RESEARCH LETTERS

Misclassification of seat belt use

In a recent article, Robertson (suppl 2):17S commented on our study of seat belts and death in a crash.1 Robertson wrote: “What is not explained adequately by the theory [about misclassification of seat belt use] is the sudden gap in police reported use by the dead and survivors that appeared in the mid-1980s”.

Robertson’s criticism seems misplaced, as we offered no theory to explain changes in the prevalence of belt use. We reported that among front seat occupants pairs in which one died or both died, the prevalence of belt use decreased from 12% in 1975 to 4% in 1980, and then rose to 40% in 1998.2 Explaining these changes, however, was not the focus of our paper. Using matched cohort methods, we noted that the risk ratio for death, comparing belted with unbelted occupants, was 0.59 using data from 1975–83, and 0.39 using data from 1986–98.3 We examined theories that might explain why these risk ratio estimates changed over time.4 We presented evidence against the theory that seat belts have become truly more effective and against the theory that estimates changed because of changes in crash characteristics. The observed change in risk ratio estimates could be explained by either, or both, of two theories:

1. Differential misclassification. Seat belt misclassification is differential when the proportion misclassified is related to the outcome (death). Risk ratio estimates could move away from their true value and toward 0 if, over time, an increasing proportion of crash survivors were classified as belted, when they were not, or an increasing proportion of those who died were classified as unbelted, when they were: this possible mechanism is illustrated with hypothetical data in the top half of table 1. (For simplicity, the table ignores the matching used in our published analysis.)

2. Non-differential misclassification. Without regard to death or survival, some belt users could be classified as not belted, or some non-users as belted, or both. Non-differential misclassification of a binary variable tends to bias risk ratio estimates toward 1.5 If non-differential error was constant over time, more recent risk ratios might also tend to be less biased, because of the influence of changing seat belt prevalence: bottom half of table 1.

For both differential and non-differential misclassification, the size and direction of any change over time in risk ratio estimates will be related to the size and direction of the errors and changes in the prevalence of seat belt use. The observed changes in risk ratio estimates alone cannot tell us which estimates are least subject to bias.

One of us has reported that there is some degree of both differential and non-differential misclassification of belt use; but the amount of error in recent data suitable for a matched-cohort analysis was so trivial, and biases toward 1 and toward 0 so balanced, that the misclassification did not appreciably influence the risk ratio estimate.7 Robertson interpreted these results as showing only that trained crash investigators were as prone to differential misclassification as police investigators.8 Whatever the correct interpretation, we and Robertson agree that additional measures of seat belt use would be useful. We hope that information from electronic crash recorders will be added to publicly available data, such as the Crashworthiness Data System (CDS). It might be feasible for the CDS to assess some crashes with a second investigator assigned to determine belt use only by vehicle inspection, without knowledge of occupant outcomes or the police report. To minimize costs, this additional investigation could be reserved for those crashes with front seat occupant pairs among whom at least one died. This would allow a matched cohort analysis to compare risk ratio estimates using three sources of belt information: (1) police reports; (2) the usual CDS investigation; and (3) an investigator who could not be biased by knowledge of the outcome.

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References
1 Robertson LS. Bias in estimates of seat belt effectiveness. Inj Prev 2002;8:263.
5 Cummings P. Association of seat belt use with death: a comparison of estimates based on data from police and estimates based on data from trained crash investigators. Inj Prev 2002;8:338–41

Bias in estimates of seat belt effectiveness

In his recent commentary entitled “Bias in estimates of seat belt effectiveness,” Robertson criticizes our study of seat and shoulder belts in relation to crash injury risk.2 He writes: “In one of the recent studies claiming high belt effectiveness, missing data on velocity changes in crashes were imputed partly from injury severity scores, again a cause imputed from an effect and then used as a control in the study, a true scientific ‘no-no’.” Robertson’s criticism is incorrect. When multiple imputation is used to deal with missing data on a covariate, the imputation model needs to preserve relationships between that covariate and other key variables that will be used in the main analysis.3 These other key variables include both exposure and outcome. In contrast, Robertson argues that measures of crash outcome should not be used to impute values on a covariate which will later enter the main analysis as a predictor of crash outcome.

In our study, velocity change during the crash (delta-V) was a clear confounder: when known, larger delta-V was associated with higher case fatality and also with greater likelihood of being unrestrained. However, delta-V was often missing, and missingness was related both to restraint use and to crash outcome, which motivated our use of imputation.

The problem with Robertson’s argument can be illustrated by considering how imputation was done under these conditions for a subject with missing data on delta-V. The form

Table 1 Hypothetical data for a cohort study of 100000 persons who crashed, classified by seat belt use and death. Percent and arrows show amount and direction of misclassification

<table>
<thead>
<tr>
<th>Misclassification type</th>
<th>True belt use prevalence</th>
<th>Belted Died Lived</th>
<th>Case fatality</th>
<th>Risk ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>None 6%</td>
<td>Yes</td>
<td>108</td>
<td>5892</td>
<td>0.0180</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2820</td>
<td>91180</td>
<td>0.0300</td>
</tr>
<tr>
<td>Differential 6%</td>
<td>Yes</td>
<td>107</td>
<td>6439</td>
<td>0.0166</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2821</td>
<td>90633</td>
<td>0.0302</td>
</tr>
<tr>
<td>Differential 35%</td>
<td>Yes</td>
<td>558</td>
<td>41621</td>
<td>0.0132</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2022</td>
<td>55799</td>
<td>0.0350</td>
</tr>
<tr>
<td>None 6%</td>
<td>Yes</td>
<td>63</td>
<td>5937</td>
<td>0.0105</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2820</td>
<td>91180</td>
<td>0.0300</td>
</tr>
<tr>
<td>Non-differential 6%</td>
<td>Yes</td>
<td>132</td>
<td>8068</td>
<td>0.0161</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2751</td>
<td>89049</td>
<td>0.0300</td>
</tr>
<tr>
<td>Non-differential 35%</td>
<td>Yes</td>
<td>408</td>
<td>35343</td>
<td>0.0114</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>1910</td>
<td>62339</td>
<td>0.0297</td>
</tr>
</tbody>
</table>

www.injuryprevention.com
of multiple imputation that we used involved drawing several delta-V values from the distribution of known values among subjects who were similar to the one with missing data. (Technically, values were drawn randomly from a bootstrap sample of these potential data donors, but since this detail affects only the variance of imputed values and not their expected value, it can be ignored here.) By Robertson’s argument, even if the subject with missing data on delta-V was known to have died in the crash, that fact should have been ignored, and he or she should have received imputed values drawn from the distribution of delta-V among other similar fatalities and survivors combined. Because most occupants survived, this implies that most of the imputed delta-V values for fatalities would have come from survivors—who, as a group, were in crashes with lower delta-V. Imputed delta-V values for fatal cases would thus have been systematically biased downward compared with known values. Imputed delta-V values for survivors would have been biased upward, because some of them came from fatal cases. In fact, among subjects with imputed values, delta-V would no longer have behaved as a confounder at all, since the imputation model would have wiped out any association between delta-V and outcome among them.

What difference does this make in terms of the relative risk estimates for restraint use? Simulation suggests that it makes. Suppose that case fatality in 10,000 crashes is considered in relation to restraint use and delta-V (dichotomized into high or low, or for simplicity). Say that in the absence of any missing data, in high-delta-V crashes, case fatality is 100/1000 among restraint users and 200/4000 in non-users. In low-delta-V crashes, case fatality is 160/4000 in restraint users and 100/1000 in non-users. Thus the true relative risk is exactly 0.4 in each delta-V stratum. Also by construction, high delta-V is associated with higher case fatality and with lower use of restraints, so that delta-V is a confounder.

Now let us consider how different analysis approaches perform, depending on the missing data mechanism. Table 1 shows three missing data patterns:

1. Delta-V is missing completely at random (MCAR): a random 40% of values are missing among all combinations of exposure, outcome, and missingness. This pattern is generally termed missing at random (MAR).

2. Delta-V is missing more often in some exposure-outcome combinations than in others. The proportions shown are those observed in our study. However, missingness does not depend on true delta-V, conditional on exposure and outcome. This pattern is termed missing not at random (MNAR).

3. Missingness on delta-V varies not only by exposure and outcome, but also by the true value of delta-V. This pattern is termed missing not at random (MNAR).

Table 2 shows the relative risk that would be obtained in each of these situations using each of three methods for handling missing data. When the analysis is restricted to cases with complete data on delta-V, the observed relative risk is biased toward 1.0 except when delta-V is missing completely at random—a situation that did not match our data and that probably rarely occurs in practice. If imputation is carried out by ignoring crash outcome when imputing delta-V values, as Robertson advocates, the relative risk is always biased. Ironically, the observed relative risks actually exaggerate the effectiveness of restraints, because the imputation method thwarted removal of some of the confounding by delta-V. When imputation of delta-V is done conditionally on crash outcome, the relative risk is unbiased under the MCAR and MAR patterns, and it is less biased than either of the other analytic approaches under the MNAR pattern. In short, both theory and simulation results indicate that the method we used to impute delta-V was sound, in contrast to Robertson’s alternative, and we stand by it.

Table 1: Missing data patterns

<table>
<thead>
<tr>
<th>Missing data pattern</th>
<th>True delta-V</th>
<th>Proportion with missing data on delta-V</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Restrained</td>
<td>Not restrained</td>
</tr>
<tr>
<td>Missing completely at random</td>
<td>Low</td>
<td>0.40</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.40</td>
</tr>
<tr>
<td>Missing at random</td>
<td>Low</td>
<td>0.37</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.53</td>
</tr>
<tr>
<td>Missing not at random*</td>
<td>Low</td>
<td>0.42</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.32</td>
</tr>
</tbody>
</table>

*See text.

Table 2: Performance of alternative approaches to handling missing data on delta-V

<table>
<thead>
<tr>
<th>Missing data pattern</th>
<th>Delta-V</th>
<th>True RR</th>
<th>Observed relative risk for restraint use</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Restrict to cases with complete data</td>
</tr>
<tr>
<td>Missing completely at random</td>
<td>Low</td>
<td>0.40</td>
<td>0.40</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.40</td>
<td>0.40</td>
</tr>
<tr>
<td>Missing at random</td>
<td>Low</td>
<td>0.40</td>
<td>0.39</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.40</td>
<td>0.69</td>
</tr>
<tr>
<td>Missing not at random*</td>
<td>Low</td>
<td>0.40</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.40</td>
<td>0.73</td>
</tr>
</tbody>
</table>

*See text.

References

No tea until three?

Scalds are the most common cause of burn injuries in preschool children.

We performed a retrospective study at the Wessex Regional Burns Unit, Salisbury, UK, which yielded information on the pattern of scald injuries in children under the age of 5 years during the period 1995–99 inclusive. These results were compared with similar studies published from the same unit from 1960–65 and 1980–85 inclusive. Altogether 276 children were admitted with scalds, and case notes were retrieved in 215 cases. Eighty five per cent of children were under the age of 3 years with the greatest proportion being in the age range of 1–2 years; 59% of scalds occurred in boys. Forty one per cent of scalds were due to a spilt hot drink. Water in hot kettles and baths accounted for only 16% and 17%, respectively.

Figures from the Child Accident Prevention Trust report for 1999 reveal that hot liquids were the cause of 70% of thermal injuries in children, with hot drinks being the single most common cause. The way in which tea and coffee are prepared appear to influence the pattern of scalds. A number of scalds occurred when the carer’s back was turned in order to fetch milk.

Figures for scald admissions show no discernible decrease over the three study periods despite the population at risk and the cause of scald injuries being clearly identified.

We suggest that the parent held child health record would be a useful tool to educate parents about the risk of spilt hot drinks in this vulnerable population. Educating health visitors to emphasise these issues, targeting playgroups and nurseries, and using the media more effectively are other ways of addressing this problem. It is imperative that more information on preventative strategies is provided if a reduction in scalds is to be seen. K Ali, J Spinks

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The World Report on Violence and Health is a watershed publication, marking a turning point in violence prevention efforts. It offers a framework to stimulate coordinated preventive action and research across types of violence; to address social, economic, and policy factors that transcend national boundaries; and to pursue violence prevention efforts on a regional or global scale.

The report presents violence as a growing, yet preventable public health problem at a time when the problem of violence is among the priority agenda items of many nations. At the United Nations (UN) meeting on UN Collabo-ration for the Prevention of Intemperonal Violence held in November 2001, the UN recognized the global and widespread impact of interpersonal violence on health, development, human rights, human security, and peace and acknowledged that the multiple and complex causes of interpersonal violence require a multidisciplinary, multicaultural re-sponse. The Americas, the Plan of Action issued at the April 2001 Quebec Summit of the Americas of the Organization of American States identified violence prevention as a prerequisite in regional efforts to strengthen democracy, create prosperity, and realize human potential (www.summit-americas.org).

In 2000, an estimated 1.6 million people worldwide died due to violence. Yet the toll of violent death offers only a partial picture of the suffering and costs to the individual and society. For every death, many more were injured and suffered from a range of physical, sexual, reproductive, and mental health problems. Violence undermines the health and wellbeing of many millions of people; it costs nations vast sums in health care, legal and criminal justice costs, absenteeism from work, and lost productivity. Violence aggravates existing inequities.

The report violence is defined as the intentional use of physical force or power, threatened or actual, against oneself, another person, or a group or community, that either results in or has a high likelihood of resulting in, injury, death, psychological harm, maldevelopment, or deprivation. The report describes the magnitude and impact of violence throughout the world. It outlines the key risk factors for violence, summarizing the types of intervention and policy responses that have been tried and what is known about their effectiveness. The analysis highlights the crucial part that public health has to play in addressing its causes and consequences.

The typology advanced characterizes the different types of violence and the links between them. It divides violence into three broad categories according to the characteristics of those committing the violent acts: self-directed violence, interpersonal violence, and collective violence. These categories are further broken down, and a chapter is devoted to each of seven topics: child abuse and neglect by caregivers, youth violence, violence by intimate partners, sexual violence, elder abuse, suicide, and collective violence. Physical, sexual, and psychological violence and acts involving deprivation or neglect are included in the typology.

Employing an ecological model, the report views violence as the result of the complex interplay of individual, relationship, social, cultural, and environmental factors. The analysis highlights the fact that the various types of violence commonly share a number of risk factors and the links between different types of violence. These links and the interaction between individual and social, and the larger social, cultural, and economic contexts have important implications for practice and policy. They suggest that addressing risk factors may contribute to decreases in more than one form of violence.

The report findings challenge injury professionals to look beyond the fragmentation that has characterized the field of violence prevention, in which research and prevention efforts for the various types of violence have often evolved separately from one another. They argue for a more coordinated search design: cohort studies, case-control studies, and cross sectional surveys. However, the evidence for falls prevention programs is sometimes directed at slips, trips, and stumbles. The section on the cost of falls provides a conscientious summary of the economic costs of older persons’ falls, but perhaps the most challenging area to incorporate into fall aetiology is that of environmental risk factors. Here the research base is smaller than that for other types of risk factors. Research in this area has typically been less rigorous or has been troubled by methodological limitations, some of which have been overlooked in this book.

The authors correctly list the hierarchy of research design: cohort studies, case-control studies, and cross sectional surveys. However, in the study of a transient risk factor such as environmental factors, even cohort studies may sometimes be limited as such risk factors can change between baseline and any fall which may occur subsequently. Another methodological limitation which was not raised is that of insufficient statistical power—some of the case-control studies presented may well have lacked power, having fairly small numbers of cases. On balance, though, the evidence presented in this chapter suggests that environmental risk factors play a part in fall aetiology at least among certain subgroups, including those who report environmental factors which interfere with their activities of daily living, among those with a particular disability, and among more vigorous older people. It is curious then, that in the final summary chapter, the evidence for home hazards as a fall risk factor is rated as weak.
Section II (strategies for prevention) covers exercise, environmental modification, footwear, assistive devices, hospitals and residential aged care facilities, medical management, medication modification, targeted strategies, and a physiological profile approach for falls prevention. This section takes a fairly clinical or individual patient approach to falls prevention which may well be the most appropriate for the intended audience. Some discussion of the population based approach would have been particularly useful contribution, as policy makers embrace the challenge of providing for our increasingly aged population. Nonetheless, this section delivers a high quality summary of evidence based falls prevention strategies. Given the opportunity for falls prevention in general practice and family medicine, the chapters on medical management and medication modification are particularly timely.

The structure of the chapter on exercise options in my view, does not give a clear overview of the evidence base for this intervention strategy. The chapter begins with an introductory summary of the key trials for and against a particular effective nature. This is followed by a section on exercise options, falls, and fall risk factors which systematically presents the results of various studies under four subsections: resistance training, endurance training, individual physiotherapy, and general exercise. Some of the most important studies in this area are not included in these sections, presumably because these were mentioned in the introductory section. These headings are a mix of exercise type (resistance, endurance, general exercise) and method of delivery (individual physiotherapy). There would have been considerable merit in including balance improvement as one of these headings, since two or three of the studies mentioned in different parts of the chapter pointed to a specific benefit of balance improvement on falls prevention. Resistance training was included as a section and yet there is no evidence that this approach reduces falls, although strength is improved. The section on individual physiotherapy reports that research is yet to examine the effect of such one-on-one training on falls outcome. I would have thought that the study by Campbell et al mentioned in the introduction could be considered one such study. This study reported a protective effect of an intervention that consisted of a selection of exercises prescribed by a physiotherapist for each participant. This chapter would have benefited greatly from a summary table of exercise programs tested in randomised trials. By using separate columns to report the impact of these programs on falls risk factors and falls outcome, the message would have been delivered more clearly.

The greatest strengths of this most welcome book are its analytic and comprehensive nature. Whatever limitations the book may have are more than compensated for by its merits. It brings together the most salient issues for falls prevention for the first time in a specialised text. It critically appraises some of the standard clinical tests, ensures that compliance is addressed in prevention programs, and introduced the concept of using physiological profiles to direct the emphasis of individually tailored prevention strategies. This authoritative book should become a well worn and dog-eared part of every falls prevention practitioner’s resource library.

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Conference Report
Conference focuses on behavior and injury control

A significant decrease in the motor vehicle death rate for Americans—90% fewer deaths per million vehicle miles between 1925 and 1995—shows that efforts to raise safety standards and change personal behavior can be highly successful. Vehicles and roads have been improved designs, while more people wear seat belts and fewer drink and drive. Using what’s been learned from similar efforts to prevent injury at both the individual and community levels was the focus of “Behavioral Approaches to Injury Control”, a January 23 conference sponsored by the Harvardview Injury Prevention and Research Center in Seattle, Washington. Experts on behavior change from around the country presented health behavior change theories, customized injury prevention messages, and strategies for including community values and policy makers in a broad approach to injury prevention. The Centers for Disease Control and Prevention, a co-sponsor of the one day conference, actively supports behavioral science approaches to injury control, said David Sleet, PhD, of the CDC’s National Center of Injury Prevention and Control. “As much as we would like to hope otherwise”, Sleet said, “most injuries cannot be resolved by introducing a vaccine-like technology, as the technology must be proven safe, adopted by people and used properly to be effective”. Proceedings from “Behavioral Approaches to Injury Control” will be posted on the HIPRC website (www.hiprc.org) in the near future.

68th RoSPA Road Safety Congress
3–5 March 2003, Blackpool, UK. 68th RoSPA Road Safety Congress Safe driving—Reducing Risks, Crashes and Casualties. The Royal Society for the Prevention of Accident’s congress will focus on recent developments in driver training, older drivers, influencing driver and pre-driver behaviour, law and enforcement, aspects of vehicle design and technology, and designing roads to help rivers. Visit www.rospa.com/road or phone +44 (0)121 248 2000 for further details.

4th Annual CAPIC Injury Prevention Conference
11 March 2003, Cardiff, UK. Details at www.capic.org.uk in due course.

Partnerships for the future
16–18 March 2003, Perth, Western Australia. 1st Asia-Pacific Injury Prevention Conference and the Australian Injury PreventionNet-work’s 6th National Conference on Injury Prevention and Control deals with issues facing developing countries and those facing indigenous people will have a specific focus but other issues will also be included. The site for registration of interest is www.cmgwest.com.au/injury.

12th International Conference on Safe Communities

Injury Researchers’ Meeting
19–21 March 2003, Dunsborough, Western Australia. This meeting, which follows the conference in Perth described above, is organised by the Injury Research Centre (University of Western Australia). It is for experienced researchers who have attended the Perth conference and is aimed at advancing injury research practice by providing a forum for a critical examination of research methods. Conference secretary: c/o Congress West Pty Ltd, CAN 079 098 829, PO Box 1248, West Perth, WA 6872, Australia, fax +61 8 9322 1734, email conves@ congresswest.co.au.

4th European Convention in Safety Promotion and Injury Control
10–11 April 2003, Paris. At this meeting, ECOSA wants to reassert the situation in Europe and to share the experiences in safety promotion and injury control measures among all partners involved. It wants to identify the successes and failures in implementing the recommendations of ECOSA’s White Book since 2001. It will in particular also look into the consequences of implementing the new provisions under the European product safety directive, the directions for enhancing safety of services, and the impact of product liability on business. The 4th European Convention will provide the platform for communication and exchange among all stakeholders involved in the consumer safety issue and will offer new insights and innovative approaches towards safety promotion in Europe. Further information: www.ecosa.org/csi/ecosa.nsf/news.

Child and Youth Health 2003
11–14 May 2003, Vancouver, British Colum-
bia. The Congress will focus attention on health issues facing children and youth within the context of the UN Special Session on Children, which immediately precedes it. It provides the international community with the setting to define opportunities and set priorities related to new knowledge development through research and the application of this knowledge to the health issues of children over the next decade. The congress will bring together child and youth health leaders, scientists, health workers, government and non-governmental organizations, and industry to identify those opportunities that are critical to moving forward on
improving the health of all children. Youth participation will be encouraged. This congress links to and is a direct response to the challenge put forward by the United Nations to address the needs of children as a priority. The call for abstracts is open until 31 October 2002. Further information: www.venuewest.com/childhealth2003 or write to Child & Youth Health 2003, c/o Congress Secretariat, Venue West Conference Services Ltd, 645–375 Water Street, Vancouver, BC, Canada V6B 5C6, tel +1 604 681 5226, fax +1 604 681 2503, email congress@venuewest.com.

Enhanced Safety of Vehicles Conference
19–23 May 2003, Nagoya, Japan. The theme of the 2003 ESV conference is “New steps towards vehicle safety enhancements”. There are 13 themes ranging from child restraint systems through vehicle design to advanced intelligent technologies. Further information about the conference can be found at www.esv2003.com.

2nd International Safe Community Conference on Cost Calculation and Cost-effectiveness in Injury Prevention and Safety Promotion
10–13 June 2003, Falun, Dalarna, Sweden. The conference will consider the costs—direct, indirect, and intangible—which injuries and accidents cause society, authorities, and individuals and present models to estimate these costs. Cost calculation methods will be discussed in a political, ethical, cultural, and socioeconomic context. Visit www.falun.se/safe2003 for further information.

XXII Congress of the International Association for Suicide Prevention

7th World Conference on Injury Prevention and Safety Promotion
6–9 June 2004, Vienna. The major objectives of the conference are strengthening violence and injury prevention as an aspect of national public health policy and programs; producing synergy of the combined efforts of various violence and injury prevention disciplines; exchanging the most recent experiences in research and practice; and facilitating participation of experts from low income countries. Further information: www.safety2004.info.