Economic inequality in adult mortality in Canada
Analyses of the Longitudinal Administrative Databank

Jacob Etches

A thesis submitted in conformity with the requirements
for the degree of Doctor of Philosophy

Graduate Department of Public Health Sciences
University of Toronto Faculty of Medicine

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Abstract

This dissertation contains two empirical papers on income and premature mortality, and one methodological paper that concerns the summary measurement of the extent of social inequalities in health.

Income dynamics and adult mortality: Canada and the USA  Chapter 4 examines the effects of income level and income drops on all-cause mortality in Canada and the United States. The Canadian data are from the Longitudinal Administrative Databank (LAD), and the US data are from the Panel Study of Income Dynamics (PSID). The LAD consists of personal income tax records for 20% of Canadian filers from 1982 through 2005. The PSID is a survey sampled in 1968 and followed annually through 1997. Analyses of the PSID confirmed previously published findings that used alternative statistical methods. The effect of income level on hazard of death is twice as large in the United States. The effects of income drops differed in Canada and the United States.

Income dynamics and adult mortality in Canada  Chapter 5 re-analyses the LAD data to refine causal inference regarding the effects of income level and income drops on all-cause mortality. Exposure at ages 40-55 is analyzed for induction times ranging from 1-18 years. Income level was defined as the mean of the previous five year period, and income drops was measured both as annual change, and as the difference between projected and
observed income. The effect of income level attenuated very little over induction time, and was not confounded by work disability. The effect of income drops also attenuated very little over induction time. Men in couple families showed a monotonic dose-response effect of income drops, and exclusion of families with potentially confounding characteristics did not affect the estimated risks. The hypothesized dependency of the effect of income drops on income level was not observed. No differences were observed between the two measures of income drops. Overall, there is strong evidence that the effect of income level on risk of death is primarily causal, while evidence for the effect of income drops is mixed.
Acknowledgements

This work would not have been possible without the support and cooperation of the Small Area and Administrative Data Division at Statistics Canada, and in particular the help of Hung Pham, Jeffrey Smith and André Bernard. I also benefitted from the generosity of René Morissette and Yuri Ostrovsky of the Business and Labour Market Analysis Division at Statistics Canada, who shared with me their work on earnings instability.

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I am also indebted to the authors of several free or open-source software products. These include \LaTeX\ and associated projects (including in particular JabRef); the R System for Statistical Computing, and the ggplot2 package by Hadley Wickham; and Graphviz and the elegant graphical user interface for OS X. The many hours others have volunteered for these projects are greatly appreciated.
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Part I

Literature review
Chapter 1

Aspects of social epidemiology

This literature review chapter lays the foundation for Chapters 4 and 5, which examine the relationship between income level and drops and subsequent mortality, and for Chapter 6 on methods for quantifying the extent of socioeconomic inequalities in health. Section 1.1 provides an overview of the relationship between socioeconomic status and health. Sections 1.3, 1.4, 1.5, 1.6 and 1.7 describe the outcome, exposure and confounding variables for the analyses in Chapters 4 and 5.

1.1 A brief introduction to research on socioeconomic status and health

The relationship between socioeconomic position and health, as understood in social epidemiology, has been usefully summarized by Lynch and Kaplan [137]. Their main points are briefly summarized here. The terms socioeconomic position, socioeconomic status (SES) and social advantage are used interchangeably, and social gradients in health refer to the positive, monotonic association of SES and health.

Systematic study of social gradients in health began in the 19th century with work by Villermé in Paris, Virchow in Upper Silesia (parts of Poland and the Czech Republic), and Farr and Engels in England. The positive correlation between most measures of
social advantage and most measures of health is one of the most consistent findings in epidemiology, and is observed across populations and periods. Social advantage tends to bestow protection from health damaging exposures and access to health enhancing resources.

Measures of health are diverse, covering positive and negative assessments of morbidity, disability, mortality, and composite quality of life measures [49, 154]. With a few exceptions, including most types of cancer [236, 75, 147], social advantage is positively associated with health and longevity, or, conversely, negatively associated with morbidity, disability and mortality [88].

Social epidemiology has drawn on Marx, Weber and Functionalist sociology to understand measures of social advantage. Lynch and Kaplan stylize Marx as defining class based on individuals’ relationship to the means of production; Weber as defining class, status and party which represent economic, social and political power structures, respectively; and Functionalisists as an American tradition of viewing social inequalities as a natural and necessary part of modern society. Weber’s emphasis on the culture and institutions associated with social hierarchy and with the “life chances” or opportunity that social advantage affords aligns well with current indicators of social advantage and with observations of health behaviours.

Indicators of social advantage commonly used in social epidemiology include education, occupation and income, and less frequently wealth [129, 123]. Area-based measures are occasionally examined in their own right (e.g. [250]), but are more often proxies for individual social advantage [172].

These indicators proxy for different resources such as literacy, numeracy, knowledge, skills, purchasing power, financial security, access to information, prestige, respect, appetite for risk and time preference. The direct and indirect effects of dimensions of socioeconomic status on health across the lifecourse are illustrated in Figure 1.1.
**FIGURE 1.1:** An exposure and resources model of the effects of the major dimensions of socioeconomic status throughout the lifecourse. Measures of health are in boxes, and determinants of health in ellipses. Solid lines indicate direct effects on exposures and resources and their consequences for health status. Dotted lines indicate indirect effects of socioeconomic status mediated by other socioeconomic indicators. Dashed lines highlight the feedback from adult health status to work and income via work disability.

Educational attainment marks the transition from a social and economic environment dominated by one’s parents’ socioeconomic status to the formation of one’s own, primarily through labour force participation. Education is a characteristic of both sexes and of the elderly, and is not affected by subsequent disability. The knowledge, reasoning skills, and skills at accessing information that education is designed to transmit may have substantial health benefits unmediated by education’s role as human capital. As human capital, education does not reward all demographic groups equally. Women and minorities typically earn less than equally educated men and majorities.

The study of occupational exposures, and their inequitable distribution, is perhaps the earliest subject in social epidemiology. Work forms the connection between education and income, and is an important source of both physical and psychosocial exposures that have the potential to harm health. Physical exposures include chemical, radiation, biological, biomechanical, noise, heat, cold and particulate exposures [174]. Psychosocial exposures include roles with high demands and low control [113, 202, 23], and those with high effort effort and low rewards [211, 210]. Both of these classic models of psy-
chosocial exposures have important associations with occupational rank. Unpaid work has received too little attention, and is poorly measured in occupational classifications, such that women, especially among older cohorts, are poorly characterized by their own occupational status.

Income is important as an indicator of one's power to purchase protection from exposures and resources to promote health. Such goods and services include housing, transportation, food, medical care, recreation, and child care. In some places and periods, water quality and availability, waste removal, and personal security are or have been private and not public goods. Individual income can, however, be a poor indicator of economic status among labour market entrants, the retired, and married women, for whom probable lifetime earnings, savings, and family income may be better measures, respectively.

Health-promoting behaviours such as making good food choices, not smoking, drinking in moderation, and exercising are usually observed to be positively correlated with socioeconomic advantage. Whether these behaviours are a consequence of one's socioeconomic status or share a common cause is not well understood, nor is their modifiability.

The health benefits of socioeconomic advantages accumulate over the life course, and transmit across generations both via the influence of parental SES and parental community socioeconomic characteristics on achieved socioeconomic status and directly. Examples of the direct transmission of socially graded health to one's children include exposure to teratogens such as lead and alcohol [171], and undernutrition in utero [16].

Despite the compelling mechanisms and strong empirical evidence for a causal effect of socioeconomic status on health, it is important to consider the plausibility of competing explanations for the association of social advantage with health. Four possible types of explanation are typically offered. These are (1) artifact, (2) non-causal, (3) social deter-
mination, and (4) health selection, as named in the influential Black Report [28].

An artifactual explanation would mean either that sampling or measurement errors create an association in datasets which does not exist in the population from which the data are sampled. The relationship would be non-causal if an association between income and premature mortality exists only because of a third variable, such as childhood health, time-preference or self-efficacy, that might be the cause of both SES and health. The artifact explanation is not plausible given number of findings and diversity of methods, and the threat of confounding, while plausibly a contributing factor, is not considered a sufficient explanation of the findings in the literature [213, 74, 88].

By elimination, therefore, the association is predominantly causal. It must be due to either or both of two pathways: social determination and health selection. Social determination refers to the pathways described above in which SES causes declines in health and increased risk of death. Health selection, often termed “reverse causality” in the US literature, refers to the pathway in which compromised health status leads to work disability and reduced income while also increasing the hazard of death (see Figure 1.1).

The scientific consensus, particularly in the epidemiological literature, is that the relationship is causal and primarily, but not exclusively, a result of the effect of income on health and longevity [70, 213, 142]. It has also been argued that health selection has been dismissed too readily and is under-researched [244]. Indeed, very few studies have tried to quantify the contribution of each pathway. One controversial attempt to do this was that of Adams et al. [2]. They observed no causal effect of income on health in their sample, though they admit that this is less surprising given that it is a retired and medically insured population that may have already experienced significant survival effects. Others have shown that selective survival by wealth during the pre-retirement period can be substantial [10].

There is a spirited debate among social epidemiologists about the relative contribu-
tions of the material, behavioural and psychosomatic pathways by which socioeconomic status “gets under the skin.” The answer likely depends on the SES indicator and the health outcome in question. There are also rich relationships between these pathways, particularly running from material conditions to health behaviour.

For the rest of this study, the emphasis will be on income, income dynamics, and premature, all-cause, adult mortality. The complexity of the wider context of socioeconomic status and health will be drawn upon only as necessary. It is assumed that the effects of income are mediated by material, behavioural and psychosomatic pathways acting on a variety of timescales.

1.2 Model of mechanism

The mechanism by which income level and income drops are hypothesized to affect risk of death is illustrated in Figure 1.2. Determinants of family earned income include marriage, births, retirement, family breakdown, job loss, family deaths and work disability. Disposable income is determined by earned income, taxes and transfers. Cash constraint is determined by disposable income and liquid an illiquid assets. Health-relevant consumption is determined by cash constraint, and non-consumption health behaviours and psychosomatic effects are determined cash constraint, births, retirement, family breakdown, job loss, family deaths, and work disability.

Estimating the mortality risks associated with income drops requires assessing the threat of confounding and the potential for effect modification by marriage, births retirement, family breakdown, job loss, and family deaths; assessing the potential for effect modification by income level; and assessing the threat of time-dependent confounding by changes in health. Confounding may be due to causes of income declines that may have effects on mortality unmediated by income decline. Such factors include marriage,
births, retirement, family breakdown, job loss, and family deaths. These factors may also act as effect modifiers by causing changes in wealth or social support, thus changing the relationship between income drops and cash constraint. Effect modification by income level is a possible consequence of the ability of liquid assets and credit (including forms of mortgage equity withdrawal) to moderate the effects of disposable income declines, such that cash constraint resulting from disposable income declines might be greater for poorer families than for richer ones. Time-dependent confounding refers to situation in which a mediator is also a determinant of the exposure. This is more often referred to as reverse-causation or health selection. Income drops are hypothesized to affect mortality via decreased health status, but decreases in health status can also lead to work-disability and income drops.

The following sections will examine the outcome, exposures, and confounders in this mechanism in more detail.
1.3 Outcome variable: premature mortality

The analyses described in Chapters 4 and 5 will focus on all-cause mortality at 41-73 years of age. Premature deaths are those that occur prior to a defined full life-span. Various approaches to defining the age that represents a full life-span have been employed, but all are arbitrary. In Manitoba, Mustard et al. examined changing regional inequalities in premature mortality defined as deaths before the age of 75 [170].

In the empirical work on income dynamics in Chapters 4 and 5, person-years of exposure have been restricted to the ages of peak labour force participation. The ages at which death is observed are simply a consequence of the length of follow-up. In Chapter 5, person-years of exposure are restricted to the ages 40-55, and the data allow follow-up periods of 1-18 years, and so deaths occur at ages 41-73.

These deaths are perhaps more premature for women than for men. Event counts, proportions and rates by cause of death, age and sex are shown in Tables 1.1, 1.2 and 1.3. Malignant neoplasms account for roughly half of female deaths, and one-fifth increasing with age to two-fifths for men. Cardiovascular disease accounts for 20% increasing with age to 30% for men, but only for 12% increasing to 21% for women. At ages 40-44 men die commonly of accidents (17%) and suicide (13%), but at older ages and for women these causes are more rare.

1.4 Exposure variable: income level

The analyses described in Chapters 4 and 5 focus on income as a measure of social advantage and premature all-cause mortality as the health outcome. A graded relationship has long been observed between greater income and lower mortality in the United States, including the seminal work by Kitagawa and Hauser [119], in which they conducted a four-month mortality follow-up of the 1960 US Census.
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<tr>
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<td>238</td>
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<td>109</td>
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<td>46</td>
<td>48</td>
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<tr>
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<td>361</td>
<td>296</td>
<td>272</td>
<td>203</td>
<td>109</td>
<td>131</td>
<td>107</td>
<td>100</td>
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<td>127</td>
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<td>- Transport accidents</td>
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<td>176</td>
<td>145</td>
<td>132</td>
<td>110</td>
<td>73</td>
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<td>58</td>
<td>54</td>
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<td>73</td>
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<td>- - Falls</td>
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<td>42</td>
<td>45</td>
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<td>56</td>
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<td>15</td>
<td>15</td>
<td>20</td>
<td>37</td>
</tr>
<tr>
<td>- - Poisoning / noxious substances</td>
<td>121</td>
<td>99</td>
<td>78</td>
<td>48</td>
<td>26</td>
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<td>40</td>
<td>44</td>
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<tr>
<td>Intentional self-harm (suicide)</td>
<td>319</td>
<td>325</td>
<td>284</td>
<td>206</td>
<td>126</td>
<td>82</td>
<td>90</td>
<td>121</td>
<td>109</td>
<td>69</td>
<td>48</td>
<td>39</td>
</tr>
<tr>
<td>Assault (homicide)</td>
<td>37</td>
<td>32</td>
<td>13</td>
<td>17</td>
<td>16</td>
<td>12</td>
<td>20</td>
<td>13</td>
<td>9</td>
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<td>4</td>
<td>5</td>
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<tr>
<td>Events of undetermined intent</td>
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<td>63</td>
<td>48</td>
<td>30</td>
<td>15</td>
<td>10</td>
<td>25</td>
<td>40</td>
<td>35</td>
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<td>Other</td>
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<td>782</td>
<td>899</td>
<td>1,275</td>
<td>199</td>
<td>309</td>
<td>398</td>
<td>560</td>
<td>649</td>
<td>951</td>
</tr>
</tbody>
</table>

**Table 1.1:** Cause of death by age and sex in Canada in 2004 [222]. Number of deaths.
### Distribution of deaths by cause by age in 2004 (%)

<table>
<thead>
<tr>
<th>Selected causes of death</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes death</td>
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<td>100</td>
</tr>
<tr>
<td>HIV disease (AIDS)</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Malignant neoplasms</td>
<td>21</td>
<td>27</td>
</tr>
<tr>
<td>- of colon, rectum and anus</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>- of pancreas</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>- of trachea, bronchus and lung</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>- of breast</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>- of prostate</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>- of the central nervous system</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>- of lymphoid / haematopoietic tissue</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Major cardiovascular diseases</td>
<td>18</td>
<td>23</td>
</tr>
<tr>
<td>- Diseases of the heart</td>
<td>16</td>
<td>20</td>
</tr>
<tr>
<td>- Ischaemic heart diseases</td>
<td>12</td>
<td>16</td>
</tr>
<tr>
<td>- - Acute myocardial infarction</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>- - Other forms of chronic IHD</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>- Other heart diseases</td>
<td>3</td>
<td>3</td>
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<tr>
<td>- Cerebrovascular diseases</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Influenza / pneumonia</td>
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<td>Chronic lower respiratory diseases</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Chronic liver disease and cirrhosis</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>- Alcoholic liver disease</td>
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<td>3</td>
</tr>
<tr>
<td>Not elsewhere classified</td>
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<td>4</td>
</tr>
<tr>
<td>Accidents (unintentional injuries)</td>
<td>17</td>
<td>12</td>
</tr>
<tr>
<td>- Transport accidents</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>- Motor vehicle accidents</td>
<td>6</td>
<td>4</td>
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<tr>
<td>- Non-transportation accidents</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>- Falls</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>- Poisoning / noxious substances</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Intentional self-harm (suicide)</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>Assault (homicide)</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Events of undetermined intent</td>
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<td>2</td>
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<tr>
<td>Other</td>
<td>13</td>
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</tr>
</tbody>
</table>

**Table 1.2**: Cause of death by age and sex in Canada in 2004 [222]. Percentage by cause of death.
# Table 1.3: Cause of death by age and sex in Canada in 2004 [222]. Crude mortality rates.

<table>
<thead>
<tr>
<th>Selected causes of death</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes</td>
<td>172.0 269.8 428.7 678.4 1,130.2 1,766.7</td>
<td>97.5 172.3 273.6 424.7 683.1 1,075.6</td>
</tr>
<tr>
<td>HIV disease (AIDS)</td>
<td>5.5 4.8 4.6 4.1 2.5 1.2</td>
<td>1.2 1.1 0.7 0.5 0.4 0.2</td>
</tr>
<tr>
<td>Malignant neoplasms</td>
<td>36.7 73.3 140.1 274.5 486.6 762.5</td>
<td>43.9 90.7 158.6 245.1 382.1 539.8</td>
</tr>
<tr>
<td>- of colon, rectum and anus</td>
<td>3.8 7.7 14.0 29.7 53.5 83.5</td>
<td>2.8 7.9 12.7 20.7 31.8 45.1</td>
</tr>
<tr>
<td>- of pancreas</td>
<td>2.2 4.1 9.9 17.8 28.4 47.4</td>
<td>1.2 2.6 5.1 10.8 19.0 32.1</td>
</tr>
<tr>
<td>- of trachea, bronchus and lung</td>
<td>7.1 19.1 38.7 83.2 160.2 254.3</td>
<td>7.2 21.8 37.8 70.7 117.3 161.6</td>
</tr>
<tr>
<td>- of breast</td>
<td>0.0 0.2 0.3 0.2 0.3 0.9</td>
<td>12.9 23.2 38.2 49.9 64.0 76.5</td>
</tr>
<tr>
<td>- of prostate</td>
<td>0.1 0.5 1.9 9.0 20.6 44.9</td>
<td>NA NA NA NA NA NA</td>
</tr>
<tr>
<td>- of the central nervous system</td>
<td>3.6 5.4 8.0 274.5 14.4 20.8</td>
<td>1.4 2.9 5.8 245.1 8.5 12.7</td>
</tr>
<tr>
<td>- of lymphoid / haematopoietic tissue</td>
<td>6.0 8.1 14.2 24.0 45.5 67.0</td>
<td>4.1 5.7 10.9 14.0 27.5 46.1</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3.4 5.9 12.7 24.9 44.8 70.2</td>
<td>1.7 2.5 5.9 11.8 24.0 41.5</td>
</tr>
<tr>
<td>Major cardiovascular diseases</td>
<td>31.6 62.6 114.4 188.0 326.3 512.4</td>
<td>11.8 19.9 32.6 62.9 120.4 222.3</td>
</tr>
<tr>
<td>- Diseases of the heart</td>
<td>27.0 54.0 98.0 159.4 265.9 402.3</td>
<td>8.4 13.1 23.8 46.1 87.6 186.6</td>
</tr>
<tr>
<td>- - Ischaemic heart diseases</td>
<td>20.6 44.1 80.2 132.4 221.7 341.4</td>
<td>5.0 8.0 16.1 33.1 65.1 120.1</td>
</tr>
<tr>
<td>- - - Acute myocardial infarction</td>
<td>10.6 24.1 42.3 67.8 114.4 167.1</td>
<td>2.5 4.0 8.5 17.2 35.9 62.4</td>
</tr>
<tr>
<td>- - - Other forms of chronic IHD</td>
<td>9.0 19.4 36.2 61.4 103.2 168.7</td>
<td>2.3 3.7 7.4 15.0 28.4 54.9</td>
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<tr>
<td>- - Other heart diseases</td>
<td>5.8 8.9 15.8 24.3 39.5 53.9</td>
<td>3.0 4.7 6.3 11.4 19.0 32.9</td>
</tr>
<tr>
<td>- Cerebrovascular diseases</td>
<td>3.6 6.0 11.5 17.6 33.6 64.9</td>
<td>2.7 6.0 6.8 12.8 25.1 41.5</td>
</tr>
<tr>
<td>Influenza / pneumonia</td>
<td>1.6 2.3 3.2 7.6 10.1 17.6</td>
<td>1.0 1.6 2.3 2.9 7.0 11.5</td>
</tr>
<tr>
<td>Chronic lower respiratory diseases</td>
<td>0.5 2.2 4.2 11.1 29.0 67.9</td>
<td>0.8 1.4 4.0 9.6 23.7 50.0</td>
</tr>
<tr>
<td>Chronic liver disease and cirrhosis</td>
<td>3.3 9.1 16.3 18.4 33.3 38.0</td>
<td>2.3 3.5 7.2 9.0 11.7 16.6</td>
</tr>
<tr>
<td>- Alcoholic liver disease</td>
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<td>1.7 2.3 4.5 5.2 5.0 6.3</td>
</tr>
<tr>
<td>Not elsewhere classified</td>
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<td>2.6 4.0 3.9 4.9 6.2 7.9</td>
</tr>
<tr>
<td>Accidents (unintentional injuries)</td>
<td>29.9 31.5 32.6 31.0 38.1 36.0</td>
<td>8.0 10.2 9.5 10.3 11.7 20.9</td>
</tr>
<tr>
<td>- Transport accidents</td>
<td>12.5 13.7 13.1 13.8 15.4 12.9</td>
<td>3.6 4.5 4.8 4.7 4.2 8.2</td>
</tr>
<tr>
<td>- - Motor vehicle accidents</td>
<td>11.0 11.5 10.9 11.9 13.3 11.4</td>
<td>3.4 4.2 4.4 4.4 3.9 8.1</td>
</tr>
<tr>
<td>- Non-transportation accidents</td>
<td>17.5 17.7 19.5 17.2 22.7 23.1</td>
<td>4.4 5.7 4.7 5.5 7.5 12.7</td>
</tr>
<tr>
<td>- - Falls</td>
<td>2.0 3.4 3.8 4.7 9.1 9.9</td>
<td>0.4 0.8 0.9 1.5 2.7 6.1</td>
</tr>
<tr>
<td>- - Poisoning / noxious substances</td>
<td>8.8 7.7 7.1 5.0 3.6 2.5</td>
<td>2.9 3.4 2.1 2.1 2.2 1.0</td>
</tr>
<tr>
<td>Intentional self-harm (suicide)</td>
<td>23.1 25.4 25.7 21.6 17.6 14.5</td>
<td>6.6 9.4 9.7 7.1 6.5 6.4</td>
</tr>
<tr>
<td>Assault (homicide)</td>
<td>2.7 2.5 1.2 1.8 2.2 2.1</td>
<td>1.5 1.0 0.8 0.7 0.5 0.8</td>
</tr>
<tr>
<td>Events of undetermined intent</td>
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<td>1.8 3.1 3.1 2.4 1.3 1.3</td>
</tr>
<tr>
<td>Other</td>
<td>21.9 35.9 62.0 82.0 126.0 226.2</td>
<td>14.3 23.9 35.3 57.5 87.6 156.4</td>
</tr>
</tbody>
</table>
Duleep studied a sample drawn from the US Current Population Survey aged 35-65 linked to Social Security records and followed for mortality from 1973 to 1978 [70, 71]. She found that income had a graded relationship to mortality at all income levels, and that this effect was robust to adjustment for health problems preceding or concurrent with the measure of income. However, when adjusted for education, above average incomes no longer showed marginal increases, despite a steep gradient for below average incomes. She also observed that a measure of mean earnings for several years was associated with greater inequality in mortality than was a single-year earnings measure.

The National Longitudinal Mortality Study, consisting of a representative million-person sample of the US population drawn from the Current Population Survey over the years 1979-1985 and followed for 1 to 7 years, showed a monotonic mortality gradient by net family income for white and black men and women aged 25-64 for most major causes of death, including cancer [194, 195].

### 1.4.1 Canadian epidemiological research

There are few population studies of income and mortality in Canada. Hirdes and Forbes used data on 2,000 45 year-old men sampled into the Ontario Longitudinal Study of Aging in 1959 and with a 10 year mortality follow-up beginning in 1969 [100]. They specified income in three levels, with the highest and lowest each representing the extreme quintile. Low income was associated with greater mortality, while high income conveyed a small benefit over the middle quintiles. This pattern was robust to adjustment for education, smoking and baseline self-rated health.

Wolfson et al. used Canadian Pension Plan (CPP) data to examine the relationship between earnings and risk of death [248]. More than 500,000 men who attained the age of 65 on or after 1979 were followed for deaths occurring between the ages of 65 and 74 in relation to earnings in the 10 to 20 years prior to age 65. The data exclude residents
of Quebec and all analyses exclude those receiving CPP disability benefits. A strong, monotonic gradient in mortality was observed. They also observe that income appears to convey a decreasing marginal survival advantage, but remains monotonic at very high levels of income.

Wolfson et al. make the argument that health selection plays at most a modest role, using as evidence the estimates shown in Table 1.4. During both 5-year survival periods, there is a positive, monotonic association between income and survival for the full sample. For the hundred thousand men who had a statistically significant rank correlation between earnings (deflated by the average industrial wage) and age, the gradient was still present, and showed marked attenuation only for the poorest quintile. Health selection is therefore not likely a sufficient explanation of the gradient, and may in fact play only a small role.

This argument against health selection is corroborated by Hanratty et al. [96], who used Swedish tax data to approach the association of income declines and mortality assuming that health selection is the relevant pathway. They examine income declines, as an indicator of decreased ability to cope with mounting health care costs, during the three years prior to death, when health care utilization increases. They divided decedents into twenty income bands, and in every band equivalized family income increased on average during the three years prior to death. Among those who did experience income

<table>
<thead>
<tr>
<th>Earnings quintile</th>
<th>All males</th>
<th>Males with increasing earnings</th>
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<td></td>
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<td>To age 74</td>
</tr>
<tr>
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<td>p(S) RR</td>
<td>p(S) RR</td>
</tr>
<tr>
<td>1 0.862 1.47</td>
<td>0.740 1.35</td>
<td>0.887 1.31</td>
</tr>
<tr>
<td>2 0.871 1.37</td>
<td>0.750 1.30</td>
<td>0.883 1.36</td>
</tr>
<tr>
<td>3 0.881 1.27</td>
<td>0.759 1.25</td>
<td>0.895 1.22</td>
</tr>
<tr>
<td>4 0.889 1.18</td>
<td>0.783 1.12</td>
<td>0.901 1.15</td>
</tr>
<tr>
<td>5 0.906 1.00</td>
<td>0.807 1.00</td>
<td>0.914 1.00</td>
</tr>
</tbody>
</table>

**Table 1.4:** Probabilities of survival (p(S)) for male CPP recipients from age 65 to age 70 and 74, for all recipients and those with an increasing adjusted earnings trend before age 65 [248]. The data are also re-calculated as relative risks (RR) of death relative to the highest income quintile.
declines in the three years prior to death, the largest relative declines were among the poorer and richer income bands.

Mustard et al. described the association of income, education, mortality and health care utilization in a 5% sample (47,935 persons) of the June 1986 population of Manitoba, with follow-up through May 1988 [173]. Although the study had limited statistical power and had a relatively short mortality follow-up, a statistically significant gradient between income and mortality was observed for ages 30-49 and 50-64, but not 65 and over, and the gradients for income and education were significant when adjusted for one another. Because both socioeconomic indicators were specified coarsely as quartiles (within age groups), it is possible that the independent effects of income and education represent some degree of residual confounding.

Wilkins et al. [246] recently reported on adjusted family income and mortality in the 10-year mortality follow-up of the 1991 Canadian Census. Income was measured as post-transfer, pre-tax total household income, expressed as a proportion of the Low-Income Cut-Off (LICO) within Census Metropolitan Areas, Census Agglomeration, small town or rural area. The LICO takes family size and community size into account. By quintile, the conditional life expectancies at 25 years were 48.3, 51.4, 52.9, 53.9, and 55.2 for men and 56.4, 58.7, 59.5, 60.1, and 60.7 for women. The range of life expectancies is large, the associations monotonic, and there is pattern of diminishing marginal survival returns on income. The conditional probabilities of surviving to age 75 make the same point, but perhaps more dramatically for men: 50.6%, 59.8%, 64.9%, 68.2%, and 72.4% for men and 71.5%, 77.5%, 80.6%, 82.0%, and 83.8% for women. The standardized relative risks of death for men aged 25 and older are 1.12, 1.21, 1.36 and 1.68 for quartiles 4 through 1 compared to 5, and for women these are 1.07, 1.13, 1.22 and 1.49. Differences by age are shown in Figure 1.3, which plots standardized rate ratios by income quintile and hazard ratios by income decile for men and women.
Figure 1.3: Wilkins et al. [246] estimated standardized rate ratios (SRR) and hazard ratios (HR) by adjusted family income quintiles and deciles, respectively, for ten-year age groups and by sex. Clear, monotonic gradients are evident throughout adulthood until early retirement, then disappear among the elderly. Although diminishing returns with income are evident, the gradient remains considerable throughout the income range. Note that percentiles were calculated for the full sample, and not within age and sex categories.
1.4.2 Measuring income

When studies have sufficiently detailed income data, such as are available from tax records, the construct and measurement of income can be more precisely specified. The definition of income is usually broken into two parts: specifying the economic unit of observation, and specifying the components of income. In order to specify the components, definitions of the dominant theories of income will be briefly described.

Economic units

The choice of economic unit answers the question “whose income?” The unit is typically individuals, individuals who receive income, families (related adults & their dependent children), spending units (groups who pool resources and consume collectively), or households (living at same address) [9, p.34]. The choice of unit also involves decisions on how income is adjusted for unit size and composition, and how units are weighted [9, p.36].

These definitions are only equivalent if all units are of the same size and members have identical incomes. This is not the case and so there are differences. According to Atkinson and Bourguignon, “nothing general can be said of the direction these differences should take.” [9, p.35]

“Individual earnings data are better adapted to positive studies focusing on the labour market, whereas household income may be more appropriate in normative studies addressing the issue of inequality in living standards, although this is influenced by the degree to which resources are shared within the household.” Note that earnings income are usually earned individually, while investment income is often jointly held [9, p.35]. The Canadian definition of a census family as related persons living together is narrower than that used by most nations (except Sweden), which use household as the recipient unit [90].
The two chief challenges to working with these definitions is correctly assigning individuals to their unit, and then deciding on how to adjust for unit size. Household size is best used to estimate “equivalised” incomes, where total income is expressed per adult equivalent. Per capita adjustments ignore economies of scale, and it is common to divide income by the square root of household size [9, p.35-6].

Buhmann et al. summarized the equivalence scale literature by expressing the problem as

\[ W = \frac{D}{S^e} \]

(1.1)

where adjusted income, or economic well-being, \( W \) is equal to disposable income \( D \) divided by household size \( S \) raised to the equivalence elasticity \( e \), which varies between 0 and 1 to express economies of scale within households (0 indicates no adjustment for household size, 1 indicates no economy of scale) [42]. Counter-intuitively, the effect of \( e \) on measures of inequality is non-monotonic (typically U-shaped with a minimum near 0.5), though as one would expect, these adjustments cause substantial shifts in the measured magnitude of inequality (though usually altering concentration rather than the rank order of nations) [60, 59]. More recent work has incorporated the ages of household members as well as their number [9, p.35-6].

Statistics Canada does not have an official equivalence scale, but that used for the Low Income Measure (LIM) is widely used. Its equivalence scale takes the oldest adult in the family as having a value of 1, and each additional person aged 16 or greater is valued at 0.4, while those aged less than 16 are valued at 0.3 [85]. For households with four persons or less, this leads to very similar results to those obtained using the standard equivalence scale adopted by the United Nations and the Luxembourg Income Study (LIS), where \( e = 0.5 \) [104]. The Organisation for Economic Cooperation and Development (OECD) does not have an official equivalence scale either, but historically used the “OECD” or
Table 1.5: Equivalent elasticities for various equivalence scales. Modified from OECD [177].

“Oxford” scale, then a modified version of this, and now it is common to use the LIS standard [177]. The differences are summarised in table 1.5.

It should also be noted that family size has been decreasing in Canada, particularly for certain cohorts [179]. This has the effect of increasing effective disposable income.

Changing household structure can have significant effects on the economic well-being of household members. For example, in Canada roughly half of the flow of children into and out of poverty is due to changing family structure, with the other half being due to changing wages and hours [184].

It is also relevant how income is shared within households. It is traditional to assume rational welfare maximization within households, but this is not always the case [182].

Another question is how to weight households. Weighting by one means that households are the unit of interest, while weighting by household size suggests an interest in individual welfare. This is not the same problem as that of equivalence scales mentioned above [9, p.35-6].

Economic well-being: permanent, transitory and Haig-Simons income

Individuals typically enter the labour force earning low wages, then earn progressively more while saving for retirement, and then retire, stop earning, and dissave. This earnings trajectory is often punctuated by periods of higher or lower earnings due to job separation, inheritance, divorce, investment etc., amounts often termed transitory income. Because of transitory income, current-year income is often a poor indicator of long-run economic
well-being. Economists often take a lifetime approach and attempt to describe permanent income, typically defined as “the amount that an individual can spend consistently with being able to maintain the same level of spending in the future,” and therefore a reflection of consumption behaviour, or economic well-being [31].¹ This literature was established by Modigliani and Friedman in the 1950s, responding to Keynes’ work on consumption [73], and is now widely used and accepted.

At the same time, this approach is counter-intuitive to some:

Most politicians do not believe that a retired couple with social security, pension and asset income of $35,000 and a backup shortstop in the major leagues making $200,000 should be lumped together because they have a permanent income of $50,000 [17].

There are also economists who deny that consumption follows the life-cycle model sufficiently well for it to be taken as a proxy for lifetime income, which would be consistent with the permanent income model. If individuals are cash-constrained, for example, they may under-consume, and then over-consume when no longer constrained [17]. The fact that there is uncertainty about future income, length of life, and future economic policy and conditions also complicates the theory and reality of the permanent income approach [73, 17].

An addition to the distinction between permanent and transitory, a popular theoretical approach to the accounting of income is what is often called Haig-Simons income (or Hicks-Haig-Simons [73]), defined as current consumption plus or minus any change in net wealth. Empirical work does not usually fully comply with the Haig-Simons income concept, which would require, for example, the imputed rental income from owner-occupied dwellings, which is difficult to estimate.

¹For a criticism of traditional economic thinking on the standard of living and well-being, see Sen [205, 206].
**Income components**

The components of income and the period over which they are measured in applied research involve compromises with respect to economic theory. Gottschalk and Smeeding state that:

Ideally income would be measured on a post-tax and transfer basis consistent with the Haig-Simons income concept of real consumption plus (or minus) change in net worth. This broad definition of income is an attempt to get closer to the distribution of lifetime utility. Income would be adjusted for economies of scale in consumption using an appropriate equivalence scale, and would cover the period over which families can smooth consumption by lending or borrowing. For families that are not credit constrained this might require measures of lifetime post-tax and transfer income adjusted for family size. At the other extreme, the relevant measure of income might be a few pay periods for families who do not have sufficient assets to smooth consumption and cannot borrow against future income. [90].

The disposable income concepts typically used by national statistical agencies fall "considerably short" of the Haig-Simons definition, excluding capital gains, imputed rents, home production, and income in kind. Leisure, indirect taxes, and benefits from public spending other than cash-transfers are also usually ignored. Transfers included in disposable income usually include only public cash and "near cash" benefits, such as food stamps. Where employers contribute to the costs of benefits such as pensions or health insurance, these are not captured. Note that while it is sometimes possible to estimate the value of non-cash transfers to the supplier, their value to households is more difficult to estimate. Including non-cash transfers reduces measured income inequality [90].

Atkinson and Bourguignon make the following comments about standard income components used in applied research [9, p.37]:

- **Total income** is not defined consistently in the literature.
- **Labour income** is complicated by issues of household labour supply and unemployment.
- **Capital income** is hard to observe (except in income tax returns), is virtual rather than real, and is typically underestimated.
Primary income is the sum of labour and capital income.

Transfer income is not part of primary income if part of redistribution system. Pensions are really capital income, but not entirely if the system is partially redistributive. Child benefits, welfare, income supplements that are non-contributory should theoretically include the imputed value of public housing, health care and education services consumed. Private transfers between households serve the same function and should be treated similarly, but cannot usually be measured.

Disposable income is usually defined to be the sum of primary and transfer income less direct taxes. It does not include geographic or other variation in price or availability of goods and services and ignores indirect taxes.

Note that labour and capital income are difficult to distinguish for the self-employed (particularly farmers), whose labour isn’t traded on a market. Brandolini and Smeeding [36] summarize a simpler set of measures as follows:

Market income is income before taxes and transfers.

Gross income is income after transfers but before taxes.

Disposable income is income after direct taxes and transfers.

1.4.3 Earnings mobility

Earnings mobility refers to changes in individual income over time. Lifetime earnings profiles are non-monotonic, peaking in the mid 40s or early 50s. In Canada, the 1980s and early 1990s saw important changes in age-earnings profiles, with opposite trends for men and women (see Figure 1.4). Earnings at labour market entry decreased and mobility decreased for entrants as well. Women further into their careers, and especially
those whose earnings were not low, saw increased mobility, while men of all earnings levels saw decreased mobility.

### 1.4.4 Summary of literature on income level

The monotonic, negative association of income with mortality is well established at working ages for men and women. In Canada, as elsewhere, the association is robust to adjustment for education and baseline health status. The gradient is sufficiently steep that the burden it identifies is as large as cancer as a segment of mortality [248]. Lack of attenuation of the effect of income on mortality during follow-up suggests that health selection plays at most a minor role. Early retirement, however, is a powerful effect modifier, such that income gradients are much steeper among early-retirees.
In the analyses of income level and income drops on mortality, it is important to exclude from the analyses early retirees since disability likely plays a role in their decision to retire, is likely associated with income level, and is likely associated with mortality. Retirement will be discussed further in Section 1.7.5.

When measuring income, the construct should be defined in accordance with the hypothesized mechanism. For effects mediated by consumption behaviour, household disposable income will be preferable in most cases. For effects due to relative status mediated by behavioural and psychosomatic effects, individual earnings or market income might be preferred. The analyses in Chapter 5 explore household disposable income assuming that consumption behaviour dominates the causal pathways. Alternatives may be explored in future research. In an attempt to approximate permanent, rather than transitory, income, income level is taken as the average of the five years preceding the exposure year.

1.5 Exposure variable: Income drops

Research on income dynamics and mortality draws on two bodies of research: research on income level and mortality, and research on job loss or unemployment and mortality. The first was discussed in Section 1.4, while the second is discussed below in Section 1.6.

The relatively scarce literature directly addressing income drops and mortality will be reviewed here. The induction period for these effects will also be discussed. There is a difference between income dynamics, which is concerned with both income increases and decreases, and income drops, which are the exposure of interest here. Studies on increases in income, such as studies of lottery winners, are not reviewed.
1.5.1 Previous epidemiological research

Benzeval and Judge [25] reviewed longitudinal studies of income and health, including several studies that had a measure of income change as an exposure and adult mortality as the health outcome. Income drops are closely related to concept of poverty. In the British Household Panel Survey, 16.5% of respondents (age range unspecified) were in poverty during any particular year, but during a six year period (1991-97), 37% experience one or more years in poverty and only 3% experience six years in poverty [25]. Analyses of spells of poverty are therefore included in the following review.

Hirdes and Forbes [100] examined men in the labour force aged 45 in 1959 in the Ontario Longitudinal Study of Aging who reported good or excellent health in 1959. The exposures were personal self-reported income level in three categories (highest quintile, lowest quintile, remainder) and any change in level by 1969, the outcome was death before 1978, and the association was estimated using logistic regression. They did not find that income level change was a statistically significant predictor of death in multivariate models, but they did find marginally statistically significant evidence that the unadjusted relative risk of death was 33% to 48% higher for those with lower income in 1969 than for those with income in the same category. Interpretation of this study’s findings is complicated by the high attrition and lack of sufficient data to allow for standard treatment of censored observations using survival analytic methods.

Kaplan and Haan [111] used data from the Alameda County Study to study income level in 1965, whether or not income fell by $10,000 by 1974, and whether death occurred between 1974 and 1983. Their sample was restricted to persons aged 50 or greater in 1965, they estimated associations using Cox regression, and found that income falls, but not level, were statistically significantly associated with increased hazard of death after adjusting for baseline health. This sample is in the process of retirement, and so income is decreasingly important as an indicator of economic security, and changes in income
are harder to interpret.

Zick and Smith [252] used data from the Panel Study of Income Dynamics to show that a spell of poverty in any of the previous three years increased the odds of death in discrete time event models adjusted for education and race. While framed as an investigation of poverty spells, this analysis does not distinguish between income level and income drops, and has very short lags. A similar analysis using Cox models produced similar findings [214].

Menchik [157] used data from the National Longitudinal Survey in the United States to show that among men (median age of 51 at baseline), baseline wealth, cumulative average earnings, and cumulative proportion of years in poverty were all associated with the odds of surviving 17 years in the expected directions. The findings were robust to stratification by baseline self-reported health. However, these experiences of poverty are not the same as the relative drops in income conceived in the present analysis. The US poverty threshold can be greater than, slightly less than, or far less than a family’s permanent income. The cumulative proportion of years in poverty is therefore invariant at very low permanent incomes, and indicates only the greatest relative drops in income for those with high permanent income. Moreover, the observed life-shortening effect of poverty may simply be revealing a non-linear association of permanent income with log-odds of dying.

McDonough et al. [156], revising previous work by Duncan [72], analyzed the effects of income dynamics and income level in the Panel Study of Income Dynamics in the United States. They developed two measures of income dynamics, income persistence and income drops, and examined the interaction of these with income level in overlapping 5 year panels. Income persistence was defined as the number of years (1-3 or 4-5) that a person spent in below $20,000 (lower), between $20-70,000 (middle), or above $70,000 (upper). 40% of the sample was always in the middle bracket, 18% were in each of
the categories “upper (1-3 or 4-5 years) and never lower,” and 11% were in each of the categories “lower (1-3 or 4-5 years) and never upper.” An income drop was defined to be any annual income less than 50% of the previous year’s income. For each of the three income levels, one or more drops was compared to no drops. The sample proportions experiencing such drops were 4, 6, and 2% in the lower, middle and upper income levels respectively. McDonough et al. observed that always low income conferred the greatest hazard, while always high income was most protective, and showed evidence that drops in income may be predictive of death.

Although not reviewed by Benzeval and Judge, Wolfson et al. [248] examined gaps in earnings histories and survival among their Canadian Pension Plan cohort (described above on page 13). They examined the earnings-survival gradient for deaths occurring at ages 65 to 70 and earnings during the 10 to 20 years prior to age 64 for those retiring after age 61, stratified by whether or not the pensioner had an interruption of their earnings history. Interruptions were defined as one or more years of zero earnings, and did not seem to have an effect on survival or modify the effect of earnings.

1.5.2 Induction periods for the effects of income dynamics

An induction period is the length of time between an exposure and the health consequences that it causes. Methods for modelling induction periods are discussed in Section 3.2. Induction periods for the effects of income dynamics will vary by cause of death. The causes of death are described by age for Canadians 40 to 69 years of age in Tables 1.1, 1.2 and 1.3.

Davey Smith has argued that social determinants of health need to be understood in terms of their latencies, and in light of the fact that most cause-specific mortality rates have remarkably constant downward trends [63]. Some causes of death have immediate biological processes, such accidents and violence, while others, such as cancer, are
**FIGURE 1.5:** Three main types of apparent income drop effects are expected. Income declines due to health problems will cause a short-run positive bias in apparent mortality risk associated with income drops. Accidental or violent deaths, in particular suicide and alcohol-related deaths, should show a causal short-run increase in apparent mortality risk, while psychosomatic and behaviourally induced or exacerbated chronic disease deaths will involve a longer latency.

defined by a slow progression. Cardiovascular deaths have a long-term chronic disease component as well as an acute component in which vascular events are triggered by acute stressors – a process referred to as “harvesting” in environmental epidemiology [203].

Iversen et al. [106] examined cause-specific mortality among persons unemployed on the day of the 1970 Danish census in the periods 1970-75 and 1975-1980. Persons on sick or maternity leave were considered employed. The unemployed showed slightly elevated relative mortality for cancer and heart disease, but substantially increased relative mortality for suicides, accidents and “other” causes of death. They found little, if any, evidence of attenuation of these risks in the second follow-up period. It is surprising that the risk of non-chronic disease deaths remains elevated for such a long period after unemployment.

Maximum and minimum induction times for the various apparent effects of income dynamics on all-cause mortality are illustrated in Figure 1.5. If the maximum induction time for health selection is less than or equal to the minimum induction time for a causal
effect on chronic disease, then the entire chronic disease effect can be estimated without
health selection bias. If there is minor overlap, then there will be only minor biases. It is
only if there is significant overlap between the induction periods that the causal effects
of income dynamics on chronic disease mortality during the follow-up period cannot be
estimated with any confidence.

In the presence of substantial overlap, estimates of the effect of income drops on all-
cause mortality for various induction periods will show a bimodal risk distribution over time
if the causal component for chronic disease deaths is substantial relative to the short-run
accident and selection effects. If the causal chronic disease risk is only moderate by
comparison, then it may only increase the level of the right tail of the risk distribution over
time, and not produce a second mode, making even qualitative inference of a long-term
causal effect on chronic disease mortality difficult.

The problem with Figure 1.5 is that it treats income dynamics only as a unique life
event, rather than as a chronic exposure. For a chronic, or recurrent, exposure the
exposure-response relationship is inevitably more complicated due to ongoing exposure,
and dimensions of exposure such as cumulative and peak effects. The analyses here do
not treat income drops as a chronic or recurrent exposure because appropriate analytic
methods could not be identified. These analyses are therefore a first step at examin-
ing the induction periods, and if strong patterns are identified, the development of more
elaborate models might be justified.

1.5.3 Economic literature on income dynamics

In economics, there are two bodies of research that address income dynamics: research
on permanent income and consumption, and research on economic inequality. In perma-
nent income theory, permanent income is the determinant of consumption, and income
dynamics are the transitory component, or noise, which obscures permanent income, as
discussed in Section 1.4.2. The economic inequality literature applies these concepts to a different problem. Trends in income inequality are decomposed into income inequality within (i.e. income dynamics) and between (i.e. permanent income inequality) income units. Cross-sectional indicators of economic inequality conflate these two, and interpretations often falsely and tacitly assume that what is measured is inequality between the permanent incomes of economic units.

Income autocorrelations within economic units will decrease with rising income dynamics (which cause income units to reorder), and increase with rising permanent income inequality (which diminishes the reordering of income units). Earnings income among Canadian men show relatively stable autocorrelations, suggesting that the permanent and transitory components of earnings dispersion are increasing roughly equally [15].

Beach et al. have used the Longitudinal Administrative Databank to decompose total variation in earnings across workers and time into a long-run inequality component between workers and an average earnings instability component over time for workers [20]. They observed that between 1982-89 and 1990-97 long-run inequality increased for men due to increasing long-run earnings inequality. The patterns they observed were not consistent with conventional explanations of the relationship of business cycle effects on inequality. They found that decreasing instability (reduced dynamics) during periods of economic growth can be greater than the associated increasing long-run earnings inequality, resulting in decreased total income variability.

Increasing transitory variance in earnings likely has different causes than increasing permanent earnings dispersion, such as labour market instability, competitive markets with high levels of firm failure and downsizing, an increase in the temporary workforce [158], or the rising importance of incentive pay [227, citing Lemieux, MacLeod and Parent]. Income instability is also generally more likely in single-earner than in multiple-earner families, with lone parents in particular at increased risk due to fewer job opportu-
nities that are compatible with their parenting responsibilities [162].

1.5.4 Summary of literature on income drops

There has been very little research directly addressing income drops and mortality. Data limitations and differences in definitions mean that most previous studies are not informative with respect to the questions addressed in Chapters 4 and 5. The exception is the work by McDonough et al. on the PSID, which failed to show the expected interaction with income level. Whether the findings from McDonough et al. can be replicated with alternative statistical models and with Canadian data is explored empirically in this study.

When using annual tax data to examine income as a determinant of mortality, there is a concern that income will decrease in the years prior to death due to health selection. It is valuable, therefore, to have the findings from Hanratty et al., showing that for all income levels, family income typically increases during the three years prior to death. As argued by Wolfson et al. [248], health selection may be playing only a very modest role as a determinant of income.

Induction periods for the effects of income drops have not been explored, but could provide evidence of a causal mechanism if hypothesized patterns are observed. The statistical power afforded by a large, longitudinal tax database will allow these patterns to be estimated here with reasonable precision. Induction periods will be discussed again in the context of job loss in Section 1.6.

The increasing transitory variance in incomes will not be directly incorporated into models. But the important differences between single-earner and dual-earner households will be reflected in Chapter 5 by analyzing couple families and single persons separately.
1.6 Related exposure variable: job loss

While there is very little literature on drops in income and subsequent mortality, there is a mature literature on the effects of job loss and unemployment on earnings and family income. There is also an established literature in social epidemiology on the effects of job loss and unemployment on adult mortality (for a more general assessment of health impacts, see [67]).

1.6.1 Job loss, unemployment & income drops

Job loss, or permanent separation, is made up of quits (voluntary), permanent layoffs (involuntary) and “other,” in roughly equal amounts [159]. Permanent layoffs are due to a shortage of work, while “other” includes a strike or lockout, return to school, illness or injury, pregnancy or adoption, retirement, work sharing, apprentice training, dismissal, or other reasons. The total permanent separation rate is stable near 20% per year, meaning that 1 in 5 workers leaves their job annually [159]. There are slightly fewer job separations during recessions because quits decrease more than layoffs increase. The total permanent separation rate is approximately matched by the annual hiring rate, though the latter has greater business cycle variability. Compared to the 1980s, the 1990s saw decreased quit rates, decreased hiring rates, and increased job tenure. There are also non-permanent separations, usually called temporary layoffs, which respond to the business cycle with very little delay [159].

Layoffs are considerably more common for men, for low-wage employees, employees of small firms, and employees in primary and construction industries [159]. Quit rates were higher for women in the 1980s, but were equal to male quit rates in the 1990s [251]. Quits have higher subsequent earnings and lower unemployment than layoffs [118].

Unemployment is defined as not working, but actively seeking work. Unemployment
rates fluctuate with the business cycle and seasonally, though the determinants of the level of unemployment are poorly understood [178]. Participation rates for women have increased dramatically over the last thirty years. Trends for men and women aged 25 to 54 (seasonally unadjusted) are shown in Figure 1.6.

Job loss is an event, while unemployment is a state. Job loss does not necessarily lead to unemployment; it can lead directly to labour market non-participation, and for multiple job holders to continued employment. Figure 1.7 shows the events and states that individuals might experience following involuntary job loss.

Galarneau and Stratychuk examined the experiences of layed off Canadian workers in the Survey of Labour and Income Dynamics for the years 1993 through 1997 [81]. Layed off workers whose job tenure had been one year or more spent an average of 16.1 weeks unemployed. 8% found a job in 1 week or less, 17% in 1 month or less, 37% in 3 months or less, 58% in 6 months or less, 67% in 9 months or less, and 74% in 12 months or less. Unemployment durations are very sensitive to the business cycle: the proportion finding a job within a year improved from 60% in 1993 to 83% in 1997. Unemployment durations were shorter for younger workers, men, those without children or living alone, those in occupations other than clerk/salesperson/service worker, those with shorter previous job tenure, those who did not receive unemployment insurance, and those who were not a visual minority. Education and earnings did not affect unemployment duration, nor did season of job loss. 84% of layed off workers who found a job within one year found a full-time job, which is close to the share of jobs that were full time, 81%. At twelve months after lay-off, the unemployment rate was 23% - much higher than in the general population, which averaged 10% during this period. Also, of those who found a job within twelve months, only 83% were employed at twelve months, with 9% unemployed and 8% no longer participating in the labour market. Of those who had not found a job within twelve months, 53% were no longer participating and 47% were unemployed. One year
**Figure 1.6:** Labour market participation, employment & unemployment rates for men and women in Canada, ages 25 to 54, seasonally unadjusted. Cansim vectors V2065047-V2065049 and V2065038-V2065040.
**Figure 1.7:** Pathways leading from full-time employment through job loss to subsequent labour market states. Hexagons indicate events, and ellipses represent states. Unemployment, non-participation, and some types of re-employment will likely involve income drops. Categories of non-participation and re-employment are meant to be illustrative rather than comprehensive. Health selection can occur in every transition, making analyses of the health effects of unemployment difficult to specify and bias difficult to eliminate.
after lay off, unemployment was more likely for older workers, less-educated workers, and lower-status occupations. 13% of layed off workers returned to school, and this group was younger, more educated, but in lower status occupations. Only 3% of layed off workers returned to school full-time. Among the re-employed, 32% earned more, 21% earned within 5%, and 47% earned less than their previous wage. The average increase in wage was 26% and the average decrease was 30%. Overall, 21% of layed off workers experienced a wage cut of 30% or more. Wages upon re-employment tend to regress to the mean, such that decreases are more likely for those with previously high wages, and increases more likely for those with previously low wages. The wage-gap was not a function of education, duration of joblessness, or family characteristics, but was partly determined by whether the new job was full-time, unionized, in the same occupation, or highly-skilled, with managers/professionals and construction workers typically faring well.

At any given time, the proportion of the unemployed who have been unemployed for twelve months or longer is referred to as the long-term unemployment ratio (LTU). Long-term unemployment ratios are variable between jurisdictions and within them over time. Canada’s long-term unemployment ratio has been low (about 10%) and relatively stable for the past 25 years, at similar levels to the United States. However, the proportion of the long-term unemployed in Canada that have been unemployed for over two years is the highest in the OECD at about 70% (men, aged 25-54, year 2000) [176]. Unemployment spells in Canada last longer than those in the US, regardless of reason for unemployment [14].

Morissette and Ostrovsky examined the effect of husbands’ permanent or temporary layoff in 1992 on family income using data from the Longitudinal Administrative DataBank [163]. On average, husbands’ earnings drop $13,100 (2002$) the year of layoff, $16,700 the following year, and five years after layoff their earnings are still $12,300 lower. Four years before layoff husbands earned an average of $49,400 and pre-layoff
after-tax family income averaged $68,200. About half of these earnings losses are offset by employment insurance and the tax system, such that after-tax family income losses are half as great. In relative terms, after-tax family income drops by 5% the year of layoff, and remains down 10% throughout the following 5 years.

Employment insurance in Canada replaces 55-60% of earnings with maximum insurable earnings based on the average industrial wage \[131, 130\]. Consumption is estimated to fall 3-6% for each 10% drop in the replacement rate, for both total expenditure as well as food and clothing. Households are more likely to report that economic hardship followed a job separation if unemployment lasted longer than 5 months, the household had zero or negative assets, if the worker had subsequently taken an unsatisfactory job, if the worker was a major breadwinner, or if the worker was single or a single parent \[40, 41\].

### 1.6.2 Job loss, unemployment & adult mortality

There are important methodological challenges in trying to estimate the causal effect of job loss on health and mortality. First, health selection may occur in several ways: (1) poor health or work disability leading to voluntary or involuntary job loss, (2) disability retirement, and (3) higher rates of job loss at unstable firms, which are often smaller and pay less, that attract less healthy workers \[227\].

Second, health problems and mortality manifest most frequently at older ages, though economic and labour market experiences likely have cumulative effects over the life-course \[63\]. Most studies lack the length of follow-up and longitudinal data on income and labour market experiences to describe both the important periods of exposure as well as the peak periods of disease manifestation \[227\].

Third, there is potential for confounding by other dimensions of socio-economic status, in particular by education and occupation \[186, 140\]. If lower education leads to poorer health and increased risk of job loss, or if occupations with greater physical and
psychosocial health hazards are also less secure, then associations between job loss and subsequent health and mortality might be non-causal. However, if job loss leads to retraining, or to improved or worsened occupational exposures, then these changes in education and occupation are playing a mediating, rather than a confounding, role.

Fourth, recurrent episodes of short-term unemployment may lead to long-term unemployment, discouraged workers, or to weakened labour force attachment [127]. These states of weakened labour force attachment are characterized by greater risk of lowered earnings, of decreased benefits, and of poorer psychosocial working conditions [185]. It may be that the acute psychological, behavioural and economic consequences of job loss for health are only part of the mechanism, and that the long-term reduction in the quality of labour market experiences following job loss explains a significant part of the effect of job losses on population health [43].

No study is likely to fully overcome all of these challenges simultaneously. Common study designs for examining the effect of job loss on health are described in Table 1.6. If the effects of job loss on health are negative, then health selection into and out of employment will upwardly bias estimates of the effects of job loss on health. If the effects of job loss on health are positive, then health selection will cause bias towards or past the null. It is clear that cross-sectional study designs will lead to highly biased estimates, and that prospective designs with baseline employment ascertainment and control for baseline health status will also suffer from upward bias, assuming negative health effects of job loss. While factory closure studies offer the best potential to control for selection bias, they are more limited than fully longitudinal population data with respect to the analysis of mediation and to external generalizability.
### Table 1.6: Study designs for examining the effect of job loss on health.

<table>
<thead>
<tr>
<th>Study design</th>
<th>Data availability</th>
<th>Potential to control for health selection into employment</th>
<th>Potential to control for health selection out of employment</th>
<th>Potential to examine latency of health effects</th>
<th>Potential to examine mediating pathways</th>
<th>External validity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross sectional association of unemployment and health status</td>
<td>Data readily available from health and labour surveys.</td>
<td>Very limited.</td>
<td>Very limited.</td>
<td>Very limited.</td>
<td>Possible, but causal inference is very theory-dependent.</td>
<td>Usually high.</td>
</tr>
<tr>
<td>Prospective longitudinal association of baseline employment status and health status during follow-up</td>
<td>Does not require detailed employment follow-up data, and could be based on administrative health or mortality records. Large sample sizes are possible.</td>
<td>Very limited.</td>
<td></td>
<td></td>
<td>Better than a cross-sectional study only with respect to mediation among health status measures from follow-up.</td>
<td>Usually high.</td>
</tr>
<tr>
<td>Prospective longitudinal association of employment status and health status</td>
<td>Longitudinal data on both labour market experiences and health status are rare.</td>
<td>Moderate: good health status at baseline can be an inclusion criterion.</td>
<td></td>
<td></td>
<td>Limited only by the quality of measurement.</td>
<td>Can be high.</td>
</tr>
<tr>
<td>Factory closure study</td>
<td>Rare, choice of control population sometimes difficult.</td>
<td>Strong: employed at baseline.</td>
<td>Strong: since all workers suffer job loss, there is no selection.</td>
<td>Good, but limited by length of follow-up and age of workers.</td>
<td>Possible, but sample homogeneity may lead to multicollinearity and identification problems.</td>
<td>Often questionable.</td>
</tr>
</tbody>
</table>
Whether job loss depends on prior health may also depend on the dimension of health or disability in question. Kraut et al. linked health utilization data in Manitoba to census data on labour force participation, and examined utilization patterns before, during and after unemployment [122]. They found that the unemployed did have elevated utilization prior to unemployment, but primarily for mental health treatment. They also observed increased utilization after a period of unemployment.

Job loss as a cause of increased mortality

Early studies by Brenner examined the ecological association between unemployment and mortality rates. He concluded that unemployment was a determinant of mortality, but his conclusions were eventually disregarded on methodological grounds [92, 238, 239]. Brenner’s results were strongly affected by the chosen time period, populations, and assumptions regarding lag structure. Much of the correlation between unemployment and mortality that he observed were the result of the recovery from the 1930s depression, and so were discounted by the research community on the grounds of omitted variable bias [198].

Subsequent analyses of the effect of job loss on mortality include baseline labour data with longitudinal survival follow-up [165, 166, 168, 106, 58, 148, 218, 225, 27, 175, 83, 120, 233, 237, 4, 143], and fully longitudinal data [164, 149, 245, 227]. For other health outcomes see Lavis et al. [127] and Burgard et al. [43].

A clear pattern for the time-dependency of the association of unemployment with mortality would be helpful in arguing for or against biological plausibility, but the pattern is mixed. Iversen et al. [106], following Moser et al. [165], show that the effect of unemployment does not diminish a great deal with time, although Moser et al. and Costa and Segnan [58] showed that immediately after unemployment there may be a healthy worker effect, such that the first five-year period is biased towards the null. A different pattern is
observed by Nylén et al. [175], who observe a large hazard ratio during the first 5 years, and lesser but appreciable, though perhaps confounded, hazard ratio in the second 5 years, and a near null finding for the subsequent 9 years of follow-up. Opposite to the findings of Moser et al., Martikainen and Valkonen [149] find that in the first year after the onset of unemployment following a period of employment, adjusted hazard ratios of all-cause mortality are generally elevated in Finnish men and women aged 25-59.

Health selection can be partially addressed through study design. Moser et al., in their 1987 paper in the BMJ, claim they control for baseline health by excluding men who are not looking for work due to health problems [167]. They also argue against selection out of work due to self-destructive health behaviours arising from “personality problems” based on the observation that the association between job-loss and mortality did not diminish in the cohort with the higher unemployment rate.

Health selection can also be examined by analysis of cause-specific mortality and appeal to biological plausibility. Although the number of death events is small in Moser et al.'s 1984 article [165], analysis of excess deaths by cause of death showed excesses for suicide and lung cancer. Suicide deaths may well be due to pre-existing mental health problems, as documented by Kraut et al. [122], and it is not likely that any social exposure could cause a short-term increase in lung cancer deaths, since malignant neoplasms typically require decades to initiate and progress to a fatal stage, and therefore should not be equally responsive during the follow-up in these studies. Costa and Segnan [58], like Moser et al. and Iversen et al., show a non-specificity of effect by cause of death. Bethune [27] observed that in both the 1970s and 1980s, in England and Wales, unemployed men appeared to face a greater increase in cancer than circulatory deaths. This pattern exists in the long-term unemployed in Finland as well [120]. Morris et al. [164] also observed that cancer and circulatory disease were elevated in British men unemployed not due to illness by the same amount, after adjusting for age, town, social class,
smoking, alcohol intake and pre-existing disease.

Martikainen et al. [148] observed that the effect of unemployment on mortality depends strongly on the duration of unemployment, with longer periods of unemployment associated with higher probability of death. While dose-response suggest that a causal mechanism is at work, it does not rebut suspicion of health selection or confounding, which could also create a dose-response association.

Although the number of deaths is very low in Moser et al. 1987 [168], there is some weak evidence that the association between unemployment and all-cause mortality is stronger at younger ages, and that job seeking men are less likely to die the year of the census, but more likely 2 years afterwards. Stefansson [225] observed a strongly decreasing age-specific relative rate with increasing age, with 25-34 year old men having a RR in the range of 5 to 10, and 65-74 year old men very close to the null. Women had modest and statistically weak effects at all ages. This pattern of greater relative risk at younger ages was also seen by Keiding et al. [117]. Bethune [27] observed peak male unemployment associations at 35-44 years, and a greater, and significant, association for women aged 16-44 than for women aged 45-59. This pattern of greater relative risk of death at younger ages within the labour force is typical of socio-economic gradients [246], and may indicate confounding or a common mechanism.

Several recent studies have examined whether mass lay-offs or adjustment for extent of firm down-sizing modifies the apparent effect of job loss on mortality. Martikainen and Valkonen [150] used Finnish data to observe that men in occupations that underwent large increases in unemployment experienced did not have greater short-term increases in mortality than men in occupations that underwent smaller increases in unemployment during the early 1990s recession. This suggests that higher mortality among the unemployed is likely to be selection out of employment based on mortality risk rather than unemployment elevating mortality risk.
Figure 1.8: Pathways leading from job loss due to mass layoff in Pennsylvania to mortality, assuming that job losses resulting from mass layoff are exogenous to health status. Adapted from Sullivan and von Wachter [227]. Rectangles indicate constructs for which they had data, ellipses represent unobserved constructs.

Martikainen et al. [143] subsequently observed that the association of baseline unemployment with 8-year mortality follow-up is attenuated when the job separation is from a firm undergoing a large reduction in its workforce, which is consistent with selection mechanisms dominating the association for these lag times. Similarly, in a large-scale firm-closure study permitting comparison between voluntary and involuntary separations, involuntary separations were not associated with increased mortality risk [224].

However, in a recent paper, Sullivan and von Wachter argued that job loss can affect mortality by (1) decreasing permanent earnings (especially for workers who are older, high-tenured, in the manufacturing sector, or living in economically depressed areas), (2) increasing earnings variability by increasing propensity of non-employment and future job loss, (3) decreasing consumption, and (4) decreasing access to health insurance [227] (see Figure 1.8). While loss of employer-based health insurance in the United States can be highly consequential, whether employer-based health benefits in Canada or other countries with universal health insurance are an important pathway by which job loss might affect mortality is not known.

Job loss as a cause of decreased mortality

Ruhm has proposed that the mortality declines across US states during recessions might be explained by a decreased conflict between work and health investment, by fewer work-
related accidents, and by the absence of work-related stress [198] (see Figure 1.9). This reverses the traditional hypothesis that the net effect of job loss on health is negative. He first observes that all-cause and cause-specific mortality are procyclical\(^2\) (with the exception of suicide) and argues that the principle causes of death and their distribution by age suggest behavioural explanations. He then uses data from the Behavioral Risk Factor Surveillance System (BRFSS) for 1987-95 to document procyclical increases in smoking and obesity and procyclical decreases in physical activity and diet quality.

Ruhm argues that despite the income associated with employment and the consequent decrease in household budgetary constraint, employment is a hazard to health because: (1) it increases the opportunity cost of time, thus raising the cost of time-intensive health behaviours such as exercise and seeking medical care; (2) health may be an input in the production of goods and services in the form of risk of physical injury and psychosocial stress, especially in cyclical sectors such as construction, as well as external health costs due to pollution; (3) risky activities may themselves be market goods, such as alcohol, consumption of which is procyclical;\(^3\) and (4) in-migrants may have negative health effects through crowding, infectious disease, motor vehicle collisions, and ignorance of local medical services, but might also have positive, non-causal net effects due to a healthy immigrant effect [198].

Ruhm’s ecological analysis suggests that across US states the mortality rate of workers aged 25-44 declines to a greater extent with increasing state-level joblessness than

\(^2\)Economists discuss a variety of cycles, including inventory cycles (less than 2 years), business or trade cycles (5-10 years), building cycles (about 20 years), and Kondratieff cycles (60 years) [30]. The terms pro-cyclical and counter-cyclical typically refer to trade cycles, which reflect “alternating periods of upward and downward movements in the aggregate level of output and employment” [33], though they are not regular in amplitude or length [128]. Cycles in unemployment are lagged on cycles in production due to lags in hiring and firing [29]. Traditional explanations of trade cycles attribute fluctuations to real shocks, monetary shocks, and the time-lags in economic decision-making [32]. Unemployment is countercyclical, while vacancies and wages are procyclical [209], where cyclicity is with reference to economic output.

\(^3\)Although there is a body of research indicating procyclical alcohol consumption, Ruhm observed non-significant countercyclicality in the BRFSS data. Among other explanations, he suggests this may be a real behavioural change reflecting changing mores regarding alcohol use and drunk driving [198].
Figure 1.9: Pathways leading from business cycle to mortality, attempting to explain why mortality rates are procyclical in the United States. Adapted from Ruhm [198]. Rectangles indicate constructs for which he had at least partial data, ellipses represent unobserved constructs. Bold arrows indicate positive effects, thin arrows indicate negative effects.
does that of older workers [198]. It is not surprising that contemporaneous associations are stronger in a group in which mortality is dominated by accidents, and Ruhm observes stronger associations with joblessness for these causes of death than for cardiovascular disease and pneumonia/influenza, and no association for cancer. However, it is perhaps surprising that these deaths decline, rather than increase as suicide does, with increasing joblessness. Since the magnitude of the beneficial effect of joblessness on mortality is reduced (by roughly 50%) when state annual mean income is added to the model, Ruhm argues that part of the net hazard of employment is mediated by income.

### 1.6.3 Summary of literature on job loss

In a recent systematic review, the evidence that the experience of unemployment is a predictor of all-cause mortality is considered strong [127]. This is based on high quality census-based cohort studies with 2 to 10 year follow-up periods, where unemployment is self-reported with respect to census day or the week prior to census day.

However, given the potential for confounding and time-dependent confounding of the association between job loss or unemployment and mortality, the unexpected patterns by cause of death, and the mixed findings of more recent studies examining mass-layoffs, skepticism should be maintained about the magnitude and perhaps even the direction of any effect. The arguments presented by Ruhm for health benefits from unemployment, though provocative, lack high-quality empirical evidence.

Examining family disposable income drops using longitudinal tax data is a more direct method of examining some of the pathways by which job loss and unemployment may affect mortality. This approach also allows one to examine whether greater income losses confer greater survival risk, though existence of a dose-response relationship would not be a sufficient rebuttal to suspicion of uncontrolled confounding by work disability, education or occupation.
1.7 Confounding variables: influences on both income and mortality

If a variable is a determinant of both the exposure and the outcome of interest, then it may create an association where there is no causal relationship, or exaggerate, conceal or reverse the estimate of a true causal association. This phenomenon is termed confounding. When the exposures are income and income dynamics, and the outcome is mortality, the most significant confounding variable is work disability. Others are life events, including family breakdown, family deaths, and retirement. One hypothesized mechanism by which such life events are thought to have effects on mortality is by disrupting social support and social networks, on which there is a significant body of research.

The term “life event” is broadly used in the social sciences to refer to changes “in demographic, educational, employment, health, or other individual circumstances locatable to a particular point in time” [204]. Here the term is used to indicate events other than changes in the subject's own health or vital status. Life events are moments when roles, social interactions, attitudes and values may change, possibly improving or diminishing health and well-being through material, behavioural or psychosomatic mechanisms.

Although confounding variables must be associated with both exposure and outcome, the association between family breakdown, family death, and retirement with income level and income drops is not substantially reviewed and is assumed.

1.7.1 Work disability

A useful overview of the incidence and prevalence of work disability in OECD countries has been provided by Prinz [187].
Definition & prevalence  The disabled population is highly heterogeneous, posing problems for definition, measurement and policy. Using standard self-report measures from national surveys in OECD countries, one-third of the disabled have a severe disability, and congenital disabilities are a small minority [187]. In the 1998/99 National Population Health Survey, 16.1% of Canadians aged 20-64 reported some activity limitation. This was similar for men and women, but higher for those aged 50-64 (24.2%) than those aged 20-49 (13.4%), and slightly higher for those with less education [187].

However, there is a surprising lack of agreement between self-reports of work-disability and receipt of work-disability benefits. Among those receiving disability benefits, 30 to 50% do not self-identify as disabled, and even in generous jurisdictions most who self-identify do not receive benefits [187]. This makes the use of health survey data relying on self-report of questionable value for characterizing beneficiaries. The disability benefit recipiency rate for Canada is 4% of working age adults (low by OECD standards), and these are evenly split between contributory and non-contributory benefit programs [187].

Disability benefits & income drops  Employment rates for the disabled are lower than for the non-disabled, negatively correlated with disability severity and age, and positively correlated with education and prevailing overall employment rates [187]. In Canada the employment rate for the disabled aged 20-64 is relatively high at 56.3% compared to 78.4% for the non-disabled [187]. Persons working with disabilities have similar earnings to those without [187].

In Canada the equivalized personal income of disabled persons is 85% of that of non-disabled persons, while equivalized household income is 91% of that of households with no disabled members [187]. These estimates are from the 1998/1999 National Population Health Survey (NPHS), and are based on self-reports of disability and income. Adjustment for determinants of income such as age, sex, education and occupation does not appear to have been performed. In other countries, relative personal income is usually
lower for severely than for moderately disabled persons [187], and the NPHS estimates may be high due to the inclusion many persons with minor disabilities.

As a variable confounding the association of income levels and drops with mortality, the important characteristic of work disability is loss of income, and not necessarily loss of employment or receipt of disability benefits. Tax-based indicators of disability benefit receipt, which are the only available indicators in the analyses presented in Chapters 4 and 5, are therefore a partial proxy, and the extent of residual confounding is difficult to predict.

Canadian disability benefit programs are highly fragmented. There are five main sources of disability income in Canada: the national Canada Pension Plan's disability benefit program (CPPD), the provincial workers’ compensation agencies (WC), the provincial social assistance disability programs (SA-D), private employment-based long-term disability (LTD) plans, and public provincial or private automobile insurance. In addition to income support programs, there are three income tax measures for the disabled: the disability tax credit (DTC), the medical expenses tax credit (METC), and the attendant care deduction (ACD; renamed the Disability Supports Deduction in 2004).

The Longitudinal Administrative Databank captures CPPD (1991+), WC (1992+), DTC (1983+) and METC (1984+). Total CPP (1982+) and total SA (1992+) are also captured. Detailed program descriptions and their capture in tax data are presented in Section 2.2.3.

In brief, CPPD covers only disabilities that completely prevent gainful employment, WC only covers work-attributable disabilities, SA-D is means-tested and covers only disabilities that completely prevent gainful employment (therefore 75% of beneficiaries are single and half have psychiatric/developmental disabilities), DTC is for very severe disabilities without reference to work, and the METC is claimed by 2.8 million people (in 2003), and therefore lacks sufficient specificity to be a useful proxy of work disability. The remaining income sources are not captured in tax data. Since SA-D is means-tested, it therefore has
differential measurement error with respect to income level as a proxy for work disability, and is therefore not suitable for the analyses presented here.

Canada spends a relatively low percentage of GDP on disability benefits. Canada spends less than the US and much less than most of Europe, by having both low rates of receipt and low benefit levels [187]. This is despite the fact that more Canadians (16.1%) self-identify as disabled than do Europeans (15.6%) or Americans (10.7%), though survey methodologies differ and biases are not known [187]. The disabled in Canada rely more on earned income than do residents of comparable countries, including the US [187].

Benefit receipt is therefore a poorer proxy of work disability in Canada than it would be in otherwise comparable countries. It has been estimated that 57% of eligible injuries do not result in a WC claim [208]. It has also been estimated that less than one-quarter of persons with disabilities received income as a result of their disability [77].

Rejection rates for disability benefit applications in Canada (i.e. CPPD) are the highest in the OECD [187]. The appeals process is also unable to promptly respond, and many disability applicants suffer financial hardship while in the application and appeals process [187]. When using benefit receipt as a proxy for work disability it is therefore essential to allow for delayed receipt of benefits.

**Work disability and mortality** Several Scandinavian studies of the mortality of disability pensioners indicate that they have significantly elevated risk of death. In a Danish study of persons aged 60-70 with eight years of follow-up, disability pension beneficiaries, and particularly men, had greater mortality than either the employed or those participating in a non-disability early retirement scheme [188].

Wallman et al. pooled several Swedish cohort studies to examine the effects of disability pension receipt on mortality [242]. Their findings were nearly identical to the more statistically powerful Swedish register-based study of the population aged 16-64 of Östergötland County [114, 115]. In Östergötland County disability beneficiaries showed
three-fold increased risk of death during a twelve year follow-up. The relative risk of death given disability benefits was greater at younger ages and greater for those receiving full rather than partial benefits. All-cause mortality was greater among those disabled due to musculo-skeletal disorders (women: HR=1.2, 95% CI: 1.1–1.3; men: HR=1.1, 1.0–1.2), psychiatric disorders (women: HR=2.4, 2.2–2.7; men: HR=3.2, 2.9–3.4), and cardiovascular disorders (women: HR=3.2, 2.8–3.6; men: HR=2.7, 2.5–2.9). Among those with psychiatric diagnoses, risk of suicide death was extremely high for women (HR=12.0, 8.4–17.2) and men (HR=5.5, 4.0–7.6). During the twelve years of follow-up, the hazard ratio appeared to be either constant or slightly u-shaped [115].

The finding in Sweden of excess mortality for causes unrelated to the diagnosis for disability eligibility is consistent with evidence that the health care utilization of early retirement disability beneficiaries in Sweden is elevated for conditions un-related to diagnosis [241].

In Norway a similar effect size among disability pensioners was observed during a six-year follow-up. However, among men, adjusting for level of education decreased the hazard ratio from 3.4 to 2.0, and for men with musculo-skeletal diagnoses adjusting for education eliminated the association of disability pension receipt with all-cause mortality [87].

These findings are consistent with the known differences among chronic diseases in terms of their contributions to disability and mortality. Manuel et al. [141] show that in Canada there are high-mortality-low-prevalence conditions, such as cancer, which make larger contributions to lost life-expectancy than to lost health-adjusted life-expectancy. At the other extreme there are low-mortality-high-prevalence conditions, such as osteoarthritis, that make large contributions to lost health-adjusted life expectancy, particularly for women, but essentially none to lost life-expectancy. These estimates were adjusted using the Health Utilities Index (HUI-3), which uses survey-elicited public preferences of various
health states, and not a direct assessment of the extent of work disability.

Not all work disability is related to chronic disease. Most lost-time injuries compensated by the Ontario Workplace Safety and Insurance Board, for example, are traumatic injuries or musculo-skeletal injuries due to cumulative exposures [249]. Tomiak et al. found that musculo-skeletal disorders and disorders of the eye and ear accounted for over 80% of self-reported activity limitation for men and women in Manitoba [231]. These disabilities may have little consequence for age-at-death, as with osteoarthritis. Because many compensable causes of work disability likely have only small effects on mortality, while a subset of the disabled experience significantly elevated mortality risk, statistical adjustment for receipt of disability benefits would not be an effective control in analyses of the effect of income on mortality. Use of disability benefit receipt as an exclusion criterion is preferable.

It is assumed that uncontrolled confounding due to work disability would be most apparent with short lags between exposure and outcome. If health status deteriorates progressively from a period where work is not affected, through a period where work is affected and income is sometimes affected, to a period where work is not possible, with probability of death rising throughout this progression, then the disease process would have to be extremely slow-paced for work-disability to regularly cause income drops more than five to ten years prior to death. If health care utilization is taken as a proxy of disability, then given that nearly one-third of health care utilization occurs in the last year of life, and of this half occurs in the last 60 days [136], it is not expected that a large number of disabled persons whose condition is potentially fatal suffer income losses many years prior to death. This is an area that deserves further research.

Defining work disability using data from the Longitudinal Administrative Databank is discussed in Section 2.2.3.
1.7.2 Social support and social networks

Many life events derive part of their importance from their disruption of social networks and social support. Home and work are where we spend most of our time, and family breakdown, family deaths, retirement and job loss can be highly disruptive of our relationships in these settings.

Social support was proposed as a determinant of physical health in the 1970s separately by Cassel and Cobb in an extension of the literature on the effects of stress. It was proposed to maintain or sustain “the organism by promoting adaptive behaviour or neuroendocrine responses in the face of stress or other health hazards, [providing] a general, albeit simple, theory of how and why social relationships should causally affect health.” The proposed adaptive behaviours include proper sleep, diet, exercise, appropriate use of alcohol, cigarettes and drugs, and seeking for and complying with medical care [101]. Social support is derived from relationships that may be intimate, instrumental, or extended. Instrumental, or effective, relationships provide practical economic, political, and logistical support. Extended relationships involve the familiar faces who may help one to get a job or to find a good doctor, but don’t provide emotional support [26].

Experimental evidence from animal studies has provided strong support for a causal effect of social support on health. The presence of a familiar same-species animal reduced the effects of experimental stress on ulcers in rats, hypertension in mice, and neurosis in goats, as well as anxiety and markers of physiological arousal in humans. Cardiovascular responses to stress are also reduced in dogs, cats, horses and rabbits when petted affectionately by humans, and human handling reduced diet-induced atherosclerosis in rabbits [101].

Social networks are thought to be important not only because of the social support they provide. Types of networks include marriage, contacts with family and friends, church membership, and formal or informal group affiliations. Early prospective studies
in Alameda County (California), Tecumseh (Michigan), Evans County (Georgia), Gothenberg (Sweden) and North Karelia (Finland) showed that the number and frequency of self-reported social relationships and contacts predicted all-cause mortality after adjustment for baseline health from self-report or physical examination [101]. In other observational research, lack of intimate emotional support has been associated with a tripling (odds ratio of 1.2 to 6.9) of mortality risk after myocardial infarction when adjusting for age, severity, and quality of care [26].

### 1.7.3 Family breakdown

Family relationships, and marriage in particular, are an important source of intimate emotional support [26]. Family breakdown, such as separation or divorce, is both a source of acute stress and likely an indicator of preceding chronic stress [155].

But as with many social risk factors, it is difficult to establish whether divorce causes ill health (causation), the unhealthy are more likely to get divorced (selection), or divorce and ill-health are co-determined but not causally related (confounding) [101]. Adding to this complexity, while several risk factors for divorce are also known risk factors for mortality, suggesting the presence of confounding, there is also some evidence that poor health can decrease the risk of divorce by incenting the maintenance of marriage, a phenomenon termed “adverse selection” [55].

There is a long literature on social networks, including marital relationships, and mortality. In the United States, never marrying is associated with a greater risk of premature mortality, and the predominant causes of excess death depend on age [112]. However, the focus on cross-sectional or baseline marital status in the social networks literature has not typically allowed for rigorous causal analysis of the effect of family breakdown.

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4The term “dissolution” is common in the discussion of divorce, and is a legal term for any of the processes by which marriages and similar unions or partnerships are terminated [151]. It is also, however, sometimes used to refer to both separation or divorce, but this is not technically correct, since it never means separation only, and is synonymous with divorce only in some jurisdictions.
Divorce may have a negative effect on mental health, increasing negative affect, diminishing sense of purpose and identity, and damaging social networks, and these, in turn, may have effects on longevity. In the Multiple Risk Factor Intervention Trial (MRFIT) study, divorced men were at increased risk of all-cause, cardiovascular, digestive system disease and accidental death, and the unadjusted association was negatively confounded by known risk factors for cardiovascular disease, despite divorced men being much more likely to smoke at baseline [155].

In Japan, the death rate is three times higher for divorced men compared to married men, which is much greater than the hazard for divorced Japanese women, men internationally, or for widowed Japanese men or women. Whereas in most countries the divorced are at higher mortality risk than the never married, the reverse is true in Japan for men and women [89]. Internationally, the smaller the proportion of persons who are divorced, the greater their risk of death relative to the married population, suggesting that a selection mechanism is at least partially responsible for the association [105]. This is also consistent with the observation in the Whitehall cohort that divorced men have an increased risk of cancer mortality that is not fully explained by their higher rate of tobacco use [24].

Although physical correlates of poor health such as obesity and short stature are not correlated with marital dissolution, risk-taking health behaviours, such as smoking and drug use, are strongly related to marital dissolution [80]. This suggests the existence of common psychosocial determinants of marriage success and longevity.

Defining family breakdown using data from the Longitudinal Administrative Databank is discussed in Section 2.2.4.
1.7.4 Family deaths

Death of a spouse, or bereavement, has been repeatedly associated with increased risk of mortality, and it is commonly thought to be a causal association. Three types of mechanism are commonly suggested: (1) emotional stress and host resistance, (2) loss of spousal social support and of social networks maintained by the spouse, and (3) loss of material or task support [145]. Whether the first and third are not elements of the second is debateable.

The causes of mortality most associated with bereavement are accident, alcohol, suicide and violence, with chronic ischaemic heart disease and lung cancer somewhat increased, and other causes only slightly increased. Risk is greater in the first six months of bereavement, among those aged less than 65, and among men [145]. These patterns are consistent with a behavioural and psychosomatic response to bereavement. Because bereavement appears to increase mortality primarily in the short-term, it may play a role in accelerating or exacerbating preexisting conditions, rather than initiating new ones. An example of this might be decreased compliance with medical care [145]. However, in the Whitehall cohort, widowers had increased ischaemic heart disease mortality even after excluding deaths in the first two years after their spouses’ deaths [24]. The effect of bereavement on mortality is proportional in education groups, but since mortality decreases with increasing education, this may mean that education is associated with resources that buffer the effects of bereavement to the same extent that they buffer the mortality effects of other health hazards [146].

There are analytically challenging sources of bias in bereavement studies. Spouses share many behavioural and environmental health exposures, such that early death of one spouse may in part only reveal that the widow(er) was already at increased risk of mortality [145]. This may explain the apparent short-term increases in mortality for chronic conditions such as cancer. Spouses may also die from the same accident [144], and dates
of death need not be identical.

Hart et al. used data from the Renfrew and Paisley study to examine confounding of the association of bereavement with cause-specific mortality [98]. All cause, CVD, CHD and stroke were adjusted for age, sex, systolic blood pressure, smoking, cholesterol, body mass index, height, social class, deprivation category, adjusted FEV1, angina, ischaemia on ECG, previous MI and bronchitis. Other causes were adjusted for age, sex, systolic blood pressure, smoking, cholesterol, body mass index, height, social class, deprivation category and adjusted FEV. They found elevated risks of death during the first and subsequent 5 year periods after bereavement for both chronic and acute causes of death, though the adjusted results for lung cancer were ambiguous and non-smoking-related cancer showed no association. The strongest associations were for accidents and violence, and these were unaffected by the adjustments and were not attenuated in the second period.

Defining family deaths using data from the Longitudinal Administrative Databank is discussed in Section 2.2.4.

1.7.5 Retirement

Definition and measurement

The paradigm case for retirement is the full-time, male, salaried employee who, at the end of a given day in his mid-60s, completely exits the labour force and begins collecting a pension. However, most labour force participants do not exit the labour force in such a clearly defined way. Because of the many different types of labour market attachment and often gradual exit, the definition of retirement is “fuzzy, to say the least,” partly explaining the lack of regular statistics on the retired population [34]. In their review of definitions of retirement, Smeeding and Quinn begin by stating that “One would be hard pressed to find a less [sic] murky term or one with fewer questions attached” [212].
Retirement is both an event and a state. While statistical agencies might be more interested in measuring the state of retirement [34], an analysis of the mortality effects of retirement clearly requires measuring the event. Measurement of the state of retirement is also complicated by the fact that many people come out of retirement. In the 2002 General Social Survey, of those who retired between 1992 and 2002 and were retired when surveyed in 2002, 22% reported having returned to paid employment at some time after their first retirement, with women more likely to have done so than men (25% versus 18%). Returning to work was strongly related to age at retirement with 28%, 14% and 17% returning if they retired at ages 50-59, 60-64 or 65+. Returning to work was also related to occupation, ranging from 16% among sales/service and trade workers to 33% among professional workers [235, p.135].

Some definitions of retirement require complete labour market withdrawal, while others allow for fully active participation [34]. Statistics Canada has a standard definition of retirement that requires the former worker to be (1) aged 55 or older, (2) not in the labour force, and (3) receiving 50% or more of their income from retirement-like sources. The development of this definition is undocumented, and moreover can only be estimated from household surveys that have detailed income and labour modules, which in Canada includes only the Survey of Labour and Income Dynamics (SLID) and the Census [34].

The Labour Force Survey (LFS) asks those not currently working when they last worked. If they last worked within the previous year, they are asked why they left their last job or business, and one of the response categories is “retired.” In 1995, for example, 4.8 million Canadians age 15 and over were not in the labour force, of whom 273 thousand had worked in the previous 12 months. Of these, 41% described themselves as retired, while 34% and 10% described themselves as layed-off or disabled, respectively. The date of retirement is taken to be the date last worked, but those aged less than 50 are excluded from estimates based on the LFS [91].
Using the SLID, Giles defined retirement separately for those aged 55-69 and those 70 and over [84]. For the 55-69 group, retirement was defined as (1) not in the labour force at any time during the previous year, (2) did not want or look for work at any time during the previous year, (3) had pension/CPP/QPP income or had five or more years of lifetime work experience. Using this definition, which is very similar to the Statistics Canada definition described above, he observed that in 1993 58% of those aged 55-70 were retired, and while for men this did not depend strongly on whether they were living with a spouse, for women 51% of those living with a spouse and 70% of those not living with a spouse were considered retired. While retirees in general have lower incomes than the general population, retired women not living with a spouse are particularly poor, with 61.6% having family income in the lowest quintile (for men this figure is 44%).

Smeeding and Quinn argue that the meaning of retirement to workers and families depends on a nation’s disability, unemployment, and retirement system, as well as their labour market institutions [212]. Any connection between health insurance and labour market participation should probably also be considered. They also conclude that the appropriate definition of retirement may depend on the policy question being analyzed.

Defining retirement using data from the Longitudinal Administrative Databank is discussed in Section 2.2.5.

**Trends in age of retirement for men and women**

Recent trends in the age of retirement for men and women in Canada according to the Labour Force Survey (LFS) are shown in Figure 1.10. In the periods 1970-1985 and 1985-1989 the labour force participation rates for men aged 55-64 in Canada declined substantially but not exceptionally when compared to other OECD nations [212]. The reductions in the age of retirement in Canada were not consistent across across industries. Reductions were strongest in government, utilities and communications and weak-
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Figure 1.10: Recent trends in the age of retirement for men and women in Canada according to the Labour Force Survey [34].

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<td>65-69</td>
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<td>139</td>
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<tr>
<td>70+</td>
<td>48</td>
<td>45</td>
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<tr>
<td>TOTAL</td>
<td>407</td>
<td>620</td>
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Table 1.8: The distribution of age at retirement for workers aged 50 or greater by self-report in the Labour Force Survey [91].

est among the self-employed. Among self-employed agricultural workers the retirement age actually increased between 1976-80 and 1991-95 [91].

A popular method for excluding the retired from analyses of working-aged adults is to restrict the analytic sample to those aged less than 60, or less than 58 [15]. But using the LFS definition, between the late 70s and early 90s the fraction of retirements at ages 50 or greater occurring before age 60 increased from 16% to 34% [91] (see Table 1.8), which suggests that this is at best a partial solution.

Association with premature adult mortality

As with job loss, analysis of retirement as a determinant of health is plagued by problems of selection bias, with selection occurring out of the labour force into retirement (with or without disability pension income), and also out of retirement at labour market re-entry.
In 2002 the General Social Survey interviewed Canadians who retired between 1992 and 2002. 71% had retired voluntarily, while 29% had retired involuntarily. 44% of involuntary and 20% of voluntary retirees attributed their retirement in part to their own health problems, and 7% and 8% to caring for family members (multiple attributions were permitted).

The attribution of retirement to health problems is slightly related to age at retirement, with 28%, 25% and 24% reporting at ages 50-59, 60-64 and 65+ respectively, and is strongly related to education, with 34%, 23% 28% and 17% reporting among those with less than high school, high school, college, and university education respectively. 27% of men and 26% of women in this sample said that if their health were better they might continue working, and 6% and 7% said the availability of suitable caregiving arrangements would have allowed for continued labour force participation [235, p.133]. With self-reported attributions of retirement decisions, there may also be social-desirability and recall biases influencing the data.

Among those who rated their health in 2002 as excellent, very good, good, or fair/poor, 27%, 22%, 20% and 10% had returned to work, respectively [235, p.135]. However, it is not known whether this strong gradient in labour market re-entry by health status is due to health selection at labour market re-entry or to causation, where returning to work improves self-rated health.

Snyder and Evans exploited a change in social security policy to perform an instrumental variable analysis of the effects of income on elderly Americans, and observed that the higher income group had higher mortality rates. In an effort to reconcile this with the literature showing the opposite association for income and mortality, they document that the younger and poorer cohort partially offset their lower income with increased levels of post-retirement employment. The authors suggest that employment is therefore beneficial to health, or conversely that retirement is a hazard [217].

Quaade et al. attempted to examine the bidirectional relationship of health and re-
Chapter 1. Social epidemiology

tirement using data from two publicly financed retirement programs in Denmark, one a
disability retirement program, and the other program was an early retirement program for
workers on long-term unemployment benefits [188]. They observed that disability ben-
eficiaries had a high relative risk of death immediately after retirement, and concluded
that disability benefits were clearly selecting those in poor health. Compared to mortality
levels in the employed and those receiving disability benefits, the early retirement benefit
population had an intermediate mortality rate that rose over time after retirement, and
they could not determine if this was a risk associated with retirement or the absence of
health selection into the labour force.

1.7.6 Immigration

Statistics Canada made large investments in research on the economic outcomes of
new immigrants, and 64 papers produced between 2002 and 2008 were reviewed by
Picot [183].

Types of immigrants

There are four classes of immigrants to Canada. The skilled economic class requires
proof of a minimum level of human capital; the family class requires a family sponsor;
the refugee class requires proof of need for refuge; and the business class, which covers
a small fraction of immigrants, requires proof of entrepreneurial intent. The number of
illegal immigrants is thought to be very small. In the early 1980s 37% of immigrants to
Canada were economic migrants, while 43% were family migrants. By 2001, 54% of
immigrants were economic migrants, while only 31% were family migrants [183]. Up to
35% of new immigrants subsequently leave Canada, and this occurs primarily during the
first two years [183].
Europe is no longer the primary region of origin for Canadian immigrants. Of immigrants who landed between 1981 and 1991, 75% were from non-European countries [54].

**Economic experiences of immigrants**

Men immigrating to Canada in the late 1970s earned about 85% of Canadian-born earnings, but after 25 years caught-up with or surpassed them. During the 1980s and 1990s, the economic situation of recent immigrants deteriorated substantially and continuously. Men immigrating between 1990 and 2004 earned only 60 to 65% of Canadian-born earnings and appear unlikely to catch-up [183]. This has been attributed to changes in country of origin, language and quality of education; to declining returns on foreign experience, such that by the late 1990s there was no return on foreign experience; and to the broad deterioration in economic outcomes for labour entrants of all kinds [183]. The widening earnings gap between recent immigrants and Canadian born workers occurred in spite of the rising education levels of immigrants, and the eventual convergence of earnings with those of Canadian-born workers grew less certain. During the 1990s adult recent immigrants’ income declined in both absolute terms and relative to that of Canadian born workers [183].

Relative and absolute low-income also became increasingly prevalent with successive cohorts. The proportion of recent immigrants with family incomes below the Low-Income Cut-Off (LICO) was 25% in 1980, 31% in 1990 and 36% in 2000, despite these years being at business cycle peaks, and despite contemporaneous decreases in low-income rates for the Canadian-born [183]. Changes in selection criteria for economic immigrants made in 1993 had small positive effects on new immigrants’ earnings, but not towards the bottom of the income distribution [183]. During the first year after immigration, 35 to 45% of immigrants experience poverty, and about one-fifth experience longer-term poverty. Those who do not enter poverty in the first year are not at high risk of entering
subsequently [183].

**Mortality of immigrants**

Life expectancy at birth is greatest for non-European immigrants and least for Canadian-born men and women. Between 1986 and 1991 non-European immigrants made much larger gains. This ordering is also true for age-specific mortality rates at working ages for men and women [54]. These findings do not take any account of the year of landing or duration of residence. Non-European immigrants will have shorter durations of residency and more recent year of landing. The explanation for the survival advantage of recent immigrants is thought to be that persons in ill-health are less likely to migrate. Potential immigrants to Canada undergo medical screening, and are also less likely than the Canadian-born to have ever smoked [54].

### 1.7.7 Summary of literature on confounding variables

Work disability, family breakdown, family deaths and retirement are all associated with decreased income and increased mortality, and therefore may confound analyses of income level or income drops and mortality. The available measures of work disability in tax data, such as are available in the Longitudinal Administrative Databank, are imperfect, and residual confounding may bias estimates. The magnitude of such biases are difficult to predict. It is possible, however, that the health conditions that typically lead to work disability do not substantially increase risk of death, and that biases may be modest. By examining the effect of including or excluding those persons who are likely to have work disability, the potential magnitude of residual confounding may be described.

Family breakdown is common, and can dramatically change equivalized family disposable income by changing the number of family members and the number of earners. Even small effects on survival could create confounding of the effects of income level or income
drops on mortality. To control for confounding, families that change structure during the period of income ascertainment will be excluded from models of the effect of income level or income drops on mortality.

Family deaths can also change family size, number of earners, and sources of income. Because bereavement is associated with mortality, the bereaved, which are a small group at working ages, will also be excluded.

Retirement involves cessation of earnings and the start of dissavings. The decision to retire is partly determined by health and expectation of survival. It is therefore important to exclude the retired, and particularly those who retire early. Typical age restrictions used to exclude the retired are not sufficient, and sources of retirement income will be used in conjunction with an young exclusion age of 55.

Recent immigrants have decreased income levels, decreased mortality rates, and perhaps increased incidence of income drops. Immigrant status may therefore confound estimates of the effect of income level and income drops on mortality towards the null, and immigrants should be excluded from the analyses.

### 1.8 Hypotheses

#### 1.8.1 Hypotheses for Chapter 4

Income level is expected to be associated with lower hazards of death, and income drops with greater hazards of death, in both Canada and the United States. Both exposures are expected to show stronger effects in the United States. The previous findings by McDonough et al. notwithstanding, higher income level is expected to reduce the effect of income drops on hazard of death in both countries.
1.8.2 Hypotheses for Chapter 5

Income level is expected to show a negative association with risk of death. Health selection requires that the onset of work disability causes income to decrease. Therefore, if health selection is a significant contributing mechanism to the association of income level and risk of death, then income level should have a greater effect in the presence of income drops. Health selection is hypothesized to play a small role, and therefore the effects of income level and income drops on risk of death are not expected to have a positive statistical interaction.

Drops in annual family disposable income during peak years of labour-force attachment are expected to increase the hazard of death over the subsequent 18 year period. The effect size and causal interpretation depend on the induction period. Effects given short induction periods are most plausibly due to health selection or to accidental deaths due to increased risk-taking. Longer term effects are more likely causal and due to an aggravation of chronic disease processes. Income level is expected to modify the effect of income drops. Notwithstanding the observations of McDonough et al. [156], higher income is expected to be associated with decreased effects of income drops due to reduced cash constraint thanks to greater assets, and so a negative statistical interaction is expected.
Chapter 2

The Longitudinal Administrative Databank

The Longitudinal Administrative Databank (LAD) is the data source for the empirical work in Chapters 4 and 5. The following sections describe the administrative data processing, cohort structure, data access, and the direct and proxy measures used for statistical modelling.

2.1 Data source

2.1.1 Data processing

When Canadians file their annual T1 personal income tax forms, Canada Revenue Agency, after a delay, provides this information to Statistics Canada. The Small Area and Administrative Data Division at Statistics Canada reconstructs family relationships each year in the T1 Family File (T1FF). Samples of the 1982 and subsequent T1FF have been linked to create the Longitudinal Administrative Databank (LAD). The LAD is a 20% simple random sample of the Canadian Population, and can therefore provide extremely precise and accurate estimates of the income sources and income dynamics of Canadians. Currently the year of death is captured from T1 returns, but the cause of death is not available in
tax data.

This data source is unique in Canada for its combination of longitudinal nature, sample size, representativeness, long coverage period, low loss to follow-up, annual measurement, use of mortality as a marker of health and detailed income descriptors. The principle limitation of the LAD is that it lacks data on constructs such as occupation, education, and health status, which might confound some estimates of association. However, disability benefit income and disability or health care tax deductions can proxy for direct measures of work disability, as discussed below.

### 2.1.2 The LAD cohort

It is helpful to visualize the data available for survival analysis of the individual-level association of income history and subsequent mortality. Because exposure at specific ages is of interest, and because the role of induction time is also of interest, it is easy to lose track of the cohorts in question and the limitations of the data.

Lexis diagrams of the LAD data are shown in Figure 2.1. The dotted horizontal lines indicate the age range for exposure, defined as 40 through 55. In Chapter 5 the five years prior to age at exposure are used as income history for estimating individual income trajectories. When exposure is defined as occurring in a given age range, then latency determines age at death, and implies different disease mechanisms and patterns of confounding and effect modification. The top panel shows the most recent exposure window. Because there may be a two-year delay in receiving disability income, the two years after exposure are used for this exclusion criterion. The middle panel shows the longest latency that can be analyzed using only data from 1992 onwards, when transfer amounts are better captured in tax data. The lower panel shows the longest latency that can be analyzed using all years of data.

Note that as lags increase, the eligible cohorts become increasingly historic, such that
Figure 2.1: Lexis diagram showing birth cohorts in LAD data that contribute to the analyses in Chapter 5. The top panel indicates the shortest latencies, the middle panel shows the longest latencies that can be analyzed with the higher quality data available starting in 1992, and the lower panel shows the longest latencies that can be analyzed using all years of data. Birth cohorts for each exposure year are annotated on the panels.
cohort effects are a potential influence when investigating different latencies. The range of year of birth for exposure years is indicated in each panel. There may be cohort effects in the population born between 1932 and 1942, such as childhood malnutrition during the depression, or expanded labour market opportunities for those with post-secondary education in the post-war period. Cohort effects will not be examined in this dissertation.

2.1.3 Analytic samples

Chapter 4 sample

The analytic sample for the Canada-US comparison presented in Chapter 4 is restricted to persons aged 45-64 at baseline following the inclusion criteria used by McDonough et al. [156]. This sample had over 500,000 persons and 40,000 deaths.

Chapter 5 sample

The analytic sample for the analyses presented in Chapter 5 is restricted to persons who are at risk of exposure to income drops at ages 40-55. These years of exposure are being referred to as person-years of exposure (PYE). Since the previous five years’ income is used to determine income level and project expected income, filers aged 35-50 in 1982 contribute the earliest exposure data for PYE in 1987. These age exclusions reduce the potential confounding by births, marriages and retirement. The LAD contains approximately 833,000 filers aged 35-50 in 1982, rising to 1.5 million in 1998, which is the start year for the final exposure assessment period. This represents 9.6 million PYE for women and 9.7 million for men. Separate models are estimated for men and women to allow for effect modification by sex. Likewise, effect modification by family type is also addressed by stratification into two groups: unattached and couple families. Single parents are excluded due to their insufficient number.
Recent immigrants are excluded since the labour market experiences and income dynamics of recent immigrants are significantly different from the rest of the population. Immigration is defined in the LAD as those landing in Canada on or after 1982 and represented 5.8% and 6.4% of PYE for women and men.

PYE are excluded if more than two of the previous five years of income were missing, which is the case for 3.0% of women and 3.1% of men. PYE are excluded if income data are missing for the exposure year, which further reduced PYE by 1.6%. Year-over-year income drops are also only estimable when the exposure year and the previous year both have non-missing income data, reducing the sample by 1.6% in those models.

Models are estimated with and without further exclusion of potential confounding variables. The following exclusions apply to the exposure year and the previous five year period: family receipt of retirement (pension/superannuation) income, death of a family member (a tax-filer or spouse), change in family structure (couple family, unattached, single parent), greater than 20% family self-employment income. Self-employment income was defined as receiving greater than 20% of total family income from self-employment sources during the exposure year or any of the previous five years. Self-employment income sources were limited partnership income, dividend income, business income, professional income, commission income, farming income, fishing income, or rental income. Total family income is defined as self-employment income plus employment income from T4 slips and other employment income.

Exclusion for work disability is based on the exposure year, any of the five previous years or any of the two subsequent years. Subsequent years’ data are used to allow for administrative delays in benefit receipt. The disability flag was set when any family member reported income indicative of work disability (CPP/QPP Disability, Worker’s Compensation, Disability Deduction Amount). Disability and retirement income were defined as family, not individual characteristics to avoid problems related to their transferability on tax
forms and to err on the side of sensitivity.

Models are also estimated using only data from 1992 onward, when transfer amounts and deaths are better captured and filing rates are higher.

### 2.1.4 Research access

Analysis of the LAD data is done remotely with the assistance of the staff in the Small Area and Administrative Data (SAAD) division at Statistics Canada. SAS code is sent by email, then checked and executed on the LAD data by SAAD staff. All output is vetted for confidentiality, which includes the suppression of tabular cell counts less than five, and the rounding of all tabular cell counts to the nearest 5. Confidentiality is also maintained by the use of disturbance weights. These weights are near 1, plus a small amount of random noise with a mean of 0, such that they do not affect model estimates but make deductions about individuals more difficult.

### 2.2 Measures

#### 2.2.1 Vital status

Coverage, representativeness, and capture of death events

The coverage and representativeness of the LAD data are high since tax filing rates are high in Canada. However, filing rates have been rising over time, with filing rates improving most for lower income, older, and female residents of Canada. Increased filing by lower income residents will change the distribution of income over time and change estimates of economic mobility, but this bias is thought to be very small [18].

Figure 2.2 shows that throughout the period 1982-2004 all age groups older than 40 became more numerous in a roughly linear way, as is shown in official population esti-
mates in Figure 2.3. At younger ages there were sharp population declines as the “bust” generation followed the postwar “boom,” followed by population recovery as the “echo” generation started entering the labour force at the end of the 1990s. Figure 2.4 shows the ratio of LAD population estimates to population estimates drawn from the Canadian census. LAD estimates of the population are very close to official estimates for all years for men and women of working age, but during the 1980s filing rates appear to have rapidly increased from rather low levels among the retired population.

Figure 2.5 shows that strong demographic changes at younger ages are reflected in death numbers, and the expected greater number of male than female deaths at ages below 80 is observed. These figures follow a very similar pattern to official figures (Figure 2.6), with the ratio of LAD to CANSIM deaths rising from 40-60% at working ages in 1982 to roughly 80% by 2004 (Figure 2.7). Among the retired, capture of death events also started from low levels but reached, and sometimes slightly exceeded, 100%. These data could be improved by linkage of the LAD to the Canadian Mortality Data Base (CMDB), but this linkage has not been possible in time for these analyses.

LAD counts of deaths are lower than CANSIM counts of deaths for two reasons. First, not all residents file taxes, and second, not all taxfilers’ deaths are captured. To estimate the under-ascertainment of taxfiler deaths directly, crude mortality (in 5 year bands, computed without regard for the timing of the denominator estimate) are estimated separately for the LAD and CANSIM data. The LAD follows the expected decreasing trend for men, but, with the exception of younger age groups, women show increasing mortality rates in the LAD (Figure 2.8). Figure 2.9 shows estimates of mortality rates using CANSIM data, which reveal steeper negative slopes for men and slow declines for women. As is clear in Figure 2.10, the ratio of LAD to CANSIM mortality rate estimates slightly exceed the ratio of LAD to CANSIM death estimates at working ages. Once the underlying trend in filing rates (and therefore LAD population estimates) are taken into account by computing
mortality rates, the retired population shows trends like the working-age population. We also see that in all years LAD mortality estimates improve with age.

A simplified picture of the ratio of LAD to CANSIM mortality rate estimates is presented in Figure 2.11, showing the ratio rising by age and by year, with an overall mean of 73%. The marked increase in the ratio of LAD to CANSIM mortality ratio with age after retirement is likely due to some combination of a decreasing filing propensity among the living and an increasing filing propensity associated with estate closure, therefore perhaps suggesting a bias in the denominator rather than improved data quality.
**Figure 2.2:** Population in LAD 2004 Register 20% sample by age, sex and year (sample weights).
Figure 2.3: Population in Canada (CANSIM II) by age, sex and year.
Figure 2.4: Population in Canada (LAD/CANSIM) by age, sex and year (LAD estimates inflated with population weights).
**Figure 2.5:** Count of deaths in LAD 2004 Register 20% sample by age, sex and year (sample weights).
Figure 2.6: Count of deaths in Canada (CANSIM II) by age, sex and year.
Figure 2.7: Ratio of count of deaths estimated using LAD data compared to official count of deaths for Canada from CANSIM data, by age, sex and year (LAD estimates inflated with population weights).
**Figure 2.8**: Mortality rate per 100,000 in LAD 2004 Register 20% sample by age, sex and year.
**Figure 2.9:** Mortality rate per 100,000 in Canada (CANSIM II) by age, sex and year.
**Figure 2.10**: Ratio of mortality rates per 100,000 based on LAD data to mortality rates based on CANSIM data, by age, sex and year.
**Figure 2.11**: Ratio of mortality rates based on LAD data to mortality rates based on CANSIM data, by year and by age. Horizontal lines drawn at 1.0, 0.8, and 0.6, as well as the mean value of 0.73. Lowess smooths illustrate the trends. Each plotting point represents one age*sex*year mortality ratio estimate.
Under-ascertainment and bias of estimates from Cox models

Estimates of crude mortality rates from the LAD for the age groups used in Chapters 5 and 4 range from about 50-80% of the official mortality rate for women and 60-80% for men, increasing by calendar year. This substantial under-ascertainment of mortality may represent a source of bias, since ascertainment may be related to income. This is not traditional loss to follow-up, since live follow-up exists until the missed estate-closure filing (in the LAD, taxpayers who file more than 6 months late are not captured in that year), but there will be some non-random missing data on vital status.

The dependent variable in survival data consists of three components: date of first contact, date of last contact, and vital status at last contact. The traditional problem with the dependent variable in survival data is loss to follow-up, in which both date of last contact and vital status at last contact are missing (e.g. [35]). Missing data are often classified as missing completely at random, missing at random, or missing not at random [132]. Simulation of loss to follow-up using this framework has shown that only missing not at random, the case where loss to follow-up is correlated with the exposure, biases estimation of exposure effects [124]. No papers could be identified that investigated the consequences of underascertainment in data where live follow-up was possible, such that the dates of first and last contact were known, but vital status at last contact were missing.

Simple simulation reveals that if income is strongly and positively associated with survival, and half of subjects are censored, 50% underascertainment of deaths can reverse or exaggerate the apparent effect of income, given strong negative and positive associations of income with underascertainment, respectively. Since income is expected to be negatively associated with underascertainment to an unknown degree, an unknown degree of attenuation of apparent income effects, or even their reversal, is anticipated in proportional hazards regressions on income alone. The consequences of underascertainment in multiple regression models are more difficult to predict.
The only comparison data on income and mortality are those from the linkage of the 1991 Census to the Canadian Mortality Database [246]. However, the linkage relied on tax data, and therefore similar biases are present. The linkage success rates reveal the potential for bias. The linkage rates were 82%, 81%, 79%, 74% and 66% for the richest through poorest quintiles, defined using the ratio of pre-tax, post-transfer household income to the relevant Low Income Cut-Off (LICO), which adjusts for family size and community size.

**Time aggregation bias**

The LAD captures year, but not month or day, of death from T1 tax form data. This causes imprecision in the measurement of date of death and introduces the problem of many tied event times to survival models. If we consider the period 1992-2004, and use the first 5 years for income ascertainment, then there are 8 years of survival data, which means 8 possible survival times, instead of 2922 possible survival times if day of death were captured.

Because it is technically possible to add month and day of death from electronic records of T1 data, the analytic benefits of this data improvement were briefly explored, and the results are described in Section 3.1.1. It was found that there was no loss in precision or bias in the estimates when using aggregated time and using the TIES=EXACT option in SAS PROC PHREG (and PROC TPHREG).

Although it is computationally intensive, it is nevertheless possible to use exact estimation in SAS with the LAD data. SAS does not allow for the use of weights in conjunction with exact treatment of ties [200, p.3239], but since the LAD weights are disturbance weights for confidentiality purposes, rather than probability weights (the LAD is a simple

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2. The original algorithm by Kalbfleisch and Prentice [108][109, p.74] scaled at \(O(n!)\), while that of Delong, Guirguis and So scales at \(O(n^2)\), where \(n\) is the number of ties [65].
random sample), this limitation has no analytic cost.

While there is no apparent analytic cost in Cox proportional hazards models when an exact likelihood is used, descriptive statistics and plots cannot be stratified more finely than by year. This is acceptable for the current analysis.

**Summary of measurement decisions on vital status**

Year of death will be used as routinely captured in LAD data. Additional capture of month and day was deemed unnecessary. Linkage of the LAD to the Canadian Mortality Database to increase capture of death events and add cause of death was not possible in time for these analyses.

### 2.2.2 Income

The definition and measurement of income were discussed in Section 1.4.2. The LAD contains very high quality data on income components for individuals and families.

**Total income**

The Canada Revenue Agency (CRA) definition of total income (TIRC) relates to taxable income only, and so LAD staff have created a variable (XTIRC) which includes both taxable and non-taxable sources of income. Both of these variables undergo small revisions periodically to reflect changes in the tax forms. For the 2003 tax data, the components of XTIRC were:

- Employment Income
  - Total Earnings from T4 Slips T4E
  - Indian Exempt Employment Income EXIND
  - Other Employment Income OEI
  - Self-Employment Income
Net Business Income BNET
Net Professional Income PFNET
Net Commission Income CMNET
Net Farming Income FMNET
Net Fishing Income FSNET

- Other Types of Income
  - Limited Partnership Income LTPI
  - Dividends XDIV
  - Interest and Other Investment Income INVI
  - Rental Income, Net RNET
  - Alimony or Separation Allowances ALMI
  - Other Income OI
  - Pension and Superannuation Income SOP4A
  - RRSP Income of individuals aged 65 and over RRSPO

- Transfers or Credits
  - Old Age Security Pension OASP
  - Canada/Quebec Pension Plan CQPP
  - Net Federal Supplements NFSL
  - Employment Insurance EINS
  - Goods and Services Tax Credit GHSTC
  - Provincial Refundable Tax Credits PTXC
  - Social Assistance SASPY
  - Workers’ Compensation WKCPY
  - Child Tax Benefits CTBI
  - Family Benefits FABEN

This is a gross income measure, since it includes earnings, capital and transfer income, but ignores taxes. A measure of disposable income is available as well (AFTAX), which is XTIRC excluding provincial and federal taxes and including the Quebec Abatement. The Quebec Abatement reduces the federal income tax payable by Quebec residents [220].
Market income

The LAD also contains a derived variable called “Market Income” (MKINC) that is defined as total income excluding government transfer payments, including Workers’ Compensation, Child Tax Benefit, Employment Insurance, CPP/QPP, etc. Market income consists of the following variables:

- Alimony or Support Income (ALMI_)
- Dividends (XDIV_)
- Earnings from T4 Slips, Total (T4E_)
- Interest and Investment Income (INVI_)
- Limited Partnership Income, Net (LTPI_)
- Other Employment Income (OEI_)
- Other Income (OI_)
- Other Pension and Superannuation Income (SOP4A)
- Registered Retirement Savings Plan Income (RRSPO)
- Rental Income, Net (RNET_)
- Self-employment, Net Income (SEI_)
- Indian Exempt Employment Income (EXIND)

Market Income plus income from government transfer payments will equal what has been defined as Total Income by SAADD (XTIRC) \[220\]. The exclusion of EI and CPP/QPP as transfers is controversial, since recipients pay premiums in order to receive the insurance and pension amounts.

Beach and Finnie \[18\] used a definition of “total employment income” or “earnings,” which they define as all wage and salary income and net self-employment income. They restrict analysis using this variable to earners aged 20-64 who were not full-time students and who received at least $1000 in earnings (1994 dollars) in order to approximate Statistics Canada’s concept of “all earners.”
Summary of measurement decisions on income

In Chapter 5 income is defined as household disposable income, using the AFTAX measure constructed by LAD staff. The use of disposable income reflects an interest in the role of consumption decisions on health as an important pathway mediating the effects of income on health and longevity. In Chapter 4, income is defined as total income, using the XTIRC measure constructed by LAD staff. The use of total income in the second analysis maximizes comparability with work of McDonough et al. [156] using data from the Panel Study of Income Dynamics in the United States.

2.2.3 Work disability

The definition and measurement of work disability were discussed in Section 1.7.1. The LAD does not have direct objective or self-reported measures of health. The LAD does have information about the receipt of disability income support and disability-related income tax measures. These are summarised in Table 2.1 and are described elsewhere [45, 46, 47, 21, 68, 134, 22, 232, 11, 48, 12, 103, 221, 169].

For the purposes of establishing that a person is disabled with respect to work, and likely to have reduced market income as a consequence of their disability, the programs must be analyzed in terms of their eligible population, their definition of disability, the approval time (delay between application to program and change in tax status), and the potential for the income, deduction or credit to appear on someone else’s tax return (typically the spouse). These programs have evolved incrementally, and some changes will affect the sensitivity or specificity of the measurement of work disability during the observation period of this study.

During the 1980s and early 1990s the prevalence of CPPD was rising quickly, as were program costs, while incidence was rising only slowly [103]. Prevalence, and costs, were reigned in by reducing eligibility in 1995 [44], resulting in lower incidence rates. Longfield
et al. have described CCPD changes during the 1990s [134]. Initial CCPD application volume rose between 1990 and 1995, then slowly declined through 2001, reaching lower levels than in 1990. The initial approval rate was relatively constant between 1990 and 1994, then dropped sharply through 1997 before rising slowly through 2003. There are three levels of appeal to CPPD decisions, and these have shown dynamic trends and remarkably high approval rates. The total number of beneficiaries increased between 1990 and 1995, then declined very slowly through 2003. The proportion of CPPD beneficiaries who are female has risen markedly since 1990, with the appearance of asymptotically approaching 50%. Note that the LAD only captures CPPD separately from total CPP starting in 1992.

While these trends were surely influenced by demographic and labour market changes, regulatory and administrative changes likely contributed as well.³ In 1995, CPPD hired additional medical advisors to reduce approval time and introduced new medical adjudication guidelines that reduced eligibility, especially for the poor, by ruling out consideration of socio-economic factors. Reforms in 1998 included a change in the number of years of contribution required for eligibility to four out of the last six years from two out of the last three or five out of the last ten. In 2001 the Federal Court of Canada decided in favour of a more generous interpretation of the definition of severe disability in the CPP legislation [134]. It is hard to predict the bias that these changes over time in CPPD incidence and prevalence might have on analyses of income drops and mortality.

The Employment Insurance (EI, formerly Unemployment Insurance) program provides sickness, maternity and parental benefits to workers who have (1) a 40% or greater decrease in regular weekly earnings, and (2) accumulated 600 insured hours in the previous 52 weeks or since the previous claim. Sickness benefits are paid for up to 15 weeks to a person who is unable to work because of sickness, injury or quarantine and is other-

³A similar set of explanatory factors is listed by Duggan and Imberman in their analysis of trends in Social Security Disability Insurance in the United States [69].
wise available for work. A medical certificate confirming the duration of the incapacity is required. Disability benefits through EI are not captured separately from other EI income, since both are entered on line 119 of the T1 personal income tax form, and were therefore not used as an indicator of work disability. EI sickness benefits will also not always represent a long-term disability. However, individuals eligible for CPPD are encouraged to apply simultaneously to EI for sickness benefits since EI sickness benefits are usually received within 28 days of application while CPPD involves a longer approval process.

**Summary of measurement decisions on work disability**

Disability benefit income received by any family member is used to exclude individuals since disability income may be transferred within households. If any income is received from CPP/QPP Disability (DSBCQF; 1992+), Worker’s Compensation (WKCPYF; 1992+), or disability deduction amount (DISDNF; 1983+) then the person is excluded from the analytic sample for being potentially disabled. Disability income during the exposure year, the prior five years, and the subsequent two years are considered. The subsequent years are used to allow for delays in approval and receipt.

Social assistance (SASPYF; 1992+) is not used because it is too difficult to establish a threshold above which disability income is likely. Medical expense deductions (MDEXCF; 1984+) are not used because this deduction is claimed very frequently and therefore has poor specificity as a measure of work disability.
**TABLE 2.1:** Disability income and disability tax measures in Canada, their properties as they relate to the proxy measurement of work disability, and their capture in the LAD.

<table>
<thead>
<tr>
<th>Category</th>
<th>Program</th>
<th>Funding Coverage</th>
<th>Beneficiaries (thousands)</th>
<th>Approval time</th>
<th>Definition of disability</th>
<th>Taxable income</th>
<th>Transferability</th>
<th>T1 fields (2004)</th>
<th>LAD capture</th>
<th>Comments</th>
<th>Mysteries</th>
</tr>
</thead>
</table>
...how do these appear in LAD? |
| Workers’ Compensation (WC) | Provincial | 67-97% workers, depending on province, nationally (excluding YT) 80% | 341 (2004), 380 (1996) | 341 (2004), 380 (1996) | Provin- cial averages for time between injury and first pay- ment | Long-term disability amount permanent impairment arising from work affecting work capacity. | No (pre- dated income tax, but included in net income) | Included at line 144 and deducted at line 250. Reported on T5007 form. | If WC and salaries/wages exceed pre-injury earnings, then the excess is treated differently. WC income is not treated differently for tax purposes whether it comes from WC directly, or is a load or advance from the employer to be repaid from future payments of WC. |

- Can represent lump sum payments for previous years; lump sum payments can also trigger retroactive filing; if total CPP greater than $6000 and age less than 60 then disability income is likely. At 60 CPP early retirement can be claimed. At age 65 disability amounts are converted to a retirement pension. It is known that many people receive CPPD but do not claim the DTC. CPPD is not lost if a person marries. After separation or divorce, CPP contributions made during marriage can be divided equally. The date of onset for payment purposes cannot be greater than 15 months prior to receipt of the application. Payments begin 4 months after the date of onset. CPPD is managed by the Income Security Programs (ISP) Branch of HRSDC. Since 1991, CPPD retroactive payments can be excluded from the year of receipt, and tax paid on them as though they had been received in the year to which they relate.

- Long-term disability amount: permanent impairment arising from work affecting work capacity. No (pre-dated income tax, but included in net income) | Included at line 144 and deducted at line 250. Reported on T5007 form. | If WC and salaries/wages exceed pre-injury earnings, then the excess is treated differently. WC income is not treated differently for tax purposes whether it comes from WC directly, or is a load or advance from the employer to be repaid from future payments of WC. |

- Regular and lump sum retroactive payments are included at line 114 (taxable, not deducted); disability payments included redundantly at line 152. Supported by T4A(P) slip (box 16). Total $1982 to present; Disability $1992 to present (LAD dictionary, page 103 says 1991-)

- Can represent lump sum payments for previous years; lump sum payments can also trigger retroactive filing; if total CPP greater than $6000 and age less than 60 then disability income is likely. At 60 CPP early retirement can be claimed. At age 65 disability amounts are converted to a retirement pension. It is known that many people receive CPPD but do not claim the DTC. CPPD is not lost if a person marries. After separation or divorce, CPP contributions made during marriage can be divided equally. The date of onset for payment purposes cannot be greater than 15 months prior to receipt of the application. Payments begin 4 months after the date of onset. CPPD is managed by the Income Security Programs (ISP) Branch of HRSDC. Since 1991, CPPD retroactive payments can be excluded from the year of receipt, and tax paid on them as though they had been received in the year to which they relate.
### Social Assistance (SA) Disability Income

**Provincial**

- 100% of population
- 350 (1996)

**Stringent definitions,** with no expectation of labour market re-entry. Not necessarily arising from work. No (considered payment of last resort), but included in net income

- Must be claimed by spouse with higher net income to maximize clawback.

- Included at line 145 and deducted at line 250.

**Total $ 1992 to present, not disability $.**

Disability likely if single and total is greater than $6000 or with dependents total is greater than $11,000. SA qualification is income and asset-tested.

Ontario replaced the Family Benefits Act with the Ontario Disability Support Program Act in June 1998. 75% or more of SA-D claimants are single (marriage or dependency can cause disqualification). Half of claimants have psychiatric disorders or developmental disabilities. Some provinces (not Ontario) have intermediate categories of disability within SA with lower levels of benefits.

### Long-term Disability (LTD) Insurance

**Private/Employer**

- ~50% of workers nationally
- 200 (2000)

**Moderate definitions,** with very high expectation of labour market re-entry. Not necessarily arising from work. Only if premiums paid by employer (which are tax deductible), and lump sum retroactive payments unclear in law. No

- Supported by T4A slip, box 28: "Other Income", footnote code 25: "Disability benefits paid out of a superannuation or pension plan" or code 07 "wage loss replacement plan". Not captured.

- LTD payments are taxable when the employee paid the premium. If the employer paid the premium, the employee is not taxed. Lump sum retroactive payments can also be taxable, but case law unclear.

### Automobile (AI) Insurance

**Disability income**

- Unknown
- No

**Included at Total $ 1992 to present, not disability.**

**Excluded**

- Not captured.

The taxability of motor vehicle insurance injury compensation income is very poorly documented in Canada.
### Chapter 2. The LAD

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2009

<table>
<thead>
<tr>
<th>Regular and Reserve Canadian Forces (CF) Veterans, Merchant Navy, RCMP civilians serving in close support; their spouses, common-law partners, children.</th>
<th>Average age 32 months (1984-5), 11 months (1986-7), approximately 5 months (2000).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veteran’s Disability Income (VAC disability pension) National</td>
<td>Compensation proportional to degree to which service caused disability (judged in fifths). CF claimant population currently increasing rapidly (2001-6).</td>
</tr>
</tbody>
</table>

Permanent disability resulting from injury or disease related to CF service. The Pension Act, s. 21, refers to "injury or disease or aggravation thereof resulting in disability". And s. 3 defines a "disability" as "the loss or lessening of the power to will and to do any normal mental or physical act". The term "aggravation" is not legislatively defined; however, it is accepted that aggravation means the permanent, as opposed to temporary, worsening of an existing disability. Diagnosis by a doctor is required.

<table>
<thead>
<tr>
<th>No</th>
<th>No</th>
<th>Excluded</th>
<th>Not captured.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal injury. General damages for pain and suffering, loss of amenities of life, loss of earning capacity, shortened expectation of life, or loss of financial support caused by the death of the supporting individual; special damages for out-of-pocket medical or hospital expenses, or accrued or future loss of earnings.</td>
<td>General and special damages from personal injury awards are generally not taxable. When damages are held on deposit, the interest becomes taxable income. No</td>
<td>Excluded</td>
<td>Not captured.</td>
</tr>
</tbody>
</table>

This includes class action settlements with the Government of Canada, as for Hepatitis C and HIV infections. Criminal injuries compensation benefits are treated similarly, though different laws apply.

#### Tort damages for personal injury

<table>
<thead>
<tr>
<th>NA</th>
<th>100% population</th>
<th>Unknown</th>
<th>Unknown</th>
</tr>
</thead>
</table>

Severely physically or mentally impaired in the tax year, and the impairment noticeably restricted the taxfiler’s activities of daily living (walking; feeding or dressing oneself; perceiving, thinking and remembering; speaking; hearing; and eliminating bodily waste), or would restrict them if not for extensive therapy, as determined by a physician. Ability to work is not considered. NA

Transferable in whole/part to supporting close relative T2201 form (Disability Tax Credit Certificate), 1983 to present (self). Requires a T2201 form (Disability Tax Credit Certificate). Two people can claim part of the same DTC. Non-refundable. Based on taxable, not net, income, therefore SA and WC do not disqualify taxfilers.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Income tax measure</td>
<td>Disability Tax Credit National Taxfilers</td>
<td>0 + time to complete T2201 form to supporting close relative NA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Two people can claim part of the same DTC. Non-refundable. Based on taxable, not net, income, therefore SA and WC do not disqualify taxfilers.
<table>
<thead>
<tr>
<th>Income tax measure</th>
<th>Medical Expenses</th>
<th>National Taxpayers</th>
<th>2003</th>
<th>0 (adaptable)</th>
<th>Non-reimbursed disability-related medical expenses and expenses reimbursed as income. Allowable expenses have been increasingly disability-related over time.</th>
<th>Transferable to spouse</th>
<th>Line 330</th>
<th>1984 to present</th>
<th>Can be expenses from any 12 month period ending during the year; health insurance premiums are eligible, including travel health insurance.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Income tax measure</td>
<td>Medical Expense Tax Credit (METC)</td>
<td>National Taxpayers</td>
<td>2827</td>
<td>0 (adaptable)</td>
<td>Provides assistance for above-average disability and medical expenses to low-income working Canadians (earning &gt; $2500; to replace lost SA benefits on labour market re-entry).</td>
<td>NA</td>
<td>As METC</td>
<td>Line 452</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Income tax measure</td>
<td>Medical Expense Supplement</td>
<td>National Taxpayers</td>
<td>165 (1997)</td>
<td>0 (adaptable)</td>
<td>Provides assistance for above-average disability and medical expenses to low-income working Canadians (earning &gt; $2500; to replace lost SA benefits on labour market re-entry).</td>
<td>NA</td>
<td>As METC</td>
<td>Line 452</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Income tax measure</td>
<td>Attendant Care Deduction (changed to Disability Supports Deduction in 2004)</td>
<td>National Taxpayers</td>
<td>0 (adaptable)</td>
<td>Requires a T929 form.</td>
<td>Disability requiring services of an attendant. (DSD: persons who need supports to enable them to attend work or school, whether or not they qualify for the DTC.)</td>
<td>NA</td>
<td>Requires a T929 form.</td>
<td>Eligibility</td>
<td>Could be deducted from non-earned income if a student.</td>
</tr>
</tbody>
</table>

**Refundable Medical Expense Supplement (1997, 2001)**

- National Taxpayers: 165 (1997), 315 (2001)
- Adaptable: 0
- Provides assistance for above-average disability and medical expenses to low-income working Canadians (earning > $2500; to replace lost SA benefits on labour market re-entry).
- Introduced in 1997. Requires a claim for either the METC or the DSD.

**Attendant Care Deduction (changed to Disability Supports Deduction in 2004)**

- National Taxpayers: 0 (adaptable)
- Requires a T929 form.
- Disability requiring services of an attendant. (DSD: persons who need supports to enable them to attend work or school, whether or not they qualify for the DTC.)
- Not captured -why not? No consequence since DTC needed for eligibility.
- The same expenses cannot be claimed for both METC and DSD. Attendant could not be spouse or common-law partner, minimum 18 years of age. ACD required DTC eligibility, while DSD requires only medically certified eligible expenses. Could be deducted from non-earned income if a student.
### Table 2.3: Family-type transition matrix for individuals aged 16+ in the Survey of Labour and Income Dynamics between the ends of 1992 and 1993 [84].

<table>
<thead>
<tr>
<th>Family type in 1993</th>
<th>End of 1992</th>
<th>End of 1993</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unattached individuals</td>
<td>13.4</td>
<td>0.8</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
<td>0.4</td>
<td>0.1</td>
<td>0.2</td>
<td>15.1</td>
<td></td>
</tr>
<tr>
<td>Couple with no children</td>
<td>0.7</td>
<td>20.6</td>
<td>0.9</td>
<td>0.0</td>
<td>0.0</td>
<td>0.6</td>
<td>0.2</td>
<td>0.3</td>
<td>23.3</td>
<td></td>
</tr>
<tr>
<td>Couple with children</td>
<td>0.8</td>
<td>0.6</td>
<td>35.7</td>
<td>0.4</td>
<td>0.2</td>
<td>1.9</td>
<td>1.4</td>
<td>0.1</td>
<td>41.1</td>
<td></td>
</tr>
<tr>
<td>Female lone-parent</td>
<td>0.2</td>
<td>0.0</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
<td>0.5</td>
<td>0.2</td>
<td>0.0</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>Male lone-parent</td>
<td>0.1</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.7</td>
<td>0.1</td>
<td>0.0</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Other economic family</td>
<td>0.7</td>
<td>0.9</td>
<td>0.6</td>
<td>0.1</td>
<td>0.0</td>
<td>11.5</td>
<td>0.4</td>
<td>0.2</td>
<td>14.4</td>
<td></td>
</tr>
<tr>
<td>Don’t know</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.1</td>
<td>0.0</td>
<td>0.2</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>15.9</td>
<td>23.1</td>
<td>37.6</td>
<td>4.3</td>
<td>1.0</td>
<td>15.0</td>
<td>2.5</td>
<td>0.8</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2.4: Family-type transitions and mean family income for individuals aged 16+ in the Survey of Labour and Income Dynamics between the ends of 1992 and 1993 [84].

<table>
<thead>
<tr>
<th>Family type in 1993</th>
<th>Same family type</th>
<th>No less than Same family type</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>%</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unattached individual</td>
<td>23,498</td>
<td>24,463</td>
<td>21,300</td>
<td>-12.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Couple with no children</td>
<td>50,143</td>
<td>50,254</td>
<td>49,629</td>
<td>-1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Couple with children</td>
<td>64,930</td>
<td>65,928</td>
<td>60,406</td>
<td>-8.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female lone-parent</td>
<td>28,688</td>
<td>30,547</td>
<td>24,215</td>
<td>-20.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male lone-parent</td>
<td>54,573</td>
<td>62,402</td>
<td>45,888</td>
<td>-26.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other economic family</td>
<td>64,034</td>
<td>63,467</td>
<td>65,484</td>
<td>3.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Don’t know</td>
<td>58,649</td>
<td>62,511</td>
<td>54,545</td>
<td>-12.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>52,890</td>
<td>54,200</td>
<td>48,514</td>
<td>-10.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### 2.2.4 Family breakdown & family deaths

The definition and measurement of family breakdown and family deaths were discussed in Sections 1.7.3 and 1.7.4. Changing family structure and size has profound effects on equivalized household income. Much of the change in family and household make-up is of irreducible complexity, and there are no widely-used typologies of non-longitudinal family or household structure. Giles cites Duncan and Hill (1985) as arguing that one should avoid analyzing families, and instead analyze individuals, ascribing to them the characteristics of their families as necessary [84].

Giles described family-type transition probabilities in Canada using the Survey of Labour and Income Dynamics (SLID) [84] (see Table 2.3). Between the ends of 1992 and 1993, 85.8% of persons aged 16 or older lived in the same family-type. Couples with and without children accounted for 56.4%, with individuals with and without children accounting for a further 17.8% and “other” accounting for 11.5%.

The stability of families is not randomly distributed. Families that change type are poorer by amounts that depend on family type (see Table 2.4). The mean family income of
couples without children is not different on average between those that entered this family type from another. Among couples with children, those entering this state from another have lower income than those who were already in this group, perhaps reflecting labour force withdrawal upon the birth of first children. New single-parents are much poorer on average than “established” single-parents, and the same is true to a lesser degree for unattached individuals.

Changing numbers of earners and dependents make longitudinal analyses of the economic conditions of families and households difficult, and the usual approach is to focus on the most frequent types of longitudinal households and to stratify the analyses by them. In an analysis of labour market-derived income dynamics and mortality, it is also desirable to exclude or control for factors or events that might determine both income and health, and so confound the observed relationship between them.

Summary of measurement decisions on family structure

Single persons and couple families are treated separately, while single-parent families are excluded since there is not sufficient statistical power to treat them separately. If family type changes during the exposure year or any of the previous five years, then thefiler is excluded. Family size is taken as the maximum reported value during this period to prevent annual variation in the reporting of dependants from affecting equivalized income. Any deaths during this period to family members of the filer are also grounds for exclusion.

2.2.5 Retirement

The definition and measurement of retirement were discussed in Section 1.7.5. For the analysis of income dynamics and premature death using tax data, as in Chapter 5, retirees are excluded using only age and receipt of retirement income as indicators.

Restricting exposure years to ages 40-55 eliminates most retirees. However, using the
LFS definition of retirement, which involves self-report of retirement given that the individual is currently out of the labour force but participated in the last 12 months, between the late 70s and early 90s the fraction of retirements at ages 50 or greater that occur before age 60 increased from 16% to 34% [91], and some of these persons likely retire at ages less than 55.

Retirement income sources and their potential to identify retirees aged less than 60 are described in Table 2.5. Prior to age 60 individuals do not qualify for public pension income through OAS/GIS or CPP/QPP [102].

In Canada there is a high level of pre-retirement RRSP withdrawal [86, 243], and so the decision on whether to use RRSP withdrawal as an indicator of retirement involves a trade-off between sensitivity and specificity. This choice is made more difficult if pre-retirement RRSP withdrawal is also thought to be an indicator of families experiencing cash constraint, and this group is of analytic interest. However, since RRSP savings are small for most families (the median value in 1999 was $20,000 [153]), it is unlikely that very many can retire before age 60 on RRSP savings alone.

Some private pensions allow for retirement with full benefits based on years of service rather than age of retirement, and some allow early retirement at 55 or earlier with reduced benefits [102]. While RPPs are a useful indicator of retirement before age 60, the fraction of the labour force covered by such plans is low and fell during the 1990s, from 37% in 1991 to 33% in 1999 [152, p.7]. However, since RPPs are more common among older workers, 56% of those aged 55-64 had RPP assets in 1999 with a median value of $151,900.

RPP assets are more common among families with higher income, and with earners employed in government, education and health. Having private pension assets (RPP or RRSP) is strongly associated with education, occupation, income, family structure, province of residence and of course with age. Families without private pension assets
also have few other assets: 70% have no home equity [153].

The incomplete population coverage of RPP as an indicator of retirement may be less serious than it at first appears for two reasons. First, workers are not likely to retire before age 60 unless they are either disabled, which can be measured through other tax measures, or because it is financially possible, and it is unlikely that it is financially possible for those without RPP assets. Second, since many families without RPP assets tend to have low income and few assets in general, it is not clear what retirement would mean for this group before the age of 60. It may be that the construct validity is questionable when the act of retirement is economically undesirable and the individual is relatively young, and that such individuals might better be described simply as out of the labour force.

Since employee RPP contributions are tax deductible (reported on line 207 separately from other tax-deferred retirement amounts), it is possible to identify those with RPP savings that include employee contributions using tax data. RPP contributions are captured in the LAD starting in 1986 [221].

Private pension savings other than RPPs and RRSPs account for only 1.3% of private pension assets in 1999 [153], and are therefore of limited use for determining retirement events in a population sample.

**Summary of measurement decisions on retirement**

Retirees are primarily excluded by restricting the age during the exposure year to ages 40 though 55. Early retirees are identified by the receipt of “other pension or superannuation income,” which captures private pension income. Personal tax-deferred retirement savings in the form of Registered Retirement Savings Plans (RRSP) are not used to indicate retirement for two reasons. First, there is a high level of pre-retirement RRSP withdrawal in Canada. Second, most RRSP savings are too small to support retirement alone, and so these withdrawals likely indicate cash constraint rather than retirement if they occur
without private pension income.
**TABLE 2.5**: Retirement income sources in Canada and their potential to identify retirees aged less than 60 [102, 78].

<table>
<thead>
<tr>
<th>Retirement income source</th>
<th>Minimum qualifying age</th>
<th>Eligible population</th>
<th>Useful indicator of retirement before age 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Old Age Security and Guaranteed Income Supplement (OAS/GIS)</td>
<td>65, means tested at 60</td>
<td>Those with 10 years of Canadian residency after 18th birthday</td>
<td>No: ineligible</td>
</tr>
<tr>
<td>Canada Pension Plan / Quebec Pension Plan (CPP/QPP)</td>
<td>60</td>
<td>Workers in Canada aged 18+ are required to pay into CPP/QPP</td>
<td>No: ineligible</td>
</tr>
<tr>
<td>Registered Pension Plans (RPP)</td>
<td>Various. 65 is a standard age for eligibility, but many plans also allow full benefits based on years of service. Many plans allow for reduced benefits up to 10 or more years before eligibility for full pension.</td>
<td>Employees with pension benefits (about 40% of workers in Canada), typically eligible for benefits after two years of paying into the plan.</td>
<td>Yes</td>
</tr>
<tr>
<td>Registered Retirement Savings Plans (RRSP)</td>
<td>RRSP investments can be withdrawn at any time, but are then taxed as income unless the withdrawal is through the Home Buyer’s Plan or the Lifelong Learning Plan, which require the withdrawals to be repaid in order to avoid having them taxed as income.</td>
<td>Tax payers who had taxable income in the previous year have the option of purchasing RRSP investments.</td>
<td>No: withdrawals may indicate cash constraint</td>
</tr>
<tr>
<td>Deferred Profit-Sharing Plans (DPSP)</td>
<td>As RRSP, but with no tax-free withdrawal programs. Savings must be distributed at termination of employment or of the plan.</td>
<td>Employees whose for-profit employers establish a DPSP.</td>
<td>No: withdrawals may indicate job loss or cash constraint</td>
</tr>
<tr>
<td>Employee savings plans</td>
<td>No restrictions.</td>
<td>Employees whose employers (typically publicly traded companies) match employees' contributions to a savings plan (typically company shares).</td>
<td>No: withdrawals may indicate cash constraint, investment decisions</td>
</tr>
<tr>
<td>Unregistered private savings and investments, including home equity</td>
<td>No restrictions.</td>
<td>No exclusions.</td>
<td>No: withdrawals may indicate cash constraint</td>
</tr>
</tbody>
</table>
Chapter 3
Aspects of biostatistics

Chapters 5 and 4 use Cox models to examine the association of income drops with subsequent mortality. Choices regarding the time scale and time origin are addressed in Sections 3.1.1 and 3.1.2. The induction period for the hypothesized effect of income drops on mortality is unknown. Methods for exploring induction periods are reviewed in Section 3.2.

3.1 Cox relative risk models

Cox [61] proposed a relative risk model for discrete and continuous survival time in which predictors had a multiplicative effect on an unspecified baseline hazard function. In many formulations it is referred to as a “proportional hazards model,” but this terminology is not correct in the presence of time-dependent covariates, and “relative risk model” or simply “Cox model” is preferred [110, p.43]. Other authors distinguish between the Cox proportional hazards model, which has only time-fixed, baseline covariates, and the time-dependent Cox model, which allows for time-dependent covariates [13].
3.1.1 Time aggregation bias

Petersen [181] has shown that time aggregation (e.g. measuring survival time in years instead of days or hours) leads to a time aggregation bias in continuous-time parametric hazard models. Approaches to dealing with tied survival times due to imprecise (i.e. aggregated) measurement in semi-parametric Cox proportional hazards models include the use of exact estimation (i.e. marginal likelihood [109, p.71-6] or averaged likelihood [228, p.48-53]), and the so-called Breslow and Efron approximations. The magnitude of the bias in the approximations depends on the number of ties as a proportion of the number at risk [76][109, p.75]. When there are many ties, the Breslow approximation performs most poorly, the Efron approximation shows considerable bias, and the exact method is preferred [5, p.129-33].

In the presence of many ties, Kalbfleisch and Prentice [109, p.75] say the exact method “still gives reasonably good estimates”, but at that time rigorous numerical investigation was not possible for lack of sufficient computing power. They referred to 7 or 8 ties at one time point as beginning “to cause difficulties” [108]. A slightly larger numerical study showed that the exact estimate appeared to be extremely close or perhaps identical to the true value when the number of ties ranged from the 30s to the high 40s [65]. However, this does not answer the question of whether there is a loss in precision when using aggregated data. Therneau and Grambsch [228, p.48-53] show an example using an original and coarsened dataset, which showed that the exact method did not give the same result for parameter estimates on the original and coarsened dataset, and that the bias was not always conservative. However, theirs was an example, not a simulation, involved a small dataset, and did not discuss bias in the estimates of precision.

1 A small simulation study was conducted to evaluate the accuracy and precision of the

---

1 The use of the term exact here follows the SAS program. S-plus, on the other hand, implements what SAS calls discrete under the name exact, and does not implement what SAS calls exact.
exact marginal likelihood in a Cox proportional hazards model\(^2\) on aggregated survival data. Exponential survival data with four correlated dummy predictors with effects of varying sign and magnitude, 8 years (3000 days) of survival time, 50% administrative right censoring and 10% loss-to-follow-up right censoring were generated. The simulation used 500 repetitions each with a sample size of 10,000. Exact estimation was performed using days and then years as the unit of survival time measurement to examine the impact of aggregation of the outcome.

The simulation results were analyzed using a paired t-test of the differences between models using aggregated and unaggregated survival data in their parameter estimates and standard errors. There was a statistically significant but trivial bias towards the null of 0.4% to 0.5% for both positive and negative coefficients, and a statistically significant but trivial conservative bias in the standard errors of 0.7% to 0.8%.

**Summary of modelling decisions on time-scale**

There is no bias in estimates of effect or in their estimated variance, and so year of death is sufficient.

**3.1.2 Choice of time origin**

Typical analyses of survival time data use study enrolment as the time origin, and days as the time scale. The choice of time origin should be determined by the analytic objectives and the nature of the data, while the time scale is usually arbitrary (see Section 3.1.1 for a discussion of time scale in terms of measurement error). Much of the survival analysis literature inaccurately refers to the choice of time origin as the choice of time scale [116].

Study enrolment is an appropriate time origin for clinical studies following patients after an intervention that alters their event hazard. But for epidemiological studies in which age-

\(^2\)SAS implements this using the algorithm proposed by Delong, Guirguis and So [65].
at-entry and time-on-study are not part of the mechanism in question, other time origins, in particular date of birth, are more appropriate [39, 121, 229].

Alternative time origins usually require delayed entry\footnote{Therneau and Grambsch [228, p.75] refer to delayed entry into the risk set as “left truncation,” as does the SAS Institute [200, p.3263]. Kalbfleisch and Prentice [110, p.12-4,23-4] use “delayed entry” and “left truncation” interchangeably, and Allison [5, p.161-5] uses the terms “late entry” and “left truncation” as equivalent. Harrell [97, p.391-2] admits that this is common usage, but prefers to distinguish between (1) delayed entry into the risk set, as would occur when using date of birth as the time origin, and (2) left-truncation, meaning that an unknown subset failed before entering the study. The similar term left-censoring is entirely different, and means that an event is known to have occurred only prior to a certain time.} into the risk set, and this can be implemented using the counting process form of the Cox model proposed by Andersen and Gill [8] and described by Therneau and Grambsch [228, p.68-77]. The SAS system has two procedures, PROC TPHREG and PROC PHREG that implement counting process syntax as an option [200, p.3241-2], though model estimates become prohibitively memory intensive with large datasets unless the MULTIPASS option is specified, which causes PHREG to recompile the risk set with each Newton-Raphson iteration rather than computing all risk sets at once. These two SAS procedures also allow the specification of an ENTRY time as an optional argument to the MODEL statement with traditional dependent-variable syntax as an alternative way of implementing delayed entry to the risk set [200, p.3229], but this gives identical results to counting process notation and uses identical computational resources in SAS version 9.1 for Windows.

In an epidemiologic cohort study sampled at exposure or a fixed time afterwards, using study enrolment as the time origin is not advised since it is highly correlated with the duration of exposure, which is often of scientific interest [37]. For most observational studies, Korn et al. [121] recommend using date of birth instead of study enrolment as the time origin and stratifying on birth cohort using the model

$$\lambda_A(a | b_0 \epsilon B_j, z) = \lambda_{0,j} A(a) e^{\beta' z} \quad (3.1)$$

where $A = a$ is the age of the individual during the follow-up period, $\lambda_A$ is the instantan-
neous rate of a death occurring at age $a$, $b_0$ is the individual's birth cohort, $B_j$ are $j$ birth cohort intervals, $\lambda_{0jA}$ is an unspecified baseline hazard for each birth cohort, and $z$ is a vector of baseline risk factors with coefficients $\beta$.

Since the baseline hazard does not need to be specified, it is very desirable to express the baseline hazard as a function of age when age is among the strongest determinants of the hazard and needs to be controlled for, as in an analysis of all-cause mortality. Stratifying by birth cohort has the advantage over adjusting for birth cohort as an explanatory variable that the hazard function need not have a multiplicative relationship among birth cohorts, as might occur due to advances in medical care [121]. The primary disadvantage is that birth cohort effects and interactions with other explanatory variables cannot be explored.

The more traditional formulation of the Cox proportional hazard model with study enrolment as the time origin and adjustment for age-at-baseline as a covariate or by stratification leads to two sources of bias. First, despite adjusting for age-at-baseline, residual confounding is likely, and because age is such a powerful determinant of most health outcomes, and correlated with many exposures, the bias can be appreciable. If the baseline hazard with age is exponential, then there is no association between age and the health outcome, and no confounding bias [121, 229]. Second, omitted variable bias in Cox proportional hazards models is known to bias estimates towards the null. Omitted variable bias is more serious for somewhat than for highly censored data, and the magnitude of the bias depends on the magnitude of the coefficient of the omitted variable. Even if age is included in the model, as with residual confounding, mis-modelling by choosing an imperfect functional form for age can result in downward bias and loss of statistical power [229].

When the age at which an event occurs is itself a predictor of health outcomes, as for age-of-diagnosis of breast cancer and subsequent mortality, the choice of study en-
rolment or date of birth as the time origin can determine even the sign of the observed relationship [56].

In addition to adjusting for the effects of age by using date of birth as the time origin, stratifying by birth cohort allows each cohort to have an independent baseline hazard on age, such that medical advances and other cohort and period effects are accounted for in a very flexible way [121, 229]. Failure to account for these can lead to the same biases as for mis-modelling of age effects.

Summary of modelling decisions on time-origin

In Chapter 5 the time-origin is date of birth. Despite the theoretical reasons for this decision, when using all-cause mortality as the event of interest, time-on-study as the outcome, and with sampling independent of exposure, there does not appear to be any significant difference between models since age has a near-perfect linear association with all-cause mortality on the log-hazard scale. Therefore, the models in Chapter 4, which were specified earlier in this manner, were not re-specified to match Chapter 5 since there did not appear to be a need to do so.

3.2 Modelling induction periods

An induction period is the length of time, or lag, between an exposure and the health consequences that it causes. The minimum induction period is the shortest time that must elapse after exposure before the health consequence can occur, and the maximum induction period is the longest time that might elapse. This meaning of induction period is synonymous with the term “latent period.” Rothman [196] subdivided the latent period into an induction period, from first exposure until initiation of the disease process, and incubation period, from initiation to clinical manifestations. This subdivision is not useful
for many disease processes and is almost always unobservable [230], and so latency and induction periods will be treated here as synonymous terms. The induction period can be thought of as a random variable at the population level, with individual level induction periods being observed instances from the population distribution, such that maximum and minimum periods are arbitrary cut-points chosen for analytic convenience.

For an acute exposure, it can be practical to look forward from exposure and describe a “window” in which health consequences might be due to the exposure, and outside of which they cannot be due to the exposure. This is the approach described by Rothman [196]. For a rate, this means that person-time only contributes to the denominator if it took place within the window. If the minimum and maximum induction times are not known, then several windows can be constructed, and Rothman recommended interpreting the window with the largest effect size as the “average” induction period.

For a chronic exposure, it is more practical to look backwards from the health event and describe a window in which exposure might have been the cause of the event. Often only a minimum induction period is specified, and a lifetime cumulative index of exposure is “lagged,” such that only cumulative exposure preceding the health event by the lag period (i.e. the minimum induction period) is used to estimate the dose-response association [50]. As with Rothman’s method, typically the largest effect size is taken to indicate the mode of the distribution of risk over time.

Salvan et al. [199] argued that using effect size rather than model fit to determine the average induction period can lead to significant bias. Greenland [93] rejected model fit as a simple criterion as well, and stated that using model fit is merely significance testing, and will therefore exaggerate the statistical significance of the estimate for the chosen induction period. He recommended that the estimates for the various simultaneously-estimated windows be examined for a regular pattern.

There are two problems with the simultaneously-estimated windows approach [93].
The first is lack of statistical power to estimate the multiple effects, which necessitates a compromise between window width and statistical power. The second is that early exposures may have some of their effects on health via subsequent exposures. Adjustment for subsequent exposures will therefore reduce earlier exposures to measures of direct effects only [190], and might also cause more bias than unadjusted estimates [193]. Models capable of overcoming the latter limitation have been suggested by Thomas [230] and Robins [191] and are discussed in Appendix Section A.2. In spite of these problems and the available solutions, Greenland still recommends the simultaneously-estimated windows approach as a method to get a rough estimate of the shape of the induction-time distribution.

**Summary of modelling decisions on induction periods**

Models that could accommodate variable induction periods for a chronic exposure and a mortality outcome were not identified. Instead, a simple approach treating income level and drops as acute exposures was implemented. For each possible lag, all eligible exposure and vital status data were used to estimate the hazard, with risk sets defined in light of the live-follow-up data. This gives estimated hazards as a function of lag, but the estimates at each lag are not independent, since they are largely based on the same exposure data and vital status data.
Part II

Thesis papers
Chapter 4

Income dynamics and adult mortality: associations in Canada and the United States
Abstract

Introduction This paper examines the association of income level and relative income drops on the hazard of death in Canada and the United States. The magnitudes of the effect of income level on hazard of death has not been directly compared, but are expected to be weaker in Canada given universal access to publicly financed health insurance. There is limited evidence in the United States that relative income drops increase the hazard of death, but perhaps primarily for those with middle-income levels.

Methods The data are from the Longitudinal Administrative Databank (LAD) and the Panel Study of Income Dynamics (PSID). The LAD is a 20% longitudinal sample of Canadian tax filers for the years 1982 through 2005, while the PSID is a closed survey cohort sampled in 1968 and followed annually through 1997. Cox hazard models were specified so as to closely replicate previously published findings using data from the PSID, and control for age, gender, family size and, in the United States, race. The sample is restricted to persons aged 45-64 at baseline.

Results Income level showed the expected negative, graded association with hazard of death in both countries. Relative to those with persistently high income, those with persistently low income had hazard ratios for all-cause mortality of 2.13 (95% confidence interval: 2.02, 2.24) in Canada and 4.39 (2.67, 7.20) in the United States. Income drops are associated with increased hazard of death in both countries at most income levels, but the patterns by income level are not consistent between the two countries.

Discussion The effect of income level on hazard of death is large in Canada but twice as large again in the United States. This is consistent with known barriers in access to effective medical care in the United States, but could also be due to greater inequalities in health behaviours, or access to or quality of non-medical public and private goods and
services that benefit health. Analyses of the PSID largely confirmed previously published findings that used alternative statistical methods, but the effect of income drops by income level were not identical in Canada and the United States. Whether this is due to differences in mechanism or to differences in data and analysis is discussed.
4.1 Introduction

This paper examines the association of income level and relative income drops on the hazard of death in Canada and the United States. Income level has been shown to have a strong, positive, and monotonic association with longevity in the United States [119, 70, 71, 194, 195] and in Canada [100, 248, 173, 246]. However, there has not been an attempt to determine whether the association is of equal magnitude in the two countries.

Relative drops in family income have been analyzed by McDonough et al. using data from the Panel Study of Income Dynamics (PSID) in the United States [156], and it was observed that income drops may be associated with mortality among adults aged 45-64 at baseline, but perhaps only among those with middle income levels. The analytic approach used by McDonough et al. was non-standard, and no subsequent research has been published confirming or refuting the findings using other analytic methods, data sources or populations. This paper reproduces the findings of McDonough et al. using routine survival analytic methods and the same PSID data, and compares these results to those obtained using identical analytic methods and personal income tax data from the Canadian Longitudinal Administrative Databank (LAD).

During the 1980s and early 1990s, Canadians experienced significantly longer durations of unemployment than did Americans, while Americans’ initially higher incidence of unemployment decreased to the more constant Canadian level. In combination, this led to a higher unemployment rate in Canada than in the United States. The increased duration of Canadian unemployment spells did not appear to be due to characteristics of the unemployed. Incident periods of unemployment in Canada, however, were more likely to be due to layoff, and less likely to be due to labour market entry than in the United States [14]. While long-term unemployment, the fraction of the unemployed that have been so for 12 months or longer, has typically also been higher in Canada than in the US [176], one would also need to know flows into other states including non-participation and involun-
tary part-time work to judge the association this has with relative income drops. These differences may mean that Canadians have greater exposure to relative earned income drops, despite a more generous unemployment insurance system.

In the United States employment and income play an important role in determining whether individuals have health insurance [79], whereas Canadians are universally insured for hospital and doctor care. Relative income drops may therefore indicate lack of access to needed medical care to a greater degree in the US than in Canada.

It is possible, therefore, that exposure to relative income drops is different between the United States and Canada, and that the effects of income drops on health and longevity are also different.

### 4.1.1 Objectives

1. Confirm McDonough et al.’s findings with survival analytic methods
2. Compare the magnitude of the income-mortality association between Canada and the United States
3. Compare the effect of income drops in Canada and the United States
4. Do income drops affect primarily the middle income group in both countries?

### 4.2 Methods

The US data are from the Panel Study of Income Dynamics, which is a closed survey cohort sampled in 1968 [99]. Poor families were oversampled. The sample was followed annually until 1997. Due to the period of the sampling, recent waves of American immigration, notably the Latino population, are poorly represented.
The Canadian data are from the Longitudinal Administrative Databank (LAD) [221]. The LAD is a 20% random sample of individuals in the T1 Family File (T1FF), which contains annual tax data for 1982 and onwards, the most recent available year being 2004. Once sampled, individuals are followed until death or loss to follow-up, and an annual top-up sample maintains representativeness. The T1FF is an annual census of all Canadians who file personal income tax (T1 form), or whose Social Insurance Number (SIN) is listed on another family member’s tax file. Both legal and common law spouses are identified by the Social Insurance Number (SIN) listed on T1 tax forms or by matching on name, address, age, sex and marital status. Children are identified from T1 data as well as supplementary files from the Child Tax Benefit and Family Allowance programs. The T1FF therefore consists of filers, who all have a reported SIN, and dependent children and spouses, some of whom have a reported SIN. Only T1FF individuals with a SIN can be sampled into the LAD.

The samples were restricted to persons aged 45-64 at baseline. The sex of taxfilers, which is not reported on the T1 form, is established by the Canada Revenue Agency by linking to the SIN master file, held by Human Resources and Skills Development Canada. Unmatched spouses are assigned a sex opposite to their spouse, and a small number of unmatched single individuals are assigned a sex randomly.

**Survival data**

Survival time was defined traditionally as time-on-study, and years until death or loss to follow-up was the outcome in Cox proportional hazards models. Although the day and month of death are reported on the T1 form, only year is captured in the LAD. Time aggregation bias is not a problem with these data when using the exact method for tied event times as implemented in SAS software (see Section 2.2.1). Subjects were right-censored when lost to follow-up, when they reached 66 years of age to exclude adults not...
of working age, and when structurally right-censored due to the end of data collection.

**Income level, income drops, and income persistence**

Income was defined as family total income. US incomes were adjusted using the Consumer Price Index (CPI-U) to 1993USD, and Canadian incomes were adjusted using the Canadian CPI, then converted to USD using the bi-national consumer expenditure purchasing power parity. Mean income level during the previous 5 years was divided into three levels with cut points at $20,000 and $70,000 1993USD. Income dynamics were dichotomized as one or more annual drops of 50% or more during the previous five years. Separate models were estimated for income persistence, defined as income that was low for 4-5 years and never high, low for 1-3 years and never high, high for 1-3 years and never low, high for 4-5 years and never low, and other. Constructs were defined so as to replicate McDonough et al.

**Analyses**

Cox models were used to estimate the effects of time-varying income level and income drops, and time-invariant age, sex, and in the US data race (black v. other). Models using PSID data are weighted using probability weights and variances are adjusted for the clustered sampling. The LAD data are a simple random sample and therefore models do not require weighting or variance adjustment.

**4.3 Results**

The LAD and PSID samples are described in Table 4.1. The ages are similar, but family size is larger in the PSID, perhaps due to the slightly earlier period of sampling. A greater proportion of PSID members died due to the longer period of follow-up. A greater propor-
Table 4.1: Sample characteristics in the Longitudinal Administrative Databank (LAD) and Panel Study of Income Dynamics (PSID).

The effects of income drops are described in Table 4.2 (see also Figure B.1). Income drops negatively affect survival, except for low-income Canadians. The effect size for the income drop effect depends on income level, but patterns differ in Canada and the United States.

The effects of income persistence are described in Table 4.3 (see also Figure B.1). Chronically low income confers a greater hazard of death than transiently low income in both Canada and the United States. Chronically high income confers a lesser hazard of death than transiently high income in both Canada and the United States. The effect of income on hazard of death is twice as great in the United States as it is in Canada.
TABLE 4.2: The effect of any 50% annual total family income drops during the previous 5 year period by mean income level during the same period in Canada and the United States. Low income is less than $20,000 1993USD, and high income is greater than $70,000 1993USD. Hazard ratios for all-cause mortality and 95% confidence limits from Cox survival models using Canadian personal income tax data (LAD) are compared to American survey data (PSID), and to the odds ratios published by McDonough et al. [156], also using the PSID data.

<table>
<thead>
<tr>
<th></th>
<th>Canada</th>
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<th>McDonough et al.</th>
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<tr>
<td></td>
<td>(LAD)</td>
<td>(PSID)</td>
<td>(PSID)</td>
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<tr>
<td>HR</td>
<td>LCL</td>
<td>UCL</td>
<td>OR</td>
</tr>
<tr>
<td>Low + drops</td>
<td>1.95 1.85</td>
<td>2.06</td>
<td>3.73 2.41 5.70</td>
</tr>
<tr>
<td>Low</td>
<td>2.31 2.19</td>
<td>2.43</td>
<td>3.35 2.22 5.06</td>
</tr>
<tr>
<td>Middle + drops</td>
<td>1.64 1.56</td>
<td>1.72</td>
<td>3.21 1.90 5.47</td>
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<tr>
<td>Middle</td>
<td>1.43 1.37</td>
<td>1.50</td>
<td>1.47 1.05 2.04</td>
</tr>
<tr>
<td>High + drops</td>
<td>1.21 1.11</td>
<td>1.32</td>
<td>1.40 0.67 2.55</td>
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<tr>
<td>High</td>
<td>1.00</td>
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TABLE 4.3: The effect of persistence of annual total family income level during the previous 5 year period in Canada and the United States. Low income is less than $20,000 1993USD, and high income is greater than $70,000 1993USD. Hazard ratios for all-cause mortality and 95% confidence limits from Cox survival models using Canadian personal income tax data (LAD) are compared to American survey data (PSID), and to the odds ratios published by McDonough et al. [156], also using the PSID data.

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</tr>
<tr>
<td></td>
<td>HR</td>
<td>LCL</td>
<td>UCL</td>
</tr>
<tr>
<td>Low 4-5y + never high</td>
<td>2.13 2.02</td>
<td>2.24</td>
<td>4.39 2.67 7.20</td>
</tr>
<tr>
<td>Low 1-3y + never high</td>
<td>1.84 1.75</td>
<td>1.93</td>
<td>3.24 1.99 5.30</td>
</tr>
<tr>
<td>Other</td>
<td>1.44 1.37</td>
<td>1.51</td>
<td>1.86 1.18 2.94</td>
</tr>
<tr>
<td>High 1-3y + never low</td>
<td>1.25 1.19</td>
<td>1.32</td>
<td>2.01 1.22 3.33</td>
</tr>
<tr>
<td>High 4-5y + never low</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Cause</td>
<td>Crude rate per 100k</td>
<td>Percent</td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>---------------------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>212</td>
<td>149</td>
<td>38</td>
</tr>
<tr>
<td>Tr/Br/Lung</td>
<td>64</td>
<td>41</td>
<td>11</td>
</tr>
<tr>
<td>Breast</td>
<td>0</td>
<td>31</td>
<td>0</td>
</tr>
<tr>
<td>Diabetes</td>
<td>19</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>153</td>
<td>39</td>
<td>27</td>
</tr>
<tr>
<td>Liver/Cirrhosis</td>
<td>18</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Accident</td>
<td>33</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Suicide</td>
<td>23</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Other</td>
<td>103</td>
<td>50</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>561</td>
<td>264</td>
<td>100</td>
</tr>
</tbody>
</table>

**Table 4.4:** Primary causes of death in Canada for men and women aged 45-64 in 2004 [222].
4.4 Discussion

4.4.1 Principal findings

Chronically low income confers a greater hazard of death than transiently low income in both Canada and the United States. Chronically high income confers a lesser hazard of death than transiently high income in both Canada and the United States. The effect of income on hazard of death is twice as great in the United States as it is in Canada.

Income drops negatively affect survival, except for low-income Canadians. The effect size for the income drop effect depends on income level, but patterns differ in Canada and the United States.

4.4.2 Strengths and weaknesses of the study

The LAD is unique in Canada for its combination of longitudinal nature, sample size, long coverage period, low loss to follow-up, annual measurement, use of mortality as a marker of health and detailed income descriptors. The coverage and representativeness of the LAD data are high since tax filing rates are high in Canada. However, filing rates have been rising over time, with filing rates improving most for lower income, older, and female residents of Canada. Increased filing by lower income residents will cause a bias in the distribution of income and in apparent mobility, but this bias is thought to be very small [18]. The long period of follow-up allows for most disease mechanisms to operate and for ages of high mortality to be achieved. The LAD data also span several business cycles, and estimates are therefore averaged across market conditions.

The PSID is a smaller survey, but also possesses annual data and a long coverage period. Capture of death events in the PSID is likely more complete due to active follow-up of respondents.

Income data is of exceptional quality in the LAD due to penalties for non-filing and late-
filing. The PSID also has very high quality data on income by component. In both data sources, the data are annual, detailed by component, and present for all family members. The effort to measure family income in longitudinal data also leads to annual measures of family dynamics.

The incomplete capture of death events in the LAD leads to misclassification of censoring status despite live follow-up in survival analyses. Since this misclassification is more likely at lower levels of income, estimates of the association of income level with mortality may be attenuated towards the null. This misclassification is also more likely among those without spouses or dependants, and so the bias may be greater for the unattached than for those in couple families. This bias is not present in the PSID due to active follow-up.

The PSID has a relatively small sample size and therefore few deaths and limited statistical power. This presents significant restrictions on model specification and on how aggressively sample homogeneity can be pursued using exclusion criteria to control confounding and effect modification.

The principle weakness of the study is the lack of comparable indicators of work disability, education and occupation in the two datasets. Work disability indicators are not equivalent, and the LAD lacks data on education and occupation. These omitted variables may lead to non-conservative biases.

There are also important differences in the time and manner of sampling. Recent immigrants are well-represented in the LAD but not in the PSID, which is a closed cohort sampled in 1968, before much of the recent Latin American immigration occurred. Immigrants have high but rapidly decreasing income volatility. There is likely also a healthy immigrant effect. The presence of immigrants in the LAD sample may therefore have attenuated the effect of income drops relative to the US. This is not necessarily a bias in either case, but reflects the difficulty of producing comparable data sources.
These models do not take account of family dynamics. Changes in the number of income earners represents an artifact, a separate mechanism, or a more complicated situation than is captured by the theoretical model for this analysis. Family dynamics may be differential between the LAD and the PSID.

The ideal lag between exposure and outcome depends on cause of death, but the LAD lacks cause of death and the PSID lacks the statistical power for any further refinement of the models. The main causes of death in Canada in 2004 are shown in Table 4.4. The induction period is very short in the Cox models presented here, but varying induction periods have been shown to result in very similar estimates or risk (see Chapter 5).

4.4.3 Implications for policymakers

The association between income level and mortality appears to be much weaker in Canada than in the United States. While the reasons for this require further investigation, this evidence is consistent with view that comparatively generous Canadian income redistribution measures and universal, publicly-financed health care protect Canadians against health risks faced by their American neighbours.

The effect of income drops on mortality also appears to be modest in Canada compared to the United States. This is consistent with there being more effective existing programs to mitigate such risks in Canada, but there are competing explanations based on uncontrolled confounding and differences in sample composition.

4.4.4 Unanswered questions and future research

This analysis pursued comparability with previously published research at the expense of optimal investigation of the causal association in question, which was pursued separately using only Canadian data (see Chapter 5). Subsequent comparative research should
build on this evidence with well-controlled models specified \textit{a priori}.  

Higher exposure to income drops was observed in Canada. This may be due to differences in unemployment experiences, differences in identification behaviour as unemployed or non-participant, or differences between the samples in their coverage of immigrants or family dynamics. If the greater exposure in Canada is a measurement artifact, it may have biased estimates towards the null, concealing effects of income drops on mortality more similar to those in the United States. In particular, it is possible that the protective effects of income drops at low income levels observed in the LAD data were due to a healthy immigrant effect.

The US data lacked power, and the estimated effects were highly dependent on the use of survey weights (results not shown), which may indicate poorly specified models. The US findings should be replicated using other American data sources.
Chapter 5

Income dynamics and adult mortality in Canada: measurement and modelling
Abstract

Introduction  This paper examines the association of family disposable income level and relative family disposable income drops with the hazard of death in Canada. The roles of various confounding factors and induction periods are explored, as is the possibility of interaction between the effects of income level and relative income drops on risk of death. If income level has a greater effect when income drops occur, then health selection may explain a significant portion of the association. If relative family disposable income drops have a greater effect on risk of death at low income levels, then higher income level may proxy for assets that buffer the effects of income drops. If income level or drops have an effect on risk of death primarily with short induction times, then health selection may explain a significant portion of these associations. If the effects of income level or income drops on risk of death are enduring or increase with increasing induction times, then the associations are either causal or confounded by enduring traits.

Methods  Longitudinal personal income tax data for Canadian families from the Longitudinal Administrative Databank, representing 20% of Canadian tax filers from 1982 though 2005, were used to estimate Cox survival models. The data were restricted to adults aged 40-55 during the exposure year and followed for mortality for 1-18 years. Potential confounding by work disability, births, marriage, family breakdown, family deaths, retirement, and immigration were controlled by excluding these groups from the analyses.

Results  Income level showed a strong negative association with hazard of death. The effect of income level was constant throughout follow-up for men and women in couple families, but decreased somewhat for unattached men and women. The effect of income drops was only clearly apparent for men in couple families, and the effect increased with the magnitude of the income drop. Very little evidence of statistical interaction between
income level and income drops was found.

**Discussion**  The magnitude of the effect of income level on risk of death is consistent with previous Canadian estimates. The lack of attenuation with increasing induction time among adults in couple families argues against a significant role for health selection in this group, while the presence of some attenuation among unattached persons may indicate greater health selection in that group. The lack of interaction between the effects of income level and income drops on risk of death further argues against a significant role for health selection. That income drops had a clear association with risk of death only for men in couple families may be due to lack of statistical power in other groups, or to greater heterogeneity of labour force attachment and economic situation in other groups. The lack of attenuation of the effects of income drops on risk of death suggests either that variable induction-time profiles by cause of death happen to sum to a constant level, or that the effects of income drops are confounded by some enduring trait. The lack of interaction between the effects of income level and income drops on risk of death suggests that the effect of income drops may not be causal via consumption behaviour, since the assets associated with higher income should moderate the effects of income drops on consumption.
5.1 Introduction

This paper examines the association of family disposable income level and relative family disposable income drops on the hazard of death in Canada. The roles of various confounding factors and induction periods are explored, as is the possibility of interaction between income level and relative income drops.

Income level has been shown to have a strong, positive, and monotonic association with longevity in Canada \[100, 248, 173, 246\]. The association is robust to adjustment for education \[173\], and is only weakly attenuated among those with continuously increasing income prior to retirement \[248\], suggesting that the association is not highly confounded by other dimensions of socio-economic status, nor is it primarily the result of health selection. The association of all-cause mortality with family income is strong at working ages for men and women, but weaker at older ages \[246\].

Longitudinal studies of income and health were reviewed by Benzeval and Judge \[25\]. Studies examining income drops and all-cause mortality are not consistent in their definitions of income drops. Studies examining long-term changes in income \[100, 111\], or history of spells in poverty \[252, 157\] are difficult to compare to the relative drops in annual income analyzed here. Relative drops in family income have been analyzed using data from the Panel Study of Income Dynamics in the United States \[156\], and it was observed that family income drops may be associated with all-cause mortality among adults aged 45-64 at baseline, but perhaps only among those with middle family income levels ($20,000 to $70,000 1993USD). These findings were replicated in Chapter 4 using PSID data, but comparable models using Canadian data showed a different pattern.

Job loss and unemployment are closely related constructs to relative income drops, and have been repeatedly associated with all-cause mortality \[127, 43\]. Important potential sources of confounding have been identified in such studies. Health selection can operate by differential job-separation and retirement, differential re-employment, differen-
tial employment at unstable firms, and differential job mobility [227]. Whether job loss depends on prior health may also depend on the dimension of health or disability in question. Kraut et al. linked health utilization data in Manitoba to census data on labour force participation, and examined utilization patterns before, during and after unemployment [122]. They found that the unemployed did have elevated utilization prior to unemployment, but primarily for mental health treatment.

Second, health problems and mortality manifest most frequently at older ages, though economic and labour market experiences likely have cumulative effects over the life-course [63]. Most studies lack the length of follow-up and longitudinal data on income and labour market experiences to describe both the important periods of exposure as well as the peak periods of disease manifestation [227].

Third, there is potential for confounding by other dimensions of socio-economic status, in particular by education and occupation [186, 140]. If lower education leads to poorer health and increased risk of job loss, or if occupations with greater physical and psychosocial health hazards are also less secure, then associations between job loss and subsequent health and mortality might be non-causal.

Fourth, recurrent episodes of short-term unemployment may lead to long-term unemployment, discouraged workers, or to weakened labour force attachment [127]. These states of weakened labour force attachment are characterized by greater risk of lowered earnings, of decreased benefits, and of poorer psychosocial working conditions [185]. It may be that the acute psychological, behavioural and economic consequences of job loss for health are only part of the mechanism, and that the long-term reduction in the quality of labour market experiences following job loss explains a significant part of the effect of job losses on population health [43].

In a recent systematic review, the evidence that the experience of unemployment is a predictor of all-cause mortality is considered strong [127]. This is based on high quality
census-based cohort studies with 2 to 10 year follow-up periods, where unemployment is self-reported with respect to census day or the week prior to census day.

However, given the potential for confounding of the association between job loss or unemployment and mortality, the frequent observation of biologically implausible increases in cancer deaths \([165, 58, 164, 27, 120]\), and the mixed findings of more recent studies examining mass-layoffs \([150, 227, 143, 224]\), skepticism should be maintained about the magnitude and perhaps even the direction of any effect.

Examining family disposable income drops using longitudinal tax data is a more direct method of examining some of the pathways by which job loss and unemployment may affect mortality. This approach also allows one to examine whether greater income drops are associated with greater risk of death, though existence of a dose-response relationship would not be a sufficient rebuttal to suspicion of confounding by work disability, education or occupation.

The most significant confounding variable of the association between income level or income drops and mortality is work disability, as mentioned above with respect to job loss. Other potential confounding factors include births, marriage, family breakdown, family deaths, retirement, immigration status, education, and occupation, which may all cause decreases in family income, and may also have effects on mortality unmediated by those changes in income.

### 5.1.1 Hypotheses

Income level is expected to show a negative association with risk of death. Health selection requires that the onset of work disability causes income to decrease. Therefore, if health selection is a significant contributing mechanism to the association of income level and risk of death, then income level should have a greater effect in the presence of income drops. Health selection is hypothesized to play a small role, and therefore the
effects of income level and income drops on risk of death are not expected to have a positive statistical interaction.

Drops in annual family disposable income during peak years of labour-force attachment are expected to increase the hazard of death over the subsequent 18 year period. The effect size and causal interpretation depend on the induction period. Effects given short induction periods are most plausibly due to health selection or to accidental deaths due to increased risk-taking. Longer term effects are more likely causal and due to an aggravation of chronic disease processes. Income level is expected to modify the effect of income drops. Notwithstanding the observations of McDonough et al. [156], higher income is expected to be associated with decreased effects of income drops due to reduced cash constraint thanks to greater assets, and so a negative statistical interaction is expected.

5.2 Methods

5.2.1 Data

The data are from the Longitudinal Administrative Databank (LAD) [221]. The LAD is a 20% random sample of individuals in the T1 Family File (T1FF), which contains annual tax data for 1982 and onwards. Data for the years 1982 through 2005 were used for this study. Once sampled for the LAD, individuals are followed until death or loss to follow-up, and an annual top-up sample maintains representativeness.

The T1FF is an annual census of all Canadians who file personal income tax (T1 form), or whose Social Insurance Number (SIN) is listed on another family member’s tax file. Both legal and common law spouses are identified by the Social Insurance Number (SIN) listed on T1 tax forms or by matching on name, address, age, sex and marital status. Children are identified from T1 data as well as supplementary files from the Child
Tax Benefit and Family Allowance programs. The T1FF therefore consists of filers, who all have a reported SIN, and dependent children and spouses, some of whom have a reported SIN. Only T1FF individuals with a SIN can be sampled into the LAD.

The sex of taxfilers, which is not reported on the T1 form, is established by the Canada Revenue Agency by linking to the SIN master file, held by Human Resources and Skills Development Canada. Unmatched spouses are assigned a sex opposite to their spouse, and a small number of unmatched single individuals are assigned a sex randomly.

**Exclusions**

Person-years of exposure (PYE) are restricted to ages 40-55. Since the previous five years’ income is used to determine income level and project expected income, filers aged 35-50 in 1982 contribute the earliest exposure data for PYE in 1987. These age exclusions reduce the potential confounding by births, marriages and retirement. The LAD contains approximately 833,000 filers aged 35-50 in 1982, rising to 1.5 million in 1998, which is the start year for the final exposure assessment period. This represents 9.6 million PYE for women and 9.7 million for men.

Effect modification by family type is addressed by stratification into two groups: unattached and couple families.\(^1\) Single parents are excluded due to their insufficient number. Recent immigrants were excluded since the labour market experiences and income dynamics of recent immigrants are significantly different from the rest of the population. Immigration is defined in the LAD as those landing in Canada on or after 1982 and represented 5.8% and 6.4% of PYE for women and men.

PYE were excluded if more than two of the previous five years of income were missing, which was the case for 3.0% of women and 3.1% of men. PYE were excluded if income

---

\(^1\)0.7 and 0.8 million common law couple person-years of exposure were excluded from couple families accidentally due to coding error. Common law status is more common over time: 1% rising to 6% for women and 7% for men between 1982 and 1998. This will be corrected in a future run.
data were missing for the exposure year, which further reduced PYE by 1.6%. Year-over-year income drops were also only estimable when the exposure year and the previous year both had non-missing income data, reducing the sample by 1.6% in those models.

Models were estimated with and without further exclusion of potential confounding variables. The following exclusions applied to the exposure year and the previous five year period: family receipt of retirement (pension/superannuation) income, death of a family member (a tax-filer or spouse), change in family structure (couple family, unattached, single parent), greater than 20% family self-employment income. Self-employment income was defined as receiving greater than 20% of total family income from self-employment sources during the exposure year or any of the previous five years. Self-employment income sources were limited partnership income, dividend income, business income, professional income, commission income, farming income, fishing income, or rental income. Total family income is defined as self-employment income plus employment income from T4 slips and other employment income.

Exclusion for work disability is based on the exposure year, any of the five previous years or any of the two subsequent years. Subsequent years’ data are used to allow for administrative delays in benefit receipt. The disability flag was set when any family member reported income indicative of work disability (CPP/QPP Disability, Worker’s Compensation, Disability Deduction Amount). Disability and retirement income were defined as family, not individual characteristics to avoid problems related to their transferability on tax forms and to err on the side of sensitivity.

Models were also estimated using only data from 1992 onward, when transfer amounts and deaths were better captured and filing rates were higher.
Survival data

The data were structured as a person-period dataset with PYE accompanied by one censoring variable for each lag. The time origin is date of birth, such that the baseline hazard is a function of age. Although the day and month of death are reported on the T1 form, only year is captured in the LAD. Time aggregation bias does not affect the results since the exact method of treating tied events is used, as discussed in Section 2.2.1.

Income level and dynamics

Income was defined family disposable income, adjusted by the Canadian consumer price index (CPI) to reflect 2005 dollars, and by the square root of family size to adjust for economies of scale in household consumption [9, p.35-6]. Family size was taken to be the greatest number reported during the PYE or the previous five years. Income level is meant to be an estimate of permanent income, which is “the amount that an individual can spend consistently with being able to maintain the same level of spending in the future,” and therefore a reflection of consumption behaviour [31].

The measurement of income level and income dynamics follows a procedure adapted from Morissette and Ostrovsky [161, 160, 162]. Income level and income dynamics in year $t_j$ were based on the income reported in years $t_{j-5}$ through $t_{j-1}$. Filers were first stratified by (1) income tertiles, based on mean of previous 5 years, (2) single versus two-adult households, and (3) five year age brackets based on filer’s age (40-44, 45-49, 50-54). A simple linear random effects model is then applied within each stratum to log equivalized family disposable income $y_{it}$ for families $i$ at year $t$:

$$y_{it} = e_i + f(A_i) + u_{it}$$

(5.1)

where $e_i$ is the family-specific random intercept, $f(A_i)$ is a quadratic function of filer’s...
baseline age $A_i$, and $u_{it}$, the family-year residual, indicates transitory earnings. This model assumes a common age-income profile within strata while allowing for filer-specific intercepts.

Because predicted values $\hat{y}_{it}$ from the mixed growth model involved shrinkage to the mean, an undesirable systematic bias is introduced such that low income levels are overestimated and high levels are underestimated. This was corrected by adjusting the predicted values so that the predicted mean of the previous five years was equal to the observed mean of the previous five years:

$$\hat{y}_{it}' = \hat{y}_{it} + (\bar{y}_{i-t-1\ldots-5} - \bar{\hat{y}}_{i-t-1\ldots-5}).$$ (5.2)

Income level $\bar{y}_{i-t-1\ldots-5}$ was categorized as tertiles. The adjusted income dynamics residuals $y_{it} - \hat{y}_{it}'$ were categorized as no drop, 1 to 15% drop, 15 to 50% drop, and 50% to 100% drop. If predicted income is slightly negative, relative change will be large and negative since observed income will typically be a large positive number. Predicted values of less than or equal to $1 were therefore set to $1. For comparison, a simple income change score of $t_{ij} - t_{ij-1}$ was also constructed using the same relative drop categories.

5.2.2 Analyses

The data were analyzed using Cox proportional hazards models. Left-truncation, right censoring and time-dependent covariates were implemented using the counting process form of the Cox model [228, p.68-77][200, p.3241-2] using PROC TPHREG in SAS [200, p.4471]. The counting process form of the Cox model allows for time origins other than study enrolment, and in this case date of birth was considered more appropriate, following Korn et al. [121]. Since the baseline hazard does not need to be specified, it is very desirable to express the baseline hazard as a function of age when age is among the
strongest determinants of the hazard and needs to be controlled for, as in an analysis of all-cause mortality.

The more traditional formulation of the Cox proportional hazard model with study enrolment as the time origin and adjusting for age-at-baseline as a covariate will be relatively unbiased if the baseline hazard function is approximately exponential, which is likely for all-cause mortality. If the baseline hazard is not exponential, estimates will also be relatively unbiased if the covariates are not correlated with age [121], but this is frequently not the case.

Effect modification by income level was addressed in multivariate models. Separate models were estimated for men and women, and for couple families and unattached filers.

5.3 Results

Person years for select Cox regressions are shown in Table 5.1. For each lag between exposure and outcome, a PYE is in the risk set if the filer was not lost to follow-up or right-censored by the final year of data collection, and did not die prior to the outcome year. The total PYE for women and men is greatest for a lag of 1, for which there are 6.8 and 7.7 million PYE respectively in unrestricted models, and lowest for a lag of 18, for which there are 246,000 and 297,000.

Disposable family income tertiles (equivalised 2005$) were cut at $24,500 and $37,500, with tertile medians of $18,000, $31,000 and $48,000 and means of $17,000, $31,000 and $55,000.\(^2\)

Income drops were defined both as a simple measure of annual change, the exposure year less the previous year, and as a deviation from a projection. Age-income trajecto-

\(^2\)Income tertiles were estimated separately for men and women, but the summary statistics accidentally pooled them. The min and max statistics show that the two were very similar, since these overlap by only $1000, which is the rounding error. This will be corrected in a future run.
ries were projected with simple growth curve models with random intercepts but a shared quadratic function of age (see Figure C.1). These models were estimated within strata defined by income tertiles, sex, 5-year age groups, and using the 5 years of tax data preceding each exposure year. Log transformation of income prior to projection was explored, but generally had very modest effects on the estimated trajectories, and slightly larger effects on the intercepts, particularly for higher-income strata. Since in all cases shrinkage to the mean was removed by centering the mean of the predicted values on the mean of the observed values, differences in the intercept due to transformation are of no consequence to the estimates of income drops for the forecasted exposure year. The models performed well for most groups, but less well for those aged 50-55, whose standard errors were higher, due to the much smaller size of these cohorts (see Figure C.2).

The first 5-year age-income profile period covers the years 1982-1986, and the last the years 1998-2002. Both of these periods are near peaks in the business cycle. Men in couple families aged 50-54 experienced substantial regression towards the mean during most phases of the business cycle. At the younger ages of 35-39 (the five years preceding an income drop at age 40), men in couple families experience strong income growth in peak periods and generally stagnant age-income trajectories during the trough years. Overall there are large differences in age-income profile across period, cohort and age, as well as between income tertiles.

Annual and projected income dynamics are symmetrically distributed around a mode of 0 (see Figure C.3). Incidence of drops is highly stable across lag and calendar year within sex, family type and income tertile strata, although drops of 0-15% become slightly less common among poor, unattached men and women with increasing lag. Table 5.2 shows the incidence of income drops.

Survival models used year of birth as the time origin and integer-years as the time-scale. Simulations (not shown) showed that for large samples there is no loss of power.
in Cox models when using integer-year as the time-scale as opposed to day-of-death, provided that exact estimation methods are used. Using year of birth as the time origin allows for non-parametric adjustment for age as a predictor of death. Income level, income drops, and their interaction, all specified as time-varying predictors, were the only predictor variables. Separate models were estimated for strata defined by family type, sex, and lag. Lag is the number of years between the exposure year and observed death events. To be in the risk set for any model, the person must have contributed exposure data and not have died or been lost to follow up before the year of death defined by the lag. Sample size is smaller for models with long lags, since many exposure periods lack the length of follow-up for these models (see Figure C.4 for a graphical summary of cohort dynamics).

Men in couple families, who contribute the most exposure years as well as more deaths than women of the same age, showed the greatest global significance test p-value (LR, Score, Wald), exceeding $p=10^{-10}$ for all but lag=18 for models with all exclusions. Unattached men and women at longer lags had marginal global significance for models with all exclusions.

In additive models (those without an interaction between income level and drops) the effect of income level was marked, as shown in Figure 5.1. While there is some decay of the effect of being in the poor income tertile with increasing lag for unattached men and women, for other groups the magnitude of the effect of income level is very stable over 18 years. Adjusting for income drops has no important effect on the effect of income level on hazard of death.

In additive models, a clear picture of dose-response for the effect of income drops was only found among men in couple families, as shown in Figure 5.2 (see Figures C.9, C.10 and C.11 for other strata). While the smallest income drops of 0-15% may decay to the null with increasing lag, larger income drops appear to have very stable associations with
hazard of death with increasing induction time.

Type 3 significance tests for the interaction between income level and income drops were not highly significant except modestly for men and women at shorter lags when the work-disabled were included (see Figure C.12). There is also weak evidence of an interaction at shorter lags among men and women in couple families when no exclusions are applied.

Beginning in 1992 the data on transfers are much more complete. The quality of the exclusions therefore increases for these years. Models restricted to these data show very similar patterns to those using data from all years, but with lower precision due to the lesser statistical power.
TABLE 5.1: Person-years of exposure (PYE; thousands) and number of deaths in select Cox models. Unrestricted models exclude only those with missing income data, and filers who immigrated on or after 1982. Fully restricted models exclude those with retirement income, family deaths, significant self-employment income, or changes in family structure during the PYE or the five previous years, and those with disability income during the PYE, the previous five years, or the following two years.

<p>| Lag | Couple families Unrestricted | | Couple families Fully restricted | | Unattached | | Unattached |
|-----|-----------------------------|----|-----------------------------|----|-----------------------------|----|
|     | PYE '000 | Deaths | PYE '000 | Deaths | PYE '000 | Deaths | PYE '000 | Deaths | PYE '000 | Deaths | PYE '000 | Deaths | PYE '000 | Deaths |
| 1   | 6,018 | 7,330 | 6,487 | 13,555 | 818 | 1,785 | 1,172 | 4,015 | 2,199 | 1,690 | 2,574 | 3,715 | 336 | 480 | 349 | 1,080 |
| 2   | 5,981 | 8,420 | 6,446 | 15,515 | 811 | 2,275 | 1,156 | 5,190 | 2,187 | 1,820 | 2,376 | 4,570 | 334 | 650 | 345 | 1,460 |
| 3   | 5,485 | 8,565 | 5,958 | 16,005 | 733 | 2,420 | 1,035 | 5,000 | 2,007 | 2,225 | 2,574 | 4,670 | 304 | 715 | 310 | 1,495 |
| 4   | 5,034 | 8,710 | 5,509 | 16,455 | 669 | 2,225 | 935 | 4,865 | 1,838 | 2,550 | 2,199 | 5,245 | 276 | 775 | 278 | 1,500 |
| 5   | 4,592 | 8,810 | 5,063 | 16,725 | 606 | 2,220 | 838 | 4,715 | 1,676 | 2,675 | 2,026 | 5,540 | 249 | 765 | 247 | 1,465 |
| 6   | 4,164 | 8,620 | 4,628 | 16,980 | 545 | 2,130 | 745 | 4,540 | 1,521 | 2,760 | 1,858 | 5,845 | 222 | 730 | 218 | 1,410 |
| 7   | 3,749 | 8,730 | 4,199 | 16,955 | 486 | 2,040 | 658 | 4,355 | 1,371 | 2,805 | 1,694 | 6,070 | 197 | 740 | 191 | 1,340 |
| 8   | 3,350 | 8,635 | 3,781 | 16,885 | 430 | 1,885 | 578 | 4,165 | 1,229 | 2,790 | 1,536 | 6,160 | 173 | 680 | 165 | 1,265 |
| 9   | 2,966 | 8,275 | 3,371 | 16,650 | 377 | 1,800 | 504 | 3,895 | 1,096 | 2,675 | 1,383 | 6,190 | 150 | 635 | 142 | 1,170 |
| 10  | 2,593 | 7,665 | 2,972 | 16,010 | 325 | 1,640 | 435 | 3,650 | 968 | 2,620 | 1,234 | 6,120 | 129 | 570 | 121 | 1,075 |
| 11  | 2,235 | 7,220 | 2,582 | 14,850 | 276 | 1,450 | 371 | 3,215 | 845 | 2,450 | 1,086 | 5,770 | 108 | 515 | 102 | 965 |
| 12  | 1,895 | 6,810 | 2,205 | 13,990 | 232 | 1,310 | 314 | 2,925 | 728 | 2,355 | 945 | 5,665 | 90 | 475 | 85 | 885 |
| 13  | 1,572 | 6,275 | 1,842 | 12,655 | 192 | 1,105 | 260 | 2,710 | 617 | 2,260 | 807 | 5,310 | 73 | 380 | 69 | 780 |
| 14  | 1,270 | 5,585 | 1,496 | 11,275 | 150 | 950 | 204 | 2,230 | 512 | 2,090 | 674 | 4,910 | 59 | 340 | 56 | 670 |
| 15  | 986  | 4,810 | 1,168 | 9,705  | 113 | 780  | 152 | 1,835 | 409 | 1,840 | 541 | 4,430 | 47 | 295 | 44 | 575 |
| 16  | 717  | 3,810 | 852  | 7,780  | 81  | 635  | 108 | 1,430 | 307 | 1,560 | 407 | 3,645 | 35 | 255 | 33 | 465 |
| 17  | 463  | 2,750 | 551  | 5,530  | 51  | 420  | 67  | 960  | 199 | 1,140 | 265 | 2,625 | 23 | 175 | 22 | 320 |
| 18  | 223  | 1,470 | 266  | 2,915  | 23  | 205  | 31  | 510  | 97  | 615  | 130 | 1,390 | 11 | 90  | 10 | 175 |</p>
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<th>Unattached</th>
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<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td></td>
</tr>
<tr>
<td>PYE '000</td>
<td>%</td>
<td>PYE '000</td>
<td>%</td>
</tr>
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<td>1,121</td>
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<td>193 48.9 290 50.1</td>
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<tr>
<td>(0-15%)</td>
<td>291</td>
<td>15.4 372 18.9</td>
<td>78 19.8 100 17.4</td>
</tr>
<tr>
<td>(15-50%)</td>
<td>309</td>
<td>16.4 338 17.1</td>
<td>84 21.3 125 21.6</td>
</tr>
<tr>
<td>(50-100%)</td>
<td>162</td>
<td>8.6 120 6.1</td>
<td>39 10.0 63 11.0</td>
</tr>
<tr>
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<td>216 54.7 317 54.9</td>
</tr>
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<td>(0-15%)</td>
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<td>107 27.1 145 25.0</td>
</tr>
<tr>
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<td>48 12.1 77 13.4</td>
</tr>
<tr>
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</tr>
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<td>53 26.9 104 33.1</td>
</tr>
<tr>
<td>(0-15%)</td>
<td>593</td>
<td>28.4 697 31.1</td>
<td>82 42.1 110 35.1</td>
</tr>
<tr>
<td>(15-50%)</td>
<td>423</td>
<td>20.3 459 20.5</td>
<td>51 26.2 80 25.6</td>
</tr>
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<td>(50-100%)</td>
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<td>102 52.2 162 51.8</td>
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<td>69 35.2 104 33.1</td>
</tr>
<tr>
<td>(15-50%)</td>
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<td>14.4 304 13.6</td>
<td>19 9.1 36 11.5</td>
</tr>
<tr>
<td>(50-100%)</td>
<td>76</td>
<td>3.7 57 2.5</td>
<td>7 3.6 11 3.6</td>
</tr>
</tbody>
</table>

**Table 5.2**: Person-years of exposure (PYE; thousands) and percentage in select Cox models. Models exclude only those with missing income data, and filers who immigrated on or after 1982. Only models with a lag of 1 year are described.
**Figure 5.1:** Effect of income level in additive models. Separate models were estimated for each combination of sex, family type, lag and exclusion set. Results shown for fully-restricted models only.
Effect of income drops (all exclusions; adjusted for income level): Male/Couple families

<table>
<thead>
<tr>
<th>Years between exposure and outcome</th>
<th>HR (compared to no drop; log scale)</th>
</tr>
</thead>
</table>

**Figure 5.2:** Effect of income drops in additive models for men in couple families. Separate models were estimated for each combination of sex, family type, lag and exclusion set. Estimates are shown for fully-restricted models only.
5.4 Discussion

5.4.1 Principal findings

The effect of family disposable income level showed the expected negative association with hazard of death when families were excluded for all potentially confounding factors including work disability income. The lowest income tertile had approximately 37-200% greater hazard of death than the highest income tertile each year during the 18 years after income level measurement. The middle income tertile had approximately 12-35% increased hazard of death compared to the top income tertile. These effect sizes are consistent with those reported from Canadian Census linkages \cite{173, 246} and for men in the Canadian Pension Plan \cite{248}.

The effect of family disposable income level appeared to be greater for unattached men, and unattached women in the poorest tertile. Analyses of the Canadian Pension Plan data also identified a greater effect of income for unmarried men \cite{248}.

The estimated hazard of death by family disposable income level is remarkably stable over time after measurement of exposure. This stability may depend on family type. Slight declines over time for men and women in couple families are consistent with a survivorship effect, but the overall stability in this group argues strongly against a dominant role for health selection. Stronger declines over time for unattached men and women in the bottom income level tertile are perhaps more consistent with a partial role for health selection or with an unknown additional risk that decays over a period of approximately 10 years.

The observation that the effect of income level is not modified by income drops is a powerful argument against an important role for health selection. This is a confirmation of the finding that among male Canadian pensioners the association between income level and mortality was barely attenuated among the subset with continuously rising in-
The models estimated for this study suggest that disposable family income drops may increase mortality for men in couple families. A relationship for other groups, which have less statistical power, is not as clear, but most estimates are in the direction of increased risk. Risk of death for men in couple families shows a clear dose-response association with increasing magnitude of drops in family disposable income. The effect size for income drops of 0-15% are quite small, but the effect sizes for income drops of 15-50% and 50-100% compared to no income drops is similar to the increased risk of death for the middle income level tertile compared to the richest income level tertile. Whereas income level tertiles offer ample statistical power and their effects on hazard of death are therefore precisely estimated, large annual income drops are uncommon, and so their effects are less precisely estimated.

Among men in couple families the mortality effects of small income drops of 0-15% appear to decay to the null after 10 years, but the effects of larger drops are very enduring. Such enduring effects for an acute exposure raise questions about whether the effect is causal, or whether the association is confounded by an enduring trait that confers both health and economic risks, such as time preference. If causal, the effect size could be maintained over time due to cause-specific mechanisms on different time-scales that happen to produce a constant effect size for all-cause mortality, or it could be maintained by income drops preceding a decreased quality of labour force attachment over a long period of time, and therefore representing the start of a long-term exposure rather than an acute one.

There was very little evidence of a dependency of the effects of relative drops in family disposable income on the level of family disposable income. Since higher income is associated with greater savings and access to credit, and these can maintain consumption during a drop in income, it is surprising that no effect modification was observed. This may
be due to insufficient statistical power, but lack of such evidence weakens the argument for a causal effect of income drops on mortality.

5.4.2 Strengths and weaknesses of the study

The LAD is unique in Canada for its combination of longitudinal nature, sample size, long coverage period, low loss to follow-up, annual measurement, use of mortality as a marker of health and detailed income descriptors. The coverage and representativeness of the LAD data are high since tax filing rates are high in Canada. However, filing rates have been rising over time, with filing rates improving most for lower income, older, and female residents of Canada. Increased filing by lower income residents will change the distribution of income over time and change estimates of economic mobility, but this bias is thought to be very small [18]. The long period of follow-up allows for most disease mechanisms to operate and for ages of high mortality to be achieved. The LAD data also span several business cycles, and estimates are therefore averaged across market conditions.

Income data is of exceptional quality due to penalties for non-filing and late-filing. Furthermore, the data are annual, detailed by component, and present for all family members. The effort to measure family income in longitudinal data also leads to annual measures of family dynamics.

The incomplete capture of death events leads to misclassification of censoring status despite live follow-up in survival analyses. Since this misclassification is more likely at lower levels of income, estimates of the association of income level with mortality will be attenuated towards the null. This misclassification is also more likely among those without spouses or dependants, and so the bias may be greater for the unattached than for those in couple families.

The lack of data on cause of death is an unfortunate limitation. Different causes of
death would be expected to show associations with income drops after differing lags. For example, suicide, alcohol poisoning, and accidents might be expected to have an acute association that diminishes over time, while heart disease might have both short-term associations due to aggravation of existing disease or changes in medical care received, as well as associations at longer lags due to disease largely initiated by the decreased income. Also, biologically implausible associations with cancer mortality, as have been observed in studies of job loss and unemployment, cannot be ruled out in the absence of data on cause of death. Despite twice winning funding to do so, we were unable to negotiate a linkage of the LAD to the Canadian Mortality Database. Further efforts to achieve this linkage may be pursued in the future.

The principal weakness of the study is the limited availability of data on the health and sociodemographic characteristics of taxfilers. Health selection in which work disability affects both income level and income drops as well as mortality is a serious inferential problem. The indicators of work disability used here were receipt of disability compensation income, allowing for delays between application and receipt. These are partial proxies of work disability. Pension disability income can only be received for disabilities that completely prevent gainful employment, worker's compensation income can only be received for disabilities attributable to exposures in the workplace, and the disability tax deduction requires disabilities affective activities of daily living without reference to ability to work. Furthermore, not all eligible persons claim or apply for these amounts, nor are all eligible claimants successful [77, 208]. Unidentified work disability is expected to lead to non-conservative bias. There is also no data about the type of disability, and patterns of labour force attachment and cause of death likely differ according to the nature of disability. In Manitoba, it was observed that mental health care utilization was elevated prior to spells of unemployment, as well as subsequently [122].

Education and occupation are determinants of income level, of income drops, and
of mortality. To the extent that their effects are not mediated by income level and income drops, they may confound estimates of the associations of income level and income drops with hazard of death. As with work disability, the lack of control for education and occupation is expected to lead to non-conservative bias.

Measuring a single instance of income drop may be identifying a long-term trait of economic precariousness in addition to the transitory experience of income decline. This might be addressed directly by more elaborate models that simultaneously adjust for multiple periods of income decline, treating it as a recurrent or chronic exposure. If income drops are a risk factor for death and are serially correlated and so the effect sizes would likely be less than additive for a cumulative exposure measure. However, the causal mechanism need not be independent between income drop events, and this makes predicting the association for a cumulative measure difficult.

In order to allow for delayed receipt of disability income, receipt of such income during the five years prior to vital status ascertainment, as well as the year of and the year after, were all treated as exogenous change to health status and potentially confounding. However, some of these instances of disability income receipt may have been endogenous, that is to say caused by the measured exposures, which would lead to over-adjustment for confounding by work-disability and bias towards the null.

5.4.3 Strengths and weaknesses in relation to other studies

Compared to Canadian census linkage studies [173, 246], the LAD has the benefit of longitudinal exposure measurement for income level and family dynamics, but lacks data on education and occupation. Compared to analyses of Canadian pension data [248], this analysis of tax data focusses on family disposable income instead of individual pensionable earnings, includes women, and examines deaths at a wider variety of ages and induction times. These studies have consistent findings, and the variety of data sources
and analytic methods provides a robust portrait of the effect of income level on hazard of death in Canada, and compelling evidence that health selection plays a small role in producing this association.

The principle precedent for this research on income drops was the excellent paper by McDonough et al. [156]. Their data source offered low statistical power, however, leading to several compromises, including a dichotomous definition of income drops, and tacit assumptions regarding the lag for their model. They also did not adjust for significant confounding factors, such as work disability. These limitations were largely overcome in this study which used a much larger data source. Furthermore, the differences observed by sex and family type in these analyses were not explored previously.

### 5.4.4 Implications for policymakers

This research reproduces and confirms the graded effects of income level on hazard of death and suggests only a small role for health selection, despite the lower income and survival rates of the work disabled. Since exposure is universal, the moderate effect sizes imply a large population health burden.

The effect sizes for larger income drops for men in couple families were similar to the effect of income level when comparing the middle tertile to the richest tertile. Income drops are not rare exposures, and are recurrent. While the hazard ratios are not large in terms of strength of evidence regarding causal inference, and there is a possibility of uncontrolled confounding, if the estimates of risk are causal the population burden could be substantial.

If the effects are causal, the external generalizability is questionable despite the strength of the LAD data. Exposure to income drops is highly contingent on labour policy and economic conditions, which vary over time and place. Policies that buffer the effects of income drops, such as health care financing, and direct housing and food assistance, are
also highly variable between jurisdictions. It is possible that when exposure is high the characteristics of the exposed will differ in ways that modify the effects of income drops on hazard of death.

Economic risk pooling can take place on many scales, and is not restricted to government programs. The number of earners in a family has great potential to reduce the magnitude of family income dynamics [160, 161].

### 5.4.5 Unanswered questions and future research

The lack of evidence for an interaction between income level and income drops is both evidence against health selection and lack of evidence that income level proxies for assets that moderate the effects of income drops. This finding should be replicated with other data, preferably with direct measures of liquid and illiquid assets.

Research on income level and hazard of death should focus on interventions. Interventions can either reduce exposure to relative differences in income level through equalization, or minimize the effects of relative differences in income level by interrupting the mechanisms that lead to health risk. Research prioritizing possible interventions based on their effectiveness, efficiency, other consequences, and feasibility in Canada is needed.

The relatively constant effects of income drops among men in couple families over time after exposure could obscure more dynamic patterns by cause of death. Alternative explanations include the possibilities that income drops are due to an enduring trait such as mental health problems, education or occupation that is associated with increased mortality, or that income drops initiate a prolonged exposure to weakened labour force attachment, leading to long-lasting excess mortality.

This study did not explicitly explore effect modification of the effect of income drops by the cause of the income drops. The goal was to isolate changes in family disposable
income that were principally due to changes in market income. However, the effects of income drops due to divorce, births, retirement and other causes may confer health risks that merit direct investigation.
Chapter 6

The summary measurement of social inequalities in health
Abstract

This paper reviews the considerations involved in quantifying the extent of the unequal distribution of health by socio-economic status in populations. Such summary measurement is required whenever groups are compared or trends over time are described with respect to social inequality in health. Thirteen measures are classified according to their health input, socio-economic input, relative or absolute scale, and their ability to detect reverse gradients, group size, re-distribution, and population size. The measures are divided into four families: effect measures, shortfall measures, heterogeneity measures and inequality measures. The most common summary measures, the rate ratio and typical regression coefficient, are argued to be inappropriate for the task of describing the extent of inequality, while others are shown to be more appropriate. Important differences between absolute and relative measures are discussed, and absolute measures are recommended for describing the extent of inequality because of the undesirable ethical assumptions implicit in relative summary measures.
6.1 Introduction

Social inequalities in health, or ‘health disparities’, refers to the unequal distribution of health across groups defined by income, education, occupational status, gender, ethnicity, skin colour, sexual orientation. This paper focuses on constructs similar to income, education and occupational status that are finely graded and at least ordinal in nature. This review benefits from excellent earlier reviews by Wagstaff, Paci and van Doorslaer [240], Kunst and Mackenbach [125], Mackenbach and Kunst [138], Manor, Matthews and Power [139], and Anand et al. [7].

Thirteen summary measures of the extent of social inequality in health are classified according to their health input, socio-economic input, relative or absolute scale, and their ability to detect reverse gradients, group size, re-distribution, and population size (see Table 6.1). Some of these properties are desirable and are therefore useful in selecting an appropriate measure. The distinction between relative and absolute measures is more difficult, because almost all measures can be calculated in either form, and because the choice involves difficult ethical decisions that are typically unacknowledged.
TABLE 6.1: Summary measures of social inequality in health: rate difference and ratio (RD, RR), Agresti’s alpha (\(\alpha\)), population attributable risk (PAR, PAR%), potential years of life or health expectancy lost (PYLL/PHEL), index of dissimilarity (ID, ID%), Pseudo-Gini (Gp), slope and relative indexes of inequality (SII, RII), generalized concentration index and concentration index (CIg, CI).

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<th>SES input</th>
<th>Effect/Shortfall</th>
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<th>Detects group size</th>
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<tr>
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<td>y</td>
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<td>n</td>
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<td>y</td>
<td>n</td>
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</tbody>
</table>
These properties will often have empirical relevance. In particular, the choice between relative and absolute inequality will often determine the result of a comparison. Consider the data used in the Acheson Report [1] as plotted in Figure 6.1. The relative rates agree with the report’s conclusion of increasing social inequalities, but the rate differences (an absolute treatment of the data) display no clear trend. Even without calculating a summary measure, it is clear that the choice between relative and absolute treatment of the data has both scientific and policy implications.

The properties that distinguish summary measures of inequality in health are classified according into properties partly drawn from the literature on the measurement of economic inequality. These properties include: (1) the nature of the health input, (2) the nature of the social hierarchy input, (3) whether the measure can detect reverse (i.e. healthy poor and sick rich) or null social gradients in risk, (4) whether the measure is sensitive to the distribution of persons on the social hierarchy input (i.e. group size), (5) whether the measure is sensitive to re-distribution of health, (6) whether the measure is sensitive to the size of the population, and (6) whether the measure is relative or absolute. Table 6.1 lists the various measures of inequality in health that we found in the literature and the properties that characterize each.

Categorical measures of health or socio-economic status can be either nominal, with unordered discrete categories, or ordinal, with theoretically ordered discrete categories. Continuous measures can be either interval measures (called cardinal by economists), such that ratios have no meaning but ratios of differences do, or ratio measures, such that ratios do have meaning [226, 95]. Unlike interval measures (e.g. degrees Celcius), the zero point on a ratio measure is not arbitrary (e.g. temperature in Kelvin). A dichotomous variable is a special case of a categorical variable.
**Figure 6.1:** All-cause, age-standardized mortality rates over time by Registrar General Social Classification for men aged 20-64 in England and Wales. The Acheson Report describes these data as they are plotted at left – in relative terms – as evidence of increasing inequality [1]. The center plot shows the absolute trends, which are much more ambiguous. The discrepancy is due to the trend in group mortality rates, shown at right. (Note that class size and occupational composition are not constant between time periods.)
**Chapter 6. Summary measurement**

### 6.1.1 Health input

Of the measures listed in Table 6.1, only Agresti's $\alpha$ requires an ordinal (e.g. self-rated health, severity of disease or disability) or dichotomous health input. Most other measures have been created for dichotomous health inputs (e.g. diseased, disabled, dead) or continuous health variables (e.g. years, quality-adjusted years). Some, including the index of dissimilarity and the concentration index, could be used for either dichotomous or continuous health inputs. It may be very difficult to compare inequality, for example, between an ordinal and a continuous health input, because no comparable summary measures are available. We will return to the nature of the health input below when we consider the important difference between measures that improve and worsen toward their zero point.

### 6.1.2 SES input

The simplest measures, the rate ratio and rate difference (referred to by Wagstaff et al. as the ‘range’ [240]), only examine extreme groups from an ordinal SES measure. This simplification ignores the functional form of risk on socio-economic status, and such measures have been called a measures of effect in order to distinguish them from measures that describe inequality across the entire socio-economic spectrum [125, 138]. Measures of effect are not sensitive to group size, and ignore the distribution of health in all intermediate groups, making them problematic as summary measures of inequality. If the socio-economic groups are of constant size, such as quintiles, the measures continue to be flawed in that they ignore the distribution health among the intermediate groups. Also, not all SES data can be easily divided into categories of constant size.

Dichotomous treatments of SES are of course a simplification of the finely graded underlying social hierarchy and are insensitive to the distribution of health within each half of the dichotomy. They are best used for the specific purpose of quantifying the change in
aggregate population health measures that would be observed were all persons to have the health experience of a reference category (i.e. the group least exposed to the risk). These measures will be called shortfall measures. The reference category is usually the most advantaged social group, but is sometimes taken to be the healthiest group. This practice is controversial because (1) the summary measure can no longer be interpreted without also stating the reference group, (2) this removes the measure’s capacity to detect reverse gradients, and (3) because the expected monotonic nature of the social gradient in health provides no reason for the practice. Like comparisons of extreme socio-economic groups, extant summary measures that assume dichotomous SES have the quality of being insensitive to changes in the proportions of the sample that are attributed to each group. While it will sometimes be of interest to discuss the shortfall in the aggregate that is attributable to social inequalities in health, these measures are lacking when it comes to ranking populations based on their inequality because they are insensitive to group size and the distribution of health within each group.

Some summary measures ignore the natural ordinality of the socio-economic hierarchy. This leads to an inability to detect reverse or null social gradients in risk, and an exaggeration of inequality when the social distribution of health is not perfectly monotonic (i.e. when health as a function of SES is everywhere increasing or everywhere decreasing), as shown in Figure 6.4 for the index of dissimilarity (ID). Departures from monotonicity are probable when the number of socio-economic categories is large or when the association of SES and health is weak. Summary measures assuming nominal SES will be termed measures of heterogeneity rather than of inequality.

Ordinal measures of SES are ideal because they capture all persons and express the natural ordinality of social hierarchies, thus assuming a monotonic relationship between the health measure and the socio-economic measure. Summary measures in which socio-economic status is assumed to be ordinal will be referred to as inequality
measures, in contrast to effect, shortfall, and heterogeneity measures.

No summary measures assume a functional form of health on socio-economic status more specific or complex than monotonic. While some use linear regression techniques, for example, this does not reflect the belief that the functional form of the association is linear, only that the linear parameter is an appropriate summary of the association.

6.1.3 Relative and absolute summary measures

Most measures can be calculated in absolute (having dimensions) or relative (dimensionless) form, but there are important normative and positive assumptions involved in choosing between these two forms, and the two forms can disagree as to which group has more or less inequality, or whether inequality is increasing or decreasing over time (see Figure 6.1). The discrepancy arises due to the fact that relative measures divide the ‘dispersion’ part of the measure by some reference quantity, usually the grand or group mean of the health measure, while absolute measures express the observed ‘dispersion’ directly. In Figure 6.1, most groups have equivalent absolute changes in mortality (class V may be an exception), and so from an absolute perspective, inequality was relatively stable. However, when the mortality rates are expressed relative to class I, both the denominator and the numerator are decreasing towards zero at the same rate, causing the relative differences to increase. This problem was addressed by Pamuk et al. in their response to the Black Report [180]. We will discuss the choice between relative and absolute measures below.

6.1.4 Sensitivity to group size

Standard epidemiological analyses are designed to investigate proposed causal models, and this goal differs from that of summarizing social inequalities in health. The following
example illustrates the incompatibility of the typical analytic model with the goal of inequality measurement. In population A 50% are exposed, while in population B only 5% are exposed. In both populations the probability of disease is 0.10 for the exposed and 0.05 for the unexposed. The standard epidemiological analysis for either population would be to calculate the probability of being diseased given exposure relative to the probability of disease for the unexposed giving a relative risk (RR) of 2 \((0.10/0.05 = 2.0)\).

However, surely one would consider population B to have a more equal distribution of health status since 95% of people in B experience the same risk, regardless of its magnitude, compared to the even split in A. The traditional analytic approach is designed to ignore the size of the two groups because that is not relevant to causal inference. An alternative way of analyzing these data would be to rank the \(n\) subjects in order of decreasing exposure, \(i = 1, 2, 3, ..., n\), and to assign to each his or her position in the cumulative proportion, \(m = i/n\). The dichotomous variable indicating exposure would be replaced by the continuous \(m\). This approach extends easily to ordinal measures of exposure, such as most indicators of socio-economic status.

6.2 Summary measures

6.2.1 Rate difference and rate ratio

The rate difference and rate ratio are effect measures that incorporate the rates of two groups. Usually the two extreme socio-economic groups are compared. Equality is assumed to be the state in which the two extreme social groups (and one assumes those in between as well) have the same rate. The rate difference is the rate for the socially disadvantaged less that for the socially advantaged group, and the rate ratio is the rate for the socially disadvantaged group divided by the rate for the socially advantaged group.

The rates for the two extreme groups must be known; in crude form for a crude statis-
tic, and in age-adjusted form for an age-adjusted statistic. The same approach could be applied to other measures of health besides rates. The main advantage of these measures is that their data requirements are minimal, they are simple to calculate and seem simple to interpret. Confidence intervals are also simple to calculate. However, these measures fail to incorporate the information provided by all intermediate groups, and do not reflect the sizes of the extreme groups. These properties can be very problematic for a summary measure of social inequality in health, and yet this is likely the most commonly used measure.

### 6.2.2 Relative and absolute population attributable risk (PAR)

These are impact measures that assume that equality means that all groups gain the rate of the most advantaged social group. They are calculated for dichotomous exposures as

\[
P_{\text{AR or } PAR}\% = \frac{I_p - I_o}{I_o} = \frac{(I_e - I_o)P_e}{I_o} = \frac{P_e(RR - 1)I_o}{I_o} \tag{6.1}
\]

and for polychotomous exposures with \(J\) exposure categories as

\[
P_{\text{AR or } PAR}\% = \frac{\sum_{j=1}^{J} P_{ej}I_{ej} - I_o}{I_o} = \frac{(\sum_{j=1}^{J} P_{ej}RR_j - 1)I_o}{I_o} \tag{6.2}
\]
Figure 6.3: Left: area A is the population attributable risk (an absolute measure). The PAR\% (the relative measure) is equal to A divided by the mean rate. Right: the polytomous PAR measures are identical to the equivalent dichotomous measures, since the weighted sum of A1, A2 and A3 is equal to A. Starting with polytomous data adds no additional information to the PAR statistic.

where $PAR$ and $PAR\%$ are the absolute and relative statistics respectively, $I_p$ is the rate for the total population, $I_o$ is the rate for the unexposed population (i.e. the most advantaged group), $I_e$ the rate for the exposed population (i.e. everyone else), $P_e$ is the proportion of the total population that is exposed, and $RR = I_e/I_o$ is the relative rate.

The $PAR\%$ is often referred to as the population attributable fraction (PAF). In equations 6.1 and 6.2, the absolute PAR is achieved by setting $\beta$ to zero, and the relative version by setting it to one. The relative version alone can be calculated with less data as $PAR\% = \frac{(RR-1)}{RR} P_e$ for dichotomous, and $PAR\% = 1 - \frac{1}{\sum_{j=1}^{J} P_{ej} RR_j}$ for polytomous exposure categories. Regression techniques could also be use to estimate the unexposed rate for an extrapolated group, such as twenty years of education. This has been called a regression-based PAR [125, 138].

Due to the multiple methods of calculation, there are several sufficient data sets. Occasionally, the following relationships will allow one to extract first the $I_o$ then the $I_e$ when one is given only the relative risk(s) (or the excess relative risk, $RR - 1$) and $P_e$ or $P_{ej}$, and can take $I_p$ from the literature. For dichotomous exposures $I_p = (1+P_e(RR-1))I_o = P_eI_e + (1-P_e)I_o$, and for polychotomous exposures $I_p = I_o \sum_{j=1}^{J} P_{ej} RR_j = \sum_{j=1}^{J} P_{ej} I_{ej}$ [219].

This is an easily calculated and interpreted shortfall measure. It can be equivalently calculated with dichotomous and polytomous rates or relative risks. Note that the PAR calculated from polytomous rates adds no additional information. This is because the
weighted sum of the rates or relative risks for subgroups of exposed subjects is equal to the overall average for the exposed. The PAR and PAR% are not sensitive to the size of the ‘exposed’ and ‘unexposed’ groups, and information is lost by treating SES as a dichotomous variable.

6.2.3 Relative and absolute Indexes of Dissimilarity (ID)

The index of dissimilarity is a measure of the amount of health that would have to be redistributed in order to achieve equality. It is calculated as follows:

\[
ID\% = \frac{\sum_{i=1}^{n} |O - E|^{\alpha}}{2(\sum_{i=1}^{n} O)}
\]

\[
ID = \frac{\sum_{i=1}^{n} |O - E|^{\alpha}}{2N} \cdot 1000
\]

where \(O\) are the observed deaths and \(E\) the expected cases or deaths in social groups \(i\), \(N\) the total number of individuals, and where \(\alpha\) is equal to one. The expected cases or deaths are calculated by multiplying the population rate by the number of individuals (or person years) in each social group. The relative statistic \(ID\%\) is the proportion of cases or deaths that must be redistributed in order to achieve equality, while the absolute statistic \(ID\) is the number of deaths per 1000 persons that must be redistributed in order to achieve equality.

For an age-adjusted statistic the observed deaths must be calculated by the direct method (double-check this), which further necessitates age-specific death rates. This measure of heterogeneity is easily calculated, and is sensitive to group size, but is very difficult to interpret as a summary measure of inequality. Because of the treatment of SES as a nominal construct, it cannot detect null or reverse social gradients in health. A given value should therefore be accompanied by a statement of whether the association
of health and SES was positive, negative or non-significant, leading to a multidimensional summary measure of inequality that is of little practical use. This measure is, however, well suited to describing social inequalities among non-ordinal groups such as those defined by race, ethnicity, religion or geography.

6.2.4 Agresti’s alpha ($\alpha$)

Agresti’s $\alpha$ is a non-parametric generalized odds ratio for ordinal data [3]. It generalizes the standard odds ratio as calculated from a $2 \times 2$ table to $r \times c$ tables. It is calculated as the ratio of the concordant ($P_c$) and discordant ($P_d$) pairs, as follows:

$$\alpha = \frac{P_c}{P_d} = \sum_{i,j} \rho_{ij} R_{ij}^{(s)} + \sum_{i,j} \rho_{ij} R_{ij}^{(d)}$$

(6.5)

where

$$R_{ij}^{(s)} = \sum_{i'>i} \sum_{j'<j} \rho_{i'j'} + \sum_{i'<i} \sum_{j'<j} \rho_{i'j'}$$

(6.6)

$$R_{ij}^{(d)} = \sum_{i'>i} \sum_{j'<j} \rho_{i'j'} + \sum_{i'<i} \sum_{j'>j} \rho_{i'j'}$$

(6.7)

where $\rho_{ij}$ is the probability that a member is classified in row $i$ and column $j$, such that for
a two-column table, \( \alpha = \frac{\rho_{11}\rho_{22}}{\rho_{12}\rho_{21}} \).

A key disadvantage of this statistic is that it is only a relative measure of association. It also treats the health data as an ordinal input, causing some loss in the precision of measurement whenever the health data were actually measured on interval or ratio scales. The advantages are that it is sensitive to group size, detects reverse and null associations of health and SES, and those familiar with the odds ratio may feel comfortable with this measure.

### 6.2.5 Relative (RII) and slope (SII) indexes of inequality

The RII and SII were introduced by Pamuk [180]. The SII is the first-order, weighted linear regression coefficient for group rates of some health condition regressed on group ridits of socio-economic status. The intent in the use of linear regression coefficients is not to accurately model the shape of the SES-health relationship, but to create an index of inequality. Group ridits are the group midpoints of the cumulative proportion for each socio-economic group when the population is ranked by increasing socio-economic status (Bross, 1958), as shown in equation 6.12. Weighting is by group size. The relative RII is the regression coefficient (SII) divided by the mean population rate. These statistics are closely related to the CI and CIg [107].

These, along with the concentration index below, are the best measures used in the literature to date. They treat SES as an ordinal input, and so assume a monotonic association of health and SES, can detect null and reverse associations, are sensitive to the size of SES groups, and can be calculated in absolute or relative form. The main disadvantage is that the novelty of these measures make the results less accessible to those who have not used them.
FIGURE 6.5: The slope index of inequality is the OLS estimate of the slope when group rates are regressed on group ridits (the cumulative midpoints of the proportion of the population), and weighted for group size. The slope index of inequality (an absolute measure) is independent of the mean, such that if a constant is added to each group rate, the slope is unaffected. The RII (a relative measure), however, would be affected since it equals the slope divided by the mean. The SII is the difference between the rates of the least and most advantaged persons, while the RII is the ratio of the rates of the least to the most advantaged persons.
6.2.6 Alternative regression methods for RII

In addition to weighted least-squares regression of standardized rates, the RII has been estimated using Poisson and logistic regression of microdata [126, 138, 64, 62]. This approach has several advantages. It is a familiar technique, software is readily available, and multivariate models that adjust for confounding variables such as age and sex are easily specified and estimated. However, the degree to which the choice of GLM model used to estimate an RII will affect the results and interpretation has not been well described.

6.2.7 Concentration Index (CI) and Generalized CI (C$^g$I)

The concentration index is related to the well-known Gini coefficient of univariate economic inequality in that it can be derived from a Lorenz curve. In a Lorenz curve individuals are ordered along the x-axis according to some rule as a cumulative proportion, and the cumulative proportion of some distributed quantity is on the y-axis, so that an equal distribution according to the ordering of individuals would be that which produces a straight line. For the Gini coefficient, individuals are ordered according to increasing income, and the distributed quantity on the y-axis is also income. For the CI and C$^g$I, individuals are ordered according to their socio-economic status, and the distributed quantity is health status. Like the Gini, the CI is a relative measure because it is derived from a Lorenz curve with a cumulative proportion of the distributed quantity on the y-axis, while the C$^g$I is an absolute measure because it is derived from a cumulative sum of the health or health burden. Therefore the C$^g$I, unlike the CI and the Gini, is not dimensionless or bounded by -1 and 1 (0 and 1 for the Gini), and is instead bounded by $\pm$ the mean health status. These relationships are shown in figure 6.6.

Kakwani et al. [107] have described the calculation of the CI and C$^g$I. For individual data, the CI can be expressed as the integral
Figure 6.6: This graphic shows the Lorenz or concentration curve for cumulative health burden by the cumulative proportion of the population ranked by increasing socio-economic status. Because those with lower SES have greater health burden, the curve is above the diagonal, which represents a uniform distribution of burden by SES. The CI is equal to twice the area between the curve and the diagonal. This is calculated for grouped data by subtracting the area of the lower triangle from the sum of the area under the dashed step-function, and multiplying the result by 2. The rectangles intersect the curve at the group ridits. For the CI, the y-axis is a proportion, summing to 1 (shown), while for the CIg it is in the original units of the health burden, summing to the population mean (not shown).
\[ CI = 1 - 2 \int_{0}^{1} L(s) ds \] (6.8)

where \( L(s) \) is the Lorenz function of persons \( s \). This is easily calculated as

\[ CI = \frac{2}{n \cdot \mu} \sum_{i=1}^{n} x_i R_i - 1 \] (6.9)

where \( x_i \) is the health score of the \( i \)th person, where \( R_i \) is the relative rank of the \( i \)th person, and where \( \mu \), the mean level of ill-health, is equal to

\[ \mu = \frac{1}{n} \sum_{i=1}^{n} x_i. \] (6.10)

For group data, if \( L(s) \) is assumed piecewise linear, CI can be calculated as

\[ CI = \frac{2}{\mu} \sum_{t=1}^{T} f_t \mu_t R_t - 1 \] (6.11)

where \( T \) social groups are ordered according to increasing social status, \( \mu_t \) is the morbidity/mortality rate of the \( t \)th social group, \( f_t \) that group’s population share, \( \sum_{t=1}^{T} f_t \mu_t \) is the mean rate of ill-health, and the relative rank of the \( t \)th group is defined to be the group ridit, such that

\[ R_t = \sum_{\gamma=1}^{t-1} f_{\gamma} + \frac{f_t}{2}. \] (6.12)
Kakwani et al. [107] show that any transfer of health from a more socially advantaged person to a less advantaged person will have an equalizing impact on CI whenever health is positively associated with social rank, and vice versa. This satisfies a basic criterion of inequality measurement analogous to the Pigou-Dalton criterion for economic inequality, as described by Sen [207]. It can also be shown that the CI is equal to the RII multiplied by twice the variance of the relative rank variable [107]. The generalized concentration coefficient is

$$CI_g = \mu \cdot CI.$$  \hspace{1cm} (6.13)

The CI and CIg can be calculated with microdata or summary data which list the number of cases/deaths, or the mean burden or health (measured on a ratio scale, such as standardized rates) for each socio-economic stratum, along with the relative population size of each stratum.

The CI and CIg use all of the observations, are sensitive to group size, detect reverse gradients and can be calculated in relative or absolute form. The graphical derivation of the CI and CIg offers many useful opportunities to visually describe complex features of trends and comparisons in the distribution of health and health burden. The CI can only be age-standardized by the direct method with grouped data. It can be standardized by the indirect method when using individual data, by doubling the area between the observed $L(s)$ and $L^*(s)$, the concentration curve achieved when person $i$’s degree of health is replaced by the average degree of health experienced in the population by persons of the same age and gender.

The CI and CIg are true inequality measures that make optimal use of health and socio-economic data. It is not particularly sensitive to small groups of highly disadvantaged persons with particularly poor health, but for the purpose of describing such small
groups, measures of effect are likely more useful than measures of inequality, and the two could be used in combination.

6.3 Conclusions and recommendations

The choice between relative and absolute summary measures of social inequality in health has received too little attention in the literature. Relative measures are more familiar and software makes them easy to estimate, but they are better suited to causal inquiry than to measuring the extent of social inequalities in health for policy purposes.

The strong and arbitrary assumption made by relative measures is made clear if one compares indicators of health status that improve towards versus away from zero. Consider the consequence of expressing the data plotted in Figure 6.1 as the probability of surviving to sixty-four, or as conditional life expectancy at twenty years, instead of as mortality rates. Unlike mortality rates, these statistics improve away from zero. The absolute measures of inequality will tell the same general story for mortality rates and for probabilities of survival and life expectancies, but the relative measures do not. Instead of having a numerator and denominator approaching zero at equal rates, they grow greater at roughly equal rates, causing the relative measure to appear to decrease, rather than to increase.

If we are interested in relative measures, clearly our interest is contingent on the health variable in question: when large values indicate greater health relative measures have a very different meaning from when small values indicate greater health. But are we interested in relative measures at all? Relative measures are more accessible: one need not know the units of a scale, and it is easier to compare relative measures of different kinds. But these are matters of convenience, and cannot be used as arguments in favour of relative measures if the choice has important implications for the results. In economic inequality research, invariance to positive scalar transformations is the primary reason for
using relative measures. But for health status, unlike economic well-being, positive scalar transformations have no practical meaning.

We have also seen that the units of the absolute inequality scale are tacitly valued according to the mean health level if one converts the absolute measure to a relative one. This debate is common in epidemiology, where the question is often asked, ‘how important is a two-fold increase in risk when the risk for the ‘unexposed’ is extremely low?’ If we were considering inequality in mortality rates, and mortality rates were extremely low, as they are for certain causes of death and for certain age-groups, the inequality would seem to be necessarily trivial, yet a relative measure might show seemingly important inequality.

One could argue that a relative measure of life expectancy implies that there is a decreasing marginal value to life years, and a relative measure of the mortality rate implies that there is an increasing marginal value to life years. Where healthy populations are older populations (and this is not always true), the increasing or decreasing marginal value of units of health will correspond to ageism. Are the fifth through tenth years of life in some way of more or less value than the fortieth through forty-fifth, or seventieth through seventy-fifth?

This type of ageism has been discussed in the context of age weights for disability adjusted life years (DALY). There is not yet a consensus as to which years should be valued most, but (a) no propositions imply equally valued units of health at all ages, (b) not all propositions involve a strictly decreasing marginal value of life years, and (c) no propositions imply a strictly increasing marginal value of life years [234]. This suggests that (1) a relative measure of social inequality in population health where the health measure is on a scale that improves towards zero will often not be consistent with our valuations of units of health; (2) a relative measure of social inequality in population health where the health measure is on a scale that improves towards zero will typically assume an arbitrary marginal value of life years, and a valuation that is inconsistent with at least some
of the proposals in the DALY literature; and (3) absolute measures of social inequality in population health are inconsistent with our valuations of units of health in the absence of age-weights, which have not been proposed in the social inequality in health literature to date. The first point argues against the interpretation in the Acheson Report that inequalities in health were increasing in England and Wales, and also against most studies that make use of the concentration index or the relative index of inequality.

Researchers should select summary measures of social inequality in health to suit their research objectives, and they should highlight the nature of the measures they report. As a public health indicator, absolute measures of inequality, without age-weights, may be preferred because the assumptions of ageism are much more transparent (all ages treated equally), and because the value of units of health are not dependent on the health of the population. There should also be greater discussion about the of age weighting in summary measures of population health more generally.

Policy makers need to be aware that although the policy objective of reducing inequalities in health seems strait-forward, measuring progress towards that goal requires technical and normative choices that can have significant effects on whether and to what degree we perceive that progress is being made. Current practice among epidemiologists and economists involves relative summary measures that will often, though not necessarily, provide a more pessimistic view than is appropriate.

A final caveat involves conflict between seemingly equivalent measures, and the limits of precision. All extant summary measures of social inequalities in health provide orderings that are at least complete and transitive. A ranking is complete if any population can be ranked against any other, and it is transitive if when population A is more equal than B, and B is more equal than C, then A is necessarily more equal than C. Less-precise alternatives are possible. One example would be an incomplete quasi-ordering, such as that in which distribution A is superior to B only if the generalized Lorenz curve of A lies
entirely inside that of B (i.e. if A dominates B). Such quasi-orderings have been explored for univariate economic inequalities, where it has been observed that there are families of measures that satisfy any given set of axioms, thus indicating a need for either more axioms or less precision. It may be that all complete and transitive orderings are more precise than any useful concept of equality.
Part III

Summary
Chapter 7

Summary

7.1 Income and mortality

The empirical and methodological work presented in this thesis concerns the relationship between socioeconomic status and health (see Section 1.1). The empirical work focusses on the relationship between family income and premature, all-cause, adult mortality (see Sections 1.2, 1.3, 1.4 and 1.5). Three broad questions are being addressed. First, does income level determine adult mortality? Second, do income drops determine adult mortality? And third, are the effects of income level and income drops the same in Canada and the United States?

If income level has a causal effect on mortality, we should question the adequacy of our policies regarding the distribution of income and the mechanisms by which income determines health. If income drops have a causal effect on mortality, we should question the adequacy of our policies regarding labour market flexibility and income security, and the mechanisms by which income drops determine health. If there are important differences between Canada and the United States in the strength of the association between income level or income drops and mortality, then policy differences between the two countries should be explored.
The absence of a statistical association between income level or income drops and mortality in the data would not necessarily imply the absence of a causal mechanism. It may be that policies in place during this period protected working age adults against the consequences of these exposures.

7.1.1 What we know about income and mortality

We know that greater income is associated with lower mortality (see Section 1.4). This is true for men and women, throughout working life, and in Canada and the United States. Income drops may be associated with greater mortality. Longitudinal income data with mortality follow-up are rare, and only one previous study examined the association, and found that in the United States income drops appeared to have a large effect on mortality, but only at middle income levels [156]. However, income drops are typically caused by job loss and subsequent unemployment, and these exposures are better studied (see Section 1.6). During the year of job loss family income drops by 5% on average, and throughout the following 5 year period remains 10% below pre-loss income [163]. Job loss is often, but not always, associated with increased mortality. Unemployment, for which data are comparatively common, is typically associated with increased mortality. After involuntary job loss, one third of workers spend less than three months unemployed, one third spend three to nine months unemployed, and the remaining third spend more than nine months unemployed [81]. The difficulty with studies of job loss and unemployment as determinants of mortality is that those in poorer health are more likely to lose their job and less likely to be re-employed. These processes of health selection are very difficult to take into account, and may bias findings such that job loss and unemployment often appear to increase mortality, but in fact do not.
7.1.2 What we don’t know about income and mortality

Two mechanisms can account for the association of income level and mortality. In the first, income level determines health and consequent mortality. In the second, health leads to work disability and decreased income, and also to increased mortality. The second mechanism is often called health selection, or reverse causation, and is a form of confounding. The relative contributions of these two mechanisms continues to be debated in the literature.

Although the effects of socio-economic status on health have been investigated in many countries, there is little cross-jurisdictional research on the effects on socio-economic status on mortality, and none on the effect of individual or family income on mortality in Canada and the United States. This is a particularly interesting comparison given the similarity of the populations and economy and the differences in income security and health care financing.

In the United States income drops were observed to have a strong association with mortality but only at middle income levels [156]. The statistical methods used in that analysis were valid, but unusual. We do not know if the US finding statistically robust, nor do we know if the US finding is reproducible in Canada.

Furthermore, for the association of income drops and mortality to be considered a causal effect of income drops on mortality, there are elements of causal inference that should be demonstrated. None of these is necessary or sufficient for there to be a causal effect, but their observation makes a causal relationship more plausible. The elements of causal inference investigated here include the strength of association, a dose-response relationship, biologically plausible induction times, economically plausible dependency on income level, persistence of the effect when controlling for health selection, and persistence of the effect when controlling for other potential confounding variables.
7.2 Methodological considerations

7.2.1 Replication of previous findings

The single comparable study of income drops as a determinant of mortality is that by McDonough et al. [156], which found that income drops had a large effect on mortality, but only at middle income levels. The replication study documented in Chapter 4 first implemented a standard Cox regression model using the same US data as were analyzed by non-standard logistic regression by McDonough et al., and then estimated identical models using Canadian data. The goals were to determine whether the US finding depends on the statistical method, whether the effect of income level similar in Canada and the US, whether the effect of income drops similar in Canada and the US, and whether the effect of drops depend on level in Canada and the US?

The data for Canada were annual income tax data for the years 1982 through 2005, while the US data were from an annual panel survey for the years 1968 through 1997. The samples were restricted to ages 45-64 at baseline, and all models were adjusted for age, sex, and family size. Models using US data were also adjusted for race, as black versus white.

7.2.2 Evidence of causality

Evidence of causality was more rigourously pursued using only the Canadian tax data, which due to the much larger sample allow more elaborate modelling. This work is documented in Chapter 5. Income level was defined as the mean family disposable income of the previous five year period, and income drops was measured both as annual change in family disposable income, and as the difference between projected and observed family disposable income. Ages at exposure to income drop were restricted to 40-55 to exclude most early retirement events. Lags of 1-18 years between exposure and death were es-
imated using separate regressions. Deaths therefore occurred at ages 41-73. Recent immigrants and those with excessive missing data were excluded.

Health selection was investigated by excluding those receiving disability benefit income. The strength of association and the existence of a dose-response relationship were investigated using four levels of income drop: none, (0-15%], (15-50%], and (50-100%]. Separate models were estimated for various induction times since biologically plausible causal mechanisms and health selection operate on different time scales. Given that families with high income will typically have greater assets, and that these should allow consumption, an important mediating factor, to be maintained after an income drop, the dependency of the effects of income drops on income level were investigated. Confounding by factors that were suspected to determine both income drops and mortality, including family structure changes, retirement, family deaths, and self-employment was investigated by estimating models that excluded persons with these characteristics.

Estimates were produced using Cox regression. All estimates were adjusted for age. Separate models were estimated for each lag, sex, and family type combination. Models were also estimated using the full data and only the data for tax years 1992 and onward, for which transfer income amounts are better captured.

7.2.3 Summary measures of the extent of social inequalities in health

Although not applied empirically in this dissertation, methods for quantifying the extent of the unequal distribution of health by socio-economic status were also explored. Such summary measurement is required when groups are compared or trends over time are described with respect to social inequality in health. Fifteen measures are classified according to their health input, socio-economic input, relative or absolute scale, and their ability to detect reverse gradients, group size, re-distribution, and population size. The measures are divided into four families: effect measures, shortfall measures, heterogene-
it measures and inequality measures. The most common summary measures, the rate ratio and typical regression coefficient, are argued to be inappropriate for the task of describing the extent of inequality, while others are shown to be more appropriate. Important differences between absolute and relative measures are discussed, and absolute measures are recommended for describing the extent of inequality because of the undesirable ethical assumptions implicit in relative summary measures.

7.3 Main findings

7.3.1 Replication of previous findings

Previous analysis of data from the American Panel Study of Income Dynamics used an unusual application of logistic regression to these survival data. The same data and measurement decisions, when analyzed using more standard extended Cox survival regression produced very similar estimates of the risk of death associated with income level and income drops. The US finding therefore does not depend on the statistical method.

7.3.2 Canada and the United States

Income level

Equivalent models using Canadian and US data suggested that the effect of income level on risk of death is twice as great in the US as it is in Canada. While the Canadian data under-ascertain death events, and this could bias the Canadian estimate towards the null if the under-ascertainment of death events is greater among those with lower income, estimates are consistent with those from the mortality follow-up of the 1991 Census of Canada [246], suggesting that any bias due to under-ascertainment is likely to be very small.
Income drops

Although the effect of income drops was statistically significant in both Canada and the United States, and the effect of income drops depended on income level in both countries, the magnitude and pattern of these effects was different. The effect of income drops on risk of death is small in Canada and slightly protective for the poor. The effect is large in the US but limited to middle incomes.

7.3.3 Evidence of causality

Income level

Well controlled models using Canadian data found that health selection is likely only a minor pathway explaining the association of income level with risk of death. Several observations support this conclusion. First, there is little decay in the effect of income level with increasing induction time, whereas decay would be expected if health selection were a major contributing pathway. Second, the effect of income level was not greater among those with income drops, regardless of induction times. For health selection to be a dominant pathway, those with health declines must experience income drops prior to death, and so those with income drops are more likely to be on a health-determined downward income trajectory, and therefore their income level should be more strongly associated with risk of death. Third, the magnitude of the effect of income drops was not substantially reduced after excluding those receiving disability benefit income.

Income drops

Evidence for a causal effect of income drops on risk of death can be divided into three categories: evidence indicating causality, evidence indicating non-causality, and ambiguous evidence.
Evidence indicating causality  Among men in couple families, the risk of death increased monotonically with increasing magnitude of relative family income drop. The effect of income drops on risk of death was not importantly affected by excluding those with disability benefit income, suggesting that the observed risks are not strongly confounded by health selection. Nevertheless, receipt of disability benefit income is a partial proxy for work disability, and residual confounding is possible. Excluding families with changes in family structure, retirement events, deaths, or significant self-employment income did not importantly affect the estimated risk associated with income drops, suggesting that these are not important confounding factors.

Evidence indicating non-causality  Income drops were hypothesized to have greater effects on risk of death among those with less income since the greater assets of those with more income allows consumption to be maintained despite a drop in income. However, there was very little evidence of a statistical interaction between income level and income drops, suggesting either that the effect is not causal or that the effect is not mediated by consumption as hypothesized. Furthermore, there was no tendency towards statistical significance of the interaction terms in models with a greater number of deaths, indicating that the failure to observe the interaction is not likely due to inadequate statistical power.

Ambiguous evidence of causality  In well-controlled models of the effects of income drops using Canadian data, the strength of the association between income drops and risk of death is relatively small as regards causal inference. However, exposure is common, and can be repeated, such that there could be a substantial cumulative population burden if the effect is causal.

Induction times between exposure to income drops and death events are hypothesized to depend on cause of death, though data on cause of death were not available. Accidents
and suicides are expected to have short induction periods, health selection to have only slightly longer induction periods, while the initiation and progression of chronic disease deaths, which account for most deaths at these ages, are expected to have a broader distribution across longer induction times. However, the typical pattern observed was of a constant, enduring effect. This is not typical of an acute exposure, and raises the possibility that the relationship is confounded by a more enduring trait. Other possibilities include that income drops mark the onset of enduring exposures to a lowered quality of labour force experience, or that cause-specific effects show more biologically plausible induction times, but happen to sum to a constant elevation in risk as a function of induction time.

### 7.4 Discussion

These analyses and previous research lead to several conclusions. First, income level is a strong determinant of mortality, and the effect is primarily causal. Second, income level likely has a much stronger effect on mortality in the US than in Canada. Third, income drops are associated with increased mortality, but evidence for causality is mixed. And fourth, income drops have not been shown to have a similar effect in the US and Canada.

#### 7.4.1 Key strengths

The Longitudinal Administrative Databank (LAD) is a 20% simple random sample of Canadian personal income tax filers, linked longitudinally with families reconstructed. These data are unique in Canada for their generalizability, sample size, longitudinal nature, low loss to follow-up, and capture of death events. Furthermore, the LAD has detailed measures of income by component, has measures that allow important sources of confounding to be controlled, has demographic measures that allow stratified analyses, spans
several business cycles, and has sufficiently long follow-up to examine relevant induction periods.

The Panel Study of Income Dynamics (PSID) in the United States has many of the same strengths and also has active follow-up of deaths.

7.4.2 Key weaknesses

The LAD is limited to some degree due to its under-ascertainment of deaths and lack of data on cause of death. It also lacks measures of occupation, education, and health status.

The PSID is principally limited by its small sample size. The PSID was also sampled in 1968, and so pre-dates recent Hispanic immigration, which may limit generalizability.

Comparisons of findings in Canada and the United States are limited by differences between the two samples. The Canadian data include more immigrants, whose incomes are lower and more dynamic and whose mortality rates are lower due to the healthy immigrant effect. This would bias estimates of income level towards the null and estimates of income drops also towards the null. Because the Canadian data were sampled and observed more recently, there may also be a greater degree of family dynamics in the Canadian data. The models comparing Canada and the United States did not exclude families whose structure changed, and it is likely that these families have greater exposure to income drops and higher mortality, leading to non-conservative bias in the effects of income drops.
7.4.3 Policy implications

Income level

This research reproduces and confirms the graded effects of income level on hazard of death and suggests only a small role for health selection, despite the lower income and survival rates of the work disabled. Since exposure is universal, the moderate effect sizes imply a large population health burden.

The association between income level and mortality appears to be much weaker in Canada than in the United States. While the reasons for this require further investigation, this evidence is consistent with the view that comparatively generous Canadian income redistribution measures and universal, publicly-financed health care protect Canadians against health risks faced by their American neighbours.

Income drops

The effect sizes for larger income drops for men in couple families were similar to the effect of income level when comparing the middle tertile to the richest tertile. Income drops are not rare exposures, and are recurrent. While the hazard ratios are not large in terms of strength of evidence regarding causal inference, and there is a possibility of uncontrolled confounding, if the estimates of risk are causal the population burden could be substantial.

If the effects are causal, the external generalizability is questionable despite the strength of the LAD data. Exposure to income drops is highly contingent on labour policy and economic conditions, which vary over time and place. Policies that buffer the effects of income drops, such as health care financing, and direct housing and food assistance, are also highly variable between jurisdictions. It is possible that when exposure is more common the characteristics of the exposed will differ in ways that modify the effects of income
drops on hazard of death.

Economic risk pooling can take place on many scales, and is not restricted to government programs. The number of earners in a family has great potential to reduce the magnitude of family income dynamics [160, 161].

The effect of income drops on mortality also appears to be modest in Canada compared to the United States. This is consistent with there being more effective existing programs to mitigate such risks in Canada, but there are competing explanations based on uncontrolled confounding and differences in sample composition.

7.4.4 Further research

The lack of evidence for an interaction between income level and income drops is both evidence against health selection and lack of evidence that income level proxies for assets that moderate the effects of income drops. This finding should be replicated with other data, preferably with direct measures of liquid and illiquid assets.

Research on income level and hazard of death should focus on interventions. Interventions can either reduce exposure to relative differences in income level through equalization, or minimize the effects of relative differences in income level by interrupting the mechanisms that lead to health risk. Research prioritizing possible interventions based on their effectiveness, efficiency, other consequences, and feasibility in Canada is needed.

The relatively constant effects of income drops among men in couple families over time after exposure could obscure more dynamic patterns by cause of death. Alternative explanations include the possibilities that income drops are due to an enduring trait such as mental health problems, education or occupation that is associated with increased mortality, or that income drops initiate a prolonged exposure to weakened labour force attachment, leading to long-lasting excess mortality.
This study did not explicitly explore effect modification of the effect of income drops by the cause of the income drops. The goal was to isolate changes in family disposable income that were principally due to changes in market income. However, the effects of income drops due to divorce, births, retirement and other causes may confer health risks that merit direct investigation.

Higher exposure to income drops was observed in Canada. This may be due to differences in unemployment experiences, differences in identification behaviour as unemployed or non-participant, or differences between the samples in their coverage of immigrants or family dynamics. If the greater exposure in Canada is a measurement artifact, it may have biased estimates towards the null, concealing effects of income drops on mortality more similar to those in the United States. In particular, it is possible that the protective effects of income drops at low income levels observed in the LAD data were due to a healthy immigrant effect.

The US data lacked power, and the estimated effects were highly dependent on the use of survey weights, which may indicate poorly specified models. The US findings should be replicated using other American data sources.
Appendix A

Methods considered but not used
A.1 Chronic exposures and dose-response relationships

Much of our knowledge about temporal variables comes from occupational exposures. These can be difficult to interpret, however, because the exposures are always extended over time and exposure levels usually varied considerably with calendar time, so that it is essential to control for total exposure. This is often difficult to do, however, because of problems of multicollinearity... and because the levels of exposure are unknown [230].

A.1.1 Terminology of dose and exposure modelling

Classical occupational and environmental epidemiology involves the analysis of chronic exposure to substances whose concentration is measured on a continuous scale. Checkoway et al. [51, p.18-21,265-8] provide the following definitions:

- **Environmental aspects of exposure** (i.e. “outside the skin”)
  
  **Exposure**: the presence of a substance in the environment external to the worker.
  
  **Concentration**: the amount of a substance per unit of environmental medium.
  
  **Intensity**: synonym for concentration.
  
  **Exposure rate**: synonym for exposure intensity.
  
  **Duration**: length of time a given intensity is maintained.
  
  **Cumulative exposure**: time-integrated exposure (i.e. sum of exposures over time).

- **Biological aspects of exposure** (i.e. “inside the skin”)
  
  **Burden**: the amount of a substance that exists in the body or in susceptible tissues; a function of the temporal pattern of exposure and of retention.
  
  **Retention**: persistence of the substance; a function of the body’s ability to absorb, metabolize and clear the substance.
  
  **Dose**: the amount of a substance that remains at the biological target during some specified time interval; a function of burden and the chosen time interval.
  
  **Dose rate**: dose expressed as a function of time; often simplified with a summary indicator such as the average or peak for analytic purposes.
  
  **Biologically active dose**: the dose related to that portion of the burden that has pathological effects, when some portion of the burden does not.
In practice, exposure intensity is usually used as a proxy, or surrogate, for dose since data for the latter are more difficult to acquire. If exposure intensity and dose are known to have a perfect linear relationship, then there is no bias or loss of precision in estimates based on exposure intensity. If the relationship is non-linear or includes error, then various biases will be introduced to estimates of effect [51, p.21]. Rich exposure data also present many options for summary indexes, which raise difficult choices:

What are the key characteristics of the... exposure profile that may be etiologically linked with disease? This is a difficult question to answer without a strong hypothesis about the biologic mechanism by which the agent of interest may act. Therefore, the best possible data on exposure intensities may fail to be associated with risk if they are summarized inappropriately [52, p.299-300].

In social epidemiology, the “substance” is usually a social construct, and analogies to burden typically would require a model of how the exposure “gets under the skin” that is more explicit than current understanding allows. There is almost no social epidemiological work so mechanistically reductive as to describe an analogue to the retention functions of occupational epidemiology. There is also a lack of understanding regarding the adequacy of exposure intensity as a proxy for dose, and this necessitates some arbitrary modelling assumptions.

Moreover, dose-response relationships are based on the definition of the target organ, tissue or gene and the specific pathological state that arises from exposure [51, p.264]. One of the hallmarks of social epidemiological exposures is the lack of such specificity. Although the textbook reductionism of occupational epidemiology is hard to replicate, even for the textbook examples this is an idealised scenario. Silica particles, for example, have multiple pathological consequences with differing induction times, as with exposure to tobacco smoke, dioxin, and asbestos [223].
A.1.2 Income dynamics as a chronic exposure

Income dynamics are expected to have complicated properties as a health exposure:

- highly variable annual dose (or dose per other time interval)
- expected mean of zero
- cumulative effects
- peak effects
- short-run health selection bias
- variable minimum and maximum induction periods depending on outcome in question
  - partial or complete recovery from exposure over time
  - short-run psychiatric and risk-behaviour-induced mortality effects
  - long run induction and exacerbation of chronic disease

Income drops are expected to be both an acute and a chronic exposure. Acute exposures are usually modelled as individual episodes, or life-events, but chronic exposures are usually simplified based on assumptions regarding their mechanism of action using indices such as time since exposure initiation, exposure duration, average exposure intensity, cumulative exposure, or maximum exposure intensity [197, p.82-9].

Single indexes of exposure cannot fully capture realistic chronic exposure mechanisms of disease causation, and the important question becomes which indexes can be used to provide reasonably unbiased estimates of which aspects of the causal mechanism. For example, if exposure is defined only as a cumulative exposure, then one assumes that increases in average exposure intensity can be offset by decreases in duration, that the induction period is not relevant, and that there is no recovery from the earliest doses.

When the minimum induction period for a disease or event, meaning the time between exposure and outcome, is known or assumed, it is appropriate to ignore outcome data during the induction period. The cost of doing so is primarily statistical power. When
statistical power is not abundant, an approach more susceptible to bias is to count outcome data in the exposed group that occurs during the induction period as belonging to the unexposed group. In a survival model, this would amount to delaying the update of the time-varying covariate indicating exposure until after the induction period has ended. Since induction periods are typically assumed with great uncertainty, this approach is generally not recommended [197, p.84].

Unlike most epidemiological risk factors that might be used as time-dependent covariates, income dynamics are expected to have only modest autocorrelation, and so their time varying nature is not so much about “updating” a baseline exposure value to avoid “time decay” of the estimated effects [6], but discovering what the current value might be. Income dynamics are also a continuous construct in which there is no unexposed period or group. This is different from many examples of chronic exposure such as tobacco or industrial carcinogens. While it is possible to define income dynamics as a categorical exposure with individuals experiencing trivial dynamics labelled as unexposed, the dose-response relationship has not been previously explored and trivial levels cannot be defined empirically.

The most common transformations of chronic exposures are cumulative, peak and average functions. Various combinations of income dynamics definitions and these chronic exposure functions are illustrated in Figure A.1. Note that if the data are analyzed in a survival model with date of birth as the time origin, time-on-study will be a strong determinant of cumulative, peak and average transformations of income dynamics, and would need to fully controlled. This is only an issue if exposure histories are left-truncated, otherwise, this is natural. This can be viewed as a problem of missing exposure data.

It is often the case that one wishes to examine cumulative exposure but also allow for other aspects of exposure history to modify this effect in a multiple regression model. Thomas [230] defines chronic exposure indices for subjects $i$ using date of birth as the
Chapter A. Methods considered but not used

**FIGURE A.1:** Chronic exposure functions for income dynamics, illustrated with random normal income dynamics data for a single family on an arbitrary scale. These functions reflect each transformation for \( T \geq t \), as they would be defined as a time-dependent covariate in a survival model. Drops and raises are defined as negative and positive income dynamics, respectively. Peak exposure is the maximum value, except for drops when it is the minimum value. Note that cumulative, peak and average transformations are strongly determined by time-on-study. The “observed” data are in the top left.
time origin for time $t$:

\begin{align*}
\text{Exposure history} & = x_i(t) ; \\
\text{Cumulative exposure} & = z_{i1} = \int_0^T x_i(t) \, dt ; \\
\text{Age at exposure} & = \int_0^T [t - \bar{t}(z_{i1})] x_i(t) \, dt ; \\
\text{Time since exposure} & = \int_0^T [T - t - \bar{T}(z_{i1})] x_i(t) \, dt ; \\
\text{Duration of exposure} & = \int_0^T (t - \bar{t}_i)^2 x_i(t) \, dt ; \\
\text{Variability in intensity} & = \int_0^T [x_i(t) - \bar{x}_i]^2 x_i(t) \, dt .
\end{align*}

where $\bar{t}(z)$ is average age at exposure for subjects with total exposure $z$, $\bar{T}(z)$ is the average time since exposure for subjects with total exposure $z$, and $\bar{t}_i$ is the average age at exposure. Note that Equations A.3-A.6 are a product of $x_i(t)$, the exposure at age $t$ for individual $i$, and therefore represent weighted cumulative exposure indices. Since these are used as covariates with $z_{i1}$, the weights are centred to avoid collinearity.

A typical analysis might involve regressing a health outcome on $z_{i1}$, and then adjusting for some of Equations A.3-A.6 to examine modifying effects of exposure history. If the regression reveals a strong effect of time since exposure, then cumulative exposure might be weighted by a function of time $f(u) = f(T - t)$ as $\int_0^T z_i(t) f(T - t) \, dt$, where $f(u)$ could be

\begin{align*}
\text{linear model: } & f(u) = u ; \quad (A.7) \\
\text{threshold model: } & f(u) = 1 \text{ if } u > \gamma, \ 0 \text{ otherwise } ; \quad (A.8) \\
\text{saturation model: } & f(u) = u/(u + \gamma) ; \quad (A.9) \\
\text{logistic model: } & f(u) = \exp(-\gamma_0 - \gamma_1 u)^{-1} . \quad (A.10)
\end{align*}
Threshold models are common in cancer epidemiology, where $\gamma$ is often five or ten years, depending on the type of cancer. Models are usually built with the simplest and most easily interpreted index that eliminates the need to the inclusion of interaction terms.

Non-linear effects can be modelled parametrically, or explored using the residuals from linear models \cite{38, 57}.

Usually decisions regarding the treatment of chronic exposures are made based on a precisely hypothesised mechanism for a particular outcome. But with income dynamics and all-cause mortality, there are many potential mechanisms and many causes of death, and very little existing research. In this context, the specification of the exposure must be more exploratory. In order to determine which types of exposure transformations are worth exploring, each type of measure from Figure A.1 will be considered in turn.

**Untransformed income dynamics** Untransformed income dynamics represent a continuous measure on either an interval or a ratio scale. Assumptions about functional form depend on modelling decisions. Typical indexes of chronic exposure are perhaps less appropriate than measures of dispersion such as the variance, which is often used to describe income dynamics in econometric analyses.

**Most recent** Modelling the most recent untransformed income dynamic assumes that each income dynamic measurement replaces the last and that there are no residual effects of income dynamics history. This is not likely a realistic assumption.

**Cumulative** For untransformed income dynamics, for which the expected value is zero, the expected value of a cumulative sum is also zero. However, as shown in Figure A.1, some individuals will have negative or positive average income dynamics, and so the cumulative sum will show the extent to which their in-
income trajectory is trending away from the typical age-income profile. These systematic changes, while interesting, are not what is meant by the term income dynamics, which refers to transitory changes in income.

**Peak** The peak exposure of untransformed income dynamics is the largest positive value (identical to the peak value of raises), or the most unusually prosperous year, untransformed income dynamics and there is no argument in the literature justifying such a measure.

**Average** Average exposure is a practical transformation for cigarette consumption, since it is a non-negative, and not a volatile exposure. For income dynamics, which is volatile and centred on 0, an average exposure transformation will typically reveal a regression to the mean pattern as a function of time-on study.

**Absolute value of income dynamics** Using the absolute value of income dynamics makes the strong assumption that the effects of transitory income changes does not depend on their sign. It is not expected that unexpected windfalls and losses have the same effect on mortality.

**Most recent** Modelling the most recent absolute value does not address the problem of ignoring income dynamics history.

**Cumulative** While taking the absolute value solves the problem of an expected value of zero for the cumulative function, the assumption of equal effects for positive and negative income dynamics does not seem realistic.

**Peak** The peak exposure intensity of the absolute value of income dynamics is a sensible exposure index if one assumes that drops and raises are equivalent and that a family’s worst economic year is an important determinant of the future health.

**Average** While taking the absolute value solves the problem of an expected value
of zero for the average function, the assumption of equal effects for positive and negative income dynamics does not seem realistic.

**Drops** Income drops are the aspect of income dynamics of most interest to the social epidemiology community. Although there is some research on the mortality consequences of income drops, there is a great deal more on the consequences of job loss, and examining drops in income is consistent with both of these literatures.

**Most recent** Modelling the most recent drops does not address the problem of ignoring income dynamics history, but is an appropriate approach to studying short-run effects if “most recent” is defined as begin within the period of increased risk of death for such mechanisms.

**Cumulative** Cumulative drops summarizes the number and size of transitory decreases in income, and is therefore a candidate indicator of chronic exposure to income dynamics. However, the number of drops is merely a function of time-on study since there is no period of non-exposure, as there might be for cigarette consumption or for an occupational exposure.

**Peak** The peak drop intensity is the family’s worst economic year. This is likely a useful indicator of economic hardship and a predictor of any mortality sequelae. It would be best to interact peak drop intensity with time elapsed since the peak occurred, such that different induction period patterns, such as those in Figure 1.5 can be observed.

**Average** If income drops are defined with respect to group-averaged age-earnings profiles with individual intercepts for years prior to the measured drop, following Morissette and Ostrovsky [161, 160, 162], then regular drops indicate that the individual’s age-earnings profile (or household income) is rising more slowly than average. The average income drop plays a similar role to the cumulative
drops measure, except that it is not as much a function of time-on-study, and therefore analytically more tractable. Cumulative exposure is attractive when the date exposure begins is datable and measured, but this is not the case for income dynamics. Nevertheless, average income drops will have a regression-to-the-mean type pattern of early variability and later stability unless a moving “window” of exposure is used to define a time-dependent average reflecting a constant number of years.

**Raises** There is no discussion of the negative consequences of positive income change, and although increases in income are thought to have positive consequences, very little work has examined transitory increases. Lottery winnings, which have received some attention [82], are not income increases but rather wealth increases.

**Most recent** Modelling the most recent raises do not address the problem of ignoring income dynamics history.

**Cumulative** Like cumulative drops, cumulative raises document the number and size of annual income raises, but since the number is not of great interest, and would need to be controlled for, the average is likely a preferable indicator.

**Peak** The peak exposure of raises is the largest positive value (identical to the peak value of untransformed income dynamics), or the most unusually prosperous year. There is some evidence that lottery windfalls are good for mental health [82]. The induction period for any effects on longevity is unknown.

**Average** If income raises are defined with respect to group-averaged age-earnings profiles with individual intercepts for years prior to the measured raise, following Morissette and Ostrovsky [161, 160, 162], then regular raises indicate that the individual’s age-earnings profile (or household income) is rising more quickly than average.
Possible non-linear associations of income drops and disease or mortality risk are illustrated in Figure A.2. It is not known which, if any, might correspond to the overall effects of income drops for any health outcome measure. The concave-up curve suggests that small drops have little impact, but as the magnitude of the income drop increases,
risk increases very rapidly. Concave down suggests the reverse: that small income drops immediately confer substantial increased risk, and that the marginal risk associated with larger drops is decreasing. The third scenario, of a sigmoidal risk profile, typical of the dose-response associations in classical toxicology, suggests that small income drops are trivial, but that at some point the marginal increases in risk decrease due to the saturation of the relevant pathways through which damage occurs. Since it is expected that assets and credit can buffer income drops, it may be that the functional form of the dose-response curve depends on permanent income level, with greater increases in risk for small income drops among those with lower permanent income.

Doll et al. observed that when smokers quit at younger ages, their excess gradually disappears, and that those quitting before age 55 see a substantial avoidance of risk. This is spite of the bias introduced by persons quitting due to poor health [66]. This time-decay of exposure effects can be incorporated into measures of cumulative exposure. Given the state of research on income drops and mortality risk, it is premature to seek this level of sophistication in measurement of exposure.

If peak and average exposure are both used to describe exposure patterns, then the non-peak average is usually used so that these indexes are independent [53]. Peak exposures can also be defined relative to an external definition (often called absolute peak exposure) or relative to the individual's exposure history (often called relative peak exposure) [53]. For example, McDonough et al. [156] defined income drops as a decrease of 50% or more from the previous year, which is a relative peak exposure definition. In occupational and environmental epidemiology, an absolute peak exposure is usually one that exceeds an established safety threshold or regulatory limit. Such a threshold for income dynamics could be arbitrarily defined based on the distribution of drops in the sample, such as a drop in the highest quintile of relative drops, which might end up being exactly the same as McDonough et al.'s relative peak definition, but has the property of fixing the
proportion of the sample that experiences the peak exposure.

One approach to eliminating the dependency of cumulative indexes of exposure is to have a moving 5 or 10 year window in which exposure is assessed. This approach assumes that the maximum induction time is equal to the exposure window width, and so this approach is not well-suited to the exploration of long induction periods. This approach is therefore more appropriate for short-run effects.

For average exposure, arithmetic and geometric means are both common. The geometric mean is only well-defined for strictly positive data, and is always less than the arithmetic mean. It is usually recommended when the data are right-skewed, in which case the arithmetic mean is not considered an adequate measure of central tendency [216]. It is defined as

\[
GM = \left( \prod_{i=1}^{n} x_i \right)^{1/n} = \exp \left( \frac{1}{n} \sum_{i=1}^{n} \ln x_i \right). \tag{A.11, A.12}
\]

The choice between the arithmetic and geometric mean will have a bearing on both the ordering of individuals with respect to exposure and also on the functional form of the association with the outcome. While the positive skew of income dynamics suggests a geometric mean, an arithmetic mean gives “extra weight” to larger income drops, which is consistent with the expectation that risk increases roughly exponentially with the magnitude of drops. Similarly, the expected functional form of income drops (expressed on a positive scale) is linear and increasing on a multiplicative risk scale, or perhaps concave up, and a geometric transformation of income dynamics would not contribute to linearity of the association or to interpretability.

There is not a need to choose a single index of exposure, since they may summarize different aspects of the exposure profile or represent different mechanisms. The more
relevant indices will improve model fit, but will not necessarily have the strongest effect sizes [199, 135].

To summarise, desirable indexes of income dynamics exposure include:

**Income dynamics** Variance of untransformed income dynamics during a fixed interval, as a categorical variable to allow non-linear associations with mortality risk;

**Largest income drop** Peak income drop during a fixed interval, as a categorical variable to allow non-linear associations with mortality risk;

**Cumulative income drops** Sum of income drops during a fixed interval, as a categorical variable to allow non-linear associations with mortality risk;

**Time since large income drops** As a categorical variable and an interaction with large income drops, indicating the time-dependency of the effect; and

**Atypical trajectories** Non-peak average income drops during a fixed interval.

When using multiple indexes of exposure, collinearity, in particular between average and cumulative measures, can be a problem [53]. Models with each separately and both should reveal whether they are too highly correlated, and if so, which is the stronger predictor.
A.2 Strutural nested failure time models

A time-dependent covariate can be affected by, and also affect, the time-dependent exposure variable, and so be both an intermediary and a confounding variable. This is sometimes called time-dependent confounding. Standard regression methods will give biased estimates of effects in this case [94, p.422]. To adjust for time-dependent confounding, Greenland [94, p.424-5] recommends the semi-parametric structural equation models and G-estimation approach to estimating causal effects proposed by Robins [192, 133]. These models can be fit using general-purpose statistical packages, including SAS [247]. However, the limitations in the measurement of health status discussed in Section 2.2.3 preclude the use of this method.

A.3 Modification of latency by dose

The modification of latency by dose can be assessed by plotting $RR(t|z)$ or $RD(t|z)$ for subgroups characterized by exposure level. Plots of $[RR(t) − 1]/\bar{z}(t)$ or $[RD(t) − 1]/\bar{z}(t)$ can make this analysis clearer by removing the vertical separation of the curves and thus making differences in shape more easily compared [230].

A.4 Exposure history as effect modification

Thomas [230] describes how aspects of exposure history such as age at exposure, exposure duration, time since exposure and variability of exposure can be treated as effect modifiers, where the effect is a simple measure of cumulative exposure.

Effect modification is modelled using statistical interaction. For example, given a continuous dependent variable $y$, and dichotomous exposure $x_1$ and effect modifier $x_2$, the
simplest and most common regression treatment is the model

\[
\hat{y} = \hat{\beta}_0 + \hat{\beta}_1 x_1 + \hat{\beta}_2 x_2 + \hat{\beta}_3 x_1 x_2
\]  
(A.13)

which describes two linear relationships simultaneously, each having a separate intercept and slope

\[
E(y|x_1, x_2 = 0) = \hat{\beta}_0 + \hat{\beta}_1 x_1,
\]
(A.14)
\[
E(y|x_1, x_2 = 1) = (\hat{\beta}_0 + \hat{\beta}_2) + (\hat{\beta}_1 + \hat{\beta}_3) x_1.
\]
(A.15)

In most cases the so-called hierarchy principle is followed, meaning that the main effects of variables involved in an interaction are included in the model. Omitting the main effect of \(x_2\) assumes that \(\beta_2 = 0\), meaning that the two linear relationships share the same intercept, or equivalently that the predicted value among the unexposed is not conditional on the effect modifier. In many cases omitting the main effect leads to absurd models.

When an effect modifier is defined only for certain exposure groups, then it is common to not include its main effect term in the model [230, 94]. For example, if age-at-first-birth (AFB) is undefined for nulliparous (P=0) women, then P and P*AFB might be regressors, with AFB not included as a main effect [94].
Appendix B

Supplementary results for Chapter 4
**FIGURE B.1**: Income drop effects in Canada and US. Odds ratios from McDonough et al. on the PSID are compared to analytically similar hazard ratios for both the PSID and the LAD.
Appendix C

Supplementary results for Chapter 5
**FIGURE C.1**: Estimated quadratic age-income profiles for men in couple families by income tertile, 5 year cohort, and 5 year period.
**Figure C.2:** Estimated standard error (SE) for quadratic age-income profiles for men in couple families by income tertile, 5 year cohort, and 5 year period.
Distribution of income drops (no exclusions; bin width = 0.05; right skew pooled at 2+)

Relative income drop = (observed − expected)/expected

Person-years of exposure
500000
1000000
1500000
2000000
−1.0 −0.5 0.0 0.5 1.0 1.5 2.0

Type
Annual
Projected

**Figure C.3:** Distribution of relative income drops based on annual change and deviance from projected income. No exclusions applied. Right skew pooled at relative increases of 2 times.
Chapter C. Supplementary results for Chapter 5

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Elches, J.

Figure C.4: Cohort dynamics by age, family type and lag between exposure and outcome. The categories survivor, death, died previously, lost to follow-up, and structural right censoring are mutually exclusive and comprehensive. Data for models using only exposure data from 1992 or after, and therefore having their first person-year of exposure in 1997, are also described. No exclusions applied.
**Figure C.5:** Incidence of exposure by income level, sex, family type, and lag between exposure and outcome. Incidence of drops is not a function of the lag between exposure and outcome. Data for models using only exposure data from 1992 or after, and therefore having their first person-year of exposure in 1997, are also described. No exclusions applied.
1987 or later, and therefore having their first person-year of exposure in 1997, are also described. No exclusions applied.

Figure C.6: Conditional probabilities of death by covariate pattern, not adjusted for age. Data for models using only exposure data from 1992 or after, and therefore having their first person-year of exposure in 1997, are also described. No exclusions applied.
There was good agreement between the likelihood ratio, score and Wald global significance tests for all estimated models (see Figure C.7). However, there were interesting differences between sex, family types, and lags.
Figure C.7: Global significance tests for survival models. Separate models were estimated for each combination of sex, family type and lag.

TPHREG Global significance by sex and family type (interaction models; all exclusions)

Years between exposure and outcome

p-value for Chi-squared test (log scale)
In terms of fit statistics [201, p.140], when the -2 log-likelihood ratio (-2LL) difference between the model without covariates and with covariates is penalized by twice the number of parameters (Akaike’s Information Criterion, AIC) or by the log of the number of events times the number of parameters (Schwartz’s Bayesian Criterion, SBC; also known as the Bayesian Information Criterion, BIC), models for men in couple families show good fit (see Figure C.8). Compared to the model with all exclusions, the model with the work-disabled included shows better fit, suggesting strong health selection, most notably among men in couple families. Women in couple families show poor fit at all lags on the SBC but adequate fit according to the AIC. For unattached men and women, the SBC shows poor fit, and the AIC is consistent with the global significance tests described above.
Figure C.8: Fit statistics for survival models. Separate models were estimated for each combination of sex, family type, lag and exclusion set.

Fit statistic (without covariates – with covariates; log scale)

-2LL
AIC
SBC

Type
Projected
Annual

-2LL
AIC
SBC

Fit statistics for PROC TPHREG models

- work disabled
- retired
- family changes
+ self-employed
+ all

Years between exposure and outcome

Couple families
Male
Female
Figure C.9: Effect of income drops in additive models for women in couple families. Separate models were estimated for each combination of sex, family type, lag and exclusion set. Estimates are shown for fully-restricted models only.
**Figure C.10:** Effect of income drops in additive models for unattached women. Separate models were estimated for each combination of sex, family type, lag and exclusion set. Estimates are shown for fully-restricted models only.
**Figure C.11:** Effect of income drops in additive models for unattached men. Separate models were estimated for each combination of sex, family type, lag and exclusion set. Estimates are shown for fully-restricted models only.
While the effect sizes are small, recall that these are single instances of income drop, and not a cumulative index of exposure to household financial uncertainty or crisis.
**Figure C.12:** Type 3 statistics for the interaction of income level and income drops in survival models. Separate models were estimated for each combination of sex, family type and lag.
**Figure C.13:** Type 3 statistics for the interaction of income level and income drops in survival models. Separate models were estimated for each combination of sex, family type and lag. P-values are plotted against the number of deaths in each model, and the lack of a trend towards statistical significance with increasing number of deaths indicates that the lack of evidence for interaction is not due to a lack of statistical power. The trend lines are quartiles of the p-value distribution from a simple quantile regression.
Figure C.14: Effect on estimates of the effect of income level of including subjects with each confounding characteristic, and of including all subjects.
**Figure C.15:** Effect on estimates of the effect of income drops of including subjects with each confounding characteristic, and of including all subjects.
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