rence of this newly defined disease, it would be difficult to detect if outcomes were determined only according to the presence or absence of injury events, as opposed to this more refined measure.

Further, in the development of multivariable etiological models, one guiding principle is never to adjust for variables unless we understand how they relate to both the exposure and the outcome. If one accepts our proposition (that we are not measuring this outcome appropriately), this means that we may not be developing appropriate etiological models. We might be considering variables as risk factors contributing to the occurrence of disease when, in fact, they are actually part of the disease process. Adjustments for these variables could

lead to biased estimates of risk being attributed to the underlying risk factors. More importantly, this would lead to inappropriate recommendations for preventive intervention.

Conclusion

The purpose of this Opinion is to share thoughts about whether injury researchers are employing the correct outcome in the conduct of etiological studies. We offer these ideas not as definitive statements based on objective information, but as possibilities that require consideration and debate. If the ideas are legitimate they are certain to have some application in future investigations.

DISSENT

Risk taking disease

Stephen Jarvis

The Opinion paper by Pickett and Hoey opens a can of worms! Any dissent from these slippery worms may prove difficult, but I hope to convince readers that this particular can should be kept shut — but then reopened from the other end!

The reasoning will follow the line — what is this new disease (risk taking disease)? — how would I know I had it? — can this disease actually be located in a specific person? — how would an epidemiologist find its associated risk factors? — how does risk taking disease stand up to accepted definitions of disease? — what alternative might improve the situation?

What is risk taking disease?

Although the authors admit that they have not 'developed this definition in any detail' it is possible to tease out a couple of summary strands describing their concept. For instance, they suggest that 'a record of an injury' may be 'only a proxy measurement for the existence of this disease'. Later they suggest 'the definition [of this disease] should be based upon exposures that foster risk and a lack of risk avoidance'.

How would I know when I've got risk taking disease

First of all, I don't seem to need an injury. Rather it seems clear that the authors would characterise this disease by measures of 'exposures that foster risk' and separate measures that capture 'ability to avoid episodes

of risks'. Perhaps this might be encapsulated as 'what hazards does the environment present and how do people react to this?' To address the second half of this question one might need to go beyond behaviour variations in the face of specified hazards to include attitudes towards risk seeking, or even further to include physical and psychosocial variations in risk perception. Whatever the refinements, however, a central dimension is the measurement of exposure to risk of injury, irrespective of whether the balance of attributability is environmental or behavioral.

How would an epidemiologist study risk taking disease?

Comparing the rate of events per unit (or degree of) exposure to postulated risk factors is a basic epidemiological tool when examining aetiology. Defining disease by exposure to risk factors is, therefore, likely to be circular. If risk taking disease is defined as above, then the relevant risk factor/exposures are those that might predict this particular combination of primary exposures (that is, risk taking disease). There is a danger that rates of this disease per unit of secondary risk factor exposure will fail to predict ultimate injury outcomes. For instance, injury and secondary exposure could be independently related to its frequency (that is the disease may be a confounder). Furthermore, in experimental studies, the disease might be 'cured' by altering the physical environment in which somebody lives!

Community Child Health, Donald Court House, University of Newcastle upon Tyne, 13 Walker Terrace, Gateshead, NE8 1EB, UK

Correspondence to: Professor Jarvis.

Can this disease actually be located in an individual?

There might be utility in talking of risk taking disease as if it affects whole societies (disease of living together), or whole environments (disease of this place), or of particular 'vectors' (disease of lorries or guns), rather than a person. The location of the disease in a person is not necessary, except insofar as the ultimate manifestation (that is injury) is represented by a breakdown in individual homeostasis. Furthermore, if this kind of social or environmental 'measles' can occur anywhere on the 'skin' of a group of people, then which individual is actually affected may be rather arbitrary (driver versus passenger versus pedestrian) and may misrepresent the 'location' of the problem.

How does risk taking disease fit accepted definitions of disease?

'In medical discourse, the name of a disease refers to the sum of the abnormal phenomena displayed by a group of living organisms in association with a specified common characteristic or set of characteristics by which they differ from the norm of their species in such a way as to place them at a biological disadvantage'.¹

In discussing his definition based on Karl Popper's 'methodological nominalism', Guy Scadding makes some interpretations pertinent to risk taking disease. First, there is the question of 'biological disadvantage'. This might appear to be a crucial problem for this disease when there are no symptoms or physical signs. However, Professor Scadding makes it clear that a disease can be represented by a poor statistical prognosis rather than current depressed health status. Thus it is, *'permissible to say that someone has a symptomless disease. Most people over middle age have symptomless athero-sclerosis. This is properly regarded as a disease, since as a group those with it have a shorter life expectancy than those who do not, although many individuals will suffer no evident ill-effects from it and live to die of some other disease'*. In this sense, persistent and detectable characteristics of individuals that convey future statistical risk among a group of them can be termed a disease.

The most cogent criticism of the risk taking disease definition, however, is Professor Scadding's insistence that diseases should not be regarded as 'the cause of illness'. He continues, *'an effect, the disease, must not be confused with its own cause'*. Here, Pickett and Hoey's question, 'what if acute injury events are really manifestations of a disease process. What if that process is real and the injury only a proxy measurement for the existence of this disease?' betrays an essentialist idea of an invisible underlying disease. Furthermore, insofar as the exposures that constitute risk taking disease genuinely do cause the illness (that is, are likely to result in injury) then it is inappropriate that they constitute the disease definition however closely they are associated with it.

In conclusion, one is really hard pressed to make risk taking disease fit any notion of a satisfactory disease definition. People with it

have no current biological disadvantage, the future group risk of disadvantage is conveyed by characteristics of their environments as much as by rather nebulous aspects of their behavior, there is no disorder of function or structure (at least at a personal level), and there is a serious possibility that an underlying 'disease' is being postulated and defined by its cause rather than its effects.

What alternative might improve this situation?

Firstly, I would return to the title of Pickett and Hoey's article 'Etiological studies of traumatic injury: are we measuring the right outcome?' Here may be part of the problem insofar as the word 'outcome' is being equated with disease. It would seem to be perfectly legitimate to conduct observational and experimental epidemiological studies to determine the aetiology of particular types of behavior with respect to hazardous environments. However, a necessary condition for much progress to be made in this direction is that the final outcome measure — that is, injury — is known to be unequivocally related to such exposure. So how could we progress?

Does injury, as defined by 'the transfer of energy at rates and in amounts above or below the tolerance of human tissues' have any definitional problems? In common usage there are deficiencies — for instance the attribution of intent in 'unintentional injury' is not only difficult, but conflicts with the required separation of disease and cause. There are also boundary problems between traumatic injury and those suffered during suffocation and poisoning. However, perhaps the most important of the criticisms of this commonly accepted definition of injury is the lack of requirement for 'biological disadvantage'. Does 'illness' necessarily follow from trivial injuries?

Misclassification of a disease is almost certain if the lower band of 'clinical severity' is left undefined. Every one of us has some minor injury during a month if recording is sensitive enough. This is homeostasis at work, and the equivalent of recording sneezes and wheezes. The crucial requirement is that those 'injury' victims with the case definition chosen will demonstrate some 'biological disadvantage' that will allow them to be accurately ascertained. The most likely current problem with our aetiological models is that we have not taken this fundamental step when capturing case material for case-control or cohort based studies of injury risk factors. For case material in observational studies it cannot be adequate to use undifferentiated emergency room attenders or hospital admissions. Either of these definitions will selectively ascertain individuals biased by propensity to come from nearby localities, from younger age groups, or from socially deprived backgrounds.³ What is more, that most powerful aetiological tool, the experimental study, will be seriously blunted when ascertainment of the 'outcomes' can vary over time due to intercurrent changes in the thresholds for attendance and admission to

hospital, often driven by access/supply factors. Do we really believe that the number of empty hospital beds causes injury?

Deaths are not much better — case fatality rates from equally severe injuries vary by place and, for severe head injuries, have probably halved in the last 10 years. What we need to know is whether children with an injury severity score of 25 or greater as a result of pedestrian/motor vehicle collisions have changed in frequency over time before claiming success for our efforts at prevention (or for any other aetiological factor!).

I would propose that the 'can of worms' be opened from this other end and that the required case definition for an injury to be used

in observational or experimental studies of injury aetiology is that it should be of a severity that can be reliably ascertained and that this severity is sufficient to lead to biological disadvantage (that is, predicts death or measurable morbidity). This is what the injury severity score and a variety of other injury severity indicators allow us to do — do we really need anything else?

1 Scadding JG. Health and disease: what can medicine do for philosophy? *J Med Ethics* 1988; 14: 118–24.

2 Robertson LS. *Injury epidemiology*. New York: Oxford University Press, 1992.

3 Walsh SSM, Jarvis SN, Towner EML, Aynsley-Green A. Annual incidence of unintentional injury among 54 000 children. *Injury Prevention* 1996; 2: 16–20.

Editorial Board Member: brief biography

ROSA GOFIN

Rosa Gofin was born in Uruguay where she received her MD from the Universidad de la Republica. She immigrated to Israel where she received the MPH degree from the Hebrew University and specialized in public health in the fields of epidemiology and community medicine.

She is at present a senior lecturer in the Department of Social Medicine of Hadassah Medical organization and the

Braun School of Public Health and Community Medicine of the Hebrew University and Hadassah and is in charge of the Mother and Child Health Service of the department and involved in teaching in the masters programme of the school for Israeli students and for international students mainly from developing countries. She is the chairperson of the teaching committee of the School of Public Health.

She is a member of national committees of the Ministry of Health and the Israeli Center for Disease Control on Trauma Surveillance and Injury Prevention, and is active in NGO organizations related to the development of programmes and activities on injury prevention. She was among the promoters for the change in the law on child car restraint use that was successfully passed in 1991.

From 1992–5 Dr Gofin was head of the Israel Epidemiology Association. Her special interests are community oriented primary care programmes for injury prevention, surveillance of injuries in emergency rooms, risk factors and outcomes of childhood injuries.