The Role of Antisocial Cognition in Antisocial Behaviour

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Thesis Declaration Form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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Overview

Antisocial behaviour is a common and significant problem. This thesis focuses on the causal role of antisocial cognition in antisocial behaviour during late childhood and adolescence and comprises three parts.

Part I is a systematic review and narrative synthesis of studies examining the causal nature of the antisocial cognition-antisocial behaviour relationship in older children and adolescents. Whilst the included studies generally support the existence of a reciprocal relationship between antisocial cognition and antisocial behaviour and suggest that antisocial cognition might constitute a causal mechanism of antisocial behaviour in adolescence, more high-quality research is needed to elucidate the causal role of antisocial cognition in antisocial behaviour.

Part II is an empirical study that uses data from the Systemic Therapy for at Risk Teens study and mediation analysis to examine the extent that one aspect of antisocial cognition, namely beliefs and attitudes supporting peer conflict, explains two robust findings in the antisocial literature known as the peer influence effect and peer selection effect. The results suggest that beliefs and attitudes supporting peer conflict could constitute a causal mechanism underlying these effects.

Part III is a critical appraisal that discusses some of the challenges associated with using mediation analysis to establish causal mechanisms in the study of antisocial behaviour and highlights some potential solutions to these problems with respect to the research questions addressed in the empirical paper in Part II of this thesis.

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Part I: Literature Review

A Systematic Review of the Causal Nature of the Relationship Between Antisocial Cognition and Antisocial Behaviour in Older Children and Adolescents

Abstract

Aims: Antisocial behaviour is a common and significant problem. Antisocial cognition has been identified as major risk factor of antisocial behaviour in older children and adolescents, however the causal nature of this relationship is not well understood. This review provides an overview and critical appraisal of research in this area to enlighten about study findings, clarify the strength of evidence and identify areas for future research.

Method: A systematic search strategy was employed to identify studies examining: (1) the reciprocal nature of the relationship between antisocial cognition and antisocial behaviour and (2) antisocial cognition as a causal mechanism of antisocial behaviour using mediation analysis.

Results: Sixteen studies satisfied the inclusion criteria. Findings from these studies generally support the conception of a reciprocal relationship between antisocial cognition and antisocial behaviour and suggest that antisocial cognition might constitute a causal mechanism in the pathway for antisocial behaviour, however significant methodological shortcomings meant that no definitive conclusions could be drawn in this respect.

Conclusion: Research in this area is still in its infancy and more high-quality studies are needed to elucidate the causal role of antisocial cognition in the antisocial behaviour pathway. This in turn could lead to more accurate theoretical models of antisocial behaviour and more effective interventions to prevent or reduce such behaviour. Recommendations for future research in this area are provided.

Introduction

Antisocial behaviour is an ambiguous construct with multiple overlapping definitions. Within the UK, the term is used by government agencies to refer to “conduct that has caused, or is likely to cause, harassment, alarm or distress to any person” (*Anti-social Behaviour, Crime and Policing Act*, 2014, p. 2). This broad definition encompasses offending behaviours in adults (i.e., criminal behaviour) and minors (i.e., delinquent behaviour), along with status offences (i.e., behaviour that is prohibited or unlawful in minors but not adults) and several related behaviours that are socially disruptive or depart from usual or accepted standards (i.e., deviant behaviour). Common examples include noise and vehicle nuisance, truancy, alcohol possession in minors, drug dealing, vandalism, and offensive, threatening or violent behaviour. Within mental health, the term is primarily used to refer to a pattern of repetitive and persistent unwanted behaviour which is indicative of a diagnosis of conduct disorder (CD; American Psychiatric Association, 2013). Critically, this behaviour is deemed to violate the basic rights of others or major age-appropriate societal norms or rules and includes violence towards people and animals, destruction of property, deceitfulness or theft, and serious violations of rules. Substantial research on antisocial behaviour has also been conducted in relation to the construct of juvenile delinquency, usually defined as the habitual committing of criminal acts by someone under the age of 18. Additionally, scholars have categorised antisocial behaviour in terms of overt and covert acts, distinguished by the degree of confrontation (Loeber & Schmaling, 1985).

At least 1.8 million incidents of antisocial behaviour took place in

England and Wales in the year ending March 2016 (Office for National Statistics, 2017). Epidemiological studies suggest that CD is the most common mental health problem in childhood and adolescence globally, with a lifetime prevalence of approximately 6.8% (5.8% female, 7.9% male) (Merikangas et al., 2010). Furthermore, CD is the most common reason for referral to child and adolescent mental health services in Western countries and research suggests that people with the condition are at greater risk of mental health problems in the future, including substance misuse and antisocial personality disorder (Keenan & Wakschlag, 2000; Nock, Kazdin, Hiripi, & Kessler, 2006). Moreover, a diagnosis of CD is strongly associated with poorer educational performance, relationship and family problems, and contact with the criminal justice system (Colman et al., 2009; Hill & Maughan, 2001).

The economic burden of antisocial behaviour on society is considerable, with the most recent government report suggesting that the cost to government agencies of responding to antisocial behaviour in England and Wales is around £3.4 billion per annum (The Police Foundation, 2010), whilst a another report by the Sainsbury Centre for Mental Health (2009) suggests that the total cost of crime attributable to people who had conduct problems in childhood is as high as £60 billion a year in England and Wales.

Antisocial behaviour typically emerges in late childhood or early adolescence, peaks in prevalence during mid-to-late adolescence, and declines thereafter (Loeber & Hay, 1997). A plethora of empirical research studies in the last 30 years has led to the identification of numerous risk

factors that appear to increase a young person's chances of developing antisocial behaviour (Joan, Cathy, & Nancy, 2001). The definition of a *risk factor* varies in the literature, however in the study of antisocial behaviour the term is generally used to refer to any attribute, characteristic or exposure of an individual that has been shown to precede antisocial behaviour and is associated with an increased likelihood of such behaviour. Well-established risk factors for self-reported and officially recorded antisocial behaviour include delinquent peer association, a history of antisocial behaviour, poor parenting practices, and low family socioeconomic status (Andrews & Bonta, 2010). The present review concerns another risk factor believed to play a significant role in the development and maintenance of antisocial behaviour in young people, namely *antisocial cognition.*

Like antisocial behaviour, there is no uniform term or definition for the construct of antisocial cognition. Andrews and Bonta (2010) defined antisocial cognition as "attitudes, values, beliefs, rationalisations and a personal identity that is favourable to crime" (p. 59). Moreover, the authors proposed specific indicators for the construct, including "identification with criminals, negative attitudes toward the law and justice system, a belief that crime will yield rewards, and rationalisations that specify a broad range of conditions under which crime is justified" (p. 59). Similarly, Butler, Fearon, Atkinson and Parker (2007) described antisocial cognitions and attitudes as characterised by "mistrust of authority figures (e.g., police, judges, lawyers), tolerance for law violations, and identification with delinquent peers and criminal a subculture" (p. 722). References to analogous conceptual constructs have been extant in the literature for many years and include

delinquent values (Thornberry, 1987), *moral beliefs* (Matsueda, 1989) and *antisocial attitudes* (Zhang, Loeber, & Stouthamer-Loeber, 1997) to name a few. Additionally, (Walters, 2012b) proposed that *criminal thinking* constitutes a core feature of antisocial cognition, which he said encapsulates different thinking styles that support a criminal lifestyle.

Whilst offering competing accounts about the way in which antisocial cognition and antisocial behaviour might be causally related, most eminent theories of crime recognise the importance of antisocial cognition in the development and maintenance of antisocial behaviour, including social control theory (Hirschi, 1969), general strain theory (Agnew, 1992), differential association theory (Akers, 1998; Sutherland, 1947), psychological inertia theorem (Walters, 2012a), the General Personality and Cognitive Social Learning (GPCSL) perspective of criminal behaviour (Andrews & Bonta, 2010) and recent revisions of the General Theory of Crime (Gottfredson, 2011; Gottfredson & Hirschi, 1990).

Concordant with these theories, large-scale cross-sectional studies have consistently shown a moderate to large association between measures of antisocial cognition and self-reported and officially recorded antisocial behaviour in school children and young offenders (Butler, Leschied, & Fearon, 2007; Butler, Parry, & Fearon, 2015; Levy, 2001; Mak, 1990; Tarry & Emler, 2007). Furthermore, longitudinal studies and narrative and meta-analytic reviews suggest that antisocial cognition is predictive of self-reported and officially recorded antisocial behaviour in high school students and justice-involved youth (Engels, Luijpers, Landsheer, & Meeus, 2004; Hubbard & Pratt, 2002; Lipsey & Derzon, 1998; Skilling & Sorge, 2014;

Wong, Slotboom, & Bijleveld, 2010), as well as general and violent recidivism in young offenders (Grieger & Hosser, 2014; Simourd & Van De Ven, 1999; Walters, 2012b). These findings have led in the identification of antisocial cognition as a major risk factor for antisocial behaviour in young people with or without a history of offending (Andrews & Bonta, 2010).

Risk factors of antisocial behaviour are generally assumed to exert their influence either directly or indirectly as part of a wider causal chain of variables or pathway. Thereby, once a risk factor relationship has been identified in the literature, the next step is to understand *how* the concerned risk factor increases the likelihood of the outcome. The last 20 years has seen a growth in studies investigating the nature of the relationship between antisocial cognition and antisocial behaviour. This research has tended to focus on the reciprocal nature of the relationship between the two variables or identification of causal mechanisms involving antisocial cognition and antisocial behaviour using mediation analysis. A reciprocal relationship is one in which two variables act as both cause and effect with respect to each other overtime, while a *causal mechanism* is “a process that connects the cause and effect and that brings about the effect” (Wikström, 2008, p. 131). Mediation analysis is the most common method for identifying causal mechanisms in the study of antisocial behaviour and provides researchers with a way of examining the extent that a third variable, or mediator (M), transmits the effect of a risk factor (X) to an outcome variable (Y) in an assumed casual sequence such that X leads to M and M leads to Y (Fiedler, Schott, & Meiser, 2011; Mackinnon, Kisbu-Sakarya, & Gottschall, 2013). It is this growing body of work that forms the focus of the current review.

The Current Review

In summary, antisocial behaviour is a common and costly problem that usually begins during late childhood or early adolescence. The identification of antisocial cognition as a major risk factor for antisocial behaviour in children and adolescents has resulted in studies examining how the two constructs might be causally related. The aim of the current review is to provide an overview and critical appraisal of this body of work, to enlighten about study findings, clarify the strength of evidence and identify areas for future research. To this end, the objective was to carry out a systematic review and evaluation of the literature pertinent to the following research question: what is the causal nature of the relationship between antisocial cognition and antisocial behaviour in older children and adolescents?

It is hoped that an improved understanding of the state of the literature regarding the causal nature of the relationship between antisocial cognition and antisocial behaviour will help clarify the causal status of the relationship and validity of current theory in this regard, which in turn could aid the development of more effective interventions to reduce and prevent antisocial behaviour.

Method

Criteria for Considering Studies for this Review

Study inclusion criteria (see Table 1) were guided by the review question. First, as a primary aim, the study had to investigate the relationship between antisocial cognition and any form of self-reported or officially recorded antisocial behaviour (i.e., symptoms characteristic of conduct disorders, delinquent behaviour, deviant behaviour and status offences),

Table 1

Study Inclusion Criteria

Population	• Older children and adolescents (i.e., aged to 10-19 years at study entry). • Studies of children under 10 years of age at study entry if the study included at least two assessments of antisocial cognition between the ages of 10 and 19 years.
Type of study design	• Prospective observational studies of the reciprocal relationship between antisocial cognition and antisocial behaviour. • Prospective observational studies focusing on the relationship between antisocial cognition and antisocial behaviour. • Experimental studies examining the effect of antisocial cognition on antisocial behaviour.
Type of outcome	• Any form of antisocial cognition, assessed using a single psychometric measure or inventory of antisocial cognition. • Any form of self-reported or officially recorded antisocial behaviour. • Any other psychometric measures or inventories of cognitive, emotional or behavioural constructs examined in relation to the antisocial cognition-antisocial behaviour relationship and relevant to the review question.

in a sample of youth (i.e., aged 10 to 19 years at study entry), as part of a reciprocal effects or mediation analysis. Studies examining a younger age group at study entry were included if they comprised at least two assessments of antisocial cognition between the ages of 10 and 19 years. Second, the study needed to comprise a prospective observational (i.e., assess variables repeatedly across two or more points in time) or experimental design. Third, the study had to comprise at least one psychometric measure or inventory of any form of antisocial cognition; however, it was not necessary for the measure or inventory to be standardised.

Exclusion criteria were also applied. Specifically, cross-sectional and longitudinal studies examining only the association or unidirectional relationship between antisocial cognition and antisocial behaviour were excluded, as such studies cannot inform about how the two variables could be causally related. Case studies, case series and proceeding papers were excluded for the same reason. Studies focusing exclusively on sexual offences, underage alcohol use or illicit substance use were also excluded given debate within the literature about whether such offences represent true forms of antisocial behaviour (Driemeyer, Yoon, & Briken, 2011). Research measuring these types of offences as part of a wider psychometric measure or inventory of antisocial behaviour, however, were not excluded. Foreign language papers were not included unless the title or abstract suggested that the citation was highly relevant to the review question.

Search Strategy for Identifying Studies

The strategy for identifying and selecting studies for inclusion in this

review was based on guidance from Petticrew & Gilbody (2004) and the Centre for Reviews and Dissemination (Tacconelli, 2010). To identify studies which met inclusion criteria, electronic databases were searched from inception using 'textword' and 'subject headings' (where possible) relating to the core concepts covered by the review question (i.e., childhood, adolescence, antisocial cognition, antisocial behaviour). Databases searched included MEDLINE (1946 to present), PsycINFO (1806 to present) and Web of Science. Search terms were initially developed for MEDLINE and modified where necessary to meet the requirements of the other databases listed above. The search terms for MEDLINE can be viewed in Appendix A. The last search was run on 18th August 2016. Language restrictions were not applied and automatic updates were enabled where possible to help ensure no future papers were missed. Other sources searched included the Internet, the international prospective register of systematic reviews, personal collections of journal articles, and the reference lists of included studies or reviews germane to the construct of antisocial cognition.

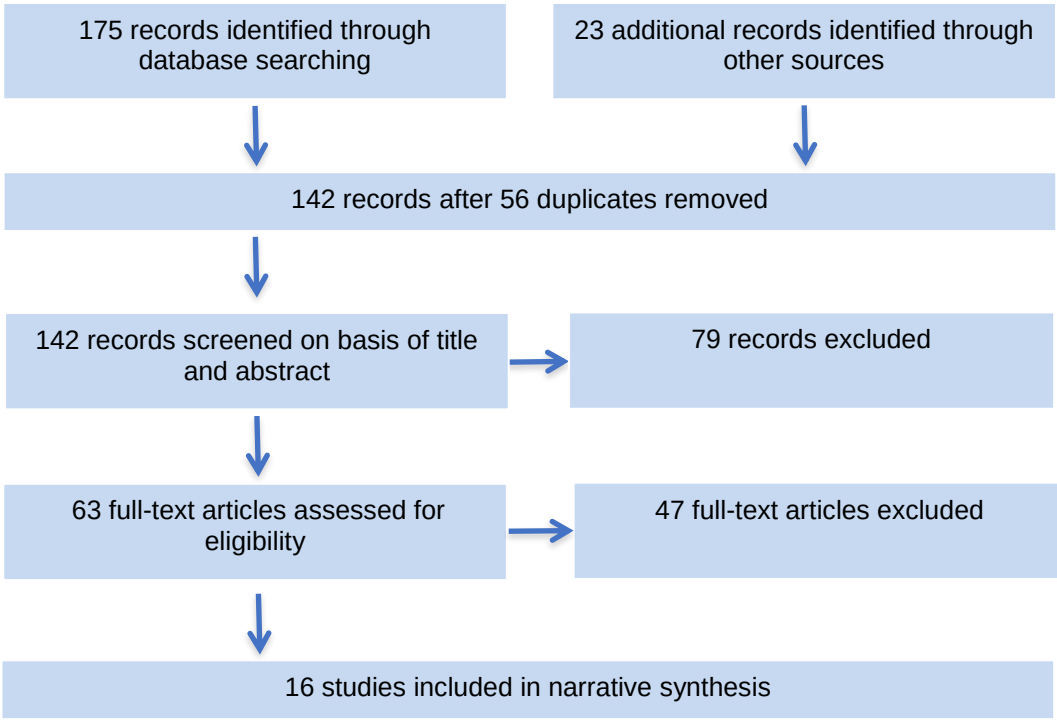
Process for Study Selection

Study selection was based on inclusion criteria and conducted in stages, as depicted by the flow-diagram in Figure 1. Citations identified from electronic database searches and other sources were imported into Endnote X5 and duplicates removed. Each reference was then screened against inclusion criteria by reading the title and abstract. Potentially eligible references were retrieved in full for further evaluation. To keep the process replicable and transparent the unsorted search results were saved and retained for future potential re-analysis. Due to time and resource

constraints, eligibility assessment was performed by a single reviewer.

Figure 1

Flow-diagram of Systematic Process for Study Selection



Quality Assessment of Included Studies

Quality assessment is fundamental to systematic reviews. Flaws in the design or conduct of studies can increase risk of systematic error (or bias) and reduce the validity of study findings. Observational studies are more susceptible to bias than experimental studies; however, many of these biases can be reduced with careful study planning. Proper interpretation of evidence also depends upon the availability and quality of descriptive information. The quality assessment of studies included in this review was guided by the Critical Appraisal Skills Programme (CASP) Cohort Study Checklist. This checklist consists of 12 questions designed to help the reader think critically about key issues related to the design and conduct of

prospective observational research. Checklists providing a summary score for the overall quality of a study have been criticised in the literature and therefore were not used (Petticrew & Gilbody, 2004; Tacconelli, 2010). Additionally, papers from Cole & Maxwell (2003), Walters (2017a) and Mackinnon and Fairchild (2009) were used to guide the quality assessment of mediation studies.

Results

Study Selection

The search of electronic databases and other sources produced a total of 198 citations. After adjusting for duplicates 142 remained. Of these, 79 studies were discarded because after reviewing the titles and abstracts it became clear that these papers did not meet the inclusion criteria. Two of these papers were discarded because they could not be feasibly translated into English. The full text of the remaining 63 citations was examined in more detail. It appeared that 47 papers did not meet the inclusion criteria as described. Specifically, 18 studies were excluded because they did not comprise a research design or address a clearly focused issue relevant to the research question, 10 because they did not include an appropriate measure of antisocial cognition or antisocial behaviour, eight because they did not examine the correct population, four because of a lack of information or clarity, four because they were unavailable for review, two because they were reviews, and one because it focused solely on substance use as an antisocial outcome. In total, sixteen studies met the inclusion criteria and were included in the systematic review.

Characteristics of Included Studies

The characteristics of included studies are summarised in Table 2. All studies comprised a prospective observational rather than experimental design. The age range of participants at study entry was 6-19 years and the smallest and largest sample size analysed comprised 324 and 1725 participants, respectively; five studies did not report the size of the sample analysed. Every study involved a secondary analysis of existing data. The studies took place between 1985 and 2016. Fourteen studies were based on data from the USA (Agnew, 1985; Halgunseth, Perkins, Lippold, & Nix, 2013; Menard & Huizinga, 1994; Rebellon, Manasse, Gundy, & Cohn, 2014; Reed & Rose, 1998; Simons, Simons, Chen, Brody, & Lin, 2007; Thornberry, Lizotte, Krohn, Farnworth, & Jang, 1994; Walters, 2015a, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013; Zhang et al., 1997), whereas the two other studies were based on data from the UK (Walters, 2015b) and Germany (Seddig, 2014).

Seven studies examined the reciprocal relationship between antisocial cognition and antisocial behaviour in late childhood and adolescence (Agnew, 1985; Menard & Huizinga, 1994; Rebellon et al., 2014; Reed & Rose, 1998; Seddig, 2014; Thornberry et al., 1994; Zhang et al., 1997) and nine studies undertook mediation analyses (Halgunseth et al., 2013; Simons et al., 2007; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). Three mediation studies also conducted moderated mediation in relation to mediated effects (Walters, 2015a, 2015b, 2016b).

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Agnew (1985) USA	Youth in Transition Survey (W1-2; 1.5yr lag), W1 *N* = 2213 male public high school students aged 15-16 at study entry.	Attitudes toward honesty and cheating: 7 self-report items (α = 0.87).	Minor, serious and status offences in past 1.5 years: (1) Total Delinquency scale (26 self-report items) and (2) Seriousness of Delinquency scale (10 self-report items).
Halgunseth et al (2013) USA	PROSPER (W1-3; control condition of second cohort; 1yr lags). *N* = 324 (50% female) youths aged 11-12 at study entry.	Delinquent-oriented attitudes: 14 self-report items (α = .89 & .91, W1 & W2).	Minor, serious and status offences in past 12 years: 12 self-report items (α = .85 & .89, W1 & W3).

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Menard & Huizinga (1994) USA	National Youth Survey (W1-5; 1yr lags). W1 *N* = 1725 (47% female) youths aged 11-17 at study entry.	Strong, moderate and weak attitudes towards illegal behaviour: 7 self-report items.	Minor and serious offences in past year: General Delinquency scale (14 self-report items).
Rebellon et al (2014) USA	New Hampshire Youth Study (W2-3; .5yr lag). *N* = 626 (59% female) youths aged 11-17 at study entry.	Attitudes towards theft and violence: 6 self-items.	Minor and serious offences in past six months: 6 self-report items.
Reed & Rose (1998) USA	National Youth Survey (W1-3; 1yr lags). W1 *N* = 1725 (47% female) youths aged 11-17 at study entry.	Attitudes towards serious theft: 2 self-report items (α = 0.86-0.87 W1-3).	Serious theft in past year: 2 self-report items (α = 0.86-0.87 across waves).

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Seddig (2014) Germany	Crime in the Modern City study (W2-5; 1yr lags). *N* = 1552 (50.7% female) students aged 14 at study entry.	Acceptance of pro-violent norms: 3 self-report items.	Serious violent offences in past year: 3 self-report items.
Simons et al (2007) USA	Family and Community Health Study (W1-2; 2yr lag). W1 *N* = 867 (54% female) African-American youths aged 10-12 at study entry.	Acceptance of deviance: 4 self-report items (α = .68-.77 W1-2).	Minor, serious and status offences in past year: conduct disorder section of DISC-IV (26 self-report items) (α = >.90 across waves).

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Thornberry et al (1994) USA	Rochester Youth Development Study (W2-4; .5yr lags). *N* = 841 (26% female) youths at high-risk of serious delinquency aged 13-14 at study entry.	Delinquent beliefs: 8 self-report items (internal consistency = 0.78-0.86 across waves).	Minor, serious and status offences in past 6 months: 28 self-report items.
Walters (2015a) USA	National Youth Survey (W1-3 & 5; 1 & 2yr lags). *N* = 1725 (47% female) youths aged 11-17 at study entry.	Attitudes toward deviance: 9 self-report items (α = .84-.85 W2-3).	Minor, serious and status offences in past year: 12 self-report items (α = .78 W5).
Walters (2015b) UK	Offending, Crime & Justice Survey (W1-4; 1yr lags). *N* = 1027 (55% female) youths without history of delinquency aged 10-18 at study entry.	PCT: 4 self-items (α = .75-.77 W1-4).	Serious offences in past year: 6 self-report items.

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Walters (2016a) USA	Pathways to Desistance Study (W1-3; .5yr lags). *N* = 1170 male youths convicted of a serious offense aged 14-18 at study entry.	General criminal thinking (i.e., PCT & RCT): (1) Moral Disengagement scale (32 self-report items; α = .90-.91 W0-1), (2) impulse control scale of Weinberger Adjustment Inventory (8 self-report items; α = .76).	Minor and serious offences in past 6 months: Self-reported Offending scale (22 self-report items).
Walters (2016b) USA	National Youth Survey (W1-4; 1yr lags). *N* = 1725 (47% female) youths aged 11-17 at study entry.	(1) Attitudes toward deviance: 9 self-report items (α = .84-.85 W2-3); (2) PCT:10 self-report items (α = .82-.85 W2-3).	Minor and serious offences in past year: 17 self-report items.

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Walters (2016c) USA	Pathways to Desistance Study (W0-2; .5yr lags). *N* = 1170 male youths convicted of a serious offense aged 14-19 at study entry.	(1) PCT: Moral Disengagement scale (32 self-report items; α = .90-.91 W0-1); (2) RCT: impulse control scale of Weinberger Adjustment Inventory (8 self-report items; α = .76).	Minor and serious offences in past 6 months: Self-reported Offending scale (22 self-report items).
Walters (2017b) USA	National Youth Survey (W1-5; 1yr lags). *N* = 1725 (47% female) youths aged 11-17 at study entry.	PCT:10 self-report items (α = .82-.85 W2-4).	Minor and serious offences in past 6 months: Self-Reported Offending scale (17 self-report items).

Table 2

Characteristics of Included Studies

Study Country	Design	AST Measure	ASB Outcome
Walters & DeLisi (2013) USA	National Longitudinal Survey of Adolescent Health (W1-4; variable lags). *N* = 812 (38% female) youth aged 11-19 at study entry.	Antisocial cognition: 9 self-report items (α = 0.88 W3).	Minor, serious and status offences in past year: 14 (W3) and 12 (W4) self-report items.
Zhang et al (1997) USA	Pittsburgh Youth Study (W1-8; .5yr & 1yr lags). W1 *N* = 1517 public schoolboys aged 7, 10 and 13 at study entry (3 cohorts).	Antisocial attitudes: Perception of Problem Behaviours scale (20 self-report items; internal consistency = 0.77-0.9 across waves), Attitude Toward Delinquency scale (15 self-report items; internal consistency = 0.73-0.89 across waves).	Minor, serious and status offences in past six months: Delinquency scale (36 self-report items), Self-report of Antisocial Behaviour scale (32 self-report items).

Note. PCT = proactive criminal thinking; PROSPER = Promoting School-community-university Partnership to Enhance Resilience; RCT = reactive criminal thinking; *N* = total sample analysed unless otherwise stated; W = assessment wave; α = Cronbach's alpha; gender split shown where available.

Summary of Main Findings: Reciprocal Relationship Studies

Two studies investigated the predictive and contemporaneous relationship between antisocial cognition and antisocial behaviour across just two points in time. Analysing data from the Youth in Transition Survey, Agnew (1985) found that beliefs favourable toward dishonesty and cheating at 15-16 years of age predicted antisocial behaviour over the next one and half years and that antisocial behaviour was contemporaneously associated with such beliefs at 16-17 years of age. Similarly, Rebellon *et al* (2014) reported a predictive and contemporaneous relationship between attitudes tolerant of theft and theft over a six-month period, in a group of participants from the New Hampshire Youth Survey who were aged 11-17 years at study entry.

Thornberry and colleagues (1994) used the first four waves of data from the Rochester Youth Development study to examine the mutual predictability of 'delinquent beliefs' and 'delinquent behaviour' in a group of 13-14-year-old youths deemed at high-risk of delinquency. The authors reported a reciprocal relationship between delinquent beliefs and delinquent behaviour over the first six months of the study, although the effect of belief on behaviour was larger. Delinquent behaviour ceased to have an impact on delinquent beliefs over the next six months, however the influence of antisocial beliefs on delinquent behaviour continued.

Zhang and colleagues (1997) noted dramatic changes in the reciprocal nature of the relationship between attitudes tolerant toward theft and violence and acts of theft and violence over a four-year period, in three groups of boys from the from the Pittsburgh Youth study who were seven, 10

and 13 years old at study entry. Specifically, the authors reported linear increases in the mean scores of both attitudes tolerant of theft and violence and acts of theft and violence across groups up to the age of 17 years, including significant increases between the ages of 11 and 14 years. When the linkages between attitudes and behaviour were examined, the authors found stronger effects of attitudes on subsequent behaviour relative to behaviour on subsequent attitudes, among boys aged 10-12 years. For boys aged 13-16 years, however, the relationship between attitudes and behaviour became more mutually predictive.

Seddig (2014) used data from the German Crime in the Modern City study to test the structure of relations between delinquent peer association, the acceptance of pro-violent norms, and violent delinquency. Pro-violent norms at ages 14, 15 and 16 years predicted violent delinquency at ages 15, 16 and 17 years, respectively. Violent behaviour also predicted pro-violent norm orientation from 14-15 years of age, but failed to do so from 15-16 or 16-17 years of age. The effect of antisocial cognition on violent behaviour was most prominent from 15-16 years of age.

Two studies reported the absence of a reciprocal relationship between antisocial cognition and antisocial behaviour in correlation-based path analyses of data from the National Youth Survey. Using the first five waves of data, Menard & Huizinga (1994) failed to find a significant predictive relationship between attitudes permissive of illegal behaviour and illegal behaviour over a period of four years. Analysing the same first three waves of data, Reed and Rose (1998) also reported the absence of a predictive

relationship between attitudes tolerant toward serious theft and serious theft once the historical effect of serious theft was controlled. Nonetheless, a complementary stage-state analysis by Menard & Huizinga (1994), in which the authors attempted to ascertain the temporal order of changes in attitudes permissive of illegal behaviour and illegal behaviour, revealed a pattern wherein changes in attitude consistently preceded changes in behaviour. Nonetheless, once a change in attitude had occurred, the influence of illegal behaviour on attitude became more pronounced than that of attitude on illegal behaviour. These effects were robust across age and sex.

In summary, six of the seven studies in this section found evidence supporting the conception of a reciprocal relationship between aspects of antisocial cognition and antisocial behaviour. Furthermore, three of these studies investigating the predictive relationship between antisocial cognition and antisocial behaviour and visa-versa suggest that the two constructs might influence each other in a dynamic rather than fixed way, with antisocial cognition having a stronger influence on antisocial behaviour during early-to-mid adolescence.

Summary of Main Findings: Mediation Studies

Two studies examined antisocial cognition as a mediator of the relationship between prior delinquency and future criminality. Using data from all four waves of the National Longitudinal Study of Adolescent Health, Walters and DeLisi (2013) ascertained that antisocial cognition (assessed using nine self-report items measuring thrill seeking, callous, deceptive, and rule breaking attitudes) at 18-26 years of age partially mediated the

relationship between self-reported delinquency at 13-19 years of age and future involvement in crime at 24-32 years of age. Walters (2016a) used data from the Pathways to Desistance study and created a composite score for *general criminal thinking* by summing standardised total scores from the moral disengagement scale (Bandura, Barbaranelli, Caprara, & Pastorelli, 1996) and Weinberger Adjustment Inventory (Weinberger & Schwartz, 1990) Impulse Control scale. General criminal thinking was found to partially mediate the relationship between self-reported antisocial behaviour and self-reported future offending in a group of male adolescent offenders over a one-year period, who were aged 14-18 years at study entry.

Antisocial cognition was investigated as a potential mediator of the relationship between delinquent peer association and self-reported antisocial behaviour (i.e., the peer influence effect) in a series of papers by Walters (2015b, 2016b, 2017b). The first of these papers examined whether *proactive criminal thinking* mediated the relationship between peer delinquency and serious offending in a group of British youths from the Offending, Crime and Justice Survey, who were aged 10-18 years at study entry and did not have a history of offending (Walters, 2015b). Proactive criminal thinking was assessed using items asking about the acceptability of stealing in given situations, which Walters claimed primarily measured techniques of neutralisation (Sykes & Matza, 1957) that allow the individual to overcome the incongruence between internalised norms and beliefs and delinquent behaviour. The results showed that proactive criminal thinking partially mediated the relationship between peer delinquency and serious offending. Moreover, this pathway demonstrated a significantly larger effect than one

where proactive criminal thinking preceded peer delinquency in the prediction of serious offending. There was no evidence that the mediated effect was moderated by sex in a moderated mediation analysis.

More recently, Walters (2016b, 2017b) examined whether antisocial cognition mediated the peer influence effect in two papers which used data from the National Youth Survey, in which participants were aged 11-17 years at study entry. In the first paper, Walters (2016b) ascertained that *criminal thought content* (i.e., attitudes towards deviance) and *criminal thought process* (i.e., proactive criminal thinking) both separately and conjointly mediated the peer influence effect in a parallel multiple mediation model. Attitudes toward deviance were assessed with nine items which asked the respondent how wrong it is for someone their age to engage in various delinquent acts, while proactive criminal thinking was measured using 10 items purported to measure neutralisation (Sykes & Matza, 1957). Again, there was no evidence that the mediated effect was moderated by sex. In a second paper, Walters (2017b) discovered that proactive criminal thinking partially mediated the peer influence effect as a first-order mediator, in a two-stage serial multiple mediation model, where *deviant identity* was a second-order mediator. Proactive criminal thinking was measured with the same 10 items used by Walters (2016b), while deviant identity was assessed using the Labelling by Parents and Labelling by Friends scales from the NYS which are completed by the child and measure reflected appraisals from the child's parents and friends, respectively. Walters (2017b) also found that a pathway where deviant identity preceded proactive criminal thinking was not predictive of delinquency.

In another study of *criminal thinking* that used data from the first three waves of the Pathways to Desistance study, Walters (2016c) demonstrated that proactive criminal thinking, measured using the moral disengagement scale (Bandura et al., 1996), and *reactive criminal thinking*, measured using Weinberger Adjustment Inventory (Weinberger & Schwartz, 1990) Impulse Control scale, partially mediated the peer influence effect and peer selection effect (i.e., relationship between offending and delinquent peer association), respectively. Walters also noted that the effect of the peer influence pathway was larger than reverse or alternate pathways whereas the peer selection pathway was not.

The remaining mediation studies investigated antisocial cognition in relation to antisocial behaviour and parent related variables. Using the first four waves of the National Youth Survey, Walters (2015a) found that *youth attitude toward deviance* mediated the relationship between *youth perception of parental attitude toward deviance* and self-reported delinquency as a second-order mediator in a two-stage serial multiple mediation model where youth perception of parental attitude toward deviance was a first-order mediator and *parental attitude toward deviance* was the independent variable. The independent and mediator variables were assessed using the same nine items asking parents or youth to indicate how much they approve or disapprove of certain antisocial acts. Additionally, Walters (2015a) demonstrated that an alternate pathway, where youth attitude toward deviance preceded youth perception of parental attitude toward deviance, was not predictive of self-reported delinquency and that the mediating effect of youth attitude toward deviance on the relationship between youth

perception of parental attitude toward deviance and self-reported delinquency was not moderated by age, race or sex.

In an earlier study, Simons and colleagues (2007) found that attitudes accepting of deviance partially mediated the relationship between parental behaviour and *conduct problems*, in a group of African-Americans from the Family and Community Health study who were aged 10-12 years at study entry. Lastly, Halgunseth and colleagues (2013) found that *delinquent-oriented attitudes* partially mediated the relationship between *parental inconsistent discipline* and self-reported *deviant behaviour*, in a group of control participants from the first three waves of data from the Promoting School-Community-University Partnerships to Enhance Resilience study, who were aged 11-12 years at study entry.

In summary, results from all nine of the studies included in this section provide evidence supporting the conception that aspects of antisocial cognition constitute a causal mechanism in adolescence that explains part of the relationship between antisocial behaviour as an outcome and history of antisocial behaviour, delinquent peer association and parent related factors, respectively. The mediating influence of antisocial cognition on these relationships may also be unaffected the age, sex or race of the individual. Furthermore, findings from studies investigating aspects of antisocial cognition in relation to other mediators suggest that the construct could form part of a causal chain of mediators that explained the peer influence effect and relationship between certain parent related variables and antisocial behaviour.

Quality Assessment of Included Studies

The studies summarised provide insight into the causal nature of the relationship between antisocial cognition and antisocial behaviour, however many had methodological shortcomings. To clarify the strength of the evidence presented, this section assesses the methodological rigour of studies according to four key aspects of research validity: internal validity, construct validity, statistical conclusion validity and external validity.

Internal validity.

Internal validity is the degree to which the results of a study can be used to make causal inferences (Warner, 2013). The internal validity of a study depends on its ability to minimise random and systematic error (i.e., bias) and satisfy the three primary criteria for causality: (1) association (i.e., the cause and effect must covary), (2) temporal precedence (i.e., the cause must precede the effect in time), and (3) non-spuriousness (i.e., the association between the cause and effect must not be produced by the association of both variables with a third variable or set of variables). Common threats to the internal validity of the studies included in this review are now highlighted.

Temporal precedence.

To achieve temporal precedence, variables must be sequentially measured and examined with no temporal overlap. Four of the seven studies investigating the reciprocal relationship between antisocial cognition and antisocial behaviour achieved temporal precedence (Menard & Huizinga, 1994; Seddig, 2014; Thornberry et al., 1993; Zhang et al., 1997). Two other

studies measured both variables over two consecutive times points, but only examined the relationship between antisocial cognition and antisocial behaviour prospectively (Rebellon et al., 2014; Reed & Rose, 1998). The remaining study employed a *half-longitudinal* research design, wherein only antisocial behaviour was measured across two successive time points (Agnew, 1985). All but one of the mediation studies also achieved temporal precedence (Halgunseth et al., 2013; Walters 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). The remaining study measured the mediator and dependent variables at a single point in time and examined the relationship between them contemporaneously (Simons et al., 2007).

***Confounding*.**

Confounding occurs when an extraneous variable (i.e., an unobserved variable that is external to the study) correlates with both the dependent variable and independent (or mediator) variable, such that it explains all or part of the relationship between the two variables. Failure to control for confounding can create a spurious association. There are many ways to reduce the possibility of confounding in observation research (for review see Hajian Tilaki, 2012). The studies included in this review all made some attempt to control for confounding by including various risk factors for antisocial behaviour as covariates in statistical models (Agnew, 1985; Halgunseth et al, 2013; Menard & Huizinga, 1994; Seddig, 2014; Rebellon et al., 2014; Reed & Rose, 1998; Simons et al., 2007; Thornberry et al., 1993; Zhang et al., 1997; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). As recommended by Cole and Maxwell (2003), eight of nine mediation studies also controlled for the potential confounding

effect of prior levels of mediator and dependent variables by including precursor measures of these variables in regression equations (Halgunseth et al., 2013; Simons et al., 2007; Walters, 2015a, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013) or excluding participants with a history of antisocial behaviour (Walters, 2015b). Additionally, seven studies performed sensitivity testing to assess the sensitivity of study findings to the effects of extraneous variables (Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). Three of these studies found that the mediating effects observed were moderately robust to the confounding effects of extraneous variables (Walters, 2015a, 2015b, 2016b, 2017b), while four others found that the mediating effects observed were low-to-moderately robust to the confounding effects of extraneous variables (Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013).

***Time lag between variables*.**

The time lag between study phases should correspond with the time needed for the casual variable to influence the outcome variable. A time lag that is too short can result in missed effects, whereas one that is too long can increase risk for confounding (Cole and Maxwell, 2013). Thereby, the time lag between study phases should be scientifically justified. A six-month or one-year time lag is considered optimal for observing antisocial behaviour (Rennison & Rand, 2007; Walters, 2017a). Thirteen of the studies included in this review were consistent with this recommendation (Halgunseth et al., 2013; Menard & Huizinga, 1994; Rebellon et al., 2014; Reed & Rose; 1998; Seddig, 2014; Thornberry et al., 1994; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Zhang et al., 1997). Agnew (1985) and Simons *et al* (2007)

had a one and a half year and two-year time lag, respectively. Finally, Walters and DeLisi (2013) had a time lag of five to six years between variables. None of the studies provided a rational for the time lag used.

***Attrition and missing data*.**

Attrition occurs when the researcher loses contact with a participant, resulting in missing data. Missing data can also result from participant nonresponse and is conceptually described in terms of three mechanisms: missing completely at random (MCAR), missing at random (MAR), and missing not at random (MNAR) (Rubin, 1976). In MNAR missingness is systematically related to the variable itself, whereas in MAR and MCAR missingness is systematically related to other observed variables or completely random, respectively. Common methods for handling missing data include listwise deletion, pairwise deletion and full information maximum likelihood (FIML) estimation. Simulation studies suggest that the potential for missing data to bias estimates of effect depends on the level and mechanism of missing data. Research suggests that when data are MNAR missingness is unlikely to bias study findings until greater than 20% of the sample, however when data are MAR or MCAR missingness is unlikely to bias study findings until greater than 60% of the sample (Kristman, Manno, & Côté, 2004). Furthermore, no missing data method will eliminate bias when data are MNAR, with listwise and pairwise deletion the worst effected (Kristman, Manno, & Côté, 2005). Understanding the level and mechanism of missing data is therefore important to determine the potential for bias from attrition and most appropriate method to handle missing data.

Seven studies included in this review reported partial or complete information about the level of attrition across study phases, which ranged from four to 17.8% (Agnew, 1985; Halgunseth et al., 2013; Menard & Huizinga, 1994; Reed & Rose; 1998; Simons et al., 2007; Thornberry et al., 1994; Zhang et al., 1997). Five studies instead provided information about the level of missing data across variables, which ranged from 57.1% to 86.3% (Walters, 2015a, 2015b, 2016b, 2016c, 2017b). Three other studies did not provide information about the level of attrition or missing data (Rebellon et al., 2014; Seddig, 2014; Walters & DeLisi, 2013). Eight studies used listwise or pairwise deletion to handle missing data (Agnew, 1985; Halgunseth et al., 2013; Menard & Huizinga, 1994; Rebellon et al., 2014; Seddig, 2014; Simons et al., 2007; Zhang et al., 1997; Walters & DeLisi, 2013) and eight used FIML estimation (Reed & Rose, 1998; Thornberry et al., Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b). Only six studies, however, tried to clarify the attrition related mechanism of missing data (Agnew, 1985; Halgunseth et al., 2013; Menard & Huizinga, 1994; Reed & Rose; 1998; Simons et al., 2007; Thornberry et al., 1994). These studies compared the baseline characteristics of participants who dropped out with those who continued, to determine if drop-out was related to other variables in the dataset. All concluded that missingness did not bias study findings.

Statistical conclusion validity.

Statistical conclusion validity (SCV) is the degree to which research data can reasonably be regarded as revealing an effect between independent and dependent variables, as far as statistical issues are concerned (Cook & Campbell, 1979). It is primarily concerned with the

avoidance of two types of conclusion error: Type-I error (i.e., the reporting of a false-positive result) and Type-II error (i.e., the reporting of a false-negative result).

The violation of assumptions of statistical tests increases the probability of making either error. Eleven studies included in this review tested and adjusted for certain violated assumptions of statistical tests, including univariate non-normality, model identification and high multicollinearity (Halgunseth et al., 2013; Reed & Rose, 1998; Seddig, 2014; Thornberry et al., 1994; Zhang et al., 1997; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). Even so, none of the included studies clarified the extent to which all major assumptions of statistical tests were met.

Low power increases the probability of making a Type-II error. Power is inversely related to sample size given other factors. None of the included studies commented on the power of statistical tests and the number of participants included in analyses was not always clear. Nonetheless, most studies were based on an initial sample of over 600 participants (Agnew, 1985; Menard & Huizinga, 1994; Rebellon et al., 2014; Reed & Rose, 1998; Seddig, 2014; Simons et al., 2007; Thornberry et al., 1994; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013; Zhang et al., 1997). The unreliable measurement of variables (i.e., with substantial amounts of measurement error) and random heterogeneity of participants can also reduce the power and precision of a study (Cole & Maxwell, 2003). Although many studies used measures with good internal consistency, the

test-retest reliability of measures in most studies was not clear.

Several studies included in this review were based on samples that were diverse in terms of age at study entry. Indeed, six studies were based on participants aged 11-17 years at study entry (Rebellon et al., 2014; Reed & Rose., 1998; Menard & Huizinga, 1994; Walters, 2015b, 2016b, 2017b). while three others were based on participants who were aged 10-18 years (Walters, 2015b) and 14-19 years at study entry (Walters, 2016a, 2016c). This is important as sample heterogeneity can reduce statistical conclusion validity by increasing the variance of findings or obscuring true relationships.

Fundamental to mediation analysis is the quantification of the indirect effect using an inferential test. Different interferential tests of the indirect effect, however, have differing degrees of power (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). One mediation study tested the statistical significance of the indirect effect using a method known to lack power (Halgunseth et al., 2013), while the remaining mediation studies used some form of bootstrapping procedure, which is currently considered one of the optimal methods for estimating the size and statistical significance of the indirect effect (Mackinnon et al., 2013; Rucker, Preacher, Tormala, & Petty, 2011).

Shared method variance can inflate estimates of effect and, thus, the probability of making a Type-I error (Cole & Maxwell, 2003). Shared method variance was a major concern across studies, since every paper measured antisocial cognition and antisocial behaviour via self-report. A handful of authors attempted to control for the effect of shared method variance by

allowing error terms to correlate across constructs in statistical models (Agnew, 1985; Reed & Rose, 1998; Simons et al., 2007; Thornberry et al., 1994; Zhang et al., 1997).

Construct validity.

Construct validity is the degree to which inferences can legitimately be made from the operationalisations within a study to the theoretical constructs on which they are based. Significantly, none of the studies reviewed employed a standardised measure of antisocial cognition with demonstrated construct validity or provided an explicit and detailed operational definition of the form of antisocial cognition under study. Furthermore, the terminology used to refer to different forms of antisocial cognition varied markedly across studies, however most studies measured these constructs using items that asked participants to indicate how much they approve or disapprove of different antisocial acts. Two other studies measured aspects of criminal thinking using a standardised measure of impulse control and items claimed by the author to measure Sykes and Matza's (1957) concept of neutralisation (Walters, 2016a, 2016c), while a final study assessed antisocial thinking using nine items related to thrill seeking, callous, deceptive and rule breaking attitudes (Walters & DeLisi, 2013).

Regarding antisocial behaviour, most studies asked participants about their involvement in various antisocial acts during a set period, to create a general score of delinquency (Agnew, 1985; Halgunseth et al., 2013; Menard & Huizinga, 1994; Thornberry et al., 1994; Walters, 2015a, 2015b, 2016a, 2016b, 2016c, 2017b; Walters & DeLisi, 2013). Four other studies examined

aspects of antisocial cognition in relation to a specific form of antisocial behaviour (Rebellon et al., 2014; Reed & Rose, 1998; Seddig, 2014; Zhang et al., 1997), while Simons *et al* (2007) assessed antisocial behaviour using a standardised measure of conduct problems in children.

External validity.

External validity is the ability to generalise study results to a more universal population (Warner, 2013). The best way to demonstrate external validity is to replicate results in different populations, places, and time periods. Sampling bias undermines external validity and occurs when a sample is collected in such a way that some members of the intended population are less likely to be included than others, resulting in an unrepresentative sample. The studies included in this review were based on samples from major population surveys or relatively large multi-centre studies. Nine papers employed a probability sampling procedure, whereby participants were gathered in a process that gives all individuals in the population of interest an equal chance of being selected (Agnew, 1985; Menard & Huizenga, 1994; Reed & Rose, 1998; Simons et al., 2007; Thornberry et al., 1994; Walters, 2015a, 2015b, 2016b, 2017b). The other studies used a non-probability sampling procedure (Halgunseth et al., 2013; Rebellon et al., 2014; Walters, 2016a, 2016c; Zhang et al., 1997) or did not provide information about sampling (Seddig, 2014; Walters & DeLisi, 2013). Only two studies were based on participants with a history of serious offending (Walters, 2016a, 2016c), whilst two others focused on juveniles at high-risk of serious delinquency (Thornberry et al., 1994; Zhang et al., 1997). Five studies used data from the National Youth Survey based on seven birth

cohorts from the 1950's and 1960's (Menard & Huizenga, 1994; Reed & Rose, 1998; Walters, 2015a, 2016b, 2017b).

Discussion

This review sought to provide an overview and critical appraisal of research examining the causal nature of the relationship between antisocial cognition and antisocial behaviour in late childhood and adolescence. The purpose was to enlighten about study findings, clarify the strength of evidence and identify areas for future research. Sixteen studies were identified in a systematic search of the literature and subsequently reviewed. Findings from these studies point to three highly tentative conclusions regarding the causal nature of the relationship between antisocial cognition and antisocial behaviour in adolescence: (1) antisocial cognition is reciprocally related to antisocial behaviour during adolescence, such that both variables act as cause and effect with respect to each other overtime; (2) the reciprocal relationship between antisocial cognition and antisocial behaviour is dynamic, with antisocial cognition having a stronger effect on antisocial behaviour during early-to-mid adolescence than visa-versa; and (3) aspects of antisocial cognition constitute causal mechanisms that explain part of the relationship between other risk factors for antisocial behaviour and antisocial behaviour, including history of antisocial behaviour, delinquent peer association, and certain parent related factors. Due to a lack of research focusing on late childhood and early adolescence, the data did not allow any tentative conclusions to be drawn about the causal nature of the antisocial cognition-antisocial behaviour relationship prior to adolescence.

These findings are concordant with several eminent theories of crime, including differential association theory (Ackers, 1998; Sutherland, 1947), psychological inertia theorem (Walters, 2012), interactional theory (Thornberry et al., 1994) and social bond theory (Hirschi, 1969). However, they are discordant with the General Theory of Crime (Gottfredson & Hirschi, 1990; Gottfredson, 2011), which suggests that aspects of antisocial cognition, like moral beliefs and values, are not important in explaining antisocial behaviour.

The abovementioned conclusions are tentative because the studies reviewed have many methodological shortcomings. That is, it could be contended that only two studies were completed with a reasonable degree of methodological rigour (Walters, 2015a, 2015b). These studies achieved temporal precedence, had an appropriate time lag for antisocial behaviour, controlled for the potentially confounding effects of other major risks factors for antisocial behaviour, conducted sensitivity testing, used data from participants recruited using a probability sampling procedure, employed optimal statistical methods, and had a large sample size with less than 20% missing data on variables. Thereby, these studies satisfied the conditions of association and temporal precedence required for causal inference and compared to other studies included in this review, were less susceptible to random and systematic biases. Two other studies were conducted in an equivalent way, but had a significant amount of missing data and failed to include information concerning the mechanism of missing data (Walters, 2016b, 2017b).

It was noteworthy that every study included in this review employed an observational rather than experimental research design. This is significant because observational research designs are unable to rule out alternative explanations for observed relationships, which is necessary to satisfy the non-spuriousness condition of causality. The lack of experimental research could relate to the ethical and practical problems associated with manipulating psychosocial variables common to the field of criminology, like antisocial beliefs and attitudes.

The strength of evidence from the research reviewed is also undermined by the widespread lack of standardised assessment and reliance on measures with unknown reliability and validity. Consequently, it is difficult to understand how effective these measures were in minimising random and systematic biases inherent to the measurement of constructs or how accurately the concepts assessed reflected the theoretical constructs on which they are based. Furthermore, the general lack of standardised assessment, absence of explicit and detailed operational definitions for constructs, and inconsistent terminology for aspects of antisocial cognition makes it is incredibly difficult to compared findings across studies.

As an aside, it is possible that the lack of standardised assessment and uniform terminology for antisocial cognition is related to the extensive use of historical data, which was collected at a time when standardised measures of antisocial cognition were not available and the construct was poorly defined. Inspecting the content of antisocial cognition measures revealed that most studies tried to capture participants' tolerance for law

violations or use of neutralisation techniques (Sykes & Matza, 1957).

Many studies measured antisocial behaviour by asking participants about their involvement in a wide range of antisocial acts. They then examined aspects of antisocial cognition in relation to a general score of delinquency. The antisocial acts enquired about usually varied markedly in terms of severity; for example, from truancy to serious assault or vandalism to firearm offences. The widespread use of global measures of antisocial behaviour is problematic because it obscures the interpretability of discrete findings and could mask important aetiological differences associated with the development of distinct types of antisocial behaviour (Burt, 2009). In other words, it reduces the overall explanatory power of the findings. The level of correspondence between the content of belief and attitude measures and measures of behaviour is also significant because it can influence the precision of estimated effects (Frymier & Nadler, 2016).

Similarly, the use of samples with a wide age range at study entry could have obscured important findings regarding the developmental relationship between antisocial cognition and antisocial behaviour through adolescence. Indeed, those studies with a narrow age range at study entry found evidence to suggest that antisocial cognition might have a greater influence on antisocial behaviour at distinct times through adolescence. Thereby, it is entirely possible that the strength of antisocial cognition as a risk factors or mediator of antisocial behaviour could differ significantly with respect to other population factors, such as sex, history of antisocial behaviour or neighbourhood. Understanding the dynamics of the relationship

between antisocial cognition and antisocial behaviour in distinct groups will be important to understand when interventions for antisocial cognition are more likely to be effective.

The use of data from major population surveys and large scale multi-centre studies helped to ensure the external validity of findings, as it meant that participants were from multiple geographical locations and settings. This was especially the case for studies that employed some form of probability sampling procedure. At the same time, most of the datasets used were derived from individuals living in the USA who were born between 1959 and 1990. Furthermore, it is notable that only two studies examined a group of participants with a history of serious antisocial behaviour. Thereby, it is unclear how well many of the findings in this review generalise to current populations outside of the USA or with a history of serious antisocial behaviour.

Finally, the strength of the findings from the studies reviewed is weakened by a lack of information about whether the assumptions of statistical tests were satisfied. Whilst most studies commented on the normality of univariate data, there are many other assumptions on which inferential statistics are founded that if violated could result in the underestimation or overestimation of effects.

Given the methodological shortcomings highlighted, it could be argued that the literature regarding the causal nature of the relationship between antisocial cognition and antisocial behaviour is still in its infancy. To advance understanding in this area, future researchers should consider the limitations

of the studies in this review. To this end, future researchers might look to achieve the following: (1) create a universal term and operational definition for the constructs of antisocial behaviour and antisocial cognition; (2) use standardised measures of antisocial cognition with demonstrated reliability and validity in children and adolescents; (3) reduce measurement error and common-method variance by constructing latent variables for antisocial behaviour using multiple sources of data most appropriate for the developmental period under question (e.g., parent and teacher reports for childhood and self-reports and official records for adolescence); (4) use specific rather than global measures of antisocial cognition and antisocial behaviour and pay greater attention to the relationship between specific forms of antisocial cognition and antisocial behaviour to unmask potential differences in aetiology; (5) confirm the external validity of current findings by collecting data from general and clinical populations with a history of antisocial behaviour, especially from outside of the USA; (6) assess a wide range of risk factors across study phases to allow greater control of potentially cofounding variables in analyses; (7) design and conduct longitudinal studies that cover the lifespan to better understand the dynamic nature of the antisocial cognition-antisocial behaviour relationship across the life course; and (8) design and conduct experimental studies to determine whether antisocial cognition causes antisocial behaviour. Such studies would help elucidate the causal role of antisocial cognition in the antisocial behaviour pathway, which in turn could help clarify the validity of current theories of antisocial behaviour and facilitate the development of more effective interventions to prevent or reduce such behaviour.

To the authors knowledge, this review had few limitations. One key limitation was that it did not include a second reviewer. This is important as research suggests that the inclusion of a second reviewer in systematic reviews can reduce errors and bias when searching for and selecting studies (McDonagh, Peterson, Raina, Chang, & Shekelle, 2013). The exclusion of two non-English papers could be considered another limitation. Future reviewers of this area might also consider focusing on moderators as well as mediators of the antisocial cognition-antisocial behaviour relationship.

Whilst this review has not been able to offer firm conclusions regarding the causal nature of the relationship between antisocial cognition and antisocial behaviour, it has provided a comprehensive and critical overview of the current state of the literature in this area. By default, the major implication of this review is that more high-quality research is needed to fully understand the causal role of antisocial cognition in the antisocial behaviour pathway.

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Part II: Empirical Paper

Testing the Role of Antisocial Cognition as a Mediator of the Peer Influence Effect and Peer Selection Effect in Older Children and Adolescents: A Longitudinal Mediation Analysis

Abstract

Aims: The peer influence and peer selection effects are two widely replicated findings in the criminological literature which are believed to be involved in the development of antisocial behaviour. Recent research suggests that antisocial cognition might constitute a causal mechanism underlying part of these effects. Building on this research, the current study investigates the extent that each effect is mediated by one aspect of antisocial cognition – *beliefs and attitudes supporting peer conflict*.

Method: Using mediation analysis, it examined whether beliefs and attitudes supporting peer conflict mediate the relationship between delinquent peer association and volume of self-reported antisocial behaviour and visa-versa, across a one-year follow-up period, in a large group of British older children and adolescents with a history of serious antisocial behaviour from the Systemic Therapy for at Risk Teens study.

Results: Consistent with study hypotheses, beliefs and attitudes supporting peer conflict partially mediated the peer influence and peer selection effects, explaining a substantial proportion of the total effect in the peer influence (i.e., 26%) and peer selection (i.e., 17%) models, respectively. Sensitivity testing revealed that the mediating effects were modestly to moderately robust to the confounding effects of unobserved covariates.

Conclusion: In conclusion, the present research suggests that beliefs and attitudes supporting peer conflict could constitute a causal mechanism underlying the peer influence and peer selection effects in older children and adolescents with a history of serious antisocial behaviour.

Introduction

Antisocial behaviour is an ambiguous construct with multiple overlapping definitions. Within the UK, the term is used by government agencies to refer to “conduct that has caused, or is likely to cause, harassment, alarm or distress to any person” (*Anti-social Behaviour, Crime and Policing Act*, 2014, p. 2). This broad definition encompasses offending behaviours in adults (i.e., criminal behaviour) and minors (i.e., delinquent behaviour), along with status offences (i.e., behaviour that is prohibited or unlawful in minors but not adults) and several related behaviours that are socially disruptive or depart from usual or accepted standards (i.e., deviant behaviour). Within mental health, the term is primarily used to describe a pattern of unwanted behaviour in children or adolescents that is symptomatic of a diagnosis of *conduct disorder* (CD; American Psychiatric Association, 2013). Critically, this behaviour is deemed to violate the basic rights of others or major age-appropriate societal norms and includes violence towards people and animals, destruction of property, deceitfulness or theft, and serious violations of rules.

At least 1.8 million incidents of antisocial behaviour took place in England and Wales in the year ending March 2016 (Office for National Statistics, 2017). Epidemiological studies indicate that CD is the most common mental health problem globally, with a lifetime prevalence of 6.8% (5.8% female, 7.9% male) (Merikangas et al., 2010). Moreover, research suggests that individuals with CD are more likely to have mental health problems (Keenan & Wakschlag, 2000; Nock, Kazdin, Hiripi, & Kessler, 2006), poorer academic achievement, relationship and family problems, and

contact with the criminal justice system (Colman et al., 2009; Hill & Maughan, 2001). The economic burden of antisocial behaviour on society is also considerable, with the most recent report suggesting that the cost to government agencies of responding to antisocial behaviour in England and Wales alone could be approximately £3.4 billion per annum (The Police Foundation, 2010).

Antisocial behaviour usually emerges in late childhood and early adolescence, peaks in mid-to-late adolescence, and decreases thereafter (Loeber & Hay, 1997). Risk-focused research in the last 30 years has led to the identification of numerous risk factors that appear to increase a young person's chances of developing antisocial behaviour (Joan, Cathy, & Nancy, 2001). In the study of antisocial behaviour, the term *risk factor* is generally used to refer to any attribute, characteristic or exposure of an individual that has been shown to precede antisocial behaviour and is associated with an increased likelihood of such behaviour. Interestingly, this body of work has consistently demonstrated a predictive relationship between delinquent peer association and antisocial behaviour and antisocial behaviour and delinquent peer association (Dishion & Owen, 2002; Gifford-Smith, Dodge, Dishion, & McCord, 2005; Monahan, Steinberg, & Cauffman, 2009; Seddig, 2014; Svensson, Burk, Stattin, & Kerr, 2012). The former of these relationships is commonly known as the *peer influence effect*, whereas the latter is referred to as the *peer selection effect*.

Once a risk factor relationship has been identified in the literature, the next step is to identify the process or *causal mechanism* that explains *how*

the risk factor is connected to the outcome and brings the outcome about. Mediation analysis is fundamental in this regard. Mackinnon, Kisbu-Sakarya, & Gottschall (2013) define a mediator (M) as a "variable that transmits the effect of an antecedent variable (X) to an outcome variable (Y) in a causal sequence such that X causes M and M causes Y" (p. 338). Mediation analysis therefore allows researchers to test putative causal mechanisms of known risk factor relationships by examining the degree to which putative mediators account for the influence of a risk factor on an outcome variable (Fiedler, Schott, & Meiser, 2011).

Eminent theories of crime offer different propositions about the causal mechanisms behind the peer influence and peer selection effects and their temporality in the antisocial behaviour pathway. Differential association theory (Akers, 1998; Sutherland, 1947), for example, suggests that young people develop antisocial beliefs and attitudes through associating with delinquent peers, which are then expressed as antisocial behaviour. On the other hand, social bond theory (Hirschi, 1969) offers a purely behavioural account of the peer selection effect, whereby antisocial behaviour directly results in delinquent peer association, as youths seek out friendships with similar others, but also acknowledges antisocial beliefs and attitudes as a cause of antisocial behaviour. Additionally, (Thornberry, 1987) interactional theory posits that antisocial beliefs and attitudes, delinquent peer association and antisocial behaviour are reciprocally related, such that all three variables influence each other in a dynamic way over time. Identifying the causal mechanisms behind the peer influence and peer selection effects is therefore important to help clarify the validity of theories of antisocial behaviour and

further understanding about how such behaviour develops, which in turn could facilitate the development of more effective interventions to prevent or reduce antisocial behaviour.

Walters recently postulated that the peer influence and peer selection effects could be transmitted by similar causal mechanisms, namely aspects of *antisocial cognition* (Walters, 2015, 2016a, 2016b, 2017b). Whilst antisocial cognition has not been universally defined, the term is generally used to refer to attitudes, values, beliefs and rationalisations and a personal identity that is supportive of crime (Andrews & Bonta, 2010). In an initial mediation analysis, Walters (2015) assessed *proactive criminal thinking* using four items asking about the acceptability of stealing in given situations and discovered that this measure partially mediated the peer influence effect in a group of youths aged 10-18 years, without a history of serious offending, from the British Offending, Crime and Justice Survey. Two successive papers also found that proactive criminal thinking, measured using 10 items asking about the acceptability of violence, breaking rules and lying in certain situations, and *attitudes toward deviance*, assessed using nine items asking about the acceptability of engaging in various antisocial acts, independently and conjointly mediated part of the peer influence effect in a sample of American youths aged 11-17 years from the National Youth Survey (Walters, 2016a, 2017b). Furthermore, Walters (2016) ascertained that proactive criminal thinking, measured using the moral disengagement scale (Bandura, Barbaranelli, Caprara, & Pastorelli, 1996), and *reactive criminal thinking*, assessed using the Weinberger Adjustment Inventory (Weinberger & Schwartz, 1990) Impulse Control scale, partially mediated the peer influence

effect and peer selection effect, respectively, in a group of male adjudicated adolescents aged 14-19 years from the Pathways to Desistance study. Considering these findings, Walters concluded that aspects of criminal thinking explain at least part of the peer influence and peer selection effects (Walters, 2015, 2016a, 2016b, 2017b).

To date, no other studies have examined aspects of antisocial cognition as mediators of the peer influence and peer selection effects. Whilst Walter's research has many strengths, his findings are weakened by certain methodological shortcomings. None of the studies employed standardised measures of antisocial cognition with demonstrated reliability and validity. It is therefore unclear if the relevant constructs were measured in a reliable or valid way. Indeed, Walters himself recognised the poor content validity of proactive and reactive criminal thinking measures (Walters, 2015, 2016a, 2016b). Moreover, the construct validity of these measures was threatened by the inadequate pre-operational explication of constructs across studies. It could also be argued that reactive criminal thinking, as described and measured in Walters (2016b), constitutes a personality or behavioural disposition rather than cognition. Thereby, the extent that aspects of antisocial cognition explain the peer influence effect is presently unclear.

Walter's extensive use of historical data from population surveys conducted in the 1970's and 1980's also raises questions about the generalisability of his findings to the present day. Moreover, the mediating role of aspects of antisocial cognition in the peer influence and peer selection effects has never been examined in relation to youths with a history of

serious antisocial behaviour or males and children under the age of 14 years, respectively. Understanding the nature of both effects is especially important in youths with serious antisocial behaviour, since this group are the most likely to continue offending across the lifespan (Burt, 2012; Leschied, Chiodo, Nowicki, & Rodger, 2008; Young, Taylor, & Gudjonsson, 2016).

Walter's failure to use standardised measures of antisocial cognition relates to his use of historical data, when such measures were unavailable. Antisocial cognition is now a recognised risk factor for antisocial behaviour, which has led to the generation of new measures of the construct and its elements in children and adolescents. One such measure is the Antisocial Beliefs and Attitudes Scale (ABAS; Butler, Leschied, & Fearon, 2007). Described as "a developmentally sensitive measure that captures young people's beliefs and attitudes towards social standards of acceptable behaviour in the context of their interpersonal relationships at home and at school" (Butler, Parry, & Fearon, 2015, p. 291), the ABAS measures empirically grounded constructs relevant to the development of antisocial behaviour in children and adolescents, which are broadly consistent with current definitions of antisocial cognition (Andrews & Bonta, 2010).The peer conflict subscale, or factor, of the ABAS, captures "the extent to which young people identify beliefs and attitudes that support engaging in conflict with peers, physical fighting, and behaving aggressively with other peers such as gang members" (Butler et al., 2015, p. 298). In two initial studies of the psychometric properties of the ABAS, Butler and colleagues discovered that the peer conflict factor predicted self-reported antisocial behaviour in primary and secondary school children in Canada (Butler et al., 2007) and the U.K.

(Butler et al., 2015), as well as self- and parent-reported antisocial behaviour in British young offenders (Butler et al., 2015). More recently, Butler and colleagues (in-press) also found that the peer conflict factor predicted new overall violent and non-violent offending, above and beyond gender, callous-unemotional traits and baseline offending, in British adolescents with a history of serious antisocial behaviour.

These findings attest to the importance of beliefs and attitudes supportive of peer conflict in the genesis of antisocial behaviour. They are also broadly consistent with research showing that children who endorse such beliefs and attitudes are more likely to engage in aggressive behaviour, as well as information processing theories which view normative beliefs and attitudes about aggression as a cognitive factor in the aetiology of aggression in children (Huesmann & Guerra, 1997; Zelli, Dodge, Lochman, & Laird, 1999). The aetiological significance of the peer conflict factor is further highlighted by the existence of the peer influence effect and well-established relationship between early persistent childhood aggression and the later development of serious conduct problems (Dishion & Patterson; Loeber, 1990). While no study has ever investigated the predictive relationship between the peer conflict factor of the ABAS and delinquent peer association, it seems likely that the two constructs are mutually predictive, on the basis that delinquent peer association could conceivably influence a person's beliefs and attitudes about peer conflict, while a person's beliefs and attitudes about peer conflict could conceivably influence their association with certain peer groups. Thereby, the extent to which a person holds beliefs and attitudes that support peer conflict could constitute a causal mechanism that

explains at least part of the peer influence and peer selection effects and warrants further investigation.

The Current Study

In summary, antisocial behaviour is a common and costly problem that usually begins during late childhood and adolescence. The peer influence and peer selection effects are two robust findings in the criminological literature, which are believed to be involved in the aetiology of antisocial behaviour. Research suggests that the peer influence and peer selection effects might be explained, in part, by aspects of antisocial cognition, however this idea requires further study. Currently, more research is needed that uses representative samples of older children and adolescents with or without a history of serious antisocial behaviour and well-standardised, psychometrically robust measures of antisocial cognition. Furthermore, the extent that beliefs and attitudes supporting peer conflict explain the peer influence and peer selection effects warrants further investigation.

The present study aimed to address this gap in the literature by investigating whether an empirically-derived aspect of antisocial cognition – the peer conflict factor from the ABAS - which is both highly relevant to delinquent peer association and predictive of antisocial behaviour in older children and adolescents, explains the peer influence and peer selection effects in a representative sample of British youth with a history of serious antisocial behaviour from the Systemic Therapy for at Risk Teens (START) study (Fonagy et al., 2013). More specifically, in two separate mediation analyses, it longitudinally determines the predictive relationships between

delinquent peer association, beliefs and attitudes supportive of peer conflict, and self-reported antisocial behaviour, before clarifying the extent that antisocial beliefs and attitudes supporting peer conflict mediate the peer influence and peer selection effects.

Based on past theory and research, in the first mediation analysis it was hypothesised that delinquent peer association would predict beliefs and attitudes supporting peer conflict, which in turn would predict self-reported antisocial behaviour and, thus, beliefs and attitudes supporting peer conflict would mediated the peer influence effect. In the second mediator analysis, it was hypothesised that self-reported antisocial behaviour would predict beliefs and attitudes supporting peer conflict, which in turn would predict delinquent peer association and, thus, beliefs and attitudes supporting peer conflict would mediate the peer selection effect.

Method

Ethical Approval

The START study was approved by the London-South East Research Ethics Committee (reference number 09/H1102/55). The ethical opinion letter can be viewed in Appendix B.

Study Design

The START study was a national multicentre pragmatic clinical randomised controlled trial, comparing the efficacy of multisystemic therapy (MST) with management as usual (MAU) in reducing risk of out-of-home placement in older children and adolescents at risk of this due to significant antisocial behaviour. A convenience sample was recruited from nine sites

across the UK between 2010 and 2013. Recruitment sites comprised services and institutions designed to manage antisocial behaviour (i.e., youth offending teams, Child and Adolescent Mental Health Services, social services, and educational services). Multi-agency panels and multisystemic teams residing at recruitment sites identified and contacted all new cases meeting eligibility criteria. Eligible consenting participants completed a battery of questionnaires at baseline, after which approximately half of the sample were randomised to MST (50.1%) or MAU (49.9%). Follow-up assessments were conducted by a research assistant at participants' homes six, 12 and 18 months later. The response rate for the START study was 76%. Of the total sample at baseline, 85% was retained for six-month assessment, 80% for 12-month assessment and 75% for 18-month assessment.

To be included in the START study, participants had to be aged 11-17 years and display significant antisocial behaviour manifested as at least one of the following criteria: (1) persistent (weekly) and enduring (≥6 months) violent and aggressive interpersonal behaviour; (2) a significant risk of harm to self or to others (e.g., self-harming, substance misuse, sexual exploitation, absconding); (3) at least one conviction and three warnings, reprimands or convictions in the past 18 months; (4) current diagnosis of an externalising disorder and a record of unsuccessful outpatient treatment; and (5) permanent school exclusion. Exclusion criteria can be viewed in Appendix C.

Participants

Participants in this study were 683 (433 male, 250 female) older children and adolescents aged 11-17 years, who underwent baseline

assessment in the START study. The average age of participants at baseline was 13.81 years (*SD* = 1.41), with nearly two thirds (65.3%) aged 11-14 years and just over one third (34.7%) aged 15-17 years. Approximately half (50.1%) of the sample received MST over MAU. Most participants were White British/European (78.3%), with the remainder classified as Black African/Afro-Caribbean (10.4%), Asian (2.3%), and Mixed/Other (7.5%). Socioeconomic status was as follows: low (62.1%), medium (26.1%), and high (9.9%). A total of 1.5% and 1.9% of participants did not provide information about their ethnicity or socioeconomic status, respectively. Nearly half (43.5%) of participants were classified as displaying significant early onset antisocial behaviour (i.e., a pattern of antisocial behaviour that included aggression and began before 11 years of age) and 56.5% were classified as late-onset (i.e., antisocial behaviour that began after 11 years of age). Additionally, 78% of participants received a diagnosis of CD based on a semi-structured diagnostic interview and standardised checklists in the START study, while 65% had committed at least one offence prior to randomisation. Analysis of demographic variables (i.e., age, gender, ethnicity, socioeconomic status) using the Mann-Whitney U-test and Chi-square test revealed no significant differences (all *p's* < .05) between treatment groups (i.e., MST vs. MAU).

Measures

Delinquent peer association six and 18 months after randomisation served as the independent and dependent variable when examining the peer influence effect and peer selection effect, respectively. Delinquent peer association was measured using the Your Friends subscale of the Self-

Report Delinquency measure (SRD; Smith & McVie, 2003). The SRD was developed in the Edinburgh Study of Youth Transitions and Crime, which began during early adolescence and involved 4300 participants. The Your Friends subscale comprises seven items asking about respondents' friends involvement in antisocial behaviour during the last six months. Antisocial behaviours covered include substance use (i.e., tobacco, alcohol, illegal drugs), truancy, theft, vandalism, identity fraud, robbery, being noisy or rude in public, burglary, fire setting, weapon possession, violence towards people or animals, forced physical sexual behaviour and drug dealing. The first three items are rated on a three-point scale (*none or I'm not sure = zero point, one or some = one point, most or all = two points*), while the remaining items are rated on a two-point subscale (*no or not sure = zero points, yes = one point*). Items were summed to form a score for delinquent peer association in the last six months. Scores theoretically ranged from zero to 20. The Your Friends subscale has demonstrated split-half reliability (Smith & McVie, 2003), as well as construct validity in relation to self-reported delinquency in early-to-mid adolescence (Smith, 2005; Smith et al., 2001) and can be viewed in Appendix D.

Volume of antisocial behaviour six and 18 months after randomisation served as the independent and dependent variable when examining the peer selection effect and peer influence effect, respectively. Volume of antisocial behaviour was measured using the Volume of Delinquency subscale of the SRD (Smith & McVie, 2003). The Volume of Delinquency subscale comprises 21 items asking about respondents' involvement in antisocial behaviour during the last six months. Antisocial behaviours covered include

truancy, running away from home, theft, vandalism, identity fraud, robbery, being noisy or rude in public, burglary, fire setting, weapon possession, violence towards people or animals, forced physical sexual behaviour and drug dealing. All items are rated on a seven-point subscale (*once = on point, twice = two points, three times = three points, four times = four points, five times = five points, between six and 10 times = six points, more than 10 times = seven points*). Items four and five also include two extra questions rated on a three-point scale (*one to two days = zero points, up to one week = one point, up to two weeks = two points, more than two weeks = three points*). Items were summed separately to form a score for volume of antisocial behaviour in the last six months. Scores could theoretically range from zero to 153. The Volume of Delinquency subscale has demonstrated split-half reliability (Smith, 2005; Smith et al., 2001), as well as concurrent validity in relation to officially recorded delinquency in early to middle adolescence (Smith et al., 2001) and can be viewed in Appendix E.

Beliefs and attitudes supporting peer conflict 12 months after randomisation served as the mediator variable when examining the peer influence and peer selection effects. Beliefs and attitudes supporting peer conflict were measured using the peer conflict factor of the ABAS (Butler et al., 2007, 2015), which comprises 10 items asking about respondent's beliefs and attitudes toward peer conflict. Examples include: (1) *Fighting is cool when you're with a group of kids,* (2) *It's ok to walk away from a fight,* and (3) *It's fun and exciting to belong to a gang*. Each item is rated on a three-point scale (*agree = two points, not sure = one point, disagree = zero points*). Items four and 10 were reverse scored and all items summed to form a total

score. Scores theoretically ranged from zero to 20. The peer conflict factor has demonstrated adequate internal consistency (Cronbach's alpha = .77) and test-retest reliability (*r* = .77) over an eight-week period (Butler et al., 2015), as well as concurrent, predictive, and construct validity in community and offending samples of older children and adolescents (Butler et al., in-press, 2007, 2015). A copy of the Peer Conflict subscale from the ABAS can be viewed in Appendix F.

The following demographic/clinical variables, all measured at baseline, were also included in this study: age, gender (*female = zero, male = one*), socioeconomic status (*low = one, medium = two, high = three*) and treatment group (*MST = one, MAU = two*).

Statistical Analysis

Data screening indicated that the distribution of all continuous variables was non-normal. Age had a normal skew and negative kurtosis, while beliefs and attitudes supporting peer conflict at baseline and 12 months after randomisation had a positive skew and normal kurtosis. The remaining variables had a positive skew and positive kurtosis.

Study hypotheses were tested using mediation analysis. Prior to each mediation analysis, the inter-correlations between variables was explored using Spearman's rank-order correlation coefficient. This test is a non-parametric version of the Pearson product-moment correlation coefficient and was conducted using a two-sided test with pairwise comparison and a Bonferroni corrected alpha level of .002. Bonferroni correction effectively controls for the inflated risk of Type 1 error (i.e., the reporting of a false-

positive result) that occurs when multiple significance tests are performed. As a rule of thumb, Cohen (1988) suggests that an *r* of .1, .3 and .5 represents small, medium and large effect sizes, respectively.

Three effects are stated in mediation analysis: direct, total and indirect. A direct effect is the degree to which a change in an upstream variable influences a change in a downstream variable without going through any other variable. In Figure 1, the direct effect of X on M, M on Y, and X on Y are represented by the path coefficients A, B and C, respectively. The total effect is equal to the sum of C + AB and reflects the extent that a change in X influences a change in Y regardless of M. The indirect effect is the product of path coefficients A and B and reflects the extent that a change in X influences a change in Y by means of M. Finally, the coefficient of C' represents the residual direct effect of X on Y after accounting for M. A full mediation process is empirically confirmed when C' equals zero and the indirect AB path (i.e., from X to Y via M) is statistically significant.

Mediation analysis was performed using path analysis in Stata 14. There are five steps involved in path analysis: model specification, model identification, model estimation, model evaluation and, if necessary, model re-specification. In short, model specification consists of specifying the relationships among variables based on past theory and research. Model identification involves determining if unique values can be found for parameter estimation. Model estimation concerns the estimation of model parameters and generation of an estimated population variance-covariance matrix for the observed variables in the specified model. Model evaluation

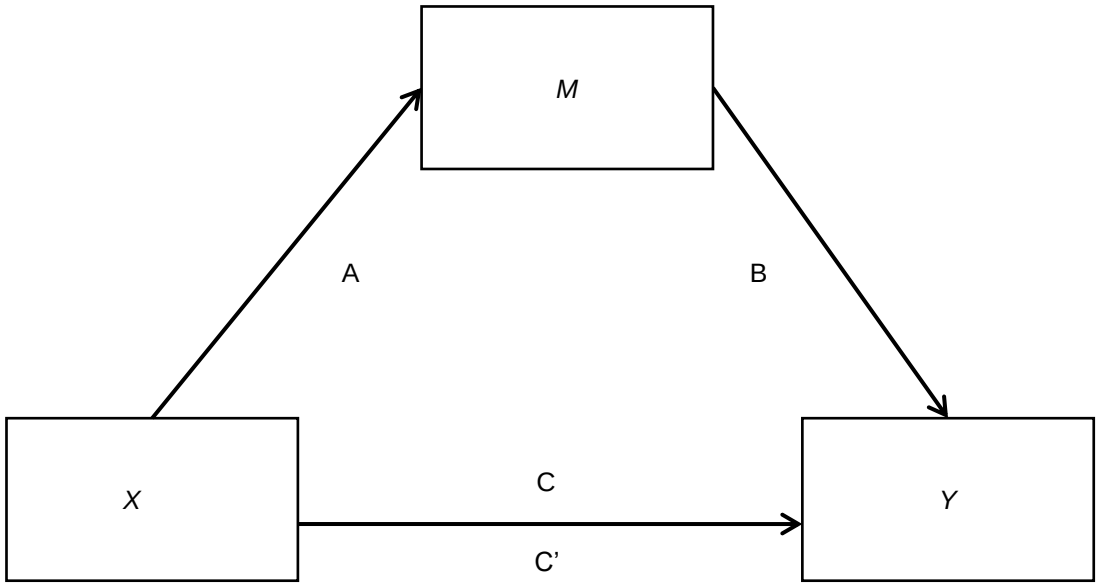

Figure 1. Conceptual path diagram for a simple mediation model.

involves testing how well the specified model reproduces the observed data, usually in terms of the degree of correspondence between the observed and model implied variance-covariance matrix. Finally, re-specification involves making changes to the model specification to improve fit.

Two mediation models were specified in this study. The peer influence model in Figure 2 tested whether antisocial beliefs and attitudes supporting peer conflict 12 months after randomisation had an indirect (mediating) effect on the relationship between delinquent peer association six months after randomisation and volume of self-reported antisocial behaviour 18 months after randomisation. Conversely, the peer selection model in Figure 3 examined whether antisocial beliefs and attitudes supporting peer conflict 12 months after randomisation had an indirect (mediating) effect on the relationship between volume of self-reported antisocial behaviour six months after randomisation and delinquent peer association 18 months after

randomisation. Baseline precursor measures of mediator and dependent variables were included in mediation models, as per Cole & Maxwell's (2003) recommendation in conducting mediation analysis. Precursor measures of mediator and dependent variables could covary within models. Demographic (i.e., age, gender, ethnicity, socioeconomic status) and clinical (i.e., treatment group) variables were included in mediation models as covariates of mediator, independent or dependent variables where they correlated with these variables.

Path models should be exactly identified or over identified, but never under identified. Under identification occurs when the number of estimated parameters exceeds the number of observations in a model. Exactly identified models have an equal number of observations and estimated parameters, whereas over identified models have a greater number of observations than estimated parameters and thus positive degrees of freedom. Rather than sample size, the number of observations in path analysis is based on the number of variables in the model (k). As explained by Norman and Streiner (2003), the specific formula for the number of observations in a path model is equal to $[k(k+1)]/2$. As there were five variables in each of the two models specified in this study, the number of observations for each model was equal to 15. The number of parameters in a model is equal to the sum of the number of direct paths, the number of variances of exogenous variables (variables not predicted by other variables), the number of covariances, and the number of disturbance terms. Based on this, the two models in this study had 14 parameters each and

were thus over identified. As none of the models had any reciprocal relations or feedback loops, they were also recursive.

The Doornik-Hansen test showed that the multivariate distribution of continuous variables in this study was significantly non-normal, X^2 (18, N = 669-683) = 2211.32, $p < .001$. Thereby, each path model was estimated using the Satorra-Bentler estimator in Stata. The Satorra-Bentler estimator provides adjusted goodness-of-fit statistics, standard errors, *p*-values, and 95% confidence intervals, which are robust to univariate and multivariate non-normality, and can be used to evaluate model fit and the statistical significance of total, direct and indirect effects.

Model evaluation involves assessing goodness-of-fit indices and the magnitude and significance of parameter estimates. Goodness-of-fit indices provide a measure of how well a specified model corresponds with the observed data. This study employed several goodness-of-fit indices, including the likelihood ratio chi-squared test, Root Mean Squared Error of Approximation (RMSEA), Comparative Fit Index (CFI) and Akaike Information Criterion (AIC). The likelihood ratio chi-squared test assesses whether the observed and model-implied variance-covariance matrices differ significantly. A non-significant chi-square value at an alpha level of .05 indicates that the variance-covariance matrices are not significantly different and thus the specified model is a good fit of the data. The RMSEA, CFI and AIC are descriptive measures of goodness-of-fit and less sensitive to sample size than the likelihood ratio chi-squared test. The RMSEA measures the discrepancy between the observed and model-implied variance-covariance

matrices, whereas the CFI compares the specified model with the null model (i.e., a model in which the variables are assumed to be uncorrelated). RMSEA and CFI values theoretically range from zero to one. Good model fit is indicated by a RMSEA value of below .06 (Hu & Bentler, 1999) and CFI value of greater than .95. The AIC also reflects the extent to which the observed and specified model variance-covariance matrices differ, but adjusts for model complexity (i.e., the number of parameters estimated). It is generally used to compare competing models; the model with a lower AIC value has a better fit with the data. The above fit indices were chosen as they provide a range of information about model fit, including absolute fit, fit adjusted for sample size, fit adjusted for parsimony, and fit relative to a null model.

If model fit is acceptable, the next step in the model evaluation process is to establish the significance of direct, indirect and total effects. An alpha level of .05 was used as the cut-off for significance for all effects. Criteria for mediation was a statistically significant indirect effect of M on the X-Y relationship and a statistically non-significant or diminished direct effect of X on Y.

Several effect size measures have been proposed for mediation analysis, however according to Walters (2017c) most of these measures fall short of satisfying one or more of the core components of an effect size indicator suggested by Cohen (1988) (i.e., sample size independence, cross-sample comparability, monotonicity and freedom from external influence). Given these limitations and as recommended by Wen and Fan (2015), this

study used the ratio of the indirect effect to the total effect as an effect size indicator for the indirect effect. The ratio of the indirect effect to the total effect is calculated by dividing the indirect effect by the total effect and is interpreted as the proportion of the total effect that is mediated (Preacher & Kelley, 2011; Wen & Fan, 2015).

Sensitivity testing was conducted using Kenny's (2013) “failsafe ef” procedure to understand how much an unobserved covariate would need to correlate with M and Y to reduce the M-Y relationship to zero and, thus, confound any observed indirect effect.

Kline (2012) recommends a ratio of 20 participants per parameter and a sample size of at least 200. Following the removal of outliers, the smallest sample analysed in a mediation analysis in this study comprised 663 participants with a ratio of 47 participants per parameter and therefore easily satisfied this recommendation. An outlier was defined as an observation on a variable that was three times greater than the interquartile range of the variable.

Multicollinearity is a phenomenon in which two or more variables are very closely linearly related. Path analysis assumes low multicollinearity among predictor variables. Multicollinearity was tested using the variance inflation factor (VIF), which indicates whether a predictor has a strong linear relationship with other predictors. Related to VIF is the tolerance statistic, which is its reciprocal (1/VIF). According to Field (2009), a VIF value of greater than 10 and tolerance value of less than .1 indicate a severe problem with multicollinearity. There was no evidence of multicollinearity between the

control and predictor variables for either of the models estimated (peer influence model: tolerance = 0.732-0.971, VIF = 1.030-1.366; peer selection model: tolerance = 0.769-0.977, VIF = 1.024-1.301).

Missing Data

Complete data for the 12 variables in this study was available for 370 participants (54.2%); 23 participants (3.4%) had missing data on one variable, 55 participants (8.1%) had missing data on two variables, 62 participants (9.1%) had missing data on three variables, 20 participants (2.9%) had missing data on four variables, 61 participants (8.9%) had missing data on five variables, and 92 participants (13.4%) had missing data on six to eight variables. Whilst seven variables had less than two percent missing data, the remaining six variables had between 19% and 33% missing data. Variables with over 30% missing data included delinquent peer association (32.5%) and volume of self-reported antisocial behaviour (31.5%) 18 months after randomisation.

Excluding cases with missing data can lead to biased parameter estimates, loss of information, decreased statistical power, increased standard errors, and weakened generalisability of findings (Dong & Peng, 2013). Missing data were therefore handled using expectation maximisation (EM) as implemented in SPSS. EM is a single imputation method, which imputes missing data with maximum likelihood values based on the observed relationships among all the variables. Values are imputed iteratively until successive iterations are sufficiently similar. Moreover, a degree of random

error is incorporated for each imputed value to reflect the uncertainty associated with the imputation (Acock, 2005).

EM depends on the assumption that the pattern of missing data is missing completely at random (MCAR) or missing at random (MAR). A pattern of missing data is MCAR when the missing values are randomly distributed throughout the dataset, whereas a pattern of missing data is MAR when the likelihood of missing data on a variable is not related to the participant's score on the variable, after controlling for other variables in the study. The current study used Little's Missing Completely at Random test to judge whether the pattern of missing data was missing completely at random. The findings were consistent with this assumption, X^2 (194, N = 461-683) = 214.39, p = .15.

Results

The results are presented in three sections. The first section presents findings from a descriptive analysis of the 12 variables in this study, while the second and third sections present findings from the correlation and mediation analysis for the peer influence effect and peer selection effect, respectively.

Descriptive Analysis

Table 1 lists descriptive statistics for the 12 variables in this study.

Table 1

Descriptive Statistics for the 12 Variables in this Study

Variable		*N*	*M*	*SD*	*Mdn*	Range	*IQR*
Age		683	13.81	1.41	14	11-17	6
PC-baseline		683	6.77	4.01	6	0-18	6
PC-12		683	6.52	3.58	6	0-19	5
DPA-baseline		683	4.95	4.65	3	0-18	5
DPA-6		683	4.76	4.18	4	0-18	4
DPA-18		681	4.58	4.19	4	0-18	4
Vol-baseline		677	19.59	17.06	15	0-86	21
Vol-6		675	15.96	14.42	12	0-72	18
Vol-18		669	8.74	8.28	7	0-41	9
Gender	Male			433 (63.4%)			
	Female			250 (36.6%)			
SES	Low			424 (63.3%)			
	Medium			178 (26.6%)			
	High			68 (10.2%)			
Treat	MST			342 (50.1%)			
	MAU			341 (49.1%)			

Note. N = number of non-missing cases; *M* = mean; *SD* = standard deviation; *Mdn* = median; *IQR* = interquartile range; Age = age at study entry; PC-baseline = beliefs and attitudes supporting peer conflict at baseline; PC-12 = beliefs and attitudes supporting peer conflict 12 months after randomisation; DPA-baseline = delinquent peer association at baseline; DPA-6

= delinquent peer association six months after randomisation; DPA-18 = delinquent peer association 18 months after randomisation; Vol-baseline = volume of antisocial behaviour at baseline; Vol-6 = volume of antisocial behaviour six months after randomisation; Vol-18 = volume of antisocial behaviour 18 months after randomisation; SES = socioeconomic status; Treat = treatment group; MST = multisystemic therapy; MAU = management as usual.

Peer Influence Effect

Correlation Analysis

Table 2 presents inter-correlations for the five variables specified in the peer influence mediation model and four demographic/clinical variables.

Table 2

Inter-correlations for the Five Variables in the Peer Influence Model and Demographic/Clinical Variables

Variable	5	6	7	8	9
1. Age	.09	-.01	.03	.14	-.10
2. Gender	.07	.11	.06	.00	.02
3. SES	.01	.04	-.04	.03	.03
4. Treat	-.03	.01	-.01	.02	.03
5. PC-baseline		.46*	.13	.37*	.20*
6. PC-12			.15	.26*	.28*
7. DPA-6				.15	.17*
8. Vol-baseline					.37*
9. Vol-18					

Note. Age = age at study entry; SES = socioeconomic status; Treat = treatment group; PC-baseline = beliefs and attitudes supporting peer conflict at baseline; PC-12 = beliefs and attitudes supporting peer conflict 12 months after randomisation; DPA-6 = delinquent peer association six months after randomisation; Vol-baseline = volume of antisocial behaviour at baseline; Vol-18 = volume of antisocial behaviour 18 months after randomisation; * =

statistically significant effect at p < .002 level (two tailed) (Bonferroni-corrected alpha: .05/30 significance tests). Pairwise deletion applied.

Eight of 30 correlations performed achieved statistical significance. There were small to medium positive correlations between independent, mediator and dependent variables and medium to large positive correlations between precursor variables and mediator/dependent variables. Demographic/clinical variables did not correlate with any other variables and were therefore excluded from the mediation analysis. The relationship between delinquent peer association six months after randomisation and beliefs and attitudes supporting peer conflict 12 months after randomisation approached significance, $r(681) = .15$, $p = .004$.

Mediation Analysis

A mediation analysis was performed to examine whether beliefs and attitudes supporting peer conflict 12 months after randomisation mediated the relationship between delinquent peer association six months after randomisation and self-reported antisocial behaviour 18 months after randomisation. Table 3 lists the results of the mediation analysis, including the unstandardised and standardised path coefficients and asymptotic *z*-test results for direct, total and indirect effects. The standardised path coefficients for the peer influence model are also shown in the corresponding path diagram in Figure 2.

The peer influence model provided a good fit for the data. The likelihood ratio chi-square test was not significant, X^2 (2) = 3.45, $p = 0.178$, while the RMSEA, CFI and AIC were .03, .99 and 21004.90, respectively. Significant paths from baseline to 12-month beliefs and attitudes supporting

peer conflict and baseline to 18-month volume of self-reported antisocial behaviour indicated stability in these constructs.

Table 3

Peer Influence Effect: Direct, Total and Indirect Effects

	Direct Effects				
Path	*B* (95% CI)	*SE B*	β	*z*	*p*
DPA-6 to PC-12 (Path A)	0.07(0.01-0.13)	0.03	.08	2.13	.03
PC-12 to Vol-18 (Path B)	0.44(0.26-0.61)	0.09	.19	4.86	<.001
DPA-6 to Vol-18 (Path C')	0.09(-0.07-0.25)	0.08	.04	1.06	.29
PC-baseline to PC-12	0.39(0.32-0.45)	0.03	.43	11.83	<.001
Vol-baseline to Vol-18	0.14(0.11-0.18)	0.02	.29	7.55	<.001
	Total and Indirect Effects				
DPA-6 to Vol-18	*B* (95% CI)	*SE B*	β	*z*	*p*
Total Effect	0.12(-0.04-0.28)	0.08	.06	1.42	.15
Indirect Effect	0.03(0.00-0.06)	0.15	.02	2.02	.04

Note. Vol-18 = volume of antisocial behaviour 18 months after randomisation; PC-12 = beliefs and attitudes supporting peer conflict 12 months after randomisation; Vol-baseline = volume of antisocial behaviour at baseline; DPA-6 = delinquent peer association six months after randomisation; PC-baseline = beliefs and attitudes supporting peer conflict at baseline. *B* (95% CI) = unstandardized beta coefficient and the lower and upper limits of the 95% confidence interval for the unstandardized coefficient (in brackets); β = standardised coefficient; *z* = asymptotic z-test; *p* = statistical significance level of the asymptotic z-test; *N* = 663.

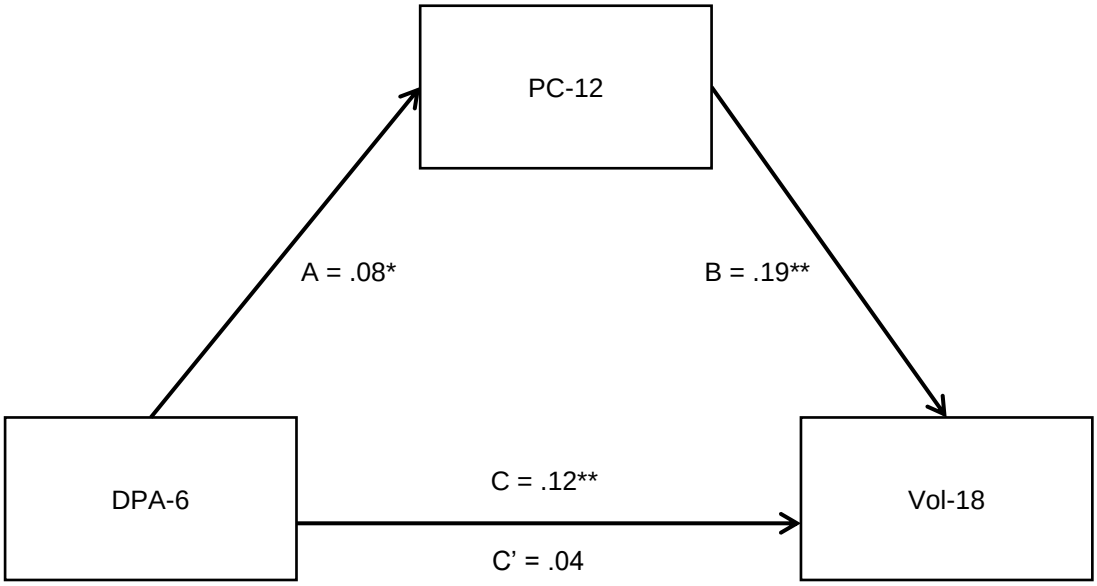

Figure 2. Path diagram for the peer influence mediation model. *N* = 663. Standardised beta coefficients are reported, control variables are not shown. DPA-6 = delinquent peer association at six months after randomisation; PC-12 = beliefs and attitudes related to peer conflict at 12 months after randomisation; Vol-18 = volume of self-reported antisocial behaviour at 18 months after randomisation; * = significant effect at $p < .05$ level (two tailed); ** = significant effect at $p < .01$ level (two tailed).

Delinquent peer association six months after randomisation had a significant direct effect on beliefs and attitudes supporting peer conflict 12 months after randomisation (Path A), but did not have a significant direct effect on volume of self-reported antisocial behaviour 18 months after randomisation (Path C'), when controlling for the effect of beliefs and attitudes supporting peer conflict and volume of self-reported antisocial behaviour at baseline. Beliefs and attitudes supporting peer conflict 12 months after randomisation also had a significant direct effect on volume of self-reported antisocial behaviour 18 months after randomisation (Path B),

when controlling for beliefs and attitudes supporting peer conflict and volume of self-reported antisocial behaviour at baseline.

The indirect path from delinquent peer association six months after randomisation to volume of self-reported antisocial behaviour 18 months after randomisation through beliefs and attitudes supporting peer conflict 12 months after randomisation was significant, however the total effect of delinquent peer association six months after randomisation on volume of self-reported antisocial behaviour 18 months after randomisation was not significant. The indirect effect accounted for 26% of the total effect of delinquent peer association six months after randomisation on volume of self-reported antisocial behaviour 18 months after randomisation.

Furthermore, the addition of beliefs and attitudes supporting peer conflict at baseline and 12 months after randomisation and volume of self-reported antisocial behaviour at baseline to the path model, caused the direct effect of delinquent peer association six months after randomisation on volume of self-reported antisocial behaviour 18 months after randomisation (Path C) to become non-significant and reduce in magnitude from $b = 0.25$, $z = 2.76$, $p = .006$ to $b = 0.09$, $z = 1.06$, $p = .29$.

Sensitivity testing revealed that unobserved covariates would need to correlate .25 with the mediator and dependent variable to eliminate the indirect effect of beliefs and attitudes supporting peer conflict on the peer influence effect observed in this study.

Peer Selection Effect

Correlation Analysis

Table 4 presents inter-correlations for the five variables specified in the peer selection mediation model and four demographic/clinical variables. Seven of 30 correlations performed were statistically significant. There were small to medium positive correlations between precursor, independent, mediator and dependent variables. Demographic and clinical variables did not correlate with any other variables and were therefore excluded from the mediation analysis.

Table 4

Inter-correlations for the Five Variables in the Peer Selection Model and Demographic/Clinical Variables

Variable	5	6	7	8	9
1. Age	.09	-.01	.04	.02	0.11
2. Gender	.07	.11	.00	.01	-.02
3. SES	.01	.04	-.02	-.03	.03
4. Treat	-.03	.01	-.01	-.01	.08
5. PC-baseline		.46*	.19*	.10	.30*
6. PC-12			.13	.20*	.33*
7. DPA-baseline				.21*	.12
8. DPA-18					.24*
9. Vol-6					

Note. Age = age at study entry; SES = socioeconomic status; Treat = treatment group; PC-baseline = beliefs and attitudes supporting peer conflict at baseline; PC-12 = beliefs and

attitudes supporting peer conflict at 12 months after randomisation; DPA-baseline = delinquent peer association at baseline; DPA-18 = delinquent peer association at 18 months after randomisation; Vol-6 = volume of antisocial behaviour at six months after randomisation; * = significant effect at p < .002 level (two tailed) (Bonferroni-corrected alpha). Pairwise deletion applied.

Mediation Analysis

A mediation analysis was performed to examine whether beliefs and attitudes supporting peer conflict 12 months after randomisation mediated the relationship between volume of self-reported antisocial behaviour six months after randomisation and delinquent peer association 18 months after randomisation. Table 5 lists the results of the mediation analysis, including the unstandardised and standardised path coefficients and asymptotic *z*-test results for direct, total and indirect effects. Standardised path coefficients for the peer selection model can also be viewed in the corresponding path diagram in Figure 3.

The peer selection model provided a good fit for the data. The likelihood ratio chi-square test was not significant, X^2 (2) = 1.505, p = .471, while RMSEA, CFI and AIC were equal to zero, .1 and 20409.51, respectively. Significant paths from baseline to 12-month beliefs and attitudes supporting peer conflict and baseline to 18-month delinquent peer association indicated stability in these constructs. Compared to the peer influence model, the peer selection model provided a marginally better fit for the data across goodness-of-fit indices, with a change in model fit per RMSEA, CFI and AIC of -.033, +.006 and -595.39, respectively.

Table 5

Peer Selection Effect: Direct, Total and Indirect Effects

	Direct Effects				
Path	*B* (95% CI)	*SE B*	β	*z*	*p*
Vol-6 to PC-12 (Path A)	0.05(0.03-0.07)	0.01	.20	5.27	<.001
PC-12 to DPA-18 (Path B)	0.16(0.06-0.26)	0.05	.14	3.20	.001
Vol-6 to DPA-18 (Path C')	0.04(0.01-0.06)	0.01	.13	2.92	.003
PC-baseline to PC-12	0.34(0.27-0.40)	0.03	.38	10.13	<.001
DPA-baseline to DPA-18	0.14(0.06-0.21)	0.04	.15	3.56	<.001
	Total and Indirect Effects				
Vol-6 to DPA-18	*B* (95% CI)	*SE B*	β	*z*	*p*
Total Effect	0.05(0.02-0.07)	0.01	.16	3.63	<.001
Indirect Effect	0.01(0.00-0.01)	0.00	.03	2.70	.007

Note. Vol-6 = volume of antisocial behaviour six months after randomisation; PC-12 = beliefs and attitudes supporting peer conflict 12 months after randomisation; DPA-18 = delinquent peer association 18 months after randomisation; PC-baseline = beliefs and attitudes supporting peer conflict at baseline; DPA-baseline = delinquent peer association at baseline; *B* (95% CI) = unstandardized beta coefficient and the lower and upper limits of the 95% corrected confidence interval for the unstandardized coefficient (in brackets); *SE B* = standardised error for the unstandardised beta coefficient; β = standardised coefficient; *z* = asymptotic z-test; *p* = statistical significance level of the asymptotic z-test; *N* = 673.

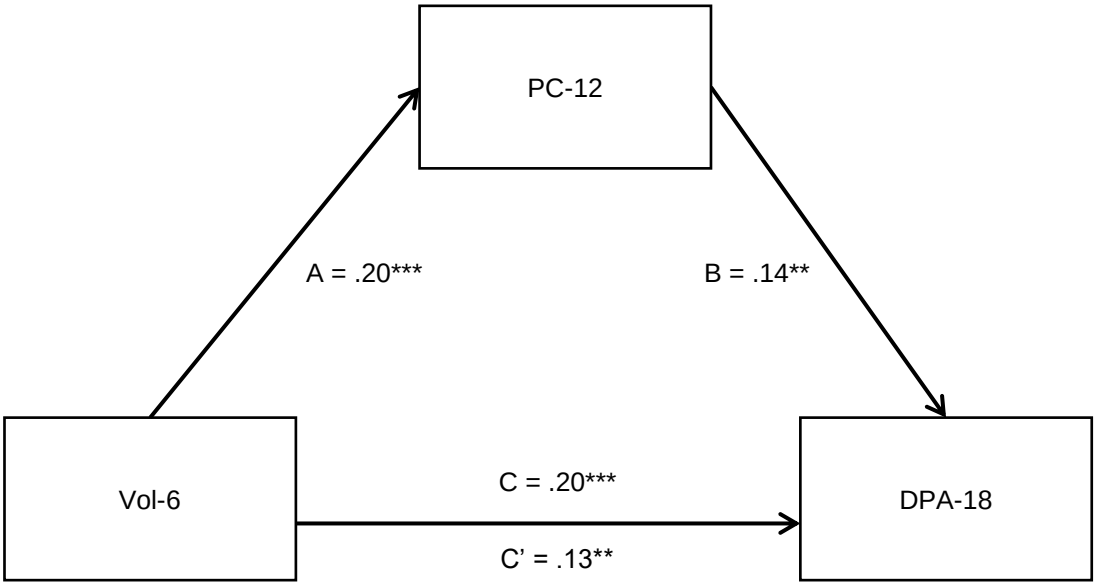

Figure 3. Path diagram for the peer selection mediation model. *N* = 673. Standardised beta coefficients are reported, control variables are not shown. Vol-6 = volume of self-reported antisocial behaviour six months after randomisation; PC-12 = beliefs and attitudes related to peer conflict 12 months after randomisation; DPA-18 = delinquent peer association 18 months after randomisation; ** = significant effect at $p < .01$ level (two tailed); *** = significant effect at $p < .001$ level (two tailed).

Volume of self-reported antisocial behaviour six months after randomisation had a significant direct effect on beliefs and attitudes supporting peer conflict 12 months after randomisation (Path A) and delinquent peer association 18 months after randomisation (Path C'), when controlling for the effect of beliefs and attitudes supporting peer conflict and delinquent peer association at baseline. Beliefs and attitudes supporting peer conflict 12 months after randomisation also had a significant direct effect on

delinquent peer association 18 months after randomisation (Path B), when controlling for the effect of beliefs and attitudes supporting peer conflict and volume of self-reported antisocial behaviour at baseline.

The indirect path from volume of self-reported antisocial behaviour six months after randomisation to delinquent peer association 18 months after randomisation through beliefs and attitudes supporting peer conflict 12 months after randomisation achieved significance, as did the total effect of volume of self-reported antisocial behaviour six months after randomisation on delinquent peer association 18 months after randomisation. The indirect effect accounted for 17% of the total effect of volume of self-reported antisocial behaviour six months after randomisation on delinquent peer association 18 months after randomisation.

The addition of beliefs and attitudes supporting peer conflict at baseline and 12 months after randomisation and delinquent peer association at baseline to the peer selection model, caused the direct effect of volume of self-reported antisocial behaviour six months after randomisation on delinquent peer association 18 months after randomisation (Path C) to reduce in significance and magnitude from $b = .06$, $z = 4.47$, $p = < .001$ to $b = .04$, $z = 2.92$, $p = .003$.

Sensitivity testing revealed that unobserved covariates would need to correlate .20 with the mediator and dependent variable to eliminate the indirect effect of beliefs and attitudes supporting peer conflict on the peer selection effect observed in this study.

Discussion

This study sought to build upon past research investigating the role of antisocial cognition as a mediator of the peer influence and peer selection effects. In two mediation analyses, it examined the extent that beliefs and attitudes supporting peer conflict mediated the relationship between delinquent peer association and volume of self-reported antisocial behaviour and visa-versa, across a one-year follow-up period in a large group of British older children and adolescents with a history of serious antisocial behaviour.

Consistent with study hypotheses, beliefs and attitudes supporting peer conflict partially mediated the peer influence and peer selection effects, when controlling for prior levels of mediator and dependent variables. Both mediation models fit the data well and the mediating effect of beliefs and attitudes explained 26% and 17% of the total effect in the peer influence and peer selection models, respectively. Sensitivity testing revealed that the mediating effects were modestly to moderately robust to the confounding effects of unobserved covariates.

Rather than a direct causal relationship between delinquent peer association and self-reported antisocial behaviour and vice-versa, this study found that delinquent peer association increased the likelihood of beliefs and attitudes supporting peer conflict, which in turn increased the likelihood of participating in antisocial behaviour. Similarly, volume of self-reported antisocial behaviour increased the likelihood of beliefs and attitudes supporting peer conflict, which in turn increased the likelihood of delinquent peer association. These findings suggest that the peer influence and peer

selection effects are partially accounted for by the same underlying casual mechanism, namely beliefs and attitudes supporting peer conflict.

Whilst the research design employed in this study cannot inform about the evolution of beliefs and attitudes supporting peer conflict, the findings support the conception that antisocial cognitions of this nature are reinforced by both delinquent peer association and participation in antisocial acts. Furthermore, although the study did not assess the peer influence and peer selection effects across repeated intervals, the direct effect of delinquent peer association on antisocial behaviour and visa-versa over the same period suggests that the two effects are reciprocally related and produced in part by beliefs and attitudes supporting peer conflict.

The results of this study both support and extend past research. The results are consonant with the wealth of literature documenting the peer influence and peer selection effects and other mediation analyses in this area, which suggest that aspects of antisocial cognition are important mediators of the peer influence (Walters, 2015, 2016a, 2016b, 2017) and peer selection (Walters, 2016b) effects. Furthermore, they are accordant with studies documenting a reciprocal relationship between the peer influence and peer selection effects (Dishion & Owen, 2002; Gifford-Smith et al., 2005; Monahan et al., 2009; Seddig, 2014; Svensson et al., 2012). Additionally, this was the first study to show that beliefs and attitudes supporting peer conflict are involved in the transmission of the peer influence and peer selection effects and by default that beliefs and attitudes supporting peer conflict could be a risk factor for delinquent peer association and visa-versa. Moreover, the

results confirm and extend the external validity of past studies (Walters, 2015, 2016a, 2016b, 2017), by reproducing similar findings using a standardised measure of a different aspect of antisocial cognition in a contemporary, mixed gender sample of older children and adolescents with a history of serious antisocial behaviour from outside the USA.

Mediation analysis is integral to theory building, since it can be used to better understand the mechanism of action through which an effect occurs. Ascertaining the mechanisms behind the peer influence and peer selection effects is pertinent, since criminological theories offer competing accounts about the way in which delinquent peer association and antisocial behaviour are causally related. The present findings are concordant with most major theories of offending to the extent that they identify antisocial cognition as a risk factor or consequence of delinquent peer association and antisocial behaviour (Andrews & Bonta, 2010; Hirschi, 1969). More specifically though, they support the prediction from differential association theory (Akers, 1998; Sutherland, 1947) that antisocial cognitions, like beliefs and attitudes supporting peer conflict, constitute important mechanisms underpinning the peer influence effect. Moreover, contrary to social bond theory (Hirschi, 1969) and the General Theory of Crime (Gottfredson, 2011; Gottfredson & Hirschi, 1990), they imply that antisocial beliefs and attitudes are involved in the transmission of the peer selection effect. Additionally, the findings are consistent with Interactional Theory (Thornberry, 1987), which specifies a dynamic, reciprocal relationship between delinquent peer association, antisocial cognition and antisocial behaviour through late childhood and early adolescence.

Improving understanding about the mechanisms responsible for the peer influence and peer selection effects could improve ability to predict, manage and reduce antisocial behaviour. The major implication of this study is that any comprehensive theory and intervention for serious antisocial behaviour in older children and adolescents should acknowledge the possible influence of beliefs and attitudes supporting physical violence or aggressive behaviour towards peers. Practically, it might be pertinent to measure a person's beliefs and attitudes about peer conflict using the peer conflict factor from the ABAS when intervening for antisocial behaviour. Intervention protocols could also be expanded to include strategies to reduce beliefs and attitudes supporting peer conflict. The current findings are especially pertinent, as research suggests that children and adolescents who are highly aggressive are more likely to be aggressive in adulthood (Burt, 2012; Leschied et al., 2008; Young et al., 2016).

This study had several strengths. First, there was correct temporal order between variables, an essential criterion for causality (Hill, 1965). Second, it controlled for prior levels of mediator and dependent variables, thereby allowing the possibility that the results were due to pre-existing differences on these variables to be ruled out. Third, it was based on a contemporary sample of children and adolescents with a history of serious antisocial behaviour, which included both genders and children younger than 14 years. Fourth, it used a standardised measure of antisocial cognition with good reliability and validity that was also highly relevant to delinquent peer association. Fifth, it used comprehensive general measures of delinquent peer association and volume of antisocial that corresponded in terms of

content, with some demonstrated reliability and validity. Sixth, the six-month follow-up was sufficient to observe changes in antisocial behaviour, but not too long to allow unobserved covariates to confound the results (Rennison & Rand, 2007; Walters, 2017a). Finally, sensitivity testing informed about the robustness of the observed indirect effects to confounding from unobserved covariates.

The study also had several limitations that should be considered when interpreting the findings. First, because it employed a non-experimental rather than experimental research design, it is not possible to advance causal inferences based on the findings (Bullock, Green, & Ha, 2010; Pirlott & MacKinnon, 2016; Stone-Romero & Rosopa, 2008). Second, the relatively wide age range of participants at study entry means that it cannot inform about when beliefs and attitudes supporting peer conflict are most likely to bring about the peer influence and peer selection effects. Third, because this study examined general antisocial behaviour, it cannot enlighten about whether certain antisocial behaviours are more likely to influence delinquent peer association or whether beliefs and attitudes supporting peer conflict are more likely to influence specific antisocial behaviours. Fourth, while both indirect effects were statistically significant, full mediation was not achieved in either mediation analysis and it could be argued that the indirect effects observed were small in size and therefore of limited importance. However, it is important to note that full mediation and large indirect effects are rare in mediation analysis in the social sciences (Kenny & Judd, 2014; Rucker, Preacher, Tormala, & Petty, 2011). The fact that the peer conflict factor

explained only 26% and 17% of the total effect in the peer influence and peer selection models, respectively, highlights that other factors are likely to be important in the transmission of both effects. Fifth, because this study examined the peer influence and peer selection effects in relation to a single mediator it is not possible to discern the relative importance, or clinical significance, of beliefs and attitudes supporting peer conflict in the transmission of each effect. Sixth, every variable in the study was measured using the self-report method, which could have inflated estimates of effect by way of shared method variance. Lastly, whilst participants in the START study were recruited from multiple locations and services in the UK, this was achieved using convenience sampling which is vulnerable to selection bias and could have reduced the representativeness of the sample.

In conclusion, the present research suggests that beliefs and attitudes supporting peer conflict could constitute a causal mechanism underlying the peer influence and peer selection effects. Whilst the findings from this study should be interpreted tentatively because of methodological shortcomings, it might be beneficial for policy makers and practitioners to consider the role of beliefs and attitudes supporting peer conflict when planning policy, developing prevention programmes, and implementing treatment.

Future research should examine the role of beliefs and attitudes supporting peer conflict as a mediator of the peer influence and peer selection effects using an experimental design, preferably alongside other psychological and social mediators in a cohort of children with a narrow age range at study entry. Mediators for inclusion in future analyses could include

other factors understood to contribute to the emergence and maintenance of antisocial behaviour; for example, low intelligence and poor problem-solving ability, antisocial personality pattern, poor family management practices (i.e., lack of parental monitoring and supervision; harsh, inconsistent or lax discipline; low parental support), poor academic performance and low school bonding (Andrews & Bonta, 2010; Day & Wanklyn, 2012). At the same time, it would be of interest to compare the peer conflict factor with other putative mediators of the peer influence and peer selection effects, like proactive criminal thinking and impulsivity. Studies like this would help clarify both the causal status and clinical significance of beliefs and attitudes supporting peer conflict to the peer influence and peer selection effects and, thus, could facilitate the development of more effective interventions to prevent or reduce antisocial behaviour.

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Part III: Critical Appraisal

Like most research on antisocial behaviour, my empirical study falls within the realm of the risk-focused research. In the last 10 years, researchers using this approach to investigate antisocial behaviour have been lambasted for their “uncritical over-interpretation” (O’Mahony, 2009, p. 103) of research findings. In response, this critical appraisal discusses the challenges associated with using mediation analysis to establish causal mechanisms in the study of antisocial behaviour. Specifically, it discusses the process of my research and makes clear why it is not possible to make casual inferences based on my findings. It then outlines three experimental mediation designs and how these could be used to investigate the research questions in my study. Finally, it highlights some of the challenges associated with the use of these designs and why it might be important for future researchers of this area to adopt a more methodologically pluralistic approach. To be succinct, I use X, M and Y to refer to independent, mediator and dependent variables, respectively.

Predicting and preventing antisocial behaviour requires an understanding of how such behaviour is caused. Risk factor research has been successful in identifying factors significantly correlated with offending and antisocial behaviour, however it has struggled to discern “which risk factors are causes and which are merely markers or correlated with causes” (Farrington, 2000, p. 7). Consequently, risk-focused research on antisocial behaviour has been criticised for being overly descriptive, conceptually incoherent, and failing to explain the causes of antisocial behaviour (Case & Haines, 2009; O’Mahony, 2009; Wikström, 2008). Recently, a handful of

authors have urged the field to move beyond the risk factor approach to a more explanatory approach that focuses on establishing the causes and causal mechanisms of offending (Case & Haines, 2009; O'Mahony, 2009; Wikström, 2008).

In accord, my study sought to improve understanding of antisocial behaviour in older children and adolescents by examining the role of beliefs and attitudes supporting peer conflict as a causal mechanism that generates the peer influence and peer selection effects. To recap, Wikström (2008) defines a causal mechanism as a "process that connects the cause and effect and that brings about the effect" (p. 131). In other words, a casual mechanism explains how a cause produces an effect. I reasoned that if I could identify part of the causal mechanism underpinning either of these effects, then this knowledge could be applied to help develop interventions able to change this mechanism to reduce or prevent antisocial behaviour.

Consistent with past research on the causal mechanisms of putative causal effects in criminology, I used mediation analysis to achieve this. Specifically, I assessed delinquent peer association, beliefs and attitudes supporting peer conflict and antisocial behaviour across four equidistant timepoints spanning an 18-month period and statistically analysed the relationships among antecedent conditions and outcome variables in two simple mediation models with a single mediator. My findings revealed a statistically significant indirect effect of beliefs and attitudes supporting peer conflict on the predictive relationship between delinquent peer association and antisocial behaviour and antisocial behaviour and delinquent peer

association, respectively. Knowing that “the mediation model is a theoretical model implying causality” (Pirlott & MacKinnon, 2016, p. 30), I concluded in my mind that beliefs and attitudes supporting peer conflict partially mediate the peer influence and peer selection effects. I soon realised, however, that this conclusion was premature for the reasons now outlined in this critical appraisal.

Prior to undertaking this research project, my understanding of mediation analysis in the social sciences was based on Baron and Kenny's (1986) seminal paper detailing their causal steps approach. The causal steps approach suggests that true mediation can be established in four steps. Essentially, if the researcher can demonstrate that there is a statistically significant correlation between X and Y, X and M, and M and Y, and a non-significant correlation of zero between X and Y after controlling for M, then the approach holds that the researcher can infer that full mediation has occurred. If there is a significant correlation between X and Y after controlling for M that is close to zero then the researcher might infer the existence of partial mediation. A full description and critique of Baron and Kenny’s (1986) causal steps approach can be found in MacKinnon, Fairchild, & Fritz (2007). The crucial point here is that their paper implies that true mediation can be determined statistically.

To appreciate why true mediation cannot be established statistically, it is necessary to understand the requirements for causal inference. As previously mentioned in this thesis, there are three primary criteria that must be satisfied to infer a cause-effect relationship between two variables: (1)

association (i.e., the cause and effect must covary), (2) temporal precedence (i.e., the cause must precede the effect in time), and (3) non-spuriousness (i.e., the association between the cause and effect must not be produced by the association of both variables with a third variable or set of variables). The gold standard research design for establishing a cause-effect relationship between X and Y is the scientific experiment. In a typical experiment for this purpose, participants are randomly assigned to conditions of X prior to the measurement of Y. A difference in the conditions of X on Y is then interpreted as evidence that X caused a change in Y. This research design can provide compelling evidence about causality as it satisfies the three criteria for causal inference; a main effect of X on Y satisfies the criterion of association, the manipulation of X prior to the measurement of Y satisfies the criterion of temporal precedence, and the random allocation of participants to conditions of X satisfies the criterion of non-spuriousness, since it balances omitted variables between conditions, which “ensures that no pre-existing individual differences between conditions account for the differences between conditions” (Pirlott & Mackinnon, 2016, p. 30).

A simple mediation model, as estimated in my research, comprises three causal effects (i.e., X on M, X on Y, and M on Y). Thereby, the abovementioned conditions that affect casual inferences about a bivariate relationship are also relevant to inferences about cause in mediation models. To argue true mediation, researchers must be able to infer causality of X on M and M on Y. In other words, for each relationship, they need to demonstrate that the casual variable is related to the outcome variable and

that the causal variable preceded the outcome variable. Furthermore, they need to show that the X-M and X-Y relationships are unaffected by other unmeasured variables. This final condition is known as the sequential ignorability assumption. For these reasons, true mediation cannot be established using a purely statistical approach. Whilst inferential statistics are necessary to determine the size and likelihood of direct and indirect effects in a sample, they cannot be used to establish temporal precedence or non-spuriousness because these are fundamentally design issues.

This has important implications for the interpretation of findings from my research. My study employed robust statistical procedures to determine the size and likelihood of direct and indirect effects. Moreover, it used a design that ensured X, M and Y were measured sequentially across six-month intervals. Thereby, it satisfied both the association and temporal precedence conditions for causal inference for both the X-M and M-Y relationships across mediation models. Nevertheless, it also used a non-experimental design. Critically, this meant that it could not prove that the X-M and M-Y relationships were unaffected by other unmeasured variables. In other words, my research violated the sequential ignorability assumption. Consequently, it is not possible to advance causal inferences on the bases of my findings.

As an aside, like most researchers using non-experimental designs to examine mediation, I attempted to bolster the internal validity of my study by exploring whether certain demographic and clinical variables, including the age, sex, socioeconomic status and treatment group of participants, were

related to X, M or Y before conducting mediation analyses. As none of these variables were related to X, M or Y, it was not necessary for me to include them as covariates in mediation models and readers of my research can be confident that none of these variables influenced the X-M and M-Y relationships observed. Nevertheless, future researchers in this area should note that trying to statistically control for all relevant confounds of the X-M and M-Y relationships is not considered an effective way of improving the internal validity of a non-experimental mediation study. This is because it is virtually impossible to be aware of and measure every potential confounding variable for a study and the sequential ignorability assumption cannot be satisfied unless the influence of every potentially confounding variable can be controlled (Stone-Romero & Rosopa, 2008).

At first glance, experimental approaches to mediation appear to offer a solution to the abovementioned difficulties of non-experimental mediation analysis. In general, there are two types of experimental mediation designs typically employed in the social sciences: *measurement-of-mediation* designs and *manipulation-of-mediator* designs (Pirlott & MacKinnon, 2016). The most common measurement-of-mediation design involves the random allocation of participants to conditions of X and sequential measurement of X, M and Y thereafter. Statistical analyses are then performed to establish the likelihood of indirect effects and any between-group differences on Y. Between-group differences in Y across conditions of X are taken as evidence that X caused a change in M which then caused a change in Y.

As applied to the peer selection research question in my study, researchers might randomly assign participants to an intervention group to reduce antisocial behaviour or control group in which antisocial behaviour can vary freely and measure beliefs and attitudes supporting peer conflict after the intervention is completed and delinquent peer association thereafter. Assuming the intervention is effective, if beliefs and attitudes supporting peer conflict mediate the peer influence effect, one might expect participants in the intervention group to exhibit significantly less antisocial behaviour between the end of the intervention and measurement of antisocial behaviour compared to the control group. This type of research design provides convincing evidence of the causal effect of X on M and X on Y; however, like non-experimental mediation designs, it cannot provide evidence of the causal effect of M on Y. Critically, the random assignment of participants to conditions of X satisfies the sequential ignorability assumption for the X-M relationship, but does not permit the elimination of alternative explanations for the M-Y relationship (Bullock, Green, & Ha, 2010; Pirlott & MacKinnon, 2016; Stone-Romero & Rosopa, 2008).

To satisfy both aspects of the sequential ignorability assumption, researchers would need to manipulate both X and M. Experimental mediation designs that involve the manipulation of M are known as manipulation-of-mediator designs. Two manipulation-of-mediator designs that involve manipulation of both M and X and which afford the strongest basis for causal inferences about the X-M and M-Y relationships are referred to as the *double*

randomisation design and *concurrent double randomisation design* (Pirlott & MacKinnon, 2016; Stone-Romero & Rosopa, 2008).

The double randomisation design comprises two experiments. The purpose of the first experiment is to demonstrate that X causes both M and Y and involves the randomisation of participants to conditions of X, whereas the second experiment aims to show that M is a cause of Y and involves the randomisation of participants to conditions of M. Using the double randomisation design to investigate the peer influence research question in my study, in the first experiment researchers might randomly allocate participants to an intervention group to reduce delinquent peer association or control group where delinquent peer association can vary freely and measure beliefs and attitudes supporting peer conflict after the intervention had finished and antisocial behaviour thereafter. The same design would be used for the second experiment, only researchers would randomly assign participants to an intervention group to reduce beliefs and attitudes supporting peer conflict or control group in which beliefs and attitudes supporting peer conflict can vary freely. Measuring X and M after the interventions are complete would also allow researchers to carry out a manipulation check, with the expectation that X and M would be significantly lower in intervention groups relative to control groups. Considering the results of each experiment together, Stone-Romero and Rosopa (2008) posit that the double randomisation design can provide compelling evidence that the X-Y relationship is mediated by M. This is because the design satisfies all three conditions for causal inference of the X-Y and M-Y relationship, if the

manipulation used for the mediator is also unconfounded (Pirlott & Mackinnon, 2016; Stone-Romero and Rosopa, 2008). If participants are randomly selected from the same population the design also permits researchers to use separate groups of participants for the first and second experiment.

The final manipulation-of-mediator design discussed here is the concurrent double randomisation design. In contrast to the double randomisation design, this design involves the simultaneous experimental manipulation of X and M in a factorial experimental design (Pirlott & MacKinnon, 2016). In the simplest version of this design, participants are randomly allocated to one of four experimental conditions reflecting combinations of the manipulated conditions of X and M. Following these manipulations, X, M and Y are measured sequentially. Like the double randomisation design, measuring X and M after the interventions would enable researchers to check the validity of the manipulations.

In answering the peer selection research question in my study, researchers using the concurrent double randomisation design might randomly assign participants to: (1) an intervention group to reduce antisocial behaviour and an intervention group to reduce beliefs and attitudes supporting peer conflict; (2) an intervention group to reduce antisocial behaviour and a control group where attitudes and beliefs supporting peer conflict can vary freely; (3) a control group where antisocial behaviour can vary freely and intervention group to reduce attitudes and beliefs supporting peer conflict; and (4) a control group where antisocial behaviour can vary

freely and a control group where attitudes and beliefs supporting peer conflict can vary freely. Once these interventions are complete, researchers would need to measure participants' level of delinquent peer association and beliefs and attitudes about peer conflict. Following this, they would need to arrange to meet participants again after a set period to measure their participation in antisocial behaviour. In contrast to other experimental mediation designs, a study like this would be analysed using Factorial Analysis of Variance. According to Pirlott & MacKinnon (2016), examining the interaction between manipulated M and X reveals the causal effects of manipulated M on X and Y and measuring the mediator after manipulated M would allow the causal interpretation of the X-M relationship. An interaction effect between manipulated M and X in my proposed hypothetical study would provide good evidence that the effects of antisocial behaviour on delinquent peer association depended to an extent on a person's attitudes and beliefs about peer conflict. Like the double randomisation design, a design like this would satisfy the first two conditions for causal inference of the X-M and M-Y relationship and substantially reduce potential for alternative explanations for these relationships, thereby permitting strong causal inferences regarding true mediation.

Nonetheless, experimental mediation designs are not without limitations. First, it is possible that manipulations of X or M could include unmeasured confounding variables. If this happened, it would not be possible to rule out alternative explanations for these relationships. For this reason, Pirlott and MacKinnon (2016) posit that researchers must "argue

persuasively for why the particular experimental manipulation did not include a covarying confounding variable" (p. 35). Second, the manipulation of M could activate other mediators of the X-Y relationship. In this event, researchers would be forced to conclude that changes in the X-Y relationship might have resulted from a combination of observed and unobserved mediators as opposed to the mediators under study. Indeed, Bullock *et al* (2010) proposes that "those who experimentally manipulate mediators should explain why they believe that each manipulation is affecting only one mediator and not others" (p. 555). Third, Bullock *et al* (2010) highlights the possibility that every person might not be influenced in the same way by manipulations of M, producing misleading inferences about indirect effects. Fourth, experimental mediation designs are often not practical or ethical, especially in the study of antisocial behaviour. Like in my study, many variables of interest in the study of antisocial behaviour are psychosocial in nature and have clinically relevant outcomes. Thus, psychosocial variables, like beliefs and attitudes or delinquent peer association, can be difficult to measure and manipulate and unethical to change.

Delinquent peer association, for example, is particularly challenging to measure accurately. Researchers could attempt to measure it directly, although this would require them to be in the presence of participants' friends most of the time. Even if this was possible, their presence would likely change the nature of the behaviour observed for obvious legal reasons. Self-report measures which ask participants about their friend's involvement in antisocial behaviour offer a solution to this problem. These types of

measures, however, are still highly susceptible to bias since they depend on participants knowledge of and willingness to state their friend's involvement in antisocial behaviour. Furthermore, it is difficult to envisage how researchers might go about manipulating variables like delinquent peer association and beliefs and attitudes supporting peer conflict or determining how strong manipulations would need to be to produce change. Whilst these constructs could be considered dynamic, is seems unlikely that changing one's attitudes and beliefs or behaviour could be achieved in the short-term.

It is also not clear how a researcher might modify the time a person spends with their peers and whether such a manipulation is ethically acceptable given that peer support is a protective factor for mental health (van Harmelen et al., 2016). A way around this might be to focus on protective factors rather than risk factors. For example, instead of trying to reduce delinquent peer association, clinicians might work with participants and their parents to increase their association with peers without a history of antisocial behaviour; although such an approach would still be challenging to implement. In addition, if researchers found a way to reduce antisocial behaviour in an experimental mediation study of the casual mechanisms behind such behaviour, then there would be little motivation to undertake the study in the first place, given that the overall purpose was to improve interventions to reduce antisocial behaviour. The purpose of the study would effectively become tautological. Moreover, excluding participants from accessing a viable and effective intervention for antisocial behaviour to ascertain whether exposure to antisocial behaviour causes stronger attitudes

and beliefs supporting peer conflict would be an ethically-questionable practice.

The above limitations mean that a single experimental mediation study is unlikely to be able to provide convincing evidence of true mediation in the study of antisocial behaviour; although it can bolster evidence for a putative causal mechanism. It is also worth noting that double randomisation and concurrent randomisation designs are incredibly rare in the antisocial behaviour literature, such that I was unable to find an example of one. The lack of experimental mediation studies on antisocial behaviour could be due to some of the challenges raised in this critical appraisal, which are not exhaustive. Even if researchers find a way to use experimental mediation designs to effectively identify causal mechanisms underlying antisocial behaviour, they might do well to consider if the value of such research is worth the considerable time, effort and expense necessary to complete it. Mediation analysis can inform about the causal mechanisms underpinning delinquent peer association and antisocial behaviour, however it could be argued that the reductionist nature of the approach over-simplifies the lived-real experiences of participants. For example, understanding why beliefs and attitudes supporting peer conflict cause people associating with delinquent groups to engage in antisocial behaviour tells us little about the value of holding such beliefs to the individual, which is arguably critical for understanding how to change such behaviour. To achieve a richer understanding of what keeps the causal mechanisms underlying the peer influence and peer selection effects in place, I would consider adopting a

position of methodological pluralism in future studies, which would allow the incorporation of qualitative methods. This seems especially pertinent when considering the current challenges associated with more robust methods for establishing true mediation.

In conclusion, true mediation cannot be established statistically and experimental research is needed to provide credible evidence on the causal mechanisms underlying known risk factor relationships in the criminological literature, like the peer influence and peer selection effects. Consequently, the results of my empirical study cannot be used to make causal inferences regarding the role of antisocial cognition as a causal mechanism behind these effects. Nonetheless, until researchers can find ethically acceptable and practical ways to adapt experimental mediation designs to study antisocial behaviour, they will inevitably continue to rely on correlational designs to understand the causal role of antisocial cognition and other putative causal mechanisms in the antisocial pathway, which are open to multiple interpretations.

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Appendix A: Search Terms for MEDLINE

Textword search terms for *adolescent/adolescence*:

1. Adolescen* or Teen* or Youth* or Minor*

MEDLINE OvidSP subject heading search terms for the above textword terms for adolescent/adolescence:

2. Adolescent (explode) or Psychology, adolescent (explode) or Minors (explode)

Textword search terms for *antisocial cognition/thinking*:

3. Antisocial cognition* or Antisocial thinking or Antisocial attitude* or Antisocial belief* or Antisocial values or Antisocial rationali?ation* or Deviant cognition* or Deviant thinking or Deviant attitude* or Deviant belief* or Deviant values or Deviant rationali?ation* or Offline cognition* or Offline latent cognitive structures

Textword search terms for *antisocial behaviour*:

4. Delinquency or Delinquent behavio?r* or Delinquent activit* or Delinquent conduct or Antisociality or Antisocial behavio?r* or Antisocial activit* or Antisocial conduct or Criminality or Criminal behavio?r* or Criminal activit* or Criminal conduct or Deviancy or Deviant behavio?r* or Deviant activit* or Deviant conduct or Juvenile offending or Externalising behavio?r* or Externalising problems or Conduct problems or Status offence*

MEDLINE OvidSP subject heading search terms for the above textword terms for *antisocial behaviour*:

5. Adolescent Behaviour (explode) or Juvenile Delinquency (explode) or Social Behaviour Disorders (explode) or Conduct Disorder (explode) or Aggression (explode) or Violence or (explode)

There were no MEDLINE OvidSP subject heading search terms *antisocial cognition/*thinking and the search was also limited to humans.

Appendix B: Confirmation of Ethical Approval

South East Research Ethics Committee
South East Coast Strategic Health Authority
Preston Hall
Aylesford
Kent
ME20 7NJ

Telephone: 01622 713097
Facsimile: 01622 885966

20 May 2009

Professor Peter Fonagy
Freud Memorial Professor of Psychoanalysis and Head of the Research Department of Clinical, Educational and Health Psychology, University College London
University College London
Psychoanalysis Unit
1-19 Torrington Place
UCL
WC1E 7HB

Dear Professor Fonagy

Full title of study: **START (Systemic Therapy for At Risk Teens): A National Randomized Controlled Trial to Evaluate Multisystemic Therapy in the UK Context**
REC reference number: **09/H1102/55**

The Research Ethics Committee reviewed the above application at the meeting held on 13 May 2009.

After the Committee's initial deliberations on your application, yourself and Dr Butler kindly joined the meeting to clarify some issues. Thank you for taking the time to do so. The following issues were clarified during the discussion:

Q **Can you deliver this? It is a very intensive process with many contacts with members of families. Do you have enough resources?**

A There is clinical provision in place within the ten established sites. A government grant of £10million has been awarded to this project. All staff have already been recruited for the ten sites. Seven sites have staff employed by NHS agencies and three have staff employed by local authorities. Collaboration was demonstrated in order to gain the funding. All systems necessary have already been developed. The study will be monitored very carefully to ensure intervention is properly delivered.

Q **Has risk assessment taken into account that you may not be able to undertake the project exactly as per the proposal?**

A One of the outcome variables is to expect site-specific differences and this should be the guiding principle of any government national roll-out.

Q **In the power calculation you have allowed for differences in sites, but vulnerable young people come from different sources. There will be a multifaceted group receiving the intervention. Has this been taken into account?**

A The power calculation is based on the success rates of the USA and Norway studies primarily recruited from offender centres. There are no figures to inform the power calculation, although most young people will probably be the same regardless of the service they come from. They will all be diligent rejecters from an early age and we have factored in that they may respond less well. Randomisation has been agreed by the funders.

The Committee were very impressed with the thought that had gone into the study, and the helpful attendance of two of the most senior members of the team; and noted that it was very helpful to have received comments from the study reviewers.

Ethical opinion

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission at NHS sites ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Approved documents

The documents reviewed and approved at the meeting were:

Document	*Version*	*Date*
The Revised Conflict Tactics Scales (CTS2)		
Beliefs and Attitudes Scale		
The Development and Well-Being Assessment - Parent Interview		
The Development and Well-Being Assessment - Interview with 11-17 year olds		
Insurance Certificate		01 July 2008
Participant Consent Form: Young Person	1.1	07 April 2009
Participant Information Sheet: Parent or Carer	1.1	07 April 2009
Participant Information Sheet: Young People aged 15-17	1.1	07 April 2009
Participant Information Sheet: Young People aged 11-14	1.1	07 April 2009
Questionnaire: Strengths and Difficulties Questionnaire		
Questionnaire: The University of New Orleans Alabama Parenting Questionnaire (APQ)		
Questionnaire: Short Mood and Feelings Questionnaire		
Questionnaire: The General Health Questionnaire		
Questionnaire: Young Person's Questionnaire Booklet		
Peer Review		
Letter from Sponsor		04 April 2009

Covering Letter		08 April 2009
Protocol	1.0	30 March 2009
Investigator CV	Professor Peter Fonagy	
Application		07 April 2009
Connors' Teacher Rating Scale - Revised (S)		
ICU (Youth Version)		
ICU (Parent Version)		
LEE scale		
The McMaster Family Assesment Device		
WASI Record Form		
The Child Attachment Interview (CAI) Protocol		
Participant Consent Form: Parent/Carer	1.1	07 April 2009
Participant Consent Form: Optional Additional Qualitative Study - Parent/Carer	1.1	07 April 2009
Participant Information Sheet: Optional Additional Qualitative Study Information for Parents	1.1	07 April 2009
Development and Well-being Assessment (Teacher Version)		

Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

Professor Katona and Dr Bhiman both declared a non-specific, non-personal interest in the study. Members agreed that Professor Katona and Dr Bhiman could remain in the meeting and contribute to the review of the study.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

09/H1102/55	Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely

Dr L. Alan Ruben
Chair

Email: nicki.watts@nhs.net

Enclosures: *List of names and professions of members who were present at the meeting and those who submitted written comments*
"After ethical review – guidance for researchers"

Copy to: *Dr O Awwenagha*

Appendix C: Exclusion Criteria for START study

Exclusion criteria:

- History or current diagnosis of psychosis.
- Generalised learning problems (clinical diagnosis) as indicated by intelligence quotient (IQ) below 65.
- Identified serious risk of injury or harm to a therapist or researcher.
- Presenting issues for which MST has not been empirically validated (i.e., substance abuse in the absence of criminal conduct or sex offending as the sole presenting issue).

Appendix D: Self-Report Delinquency Your Friends subscale

This measure has been removed to avoid copyright infringement.

Appendix E: Self-Report Delinquency Volume of Delinquency subscale

This measure has been removed to avoid copyright infringement.

Appendix F: ABAS Peer Conflict subscale

BELIEFS AND ATTITUDES SCALE				
INSTRUCTIONS: Listed below are statements about people's beliefs and attitudes. Please circle whether you ***AGREE*** **(A)** are ***NOT SURE*** **(NS)** or ***DISAGREE*** **(D)** with each statement.		**Agree**	**Not Sure**	**Disagree**
6	It's fun and exciting to belong to a gang.	A	NS	D
14	Fighting is cool when you're with a group of teenagers.	A	NS	D
20	Blaming other teenagers is a good way to avoid getting into trouble.	A	NS	D
24	It's OK to walk away from a fight.	A	NS	D
27	Being in a gang stops you from getting picked on.	A	NS	D
32	Some young people deserve to be picked on.	A	NS	D
42	Sometimes it's good to carry a weapon to protect yourself.	A	NS	D
47	You have to hurt the other person before he hurts you.	A	NS	D
50	Teenagers feel better when they know they can win a fight.	A	NS	D
65	Fighting is wrong, even when somebody is really bothering you.	A	NS	D