

PostScript

LETTER

Excess in cardiovascular events on Mondays: could atherosclerotic plaques be more vulnerable after the weekend because of alcohol related cytokine dysregulation?

Barnett and Dobson performed meta-analysis of data about weekly distribution of cardiovascular events and found a slight excess in events on Mondays, more pronounced in men or associated with greater alcohol consumption.¹ However, a debate about the underlying mechanisms is wide open.

The meta-analysis provided little evidence to support the possibility that registration data collection or recording procedures play a significant part in the phenomenon. Another part of the hypothesis lies in regard to increased stress on Mondays, particularly in employed people. A lack of data on work schedules in the original studies prevents a firm conclusion regarding this issue. Today, a growing number of people do not have a classic workweek with completely free weekends, work in shifts is common, and the line between work and free periods seems to be blurred. Furthermore, anger, quarrelling, sleep disturbances and other emotional stresses are well recognised triggers of cardiovascular events² and, besides some habitual differences between extreme population subgroups, a random distribution of such triggers over the week probably exists in the general population. Last but not least, emotional stress seems to be more important for triggering cardiovascular events in women,² whereas a Monday peak is present only in men.¹

Increased alcohol consumption during weekend remains an appealing explanation. However, both alcohol consumption and deaths related to alcohol intoxication are at their lowest on Mondays, and peak on weekend days.³ So, what may cause this hypothetical, approximately one day delay in cardiovascular risk on Monday?

During alcohol hangover, an increase in concentrations of interleukin 12 (IL12) and interferon γ (INF γ) has been seen.⁴ Inflammation and its reactants are now regarded as markers of increased risk of acute coronary incident, but also as possible contributors to the initiation of that very incident. Increased serum IL12 and both serum and intracellular INF γ , and associated increase in T-helper 1 activity are found in patients with unstable coronary disease.^{5,6} Perhaps, it takes some time before impaired cytokine and T-helper 1 function induce plaque vulnerability, and coupled with other consequences of alcohol consumption, for example, increased coagulability and likelihood of ventricular arrhythmias, increase cardiovascular risk, which usually falls on Monday.

Alcohol induced cytokine dysregulation and subsequent plaque destabilisation may be the core mechanisms of delayed cardiovascular risk on Mondays, but extensive research is necessary to confirm and to further develop this notion. Although Monday excess accounts only for less than 1 event in 100, in terms of epidemy

of coronary disease it is highly significant rather than slight.

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BOOK REVIEWS

Dicing with death. Chance, risk and health

S Senn. Cambridge: Cambridge University Press, 2003, £14.99, pp 264. ISBN 0-521540232

The author introduces in an intuitive way most statistical methods currently used in the medical research literature. By means of practical examples, a good deal of historical background, and rarely a mathematical formula, the author leads the reader through both basic and more advanced concepts. The book starts from regression to the mean and Simpson's paradox, goes through Bayesian ideas, experimental design in clinical trials, relative risks and odd ratios, all the way to case-control studies, Cox proportional hazards model, meta-analysis, and evidence based medicine. It gives the reader these, and many more, essential biostatistics concepts in a natural and intuitive way.

The text is permeated with historical references, helping to put methods and famous names into context and, more importantly, to understand how concepts evolved and how they relate to each other.

The text flows with an easiness rarely seen in statistical books. More technical sections can be skipped, so that maths adverse readers can avoid formulas altogether, with minimal prejudice to the flow.

Statisticians like me will enjoy reading it, not just for the informal treatment of our subject and extensive historical background, but also to draw on the author's vast experience via his accessible examples. However, the main merit of the book lies in making technical concepts easy for non-mathematicians to understand. While introductory statistical books abound, few are able to make methods, drawbacks, and closely

related alternatives as clear as this one. By focusing on concepts illustrated by real life examples, the author manages to make readers forget about practical technicalities, which so often hamper the good understanding of statistics by researchers and the general public. At times when statistics permeate our lives, not just through medical research but also in the general media, this clarity is very welcome.

Renée X de Menezes

A short introduction to epidemiology, 2nd edn

Neil Pearce. Wellington: Centre for Public Health Research, 2005, pp 130. ISBN 0-476-01236-8. An electronic copy in pdf form can be downloaded for free from the web site <http://publichealth.massey.ac.uk>

Neil Pearce's new textbook provides an overview of major types of epidemiological study designs and their key strengths and weaknesses. At the start, Pearce emphasises that, compared with randomised controlled trials, observational studies require special attention to sources of bias, and the book's content and organisation reflect this awareness. It is logically organised to explore study design types (incidence studies, prevalence studies, ecological studies), study design issues (precision, validity, effect modification), and the analysis and interpretation of results (causal inference). This format will help students to see the forest rather than the trees and to understand the relation between different epidemiological methods.

But Pearce himself poses a sensible question in his preface: "Who needs another introductory epidemiology text?" In response, Pearce maintains that his text is worthwhile because it is shorter than other texts, because it presents a variety of methods in a structured and systematic manner, and because the field of epidemiology is changing rapidly to tackle disease phenomena on a global scale. While the book succeeds admirably on the first two qualities, it falls short on providing a substantial foundation for epidemiology students to understand the need for new methods and theories for global public health. For example, chapter 1 highlights the need for a "problem based" approach to teaching epidemiology, but, apart from a handful of sidebar examples, the didactic approach of subsequent chapters fails to put this recommendation into practice.

Nevertheless, the book's brevity and organisation make it accessible and practical both as an introductory textbook and as an introduction to epidemiology for those who want to read the epidemiology literature critically but not necessarily to practise it. Familiar mathematical equations are provided for calculating confidence intervals and χ^2 values, although more for illustration than for application (unless the reader has already taken biostatistics). The one important omission is the lack of a glossary, which would have added to the book's value as a user friendly reference tool.

Mark Parascandola