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Chronic Diseases and Injuries in Canada
Public Health Agency of Canada
785 Carling Avenue
Address Locator 6806B
Ottawa, Ontario K1A 0K9

Fax: (613) 941-2057
E-mail: cdic-mcbc@phac-aspc.gc.ca

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Influence of viewing professional ice hockey on youth hockey injuries

G. Keays, MSc (1); B. Pless, MD (2)

This article has been peer reviewed.

Abstract

Introduction: Most televised National Hockey League (NHL) games include violent body checks, illegal hits and fights. We postulated that minor league players imitated these behaviours and that not seeing these games would reduce the rate of injuries among younger hockey players.

Methods: Using a quasi-experimental design, we compared 7 years of televised NHL matches (2002–2009) with the year of the NHL lock-out (2004/2005). Data from the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP) were used to identify the injuries and to ascertain whether they were due to intentional contact and illegal acts including fights.

Results: We found no significant differences in the proportions of all injuries and those involving intentional contact, violations or illegal acts among male minor league hockey players during the year when professional players were locked out and the years before and after the lock-out.

Conclusion: We concluded that not seeing televised NHL violence may not reduce injuries, although a possible effect may have been obscured because there was a striking increase in attendance at equally violent minor league games during the lock-out.

Keywords: *adolescent, males, television viewing, violence, sports injuries, hockey*

Introduction

''Sure you try what they do. You see them do all sorts of things and get away with it.'' So said a 12-year-old hockey player being interviewed on Canadian television following the blind-side hit that concussed National Hockey League (NHL) star, Sidney Crosby, removing him from play for nearly eleven months. Recent deaths of NHL enforcers—players whose main role is to fight—have fuelled the debate regarding ice hockey violence.

The influence of the media on the behaviour of viewers has been the subject of controversy since the 1950s.[1–3] In particular, disagreement remains about whether viewing violence on TV has a negative effect on children. In 1975, Rothenberg was convinced by 146 studies ''that violence viewing produces increased aggressive behaviour in the young.''[4] More recent reports, however, including systematic reviews and meta-analyses, have reached varying conclusions ranging from no effect[5] to clearly harmful.[6–11] Nevertheless, the American Psychological Association[12] and the American Academy of Pediatrics[13] assert that the bulk of the evidence points to negative effects.

Although most televised violence seen by children is presented in cartoons or action dramas, it is also evident in many sports broadcasts. Ice hockey, in particular, has a reputation for combining skilful play with aggression. It has the highest rate of sport injuries for boys[14] and is second only to football as a cause of catastrophic spinal injuries.[15] The amount of violence typically found on hockey broadcasts is striking: about 40% of NHL games include at least one fight[16] and about 16% of all severe injuries (e.g. those that force a player to leave the game) are caused by behaviours resulting in a penalty or suspension.[17] Minor professional hockey leagues, viewed by many as the most violent in hockey, generally have three to four fights per game.[18] Checking from behind—an action usually associated with severe injuries—only became illegal in 2000,[19] and there is still controversy about what to do about deliberate hits aimed at the head (''head shots'').[20] The macho aspect of professional hockey delayed the introduction of helmets until 1979[21] and continues to delay compulsory visor use.[22] In minor hockey, both have been obligatory for many years.

The behaviour of children and youth playing in minor leagues seems to be influenced by their watching televised NHL games.[23–27] A survey showed that 90% reported having learned a ''behaviour, technique or skill'' from watching professional hockey players. In addition, 56% stated they had copied illegal tactics of professional players at least once during the current hockey season.[28] Another survey indicated that high school hockey players who chose aggressive NHL players as role models were more likely to assault others during games.[29] More recently, a

Author references:

1. McGill University Health Centre, Montreal Children's Hospital, Montréal, Quebec, Canada
2. Department of Pediatrics, Epidemiology and Biostatistics, McGill University, Montréal, Quebec, Canada

Correspondence: Glenn Keays, McGill Health Centre, Montreal Children's Hospital, 2300 Tupper, Room CB-27, Montréal, QC H3H 1P3; Tel.: 514-412-4400 ext 2316; Fax: 514-412-4477; Email: glenn@keays.ca

report commissioned by the ministry of sports in British Columbia noted that 27% of the 144 young hockey players surveyed imitated illegal hits they had seen after watching NHL players.[30]

Accordingly, we concluded there was a reasonable basis for postulating that *not* watching professional hockey on TV would improve the behaviour of younger players such that there would be fewer injuries. To examine this hypothesis, we took advantage of a natural experiment: during the winter of 2004/2005, owners locked out NHL players during a contract dispute. As a result, except for replays of old NHL games in April 2005 and junior league championship games at the end of May, there was no hockey on Canadian television. We investigated whether the absence of televised professional hockey during this season was associated with a lower rate of injuries among minor league players.

Methods

Our study was restricted to boys playing organized hockey in formal minor leagues in Canada throughout seven successive seasons beginning in 2002/2003. Minor leagues are categorized as peewees, bantams, or midgets according to the age of the players.[31]

We considered only those injuries that occurred during the regular NHL season. The Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP)[32,33] provided details concerning the injuries. CHIRPP is an injury surveillance system situated in 14 emergency departments in seven provinces. It gathers information from parents of patients (or older patients) regarding the circumstances of the injury and includes medical details such as the nature of the injury, the body part and the treatment.

We used several definitions to describe the cause or mechanism of the injury. Initially, we compared all injuries to ''contact-related injuries,'' which include all types of contact, intentional or not. Then, we analysed two specific types of contacts. The first, ''injuries due to illegal contact,'' refers to those cases caused by an illegal hit (or act), as defined by Hockey Canada:[31] elbowing (extending elbow in a manner to cause injury), cross-checking (using the shaft of the stick to forcefully check an opponent), checking from behind, boarding (checking a defenceless opponent so as to cause him to impact the boards violently), checking to the head, kneeing (leading with the knee to make contact with the opponent), slashing (any forceful or powerful chops with the stick on an opponent's body), tripping (placing the stick, knee, foot, arm, hand or elbow in way that causes the opponent to trip or fall), roughing, or any acts of violence such as fights, altercations and deliberate punches. The second category, ''injuries due to fights,'' includes all injuries resulting from fights, altercations and deliberate punches.

To calculate rates, we obtained from Hockey Canada, for each year of study, the numbers of boys aged 11 to 17 years registered in each of the minor hockey leagues and expressed the proportion as numbers of injuries per 1000 registered male players in this age group in all the cities with pediatric CHIRPP centres. Confidence intervals for individual rates and individual proportions were calculated using the Poisson test.

Results

From September to April in the years 2002 to 2009, CHIRPP reported 14 717 hockey injuries for 11- to 17-year-old boys. Of the injured, 24% were peewees (11- to 12-year-olds), 39% were bantam (13- to 14-year-olds) and 37% were midgets (15- to 17-year-olds). During most years, at each level, about 70% of the injuries were contact related. For all age levels combined, the rates per 1000 registered players varied from 19.0 to 24.9 for any injury and from 13.7 to 18.4 for those judged to be contact related (Table 1). The data do not reveal, however, any pattern or trend over time nor any evidence that the proportion of injuries changed markedly when the lock-out year is compared with the preceding or following years. The same is true when these data are examined for each league or age group.

Although not statistically significant, Table 2 shows a consistent pattern indicating slightly more injuries arising from acts that were judged to be dangerous, that is, intentional or illegal, during the lock-out year.

Figure 1 shows attendance records at minor professional league games before, during and after the lock-out. We reasoned that, deprived of NHL games on TV, avid fans would compensate by attending these games, some of which were televised. The figure clearly shows that there was a peak in attendance at these games during the lock-out; what the figure does not reveal is that many contend that spectators attend these games in part because of their violence.[34,35] Players and coaches of these teams accept that the ''goon'' (who play hockey with an emphasis on intimidation and violence) is part of the games' appeal.[36,37]

Discussion

Professional hockey is violent because it relies on aggressive play. In *Violence and Sport*, Smith[28] defines aggression as ''any behaviour designed to injure another person, psychologically or physically.'' It is physical violence that typifies much of professional hockey. Robidoux and Trudel[38] observe that ''body-checking is an example of the regulated use of physical force to gain an advantage … it clearly leads to an increase in injuries.'' Several previous studies suggest that observing the behaviour of professionals during televised hockey matches influences young hockey players.[25,28–30,39,40] Contrary to what we expected, however, we found no consistent difference between rates of injuries of all kinds when youngsters were *not* watching NHL games on TV versus seasons when they were. Nonetheless, the belief that young players imitate viewing violence on TV remains plausible and prompted us to search for an explanation.

One explanation is that the behaviours related to youth hockey injuries are so deeply ingrained that they are not likely to change after only one year during which they were not reinforced by viewing the actions of professional players. A second possible explanation is that, by way of compensation, during the lock-out junior players attended more minor professional

TABLE 1
Approximate rates[a] of all hockey injuries and contact-related injuries by league (age group) and season per 1000 minor league players (11–17 years), all CHIRPP centres, Canada

Hockey season	Registered players,	All injuries			Contact-related injuries		
	n	n	Rates/1000 (95% CI)		n	Rates/1000 (95% CI)	
PEEWEES (11–12 years)							
2002/2003	32561	596	18.3	(16.9–19.8)	440	13.5	(12.3–14.8)
2003/2004	34541	508	14.7	(13.5–16.0)	356	10.3	(9.3–11.4)
2004/2005	**32339**	**492**	**15.2**	**(13.9–16.6)**	**362**	**11.2**	**(10.1–12.4)**
2005/2006	35492	449	12.7	(11.5–13.9)	322	9.1	(8.1–10.1)
2006/2007	33526	482	14.4	(13.1–15.7)	356	10.6	(9.6–11.8)
2007/2008	32235	525	16.3	(14.9–17.7)	392	12.2	(11.0–13.4)
2008/2009	34354	523	15.2	(14.0–16.6)	378	11.0	(9.9–12.2)
BANTAMS (13–14 years)							
2002/2003	30116	939	31.2	(29.2–33.2)	682	22.6	(21.0–24.4)
2003/2004	30448	861	28.3	(26.4–30.2)	624	20.5	(18.9–22.2)
2004/2005	**30848**	**833**	**27.0**	**(25.2–28.9)**	**604**	**19.6**	**(18.1–21.2)**
2005/2006	33332	761	22.8	(21.3–24.5)	558	16.7	(15.4–18.2)
2006/2007	31249	731	23.4	(21.7–25.1)	535	17.1	(15.7–18.6)
2007/2008	30049	754	25.1	(23.4–26.9)	558	18.6	(17.1–20.2)
2008/2009	32978	854	25.9	(24.2–27.7)	619	18.8	(17.3–20.3)
MIDGETS (15–17 years)							
2002/2003	28023	721	25.7	(23.9–27.7)	544	19.4	(17.8–21.1)
2003/2004	28152	837	29.7	(27.8–31.8)	614	21.8	(20.1–23.6)
2004/2005	**28597**	**738**	**25.8**	**(24.0–27.7)**	**562**	**19.7**	**(18.1–21.3)**
2005/2006	32615	715	21.9	(20.4–23.6)	510	15.6	(14.3–17.0)
2006/2007	32070	813	25.4	(23.7–27.1)	577	18.0	(16.6–19.5)
2007/2008	29963	777	25.9	(24.2–27.8)	570	19.0	(17.5–20.6)
2008/2009	34970	808	23.1	(21.6–24.7)	601	17.2	(15.9–18.6)
ALL PLAYERS (11–17 years)							
2002/2003	90700	2256	24.9	(23.9–25.9)	1666	18.4	(17.5–19.3)
2003/2004	93141	2206	23.7	(22.7–24.7)	1594	17.1	(16.3–18.0)
2004/2005	**91784**	**2063**	**22.5**	**(21.6–23.5)**	**1528**	**16.6**	**(15.9–17.5)**
2005/2006	101438	1925	19.0	(18.2–19.9)	1390	13.7	(13.0–14.5)
2006/2007	96844	2026	20.9	(20.1–21.9)	1468	15.2	(14.4–16.0)
2007/2008	92248	2056	22.3	(21.4–23.3)	1520	16.5	(15.7–17.4)
2008/2009	102302	2185	21.4	(20.5–22.3)	1598	15.6	(14.9–16.4)

Sources: Canadian Hospitals Injury Reporting and Prevention Program[32]; Hockey Canada (http://www.hockeycanada.ca/index.php/ci_id/23952/la_id/1.htm).

Abbreviations: CHIRPP, Canadian Hospitals Injury Reporting and Prevention Program; NHL, National Hockey League.

Notes: 2004/2005 (bolded) was the year when owners locked out NHL players during a contract dispute. As a result, except for replays of old NHL games in April 2005 and junior league championship games at the end of May, there was no hockey on Canadian television.

[a] Injuries treated in children's hospital emergency departments do not necessarily parallel the denominator data of registered players. Thus, the rates we used are not "true" rates in that the numerators and denominators are from different populations.

league games. Paradoxically perhaps, these are widely regarded as even more violent than NHL games,[34–37] and it is noteworthy, as Figure 1 shows, that there was a striking increase in attendance at these games during the lock-out.[41,42] Thus, exposure to violence may have remained much the same for the entire period of the study.

TABLE 2
Proportions of injuries due to illegal acts and fights during organized hockey, by minor hockey league and year, 2002/2003 to 2008/2009

Hockey season	All injuries,	Injuries due to illegal acts[a]		Injuries due to fights[b]	
	n	%	(95% CI)	%	(95% CI)
PEEWEES (11–12 years)					
2002/2003	596	22.5	(18.1–26.9)	0.5	(0.0–1.3)
2003/2004	508	16.7	(12.5– 21.0)	0.2	(0.0–0.8)
2004/2005	**492**	**27.4**	**(22.3–32.7)**	**1.2**	**(0.0–2.5)**
2005/2006	449	25.4	(20.1–30.7)	0.4	(0.0–1.3)
2006/2007	482	21.8	(17.0–26.7)	0.2	(0.0–0.8)
2007/2008	525	26.5	(21.6–31.5)	0.6	(0.0–1.5)
2008/2009	523	22.9	(18.3–27.7)	0.2	(0.4–0.7)
BANTAMS (13–14 years)					
2002/2003	939	17.1	(14.0–20.4)	0.2	(0.0–0.7)
2003/2004	861	13.8	(10.8–16.9)	0.6	(0.0–1.3)
2004/2005	**833**	**18.7**	**(15.3–22.3)**	**1.0**	**(0.1–1.9)**
2005/2006	761	18.3	(14.7–21.9)	0.4	(0.0–1.0)
2006/2007	731	18.5	(14.8–22.2)	0.8	(0.0–1.7)
2007/2008	754	16.4	(13.0–20.0)	0.9	(0.1–1.9)
2008/2009	854	17.6	(14.3–21.0)	0.5	(0.0–1.1)
MDGETS (15–17 years)					
2002/2003	721	17.2	(13.6–20.9)	1.9	(0.7–3.3)
2003/2004	837	19.5	(16.0–23.1)	1.9	(0.7–3.2)
2004/2005	**738**	**23.2**	**(19.2–27.2)**	**2.7**	**(1.2–4.3)**
2005/2006	715	19.3	(15.5–23.2)	1.3	(0.2–2.4)
2006/2007	813	17.2	(13.9–20.7)	1.6	(0.5–2.8)
2007/2008	777	19.9	(16.3–23.7)	2.1	(0.8–3.4)
2008/2009	808	19.3	(15.8–22.9)	1.6	(0.5–2.8)
ALL PLAYERS (11–17 years)					
2002/2003	2256	18.6	(16.5–20.7)	0.8	(0.4–1.4)
2003/2004	2206	16.6	(14.6–18.7)	1.0	(0.5–1.6)
2004/2005	**2063**	**22.4**	**(20.1–24.8)**	**1.6**	**(1.0–2.4)**
2005/2006	1925	20.3	(18.0–22.7)	0.7	(0.3–1.3)
2006/2007	2026	18.8	(16.6–21.0)	1.0	(0.5–1.6)
2007/2008	2056	20.3	(18.1–22.7)	1.3	(0.7–1.9)
2008/2009	2185	19.5	(17.4–21.7)	0.8	(0.4–1.4)

Source: Canadian Hospitals Injury Reporting and Prevention Program[32]; Hockey Canada (http://www.hockeycanada.ca/index.php/ci_id/23952/la_id/1.htm)

Abbreviation: NHL, National Hockey League.

Notes: 2004/2005 (bolded) was the year when owners locked out NHL players during a contract dispute. As a result, except for replays of old NHL games in April 2005 and junior league championship games at the end of May, there was no hockey on Canadian television.

[a] Illegal acts: hooking, tripping, holding, cross-checking, checking from the back, slashing, elbowing, boarding, checking to the head, kneeing, slashing, roughing.

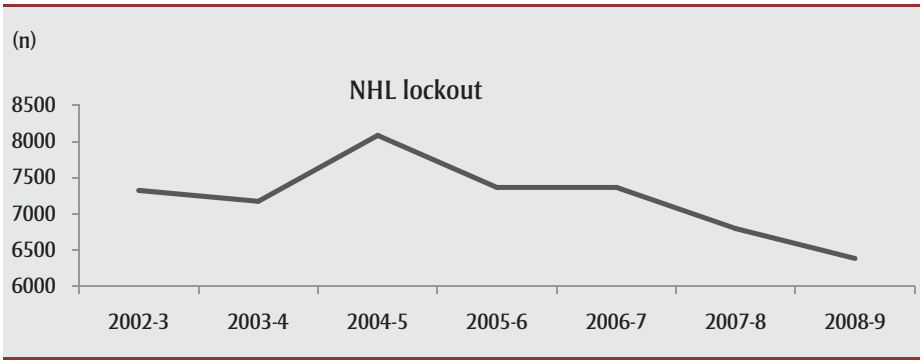
[b] Fights and altercations.

Limitations

We acknowledge several limitations. First, CHIRPP data only include a portion of all injuries across Canada, which cannot be regarded as a genuine sample of these injuries. The injuries treated in children's hospital emergency departments do not necessarily parallel the denominator data of registered players. Thus, we accept that the rates we used are not true rates in that the numerators and denominators are from somewhat different populations. However, it is the relative comparisons that we were examining and there is no reason to believe that the relationship changed over the study period.

A second limitation is that there is often insufficient detail in CHIRPP reports to be certain whether an injury was caused by

FIGURE 1
Attendance records from two minor professional hockey leagues (North American Hockey League and the American Hockey League) between the 2002/2003 and 2008/2009 hockey seasons



Sources: www.theahl.com, www. lnah.com.

Abbreviation: NHL, National Hockey League.

an aggressive or illegal act, and there are missing data. However, all records are coded centrally by trained coders and the information regarding the nature of injury and level of treatment is generally consistent over time. Again, unless there is reason to assume a change in these variables over time, our comparisons are justified.

Third, we did not attempt to verify that all our subjects actually watched televised NHL games between 2002 and 2008. However, the Canadian Broadcasting Corporation (CBC) recently announced *Hockey Night in Canada* as its highest rated show, estimating that 78% of Canadians aged 25 to 54 years watch NHL games.[43] If we apply the same proportion to our target group of 11- to 17-year-old male adolescents living in Canada and note that NHL hockey games were not only broadcast by the CBC, we can comfortably assume that there are at least one million boys of that age watching the NHL regularly. Moreover, given the extent to which ice hockey is part of Canadian culture, it would be surprising if most games involving home teams were not also watched. In addition, we believe it reasonable to assume that, except for the lock-out season where there was nothing to watch, the proportion of young spectators remained the same over the study years.

Finally, although we cannot be certain that young hockey players were part of the increase in attendance in minor professional leagues during the lock-out, it seems reasonable to assume that they were. Although attendance went up significantly, even if this included children and adolescents it would not come close to the number of children and adolescents who watch televised hockey.

Although not statistically significant based on Jonckheere trend test ($p = .099$), it is worth noting that the data in Table 1 suggest a small decline in these injuries over time. If true, this development may represent the success of various preventive initiatives or a decreased propensity to go to emergency departments when an injury occurs.

Conclusion

In spite of a reasonable hypothesis, we failed to demonstrate that not viewing the violence that typifies so much of professional hockey has a beneficial effect on the behaviour of young players. Specifically, we found no significant differences in the rates of injuries during one year when professional players were locked out and there were no televised hockey broadcasts. However, the effect may have been partly obscured by compensatory viewing of even more violent junior league games.

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References

1. Maccoby EE. Television: its impact on school children. Public Opin Quart. 1951;15(3):421–4.
2. Bandura A, Walters RH. Social learning and personality development. New York: Holt, Rinchast and Winston; 1963.
3. Berkowitz L. Aggression: a social psychological analysis. New York: McGraw-Hill; 1962.
4. Rothenberg MB. Effect of television violence on children and youth. JAMA. 1975;234(10):1043–6.
5. Ferguson CJ. Media violence: miscast causality. 2002. Am Psychol. 2002;57(6–7):446–7.
6. Ybarra ML. Linkages between depressive symptomatology and Internet harassment among young regular Internet users. Cyberpsychol Behav. 2004;7(2):247–57.
7. Villani S. Impact of media on children and adolescents: a 10-year review of the research. J Am Acad Child Psy. 2001;40(4):392–401.
8. Bar-on ME. The effects of television on child health: implications and recommendations. Arch Dis Child. 2000;83(4):289.
9. Browne KD, Hamilton-Giachritsis C. The influence of violent media on children and adolescents: a public-health approach. Lancet. 2005;365(9460):702–10.
10. Hopf WH, Huber GL, Weiss RH. Media violence and youth violence: a 2-year longitudinal study. J Media Psychol. 2008;20(3):79–96.
11. Huesmann LR, Taylor LD. The role of media violence in violent behavior. Annu Rev Publ Health. 2006;27:393–415.
12. McIntyre JJ; American Psychological Association. On the impact of media violence on children [Internet]. Testimony before the United States Senate Committee on Commerce, Science, and Transportation. 2007 [cited 2011 Sep 7]. Available from: http://www.apa.org/about/gr/pi/advocacy/2007/mcintyre-media.aspx

13. Council on Communications and Media. From the American Academy of Pediatrics: policy statement--media violence. Pediatrics. 2009;124(5):1495–503.

14. Benson BW, Meeuwisse WH. Ice hockey injuries. Med Sport Sci. 2005;49:86–119.

15. Boden BP, Jarvis CG. Spinal injuries in sports. Neurol Clin. 2008;26(1):63–78.

16. Singer, DM. Hockey Fights [Internet]. [cited 2011 Sep 7]. Available from: http://www.hockeyfights.com

17. TSN. NHL injuries [Internet]. [cited 2011 Sep 7]. Available from: http://www.tsn.ca/nhl/injuries/

18. NHL fights [Internet]. [cited 2011 Sep 7]. Available from: http://dropyourgloves.com/Fights/LeagueFights.aspx?League = 1

19. IIHF. IIHFederation Rule Book, 2010–2014 [Internet]. Zurich (CH): The International Ice Hockey Federation; 2010 [cited 2011 Sep 7]. Available from: http://www.iihf.com/iihf-home/sport/iihf-rule-book.html

20. Associated Press. NHL GMs form committee to study headshots [Internet]. Ottawa (ON): The National Hockey League; 2009 Nov 11 [cited 2011 Sep 7]. Available from: http://www.nhl.com/ice/news.htm?id = 505769

21. Duhatsche E. Should visors be mandatory in the NHL? [Internet]. Toronto (ON): The Globe and Mail; 2011 Nov 22 [cited 2012 Jan 7]; Available from: http://m.theglobeandmail.com/sports/hockey/globe-on-hockey/should-visors-be-mandatory-in-the-nhl/article1951461/?service = mobile

22. Anderson D. Sports of the times; the great visor debate in hockey [Internet]. New York: The New York Times; 2000 Mar 22 [cited 2012 Jan 7]; Available from: http://www.nytimes.com/2000/03/22/sports/sports-of-the-times-the-great-visor-debate-in-hockey.html

23. Cusimano MD. Canadian minor hockey participants' knowledge about concussion. Can J Neurol Sci. 2009;36(3):315–20.

24. Gee CJ, Leith LM. Aggressive behavior in professional ice hockey: a cross-cultural comparison of North American and European born NHL players. Psychol Sport Exerc. 2007;8(4):567–83.

25. Nash JE, Lerner E. Learning from the pros: violence in youth hockey. Youth Soc. 1981;13(2):229–44.

26. Kerr JH. Book review: Aggression in the sports world: a social psychological perspective. Int J Sport Manage Market. 2009;5(4):477–8.

27. Smith MD. Violence and injuries in ice hockey. Clin J Sport Med. 1991;1(2):104–9.

28. Smith MD. Violence and sport. Toronto (ON): Butterworths; 1983.

29. Smith MD. Significant others' influence on the assaultive behavior of young hockey players. Int Rev Sociol Sport. 1974;9(3):45.

30. Pascall B, White S. Eliminating violence in hockey. Vancouver (BC): Ministry of Small Business, Tourism and Culture; 2000 May.

31. Hockey Canada. Articles, by-laws, regulations, history as adopted at Ottawa, December 4, 1914 and amended to June 2009. Effective 2009–2010 Season [Internet]. Canada: Hockey Canada; 2009 [cited 2011 Sep 7]. Available from: http://www.hockeycanada.ca/index.php/ci_id/66601/la_id/1.htm

32. Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP) [Internet]. Ottawa (ON): Public Health Agency of Canada; 2011 [cited 2011 Sep 7]. Available from: http://www.phac-aspc.gc.ca/injury-bles/chirpp/index-eng.php

33. Mackenzie SG, Pless IB. CHIRPP: Canada's principal injury surveillance program. Inj Prev. 1999;5(3):208–13.

34. Zhang JJ, Lam ET, Connaughton EP, Bennett G, Pease DG, Pham UL, etc. Variables affecting spectator enjoyment of minor league hockey games. Int J Sport Manage Market. 2004;5:1–26.

35. Andrew DP, Koo GY, Hardin R, Greenwell TC. Analysing motives of minor league hockey fans: the introduction of violence as a spectator motive. Int J Sport Manag Market. 2009;5:73–89.

36. Andrijiw AM. Life after hockey: an examination of athletic career transition and the National Hockey League's career transition program. Saint Catherine (ON): Faculty of Applied Health Sciences, Brock University; 2010.

37. Conacher B. As the puck turns: a personal journey through the world of hockey. Mississauga (ON): Wiley; 2007.

38. Robidoux M, Trudel P. Hockey Canada and the bodychecking debate in minor hockey. In: David Whitson D, Gruneau R, editors. Artificial ice: hockey, culture, and commerce. Toronto (ON): University of Toronto Press; 2006:101–22.

39. Reid SR, Losek JD. Factors associated with significant injuries in youth ice hockey players. Pediatr Emerg Care. 1999;15(5):310–3.

40. Smith M. From professional to youth hockey violence: the role of the mass media. In: Gamon MA, editor. Violence in Canada. Toronto (ON): Methuen; 1978:269–81.

41. Latour G. La LNAH à l'heure des choix [Internet]. Hockey Zone Plus. 2006 Mar 29 [cited 2011 Sep 7]. Available from: http://www.hockeyzoneplus.com/semipro/sp129_f.htm

42. AHL attendance surpasses 7 million [Internet]. 2005 May 22 [cited 2011 Sep 7]. Available from: http://theahl.com/ahl-attendance-surpasses-7-million-p136423

43. Number 1 NHL broadcast in Canada. CBC report, 2012 [cited 2012 Mar 28]. Available from: http://www.cbc.ca/revenuegroup/images/HNIC%20-%20Fall%202011.pdf

How active are children in Toronto? A comparison with accelerometry data from the Canadian Health Measures Survey

M. R. Stone, PhD (1); G. E. Faulkner, PhD (2); R. N. Buliung, PhD (3)

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Abstract

Introduction: The Canadian Health Measures Survey (CHMS) is the most comprehensive direct health measures survey ever conducted in Canada. Results show that the majority of children and youth (93%) do not meet current physical activity recommendations for health. CHMS data have not yet been considered alongside an independent sample of Canadian youth; such a Canadian-context examination could support CHMS results and contribute to discussions regarding accelerometry data reduction protocols.

Methods: From 2010 to 2011, valid accelerometry data were collected on 856 children living in the Greater Toronto Area (GTA). Where possible, data presentation and analyses were aligned with the CHMS protocol such that physical activity outcomes could be compared.

Results: Overall, trends were similar, with some deviations likely due to contextual and sampling differences and differences in data collection/reduction protocols regarding accelerometer model selection, wear time, activity intensity thresholds and epoch.

Conclusion: The similar trends support the notion that physical inactivity is an ongoing problem in communities across Canada.

Keywords: *ActiGraph, accelerometer, physical activity, sedentary behaviour, obesity, public health, youth, CHMS*

Introduction

Regular physical activity in childhood is associated with many physical, physiological and mental health benefits.[1] Canada's physical activity guidelines suggest children and adolescents aged 5 to 17 years accumulate at least 60 minutes of moderate-to-vigorous physical activity (MVPA) each day.[2] There is also evidence that they should engage in vigorous physical activity (VPA) at least 3 days a week.[2] While self-report and pedometer data have provided some evidence of national physical activity trends over time,[3] direct, objective assessments using accelerometry (on a national scale) have been absent until recently.

March 2011 saw the release of physical activity and sedentary behaviour data collected on a nationally representative sample of Canadian children and youth (n = 1608; boys = 809; girls = 799) as part of the Canadian Health Measures Survey (CHMS).[4] Actical accelerometers (Phillips – Respironics, Oregon, US) were used to capture minute-by-minute data over 7 consecutive days. Information was extracted using quality control and data reduction decisions[5] on the amount of time children and youth typically spend sedentary and in light, moderate and vigorous intensity physical activity; the amount of time spent in MVPA; the average number of steps taken per day; and the percentage of children attaining selected physical activity criteria. Results indicated that very few (7%) achieve recommended levels of physical activity (with more boys achieving guidelines than girls), and many spend a significant portion of their day sedentary (average of 8.6 hours per day).[4]

The CHMS is the most comprehensive direct health measures survey conducted in Canada. In addition to national estimates of physical activity levels, the study has also shed light on the declining levels of fitness observed in Canadian youth over the past few decades.[6] These data have received considerable public interest and media attention. They have also fuelled national campaigns (i.e. ParticipACTION; www.participaction.com) to increase population-wide levels of physical activity in children and youth. Comparisons with nationally representative data from the United States[7] revealed similar trends in physical activity and sedentary behaviour, despite some contextual, sampling and methodological differences between the two datasets. However, to our knowledge CHMS data have yet to be compared with an independent sample of Canadian children and youth. It would be relevant to verify their accuracy, given the widespread dissemination of the CHMS findings and their impact on research, policy and practice across Canada.

The aim of this study is to present accelerometer data from another study,

Author references:

1. School of Health and Human Performance, Dalhousie University, Halifax, Nova Scotia, Canada
2. Faculty of Kinesiology and Physical Education, University of Toronto, Toronto, Ontario, Canada
3. Department of Geography, University of Toronto Mississauga, Mississauga, Ontario, Canada

Correspondence: Michelle R. Stone, School of Health and Human Performance, Dalhousie University, 6230 South Street, Halifax, NS B3H 4R2; Tel.: (902) 494-1167; Fax: (902) 494-1084; Email: michelle.stone@dal.ca

Project BEAT, in a similar fashion to the way CHMS data have been reported, and discuss similarities and discrepancies between the two datasets.

Methods

Data source

Project BEAT (Built Environment and Active Transport; www.beat.utoronto.ca) is a large-scale, multidisciplinary and mixed method study examining how the built environment influences the way elementary school children travel to school in Toronto, Ontario. In January 2010 all elementary schools with Grade 5 and 6 students within the Toronto District School Board (n = 469) received an invitation to participate. Out of the pool of interested schools (54 responded, 40 of which were interested; response rate = 11.5%), 16 were selected because they varied with respect to built form (suburban looping street layout versus urban grid-based street layout) and socio-economic status (SES; low- and high-income households based on median household income reported in the 2006 Canada Census). Half of the surveyed schools were low-SES schools, and the other half were high-SES schools. Consent was obtained from participating school boards, individual schools, parents and students. Student participation was voluntary. Ethics approval from the Toronto District School Board and University of Toronto Ethics Committee was received.

Participants

Of the 1704 students enrolled in Grades 5 through 6 at the 16 participating schools, 1027 (60.3%; boys, n = 478; girls, n = 549) completed the travel behaviour survey and were given consent to participate in the study by their parents/guardians; missing responses resulted from parent or student refusal. Prior to any data collection, children completed an assent form (n = 1001; 26 students were absent for data collection). Height and weight were measured to calculate body mass index (BMI), and accelerometer-measured physical activity data were collected. For inclusion in data analysis, each child was required to wear an accelerometer for a minimum of 10 hours for at least 3 weekdays and 1 weekend day. A string of 30 minutes of consecutive zeros was classified as non-wear time/sleep time; these periods (most of which occurred during sleep) were removed from the analyses. Biologically implausible data were assessed to determine whether to include files in the final analyses. Of the 1001 children who wore an accelerometer, 95.8% had at least 1 valid day of data and 85.5% had at least 3 weekdays and 1 weekend day of valid data (n = 856; boys = 389, girls = 467; Table 1). This article is therefore based on these 856 participants (mean age [standard deviation] 11.1 [0.6] years), who met these inclusion parameters. This final response rate (856/1704 = 50.2%) is consistent with other active-consent studies with Canadian elementary school students.[8] Using age- and sex-specific BMI cut-points provided by the International Obesity Task Force,[9] participants were classified as normal weight, overweight or obese (Table 2).

Measurement of physical activity and sedentary behaviour

Children's physical activity was measured for seven days using an accelerometer (ActiGraph GT1M; Pensacola, FL, United States). The ActiGraph series are the most commonly used devices in the field, and they have moderate to good validation in children.[10] Prior to data collection, the intra-unit and inter-unit variability of all ActiGraph monitors (n = 120) was tested using a standardized treadmill protocol. The coefficients of variation were within acceptable limits.[11,12]

We used a 5-second epoch to capture rapid transitions in activity typical in children and related to health outcomes.[13] Children were asked to wear their accelerometer consistently; they were asked to only remove the device when engaging in water-based activities. The monitors were initialized to start collecting data at 12:00 A.M. on the day they were handed out to participants. The first day was excluded from data analyses to

TABLE 1
Distribution of Project BEAT and CHMS participants, by valid days of accelerometer wear (10 or more wear hours), age group and sex

	Number of valid days of accelerometer wear, %									
Study, Age group	0[a]	1	2	3	4	5	6	7	≥ 1	≥ 4[b]
Project BEAT										
10–12 years										
Total	4.2	1.8	1.6	6.9	2.1	7.9	21.9	53.6	95.8	85.5
Boys	4.1	2.6	1.7	7.6	1.7	8.4	20.5	53.3	95.9	84.0
Girls	4.3	1.1	1.5	6.3	2.4	7.4	23.0	53.9	95.7	86.8
CHMS										
Total[c]	4.6	2.9	3.6	4.1	8.2	12.7	24.0	39.8	95.4	84.7
6–10 years										
Boys	2.7	2.4	3.2	1.5	6.4	11.5	24.7	47.7	97.3	90.2
Girls	4.2	2.4	2.1	1.8	6.6	13.4	22.1	47.4	95.8	89.5
11–14 years										
Boys	4.4	2.0	1.7	5.1	6.4	11.9	30.5	38.0	95.6	86.8
Girls	3.2	2.8	3.6	2.1	7.8	12.1	23.1	45.2	96.8	88.3

Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS)[4]

Abbreviations: BEAT, Built Environment and Active Transport; CHMS, Canadian Health Measures Survey.

[a] Agreed to wear accelerometer, but returned device with no valid data (invalid wear or device malfunctioned).

[b] Three weekdays and one weekend day.

[c] Total includes additional age group (aged 15–19 years) sampled in CHMS. Remainder of table reflects results for those aged 6–10 years and 11–14 years, in light of Project BEAT's sample demographics (aged 10–12 years).

TABLE 2
Descriptive characteristics of Project BEAT and CHMS participants, by age group and sex

	Project BEAT		Canadian Health Measures Survey (CHMS)			
	10–12 years		6–10 years		11–14 years	
Characteristics	Boys	Girls	Boys	Girls	Boys	Girls
Sample size, n	389	467	369	340	256	248
Mean age, years	11.0	11.1	8.2	8.1	12.5	12.3
Mean height, cm	147.2	147.5	133.9	131.6	158.9	156.9
Mean weight, kg	42.3[a]	40.9	32.5	29.9	52.1	50.6
Mean BMI, kg/m^2	19.3[a]	18.6	17.8	17.0	20.3	20.4
BMI category, %[b]						
Normal	67.4[a]	73.9	74.4	82.5	72.5	70.5
Overweight	21.9	21.6	17.1[E]	12.6[E]	21.5	23.0[E]
Obese	10.8[a]	4.5	8.1[E]	4.9[E]	6.0[E]	6.5[E]

Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS).[4]

Abbreviations: BEAT, Built Environment and Active Transport; BMI, body mass index; CHMS, Canadian Health Measures Survey.

[a] Significantly different from girls; $p < .05$.

[b] International Obesity Task Force classification.[9]

[E] Use with caution.

control for any participant reactivity and because they were often handed out mid-day. Data collection took place during the Spring/Summer (April to June) and Fall (September to December) school period to limit any seasonal effect.

Time spent at various levels of movement intensity (sedentary, light, moderate, vigorous, hard) was classified according to published thresholds in children[14] and used to determine accumulated minutes of sedentary behaviour; light, moderate, vigorous and hard activity; and MVPA. The percentage of time spent sedentary, in light intensity activity and in MVPA were calculated using wear time data (the percentage of time spent in hard intensity activity was <1%, and therefore not reported). The proportion of children attaining different physical activity targets was examined in line with CHMS analyses. For example, Canadian and World Health Organization (WHO) physical activity guidelines recommend 60 minutes of MVPA each day.[2,15] Like the CHMS analyses, adherence was defined as the probability of accumulating at least 60 minutes of MVPA at least 6 days a week. The probability of accumulating at least 30, 60 and 90 minutes of MVPA on at least 1, 2, 3, 4, 5 or 6 days of the week was also calculated. The probability of accumulating any VPA (at least 5, 10 and 20 minutes) on at least 1, 2, 3, 4, 5 or 6 days of the week was also calculated. Minimal activity was assumed on missing days.

Statistical analyses

All analyses were conducted using SPSS version 19.0 for Windows (IBM, Armonk, NY, US) and were based on data for participants with at least 4 valid days. Similar to CHMS output, comparisons of physical activity intensity and duration were made according to gender and body weight classification (normal weight, overweight and obese)[9] using mixed-model ANOVAs with pair-wise contrasts. Differences between estimates were tested for statistical significance ($p < .05$).

Results

Participants

Table 1 shows a comparison of accelerometer wear by age group and sex between the studies. Table 2 shows the demographic characteristics (gender distribution, mean age, height, weight and BMI) for Project BEAT and CHMS participants.

Hours spent sedentary or in light intensity activity

Project BEAT collected an average of 16.7 hours per day of valid accelerometer data. Children spent an average of 13.3 hours (or 79.6% of that period) sedentary (790 minutes for boys, 802 minutes for girls; Table 3), a percentage nearly 20% higher than that identified by Colley et al.[4] using 2007 to 2009 CHMS data (62%). Similar to the CHMS, time spent sedentary did not differ by gender or weight classification. While the CHMS dataset did demonstrate differences between genders according to weight classification (with normal weight boys significantly less sedentary than normal weight girls, $p < .05$), there was no such relationship in Project BEAT. Project BEAT participants spent another 2.9 hours of their day (17.4% of wear time), on average, in light intensity activity (versus 4 hours per Colley et al.[4]); only in Project BEAT did gender differences in the accumulation of light intensity activity appear, with boys accumulating an average of 20 minutes more light intensity activity per day than girls ($p < .05$, Table 3). In both datasets, children classified as either overweight or obese accumulated a similar amount of light intensity activity per day compared to normal weight children.

Moderate-to-vigorous and vigorous activity

Boys achieved just over half the recommended levels of MVPA per day (35 minutes) while girls attained just 24 minutes per day, findings lower than those reported by Colley et al.[4] based on the CHMS (average of 61 and 47 minutes, respectively), yet similar with respect to gender differences. As observed in the CHMS, overweight and obese boys in Project BEAT accumulated less MVPA (32 and 26 minutes per day, respectively) compared with boys who were normal weight (38 minutes). Unlike the CHMS, this gradient was also observed in girls; girls classified as being overweight or obese accumulated 4 to 5 minutes less MVPA per day compared with normal weight girls (Table 3).

Project BEAT and CHMS data both revealed that the vast majority of all

TABLE 3
Average daily minutes of activity (at various levels of intensity) of Project BEAT and CHMS participants, by gender, age and BMI category

Study, Sex, Age, BMI category	Intensity of activity, average minutes/day					
	Sedentary	Light	Moderate	Vigorous	Hard	MVPA
Project BEAT						
Boys						
Age 10–12 years	790	185[a]	27[a]	7[a]	1	35[a]
BMI category						
Normal weight[b]	786	185[a]	29[a]	8[a]	1	38[a]
Overweight	796	184	25[c]	6[c]	<1	32[c]
Obese	800	186	21[c]	4[c]	<1	26[c]
Girls						
Age 10–12 years	802	165	18	5	<1	24
BMI category						
Normal weight[b]	799	165	19	5	1	25
Overweight	808	163	16[c]	4[c]	<1[c]	21[c]
Obese	830	174	16	3[c]	<1[c]	20[c]
CHMS						
Boys						
Age 6–10 years[b]	445	298	67[a]	2	–	69[a]
Age 11–14 years	524[c]	252[c]	58[a]	2	–	59[a]
BMI category[d]						
Normal weight[b]	500[a]	262	64[a]	2	–	65[a]
Overweight	524	260	50[c]	1[c]	–	51[c]
Obese	536	248	43[c]	<1[c]	–	44[c]
Girls						
Age 6–10 years[b]	446	306	56	2	–	58
Age 11–14 years	527[c]	250[c]	46[c]	2[E]	–	47[c]
BMI category[d]						
Normal weight[b]	524	249	46	2	–	48
Overweight	515	262	43	1[E]	–	44
Obese	544	263	47	<3	–	48

Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS).[4]

Abbreviations: BEAT, Built Environment and Active Transport; BMI, body mass index; CHMS, Canadian Health Measures Survey; MVPA, moderate-to-vigorous physical activity.

[a] Significantly different from estimate for girls ($p < .05$).

[b] Reference category; International Obesity Task Force classification.[9]

[c] Significantly different from estimate for reference category ($p < .05$).

[d] Includes additional age group (aged 15–19 years) sampled in CHMS. Remainder of table reflects results for those aged 6–10 years and 11–14 years, in light of Project BEAT's sample demographics (age 10–12 years).

[E] Use with caution.

MVPA is accumulated at moderate intensity (80% and 97%, respectively). Around 4.3% of children in Project BEAT accumulated at least 20 minutes of VPA at least 3 days a week, a result quite similar to CHMS findings (4%) (Figure 1).* Project BEAT data showed a significantly greater proportion of boys than girls meeting this target (7.1% and 1.9%, respectively; $p < .05$); gender comparisons were not made in the CHMS cohort. A little more than a quarter of Project BEAT children (27.1%) accumulated at least 10 minutes of VPA on at least 3 days of the week (35.1% of boys and 20.4% of girls; $p < .05$). Nearly two-thirds of children (64.7%) accumulated at least 5 minutes of VPA on at least 3 days of the week (72.8% of boys and 57.9% of girls; $p < .05$), findings that are proportionately higher compared to CHMS observations (Figure 1).

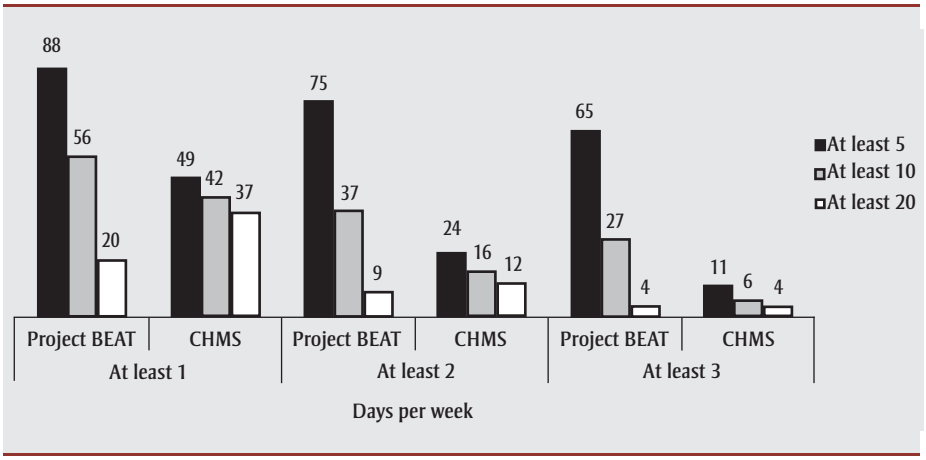
While the vast majority of children in both datasets did not meet the current physical activity recommendations of at least 60 minutes of MVPA at least 6 days a week, the proportion achieving recommendations was lower in Project BEAT (< 1%; 0.5% of boys, no girls) in comparison to the CHMS (6.7% of children; 9.0% of boys, 4.1% of girls) (Table 4). Like the CHMS, the difference in the proportion of Project BEAT children who met guidelines on at least 3 days a week compared to at least 6 days a week was much greater for boys than girls (13.3% and 2.1% increase, respectively; Figure 2).

In both datasets, considerably higher percentages of children accumulated 30 minutes of MVPA per day; in Project BEAT, 22.6% of boys and 5.4% of girls do so at least 6 days a week (CHMS: 29.0% and 21.3%, respectively; Table 4). Like the CHMS (82.6%), the majority of boys in Project BEAT (71.8%) accumulated 30 minutes of MVPA at least 3 days a week; yet unlike the CHMS (72.6%), this was not the case for girls (36.9%). In fact, just over half of Project BEAT girls (52.6%) only managed to accumulate 30 minutes of MVPA on 2 or more days of the week.

Not a single child in Project BEAT (and fewer than 2% in the CHMS) accumulated at least 90 minutes of MVPA at least 6 days of the week and only 2% met these criteria on at least 2 days of the week (3.3% of boys, 0.9% of girls; $p < .05$, Table 4). The proportion of children attaining these standards rose to 16.8% for at least 1 day of the week, with approximately 10% more boys than girls doing so (22.3% and 12.3%, respectively; $p < .05$), an increase

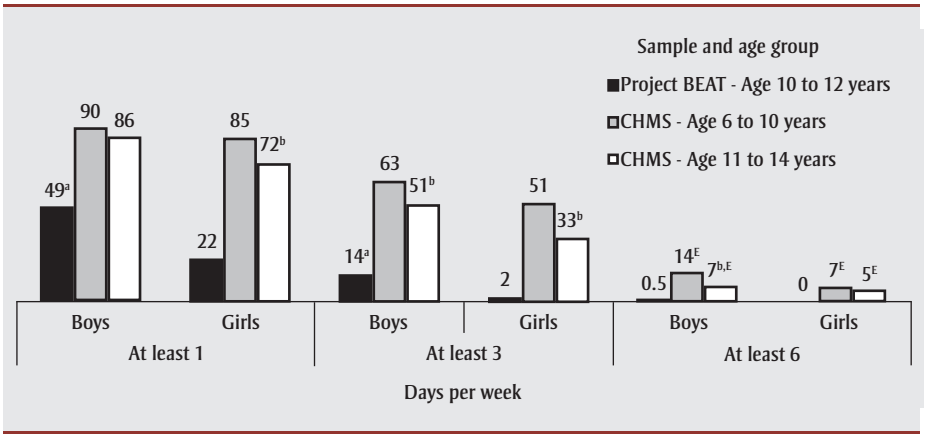
* The CHMS results in Figure 1 are based on children and youth aged 6–19 years, whereas Project BEAT results are based on children aged 10–12 years.

FIGURE 1
Percentage of Project BEAT participants (10–12 years) and CHMS participants (6–19 years) with at least 5, 10 and 20 minutes of vigorous physical activity a day, by number of days a week



Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS).[4]

FIGURE 2
Percentage of Project BEAT participants (10–12 years) and CHMS participants (6–10 years and 11–14 years) with at least 60 minutes of moderate-to-vigorous physical activity, by days per week and by sex



Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS).[4]

[a]Significantly different from estimate for girls ($p < .05$), Project BEAT.

[b]Significantly different from estimate for 6- to 10-year olds of same sex ($p < .05$), CHMS.

[E]Use with caution.

much lower than that reported in the CHMS dataset (60%; Table 4).

Discussion

To our knowledge, this is the first attempt to compare accelerometer-measured physical activity data from a large sample with results from a nationally representative dataset (the CHMS, n = 1608).[4] Overall, findings were broadly consistent (i.e. similar trends appeared) with some deviations probably due to differences in data collection and reduction protocols and in sample demographics.

Strengths of this study include the large sample size (856 children) and use of an objective measure of physical activity to examine multiple aspects of physical activity behaviour. Our collection of high-frequency physical activity data was particularly appropriate for quantifying children's activity behaviour.[13] In addition, a relatively low number of participants were excluded due to invalid accelerometer wear (less than 15%). The biggest limitation of the study is the contextual, sampling and methodological differences between the two datasets, which posed challenges in making direct comparisons. For example, the narrow age range of children sampled and the investigation of Toronto neighbourhoods in Project BEAT was quite different to the national data collection strategy of the CHMS. These issues limit the generalizability of findings to other age groups and geographic locations. While both studies present objectively measured data, the accelerometer measurement and data reduction protocols differed. The lack of standardized physical activity measurement and data reduction protocols in the field are a limitation to any study that attempts to compare results to an independent dataset. Despite these differences, overall trends were similar between the two studies, which supports the notion of very few children (< 10%) accumulating enough daily activity for health benefits and too many spending a significant amount of their day sedentary.

With methodological differences between Project BEAT and the CHMS acknowledged, the similar trends in findings are sobering. The Canadian physical activity guidelines for children and youth (which are in line with the World Health Organization Global Physical Activity Recommendations[15]) encourage children and youth to accumulate at least 60 minutes of MVPA every day.[2] CHMS data illustrate that only 7% achieve these recommendations, while Project BEAT data show that even less do so (< 1%). In fact, not a single girl managed to accumulate at least 60 minutes of MVPA every day of the week, based on Project BEAT data. Perhaps just as sobering is the finding that only 13.2% of children in Project BEAT managed to attain at least 30 minutes of MVPA on at least 6 days of the week, lower than the CHMS results at 25.3%. BEAT and CHMS data both illustrate that children and youth spend the majority of their day sedentary (anywhere from 62% to 80%).

TABLE 4
Percentage of Project BEAT participants (10–12 years) and CHMS participants (6–19 years) attaining selected physical activity criteria

	Days active out of 7, %						
Minutes of MVPA	**0**	**≥ 1**	**≥ 2**	**≥ 3**	**≥ 4**	**≥ 5**	**≥ 6**
Project BEAT							
≥ 30							
Total	22.0	78.0	65.2	52.8	40.9	25.0	13.2
Boys	11.0*	89.0*	80.3*	71.8*	59.5*	39.2*	22.6*
Girls	31.1	68.9	52.6	36.9	25.3	13.1	5.4
≥ 60							
Total	66.0	34.0	15.4	7.5	3.4	1.4	0.2
Boys	51.0*	49.0*	26.2*	13.8*	6.2*	2.8*	0.5
Girls	78.5	21.5	6.4	2.1	1.1	0.2	0.0
≥ 90							
Total	83.2	16.8	2.0	0.4	0.1	0.0	0.0
Boys	77.7*	22.3*	3.3*	0.5	0.3	0.0	0.0
Girls	87.7	12.3	0.9	0.2	0.0	0.0	0.0
CHMS							
≥ 30							
Total	5.1	94.9	87.6	77.7	64.5	47.1	25.3
Boys	3.3	96.7*	91.1*	82.6*	70.1*	52.6*	29.0*
Girls	6.9	93.1	83.9	72.6	58.4	41.2	21.3
≥ 60							
Total	20.2	79.8	61.3	44.4	29.3	16.6	6.7
Boys	14.8	85.2*	69.5*	52.9*	36.4*	21.5*	9.0*
Girls	26.1	73.9	52.6	35.4	21.7	11.3	4.1[E]
≥ 90							
Total	40.9	59.8	35.1	20.1	10.7	5.0[E]	1.7[E]
Boys	33.7	66.3*	42.5*	26.0*	14.7*	7.1*[E]	2.5*[E]
Girls	47.1	52.9	27.3	13.7	6.5[E]	2.7[E]	<2

Sources: Built Environment and Active Transport (BEAT) Project (2010–2011); 2007–2009 Canadian Health Measures Survey (CHMS).[4]

Abbreviations: BEAT, Built Environment and Active Transport; CHMS, Canadian Health Measures Survey; MVPA, moderate-to-vigorous physical activity.

* Significantly different from estimate for girls ($p < .05$).

[E] Use with caution.

Comparing CHMS and BEAT accelerometry procedures

Proportions attaining physical activity recommendations differed in both studies. Three methodological differences highlight the lack of standardization in accelerometry-based physical activity measurement protocols, which continues to make comparability between studies difficult.

1. The accelerometer wear protocols differed

In Project BEAT, participants were asked to wear their accelerometer while awake and asleep to maximize compliance and thus boost the probability of generating a large sample of participants with valid data for inclusion in data analyses. The CHMS requested that participants wear their accelerometer during waking hours only. In Project BEAT, the decision was made to exclude periods of 30 consecutive minutes of zero counts (most of which occurred during sleep time), whereas the CHMS used a less conservative approach. These decisions affect wear time and thus explain the difference in average wear times (Project BEAT: 16.7 hours; CHMS: 13.6 hours). These discrepancies also provide some rationale for differences seen in the proportion of the day spent sedentary and in light intensity activity and MVPA between the two datasets. For example, Project BEAT data show that children spend 79.6% of their day sedentary. Another 17.4% is spent in light intensity activity, with MVPA only contributing to 3% of the daily profile. In the CHMS dataset, children spent an average of 62% of their waking hours sedentary, with another 29.4% and 8.6% spent in light intensity activity and MVPA, respectively.

2. The accelerometer devices and activity intensity classification thresholds differed

Project BEAT used ActiGraph GT1M accelerometers to monitor physical activity behaviour, while the CHMS used Actical accelerometers. While the GT1M model is one of the most validated and widely used devices of their kind, a possible limitation is that it measures acceleration in the vertical plane only; the Actical device is omni-directional, allowing it to capture a wider range of movement than a uni-axial device and capture non-ambulatory activities. Despite the theoretical advantage of the Actical accelerometer, in reality both accelerometers provide similar information given that the majority of movement is detected in the vertical plane.[16] Each accelerometer model provides a unique dimensionless activity count over a user-defined interval (i.e. between 1 and 60 seconds). These raw data are converted to useable information using calibration research that generates model-specific activity intensity thresholds. Consequently, time spent sedentary and in light, moderate, vigorous and hard intensity activity can be computed. The CHMS activity intensity thresholds for the Actical were derived from calibration work in children[17,18] and adults,[18] and the Project BEAT thresholds only from calibration trials with children.[14]

Metabolic energy turnover (MET) values are often used to express the intensity of physical activity according to intensity categories; a compendium of energy costs for a variety of children's activities is available.[19] In most studies like the CHMS, moderate intensity is defined as 3 METs or more. However, more recent evidence suggests that a threshold of 4 METs or

more may be more appropriate for describing moderate or higher intensity activity in children[20–23] and for determining relationships between activity and health outcomes.[14] Indeed, the National Health and Nutrition Examination Survey (NHANES) in the U.S. uses a moderate intensity threshold based on 4 METs to classify MVPA in children[24]; in the BEAT study, the threshold for moderate intensity was also based on 4 METs. Our use of a more stringent threshold to classify MVPA (a decision made before the release of the CHMS findings) likely explains the lower levels of MVPA observed (and fewer children meeting guidelines) in Project BEAT data compared with CHMS data.

Reports in other countries, for example, NHANES[7] in the U.S. and the Avon Longitudinal Study of Parents and Children (ALSPAC)[25] in England, support these results. It is of particular interest to compare Project BEAT data with the ALSPAC data since both studies used the same thresholds to classify moderate and vigorous intensity activity: we see very similar proportions of children achieving the 60 minutes of MVPA per day guidelines (BEAT at < 1% and ALSPAC at 2.5%) and similar average levels of MVPA (BEAT at 29 minutes per day and ALSPAC at 20 minutes per day).

3. The user-specified data collection interval differed

Project BEAT used a 5-second epoch to capture the short and sporadic bursts of activity that are typical in children,[13] whereas the CHMS captured physical activity data at 1-minute intervals. The influence of epoch length on physical activity data has been discussed at length: shorter epochs capture more MVPA, and longer epochs ''dilute'' the intensity of the data[26,27] and therefore affect the proportion of children attaining PA guidelines.[28] Some have found significant epoch effects for hard and very hard activity,[26] and others for all intensities.[29] Using direct observation, McClain at al.[30] showed that a 5-second epoch provided the least discrepant estimates of MVPA in fifth grade children compared with 10-, 15-, 20-, 30- and 60-second epochs. Indeed, there is strong support for utilizing a 5-second epoch to truly capture children's spontaneous, discontinuous patterns of activity.[13,29,30]

Project BEAT's finding of a greater proportion of children accumulating at least 5, 10 and 20 minutes of vigorous intensity activity on one or more days in comparison to the CHMS results could be a reflection of utilizing a shorter epoch to capture and express accelerometer data. The discrepancies were more apparent for lower levels of VPA (at least 5 and 10 minutes); in fact, when examining those accumulating at least 20 minutes of VPA per day, the proportions were nearly identical between datasets (around 4% for each). The epoch effect may be diluted at the upper extremes of daily VPA accumulation and have a less significant impact on levels of MVPA than accelerometer intensity thresholds, given levels were somewhat lower in Project BEAT compared with the CHMS dataset.

Conclusion

Using data from Project BEAT, this study demonstrates that the low levels of physical activity and high levels of sedentary behaviour amongst Canadian youth, as reported in the CHMS, do occur in an independent sample of Canadian youth. Accelerometry data in both datasets show that the majority of children and youth do not meet current physical activity recommendations and spend a significant proportion of their day sedentary. These similarities have been established despite contextual, sampling and methodological differences between the two datasets, limitations that have been noted and discussed, and also presented as three methodological considerations in analyzing accelerometry data. That both datasets reveal similar trends in physical activity and inactivity behaviour among Canadian children and youth is encouraging for purposes of validation, yet disheartening given the ramifications of such inactivity on health. The consistency of data from the CHMS and that on a sample of children from the Greater Toronto Area, where conditions might be most conducive for physical activity in terms of facilities and resources, supports the notion that physical inactivity is most likely an ongoing problem across Canada.

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References

1. Janssen I, LeBlanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. Int J Behav Nutr Phys Act. 2010;7:40.

2. Canadian Society for Exercise Physiology, ParticipACTION. Canadian Society for Exercise Physiology and ParticipACTION share new research to inform Canadians of physical activity levels required. Press release. 2010 May 12. Available at: http://files.participaction.com/pressreleases/en/csep_pac-nationalrelease-final-en.pdf

3. Katzmarkyk PT, Ardern CI. Physical activity levels of Canadian children and youth: current issues and recommendations. Can J Diabetes. 2004;28:67–78.

4. Colley RC, Garriguet D, Janssen I, Craig CL, Clarke J, Tremblay MS. Physical activity of Canadian children and youth: accelerometer results from the 2007 to 2009 Canadian Health Measures Survey. Health Reports (Statistics Canada, 82-003) 2011; 22:1–10.

5. Colley R, Connor Gorber S, Tremblay MS. Quality control and data reduction procedures for accelerometry-derived measures of physical activity. Health Rep. 2010; 21:63–9.

6. Tremblay MS, Shields M, Laviolette M, Craig CL, Janssen I, Gorber SC. Fitness of Canadian children and youth: results from the 2007–2009 Canadian Health Measures Survey. Health Rep. 2009;21:7–20.

7. Troiano RP, Berrigan D, Dodd K, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. Med Sci Sports Exerc. 2008;40:181–8.

8. Veugelers PJ, Fitzgerald AL. Prevalence of and risk factors for childhood overweight and obesity. CMAJ. 2005;173:607–13.

9. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ. 2000;320:1240.

10. De Vries SI, Van Hirtum HW, Bakker I, Hopman-Rock M, Hirasing RA, Van Mechelen W. Validity and reproducibility of motion sensors in youth: a systematic update. Med Sci Sports Exerc. 2009;41: 818–27.

11. Chen KY, Bassett DR, Jr. The technology of accelerometry-based activity monitors: current and future. Med Sci Sports Exerc. 2005;37:S490–500.

12. Welk GJ. Principles of design and analyses for the calibration of accelerometry-based activity monitors. Med Sci Sports Exerc. 2005;37:S501–11.

13. Stone MR, Rowlands AV, Middlebrooke AR, Jawis MN, Eston RG. The pattern of physical activity in relation to health outcomes in boys. Int J Pediatr Obes. 2009; 4:306–15.

14. Stone MR, Rowlands AV, Eston RG. Relationships between accelerometer-assessed physical activity and health in children: impact of the activity-intensity classification method. J Sports Sci Med. 2009;8:136–43.

15. World Health Organization. Global recommendations on physical activity for health. Geneva (CH): World Health Organization; 2010.

16. Corder K, Brage S, Ekelund U. Accelerometers and pedometers: methodology and clinical application. Curr Opin Clin Nutr Metab Care. 2007;10:597–603.

17. Puyau MR, Adolph AL, Vohra FA, Zakeri I, Butte NF. Prediction of activity energy expenditure using accelerometers in children. Med Sci Sports Exerc. 2004;36: 1625–31.

18. Wong SL, Colley R, Connor Gorber S, Tremblay M. Actical accelerometer sedentary activity thresholds for adults. J Phys Act Health. 2011;8:587–91.

19. Ridley K, Olds TS. Assigning energy costs to activities in children: a review and synthesis. Med Sci Sports Exerc. 2008;40: 1439–46.

20. Harrell JS, McMurray RG, Baggett CD, Pennell ML, Pearce PF, Bangdiwala SI. Energy costs of physical activities in children and adolescents. Med Sci Sports Exerc. 2005;37:329–36.

21. Mattocks C, Leary S, Ness A, Deere K, Saunders J, Tilling K, et al. Calibration of an accelerometer during free-living activities in children. Int J Pediatr Obes. 2007;2:218–26.

22. Reilly JJ, Penpraze V, Hislop J, Davies G, Grant S, Paton JY. Objective measurement of physical activity and sedentary behaviour: review with new data. Arch Dis Child. 2008;93:614–9.

23. Treuth MS, Schmitz K, Catellier DJ, McMurray RG, Murray DM, Almeida MJ, et al. Defining accelerometer thresholds for activity intensities in adolescent girls. Med Sci Sports Exerc. 2004;36:1259–66.

24. Trost SG, Pate RR, Sallis JF, Freedson PS, Taylor WC, Dowda M, et al. Age and gender differences in objectively measured physical activity in youth. Med Sci Sports Exerc. 2002;34:350–5.

25. Riddoch CJ, Mattocks C, Deere K, Saunders J, Kirkby J, Tilling K, et al. Objective measurement of levels and patterns of physical activity. Arch Dis Child. 2007;92:963–9.

26. Nilsson A, Ekelund U, Yngve A, Sjoestroem M. Assessing physical activity among children with accelerometers using different time sampling intervals and placements. Pediatr Exerc Sci. 2002;14:87–96.

27. Trost SG, McIver KL, Pate RR. Conducting accelerometer-based assessments in field-based research. Med Sci Sports Exerc. 2005;37:S531–43.

28. Ojiambo R, Cuthill R, Budd H, Konstabel K, Casajus JA, Gonzalez-Aguero A, et al. Impact of methodological decisions on accelerometer output variables in young children. Int J Obes (London). 2011;35: S98–103.

29. Edwardson CL, Gorely T. Epoch length and its effect on physical activity intensity. Med Sci Sports Exerc. 2010;42:928–34.

30. McClain JJ, Abraham TL, Brusseau TA Jr., Tudor-Locke C. Epoch length and accelerometer outputs in children: comparison to direct observation. Med Sci Sports Exerc. 2008;40:2080–7.

Cancer incidence, mortality and survival trends in Canada, 1970–2007

L. Kachuri, MPH (1); P. De, PhD (1, 2); L. F. Ellison, MSc (3); R. Semenciw, MSc (4); The Advisory Committee on Canadian Cancer Statistics

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Abstract

Introduction: Monitoring cancer trends can help evaluate progress in cancer control while reinforcing prevention activities. This analysis examines long-term trends for selected cancers in Canada using data from national databases.

Methods: Annual changes in trends for age-standardized incidence and mortality rates between 1970 and 2007 were examined by sex for 1) all cancers combined, 2) the four most common cancers (prostate, breast, lung, colorectal) and 3) cancers that demonstrate the most recent notable changes in trend. Five-year relative survival for 1992–2007 was also calculated.

Results: Incidence rates for all primary cancer cases combined increased 0.9% per year in males and 0.8% per year in females over the study period, with varying degrees of increase for melanoma, thyroid, liver, prostate, kidney, colorectal, lung, breast, and bladder cancers and decrease for larynx, oral, stomach and cervical cancers. Mortality rates were characterized by significant declines for all cancers combined and for most cancers examined except for melanoma and female lung cancer. The largest improvements in cancer survival were for prostate, liver, colorectal and kidney cancers. While the overall trends in mortality rates and survival point to notable successes in cancer control, the increasing trend in incidence rates for some cancers emphasize the need for continued efforts in prevention.

Keywords: *cancer surveillance, incidence, mortality, survival, risk factors*

Introduction

At the beginning of 2007, nearly 750 000 Canadians had a diagnosis of cancer in the previous 10 years.[1] Cancer is the leading cause of death in Canada,[2] with 82% of all cancer deaths occurring in those aged 60 years and over.[1] By 2036, about 10.9 million Canadians will be aged 65 years or older,[3] which will lead to more new cancer cases and create significant demands for cancer care.

An examination of historical cancer trends can help us predict future patterns of this disease and evaluate progress in cancer control, thus allowing public health professionals to reinforce existing cancer prevention and control activities.

This analysis examines long-term trends for (1) all cancers combined, (2) the four most common cancers in Canada (prostate, female breast, lung, colorectal), and (3) those cancers shown to have the most notable changes in their incidence or mortality trends in the past decade (stomach, liver, thyroid, larynx, melanoma, bladder, kidney, cervix). To our knowledge, this is the most up-to-date and comprehensive examination of long-term Canadian cancer trends. As such, it can be used to compare with reported trends in other countries. More importantly, trends are discussed in the context of major cancer risk factors and associated health behaviours to provide perspective on the possible determinants of disease.

Methods

Data sources

We took cancer incidence data from 1992 to 2007 from the July 2010 version of the Canadian Cancer Registry, a person-oriented, population-based database.[4] Data for the earlier period, from 1970 to 1991, are from the National Cancer Incidence and Reporting System, a tumour-oriented database established in 1969.[5] Mortality data were from the Canadian Vital Statistics Death Database. Population estimates were from Statistics Canada's Demographic Estimates Compendium 2010.[6]

We created a file containing records of invasive cancer cases for all ages and in situ bladder cancer cases (except from the province of Ontario) using the International Agency for Research on Cancer multiple primary coding rules.[7] Cancer cases were classified based on the *International Classification of Diseases for Oncology, 3rd Edition*.[8] Cancer group definitions are provided elsewhere.[1] For cancer deaths, the underlying causes of death were selected according to the *International Classification of Diseases* and classified to version 10 (ICD-10).[9]

Author references:

1. Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
2. Cancer Control Policy, Canadian Cancer Society, Toronto, Ontario, Canada
3. Health Statistics Division, Statistics Canada, Ottawa, Ontario, Canada
4. Chronic Diseases Prevention and Control, Public Health Agency of Canada, Ottawa, Ontario, Canada

Correspondence: Prithwish De, Canadian Cancer Society, 55 St Clair Ave West, Suite 300, Toronto, ON M4V 2Y7; Tel.: 416-934-5335; Fax: 416-961-4189; Email: prithwish.de@cancer.ca

Statistical analysis

We calculated age-specific rates for each year and then standardized them to the 1991 Canadian population to obtain the age-standardized incidence rates (ASIR) and mortality rates (ASMR). Trends over the short and long terms were analyzed by calculating the annual percent change (APC) and average annual percent change (AAPC) in rates, respectively, using Joinpoint version 3.5.1 software.[10] Joinpoint uses piecewise regression to model the change in ASIR and ASMR on the log scale. While other approaches, such as a polynomial fit to the data, could be used, Joinpoint characterizes trends more succinctly by transforming the slope of each segment into an average percent change.[11] A minimum of 5 years of data before and after a point of change was required to identify a new trend. Models were tested using the Monte Carlo permutation method ($p = .05$ level of significance). Any statistically significant changes in trend are described here as ''decreasing'' or ''declining'' or, conversely, ''increasing.''

Relative survival analyses were based on a publicly available algorithm[12] which we adapted slightly. The focus of this analysis was on all primary cancer cases aged 15 to 99 years at diagnosis. Mortality follow-up through December 31, 2007, was determined by record linkage of the Canadian Cancer Registry to the Canadian Vital Statistics Death Database and from information reported by provincial/territorial cancer registries. Data from Quebec were excluded because the method of ascertaining the date of diagnosis of cancer cases in this province differed from that of the other provinces[13,14] and because of issues in correctly ascertaining the vital status of cases diagnosed in Quebec within the Canadian Cancer Registry. We derived 5-year relative survival ratio (RSR) estimates using the cohort method for 1992 to 1994, 1996 to 1998, and 2000 to 2002 and the period method for 2005 to 2007. Because more recent data were unavailable, expected survival data for 2005 to 2007 (used in the derivation of relative survival) were assumed to be the same as in 2000 to 2002. Further information on the survival methodology used is provided elsewhere.[15]

Results

In 2007, 85 430 new cancer cases and 36 569 cancer deaths were reported in males, and 78 099 new cases and 33 026 deaths occurred in females. Together, the most frequently diagnosed cancers (prostate, female breast, colorectal and lung) accounted for 55% and 52% of all new cancer diagnoses in males and females, respectively, as well as 50% and 51% of cancer deaths in each sex.

Trends in incidence and mortality

All cancers combined

Table 1 shows the APCs and AAPCs for cancer incidence. The ASIRs for all cancers combined for 1970 and 2007 were higher in males (1970: 330.4 per 100 000; 2007: 463.2 per 100 000) than in females (1970: 272.0 per 100 000; 2007: 362.3 per 100 000). The rate increased at an average of 0.9% per year in males and 0.8% per year in females over the study period.

The APCs and AAPCs for cancer mortality are shown in Table 2. As for incidence, the ASMR for all cancers combined was higher in males (1970: 228.4 per 100 000; 2007: 200.1 per 100 000) than in females (1970: 152.1 per 100 000; 2007: 141.2 per 100 000) but decreased at a rate of 0.3% per year in males and 0.2% per year in females over the study period.

Selected cancers

Between 1970 and 2007, there was an overall upward trend in male incidence rates (Figure 1) for melanoma (AAPC: 3.7%) and for thyroid (3.6%), liver (3.5%), prostate (2.2%), kidney (1.8%), colorectal (0.6%) and bladder cancers (0.4%) but a declining trend for larynx (0.8%), oral (1.4%) and stomach (2.1%) cancers. Incidence rates increased in females for thyroid (AAPC: 4.4%), lung (4.4%), melanoma (2.9%), kidney (2.1%), liver (1.9%), breast (0.5%) and bladder cancers (0.5%) while decreasing AAPCs were observed for cervix (2.5%) and stomach cancers (2.3%).

For most cancers, mortality rates between 1970 and 2007 were characterized by statistically significant decreases (Figure 2) with the exception of female lung cancer (AAPC: 4.0%) and melanoma (AAPC: 2.3% for males, 0.8% for females), for which increases were observed.

The trends for certain cancers are worth highlighting. For example, the prostate cancer incidence rate peaked twice, in 1993 and 2001 (Table 1 and Figure 1). Following the first peak, the incidence rate decreased (APC: 5.2%) until 1997, after which the rate climbed at 3.9% per year to a second peak in 2001, followed by a period of non-significant decline. We observed only one period of increase in the mortality rate for this cancer, between 1977 and 1993, which preceded a continuous decline that has further accelerated since 2001 (Table 2).

The incidence rate of lung cancer increased by 3.7% per year in males between 1970 and 1983. This was followed by a period of non-significant change until 1990 when the incidence rate started declining (Table 1). In females, the incidence rate has been increasing since 1970 but has slowed from 8.4% per year (1970–1983) to 3.8% per year (1983–1992) and finally to 1.4% per year (1992–2007). Lung cancer mortality in males followed a trajectory similar to incidence: the rate increased (2.7% per year) until 1983, remained stable (0.0% per year) from 1983 to 1992, and then began declining at an annual rate of 2.2% (Table 2). In contrast, the lung cancer mortality rate in females has continued to increase since 1970, from 6.9% per year (1970–1985) to 3.6% per year (1985–1994) and finally 1.0% per year (1994–2007).

Larynx cancer incidence rates increased from 1970 until 1980 in males (APC: 3.6%) and until 1989 in females (3.2% per year). Male incidence rates declined at 1.0% per year from 1980 to 1992, after which the decrease accelerated to 3.4% per year. Female incidence rate declined at an annual rate of 3.1% since 1989. The mortality rate increased from 1970 until 1988 for males (0.8% per year) and until 1991 for females (1.9% per year), followed by significant declines in both sexes.

Bladder cancer incidence rates increased from 1970 to 1981 (males: 3.3% per year; females: 3.5% per year), but the trend

TABLE 1
Annual percent change and average annual percent change in age-standardized incidence rates per 100 000 for selected cancers, Canada[a], 1970–2007

Cancer type	ASIR		Trend 1		Trend 2		Trend 3		Trend 4		Trend 5		Trend 6		AAPC (95% CI)
	1970	2007	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	(1970–2007)
Males															
All cancers	330.4	463.2	1970–1983	2.6* (2.3, 2.9)	1983–1989	0.0 (−1.0,1.0)	1989–1993	2.3* (0.3, 4.4)	1993–1997	−2.2* (−4.0, −0.3)	1997–2001	1.1 (−0.7, 3.0)	2001–2007	−0.6* (−1.2, 0.0)	0.9* (0.5, 1.3)
Prostate	53.8	124.7	1970–1989	2.8* (2.6, 3.1)	1989–1993	9.7* (6.4,13.1)	1993–1997	−5.2* (−7.8, −2.6)	1997–2001	3.9* (1.2, 6.7)	2001–2007	−0.7 (−1.5, 0.1)			2.2* (1.6, 2.7)
Colorectal	47.8	60.4	1970–1984	2.2* (1.9, 2.5)	1984–1996	−0.6* (−1.0, −0.3)	1996–2000	1.4 (−0.9, 3.7)	2000–2007	−0.7* (−1.3, −0.2)					0.6* (0.3, 0.9)
Lung	59.3	67.8	1970–1983	3.7* (3.4, 4.0)	1983–1990	−0.4 (−1.2, 0.4)	1990–2007	−1.9* (−2.0, −1.7)							0.3* (0.1, 0.5)
Bladder	24.5	26.9	1970–1981	3.3* (2.5, 4.0)	1981–2007	−0.7* (−0.9, −0.6)									0.4* (0.2, 0.7)
Thyroid	1.5	5.2	1970–1997	2.4* (1.9, 2.8)	1997–2007	6.9* (5.7, 8.1)									3.6* (3.1, 4.0)
Larynx	6.9	4.8	1970–1980	3.6* (2.5, 4.6)	1980–1992	−1.0* (−1.8, −0.3)	1992–2007	−3.4* (−3.9, −3.0)							−0.8* (−1.2, −0.4)
Liver	2.0	6.2	1970–2007	3.5* (3.3, 3.7)											3.5* (3.3, 3.7)
Melanoma	3.4	13.7	1970–1986	6.2* (5.4, 7.1)	1986–2007	1.9* (1.6, 2.2)									3.7* (3.3, 4.1)
Oral	20.6	12.9	1970–1978	0.2 (−0.8, 1.1)	1978–1992	−1.7* (−2.1, −1.3)	1992–1998	−3.5* (−5.2, −1.8)	1998–2007	−1.0* (−1.7, −0.3)					−1.4* (−1.8, −1.0)
Stomach	23.4	10.4	1970–1983	−1.3* (−1.7, −0.9)	1983–2007	−2.6* (−2.7, −2.4)									−2.1* (−2.3, −2.0)
Kidney	7.8	15.1	1970–1977	1.2 (−0.3, 2.6)	1977–1989	4.0* (3.4, 4.6)	1989–1998	−0.3 (−1.0, 0.4)	1998–2007	1.4* (0.9, 2.0)					1.8* (1.4, 2.2)
Females															
All cancers	272.0	362.3	1970–1981	1.6* (1.2, 1.9)	1981–2007	0.4* (0.4, 0.5)									0.8* (0.7, 0.9)
Breast	77.1	98.4	1970–1998	0.9* (0.8, 1.1)	1998–2007	−0.7* (−1.2, −0.2)									0.5* (0.4, 0.7)
Colorectal	44.5	40.6	1970–1983	1.0* (0.6, 1.4)	1983–1996	−1.5* (−1.8, −1.1)	1996–2000	1.3 (−1.8, 4.4)	2000–2007	−0.8* (−1.6, −0.1)					−0.2 (−0.6, 0.2)
Lung	9.3	47.2	1970–1983	8.4* (7.9, 8.9)	1983–1992	3.8* (3.2, 4.4)	1992–2007	1.4* (1.2, 1.5)							4.4* (4.2, 4.6)
Bladder	6.7	7.9	1970–1981	3.5* (1.9, 5.1)	1981–2007	−0.4* (−0.6, −0.2)									0.5* (0.1, 0.9)
Thyroid	3.9	18.0	1970–1989	1.8* (1.4, 2.2)	1989–1994	6.9* (3.5, 10.4)	1994–1998	1.9 (−2.7, 6.7)	1998–2002	12.5* (8.2,17.0)	2002–2007	6.9* (5.4, 8.3)			4.3* (3.5, 5.1)
Larynx	0.7	1.0	1970–1989	3.2* (2.3, 4.1)	1989–2007	−3.1* (−3.8, −2.3)									0.1 (−0.5, 0.6)
Liver	0.9	1.7	1970–2007	1.9* (1.6, 2.2)											1.9* (1.6, 2.2)
Melanoma	4.1	11.3	1970–1980	7.4* (5.8, 9.1)	1980–2007	1.3* (1.0, 1.5)									2.9* (2.4, 3.3)
Oral	5.1	5.4	1970–1979	1.4 (0.0, 2.9)	1979–2007	−0.4* (−0.6, −0.2)									0.0 (−0.3, 0.4)
Cervix	19.4	7.8	1970–1988	−3.3* (−3.6, −3.0)	1988–2007	−1.8* (−2.1, −1.5)									−2.5* (−2.7, −2.3)
Stomach	11.2	4.8	1970–1986	−1.9* (−2.3, −1.5)	1986–1991	−4.2* (−7.2, −1.1)	1991–2007	−2.0* (−2.4, −1.7)							−2.3* (−2.7, −1.8)
Kidney	3.7	9.0	1970–1980	1.4* (0.1, 2.7)	1980–1987	6.7* (4.4, 8.9)	1987–1997	−0.1 (−1.0, 0.8)	1997–2007	1.8* (1.2, 2.5)					2.1* (1.5, 2.7)

Abbreviations: AAPC, average annual percent change; APC, annual percent change; ASIR, age-standardized incidence rates; CI, confidence interval.

[a] Excluding Quebec.

* Two-sided $p < .05$.

TABLE 2
Annual percent change and average annual percent change in age-standardized mortality rates per 100 000 for selected cancers in Canada[a], 1970–2007

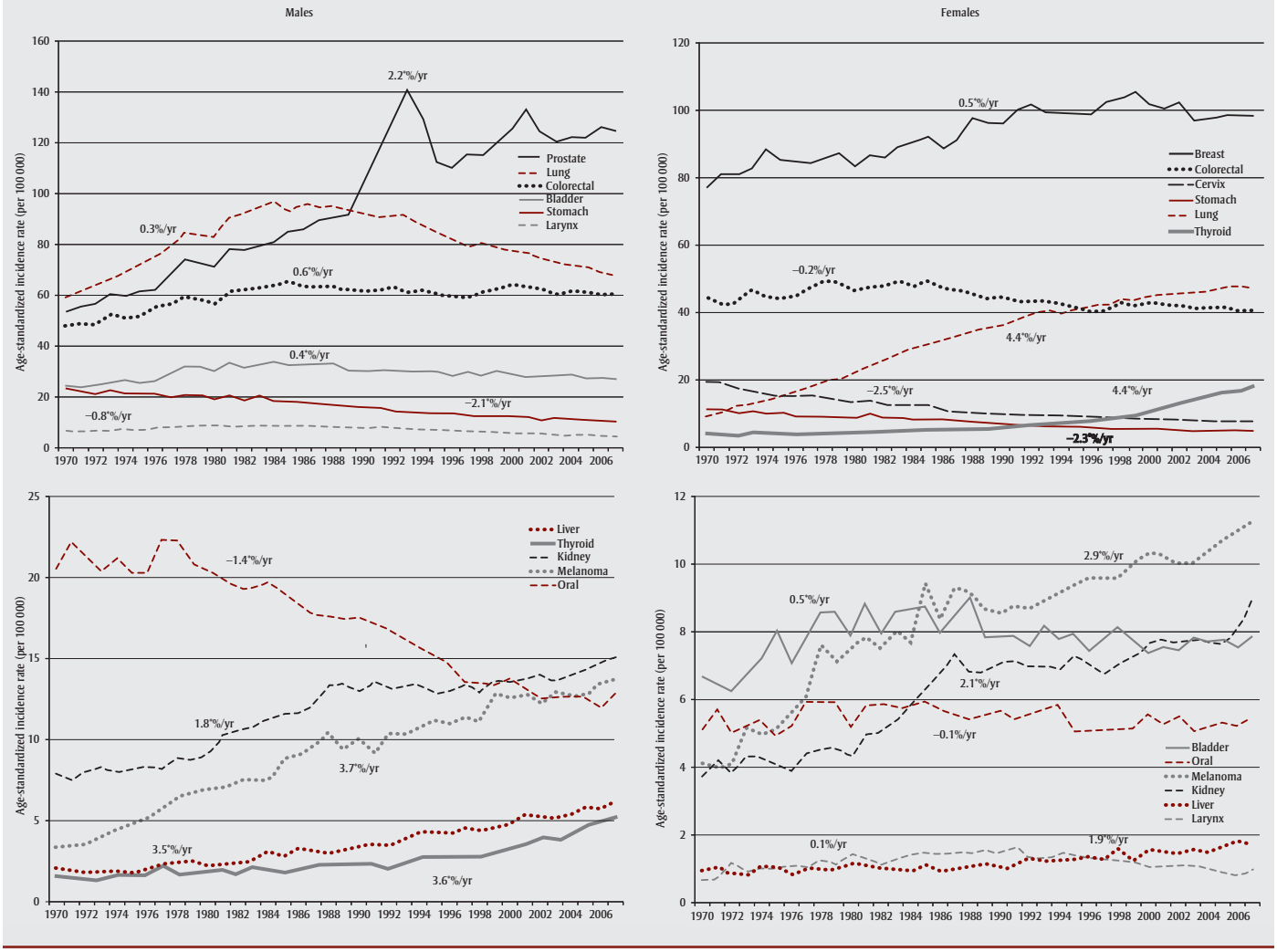
Cancer type	ASMR		Trend 1		Trend 2		Trend 3		Trend 4		AAPC (95% CI)
	1970	2007	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	Year	APC (95% CI)	(1970–2007)
Males											
All cancers	228.4	200.1	1970–1988	0.7* (0.6, 0.7)	1988–2001	−0.9* (–1.0, –0.8)	2001–2007	–2.0* (–2.3, –1.7)			–0.3* (–0.4, –0.3)
Prostate	25.4	20.4	1970–1977	–0.2 (–1.3, 0.8)	1977–1993	1.4* (1.1, 1.7)	1993–2001	–2.3* (–3.0, –1.6)	2001–2007	–4.2* (–5.1, –3.4)	–0.6* (–0.9, –0.3)
Colorectal	33.9	24.4	1970–1988	0.0 (–0.2, 0.2)	1988–2003	–1.1* (–1.4, –0.8)	2003–2007	–2.5* (–4.1, –0.9)			–0.7* (–0.9, –0.5)
Lung	55.0	57.0	1970–1983	2.7* (2.4, 3.0)	1983–1992	0.0 (–0.5, 0.5)	1992–2007	–2.2* (–2.3, –2.0)			0.0 (–0.1, 0.2)
Bladder	9.0	6.9	1970–2007	–0.7* (–0.8, –0.6)							–0.7* (–0.8, -0.6)
Thyroid	0.6	0.4	Not applicable: small number of deaths								
Larynx	3.1	1.8	1970–1988	0.8* (0.4, 1.3)	1988–2001	–2.3* (–2.9, –1.7)	2001–2007	–6.0* (–7.8, –4.2)			–1.4* (–1.4, –1.0)
Liver	1.8	3.1	1970–1982	0.1 (–1.7, 1.8)	1982–1987	8.6* (0.3, 17.6)	1987–1991	–5.2 (–15.0, 5.7)	1991–2007	2.4* (1.7, 3.2)	1.6 (0.0, 3.3)
Melanoma	1.3	2.8	1970–1985	4.2* (3.0, 5.3)	1985–2007	1.1* (0.6, 1.5)					2.3* (1.8, 2.8)
Oral	6.3	4.1	1970–1991	–0.5* (–0.8, –0.1)	1991–2007	–2.5* (–3.0, –2.0)					–1.3* (–1.6, –1.0)
Stomach	23.3	6.5	1970–2007	–3.3* (–3.4, –3.3)							–3.3* (–3.4, –3.3)
Kidney	4.7	5.0	1970–1992	0.8* (0.5, 1.1)	1992–2007	–0.7* (–1.1, –0.2)					0.2 (–0.1, 0.4)
Females											
All cancers	152.1	141.2	1970–1977	–0.6* (–1.1, –0.1)	1977–1988	0.5* (0.2, 0.8)	1988–2002	–0.3* (–0.4, –0.1)	2002–2007	–1.2* (–1.8, –0.6)	–0.2* (–0.4, –0.1)
Breast	30.7	21.8	1970–1982	–0.4* (–0.7, –0.1)	1982–1986	2.0 (–0.4, 4.4)	1986–1994	–1.0* (–1.6, –0.4)	1994–2007	–2.4* (–2.7, –2.2)	–1.0* (–1.3, –0.7)
Colorectal	28.6	16.3	1970–1986	–1.2* (–1.5, –1.0)	1986–2007	–1.7* (–1.8, –1.5)					–1.5* (–1.6, –1.3)
Lung	8.3	36.1	1970–1985	6.9* (6.5, 7.4)	1985–1994	3.6* (2.9, 4.2)	1994–2007	1.0* (0.8, 1.3)			4.0* (3.8, 4.3)
Bladder	2.8	2.1	1970–1996	–1.2* (–1.5, –0.8)	1996–2007	0.3 (–0.7, 1.3)					–0.7* (–1.1, –0.4)
Thyroid	1.0	0.4	Not applicable: small number of deaths								
Larynx	0.4	0.4	1970–1991	1.9* (0.8, 3.0)	1991–2007	–2.7* (–4.0, –1.4)					–0.1 (–0.9, –0.7)
Liver	0.7	0.8	1970–1989	1.2* (0.3, 2.0)	1989–1994	–6.3 (–13.2, 1.1)	1994–2007	1.9* (0.7, 3.1)			0.4 (–0.8, 1.5)
Melanoma	1.1	1.6	1970–1983	2.0* (0.7, 3.3)	1983–2007	0.2 (–0.2, 0.6)					0.8* (0.3, 1.3)
Oral	1.9	1.5	1970–2007	–0.6* (–0.8, –0.3)							–0.6* (–0.8, –0.3)
Cervix	7.3	1.9	1970–1976	–7.7* (–9.8, –5.6)	1976–2007	–3.1* (–3.3, –2.8)					–3.8* (–4.2, –3.5)
Stomach	11.3	2.9	1970–1977	–5.3* (–6.7, –3.9)	1977–2007	–3.1* (–3.3, –2.9)					–3.5* (–3.8, –3.2)
Kidney	2.2	2.3	1970–1978	–1.1 (–2.8, 0.8)	1978–1988	2.1* (0.8, 3.4)	1988–2007	–0.8* (–1.2, –0.5)			–0.1 (–0.6, 0.4)

Abbreviations: AAPC, average annual percent change; APC, annual percent change; ASMR, age-standardized mortality rates; CI, confidence interval.

[a] Excluding Quebec.

* Two-sided $p < .05$.

FIGURE 1
Average annual percent change in age-standardized incidence rates for selected cancers in males and females, Canada, 1970–2007



Note: Joinpoint analyses with up to 5 joinpoints were based on age-adjusted rates (per 100 000 persons).

[a] Excluding Quebec.

* Two-sided $p < .05$.

reversed in 1981 when incidence rates began decreasing (males: 0.7% per year; females: 0.4% per year). Mortality rates, on the other hand, have decreased over the entire study period for males (0.7% per year) and from 1970 to 1996 for females (1.2% per year).

The overall incidence rate of kidney cancer in males increased over two periods, 1977 to 1989 and 1998 to 2007. We observed two similar periods of increase in females, from 1980 to 1987 and from 1997 to 2007. In contrast, the male mortality rate increased from 1970 to 1992 but has been decreasing since then, while the female mortality rate increased from 1978 to 1988 and has since declined.

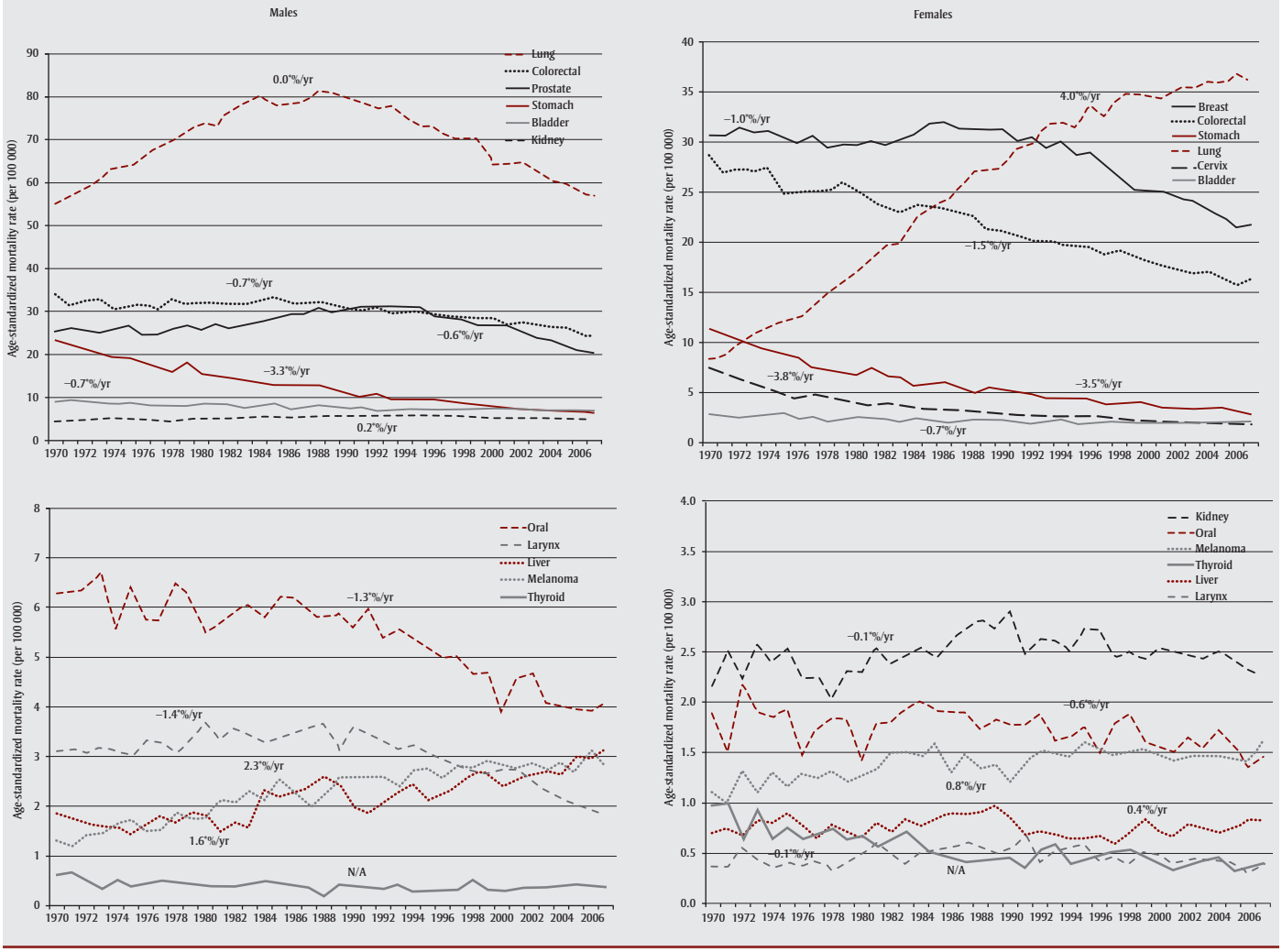
The incidence rate of thyroid cancer has been steadily increasing since 1970 in both sexes. In males, the rate increased at 2.4% per year between 1970 and 1997 and then accelerated to 6.9% per year until 2007. More notably, in females the incidence rate has varied from 1.8% per year between 1970 and 1989, 6.9% per year between 1989 and 1994, 12.5% per year between 1998 and 2002, and more recently (2002–2007), 6.9% per year. Thyroid cancer mortality rates were too low to permit a Joinpoint analysis.

Trends in survival

Between 1992 to 1994 and 2005 to 2007, the 5-year age-standardized RSR for all cancers combined rose by 6.8 percentage points to 62% (Table 3). Larger gains in survival were seen for males than females (8.5 vs. 5.0 percentage points) over this period, resulting in considerable narrowing in the previous gap.

The degree of improvement in the 5-year RSR varied considerably for individual cancers. The largest improvements of approximately 8 to 10 percentage points were for prostate, liver, colorectal and kidney cancers. Small improvements of 2

FIGURE 2
Average annual percent change in age-standardized mortality rates for selected cancers in males and females, Canada[a], 1970–2007



Abbreviation: N/A, not applicable.

Note: Joinpoint analyses with up to 5 joinpoints were based on age-adjusted rates (per 100 000 persons).

[a] Excluding Quebec.

* Two-sided $p < .05$.

to 3 percentage points were observed for lung, larynx, cervical and oral cancers. There was no apparent improvement for bladder cancer over the study period. Disparities between the sexes in survival gains favoured females (data not shown) and included oral (3.7% females vs. 1.1% males), larynx (4.1% vs. 1.5%), lung (3.5% vs. 1.3%) and stomach cancers (6.3% vs. 4.5%).

Discussion

Over the nearly 40-year period between 1970 and 2007, incidence rates for all cancers combined increased significantly in both Canadian males and females. While rates have stabilized in males since 1993, in females the overall incidence rate appears to have started plateauing only recently. These overall trends were driven largely by the three most common cancers in males (i.e. lung, prostate and colorectal) and in females (i.e. breast, lung and colorectal).

Cancer mortality rates in both sexes peaked in 1988 and have since declined largely due to reductions in mortality rates in the four leading causes of cancer death (i.e. lung, colorectal, prostate and breast cancers). The gains in 5-year RSR since the period 1992 to 1994 for all cancers combined and for selected cancers suggest improvements in treatment and early detection of certain cancers as well as advances in supportive and general medical care.

Trends in leading cancers

Prostate cancer

Little is known about the risk factors for prostate cancer aside from age. Although androgens are critical for prostate cancer growth, it is unclear whether high androgen levels can promote cancer initiation.[16] A link with physical activity has been

TABLE 3
Five-year age-standardized relative survival ratios for selected cancers[a] by time period, Canada[b], 1992–2007

Cancer type	RSR[c], % (95% CI)				Change[d]
	1992–1994	1996–1998	2000–2002	2005–2007	1992–1994 to 2005–2007
All cancers	56 (55–56)	57 (57–57)	60 (60–61)	62 (62–63)	6.8
Male	54 (54–54)	55 (55–56)	60 (60–60)	62 (62–63)	8.5
Female	57 (56–57)	58 (58–59)	60 (60–60)	62 (62–63)	5.0
Prostate	87 (86–87)	90 (89–90)	94 (94–95)	96 (96–97)	9.8
Female breast	82 (82–83)	85 (85–86)	87 (86–87)	88 (87–88)	5.6
Colorectal	56 (55–56)	58 (57–59)	61 (60–61)	64 (64–65)	8.6
Lung	14 (13–14)	15 (14–15)	15 (15–15)	16 (16–17)	2.6
Bladder	73 (72–74)	71 (70–72)	71 (70–72)	72 (71–73)	−0.3
Thyroid	93 (92–94)	94 (93–95)	96 (96–97)	98 (97–98)	4.9
Larynx	62 (60–64)	63 (61–65)	62 (59–64)	64 (61–66)	1.9
Liver	10 (8–11)	12 (11–14)	17 (15–18)	18 (17–20)	8.7
Melanoma	84 (83–86)	87 (86–87)	89 (88–90)	89 (89–90)	4.9
Oral	60 (59–61)	59 (58–61)	61 (59–62)	62 (61–64)	2.3
Cervix	70 (68–71)	70 (69–72)	73 (71–75)	72 (70–73)	2.2
Stomach	19 (18–21)	22 (20–23)	22 (21–23)	25 (23–26)	5.1
Kidney	60 (58–61)	62 (60–63)	64 (63–65)	67 (66–69)	7.7

Abbreviations: CI, confidence interval; RSR, relative survival ratio.

[a] For persons aged 15 to 99 years at diagnosis.

[b] Excluding Quebec.

[c] Results derived using both the cohort (1992–1994, 1996–1998 and 2000–2002) and period (2005–2007) methods.

[d] Absolute difference in percentage points.

suggested[17] but the evidence remains inconclusive.[18,19] Obesity is only weakly associated with the development of prostate cancer, but there is some suggestion that it could increase the risk of death and metastasis.[20–22]

Despite uncertainty about the benefits and risks of prostate cancer testing using the prostate-specific antigen (PSA) test, its use is widespread.[23] According to national health surveys, the proportion of males aged 35 plus who had ever had a PSA test was 53.8% in 2008.[24] Two recent randomized trials have not confirmed PSA as a viable population-based screening tool for reducing prostate cancer deaths,[25,26] and it is not currently recommended in Canada as a population-based screening test. Nonetheless, the prostate cancer incidence rate in Canada rose sharply following the introduction of the PSA test in 1988. The incidence rate peaked in 1993 and then again in 2001. This second date could be explained by the publicity that year surrounding the then federal health minister's disclosure that he had been diagnosed with prostate cancer.

The prostate cancer mortality rate in Canada has declined since 1995, returning to pre-1970 levels in 2007. Early detection of prostate cancer through widespread screening is believed to have contributed to the decreasing mortality trend in the United States,[27] and there is some suggestion that a similar phenomenon is responsible for the mortality and survival trends in Canada.[28] The nearly 10 percentage point gain in the 5-year RSR since 1992 to 1994 may also, to some degree, be explained by the greater availability of effective hormonal therapy for early and advanced-stage disease in the mid-1980s[29] followed by the introduction of watchful waiting and advances in combined radiation and hormonal therapy for prostate cancer which occurred in the 1990s.[30]

Breast cancer

The female breast cancer incidence rate in Canada rose steadily at 0.9% per year between 1970 and 1998, after which the rate started to decline at 0.7% per year. Trends in breast cancer incidence likely reflect long-term changes in hormonal factors (e.g. early age at menarche, late age at menopause, breastfeeding, oral contraceptive use, hormone replacement therapy use) and the increasing uptake of mammography screening, especially throughout the 1980s.[31] The first provincial organized breast cancer screening program was implemented in Canada in 1988, and all 10 Canadian provinces had established programs by 1998.[32] While all provincial programs offer mammography screening to women aged 50 to 69 years, some are also open to those in their 40s and those older than 69 years.[32]

National health surveys show that the proportion of postmenopausal females aged between 50 and 69 years who self-report having a mammogram within the previous two years has increased from 40.5% in 1990 to 72.5% in 2008.[32] The brief decline in the breast cancer incidence rate between 1998 and 2005 could be due to the exhaustion of undiagnosed prevalent cases as a result of screening and/or to a reduction in breast cancer risk as a result of postmenopausal women avoiding hormone replacement therapy following reports from the Women's Health Initiative and earlier investigations that highlighted the associated risks.[33]

Although postmenopausal obesity and alcohol consumption can increase breast cancer risk[34,35] and physical activity can reduce risk,[36] the impact of these factors in the Canadian context is not clear.

The breast cancer mortality rate started declining in 1986 at 1.0% per year and accelerated to 2.4% per year after 1994. The lower mortality and improved survival likely resulted from the increasing use of opportunistic mammography testing prior to the establishment of provincial screening programs, the increasing use of hormonal and adjuvant chemotherapy[37,38] and the shift in clinical practice to breast-conserving surgery and lumpectomy.[39,40]

Lung cancer

Smoking is a causal factor in the development of lung, oral cavity and larynx

cancers, among others.[41] The effects of smoking tobacco on lung cancer incidence are observed only after a latency period of approximately 25 years.[42] The prevalence of smoking in Canada has decreased substantially between 1965 and 2007 from 61% to 20% in males and from 38% to 18% in females aged 15 plus years.[43] After reaching a peak in 1965, tobacco use dropped in response to the widely publicized negative health effects of cigarette smoking reported by the U.S. Surgeon General.[44] This resulted in a decline in the male lung cancer incidence rate after this peaked in 1983 and in male mortality rate after this peaked in 1988. By 2007, the male lung cancer incidence rate had fallen to nearly the same level as in 1970.

The lung cancer mortality rate in females, on the other hand, has continued to increase, albeit at a slower pace since the mid-2000s. Smoking rates in females started to decrease about 15 years after those in males, remaining between 37% and 39% until 1979.[43] Though there is still an upward trend in lung cancer incidence and mortality rates in Canadian females, encouraging U.S. data show that the female lung cancer death rate in that country is decreasing following a plateau.[45]

Colorectal cancer

Colorectal cancer is associated with several modifiable risks including obesity, physical inactivity, consumption of red and processed meat and smoking.[46] The prevalence of obesity (i.e. body mass index $\geq$ 30 kg/m^2) in Canadian adults has increased from 13.8% to 23.9% over the 30 years until 2007/2009.[47,48] Prevalence was higher in females (15.9%) than in males (11.5%) in 1978/1979, but this pattern has now reversed such that slightly more males (24.2%) than females (23.6%) were considered obese in 2007/2009.[47,48]

The colorectal cancer incidence rate in males has returned to a level seen in the early 1980s, while in females the rate is now lower than that in the 1970s. The decline in the male death rate began in 1988, while in females the rate continued a decline that began before 1970. These differing trends suggest different risk factors. It has been suggested that increasing use of hormone replacement therapy in women prior to the early 2000s may have contributed to the declining risk of colon cancer in this sex.[34,49]

The decline in colorectal cancer death rates in both sexes began before the growing uptake of screening through the organized programs largely implemented across Canadian provinces in the past six years. Testing of occult blood in the stools of average-risk individuals aged 50 plus years[50] and colonoscopy for high-risk individuals have been the predominant approaches for the early detection and removal of pre-cancerous polyps,[51] aimed at lowering colorectal cancer incidence and mortality. Currently, the average participation rate for those aged between 50 and 74 years in provincial organized screening programs is 32.2%.[52] Greater uptake of screening will likely further reduce colorectal cancer incidence and mortality rates in Canada.

Emerging trends in other cancers

Thyroid cancer

Thyroid cancer has been one of the most rapidly increasing cancers in Canada in recent years.[1,53] The steep upward trend could be due to the increasing use of diagnostic technologies such as fine-needle aspiration for the detection of subclinical tumours, increased exposure to diagnostic ionizing radiation that could promote the initiation of new tumours, or increased exposure to an as yet unidentified environmental risk factor.[54,55] Ionizing radiation remains the most established risk factor for thyroid cancer, but mounting evidence points to a possible role of body weight and female reproductive factors, both of which probably operate in carcinogenesis through hormonal pathways. Despite the growing incidence rate, thyroid cancer mortality rates have remained low and the 5-year RSR in both sexes (98%) is the highest of all the major cancers.

Liver cancer

The most common type of primary liver cancer, hepatocellular carcinoma, is associated with low survival and high mortality. Between 1970 and 2007, incidence of liver cancer in Canada has increased faster in males (3.5% per year) than in females (1.9% per year). Gender differences in incidence may be due to the different distribution of liver cancer risk factors, such as heavy drinking (i.e. above the low-risk drinking guidelines[56]), smoking and hepatitis infection. Population-based estimates show that the heavy drinking rate in Canada has increased between 1989 and 2007 from 18.9% to 25% for males and 7.2% to 9.6% for females.[57,58]

In developed countries the greatest burden of liver cancer is due to chronic hepatitis C infection rather than hepatitis B virus (HBV) infection, which is more prevalent in other parts of the world.[59] A link has also been hypothesized between obesity and liver cancer, which is believed to occur through non-alcoholic fatty liver disease.[60] Such an association places greater importance on the increasing obesity rate in Canada, which has climbed from 14% in the late 1970s to 24% in 2007/2009.[47,48]

Melanoma

Ultraviolet radiation can cause all forms of skin cancer.[61] Although the increasing incidence of melanoma in Canada could be in part due to better detection,[62,63] it more likely reflects greater recreational UV exposure from sun and artificial tanning. The prevalence of tanning is about 49% in Canadian women and 28% in Canadian men aged 16 to 24 years according to the 2006 National Sun Survey.[64]

Of all the major cancers, melanoma has had the second greatest increase in mortality rate (after liver cancer in males and lung cancer in females) since 1970. Although the mortality rate in females has remained essentially unchanged since 1983, in males the mortality rate rose by 1.1% per year over a similar time period (1985–2007). The lower 5-year RSR[65] and the higher proportion of more advanced-stage cases in males[66] reflects the higher melanoma mortality rate in men compared with women. However, the upward rise in male mortality has been diminishing, possibly due to improved survival through earlier detection and better treatments for melanoma including surgical resection.[67]

Kidney

The reason behind the increasing kidney cancer incidence rate, while not clear, could reflect several changes including the availability of newer diagnostic techniques[68,69] as well as the increased prevalence of obesity and hypertension, both of which are important risk factors.[70] In fact, 55% of kidney cancers in Canadian males and 27% in females may be attributable to being overweight or obese.[71]

Trends in cancers with decreasing rates

Stomach, cervix, oral, larynx, bladder

Smoking is an important risk factor shared by stomach, oral, larynx, bladder and cervical cancers. The decreasing incidence and mortality trends for these cancers can be largely explained by trends in smoking, which dropped dramatically after 1965 for males and after 1979 for females in Canada.[43] Changes in other risk factors have also influenced observed trends. For example, the decline in stomach cancer rates since the 1970s resulted from improvements in diet including higher intakes of fruits and vegetables and lower intake of salt-preserved foods,[72] and more recently, an increased recognition and treatment of *Helicobacter pylori* bacterium infection, a key stomach cancer risk factor.[73]

Cervical cancer incidence and mortality rates in Canada continued to decline during the study period due to the widespread use of the Papanicolaou (Pap) test screening introduced in 1949.[74] As a complement to Pap screening, immunization of females aged 9 to 26 years with a human papillomavirus (HPV) vaccine (approved in Canada in 2008[75]) is expected to further reduce the long-term incidence and mortality rates. With the growing recognition of HPV in the etiology of certain oral cancers, such as those arising in the tonsils and oropharynx,[76,77] HPV immunization could also help shape future oral cancer trends in Canada.

Limitations

Our analysis had several limitations. First, we attempted to explain observed cancer trends with regard to population-based data on risk factors that are largely cross-sectional and mostly self-reported. Second, because of data availability, we were able to consider only a subset of modifiable lifestyle factors that influence disease rates. Moreover, we considered only modifiable risk factors that may be etiologically relevant to adult-onset cancers but not those unique to pediatric and adolescent cancers. Third, the data sources, methods for cancer registration, as well as completeness and accuracy of data used for deriving incidence estimates can vary across Canada.[1] Such differences can lead to minor under- and overestimates of disease rates, which are discussed more fully elsewhere.[1] Finally, relative survival estimates for the years 2005 to 2007 may be overestimated due to the necessity of using expected survival data from an earlier time period in their derivation. The effect would likely be greatest for cancers with older case distributions such as prostate cancer.

Conclusion

The downward trends in incidence rates for certain cancers and mortality rates for most cancers in Canada support the success of various strategies including cancer screening, prevention through lifestyle and behavioural changes, and improvements in environmental hygiene. Despite these successes, the need for reinforcing primary prevention remains important as several cancers continue to show stable or rising incidence trends.

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Conflict of interest: none.

References

1. Canadian Cancer Society's Steering Committee on Cancer Statistics. Canadian Cancer Statistics 2011. Toronto (ON): Canadian Cancer Society; 2011.

2. Statistics Canada. Leading causes of death in Canada, 2009 [Internet]. Ottawa (ON): Minister of Industry; 2012 [cited 2012 Sept 19] [Statistics Canada, Catalogue No.: 84-215-X). Available from: http://www.statcan.gc.ca/bsolc/olc-cel/olc-cel?catno=84-215-x&lang=eng

3. Statistics Canada. Population projections for Canada, provinces and territories, 2009 to 2036 [Internet]. Ottawa (ON): Minister of Industry; 2010 [cited 2011 Aug 5]. [Statistics Canada, Catalogue No.: 91-250-X]. Available from: http://www.statcan.gc.ca/daily-quotidien/100526/dq100526b-eng.htm

4. Statistics Canada. Cancer incidence in Canada 2007 and 2008 [Internet]. Ottawa (ON): Minister of Industry; 2010 [cited 2011 Aug 5]. [Statistics Canada, Catalogue No.: 82-231-X]. Available from: http://www.statcan.gc.ca/pub/82-231-x/82-231-x2009001-eng.htm

5. Band P, Gaudette LA, Hill GB, Holowaty EJ, Huchcroft SA, Johnston GM, et al. The making of the Canadian Cancer Registry: cancer incidence in Canada and its regions, 1969-1988. Ottawa (ON): Canadian Council of Cancer Registries, Health and Welfare Canada, Statistics Canada, 1993.

6. Statistics Canada. Demographic estimates compendium, 2010. CANSIM Table 051-0001, released on September 29, 2010. Ottawa (ON): Minister of Industry; 2010.

7. Parkin DM, Chen VW, Ferlay J, Galceran J, Storm HH, Whelan SL. Comparability and quality control in cancer registration. Lyon (FR): World Health Organization; 1994.

8. Fritz A, Percy C, Jack A, Shanmugarathan K, Sobin L, Parkin DM, et al., editors. International classification of diseases for oncology, 3rd ed. Geneva (CH): World Health Organization; 2000.

9. International statistical classification of diseases and related health problems, 10th rev. Geneva (CH): World Health Organization; 1992.

10. Joinpoint regression program, v.3.5.1 [computer program]. Rockville (MD): Statistical Research and Applications Branch, National Cancer Institute; 2011.

11. Kim HJ, Fay MP, Feuer EJ, Midthune DN. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med. 2000;19:335–51.

12. Dickman PW. Estimating and modelling relative survival using SAS [Internet]. [Stockholm (SE)]: [publisher unknown]; [cited 2010 Jan 4]. Available from: http://www.pauldickman.com/rsmodel/sas_colon

13. Ellison LF, Gibbons L; Canadian Cancer Survival Analysis Group. Five-year relative survival from prostate, breast, colorectal and lung cancer. Health Rep. 2001;13(1): 23–34.

14. Louchini R. La survie au cancer pour les nouveaux cas déclarés au Québec en 1992. Quebec (QC): Institut national de santé publique du Québec; 2002.

15. Cancer Survival Statistics [Internet]. Ottawa (ON): Statistics Canada; 2011 [cited 2011 Jul 5]. [Statistics Canada, Catalogue No.: 82-226-X]. Available from: http://www.statcan.gc.ca/pub/82-226-x/82-226-x2011001-eng.htm

16. Wang D, Tindall DJ. Androgen action during prostate carcinogenesis. Methods Mol Biol. 2011;776:25–44.

17. Vainio H, Bianchini F, editors. Weight control and physical activity. Lyon (FR): International Agency for Research on Cancer; 2002. (Vainio H, editor. IARC handbook of cancer prevention; vol 6).

18. Pan SY, DesMeules M. Energy intake, physical activity, energy balance, and cancer: epidemiologic evidence. Methods Mol Biol. 2009;472:191–215.

19. Friedenreich CM, Neilson HK, Lynch BM. State of the epidemiological evidence on physical activity and cancer prevention. Eur J Cancer. 2010;46(14):2593–604.

20. MacInnis RJ, English DR. Body size and composition and prostate cancer risk: systematic review and meta-regression analysis. Cancer Causes Control. 2006;17(8): 989–1003.

21. Giovannucci E, Liu Y, Platz EA, Stampfer MJ, Willett WC. Risk factors for prostate cancer incidence and progression in the health professionals follow-up study. Int J Cancer. 2007;121(7):1571–8.

22. Gong Z, Agalliu I, Lin DW, Stanford JL, Kristal AR. Obesity is associated with increased risks of prostate cancer metastasis and death after initial cancer diagnosis in middle-aged men. Cancer. 2007;109(6): 1192–202.

23. Levy I. Prostate cancer: the epidemiologic perspective. Can J Oncol. 1994;4 Suppl 1:4–7.

24. Statistics Canada. Canadian Community Health Survey, Cycle 4.1, 2007 [computer file]. Ottawa (ON): Health Statistics Division; 2007.

25. Andriole GL, Crawford ED, Grubb RL 3rd, Buys SS, Chia D, Church TR, et al. Mortality results from a randomized prostate-cancer screening trial. N Engl J Med. 2009;360(13): 1310–9.

26. Schroder FH, Hugosson J, Roobol MJ, Tammela TL, Ciatto S, Nelen V, et al. Screening and prostate-cancer mortality in a randomized European study. N Engl J Med. 2009;360(13):1320–8.

27. Hankey BF, Feuer EJ, Clegg LX, Hayes RB, Legler JM, Prorok PC, et al. Cancer surveillance series: interpreting trends in prostate cancer--part I: evidence of the effects of screening in recent prostate cancer incidence, mortality, and survival rates. J Natl Cancer Inst. 1999;91(12):1017–24.

28. Nam RK, Klotz LH. Trends in prostate cancer screening: Canada. In: Ankerst DP, Tangen CM, Thompson IM, editors. Current clinical urology: prostate cancer screening, 2nd ed. New York (US): Humana Press; 2009. pp. 23–33.

29. Meng MV, Grossfeld GD, Sadetsky N, Mehta SS, Lubeck DP, Carroll PR. Contemporary patterns of androgen deprivation therapy use for newly diagnosed prostate cancer. Urology. 2002;60 (3 Suppl 1):7–11.

30. Kupelian PA, Buchsbaum JC, Elshaikh MA, Reddy CA, Klein EA. Improvement in relapse-free survival throughout the PSA era in patients with localized prostate cancer treated with definitive radiotherapy: year of treatment an independent predictor of outcome. Int J Radiat Oncol Biol Phys. 2003;57(3):629–34.

31. Holford TR, Cronin KA, Mariotto AB, Feuer EJ. Changing patterns in breast cancer incidence trends. J Natl Cancer Inst Monogr. 2006;(36):19–25.

32. Shields M, Wilkins K. An update on mammography use in Canada. Health Rep. 2009;20(3):7–19.

33. De P, Neutel CI, Olivotto I, Morrison H. Breast cancer incidence and hormone replacement therapy in Canada. J Natl Cancer Inst. 2010;102(19):1489–95.

34. Reeves GK, Pirie K, Beral V, Green J, Spencer E, Bull D. Cancer incidence and mortality in relation to body mass index in the Million Women Study: cohort study. BMJ. 2007;335(7630):1134.

35. Alcohol consumption and ethyl carbamate. Lyon (FR): International Agency for Research on Cancer; 2010. (IARC monographs on the evaluation of carcinogenic risks to humans; vol 96).

36. Friedenreich C. The role of physical activity in breast cancer etiology. Semin Oncol. 2010;37(3):297–302.

37. Harlan LC, Abrams J, Warren JL, Clegg L, Stevens J, Ballard-Barbash R. Adjuvant therapy for breast cancer: practice patterns of community physicians. J Clin Oncol. 2002;20(7):1809–17.

38. Mariotto A, Feuer EJ, Harlan LC, Wun LM, Johnson KA, Abrams J. Trends in use of adjuvant multi-agent chemotherapy and tamoxifen for breast cancer in the United States: 1975–1999. J Natl Cancer Inst. 2002;94(21):1626–34.

39. Edwards BK, Brown ML, Wingo PA, Howe HL, Ward E, Ries LA, et al. Annual report to the nation on the status of cancer, 1975–2002, featuring population-based trends in cancer treatment. J Natl Cancer Inst. 2005;97(19):1407–27.

40. NIH consensus conference. Treatment of early-stage breast cancer. JAMA. 1991; 265(3):391–5.

41. Tobacco smoke and involuntary smoking. Summary of data reported and evaluation. Lyon (FR): International Agency for Research on Cancer; 2004. (IARC monographs on the evaluation of carcinogenic risks to humans; vol 83.)

42. Kleinsmith LJ, editor. Principles of cancer biology, 1st edition. San Francisco (CA): Benjamin Cummings; 2005.

43. Reid J, Hammond D. Tobacco use in Canada: patterns and trends, 2011 edition. Waterloo (ON): Propel Centre for Population Health Impact, University of Waterloo; 2011.

44. The Surgeon General's Advisory Committee on Smoking and Health. Smoking and health: report of the Advisory Committee to the Surgeon General of the Public Health Service. Washington (DC): US Department of Health, Education and Welfare, 1964. (Public Health Service Publication, No. 1103.)

45. Kohler BA, Ward E, McCarthy BJ, Schymura MJ, Ries LA, Eheman C, et al. Annual report to the nation on the status of cancer, 1975–2007, featuring tumors of the brain and other nervous system. J Natl Cancer Inst. 2011;103(9):714–36.

46. Food, nutrition, physical activity, and the prevention of cancer: a global perspective [Internet]. Washington (DC): World Cancer Research Fund/American Institute for Cancer Research; 2007 [cited 2012 Sept 19]. Available from: http://www.dietandcancerreport.org/cancer_resource_center/downloads/Second_Expert_Report_full.pdf

47. Tjepkema M. Adult obesity. Health Rep. 2006;17(3):9–25.

48. Statistics Canada. Canadian Health Measures Survey: Cycle 1 data tables. Ottawa (ON): Minister of Industry; 2010.

49. Newcomb PA, Zheng Y, Chia VM, Morimoto LM, Doria-Rose VP, Templeton A, et al. Estrogen plus progestin use, microsatellite instability, and the risk of colorectal cancer in women. Cancer Res. 2007;67(15):7534–9.

50. Mandel JS, Church TR, Bond JH, Ederer F, Geisser MS, Mongin SJ, et al. The effect of fecal occult-blood screening on the incidence of colorectal cancer. N Engl J Med. 2000;343(22):1603–7.

51. Schatzkin A, Freedman LS, Dawsey SM, Lanza E. Interpreting precursor studies: what polyp trials tell us about large-bowel cancer. J Natl Cancer Inst. 1994;86(14): 1053–7.

52. Pan-Canadian System Performance Working Group. 2010 system performance report. Toronto (ON): Canadian Partnership Against Cancer; 2010.

53. Liu S, Semenciw R, Ugnat AM, Mao Y. Increasing thyroid cancer incidence in Canada, 1970–1996: time trends and age-period-cohort effects. Br J Cancer. 2001; 85(9):1335–9.

54. Kent WD, Hall SF, Isotalo PA, Houlden RL, George RL, Groome PA. Increased incidence of differentiated thyroid carcinoma and detection of subclinical disease. CMAJ. 2007;177(11):1357–61.

55. How J, Tabah R. Explaining the increasing incidence of differentiated thyroid cancer. CMAJ. 2007;177(11):1383–4.

56. Butt P, Beirness D, Cesa F, Gliksman L, Paradis C, Stockwell T. Alcohol and health in Canada: a summary of evidence and guidelines for low-risk drinking. Ottawa (ON): Canadian Centre on Substance Abuse; 2011.

57. CANSIM Table 105–0501. Health indicator profile, annual estimates, by age group and sex, Canada, provinces, territories, health regions (2011 boundaries) and peer groups, occasional [data file]. Ottawa (ON): Statistics Canada; [cited 2012 Sept 19]. Available from: http://www5.statcan.gc.ca/cansim/a05?lang=eng&id=1050501

58. Ahmad N, Flight J, Singh VA, Poole N, Dell CA. Focus on gender - a national survey of Canadians' use of alcohol and other drugs - Canadian Addiction Survey (CAS). Ottawa (ON): Health Canada; 2008.

59. Parkin DM. The global health burden of infection-associated cancers in the year 2002. Intl J Cancer. 2006;118(12):3030–44.

60. Chuang SC, La Vecchia C, Boffetta P. Liver cancer: descriptive epidemiology and risk factors other than HBV and HCV infection. Cancer Lett. 2009;286(1):9–14.

61. Solar and ultraviolet radiation. Lyon (FR): International Agency for Research on Cancer; 1992. (IARC monographs on the evaluation of carcinogenic risks to humans; vol 55.)

62. Jemal A, Devesa SS, Hartge P, Tucker MA. Recent trends in cutaneous melanoma incidence among whites in the United States. J Natl Cancer Inst. 2001;93(9):678–83.

63. Welch HG, Woloshin S, Schwartz LM. Skin biopsy rates and incidence of melanoma: population based ecological study. BMJ. 2005;331(7515):481.

64. The Ontario Sun Safety Working Group. Insight on cancer: sun exposure and protective behaviours in Ontario. An Ontario report based on the 2006 Second National Sun Survey. Toronto (ON): Canadian Cancer Society; 2010 [cited 2011 Jun 13]. Joint publication of Cancer Care Ontario. Available from: http://uvnetwork.ca/InsightonCancerSunExposureandProtectiveBehavioursinOntarioFinal.pdf

65. Joosse A, de Vries E, Eckel R, Nijsten T, Eggermont AM, Holzel D, et al. Gender differences in melanoma survival: female patients have a decreased risk of metastasis. J Invest Dermatol. 2011;131(3):719–26.

66. Howlader N, Noone AM, Krapcho M, Neyman N, Aminou R, Waldron W, et al., editors. SEER Cancer Statistics Review, 1975–2008. Bethesda (MD): National Cancer Institute; 2011 [cited 2011 Dec 13]. Available from: http://seer.cancer.gov/csr/1975_2008/

67. Morton DL. Current management of malignant melanoma. Ann Surg. 1990;212(2): 123–4.

68. Jayson M, Sanders H. Increased incidence of serendipitously discovered renal cell carcinoma. Urology. 1998;51(2):203–5.

69. Leslie JA, Prihoda T, Thompson IM. Serendipitous renal cell carcinoma in the post-CT era: continued evidence in improved outcomes. Urol Oncol. 2003;21(1):39–44.

70. Chow WH, Gridley G, Fraumeni JF Jr, Jarvholm B. Obesity, hypertension, and the risk of kidney cancer in men. N Engl J Med. 2000;343(18):1305–11.

71. Pan SY, Johnson KC, Ugnat AM, Wen SW, Mao Y. Association of obesity and cancer risk in Canada. Am J Epidemiol. 2004; 159(3):259–68.

72. Howson CP, Hiyama T, Wynder EL. The decline in gastric cancer: epidemiology of an unplanned triumph. Epidemiol Rev. 1986;8(1):1–27.

73. Schistosomes, liver flukes and Helicobacter pylori. Lyon (FR): International Agency for Research on Cancer; 1994. (IARC monographs on the evaluation of carcinogenic risks to humans; vol 61.)

74. Liu S, Semenciw R, Probert A, Mao Y. Cervical cancer in Canada: changing patterns in incidence and mortality. Int J Gynecol Cancer. 2001;11(1):24–31.

75. National Advisory Committee on Immunization. Statement on human papillomavirus vaccine. Can Commun Dis Rep. 2007;33(2):1–32.

76. A review of human carcinogens: biological agents. Lyon (FR): International Agency for Research on Cancer; 2012. (IARC monographs on the evaluation of carcinogenic risks to humans; vol 100b.)

77. Auluck A, Hislop G, Bajdik C, Poh C, Zhang L, Rosin M. Trends in oropharyngeal and oral cavity cancer incidence of human papillomavirus (HPV)-related and HPV-unrelated sites in a multicultural population: the British Columbia experience. Cancer. 2010;116(11):2635–44.

Parenting disability, parenting stress and child behaviour in early inflammatory arthritis

*P. Zelkowitz, EdD (1); K. J. Looper, MD (1); S. S. Mustafa, PhD (1); M. Purden, PhD (2); M. Baron, MD (3); McGill Early Arthritis Research Group**

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Abstract

Introduction: Our study examines the association between the disease characteristics of inflammatory arthritis and patients' self-perception of mental health, parenting disability, parenting stress and child behaviour in early inflammatory arthritis (EIA).

Methods: Patients in the early phase (more than 6 weeks, less than 18 months) of inflammatory arthritis were recruited from a larger EIA registry that recorded sociodemographic data and measures of pain, physical functioning and disease activity. Patient-perceived parenting disability, parenting stress, depression and children's behaviour problems were assessed using the Parenting Disability Index, Parenting Stress Index, Center for Epidemiologic Studies - Depression Mood Scale and Child Behavior Checklist, respectively.

Results: Pain, physical dysfunction, number of tender joints and physician global assessment of disease activity were associated with parenting disability. Self-report measures of parenting disability were associated with those of depression and parenting stress. Parenting stress was associated with children internalizing and externalizing behaviour problems while parenting disability was associated with children externalizing behaviour problems.

Conclusion: This study suggests a possible reciprocal relationship among physical aspects of disease activity, parenting disability and parent and child distress in EIA.

Keywords: *parenting disability, parenting stress, child behaviour, arthritis, physical functioning, pain*

Introduction

Arthritis comprises more than a hundred rheumatic conditions involving joints and their surrounding tissues. Inflammatory arthritis occurs when joints get inflamed because of immune system disruptions. It is a painful disabling condition associated with impairment in psychological and social functioning.[1,2] Rheumatoid arthritis (RA) is the most common form of inflammatory arthritis and is characterized by chronic destructive synovitis. Undifferentiated arthritis is one that does not fulfill disease classification criteria and can ultimately either resolve or else evolve to full-blown rheumatoid arthritis.

Physical illness in a parent can affect children in many ways. The parent may be unable to perform usual childcare tasks and may, in fact, expect children to take on additional household responsibilities.[3] Moreover, the parent may be emotionally unavailable due to pain, fatigue and preoccupation with the disease.[4,5] Family stress related to possible loss of income or marital conflict around changing roles and division of labour may also take its toll on the parent-child relationship.[6] Children's adjustment is most likely to be affected if the ill parent exhibits psychological distress[4] and if parenting behaviour is affected.[5]

Research in families with a parent suffering from arthritis has been limited, but what is available indicates significant negative effects. For example, an exploratory study by Grant et al.[7] showed that parents and grandparents with RA experience difficulties with instrumental childcare tasks such as lifting a child. Katz et al.[8] found that women with RA experience disability in parenting activities and hence perform fewer parenting functions. Backman et al[9] qualitatively examined the experiences of mothers living with arthritis and described the impact of inflammatory arthritis on motherhood as dramatic, with both positive and negative influences. They described participation in the mothering role as fluctuating and influenced by the support available and the unpredictable balance of fatigue and energy.[9] This resulted in the family being more cohesive at certain times and feeling regret about lost family activities at other times.[9]

* M. Starr, MD; M. Gagné, MD; M. Stein, MD; H. Kang, MD; M. Kapusta, MD; F. Couture, MD; M. A. Fitzcharles, MD; B. Garfield, MD; H. A. Ménard, MD; L. Berkson, MD; C. Pineau, MD; A. Gutkowski, MD; M. Zummer, MD; J. P. Mathieu, MD; S. Mercille, MD; S. Ligier, MD; J. Krasny, MD; C. Bertrand, MD; S. Y. Yuen, MD; J. Schulz, MD.

Author references:

1. Department of Psychiatry, McGill University, Montréal, Quebec, Canada
2. Department of Nursing, McGill University, Montréal, Quebec, Canada
3. Department of Rheumatology, McGill University, Montréal, Quebec, Canada

Correspondence: Karl Looper, Department of Psychiatry, McGill University, Jewish General Hospital, 3755 Chemin de la Côte-Ste-Catherine, Montréal, QC H3T 1E2; Tel.: 514-340-8222; Fax: 514-230-8126; Email: karl.looper@mcgill.ca

Arthritis patients often perceive themselves as functioning inadequately with regard to their children and grandchildren.[10] One study reported that children of RA patients had more daily hassles, smaller social support networks and poorer social adjustment than did the controls.[11] Adolescent children of a parent with RA reported lower self-esteem than those of well parents.[12]

Much of the research in this area has consisted of small, qualitative studies. Moreover, it usually involved patients who have been ill for many years. However, parenting issues may be different in early versus longstanding inflammatory arthritis. Clearly needed is research examining the relationship between parenting and children, mental health, and illness outcomes early in the disease trajectory, with the aim of developing treatment interventions directed at specific factors in the early stage of illness to improve subsequent health and parenting outcomes.

To that end, we undertook this study to explore the associations between disease variables such as pain, physical dysfunction and disease activity, mental distress, parenting factors such as parenting ability and parenting stress, and child behavioural measures during the first 18 months of the parents' inflammatory arthritis.

Methods

Participants

Two hundred and fifty-seven patients were enrolled in the McGill Early Arthritis Registry (McEAR) between March 2006 and May 2009. Referrals to the McEAR come from 21 rheumatologists working in Montréal, Quebec. The participating rheumatologists, each of whom works in a private office or outpatient hospital clinic, were asked to recruit all new patients with early inflammatory arthritis (EIA) who fit the inclusion criteria, that is, they had newly diagnosed inflammatory arthritis, defined as one or more joints with inflammation that lasted a minimum of 6 weeks to a maximum of 18 months. (Disease duration was assessed by asking the patient ''When did you first start to have this episode of joint pain?'' and ''Have you ever had episodes of similar pain and swelling in your joints before this episode?'') Patients were 18 years or older, spoke English or French, and agreed to periodic physical and laboratory examinations as well as to completing questionnaires assessing demographics, disability, pain and psychosocial factors related to their illness. Exclusion criteria included clinical evidence of remote joint damage suggestive of a previous episode of RA, any rheumatic diagnosis other than RA or undifferentiated inflammatory arthritis, severe functional limitation from a disease other than arthritis, and any disorder that compromised the ability to give informed consent.

Of the 257 patients enrolled in the McEAR, 80 had children younger than 18 years of age living with them. All the 257 patients enrolled in the McEAR were invited to participate in psychosocial studies, which involved a home visit by interviewers, and 104 (40.5%) agreed to do so. Of the 104 participants, 29 had children aged less than 18 years living with them. All of those 29 parent participants agreed to participate and constituted the sample for this study, a response rate of 36% (29/80 of the McEAR participants who had children under 18 years) when considering the McEAR as a whole and 100% of those who agreed to the psychosocial study.

All patients in the registry were seen by one of the two study nurses who met them at the office of the referring rheumatologist with whom the clinical care remained. (This made it easier for those patients who lived on or near the island of Montreal.) Each nurse was trained to perform a complete tender and swollen joint count. Nurses, physicians and patients were not blind to one another's evaluations.

A trained interviewer, blind to patients' information recorded in the main registry database, arranged to see the patient at home within 10 days of the registry visit for this study. The trained interviewer obtained consent to proceed with the interview and assisted the patients in filling out the study-specific questionnaires. Patients received $25 for each meeting with the study interviewer. All patients in the McEAR signed an informed consent and the study was approved by the Institutional Review Boards of McGill University and the Jewish General Hospital.

Illness outcome measures

Physical functioning

We measured physical functioning using the Medical Outcomes Study Short-Form 36 (SF-36)[13] which, with its good psychometric properties, has been frequently used to measure health-related quality of life. It consists of eight domains: physical functioning, social functioning, role limitations (physical problems), role limitations (emotional problems), mental health, vitality, bodily pain and general health perceptions. Scores range from 0 (worst) to 100 (best). Although the 8 scales are combined into 2 summary measures, one of which, the Physical Component Score (SF36-PCS), provides an overall estimate of physical health, we chose to measure physical function using the physical functioning domain score. We measured pain more specifically using the McGill Pain Questionnaire (MPQ). [14]

Pain

Pain was assessed using the Short-Form MPQ,[15] which contains 11 items related to the sensory dimension of pain and 4 related to the affective dimension. Each descriptor is ranked on a four-point intensity scale (0–3; none to severe), and total scores range from 0 (no pain) to 45 (severe pain). The MPQ has been extensively used and has sound psychometric properties. The total pain score was used in this study.

Disease activity

We assessed disease activity using joint counts, with the number of swollen and tender joints determined according to the American College of Rheumatology joint count.[16] We also measured blood level of the acute-phase reactant, C-reactive protein (CRP). Number of swollen joints, number of tender joints, patient global assessment of disease activity and blood CRP level together make up the Disease Activity Score in 28 joints (DAS28).[17–20]

DAS28, based on 28 specific joints that are a subset of the American College of Rheumatology criteria, was developed[17] and validated[18] for patients with RA and is considered to be a valid measure of disease activity.[18] The validation criteria included correlations with a selected group of other disease variables (correlational validity), with physical disability and judgment of a group of rheumatologists in clinical practice (criterion validity I and II, respectively) and with the radiographically determined damage of hands and feet (construct validity).[18] DAS28, however, excludes joints of the ankles and feet.[21]

In our study, we completed the patient global assessment of disease activity using an 11-point visual analogue scale that ranged from 0 (best) to 10 (worst) with reference to the past week. The rheumatologist rated the physician global assessment of disease activity using a single 11-point numerical rating scale where 0 meant ''no arthritis activity'' and 10 meant ''worst arthritis.''

Psychosocial measures and parenting and child functioning measures

Center for Epidemiologic Studies - Depression Mood Scale

The Center for Epidemiologic Studies – Depression Mood Scale (CES-D) is a 20-item self-report scale designed to measure depression in the general population.[22] Answers are based on how frequently in the previous week each item was experienced. Scores range from 0 to 60, with higher scores indicating greater depression. A cut-off score of 16 is the requirement for identifying depression, but in chronic disease such as RA, cut-offs of 19 have been recommended.[23,24] In our study, we used the total score (continuous variable) to measure symptom severity.

Parenting Stress Index

The Parenting Stress Index (PSI) (Short Form)[25] is a 36-item self-report measure assessing parental distress. It has 3 subscales, Parental Distress, Parent-Child Dysfunctional Interaction and Difficult Child. Each item is rated from 1 (''strongly disagree'') to 5 (''strongly agree''). Higher scores indicate greater stress. Cronbach's α ranges from .88 to .95, and the scale has construct validity compared to measures of children's behaviour problems.[25] We used the total score in this study.

Parenting Disability Index

The Parenting Disability Index (PDI) was developed as a measure of parenting function and disability and validated in women with RA.[8] The 27 items on the scale are scored from 0 to 3, with 0 meaning no disability and 3 meaning inability to perform parenting tasks. The PDI is the mean difficulty level across the domains within the parent cohort/child age group. Two summary scores were developed; the summary score used in this study is the modified PDI (MPDI).[8] This scale was validated in an RA population.

Child Behavior Checklist

The Child Behavior Checklist (CBCL)[26,27] was used to assess children's behaviour problems, using the relevant versions for children aged 1.5 to 5 years and 6 to 18 years. Parents rate their children's behaviour over the previous 2 months. Items are scored 0 for ''not true'' to 2 for ''very or often true.'' Both versions have 3 broadband (summary) scores representing internalizing problems, externalizing problems and total problems. Internalizing problems include anxiety, depression and somatic symptoms, while externalizing problems include conduct problems, hyperactivity and aggression. Test-retest reliabilities range from .88 to .91 for the broadband scales, and the interrater reliability for the CBCL is .72 for internalizing problems and .85 for externalizing problems.[26] We used internalizing and externalizing scores for both age groups. To obtain comparable data, if there were 2 or more children residing at home, parents were asked to rate the behaviour of the child closest in age to 10 years.

Statistical analysis

Because of the small sample size (n = 29), data analyses in this study must be considered exploratory. Data were analyzed using SPSS version 17 for Windows (IBM, Chicago, IL, US). Descriptive statistics were used to summarize baseline characteristics of the EIA patients. Pearson correlations were used to calculate the associations between the continuous study variables. Missing data was not imputed, and we did not include cases for which data were missing in our calculations.

Results

The mean age of our study participants was almost 42 years; 20 (69%) were female and 23 (79%) were married/cohabiting. A total of 21 (72%) participants were employed; 14 were working full time, 3 were on sick leave, 2 were working part time and 2 were self-employed. The total yearly household income of more than half of the participants was over $60,000. The sample was highly educated with 20 (69%) college, university or postgraduate educated. Out of our 29 participants, 15 (52%) had 1 child, 10 (35%) had 2 children and 4 (14%) had 3 children aged under 18 years living at home. Our participants had an average disease duration (standard deviation) of 8.24 (3.65) months. Four participants (14%) had a CES-D score above the cut-off of 19. (See Table 1.)

The average age of the target child was 10.6 years. The proportion of boys and girls was almost equally distributed along the sample, and 48% of the target children (those closest in age to 10 years) were girls. Three children scored above the clinical cut-off of 60 on CBCL internalizing and externalizing problems.

Parenting stress (PSI) was significantly correlated only to CES-D total depressive mood score. On the other hand, patient-perceived parenting disability (MPDI) was correlated to all the disease measures: physician global assessment of disease activity, number of tender joints, physical dysfunction (SF-36) and pain (MPQ) in addition to depressive mood (CES-D). Patient-perceived children's externalizing and internalizing behaviour problems (CBCL, all ages) showed significant correlation to physician global assessment of disease activity. Parental depressive symptoms were not associated with children's behaviour problems. Table 2 shows the

TABLE 1
Characteristics of study participants (N = 29)

Study variable	Frequency, n (%)	Mean (SD)	95% Confidence interval
Demographics			
Age, years		41.97 (7.95)	26–57
Female	20 (69)		
Married/co-habiting	23 (79)		
Employed	21 (72)		
Yearly household income			
< $60,000	11 (38)		
≥ $60,000	17 (59)		
Education level			
High school or less	9 (31)		
College or more	20 (69)		
Number of children			
1	15 (52)		
2	10 (35)		
3	4 (14)		
Age of target child[a]		10.60 (5.10)	1.00–18.00
Gender of target child[a]: girl	13 (48)		
Disease characteristics			
Duration, months		8.24 (3.65)	4.00–18.00
SF-36 physical functioning		60.04 (28.90)	5.00–100.00
MPQ total pain		8.60 (11.17)	0.00–45.00
Number of swollen joints		9.19 (9.56)	0.00–39.00
Number of tender joints		16.00 (12.76)	0.00–43.00
CRP, mg/L		22.87 (22.49)	0.30–69.00
DAS28		5.30 (1.80)	2.61–8.08
Physician global assessment of disease activity		3.96 (2.71)	0.00–10.00
Psychosocial variables			
CES-D total depressive mood		10.03 (10.58)	0.00–41.00
PSI		63.89 (19.43)	36.00–100.00
MPDI for all ages		0.65 (0.61)	0.00–1.95
CBCL, externalizing problems, all ages		47.96 (8.18)	34.00–65.00
CBCL, internalizing problems, all ages		50.55 (11.39)	33.00–78.00

Abbreviations: CBCL, Child Behavior Checklist; CES-D, Center for Epidemiologic Studies - Depression Mood Scale; CRP, C-reactive protein; DAS28, Disease Activity Score in 28 joints; MPDI, Modified Parenting Disability Index; MPQ, McGill Pain Questionnaire; PSI, Parenting Stress Index; SF-36, Medical Outcomes Study Short Form 36; SD, standard deviation.

[a] To obtain comparable data, if there were 2 or more children residing at home, parents were asked to rate the behaviour of the child closest in age to 10 years.

relationships between parents' physical and mental health and parenting and child behaviour problems.

Parenting stress (PSI) was significantly correlated to parenting disability (MPDI) and children's internalizing and externalizing (CBCL) problems. Patient-perceived parenting disability (MPDI) showed significant correlation to children's externalizing (CBCL) problems in addition to parenting stress. Table 3 shows the interrelationships between parenting and child behaviour variables.

Other disease-related variables including the number of swollen joints, CRP and the DAS28 did not show significant correlations with any of the parenting or child behaviour measures.

Discussion

Parenting is both physically and emotionally extremely demanding.[28] Given the scarcity of literature on how chronically ill arthritis patients who have young children manage both their illness and parenting, we sought to examine the relationship between arthritis in its early stages (between 6 weeks and 18 months) and patients' self-rated distress, ability to perform parenting tasks and perception of their child's behaviour.

The main findings of this study indicate that arthritis, even in its early stages, does interfere with parenting. Our patients suffered increased parenting disability with the increase in multiple measures of disease activity including pain, physical dysfunction, number of tender joints and physicians' assessment of disease activity. Patients reported having difficulties with, among others, bending, outdoor activities or having other children in their home. This perceived inability to parent adequately may have resulted in feelings of distress. This is suggested by the fact that parenting disability was highly correlated with parenting stress and both of these factors were related to CES-D depressive mood score.

These findings are consistent with previous research in more advanced RA patients.[7,8] These studies found that many patients had problems with parenting, particularly with tasks related to physical care activities such as lifting a child from the floor and keeping up with children. These problems are often attributed to physical as well as psychological issues such as anxiety, depression and guilt. White et al.[29] also found that for mothers with RA, more fatigue was a significant predictor of greater frequency and intensity of daily parenting problems and greater difficulty monitoring their children's whereabouts.[29] Mothers with RA had more problems monitoring their child if they were more depressed and experiencing an exacerbation.[29] Our results also agree with those of a very recent study[30] that investigated the impact of systemic lupus erythematosus on mothering abilities. The authors reported that greater fatigue and func-

TABLE 2
Pearson product moment correlations (*r*) among study variables[a]

Statistics		Parental health disease measure				
		CES-D total depressive mood	Physician global assessment of disease activity	Number of tender joints	SF-36 physical functioning	MPQ total pain
Parenting variables						
Parenting Stress Index (PSI)	*r*	.565**	.360	.039	−.149	.045
	p	.002	.077	.874	.488	.834
	n	27	25	19	24	24
Parenting Disability Index (MPDI)	*r*	.716**	.648**	.541*	−.608**	.455*
	p	<.001	.001	.025	.003	.038
	n	24	22	17	21	21
Child behaviour problems (CBCL)						
Internalizing	*r*	.348	.503*	.017	−.148	−.020
	p	.113	.020	.951	.546	.934
	n	22	21	15	19	19
Externalizing	*r*	.363	.441*	.016	−.274	−.092
	p	.074	.035	.950	.218	.684
	n	25	23	18	22	22

Abbreviations: CBCL, Child Behavior Checklist; CES-D, Center for Epidemiologic Studies - Depression Mood Scale; MPDI, Modified Parenting Disability Index; MPQ, McGill Pain Questionnaire; PSI, Parenting Stress Index; SF-36, Medical Outcomes Study Short Form 36.

[a] Data on depression, pain, physical functioning, parenting stress, parenting disability, and children's behaviour problems based on patients' perceptions.

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

tional disability resulted in higher PDI scores in mothers with children aged less than 18 years living with them. The mean disease duration of those mothers was 7 years. In our study we were able to detect the association between patient-perceived parenting disability and disease variables within 8 months, on average, from the onset of illness.

The patients' reduced parenting efficacy and concomitant psychological distress also affected their children. Children whose parents had more physician-rated disease activity were rated by those parents as exhibiting more externalizing and internalizing behavioural problems. Parenting stress was associated with both internalizing and externalizing problems while parenting disability was correlated only with externalizing problems. It is possible that children whose parents are less well able to supervise and care for them will be prone to more non-compliant, aggressive behaviour. In a study by Welch et al.[31] parents recently diagnosed with cancer did not detect emotional or behavioural problems on the CBCL scale in their children; however, their children, especially adolescent girls, reported symptoms of anxiety/depression and aggressive behaviour.[31] These findings indicate that future studies should include children's self-reports along with their parents' in order to achieve a more complete picture of child distress in response to parental chronic illness.

That physician global assessment of disease activity—and not measures of function, pain or joint counts—relates to the patients' perceptions of their children's externalizing and internalizing behaviour could indicate that patients' reports of distress about parenting have some influence on physicians' global assessment. Rheumatologists could be invited to refer patients to allied health care professionals for further assessment on an individualized basis.

TABLE 3
Pearson product moment correlations (*r*) between parenting and child behaviour variables

Statistics		Parenting variables		Child behaviour variables	
		PSI	MPDI	CBCL – internalizing	CBCL – externalizing
Parenting Stress Index (PSI)	*r*	—	.441*	.537**	.572**
	p	—	.031	.010	.003
	n	—	24	22	25
Parenting Disability Index (MPDI)	*r*	—	—	.232	.486*
	p	—	—	.325	.022
	n	—	—	20	22
Child behaviour problems (CBCL) Internalizing	*r*	—	—	—	.759**
	p	—	—	—	.000
	n	—	—	—	22

Abbreviations: CBCL, Child Behavior Checklist; MPDI, Modified Parenting Disability Index; PSI, Parenting Stress Index.

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Limitations

Our study was limited by a small sample size (n = 29). Only 30% of the participants in the McEAR registry had children under 18 living at home. Since our study was concerned with parenting, we were unavoidably limited to those patients with children living at home with them. We thus lacked power to detect significant associations among the study variables. For example, it was not possible to explore differences in employment status as well as other non-work responsibilities that may have affected parenting stress and mood. Nevertheless, we were able to demonstrate significant relationships between parental mood and parenting,

as well as between disease activity and parenting behaviour.

This study was cross-sectional and therefore did not allow for estimation of the causal direction of the observed associations. Longitudinal studies are required to assess how disease progression may affect the parent-child relationship as well as child outcomes. However, based on previous research reporting that disability in valued life activities is a strong predictor of the subsequent development of depressive symptoms and that depressive symptoms lead to lower parental functioning,[32] we speculate that disability in parenting activities would be associated with psychological distress with consequent negative effects on the children.

A third limitation of our study is the use of parental reporting of their own psychological and physical symptoms as well as child outcomes, which may result in shared method variance. Future research should follow the families longitudinally to see how disease progression or improvement following a course of treatment affects the parent-child relationship and child outcomes. Moreover, it would be useful to obtain information from the children themselves and also to have independent ratings by teachers or other knowledgeable informants.

Finally, recent research suggested that the DAS28 may underestimate disease activity in some RA patients with disease onset mainly in the feet and particularly during the first 2 years of the disease.[21]

Conclusion

Our study pointed towards the potential inability of parents to provide their children with quality care due to their arthritis-induced pain, physical dysfunction and disease activity. This patient-perceived physical impairment was associated with psychological distress in the parents and patient-perceived behaviour problems in the children, which highlights the interrelatedness and complexity of the relationships between parents' physical health, psychological health and parent-child interactions. A multifaceted approach to the care for parents with arthritis is called for. It is important to manage not only the physical symptoms of arthritis, but also the emotional distress attendant upon the pain and functional impairment associated with the disease. Backman et al. [9] suggested some strategies that might help parents with arthritis fulfill their parenting role. These include adjusting expectations, adaptive or alternative approaches to performing parenting tasks, public health interventions and credible advice grounded in the experiences of parents living with arthritis.[9] Reframing and explaining illness behaviour to children[33] and encouraging mature child behaviour[34] could also be beneficial.

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References

1. Anderson KO, Bradley LA, Young LD, McDaniel LK, Wise CM. Rheumatoid arthritis: review of psychological factors related to etiology, effects, and treatment. Psychol Bull. 1985;98(2):358–87.

2. Fex E, Larsson BM, Nived K, Eberhardt K. Effect of rheumatoid arthritis on work status and social and leisure time activities in patients followed 8 years from onset. J Rheumatol. 1998;25(1):44–50.

3. Lundwall RA. Parents' perceptions of the impact of their chronic illness or disability on their functioning as parents and on their relationships with their children. Fam J. 2002;10(3):300–7.

4. Korneluk YG, Lee CM. Children's adjustment to parental physical illness. Clin Child Fam Psychol Rev. 1998;1(3):179–93.

5. Sigal JJ, Perry JC, Robbins JM, Gagne MA, Nassif E. Maternal preoccupation and parenting as predictors of emotional and behavioral problems in children of women with breast cancer. J Clin Oncol. 2003; 21(6):1155–60.

6. Rolland JS. Parental illness and disability: a family systems framework. J Fam Ther. 1999;21(3):242–66.

7. Grant MI, Foster NE, Wright CC, Barlow JH, Cullen LA. Being a parent or grandparent with back pain, ankylosing spondylitis or rheumatoid arthritis: a descriptive postal survey. Musculoskeletal Care. 2004; 2(1):17–28.

8. Katz PP, Pasch LA, Wong B. Development of an instrument to measure disability in parenting activity among women with rheumatoid arthritis. Arthritis Rheum. 2003;48(4):935–43.

9. Backman CL, Smith L del F, Smith S, Montie PL, Suto M. Experiences of mothers living with inflammatory arthritis. Arthritis Rheum. 2007;57(3):381–8.

10. Barlow JH, Cullen LA, Foster NE, Harrison K, Wade M. Does arthritis influence perceived ability to fulfill a parenting role? Perceptions of mothers, fathers and grandparents. Patient Educ Couns. 1999;37(2): 141–51.

11. Turner Cobb JM, Steptoe A, Perry L, Axford J. Adjustment in patients with rheumatoid arthritis and their children. J Rheumatol. 1998;25(3):565–71.

12. Hirsch BJ, Moos RH, Reischl TM. Psychosocial adjustment of adolescent children of a depressed, arthritic, or normal parent. J Abnorm Psychol. 1985; 94(2):154–64.

13. McHorney CA, Ware JE Jr, Raczek AE. The MOS 36-Item Short-Form Health Survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. Med Care. 1993; 31(3):247–63.

14. Melzack R. The McGill Pain Questionnaire: major properties and scoring methods. Pain. 1975;1(3):277–99.

15. Melzack R. The short-form McGill Pain Questionnaire. Pain. 1987;30(2):191–7.

16. Felson DT, Anderson JJ, Boers M, Bombardier C, Chernoff M, Fried B, et al. The American College of Rheumatology preliminary core set of disease activity measures for rheumatoid arthritis clinical trials. The Committee on Outcome Measures in Rheumatoid Arthritis Clinical Trials. Arthritis Rheum. 1993;36(6):729–40.

17. van der Heijde DM, van't Hof MA, van Riel PL, Theunisse LA, Lubberts EW, van Leeuwen MA, et al. Judging disease activity in clinical practice in rheumatoid arthritis: first step in the development of a disease activity score. Ann Rheum Dis. 1990; 49(11):916–20.

18. van der Heijde DM, van't Hof MA, van Riel PL, van Leeuwen MA, van Rijswijk MH, van de Putte LB. Validity of single variables and composite indices for measuring disease activity in rheumatoid arthritis. Ann Rheum Dis. 1992;51(2):177–81.

19. van der Heijde DM, van Riel PL, van Leeuwen MA, van't Hof MA, van Rijswijk MH, van de Putte LB. Prognostic factors for radiographic damage and physical disability in early rheumatoid arthritis. A prospective follow-up study of 147 patients. Br J Rheumatol. 1992;31(8):519–25.

20. Wijnands MJ, van't Hof MA, van Leeuwen MA, van Rijswijk MH, van de Putte LB, van Riel PL. Long-term second-line treatment: a prospective drug survival study. Br J Rheumatol. 1992;31(4):253–8.

21. Bakker MF, Jacobs JW, Kruize AA, van der Veen MJ, van Booma-Frankfort C, Vreugdenhil SA, et al. Misclassification of disease activity when assessing individual patients with early rheumatoid arthritis using disease activity indices that do not include joints of feet. Ann Rheum Dis. 2012;71(6):830–5.

22. Radloff LS. The CES-D scale. A self-report depression scale for research in the general population. Appl Psychol Meas. 1977;1(3): 385–401.

23. Martens MP, Parker JC, Smarr KL, Hewett JE, Slaughter JR, Walker SE. Assessment of depression in rheumatoid arthritis: a modified version of the center for epidemiologic studies depression scale. Arthritis Rheum. 2003;49(4):549–55.

24. McQuillan J, Fifield J, Sheehan TJ, Reisine S, Tennen H, Hesselbrock V, et al. A comparison of self-reports of distress and affective disorder diagnoses in rheumatoid arthritis: a receiver operator characteristic analysis. Arthritis Rheum. 2003;49(3): 368–76.

25. Abidin RR. Parenting stress index short form, 3rd ed: test manual. University of Virginia (36-item version). Odessa (FL): Psychological Assessment Resources, Inc; 1995.

26. Achenbach TM. Child behavior checklist for ages 6–18. Burlington (VT): University of Vermont, Research Center for Children, Youth, & Families; 2001.

27. Achenbach TM, Ruffle TM. The Child Behavior Checklist and related forms for assessing behavioral/emotional problems and competencies. Pediatr Rev. 2000;21(8): 265–71.

28. Umberson D. Relationships with children: explaining parents' psychological well-being. J Marriage Fam. 1989;51(4):999–1012.

29. White CP, White MB, Fox MA. Maternal fatigue and its relationship to the caregiving environment. Fam Syst Health. 2009; 27(4):325–45.

30. Poole JL, Rymek-Gmytrasiewicz M, Mendelson C, Sanders M, Skipper B. Parenting: the forgotten role of women living with systemic lupus erythematosus. Clin Rheumatol. 2012;31:995–1000.

31. Welch AS, Wadsworth ME, Compas BE. Adjustment of children and adolescents to parental cancer. Parents' and children's perspectives. Cancer. 1996;77(7):1409–18.

32. Pakenham KI, Cox S. Test of a model of the effects of parental illness on youth and family functioning. Health Psychol. 2011; forthcoming.

33. Allaire S. How a chronically ill mother manages. Am J Nurs. 1988;88(1):46–9.

34. White CP, Mendoza J, White MB, Bond C. Chronically ill mothers experiencing pain: relational coping strategies used while parenting young children. Chronic Illn. 2009;5(1):33–45.

Population-based surveillance of asthma among workers in British Columbia, Canada

M. Koehoorn, PhD (1); L. Tamburic, BSc (2); C. B. McLeod, PhD (2); P. A. Demers, PhD (3); L. Lynd, PhD (4); S. M. Kennedy, PhD (1)

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Abstract

Introduction: Population-based health databases were used for the surveillance of asthma among workers in British Columbia for the period 1999 to 2003. The purpose was to identify high-risk groups of workers with asthma for further investigation, education and prevention.

Methods: Workers were identified using an employer-paid health premium field in the provincial health registry, and were linked to their physician visit, hospitalization, workers' compensation and pharmaceutical records; asthma cases were defined by the presence of an asthma diagnosis (International Classification of Diseases [ICD]-9-493) in these health records. Workers were assigned to an ''at-risk'' exposure group based on their industry of employment.

Results: For males, significantly higher asthma rates were observed for workers in the Utilities, Transport/Warehousing, Wood and Paper Manufacturing (Sawmills), Health Care/Social Assistance and Education industries. For females, significantly higher rates were found for those working in the Waste Management/Remediation and Health Care/Social Assistance industries.

Conclusion: The data confirm a high prevalence of active asthma in the working population of British Columbia, and in particular, higher rates among females compared to males and in industries with known respiratory sensitizers such as dust and chemical exposures.

Keywords: *population surveillance, occupational diseases, asthma, British Columbia*

Introduction

Exposure to occupational hazards accounts for a significant proportion of the national and global burden of disease, which could be substantially reduced through recognition, measurement and controls. Work-related asthma is considered to be the most common work-related respiratory disease in industrialized countries.[1] Occupational exposures, including organic and inorganic dusts, and biological agents, such as flour/grains, plants, fur, feathers, fungi and various types of woods, are important risk factors for both the initiation and aggravation of adult asthma.[2,3] The American Thoracic Society,[4] in their review of the literature based largely on studies in industrialized countries, estimated that approximately 15% of asthma was due to occupational exposures, although other estimates of the attributable risk proportions are as high as 29% and 36.5%.[5,6] Workers' compensation statistics often do not reflect this level of risk in the population.[7] There are limitations in the use of accepted workers' compensation claim data for surveillance, in particular the ability of the data systems to ascertain cases of disease due to underreporting,[8] a lack of recognition of the relationship between some exposures and health outcomes, or emerging relationships that are not yet recognized without systematic data collection.

The National Institute for Occupational Safety and Health (NIOSH) in the United States highlighted the need for improved surveillance research methods.[9–11] NIOSH specifically mentions the use of linked data sources, such as administrative and health care data, to identify populations that may not be well captured in existing surveillance systems. We investigated multiple administrative health databases for population-based surveillance of asthma rates by industry of employment among workers in the Canadian province of British Columbia. We also investigated the face validity of this surveillance approach by investigating asthma rates among high-risk groups using an exposure matrix.[2] We hypothesized that the rate would be higher among workers in industries with suspected or known allergens such as wood dust (i.e. wood and paper manufacturing), moulds or endotoxins (e.g. schools) and latex/glutaraldehyde or industrial cleaning agents (e.g. health care services).[2,12,13]

We had access to health databases for population health and health services

Author references:

1. School of Population and Public Health, University of British Columbia, Vancouver, British Columbia, Canada
2. Centre for Health Services and Policy Research, University of British Columbia, Vancouver, British Columbia, Canada
3. Occupational Cancer Research Centre, Cancer Care Ontario, Toronto, Ontario, Canada
4. Pharmaceutical Sciences, University of British Columbia, Vancouver, British Columbia, Canada

Correspondence: Mieke Koehoorn, School of Population and Public Health, University of British Columbia, 2206 East Mall, 1st Floor, Vancouver, BC V6T 1Z3; Tel.: 604-822-5756; Fax: 604-822-4994; Email: mieke.koehoorn@ubc.ca

research via Population Data BC.[14] The data included longitudinal, person-specific, de-identified health data on British Columbia's 4.5 million residents across multiple data sources. The use of these linked administrative health databases provided a novel approach to work-related health surveillance, beyond workers' compensation statistics. As surveillance tools, the databases can provide evidence of the relationship between exposures and disease outcomes for further investigation of high-risk groups and recognition of work-related illness, as well as education and prevention efforts.

Methods

Study population

Using the provincial health registry, we identified individuals aged 15 to 64 years who had been continuously registered (i.e. living in the province) for a minimum of 3 years at entry into the study. The study population excluded individuals with missing data on sex or with a diagnosis of chronic obstructive pulmonary disease (International Classification of Disease, version 9 [ICD-9] codes 491, 492, 496) given the potential overlap between asthma and chronic obstructive lung disease diagnoses in older adults. Among continuously registered residents, we identified workers using an employer-paid health premium code in the health registry. As a dynamic study population, individuals could enter the study at any time from 1999 to 2003 providing they met the inclusion criteria.

Exposure groups

The employer-paid health premium codes were used to assign a standardized industry of employment (North American Industry Classification System or NAICS code[15]). To identify industries with known or suspected allergen exposures, defined here as at-risk or high-risk industries, we used an asthma-specific job exposure matrix[2] previously developed for population-based studies. All matches between the matrix and our study sample were reviewed by investigators (MK, PD) with expertise in occupational asthma and occupational hygiene as well as knowledge of British Columbia industries and their exposures.

Health data sources

Health data on physician visits and hospitalizations, workers' compensation claims and filled prescriptions for the population of British Columbia were available to researchers for approved projects from the Ministry of Health, WorkSafeBC and PharmaNet, respectively, via Population Data BC. Data are linkable at the individual level but were provided to researchers with personal identifiers removed from the records. Use of the data for this project was governed by data access and confidentiality agreements between the data stewards and the researchers, as well as by the Behavioural Ethics Research Board of the University of British Columbia (Certificate #B05-0664). Given a universal health care system for physician visits and hospitalizations, over 93% workforce coverage by the workers' compensation system and a provincial prescription database (all prescriptions filed in pharmacies in British Columbia), the health data are considered comprehensive for provincial residents. Follow-up of asthma outcomes was limited to the study years given the availability of health records across all data sources and the availability of industry of employment codes in the provincial health registry.

Case definition

Physician-diagnosed asthma was identified using the ICD-9 code 493. The case definition was met if a worker had one diagnosed hospitalization, two diagnosed physician visits in a 12-month period, one diagnosed workers' compensation claim, or two prescriptions for any asthma-related drugs confirmed by at least one diagnosed physician visit within a 12-month period.[7] In the event of two physician visits or two prescriptions within 12 months but spanning calendar years, the year of the first visit or prescription was attributed to the asthma case. Asthma-related drugs were defined by the pharmacist on the research team (LL) and extracted based on the Drug Identification Number (list available upon request).

For the purposes of surveillance, we were interested in ''active'' asthma among the working study population during each year of follow-up. Focusing on active asthma is important as both asthma onset (incident cases during follow-up) and aggravation of existing asthma (prevalent cases requiring health care during follow-up) are compensable conditions associated with workplace exposures in British Columbia. Individuals who met the case definition before entry into the study were prevalent cases. Individuals who met the case definition during the study follow-up period (i.e. were asthma-free for a minimum of three years before entry into the study) were incident cases. Incident cases were considered active asthma in the year they became an incident case and each subsequent year that they had a health care contact for asthma. Prevalent cases were considered active asthma in each year that they had a health care contact for asthma. Active asthma was defined as contact with the health care system (physician visit, hospitalization, prescription or compensation claim) during the year.

Analysis

Rates of active asthma were calculated per year of follow-up using Stata version 10.1 (StataCorp, College Station, TX, US). Age- and sex-adjusted rates with 95% confidence intervals (CIs) were compared by industry of employment groups and for high-risk versus low-risk industries.

Results

Study population

A total of 2.7 million residents of working age were continuously registered for health services during the study period, 1999 to 2003. Less than 0.3% of individuals were excluded for missing data on sex (n = 4001) and for a diagnosis of chronic obstructive pulmonary disease (n = 3456). Altogether, 908 896 workers were identified by industry of employment using the employer-paid health premium codes. They represented 33% of the registered population but 60% of the working population in British Columbia.[16] This method of identifying a population-based

working cohort using the employer-paid health premium codes underrepresents the self-employed, small worksites (i.e. < 5 employees) and females as these groups have lower rates of employer-paid health premiums.

In 2003, females made up 42.8% of the included working study population, compared to 47.2% of the overall British Columbia workforce[17] and to 50.6% in the continuously registered population from which the working population was drawn. The mean age of the working study population was 42.1 years; this compares to an average age of 40.7 years among workers in the province [18] and to 39.4 years among the registered population.

The included working study population was distributed over 843 distinct industrial sectors. Males were concentrated in Wood and Paper Manufacturing (14.8% in 1999 and 13.4% in 2003), Public Administration (11.7% and 11.0%) and Transport and Warehousing (8.2% and 7.7%). Females were concentrated in Health Care and Social Assistance (22.8% and 23.3%) and Education (14.9% and 14.8%). This is comparable to the top industries of employment by gender in the overall provincial workforce, with the exception of Trades for both genders, Construction among males and Accommodation and Food Services among females.[19]

Active asthma rates

Overall, we identified a total of 41 966 cases of asthma, of which 30 080 were prevalent at the time of entry into the study (for a prevalence rate of 33.1 cases per 1000 workers) and 11 886 were new cases identified during the study follow-up period (for a cumulative incidence rate of 13.1 cases per 1000 workers). The majority of asthma cases were identified through physician visits (20.8%) or a combination of prescriptions with a physician visit (55.2%). Only 302 cases (0.7%) were captured by a workers' compensation claim.

Rates of active asthma for the years 1999 to 2003 (Table 1) ranged from 22.3 to 26.2 cases per 1000 male workers, and from 33.7 to 40.6 cases per 1000 female workers. A small annual increase was observed from 1999 through to 2003. Age-adjusted active asthma rates by industry are shown for the year 2003 only, as results for 1999 to 2002 were similar (Table 2). For males, the active asthma rate was significantly higher than the overall rate in the working population for workers employed in Wood and Paper Manufacturing (including Sawmills), Health Care and Social Assistance, and Schools industries. Higher rates (although 95% confidence interval [CI] included the rate for the working population) were also observed for male workers in the Utilities; Transport and Warehousing; Educational Services; Mining, Oil and Gas; Finance and Insurance; and Public Administration industries. For females, the active asthma rate was significantly higher in the Waste Management and Remediation and Health Care and Social Assistance (including general hospitals and nursing care facilities) industries. Higher rates (95% CI overlapped with the overall rate in the working population) were also observed for the female workers in the Public Administration, Information and Cultural, and Educational Services industries.

During follow-up, rates of active asthma for ''at-risk'' industries among males ranged from 25.2 to 28.6 cases per 1000 workers and were higher than those observed for ''low-risk'' industries. Rates for ''at-risk'' industries among females ranged from 35.2 to 41.6 cases per 1000 workers. While rates tended to be slightly higher for at-risk industries compared to low-risk industries for females, the differences were not as consistent or as high as that observed for males.

Discussion

The purpose of this study was to investigate the feasibility of using linked health data for population-based surveillance of asthma among workers and to investigate at-risk industry groups for on-going monitoring and future prevention efforts. To do this we estimated the active asthma rate among a population-based workforce sample by industry of employment and among at-risk industry groups with known or suspected allergen exposures.

The use of an active asthma measure for the surveillance of work-related disease does not enable comparisons with many other studies using more traditional measures of asthma incidence and prevalence for population-based estimates. Nevertheless, our overall annual rate of active asthma of approximately 30 cases per 1000 workers is consistent with an

TABLE 1
Active asthma[a] rates per 1000 workers, British Columbia, Canada, 1999–2003

	1999 Rate (95% CI)	2000 Rate (95% CI)	2001 Rate (95% CI)	2002 Rate (95% CI)	2003 Rate (95% CI)
Males	22.3 (21.8–22.7)	23.7 (23.2–24.1)	25.0 (24.5–25.5)	25.5 (25.0–25.9)	26.2 (25.7–26.7)
High risk industries	25.2 (24.1–26.3)	27.1 (25.9–28.2)	27.8 (26.7–29.0)	28.5 (27.3–29.6)	28.6 (27.4–29.8)
Low risk industries	21.5 (20.9–22.0)	22.8 (22.3–23.3)	24.3 (23.8–24.9)	24.8 (24.2–25.3)	25.6 (25.0–26.2)
Females	33.7 (33.0–34.4)	36.1 (35.4–36.8)	37.4 (36.7–38.1)	38.2 (37.5–38.9)	40.6 (39.9–41.3)
High risk industries	35.2 (33.7–36.6)	36.0 (34.5–37.5)	38.0 (36.5–39.4)	38.2 (36.8–39.7)	41.6 (40.1–43.0)
Low risk industries	33.3 (32.5–34.1)	36.1 (35.3–36.9)	37.3 (36.5–38.1)	38.2 (37.4–39.0)	40.2 (39.4–41.0)

Abbreviations: CI, confidence interval.

[a] Active asthma is defined by a physician visit, hospitalization, workers' compensation claim or prescription for asthma. It includes incident cases and prevalent cases who had at least one of these health care contacts in the year of follow-up.

TABLE 2
Age-adjusted active[a] asthma rates per 1000 workers, British Columbia, Canada, 2003

Industry, Subsector[b]	Males Rate (95% CI)	Industry, Subsector[b]	Females Rate (95% CI)
Overall	26.5 (26.0, 27.0)	Overall	40.6 (39.9–41.3)
Utilities	31.9 (26.3–37.6)	**Waste Management, Remediation**	**47.3 (40.8–53.8)**
Transport and Warehousing	30.2 (26.0–34.4)	**Health Care and Social Assistance**	**45.6 (43.7–47.4)**
Wood and Paper Manufacturing	**30.2 (28.7–31.7)**	**Nursing Care Facilities**	**47.0 (40.8–53.3)**
Sawmills	**30.8 (28.5–33.2)**	**General Hospitals**	**42.9 (40.9–45.0)**
Health Care and Social Assistance	**29.8 (26.8–32.8)**	Specialty Hospitals	41.6 (36.8–46.4)
General Hospitals	27.8 (24.0–31.7)	Public Administration	42.7 (40.3–45.0)
Educational Services	28.1 (26.0–30.1)	Municipal Public Administration	43.2 (38.2–48.1)
Schools	**29.6 (27.0–32.3)**	Information and Cultural Industries	41.6 (37.9–45.2)
Universities	26.2 (21.8–30.5)	Educational Services	41.5 (39.5–43.5)
Mining, Oil and Gas	27.9 (22.8–33.0)	Colleges	44.5 (38.5–50.5)
Finance and Insurance	27.3 (24.6–30.0)	Universities	41.7 (36.6–46.8)
Public Administration	26.9 (25.2–28.7)	Schools	41.5 (39.1–43.8)
Municipal Public Administration	26.7 (24.0–29.5)	Mining, Oil and Gas	40.6 (23.1–58.1)
Information and Cultural Industries	26.2 (23.9–28.6)	Utilities	40.2 (28.9–51.6)
Telecommunications	26.0 (23.9–28.6)	Professional/Scientific/Technical Services	39.3 (35.5–43.1)
Transportation	26.0 (24.1–28.0)	Finance and Insurance	38.0 (35.7–40.2)
Real Estate	26.0 (21.0–31.1)	Arts, Entertainment, Recreation Services	37.7 (30.8–44.7)
Waste Management, Remediation	25.9 (21.6–30.3)	Retail Trade (General)	37.1 (32.0–42.2)
Accommodation and Food Services	25.3 (22.0–28.6)	Accommodation and Food Services	36.7 (33.0–40.3)
Wholesale	24.7 (22.8–26.6)	Wholesale	36.5 (32.5–40.5)
Retail (Foods and Goods)	24.5 (22.7–26.3)	Retail (Foods and Goods)	36.4 (33.6–39.2)
Metals, Machinery	24.5 (22.5–26.4)	Real Estate	34.7 (27.6–41.9)
Food and Textiles Manufacturing	23.9 (20.7–27.1)	Wood and Paper Manufacturing	33.8 (29.7–37.8)
Professional/Scientific/Technical Services	23.7 (21.5–26.0)	Warehousing	33.8 (27.1–40.4)
Computer Systems Design	27.8 (22.7–32.8)	Transportation	33.6 (29.8–37.4)
Arts/Entertainment/Recreation Services	23.1 (18.4–27.8)	Construction	32.6 (24.4–40.7)
Agriculture, Forestry	22.8 (19.4–26.1)	Metals, Machinery	30.9 (26.1–35.7)
Construction	22.6 (20.4–24.9)	Food and Textiles Manufacturing	30.7 (25.7–35.8)
Retail (General)	21.7 (17.2–26.2)	Management	30.5 (16.9–44.1)
Management	20.7 (10.8–30.7)	Agriculture, Forestry	25.1 (16.4–33.8)

Abbreviation: CI, confidence interval.

Note: grey shading, rate is higher than overall rate in working population; bold, 95% CI does not include the overall rate.

[a] Active asthma is defined by a physician visit, hospitalization, workers' compensation claim or prescription for asthma. It includes incident cases and prevalent cases who had at least one of these health care contacts for asthma in 2003.

[b] North American Industry Classification System (NAICS) Coding[15] with subsectors where sample size allows.

overall rate of 3% (or 30 cases per 1000) for active asthma (as defined by an asthma attack in the past year) observed in the European Community Respiratory Health Survey (ECRHS).[20] Our study definition of active asthma includes incident as well as prevalent cases requiring health care, and we expected our estimates to fall within the range of previous incidence and prevalence studies. An asthma incidence rate of 3% (or 30 cases per 1000 workers) among high-risk or exposed occupations in the ECRHS II study [21] falls within the range we observed in our study among high-risk groups of male and female workers. Estimates of the prevalence of work-related asthma using administrative data from a sample of the labour force in the Canadian province of Manitoba found rates as high as 48 cases per 1000 workers among some occupational groups,[12] close to our highest estimate observed among women in high-risk groups. Observed differences with previous studies may be attributed to different case ascertainment definitions, trying to compare with an annual active asthma rate, and the use of industry versus occupation to assign exposure risk. Finally, a study of new-onset adult asthma among a working sample in the province of Alberta found an incidence rate of 1.6% (or 16 cases per 1000)[13] over a 10-year

follow-up period. Although higher than our estimate of cumulative incidence at 13 cases per 1000 workers, this may be explained by differences in the definition for case ascertainment (one physician visit for asthma in the Alberta study). Collectively, our surveillance system produced estimates that were within the range of other studies despite differences in methods, case definitions and workforce characteristics.

The higher rate of asthma among female workers compared to male workers is consistent with surveillance results from the neighbouring province of Alberta,[13] as well as self-reported estimates from national health surveys[22] and those from other jurisdictions.[5] The Alberta study relied on health records similar to our methodology for case ascertainment among a worker population and found twice the incidence rate of adult asthma among females compared to males,[13] consistent with our findings. Again, higher rates in the current study may be due to our case definition of active asthma in the follow-up year, but females having almost twice the rate of males overall was consistent with our findings. Possible occupational explanations for the gender difference in a working population include more females working in high-risk jobs (i.e. teaching or health care)[17] or in high-risk jobs with exposures that are not as obvious or amenable to personal protective equipment, leading to more active asthma symptoms and medical attention (i.e. food services versus wood and paper products[23]). It may also reflect a gender difference associated with the ''healthy worker effect'' whereby males have a ''stronger healthy worker hire effect'' while females have a ''stronger healthy worker survivor effect.''[24,25] With a strong healthy worker hire effect, males with childhood-onset asthma or existing adult-onset asthma would be less likely to be hired into high-risk jobs. With a stronger healthy worker survivor effect, females would be more likely to be in the workforce with asthma (although less likely to remain at work over the longer term). It may also be plausible that women are more likely to work with asthma symptoms. Evidence suggests that socioeconomic factors may differentially impact vulnerable groups such as females with less job mobility, placing them at increased risk for adverse effects of workplace exposures.[24,26]

Our study identified industries with higher than average rates of active asthma. Industries dealing with wood/wood dust as well as individuals working in schools and health services were at an increased risk of asthma compared with those working in other industries. This is in keeping with known or suspected exposures related to dusts, moulds/endotoxins, and latex/glutaraldehyde or industrial cleaning agents.[2] It is also consistent with surveillance studies investigating high-risk groups by occupation, including the Manitoba and Alberta studies that identified a higher risk among workers in teaching and related occupations.[12,13] The previous studies were able to identify other high-risk groups likely due to differences in type of employment by province (i.e. forestry is a major industry in British Columbia) but also due to a finer level of detail for exposures by occupation. For example, the Alberta study was able to identify a higher risk among workers in industries dealing with flour/food, fibreglass and vehicles. Similarly, the Manitoba study identified higher risks among fabricating, installing and repairing occupations of electrical, electronic and related equipment.

Conversely, some industrial groups such as Metals/Machinery or Manufacturing (Food and Textiles) did not appear at an increased risk relative to other industries. The reliance on industry of employment as a surrogate measure of exposure for at-risk jobs in this surveillance project may be subject to more misclassification for these types of industries. Industrial groups such as Manufacturing may be made up of multiple and diverse occupational groups. Some have known or suspected exposures such as textiles, wood dust or metal working fluids, but others do not (i.e. beverage manufacturing), resulting in a conservative bias on the estimates. Industries such as Educational Services or Health Services, on the other hand, may be dominated by several large occupational groups such as teachers or nurses/cleaners all with known or suspected allergens (e.g. mould or chemical irritants), resulting in better estimates of exposure using this method of at-risk industries of employment. Overall, the misclassification of exposure using industry of employment exerts a conservative bias attenuating the risk estimates. Ultimately, it would be ideal to improve this surveillance method with more detailed occupation information at the population level. This is currently not captured by any of the administrative datasets with the exception of the workers' compensation claim records. Including occupation of employment in medical registries or health records/databases would be an invaluable source of data for surveillance studies, as concluded by others involved in similar work.[27] Novel approaches to obtaining occupation and industry of employment data from administrative databases for population-based health surveillance are also warranted, as was done by Cherry et al. in Alberta.[13]

While Ministry of Health employer coding is assumed to be valid for billing purposes, there may still be limitations in terms of assigning exposure based on the industry of employment codes. Not all individuals within an industry are working in an occupation with exposures, and we were not able to investigate high-risk *occupational* groups. We relied on an exposure matrix to investigate at-risk *industrial* groups; some misclassification with bias towards the null is unavoidable using this approach and may explain why we did not observe greater differences between the high- and low-risk groups. It is also possible that the misclassification is greater for women than men using industry coding for exposure as women may work in more diverse occupations in high-risk industries such as construction.[28] This would explain the smaller observed differences in asthma rates between high- and low-exposed industrial groups for women.

There is also some uncertainty about whether the industry of employment at the time of case ascertainment is the industry of exposure. Current symptoms may be as a result of past exposures (i.e. employment in an industry other than the current one). Our population-based

surveillance approach overcomes some of the biases associated with the healthy worker effect in previous occupational cohort studies and provides information about asthma in the working population by following workers forward even after they leave the workforce (or if they change industries). Further, as we have emphasized, women as well as self-employed workers or those who work for small employers are underrepresented using the employer-paid health premium codes in the provincial medical registry.

The years of follow-up were limited based on the availability of the industry coding. Workplace processes (and exposures) have not changed so dramatically over the past decade as to render these findings irrelevant for workers in the same industries today (i.e. forestry workers are still exposed to wood dust, teachers to moulds or endotoxins, cleaners to industrial cleaning agents, and metal workers to metal working fluids), and the job exposure matrix used in this study is based on known risk factors for asthma still present in industries. The retrospective data available for this study represent the only known source of employment codes for a large proportion of the working population, linked to multiple health databases. An advantage of using population-based administrative data is that the larger number of individuals involved allows for robust analysis of age, sex and other demographic trends, compared to data limited by sample size. Another advantage is that the ability to link multiple health databases improves case ascertainment for occupational outcomes.[29]

We are not suggesting that all of the cases of asthma among workers in our study are work-related, but it appears that workers' compensation claims data underestimate asthma in the population, necessitating the use of additional data sources to capture asthma among workers and identify work groups with higher rates. Less than 1% of our cases were captured by the workers' compensation system as an accepted claim[7] despite a recognized population-attributable risk estimate of asthma associated with occupational exposures of 15%,[4] including Canadian estimates.[30,31] Given the face validity associated with higher rates among industries with known allergens, the current study offers a surveillance tool for ongoing monitoring among at-risk groups, as well as evidence of the need for additional education on the association between workplace exposures and asthma morbidity. The study also offers a tool for identifying new or emerging at-risk groups for further investigation, such as the workers in the waste management industry and the public sector, as we observed.

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References

1. Kogevinas M, Anto JM, Sunyer J, Tobias A, Kromhout H, Burney P. Occupational asthma in Europe and other industrialised areas: a population-based study. European Community Respiratory Health Survey Study Group. Lancet. 1999 May 22; 353(9166):1750–4.

2. Kennedy SM, Le Moual N, Choudat D, Kauffmann F. Development of an asthma specific job exposure matrix and its application in the epidemiological study of genetics and environment in asthma (EGEA). Occup Environ Med. 2000;57:635–41.

3. Le Moual N, Kennedy SM, Kauffmann F. Occupational exposures and asthma in 14,000 adults from the general population. Am J Epidemiol. 2004;160(11):1108–16.

4. Balmes J, Becklake M, Blanc P, Henneberger P, Kreiss K, Mapp C, et al. American Thoracic Society statement on occupational contribution to the burden of airway disease. Am J Respir Crit Care Med. 2003;167(5):787–97.

5. Karjalainen A, Kurppa K, Martikainen R, Klaukka T, Karjalainen J. Work is related to a substantial portion of adult-onset asthma incidence in the Finnish population. Am J Respir Crit Care Med. 2001;164(4):565–8.

6. Arif AA, Whitehead LW, Delclos GL, Tortolero SR, Lee ES. Prevalence and risk factors of work related asthma by industry among United States workers: data from the third national health and nutrition examination survey (1988–94). Occ Env Med. 2002;59(8):505–11.

7. McLeod CB, Bogyo T, Demers P, Edeer D, Hertzman C, Kennedy S, et al. Asthma in British Columbia [Internet]. Vancouver (BC): Centre for Health Services and Policy Research, University of British Columbia; 2007 Jan [cited 2011 Jul 19]. Available from: http://pwhr.sites.olt.ubc.ca/files/2012/03/Asthma-Report-2007.pdf

8. Kraut A. Estimates of the extent of morbidity and mortality due to occupational diseases in Canada. Am J Ind Med. 1994;25(2):267–78.

9. National Institute for Occupational Health and Safety (NIOSH). Tracking occupational injuries, illnesses, and hazards: the NIOSH surveillance strategic plan. Cincinnati (OH): National Institute for Occupational Health and Safety; 2001. Report No.: DHHS (NIOSH) Publication Number 2001–118.

10. Souza K, Steege AL, Baron SL. Surveillance of occupational health disparities: challenges and opportunities. Am J Ind Med. 2010;53:84–94.

11. National Occupational Research Agenda (NORA). Traumatic occupational injury research needs and priorities: a report by the NORA traumatic injury team. Cincinnati (OH): National Institute for Occupational Health and Safety; 1998. Report No.: DHHS (NIOSH) Publication Number 98–134

12. Kraut A, Walld R, Mustard C. Prevalence of physician-diagnosed asthma by occupational groupings in Manitoba, Canada. Am J Ind Med. 1997;32(3):275–87.

13. Cherry N, Beach J, Burstyn I, Fan X, Guo N, Kapur N. Data linkage to estimate the extent and distribution of occupational disease: new onset adult asthma in Alberta, Canada. Am J Ind Med. 2009; 52(11):831–40.

14. Population Data BC. Research data access framework [Internet]. Vancouver (BC): Population Data BC, University of British Columbia. [cited 2011 Jul 20]. Available from: http://www.popdata.bc.ca/dataaccess/rdaf

15. Statistics Canada. North American Industry Classification System (NAICS) 2002. Ottawa (ON): Government of Canada, Ministry of Industry; 2003.

16. Tamburic L, McLeod C, Xu F, Demers P, Kennedy S, Lynd L, et al. Asthma in BC workers: surveillance using new occupational research capacity in the BC Linked Health Database. Canadian Association for Research on Work and Health (CARWH) conference (poster presentation); June 15–17 2008; Montreal (QC). [cited 2011 Jul 20]. Available from: http://pwhr.sites.olt.ubc.ca/files/2012/03/Asthma-poster-from-CARWH-2008.pdf

17. BCStats. Employment by class of worker, industry, and gender, BC - annual averages (thousands of persons) [Internet]. Ottawa (ON): Statistics Canada - Labour Force Survey - CANSIM Table 282-0012; [cited 2012 May 14]. Available from: http://www.bcstats.gov.bc.ca/Files/16005079-7667-4c0a-ac45-bd3316341a4c/EmploymentbyClassofWorker.pdf

18. BCStats. BC's Ageing Workforce. Victoria (BC): Infoline [Internet]. 2010 Aug 13 [cited 2011 Jul 20];10(32):3–5. Available from: http://www.bcstats.gov.bc.ca/Files/796b76ad-c446-46b7-b6b4-362f68b51f8d/InfolineBCsAgeingWorkforce.pdf

19. BCStats. British Columbia employment by detailed industry, annual averages [Internet]. Victoria (BC): Government of British Columbia; 2011 Feb 8 [cited 2011 Jul 20]. Available from: https://www.bcstats.gov.bc.ca/Files/2bd86b08-a46b-4ed3-88e6-b14e27bda5b3/EmploymentbyIndustry.pdf

20. European Community Respiratory Health Survey. Variations in the prevalence of respiratory symptoms, self-reported asthma attacks, and use of asthma medication in the European Community Respiratory Health Survey (ECRHS). Eur Respir J. 1996 Apr;9(4):687–95.

21. Kogevinas M, Zock JP, Jarvis D, Kromhout H, Lillienberg L, Plana E, et al. Exposure to substances in the workplace and new-onset asthma: an international prospective population-based study (ECRHS-II). Lancet. 2007;370(9584):336–41.

22. Garzia NA. Surveillance of asthma in relation to work among Canada's adult population [dissertation]. Vancouver (BC): University of British Columbia; 2008 [cited 2011 Jul 20]. Available from: https://circle.ubc.ca/handle/2429/2471

23. BCStats. Earnings & Employment Trends, June 2010: Woman enough to do the job: women's integration into traditionally male-dominated occupations. Victoria (BC): Government of British Columbia; 2010 Jun [cited 2012 May 14]. Available from: http://www.bcstats.gov.bc.ca/Files/669c1e5b-5a2d-44f1-996d-77e30c10df9c/WomanEnoughtoDotheJobWomensIntegrationintoTraditionallyMale-DominatedOccupations.pdf

24. Le Moual N, Kauffmann F, Eisen EA, Kennedy SM. The healthy worker effect in asthma: work may cause asthma, but asthma may also influence work. Am J Resp Crit Care. 2008;177(1):4–10.

25. Lea CS, Hertz-Picciotto I, Andersen A, Chang-Claude J, Olsen JH, Pesatori AC, et al. Gender differences in the healthy worker effect among synthetic vitreous fiber workers. Am J Epidemiol. 1999;150(10):1099–106.

26. Marabini A, Dimich-Ward H, Kwan SY, Kennedy SM, Waxler-Morrison N, Chan-Yeung M. Clinical and socioeconomic features of subjects with red cedar asthma. A follow-up study. Chest. 1993;104(3):821–4.

27. Suarthana E, McFadden JD, Laney AS, Kreiss K, Anderson HA, Hunt DC, et al. Occupational distribution of persons with confirmed 2009 H1N1 influenza. J Occup Environ Med. 2010;52(12):1212–6.

28. Statistics Canada. Public use microdata file (PUMF): Labour Force Survey – Data File (LFS). Ottawa (ON): Statistics Canada; [cited 2011 Mar 15]. Available from: http://www.statcan.gc.ca/cgi-bin/imdb/p2SV.pl?Function = getDatafileData&Item_Id = 55456&lang = en&db = imdb&adm = 8&dis = 2

29. Smith GS, Veazie MA, Benjamin KL. The use of sentinel injury deaths to evaluate the quality of multiple sources of reporting of occupational injuries. Ann Epidemiol. 2005;15(3):219–27.

30. Tarlo SM, Leung K, Broder I, Silverman F, Holness DL. Asthmatic subjects symptomatically worse at work: prevalence and characterization among a general asthma clinic population. Chest. 2000;118(5):1309–14.

31. Johnson AR, Dimich-Ward HD, Manfreda J, Becklake MR, Ernst P, Sears MR, et al. Occupational asthma in adults in six Canadian communities. Am J Respir Crit Care Med. 2000;162(2):2058–62.

Unintentional injury mortality and external causes in Canada from 2001 to 2007

Y. Chen, PhD (1); F. Mo, PhD (2); Q. L. Yi, PhD (1); Y. Jiang, MSc (2); Y. Mao, PhD (2)

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Abstract

Introduction: To understand the distribution pattern and time trend of unintentional injury mortalities is crucial in order to develop prevention strategies.

Methods: We analyzed vital statistics data from Canada (excluding Quebec) for 2001 to 2007. Mortality rates were age- and sex-standardized to the 2001 Canadian population. An autoregressive model was used for time-series analysis.

Results: Overall mortality rate steadily decreased but unintentional injury mortality rate was stable over the study period. The three territories had the highest mortality rates. Unintentional injury deaths were less common in children than in youths/adults. After 60, the mortality rate increased steadily with age. Males were more likely to die of unintentional injury, and the male/female ratio peaked in the 25- to 29-year age group. Motor vehicle crashes, falls and poisoning were the three major causes. There was a substantial year after year increase in mortality due to falls. Deaths due to motor vehicle crashes and drowning were more common in summer months, and deaths caused by falls and burns were more common in winter months.

Conclusion: The share of unintentional injury among all-cause mortality and the mortality from falls increased in Canada during the period 2001 to 2007.

Keywords: *age standardization, burn, Canada, consumer product, drowning, fall, mortality, poisoning, unintentional injury, suffocation, vehicle traffic crash, vital statistics*

Introduction

Injuries are among the leading causes of death and disabilities worldwide.[1] They represent about 16% of the global burden of disease[2] and are the leading cause of death in people aged under 60 years.[3] In 2004, World Health Organization estimated that injuries caused over 5 million deaths per year, of which 3.9 million were unintentional.[4] Compared to many other diseases, injuries affect more young people, and therefore result in more years of life lost.[1] In Canada, the total economic burden of injury was about $20 billion in 2004, of which $16 billion was as a result of unintentional injuries.[5]

Unintentional injury is any injury that is not caused on purpose or with intention to harm. Since not all unintentional injuries are random events and some of them can be prevented, it is not usually appropriate to use the word "accident" to define unintentional injury. Unintentional injury can be further classified according to external causes such as motor vehicle collisions, falls, poisoning, drowning, suffocations and so on.[6] Unintentional injuries may be work-related or sports-related. Different types of unintentional injury may have unique patterns in different subpopulations, for example, motor vehicle crashes are most common among young people[7] while falls are more likely to cause a fatal outcome among the elderly.[8] Monitoring the changing patterns of overall and cause-specific unintentional injury mortalities gathers information essential to developing new programs on unintentional injury prevention and intervention and modifying existing ones. In this study, we conducted a descriptive analysis of vital statistics data to investigate the distribution patterns and time trends of overall and cause-specific unintentional injury mortalities in Canada (excluding Quebec).

Methods

The study was based on mortality data from the Canadian Vital Statistics Death Database (excluding deaths registered in the province of Quebec, since these were not available on the Data Extraction and Analysis System, Public Health Agency of Canada) for the period from January 1, 2001, to December 31, 2007. Death statistics are based on information abstracted and compiled from death certificates, and are provided to Statistics Canada by the vital statistics registrars in each province or territory. The mortality data in this analysis are coded using the *International Classification of Diseases, 10th Revision* (ICD-10), in which external causes are classified under a series of alphanumeric codes, V01–Y98. These codes were used to identify unintentional injury deaths (ICD-10: V01–X59, Y85–Y86) and unintentional

Author references:

1. Department of Epidemiology and Community Medicine, University of Ottawa, Ottawa, Ontario, Canada
2. Consumer Product Safety and Injury Risk Assessment Program Working Group, Science Integration Division, Centre for Chronic Disease Prevention and Control, Public Health Agency of Canada Ottawa, Ontario, Canada

Correspondence: Yue Chen, Department of Epidemiology and Community Medicine, Faculty of Medicine, University of Ottawa, 451 Smyth Road, Ottawa, ON K1H 8M5; Tel.: 613 562-5800 ext. 8287; Fax: 613 562-5465; Email: ychen@uottawa.ca

injury deaths by cause including motor vehicle traffic crashes (V02–V04 [.9], V09.2, V12–V14 [.3–.9], V19 [.4–.6], V20–V28 [.3–.9], V29 [.4–.9], V30–V79 [.4–.9], V80 [.3–.5], V81–V82 [.1], V83–V86 [.0–.3], V87 [.0–.8], V89.2); pedal cycle (ICD-10: V10–V14, V16–V19); pedestrian (traffic-related) (ICD-10: V02–V04 [.1], V09.2, V09.3); recreation boating (ICD-10: V90.2–V90.8, V91.2–V91.8, V92.2–V92.8, V93.2–V93.8, V94.2–V94.8); drowning (ICD-10: V90, V92, W65–W74); falls (ICD-10: W00–W19); burns or fire (ICD-10: W85–W91, X00–X19); suffocations (ICD-10: W75–W84); poisoning (ICD-10: X40–X49) and other unintentional causes.

We took annual population estimates from Statistics Canada's annual demographic statistics.[9,10] Age- and sex-standardized mortality rates were calculated using the direct method with reference to the 2001 Canadian population. For each province and for the three territories (Northwest Territory, Yukon and Nunavut) combined, average overall mortality rates were calculated for the 7-year study period, that is, the total number of deaths during the period divided by the sum of the annual populations, which is equivalent to a weighted average of annual rates using the annual population as a weight.

To explore time trends for unintentional injury mortality rates from 2001 to 2007 and seasonal pattern for cause-specific unintentional injury mortalities in males and females, we conducted time-series analysis. The mortality rates were calculated by using average annual population as the denominator since there were no monthly population estimates. Because the numbers of death per month were small and could not be further stratified by age, we conducted age and sex-standardization by using the ratios of annual standardized versus crude rates (crude monthly rate × standardized/crude mortality for the year). The adjusted monthly rates were then plotted to visually display their time trends and seasonal patterns. Autoregressive models were fitted to determine the associations of secular year and month with various unintentional injury mortalities. In the models, the first order autocorrelation was considered, with monthly rates being dependent variables and year and month indicators being independent variables.

All analyses were conducted using SAS version 9.1 statistical software (SAS Institute Inc., Cary, NC, US).

Results

There were a total of 51 178 deaths due to unintentional injuries, which accounted for 4.2% of all deaths in Canada excluding Quebec during the study period from 2001 to 2007. The age- and sex-standardized mortality for all causes steadily decreased from 702 per 100 000 in 2001 to 631 per 100 000 in 2007 ($p < .001$) while the mortality due to unintentional injuries was relatively stable year after year ($p = .571$). As a result, the proportion of unintentional injury mortality versus all mortality increased significantly ($p = .003$) in the 7-year period. Males accounted for 61.1% of all unintentional injury deaths. However, males and females had similar time trends for unintentional injury mortality, overall mortality (all causes combined) and their ratio (Table 1).

After age- and sex-standardization, the three territories combined had the highest overall mortality (842.3 per 100 000) and unintentional injury mortality (69.1 per 100 000) (Table 2). British Columbia had the lowest overall mortality (626.8 per 100 000) while Newfoundland and Labrador had the lowest mortality due to unintentional injuries (24.8 per 100 000). The unintentional injury mortality for the three territories was almost triple that of

TABLE 1
Crude and standardized mortality by calendar year, total and by sex, Canada (excluding Quebec), 2001–2007

Calendar year	Crude mortality		Standardized mortality[a]		
	All causes, per 100 000	Unintentional injury, per 100 000	All causes, per 100 000	Unintentional injury, per 100 000	Unintentional injury/All causes, %
Total					
2001	701.8	28.7	702.1	28.7	4.09
2002	704.3	29.9	692.3	29.6	4.28
2003	710.3	29.5	685.3	28.8	4.20
2004	702.2	28.7	664.6	27.7	4.27
2005	708.5	30.1	656.8	28.6	4.35
2006	699.2	30.5	633.6	28.5	4.50
2007	709.7	32.0	631.0	29.7	4.71
Male					
2001	725.0	36.4	879.5	39.9	4.54
2002	720.3	37.1	857.7	40.6	4.73
2003	729.5	36.2	851.2	39.3	4.62
2004	718.7	35.2	824.1	37.8	4.59
2005	722.6	37.3	811.2	39.6	4.88
2006	715.1	36.8	783.2	38.5	4.92
2007	725.9	39.0	778.3	40.4	5.19
Female					
2001	679.1	21.2	572.7	18.5	3.23
2002	688.5	22.8	571.3	19.7	3.45
2003	691.4	22.9	564.3	19.3	3.42
2004	686.0	22.4	547.8	18.4	3.36
2005	694.5	23.0	650.3	18.9	2.91
2006	683.5	24.3	626.4	19.4	3.10
2007	693.8	25.0	624.7	20.0	3.20

[a] Standardized according to the entire 2001 Canadian population.

Newfoundland and Labrador. Ontario also had a low unintentional injury mortality (26.3 per 100 000), which was similar to Newfoundland and Labrador, but all-cause mortality was very different in the two provinces, 655.9 per 100 000 in Ontario versus 802.9 per 100 000 in Newfoundland and Labrador. The differences between the other provinces were relatively small for both all-cause mortality and unintentional injury mortality (Table 2). Table 2 also shows that the unintentional injury mortality and the ratio of unintentional injury versus all-cause mortalities were higher in males than in females across all provinces/territories.

Unintentional injury deaths were less common among children (< 7 per 100 000) than among youths and adults (Table 3). Unintentional injury mortality was generally similar for those aged 15 to 59 years (28.5–37.7 per 100 000 in males and 8.5–12.7 per 100 000 in females). After age 60 years, mortality increased steadily with age from 35.3 per 100 000 in the 60- to 64-year age group to 801.0 per 100 000 in the 90-year-plus age group in men and from 14.4 to 663.1 per 100 000 in women. In all the age groups, males were more likely to die of unintentional injuries (Table 3). The male to female mortality ratio increased with age after 5 to 9 years (1.34), peaked at 25 to 29 years (3.76) and then steadily decreased with age. Table 3 also shows that of unintentional injury deaths from identified causes, motor vehicle traffic crashes were most common in males, with a mortality rate of 10.2 per 100 000, followed by falls (7.7 per 100 000) and poisoning (5.1 per 100 000). In females, the first three identified causes for unintentional injury death were falls, motor vehicle traffic crashes and poisoning with the mortality rates being 7.9, 4.5 and 2.2 per 100 000, respectively. Cause-specific unintentional injury mortalities were all higher in males than in females except for fall mortality (Table 3). Overall, falls accounted for 26% of all unintentional injury deaths, motor vehicle traffic crashes for 24% and poisoning for 12% (Figure 1).

TABLE 2
Average mortality by province/territory, Canada (excluding Quebec), total and by sex, 2001–2007

Province	Crude mortality		Standardized mortality[a]		
	All causes, per 100 000	Unintentional injury, per 100 000	All causes, per 100 000	Unintentional injury, per 100 000	Unintentional injury/All causes, %
Total					
British Columbia	715.7	32.1	626.8	30.1	4.80
Alberta	582.4	27.8	660.7	28.7	4.34
Saskatchewan	896.0	42.5	693.8	37.5	5.41
Manitoba	843.7	38.1	720.3	34.7	4.82
Ontario	680.7	27.0	655.9	26.3	4.01
New Brunswick	831.2	38.3	713.2	35.3	4.95
Nova Scotia	871.2	35.6	733.9	31.8	4.33
Prince Edward Island	837.9	35.5	703.7	32.4	4.60
Newfoundland and Labrador	841.0	25.2	802.9	24.8	3.09
Territories[b]	396.5	52.3	842.3	69.1	8.20
Male					
British Columbia	741.7	42.0	769.8	42.5	5.52
Alberta	603.3	37.3	822.0	41.0	4.99
Saskatchewan	926.4	53.3	881.0	52.8	5.99
Manitoba	850.1	44.9	903.0	46.6	5.16
Ontario	691.7	31.5	808.1	35.0	4.33
New Brunswick	856.7	49.8	906.6	50.9	5.61
Nova Scotia	892.3	43.1	921.3	44.3	4.81
Prince Edward Island	852.6	43.4	895.2	45.3	5.06
Newfoundland and Labrador	905.2	32.9	1022.9	34.7	3.39
Territories[b]	486.9	74.8	1023.9	92.1	9.00
Female					
British Columbia	690.0	22.3	520.0	18.6	3.58
Alberta	561.0	18.1	543.9	17.6	3.24
Saskatchewan	866.1	31.9	557.7	23.5	4.21
Manitoba	837.5	31.4	587.7	24.4	4.15
Ontario	670.1	22.7	544.7	18.7	3.43
New Brunswick	806.5	27.2	572.9	21.4	3.74
Nova Scotia	851.2	28.4	596.4	20.7	3.47
Prince Edward Island	823.9	28.0	566.9	20.1	3.55
Newfoundland and Labrador	778.6	17.8	647.5	15.3	2.36
Territories[b]	300.5	28.5	695.6	45.2	6.50

[a] Standardized according to the entire 2001 Canadian population.

[b] Yukon, Northwest Territory, Nunavut.

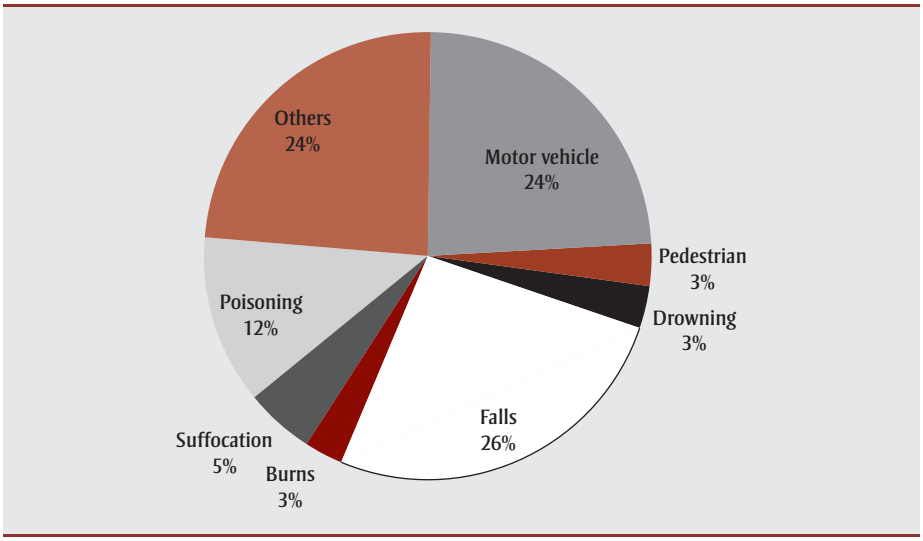
Although unintentional injury mortality increased sharply after 60 years of age, it comprised a much higher proportion of all deaths in younger age groups (Table 4), peaking at age 15 to 19 years (45.2%) for both males (46.7%) and females (41.8%), then gradually decreasing (Table 4).

We investigated major external causes for unintentional injury mortality separately, including motor vehicle traffic crashes, falls, poisoning, pedestrian (traffic-

TABLE 3
Mortality and male to female ratio for mortality due to unintentional injury by age and external causes, Canada (excluding Quebec), 2001–2007

	Mortality, per 100 000		Ratio Male:Female
	Male	Female	
Age group, years			
0–4	7.5	5.3	1.42
5–9	4.3	3.2	1.34
10–14	6.2	3.5	1.77
15–19	29.0	12.0	2.42
20–24	37.7	12.1	3.12
25–29	32.0	8.5	3.76
30–34	28.5	8.6	3.31
35–39	30.4	9.8	3.10
40–44	33.1	10.6	3.12
45–49	34.8	11.9	2.92
50–54	33.3	11.5	2.90
55–59	33.3	12.7	2.62
60–64	35.3	14.4	2.45
65–69	42.8	21.0	2.04
70–74	56.9	32.3	1.76
75–79	98.0	61.4	1.60
80–84	187.2	122.5	1.53
85–89	362.9	271.4	1.33
≥90	801.0	663.1	1.21
External causes			
Motor vehicle crashes	10.2	4.5	2.3
Pedestrian (traffic-related)	1.3	0.8	1.6
Pedal cycle	0.4	0.1	5.4
Recreation boating	0.2	0.0	10.4
Drowning	1.5	0.4	4.3
Falls	7.7	7.9	1.0
Burns	1.1	0.6	1.9
Suffocation	1.6	1.1	1.4
Poisoning	5.1	2.2	2.3
Others	7.9	5.6	1.4

FIGURE 1
Proportion of unintentional injury deaths by cause in Canada excluding Quebec, 2001–2007



related), drowning, burns, and suffocation in males (Table 5; Figure 2) and in females (Table 6; Figure 3). There was a substantial year after year increase in mortality as a result of injuries due to falls in both males ($p < .01$) and females ($p < .01$). For other type of injuries, age-standardized mortalities either decreased slightly (burns and drowning in males, motor vehicle crashes and burns in females) or showed no significant changes. The risk of death caused by motor vehicle traffic crashes and drowning was significantly higher in summer months and was more marked in males than females. Deaths caused by falls and burns were more common in winter months. More poisoning deaths could be seen in March and April ($p < .05$) and pedestrian accident deaths in September and October ($p < .05$) and November and December ($p < .01$) when compared with January and February. There was no significant difference between the month periods for suffocation.

Discussion

Our study demonstrated that age- and sex-standardized mortality from all unintentional injuries was stable during the 7-year study period whereas overall mortality declined approximately 10% and the proportion of unintentional injury versus overall mortalities increased from 4.1% to 4.7%. This indicated that the share of unintentional injury in all causes for mortality is on the increase in Canada. All unintentional injury mortality in males, as well as cause-specific injury mortalities but with the exception of fall mortality, exceeded those in females.

The three territories had the highest overall mortality and unintentional injury mortality. The unintentional injury versus overall mortality ratio was almost double in the three territories compared with the nine provinces. A previous population-based case-control study conducted in the Northwest Territories demonstrated that being male, aged over 14 years, living in remote communities, living in the far north, and being Aboriginal were risk factors for injury mortality.[11] There is a higher proportion of Aboriginal people in the territories compared with the rest of Canada. A study conducted among Albertan children showed that Aboriginal children had a significantly higher risk for both intentional and unintentional injury deaths.[12] Injury mortality rates among Indigenous people in the United States and Australia are approximately 2 to 3 times greater than rates for non-Aboriginal populations.[13]

Motor vehicle traffic crashes and falls were two major causes for unintentional injury deaths in Canada. The former was a more common cause in males than females and was a main reason for the markedly increased mortality from unintentional injuries in youths and young adults. The mortality due to motor vehicle traffic crashes changed little year after year during the study period in males and declined slightly in females. However, the data showed a clear seasonal pattern with a significantly increased risk in summer and more so in males than in females. During the traditional summer vacation

TABLE 4
Overall and unintentional injury mortality (per 100 000) by age and sex, Canada (excluding Quebec), 2001–2007

Age group, years	Total			Male			Female		
	All causes, per 100 000	Unintentional injury, per 100 000	Unintentional injury/All causes, %	All causes, per 100 000	Unintentional injury, per 100 000	Unintentional injury/All causes, %	All, per 100 000	Unintentional injury, per 100 000	Unintentional injury/All causes, %
0–4	124.4	6.43	5.2	135.4	7.5	5.5	112.9	5.3	4.7
5–9	11.7	3.73	31.9	13.0	4.3	33.1	10.3	3.2	30.8
10–14	14.3	4.89	34.2	16.2	6.2	38.3	12.3	3.5	28.2
15–19	45.9	20.78	45.2	62.1	29.0	46.7	28.8	12.0	41.8
20–24	60.0	25.19	42.0	85.8	37.7	43.9	33.1	12.1	36.6
25–29	58.5	20.35	34.8	82.6	32.0	38.7	34.0	8.5	25.0
30–34	69.6	18.60	26.7	92.1	28.5	30.9	46.8	8.6	18.4
35–39	95.9	20.12	21.0	122.1	30.4	24.9	69.4	9.8	14.1
40–44	140.6	21.91	15.6	174.9	33.1	18.9	106.0	10.6	10.0
45–49	221.8	23.35	10.5	272.9	34.8	12.8	170.8	11.9	7.0
50–54	349.8	22.34	6.4	431.0	33.3	7.7	269.7	11.5	4.3
55–59	545.1	22.92	4.2	674.1	33.3	4.9	417.8	12.7	3.0
60–64	881.9	24.67	2.8	1092.7	35.3	3.2	677.4	14.4	2.1
65–69	1406.4	31.51	2.2	1751.8	42.8	2.4	1082.9	21.0	1.9
70–74	2270.4	43.83	1.9	2850.2	56.9	2.0	1755.2	32.3	1.8
75–79	3711.0	77.39	2.1	4684.2	98.0	2.1	2953.0	61.4	2.1
80–84	6157.3	147.78	2.4	7759.1	187.2	2.4	5130.4	122.5	2.4
85–89	10708.7	302.40	2.8	13173.0	362.9	2.8	9445.9	271.4	2.9
≥ 90	20590.7	700.09	3.4	23399.0	801.0	3.4	19562.0	663.1	3.4

months, people may drive longer distances while on vacation and teens and young adults may have more opportunities to drive and ride in cars,[1] and hence are more likely to be exposed to vehicle- and traffic-related risk factors. Effective interventions on motor vehicle crashes are most important for reducing unintentional injury mortality among youths and young adults, especially for males.

Mortality due to falls was the only cause-specific mortality that showed a steady increase during the study period, and it was slightly more common in females than males. Fall injury accounted for about one-third of all unintentional injury deaths in adults, and was the principal reason for the dramatic increase in mortality due to unintentional injury with age in the elderly. Worldwide, motor vehicle crashes account for 33% while falls only account for 11% of unintentional injury death,[1] but in this study they

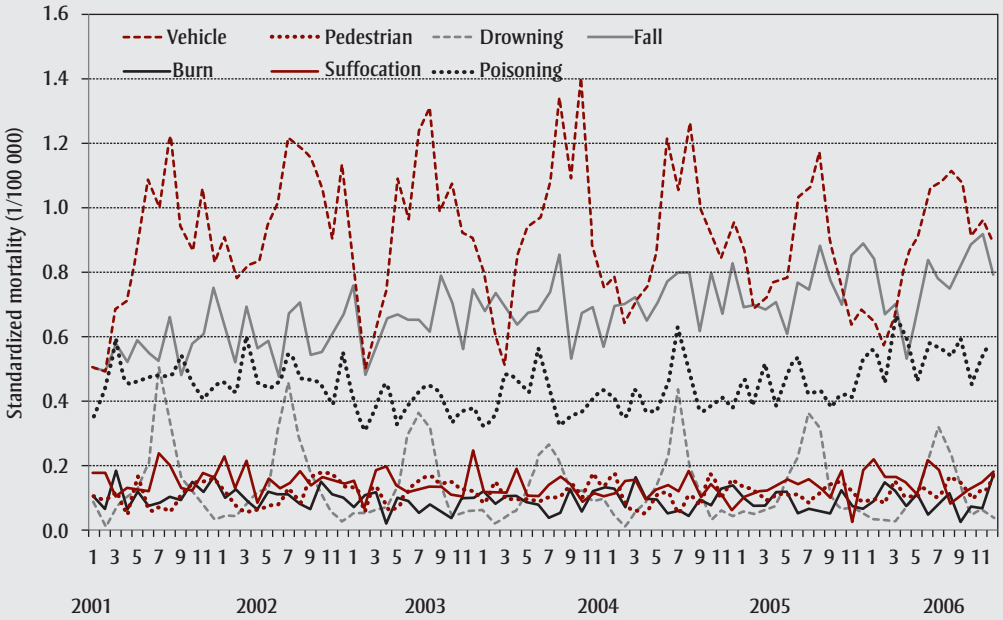
TABLE 5
Results of time-series analysis (autoregressive model) for major types of unintentional injury mortality (per 100 000) in males

Variable	Regression coefficient (Standard error)						
	Motor vehicle	Falls	Poisoning	Drowning	Pedestrian	Burns	Suffocation
Secular year	–0.0080 (0.0072)	0.0342 (0.0039)**	0.0090 (0.0062)	–0.0069 (0.0023)**	0.0012 (0.0016)	–0.0028 (0.0014)*	–0.0023 (0.0021)
Month							
1–2	–0.4768 (0.0490)**	–0.0742 (0.0280)**	–0.0729 (0.0325)*	–0.2976 (0.0170)**	0.0011 (0.0118)	0.0311 (0.0105)**	–0.0124 (0.0150)
3–4	–0.4310 (0.0490)**	–0.0936 (0.0280)**	0.0354 (0.0318)	–0.2743 (0.0169)**	–0.0173 (0.0118)	0.0310 (0.0105)**	–0.0116 (0.0150)
5–6	–0.1822 (0.0484)**	–0.0701 (0.0287)*	–0.0053 (0.0281)	–0.1435 (0.0177)**	–0.0026 (0.0121)	0.0167 (0.0111)	–0.0227 (0.0154)
7–8	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
9–10	–0.1600 (0.0484)**	–0.0521 (0.0287)	–0.0310 (0.0281)	–0.2383 (0.0177)**	0.0336 (0.0121)**	0.0138 (0.0111)	–0.0306 (0.0154)
11–12	–0.2846 (0.0490)**	0.0029 (0.0280)	–0.0243 (0.0317)	–0.2806 (0.0169)**	0.0281 (0.0118)*	0.0449 (0.0105)**	–0.0202 (0.0150)

* $p < .05$

** $p < .01$

FIGURE 2
Monthly standardized mortality due to different types of unintentional injury in males, Canada (excluding Quebec), 2001–2007



were 24% and 26%, respectively (Figure 1). The aging process and low levels of bone mineral density are closely associated with the severity of an injury and the consequence of the fall.[8,14–19] However, we do not know if aging and bone mineral density are the main reasons for the steady increase in mortality in the 7-year study period. Other factors such as medication use, especially in older people,[20] overweight and obesity,[21,22] engagement in physical activity,[23,24] utilization of medical products and day-to-day activities[25–28] warrant further investigation for possible impact on the uptrend of mortality from falls in the Canadian population. Our study also showed a seasonal pattern for fall mortality, that is, it was the highest in November and December. One study from the United States found that fall injuries were associated with holiday decorating or related activities, which is also likely in our current context.[29] Weather is probably another important reason.[30,31]

Poisoning was the third leading cause of unintentional injury mortality in Canada, and accounted for 14% of unintentional injury deaths in males and 10% in females (data not shown). Unintentional poisoning may be work-related, and other common agents are household chemicals and pesticides, medications and plants.[32–35] Other causes of unintentional deaths such as drowning and burns were less common. Our data showed that deaths due to drowning most frequently happened in summer, and that males versus females and children versus adults accounted for a higher proportion of drowning-related deaths. Most drowning accidents are related to recreation or leisure. Our data also showed an increased mortality due to burns in winter but no seasonal variations for suffocation.

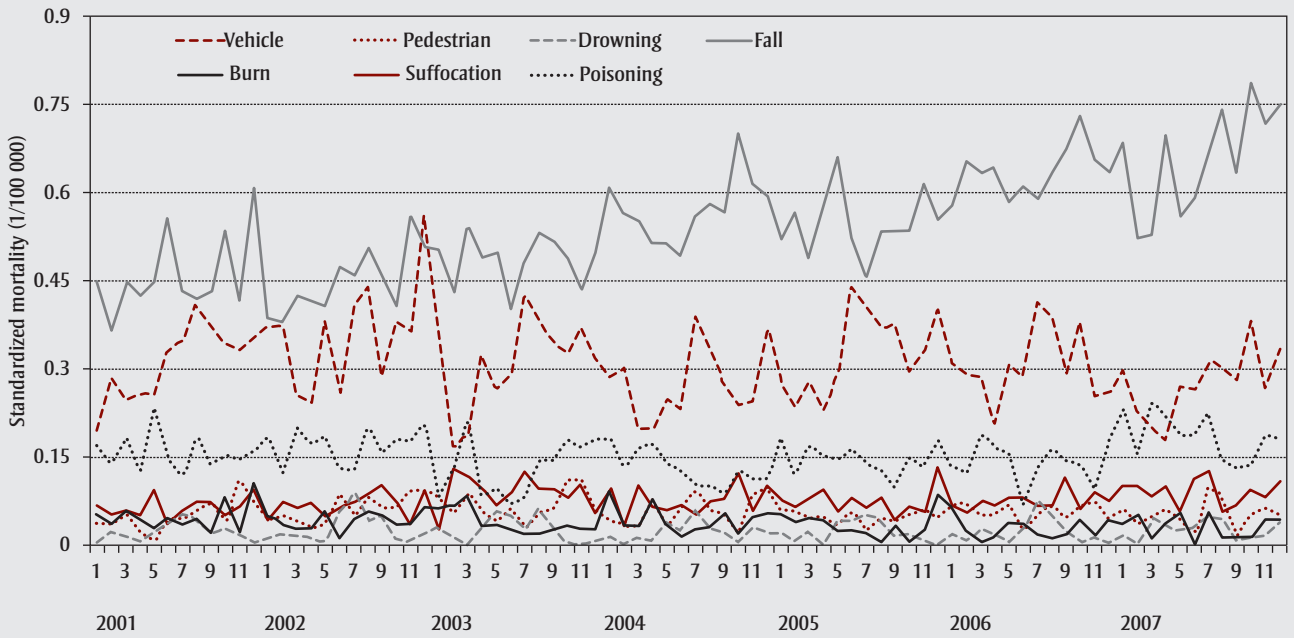
TABLE 6
Results of time-series analysis (autoregressive model) for major types of unintentional injury mortality (per 100 000) in females

	Regression coefficient (Standard error)						
Variable	Motor vehicle	Falls	Poisoning	Drowning	Pedestrian	Burns	Suffocation
Secular year	−0.0069 (0.0033)*	0.0365 (0.0034)**	0.0016 (0.0025)	−0.0001 (0.0007)	−0.0006 (0.0012)	−0.0026 (0.0009)**	0.0028 (0.0011)*
Month							
1–2	−0.0984 (0.0217)**	−0.0277 (0.0230)	−0.0005 (0.0157)	−0.0360 (0.0049)**	−0.0073 (0.0081)	0.0216 (0.0067)**	−0.0079 (0.0080)
3–4	−0.1435 (0.0217)**	−0.0158 (0.0230)	0.0297 (0.0156)	−0.0317 (0.0049)**	−0.0094 (0.0081)	0.0097 (0.0067)	0.0011 (0.0080)
5–6	−0.0859 (0.0211)**	−0.0204 (0.0228)	0.0038 (0.0148)	−0.0182 (0.0048)**	−0.0155 (0.0079)	0.0040 (0.0070)	−0.0074 (0.0082)
7–8	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
9–10	−0.0516 (0.0211)*	0.0284 (0.0228)	−0.0046 (0.0148)	−0.0326 (0.0049)**	−0.0063 (0.0079)	0.0022 (0.0069)	0.0007 (0.0082)
11–12	−0.0422 (0.0217)	0.0388 (0.0230)	0.0094 (0.0156)	−0.0357 (0.0049)**	0.0153 (0.0081)	0.0174 (0.0067)*	0.0029 (0.0080)

* p value < .05

** p value < .01

FIGURE 3
Monthly standardized mortality due to different types of unintentional injury in females in Canada (excluding Quebec), 2001–2007



Limitations

Death registration is mandatory in Canada and therefore there is minimal missing vital statistics data. However, the underlying cause, defined as the disease or injury that initiated the chain of events leading directly to death, is considered. Some people might not die instantly after an injury, and subsequent conditions (e.g. heart failure) might be coded as primary cause of death. This method of death registration relies on medical examiners or coroners' judgment; it may happen that an injury is closely related to the death but is not considered the underlying cause. Since secondary diagnoses are excluded, it may underestimate the true burden of unintentional injury mortality. Miscoding and data entry errors may also result in misclassification of information on cause of deaths and external causes of injuries. In addition, the study period of 7 years is relatively short.

Conclusion

Overall unintentional injury mortality changed little from year to year while overall mortality showed a steady decline in Canada. The three territories had the highest unintentional injury mortality, both absolutely and as a share of overall mortality. Motor vehicle traffic crashes and falls were the leading causes of injury death. Fall mortality was the only type of unintentional injury mortality that showed an annual increase. Death from fall injury was more common in females than males while other types of injury death were stable and were more common in males than in females. There were seasonal patterns for some types of unintentional injury mortalities such as higher risks of death due to motor vehicle traffic crashes and drowning in summer and falls and burns/fire in winter. The increasing share of overall unintentional injury mortality versus all-cause mortality and the increasing trend for fall mortality call for more research on risk factor identification and effective interventions.

Acknowledgments

We would like to thank the following for their role in the "Consumer product- related injury and risk assessment" project: Dr. Howard Morrison, senior supervisor and director of the project; Mr. Doug Hopkins, manager of the project; and Ms. Caroline Da Silva, responsible for the management and coordination of the project.

References

1. Chandran A, Hyder AA, Peek-Asa C. The global burden of unintentional injuries and an agenda for progress. Epidemiol Rev. 2010;32:110–20.

2. Krug EG, Sharma GK, Lozano R. The global burden of injuries. Am J Public Health. 2000;90:523–6.

3. Peden MM, McGee K, Krug E, editors. Injury: a leading cause of the global burden of disease, 2000. Geneva (CH): World Health Organization; 2002.

4. Violence, injuries and disability biennial report, 2006–2007. Geneva (CH): World Health Organization; 2008.

5. Mulholland E; Advisory Committee on the Economic Burden of Injury in Canada. The economic burden of injury in Canada. Toronto (ON): SMARTRISK; 2009.

6. Harrison JE. Injury classification: balancing continuity and utility. Inj Control Safety Promot. 2000;7:51-63.

7. Curry AE, Hafetz J, Kallan MJ, Winston FK, Durbin DR. Prevalence of teen driver errors leading to serious motor vehicle crashes. Accid Anal Prev. 2011;43:1285-90.

8. Kannus P, Parkkari J, Niemi S, Palvanen M. Fall-induced deaths among elderly people. Am J Public Health. 2005;95:422-4.

9. Statistics Canada. Annual Demographic Estimates: Canada, Provinces and Territories [Internet]. Ottawa (ON): 2011 Sep. [Statistics Canada, Catalogue No.: 91-215-X]. Available from: http://www.statcan.gc.ca/pub/91-215-x/91-215-x2011000-eng.htm

10. Statistics Canada. Annual Demographic Statistics, 2005. Ottawa (ON): 2006. [Statistics Canada, Catalogue No.: CS91-213/2005].

11. Mo D. Injury mortality risk assessment and targeting the subpopulations for prevention in the Northwest Territories, Canada. Int J Circumpolar Health. 2001;60:391–9.

12. Harrop AR, Brant RF, Ghali WA, Macarthur C. Injury mortality rates in Native and non-Native children: a population-based study. Public Health Rep. 2007;122:339–46.

13. Stevenson MR, Wallace LJ, Harrison J, Moller J, Smith RJ. At risk in two worlds: injury mortality among indigenous people in the US and Australia, 1990–92. Aust N Z J Public Health. 1998;22:641–4.

14. Rauh MJ, Nichols JF, Barrack MT. Relationships among injury and disordered eating, menstrual dysfunction, and low bone mineral density in high school athletes: a prospective study. J Athl Train. 2010;45:243–52.

15. Sakai A, Oshige T, Zenke Y, Yamanaka Y, Otsuka H, Nakamura T. Shorter unipedal standing time and lower bone mineral density in women with distal radius fractures. Osteoporos Int. 2009;21:733–9.

16. Sone T. [Increase in bone mineral density and its effect on fracture risk]. Clin Calcium. 2005;15:625–9.

17. Hartholt KA, Stevens JA, Polinder S, van der Cammen TJ, Patka P. Increase in fall-related hospitalizations in the United States, 2001–2008. J Trauma. 2011;71:255–8.

18. Hartholt KA, van Beeck EF, Polinder S, van der Velde N, van Lieshout EM, Panneman MJ, et al. Societal consequences of falls in the older population: injuries, healthcare costs, and long-term reduced quality of life. J Trauma. 2011;71:748–53.

19. Melton LJ 3rd, Riggs BL. Risk factors for injury after a fall. Clin Geriatr Med. 1985;1:525–39.

20. Hartikainen S, Lonnroos E, Louhivuori K. Medication as a risk factor for falls: critical systematic review. J Gerontol A Biol Sci Med Sci. 2007;62:1172–81.

21. Finkelstein EA, Chen H, Prabhu M, Trogdon JG, Corso PS. The relationship between obesity and injuries among U.S. adults. Am J Health Promot. 2007;21:460–8.

22. Hu HY, Chou YJ, Chou P, Chen LK, Huang N. Association between obesity and injury among Taiwanese adults. Int J Obes (Lond). 2009;33:878–84.

23. Appleby PN, Allen NE, Roddam AW, Key TJ. Physical activity and fracture risk: a prospective study of 1898 incident fractures among 34,696 British men and women. J Bone Miner Metab. 2008;26:191–8.

24. Gill DP, Zou GY, Jones GR, Speechley M. Injurious falls are associated with lower household but higher recreational physical activities in community-dwelling older male veterans. Gerontology. 2008;54:106–15.

25. Belechri M, Petridou E, Trichopoulos D. Bunk versus conventional beds: a comparative assessment of fall injury risk. J Epidemiol Community Health. 2002;56:413–7.

26. Dellinger AM, Boyd RM, Haileyesus T. Fall injuries in older adults from an unusual source: entering and exiting a vehicle. J Am Geriatr Soc. 2008;56:609–14.

27. O'Neil J, Steele GK, Huisingh C, Smith GA. Escalator-related injuries among older adults in the United States, 1991–2005. Accid Anal Prev. 2008;40:527–33.

28. Stevens JA, Thomas K, Teh L, Greenspan AI. Unintentional fall injuries associated with walkers and canes in older adults treated in U.S. emergency departments. J Am Geriatr Soc. 2009;57:1464–9.

29. Centers for Disease Control and Prevention (CDC). Fall-related injuries during the holiday season–United States, 2000–2003. MMWR Morb Mortal Wkly Rep. 2004;53:1127–9.

30. Jacobsen SJ, Sargent DJ, Atkinson EJ, O'Fallon WM, Melton LJ 3rd. Contribution of weather to the seasonality of distal forearm fractures: a population-based study in Rochester, Minnesota. Osteoporos Int. 1999;9:254–9.

31. Wareham K, Johansen A, Stone MD, Saunders J, Jones S, Lyons RA. Seasonal variation in the incidence of wrist and forearm fractures, and its consequences. Injury. 2003;34:219–22.

32. Miech R, Koester S, Dorsey-Holliman B. Increasing US mortality due to accidental poisoning: the role of the baby boom cohort. Addiction. 2010;106:806–15.

33. Yoon YH, Stinson FS, Yi HY, Dufour MC. Accidental alcohol poisoning mortality in the United States, 1996–1998. Alcohol Res Health. 2003;27:110–8.

34. Kivisto JE, Parkkari J, Mattila VM, Hoppu K. Poisoning deaths among Finnish children from 1969 to 2003. Acta Paediatr. 2009;98:1661–6.

35. Meyer S, Eddleston M, Bailey B, Desel H, Gottschling S, Gortner L. Unintentional household poisoning in children. Klin Padiatr. 2007;219:254–70.

Report summary – Health-Adjusted Life Expectancy in Canada: 2012 Report by the Public Health Agency of Canada

The Public Health Agency of Canada Steering Committee on Health-Adjusted Life Expectancy (1)

Health-adjusted life expectancy is an indicator of the average number of years that an individual is expected to live in a healthy state. It is a summary measure that combines both quantity of life and quality of life. In other words, it combines mortality and morbidity experience into a single summary measure of population health. It can be used to measure the burden of disease and injury in the population, risk factors and the performance of public health efforts.

This report, entitled *Health-Adjusted Life Expectancy in Canada: 2012 Report by the Public Health Agency of Canada*,[1] provides estimates of health-adjusted life expectancy among Canadians with and without selected chronic diseases (diabetes and cancer) and chronic conditions (hypertension), and by socio-economic status (income). Estimates are provided for females and males and for people of different ages.

Low socio-economic status is associated with a loss in health-adjusted life expectancy. In 2001, Canadian women and men in the top one-third income group had a health-adjusted life expectancy at birth of 72.3 years and 70.5 years, respectively. Compared with being in the highest income group, being in the bottom one-third income group was associated with a loss of health-adjusted life expectancy at birth of 3.2 years for women and 4.7 years for men.

Chronic diseases and conditions also are associated with a significant loss in health-adjusted life expectancy. The estimates of health-adjusted life expectancy by chronic disease status in this report were calculated based on the mortality and morbidity experience of people with and without diabetes and/or hypertension (high blood pressure) for the 2004–2006 period and of people with and without cancer for the 2002–2005 period. According to the results of this study, the diabetes cohort at age 55 had a loss in health-adjusted life expectancy of 5.8 years for women and 5.3 years for men. The cohort of people with high blood pressure at age 55 had a loss of 2.0 years and 2.7 years for females and males, respectively. The cancer cohort at age 65 had a loss in health-adjusted life expectancy of 10.3 years for women and 9.2 years for men.

This report provides information for use in public health research, policy development and practice. Future reports could extend the scope to include health-adjusted life expectancy by behavioural risk factor status (such as obesity, physical inactivity and smoking).

The full report is available at http://www.phac-aspc.gc.ca/cd-mc/hale-evas-pdf-eng.php

References

1. Public Health Agency of Canada Steering Committee on Health-Adjusted Life Expectancy. *Health-Adjusted Life Expectancy in Canada: 2012 Report by the Public Health Agency of Canada*. Ottawa (ON): Public Health Agency of Canada; 2012. Available from: http://www.phac-aspc.gc.ca/cd-mc/hale-evas-eng.php

Author references:

1. Members of the Public Health Agency of Canada Steering Committee on Health-Adjusted Life Expectancy: Priya Bakshi, Bernard C. K. Choi (Chair), Alan Diener, Eric Driscoll, Joellyn Ellison, XiaoHong Jiang, Albert Kwan, Lidia Loukine, Wei Luo, Howard Morrison, Heather Orpana, August J. Saaltink, Robert Semenciw, Feng Wang, Chris Waters, Carl Yue, Rita Zhang.

Correspondence: Dr. Bernard C.K. Choi, Senior Research Scientist, Centre for Chronic Disease Prevention, Public Health Agency of Canada, A/L 6806B, 785 Carling Avenue Ottawa ON K1A 0K9; Tel.: 613-957-1074; Email: Bernard.Choi@phac-aspc.gc.ca

CSEB Student Conference 2012 abstract winners

Preface

Chronic Diseases and Injuries in Canada (*CDIC*) was pleased to manage, once again, the student abstract contest for the Canadian Society for Epidemiology and Biostatistics (CSEB) Student Conference, which was held at the University of Saskatchewan in May 2012. An editorial panel from the Public Health Agency of Canada judged 42 abstract submissions and selected the top 7 to be published in this issue of the journal.

The editorial panel consisted of the following members:

- Howard Morrison, PhD, Editor-in-Chief, *CDIC*
- Kenneth Johnson, PhD, Senior Epidemiologist
- Ania Syrowatka, MSc, Epidemiologist
- Michelle Tracy, MA, Managing Editor, *CDIC*

The selected abstracts were judged on their originality, clarity, scientific and technical excellence, and potential impact. The following questions helped guide the judges:

1. Is it relevant to chronic diseases and/or injuries?
2. Are the data Canadian, or if not, do the authors place the issue in the context of Canada?
3. Does the study have national relevance? Local studies are of interest only to the extent that they provide sufficient details to allow them to be useful to other, non-local researchers.
4. Does the study address a significant public health issue?
5. Is the study scientifically rigorous?
6. Are the methods and/or results novel, or is it a significant improvement on previous studies of the same issue?
7. Can you imagine any reasonable circumstance where a different author would reference this study?

Since 2009, *CDIC* has collaborated with CSEB to foster publishing opportunities for students. *CDIC* is proud to collaborate with CSEB again this year and to encourage students in their publishing efforts. On behalf of the *CDIC* editorial team, I would like to thank all students who submitted their abstracts and to congratulate the winners. Having one's abstract published in a peer-reviewed journal is a good place to start in science publishing! We look forward to seeing future submissions of full research articles.

Michelle Tracy, MA
Managing Editor, *Chronic Diseases and Injuries in Canada*

The effect of gender and geography on the self-rated mental health of single parents

A. Banerjee, MBBS (1); B. Janzen, PhD (1)

Introduction: Single parents, one of the most socio-economically vulnerable groups in Canada, consistently report poorer mental health compared with cohabiting parents. However, most of the studies examining the mental health of single parents typically fail to consider whether they live in urban or rural areas, or else focus exclusively on urban dwellers. This is despite that 1) a growing body of research points to place (e.g. rurality) as a determinant of health and 2) in 2006, just over 13% of families residing in rural Canada were headed by a single parent. In addition, little is known about the mental health of single fathers despite their increasing numbers in Canada.

Objective: To determine if the mental health of single parents varies by gender and/or rurality and what factors (e.g. economic, social) contribute to variations in mental health by gender and/or rurality.

Methods: From Statistics Canada's 2007/2008 Canadian Community Health Survey (Master file), we selected for analysis a subsample of 18- to 64-year-old single parents with at least one child aged under 25 years living with them. The dependent variable was self-rated mental health, and the primary independent variables were sex and residence, the latter based on Statistics Canada's metropolitan influenced zone (MIZ) classification. Covariates included age, employment status, household income, home ownership, food security and sense of belonging to a community. A series of univariate, bivariate and multivariable logistic regression analyses were conducted to answer the research questions. Sampling weights and a bootstrap variance estimation program were used to address the complex sampling strategy.

Results: The sample (weighted) consisted of 1 024 856 single parents. Single mothers made up 81% of the sample, and the majority of single parents (86%) lived in urban areas. Overall, 9.3% of single mothers and 7.0% of single fathers reported fair/poor mental health. The proportion of single fathers with fair/poor mental health was 6.7% in Census Metropolitan Areas/Census Agglomerations (CMAs/CAs), 11.0% in strong/moderate MIZ and 4.6% in weak/no MIZ. Among single mothers, the proportion with fair/poor mental health was 9.5% in CMA/CA, 7.9% in strong/moderate MIZ and 8.2% in weak/no MIZ. Inspection of the preliminary results are suggestive of variation in self-rated mental health and access to economic/social resources (e.g. employment, food security, sense of belonging) according to gender and/or degree of rurality; however, additional analyses applying the appropriate variance estimation techniques are required to determine whether these differences are statistically significant. In addition, multiple logistic regression needs to be conducted to determine if any observed gender/residence differences in single parents' mental health remain statistically significant after adjustment for key covariates.

Conclusion: The results of the study enhance understanding of the diverse experiences of Canadian single parents and inform the development of more targeted policies directed at improving their mental health.

Keywords: health policy, social policy, mental health, social epidemiology, behavioural epidemiology, Canadian Community Health Survey

Author references:

1. Department of Community Health and Epidemiology, College of Medicine, University of Saskatchewan, Saskatchewan, Canada

Correspondence: Ankona Banerjee; Email: asb426@mail.usask.ca

Sitting, screen time and suicide: the relationship between sedentary activity and suicide ideation in Canadian adolescents and young adults

M. A. Bélair, BSSc (2); I. Colman, PhD (2)

Introduction: Suicide is the second leading cause of death among 15- to 24-year-olds in Canada. At 21.4%, suicide rates among adolescents have remained constant despite declining rates in other developed countries. More than 50% of adolescents who commit suicide have a major depressive disorder.

Objective: To investigate whether a link exists between sedentary activity and suicide ideation in Canadians aged 15 to 24 years.

Methods: Using an initial sample of 8356 from the Canadian Community Health Survey (CCHS), Cycle 4 (2007/2008), 7914 adolescents and young adults aged 15 to 24 years were included in the analysis. We conducted Breslow-Day tests for effect modification to determine the need for stratification and multivariate logistic regression analysis to assess the relationship between sedentary activity and lifetime suicide ideation. Sedentary activity was classified into three categories: 0 to 15, 15 to 34 and 35 plus hours per week.

Results: Those who were sedentary 15 to 34 h/wk had odds ratio (OR) of lifetime suicidal ideation 1.18 times higher (95% confidence interval [CI]: 0.99–1.41) than those who were sedentary 0 to 15 h/wk, while those who were sedentary 35+ h/wk had OR 1.41 times higher (95% CI: 1.15–1.74) than those in the least sedentary group. When controlling for sex, age, self-perceived health, self-perceived mental health and body mass index (BMI), as well as modelling an interaction between sex and self-perceived health and between sex and BMI, the relationship between suicide ideation and sedentary activity for youth and young adults in the 35+ h/wk exposure category remained significant with an adjusted OR of 1.33 (95% CI: 1.06–1.68), whereas that for those in the 15 to 34 h/wk exposure category was non-significant at 1.11 (95% CI: 0.92–1.35). To interpret the interaction terms, we explored sex-stratified models. For males reporting poor/fair self-perceived health, OR of lifetime suicidal ideation was 1.26 (95% CI: 0.82–1.26) whereas for females OR was 2.33 (95% CI: 1.68–3.23) compared with the reference (good/very good/excellent self-perceived health). A 10-unit increase in BMI decreased odds of lifetime suicide ideation for males by 0.97 (95% CI: 0.73–1.28), whereas it increased odds of lifetime suicide ideation by 1.58 times (95% CI: 1.29–1.92) for females.

Conclusion: A relationship exists between sedentary activity levels and lifetime suicide ideation among youth and young adults with 35 or more hours of sedentary activity per week. This is of concern as a greater proportion of adolescents and young adults spends more time being sedentary. However, the cross-sectional nature of the CCHS does not permit us to comment on the direction of this relationship. Further research using longitudinal data is recommended.

Keywords: mental health, Canadian Community Health Survey, depression, suicide

Author references:

2. Department of Epidemiology and Community Medicine, University of Ottawa, Ottawa, Ontario, Canada

Correspondence: Marc-André Bélair; Email: mabelair@mabelair.com

Health status and service use among homeless individuals with mental illness: consistency between self-report and administrative health records in the At Home/Chez Soi Multi-site Trial

A. Hinds, MSc (3); J. Distasio, PhD (4); P. J. Martens, PhD (3, 5); M. Smith, MSc (5)

Introduction: Homeless individuals with poor health use health care services frequently.

Objective: To examine health status, health care and prescription drug use among mentally ill, homeless individuals and compare self-report and administrative data claims to estimate the degree of agreement between the two sources.

Methods: Baseline survey data from 100 participants of the Winnipeg site of the Mental Health Commission of Canada's At Home / Chez Soi research project were linked to de-identified administrative health records stored in the Repository at the Manitoba Centre for Health Policy. We analyzed demographic characteristics, homelessness histories and health service use as well as disease status for asthma, hypertension, arthritis and diabetes (using previously validated definitions). Participants were similarly classified using their survey responses. The degree of agreement between the two data sets was evaluated using cross tabulations and the kappa [κ] statistic.

Results: There was 100% linkage of surveyed homeless people with the Repository data. In one year, 97% of participants had at least one ambulatory physician visit, with an age- and sex-adjusted rate of 14.82 per person-years (overall Manitoba rate = 4.99 per person-years); 34% were hospitalized (adjusted hospital separation rate = 491 per thousand person-years versus the Manitoba rate of 137 per thousand person-years); and 95% filled at least one prescription with 65% of drugs targeting the nervous system (the majority were psycholeptics). The degree of agreement between the disease-related data sources ranged from poor (κ = 0.27) for arthritis to moderate (κ = 0.57) for hypertension. Individuals were more likely to be classified as having one of the four selected conditions based on the administrative data than on the survey data.

Conclusion: Compared to the general population, homeless participants had high health service use and high prescription drug use. There was poor to moderate agreement between the two data sources with regard to disease detection. Researchers studying homeless persons with mental illness should consider using multiple data sources to estimate disease prevalence and health service use.

Keywords: mental health, epidemiological methods, health care services use, administrative health records, homelessness

Author references:

3. Department of Community Health Sciences, University of Manitoba, Winnipeg, Manitoba, Canada
4. Institute of Urban Studies, University of Winnipeg, Winnipeg, Manitoba, Canada
5. Manitoba Centre for Health Policy, University of Manitoba, Winnipeg, Manitoba, Canada

Correspondence: Aynslie Hinds; Email: umhinds0@cc.umanitoba.ca

Modelling costs of episodes of care for exacerbations of chronic obstructive pulmonary disease

J. P. Kuwornu, MSc (6); L. M. Lix, PhD (6, 7); J. M. Quail, PhD (6, 7); E. Wang, MSc (7); M. Osman, MA (7)

Introduction: An episode of care (EoC) is the cluster of health care services associated with an acute or chronic condition. EoCs are used to examine variations in cost and use of different treatment pathways. Predictive models of EoC utilization and costs can be used to identify the pathways that result in optimal treatment outcomes. However, a major issue associated with making valid and accurate predictions is the selection of an appropriate statistical model.

Objective: To compare different statistical models for predicting EoC costs for exacerbations of chronic obstructive pulmonary disease (COPD).

Methods: The study data included hospital separations, physician billing claims, prescription drug records and population registration files from Saskatchewan. The study cohort was made up of individuals aged 35 years and older with a COPD diagnosis in hospital or physician claims. We identified EoCs initiated by a hospitalization with a primary diagnosis of COPD from 2000/2001 to 2009/2010, and calculated total hospital, physician and drug costs for each EoC, adjusting for inflation. We compared marginal generalized estimating equation (GEE) and random effects models with a gamma or negative binomial distribution for mean EoC cost and quantile regression model for median EoC costs. Covariates included demographic, socio-economic and disease-related variables.

Results: From the study cohort (n = 41 848), we identified 20 999 EoCs for COPD exacerbations initiated by hospitalisation. Average age of those who had COPD EoCs was 71 (standard deviation [SD]: 12) years, and 53% were male. Total costs were highly skewed. The median total cost was $4,506, while the mean (SD) was $7,968 ($13,354). The median was higher for episodes (n = 2400) in which the individual died ($8,380) than not die (n = 18 599; $4,400). The random effects and GEE models failed to converge for the gamma distribution. The model with a negative binomial distribution fit the data well based on the deviance statistic. All covariates in this model were statistically significant except for sex (p = .8179) and age (p = .0610).The quantile regression model also converged; only the Charlson comorbidity score was not statistically significant (p = .5791).

Conclusion: Quantile regression and marginal models with negative binomial distribution appear to be valid approaches to addressing the small proportion of individuals with high EoC costs for COPD. The models produced different results about the significance of the covariates. The choice of models will influence the patient characteristics associated with health care costs and treatment trajectories, and could lead to different conclusions about optimal treatment pathways for COPD patients.

Keywords: longitudinal analysis, epidemiological methods, respiratory epidemiology

Author references:

6. School of Public Health, University of Saskatchewan, Saskatoon, Saskatchewan, Canada
7. Health Quality Council, Saskatoon, Saskatchewan, Saskatchewan, Canada

Correspondence: John Paul Kuwornu; Email: paul.kuwornu@usask.ca

Characteristics associated with increased pain and low functional recovery three to five years following total knee arthroplasty

J. E. Mollins, MSc (8); C. A. Jones, PhD (9, 10); M. Clark, MD (11); L. Beaupre, PhD (9, 10)

Introduction: The incidence of total knee arthroplasty (TKA) performed in Canada is steadily increasing; however, 9% to 19% of all TKA patients experience little or no improvement in physical function and pain relief postoperatively. A lack of consensus exists about the factors associated with these poor outcomes. Determining baseline characteristics and demographics associated with increased pain and negated postoperative functional status could assist in identifying patients who are less likely to benefit from this operation. If these factors are modifiable, they could be addressed before TKA to improve postoperative outcome; if not, patients could be given realistic postoperative expectations.

Objective: To identify modifiable and non-modifiable baseline patient demographics associated with poor pain and physical function scores on the Western Ontario McMaster Osteoarthritis Index (WOMAC) at 3 to 5 years following TKA.

Methods: This was a secondary analysis of prospectively collected data from the Alberta Arthroplasty Study, a large randomized clinical trial. We performed initial descriptive analyses and compared baseline scores between responders and non-responders as well as univariate linear regression for the following independent variables: age, gender, group allocation, body mass index (BMI), categorical comorbidities (≤ 2 or ≥ 3 conditions), presence of back pain, diabetes status, presence of lung disease, smoking status, baseline Medical Outcomes Study 36-item Short Form (SF-36) mental health (MH) scores, baseline WOMAC physical function scores and baseline WOMAC pain scores. This initial model building step was performed twice: once with WOMAC pain scores and once with WOMAC function scores as the dependent variable. A multivariate regression was then developed using purposeful selection techniques. Final stability of the model was assessed using forward and backward stepwise regression methods to determine agreement among significant variables; variance inflation factors were calculated to test for collinearity.

Results: A total of 388 patients consented to further evaluation 3 to 5 years after TKA. We observed significant improvements in both WOMAC pain and function scores. In the multivariate analyses, older age, presence of back pain, and overweight or obesity were indicators of both worse pain levels and inferior functional status. Better preoperative WOMAC pain and SF-36 MH scores were associated with improved postoperative pain levels. Higher WOMAC function and SF-36 MH scores at baseline were predictive of better functional outcomes post-TKA. Coefficients of determination (R^2) were 0.15 for the pain model and 0.19 for the function model.

Conclusion: Older age is associated with poorer pain outcomes several years post-TKA; however, older individuals experienced changes in pain similar to that of younger patients. Thus, older age should not be a limiting factor when considering candidates for TKA. Increased BMI was also a significant predictor of long-term pain and function scores. Interventions to manage BMI and back pain should be considered preoperatively to maximize TKA outcomes. Based on the identified risk factors, patient expectations may be revised regarding outcomes. Low R^2 values indicate limited ability of the model to predict patient outcomes 3 to 5 years following surgery. Future research may consider including more psychosocial variables in medically based models when assessing TKA outcomes.

Keywords: clinical epidemiology, longitudinal analysis, aging

Author references:

8. Faculty of Rehabilitation Medicine, University of Alberta, Edmonton, Alberta, Canada
9. Department of Physical Therapy, University of Alberta, Edmonton, Alberta, Canada
10. Alberta Innovates - Health Solutions, Edmonton, Alberta, Canada
11. Department of Surgery, University of Alberta, Edmonton, Alberta, Canada

Correspondence: Juliana Mollins; Email: mollins@ualberta.ca

Variable importance measures for non-normal data: an application to patient-reported outcomes on their health-related quality of life

T. T. Sajobi, PhD (12); B. M. Dansu, PhD (12); L. M. Lix, PhD (12)

Introduction: Health-related quality of life (HRQOL) measures are widely used in clinical trials to assess the effectiveness of new treatments across physical, psychological and social domains. Variable importance measures derived from descriptive discriminant analysis (DDA) and multivariate analysis of variance (MANOVA) procedures have been developed for evaluating the importance of domain for HRQOL data collected at a single time point. This includes standardized discriminant function coefficients, discriminant ratio coefficients, and *F*-to-remove statistics. However, these measures may result in inconsistent rank ordering of domains in HRQOL data characterized by non-normal distributions.

Objective: To develop and apply measures of relative importance derived from DDA and MANOVA procedures based on trimmed means and Winsorized covariances for evaluating domain importance in non-normal multivariate data.

Methods: DDA and MANOVA procedures that are insensitive (i.e. robust) to departures from multivariate normality assumption were developed by replacing the least squares estimates of means and covariances with trimmed means and Winsorized covariances, respectively. Variable importance measures derived from the coefficients of these robust DDA and MANOVA procedures were used to rank order variables in non-normal multivariate data. Variable importance measures based on least squares and robust estimators were illustrated using data from the Manitoba Inflammatory Bowel Disease Cohort Study, an on-going longitudinal cohort study that is investigating psychosocial predictors of health outcomes. Study participants with self-reported active (n = 265) and inactive (n = 116) disease were compared on the four domains of the disease-specific inflammatory bowel disease questionnaire (IBDQ) and the eight domains of the Medical Outcomes Study 36-item Short Form (SF-36) Questionnaire that measured the physical and mental aspects of participants' health and well-being.

Results: When measures of relative importance based on least squares estimators were used to evaluate domain importance, the IBDQ bowel symptom and the SF-36 general health domains were identified as the most important domains. In contrast, the IBDQ emotional health and the SF-36 general health domains were identified as the most important domains that discriminate between active and inactive disease groups when variable importance measures based on trimmed means and Winsorized covariances were used to evaluate domain importance. The rank ordering of the remaining domains varied across variable importance measures and estimation methods.

Conclusion: These relative importance measures can be used to choose a parsimonious subset of domains that best discriminate between groups in non-normal HRQOL data. Further research is needed to investigate the properties of these measures under a variety of data analytic conditions.

Keywords: biostatistics, population, public health

Author references:

12. School of Public Health, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

Correspondence: Tolulope Sajobi; Email: tts229@mail.usask.ca

Women's health care utilization among HIV-positive women on ART in British Columbia*

X. Wang, MPH (13); K. A. Salters, MPH (14); H. Wang, MSc (14); W. Zhang, MSc (14); N. Pick, MD (15, 16); J. S. Montaner, MD (14, 15); R. S. Hogg, PhD (13, 14); A. Kaida, PhD (13)

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Introduction: Women make up more than 50% of HIV-positive (HIV+) population globally and about 22% nationally. That there are experiences unique to women living with HIV has been well established. Compared with their HIV-negative counterparts, HIV+ women are more likely to have abnormal gynecological conditions and menopause-related problems such as osteoporosis. However, several studies have suggested that medical care specific to women was underutilized among HIV+ women despite that appropriate use of women's health care has been shown to reduce HIV-related burden of diseases.

Objective: To estimate the prevalence and covariates of women's health care utilization among HIV+ women who ever received antiretroviral therapy (ART) in British Columbia (BC).

Methods: The Longitudinal Investigations of Supportive and Ancillary Health Services (LISA) study is a study of people living with HIV who have ever received ART in various BC clinics. The cross-sectional interview data on sociodemographic factors, supportive service use and quality of life were linked to the longitudinal HIV clinical data available through the provincial Drug Treatment Program. For this analysis, inclusion was restricted to LISA participants who identified as female. The outcome measure was current utilization of women's health care. The answers were dichotomous (yes vs. no) based on responses to the LISA survey question, ''I have a physician who I see regularly for women's health care.'' Independent covariates included demographic variables (age, ethnicity, health authority, rural/urban residency, marital status), sociodemographic variables (education, employment, income, housing stability, food security), psychosocial variables (stigma, perceived neighbourhood problems or cohesion, quality of life), substance-use variables (alcohol use, illicit drug use, drug injection), sexual health variables (sexual activity, condom use, sex trade history, pregnancy intention, number of births, history of sexually transmitted infections, abnormal Pap smear in last 6 months), mental health variables (symptoms of depression), and HIV clinical variables (ART status, CD4 cell count, plasma viral load, viral load suppression, duration of immunosuppression). Bivariate analyses and multivariable logistic regression analyses were conducted to identify factors associated with women's health care utilization.

Results: Of the 231 women participants, 77% regularly accessed women's health care. Median age was 41 years, 49% reported Aboriginal ancestry, 72% had an annual income less than $15,000, 62% had stable housing and 23% were food secure. In the multivariate analysis, factors associated with women's health care utilization included not living in Vancouver Island Health Authority (odds ratio [OR] = 0.12, 95% confidence interval [CI]: 0.04–0.37), no current illicit drug use (OR = 0.42, 95% CI: 0.19–0.92), higher annual income (OR = 6.73, 95% CI: 1.85–24.54) and increased provider trust (QoL scale) (OR = 1.03, 95% CI: 1.00–1.05).

Conclusion: Despite a relatively high prevalence of women's health care utilization among HIV+ women on ART in BC, a health service gap persists along geographic and social axes. To effectively integrate women's health care into routine HIV care, programs and services need to be tailored to women's needs by addressing social and structural determinants of health.

Keywords: social epidemiology, behavioural epidemiology, women's health, health services research

Author references:

13. Faculty of Health Sciences, Simon Fraser University, Burnaby, British Columbia, Canada
14. BC Centre for Excellence in HIV/AIDS, Vancouver, British Columbia, Canada
15. Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada
16. Oak Tree Clinic, BC Women's Hospital and Health Centre, Vancouver, British Columbia, Canada

Correspondence: Xuetao Wang; Email: wangxtk@gmail.com

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