Reneece 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 under-
stand how they relate to both the exposure and
the outcome. If one accepts our proposition
(that we are not measuring this outcome appro-
priately), 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 impor-
tantly, this would lead to inappropriate recom-
mandations 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 etiologic 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 appli-
cation 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 in-
stance, injury and secondary exposure could be
independently related to its frequency (that is
the disease may be a confounder). Further-
more, in experimental studies, the disease
might be ‘cured’ by altering the physical
environment in which somebody lives!
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 homoeostasis. 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 it, 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 homoeostasis 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. 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 etiology 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?


**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.