0.774 (0.562, 0.986)***	−0.003 (−0.009, −0.004)	$F(1, 146) = 0.612$	.435	.315***	.225***
Percentage of minorities	23	97	0.758 (0.536, 0.979)***	−0.008 (−0.015, −0.001)*	$F(1, 95) = 4.534$	.036*	.377***	.151***
Research design descriptors								
Type of measurement					$F(3, 133) = 3.194$	.026*	.336***	.155***
Self-report (RC)	10	34	0.545 (0.229, 0.861)***					
Parent-report	1	1	0.057 (−1.211, 1.324)	−0.488 (−1.751, 0.775)				
Official records	16	82	1.005 (0.763, 1.247)***	0.460 (0.086, 0.835)*				
Other	9	20	0.533 (0.154, 0.912)**	−0.012 (−0.493, 0.469)				
(2) Aggression								
Sample descriptors								
Percentage of males	15	25	0.563 (0.350, 0.776)	−0.007 (−0.014, −0.0004)*	$F(1, 22) = 4.793$	.039*	.017	.116**
Percentage of minorities	10	16	0.742 (0.418, 1.065)	−0.006 (−0.016, 0.003)	$F(1, 14) = 2.061$	.173	.046	.146*
Research design descriptors								
Type of measurement					$F(4, 20) = 1.942$	.143	.005	.165**
Self-report (RC)	7	10	0.567 (0.243, 0.891)**					
Parent-report	3	3	0.593 (0.174, 1.012)**	0.026 (−0.398, 0.451)				
Teacher report	5	7	0.881 (0.530, 1.232)***	0.314 (−0.037, 0.665)				
Peer report	1	7	0.022 (−0.876, 0.920)	−0.545 (−1.500, 0.410)				
Other	3	3	0.349 (−0.188, 0.886)	−0.218 (−0.845, 0.410)				
Risk factor characteristics								
Type of factor					$F(1, 23) = 0.151$	.701	.017	.178**
Static factors (RC)	2	2	0.685 (−0.016, 1.386)					
Dynamic factors	13	23	0.544 (0.274, 0.814)***	−0.141 (−0.893, 0.610)				
Age period in which risk factors are present					$F(2, 22) = 2.030$	.155	.026	.114
Childhood (0–12) (RC)	5	7	0.773 (0.434, 1.112)***					
Adolescence (12–23)	10	16	0.428 (0.170, 0.687)**	−0.345 (−0.709, 0.019)				
Life-course (0–23)	2	2	0.252 (−0.413, 0.917)	−0.092 (−0.792, 0.607)				
(3) Alcohol/drug abuse								
Sample descriptors								
Percentage of males	19	57	0.436 (0.273, 0.599)***	−0.006 (−0.012, 0.001)	$F(1, 55) = 2.604$	.112	.022*	.090***
Percentage of minorities	14	47	0.445 (0.273, 0.616)***	−0.001 (−0.007, 0.006)	$F(1, 45) = 0.080$	.778	.021*	.073***
Risk factor characteristics								
Type of factor					$F(1, 55) = 1.080$	.303	.024*	.081***
Static factors (RC)	8	23	0.344 (0.113, 0.576)**					
Dynamic factors	12	34	0.489 (0.297, 0.680)***	0.145 (−0.134, 0.424)				
Age period in which risk factors are present					$F(2, 52) = 6.296$	.004**	.013	.128***
Childhood (0–12) (RC)	1	2	−0.407 (−0.914, 0.100)					
Adolescence (12–23)	15	48	0.441 (0.243, 0.638)***	0.847 (0.366, 1.328)***				
Life-course (0–23)	4	5	0.388 (0.075, 0.701)*	0.795 (0.239, 1.350)**				
Subdomain of risk factors					$F(4, 52) = 0.570$	.685	.026**	.098***
Only alcohol abuse (RC)	10	17	0.397 (0.182, 0.612)***					
Only drug abuse	10	23	0.455 (0.239, 0.671)***	0.058 (−0.115, 0.232)				
Alcohol and/or drug abuse	9	12	0.467 (0.213, 0.722)***	0.071 (−0.228, 0.369)				
Smoking	2	4	0.264 (−0.056, 0.584)	−0.133 (−0.415, 0.150)				
Gambling	1	1	0.239 (−0.325, 0.803)	−0.158 (−0.702, 0.386)				
(5) Relationship								
Sample descriptors								
Percentage of males	19	51	0.386 (0.223, 0.549)***	−0.002 (−0.007, 0.004)	$F(1, 49) = 0.282$	.598	.132***	.051
Percentage of minorities	15	45	0.419 (0.271, 0.567)***	−0.006 (−0.011, −0.001)*	$F(1, 43) = 6.454$	.015*	.147***	.017
Risk factor characteristics								
Type of factor					$F(1, 49) = 2.640$	.111	.138***	.036
Static factors (RC)	3	3	−0.016 (−0.537, 0.504)					
Dynamic factors	19	48	0.413 (0.257, 0.569)***	0.429 (−0.102, 0.961)				
Age period in which risk factors are present					$F(2, 45) = 3.507$	.038*	.100***	.110*
Childhood (0–12) (RC)	7	12	0.700 (0.385, 1.015)***					
Adolescence (12–23)	16	35	0.281 (0.064, 0.498)*	−0.419 (−0.742, 0.095)*				
Life-course (0–23)	1	1	0.135 (−0.720, 0.989)	−0.565 (−1.464, 0.334)				
(6) Emotional and behavioral problems								
Sample descriptors								
Percentage of males	32	150	0.383 (0.273, 0.493)***	−0.003 (−0.008, 0.001)	$F(1, 148) = 2.282$	.133	.061***	.063***
Percentage of minorities	24	110	0.412 (0.277, 0.547)***	−0.003 (−0.007, 0.002)	$F(1, 108) = 1.618$	.206	.066***	.070***
Risk factor characteristics								
Type of factor					$F(1, 148) = 2.581$	.110	.059***	.066***
Static factors (RC)	5	6	0.630 (0.294, 0.967)***					
Dynamic factors	31	144	0.353 (0.239, 0.466)***	−0.278 (−0.620, 0.064)				

(continued on next page)

Table 3 (*continued*)

Moderator variables	# Studies	# ES	Intercept (95% CI)/mean *d* (95% CI)	β (95% CI)	*F* (df1, df2)[a]	*p*[b]	Level 2 variance	Level 3 variance
Age period in which risk factors are present					$F(2, 132) = 1.236$	.294	.066***	.065***
Childhood (0–12) (RC)	14	52	0.378 (0.228, 0.529)***					
Adolescence (12–23)	22	80	0.324 (0.192, 0.456)***	−0.054 (−0.206, 0.097)				
Life-course (0–23)	2	3	0.688 (0.203, 1.173)**	0.310 (−0.187, 0.807)				
Subdomain of risk factors					$F(3, 146) = 0.755$	.521	.062***	.067***
Internalizing problems (RC)	14	38	0.295 (0.144, 0.447)***					
Externalizing problems	28	77	0.392 (0.269, 0.516)***	0.097 (−0.407, 0.235)				
Internalizing and/or externalizing problems	4	5	0.455 (0.076, 0.834)*	0.160 (−0.233, 0.553)				
Psychopathy-related traits	11	30	0.376 (0.209, 0.543)***	0.081 (−0.093, 0.255)				
(7) School/employment								
Sample descriptors								
Percentage of males	21	63	0.296 (0.190, 0.403)***	−0.003 (−0.007, 0.001)	$F(1, 61) = 2.164$	.146	.050***	.027*
Percentage of minorities	14	40	0.335 (0.198, 0.472)***	0.001 (−0.003, 0.005)	$F(1, 38) = 0.145$	.706	.062***	.026
Risk factor characteristics								
Type of factor					$F(1, 61) = 2.713$	.105	.046***	.029
Static factors (RC)	11	17	0.196 (0.032, 0.360)*					
Dynamic factors	19	46	0.337 (0.219, 0.455)***	0.141 (−0.030, 0.313)				
Age period in which risk factors are present					$F(2, 57) = 0.104$	.902	.051***	.015
Childhood (0–12) (RC)	8	15	0.299 (0.120, 0.477)**					
Adolescence (12–23)	16	39	0.255 (0.140, 0.370)***	−0.044 (−0.243, 0.156)				
Life-course (0–23)	3	6	0.030 (−0.257, 0.316)	−0.014 (−0.331, 0.303)				
Subdomain of risk factors					$F(4, 58) = 2.015$	.104	.047***	.021
Work-related factors (RC)	4	7	0.147 (−0.091, 0.385)					
Poor academic achievement	19	30	0.254 (0.128, 0.380)***	0.107 (−0.147, 0.360)				
Poor academic behavior	11	16	0.472 (0.306, 0.639)***	0.325 (0.053, 0.598)*				
Attitude-/interest-/motivation-related factors	4	7	0.259 (0.015, 0.502)*	0.111 (−0.223, 0.446)				
Other	3	3	0.227 (−0.109, 0.555)	0.076 (−0.319, 0.471)				
(8) Other								
Sample descriptors								
Percentage of males	15	25	0.288 (0.108, 0.467)**	−0.003 (−0.013, 0.007)	$F(1, 23) = 0.440$	.514	.015*	.075*
Percentage of minorities	14	21	0.318 (0.154, 0.482)***	−0.002 (−0.007, 0.003)	$F(1, 19) = 0.676$	.421	.012	.051
Risk factor characteristics								
Type of factor					$F(1, 25) = 9.948$	.004**	.000	.083**
Static factors (RC)	12	11	0.194 (0.020, 0.368)*					
Dynamic factors	15	9	0.373 (0.196, 0.551)***	0.179 (0.062, 0.296)**				
Age period in which risk factors are present					$F(2, 21) = 0.207$	.815	.010*	.076
Childhood (0–12) (RC)	2	4	0.116 (−0.391, 0.623)					
Adolescence (12–23)	9	14	0.282 (0.078, 0.486)*	0.167 (−0.380, 0.713)				
Life-course (0–23)	6	6	0.262 (0.032, 0.492)*	0.147 (−0.410, 0.703)				
Subdomain of risk factors					$F(7, 19) = 2.394$	.062	.000	.077**
Violence experience/violent victimization (RC)	3	4	0.145 (−0.174, 0.463)					
Personality-related traits	2	4	0.060 (−0.397, 0.518)	−0.085 (−0.642, 0.473)				
Cultural adjustment difficulties	2	4	0.408 (−0.045, 0.860)	0.263 (−0.290, 0.817)				
Financial management problems	1	2	−0.167 (−0.829, 0.495)	−0.312 (−1.046, 0.423)				
Negative biosocial interaction	2	2	0.156 (−0.312, 0.624)	0.011 (−0.555, 0.577)				
Experience of negative or stressful life-events	3	3	0.356 (0.101, 0.611)**	0.211 (−0.130, 0.553)				
Traditional strain	3	3	0.469 (0.208, 0.730)**	0.324 (−0.020, 0.668)				
Other	4	5	0.538 (0.293, 0.783)***	0.394 (0.072, 0.715)*				
(9) Family								
Sample descriptors								
Percentage of males	36	257	0.265 (0.199, 0.330)***	−0.001 (−0.003, 0.002)	$F(1, 255) = 0.221$	.639	.078***	.017**
Percentage of minorities	29	198	0.186 (0.123, 0.249)***	0.000 (−0.002, 0.001)	$F(1, 196) = 0.156$	.693	.073***	.009
Risk factor characteristics								
Type of factor					$F(1, 271) = 4.497$	.035*	.070***	.017**
Static factors (RC)	35	167	0.285 (0.215, 0.356)***					
Dynamic factors	27	106	0.193 (0.111, 0.274)***	−0.093 (−0.179, −0.007)*				
Age period in which risk factors are present					$F(2, 259) = 2.280$	.104	.070***	.016**
Childhood (0–12) (RC)	13	64	0.252 (0.149, 0.354)***					
Adolescence (12–23)	17	57	0.164 (0.055, 0.272)**	−0.088 (−0.228, 0.052)				
Life-course (0–23)	32	141	0.295 (0.221, 0.370)***	0.044 (−0.061, 0.149)				
Subdomain of risk factors					$F(3, 269) = 7.351$	<.001***	.062***	.020***
Mother-related risk factors (RC)	16	62	0.061 (−0.040, 0.162)					
Father-related risk factors	7	12	0.244 (0.057, 0.430)*	0.183 (−0.011, 0.376)[c]				
Sibling-related risk factors	7	13	0.312 (0.132, 0.491)***	0.251 (0.055, 0.446)*				
Family-related risk factors	37	186	0.303 (0.234, 0.372)***	0.242 (0.140, 0.345)***				

Table 3 (*continued*)

Moderator variables	# Studies	# ES	Intercept (95% CI)/mean *d* (95% CI)	β (95% CI)	*F* (df1, df2)[a]	*p*[b]	Level 2 variance	Level 3 variance
(10) Neurocognition/physiology (static)								
Sample descriptors								
Percentage of males	27	132	0.239 (0.161, 0.317)***	−0.004 (−0.006, −0.002)**	$F(1, 130) = 11.115$	.001**	.011***	.025***
Percentage of minorities	19	107	0.190 (0.082, 0.297)***	0.000 (−0.003, 0.002)	$F(1, 105) = 0.041$	.841	.015***	.037***
Risk factor characteristics								
Subdomain of risk factors					$F(8, 123) = 4.675$	<.001***	.004	.042***
General intelligence (RC)	15	22	0.278 (0.169, 0.387)***					
Verbal intelligence	21	40	0.274 (0.174, 0.374)***	−0.004 (−0.082, 0.074)				
Spatial intelligence	8	17	0.257 (0.141, 0.372)***	−0.021 (−0.113, 0.072)				
Numeric reasoning	5	8	0.292 (0.165, 0.419)***	0.015 (−0.087, 0.116)				
Attention problems	5	6	0.246 (0.037, 0.455)*	−0.032 (−0.242, 0.179)				
Low resting heart rate	4	4	0.650 (0.446, 0.853)***	0.372 (0.175, 0.570)***				
Cognitive flexibility	4	10	0.001 (−0.161, 0.163)	−0.277 (−0.432, −0.122)***				
Other nonverbal intelligence factors	7	8	0.252 (0.110, 0.394)***	−0.026 (−0.150, 0.099)				
Other neurocognitive/ physiological factors	7	17	0.166 (0.032, 0.299)*	−0.112 (−0.238, 0.014)				
(11) Background (static)								
Sample descriptors								
Percentage of males	22	30	0.199 (−0.048, 0.446)	−0.002 (−0.014, 0.009)	$F(1, 28) = 0.160$	.692	.066**	.224***
Percentage of minorities	22	29	0.190 (−0.037, 0.418)	−0.004 (−0.012, 0.004)	$F(1, 27) = 0.871$	.359	.030	.216***
Risk factor characteristics								
Subdomain of risk factors					$F(1, 28) = 4.419$	.045*	.055*	.172***
Being male (RC)	11	11	0.433 (0.142, 0.725)**					
Being non-Caucasian	15	19	0.096 (−0.153, 0.345)	−0.337 (−0.666, −0.009)*				
(12) Attitude								
Sample descriptors								
Percentage of males	6	19	0.208 (0.006, 0.410)*	0.005 (−0.007, 0.017)	$F(1, 17) = 0.754$	.397	.046***	.032
Percentage of minorities	5	17	0.161 (−0.063, 0.384)	−0.003 (−0.014, 0.008)	$F(1, 15) = 0.354$	.561	.047***	.035
Risk factor characteristics								
Age period in which risk factors are present					$F(1, 17) = 1.4552$	.244	.047***	.024
Childhood (0–12) (RC)	1	4	−0.024 (−0.457, 0.409)					
Adolescence (12–23)	5	15	0.251 (0.043, 0.458)*	0.275 (−0.206, 0.755)				
(13) Physical health								
Sample descriptors								
Percentage of males	6	14	0.152 (−0.482, 0.786)	0.021 (−0.053, 0.094)	$F(1, 12) = 0.367$	.556	.015	.395*
Percentage of minorities	4	10	−0.305 (−1.622, 1.012)	0.021 (−0.022, 0.064)	$F(1, 8) = 1.288$	.289	.000	.528**
Risk factor characteristics								
Type of factor					$F(1, 12) = 0.760$	.401	.015	.365*
Static factors (RC)	4	11	0.236 (−0.444, 0.915)					
Dynamic factors	2	3	−0.249 (−1.252, 0.754)	−0.485 (−1.696, 0.727)				
Age period in which risk factors are present					$F(2, 11) = 1.089$	.370	.021	.225
Childhood (0–12) (RC)	2	2	0.444 (−0.319, 1.206)					
Adolescence (12–23)	3	4	−0.085 (−0.735, 0.566)	−0.528 (−1.324, 0.268)				
Life-course (0–23)	2	8	0.022 (−7.409, 0.785)	−0.422 (−1.500, 0.657)				
(14) Neighborhood								
Sample descriptors								
Percentage of males	8	12	−0.021 (−0.565, 0.523)	0.008 (−0.031, 0.047)	$F(1, 10) = 0.213$	.654	.154***	.339
Percentage of minorities	10	16	−0.080 (−0.492, 0.332)	0.002 (−0.012, 0.016)	$F(1, 14) = 0.087$	.773	.096***	.286
Risk factor characteristics								
Type of factor					$F(1, 14) = 0.028$	.869	.096***	.288
Static factors (RC)	7	11	−0.105 (−0.598, 0.388)					
Dynamic factors	3	5	−0.035 (−0.787, 0.718)	0.070 (−0.829, 0.970)				
Age period in which risk factors are present					$F(2, 13) = 1.780$	.207	.094***	.203
Childhood (0–12) (RC)	1	1	0.810 (−0.421, 2.040)					
Adolescence (12–23)	4	6	0.007 (−0.566, 0.580)	−0.803 (−2.160, 0.555)				
Life-course (0–23)	5	9	−0.317 (−0.826, 0.192)	−1.127 (−2.459, 0.205)				

Note. # studies = number of studies; # ES = number of effect sizes; mean *d* = mean effect size (*d*); CI = confidence interval; β = estimated regression coefficient; Level 2 variance = variance between effect sizes from the same study; Level 3 variance = variance between studies.

[a] Omnibus test of all regression coefficients in the model.

[b] *p*-Value of the omnibus test.

[c] Not significant at the .05 level, but a trend towards significance was observed, $t(10) = 1.861$, $p = .064$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

moderating effect of the age period in which risk factors are present. Effects of relationship risk factors were smaller during adolescence than during childhood. Percentage of males and type of factor did not significantly moderate the relationship effect.

3.2.5. Emotional and behavioral problems

None of the variables tested (i.e., percentage of males, percentage of minorities, subdomain of risk factors, type of factor, and age period in which risk factors are present) significantly moderated the effect of this risk domain.

3.2.6. School/employment

None of the variables tested (i.e., percentage of males, percentage of minorities, subdomain of risk factors, type of factor, and age period in which risk factors are present) significantly moderated the effect of this risk domain.

3.2.7. Other

We only found a moderating effect of type of factor. Dynamic risk factors yielded a larger effect than static risk factors. No significant moderating effect was found of percentage of males, percentage of minorities, subdomain of risk factors, and age period in which risk factors are present.

3.2.8. Family

First, we found that the effect of family risk factors for persistent offending was moderated by the family member. The effects of sibling-related risk factors and family-related risk factors (i.e., parent not specified) were larger than mother-related risk factors. Although the mean effect of father-related risk factors did not significantly differ from the effect of mother-related risk factors, there was a trend showing that the effect of father-related risk factors was larger. Further, we found that the effect of dynamic risk factors was smaller than the effect of static risk factors. We found no significant moderating effect of the variables percentage of males, percentage of minorities, and age period in which risk factors are present.

3.2.9. Neurocognition/physiology (static)

We found that effects of risk factors for persistent offending decreased when the percentage of males in the samples increased. We also found a moderating effect of two subdomains of risk factors. The effect of the subdomain low resting heart rate was higher than the effect of the subdomain general intelligence, whereas the effect of the subdomain cognitive flexibility was lower (and not significant) than the effect of the subdomain general intelligence. No significant moderating effect was found of percentage of minorities.

3.2.10. Background (static)

A moderating effect was found of the subdomain of risk factors for persistent offending. The effect of being non-Caucasian was smaller than the effect of being male. No significant moderating effect was found of percentage of males and percentage of minorities.

3.2.11. Attitude

None of the variables tested (i.e., percentage of males, percentage of minorities, and age period in which risk factors are present) significantly moderated the effect of this risk domain.

3.2.12. Physical health

None of the variables tested (i.e., percentage of males, percentage of minorities, type of factor, and age period in which risk factors are present) significantly moderated the effect of this risk domain.

3.2.13. Neighborhood

None of the variables tested (i.e., percentage of males, percentage of minorities, type of factor, and age period in which risk factors are present) significantly moderated the effect of this risk domain.

4. Discussion

The present study aimed to generate more specific knowledge on the association between different types of risk factors and life-course persistent offending by summarizing the effects of risk factors meta-analytically. More specifically, we quantitatively examined the effects of several risk domains (i.e., groups of more or less similar risk factors) for life-course persistent offending relative to adolescence-limited offending. Furthermore, we examined whether the effects of these risk domains were moderated by sample descriptors, research design descriptors, and risk factor characteristics.

4.1. Overall effect of domains of risk factors

A significant overall effect size was found for 11 of the 14 domains of risk factors, which ranged from Cohens' $d = 0.200$ (for the attitude domain) to $d = 0.758$ (for the criminal history domain). The (static) background domain, the physical health domain, and the neighborhood domain showed no significant overall effect, meaning that background characteristics, rates of physical health problems, and neighborhood characteristics of life-course persistent offenders were relatively similar to those of adolescence-limited offenders. We found that levels of risk factors in most domains were significantly higher in life-course persistent offenders than in adolescence-limited offenders. In general, these findings confirm that exposure to risk factors in different domains increases the chance for persistence in offending, indicating that multiple risk domains are involved in delinquent behavior (e.g., Loeber, Burke, & Pardini, 2009; Loeber, Farrington, et al., 2008; Loeber, Slot, et al., 2008; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002).

The strongest effects were found in the criminal history and aggression domains. This was to be expected, since Moffitt (1993, 2006) stated in her dual taxonomy of offending behavior that life-course persistent offenders primarily differ from adolescence-limited offenders in that they exhibit higher levels of antisocial behavior. The next strongest effects were found in the following risk domains: alcohol/drug abuse, sexual behavior, relationship, emotional and behavioral problems, and school/employment, which are encouraging findings because behavior related to these risk domains are dynamic in nature, and can therefore be targeted by intervention strategies. Further, it is interesting to note that compared to other risk domains, we found a relatively small effect in the family domain both during childhood and adolescence. This indicates that differences in family problems between life-course persistent offenders and adolescence-limited offenders are relatively small. In fact, and as presented in Table 1, there are several other individual and social risk domains of which the effect is larger compared to the effect of the family domain (indicated by significant d values exceeding 0.250). However, important to note is that the rather small effect of the family domain does not imply that familial factors are *in general* weakly associated with life-course persistent offending. In the present study, we examined the effect of risk domains for persistent offending relative to adolescence-limited offending, and we therefore cannot make inferences about the general importance of the family and other risk domains. In order to make such inferences, future research is necessary in which the effects of risk domains for life-course persistent offending are examined relative to non-offenders (i.e., normal controls).

Also interesting is the finding of a relatively small effect of the domain comprising neurocognitive and physiological risk factors ($d = 0.243$). In recent years, there has been increasing evidence of a link between neuropsychological impairments and (severe) antisocial outcomes (e.g., Ishikawa & Raine, 2002; Moffitt, 1993; Morgan &

Lilienfeld, 2000; Raine, 1993). In the present study, we found a rather small effect in the neurocognitive domain, implying that exposure to neurocognitive and physiological risk factors is only somewhat higher in life-course persistent offenders than in adolescence-limited offenders. Although this effect was small, it is possible that an interaction between children's neuropsychological impairments and a disadvantaged environment may lead to the life-course persistent pattern of antisocial behavior, as proposed by Moffitt (1993, 2006). On the other hand, this rather small effect may also imply that a true categorical distinction between the life-course persistent and adolescence-limited offender profile based on the nature of the developmental antecedents is not very likely. This reasoning is in line with Fairchild et al. (2013), who concluded that there are similarities in brain structure and function, neuropsychological performance, and cortisol reactivity between childhood-onset and adolescence-onset forms of conduct disorder. However, it should be noted that we found a moderating effect of subdomains of risk factors within the neurocognitive/physiology risk domain, indicating that the effect of risk factors for life-course persistent offending relative to adolescence-limited offending is not the same for all neurocognitive risk factors. Specifically, we found a relatively large mean effect for risk factors pertaining to a low resting heart rate ($d = 0.650$) and despite the low number of risk factors in this subdomain, this result is in line with previous studies showing a robust relation between low resting heart rate and antisocial behavior (e.g., Ortiz & Raine, 2004; Portnoy & Farrington, 2015). It should also be noted that most studies in this research area are based on male samples, and therefore an empirical evaluation of the association between children's neuropsychological impairments and antisocial outcomes in female samples is an important topic for future research.

Based on the results of the trim and fill analyses, we found indications for missing data in 10 of the 14 risk domains, meaning that the true overall effect of these domains may be different from the overall effects that were estimated in the present meta-analytic study. For some domains small effect sizes were underrepresented (the adjusted mean d was smaller than the observed mean d), whereas for most other domains large effect sizes were underrepresented (the adjusted mean d was larger than the observed mean d). It is important to note that the adjusted mean effect sizes that were produced by the trim and fill analyses should not be regarded as true mean effects. Terrin, Schmid, Lau, and Olkin (2003) showed in their simulation study that in case of between-study heterogeneity, an inappropriate adjustment for publication bias may be produced by the trim and fill analysis where in reality no bias exists. These findings were later supported by Peters, Sutton, Jones, Abrams, and Rushton (2007), who also conducted a number of simulated meta-analyses and reached the same conclusion. Therefore, the differences between the adjusted and the observed mean effect sizes found in the present study should be interpreted as indicative of a biasing effect due to, for example, publication or selection bias. This biasing effect is considerable for the criminal history domain (Δ mean $d = 0.347$), the physical health domain (Δ mean $d = 0.242$), and the background domain (Δ mean $d = 0.225$), but small for the other domains (Δ mean $d < 0.130$), indicating that the mean effects of seven risk domains would not change substantively had the missing data been found. However, this biasing effect should be taken into account and warrants some caution when interpreting the effects of the risk domains.

4.2. Moderating effects of risk factor characteristics

First, moderator analyses showed that the effects of two risk domains were moderated by the age period in which risk factors were present. In the relationship domain we found a significant mean effect of risk factors for life-course persistent offending relative to adolescence-limited offending, but this effect was largest during childhood. These results are in line with the work of Loeber et al. (1993), who also found that risks related to relationships with parents and other children were more salient in childhood. In the alcohol/drug abuse domain we found a significant mean effect of risk factors present during adolescence and over the life course, but a significant mean effect was not found for risk factors during childhood. This may be explained by the small number of effect sizes (only two) in the childhood category, and therefore, it is not appropriate to conclude that there is no effect of alcohol- and drug-related risk factors during childhood. Peleg-Oren, Saint-Jean, Cardenas, Tammara, and Pierre (2009) showed that an onset of alcohol use prior to the age of 13 is indeed associated with future delinquent behavior, and therefore, the current result should be interpreted with caution.

Further, we found a moderating effect of the type of risk factor (dynamic versus static) within the family domain. Static (i.e., non-treatable) family risk factors, such as a low family SES, a parental history of criminality, a child entering a single parent family at birth, and a large family size, showed larger effects than dynamic factors, such as family relationship problems, low positive parenting, poor parental monitoring, and poor parental discipline practices. This indicates that differences in static family risk factors between life-course persistent and adolescence-limited offenders were relatively large, whereas the offender groups differed less on dynamic family risk factors. This result is not surprising, since static risk factors in general are more strongly associated with future offending behavior and recidivism than dynamic risk factors (Van der Put et al., 2011). However, in the present study, we only found a moderating effect of risk factor type within the family and "other" risk domain (i.e., the domain of risk factors that could not be classified in any other risk domain). This may be explained by low numbers of static and/or dynamic risk factors in several risk domains leading to insufficient statistical power for detecting a moderating effect of the type of risk factor.

In the family domain, the effects of father-, sibling-, and family-related risk factors were remarkably stronger than the non-significant effect that we found for mother-related factors. This indicates that the effect of fathers, siblings, and the family as a whole is greater for the life-course persistent group than for the adolescence-limited group, whereas the effect of mothers is approximately equal for both offending groups. The effect of father-related risk factors may be explained in the light of the overrepresentation of boys in the samples of the primary studies. Fathers are particularly likely to be involved with their sons (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000), who are more prone to delinquency than daughters (Loeber & Hay, 1997). Related to this, fathers are important role models of gender-appropriate behavior for their sons, and since antisocial behavior is especially exhibited by boys and men, in particular male role models may be influential in the development of this negative behavior (DeKlyen, Speltz, & Greenberg, 1998). As for the effect of sibling-related risk factors, our results are in line with previous research demonstrating that siblings influence delinquency, because they also may act as a deviant role model (e.g., Fagan & Najman, 2003; Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001). However, it must be noted that there were only 12 father-related and 13 sibling-related risk factors and that father-related risk factors have not been widely studied yet.

4.3. Moderating effects of sample and research design characteristics

The present study showed that the effect of both the aggression and the neurocognition/physiology risk domains decreased as the percentage of males in samples increased. This means that the effect of risk factors constituting these two domains are larger for females than for males, suggesting that differences in rates of aggression and neurocognitive/physiological problems between life-course persistent offenders and adolescence-limited offenders are larger for females than for males. This implies that particular risk factors determining a female persistent offending trajectory may be different from risk factors determining male persistent delinquency, which has been an important issue in the literature on the etiology of crime (e.g., Fergusson et al.,

2000; Hipwell et al., 2002; Keenan & Shaw, 1997; Silverthorn & Frick, 1999). Because primary research is based on predominantly male samples, it is well possible that effects of other risk domains also differ for male and female offenders. It is therefore important that more research is directed on determining risk factors for persistent offending that are of particular relevance for females, even though rates of (persistent) delinquent behavior are lower for females than for males (Loeber & Keenan, 1994).

Further, we found a moderating effect of cultural minorities on the effect of the criminal history and relationship domains. The effects of these two risk domains decreased when percentages of different cultural minorities in samples increased. In addition, we found no significant mean effect of the factor "being non-Caucasian" in the background domain, implying that this factor cannot be regarded as a risk for persistent offending relative to adolescence-limited offending. These results imply that belonging to a cultural minority does not need to be a risk by itself, and that effects of different risk factors for persistent offending varies between offenders of different cultural minorities. We propose that this variation may be explained, at least to some extent, by assuming that juveniles of different cultural minorities are affected by different socio-environmental influences during their lives. This supports the notion of Moffitt (1993, 2006), as she states that a life-course persistent offending pattern is produced by an interaction between neurological deficits and the physical and social environment in which juveniles are raised. Given the fact that most primary studies were conducted in Western countries and that only few cultural groups were examined, issues of cultural and ethnic differences in risk factors for life-course persistent delinquency have only been scarcely explored. A further exploration of the present results is recommended in future studies to gain a better understanding of which risk factors contribute to life-course persistent delinquency in different ethnic or cultural groups, and why there are differences in effects of (domains of) risk factors between these groups.

As for moderating effects of research design characteristics, we found that the overall mean effect of the criminal history domain was moderated by the way in which delinquency was measured. The mean effect of risk factors based on official data was remarkably larger than the mean effect of risk factors based on self-reported data, and this may be indicative of the problem of underreporting in self-reported data (e.g., Babinski, Hartsough, & Lambert, 2001; Huizinga & Elliott, 1986; Lafayette, Frankle, Pollock, Dyer, & Goff, 2003). Although it is possible that offenders of the life-course persistent trajectory deliberately conceal information on the number and seriousness of committed delinquent acts, recent research showed that the vast majority of convicted individuals is willing to self-report about the conviction of a criminal offense (Auty et al., 2015). Therefore, Auty and colleagues propose that it is more likely that offenders are having problems in accurately recalling criminal offenses that they have committed during their lives, and this may be especially the case for life-course persistent offenders with a long history of numerous offenses. When interpreting the mean effect of the criminal history domain, it is important to be aware of such validity issues in the measurement of delinquency.

4.4. Limitations

Several limitations of the present study should be mentioned. First, the variation in the way offending trajectories were defined in the primary studies prompts caution in interpreting the results obtained in the present meta-analytic study. There were marked differences between primary studies in criteria and methods that were used to distinguish a life-course persistent offending trajectory from an adolescence-limited offending trajectory, leading to heterogeneity in the two offending groups that were compared in the present study. These differences in criteria and methods make it more difficult to determine the true mean effect of domains of risk factors for life-course persistent delinquency relative to adolescence-limited delinquency.

Second, although we considered the effects of domains of risk factors for persistent delinquency as outcome variables and potential moderator variables as explanatory variables in the analyses, the present study does not permit conclusions about causality. In addition, it has been widely acknowledged that risk factors are not present in isolation, but coexist and interact with other risk factors. This accumulation of risk factors means that the more risk factors a juvenile is exposed to, the greater the likelihood of future delinquency will be (e.g., Loeber, Farrington, et al., 2008; Loeber et al., 2009; Prinzie, Hoeve, & Stams, 2008; Sameroff, Bartko, Baldwin, Baldwin, & Seifer, 1998; Van der Laan & Blom, 2006). Moreover, juveniles who are exposed to an accumulation of risks in multiple domains, rather than to risks in a single domain, are at greater risk for later negative outcomes (Loeber, Farrington, et al., 2008; Loeber, Slot, et al., 2008; Loeber et al., 1993; Stouthamer-Loeber et al., 2002). However, in the present study, we were primarily interested in the mean effect of individual risk domains, and we therefore classified each risk factor into one of 14 mutually exclusive risk domains. In this way, we were able to conduct a separate meta-analysis for each risk domain in order to determine the mean effect of groups of (more or less) similar risk factors for life-course persistent offending relative to adolescence-limited offending. Consequently, we were not able to examine which combination of risk domains (or risk factors) may be especially predictive of life-course-persistent offending and this should be examined in future research.

Third, it must be noted that the trim and fill method is not a perfect approach to account for missing data, and there are several methodological difficulties regarding this method. First, Nakagawa and Santos (2012) mentioned that this method has originally been designed for meta-analyses where independence of effect sizes can be assumed. Second, Peters et al. (2007) and Terrin et al. (2003) pointed out that the performance of the trim and fill method is limited when effect sizes prove to be heterogeneous. Third, Egger, Davey-Smith, and Altman (2001) noted that the application of the trim and fill method could mean adding and adjusting for non-existent effect sizes in response to funnel plots that are asymmetrical, simply because of random variation. Despite these shortcomings, there is no best method for detecting and handling missing data in meta-analysis, and every method has its own limitations and drawbacks. The considerable and ongoing debate in scientific literature about the appropriateness of different methods for detecting and handling missing data reflects the importance of interpreting the results from the trim and fill method in the present study with caution.

A final limitation is that many moderator analyses were based on a small number of effect sizes, implying a low statistical power to detect unique contributions of moderating variables in multiple moderator models. Due to this statistical power problem, we chose not to examine multiple moderator models.

4.5. Implications for clinical practice

The current study has a number of implications for clinical practice. First, the results showed that life-course persistent offenders experience much more problems in multiple domains than adolescence-limited offenders. Therefore, in order to prevent that adolescent-onset offenders will become life-course persistent offenders, multifaceted interventions that target problems in multiple risk domains should be offered by professionals from different disciplines. Because of the diverse and specific needs of young offenders, effective collaboration between clinical experts from multiple disciplines is essential.

Second, we found a rather small effect of the family risk domain compared with effects of other risk domains, indicating that the potential of strong family-focused interventions for the prevention or treatment of persistent delinquency may be relatively low. In clinical practice, there are many family-focused interventions, but in light of the present results, clinicians should pay serious attention to individual and social risk domains in addition to the family risk domain. This is

supported by previous research showing positive effects of multimodal programs targeting a variety of criminogenic needs of delinquent juveniles (e.g., De Vries, Hoeve, Assink, Stams, & Asscher, 2014; Lipsey, 1992, 1995; Lipsey & Wilson, 1998). Further, interventions addressing family problems should rather focus on the father, the siblings, and the family as a system than merely on the mother, since our results suggest that fathers and siblings may be more influential in developing life-course persistent offending than mothers.

Third, the results suggest that intervention strategies specifically addressing problematic social interactions (especially with peers) are particularly promising in preventing persistent delinquency when these interventions are offered in a person's childhood years. Therefore, investing in the development and improvement of early intervention efforts targeting these problems seems beneficial. However, clinicians should keep in mind that problems in other risk domains (e.g., emotional and behavioral problems, school problems, and family problems) should be addressed simultaneously in early intervention efforts. Further, since effects of the aggression and neurocognition/physiology risk domains were larger for females than for males, we suggest that interventions should be at least to some extent gender-specific, so that differences in needs between male and female offenders can best be addressed. Similar suggestions have been proposed by other researchers who advocate the development of interventions specifically for girls (e.g., Chesney-Lind, 2005; Hipwell & Loeber, 2006). In terms of cultural differences between offenders, it is also important that interventions be culturally matched to offending juveniles, since the current results reveal a difference in effect of the relationship risk domain between Western and non-Western juvenile offenders. Clinical practitioners should be aware that interventions generally considered as effective for Western juvenile offenders may be less effective or even ineffective for non-Western juvenile offenders. However, future research on the effectiveness of culturally-adapted treatment is necessary, since researchers are divided on this issue (see, for instance, Kumpfer, Alvarado, Smith, & Bellamy, 2002).

A final implication of our study concerns the improvement of risk assessment procedures. Accurate risk assessment is crucial for identifying those adolescent-onset offenders who are at significant risk for becoming a life-course persistent offender, so that interventions are offered to those who would benefit most. To be effective, risk assessment instruments should be based on risk factors that are most strongly associated with life-course persistent offending. In general, our results suggest that these instruments should particularly assess factors relating to criminal history, aggressive behavior, alcohol/drug abuse, sexual behavior, social relationships (especially with peers), emotional and behavioral problems, and school/employment. Risk factors in other risk domains should also be assessed, but should weigh less in determining the risk, since these factors are less associated with life-course persistent offending. However, since we found moderating effects of gender, age, and cultural minorities within several risk domains, implementing a general risk assessment instrument is likely to produce suboptimal risk estimates in different populations. Therefore, risk assessment instruments should be specifically tailored to different groups of offending juveniles by conducting proper validation research in different populations, so that only the most relevant risk factors are properly weighed in estimating the risk for life-course persistent offending (see also the work of Van der Put et al., 2011, 2014 who suggest applying tailored risk assessment strategies).

5. Conclusion

The present review contributes to the literature on risk factors for persistent delinquent behavior of juveniles by examining the effect of several domains of risk factors for life-course persistent offending, relative to adolescence-limited offending. The largest effects were found for domains consisting of risk factors pertaining to criminal history, aggressive behavior, alcohol/drug abuse, sexual behavior, social relationships, emotional/behavior problems, and school/employment, whereas relatively smaller effects were found for family, neurocognitive, and neighborhood-related risk factors. In addition, we found moderating effects of gender, age, and ethnicity within several domains of risk factors. Overall, the results showed that the prevalence of risk factors in multiple domains was larger for life-course persistent than for adolescence-limited offenders, implying that most risk domains play a more or less significant role in the development of persistent delinquent behavior. Contrary to the developmental taxonomy of Moffitt (1993) and in line with Fairchild et al. (2013), we conclude that the difference between life-course persistent and adolescence-limited offenders may be rather quantitative than qualitative. The findings of the present study provide avenues for more focused future research and the development and improvement of both intervention services and risk assessment procedures.

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Contributors

Mark Assink participated in the design of the study, searched for primary studies, coded all primary studies, conducted all statistical analyses, and drafted the manuscript. Claudia E. van der Put and Machteld Hoeve participated in the design of the study and critically reviewed the manuscript. Sanne L. A. de Vries coded 10 primary studies, participated in determining the interrater agreement, and critically reviewed the manuscript. Geert Jan J. M. Stams and Frans J. Oort critically reviewed the manuscript. All authors contributed to and approved the final version of the manuscript.

Conflict of interest

All authors declare that they have no conflicts of interest.

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Supplementary data

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