

Evolution, hypotheses, and the question of whether humans are still evolving.

Editorial published in *Bioscience Hypotheses*: 2(4) 2009: 193 - 197

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ABSTRACT.

A central requirement of this journal is that ideas should be testable. Can evolutionary ideas be tested? Ones about the past evolutionary history can be, but only if the hypothesis extends beyond describing what has happened into its present day implications. Evolutionary mechanisms can clearly be tested if they apply to fast-growing species, or provide specific tests of outcomes that would not otherwise be expected. But the future path of evolution, and especially of human evolution, is a more fraught area. There are still strong selective pressures on humans even in the affluent, urban West, deriving from pre-reproductive mortality, family size and age, and reproductive success. I skim the evidence that all three factors have substantial genetic components, and hence are likely to be the subject of future human evolution, and challenge readers to consider what testable hypotheses about human evolution these forces suggest.

TEXT

Several papers in this issue of the Journal address issues of evolution. In this year of Darwinian celebration, it is appropriate that we have some evolutionary papers. But Evolutionary hypotheses pose a unique challenge to science, and to this Journal. How can they be tested?

One of the central editorial policies of *Bioscience Hypotheses* is that ideas should, in principle, be testable (1,2). I think this is a very important criterion. Science is about explaining the world, technology about using it. Both are practical endeavours. To be useful, new scientific ideas must have practical consequences. These can be tested. They might be extraordinarily hard to test. Testing them might require samples or equipment that we do not yet possess. But untestable ideas rarely lead to new insights.

So, how does one test evolution?

In some systems, evolution can be seen to happen, and so predictions of its mechanism or path can be made and tested. This is clearly true for fast-growing species such as bacteria. The adaptation of bacterial strains to modern medicine is a classic case: selection by medical use of antibiotics drives bacterial populations to evolve resistance genes which would be at a selective disadvantage in the absence of antibiotics: both selection for and against those genes can be demonstrated experimentally (3). Charrabarty et al's (4) studies of bacterial genome structure emphasises that evolution of new strains is still occurring, and makes specific predictions about the resulting genetic structure. Burattini et al (5) make specific, quantitative predictions about the evolution of viral populations, based on models of viral populations as they compete for proliferation within a host and transmission between hosts.

The evolution of fast-growing animal strains can also be tested, and formed a central part of the argument Darwin made in *The Origin of Species*: Darwin was particularly taken with the evolution of breeds of pigeon under intense artificial selection, as well as his famous finches. But we cannot (usefully) make predictions about hundreds of generations of human evolution. We can only look back and say 'this happened'. And for almost all of historical evolution of any species, we can only look at what happened. Is this a hypothesis, or an observation? What predictions does 'this happened' make?

(This is not to decry palaeontology. Accurate description of fossils, whether physical or molecular, and using the descriptions to rigorously reconstruct the organisms as they were in

life is a painstaking and skilled area of science attracting true intellectual giants. But it is description, not prediction. It explains where we have been, but it does not predict where we are going.)

In fact, evolutionary theory can make specific, testable predictions. Seth Roberts' ideas on weight control and diet (see ref 6 and references therein) were derived from the understanding of how we evolved from Pleistocene ancestors. The 'Evo-Devo' school of biology, that seeks to link evolutionary and developmental biology, to explain how current developmental programmes evolved, and hence their characteristics today, and also makes testable predictions about development and disease. Greve et al (7) suggest that post-reproductive individuals provide support for the population through their experience, and that this should be detectable by looking at such help across kinship groups. Aarsen (8) discusses how the new mutations in an individual alter the classic ideas of relatedness, and that this explains our preference for saving our children over our brothers or sisters (which classic evolutionary theory would predict are equally valuable to the long-term survival of our genes.) Both these build on a long tradition of evolutionary mechanisms deriving from population genetics, although with different styles of argument.

These go beyond description of evolutionary history. They attempt to describe *mechanism*. This is a risky process. Authors (and by association this Journal) risk being accused of making up what Stephen J. Gould famously called 'Just-So Stories' after the origin fables of Rudyard Kipling, stories that are plausible but no more testably true than a thousand others. And this is why testability is central.

So Bioscience Hypotheses does accept evolutionary papers, even on species as long-lived as humans, with the repeated caveat that they should, to some extent, have testable implications and not be more 'this happened' descriptions.

But there is one area of human evolution which has not, yet, been addressed by a hypothesis, published here, and so I pose this to you, our readership, for your consideration. The past of human evolution continues to be explored, and its implications in our present physiology and anatomy discussed. But what of its future?

It is often said that we have reached the end of human evolution (see for example refs 9-12). The argument is that selective forces that wean out unfit individuals no longer operate in a society where the average age of death is three times the average age of parenthood, and where chasing down food or defending against the elements is not relevant to individual survival. Even those forces that do kill many people today, such as smoking-induced disease, will not affect our evolution (it is argued) as those forces change too fast. Evolution requires multiple generations to act, except for the most extreme, extinction-inducing events, whereas the impact of smoking (as measured by lung cancer incidence, which is the most direct and obvious cause of smoking-related death) is already declining just three generations after cigarette smoking was encouraged among the soldiers of World War One as a tranquilizer. Will the coming generation be adapted to ultra-fast mobile phone keypad operation, or have an inherited ability to program a VCR? It is implausible that such a clumsy interface as text messaging will be around in 100 years' time, and VCRs are already becoming obsolete. What selective forces are left (so the argument goes) change so fast that no significant adaptation can occur at the level of population genetics before the force itself has changed. In short, humans will not evolve any more unless we are, to paraphrase the famous words of a US general, sent back to the stone age.

Steve Jones also believes that the human mutation rate is falling due to a lack of older fathers (9): this seems unlikely, given the amount of variation already in the human gene pool, and the increasing age of parents in some parts of the population compared to historical times.

I believe that humans are evolving, and probably very fast. Certainly the advent of agriculture put huge selective pressures on Neolithic populations (13), and Cochran and Harpending argue that this selection may still be going on. What is in question is not the fact of evolution, but the nature of the selective forces involving, and hence what we are evolving into.

Firstly, the facts of selection. The majority of the world's population does not live in Western affluence. Much of it lives in conditions of great poverty, in which malnutrition, infectious disease and acute injury (from causes ranging from personal brutality to war) are the major causes of death. These operate at all stages of life, and there can be no doubt that any genetic trait that enhances survival of these causes of morbidity and mortality will be selected strongly. An objective view of human evolution could discount the 10% of the species that live in the wealthy affluence of Western urban civilization and consider the 90% that do not. Cochran and Harpending argue that the same conditions applied in even the most affluent of European countries until no later than 2 centuries ago: as Cochran has commented, "I have seen people explain that everyone was always prosperous in Europe. And I'm thinking – you are not Irish, are you?" (14). Although the 19th century Irish famines are the examples best known in America, all 'Western' countries have had agricultural crises when some of the population starved. Until the last century all these selective factors affected 99% or more of humanity, and the effect of that selection should be with us today.

However, readers of this journal are likely to be affluent (and old – if you can read about infant mortality then you are too old to be affected by it). They are not living in historical times. What of human evolution for them, in the 21st century? Has that stopped? I believe it has not, for reasons summarised below. In the spirit of the Journal (15), these are not completed ideas. I throw them out to our readership to develop or refute.

Even in countries with Western levels of affluence, selection is happening on at least three levels: preproductive mortality, reproductive dynamics and reproductive success.

If someone dies before they can reproduce, then, to the extent that that cause of death has any genetic component, those genes will be selected against. Pre-reproductive mortality continues to be significant even in the affluent West. Figure 1 shows the major causes of death before age 25 in the UK (The average age of first-time mothers in the UK was 27.1 years in 2000, up from 25.7 a decade before (16)). This translates to an annual mortality rate of 0.055% for males, 0.035% for females. If this sounds too low to be of evolutionary significance, the annual mortality rate from malaria in pre-industrial the Mediterranean was probably around 0.12%, and that was a selective force that is credited with causing the spread of thalassaemia, sickle cell haemoglobin and G6PDH deficiency genes throughout that region(17)¹ because the partial resistance to malaria that heterozygotes show more than compensated for the minor selective disadvantage they cause in heterozygotes and severe disadvantages in homozygotes. So there is enough selective force there, even in the presence of Westernized medicine, technology and risk-avoiding legislation, to shift the gene pool. There is clearly a large pool of genetic variation in these populations. Therefore selection and evolution is happening.

The question is – selection for what? Many of these causes of death might seem unlikely targets for evolution. The dominant causes of death are external ones, ie causes outside the body such as drowning, assault, self-harm or traumatic accident (See Figure 2). How does one select against being killed in a road traffic accident? Resistance to 150 kilometer-per-hour impact is unlikely to be a heritable feature of human physiology. But recovery from massive trauma does vary substantially between individuals, and in animal models there is a substantial genetic component to this. In addition, the need to avoid or seek exposure to the chances of traffic accident (specifically the desire to drive very fast and unsafely) is a psychological trait, relating to risk taking and a variety of subtle factors about our need for a place in a society of our peers. Death from self-harm, more common than death from assault, despite the popular media coverage of the latter in the UK, is clearly highly dependent on psychological factors. All psychological traits have strong heritability (18-20), and specifically

¹ I have not found any reliable figures on pre-industrial deaths from Malaria: disease definitions change and statistics on the causes of mortality are not collected. However the Center for Disease control estimates today that ~1 million people die each year of malaria in sub-Saharan Africa (<http://www.cdc.gov/malaria/facts.htm>), which with a combination of poor medical infrastructure and poor nutrition is probably similar to the pre-industrial Mediterranean, out of a current population of 809 million people (<http://hivinsite.ucsf.edu/global?page=cr09-00-00>), ie a death rate of 0.12%

risk-taking behaviour is found to be more influenced by genes than by environment (21). In an environment where motor cycle crashes were a significant cause of pre-reproductive death, risk-taking behaviour could be selected against.

Secondly, family size and average parental age varies substantially, and to the extent that this is affected by genetic traits, this will be selected. A population where the average couple has four children when they are between the ages of 20 and 25 will increase 10-fold (in absence of death from anything but old age) in about 75 years, whereas one where the average couple has 3 children between 30 and 40 will take 120 years to achieve this growth. The former will out-compete the latter, providing all the children survive to reproduce themselves. And obviously, a population where each couple has just two children will gradually decline as some of those children will die before reproducing. Many Western countries provide substantial financial incentives, through tax concessions, state subsidy, or direct payment to parents, to encourage people to have children to combat just this problem, although this is a means of reversing trends of declining tax revenues and increasing healthcare costs than an attempt to combat species evolution.

Choices of reproductive age and family size are complex, and heavily influenced by social expectations and personal opportunities. The affluent Western middle class may choose to wait until their university education and post-graduate professional training is complete before starting a family: such opportunities are available to few in sub-Saharan Africa. But they are also influenced by psychological factors, and as I mentioned above all have high heritability. In the affluent west, where choices are available, then choices that lead to late reproduction and few children may well be selected against, because over many generations those making such choices will contribute fewer children to the gene pool than those taking the option to reproduce early. (This is not a new observation – scientists in the 1930s feared that the world would be taken over by the fast-breeding, together with whatever genotype was correlated with that.)

Lastly, reproductive success. This does not just mean fertility, but success in having children that survive to breeding age. Here again there is substantial variation in our population. What contributes to reproductive success? Cochrane and Harpending point out that in most historical periods being at the top of the social tree lead to reproductive success. Positive attributes such as intelligence are correlated with such social success, as are undeniably genetically-linked anatomical features such as height (see ref 22 and refs therein). The drive in human evolution towards large brains is indisputable, despite the metabolic and anatomical cost they impose (23), so intelligence must have been of some advantage to our ancestors. But the nature and extent of that selection is hard to quantify. Part of the problem is that there is a dearth of other ways to analyse the problem. Statements that brains are metabolically expensive, and that large brains can only be evolved when other tissues reduce their demand on a limited metabolic energy supply, can become circular, as there is limited evidence that the brain is more metabolically expensive than (say) the gut apart from their relative size, which is what we are trying to explain. Mau et al (24) suggests that specific biochemical markers of energy density as well as overall bulk can be used to support or refute the 'expensive tissue' hypothesis with comparisons over a wide range of species.

Intelligence is clearly highly heritable: there is a wealth of evidence that differences in intelligence between individuals are largely related to the genetic differences between them (22, 25, 26). (Why this is denied so vehemently is a larger matter than we can address here, but approaches such as Aron's (27) might help to side-step the argument by provide a molecular mechanism for the evolution of intelligence, one which predicts specific patterns in NCAM polymorphism in relation to intelligence which can be tested in the laboratory independently of other tests of cognitive function.) So if intelligence does relate to reproductive success or family size or pre-reproductive death, it should be selected.

But criminality also relates to social and economic success in some circles. Several studies have suggested that top performing investment bankers have more than trace of psychopathy in their mental makeup (28), and (until the credit crunch of 2008) were considered highly successful people. Criminality in general (29) is probably heritable, although the reasons for this are controversial. (Interestingly, the ability to recover from being a victim of crime is also

genetically influenced (30), although whether this includes economic crime as well as assault has not been examined yet.) So although the psychological traits that confer social success are almost certainly heritable, and hence are likely to be selected for, it is not at all clear what those traits are.

On the negative side, sustained alcohol or other drug abuse is linked to low fertility (31-33), as well as automobile crashes and other trauma, and ample evidence suggests that risk of substance addiction (if that substance is available in the social environment the individual is exposed to) is strongly heritable. This probably includes food addiction (34), leading to obesity, itself a significant factor in premature death (ie death in the reproductive period) as well as reproductive success. Psychological stress is often cited as a factor reducing fertility, although studies do not find a pronounced effect, and the stress has to be substantial (35, 36). Unlike specifics of our environment – DDT, VCR controls, jet noise – stress at change in our environment is likely to be with us for generations.

These factors are not independent, and are not independent of the social factors that humans create around themselves. This makes dissecting specific genetic effects difficult, even effects of broad traits themselves influenced by many genes. But the overall message is clear. Human evolution cannot have stopped. Yes, antibiotics and aseptic technique and clean drinking water mean that selection for resistance to bacterial infection is now minor for those that can afford the drugs, and the water. Myopia, deafness, colour-blindness, many congenital abnormalities, these are no longer selected against as obviously as they were in the past. Physical appearance, with the exception of overall body size, is so easily disguised in the West that only the disfigured can fail to reproduce. But humans still die, and those that do not reproduce at very different rates, and many of the reasons for this have some heritability.

Many of the features I have mentioned about are related to chemicals we have introduced into the environment (endocrine disruptors, drugs) or psychological factors (psychopathy, risk aversion, stress tolerance). Does this mean that psychological evolution is now a dominant target for selection, or drug-related neurochemistry, or the ability to more efficiently metabolise synthetic chemicals (which will also be selected in the children foraging in waste tips on the fringes of developing world megacities). Are we evolving faster, more wasteful metabolisms in a world where food is unlimited? After a million years when intelligent, participative cooperation was the key to success, are we selecting for 'domesticated' submission to authority (as Cochrane and Harpending have hinted) or selecting for psychopaths?

What are your hypotheses about how we are evolving? And, most critically, how would you test them?

ACKNOWLEDGEMENTS

Many thanks to Bruce Charlton for many insightful discussions on the nature of human evolution. My thoughts here, however, are not his fault.

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FIGURE LEGENDS

FIGURE 1.

Total deaths in UK of people between ages of 0 and 25, 2001 to 2005 inclusive. A. number of deaths by cause of death. Y axis: number of deaths in 5 year period. X axis: cause of death. ICD 10 codes are listed together with a brief description. B. number of deaths in age group 0-25 years as a fraction of all deaths, by cause. Y axis: $(\text{number of deaths age 0-25})/(\text{all deaths})$ 2001 to 2005 inclusive. X axis: cause of death. Data from UK Office for National Statistics Mortality Statistics: Cause (Series DH2):<http://www.statistics.gov.uk/statbase/Product.asp?vlnk=618>

FIGURE 2

Total deaths in period 2001 to 2005 inclusive in the UK from 'external causes'. Y axis: number of deaths. X axis: causes, with ICD 10 codes. 'Other external causes' are all the residual causes in ICD codes V01-Y89 other than those shown explicitly as separate bars.

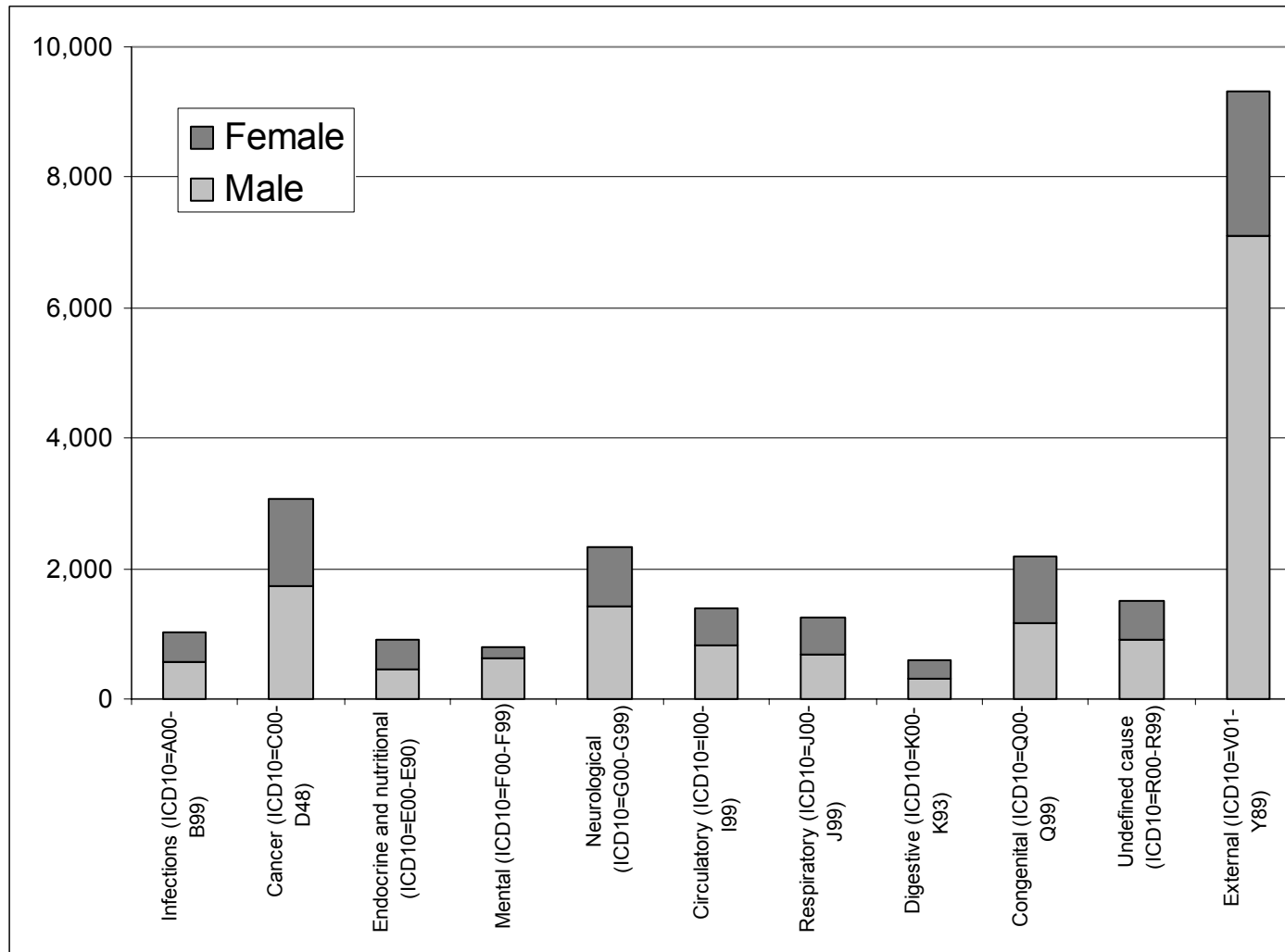


FIGURE 1A.

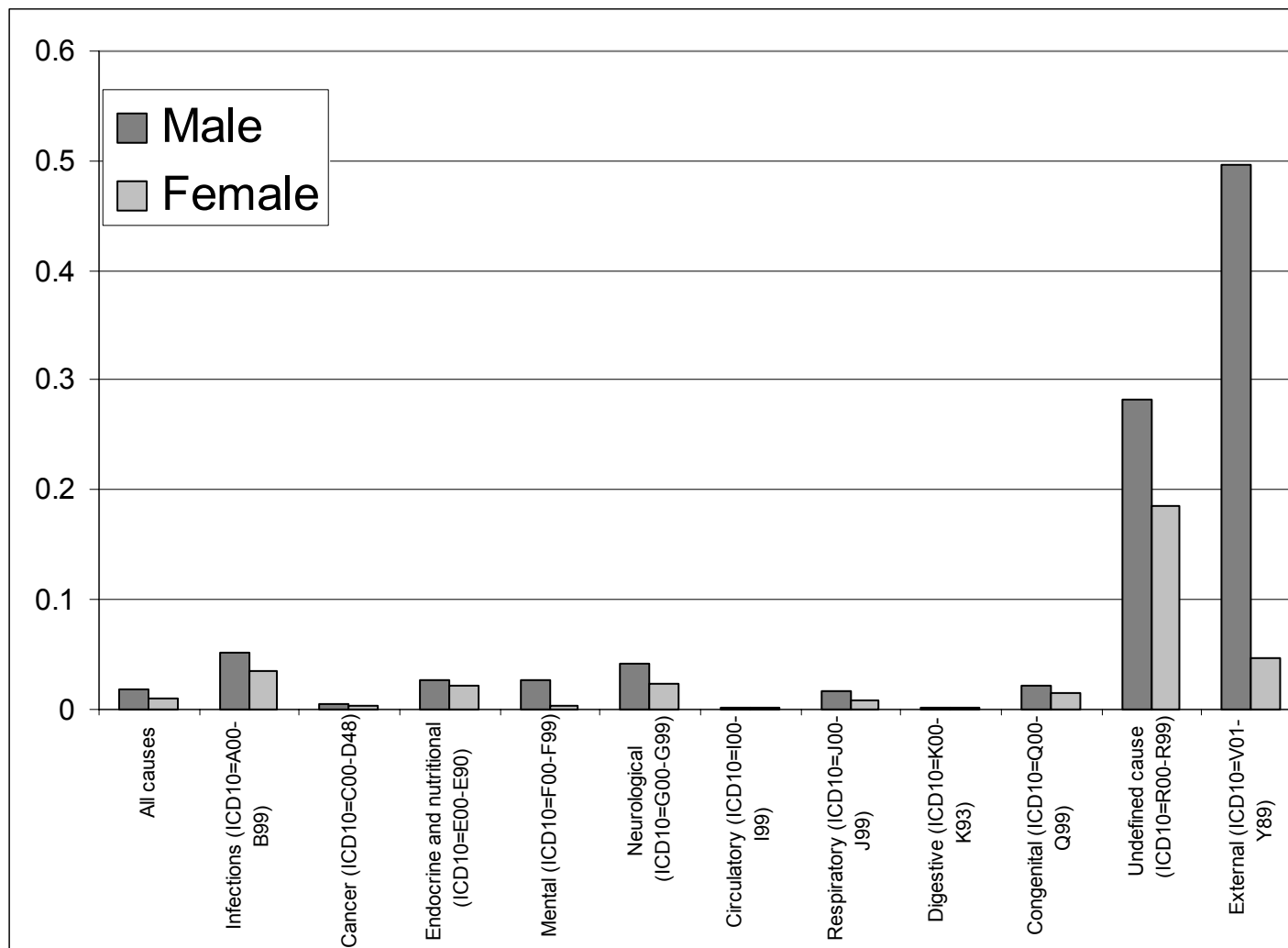


FIGURE 1B

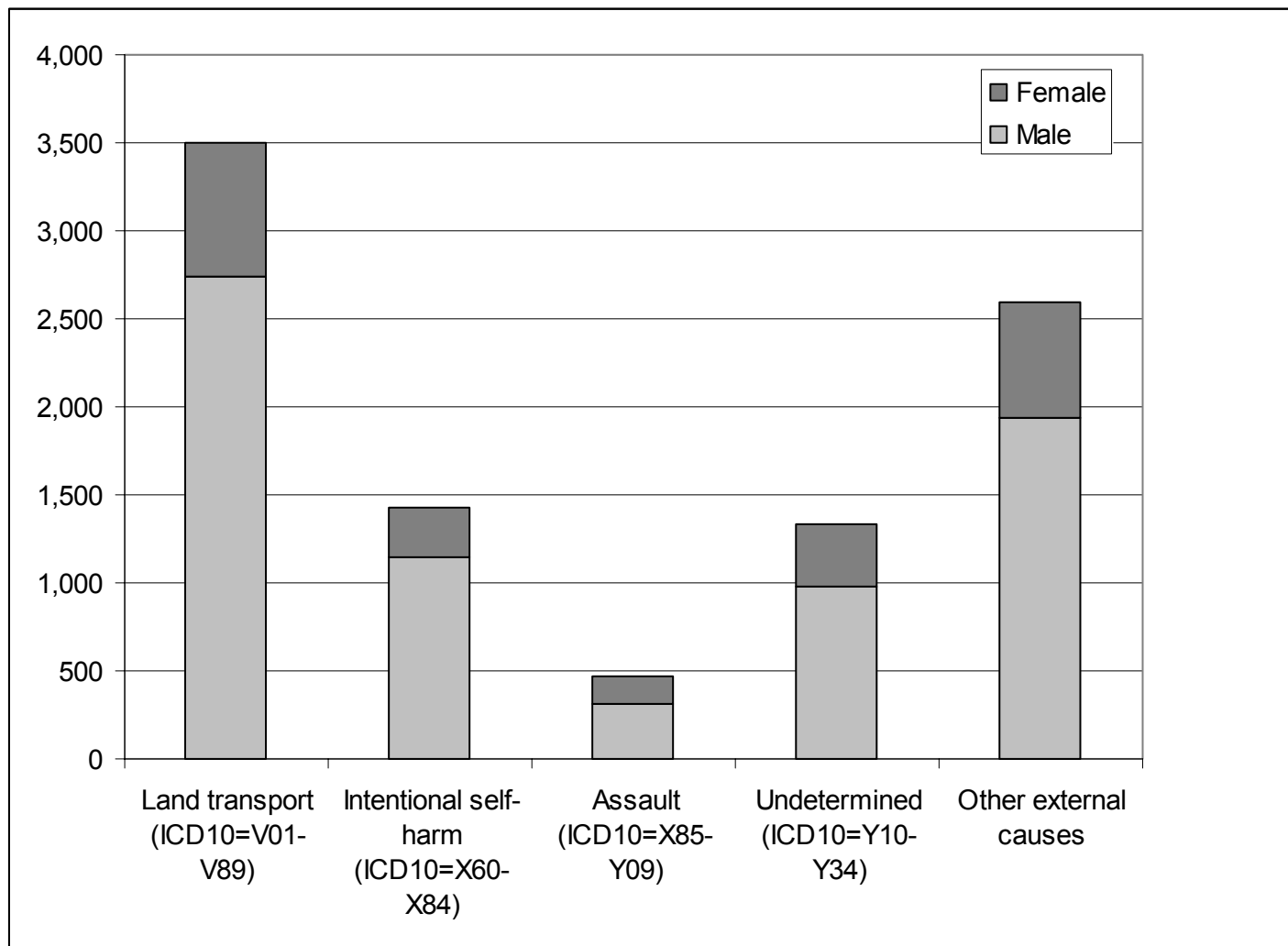


FIGURE 2