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Original Article

Someone to live for: effects of partner and dependent children on preventable death in a population wide sample from Northern Ireland

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ABSTRACT

How to allocate resources between somatic maintenance and reproduction in a manner that maximizes inclusive fitness is a fundamental challenge for all organisms. Life history theory predicts that effort put into somatic maintenance (health) should vary with sex, mating and parenting status because men and women have different costs of reproduction, and because life transitions such as family formation alter the fitness payoffs from investing in current versus future reproduction. However, few tests of how such life history parameters influence behaviours closely linked to survival exist. Here we examine whether specific forms of preventable death (accidents/suicides, alcohol-related causes, and other preventable diseases) are predicted by marital status and dependent offspring in a modern developed context; that of Northern Ireland. We predict that men, non-partnered individuals and individuals who do not have dependent offspring will be at higher risk of preventable death. Running survival analyses on the entire adult population (aged 16–59, $n = 927{,}134$) controlling for socioeconomic position (SEP) and other potential confounds, we find that being single (compared to cohabiting/married) increases risk of accidental/suicide death for men (but not for women), whereas having dependent children is associated with lower risk of preventable mortality for women but less so for men. We also find that the protective effect of partners is larger for men with low SEP than for high SEP men. Findings support life history predictions and suggest that individuals respond to variation in fitness costs linked to their mating and parenting status.

1. Introduction

1.1. Background

Individual health can have large impact on fitness outcomes. All else equal, healthier individuals, those in good physical and mental condition, are more attractive as partners and more likely to conceive and successfully raise offspring. Better health is also associated with a longer life span which enables a higher number of reproductive opportunities and/or a higher cumulative investment in offspring. There are thus clear fitness benefits associated with investment in health. However, because a unit investment in health (somatic effort) cannot also be invested in other fitness enhancing activities, individuals face a trade-off between investing in health and other fitness generating activities, i.e. mating or parenting effort (Stearns, 1992). Life history theory predicts that individuals should be more likely to participate in activities that are detrimental to health or likely to shorten life span when such activities are expected to lead to reproductive benefits. This might in part explain why many behaviours that greatly increase morbidity and risk of death are common. In developed countries smoking, poor diet, excessive alcohol consumption and accidental deaths related to risk-taking behaviours are leading causes of premature mortality (Mokdad, Marks, Stroup, & Gerberding, 2004) and widely practiced even when they are known to be harmful and resources to prevent them are available (Buck & Frosini, 2012; Hill, 1993). Understanding that human behaviour has been shaped by natural selection to maximize inclusive fitness, not individual health or longevity, provides an ultimate explanation for why achieving behavioural change continues to prove challenging.

The extent to which individuals engage in activities detrimental to health varies greatly. Life history theory predicts that individuals will incur different fitness costs and benefits from shifting resources from somatic maintenance to reproduction depending on their sex, resource access and life stage (marital and parenting status). Much of the variation in preventable death outcomes can be explained by socioeconomic factors (Marmot, 2005). Socioeconomic differences in health effort and mortality outcomes have previously been explored from an evolutionary perspective (e.g. Nettle, 2010a, 2010b, 2011; Pepper & Nettle, 2014). One proposed explanation for these patterns is that individuals with lower socioeconomic position (SEP) have less to gain from investing in preventative health efforts since it is less likely that they will live to reap subsequent benefits (Nettle, 2010b). This prediction rests on the assumption that low SEP individuals have higher extrinsic mortality, i.e. mortality not easily mitigated by individual effort. In this paper, we focus on less studied factors related to reproduction—mating and parenting status—that should

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shift fitness benefits associated with health investments. Below we lay out the life history predictions for these factors and review existing evidence from the public health literature.

1.2. Sex, mating status and health effort

Men and women differ in a number of ways with regard to reproduction. These differences impact health and risk-taking behaviour. Because females invest most in physiological reproduction (i.e. gestation and lactation), males are anticipated to compete over females. Such competition may include hazardous, or risk-taking behaviours as a form of mating effort e.g. physical violence (Kruger & Nesse, 2006; Williams, 1966). Risk-taking males might be attractive to females as they display physical prowess or ability to outcompete other men (Daly & Wilson, 2001). While women are constrained to a maximum of one reproductive event per year, men can father children by several women simultaneously. Generally, successful men can thus increase fitness by attaining multiple mates in a way that females cannot (but see Brown, Laland, & Borgerhoff Mulder, 2009; Kokko & Jennions, 2008). This means that for men, the mean return in reproductive success of engaging in competitive activities might be higher on average than a less competitive strategy, even though the majority of men will suffer the adverse outcome rather than the rarer payoff (Trivers, 1972). Females, on the other hand, should generally have lower fitness payoffs from hazardous forms of mating effort. Importantly, male–male competition need not be violent, nor lead to mortality. Both under which conditions higher intrasexual competition is expected and the nature of such competition (whether higher competition should lead to more violence) has recently been questioned (Kokko & Jennions, 2008; Schacht, Rauch, & Borgerhoff Mulder, 2014). However, sex differences in mortality have been reported to be highest in years of high mating competition, higher among never married and low SEP individuals (Kruger & Nesse, 2006). An illustrative example of sex differences in mortality comes from communities where no individuals engage in mating or reproductive effort (monasteries of monks and nuns): Luy (2003) found that sex-specific mortality was only slightly lower for nuns than monks, and this difference was due to a longer life span of the non-reproductive men, rather than a shorter life span of the non-reproductive women. The sex differences in cost of reproduction lead to the expectation that single individuals (and in particular men), should be more likely to engage in health neglect and risk-taking than women or coupled individuals.

1.3. Parenting status and health effort

Once a mate has been found and family formation has begun, individuals have lower returns to costly forms of mating effort. Individuals are predicted to benefit more from avoiding illnesses and injury when they care for dependent offspring, at least when parental care is important for child outcomes. Sear and Mace (2008) and Lamb (2004) have shown that offspring are more likely to survive and have positive health outcomes if mothers and fathers—the latter at least in western contexts—are present. Because mothers invest most in offspring and their loss is more detrimental to children, mothers are predicted to take more precautions with their health than fathers. However, men also alter behaviour from mating to parenting effort over the life course. Testosterone, the reproductive hormone associated with violent and risk-prone behaviour, is lower in married than non-married men, decreases with fatherhood (Gettler, McDade, Feranil, & Kuzawa, 2011; Gray, Kahlenberg, Barrett, Lipson, & Ellison, 2002) and is lower in fathers who invest more in offspring (Alvergne, Faurie, & Raymond, 2009; Muller, Marlowe, Bugumba, & Ellison, 2009). Some recent evidence has shown similarities in functional brain activation in primary caregiving mothers, secondary caregiving heterosexual fathers and primary caregiving homosexual fathers in response to infant stimuli (Abraham et al., 2014). Thus, changes in testosterone level and malleability of brain functions shed light on the proximate mechanisms that regulate male allocation to parenting versus mating effort. Whether such changes translate to health behaviour with impact on life span and survival in modern contexts is less well understood.

1.4. Public health evidence

Public health studies examining differences in preventable mortality between married and non-married individuals, and those with and without children can provide useful insights into variation in health allocations. These studies, often based on large-scale demographic data from western countries, generally find that married individuals have better overall health (Schoenborn, 2004) and lower mortality than non-married peers (Ben-Shlomo, Smith, Shipley, & Marmot, 1993). However, a large proportion of the literature on marital status and mortality focuses on either overall mortality (Johnson, Backlund, Sorlie, & Loveless, 2000) or specific death outcomes (e.g. ischemic heart disease) (Manor, Eisenbach, Israeli, & Friedlander, 2000) that are difficult to interpret as they are not linked to particular individual behaviours. Less is known about cause-specific mortality (Silventoinen, Moustgaard, Peltonen, & Martikainen, 2013) or preventable diseases. Some evidence suggests that not being married incurs higher risk of accidental death (Burrows, Auger, Gamache, & Hamel, 2012), suicide (O'Reilly, Rosato, Connolly, & Cardwell, 2008; Qin, 2000; Silventoinen et al., 2013) and alcohol-related death (Connolly, O'Reilly, Rosato, & Cardwell, 2011; Koskinen, Joutsenniemi, Martelin, & Martikainen, 2007). It has also been suggested that marriage decreases risk of all-cause mortality more for men than for women (Kaplan & Kronick, 2006; Kposowa, 2000; Shor, Roelfs, Bugyi, & Schwartz, 2012; Staehelin, Schindler, Spoerri, & Zemp Stutz, 2012 but see Lund et al., 2002) but few examples of a sex-specific effect of marriage on preventable mortality outcomes exist.

There are also associations between parity or dependent children and mortality. Women without children have higher risk of all-cause mortality (Jaffe, Neumark, Eisenbach, & Manor, 2009), accidental and alcohol-related death (Grundy & Kravdal, 2010) than women with children. In a Danish study, having a child less than two years old decreases risk of suicide death for women, but not for men (Qin, 2000). Studies examining the effect of children on men's risk of death are scarce, and attempts to adjust for living arrangements when examining the relationship between parity or dependent children and mortality are often poor (Koskinen et al., 2007). However, a study of Swedish men found that childless men who were cohabiting with a partner had higher risk of death from suicide, external violence and addiction, compared to men who were custodial fathers and cohabiting with a partner (Ringbäck Weitoft, Burström, & Rosén, 2004).

1.5. Aims

Overall, non-married and childless individuals appear to have higher risks of accidental, alcohol-related deaths, suicide and all-cause mortality. However, at present it is difficult to draw any firm conclusion about these effects, because confounds and ages of individuals vary substantially between studies. It is important to test effects of cohabitation/marital status and dependent children alongside each other as it is otherwise difficult to isolate effects. Many studies also include adults beyond pensionable age (e.g. Connolly et al., 2011; O'Reilly et al., 2008). Because selection pressures should be stronger on traits that exhibit themselves earlier in life (Medawar, 1952; Williams, 1957), and these behaviours are assumed to be related to reproductive trade-offs, we are interested in the effect of marriage and offspring in individuals in young and middle age, rather than throughout the life course.

Furthermore, few studies compare outcomes that are linked to risk-taking behaviour to those related to health neglect caused by more long-term habits. Risk is the unpredictability in outcome of a significant behaviour (Winterhalder, 2007) and different from hazardous or unsafe behaviours that are highly likely to have a negative impact on individual

health or survival. Risk-taking behaviours (e.g. reckless driving or violence), and health neglect that lead to health deterioration over time (e.g. smoking or drinking) could be considered to belong to separate domains, but life history theory predicts that both types of behaviours should be higher in men, non-partnered individuals and those without dependent children. Another important contribution from life history theory to public health is the prediction that the effects of sex, marital status and dependent offspring on likelihood of health effort might vary with the individual's life history strategy. If men with lower SEP (a reliable proxy for faster life history pace (e.g. Nettle, 2010a)) engage in risky behaviours as mating effort (because they do not have wealth to attract mates with) then the sex difference in mortality should be larger among low SEP than high SEP individuals. We might also predict that shifting from mating to parenting effort should lead to a larger reduction in preventable mortality risk among low SEP men than high SEP men, since the latter should be less likely to have had a mating strategy that involved risk-taking and health neglect to begin with.

We use the Northern Ireland Mortality Study (NIMS) with data on the entire adult population (aged 16–59, $n = 927{,}134$) enumerated in the 2001 Census to examine risk of death during a follow up period of nearly 9 years. We examine effect of marriage or partner cohabitation (henceforth referred to as "cohabitation") instead of relying only on marital status, as cohabiting with a partner should also imply less mating effort. Rather than parity, often used in other studies, we test effects of dependent children in the household as it captures whether offspring is currently dependent on the parent as a carer. We predict that cohabiting with a partner and dependent children should both decrease risk of preventable death, that the protective effect of a partner should be larger for men (who should alter their health/risk behaviour more when no longer looking for a mate), and the effect of dependent offspring larger for women (who are the main carers).

2. Methods

2.1. Data

Northern Ireland Mortality Study (NIMS) is a database from Northern Ireland handled by Northern Ireland Statistics and Research Agency (NISRA). The database comprises in total c. 1.6 million individuals who resided in Northern Ireland on Census day, 29th of April 2001, with an 8.7 year follow up period. The data are held in a safe setting by NISRA and made available for this study. No ethical permission was required.

2.1.1. Preventable mortality outcomes

Our data comprise every death that occurred in Northern Ireland during the study period (see above) to a person aged 16–59 in 2001, enumerated at the 2001 Census. Causes of death were recorded by the GP as international classifications of diseases (ICD-10 codes). We analyze all death outcomes deemed preventable (as opposed to amenable to health care, or unavoidable) according to the classification by Page et al. (2006). Three large categories of preventable deaths emerge in our sample (for a complete list of ICD-10 codes for each category, see Wheller et al. (2007):

i, Accidental deaths and suicides. Deaths in this category are mostly falls, traffic accidents and undetermined intent (potential suicide), violence, homicide, or exposure to drugs. Although suicides (n = 890) and accidental deaths (n = 1256) are distinct causes, they are collapsed here because they share similarities such as being instantaneous, and also because they are sometimes difficult to distinguish. Some accidental deaths ($n = 43$ of $n = 2146$) involved alcohol as a contributing cause. These deaths were included both in the present and following category. Our definition differs slightly from the Page et al version as we also included deaths from car accidents with "car or other heavy vehicle" (ICD-10 codes V.43 and V.44, n = 86), accidental poisoning (X.44, n = 37) and a few rare (n < 10) disclosive deaths). However when running models for all preventable deaths, we follow the Page et al definition for sake of comparability to other published studies.

ii, Alcohol-related deaths. The majority of this group consists of liver-related diseases (cirrhosis of the liver) caused by long-term excessive alcohol consumption. Although death from alcohol is likely to be caused by long-term excessive drinking that deteriorates health over time, alcohol-related death is not negligible among younger individuals (see supplementary material, S1).

iii, Other preventable causes. These are all other medical causes that are deemed preventable by medical intervention or public health initiatives. Among the other diseases ($n = 5907$), the single largest category are lung cancer deaths ($n = 1572$), other preventable cancers ($n \sim 2400$), diseases of the circulatory system ($n \sim 365$), diseases of the respiratory system ($n \sim 500$), and diseases of the digestive system ($n \sim 250$) also contributing. The numbers given here are not exact in order to protect anonymity.

2.1.2. Independent variables

Our sample includes all individuals aged 16–59 at the 2001 Census. The lower age restriction was set because only individuals age 16 or over have socioeconomic data. Age was capped at 59 because we are interested in behaviours that might be associated with reproductive effort, and because selection pressures should be stronger for traits exhibited earlier in life. Capping age at 59 generates an age-span where deaths go up to 67, when individuals might still suffer mortality from lag from e.g. alcohol or health neglect, but not too old to capture accidental deaths correlated with old age rather than mating effort. For mating status we combine marital status and current living arrangements, as to measure cohabitation with partner rather than just marital status. Dependent offspring are children (aged 18 or younger) who reside in the household at the time of the 2001 Census. Because some individuals are themselves children cohabiting with their parents, we constructed the following categories: coresident with parents (ego is him/herself a child in the household), and for adults: no dependent children in the household, or 1 or more dependent children in the household. Because having no dependent children in the household can mean that offspring have moved out, we split the "no children in household" category into younger (aged 44 and younger) and older (aged 45 and older). Forty-four was chosen as the cut-off point as at this age most women who will have children have during their life time will have reproduced. We include data on a number of different socioeconomic variables: household car access (0, 1 or 2 or more), housing tenure (social housing, privately rented or privately owned), education (no education, lower secondary (1 A level or GCSEs), upper secondary (2 A-levels or more), or university degree or higher. We also include economic activity at Census (active, unemployed, student, retired, homemaker, permanently ill or other inactive). Economic activity was a better fit than socioeconomic class, and better reflects current resource access. For models run separately by SEP, we created an index based on tenure, car access and education, where each increasing level of SEP corresponded to one point, rendering a scale of increasing SEP from 0 to 7. Lastly, we include area of residence (Belfast, Derry, towns, rural areas) and community background (Catholic, Protestant, none/other). We exclude people ($n \sim 21\ 000$) who reside in communal establishments since these individuals lack socioeconomic variables. Ethnicity was not included since less than 0.4% had another ethnicity than "white". Northern Ireland has had very low levels of immigration as a result of recent domestic unrest associated with sectarian conflict (for full descriptive statistics by sex, see SM, Table S1 and S2).

2.2. Statistical analysis

We run Cox proportional hazard models, a type of survival analysis, with preventable mortality events as outcomes, separately for men and women (with a total 7,967,318 person–years at-risk). Cox models are semi-parametric and can handle "right" censored data (i.e. those that are still alive at the end of the study period). The proportional hazards (PH) assumption, that the hazards do not change over the study period, was checked according to Box-Steffensmeier and Zorn (2002) and fulfilled for all variables apart for age. However, an increased risk of death with age was expected, and a large sample size is more likely to result in a significant slope of the residuals and thus violate the PH assumption. After inspection of the Schoenfeld residuals we conclude that the overlap of age categories is very minor, and we proceed without altering the models. We rely on Akaike Information Criterion, AIC, to compare models (Burnham & Anderson, 2002) where at least 2 points lower AIC value implies a better fit. We tested for differences between electoral ward (c. 2900 people) by running models with shared frailty (i.e. random intercepts for survival models), but once individual SEP was controlled for there was no significant variation in preventable mortality between wards (models with shared frailty had worse fit) and thus no reason to adjust for clustering.

3. Results

The absolute risk of preventable death is low: 1.3% of men and 0.6% of women who were aged 16–59 in 2001 died from preventable causes in the following 8.7 years. Preventable causes of death are responsible for almost half (49.8%) of the total number of deaths in this age group. Overall, 57% of men are cohabiting with a partner, however only 16% of all preventable deaths are to cohabiting men (see Table 1). Among women there is no such overrepresentation of preventable deaths among non-cohabiting women (58% are married, and 59% of preventable deaths are to cohabiting women). Thirty-seven percent of men and 46% of women co-reside with dependent children, and for both sexes mortality is lower for these individuals (27% and 32% respectively). Individuals with lower SEP were markedly over-represented for all causes of preventable death (see supplementary material, Table S1 and S2).

3.1. Sex differences in risk of preventable death

As predicted, the risk of death is higher for men than women for all types of preventable mortality. Sex differences in mortality are high and largest for accidental/suicide death where women have 70% lower hazards of death than men (HR 0.30, 95% CI 0.27–0.34). Women have 55% lower hazards of alcohol-related death and 34% lower hazards of other preventable diseases than men (see Table S3).

3.2. Effect of marital status on preventable death

Single men have higher hazards of mortality than cohabiting men for accident/suicide death, but not for alcohol-related death, or death from other preventable diseases (Table 2). The hazard ratios are large for accidental/suicide death for single (HR 1.58, 95% CIs 1.32–1.88) and formerly married (HR 1.68, 95% CIs 1.39–2.02), compared to currently cohabiting men. Formerly married/cohabiting men have higher risk of death than cohabiting men for all causes. Formerly married/cohabiting women have 29% higher hazards for alcohol-related death than currently married/cohabiting women, but surprisingly slightly lower hazard of other preventable death than married/cohabiting women (see Table 2). All models with an interaction term for marital status and sex fit the data better than the main model, as they have at least 3 points lower AIC (see SM, Table S4 and S5). These interactions reveal that the effect of partner cohabitation is larger for men than for women for all outcomes.

3.3. Effect of dependent children on preventable death

Young men without dependent children do not have higher hazards of accidental death; however, for alcohol-related death, men without dependent children have 63–95% higher hazards (depending on age) than men with dependent children (see Table 2). Women who do not have dependent children have higher hazards of any preventable death than women who have dependent children. The decreased risk of death for women with dependent children is especially large for accidents/suicides (women aged 16–44 without dependent children have 41% increased hazards) and alcohol-related deaths (143% higher hazards) than those caring for at least one child. Interaction models showed that there were no significant differences in the effect of children by sex for accidental/suicide or alcohol-related death, but that for other preventable diseases: women without dependent children had higher hazards compared to peers with children, as compared to the difference in hazards between men with and without children (see Tables S4 and S5).

3.4. Effect of partner cohabitation and dependent children by socioeconomic position (SEP)

We run models separately by SEP to examine whether effects of partner and dependent offspring vary by level of resources and

Table 1
Distributions of deaths by marital status, dependent children and socioeconomic position (SEP).

	Total sample		Accidental/Suicide death		Alcohol-related death		Other preventable death		Preventable deaths (total)	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
	$n = 454497$	$n = 472653$	$n = 1609$	$n = 537$	$n = 827$	$n = 435$	$n = 3635$	$n = 2272$	$n = 5914$	$n = 3161$
Marital status										
Single	38%	31%	46%	29%	29%	15%	19%	15%	57%	17%
Married/Cohabiting	57%	58%	42%	46%	41%	50%	66%	64%	16%	59%
Formerly married	6%	11%	12%	25%	30%	35%	15%	21%	27%	24%
Dependent children in household										
1 child or >	37%	46%	28%	41%	20%	35%	28%	30%	27%	32%
No child (& aged 44 or <)	16%	14%	18%	14%	18%	14%	6%	5%	10%	8%
No child (& aged 45 or >)	18%	20%	23%	30%	48%	46%	58%	58%	48%	52%
Coresiding with parents	29%	20%	31%	15%	14%	5%	9%	7%	15%	8%
By SEP										
Low SEP	33%	34%	54%	55%	73%	67%	56%	58%	58%	59%
High SEP	67%	66%	46%	45%	27%	33%	44%	42%	42%	41%

SEP is an index variable based on highest level of education, housing tenure, household car access. Presence of dependent children in household is split into two, (for individuals aged 44 and younger, or, aged 45 and older). Coresiding with parents implies ego is him/herself a child/youth in the household and coresiding with parents.

Table 2
Cox regressions for preventable death outcomes.

	Accidental death/suicide		Alcohol-related death		Other preventable death		Preventable deaths total	
	Men	Women	Men	Women	Men	Women	Men	Women
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Marital status								
Married/Cohabiting	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Single	1.58 (1.32, 1.88)	1.17 (0.87, 1.58)	1.05 (0.84, 1.32)	0.84 (0.60, 1.18)	1.01 (0.90, 1.14)	0.97 (0.83, 1.14)	1.10 (1.01, 1.21)	0.96 (0.84, 1.10)
Formerly married	1.68 (1.39, 2.02)	1.65 (1.30, 2.11)	1.80 (1.48, 2.21)	1.29 (1.01, 1.65)	1.12 (1.00, 1.24)	0.89 (0.79, 0.99)	1.32 (1.21, 1.43)	1.02 (0.93, 1.13)
Dependent children in household								
1 child or >	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
No child (& aged 44 or <)	1.03 (0.86, 1.24)	1.41 (1.06, 1.89)	1.63 (1.25, 2.14)	2.43 (1.76, 3.37)	1.19 (0.99, 1.42)	1.28 (1.03, 1.59)	1.27 (1.13, 1.42)	1.54 (1.32, 1.80)
No child (& aged 45 or >)	1.29 (1.06, 1.58)	1.51 (1.11, 2.07)	1.95 (1.53, 2.47)	1.94 (1.44, 2.62)	1.23 (1.03, 1.46)	1.86 (1.48, 2.34)	1.28 (1.19, 1.39)	1.30 (1.17, 1.45)
Coresiding with parents	0.86 (0.69, 1.07)	1.32 (0.90, 1.95)	2.07 (1.54, 2.80)	2.05 (1.21, 3.47)	1.17 (1.07, 1.28)	1.18 (1.05, 1.34)	1.27 (1.12, 1.43)	1.82 (1.51, 2.19)

Models control for age, economic activity, highest level of education, housing tenure, household car access, community background (Catholic, Protestant, none), and residence (Belfast, Derry, small town, rural. Presence of dependent children in household is split into two, as some children of older individuals could have moved out (i.e., no children in household (for individuals aged 44 and younger, or aged 45 and older). Coresiding with parents implies ego is him/herself a child/youth in the household and coresiding with parents. HR = hazard ratios, CI = confidence intervals. n = 454497 for men and n = 472653 for women.

education. A low SEP is strongly correlated to a faster life history pace, higher mortality and more health neglect and risk-taking behaviour. It is therefore likely that any effects of partner or dependent children might interact with SEP. Because running separate models by SEP limits sample size, we examine accidental deaths and alcohol-related deaths combined. The AIC comparisons for SEP interactions can be found in the Supplementary material (Tables S6 and S7). Being single (compared to cohabiting) is associated with a larger hazard of accidental/suicide/alcohol death among low SEP men, than among high SEP men (Table 3). In other words, men with low access to resources have a higher relative disadvantage of not cohabiting than better off men as compared to their cohabiting peers. For other preventable causes of death, there are no significant differences between single and cohabiting men for either high SEP or low SEP. Among women, there is no difference in the effect of partner cohabitation among either high or low SEP individuals (Table 3, SI S6 and S7). Low SEP men with no dependent children have greater hazards of accidental/suicide/alcohol death compared to peers with children, than do high SEP men with no dependent children. Women who do not have dependent children have higher hazards of accidental/suicide/alcohol death regardless of SEP, but for other preventable diseases only low SEP women without children have higher mortality hazards (Table 3).

4. Discussion

4.1. Main findings

We find that cohabitation with partner and dependent children overall decrease preventable mortality risks but that these effects vary by sex, SEP and between different types of preventable death. Our findings, based on nearly every adult in Northern Ireland, are novel as they concern outcomes related both to risk-taking and health neglect, and test heterogeneity of effects by sex and SEP. Effects are mostly in the predicted directions. The largest sex differences as well as the largest protective effects of partner cohabitation are for accidental or suicide death. This category of deaths is instantaneous and limits doubt about time lags from behaviour to death. That not having a cohabiting partner was associated with a higher increased risk of death for men than women is in line with the prediction that men act in ways that incur higher mortality risks when searching for a partner. It confirms some previous findings (e.g. Kposowa, 2000) but is here extended for multiple preventable mortality outcomes. Dependent children in the household had as expected protective effects on all preventable mortality outcomes for women, but for men effects varied depending on the mortality outcome: young men without dependent children had higher risks for alcohol-related deaths, but

Table 3
Cox regressions for preventable death outcomes by socioeconomic position (SEP).

	Accidental, suicide or alcohol-related death				Other preventable death			
	Men		Women		Men		Women	
	Low SEP	High SEP	Low SEP	High SEP	Low SEP	High SEP	Low SEP	High SEP
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Marital status								
Married/Cohabiting	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Single	1.57 (1.33, 1.86)	1.35 (1.04, 1.76)	1.08 (0.83, 1.42)	1.25 (0.83, 1.89)	1.09 (0.95, 1.25)	0.98 (0.78, 1.24)	1.01 (0.84, 1.22)	1.01 (0.75, 1.37)
Formerly married	2.06 (1.76, 2.41)	1.82 (1.37, 2.41)	1.64 (1.35, 2.00)	1.56 (1.10, 2.21)	1.18 (1.04, 1.33)	1.10 (0.88, 1.37)	0.89 (0.78, 1.02)	1.05 (0.84, 1.32)
Dependent children in household								
1 child or >	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
No child (& aged 44 or <)	1.37 (1.12, 1.68)	1.04 (0.81, 1.34)	2.08 (1.56, 2.70)	1.67 (1.15, 2.41)	1.36 (1.07, 1.72)	1.04 (0.78, 1.39)	1.64 (1.23, 2.19)	1.04 (0.75, 1.45)
No child (& aged 45 or >)	1.60 (1.31, 1.96)	1.46 (1.15, 1.84)	1.84 (1.39, 2.43)	1.45 (1.03, 2.06)	1.26 (1.10, 1.43)	1.10 (0.97, 1.25)	1.22 (1.03, 1.44)	1.12 (0.93, 1.34)
Coresiding with parents	1.18 (0.94, 1.47)	1.07 (0.78, 1.48)	1.26 (0.81, 1.95)	1.51 (0.89, 2.55)	1.17 (0.94, 1.46)	1.36 (0.99, 1.86)	1.54 (1.13, 2.10)	2.11 (1.44, 3.10)

Models control for age, economic activity, SEP index*, community background (Catholic, Protestant, other/none), and residence (Belfast, Derry, small town, rural). Coresiding with parents implies ego is him/herself a child in the household and coresiding with parents; SEP index is based on highest level of education, housing tenure, and household car access. HR = hazard ratios, CI = confidence intervals. n = 454497 for men and n = 472653 for women.

offspring had no effect on young men's hazard for accidents/suicides or other preventable diseases.

Among both men and women, low SEP individuals have higher protective benefits of caring for dependent offspring than high SEP individuals. Low SEP men also have higher comparative protective effects of having a cohabiting partner than high SEP men. This difference may be interpreted as individuals with low access to resources being more likely to compete for mates through health adverse behaviours, whereas high SEP individuals might compete for mates in ways that do not result in death e.g. through wealth. It is often assumed that it is a favourable strategy for men to engage in hazardous behaviours. This might only be true when undesirable outcomes are far off, sex ratio are skewed (Kokko & Jennions, 2008; Schacht et al., 2014) or for a certain stratum of society where females favour such behaviour. In other (high SEP) strata the opposite, risk-averse, (e.g. investing in education) might be desirable behaviours. Our results confirm some previous findings showing a particular heightened risk of all-cause mortality with divorce (Hemström, 1996), and parity (Kotler & Wingard, 1989) among women with low SEP. It is also notable that the effect of marriage/cohabitation is actually stronger among low SEP men, even though poorer people tend to have weaker marital stability than those who are better off (O'Connor, Pickering, Dunn, Golding, & Team, 1999). It is interesting to note that low SEP individuals, who are much more likely to favour early birth, face higher risk of death if not having a partner or delaying birth, than do high SEP individuals.

These results are noteworthy especially since it has been debated whether a trade-off between mating and parenting effort exists (Stiver & Alonzo, 2009). In humans, investing in own health might increase both offspring's prospects and the individual's ability to attract mates. We are not explicitly testing whether such a trade-off exists but demonstrate that mating and parenting status is associated with mortality outcomes in the predicted directions, and that these effects are higher among low SEP individuals.

4.2. Reverse causality and alternative explanations

Despite using longitudinal data, many studies examining the effect of marital status and children on mortality might suffer from issues of reverse causality (Cheung, 2000). It is possible that single individuals are more likely to die from risky causes if they are on a life history trajectory that is associated with higher future discounting and means that they are less likely to form and maintain relationships. Such strategies might be shaped early in life well before the baseline of these studies. Our study is not exempt from this issue, but does include multiple outcomes and substantial socioeconomic controls, including an aspect of long-term illness in the "economic activity" variable, accounting somewhat for the possibility that healthier individuals might be more likely to be partnered/have children. Selection bias into marriage and protective effects of partner or children are not mutually exclusive, and it is possible that both processes contribute to some extent to the observed patterns. Ultimately, to establish causality data on health behaviour before and during marriage and entry to parenthood is needed.

There might also be various constraint-based explanations for lower risk of death when caring for children, if childcare limits opportunities to partake in hazardous behaviours that might otherwise pose a risk. Another possible causal pathway is that social support of a cohabiting partner might lead to higher probability of survival. Data on social support networks could shed light on whether individuals are being more cautious when in a relationship, or if effects are due to the behaviour of their partner (Grundy & Kravdal, 2010). Furthermore, grief after the death of a spouse might contribute to higher risk of death for formerly married individuals; there is some evidence that widowed men are more likely to increase alcohol consumption (Eng, Kawachi, Fitzmaurice, & Rimm, 2005), and have higher impulsivity (Stroebe, Schut, & Stroebe, 2007) following the death of their partner.

4.3. Limitations

It is unfortunate that we cannot determine whether the dependent children are the individuals' biological children, especially because men are probably more likely to be co-residing with non-biological children of a new partner. This might in part explain why caring for offspring mattered more for women. A limitation of how the data were collected is that cohabitation status and presence of children in household are not necessarily the same at the time of death as when it was measured. It might be problematic that the co-variants are not time-varying if life history traits such as age at marriage and first birth vary with SEP. However, as we are measuring both instantaneous deaths and death from behaviours with longer time-lags, run analyses separately by SEP and have data on the entire adult population, we are fairly confident that this type of issue would not seriously affect our results.

4.4. Future directions

We have shown that there is substantial variation in risk of multiple preventable mortality outcomes with marital status and dependent children and discussed how the differences in magnitude of these effects might reflect different fitness costs and benefits individuals incur. The detail and scale of this database has enabled us focus on mortality closely linked to behaviour, such as accidental and alcohol-related death, and test how predictions vary by life history pace. Evolutionary theory can make important contributions to public health (Gibson & Lawson, 2014; Hill, 1993; Lawson & Uggla, 2014; Nettle, 2010b; Pepper & Nettle, 2014) for example by providing ultimate explanations for socioeconomic determinants of health behaviours and outcomes. We have shown that life history theory can apply ultimate explanations to predict associations between mortality risk and mating and parenting status in a modern developed population. Future research should aim to test whether these effects are robust across different ecologies with varying norms related to gender roles, marriage and childrearing.

Supplementary materials

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.evolhumbehav.2014.07.008.

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