Pathways to Early Mortality for Serious and Violent Young Offenders

by

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B.A., Simon Fraser University, 2017

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in the
School of Criminology
Faculty Arts and Social Sciences

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Abstract

Early mortality is significantly more common among offenders compared to non-offenders but less is known about the specific mechanisms that increase this risk. To address this gap, Tremblay and Paré (2003) specified three pathways to early mortality in offender populations. The general-hazard model suggests that early mortality is driven by low self-control. The occupational-hazard model attributes early mortality to the hazards directly associated with offending. The strain-hazard model emphasizes the prevalence of self-inflicted deaths resulting from differential exposure to strain. Using self-report, official, and social network data from the Incarcerated Serious and Violent Young Offender Study, the current study operationalized and tested these models. Findings from multinomial logistic regression analyses showed no support for the general-hazard model but identified several predictors of early mortality across the strain-hazard and occupational-hazard models, including negative self-identity, parental dysfunction, and youth offending. Social network findings showed partial support for the occupational-hazard model. Specifically, the accumulation of criminal social capital may protect against early mortality outcomes. Recommendations for policy and practice are made with reference to these findings.

**Keywords:** developmental and life course criminology, early mortality, serious and violent young offenders, social network analysis
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# List of Acronyms

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<th>Description</th>
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<tr>
<td>AL</td>
<td>Adolescence limited</td>
</tr>
<tr>
<td>DLC</td>
<td>Developmental and life course</td>
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<tr>
<td>LCP</td>
<td>Life course persistent</td>
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<td>LSC</td>
<td>Low self-control</td>
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<td>SNA</td>
<td>Social network analysis</td>
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Chapter 1.

Introduction

Criminal offending is associated with many negative consequences across the life course (Laub & Sampson, 1988; West & Farrington, 1977). Not only does prolonged conflict with the law directly lead to negative consequences (e.g., incarceration), but the lifestyles of offenders are often characterized by adversity, conflict, and marginalization. Recently, researchers have begun to focus on early mortality as a consequence of offending, showing that early mortality is significantly more common among offenders compared to the general population (e.g., Coffey, Veit, Wolfe, Cini, & Patton, 2003; Daigle & Naud, 2012; Repo-Tiihonen, Virkkunen, & Tiihonen, 2001; Teplin, McClelland, Abram, & Mileusnic, 2005). Despite this, few studies have explored the specific mechanisms that contribute to offender mortality, especially beyond the bivariate level. Additionally, previous research has been limited by small sample sizes, low-risk control groups, and a failure to measure theoretically-informed risk factors associated with early mortality (Piquero, Farrington, Shepherd, & Auty, 2014). As a result, practitioners have been provided with little information as to the risk factors that may help distinguish which specific offenders may be at risk of early mortality.

As result of previous research limitations, there remains a lack of consensus regarding the theoretical explanations for offenders’ differential mortality risk. For example, research from developmental and life course (DLC) criminology found an association between particular offending characteristics and early mortality, suggesting that offending and mortality may be directly linked (e.g., Zane, Welsh, & Zimmerman, 2018). Other studies suggested that mortality is not caused by offending directly, but instead by particular individual and social risk factors that underlie the lifestyles of offenders (e.g., Piquero et al., 2014). Such findings challenge offending-based theories, suggesting that underlying propensities for engaging in riskier behaviours are more helpful in explaining early mortality risk among offenders.

Tremblay and Paré’s (2003) pathway perspective on early mortality bridges the gap between offending-based theories and propensity-based theories. Briefly, Tremblay and Paré specified three pathways to early mortality within offender populations. The
first pathway (or “model”), referred to as the general hazard model, suggests that some offenders may experience higher mortality rates as a consequence of “unhealthy proximal behavior” caused by underlying characteristics such as low self-control (Tremblay & Paré, 2003, p. 300). The second pathway—the occupational hazard model—argues that offenders experience higher mortality rates than non-offenders as result of the hazards directly associated with their offending. Finally, the strain hazard model posits that some offenders, particularly persistent offenders, are differentially exposed to aversive life conditions (i.e., strains) that make early mortality outcomes more likely. Ultimately, Tremblay and Paré’s theory presents a helpful framework for understanding how young offenders may experience early mortality, not exclusively through their involvement in criminal behavior, but also through an array of social and individual risk factors. This theory serves as the theoretical underpinning of the current study.

1.1. Aim of the current study

The current study aims to fill several gaps in the literature on offending and mortality. First, responding to previous researchers’ calls for more theoretically-supported research on the topic (e.g., Nieuwbeerta & Piquero, 2008), the current study uses Tremblay and Paré’s (2003) pathway perspective to examine a variety of theoretically-identified adolescent risk factors and offending characteristics related to early mortality. Second, in response to the lack of research exploring the specific mechanisms related to early mortality within offender populations, the current study explores risk factors for early mortality using a large high-risk sample of serious and violent young offenders. The sample under examination is particularly relevant given that serious and violent offenders commit a disproportionate amount of crime (Loeber & Farrington, 1999), cause substantial costs to the criminal justice system (Easton, Furness, & Brantingham, 2014; Hoddenbagh, Zhang, & McDonald, 2009), and are at high likelihood of experiencing early death (Teplin et al., 2005). Additionally, the sample addresses issues in previous longitudinal research regarding low base rates of individuals that experience early mortality (Piquero, Farrington, & Shepherd, 2014) and provides a unique opportunity to address the “differential rates and types of mortality that would be obscured if comparisons are drawn only between offenders and non-offenders” (Nieuwbeerta & Piquero, 2008, p. 280). Finally, the current study explores the social
networks of deceased offenders using social network analysis (SNA)—a theoretical and analytic tool that has been underutilized in the existing body of literature. SNA was used to more thoroughly operationalize Tremblay and Paré’s occupational-hazard model by capturing specific social dynamics related to offending (e.g., co-offending, conflict, victimization). In doing so, the current study also addresses a key limitation in previous mortality research; that is, traditional variable-oriented studies have typically relied on examining individual-level traits that differentially expose offenders to early mortality, but have often ignored how an individual’s embeddedness within a social environment may contribute to that risk. As such, the use of SNA as a tool to explore early mortality not only serves to better test Tremblay and Paré’s theory, but also contributes to the broader body of literature by examining domains of risk factors that traditional variable-oriented models have neglected.

The main objective of the current study is to empirically examine each of Tremblay and Paré’s (2003) three pathways to early mortality. Although Tremblay and Paré’s theory is widely-discussed in the offender mortality literature, no known studies have empirically tested each of its components together. The current study not only evaluates the empirical validity each pathway independently, but also provides a head-to-head test of which pathway, if any, is most informative of early mortality in a sample of serious and violent young offenders. In doing so, the study aims to answer the following research questions, each of which are informed by Tremblay and Paré’s hypotheses:

1) Are adolescents who exhibit riskier lifestyle patterns also more likely to experience early mortality?
2) Do higher levels of strain in adolescence influence the likelihood of experiencing early mortality?
3) Does a youth’s exposure to conflict and criminal opportunity influence their likelihood of experiencing early mortality?
4) Which of Tremblay and Paré’s three pathways is most strongly related to early mortality among a sample of serious and violent young offenders?

1.1.1. Overview of chapters

This thesis is comprised of eight chapters, including the current introductory chapter. Chapter 2 begins by providing an overview of developmental and life course
criminology’s contribution to early mortality literature, and discusses the debate surrounding general vs. specific theories on early mortality. Next, Tremblay and Paré’s (2003) pathway perspective is explained in detail and key findings from previous studies on adolescent risk factors for early mortality are discussed in reference to Tremblay and Paré’s three models. Chapter 3 provides a brief overview of the fundamental principles of SNA and discusses its relevance to offender mortality research. This chapter is centred on the idea that SNA provides the necessary methodological and theoretical tools to test and extend Tremblay and Paré’s theoretical perspective—particularly the occupational-hazard model. Specific focus in this chapter is given to prior criminological research that has explored peer networks, co-offender networks, and victimization networks. Chapter 4 discusses the methodology of the current study. Chapters 5 and 6 detail the results of the model comparisons and the analysis of the social network data, respectively. Finally, Chapter 7 discusses the main implications of the study’s findings, addresses the study’s limitations, highlights directions for future research, and summarizes the main conclusions and contributions of the study.
Chapter 2.

Offending and early mortality

A body of literature on the relationship between offending and mortality has begun to develop over the last two decades. This research shows that early mortality—regardless of cause—is significantly more common among offenders compared to the general population, and this is true regardless of whether focusing on adolescent offender samples (e.g., Chassin Piquero, Losoya, Mansion, & Schubert, 2013; Coffey, Veit, Wolfe, Cini, & Patton, 2003; Lattimore, Linster, & MacDonald, 1997; Sailas, Feodoroff, Lindberg, Virkkunen, Sund, & Wahlbeck, 2006; Teplin et al., 2005) or adult offender samples (e.g., Daigle & Naud, 2012; Laub & Vaillant, 2000; Nieuwbeerta & Piquero, 2008; Repo-Tiihonen, Virkkunen, & Tiihonen, 2001; Tikkanen, Holi, Lindberg, Tiihonen, & Virkkunen, 2009). Despite the unanimity of this finding, fewer studies have examined the specific mechanisms that contribute to early mortality within offender populations. This has led to debate among scholars regarding the theoretical explanations behind the disproportionate rates of mortality observed across offender populations. Much of this debate centres on whether there are specific “pathways” to early mortality.

The notion of different pathways is deeply rooted in developmental criminology (Loeber & Le Blanc, 1990; Loeber & Hay, 1994). For example, Moffitt’s (1993) dual taxonomy perspective described different pathways to offending for adolescence limited (AL) and life course persistent (LCP) offenders, and this theory has received widespread empirical attention (e.g., Barnes, Beaver, & Boutwell, 2011; Piquero & Brezina, 2006; Sampson & Laub, 2003). The notion of different pathways to early mortality is also plausible, but such assertions have been almost entirely conceptual. Specifically, there is debate in the literature regarding whether offending directly implicates risk for early mortality or whether there exists a set of risk factors responsible for criminal behavior as well as early mortality. Essentially, this debate amounts to whether general or specific theories of early mortality are required. In line with propensity theories like Gottfredson and Hirschi’s (1990) general theory of crime, general theories suggest that offenders have an underlying propensity to engage in riskier lifestyles (e.g., higher substance usage, riskier sexual behaviors), and this contributes to both an increased risk of
offending and an increased risk of early mortality (Gottfredson & Hirschi, 1990; Moffitt et al., 2011; Tremblay & Paré, 2003). On the other hand, specific theories suggest that there is something unique about involvement in criminal behavior that directly increases the likelihood of early mortality. From this perspective, the extent of a person's criminal behavior is a key theoretical risk factor to measure in order to better understand early mortality (Sampson & Lauritsen, 1990; Singer, 1981; Tremblay & Paré, 2003).

The debate between general versus specific theories of early mortality in some ways also resembles debates with developmental and life course (DLC) research regarding the relative importance of state dependence versus population heterogeneity perspectives (see Nagin & Paternoster, 2000). The state dependent perspective suggests that changes occurring outside of the individual, such as strain (Agnew, 1992) or labelling (Akers 1985) help predict continued offending, whereas population heterogeneity perspective proposes that criminal behaviour results from individual propensity effects (e.g., Gottfredson & Hirschi, 1990). However, other researchers such as Moffitt (1993) have argued that these are not mutually exclusive perspectives. Instead, Moffitt argued that AL offenders were motivated by environmental factors (e.g., social immaturity, social learning, and reinforcement) and would only continue to offend in adulthood if they were “ensnared by consequences of antisocial behaviour” (e.g., becoming gang involved, drug-dependent, labelled) (Moffitt, 1993, p. 61). As such, Moffitt viewed AL offenders to be state-dependent and LCP offenders to be explained by an underlying propensity for engaging in criminal behavior (largely stemming from neurological deficits and early childhood adversity).

Similar to Moffitt's (1993) developmental taxonomy perspective, Tremblay and Paré (2003) presented a different perspective on the debate surrounding early mortality that accommodated both general and specific explanations. Tremblay and Paré suggested that there may be multiple pathways to early mortality in offender populations and these pathways were explained both by general and specific risk factors. Specifically, Tremblay and Paré suggested that some offenders may die prematurely as a result of underlying characteristics, whereas others experience early mortality as a result of factors directly, or indirectly, related to offending. Ultimately, this perspective acknowledges the variation in both offending and lifestyle across offender populations, and suggests that focusing on only a single risk factor domain may neglect important differences between offenders.
2.1. Tremblay and Paré’s Pathway Perspective

Tremblay and Paré (2003) presented three explanatory models on early mortality—(1) the general-hazard model, (2) the occupational-hazard model, and (3) the strain-hazard model. Each of these three models explains early mortality outcomes through a different set of risk factor domains, and draws from what may be considered to be competing theoretical perspectives. Specifically, the general hazard model captures the “general” theory perspective that the propensity for risky lifestyles leads to an increased risk of early mortality; the occupational hazard model draws from “specific” theories that support a more direct link between criminality and mortality (e.g., the victim-offender overlap); and the strain-hazard model contains aspects of both general and specific perspectives. In sum, Tremblay and Paré’s theory suggests that focusing on either specific or general explanations for early mortality alone may neglect important predictive risk factors. Instead, a mixed approach is beneficial as it allows for the examination of a broader variety of social, individual, and criminogenic risk factors. To better elucidate this perspective, each of the three pathways is presented in detail below.

The general-hazard model suggests that offenders experience higher mortality rates because of “unhealthy proximal behaviour” rather than because of offending (Tremblay & Paré, 2003, p. 300; also see Laub & Vaillant, 2000, p. 102). Informed by Gottfredson and Hirschi’s (1990) general theory of crime, this pathway predicts that the relationship between offending and early mortality is accounted for by low self-control (LSC). Specifically, individuals with LSC are more likely to engage in riskier activities that promote immediate pleasure regardless of the negative consequences that behavior may bring to the individual or to others (Hirschi & Gottfredson, 1994). Tremblay and Paré (2003) therefore suggested that higher mortality rates in offender populations are not caused by offending directly, but rather are a reflection of the risky underlying personality traits of offenders. For this reason, offenders are expected to be at a higher risk for all types of mortality, and the difference between offenders and non-offenders is best observed in the mortality rate, rather than in the particular types of death that offenders experience. This pathway falls in line with propensity-based theories and is supported, at least in part, by research that shows an association between risky behaviours and early mortality in offender populations.
Tremblay and Paré (2003) specified the occupational-hazard model to outline how the hazards associated with involvement in criminal activity directly influence the likelihood of experiencing mortality. Specifically, high-rate chronic offenders are predicted to experience higher mortality rates as a result of more exposure to conflict (e.g., with victims, co-offenders) across the life course. As offenders progress in their criminal careers and engage in more high-stake criminal activities, the likelihood of experiencing early mortality is predicted to increase as they become “viewed as prime incapacitation targets for other offenders, as well as for victims and hostile third parties” (Tremblay and Paré, 2003, p. 305). This hypothesis may be viewed as an extension of the victim-offender overlap phenomenon, where offenders who engage in riskier, and more chronic, offending patterns are more likely to experience victimization throughout the life course (e.g., Dobrin, 2001; Gottfredson, 1984; Wolfgang, 1958; Wolfgang, Thornberry, & Figlio, 1987).

The final model proposed by Tremblay and Paré (2003) is the strain hazard model. In line with Agnew’s (1992) general strain theory, this model suggests that offenders (particularly persistent offenders) are differentially exposed to aversive life conditions which cause “inner turmoil and misery” (Tremblay and Paré, 2003, p. 320) and ultimately lead to early mortality. This psychological or emotional strain may result directly from continuous involvement in crime (e.g., being in constant competition for resources and opportunities) or may be a product of the aversive social and environmental circumstances that offenders are often subjected to (e.g., social stigma, marginalization, poor living conditions). Other researchers have referred to this pathway as an “indirect causal model” (van de Weijer et al., 2016, p. 92) where offenders are predicted to die prematurely, not as a direct consequence of offending but because of circumstances that result as a consequence of prolonged conflict with the law. Whereas the general hazard model suggests that offenders experience higher mortality rates, regardless of mortality cause, the strain-hazard model emphasizes the prevalence of self-inflicted deaths in offender populations—particularly deaths related to suicide or substance abuse (e.g., overdoses, illnesses caused by excessive substance use).

In sum, Tremblay and Paré’s three theoretical models suggest that it is possible that both general and specific theories are applicable to early mortality. It is possible that some offenders experience early mortality as a result of their propensity for making riskier lifestyle choices (i.e., the general-hazard model) whereas others die prematurely
as a consequence of offending (i.e., the occupational-hazard model), or a combination of both (i.e., the strain-hazard model). As such, Tremblay and Paré’s theory provides a strong guidance to the exploration of early mortality risk as it allows for the examination of a broad range of theoretically-informed risk factors. Although the empirical validity of the theory remains largely untested, elements of each of the three pathways have been examined in prior empirical studies, which provides at least a preliminary understanding of the support for or against each pathway.

The sections below review prior research on offender mortality and are organized according to whether they support the general-hazard, occupational-hazard, or strain-hazard models. This chapter is primarily focused on research that has explored adolescent risk factors for early mortality within offender populations. There are two main reasons for this. First, Tremblay and Paré’s perspective was not intended to be applied to mortality outcomes across all populations. The models were specifically focused on explaining the mechanisms that contribute to mortality among offenders, and for this reason, research from general population samples are not directly applicable. Second, although Tremblay and Paré’s theory was not intended for adolescent offenders specifically, focusing on adolescent risk factors for early mortality is important as it avoids the artificial inflation of early offending that may accompany retrospective data drawn from adult offenders (also referred to as Robins’ paradox; see Robins, 1978). That said, research that draws from adult offending data is reviewed in the context of the occupational-hazard model to explore the hypothesis that mortality increases as a function of the seriousness of offending across the life course.

### 2.2. Research supporting the general-hazard model

The general-hazard model specifies that early mortality in offender populations is caused by risky behaviours that result from higher levels of LSC. As such, research that supports an association between LSC and early mortality while controlling for offending supports this model. However, few studies directly measured and tested the relationship between LSC and early mortality. As such, the current section also reviews research that has explored the association between early mortality and risky behaviours that have been shown in prior research to be associated with LSC.
Lattimore, Linster, and MacDonald (1997) were among the first to explore predictors of early mortality among young offenders. Sampling from two cohorts of serious and chronic adolescent male offenders from the California Youth Authority Project, Lattimore et al. (1997) examined the impact of demographic (e.g., ethnicity, neighbourhood), social (e.g., family upbringing), and criminogenic (e.g., violent arrest) risk factors on early mortality. Homicide was the most common cause of death for all youth in the sample and rates of homicide among the participants far exceeded the (already high) national homicide victimization rates at the time. The authors also discovered that the odds of experiencing homicide were significantly higher for youth that were Black, were living in Los Angeles (an area known for high rates of violent crime and gang activity), and had a history of gang involvement, violence, and drug arrests. Although these findings could be argued to support a direct link between serious offending and early mortality, Lattimore et al. instead viewed this constellation of risk factors as an indicator of an overarching risky lifestyle established early in the life course. Specifically, the authors argued that early mortality risk was a trait that was established prior to a youth’s involvement in the criminal justice system, rather than a risk factor that resulted from prolonged serious offending (Lattimore et al., 1997, p. 206). In other words, the authors suggested that any relationship between offending and mortality was explained by the underlying characteristics of the offenders. However, this assertion was not directly tested in their study, leaving much to question about the relationship between risky lifestyles and early mortality.

Building from Lattimore et al.’s (1997) early exploration of adolescent risk factors for early mortality, Piquero, MacDonald, Dobrin, Daigle, and Cullen (2005) examined the extent to which LSC was related to homicide victimization risk in a sample of parolees released from the California Youth Authority (i.e., the same data used by Lattimore et al., 1997). Controlling for social (e.g., family risk), demographic (e.g., ethnicity), and criminogenic traits (e.g., early onset offending), the authors found that being Black and having higher levels of LSC were predictive of homicide victimization. Additionally, early onset of offending (i.e., youth who experienced an arrest at or prior to age 14) and total number of arrests (while on parole) were not predictive of homicide victimization. Such findings provided partial support for the general-hazard model hypothesis (and Lattimore

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1 The national homicide rate during the follow-up period of the study was between 104-184/100,000, and the estimated homicide victimization rate of the participants in the study was 580/100,000.
et al.’s (1997) hypothesis) that early mortality risk may be related to an underlying propensity for engaging in a risky lifestyle, rather than offending itself.

Farrington, Loeber, Stallings, and Homish’s (2012) study also provided partial support for the general-hazard model. One of the purposes of this study was to predict homicide victimization using a broad set of explanatory and behavioural childhood and adolescent risk factors. Drawing from a sample of approximately 850 adolescent males who participated in the Pittsburgh Youth Study, Farrington et al. found that low academic achievement, low levels of guilt, high levels of hyperactivity, and callous-unemotional traits were significant explanatory predictors of homicide victimization. Additionally, negative behaviour in school (e.g., being suspended, truant) and peer delinquency were found to be significant behavioural predictors of homicide victimization. These findings suggest that homicide victims in the sample may have engaged in riskier behaviors during adolescence and may have had a higher propensity for engaging in those behaviours as a result of particular individual-level traits (e.g., high levels of hyperactivity, low levels of guilt, callous-unemotional traits). Although the authors did not analyze the relationship between offending and homicide victimization while controlling for these risk factors, their findings support the general-hazard model hypothesis that early mortality may not necessarily be a consequence of the offences individuals commit, but rather a reflection of certain underlying personality traits that predispose individuals to having riskier lifestyles (Tremblay and Paré, 2003).

Chassin, Piquero, Losoya, Mansion, and Schubert (2013) addressed an important gap in the early mortality literature by comparing the impact of static (demographic characteristics, personality, historical variables) and dynamic risk factors (factors that were more proximal, temporally, to the individual’s death) in a large sample ($n = 1354$) of adolescent (age 14–19) male and female offenders enrolled in the Pathways to Desistance study. Chassin et al. found that youth in the sample who had a lifetime history of meeting the criteria for a substance use disorder were more likely to experience early mortality. Additionally, current substance use was a significant dynamic predictor of early mortality, suggesting that risky behaviours both past and present have implications for early mortality. Despite this, individual characteristics of the offenders, such as LSC, were not tested in the models, raising the question of whether substance
use behaviours of the deceased youth in the sample were driven by individual characteristics or other environmental factors (e.g., family upbringing).²

Research examining the relationship between offending and mortality into later stages of the life course has also shown support for aspects of the general-hazard model. Laub and Vaillant (2000) extended the Gluecks’ Unravelling Juvenile Delinquency (UJD) study to examine the impact of adolescent and childhood risk factors for early mortality up to age 65. The authors discovered that unhealthy behaviors such as alcohol abuse and poor self-care had a larger impact on early mortality risk than certain adolescent risk factors (e.g., education level, dysfunctional upbringing) and juvenile delinquency. The latter finding is particularly important in terms of supporting the general-hazard model as it suggested that the relationship between offending and mortality may be mediated by the presence of certain lifestyle risk factors. However, the authors also acknowledged that their findings do not address whether those risky behaviours were caused more by underlying individual-level characteristics (e.g., impulsivity) or certain childhood risk factors (e.g., parental maltreatment or abuse) (Laub & Vaillant, 2000, p. 101).

In sum, findings across the aforementioned studies provide partial support for the general-hazard model. Specifically, two studies identified a direct relationship between certain individual characteristics (e.g., hyperactivity, LSC) and early mortality (Farrington et al., 2012; Piquero et al., 2005), and others found that certain risky or unhealthy behaviours predicted early mortality after controlling for offending or delinquency (Chassin et al., 2013; Laub & Vaillant, 2000). Together, these findings may suggest that some offenders experience early mortality, not as a consequence of their offending, but as a result of a higher propensity for engaging in risky behaviours. However, given the limited amount of research that has explored this hypothesis, the specific nature of the relationship between risky lifestyles and early mortality has not been determined with certainty.

² This question is also addressed further in section 2.4 of this thesis
2.3. Research supporting the occupational-hazard model

Although there is partial support for the general-hazard model in the literature, other studies have found a direct link between offending trajectories and early mortality after controlling for childhood and adolescent risk factors. For example, Piquero et al. (2014) tracked a sample of 411 males enrolled in the Cambridge Study in Delinquent Development (CSDD) into their late 50s to examine the extent to which mortality rates differed across offending trajectory groups. Results showed that high-rate chronic offenders were the most likely group to die by the end of the follow up period compared to all other offender groups (i.e., very low-rate chronic, low-adolescence-peak, high-adolescence-peak, high-rate), even after controlling for individual behaviours, personality characteristics, and environmental risk factors. Similarly, drawing from a large sample (n = 4615) of male and female offenders recruited to the Criminal Careers and Life Course Study in the Netherlands, Nieuwbeerta and Piquero (2008) discovered that high-rate persistent offenders were more likely to experience early mortality during the 25-year follow-up period than any other group of offenders in the sample. More recently, Zane, Welsh, and Zimmerman (2018) also found a direct relationship between offending and early mortality in a sample of boys (age 5–13) originally recruited to the Cambridge-Sommerville Youth Study in 1939. Specifically, the authors discovered that LCP offenders died significantly earlier than AL offenders, and that childhood risk factors (e.g., temperament, family dysfunction) were not significant predictors of mortality at any stage of the life course when controlling for offending. However, when examining unnatural deaths alone (as opposed to all-cause mortality), the relationship between LCP offending and mortality was confounded by alcoholism, suggesting that a general-hazard effect may have been present for particular types of mortality in the sample.

In addition to findings of association between offending trajectories and early mortality, other studies have shown an association between early mortality and particular offending characteristics. Kjelsberg and Laake (2010) explored predictors for mortality in a sample of 1112 male and female offenders randomly selected from the Norwegian National Crime Register. Earlier age of first conviction and higher rates of drug related

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3 Offending trajectories were calculated based on a follow-up period where most participants were age 40.

4 However, the authors did not control for other known adolescent and childhood risk factors
crimes were found to be significant predictors of early mortality for both male and female offenders, and higher crime diversity was a significant predictor for male offenders only. Similarly, Farrington et al. (2012) found that homicide victims were more likely to carry guns, use a weapon in the commission of an offence, and to have sold hard drugs compared to non-deceased offenders. Such findings support the occupational-hazard model hypothesis that prolonged engagement in more serious criminal activities may be a risk factor for early mortality, as serious offenders may be more likely to be viewed as a threat by others vying for those same opportunities.

The occupational-hazard model proposes that the risk of experiencing violent mortality increases as offenders engage in more frequent and serious offending across the life course (Tremblay & Paré, 2003). Specifically, as offenders progress throughout their criminal careers, they are more likely to pursue serious criminal opportunities that provide higher-returns, increasing their likelihood of being involved in crime-related disputes (Tremblay & Paré, 2003, p. 308). For this reason, gang affiliation may also be an important predictor of early mortality under the occupational-hazard framework. This claim is supported by a large body of literature that shows that many gangs are structured on a status system, and losses or gains in status among gang members have the potential to escalate into inter-gang disputes under certain micro-level contexts (e.g., Short & Strodtbeck, 1965; Horowitz & Schwartz, 1974). Additionally, youth gang affiliation has been shown to directly increase victimization risk by exposing young offenders to more dangerous situations (DeLisi, Barnes, Beaver, & Gibson, 2009). For this reason, the link between gang affiliation and early mortality may indicate that early mortality is directly attributed to specific types of offending (i.e., occupational-hazard model), rather than an underlying risky lifestyle (i.e., general-hazard model).

Several studies have found a link between gang affiliation and early mortality. As previously mentioned, Lattimore et al. (1997) discovered that youth gang affiliation was a strong predictor of early mortality in the sample. Although the authors interpreted this finding as an indicator of an underlying risky lifestyle, it may also suggest that there is something particular about gang offending that directly increases an offender's mortality risk. Given that Lattimore et al. did not control for individual characteristics of offenders, it is unknown whether the relationship between gang activity and mortality is attributed to individual characteristics or other proximal behaviours. To help answer this question, Chassin et al. (2013) showed that being an active gang member and carrying a gun
were significant dynamic risk factors for early mortality in a sample of serious and violent young offenders, even after controlling for individual and social risk factors such as impulse control, substance use, and parental education level. These findings suggested that as young offenders participate in offences that are more serious and violent in nature, the risk of experiencing mortality may increase as a direct result of those offences.

In sum, several studies found at least partial support for an occupational-hazard model for early mortality. Specifically, studies suggested that prolonged high-rate offending and engaging in riskier types of offences may directly increase an offender's mortality risk. Such findings challenge the risky lifestyle hypothesis posed by previous mortality researchers (e.g., Lattimore et al., 1997), and suggest that offending and mortality may be directly linked. However, in order to make claims about the implications of these findings for Tremblay and Paré’s theory as a whole, research exploring the third and final pathway to early mortality must also be addressed. This pathway—the strain-hazard model—asserts that some offenders may experience early mortality as a consequence of strains caused by both offending and lifestyle.

2.4. Research supporting the strain-hazard model

Identifying research that has directly explored the strain-hazard model poses a unique challenge as indicators of strain may overlap with risk factors captured by the general-hazard and occupational-hazard models. For example, previous research has suggested that some adolescents may engage in certain risky behaviours (e.g., substance abuse) as means of coping with negative emotions caused by strain (e.g., Agnew, 2006; Baron, 2006) whereas other adolescents may engage in those same behaviours as a result of exhibiting higher levels of LSC (e.g., Gottfredson & Hirschi, 1990). Additionally, as outlined by Tremblay and Paré (2003), offenders who engage in higher rates of offending may also experience higher levels of strain as a result of being in constant conflict with the law, and having a lack of meaningful life pursuits. For this reason, the current section reviews research that has examined particular sources of strain—rather than behaviours that may be linked to strain—in childhood or adolescence.
Agnew (1985) suggested that family-related strains were among the most impactful types of strains faced by young offenders, and other researchers have extended this hypothesis, arguing that family-related strains also may be a risk factor for early mortality. Specifically, bivariate findings from Lattimore et al.'s (1997) study showed that offenders who experienced higher levels of caregiver neglect during childhood were more likely to experience early mortality. Additionally, Farrington et al. (2012) found that youth who had a broken family, a family on welfare, and/or a father with behavioural problems were more likely to experience homicide victimization. Finally, van de Weijer, Bijleveld, and Huschek’s (2016) study, which examined 500 male and female youth born to high-risk families in the Netherlands between 1918 and 1959, showed that the relationship between offending and mortality was spurious after accounting for the familial backgrounds of the participants. Specifically, when comparing offenders to a sample of non-offenders from high-risk, low social class families, the researchers found no significant differences between the two groups in early mortality risk; however, both the offenders and high-risk non-offenders were significantly more likely to experience early mortality than members of the general cohort sample (i.e., a lower-risk control group). Although the authors did not directly measure strain in this study, their findings suggest that individuals from high-risk families may be at a higher risk of experiencing early mortality, and this risk is not attributed to their patterns of offending.

Together, the findings across the aforementioned studies support the hypothesis that higher levels of family-related strains in early childhood or adolescence may increase an offender’s risk of experiencing early mortality. However, given the limited amount of research on this topic, the specific nature of this relationship remains inconclusive. Additionally, it is also important to note that other studies have produced contradictory results, showing no association between family dysfunction and early mortality when controlling for offending and other relevant risk factors (e.g., Piquero et al., 2005). Although these findings may be partially attributed to differences in the measurement of family risk (i.e., Piquero et al. created an eight-item index of family risk, rather than examining individual family risk factors), this contradictory evidence still indicates the need for more research on the relationship between family-related strain and early mortality risk.
In addition to family-related strains, individual strains may also be related to early mortality. Using official data from Finland, Sailas et al. (2006) examined risk factors for early mortality among serious young offenders (age 15–21) sentenced to prison between 1984 and 2000. Like most previous studies, young offenders in this sample experienced disproportionately high rates of early mortality compared to the general population (over 11% of the sample was deceased by the end of the follow-up period). However, Sailas et al. (2006) also discovered that young offenders with psychiatric disturbances were more likely to experience early mortality than young offenders without psychiatric issues. Furthermore, the majority of deaths of offenders with psychiatric disturbances were unnatural, violent, and occurred while under the influence of substances. Although this study did not control for other underlying individual or social risk factors, its findings may suggest that the strains caused from prolonged psychological distress may increase the likelihood of experiencing mortality for some offenders.

In addition to strains related to family or social background, several studies have suggested that the ethnic background of offenders may also have implications for early mortality. One finding that appears to be nearly unanimous across studies on early mortality is that Black offenders experience early mortality at significantly higher rates than White offenders (Chassin et al., 2013; Lattimore et al., 1997; Piquero et al., 2005). Most researchers have interpreted this finding from a strain perspective. Extensions of general strain theory have shown that ethnic minorities are exposed to strains that are distinctly different from those experienced by ethnic majorities, and those strains are directly relevant to delinquency (Pérez, Jennings, & Glover, 2008). Similarly, researchers have suggested that ethnicity-specific strains may also increase the risk of experiencing early mortality. For example, Piquero et al. (2005) suggested that disproportionate rates of young Black homicide victims in their sample may have been linked to environmental strains such as neighbourhood disorganization and the lack of legitimate opportunities in those neighbourhoods. Chassin et al. (2013) echoed this perspective by pointing to the “highly segregated residential placement” (p. 693) of Black adolescents in the United States to contextualize the disproportionate rate of Black adolescent homicide victims in their study. Specifically, the authors suggested that living in neighbourhoods with high levels of poverty, unemployment, and residential instability may increase the likelihood of being exposed to violence and adversity throughout the life course. Additionally, Farrington et al. (2012), suggested that race may predict homicide victimization as a
result of racial differences across specific risk factors related to strain in the study, such as having a broken family and being on welfare (p. 153).

Although the research on strain and mortality is limited, findings across the aforementioned studies support the hypothesis that, for some offenders, the cumulative effects of strain may directly increase their likelihood of experiencing early mortality. Particular types of strains that have been identified by previous researchers as being important for early mortality include family-related strains, individual strains, and ethnic-specific strains. Despite this, few studies have explored these sources of strain while controlling for other underlying social or individual risk factors. As such, more research is needed on the relationship between strain and early mortality in offender populations.

2.5. Summary of the evidence for Tremblay and Paré’s theory

Key findings from previous studies on early mortality and offending have shown support for aspects of each of Tremblay and Paré’s three theoretical models. Table 1 provides a summary of the key findings from the studies examined in this chapter and is organized across the three models. As displayed in this table, and discussed throughout the chapter, findings across many studies may be interpreted within the context of multiple models. For example, Lattimore et al.’s (1997) discovery that being gang involved, violent, and living in a crime-ridden area increased mortality risk could be interpreted as symptom of an underlying risky lifestyle (i.e., the general-hazard model), or as an indicator of the occupational hazards associated with more serious types of offending. For this reason, it is difficult to identify which of the three models has received the most empirical support to-date. That said, it appears that the strain-hazard model has received the least direct empirical support.
Table 1. Summary of key research findings across Tremblay and Paré’s three pathways

<table>
<thead>
<tr>
<th>Source</th>
<th>General-hazard model</th>
<th>Occupational-hazard model</th>
<th>Strain-hazard model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chassin et al. (2013)</td>
<td>Current and historic substance use were significant predictors of early mortality</td>
<td>Gang membership and gun carrying were significant predictors of early mortality</td>
<td>Black adolescent offenders were more likely to experience early death—especially by homicide</td>
</tr>
<tr>
<td>Farrington et al. (2012)</td>
<td>Explanatory predictors of homicide: low academic achievement, low guilt, hyperactivity, callous-unemotional traits, Behavioural predictors of homicide: school behavioral problems, peer delinquency</td>
<td>Homicide victims more likely to carry guns, use a weapon in commission of offence, and to have sold hard drugs</td>
<td>Explanatory predictors of homicide: broken family, father behavioural problems, family on welfare, Behavioural predictors of homicide: bad relationship with peers and parents</td>
</tr>
<tr>
<td>Kjelsberg &amp; Laake (2010)</td>
<td>DNT</td>
<td>Earlier age of first conviction, drug crimes, crime diversity were predictors of early mortality</td>
<td>DNT</td>
</tr>
<tr>
<td>Lattimore et al. (1997)</td>
<td>Being gang involved, being violent, and living in LA were predictors of mortality Drug abuse did not predict mortality</td>
<td>Youth gang involvement and drug arrests positively associated with homicide risk</td>
<td>Bivariate correlation between caregiver neglect and all-cause mortality Homicide victims more likely to be Black</td>
</tr>
<tr>
<td>Laub &amp; Vaillant (2000)</td>
<td>Alcohol abuse and poor self-care were predictors of early mortality</td>
<td>Juvenile delinquency not related to early mortality</td>
<td>Dysfunctional upbringing not related to early mortality</td>
</tr>
<tr>
<td>Nieuwbeerta &amp; Piquero (2008)</td>
<td>DNT</td>
<td>High-rate persistent offenders were the most likely group to die by the end of the follow up period</td>
<td>DNT</td>
</tr>
<tr>
<td>Piquero et al. (2005)</td>
<td>LSC predictive of both homicide victimization and violent offending</td>
<td>Early onset of offending and number of arrests were not predictive of homicide victimization</td>
<td>Homicide victims more likely to be Black Family risk not predictive of homicide victimization</td>
</tr>
<tr>
<td>Piquero et al. (2014)</td>
<td>Risky behaviours and characteristics not significantly associated with early mortality</td>
<td>High-rate chronic offenders were the most likely group to die by the end of the follow up period</td>
<td>Family upbringing not significantly associated with early mortality</td>
</tr>
<tr>
<td>Sailas et al. (2006)</td>
<td>DNT</td>
<td>DNT</td>
<td>Young offenders with psychiatric disturbances were more likely to experience early mortality</td>
</tr>
<tr>
<td>van de Weijer et al. (2016)</td>
<td>DNT</td>
<td>No relationship between offending and mortality</td>
<td>Relationship between offending and mortality was spurious after controlling for social backgrounds of offenders</td>
</tr>
<tr>
<td>Zane et al. (2018)</td>
<td>Alcoholism confounded relationship between offending and unnatural mortality Substance use and temperament not predictors of all-cause mortality</td>
<td>An earlier and more persistent pattern of offending was associated with premature mortality in the later stages of the life-course</td>
<td>Dysfunctional family upbringing was not a significant predictor of mortality</td>
</tr>
</tbody>
</table>

Green = findings in support for model; Red = findings against model; DNT = did not test
The challenge of cataloguing research findings across Tremblay and Paré’s three models highlights a larger limitation of the literature on early mortality and offending; that is, the disconnect between the conceptualization and operationalization of Tremblay and Paré’s theory. Tremblay and Paré conceptualized three potential pathways to early mortality, but few studies have appropriately operationalized these models. Previous researchers have referred to this as a problem of “map” versus “terrain” (Cooke, Hart, Logan, & Michie, 2012). Tremblay and Paré provided a description of the “terrain” surrounding early mortality: the concept of distinct pathways. However, it is the role of researchers to “map” this terrain by appropriately capturing the specific pathways that Tremblay and Paré conceptualized. In effect, any comparison of the support for and against each of the pathways (and debating the merits of one versus the other) is insufficient until each model is appropriately operationalized.

The current study takes a step towards this goal by directly operationalizing and comparing each of Tremblay and Paré’s models, taking into consideration the limitations of previous studies. One of the current study’s main contributions in this respect is its inclusion of social network analysis (SNA) as a means of more accurately capturing the occupational-hazard model. Whereas previous studies have relied on official data to explore the relationship between offending and mortality, SNA allows for a more direct examination of whether certain social mechanisms related to offending (e.g., exposure to conflict) are associated with early mortality (as predicted by Tremblay and Paré). As such, the use of SNA in the current study not only allows for the modelling of a key aspect of Tremblay and Paré’s theory, but also has important implications for the broader debate in the literature surrounding general versus specific theories of early mortality.
Chapter 3.

Social networks and early mortality

3.1. Social network analysis: A brief overview

SNA is an interdisciplinary research practice devoted to the study of relationships among interacting units (Wasserman & Fraust, 1994). With respect to criminology (see Bouchard & Malm, 2016), these units typically refer to specific individuals (e.g., gang members, terrorists, white collar criminals) or groups (e.g., gangs, terrorist organizations, companies). Individuals and groups may be connected to one another through one or more types of ties, and those ties may be positive (e.g., friendship, co-offending relationships) or negative (e.g., conflict, victimization). Together, this combination of actors and relations (also referred to as “ties”) is what constitutes a social network (Knoke & Yang, 2008), and at a local level, the total amount of actors (referred to as “alters”) and ties directly connected to a specific actor (or “ego”) is referred to as an “ego-network” (Borgatti, Mehra, Brass, & Labiaca, 2009).

Typically, networks are represented visually through sociograms (Moreno, 1934), and network measures are used to analyze the properties of a network and positions of actors within it. For example, density captures the level of cohesion present in a network, whereas measures of centrality are typically used to capture the positioning or influence of actors within a network (McGloin & Kirk, 2012). These measures also can be used to explore variation across individuals or groups with respect to the ways they are positioned or embedded in their own local social structures or ego-networks (Hanneman & Riddle, 2005). In sum, SNA allows for the measurement and representation of the local and broader social structures in which actors are embedded, and seeks to explain how those social structures impact certain behaviours or outcomes (Knoke & Yang, 2008).

There are several key assumptions that underlie SNA. First, it is assumed that the mapping and analysis of patterned relations among actors typically offers more insight into actors’ behavior than key attributes such as gender, age, or ideology (Knoke
Moreover, it is assumed that the structural mechanisms of social networks (e.g., how cohesive the network is, how information travels through a network) affect the behaviors of the actors embedded within the network (Knoke & Yang, 2008). Additionally, SNA treats social networks as dynamic units that are continually changed by the interactions of the actors embedded within them (Knoke & Yang, 2008). As such, actors within social networks are considered to be interdependent, rather than isolated entities operating in a vacuum (Bouchard & Malm, 2016).

The assumption of interdependence has key implications for traditional theories on criminal behavior that relied solely on individual characteristics to explain offending outcomes (McGloin & Kirk, 2010). Across a wide range of theories like labeling theory, social bond theory, and social learning theory, relationships are critical, but those theories do not expand on the importance of such relationships beyond the individual level (Sierra-Arevalo & Papachristos, 2015). Such theories treat the individual as autonomous as if they are in complete control of their relationships and the extent to which such relationships provide opportunities for crime. Social network perspectives, on the other hand, acknowledge the interdependence between individuals; a single actor’s opportunities for criminal behavior may be constrained or enhanced according to the behavior of others (McGloin & Kirk, 2010).

SNA stems from decades of research traditions (e.g., sociometry, ethnography, survey research, archival analysis) across multiple disciplines (Wellman & Wasserman, 2000, p. 351) and has now established itself as a prominent part of mainstream criminology (see Bouchard & Malm, 2016). SNA is an essential part of criminology as it provides theoretical perspectives and methodological techniques that offer unique ways of building upon traditional methods of measuring and analyzing social influence (Bouchard & Malm, 2016). SNA also offers a unique theoretical perspective on the social mechanisms that influence crime, and as such, has been particularly useful in the study of co-offending relationships, delinquent peer relationships, and the diffusion of victimization across offender networks.

In the context of the current study, SNA provides a useful framework for testing and expanding upon Tremblay and Paré’s occupational hazard model. It provides the necessary analytic tools to measure the nature of offenders’ relationships with other offenders (e.g., co-offending, victimization, conflict), their exposure to more dangerous
individuals (e.g., gang members), as well as their positioning and level of influence in the context of their broader criminal network. Ultimately, the inclusion of social network data and measures allows for a more nuanced evaluation of Tremblay and Paré’s hypothesis that early mortality may increase as a function of the level of conflict present in an offender’s network. Additionally, social network data allow for the exploration of potential protective factors for early mortality related to an offender’s social relationships (e.g., the positive relationships in an offender’s network). To effectively explore these potential associations, three types of relations are of interest to the current study: (1) social (or “peer”) ties, (2) co-offending ties, and (3) victimization and conflict ties. Here, a brief overview of the relevant literature on each of these network ties is presented.

3.2. Peer networks

Although the advancement of social network data collection and analysis has impacted many fields of study, it is argued that peer-effect studies have been one of the most noticeably impacted (Bouchard & Malm, 2016). This is largely due to the fact that theories on delinquency have long suggested that social network dynamics are greatly influential on human behaviour (e.g., Krohn, 1986), but were unable to properly test these hypotheses without the availability of social network data. For example, as mentioned by Bouchard and Malm (2016), differential association theory is rooted in a network perspective but was evaluated solely using proxies for social influence for over 50 years (pp. 3-4). Now, by drawing from social network theory and methodology, researchers have the tools to properly evaluate longstanding theories of crime and delinquency, particularly those focused on the etiological role of peers and social relationships.

Peer effects may have important implications for early mortality risk among offenders. Specifically, findings across previous peer network studies raise the question of whether greater cohesiveness in the networks of young offenders exacerbates or mitigates early mortality risk. A higher level of network cohesion may mitigate early mortality risk by increasing the amount of social support and resources available to a young offender. Specifically, this could protect against strain-related mortality outcomes such as overdose and suicide (i.e., by providing more emotional support and access to external resources) as well as violent victimization (i.e., by providing more peers to defend against violent threats). However, findings across several studies also suggest
that greater cohesion in delinquent peer networks may increase an adolescent’s risk of experiencing early mortality by pressuring them to participate in dangerous activities that they would have otherwise not engaged in. For example, Haynie (2001) examined the association between structural components of adolescent friendship networks and delinquency in a sample of approximately 20,000 students (grades 7–12) enrolled in the Add Health study. She discovered that greater cohesion within peer networks facilitated a common identity among the members of the group, particularly when the group was comprised of delinquent youth. Haynie suggested that greater cohesion in the social network constrained the behaviour of the network’s members, pushing them to act in accordance with the dominant behaviour of the network (Haynie, 2001, p. 1048; also see Haynie, 2002). Similarly, Weerman (2011) examined the effects of peer behaviour on delinquency by drawing from two waves of longitudinal data on 1,156 students who participated in the Netherlands Institute for the Study of Crime and Law Enforcement (NSCR) study. Weerman found that closeness and loyalty were defining features of both delinquent and non-delinquent peer networks, and that some adolescents had a tendency to adapt their behaviour to be congruent with the average level of behaviour in their peer network. Additionally, Baerveldt, van Rossem, and Volker (2008) found that youth across 16 Dutch high schools (n = 859) had a propensity to adopt a similar level of delinquency as their peers in their network. Together, the findings across these studies suggest that adolescents tend to be embedded in cohesive peer networks and may feel pressured to conform to the behavioural norms of that network under certain social and environmental circumstances.

In addition to findings of support for the influence of social networks on peer delinquency, several studies have suggested that peer selection may be a driving force behind patterns of delinquency in peer networks. Specifically, the peer selection hypothesis specifies that youth have tendencies to select friends with a similar propensity for delinquency as themselves (Haynie, Doogan, & Soller, 2014). Knecht, Burk, Weesie, & Steglich (2008) found support for this hypothesis in their peer selection study, which drew from a large sample of youth (n = 3041) across 14 schools in the

5 In this study, social network data were gathered by interviewing each student and asking them to identify their best male and female friends (up to 5 nominations per sex).
6 However, Weerman also stressed that network influence is only a partial explanation for adolescent behavioural adaptation and that individual and social factors should also be considered.
Netherlands. Specifically, the researchers discovered that peer selection played a more significant role than peer influence in explaining similarities in alcohol use in adolescent friendship networks. Similarly, Haynie et al. (2014) found that that both male and female adolescents had a tendency to select friends based on homophilous behavioural traits (e.g., similar level of delinquency), and that those selection effects were particularly strong in female peer networks. From an early mortality perspective, these findings raise the question of whether higher rates of mortality among certain groups of offenders may be attributed to a peer selection process. In other words, just as Haynie et al. (2014) observed homophily in delinquency when examining peer selection, actors in risky social networks may also experience consequence homophily as a result of selecting peers with similar underlying risk factor profiles as themselves. Such questions have not yet been tested from a social network perspective.

3.3. Co-offender networks

Offending is an inherently social "profession" that often relies on co-offending partnerships (Reiss, 1986). However, the processes by which those co-offending relationships are formed, and how those relationships shape the behaviours of offenders, have rarely been examined from a social network perspective (Bouchard & Malm, 2016). This is surprising given that it has long been known that co-offender selection is not a random process (e.g., Tremblay, 1993; Warr, 1996). Tremblay’s (1993) theory on the search for suitable co-offenders argued that offenders must search for co-offenders that balance two competing needs: the need for trust and protection, and the need to advance their criminal career through new opportunities. The former is achieved by working with individuals who are trusted by the offender, whereas the latter is achieved by working with individuals who are less-known to the offender but are specialized in particular skills or connected to advantageous criminal enterprises. If this balancing act in the search for co-offenders does exist, co-offending networks of offenders may include a combination of well-known trusted peers and lesser-known associates. Few studies, however, have tested this theory using social network techniques.

Although research on co-offender networks is limited, there is some support for components of Tremblay’s (1993) theory. Malm, Bouchard, Decorte, Vlaemynck, and Wouters’s (2017) study, which examined the relationship between co-offending networks
and risk perception among cannabis growers, found that cannabis growers felt more protected from law enforcement detection when they were embedded in large cohesive networks with strong ties. Additionally, research on inter- and intra-gang violence has suggested that certain co-offending relationships may be influenced by homophily characteristics such as shared gang affiliation (McCuish, Bouchard, & Corrado, 2015)—a characteristic that also has been suggested to be an indicator of trust in previous network studies (see Weerman, 2003). Additionally, McGloin, Sullivan, Piquero, and Bacon (2008) discovered that the co-offending relationships between young offenders were relatively short-lived and ephemeral, and that young offenders tended not to re-offend with the same individuals. Such findings suggest that cohesiveness and trust are not the only driving forces behind co-offender selection, particularly for young offenders.

Findings from research on co-offending networks raise several key questions with respect to early mortality risk in young offender populations. For similar reasons as were mentioned in the context of peer-effect studies, it is possible that being embedded in more cohesive co-offending networks is a mitigating factor for early mortality risk as it offers offenders more access to support and protection. However, it is also possible that being a member of a large cohesive criminal network may foster a false sense of security that leaves offenders vulnerable to potential acts of victimization—similar to how cannabis growers were found to be less concerned about law enforcement detection when they were embedded in large, cohesive networks (Malm et al., 2017). Additionally, given the ephemeral and volatile nature of some co-offending relationships, particularly in adolescent offender networks (e.g., McGloin et al., 2008), it is possible that co-offending relationships devolve into conflict or victimization over time as certain actors become more desperate for new criminal opportunities. Such questions have not yet been answered in the existing body of literature.

3.4. Victimization and conflict in offender networks

Violence and victimization are not randomly distributed phenomena. Prior research shows that small populations of individuals who chronically engage in crime and delinquency experience the vast majority of victimization and violence (e.g., Kennedy, Piehl, & Braga, 1996; Loeber and Farrington, 2011). Specifically, previous researchers have suggested that violence is reciprocal exchange, as violent offenders will often experience retaliatory victimization as a result of their offending (Singer, 1986).
Furthermore, populations that experience the highest levels of violence are relatively homogeneous across age and ethnicity (Papachristos, Braga, & Hureau, 2012). Such findings are consistent across official data sources in Canada and the United States where young (age 18–24) minority (e.g., Indigenous, Black) males are consistently cited as the most common victims of homicide (David, 2017; Harper, Lynch, Burris, & Smith, 2007). Together, these findings demonstrate that victimization risk is differentially distributed (at least at the aggregate level) across offender populations. However, to understand the individual mortality risk-level of offenders, a more thorough examination of offenders’ social networks is necessary.

Previous social network studies exploring offender victimization demonstrated that the concentration and diffusion of violence is highly influenced by individual and group relationships (e.g., Papachristos, 2009; Papachristos, Braga, Piza, & Grossman, 2015; Papachristos, Wildeman, & Roberto, 2015). This is particularly evident in studies on gang networks where incidents of violent victimization created institutionalized networks of conflict and violence that placed individuals within those networks at a high risk for experiencing victimization, regardless of their individual motives (Papachristos, 2009). Additionally, studies drawing from general offender samples show that both direct and indirect exposure to victims of targeted violence (Papachristos et al., 2015b) and gang members (Papachristos et al., 2015a) significantly increased the odds of being victimized, even after controlling for key demographic variables (e.g., gender, age, gang membership). Together, these findings suggest that the likelihood being victimized is not necessarily dependent on the individual actions or motives of offenders but is instead influenced by who those offenders are exposed to within their social networks. This process is commonly referred to as social contagion and reiterates the importance of using SNA to uncover the group-level processes that expose individuals to risk for early mortality.

The social contagion model (see Burt, 1987) is a theoretical model used to examine processes of social change within social networks. Social contagion refers to the ways in which particular behaviours or phenomena (e.g., imitation, competition, communication) diffuse or transmit across populations (Papachristos et al., 2015b, p. 144). In the context of victimization research, the social contagion model suggests that violent victimization is a product of direct or indirect exposure to victims or other risky actors (e.g., gang members) within a social network. In this sense, violence, much like a
pathogen, is considered contagious to the extent that it travels through social network pathways and infects actors within the network. As such, collecting social network data from high-risk offenders offers a unique opportunity to analyze the diffusion of conflict and violence and identify individuals within offender networks who may be at risk of being victimized.

In the context of the current study, the social contagion model offers a useful theoretical tool to interpret the potential interconnection of deceased participants in a social network. Specifically, the social cohesion model would suggest that higher levels of connectivity between (now) deceased participants may have been one of the driving risk factors behind early mortality. This is particularly relevant to Tremblay and Paré's occupational-hazard and strain-hazard hypotheses. It is possible that greater cohesion in the ego-networks of deceased offenders may indicate a higher occupational-hazard risk, particularly when those offenders are embedded in networks with high levels of gang associations. Actors who have conflicts with victims of gang-related homicide may experience residual blow-back from that victim's death, regardless of whether or not they were directly involved (e.g., Papachristos, 2009; Papachristos et al., 2015a). Additionally, strain-driven deaths such as overdoses and suicides travel pathogenically, either as a result of drug contamination (in the case of drug overdoses) or residual grief (e.g., the death of a close friend or family member driving an individual to risky drug use or suicide). Such instances are unfortunately evidenced in current world events such as the North American opioid epidemic, and the numerous suicide epidemics across many of Canada’s First Nation communities (e.g., Randhawa, 2017). Ultimately, the social contagion model offers a unique perspective that may provide additional insight into the potential diffusion of early mortality across serious and violent young offender networks.

### 3.5. SNA as a tool to explore early mortality

Network findings across peer-effect, co-offender, and victimization studies clearly demonstrate the importance of considering the social context of offenders when examining changes in behaviours or outcomes. For this reason, SNA is also important for exploring differential mortality risk in offender populations. Rather than examining individual-level traits that may predispose an offender to experiencing early mortality, SNA allows for a direct measurement of how an individual’s embeddedness in a particular social environment may contribute to their mortality risk. This approach also
allows for a direct measurement of specific relationships related to offending (e.g., peer relationships, co-offending relationships, conflict and victimization), which is necessary to test Tremblay and Paré’s occupational-hazard hypothesis that exposure to conflict and criminal opportunity influences an offender’s likelihood of experiencing early mortality. More broadly, SNA may be an important methodological and theoretical tool for understanding why certain offenders are disproportionately more likely to experience early mortality.
Chapter 4.

Methodology

4.1. Study Design

The current study draws from data collected for the Incarcerated Serious and Violent Young Offender Study (ISVYOS), the largest and longest-running study on young offenders in Canada. Since its inception in 1998, the study has received four consecutive grants from the Social Sciences and Humanities Research Council (SSHRC). When originally initiated, the study used a cross-sectional design where incarcerated young offenders were interviewed at one time period and were asked to provide retrospective accounts of past life-events and behaviours. In 2012, the project was adapted to include a longitudinal component that involved tracking the criminogenic traits of all youth in the sample into adulthood to examine offending outcomes throughout the life course. Whereas previous study waves involved interviewing participants, the longitudinal component of this project relies on data from official records.

The inclusion of both cross-sectional and longitudinal data is appropriate for the current study as it allows for the examination of adolescent risk factors that may be predictive of early mortality throughout various stages of the life course. As previously noted, previous studies on offender mortality have often relied on cross-sectional data alone (e.g., Teplin et al., 2005), or official data sources that do not include a broad set of theoretically-informed risk factor variables (e.g., Sailas et al., 2006). Such limitations have left much to question regarding the specific mechanisms that are associated with early mortality high-risk in young offender populations. The inclusion of both cross-sectional data and longitudinal data in the current study provides a unique opportunity to address this gap in the literature.

4.2. Sample and procedure

The current study draws from data collected from two cohorts of young offenders who were interviewed for the purposes of the ISVYOS. Permission to conduct the
ISVYOS was granted by both the Ministry of Children and Family Development (MCFD)—the caregiver to all incarcerated youth in BC—as well as by Simon Fraser University’s Ethics Review Board. The main purpose of the ISVYOS is to collect self-report and official data pertaining to risk factors and offending pathways associated with serious and violent offending across the life course. In addition to using self-report and official record data, the current study also extended the ISVYOS by collecting and analyzing social network data on a subsample of participants.

Self-report data were collected via one-on-one interviews with incarcerated youth in closed and open custody centres across British Columbia, Canada. These interviews were standardized and systematic as to promote both quality and consistency of data. Specifically, research assistants (RAs) approached youth while on their custody center unit to invite them to participate in the study. Youth were eligible to participate in the study if they were English-speaking, demonstrated an understanding of the interview questions, and were willing to provide accurate information. Approximately five percent of youth declined to participate. If youth wished to participate, RAs brought them to a private interview room to ensure confidentiality. To obtain assent, participants were read and given a copy of an information sheet explaining the purpose of the study, how information would be collected (e.g. interview and file information), and that all information would be kept confidential unless the participant made a direct threat against themselves or someone else. Participants signed a form signifying that they understood the details of the study and that they could withdraw at any time.

The longitudinal component of the study involved the systematic collection of official criminal history data. These data were collected via the BC Corrections Network (CORNET)—a database used to track all provincial offenders in BC. Specifically, the CORNET database was used to record criminal convictions and custody placements/movements for each participant in the study. This data collection process also allowed for the recording of major life events such as whether or not a participant was deceased.

A description of the total sample is provided in Table 2. The total sample includes 881 serious and violent young offenders, 547 of which were interviewed between 1998 and 2001 during the first wave of the ISVYOS, and 334 of which were interviewed between 2005 and 2011 during the second wave of the study. In Canada, youth who are
incarcerated in youth custody centres must be between the ages of 12 and 17, but under extraordinary circumstances (e.g., to maintain the safety of the young offender), individuals may be allowed to remain in youth custody for several years after. For this reason, members sample were between the ages of 12 and 21 at the time of their first interview ($M = 16.4$). Unlike some previous studies on early mortality (e.g., Lattimore et al., 1997), the sample includes a representation of both male ($n = 698$) and female ($n = 183$) participants. Among all participants with available ethnicity data ($n = 869$), 25.6% self-identified as Indigenous ($n = 225$), 59.2% self-identified as White ($n = 520$), and 14.1% self-identified as a non-Indigenous minority\(^7\) ($n = 124$). As such, the sample reflects the well-known overrepresentation of Indigenous Peoples in the Canadian justice system (Malakieh, 2018).

Table 2 also reflects the high-risk nature of the sample. For example, 34.8% of participants had used drugs before the age of 12, and on average participants had used 1.5 hard drugs (cocaine, crack cocaine, crystal meth, and heroin) by the time of their first interview. Just over half of the sample had been either sexually or physically abused, and most participants (61.9%) had experienced at least one form of parental dysfunction. As previously stated, this sample includes the most serious and violent young offenders in the province at the time, and this is reflected by the average age of first conviction (14.7), average number of youth convictions (11.4), and average time spent in youth custody (10.2 months).

\(^7\) Non-indigenous minority includes all offenders who did not identify as White or as Indigenous. Common examples include South Asian, East Asian, Black
At the time of coding, 63 participants were identified as deceased in the sample, but only 55 had available offending and interview data to analyze. This amounts to 6.2% of the total sample. The average age of death in the sample was 24.6. Details involving the cause of death were available for only 38 cases. These data showed that 18 participants died of homicide, 5 of suicide, 9 of drug-related causes (including overdoses and health problems directly related to substance use), and 6 of other causes (e.g., motor vehicle accidents, cancer). 55.6% \((n = 10)\) of the homicide cases were gang-related, and all homicide victims were male. Whereas homicide was the most common type of known death in the male population, the most common type of death among females was drug-related \((62.3\%, n = 5)\). However, it is important to note that the data on causes of death among participants are likely biased as it may be more likely for the cause of death to be reported in CORNET if it were a homicide, as opposed to other more common deaths such as drug overdoses and suicides. For this reason, it is expected that the remaining 25 unknown deaths were not related to homicide.
4.2.1. Social network data

The current study extends the ISVYOS by collecting social network data on a subsample of participants. Specifically, social network data were collected on all deceased offenders in the sample without a sealed criminal record \((n = 54)\). A control group was then created using the alters within the total deceased offender network. Although alters in the total network of the deceased participants included individuals that were and were not part of the ISVYOS, ethical requirements prohibited the ISVYOS from examining the networks of alters that were not part of the original study. To this point, there was a total of 38 alters in the ego-networks of deceased offenders that were coded as part of the larger ISVYOS network project.\(^8\) Thus, the current study also includes the ego-networks of what were originally solely the alters of the deceased subsample.

There are several caveats for the control sample. First, whereas deceased egos were sampled independently, members of the control group were selected based on their connection to one or more of the deceased egos. As such, this control group must be interpreted within the context of the deceased offender network. Ultimately, the inclusion of these 38 controls helps answer the question of why some offenders in risky social networks do not experience early mortality, despite being directly connected to offenders who do. Secondly, the control group only includes individuals from Cohort II of the ISVYOS, whereas the deceased subsample includes participants from both Cohort I and Cohort II. Typically, when comparing the two cohorts, Cohort I is older than Cohort II; however, because the only persons included in Cohort I in the current study are those that are deceased, there are no significant differences in the length of time that data were available when comparing across subsamples.\(^9\) Secondly, the ISVYOS network project included a high proportion of gang members, and as such, the control sample contains a higher proportion of gang members than would be expected from randomly sampling from the total ISVYOS sample. Although this may dilute potential differences related to gang associations and early mortality in the network, the inclusion of this high-risk control group ensures that other differences in mortality risk between the two groups

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\(^8\) This project includes the ego-networks of 100 offenders who committed offences in Surrey, British Columbia.

\(^9\) Confirmed by running independent samples t-test \((t(79) = 1.16, p = 0.25)\)
are also likely to be observed when comparing deceased offenders to a lower-risk group of offenders.

The criminal and non-criminal associations of the offenders, both in prison and in the community, were collected from CORNET. Prison-related data in CORNET were drawn from custody officer reports, institutional incident reports, case management reports, risk assessments, emails relating to the inmates, and miscellaneous prison documents (e.g., inmate complaint forms, injury forms). Community-related data were taken from probation officer reports, pre-sentencing reports, risk assessments, and practitioner emails. Additionally, CORNET provides an updated list of in-prison contact-concerns (i.e., individuals whom the offender may have conflicts with) as well as court ordered no-contact orders pertaining to both prison and the community. All network data were recorded in “edgelist” format (see Borgatti, Everett, & Johnson, 2013).

A deductive coding process was employed to record the criminal and non-criminal ties in each offender’s ego-network independently. For each instance in which the name of an associate was presented in CORNET, a tie was coded between the offender (ego) and the individual they were connected to (alter). It was also recorded whether the interaction occurred in prison or in the community, and whether the tie was related to co-offending, victimization, conflict, or a social interaction. Co-offending ties included any interaction directly related to the commission of an offence in the community or in prison, including non-criminal offences that result in a charge or official warning in a correctional institution (e.g., tampering with prison cells, inmate intimidation). Victimization ties included interactions in which the offender was either victimized by individual or perpetrated an act of victimization on another individual. This included incidents of verbal victimization (e.g., intimidation, verbal abuse), physical victimization (e.g., assaults), and emotional victimization (e.g., peer manipulation in prison). Although victimization ties were presented as non-directed in the total combined network, directed victimization ties of the deceased sample were also presented separately to contextualize the findings of the current study. Conflict ties were recorded for interactions that were mutually conflictual in nature but did not exhibit a clear direction of victimization. This includes physical and verbal fights, as well as generalized conflict between actors in a network (e.g., conflicts related to gang activity, peer issues

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10 This is necessary as all other network ties included in the total network are also non-directional.
in prison). Social ties—also referred to as “peer” ties—were recorded for all interactions that were not directly related to an offence and were not conflictual in nature (e.g., socializing in the community or in prison). Finally, in all instances where the nature of the interaction between the ego and the alter was unclear or unspecified, the ties were labelled as “unknown.”¹¹ The date and location of each tie was also recorded, along with a brief description of the interaction.

In addition to collecting data on the ties between offenders, attribute data on actors in the network (both egos and alters) were also coded. Information from CORNET was used to identify the gender of the actors in the network, and in cases where the gender of the actor could not be confirmed, their gender was coded as “unknown.” CORNET was also used to record whether actors in the network were known to have past or present gang membership. Data from ISVYOS interviews were then used to supplement gang membership status for all actors in the network who were also participants of the ISVYOS and had answered gang-specific interview questions. Given that neither data source provided a totally comprehensive assessment of gang membership for all actors in the network, gang membership of network actors is best interpreted as “known” vs. “unknown.”

Complying with ethical requirements of the ISVYOS, all social network data were anonymized before extracting them from the secure servers they were collected on.¹² A key component of the anonymization process involved identifying whether an individual in one ego-network was the same individual as in another ego-network. Actors in multiple ego-networks with the same first and last name, or the same correctional service identification number, were coded as a single actor in the total combined network. In instances where only a single name was provided, the date and the location of the interactions were used to verify the actor’s identity. In any instance where it was not definitive whether actors were the same individual or different individuals, they were coded as separate actors in the network. As a result of this conservative coding approach, the data likely underestimate the number of mutual ties among the egos.

¹¹ Unknown ties were subsequently excluded from analyses on the final network

¹² All data were collected and stored on computers at a secure custody centre in British Columbia
Several additional coding decisions are important to note in the current study. Given that the purpose of the study was to examine risk factors for early mortality related to the social networks of offenders, ties to family members were not included in the network unless the tie between the ego and that family member was related to co-offending, victimization, or conflict. In other words, regular familial relationships were not of interest to the current study. The inclusion of such ties would only dilute the network as social interactions with family members are recorded in CORNET more frequently than interactions with non-family members in the community. For similar reasons, connections to the romantic partners of the offenders were not included in the network unless the tie connecting them to the ego was related to co-offending, victimization, or serious conflict (e.g., domestic fight that resulted in police intervention). 

4.3. Measures

A combination of self-report data, official data, and social network data were used to capture Tremblay and Paré’s (2003) general-hazard, the strain-hazard, and occupational-hazard models. Specifically, self-report and official data were first used to identify specific risk factors and demographic characteristics associated with early mortality in each of the three pathways. Social network data were then used to expand on Tremblay and Paré’s perspective with particular focus on the occupational hazard model. Given that the network dataset represents a subsample of the main sample examined in the current study, the network data were analyzed separately from the self-report and official record data. Additionally, as a result of differences between the interview questionnaires across the two cohorts, only cases where self-report data were available on cross-cohort variables were included in the current study. Cases with sealed criminal histories were also excluded as they provided no offending data to analyze.

4.3.1. Mortality

The current study used recordings in CORNET to identify deceased participants in the sample. Specifically, the code (“DE”) is used on an offender’s criminal history sheet to denote when they are deceased. To determine which of the participants were

13 Minor conflicts/arguments were not included in the network
deceased, the criminal history sheets of all offenders without sealed criminal records were checked systematically. This process resulted in the identification of 63 deceased participants; however, for eight of the deceased participants, interview data were not available, typically due to an incomplete interview. These participants were excluded when examining Tremblay and Paré’s (2003) pathway model. At the time of data collection, participants in Cohort I were followed up with until age 34-39 (depending on birth year) and participants in Cohort II were followed up with until age 26-31.

Recognizing that non-deceased offenders are not a homogenous group, the control sample was divided into two groups: (1) those who incurred a conviction for a violent offense as a youth (i.e., between the ages of 12–17) \((n = 520)\) and (2) those who had never incurred a conviction for a violent offense as a youth \((n = 306)\). The age of 17 was used as a cut-off point to eliminate any potential overlap with the adult criminal justice system and to ensure that all risk factors included in the model were measured during adolescence. A violent offense was defined as any criminal offense where hands-on violence was used or a weapon was presented, but not necessarily used, to harm the victim. The decision to separate non-deceased offenders into violent and non-violent subgroups was made for several reasons. First, previous research has demonstrated that violent offenders exhibit a higher likelihood of being victimized than non-violent offenders (Lattimore et al., 1997). Given the high prevalence of violent deaths in the sample (28.6% of deceased cases were a result of homicide), it was of interest to examine whether the deceased offenders differed from the higher-risk (i.e., violent) offenders in any of the pathways tested. Second, other researchers noted that the highest-rate and most serious offenders exhibit “a wide range of negative life outcomes” and “tend to sort themselves into high-risk situations” (Nieuwbeerta and Piquero, 2008, p. 280; also see Forde and Kennedy, 1997). Given that an aim of the study is to explore potential differences in lifestyle risk factors that contribute to early mortality, separating violent offenders from non-violent offenders provides a better opportunity to explore the nuances between offending and mortality.

4.3.2. General-hazard model

Although a direct measurement of LSC was not available from the current data, the general-hazard model was captured by measuring four indicators of a risky lifestyle that have been shown to be associated with LSC in previous research (e.g., Baron,
2003; Piquero et al., 2005). Hard drug versatility was measured on a four-item scale based on participants’ self-reporting of having ever used cocaine, crack-cocaine, heroin, and crystal meth. It was decided to measure hard drug versatility, as opposed to general substance use versatility because “softer drugs” (e.g., cannabis) and alcohol are used more normatively in the general youth population.\textsuperscript{14} Drug use and skipping school were considered to have an early onset if they occurred before the age of 12. Twelve was set as the cut-point to establish temporal order between the risk factor and involvement in the youth criminal justice system.\textsuperscript{15} The age of 13 was used as the cut-off point for early sexual activity due to a low base-rate of youth engaging in sexual activity before the age of 12.

### 4.3.3. Strain-hazard model

To test whether external and internal sources of strain help explain early mortality risk in offender populations, four measures of strain were examined. To eliminate any potential overlap with the other two models, only explanatory (as opposed to behavioural) risk factors were included. Family-related strain was captured by measuring whether participants reported being kicked out of their home before the age of 13,\textsuperscript{16} as well as by the level of parental dysfunction reported by the youth. Parental dysfunction was measured on a scale of six indicators, specifying whether a biological parent of the participant (i.e., mother, father) had exhibited (1) a history of alcohol abuse, (2) a history of drug abuse, (3) physical abuse, (4) sexual abuse, (5) mental illness, and (6) a criminal record. Individual strain was captured via a dichotomous indicator of self-reported physical or sexual abuse, as well as by the level of negative self-identity reported by the youth. Negative self-identity was measured using Schneider’s (1990) Good Citizen Scale: a 15-item scale ($\alpha = .72$) that assesses subjects’ perceptions of themselves. Examples of items included on this scale include the level to which a youth views

\textsuperscript{14} Health Canada (2017) statistics show that 26.9% of youth age 15-19 reported a lifetime history of cannabis use and 64.2% reported using alcohol in their lifetime (Health Canada, 2017). Comparatively, only 2.6% of youth reported ever using cocaine/crack cocaine and only 1.0% reported ever using crystal meth.

\textsuperscript{15} In Canada, youth cannot be held criminally responsible before the age of 12.

\textsuperscript{16} Similar to early onset sexual activity, the age of 13 was used as result of a low base rate at age 12.
themselves as polite vs. rude, weak vs. tough, attractive vs. unattractive. Higher scores on this scale indicate higher levels of negative self-identity.

### 4.3.4. Occupational-hazard model

Three measures were included to test the occupational-hazard model. To account for the level of exposure to criminal opportunity within the youth criminal justice system, the total number of youth convictions (i.e., convictions between the ages of 12-17) and the total number of months spent in prison during adolescence were measured. These data were gathered from official criminal history records logged in CORNET. The purpose of including these data were to test whether a participant’s likelihood of experiencing mortality increased as a function of a greater degree of involvement in offending. Additionally, based on previous research that showed an association between earlier onset of offending and early mortality (e.g., Kjelsberg & Laake, 2010) the age of a youth’s first conviction was recorded. To test the hypothesis that more exposure to conflict increases early mortality risk, self-report data were used to measure whether the youth had engaged in physical fights on a weekly basis. However, self-reported fighting data only reflected a subject’s fighting profile at one point in their adolescence, and for this reason, social network measures were also used to more accurately capture a youth’s exposure to conflict across the life course.

### 4.3.5. Social network measures

Most previous studies have explored the relationship between offending and mortality by examining differences in offending trajectories captured via official data sources. Although these studies provide important information regarding differences in the offending profiles of deceased and non-deceased offenders, they do not address a key component of Tremblay and Paré’s (2003) occupational-hazard model. Tremblay and Paré hypothesized that as offenders progress in their criminal careers, they are more likely to be exposed to conflict and victimization and may die prematurely as a direct consequence of that heightened exposure. Although variable-oriented approaches may highlight criminogenic risk factors associated with mortality, they do not account for how an offender’s embeddedness within a particular social environment may contribute to early mortality. SNA provides the necessary theoretical and analytical tools to capture
the social dynamics related to offending and examine whether certain types of relationships may impact an offender’s risk of experiencing early mortality.

Two measures of centrality—degree centrality and betweenness centrality—were used to test whether an actor’s level of influence in the network was associated with their mortality risk. In non-directed networks, degree centrality measures the total number of immediate connections surrounding an actor in the network. In directed networks, in-degree measures the number of incoming ties (i.e., ties initiated by other actors in the network) and out-degree measures the number of outgoing ties (i.e., ties initiated by the actor being observed). Participants with higher degree centrality are more visible in the network and are directly connected to a higher number of other actors. As such, comparisons in degree centrality test whether higher levels of connectivity, or visibility, in risky social networks increase an actor’s mortality risk. Betweenness centrality is a measure of brokerage in that it measures an actor’s ability to connect previously unconnected actors. Specifically, betweenness centrality measures the “degree to which a point falls on the shortest path between others” in a network (Freeman, 1977, p. 35). In the context of the occupational-hazard model, betweenness centrality is an important measure of influence as it tests whether higher levels of brokerage increase an actor’s mortality risk (i.e., by getting caught in the middle of offender disputes), or protects against mortality by increasing the amount of information and resources available to the offender. Both betweenness centrality and degree centrality have been commonly used to detect key players in social networks in previous social network studies (e.g., Borgatti, 2006; Hashimi & Bouchard, 2017; Morselli, 2010).

In addition to examining the level of influence of the egos in the network, several measures of cohesion were also included to examine differences across the ego-networks of the offenders. Specifically, three measures of cohesion were included to test whether more cohesive ego-networks protect against early mortality (e.g., by increasing the level of trust and communication in the network) or increase mortality risk (e.g., by increasing the risk of vicarious victimization or conflict exposure). The first measure, effective size, captures the number of non-redundant ties in an offender’s ego-network. Specifically, effective size measures the difference between an ego’s degree and the average degree of the alters in their network. Higher effective size reflects a higher number of “structural holes” in the network (Burt, 1992; Malm, Bouchard, Decorte, Vlaemynck, & Wouters, 2017). This means that ego’s with higher effective size have
more control over the flow of communication in their ego-network, as their network is less cohesive. Consequently, lower effective size indicates a more cohesive ego-network. Average nodal degree reflects a ratio of the sum of ties between the ego and all alters in the ego-network network to the total number of alters in their ego-network. Higher average nodal degree indicates stronger ties (positive or negative) between the ego and the alters in the network. Finally, density was calculated by summing the number of present ties in an ego-network and dividing the sum by the total number of possible ties in that ego-network. The total number of possible ties (PT) was calculated as follows:

\[
PT = \frac{n(n-1)}{2}
\]

where \( n \) is the number of nodes in the ego-network.

Finally, to test Tremblay and Paré’s hypothesis that higher levels of conflict increase early mortality risk, the nature of ties between actors in the network was examined. Specifically, two categories of ties were measured: negative and positive criminal social capital ties. Negative ties were captured by summing the total number of conflict ties and victimization ties in each ego’s immediate network. As such, a higher number of negative ties indicates a higher total exposure to conflict and victimization. Positive criminal social capital was measured by summing the co-offending ties and social prison ties in an offender’s network. This measure provides a count of the number of positive associations with other offenders in the network. Additionally, to control for network size, the proportion of negative ties to all ties in an offender’s ego-network was measured. Finally, the proportion of gang members to all alters in each offender’s ego-network was measured to test the hypothesis that offenders are at a higher risk for experiencing early mortality when they are connected to riskier actors. This hypothesis has been supported by previous social network research which suggests that exposure to gang members in a social network may increase the likelihood of being violently victimized, regardless of an actor’s individual motives (e.g., Papachristos, 2009).

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17 Only ties directly connecting the ego to an alter were measured. Ties between alters were not included.

18 Social community ties were excluded from this measure to ensure that only ties with other offenders were captured. (i.e., some community social ties may not be related to offending)
4.4. Analytic strategy

4.4.1. Self-report and official data

The self-report and official data were first analyzed to explore risk factors for early mortality related to each of Tremblay and Paré’s three pathways. The first step of the analyses involved addressing missing data using the multiple imputation chained procedure for Stata (StataCorp, 2013). Using all independent and control variables in the analyses, ten imputations were conducted for each item, and the average imputed values were reported for each case with missing data. After missing data were addressed, bivariate analyses were conducted to assess whether the deceased group differed from either of the non-deceased groups on any of the measures used to capture the three different pathways. Subsequently, three multinomial logistic regression (MLR) models were produced, each testing one of Tremblay and Paré’s three pathways individually. These models included the same items tested at the bivariate level while controlling for gender and ethnicity. A final MLR model was produced using the strongest bivariate predictors of early mortality across all three pathways. This model ultimately provided a comparison of the relative importance of variables from different pathways. Finally, to test the robustness of the results, a sensitivity analysis was conducted by testing each of the aforementioned models in a logistic regression with early mortality as the outcome of interest. In these models, a binary measure of adolescent violence (1 = incurred violent conviction as youth; 0 = no violent conviction as youth) was included as an additional measure of control. All analyses were conducted using Statistical Package for the Social Sciences (SPSS).

4.4.2. Social network data

Following the analysis of the self-report and official data, SNA was used to more appropriately capture components of the occupational-hazard model and supplement findings from traditional variable-oriented approaches to studying risk factors for early mortality. The first step in the analysis involved conducting a components analysis of the total combined network of the deceased and control egos. The purpose of this analysis was to identify and subsequently isolate the main component of the network. Isolating the main component was important given the sampling criteria for the control group. Specifically, whereas the deceased offenders were sampled irrespective of their
connection to any other offenders, participants of the control group were selected based on their connection to one or more deceased egos. As a result of this sampling criteria, the control group had a higher likelihood of being included in the main component\(^{19}\) (i.e., because the network sizes of the deceased egos in the main component were larger than the deceased egos not included in the main component). For this reason, comparisons between deceased and non-deceased egos in the full network (i.e., all components) may be biased in favor of the control group. Isolating the main component reduces this bias and ensures that the deceased and non-deceased egos are drawn from the same population.

After the components analysis was conducted, the main component was examined at the descriptive level. Next, bivariate analyses were conducted to compare the deceased and non-deceased egos included in the main component. The network influence of the egos was compared by examining differences in degree centrality and betweenness centrality of deceased and non-deceased participants. To compare the level of cohesiveness across the groups, the effective size, density, and average nodal degree were used. To test whether particular types of ties are associated with early mortality in the network, the average number of criminal social capital ties, negative ties, and proportion of negative ties to all known ties in an offender’s ego-network were compared across deceased and non-deceased groups. Finally, the number and proportion of gang members in the ego-networks of offenders were compared to assess whether exposure to riskier actors within a local social structure was associated with early mortality risk. As an additional measure of control, analyses were conducted using both age-adjusted and non-age-adjusted scores, and controlling for age did not result in major changes to the results. For ease of interpretation, all results displayed in Chapter 6 include non-age-adjusted scores, but age-adjusted scores are presented in Appendix A for interested readers.

After comparing offenders in the main component of the total network, the directed victimization network was examined separately. The purpose of examining the victimization network separate from the total network was to test whether patterns in the direction of victimization among offenders were associated with mortality risk. For the same reasons as previously noted, a components analysis was first conducted to isolate

\(^{19}\) Confirmed via chi-square analysis ($\chi^2 = 15.75, p < .001$)
the main component of the network. Subsequently, bivariate analyses were conducted to compare deceased and non-deceased participants with respect to the level of incoming victimization (in-degree), outgoing victimization (out-degree), and proportion of outgoing victimization in their respective ego-networks. Betweenness centrality was also examined to test the extent to which an actor was “caught in the middle” of offender disputes. Cohesion was compared by examining the effective size, density, and average nodal degree of deceased and non-deceased (victimization) ego-networks. Finally, the number of, and proportion of, gang members in each participant’s ego-network was compared to assess whether conflicts with riskier actors impact mortality risk. All network analyses were conducted using UCINET 6 (Borgatti, Everett, & Freeman, 2002) and sociograms were produced using Netdraw (Borgatti, 2002).
Chapter 5.

Results: Examining and comparing the three pathways

5.1. Comparison of deceased and non-deceased offenders

Bivariate analyses were conducted to identify potential differences between the deceased and non-deceased groups. Findings from these analyses are displayed in Table 3. Consistent with the total sample, the deceased sample was predominantly male (80.0%, \(n = 44\)) and the gender composition of the three groups did not differ. There was a significant difference in ethnicity across the three groups. Post hoc testing\(^{20}\) revealed that the violent group was comprised of significantly fewer Indigenous participants than the deceased group and non-violent group. In addition to differences in the demographic characteristics of the three groups, risk factors associated with the pathways also differed between groups. Although the deceased group did not significantly differ on any of the general-hazard items tested, non-violent offenders used a significantly higher number of hard drugs in adolescence (\(M = 1.7, SD = 1.4\)) than the violent offenders in the sample (\(M = 1.4, SD = 1.4\)). With respect to the strain items tested, the deceased offenders and the violent offenders exhibited significantly higher levels of negative self-identity than the non-violent offenders. Significant differences were also observed across groups for the risk factors used to operationalize the occupational-hazard model. On average, deceased offenders and violent offenders exhibited a significantly earlier age of conviction than non-violent offenders. Similarly, deceased and violent offenders had a significantly higher number of convictions during adolescence and spent significantly more time in youth custody than the non-violent offenders. These findings were further investigated by examining the differences in means over each year in adolescence. As displayed in Figure 1, the significant difference in convictions between deceased and non-violent offenders occurred at age 15. Additionally, deceased youth spent significantly more time in custody than non-violent youth between the ages of 15 and 17.

\(^{20}\) Post hoc tests were conducted via cellwise residual analysis using the Bonferroni correction (see Garcia-Perez & Nunez-Anton, 2003).
Figure 1. Comparison of youth offending measures by year
### Table 3. Bivariate comparison of groups

<table>
<thead>
<tr>
<th></th>
<th>Deceased</th>
<th>Non-Deceased (Non-Violent)</th>
<th>Non-Deceased (Violent)</th>
<th>(\chi^2/F, p, \Phi/\eta^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender (male = 1)</strong></td>
<td>80.0 (44)</td>
<td>79.0 (241)</td>
<td>79.3 (413)</td>
<td>.03, ns, .01</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td>9.85, p &lt; .05, .11</td>
</tr>
<tr>
<td>Indigenous</td>
<td>32.7 (18)</td>
<td>30.5 (92)</td>
<td>22.5 (116)</td>
<td></td>
</tr>
<tr>
<td>Non-Indigenous minority</td>
<td>16.4 (9)</td>
<td>11.3 (34)</td>
<td>15.9 (82)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>50.9 (28)</td>
<td>58.1 (176)</td>
<td>61.6 (317)</td>
<td></td>
</tr>
<tr>
<td><strong>General-hazard pathway</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early drug use</td>
<td>56.4 (31)</td>
<td>68.5 (209)</td>
<td>64.1 (334)</td>
<td>3.65, ns, .06</td>
</tr>
<tr>
<td>Skip school early</td>
<td>80.0 (44)</td>
<td>81.0 (247)</td>
<td>79.7 (415)</td>
<td>.21, ns, .02</td>
</tr>
<tr>
<td>Early sexual activity</td>
<td>74.5 (41)</td>
<td>76.4 (233)</td>
<td>73.1 (381)</td>
<td>1.08, ns, .04</td>
</tr>
<tr>
<td>Hard drug versatility</td>
<td>1.7 [1.5]</td>
<td>1.7 [1.4]c</td>
<td>1.4 [1.4]b</td>
<td>4.63, p &lt; .01, .01</td>
</tr>
<tr>
<td><strong>Strain-hazard pathway</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kicked out of home early</td>
<td>9.1 (5)</td>
<td>12.1 (37)</td>
<td>12.7 (66)</td>
<td>.60, ns, .03</td>
</tr>
<tr>
<td>Abuse</td>
<td>60.0 (33)</td>
<td>48.5 (148)</td>
<td>51.1 (266)</td>
<td>2.50, ns, .05</td>
</tr>
<tr>
<td>Parental dysfunction</td>
<td>1.6 [1.4]</td>
<td>1.2 [1.3]</td>
<td>1.3 [1.3]</td>
<td>2.76, ns, .01</td>
</tr>
<tr>
<td>Negative self-identity</td>
<td>50.2 [8.8]b</td>
<td>46.8 [9.6]a</td>
<td>48.6 [9.0]b</td>
<td>5.33, p &lt; .01, .01</td>
</tr>
<tr>
<td><strong>Occupational-hazard pathway</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weekly fighting</td>
<td>20 (11)</td>
<td>23.6 (72)</td>
<td>22.6 (118)</td>
<td>.36, ns, .02</td>
</tr>
<tr>
<td>Age of first conviction(^\dagger)</td>
<td>14.7 [1.4]b</td>
<td>15.4 [1.7]a,c</td>
<td>14.3 [1.4]b</td>
<td>49.85, p &lt; .001, .10</td>
</tr>
<tr>
<td>Youth convictions (total)(^\dagger)</td>
<td>13.3 [9.8]b</td>
<td>8.5 [7.8]a,c</td>
<td>12.8 [8.7]b</td>
<td>26.46, p &lt; .001, .06</td>
</tr>
<tr>
<td>Youth custody time (months)(^\dagger)</td>
<td>12.0 [9.9]b</td>
<td>6.6 [7.3]a,c</td>
<td>12.0 [9.9]b</td>
<td>36.31, p &lt; .001, .08</td>
</tr>
</tbody>
</table>

*ns = not significant at .05 level

\(^{\dagger}\)Levene’s test violated. Dunnet T3 used
\(^a\)Indicates significantly different from Deceased.
\(^b\)Indicates significantly different from Non-Deceased (Non-Violent).
\(^c\)Indicates significantly different from Non-Deceased (Violent).
5.2. Multivariate models

Next, the risk factors associated with Tremblay and Paré’s pathway theory were examined in a series of multinomial logistic regression (MLR) models. All risk factors examined at the bivariate level were included in the multivariate models, regardless of their bivariate significance, to examine the potential impact of each item while controlling for relevant demographic characteristics. The deceased group was selected as the reference group for all multivariate models to identify potential differences between the deceased and both non-deceased groups. After operationalizing and testing each model separately, the strongest bivariate predictors of each model were included in a final model to test which of the three pathways was most strongly predictive of early mortality in the sample. Findings for each MLR model are found in Table 4.

The first MLR model tests the general hazard items (early onset drug use, early onset skipping school, early onset sexual activity, hard drug versatility) while controlling for ethnicity and gender. The model was significant ($\chi^2(14) = 27.15, p < 0.05$) and was an adequate fit for the data. However, consistent with the bivariate findings, there were no significant predictors of early mortality in this model.

The second MLR model tested the strain items while controlling for gender and ethnicity. The model was significant ($\chi^2(14) = 31.19, p < 0.01$) and provided an adequate fit for the data. Consistent with findings at the bivariate level, negative self-identity remained a significant predictor of early mortality at the multivariate level. Specifically, a one unit increase in negative self-identity decreased the odds of being a non-violent offender compared to being a deceased offender by approximately 4% (OR = 0.96, $p < 0.05$). Unlike findings at the bivariate level, however, parental dysfunction emerged as significant predictor of early mortality after controlling for other sources of strain and demographic characteristics. The odds of being a non-violent offender compared to a deceased offender decreased by approximately 23% with a one unit increase in parental dysfunction (OR = 0.77, $p < 0.05$).
Table 4. Multinomial logistic regression models predicting early mortality

<table>
<thead>
<tr>
<th></th>
<th>General-hazard model</th>
<th>Strain-hazard model</th>
<th>Occupational-hazard model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-Violent OR (95% CI)</td>
<td>Violent OR (95% CI)</td>
<td>Non-Violent OR (95% CI)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>.82 (.43–1.57)</td>
<td>0.57 (0.30–1.06)</td>
<td>.90 (.47–1.75)</td>
</tr>
<tr>
<td>Non-Indigenous minority</td>
<td>.56 (.24–1.33)</td>
<td>.73 (.33–1.65)</td>
<td>.49 (.21–1.16)</td>
</tr>
<tr>
<td>Male</td>
<td>1.05 (.49–2.22)</td>
<td>0.81 (0.42–1.78)</td>
<td>.76 (.38–1.71)</td>
</tr>
<tr>
<td>General-hazard</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early drug use</td>
<td>.54 (.28–1.03)</td>
<td>.72 (0.38–1.36)</td>
<td>—</td>
</tr>
<tr>
<td>Skip school early</td>
<td>1.11 (.51–2.39)</td>
<td>1.16 (0.55–2.44)</td>
<td>—</td>
</tr>
<tr>
<td>Early sexual activity</td>
<td>1.06 (.51–2.19)</td>
<td>1.28 (0.64–2.58)</td>
<td>—</td>
</tr>
<tr>
<td>Hard drug versatility</td>
<td>1.05 (.84–1.31)</td>
<td>0.86 (0.70–1.07)</td>
<td>—</td>
</tr>
<tr>
<td>Strain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kicked from home early</td>
<td>—</td>
<td>—</td>
<td>1.88 (.68–5.21)</td>
</tr>
<tr>
<td>Abuse</td>
<td>—</td>
<td>—</td>
<td>.73 (.39–1.37)</td>
</tr>
<tr>
<td>Parental dysfunction</td>
<td>—</td>
<td>—</td>
<td>.77 (.62–.97)*</td>
</tr>
<tr>
<td>Negative self-identity</td>
<td>—</td>
<td>—</td>
<td>.96 (.93–.99)*</td>
</tr>
<tr>
<td>Occupational-hazard</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fight weekly</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Age of first conviction</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Youth convictions</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Overall % predicted</td>
<td>58.1%</td>
<td>58.5%</td>
<td>63.6%</td>
</tr>
<tr>
<td>$\chi^2$</td>
<td>27.15*</td>
<td>31.19**</td>
<td>116.24***</td>
</tr>
<tr>
<td>2-log likelihood</td>
<td>559.54</td>
<td>1292.09</td>
<td>1061.45</td>
</tr>
<tr>
<td>Nagelkerke R2</td>
<td>.04</td>
<td>.04</td>
<td>.13</td>
</tr>
</tbody>
</table>

Reference category for all regression models: Deceased

aReference group: White

*(p < .05) **(p < .01) ***{(p < .001)
Unlike the previous two models, the occupational-hazard model did not include all of the items tested at the bivariate level. As a result of multicollinearity between youth convictions and youth incarceration time, one of the two items had to be removed from the model. To decide which item to exclude, two MLR analyses were conducted with each of the items individually, and the item with the weakest effect on the model was removed. Youth incarceration time was excluded from the model as it provided a slightly smaller odds ratio than youth convictions, though both items produced a similar overall effect on the model. The total model was significant ($\chi^2(12) = 116.24, p < .001$) and adequately fit the data. Consistent with findings at the bivariate level, youth convictions remained a significant predictor of early mortality. Specifically, an increase in number of youth convictions decreased the odds of being a non-violent offender compared to being deceased (OR = 0.95, $p < .05$). Whereas age of first conviction differed significantly between deceased and non-violent offenders at the bivariate level, age of first conviction was only a significant predictor of mortality when comparing the deceased and violent offender groups. Specifically, the odds of being a violent offender compared to a deceased offender decreased by approximately 22% with a one unit increase in age of first conviction (OR = .78, $p < .05$).

To compare the effects of risk factors across the three pathways at the multivariate level, a final MLR model was produced using all significant or marginally significant ($p < .10$) bivariate predictors of early mortality across all pathways. As shown in Table 5, the final model was significant ($\chi^2(14) = 137.79, p < .001$) and produced a higher overall percentage of correct classifications (64.5%) than the previous three MLR models. Negative self-identity and total number of youth convictions remained significant predictors of early mortality when comparing the deceased and non-violent groups. The odds ratios of these items also remained substantively the same. Additionally, when comparing deceased and violent offenders, age of first conviction remained a significant predictor of early mortality. Parental dysfunction was no longer a significant predictor of early mortality when controlling for other criminogenic and lifestyle risk factors, and unlike the previous models, non-Indigenous minority status emerged as a significant predictor of early mortality. Specifically, being a non-Indigenous minority compared to being White decreased the odds of being in the non-violent group compared to the deceased group (OR = .37, $p < .05$).
Table 5. Final MLR model comparing strongest predictors across all pathways

<table>
<thead>
<tr>
<th></th>
<th>Non-Violent OR (95% CI)</th>
<th>Violent OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>.89 (.46–1.72)</td>
<td>.61 (.32–1.16)</td>
</tr>
<tr>
<td>Non-Indigenous minority</td>
<td>.37 (.15–.88)*</td>
<td>.73 (.31–1.68)</td>
</tr>
<tr>
<td>Male</td>
<td>.97 (.45–2.10)</td>
<td>.90 (.43–1.90)</td>
</tr>
<tr>
<td><strong>General-hazard</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early drug use</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Skip school early</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Early sexual activity</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Hard drug versatility</td>
<td>1.05 (.85–1.30)</td>
<td>.89 (.72–1.10)</td>
</tr>
<tr>
<td><strong>Strain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kicked from home early</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Abuse</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Parental dysfunction</td>
<td>.82 (.65–1.02)</td>
<td>.85 (.69–1.06)</td>
</tr>
<tr>
<td>Negative self-identity</td>
<td>.96 (.93–.99)*</td>
<td>.98 (.95–1.02)</td>
</tr>
<tr>
<td><strong>Occupational-hazard</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fight weekly</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Age of first conviction</td>
<td>1.15 (.89–1.49)</td>
<td>.76 (.59–.97)*</td>
</tr>
<tr>
<td>Youth convictions</td>
<td>.96 (.91–.99)*</td>
<td>.97 (.94–1.02)</td>
</tr>
</tbody>
</table>

Overall % predicted: 64.5%

χ²: 137.79***

2-log likelihood: 1339.16

Nagelkerke R²: .18

Reference category for model: Deceased

*Reference group: White

*(p < .05) ***(p < .01) ****(p < .001)

To test the robustness of the multivariate results, a sensitivity analysis was conducted for each multivariate model by including the same measures in a logistic regression while controlling for youth violence. In all models, early mortality is the outcome of interest (i.e., deceased = 1; non-deceased = 0). As shown in Table 6, only one item across all models was significant at the .05 level. Specifically, in the occupational-hazard model, an increase in youth convictions increased the log odds of being deceased by .04.
Table 6. Binary logistic regression models predicting early mortality (sensitivity analysis)

<table>
<thead>
<tr>
<th></th>
<th>General-hazard model</th>
<th>Strain-hazard model</th>
<th>Occupational-hazard model</th>
<th>Final model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>B (SE)</td>
</tr>
<tr>
<td>Ethnicity(^a)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>.42 (.32)</td>
<td>.33 (.32)</td>
<td>.37 (.32)</td>
<td>.36 (.32)</td>
</tr>
<tr>
<td>Non-Indigenous minority</td>
<td>.41 (.41)</td>
<td>.47 (.41)</td>
<td>.41 (.41)</td>
<td>.60 (.42)</td>
</tr>
<tr>
<td>Male</td>
<td>.08 (.36)</td>
<td>.19 (.37)</td>
<td>-.07 (.36)</td>
<td>.08 (.37)</td>
</tr>
<tr>
<td>Violent conviction (1 = yes)</td>
<td>-.04 (.29)</td>
<td>-.14 (.29)</td>
<td>-.12 (.31)</td>
<td>-.18 (.31)</td>
</tr>
<tr>
<td>General-hazard</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early drug use</td>
<td>.44 (.32)</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Skip school early</td>
<td>-.13 (.37)</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Early sexual activity</td>
<td>-.18 (.35)</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Hard drug versatility</td>
<td>.07 (.46)</td>
<td>—</td>
<td>—</td>
<td>.06 (.10)</td>
</tr>
<tr>
<td>Strain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kicked from home early</td>
<td>—</td>
<td>-.58 (.50)</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Abuse</td>
<td>—</td>
<td>.28 (.31)</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Parental dysfunction</td>
<td>—</td>
<td>.20 (.11)†</td>
<td>—</td>
<td>.18 (.11)†</td>
</tr>
<tr>
<td>Negative self-identity</td>
<td>—</td>
<td>.03 (.02)†</td>
<td>—</td>
<td>.03 (.02)</td>
</tr>
<tr>
<td>Occupational-hazard</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fight weekly</td>
<td>—</td>
<td>—</td>
<td>-.19 (.35)</td>
<td></td>
</tr>
<tr>
<td>Age of first conviction</td>
<td>—</td>
<td>—</td>
<td>.11 (.12)</td>
<td>.10 (.12)</td>
</tr>
<tr>
<td>Youth convictions</td>
<td>—</td>
<td>—</td>
<td>.04 (.02)*</td>
<td>.03 (.02)</td>
</tr>
<tr>
<td>Overall % predicted</td>
<td>93.7</td>
<td>93.7</td>
<td>93.7</td>
<td>93.7</td>
</tr>
<tr>
<td>χ2</td>
<td>4.76</td>
<td>11.27</td>
<td>6.315</td>
<td>11.93</td>
</tr>
<tr>
<td>2-log likelihood</td>
<td>405.68</td>
<td>399.16</td>
<td>403.60</td>
<td>397.98</td>
</tr>
<tr>
<td>Nagelkerke R2</td>
<td>.02</td>
<td>.03</td>
<td>.02</td>
<td>.04</td>
</tr>
</tbody>
</table>

Reference category for all regression models: Deceased
\(^a\)Reference group: White
\(^*(p < .05)\) **(p < .01) ***(p < .001)
Chapter 6.

Results: Network risk factors for early mortality

As a result of limitations in capturing aspects of the occupational-hazard model through a traditional variable-oriented approach, SNA was used to more appropriately operationalize and test the occupational hazard model. Specifically, SNA was used to capture key social dynamics related to offending and test whether exposure to high-risk situations and actors increases mortality risk. This was accomplished by examining the social network of 92 offenders, 54 of whom were deceased.

6.1. Total social network

The first step of the analysis involved conducting a components analysis on total network. The total network—shown in Figure 2—includes the ego-networks of 92 offenders, 54 of whom are deceased. Of these 92 egos, 74 were included in the main component (marked in red). The remaining 18 egos that were not included in the main component were all deceased and had significantly smaller networks than the egos included in the main component. The main component—displayed in Figure 3—is comprised of the combined ego-networks of 36 deceased and 38 non-deceased offenders. As shown in Table 7, the main component of the network includes 7804 ties, 76.6% \((n = 5982)\) of which occurred in prison. Approximately 61.3% of ties in the network are social, 12.3% are related to co-offending, and 26.4% are related to either victimization or conflict. Of the 3608 actors in the network, 86.0% are male, and 5.5% are known to have (at some point in their life course) been a gang member. The density of the network is 0.002 and the average degree is 1.86.
Figure 2. Components analysis of total network (main component highlighted in red)

*Note.* □ = Deceased ego. △ = Non-deceased ego. O = Alter
Figure 3. Main component of total network
Note. Egos marked in red; alters marked in blue.
Table 7. Description of main component of total network

<table>
<thead>
<tr>
<th></th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Network ties</td>
<td>7804</td>
</tr>
<tr>
<td>Social</td>
<td>61.3 (4784)</td>
</tr>
<tr>
<td>Co-offending</td>
<td>12.3 (957)</td>
</tr>
<tr>
<td>Victimization</td>
<td>16.2 (1266)</td>
</tr>
<tr>
<td>Conflict</td>
<td>10.2 (797)</td>
</tr>
<tr>
<td>Total community</td>
<td>23.3 (1822)</td>
</tr>
<tr>
<td>Total prison</td>
<td>76.7 (5982)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acts</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Alters</td>
<td>3608</td>
</tr>
<tr>
<td>Males</td>
<td>86.0 (3104)</td>
</tr>
<tr>
<td>Gang members</td>
<td>5.5 (200)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Network cohesion</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Density</td>
<td>.002</td>
</tr>
<tr>
<td>Average degree</td>
<td>1.86</td>
</tr>
</tbody>
</table>

After examining the main component at the descriptive level, bivariate analyses were conducted to compare the influence and ego-network cohesion of the deceased and non-deceased offenders in the main component of the network. First, the influence of the egos was compared by examining differences in degree centrality and betweenness centrality. As shown in Table 8, the non-deceased egos had significantly higher degree centrality and higher betweenness centrality than the deceased egos. Such findings correspond with the sociogram in Figure 3 where non-deceased egos (marked by a red triangle) are more visibly embedded near the centre of the network. To assess the level of cohesiveness across deceased and non-deceased ego-networks, the effective size, density, and average nodal degree were compared. The average effective size of the non-deceased ego-networks was significantly larger than the deceased ego-networks, suggesting that there are more “structural holes” (Burt, 1992) present in the ego-networks of the control group. Furthermore, non-deceased egos had significantly higher average nodal degree, indicating that they were more strongly connected to the actors in their respective ego-networks. The density of the ego-networks did not differ across deceased and non-deceased participants.

Next, the nature of the relationship between the egos and alters was examined to test whether particular types of ties are associated with early mortality in the network.
First, the average raw number of (positive) criminal social capital ties and negative ties were compared across the two groups. As displayed in Table 8, non-deceased offenders had a significantly higher number of criminal social capital ties in their ego-networks than deceased offenders. The non-deceased egos were also observed to have a higher number of negative ties, but this difference was not significant at the .05 level ($p = .068$). To control for the size of the ego-networks, the proportion of negative ties to all known ties was also compared across groups. The ego-networks of deceased offenders contained a significantly higher proportion of negative ties ($M = .43$) than non-deceased egos ($M = .33$). No significant differences were observed with respect to the number or proportion of gang members in the ego-networks of the offenders.

### Table 8. Bivariate comparison of deceased and non-deceased egos in main component of total network

<table>
<thead>
<tr>
<th></th>
<th>Deceased</th>
<th>Non-Deceased</th>
<th>$t$, $p$, $d$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Centrality</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree centrality†</td>
<td>.01 (.09)</td>
<td>.02 (.01)</td>
<td>$t(72) = 3.07$, $p &lt; .01$, .68</td>
</tr>
<tr>
<td>Betweenness centrality†</td>
<td>.03 (.02)</td>
<td>.04 (.03)</td>
<td>$t(72) = 2.78$, $p &lt; .01$, .65</td>
</tr>
<tr>
<td><strong>Cohesion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective size</td>
<td>42.65 (32.32)</td>
<td>72.32 (49.19)</td>
<td>$t(72) = 3.01$, $p &lt; .01$, .50</td>
</tr>
<tr>
<td>Density</td>
<td>.03 (.04)</td>
<td>.03 (.02)</td>
<td>$t(72) = .31$, ns, .07</td>
</tr>
<tr>
<td>Average nodal degree</td>
<td>1.17 (.20)</td>
<td>1.41 (.34)</td>
<td>$t(72) = 3.62$, $p &lt; .01$, .84</td>
</tr>
<tr>
<td><strong>Negative vs positive ties</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Criminal social capital ties</td>
<td>16.39 (27.58)</td>
<td>65.11 (90.83)</td>
<td>$t(72) = 3.04$, $p &lt; .01$, 1.03</td>
</tr>
<tr>
<td>Negative ties</td>
<td>18.97 (16.74)</td>
<td>27.08 (19.14)</td>
<td>$t(72) = 1.91$, ns, .35</td>
</tr>
<tr>
<td>Proportion negative ties</td>
<td>.43 (.19)</td>
<td>.33 (.14)</td>
<td>$t(72) = -2.48$, $p &lt; .01$, .27</td>
</tr>
<tr>
<td><strong>Gang members</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of gang members</td>
<td>4.78 (3.82)</td>
<td>3.94 (4.10)</td>
<td>$t(72) = -.08$, ns, .02</td>
</tr>
<tr>
<td>Proportion gang members</td>
<td>.12 (.13)</td>
<td>.08 (.08)</td>
<td>$t(72) = -1.75$, ns, .42</td>
</tr>
</tbody>
</table>

*Note.* †indicates normalized scores used
6.2. Victimization network

Given that deceased offenders were found to have a significantly higher proportion of negative ties in their ego-networks, the directed victimization network of the sample was examined separately to provide more context on the relationship between negative associations and early mortality. Whereas the total social network of the sample represents all ties as non-directional, the victimization network only includes instances of victimization where there was a clear direction. This means that instances of mutual (i.e., non-directional) conflict are not included in this network. In order to compare deceased and non-deceased egos in this network, a components analysis was first conducted to isolate the main component. A visualization of this components analysis is displayed in Figure 4. The components analysis revealed a main component (marked in blue) comprised of 30 egos, 9 of which are deceased. The main component—shown in Figure 5—includes 754 ties among 654 actors. 84.2\% (n = 635) of the victimization in the main component occurred in prison, and 89.0\% (582) of the actors in the component are male. The density of the main component is .002 and the average degree is 1.15.
Figure 4. Components analysis of victimization network (main component highlighted in blue)

Note. □ = Deceased ego. △ = Non-deceased ego. ○ = Alter
Figure 5. Main component of victimization network

*Note.* Arrows indicate direction of victimization.

Next, bivariate analyses were conducted to compare the deceased and non-deceased egos in the main component of the victimization network. Findings from these analyses are displayed in Table 9. Network influence was tested by comparing the in-degree centrality, out-degree centrality, and betweenness centrality of the egos. Results showed no significant differences across the two groups on any of the centrality measures. To control for network size, the proportion of outgoing victimization to all victimization in each offender’s ego-network was compared across the two groups. However, no significant differences were noted. Similarly, no significant differences were found when comparing ego-network cohesion (i.e., effective size, density, and average degree) or exposure to gang members in the network. Together, these findings suggest that deceased and non-deceased egos exhibited similar victimization profiles.

Table 9. Bivariate comparison of deceased and non-deceased egos in main component of victimization network

<table>
<thead>
<tr>
<th></th>
<th>Deceased</th>
<th>Non-Deceased</th>
<th>t, p, d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Centrality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-degree centrality†</td>
<td>.023 (.011)</td>
<td>.025 (.013)</td>
<td>t(28) = .22, ns, .08</td>
</tr>
<tr>
<td>Out-degree centrality†</td>
<td>.006 (.004)</td>
<td>.005 (.006)</td>
<td>t(28) = -.38, ns, .16</td>
</tr>
<tr>
<td>Betweenness centrality†</td>
<td>.001 (.001)</td>
<td>.001 (.002)</td>
<td>t(28) = -.28, ns, .12</td>
</tr>
<tr>
<td>Proportion of outgoing victimization</td>
<td>.78 (.14)</td>
<td>.83 (.16)</td>
<td>t(28) = .79, ns, .32</td>
</tr>
<tr>
<td>Cohesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective size</td>
<td>19.51 (5.46)</td>
<td>19.62 (9.27)</td>
<td>t(28) = .03, ns, .01</td>
</tr>
<tr>
<td>Density</td>
<td>.001 (.003)</td>
<td>.002 (.004)</td>
<td>t(28) = .46, ns, .19</td>
</tr>
<tr>
<td>Average nodal degree</td>
<td>.021 (.043)</td>
<td>.047 (.082)</td>
<td>t(28) = .85, ns, .38</td>
</tr>
<tr>
<td>Gang</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of gang members</td>
<td>2.56 (1.34)</td>
<td>1.95 (1.68)</td>
<td>t(28) = -.92, ns, .36</td>
</tr>
<tr>
<td>Proportion gang members</td>
<td>.21 (.14)</td>
<td>.18 (.23)</td>
<td>t(28) = -.39, ns, .18</td>
</tr>
</tbody>
</table>

Note. † indicates normalized scores used
Chapter 7.

Discussion

Previous studies have shown that early mortality is more common among offenders compared to the general population, but less is known about the specific causal mechanisms that underlie mortality risk in offender populations. Typically, two explanations for early mortality have been proposed: (1) specific offending-based theories that suggest that offending and mortality are directly linked, and (2) general theories that suggest that early mortality and offending are correlated only because they share underlying individual and social risk factors that act as causal factors for both outcomes. Few studies, however, have approached the examination of mortality risk factors using a theoretical framework that specifies multiple pathways to early mortality. The current study used a sample of serious and violent male and female offenders from British Columbia, Canada to address this gap by operationalizing and testing Tremblay and Paré’s (2003) pathway perspective on early mortality. This theory suggests that there are multiple pathways (also referred to as “models”) to early mortality in offender populations, which are characterized by general individual-level risk factors and risk factors specifically related to offending. The general-hazard model argues that some offenders experience early mortality as a result of risky behaviours caused by higher levels of LSC; the occupational-hazard model suggests that offenders experience higher mortality as a result of hazards directly related to offending; the strain-hazard model posits that some offenders are differentially exposed to aversive life conditions that make early mortality outcomes more likely.

To test Tremblay and Paré’s theory, each of their proposed models were compared at the multivariate level using a combination of self-report and official data. As a result of limitations in using individual-level risk factors to operationalize the occupational-hazard model—namely, that individual-level offending variables often ignore how an individual’s embeddedness within a particular social environment may also contribute to mortality—SNA was used to supplement findings from the model comparison. The use of SNA allowed for a more comprehensive measurement of an offender’s exposure to conflict, and captured the social context related to offending.
Such measures have been largely ignored in traditional variable-oriented studies on early mortality, and as such, the current study has important implications for both Tremblay and Paré’s theory, and the broader debate surrounding early mortality in offender populations. Although findings from the study lacked support for the general-hazard model, risk factors across the strain-hazard and occupational-hazard models—including negative self-identity, family-related strain, and youth offending—were significant predictors of early mortality. Social network findings also showed partial support for the occupational-hazard model. Specifically, the accumulation of criminal social capital may protect against early mortality outcomes.

### 7.1. Mortality characteristics of the sample

The study began by identifying deceased members of a sample of 881 serious and violent young offenders. After an approximately 17 year follow up period, 6.2% of participants in the sample were deceased (n = 55), and the average age of death was 24.6. Although mortality rates and ages vary across studies—largely due to differences in follow-up period and sample composition—the rate of death in the current study is higher than most previous studies with similar exposure periods (e.g., Daigle & Naud, 2012; Lattimore et al., 1997), and the average age of death is comparable to studies that have focused on high-risk young adult offender samples (e.g., Sailas et al., 2006). The prominence of early mortality raises the issue of not simply ensuring that mortality is controlled for in life course research (i.e., to inform exposure time; Eggleston et al., 2004); rather, these findings support previous researchers’ claims that early mortality is not a rare event in high-risk offender populations and, as such, should be actively considered in research on offending and criminal careers (Lattimore et al., 1997, p. 206). Given the serious and high-risk nature of the sample, their mortality characteristics are not representative of all young offenders in Canada, but clearly demonstrate that serious and violent individuals are disproportionately at risk for early mortality. From a research standpoint, these findings also highlight the unique opportunity that high-risk offender samples provide for studying the mechanisms that underlie mortality risk as they (unfortunately) provide a high base rate of mortality as well as offending.

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21 Depending on birth year, participants in Cohort I were followed up with until age 34-39 and participants in Cohort II were followed up with until age 26-31
7.2. Interpreting findings across the three pathways

7.2.1. The general-hazard model

The first of Tremblay and Paré’s models that was examined was the general-hazard model. Drawing from Gottfredson and Hirschi’s (1990) general theory of crime, the general-hazard model suggests that early mortality is caused by risky proximal behaviours that result from higher levels of low self-control in offender populations. In other words, the relationship between offending and early mortality is spurious once accounting for the relationship between factors such as low self-control. The general-hazard model was captured by examining four risky lifestyle indicators that have been associated with LSC in previous research (e.g., Baron, 2003; Piquero et al., 2005): (1) hard drug versatility, (2) early onset drug use, (3) early onset skipping school, (4) early onset sexual activity. Descriptive findings from the total sample demonstrated that there was variability in these measures across the total sample (see Table 2), but despite this variability, these factors were not associated with early mortality at the bivariate or multivariate level. As such, the study found no support for the general-hazard model in the sample.

Findings from the general-hazard model contradicted previous studies that have found a link between LSC and early mortality (e.g., Piquero et al., 2005), as well as risky behaviours and early mortality in offender populations (e.g., Chassin et al., 2013; Farrington et al., 2012; Laub & Vaillant, 2000). Whereas these studies suggested that offenders who have a higher propensity for engaging in risky behaviours are more likely to experience early mortality (regardless of offending), deceased and non-deceased offenders in the current sample did not differ with respect to lifestyle risk. There are several potential reasons for this difference in findings. First, whereas previous studies examined the presence of lifestyle risk factors across a fuller period of adolescence, the current study aimed to capture more variability in lifestyle risk across the sample by measuring the early onset of risky behaviours. For this reason, the findings from the current study do not negate the possibility that the relationship between risky lifestyles and early mortality is established later in adolescence. Secondly, there are distinct differences in the measurement of the behavioural risk factors included in the current study compared to others that have examined similar risk factors. For example, rather than measuring hard drug versatility or early onset of drug use, Chassin et al. (2013)
measured symptoms of drug dependency and history of a substance use disorder. Similarly, whereas the current study measured the early onset of truancy, Farrington et al. (2012) measured specific behavioural problems in school and only examined homicide risk, as opposed to all-cause mortality risk. As such, it is possible that differences between findings from the current study and others are largely related to differences in the operationalization of the models tested.

With respect to drug use in particular, null findings from the current study are congruent with previous studies where no association between drug abuse and early mortality was found (e.g., Lattimore et al., 1997; Piquero et al., 2014). However, despite these null findings, efforts by policy-makers and practitioners to mitigate the dangers of substance use in high-risk populations (including young offender populations) should be of continued importance. This is particularly important in British Columbia where 1,514 individuals died of illicit drug overdoses in 2018—a per capita rate of approximately 30 per 100,000 (BC Coroner’s Service, 2019). However, from risk assessment perspective, the findings from the current study suggest that focusing predominantly on risky behaviours such as drug abuse may not necessarily be an effective way to assess all-cause mortality risk in high-risk groups of offenders. Instead, practitioners may be served better to consider a broader variety of social and criminogenic risk factors, as indicated by findings across the strain-hazard and occupational-hazard models.

7.2.2. The strain-hazard model

The second model examined was the strain-hazard model. Drawing from Agnew’s (1992) general strain theory, the strain-hazard model suggests that the differential exposure to strain in offender populations causes some offenders to experience internal despair which may ultimately lead to early (and often self-inflicted) mortality. Tremblay and Paré (2003) also posited that strain may result directly from continuous involvement in crime or may have independent or conditional effects on mortality (pp. 314-315). Findings from the current study supported both sides of this hypothesis. Two sources of strain were found to be associated with early mortality in the strain-hazard model. Specifically, an increase in negative self-identity and an increase in parental dysfunction both increased the odds of being a deceased offender compared to a non-violent offender. When examining these items in the context of a final model, which included risk factors from each of the three pathways, negative self-identity
remained a significant predictor of early mortality, whereas parental dysfunction was no longer significant after controlling for other criminogenic and lifestyle risk factors that were relevant to early mortality.

The relationship between individual and family-related strains and early mortality may support Tremblay and Paré’s (2003) hypothesis that higher rates of self-inflicted deaths in offender populations are driven by the emotional suffering that offenders experience as a result of cumulative exposure to strain. Specifically, youth who thought more negatively of themselves, and/or did not have positive parental support, may have been more likely to end their lives prematurely. Additionally, consistent with Agnew’s (2006) prediction that children and youth would use delinquent behaviours as a means of alleviating negative emotions caused by strain, youth in the sample who experienced higher levels of individual and family-related strains may have also been more likely to turn to dysfunctional and dangerous coping mechanisms such as drug abuse. This hypothesis has also been supported by previous theorists (e.g., Baumeister, 1990), as well as empirical research that has shown a significant positive association between low self-esteem and substance abuse in high-risk youth populations (e.g., Gordon & Caltabiano, 1996; Wild, Flisher, Bhana, & Lombard, 2004). However, given that drug abuse also has been shown to be attributed to underlying individual characteristics such as LSC (Baron, 2003), these findings may also suggest that the strain-hazard and general-hazard models are not mutually exclusive. As Tremblay and Paré (2003) suggested, it is possible that some offenders experienced drug-related deaths as a result of a higher propensity for engaging in risk-taking behaviours, whereas others experienced drug-related deaths as a result of coping with negative emotions caused by strain. For this reason, future research should consider examining the interaction between the general-hazard and strain-hazard models with respect to particular mortality outcomes (e.g., drug-related deaths).

Given that the relationship between family dysfunction and early mortality was no longer significant after controlling for youth offending in the final model, it is also possible that youth with more dysfunctional parents may have been driven to engage in criminal offending at a higher rate; this may have subsequently increased their risk of experiencing early mortality. Indeed, other studies using the ISVYOS data showed that a greater degree of family dysfunction was significantly associated with a high-rate persistent offending trajectory measured from ages 12-29 (McCuish & Corrado, 2018).
Such findings are congruent with Lemert’s (1967) strain-desistence theory\(^{22}\) which suggests that offenders who engage in higher rates of criminal activity may find their lives less meaningful over time and may feel constrained by their criminal lifestyle. When offenders do not attribute meaning to their lives, they may be more willing to engage in more dangerous criminal activities that increase their risk of early mortality. Furthermore, as offenders feel more apathetic or discouraged about their lives over time, it is possible that some may choose to end their life prematurely through suicide or excessive drug use (Tremblay and Paré, 2003). Lemert’s (1967) strain-desistence theory ultimately merges the strain-hazard and occupational-hazard perspectives by considering offending as a source of strain. For this reason, this interpretation is also relevant to the occupational-hazard model and reinforces that pathways to early mortality may not be mutually exclusive.

In sum, strain may play an important role in influencing early mortality risk in offender populations. As such, early assessments of family-related strains, as well as the internalization of strain (e.g., youths’ perceptions of themselves), may assist practitioners in identifying youth who may be at an increased risk for experiencing mortality later in life. Given that the current study identified parental dysfunction as a risk factor for early mortality, intervention and treatment programs aimed at supporting parents from high-risk families who experience strains of their own (e.g., criminal involvement, substance use, mental health issues) may be an effective way to reduce the likelihood of their children engaging in criminal behaviour and experiencing early mortality later in life. Also, as suggested by previous research on high-risk adolescent samples, a more supportive family environment may be important for a child’s security, which in turn may help produce a more positive self-image that protects against early mortality (Sharaf, Thompson, & Walsh, 2009). Furthermore, programs aimed at shifting youths’ negative self-perceptions and increasing their resiliency and self-esteem may reduce maladaptive coping mechanisms and dangerous behaviours that lead to self-inflicted mortality outcomes. This claim is supported by general population studies that have shown that programs aimed at increasing adolescents’ self-esteem are effective in reducing maladaptive behaviours and negative life outcomes (see Mann, Hosman, Schaalma, & de Vries, 2004).

\(^{22}\) which Tremblay and Paré (2003) drew from to specify the strain-hazard model
It is important to note that one of the common critiques of self-identity research is that small effect sizes may not justify investing time and money into programs aimed at improving adolescents’ self-esteem (Baumeister, Campbell, Krueger, & Vohs, 2003). Despite this, other researchers have argued that it is important not to underestimate the effects of self-esteem as even small effect sizes can have major impacts on behaviours and life outcomes over time (Trzesniewski et al., 2006). Specifically, adolescents who think more negatively about themselves may be more likely to experience adjustment problems in adulthood, which can negatively impact future opportunities and life outcomes (Trzesniewski et al., 2006, p. 387). Additionally, other researchers have highlighted the overlap between negative self-identity and persistent offending, suggesting that prosocial identity is also an important factor in the desistance process (Na & Paternoster, 2019; Rocque, Posick & Paternoster, 2016). As such, reducing young offenders’ negative self-perception may be an important way to reduce both offending and early mortality risk, and also may have many other tangential benefits such as reducing adjustment problems.

Ultimately, findings across the strain-hazard model suggest that both individual and family-related strains may be important for reducing early mortality risk among young offenders. However, what remains unaddressed by these findings is how differences in the lived-experiences among offenders may impact their exposure to strain across the life course. Findings from the final model, as well as those from previous mortality studies (e.g., Chassin et al., 2013; Lattimore et al., 1997; Piquero et al., 2005), suggest that this is particularly important to consider within the context of ethnic identity.

**Ethnic-specific strains and early mortality**

The final model, which compared the effects of risk factors from each of the three pathways, highlighted an additional predictor of early mortality that was not reflected by previous models. Specifically, after controlling for demographic characteristics and key risk factors across multiple domains, being a non-Indigenous minority compared to being Caucasian decreased the odds of being non-violent compared to being deceased. As previously addressed in Chapter 2, previous studies have often found that ethnic minority groups are disproportionately at risk of experiencing early mortality (e.g., Chassin et al., 2013; Lattimore et al., 1997; Piquero et al., 2005), and these findings
have often been interpreted from a strain perspective. Similarly, it is possible that some youth in the current study may have experienced early mortality as a result of ethnic-specific strains that were not captured by the current study. This interpretation must be considered within the sociopolitical context of the sample jurisdiction.

The relationship between non-Indigenous minority status and mortality may be related to differences in the lived experiences and available opportunities of non-Indigenous minority youth (e.g., South Asian, East Asian, Black) in British Columbia compared to their Caucasian counterparts. British Columbia is a multicultural province with a growing population of residents with non-European origins,\(^{23}\) and this is largely attributed to increases in immigration over the past few decades (Statistics Canada, 2016). Research from Canada and internationally has shown that challenges associated with immigration can place strain on multiple generations of families, often limiting available opportunities and quality of life (Arredondo-Dowd, 1981; Choudhry, 2001; Khan & Watson, 2005). Beyond the effects of immigration, individuals from ethnic-minority groups may also experience racial prejudice across their life course, the negative effects of which are both numerous and widely-known. Evidence of such effects may be reflected by current socioeconomic divisions in Canada, as visible minority groups are less likely to be employed compared to non-racialized groups, despite having higher average levels of educational attainment (Canadian Council on Social Development, 2000). As such, it is possible that some youth experienced early mortality as a result of cumulative exposure to strains that were unique to their lived experience as a minority in Canada. Specifically, some ethnic-minority youth may have had fewer legitimate opportunities for acquiring income and may have resorted to illegitimate, and riskier, opportunities that increased their risk of experiencing early mortality. Alternatively, some youth may have learned to cope with these strains through maladaptive coping mechanisms such as drug abuse, and others may have resorted to ending their lives prematurely (as posited by Tremblay and Paré’s strain-hazard model).

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\(^{23}\) According to the most recent census data, 18.0% of the provincial population consisted of residents with east and Southeast Asian origins and 9.1% of residents with South and West Asian origins (Statistics Canada, 2016).
7.2.3. The occupational-hazard model

The occupational-hazard model predicts that more frequent and serious criminal offending increases the likelihood of early mortality by increasing offenders’ exposure to conflict. The current study found partial support for this hypothesis. Consistent with the occupational-hazard model hypothesis, when comparing deceased and non-violent offenders, an increase in the number of youth convictions was found to increase the odds of early mortality. However, when comparing deceased and violent offenders, an increase in age of first conviction was found to decrease the odds of early mortality. This finding directly opposes the occupational-hazard model hypothesis that an earlier onset of offending increases the risk of experiencing early mortality. Both findings have important implications for Tremblay and Paré’s (2003) theory as well as for criminal justice policies and practices.

Previous studies found a direct link between high-rate chronic offending in adulthood and early mortality (e.g., Nieuwbeerta & Piquero, 2008; Piquero et al., 2014; Zane et al., 2019). These findings are consistent with Moffitt’s (1993) developmental taxonomy, where LCP offenders are predicted to experience more negative life outcomes as result of prolonged high-rate offending. The current study extends these findings, suggesting that the relationship between high-rate offending and mortality may be established in adolescence. Specifically, when comparing deceased and non-violent offenders, offenders who were convicted of a higher number of offences between the ages of 12 and 17 were more likely to experience early mortality, and that relationship was not confounded by adolescent drug abuse, family or individual strain, or demographic risk factors. Such findings may support the hypothesis that although not all offenders experience early mortality, those that offend more chronically during adolescence may expose themselves to more opportunities for conflict and victimization throughout the life course, and this is not attributed to differences in age of onset. It is possible that adolescents who engage more frequently in criminal activities may be seen as a threat by other offenders, and may be more likely to be targeted by those offenders compared to adolescent offenders who maintain a less “visible” profile. This may also explain why weekly fighting in adolescence was not a significant predictor of early mortality, as being victimized may be more related to a youth’s criminal activity than to their propensity for engaging in physical conflict in adolescence. However, as previously mentioned, the association between high-rate offending in adolescence and early
mortality may also support Lemert’s (1967) hypothesis that frequent offending and contact with the criminal justice system is a source of strain itself. The theoretical overlap between the strain-hazard and occupational-hazard models reiterates the importance of approaching early mortality research from a theoretical perspective that allows for multiple (non-mutually exclusive) pathways to early mortality.

When comparing the offending characteristics of deceased and violent offenders, deceased offenders were found to have a later age of onset, despite offending at a similar rate as violent offenders. Whereas previous studies have supported the occupational-hazard model hypothesis that earlier age of onset is a risk factor for early mortality (e.g., Kjelsberg & Laake, 2010), findings from the current study propose that having an earlier start to the criminal career does not necessarily increase an offender’s risk of experiencing early mortality. Instead, deceased offenders may have overcompensated for their later start to offending by engaging in crime at a similar rate as offenders who had more experience than them (i.e., violent offenders). By being particularly active in the years between age of onset and death, deceased offenders may have taken on too much too soon, and may not have had the criminal social capital or experience to handle the consequences that may have arose from their offending. This may be particularly important to consider with respect to violent mortality outcomes, as previous research on high-risk youth samples has shown that higher levels of social capital may help protect against victimization (McCarthy, Hagan, & Martin, 2002). Such claims are also partially supported by network findings from the current study, which are further discussed in section 7.3 of this chapter.

It is also important to consider the possibility that the difference in age of onset between violent offenders and deceased offenders reflects a key difference in the sampling criteria for the two groups. Specifically, whereas members of the violent group were selected based on similarities in their offending characteristics (i.e., the commission of one or more violent offences), the deceased group is comprised of both violent ($n = 36$) and non-violent ($n = 19$) offenders. Considering that life course research has long showed that violent offenders tend to engage in crime earlier than non-violent offenders (e.g., Elliot, 1994), the difference in age of first conviction between the deceased and violent groups may have been driven by the non-violent offenders in the deceased group.
Serious and violent offenders commit a disproportionate amount of crime and cause substantial costs to the criminal justice system (Easton, Furness, & Brantingham, 2014; Hoddenbagh, Zhang, & McDonald, 2009; Loeber & Farrington, 1999). As such, a major goal of developmental and life course criminology has been to prevent and reduce high-rate chronic offending through evidence-based assessment and intervention strategies. Findings from the occupational-hazard model suggest that such efforts are important not only from a criminological perspective, but also from broader public health framework. Specifically, programs and strategies aimed at preventing or reducing the frequency of adolescent offending may not only reduce the expenditure of criminal justice system resources, but may also assist in preventing early mortality outcomes. This message should be communicated clearly to both policy makers and practitioners, and should be included as one of the core goals of early prevention and intervention strategies aimed towards high-risk youth. Other researchers have echoed this opinion, suggesting that by the time youth are already involved in the criminal justice system, the chances of reducing early mortality risk are substantially reduced (e.g., Lattimore et al., 1997). As such, early mortality prevention should not only be a priority for criminal justice professionals, but also for practitioners outside of the criminal justice system (e.g., education system, health care system, social services). Additionally, these strategies should not only account for individual-level risk factors but should consider the social context in which individuals are embedded in. To capture this social context, a social network approach is necessary.

7.2.4. Implications of sensitivity analysis findings

To test the robustness of the findings from the MLR analyses, each of the previously tested models were included in a binary logistic regression analysis with early mortality as the outcome of interest. Instead of disaggregating the control sample into violent and non-violent sub-groups, youth violence was used as a control measure in all models. Consistent with findings from the MLR analyses, none of the risky lifestyle indicators were predictive of early mortality when comparing deceased and non-deceased offenders. As such, these findings provide no support for the general-hazard model hypothesis that a higher propensity for engaging in risky behaviours increases the risk of early mortality in offender populations. Additionally, consistent with MLR findings from the occupational-hazard model, findings from the sensitivity analysis also showed
that offenders who incurred more convictions during adolescence were more likely to experience early mortality. However, when examining the final model—which included risk factors across all domains—a higher number of adolescent convictions was no longer significantly predictive of early mortality. As such, findings from the sensitivity analyses only provide partial support for Tremblay and Paré’s (2003) occupational-hazard hypothesis that offending and mortality are directly linked.

Despite providing partial support for the MLR findings across the general-hazard and occupational-hazard models, findings from the sensitivity analyses were inconsistent with MLR findings across the strain-hazard model. Specifically, none of the indicators of strain were predictive of early mortality when comparing deceased offenders to non-deceased offenders. Although these findings help justify the decision made in the current study to disaggregate offenders into violent and non-violent sub-groups, they may also suggest that the items included in the model do not sufficiently capture the risk profiles of the deceased offenders. Specifically, these findings raise the question of whether early mortality risk may be better examined by measuring offenders’ total exposure to strain, rather than measuring specific indicators of strain. Although testing this hypothesis is beyond the scope of the current study, future studies should consider addressing this question by measuring strain exposure using a scale-oriented approach. Such research may provide important insight into the underlying mechanisms that drive early mortality outcomes in offender populations, and may have key implications for Tremblay and Paré’s (2003) theory.

7.3. Unpacking the social networks of deceased and non-deceased offenders

The final component of the current study involved examining the social networks of deceased and non-deceased offenders using SNA. The purpose of using SNA was to more accurately test Tremblay and Paré’s theory and to address a key limitation in previous research on offending and mortality. That is, previous studies have predominantly relied on examining individual-level traits that may differentially expose someone to opportunities for both offending and mortality but have often ignored how an individual’s embeddedness within a particular social environment may also contribute to mortality. Capturing these broader social influences through SNA may be key for understanding why higher-rate offenders are disproportionately more likely to experience
early mortality. To explore this hypothesis, a subsample of deceased \( (n = 54) \) and non-deceased \( (n = 38) \) offenders were compared with respect to their level of social influence, social cohesion, criminal social capital, and exposure to conflict and victimization. Specifically, this network sample included all members of the ISVYOS who were deceased and had available network data, as well as a select group of ISVYOS participants who were connected to one or more deceased participants and whose networks had previously been coded for a network project. Ultimately, the non-deceased participants were included in the network sample to explore why some offenders do not experience early mortality despite being criminally connected to offenders who do.

7.3.1. Network differences between deceased and non-deceased offenders

Deceased and non-deceased offenders were first compared with respect to network influence and ego-network cohesiveness. Despite there being no differences in the amount of available data for the two groups, non-deceased offenders had a significantly greater number of ties and were more likely to be positioned in brokership roles in the total network. Similarly, when examining ego-network cohesion, non-deceased offenders were found to have more “structural holes” (Burt, 1992) in their ego-networks, while also being more strongly connected to the alters in their ego-networks (i.e., higher average nodal degree). Together, these findings suggest that offenders who were more connected and had more control over the flow of communication, both in the total network and their respective ego-networks, were also less likely to experience early mortality. However, given that the total network contained a combination of positive and negative ties, these findings must be interpreted within the context of the nature of the relationships between actors in the network. For this reason, deceased and non-deceased egos were also compared with respect to their accumulation of negative versus positive criminal social capital ties.

Bouchard and Nguyen (2010) asked the question “is it who you know, or how many that counts?” (p. 130). The current study considered this question in the context of early mortality risk by examining both the quantity and nature of relationships in the ego-networks of deceased and non-deceased offenders. When comparing the two groups, non-deceased offenders had a significantly higher number of positive criminal social capital ties, but were no different than deceased offenders with respect to the total
number of negative relationships they had formed. However, deceased offenders averaged a significantly higher proportion of negative ties in their ego-networks, suggesting that early mortality risk may not be directly related to an offender’s total exposure to conflict or victimization but is instead relative to the amount of positive criminal social capital an offender has accrued. These findings suggest that who an offender knows and how many connections they have are important for early mortality risk.

Findings from the total network comparisons partially support the occupational-hazard hypothesis. Although total exposure to conflict and victimization across the study period was not predictive of early mortality, offenders who grew their positive criminal social capital appeared to be more protected against early mortality outcomes. There are several potential reasons for this. In the context of violent mortality, offenders who were connected to a higher total number of trusted criminal associates, and were more strategically positioned in the total network, may have had more access to information about potential threats of victimization against them. This may have enabled them to take necessary precautions to avoid or prepare for threats when necessary. This may be particularly relevant for correctional settings where violence is common (Bowker, 1980; Irwin, 1980) and access to information is often important for inmate safety. Additionally, offenders who accrued more positive criminal social capital may have been able to rely on trusted criminal associates to defend or protect them against incoming threats, as has been suggested by previous studies on social capital in high-risk youth samples (e.g., McCarthy et al., 2002). Comparatively, as a result of having a weaker support network, deceased offenders may have been forced to deal with conflict themselves, which may have increased their likelihood of being targeted by other offenders vying for criminal opportunities, as posited by Tremblay and Paré (2003).

Findings from the total network comparisons may also have implications for strain-related mortality in the sample. Drawing from Lemert’s (1967) strain-desistence theory, Tremblay and Paré emphasized that offenders were more likely to suffer from the effects of criminogenic strains when they “did not benefit from the support that intimate interactions with other co-offenders can provide” (p. 314). As such, offenders with fewer positive ties in their ego-networks may have been more likely to use maladaptive coping mechanisms like drug abuse to mitigate the cumulative impact of criminogenic strains. Over time, offenders with fewer positive ties in their personal networks may have also
experienced feelings of loneliness or despair, and may have been more likely to end their lives prematurely in response to those emotions. Contrastingly, offenders who accrued more positive criminal social capital over their life course may have had more access to social supports, resources, and positive reinforcements to support them through the strains of living a criminal lifestyle. Given that type of death was not controlled for in the current study, additional research is needed to tease apart this relationship.

What remains unknown from these results is whether the direction of victimization in the network, or the types of individuals that offenders victimized (or were victimized by), were responsible for the relationship between negative criminal ties and early mortality risk. To investigate this question, a separate analysis of the directed victimization network of the sample was conducted.

7.3.2. Unpacking the relationship between negative criminal ties and early mortality risk

To further investigate the relationship between negative criminal ties and early mortality, deceased and non-deceased offenders were compared with respect to their degree and proportion of outgoing and incoming victimization ties, their positions in the network, their ego-network cohesion, and their exposure to gang members in the network. Results showed no differences between the two groups on any of the measures tested, suggesting that deceased and non-deceased egos in the sample exhibited similar victimization profiles; however, it is again important to note that the similarities in victimization profiles did not equate to similarities in criminal social capital networks. The findings indicate that offenders experiencing early mortality are not differentially exposed to victimization; rather, they may lack the types of social support networks that counterbalance this victimization and help cope with these victimization experiences. Put differently, individuals that experienced early mortality were not dealing with differential exposure to victimization; rather, they were dealing with comparatively weaker criminal social capital resources for coping with this victimization. Unfortunately, it is too early to interpret whether the social support is positive (e.g., emotional support) or negative (e.g., provides the criminal capital to negatively retaliate against the individuals that an offender is in conflict with). These findings pose interesting questions,
both for the victim-offender overlap literature, as well as for Tremblay and Paré’s occupational-hazard model.

The victim-offender overlap literature suggests that offenders who frequently engage in acts of victimization are more likely to be victimized themselves (e.g., Piquero et al., 2005) and previous social network studies have shown that exposure to risky actors (e.g., gang members) in criminal networks can increase the likelihood of experiencing violent mortality (e.g., Papachristos et al., 2015a). Additionally, the occupational-hazard model hypothesizes that as an offender perpetrates more victimization onto others and establish a higher number of negative relationships with risky offenders, their likelihood of experiencing early mortality may increase (Tremblay and Paré, 2003). However, null findings from the current study indicate that more frequent perpetration of victimization in a risky criminal network does not necessarily increase an offender’s risk of experiencing early mortality, nor does engaging in victimization with riskier offenders. Instead, these findings support results from the total network analysis that the risks posed by engaging in victimization and conflict may be relative to the amount of positive criminal social capital in an offender’s network. These findings have key implications for policy and practice in the criminal justice system.

### 7.3.3. Implications for policy and practice

Results from the network analysis highlight the importance of considering the social embeddedness of offenders when assessing early mortality risk. Monitoring the social networks of offenders—including the nature of their relationships with other offenders—may assist criminal justice practitioners in assessing which specific offenders may be at a heightened risk of experiencing early mortality. Offenders who are disliked by their peers, or are socially isolated, and experience frequent conflict or victimization may be at a higher risk for experiencing early mortality, and may be prime candidates for intervention and support services. For this reason, risk assessment tools, should be modified to capture network-related risk factors for early mortality, particularly in correctional facilities where data on offender interactions are readily available. For example, the Corrections Needs Risk Assessment (CRNA)—a tool used to guide correctional staff in providing appropriate level of supervision and intervention based on an offender’s criminogenic history and needs—could be adapted to include an assessment of an offender’s standing in the context of the current network of offenders.
at a specific correctional facility. Using self-nomination data from offenders upon intake, and updating these data using records of offender interactions on the units, correctional staff may be better equipped to assess an offender’s likelihood of experiencing conflict, victimization, or social isolation on the unit. Ultimately, these network-informed risk assessments may assist staff in directing resources and services towards the offenders who are in most need of support.

One of the key findings from the current study is that positive criminal associations may be key protective factors against early mortality in risky criminal networks. Although the underlying nature of the relationship remains unknown (e.g., whether positive criminal associations are helpful for coping with the strains associated with offending, or helpful for defending against threats of violence), this finding may have important implications for treatment and intervention programs aimed at reducing early mortality among high-risk offenders. Of course, programs and policies should not be designed to help offenders grow their criminal networks; however, focusing solely on decreasing offenders’ total exposure to conflict and victimization may also not be an effective way to reduce early mortality risk. Instead, practitioners could use social network data to identify persons in an offender’s ego-network that are (a) positively connected to the ego and (b) not connected or only weakly connected to their broader criminal network. Such persons may be both a positive source of social support for the offender and may be less likely to expose the offender to opportunities for conflict or co-offending.

Early mortality risk may also be mitigated by helping offenders transform negative relationships into positive ones, and one way to accomplish this goal may be through restorative justice (RJ) programs. RJ programs have been shown to be effective in reducing recidivism (see Latimer, Dowden, & Muise, 2005), and research in correctional settings suggests that RJ programs are also effective in helping offenders develop conflict resolution skills and improving the overall experience of imprisonment (Dhami, Mantle, & Fox, 2009). By helping mediate conflicts between offenders and facilitating offenders’ understanding of the consequences of victimization and conflict, RJ programs may ultimately help reduce the proportion of negative criminogenic relationships in an offender’s social network.
In addition to RJ programs, programs that help foster social competency in young offenders may also be an effective way to minimize the accumulation of negative criminogenic ties. Previous research has shown that more frequent involvement in the criminal justice system disrupts the healthy psychosocial development of youth and hinders young offenders’ ability to establish positive interpersonal relationships (Steinberg, Chung, & Little, 2004). As such, programs that aim to facilitate healthy social relationships between offenders, as well as will pro-social peers, may help minimize the development of negative relationships across an offender’s life course. These programs may be particularly important before, or during the early stages of, a youth’s involvement in the criminal justice system when their psychosocial make-up is more amenable to change.

Ultimately, SNA demonstrates that individuals are not completely autonomous beings who are in complete control over their relationships, or the way those relationships influence their behaviours. Instead, human behaviours and life outcomes are inherently influenced by social relationships and experiences. Considering that many early mortality outcomes are socially-driven (e.g., homicides, drug-overdoses), policies and programs that neglect the social embeddedness of offenders may not be effective in reducing early mortality risk. That said, social network research on early mortality in offender populations is in its infancy and more research is needed to better understand which specific programs and policies may be effective in reducing early mortality risk.

7.4. Study limitations and future research

Findings from the current study should be considered in the context of the study’s limitations. To best address these limitations, the current section is organized across the study’s two main components. Along with limitations, implications for future research are provided for both components of the study.

7.4.1. Evaluating Tremblay and Paré’s pathway perspective: Limitations and future research directions

Operationalizing and testing Tremblay and Paré’s pathway perspective brought about several challenges. First, Tremblay and Paré’s perspective was originally specified to explain early mortality outcomes in general offender populations. Given that the
current study uses data from a high-risk sample of serious and violent young offenders, it is expected that the models reflect less variation between deceased and non-deceased offenders than would be expected if examining a broader sample of offenders from various age groups and risk levels. Secondly, limited data on cause of death in the sample restricted the ability to test Tremblay and Paré’s models on particular mortality outcomes. This is particularly important to consider in the context of the occupational-hazard and strain-hazard models, which were specified to explain specific mortality outcomes. Nevertheless, these were the two models that received most support in the analyses. Additional research on specific-cause mortality risks may help further validate Tremblay and Paré’s theory and assist practitioners in reducing specific mortality outcomes (e.g., drug overdose, suicide, homicide). Finally, the available data did not allow for a direct measurement of participants’ level of self control, nor did they account for all potential sources of strain (e.g., socioeconomic status, living conditions, neighbourhood disorganization). As such, it is possible that findings from the general-hazard and strain-hazard model may have differed if examining self-control directly, or other sources of strain.

Offenders within the non-deceased groups will also become deceased over time. This poses a challenge for all mortality research, as changes to treatment and reference groups also have the potential to change results. Additionally, given that risk factors in the current study were only measured at one time period in adolescence, it is possible that these risk factors had a stronger relationship with cases of mortality that occurred earlier rather than later in the life course. For this reason, mortality data should be continuously collected for longitudinal samples, and risk factors for mortality should be evaluated across multiple stages of the life course. This longitudinal approach may help capture the dynamic nature of a person’s life and identify additional risk factors that are distinct to specific stages of the life course. Despite the fact that the current study did not examine the full life course of the sample, it still provides a strong jumping off point for future research on early mortality in high-risk offender samples.

Finally, although the current study identified several risk factors when comparing deceased and non-violent offenders, deceased offenders and violent offenders exhibited similar risk factor profiles across the three models. These findings raise the important question of whether violent offending may be a potential risk factor for early mortality across the life course. Specifically, it is unknown if, over a longer exposure period,
members of the violent group will experience differential mortality rates compared to the non-violent group. Although victim-offender overlap studies have suggested that offenders who enact more violence will be more likely to be violently victimized (e.g., Piquero et al., 2005), the hypothesis remains untested with respect to all-cause mortality risk. To empirically test this hypothesis, future studies with longer follow-up periods should consider examining differences in the mortality rates and specific risk factor traits between violent and non-violent offenders. Doing so may have key implications for Tremblay and Paré’s theory, and for the broader literature on offending and early mortality.

7.4.2. Using network analysis to explore early mortality risk: Limitations and future research directions

As a result of collecting social network data from official community and correctional service reports in CORNET, there are likely differences in the amount of, and quality of, data across cases. Specifically, offender relationship ties are not systematically recorded in CORNET, and the amount of available data is largely dependent on the level of detail that practitioners include in their reports. This means that offenders who attended programs and institutions with stricter reporting requirements may have had more information recorded about their interactions with other offenders. However, given that the members of the control group were sampled based on their connection to deceased offenders in the network—often as a result of attending the same programs or institutions—it is expected that this bias is equal across both groups. Regardless of potential differences between available data on specific offenders, it is expected that criminal ties of all offenders in the sample were underestimated. This is particularly true for community-related ties as criminal justice professionals in the community typically receive minimal information about an offender’s associations in the community. Despite this, it is expected that most incidents of victimization or conflict that resulted in official criminal justice system involvement were recorded as these incidents typically require official documentation in CORNET.

An additional limitation involves the representation of time in the current study. Specifically, offenders in the sample were followed for an average of 14 years, and the networks represented in this study reflect the total number of ties established during that time period. Given that the prevalence of mortality increases over time, it is possible that
some network differences between deceased and non-deceased offenders may have been present at particular points in time, but were not reflected across the entirety of the life course. For this reason, additional differences between deceased and non-deceased offenders may not have been captured by the current study. Additionally, given that the temporal precedence of relationships in an offender’s ego-network was not addressed, it is unknown whether having a higher number of positive criminal ties protects offenders from experiencing serious victimization and conflict, or whether more frequent conflict and victimization inhibits offenders from being able to generate positive relationships. Answering this question in future research may help contextualize the relationship between offending and early mortality and help identify particular stages of the life course in which offenders may be more susceptible to treatment and intervention measures.

Finally, given that network risk factors for early mortality were only explored at the bivariate level, the current study does not account for potential confounding factors for early mortality. As such, it is possible that network differences between deceased and non-deceased offenders in the study may have been attributed to individual-level traits of offenders that were not measured. Future studies with larger sample sizes can address this limitation by conducting multivariate analyses that control for both network risk factors and individual traits.

7.5. Summary and conclusion

Using data on adolescent risk factors for early mortality in a sample of serious and violent young offenders, findings from the current study support Tremblay and Paré’s (2003) theory that there are multiple pathways to early mortality in offender populations. Multivariate analyses showed no support for the general-hazard model, but identified several predictors of early mortality across both the strain-hazard and occupational-hazard models, including negative self-identity, parental dysfunction, and youth convictions. Additionally, after controlling for risk factors across all models, being a non-Indigenous minority increased the risk of early mortality, suggesting that differences in the lived-experiences across ethnic groups may also be associated with early mortality risk. As such, the current study helps bridge the gap between offending and propensity-based perspectives on early mortality and demonstrates the importance of
accounting for underlying characteristics that may predispose offenders to experiencing early mortality, as well as factors that related to offending.

With respect to policy and practice, findings across the strain-hazard and occupational-hazard models suggest that early mortality risk assessment and intervention strategies should acknowledge the impact of individual and family-related strains on early mortality, as well as strains that may result from high-rate chronic offending. Additionally, findings from the combined pathway model suggest that the specific needs of high-risk groups—including chronic adolescent offenders and ethnic minority youth—should be actively considered in policies and programs aimed at reducing offender mortality risk. Unfortunately, few studies on risk assessment and intervention strategies for early mortality have been conducted, and more research is needed to support policy-makers and practitioners in the field. Such research is particularly important with respect to specific mortality outcomes such as drug-overdose deaths, which are currently rampant in North America. Despite the lack of research on early mortality prevention strategies, findings from the current study clearly indicate that the risk of experiencing early mortality should be communicated to young offenders as early and as clearly as possible, as has been suggested by previous researchers in the field (Lattimore et al., 1997).

In addition to providing the first comprehensive comparison of Tremblay and Paré’s three models, the current study also demonstrated the important practical and theoretical applications of SNA in the context of early mortality research. Social network data were used to more test whether greater exposure to conflict and victimization across the life course was associated with early mortality (as posited by the occupational-hazard model). Findings showed partial support for the occupational-hazard model as deceased offenders were found to have a higher proportion of—but not total number of—negative criminal ties in their ego-networks. Comparatively, non-deceased offenders had accumulated more positive criminal social capital across their criminal careers, suggesting that positive criminal ties may help insulate offenders from the risks posed by negative actors in a criminal network. These results suggest that intervention strategies that foster healthy conflict resolution skills and psychosocial development may help protect against early mortality by reducing the proportion of negative criminogenic ties in offenders’ networks. More generally, findings from the study demonstrate that policies, practices, and future research must actively consider
how an individual’s embeddedness within a particular social environment may contribute to early mortality. Focusing on individual risk factors alone ignores the principle of interdependence, and as such, may neglect key risk and protective factors associated with early mortality.
References


Appendix A.

Age-adjusted network analyses

<table>
<thead>
<tr>
<th></th>
<th>Deceased</th>
<th>Non-Deceased</th>
<th>t, p, d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
</tr>
<tr>
<td>Centrality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree centrality*</td>
<td>.01 (.01)</td>
<td>.01 (.01)</td>
<td>t(71) = 2.25, p &lt; .05, .52</td>
</tr>
<tr>
<td>Betweenness centrality*</td>
<td>.02 (.02)</td>
<td>.03 (.02)</td>
<td>t(71) = 1.18, p &lt; .10, .41</td>
</tr>
<tr>
<td>Cohesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective size†</td>
<td>3.38 (3.03)</td>
<td>5.06 (3.49)</td>
<td>t(71) = 2.19, p &lt; .05, .51</td>
</tr>
<tr>
<td>Density</td>
<td>.03 (.04)</td>
<td>.03 (.02)</td>
<td>t(72) = .31, ns, .07</td>
</tr>
<tr>
<td>Average nodal degree</td>
<td>1.17 (.20)</td>
<td>1.41 (.34)</td>
<td>t(72) = 3.62, p &lt; .01, .84</td>
</tr>
<tr>
<td>Negative vs positive ties</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Criminal social capital ties†</td>
<td>1.24 (2.01)</td>
<td>4.46 (5.59)</td>
<td>t(71) = 3.25, p &lt; .01, .76</td>
</tr>
<tr>
<td>Negative ties†</td>
<td>1.54 (1.52)</td>
<td>1.95 (1.54)</td>
<td>t(71) = 1.16, ns, .27</td>
</tr>
<tr>
<td>Proportion negative ties</td>
<td>.43 (.19)</td>
<td>.33 (.14)</td>
<td>t(72) = -2.48, p &lt; .01, .27</td>
</tr>
<tr>
<td>Gang members</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of gang members</td>
<td>4.78 (3.82)</td>
<td>3.94 (4.10)</td>
<td>t(72) = -.08, ns, .02</td>
</tr>
<tr>
<td>Proportion gang members</td>
<td>.12 (.13)</td>
<td>.08 (.08)</td>
<td>t(72) = -1.75, ns, .42</td>
</tr>
</tbody>
</table>

Note. † indicates age-adjusted; * indicates age adjusted and normalized.
### Age-adjusted comparison of deceased and non-deceased egos in main component of victimization network

<table>
<thead>
<tr>
<th></th>
<th>Deceased M (SD)</th>
<th>Non-Deceased M (SD)</th>
<th>t, p, d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Centrality</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-degree centrality*</td>
<td>.005 (.003)</td>
<td>.004 (.005)</td>
<td>t(25) = .51, ns, .22</td>
</tr>
<tr>
<td>Out-degree centrality*</td>
<td>.017 (.007)</td>
<td>.018 (.010)</td>
<td>t(26) = -.31, ns, .13</td>
</tr>
<tr>
<td>Betweenness centrality*</td>
<td>.001 (.001)</td>
<td>.001 (.001)</td>
<td>t(28) = -.15, ns, .00</td>
</tr>
<tr>
<td>Proportion of outgoing vict.</td>
<td>.78 (.14)</td>
<td>.83 (.16)</td>
<td>t(28) = .79, ns, .32</td>
</tr>
<tr>
<td><strong>Cohesion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective size†</td>
<td>1.4 (.38)</td>
<td>1.5 (.75)</td>
<td>t(25) = .45, ns, .17</td>
</tr>
<tr>
<td>Density</td>
<td>.001 (.003)</td>
<td>.002 (.004)</td>
<td>t(28) = .46, ns, .19</td>
</tr>
<tr>
<td>Average nodal degree</td>
<td>.021 (.043)</td>
<td>.047 (.082)</td>
<td>t(28) = .85, ns, .38</td>
</tr>
<tr>
<td><strong>Gang</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of gang members</td>
<td>2.56 (1.34)</td>
<td>1.95 (1.68)</td>
<td>t(28) = -.92, ns, .36</td>
</tr>
<tr>
<td>Proportion gang members</td>
<td>.21 (.14)</td>
<td>.18 (.23)</td>
<td>t(28) = -.39, ns, .18</td>
</tr>
</tbody>
</table>

*Note.* † indicates age-adjusted; * indicates age adjusted and normalized