Earth, Air, Fire and Water: Moral Responsibility and the Problem of Global Drug Resistance

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DECLARATION

I, the undersigned, hereby declare that the work contained in this dissertation is my own original work and that I have not previously submitted it to any university for a degree, in its entirety or in part.

Signature.

Date.

Abstract

In this dissertation, I grapple with the problem of global drug resistance and moral responsibility which, as far as I am aware, has so far not been presented as a topic of ethical inquiry. It represents a conundrum involving three major factors: microbial adaptation and change, human social factors and environmental changes. Drug resistance is a phenomenon in which certain microorganisms, when exposed to antimicrobial agents, may acquire the beneficial trait of drug resistance which ensures a better potential for their survival. The acquired trait of drug resistance I argue renders such microorganisms *'supra-natural'*. *Supra-natural* is a term I coin for entities that have been imposed upon nature by human design; they do not follow the natural evolutionary processes of adaptation and change. Drug resistance is classified as an emerging infectious disease. Human social factors and environmental change (particularly population growth, density and consumerist practices) enhance the rise of emerging infectious diseases. Through such increasing destructive practices, stress is placed on the environment. Environmental stress facilitates the rise of new and old infectious diseases and the spread of drug resistant *supra-natural* microorganisms. Thus, our ability to treat successfully illnesses and injuries in humans, animals and plants is increasingly impaired. Morally, we are responsible for the problem of global drug resistance. Drug resistant microorganisms exist in nature and concerning this, we can do nothing. At best, we can only try to control the problem using prudential measures. The problem of global drug resistance represents both a biomedical ethical and an environmental ethical issue. Is there a way out of the human-nature debate? Through Bryan Norton's enlightened anthropocentrism, I identify the ways in which his thesis may be applied to the problem of human and environmental concerns and show its applicability in broadening the parameters of biomedical ethics education to include environmental concerns.

Key words: biomedical ethics, environmental ethics, drug resistance, *Supra-natural'* microorganisms, ethics education, enlightened-anthropocentrism.

Abstrak

In hierdie proefskrif bespreek ek die probleem van die verskynsel dat mikro-organismes op 'n globale skaal weerstand begin bied teen mediese middels (globale middel-weerstandigheid) en die morele verantwoordelikheid wat dit oproep – 'n probleem wat, na my beste wete, nog nooit aangebied is as 'n tema van eties-filosofiese ondersoek nie. Dit verteenwoordig 'n kompleks van drie belangrike oorwegings: mikrobiese aanpassings en veranderinge, menslike sosiale faktore, en omgewingsveranderinge. Middel-weerstandigheid is 'n verskynsel waarin sekere mikro-organismes, wanneer hulle blootgestel word aan antimikrobiese agente, die (vir hulself) voordelige kenmerk kan bekom van weerstandigheid teen die middel; iets wat 'n beter potensiaal vir hul eie oorlewing verseker. Hierdie bekomde kenmerk (middel-weerstandigheid) maak, volgens my argument, sulke mikro-organismes '*supra-natuurlik*'. *Supra-natuurlik* is 'n term wat ek munt vir entiteite wat aan die natuur blootgestel is as gevolg van menslike ontwerp; hulle volg nie die natuurlike evolusionêre prosesse van adaptasie en verandering nie. Middel-weerstandigheid word geklassifiseer as 'n opkomende aansteeklike siekte. Menslike sosiale faktore en omgewingsveranderinge (veral bevolkingsgroei, -digtheid and verbruikerspraktyke) vergroot die opkoms van aansteeklike siektes. Deur sodanige toenemende destruktiewe praktyke word stres geplaas op die omgewing. Omgewingstres fasiliteer die opkoms van nuwe en ou aansteeklike siektes asook die verspreiding van weerstandige *supra-natuurlike* mikro-organismes. Ons vermoë om siektes en beserings van mense suksesvol te behandel, word gevolglik toenemend ondermyn. Moreel gesproke is ons verantwoordelik vir die probleem van globale middel-weerstandigheid. Middel-weerstandige mikro-organismes bestaan in die natuur, en aan daardie feit as sodanig kan ons niks doen nie. Ons kan, ten beste, probeer om die probleem te beheer deur middel van verstandige maatreëls. Die probleem van globale middel-weerstandigheid verteenwoordig sowel 'n biomedies-etiese as 'n omgewingsetiese kwessie. Is daar 'n uitweg uit die mens-natuur debat? Ek identifiseer, met 'n beroep op Bryan Norton se swak antroposentrisme, maniere waarop sy tese toegepas sou kon word op die probleem van menslike en omgewings-oorgwegings Ek wys ook op die toepaslikheid daarvan vir die verbreding van die parameters van biomediese etiek-opvoeding ten einde omgewingsoorwegings deel van lg. te maak.

Kernbegrippe: biomediese etiek, omgewingsetiek, middel-weerstandigheid, '*Supra-natuurlike*' mikro-organismes, etiek-opvoeding, swak antroposentrisme.

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Dedication

To Joum

You are all my reasons.

....there is no higher life. This is the only life there is. Which we share with animals.....

That's the example I try to follow:

To share some of our human priviledge with the beasts.

J. M. Coetzee 1999:74

Table of Contents

Introduction

... earth, air and ocean, are the eternal witnesses of the acts we have done ...
No motion impressed by natural causes, or by human agency, is ever obliterated.

Charles Babbage 1838.

At first glance, the reader might wonder about the title of this dissertation, *Earth, Air, Fire and Water* in connection with its subtitle, *Moral Responsibility and the Problem of Global Drug Resistance.* 'Earth, Air, Fire and Water' refer to the four elements that so greatly influenced pre-Socratic thought. These elements and their processes were of great concern to the ancient 'natural' philosophers. For example, Heraclitus is remembered for his theory of consistent flow in which he asserts that 'everything flows, therefore we cannot step twice into the same river' meaning of course, that nature is in a constant state of change. He is also remembered for such seeming obscure 'dark sayings' such as 'nature loves concealment' (Solomon and Higgins 1996: 27). And indeed, nature does change and in a sense, conceals. But this dissertation is not grounded in a call back to naturalistic philosophy. Rather, it is a call to see through the vehicle of drug resistance the ways in which we are morally responsible for changes, seemingly concealed, in the earth, air, fire and water and the consequences of our actions upon nature. Broadly, it might be said that this work represents an 'enlightening' of these ancient concepts through which our moral responsibility to nature becomes better defined.

Approach to the Problem

In this work, I explore moral responsibility and the problem of global drug resistance. As far as I am aware, nothing has been written concerning this problem from the disciplines traditionally included in applied ethics: biomedical, business and environmental. My approach to the problem is not constructed in the context of individual moral intentions or prescriptions found in traditional moral theory. Rather, it is framed in the conviction that the fundamental lesson of global drug resistance is that it teaches us how, in not considering nature, we failed to regard the fact that our actions affect entities and life systems beyond ourselves. Thus, I set out to make my case that our moral responsibility for drug resistance turns on the fatal flaw of separating ourselves from the rest of nature.

When I began this dissertation, I did so in the perspective of biomedical ethics. I thought that the problem of global drug resistance resided in the misuse of antimicrobials by doctors and patients. Thus, if these problems were ethically addressed from any traditional bioethical perspective, the problem of global drug resistance could be resolved. Likewise, it could have taken a more business ethics slant, because, as I will show the role of industry in the promotion and distribution of antimicrobials plays a major part in increasing drug resistance. I felt that there was probably an environmental component involved as well. Simply watching television news of changing weather patterns and the rise of infectious diseases in new places pointed to what I considered an obvious link: changing environments opportune the rise of new diseases that we are unable to manage effectively because of drug resistance.

In deeper exploration, while it is clear that moral responsibility for global drug resistance remains in the human camp, how would I frame it? From a biomedical ethics position, in the application of antimicrobial therapy, we initiated the trait of drug resistance in microorganisms; in the misuse of antimicrobials, we contribute to their expansion. From the perspective of business ethics, the over-use of antimicrobial products in human and industrial uses adds more and more opportunities for the spread of drug resistance. From an environmental ethics viewpoint, in imposing the trait of drug resistance onto naturally evolving microorganisms, we ruptured the process of natural evolution.

While I will point to remedial measures for the containment of drug resistance, I choose not to frame it in prescriptions of moral intentions, of individual right or wrong actions. Rather, I will assert that the fundamental problem in global drug resistance, and our inferred moral responsibility for it, points to elemental flaws in the ways in which we view ourselves as part of the world. Thus, it is in this larger framework of an enlightened worldview that I make my case.

Broadly, in this work I investigate if there is some way I can connect the terms health and environment in the context of drug resistance. I do not believe from a solely biomedical or business ethics perspective that I can significantly address the problem. This is because, as based on strong anthropocentrism, humans and human interests are necessarily considered as the overriding value. In this regard, while proscriptions against harming non-humans are present, they are likely framed from the perspective of ways in which harm might affect our character, or our interests. Because of these factors, I turn to an exploration into environmental ethics, a discipline in which I had virtually no background. This required amongst other things, trying to acquire a basic understanding of ecology (which proved to be an enterprise in itself), as well as to up-date long ago learned fields of biology, zoology and microbiology. However, not until I had grappled with aspects of all these could I appreciate the extent of the danger the trait of drug resistance poses to the stability of the biotic community. With this knowledge, my first hurdle was identified. I felt I needed to show how three seemingly diverse systems (the microbial, the human and the environmental) are in fact connected.

Delving into environmental ethics led to the acceptance of what I consider as the major premise of all environmentalists: In arrogance or ignorance, we have inflicted grave damage upon nature and we are morally obliged to stop destructive practices and, as best we can, undo the harm that we have caused. This then led me to consider practical aspects concerning global drug resistance, namely how to expand worldviews to include environmental concerns in the context of teaching biomedical ethics. For example, I did not see how I could construct a teaching curriculum in biomedical ethics within environmental ethical value theories that afford intrinsic value to (or are derived) from nature. This is not to infer that it could not be done, rather that it would be difficult to accomplish in a purely practical perspective such as time available and subject matter to be addressed. Likewise, I did not see how I could address the same problems within a strongly anthropocentric position without diminishing the environmental component. This led me to accept the enlightened anthropocentric position of Bryan G. Norton (1987) in which I found I could more easily accommodate the human-nature conundrum. This is because he reconceptualises the problem pragmatically. Norton retains the intrinsic value of humans while shifting the focus to, through environmental ethics, an enlightenment of our preferences. So enlightened, we ideally will develop a broader worldview: an environmental conscience.

Drug resistance as I will show, is with us, but out of our reach. Its emergence was anticipated from the outset. We lent a deaf ear to the warning. Sins of omission are morally as wrong as those of commission. It is too late to lament. Now our moral responsibility is to prevent the emergence of new drug resistant microorganisms. This can only be done through the wedding of environmental and human concerns, through a sort of merger between the so far artificially kept distinctive disciplines of applied ethics.

I will show that drug resistance affects both human and environmental health. Biomedical ethics, as it is conceived, taught and practised – exclusively concerned about *human* health – fails to consider the untoward effects of medical practices on the health of the environment. Likewise, because of the damages inflicted by humans to nature, environmental ethics (at least the nonanthropocentric brands) runs the risk of narrowing its concerns strictly to 'nature' at the expense of human interests. In its weaknesses and strengths, enlightened anthropocentrism provides us with a bridging tool to incorporate environmental concerns within our worldviews. If we are not made aware of the human impact of our actions on nature there is little if any chance that we will ever consider changing our behaviours. Although knowledge of what we ought to do is no guarantee that we will, it is a first and necessary step.

I make no claim to be an expert in any of the fields of applied ethics. But through all its twists and turns, I hope that my exploration into the problem of global drug resistance will affirm what I set out to prove, that we have a moral responsibility to expand our worldviews personally and through education to include environmental concerns. If our views concerning nature are so enlightened, then we may consider that our actions or inactions affect not only ourselves, but also all things in and of nature, our Earth, our home.

Overview of Chapter Content

In Part one, I prove that there are three major factors contributing to increased numbers of emerging and re-emerging infectious diseases and an increased prevalence of drug resistance. These factors are microbial adaptation and change, human social factors and environmental changes. I argue that the problem of drug resistance cannot be understood unless these three factors are seen not as separate pieces, but as integral parts of a larger whole. I first conceive these factors broadly as an ecological triangle, framing the emergence of infectious disease and drug resistance by way of interactions of the causal agent, the human or animal host and the social and biological environment.

Because diseases newly resistant to available therapies are now classified as an emerging infectious disease, in Chapter 1, I first show how in our social and cultural evolution, concepts of health and disease have developed as social constructs. These constructs, I illustrate, blend with other human forces such as power and domination. The problems of emerging infectious diseases and drug resistance, I argue, should not be considered just a human malady; rather they represent an illness of the whole. In describing how predominately Western worldviews contributed to the process of separating us from nature, I also show that it negated the idea of our interrelationships and interdependency with nature.

In Chapter 2, I turn to the first leg of the ecological triangle, microbial adaptation and change. I set out to explain the world of microorganisms. I try to show that in Darwin's idea of natural selection lies the mechanisms of drug resistance. As microorganisms have a fluid matrix of reproductive potentials from which to draw, I identify some of the different types of reproductive means that they might use to acquire the trait of drug resistance. One of the difficulties in understanding drug resistance is that, while scientists know a great deal about the reproductive potentials of microorganisms, in some cases, as I point out, the precise ways in which the trait of drug resistance is transferred remains unknown.

A second and relevant point I wish to emphasise is that we seem to know that environmental stress does affect the evolutionary properties of microorganisms, we do not know exactly how. I conclude that since it appears we do not know what we may need to know concerning drug-resistant microorganisms in particular then we should proceed cautiously when, for example, remedial measures of control might be addressed. I then turn to the benefits microorganisms bring to the biotic community. The significance in microbial numbers, diversity and adaptability when compared with the human model I feel important to address. This is because in placing greater stress on the environment, more opportunities are provided for the flourishing of microorganisms: for instance, greater human populations will all in all equal greater human population density which will equal greater opportunities for microbial adaptation and change.

I indicate that antimicrobial drug resistance is a serious problem affecting the biotic community because it has permeated the biosphere. As I point out, because drug resistance is now a global problem, it requires a common global will towards its containment. This, I suggest, will only be sporadically achieved if at all.

Finally, I affirm the observation that we owe our existence to microorganisms; they sustain our lives and the life of our planet. Through the initiation of the trait of drug resistance we have imbalanced the evolutionary forces of nature. In addition, I show that our actions assist the evolution of new infectious disease as microorganisms seek new hosts in which to follow their natural biologic-evolutionary models of adaptation and change.

In Chapter 3, I seek to call attention to human social factors that contribute to the rise of infectious disease and drug resistance. The ways in which this happens are many and diverse. Some of the major contributing factors I identify are an increasing human population, the production, consumption and distribution of goods and the globalisation of what is perceived as the 'good life' which results in growing consumerist practices. Such factors I show, contribute to greater environmental stress. I address practices in industry and medicine that contribute to increasing drug resistance noting divergent social and cultural practices in the prescribing of antimicrobial therapies. From sub-therapy to over-demand, the misuse of antimicrobials in medicine is varied and largely unrestrained. I then turn to pesticide use because it runs parallel to that of drug resistance: insects also acquire resistance. As in microorganisms, the acquisition of the trait of pesticide resistance by insects and other living entities is a positive adaptive trait used in attaining the 'survival of the fittest'.

I voice a concern about the use of antimicrobials in the food-animal industry. Food industries rely on large human consumption of their products and so try to produce the maximum yield in any food-animal. Because of the often-adverse conditions under which food-animals are produced, antimicrobials are used to bolster animal weight and assist their immune systems. The role of the pharmaceutical industry in drug resistance is also addressed, particularly in marketing practices that point to profit as their major objective. Overall, in consideration of such factors, I seek to provide the reader with a glimpse of the complexity of the problem of global drug resistance.

Factors such as diverse cultural practices in prescribing, global counterfeit trade in antimicrobials, wide disparities in global distribution and availability, plus cultural perceptions of antimicrobials point to some of the difficulties in conceiving ways in which we might try to contain drug resistance. These and other considerations I consider indicate a global tendency to consider antimicrobials as simply benign commodities to be demanded, traded, negotiated, misused and abused.

Then in Chapter 4, I turn to the third leg of the triangle: environmental concerns. Our ability to treat successfully illnesses caused by microorganisms is impaired because of drug resistance. Although the most common culprits in this regard are members of the bacterial kingdom, to understand the broader picture, I use examples of viral infections to illustrate the human-nature relationship. The principle of drug resistance applies equally; viruses also may build up resistance to antiviral therapies. I then identify the circular way in which we acquire new infectious diseases showing how, for example, global warming and other environmentally destructive activities contribute to changing weather patterns that in turn influence animal, plant, insect and microbial populations. The normal reproductive patterns of many animals change with shifting weather patters such as the El Niño phenomenon, which is largely attributed to global warming. This results in increased numbers of a given species. Concurrently, because of our need for new habitats due to population expansion, we encroach upon the habitats of other species. These species carry their own diseases. But we create fresh opportunities for microorganisms to acquire us as new hosts in cross-species microbial disease transfer.

Delving into the world of an 'old' disease, *V. cholerae,* I demonstrate how environmental instability, poverty (including lack of adequate sanitation facilities), and drug resistance are all linked. But the problems in measuring disease in relation to exposure rates are conundrums, and I try to show that the tendency in traditional medical practice is to focus on a causative agent rather than inclusion of ecology-related implications.

I then proceed to show examples of how the degradation of some environmental components, soil and air, contribute to the rise in new diseases and the spread of drug resistance. The example of soil illustrates (amongst other things) that there is a connectedness between soil degradation and the rise of epidemics. An interesting spin off - new drug testing by pharmaceutical companies - links environmental ethical concerns with an area seemingly remote as research ethics. Global warming is another example of this connectedness. This phenomenon, as I show, results in previously unheard of new and old diseases appearing in different habitats and flourishing in newly found hosts.

The finding of drug-resistant microorganisms in species that have had, as far as is known, no contact with antimicrobials is important to discuss because the routes and acquisition of drug-resistant microorganisms in the environment remain a concern. That supposedly 'wild' animals have acquired drug-resistant microorganisms supports my observation that nothing in the world remains untouched by human influence. In my discussion about woodmice and badgers that have somehow acquired drug-resistant microorganisms, I wish to link the human impulse to eradicate things that we perceive as possibly dangerous to ourselves with thoughts about biological diversity. I contrast Professor G. Bell's (2001: 2413-2418) hypothesis, 'the neutral theory of biodiversity', with that of a laboratory experiment which concludes that a heterogeneous environment supports a flourishing of diverse life forms, as opposed to a homogenous one in which one life form eliminates all others. For animals that carry drug-resistant microorganisms, eradication of whatever species is not the answer. But I point out that a major difficulty exists because we do not know to what extent drug-resistant microorganisms exist in nature, only that they do. Overall, I try to show that links between the environment, environmental stress, emerging infectious disease and drug resistance is evidenced in many ways and that it is perilous to believe otherwise.

Having made my case for the three factors contributing to global drug resistance namely, microbial adaptation and change, human social factors and environmental changes I then dismantle the idea of a well-formed triangle. While retaining the major connections, I suggest that the idea of a triangle should be expanded conceptually to include their complex relationships of interconnectedness and interdependency.

Proceeding to Part 2, I turn to an ethical analysis of the problem of global drug resistance.

Chapter 5 begins the inquiry. I start by identifying that within environmental ethics, there have been gradual shifts in conceiving human responsibility towards nature. I choose to focus on the human-nature problem in general terms, first asking just what is involved in the concept of 'nature'. My idea in this regard is that if a definition could be reached, then we might find a way to separate 'natural' entities existing in nature from 'unnatural' entities. I wish to find a way in which drug-resistant microorganisms can be set apart from naturally occurring life forms. This is because, as I will later argue, drug-resistant microorganisms pose a threat to the biotic community.

I proceed to argue that what we call 'nature' is not 'natural'. By this, I mean that, because we have disastrously encroached upon and affected nature, even what we term 'wild' nature is no longer wild. A problem I raise is that we fail to associate our perceptions or awareness of changes in nature beyond a simple observation. This, I suggest, is compounded because we are not informed of, for example, the vast amount of development within the sciences that also influences nature. All in all, then I consider it better to view all nature as unnatural. I demonstrate how in early crop production we created new plants but at least we stayed within the boundaries of species. Such agricultural 'tinkering', I argue, did not present a harm to us or to nature because an unwanted or dangerous crop could be eradicated. But drug-resistant microorganisms, I suggest, exceed this consideration.

I then devise the term *'supra-natural'* to refer to any species that has been by human design imposed upon nature. 'Supra' means that entities are beyond or more than natural; they are imposed upon nature. In this formulation, 'natural' refers to "entities existing in or caused by nature" (Thompson: 1995: 907). As an example, species that have evolved over time in evolutionary processes may be considered 'natural' species. Applied to drug resistance then, antimicrobial drugs initiated the trait or property of drug resistance. The acquisition of this trait or property has led to the development of species (sub-species, strains or isolates) of new microorganisms we now refer to as 'drug-resistant'. Such *supra-natural* entities do not follow the natural order of evolution and change over time.

As an example of this, I use the genus and species of *Staphylococcus aureus*. *Staphylococcus aureus* in this sense are naturally occurring microorganisms. On the other hand, methicillin-resistant *Staphylococcus aureus* microorganisms are *supra-natural* (imposed upon nature); they would not have occurred in nature if we had not implemented antimicrobial therapy. In this way, in the context of nature, I make the distinction by which we can separate some living entities from others.

I then discuss what I consider general trends in our perceptions of technology: things are good if our immediate gratifications are satisfied and we tend not to look beyond our initial satisfactions. Using oil-eating microbes and drug-resistant microorganisms, I show that both may change the fabric of nature but they are perceived differently.

The phenomenon of drug resistance was known from the time of the inception of antimicrobial therapy. Continuing onwards, I ask the question if it should make any moral difference if a life form is 'created' by purpose or by accident. From the perspectives of Kantianism and consequentialism, I argue that for the former, the conclusion that there was *unequivocal* good-will behind the act of antimicrobial therapy is not evident.[1] Concerning the latter, I suggest that the overall consequences are yet to be determined. However, so far the leaning is towards untoward consequences. But my conclusion is that from either purpose or intent *that* such entities exist or will come into existence remains a fact. We bear the moral responsibility for their existence just as we will bear the moral responsibility for the admission into nature of any other *supra-natural* entities. The imposition upon nature of *supra-natural* entities, I argue, provides good reason to introspect on our human-nature relationship.

Then I turn to address the topic of ecological unity, which focuses on relationships between and within the earth's closed system. I point to some objectives within environmental ethics, the first being the ways in which we might extend moral considerability to nature and nonhuman others. As anthropocentrism confines intrinsic value to only members of the human species, to design a nonanthropocentric moral theory that includes nature and her entities is fraught with difficulties. A few examples of these are: Should all species be considered of equal value? Should there instead be a hierarchy of value? Is it possible for humans to be the loci of value and have nature as the source of value? Moreover, on what basis might entities be included in such theoretical approaches?

I argue that I have a solution to the latter question. Drug-resistant microorganisms differ from naturally evolving life forms because they were / are superimposed upon nature. Because they are *supra-natural*, they may be excluded from the rest of nature. The trait or property of drug resistance serves as a sufficient property or trait on which to separate drug-resistant microorganisms from other life forms.

As I discussed earlier, drug resistance threatens to return human healthcare 'back to the pre-antibiotic age' (Brundtland 2000). So it might be asked if my concern is only confined to humans. I argue that it is not. I then evoke Aldo Leopold's ([1949] 1966: 32) maxim:

> *A thing is right when it tends to preserve the stability, integrity and beauty of the biotic community. It is wrong when it tends otherwise.*

I proceed to identify the ways in which drug resistance threatens the biotic community. I argue that the trait of drug resistance is an advantageous mutable trait. In other words, if other microorganisms acquire this trait they will have a better chance of survival. Because of the reproductive potentials of microorganisms, they could acquire this trait in a variety of ways (such as I show in Chapter 2). This is bounded by only their genes and subject to environmental stress. While I offer a hypothetical example of a world in which all microorganisms acquire this trait, in doing so I ask the reader to consider that it might not be entirely ungrounded. This is because, as I argue, drug-resistant microorganisms *do exist* in nature, the survival trait of drug resistance *is mutable,* things in nature *are united* ecologically and we *continue* to devalue nature.

Finally, I conclude that this combination of factors gives good reason to consider that the property of drug resistance and increasing drug resistance to antimicrobial therapy threatens the 'stability, integrity and beauty of the biotic community'.

In the last section of Chapter 5, I turn to identify some lessons we may learn from drug resistance. These include such considerations as our tendency to think that all technological applications result in only benign consequences. In overviewing insights gained from the exploration into drug resistance, I point out grave problems in assigning moral responsibility to particular individuals and particular places. This is because, as I try to show, there are globally gross disparities in knowledge, economics, characters, cultural perceptions of diseases, the role of antimicrobials and so forth. Moreover, as I identify in a report from the Fogarty Center (Levy *et al.* 1987), global movements aimed at understanding the scope of drug resistance are often hamstrung by the interests of big business. Nonetheless, I continue to propose some remedial measures for the containment of drug resistance. I conclude that while these prudential measures are of vital importance, they turn on a higher moral level: our moral responsibility is to re-think our worldviews concerning nature as only a commodity and to see our interconnectedness with her.

In Chapter 6, I continue supporting the idea of system connectedness and look to some positions in environmental ethics that would inform the human-nature debate. There are many and varied approaches in environmental ethics. My choices for inclusion are based on some of the ways intrinsic value to nature are formulated as well as particular references found within them that, in one way or another, include microorganisms.

Concerning the arguments of a biocentric egalitarian, Paul Taylor, I consider that my argument distinguishing *supra-natural* from natural life forms might provide sufficient reason to challenge the inclusion of all living entities as equal in worth or value. Although, as I point out, Taylor insists that his theory can be applied in practice, I find this hard to realise. While I believe I understand his point that we should not do grievous harm to living entities, it is difficult in the case of pathogenic microorganisms to identify the boundaries of 'harm'. I show that while Taylor argues that (within limits) we can protect ourselves because we are moral agents, we are likewise prohibited from destroying species. This is to avoid species-partiality. A way around this, I suggest, is to argue that in containing drug resistance we are acting on the behalf of other species as well, thus actions move beyond human-only considerations. However, I point to an irony because factually, we have no means by which we can protect ourselves or other species; drug-resistant microorganisms are existent and there is nothing we can do about that. Our only options are prudential measures to limit further dissemination.

As I point out, a major hurdle remains. Taylor accords 'life' as the fundamental value grounding the equality of living entities. Drug-resistant microorganisms are alive. So then I am bound to consider ways in which this hurdle might be overcome. I find one possible solution. Throughout his text he refers to things 'natural': natural systems, natural creatures, natural states and so forth. If my assumption is correct, he refers to natural value existing in the products and processes of natural evolution. Again, I argue that delineating *supra-natural* life from natural life serves to delineate certain species from other naturally occurring ones. So the fundamental value of life is retained but it is restricted to only natural / naturally evolving life forms. In an attempt to answer Taylor's question concerning conflicts rising from 'respect for persons' in the domain of human ethics and 'respect for nature' in the domain of environmental ethics, I argue that my thesis still holds. In acting to control drug resistance we act on behalf of the biotic community. Moreover, I argue that in delineating between species natural and species *supra-natural*, a way is provided by which we can act without being species-biased. However, a weak point in my argument is that by giving the fundamental value of life to only life forms that naturally occur, I would exempt, for example, human and nonhuman clones.

I then turn to overview Callicott's position principally in terms of his support for Aldo Leopold's *Land Ethic*. From a preservationist perspective, while I support the goal of preserving what we can of 'wild' nature, it is because life-systems connect that I do not consider any of nature to be 'wild' anymore - a point raised earlier. I then present an argument from Passmore and Callicott's rebuttal to it. This involves Passmore's objections to the 'community' concept expounded by many environmentalists. In this context, he argues that bacteria do not recognise mutual obligations or recognise mutual interests. I focus on Callicott's (1989: 71) rebuttal in which he made the claim:

> *... all living things are united ecologically and all share a common interest in life itself, the desire to live an to be let alone.*

I then proceed to provide support for his claim of ecological unity, but object to his use of 'desire' with reference to non-human entities in phrases like: '... desire to live and to be let alone'. For me, it is a difficult concept to grasp that microorganisms have 'desires' at least if perceived as a cognitive function. Rather, I suggest they may have affinities (towards something) conceived rather in a biological sense.

I consider that Passmore's argument asserting that only humans are capable of generating obligations is an important point to overview. Certainly anyone who recognises that we have acted and do act environmentally wrongly will admit that attitudes towards nature must change. But how do we frame considerations of nature in an anthropocentric perspective? As I see it, unless we find a way to value nature in a non-instrumental way, she will always remain subject to our over-riding interests.

In concluding this chapter, I turn to a discussion principally surrounding one of Holmes Rolston III's topics: *Good kinds, bad kinds and good-of-their-kinds* (Rolston: 1988: 101-104) presented in his 1988 publication *Environmental Ethics: Duties to and Values in The Natural World*. First, concerning the rise of emerging infectious diseases, I will show that a contemporary exploration into the world of the *Chlamydia* species reveals remarkable increase in our knowledge concerning its potential for adaptation and change since the publication of his work. So enlightened, I will ask if in light of new information this changes his various claims concerning the bighorn sheep / *Chlamydia* problem(*ibid*: 53, 66, 102-103, 182-184). Importantly, because of their reproductive diversity and adaptation, as I will show informing Rolston's example of *Chlamydia,* there are incredible ways in which they adapt naturally to whatever natural systems offer for their continued existence.

I assert, contrary to Rolston's argument (*ibid*: 183) concerning 'interfering' with nature, that such 'interferences' may be the right thing to do and they should be evaluated on a case-by-case basis. Finally, in the context of drug resistance I suggest that the admission of *supra-natural* entities as a species imposed upon nature may alters the composition of the systems natural value. Drug resistance, I will assert, exists as a human-imposed process that will catastrophically affect natural systems. Rolston, as I will show, argues that value is generated in the system of evolutionary natural processes. In other words, the system generates its own value' (*ibid*: 186-189). In this regard, I suggest since the system is altered because of human design (the admission of non-naturally evolving life forms that affect the system); its value may be retained however, it is altered.

In reviewing some of these perspectives, I try to show that the problem of global drug resistance cannot be easily sorted-out. Each of the thinkers I overview point us in some direction and meet at least the philosophical requirement to ask questions and seek answers.

In the final chapter, I turn to the 'enlightened' anthropocentric argument in environmental ethics presented by Bryan Norton (1987). Norton's approach to environmental ethics, emphases the primacy of considered or 'enlightened' preferences over felt preferences. It retains humans as value loci. Thus, while remaining anthropocentric, it is presented in a weakened, or better put 'enlightened' or 'prudential' form (Brennan and Lo 2000). I will discuss why global drug resistance represents what Norton (1991: 207-214) terms a third generation environmental problem, one with a strong medical twist. Norton, in calling for a shift in our worldviews concerning nature, supports my contention that, amongst other actions towards environmental protection and preservation[2], environmental education should form a part of all learning situations. The containment of drug resistance is the only option available by which we can try to control its further impact upon the biotic community.

Then I assert that the traditional parameters of biomedical ethics are insufficient to accommodate environmental concerns. However, Norton's enlightened anthropocentric approach, I believe, provides the foundation upon which environmental concerns can be accommodated in the context of teaching biomedical ethics.

A weak point in Norton's argument, I will suggest, is found in that fact that he does not acknowledge the power of some of our felt preferences, one of which is our deep desire or need to become well when we are ill. The intense emotion involved in this, be it rational or irrational, may lead us to the consumption of antimicrobials be they mal-, ill-, sub- or over-prescribed. If we cannot afford a full regimen, we will consume whatever we can afford. If we consume counterfeit or sub-dosed antimicrobials, it is our misfortune for we are only looking for a way of becoming well. As the psychological desire of wellness is a primeval 'felt preference', it will be difficult to be informed by any enlightened perspective simply because of its profound emotional grounding.

Norton's pragmatic approach to environmental issues identifies a critical link between the way we view the world and our behavior, one so intense that how we view the world, or our worldview, will largely determine our actions. Thus, the fundamental justification for changing our worldview is that making such a change is the only realistic way to alter sufficiently our harmful environmental behaviors such as the imprudent use of antimicrobial therapies which impact adversely on the biotic community.

Introduction to Part 1: Earth, Air, Fire and Water

There is growing concern about the increasing rise of infectious diseases and our inability to treat them successfully because of growing drug resistance. While many sources[3] allude to connections between human health and that of the environment, others[4] simply pass over any connections and focus on only the medical aspects inherent in a particular infectious disease. Some thinkers, particularly biologists, do look beyond the discipline of medicine and explicitly point to the connection between human health and entities in nature. For example, Murphy (2000: 2) states: 'Nearly all emergent disease incidences of the past 10 years have involved zoonotic agents'. Therefore, there is a slight movement extended beyond traditional conceptions of causes or factors implicated in the rise of infectious diseases and drug resistance. Yet the traditional mode of conceptualising disease and drug resistance remains largely confined to a particular slant; the medical is set aside from the environmental. Another way of putting it might be to say that humans are separated from the rest of nature.

In part one of this dissertation, I set out to prove that there are three major factors contributing to increased numbers of emerging and re-emerging infectious diseases and an increased prevalence of drug resistance.[5] These factors are:

1. Microbial adaptation and change
2. Human social factors
3. Environmental changes

My primary aim in the first part of this work is to show the interconnectedness of these three factors that are largely, but not entirely, based on science. I will try to show that the problem of drug resistance (an emerging infectious disease) cannot be understood unless these three factors are seen not as separate pieces, but as integral parts of a larger system. The success (or failure) of this work lies in the recognition of this interconnectedness.

These three factors may be broadly conceived as an ecological triangle, framing the emergence of infectious disease and drug resistance by way of interactions of the causal agent, the human or animal[6] host and the social and biological environment. Agents involved in infectious diseases and drug resistance are diverse. They include viruses, fungi, protozoa, helminths, prions[7] and bacteria. Medically, drug resistance is of most obvious importance in its effect on our ability to treat successfully infectious diseases caused by bacteria. In addition, it also influences complications that arise in disease processes caused by other vectors, in which antimicrobials are also generally the treatments of choice. However, drug resistance spins out to domains beyond traditional medical practice; it affects, for instance, water purification, the farm-animal, pesticide, fungicide and related industries as well as new technology such as genetic engineering. And importantly, all of these affect the environment.

Because infectious disease and drug resistance are entwined, I will look at the two together in the first Chapter of Part 1. This will set the stage for further chapters in which I will enlarge upon aspects of the ecological triangle, namely microbial adaptation and change, human social factors, and environmental changes.

Chapter 1: Infectious Disease and Drug Resistance

One can properly think of most human lives as caught in a precarious equilibrium between the microparasitism of disease organisms and the macroparasitism of large-bodied predators, chief among which have been other human beings.

William McNeil 1996: 24.

Introduction

Weinstein (2000: 67) claims that drug resistance is human-made, a human 'social disease' as he puts it. In other words, he implies that drug resistance is due to some malady, an illness or disorder in human society. I accept this claim as true, and I will identify good reasons to justify and defend it.

It is important from the onset to identify the connections between the terms 'drug resistance', 'infectious', and 'disease'. Drug resistance refers to the ability of microorganisms to acquire resistance to the majority of (and possibly all) available antimicrobial agents – in other words, 'a pathogenic microorganism ceases to be killed or inhibited by a particular drug or drugs' (UKST 1997 – 1998). 'Infectious' is defined by one medical dictionary as 'Denoting a disease due to the action of a microorganism,' while 'disease' is defined as 'An interruption, cessation or disorder of bodily functions, systems or organs' (Dirckx 1997: 440, 242).

These terms are important to the topic of drug resistance because diseases newly resistant to currently available drug therapy have recently been classified under

'Emerging Infectious Diseases' (CDC 2001: USDD 2002), powerfully illustrating their relationship.

Both disease and drug resistance are included in the practice of medicine, and the practice of medicine is informed by societal concepts of health and disease. Disease as a concept, as I will show, is a fluid notion changing over time and place. This chapter will include an excursion into how infectious diseases emerged, the many meanings of disease, how 'disease' changes conceptually, how power influences concepts of disease, the ways in which drug resistance now fits into this context, and the extent to which drug resistance has emerged as a global societal problem.

What is an 'Infectious Disease'?

Perhaps in an attempt to sidestep the definition of disease, the World Health Organisation (WHO 1996 a) instead chose to define 'health'. 'Health,' we are informed, 'is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity,' a definition Hudson (1993: 45) considers a 'semantic and logical quagmire'. This is an interesting thought, because if its antonym is applied the majority of the world's human population are 'diseased,' for the qualification of 'complete' is both elusive and subjective. But I will set aside that argument, rather pointing out that the expanded WHO definition is significant in that it serves to broaden the concept of health to include societal factors.

Admittedly, the inclusion of societal factors is important. However, I wish to go a step further and broaden the definition to include the biotic community.[8] This is because, in keeping with the ecological triangle, the environment plays a critical role in our understanding of the nature of infectious disease and drug resistance. Rephrasing the WHO's definition of health and referring back to the dictionary definitions of 'infectious' and 'disease' noted earlier, a formulation could read:

> *Disease is (1) a state of disorder in any member of the biotic community's integrity, including their physical, mental, or social well-being; (2) any injury, ailment, deformity, or adverse condition, in any living organism, system, or sphere, including an interruption, cessation, or disruption of their optimal functioning attributable to microbial action.*

With this broad definition in hand, a useful starting point in understanding the relationship between humans and infectious disease is to look back to our natural evolution. This will show how we in our humanness set in motion factors that induced the spread of infectious diseases and, combining with other factors, would later result in drug resistance. In addition, it will become clear that drug resistance is inextricably linked to socially and historically constructed social practices[9], including the practice of medicine. The importance of this lies in showing how through certain repetitive actions we, in our humanness, contributed to the rise of diseases, particularly infectious diseases.

Early Humans and Infectious Disease

Authors such as Ewald (1994) and Cockburn (1967) identify three characteristic eras that in historical sequence show distinctive disease patterns. They are the pre-agricultural, agricultural and modern industrial eras. I will examine these distinctions and show that within them are recurrent themes that have not changed significantly over time. Importantly, in spite of time and space, it is in the perpetuation of many of the practices of our ancestors that the moral responsibility for drug resistance finds relevance today.

Concerning our ancient ancestors and infectious disease, while archaeology and palaeopathology[10], a branch of archaeology developed late in the 19th century, can help us to some extent in the re-creation of ancient models of society, they can only suggest that prehistoric and early human models of behaviour have not altered dramatically from those times until now. Concerning diseases *per se*, palaeopathology in particular informs us with some certainty about some and conjecturally about other types of ancient diseases, but it is impossible to know everything. This is because the genetic features of the causative microorganisms (like pathogenic macro organisms) are no longer present.[11]

Humans are of course comparative newcomers in evolutionary terms. Around two million years ago, humans evolved to the point at which larynxes and brains made speech possible. Our instincts and propensity for grouping, for being 'social creatures' as Aristotle observed, is genetically endowed. With these instincts came both advantages and disadvantages. In the early Palaeolithic era (about 30 000 to 35 000 years ago), numerous groups of humans spread globally and the world human population was about one million (Star 1973). By this time, humans had progressed to a significant extent, both biologically and culturally.[12] The biological divisions of 'childhood' and 'adolescence' were established, a few women and men reached 'old age,' and the female menopause, the first in mammalian history, was established (Diamond 1996: 130).

For nearly four million years, constantly evolving humans roamed the continents in small nomadic tribes. Forming a cooperative tribe was essential and from early bonds, societies and cultures arose changing over millions of years of evolution (Plotkin 1986). The hominoid was not 'intellectual' at that time. The adaptation of group behavioural strategies such as defection (selfish individualism), altruism[13], and 'tit for tat' strategies (Axelrod and Hamilton 1981: 1390-1396) enabled our early ancestors to survive. From these loose bonds developed tribal formations, an instinctive drawing together resulting from genetic modification and natural selection.

Archaeologists such as Mithen (1996) argue that the archaic human mind appears to be one in which social behaviours were relatively isolated from interaction with the natural world, including other human tribes and material culture. If so, this inclination plus low human population density meant that early humans rarely encountered other tribes (by choice or not) and were therefore less likely to contract infectious diseases. Nomadic travel also had the advantage that human excrement was spread over a large area reducing possible exposure to contaminated faeces or urine. Early hunter-gatherer societies certainly did not have much of a problem with waste disposal; nomadic lifestyles and low population density permitted wastes and even cast-off possessions to be disregarded with impunity. Material possessions were few, and soon recycled in the rapid biodegradation of primary organic debris.

Adaptive patterns of behaviour become increasingly important and complex as one ascends the scale of living things. The archaeological picture changed dramatically around 40 000 to 50 000 years ago with the appearance of behaviourally modern humans. The suggested catalyst according to Barber and Peters (1992: 305-352) was the development of a full language system, which in combination with biological changes, made sophisticated communication possible. As humans, we developed the ability to convey specific information, distinguish between past, present and future, cogitate symbolically ponder abstractly, imagine, and reason. Thus, in the late Stone Age, there was an abrupt and dramatic change in subsistence patterns, tools and symbolic expression.

In higher animals such as humans, adaptations expressed themselves in instincts, tastes and habits that would be group-beneficial, for example, resource sharing and the avoidance of communal dangers. There was undoubtedly a social hierarchy based on gender as in all primate societies, and the rudiments of a social system were further developed during this time. Supernatural beliefs appeared in the late Palaeolithic (old Stone Age). Apart from chiefs or tribal leaders, the social group was dominated by 'witch doctors' or 'shamans' to whom was attributed the power of communication with spirits. It is during this era that myths appeared (De Laet 1994: 640).

From the broad cultural stages of the Palaeolithic (9000-4000 BCE), to the Neolithic (4000-1200 BCE) to the Protohistoric (1200 - beginning of CE) to current times, disease has always been with humans.[14] With the shift to agriculture and sedentism, humans lived in closer contact with each other, with water-sources, with plants gathered and cultivated, and with animals hunted and domesticated. Closer contact with all these meant closer contact with the microorganisms they carried. Environmental conditions and biological and cultural human development led to development of agriculture, fishing, and animal husbandry. For example, agriculture spread to ancient Greece by about 6000 BCE from the Near East, with evidence of both plant and animal domestication (Bender 1975: 13).[15] At first livestock consisted mainly of sheep and goats with the main crops being emmer, barley and einkorn (*ibid*: 20). Soil erosion too resulted from human exploitation of the land, with, according to archaeologists van Andel, Zagger and Demitrack (1990), a major phase of soil erosion occurring after the advent of farming. Further, they give evidence that after the first millennium BCE there was serious intermittent soil erosion in many places, 'compatible with a model of the control of the timing and intensity of landscape destabilisation by local economic and political conditions' (*ibid*).

Water-sources were critical factors in human development with archaeological evidence revealing constructed human settlements in Europe and Asia over 12 000 years ago, most built near water sources. Then as now, water availability helped to determine both where and how people live and influenced the way in which they related to each other, for good or ill.

There is no doubt that certain epidemic diseases plagued our ancient ancestors from time to time, but throughout human history there have been epochs when states of equilibrium existed between people and microorganisms. During prehistoric times (which changed dramatically with the advent of the trade routes) physical and social conditions of the majority of humankind may well not have been ideal, but neither were they in a constant state of crisis. In spite of harsh conditions, by the end of the Palaeolithic our ancestors selectively propagated plants and animals, cultivated the soil and tended domestic animals, and expanding populations depleted or wiped out large game resources by effective hunting (Todd 1987: 265-267) – just as humans do today.

By the end of the Palaeolithic, in various parts of the world, humans reached a 'carry capacity' of five humans per 1 000 square kilometres of earth (Bender 1975: 77) and people were experimenting with new methods of growing subsistence grains. The shift from hunter-gatherers to sedentism meant increasing dependence on cereal grains as an energy source as well as a dietary shift based on preference. The dietary changes inevitably resulting from sedentism were not without consequences to human health. Reliance on grains as a staple decreased dietary breadth and reduced consumption of the fruits and vegetables that, together with insects and small mammals, had been primary food sources throughout primate and hominoid evolution. This nutritional departure was unparalleled; no other free-living animals routinely consumed cereal grains

(Milton 1993: 87). It is important to understand that the common grains of our ancestors were different from ours today, mainly they were smaller, difficult to harvest and to digest (Zohary 1969: 67), so they could not be consumed unless cooked, milled, winnowed, or threshed.

While the control of fire is claimed to be the earliest human technology (Mumford in Rifkin 1999: 7), the technology involved in the development of agricultural practices must be recognised as a major factor in the increase of infectious diseases. Why is this so? The production of grains for consumption resulted in a vast array of dietary resources and prepared the way for enormous expansion of human populations, trade routes and commerce, and tremendous societal changes. This incredible cultural adaptation was not merely quantitative, but a significant change from all earlier human behaviour, reflecting a major qualitative transformation. It was as Klein (2000: 33) says, a 'creative revolution' that exhibited technological ingenuity, social formations and ideological complexity.

Increased grain production effected by hybridisation influenced human population growth, land control and distribution, and interest in the development of trade routes. When grain became a staple, large populations of people were needed for sowing seed and reaping harvests. Land had to be cleared, water-sources diverted for crops, storage facilities constructed for excess, and so on. Land, water sources, and the people who worked them resulted in divisions of labour as well as the relegation of societal roles, including the creation of power bases of those who controlled such factors.

Other contributions to the rise in infectious diseases were through domestication of animals, problems with waste management, and increased numbers of humans. With the initiation of grains in human diet, meat was consumed less than in the earlier Stone Age. This, as Cohen *et al.* (1984: 112) speculate, is a probable cause of the decrease in stature and overall nutritional status that accompanied humans in transition from foraging to agriculture. Yet meat remained important, and as humans domesticated animals, from horses to cows to pigs to fowl[16], the microorganisms the animals carried spread to humans. Human settlements also provided a haven for other birds and animals drawn there because of human food stocks and the (mis)-management of wastes.

Certainly, the establishment of permanent agricultural societies brought with it problems with waste management. Simply leaving garbage where it lay, as was the habit of primitive hunter-gatherers, would cause problems in a settled society because human and food wastes and crop debris would decompose, attract insects and scavengers, and build up to nuisance levels. We know little about early human domestic habits, but compared with contemporary standards, management of waste was virtually non-existent. In the early age of sedentism, continued residence in one area must have necessitated cursory attempts at garbage control, but this resulted (at best) in waste being dumped in the surrounding countryside or covered over with layers of earth (Priestly 1968: 255). 'Sanitation' or public health as such did not exist as a concept, so contaminated human and animal waste often made its way into water supplies. Thus, entire settlements were always under threat from water-borne (diarrhoeas, dysenteries), water-washed (infectious skin and eye diseases, louse-borne typhus), water-based (schistosomiasis, guinea worm) and water-related (malaria, dengue fever) diseases (McGarry and Mara 1977).

This then is how microorganisms present in the hair, fur, skin, feathers, milk, and flesh of animals and birds found, in the blood of humans, a new environment to grow and flourish. The dust of animal fodder proved to contain microorganisms, such as anthrax, Q fever and tuberculosis, which were inhaled by humans. Sod breaking gave insects and plant-living microorganisms such as scrub typhus the chance to seek human hosts. Slash and burn techniques exposed human populations to mosquitoes such as *Anopheles gambiae*, which carries malaria. And let us not forget the increased presence of other humans and living conditions that were conducive to the spread of infectious diseases.

This brief glimpse into the relationship between early human behaviour and infectious disease reveals at least three major issues related to the rise of infectious disease and subsequent drug resistance. They are golden threads that we will see recurring and entwining in different ways as the narrative of disease and drug resistance unfolds:

- Our social behaviour, our *humanness*, contributed to the advent and spread of infectious disease.
- Our well-being has historically been grounded in environmental domination.
- There is not an instance in history in which the introduction of a new technology has had only benign consequences (Rifkin 1999: 70).

Disease, Societal Shifts and the Rise of Disease Networks

We do not know for certain about exact models of power in earliest hominoid development, but we can reasonably surmise a gender hierarchy. Moreover, then as now, whoever controlled the land, resources and technology probably controlled the means by which they could advance their power. Power, disease, and the means to affect societal behaviour, as we shall see, are all interrelated.

During the Neolithic the 'chief' became the 'king,' whose function became hereditary and whose powers were increasingly of a military nature. Likewise, in society's transition the powers of the traditional healers or shamans gradually became coupled with secular, economic and political powers. The transition from hunter-gatherer, with a practice of sharing produce equally, to food production replaced interdependency and reciprocity with competition for the possession of the greatest possible quantity of resources. According to De Laet (1994: 644), 'the advent of the concept of property conduced theft, plunder, and war'. This may have represented the introduction of moral dilemmas into human society and the origin of questions concerning right and wrong, good and bad, justice, and distribution of wealth. As trade and commerce developed (in conjunction with power bases from tribes to communities to nation-states), infectious diseases also spread. Such networks grew during and with the transition from agrarian societies to industrialisation.

In the mid-17th century, the 'medicalisation'[17] of the West coincided with the great ages of American and European expansion: the two phenomena are inexorably intertwined. From the beginnings of a global economy came the beginnings of mass consumerism. Mass consumerism served largely to negate the basic needs of human survival and in its place create the possibility of achieving material desires. Countries targeted for 'expansionism,' according to Watts (1997: viii), formed part of a larger scheme called 'development'. An unintended consequence of this development was the formation of identifiable disease networks, which like the old Portuguese trading network spanned the world. A second consequence was the rape of the earth in an attempt to meet increasing industrial needs. For example, natural resources such as coal and gas were necessary to keep the system operational. This of course runs concurrent with human desires such as the ever-increasing demand for products (furs, spices, exotic merchandise) designed to enable the continuation of a 'civilised' lifestyle.

Attempts to control disease networks fell under the domain of public health. During the early days of colonisation and imperialism, public health practices in 'conquered' lands depended to a greater or lesser extent on commercial interest, but the overall mortality rates of people in such countries remained extremely high (*ibid*: 7). However, in the West during the 19^{th} and 20^{th} centuries there was a general improvement in mortality from infectious diseases (although there was no simultaneous decline in their incidence). It is hypothesised that this decline in mortality was attributable to various factors based on technology, principally the application of sound public health practices (such as sanitation, urban planning, improved food hygiene, water purification and medical practices, notably the use of antimicrobials (Gostin 2000: 20).

The propagation and acceptance of the idea that human technology, embedded in industrialisation (or vice versa), and 'progress' were 'cure-alls' resulted in a general complacency regarding the consequences of technology on the part of the West. After World War II, some social changes further reduced mortality rates from infectious diseases. These included a decline in Western birth rates (which subsequently reduced food and housing demands), changes in personal hygiene practices (King 1958), and the rise of medical and scientific technology, particularly the advent of antimicrobial therapies.

Certainly great strides were made in the prevention and control of infectious diseases, particularly in the West. From successes such as the near-eradication of polio, the obliteration of smallpox, and the advent of vaccines to prevent diseases, there was a general lull in Western public health development. There was a myth, it seemed, that infectious diseases had largely been conquered. For example, the Nobel prize-winning author of a 1962 textbook, the *Natural History of Infectious Disease*, wrote:

> *At times one feels that to write about infectious disease is almost to write about something that has passed into history* (Burnet 1962: 3).

Fortified by such assurances, Western public health systems in the mid-20th century fell into decline as emphasis shifted to the so-called degenerative diseases or diseases of 'ageing societies' (Raleigh 1999: 983). Of course, this 'great conquest' was seen in the ethnocentric perspective of the West, ignoring the developing world and based naively on the premise that no new pathogens would emerge and on failure to realise that drug resistance has the potential profoundly to affect the treatment of emerging, re-emerging, and even 'old' infectious diseases.

Disease Causes

What were some early thoughts concerning the cause of disease? Certainly all is conjecture, but we can safely say that our early human ancestors had no concept of disease *per se*. How could one explain a healthy body one day and an illness the next? Much as ours are today, our early ancestors' interactions with the world were shaped by the way they saw it, in the same manner they perceived that the world was shaped by their interactions. To alleviate fears of the unknown, including disease and pestilence, mythic thought developed as an explanation for the unknown unfathomable world. In mythic thought, rationalisation for causes of disease was developed and personified by creatures and happenings in the real world. Thus, mythic figures took the form of beasts and humans and involved natural events. Such characters adopted the persona of humans; they replicated actions and inactions of human will and character – things that humans knew best. The myths suggest that early humans conceived the causes of diseases to be acts of retribution from higher powers – divine punishment from the gods for some misdeed, as Pindar (518-438 BCE) describes:

> *Thus now, he saw that son of Eilatos, Iskhys, the stranger share her bed of love, that impious treachery; and sent his sister storming in resistless anger to Lakereia, where by the high banks of Boibas the maiden had her home. And fate of a far other kind turned to her ruin and smote her down: and many a neighbour, too, suffered alike and was destroyed beside her; as when on the mountain from one small spark a raging fire leaps up, and lays in ruin all the widespread forest. He took the child to the Kheiron, that he teach him to be a healer for mankind of all their maladies and ills.*

Conceiving 'Disease'

Concepts concerning the cause of disease transformed with the evolution of human culture and remained inseparable from whatever system of beliefs was present in any given society, not much different from today. For example, ancient Mesopotamians believed an individual god ruled each body organ, just as they believed in a multitude of gods interacting as forces in their daily lives. Thus, should an organ become diseased, it was necessary to pray and sacrifice to appease the offended god. If by chance it healed, they offered further prayers and sacrifices. Today pilgrims travel to Lourdes and other holy shrines in search of a cure. The Hippocratics relied on the four humours in their theoretical practice which included the ancient philosophical elements of earth, air, fire and water. With the first attempts to address diseases in an objective manner, they are credited with the first major shift away from thinking of disease as 'sin' on the part of the patient or divine retribution. However, such old beliefs[18] may never be entirely eradicated and beliefs primal or otherwise may be reinforced and manipulated by prevailing ideologies.

In the 19th century, thoughts concerning disease aetiology fell under the influence of two developments that served as both a philosophical and an empirical basis for the biomedical approach to disease characteristic of modern medical practice. The first was the 'Cartesian revolution,' which gave rise to the idea that the mind and body were independent of each other. The second was the doctrine of specific aetiology or 'germ theory,' which was derived from the discovery of the microbiological origins of infectious disease. Together, they effectively denied the influence of, separately or together, any psychological, social, political, economic or environmental causes of disease.

According to Cartesian theory, the body, conceived as a mechanical device, was an apparatus whose illness needed to be corrected through manipulation of its parts by mechanical experts (the medical professionals), a notion historically reinforced by Galen. 'Cure' was effected through the neutralisation of particular adverse elements or by a modification of the physical process involved in a particular disease. While the Cartesian and the germ theory approaches to disease are still evident in Western medical practice today, they have been progressively challenged by multicausal models (Luke 1991; Eyles 1994).

Today the existence of infective agents such as microorganisms is seen as such an obvious truth that it hardly needs stating. For example, people are aware that they risk infection if injuries are left unattended and exposed to the environment. Two hundred years ago, such precautions were not so obvious. The threat posed by microorganisms was not part of common understanding, since such organisms had not been identified. This does not mean that until the introduction of the germ theory people were oblivious to the hazards of leaving an open wound untreated, but their conception of what would happen if they did so was very different from the idea of infective agents. Societies have approached the problems of infectious diseases, wounds, and illnesses in many different ways, and these approaches all made perfect sense to the people involved, however strange, if not irrational, they may seem from our vantage point at the beginning of the 21st century.

Modern medicine as practised today may seem to be qualitatively different from these earlier applications because it is based on the 'rationality of modern medical science'. The evidence to support this position is compelling given that medical science purports to be able to diagnose, treat and cure infectious diseases that have affected humans for thousands of years. However, the rise of drug resistance serves to show the fragility of complete reliance on this model. Another difficulty with this approach is that it only tells half of the story. This is because both medicine and science exist in social contexts that serve to limit as well as challenge their activities. A valid example is found in the Henle-Koch model of germ theory.

During the last two decades of the 19th century, scientists using the Henle-Koch model of germ theory discovered agents responsible for many infectious diseases, such as tuberculosis, plague, syphilis and cholera. Until then disease had generally been blamed on either a 'sin' on the part of the patient, a 'miasma' (atmospheric components made up of malodorous and poisonous particles generated by the decomposition of organic matter) (Risse 1993: 18), or an ill of deliberate intent usually caused by those outside a patient's particular cultural circle. The Henle-Koch model of germ theory can and has been extended, and often overextended, to account for other processes. And it can be manipulated by those in power to grasp public imagination and can become part of a public and popular mythology (Luckin 1984).

In the creation of such popular myths, the germ theory has been used both to label certain individuals or groups as potentially dangerous and as a metaphor for social persecution where the undesired group is perceived as germs or as nonhuman animals – germ-carriers infecting the wider society or its more powerful members. Creation of such popular myths requires building on common fears, the basis of which probably rests in the fact of death, the great unknown, and thus by association involves the medical classifications of 'disease' and 'health'.

For those in quest of power, popular myths may be used as a tool to classify members of a real or perceived oppositional power base, or simply undesirable members of society, as being nonhuman (germs) or sub-human (animals). This is because inherent in the germ theory is the perception that all germs and their possible animal sources should be eradicated. Thus, the classification of groups of people as 'vermin,' 'pigs,' 'rats,' or 'lice' serves to suppress any moral feelings concerning their treatment. 'Rational' man becomes irrational. In these mysterious and seemingly uncontrollable tragedies, the thin veneer of human reason peels back to expose a dark undersurface capable of inexplicable atrocities. For example, during the plague epidemics, people sought to blame others; scapegoating was rampant, and xenophobia was the norm. This was reinforced by those in power through mediasised ideological constructs, or, as Thompson (1990: 7) puts it, 'utilising meaning mobilised in the service of power'.

The Limits of Medical Knowledge

What were the circumstances in which medical knowledge developed, and how did medical knowledge influence social practices? Our starting point must be the inexorable link between modern medicine and the world of science, the combination of which has culminated in the dominance of what has been described as 'Western scientific medicine'. Conventional accounts of the development of Western science stress its emergence out of intentionalism and magic.

Although applauding prescientific thinkers for their energy in trying to understand change in the natural world around them, we recognise that science and the work of scientists starts to come into existence when the metaphysics of explaining things through external forces are replaced by approaches emphasising direct observation and experiment. Seeking out the regularities of phenomena and explaining why they should be so allows science to be both rational and (arguably) neutral. Such understanding forms the basis for technological innovation. However, as some historians and philosophers of science have pointed out, the idea of a simple distinction between irrational prescience and rational science is not always convincing.

Thomas Khun (1970) points out that the way in which science operates is very far from a rigorous objective assessment of evidence. Instead, he argues, scientific ideas are organised into a definable paradigm of ideas that create a state of what he calls 'normal science'. Such paradigms define the areas of acceptable knowledge and most scientists work within the framework of ideas provided by such approaches. This means that theories that work in accordance with the paradigm are regarded as the basis of scientific investigation. Where problems of evidence occur they are treated as anomalies to the paradigmatic understanding. New phenomena can only provide the basis for a new theory when the conceptual understanding necessary for the new theory has been established. When scientific change does occur, it is often the result of a crisis in existing theory that brings about a radical change in the ideas, which Khun calls a paradigm shift. According to his theory, this, rather than the empirical falsification of theories, is what takes place in the development of science.

The idea that scientific knowledge can be understood in terms of successive paradigms, each one replacing the last, has been applied to the development of Western medicine where, as Hudson (1993) notes, there has been a progressive displacement of the patient from the centre of medical interest. He points out that prescientific medicine can be characterised as 'bedside' medicine because the doctor had to pay particular attention to the complaints of patients who were under his (it was always a male) medical care and from whom he derived his living. For example, the nature of one influential model of prescientific medical knowledge was based on imbalances in the four humours, sanguine (yellow bile), melancholic (black bile), choleric (blood) and phlegmatic (phlegm). Diagnosis and treatment were seen in terms of restoration of the balance of these bodily humours, as illness was synonymous with the symptoms reported and not the outward sign of anything else. Thus, the focus of medical activity was of necessity centred on individual patients and their concerns. This system of medicine, originating with the Greeks and Romans, formed the basis of scientific medical knowledge well into the 17th century.

The shift from a patient-centred or bedside approach to 'hospital medicine' occurred, according to Hudson (*ibid:* 44-50), during the 19th century. Under hospital medicine, the patient's physical body became crucially important in aiding the understanding of disease. The way in which medicine was practised also changed with the ways in which the physical examination was used by doctors. It was perceived as a more objective method of investigation than the personal accounts of patients. As the 19^{th} century progressed, medical science developed a number of methods for investigating the bodies of patients, such as the stethoscope and X-ray. In addition, laboratory medicine became part of a doctor's routine investigations.

Such technological developments meant that the body could be examined rather than only observed. Pathologies could be quantified, leading to standardised methods of physical functioning. Such shifts resulted in the essential 'dualism' of the body and mind in medical practice where the individual's body is treated as separate from his or her understanding of it (Elliott 1999: 30). The negative effects of such approaches have given rise to various attempts at reconceiving the practice of medicine, the subject of the following section.

Medical Practice and Disease as Forms of Social Control

Much of the discussion concerning the reconception of medical practice (and thus disease concepts) is attributable to Michael Foucault who, amongst his other insights, recognises that the development of modern medicine has taken the particular route that it has because it simultaneously constructs its own object of enquiry and comes up with ideas to explain and deal with it. Two examples will suffice: to the prescientific physician, the evidence for the existence of humour was as compelling as the modern doctor's acceptance of laboratory blood results; just as medieval anatomists using Galen's account of the human body could 'see' what he had told them was there because that was what they were supposed to see. Medicine, then, provides internally both inquisitional objects and answers. Furthermore, Foucault (1973) identifies how in the creation of hospitals came what he describes as the 'clinical gaze' which established the idea that disease was a discrete phenomenon of the human anatomy. He (*ibid*: 196) claims the gaze is a way of seeing and understanding that becomes identical with the thing itself.

For Foucault, there are no fixed meanings or even the possibility of an appeal to an external reality. For this reason, he has often been identified with a theoretical approach known as 'social constructionism'. In this way of thinking, interest focuses more on how health and illness are created and understood by society and social processes than on seeking to find their biological basis. Similarly, Turner (1995) and Douglas (1970) maintain that in many cultures the body has been perceived as an image of society. As a result, notions about the body will often relate to prevailing ideas about society.

In a different way, Foucault (1973) makes the point that it is not only how medical science sees the body that is affected by discourses of knowledge, but also how people themselves view their own bodies. For example, the shift from traditional agricultural to industrialised societies was marked by a shift in people's conception of their bodies, from one of 'fleshy' to one of 'mindful' (Shilling 1993). What this implies is that instead of the body being just an object synonymous with the person, a central role is placed on the mind in directing not only what the body does but also the responsibility for its actions. The rise of the 'mindful body' itself changed the nature of health and illness as new 'problems' and new 'solutions' became commonplace in medicine. Clearly identifying this, Foucault (1973) points out that in the 19^{th} century a concern developed regarding the nature of sexuality, the problem of the 'hysterical woman' and the 'masturbating child' – tendencies that it was thought would harm the health of the nation if not countered. Foucault's analysis of the social construction of masturbatory concerns is reinforced by a report by Watts (1997: 104) in which he identifies how in the 'medicalisation' of Western imperialist practices, the medical establishment of the time first wrote about syphilis for profit and then promoted abstinence from masturbation. Thus, he claims, the medical establishment contributed to the epidemic spread of syphilis.

Nations may also be conceived in terms of health or disease. An example of this may be found in Nazi Germany where, authors Annas and Grodin (1992: 269) state:

> *... the conduct of those who worked in the concentration camps was guided by a biomedical paradigm of the moral danger facing the nation ... the paradigm of the states' facing a physical threat to its overall well-being that could be alleviated only by medical interventions is reflected in the medical literature and training of healthcare personnel both before and during the war.*

In other words, before and during World War II, the body of Germany was 'sick'. To cure the nation-body, most German medical professionals capitulated to the prevailing Nazi ideology eradicating the alleged 'cause' of its diseased state.

In addition, the works of Foucault have challenged ways of viewing what is accepted as normal and benign in that it is a product of our own contemporary imagination or the 'fabrication' of discourses. The challenge then is to locate the operation of 'micro power'. Ultimately, Foucault is interested in how power permeates every aspect of society to the degree that everybody was (and is) involved in the exercise of it. For example, in his studies of madness (1965) and penal policy Foucault demonstrated that far from there having been societal progress towards more humane management of the mentally ill and prisoners, psychiatry and penology have in fact developed from increasingly stringent control and more invasive tactics. Thus, we can see how medicine is directly involved in issues of social control. Most societies have to have some form of generally acceptable value systems or forms of social control if they are to remain relatively stable. This by definition means that there will be people who refuse to or cannot fit into the system, as well as people who for some reason society believes (or comes to believe, sometimes by purposeful manipulation) exist outside societal norms. Such people become seen and are often treated as societal deviants.

Various groups at various historical periods have been viewed as 'social deviants' – defined primarily by the prevailing societal norms (*e.g.* homosexuals, alcoholics, blacks). Scambler (2000: 171) defines deviance as 'non-conformity to a norm or set of norms which is accepted by a significant proportion of a society's citizens or inhabitants'. Deviant behaviour then may be considered as a behaviour that, as soon as it has become public knowledge, is routinely subject to sanctions – to punishment, correction or treatment. As many of Foucault's works identify, medicine has been involved in the construction and maintenance of forms of social control through socially sanctioned authority to define both medical problems and deviant behaviour. Through his archaeology and genealogy, we may again turn to Foucault who identifies through *power* how our Western societies have used normalisation as a tool to increase institutional power and to objectivise the individual, particularly those considered to represent deviants from the norm.

Drawing concepts of illness and disease as forms of deviant behaviour dates from the 1950s, principally through the work of Parsons (1951). He argues that illness is a form of social deviance because it disrupts the social system by inhibiting people's performance of their socially constructed societal roles. If such disruptions can be minimised, then the behaviour associated with the illness – which unlike other forms of deviant behaviour cannot be prevented by the threat of sanctions – must be controlled. Control is exercised through the prescription of social roles for the diseased and for medical professionals. It is through these socially created roles that rights and responsibilities of those designated as diseased and those responsible for their treatment are created.

According to Parsons (*ibid*: 81), rights of the deviant ill include exemption from their societal roles and from responsibility for their current deviant state. Correspondingly, their responsibilities include the obligation to want to become well as soon as possible and to co-operate and consult with medical experts when necessary. Failure to meet either or both of these obligations may lead to the accusation that these people are individually responsible for the continuation of their illness, and ultimately the withdrawal of their rights to any sick role. In these and other ways, the role of medicine includes the concept of social control. Continued further, Freidson (1970) contends that it is in the interest of medicine to pursue actively social constructions because it enhances the demands for the skills of medical practitioners: 'medicine's monopoly includes the right to create illness as an official social role' (*ibid*: 56). While one may argue against his position, he does identify that the practice of medicine includes power and responsibilities that extend beyond merely scientific origins.

In modern societies, doctors are generally responsible for collectively constructing and individually selecting and applying diagnostic labels as part of any society's social structure. It is recognised that the application and communication of some diagnoses have serious and unwelcome consequences for patients, most notably when the medical diagnosis is personally or socially stigmatising.

Stigmatising conditions are any deviant conditions that set their victims apart from the 'normal' in a society. Thus, in this context, people suffering from certain diseases (*e.g.* AIDS, cancer, psoriasis, mental illness) or who are disease carriers (*e.g.* plague, typhus, cholera) have been in the past and often continue to be, labelled as deviants. Such 'deviants' tend to be rejected or shunned to varying degrees by others. Another consequence of labelling is that the stigma attached to the illness evolves to dominate perceptions of the person suffering from it, and this affects how the bearer of the stigma is treated by others. In this way, the deviant illness becomes the focal point whilst the person's social identity, including his or her past, can be subjugated (Scambler 2000: 173).

The societal implications of the practice of medicine as a form of social control, the labelling of persons as 'deviant illnesses' or carriers of disease, the ways in which perverse power intersects with disease perceptions, the changing concept of disease, are all important considerations in understanding the contemporary problem of emerging and re-emerging infectious disease and drug resistance.

Disease Policies and Perceptions

After diseases are identified and labelled, they are inevitably classified. Medicine and science, as we have seen, play a major role in the identification and classification of disease. From disease classifications, official responses to disease are manifested in policies. Policies are usually under the management and control of governmental agencies. Such departments of course include people in the roles of politicians, scientists and health care personnel.

From early times until now, those who hold power have determined the official response to disease, although admittedly epidemiological contexts differ.

Generally, the powerful in society tend to claim that the disease in question targets only one particular set of people, while others are spared. While the history of Europe's plague epidemics serves as a well-known paradigm (the Jews having been the main targets of accusation), the 17^{th} century cholera epidemics reveal a construction designed to deny its existence. In England, the second and third cholera pandemics were enhanced by the concomitant industrial revolution, which caused a vast migration of people from the countryside to the cities in search of jobs. One consequence was the unregulated growth of tenements and slums. These workers (poor and generally uneducated) were considered expendable by those in power.

For example, Watts (1977: 107) points out that thoughts of supplying fresh water and removing waste was furthest from developers' minds as 'industrialists cut corners in order to maximise profits'. This meant that the living conditions of the workers were conducive to disease outbreaks, and no remedial measures were put into place as workers were considered (and for some time were) easily replaceable. Because such workers were socially constructed to represent the dregs of society (uneducated, ill-mannered, immoral) it was easy for those in authority to pursue their power bases unchallenged. Thus, when the cholera epidemic reached London, it struck the working class. However, nothing was done to ameliorate it and in fact its existence was denied. For example, in London officials were reluctant to quarantine ports or even incoming ships lest the emerging textile industry be harmed. Watts (*ibid*) explains that the local administration made the claim that there was no cholera in England. This unwritten policy remained in effect for almost 20 years and did little to limit the extent of the epidemic, or its impact on the working class.

Thus, the meanings of disease and the diagnostic expressions accompanying them ultimately find their meaning in what is done (or not done) with them rather than what may be said (or unsaid) about them. As Temkin (1977: 77) puts it:

> *Disease ... is thought of as the situation requires. The circumstances are represented by the patient, the physician, the public health, the medical scientist, the pharmaceutical industry, and last but not least, the disease itself ... our thinking about disease is not only influenced by internal and external factors, it is also determined by the disease situation in which we find ourselves.*

These thoughts find relevance to our discussion, because as already mentioned, diseases newly resistant to available drug therapy have recently been classified as 'Emerging Infectious Diseases'. At the time of writing, there is no documentation concerning any quarantine or isolation practices relating to diseases newly resistant to available drug therapy. (Multi-drug-resistant tuberculosis (MDR TB) is not considered an infectious disease *newly* resistant to available drug therapy. Quarantine and isolation measures for MDR TB exist in varying degrees world-wide.) There are, however, practices in place aimed at identification of carriers of drug-resistant microorganisms, particularly that of *Staphylococcus aureus* in European Union countries (van Bogaert 2003, personal communication). (*S. aureus* is a particular threat in nosocomial infections as well as a complicating factor in postoperative procedures).

Such hospital policies are not currently practiced in South Africa. Yet they may become more widespread as surveillance methods are implemented. Like quarantine, the use of such surveillance methods may be intended for protection of the general population and as such would be subject to the same considerations concerning exploitation, misapplication and ethical breaches that have been evidenced throughout history. The use of quarantine, isolation, and other methods of infectious disease control are open to a myriad of abuses, including abuses of rights.

Emerging Infectious Disease

As defined by the Centres for Disease Control (CDC 2001), Emerging Infectious Diseases[19] (henceforth EID) are 'diseases of infectious origin whose incidence in humans has increased within the past two decades or threatens to increase in the near future'. The 'incidence in human' reference is expanded by the United States Department of Defence (USDD) to also include '... animal, and plant infections' (USDD 2002: 1). Adopting the USDD definition of EID to our earlier definition of disease, it now reads as follows:

> *A state of disorder (dis-ease) threatening the integrity of any member of the biotic community including their physical, mental, or social well-being; any injury, ailment, deformity, or adverse condition in any living organism, system, or sphere including an interruption, cessation, or disruption of their optimal functioning attributable to microbial action whose incidence in humans, plants and animals has increased within the past two decades or threatens to increase in the near future.*

Under the heading of human EID (CDC 2001; USDD 2002; 1)[20] are various sub-classifications:

- Completely new diseases
- Reintroduced old diseases
- Old diseases occurring in new places
- Old diseases occurring in new populations

- New diseases with increased virulence
- *Diseases newly resistant to available drug therapies* (My emphasis added).

Two issues immediately come into focus when looking at this list. The first is that classifying 'diseases newly resistant to available drug therapies' as EID can work, as we have seen, for good or ill according to a particular disease 'situation'. In other words, we will socially construct from our experiences the meaning of 'diseases newly resistant to available drug therapies'. Our disease 'experience,' as I have tried to show earlier in this chapter, consists largely of our worldviews. Moreover, our worldviews are often influenced or mediasised by those in powerful positions. Importantly, our worldviews generally reflect the predominant perspective of humans as the centre of the universe.

Returning to the second shift in disease classifications, we see slight movement (intentional or otherwise) away from the classic conception of disease as caused by a single agent. This fits into our ecological triangle (microbial adaptation and change, human social factors and environmental changes) because in establishing the connections some questions immediately come to mind. Why are old diseases occurring in new places and in new populations? Why are there new diseases at all? Why are old diseases being reintroduced? What factors contribute to these occurrences? Why are diseases not responding to available drug therapy?

So far, I have provided an overview of the relationship between humans and infectious disease, and pointed out some of the ways in which we have historically conceived disease, and how power influences our conceptions. Now I turn to the final focus of this chapter, in which I identify the scope of global drug resistance. This is by intent a broad glimpse of some infectious disease types, vectors, geographical distributions, economic gaps, cultural practices, implied relationships with power, and human interactions. I want to emphasise that there are numerous reasons for the rise in infectious diseases and drug resistance. Moreover, I will show the problem extends beyond the human: an emerging infectious disease rising anywhere is a problem for the biotic community everywhere.

The Scope of Drug Resistance

The scope of drug resistance is broad and includes aspects ranging from 'culture' to 'nature'. Some critical features follow:

From a human health perspective, the past World Health Organisation's Director-General G. H. Brundtland (2000) stated:

> *Used wisely and widely, the drugs we have today can be used to prevent the infections of today and the antimicrobial-resistant catastrophes of tomorrow. However, if the world fails to mount a more serious effort to fight infectious diseases, antimicrobial resistance will increasingly threaten to send the world back to a pre-antibiotic*[21] *age. Our grandparents lived during an era without effective antibiotics. We don't want the same situation for our grandchildren.*

Drug resistance threatens to reverse medical progress. Diseases that were once easily curable (*e.g.* sore throats and ear infections) are in danger of becoming incurable. Increasing drug resistance could rob us of our ability to cure illnesses and stop epidemics (WHO 2000 c). Despite human ingenuity in developing drugs to fight illnesses, nature has proved to be infinitely more resourceful. The facts are grim. In 1998, 10 million people worldwide died of infectious diseases, of which 85% were acute respiratory infections, AIDS, diarrhoea or malaria (*ibid*).

In 2000, there were an estimated two million deaths due to tuberculosis (TB) worldwide, 98% of them in developing countries (WHO 2000 b). TB accounts for 25% of preventable deaths in adults in developing countries, but with the global rise in multidrug-resistant tuberculosis (MDR TB), and in combination with HIV, figures will continue to rise dramatically in the next few years (Ravigilone 1999). The independent states of the former Soviet Union have been hit particularly hard by MDR TB. For example, in Latvia MDR TB accounted for 25% of all reported TB cases in 1998 (WHO 1998).

Increasing numbers of patients show primary resistance to AZT and other antiretrovirals (Mellors and Kuritzkes 1998; WHO 1996 b). Reports from the *Southern African Journal of HIV Medicine* indicates this disturbing rise, noting results from the USA National Institute of Health study that showed 'an alarming incidence of resistance mutations … The genotype results showed emergence of new mutations in 3 of 8 individuals on interrupted efavirenz[22]-based regimens' (Martins 2003: 14).

Hospitals and other health care settings often harbour multi-resistant organisms that emerge due to the often-inappropriate use of antimicrobials. Intensive care settings may be the foci of the most virulent of microorganisms. Health care-associated exposures to organisms have been subjected to selective antimicrobial pressure and development of resistant microorganisms is a common source of serious complications (Tenover and Hughes 1996: 2335). In the USA, 140 000 deaths per year have been attributed to nosocomial[23] infections (*ibid*: 2337).

Although strides have been made in increasing public awareness of drug resistance these have primarily been in the developed countries that have sufficient infrastructure to confront the mounting public health crisis (Marchese and Schito 2001: 173). And it must be added that in the USA such awareness campaigns disintegrated with the 2001 dissemination of anthrax.[24] The antimicrobial of choice in anthrax treatment is (or perhaps rather was) ciprofloxacin, commonly called 'Cipro'. Levy (2002: 319) describes the resulting problems of stockpiling:

> *There is no control over how the consumer will respect an acquired antibiotic. A 60-day supply is far more than any antibiotic stockpiles I have ever seen in medicine cabinets. The run on Cipro thus becomes a further threat to our national health and welfare. Ingestion of this drug by people who stockpiled it will cause significant change in the microbiological environment. Treatment of other diseases now is compromised.*

Thus, it becomes evident that even in countries with sufficient resources and public health infrastructure to manage drug resistance 'things fall apart' when other dangers enter the equation. For faced with the prospect of a real or perceived illness or a real or perceived threat to wellness, rational approaches to antimicrobial therapy appear to disintegrate, a problem I will address in a later chapter.

Antimicrobials are unique therapies. It is significant to note that any individual use affects the microbial environment and thereby the greater physical environment including other living and non-living things. Because of this, emergence of antimicrobial drug resistance becomes a subsequent problem not only for the individual consuming a drug but also for anyone or anything sharing the environment.

The problem of drug resistance was partially addressed by the development in the 1960s of a succession of effective new antimicrobial therapeutic agents. However, in recent years the rate of development of such agents has slowed significantly. At the same time, there has been rapid and extensive development of antimicrobial resistance (EC 1999). Except for Linezolid (Stevens *et al.* 2002: 1481-1490), no truly novel antimicrobial drugs have been launched in more than 10 years, leading to increasing problems in finding effective antimicrobial chemotherapy for diseases caused by a number of major bacterial pathogens.

The development of antimicrobial resistance in many pathogenic microorganisms poses one of the most serious problems in the control of infectious diseases (CISET 1995; Fidler 1997; WHO 2000 b; WHO 2000 c). Global migration, known and unknown vectors, the increased use of antibiotics in stock feeds, and an environment under stress are some reasons for the global spread of pathogenic microorganisms, viruses, fungi, and so on (WHO 1997). Drug resistance is not a passing trend but likely to be a permanent feature in the fight against infectious diseases.

On one level, the answer to the problem of global drug resistance would appear to lie in the prudent use of antimicrobials. In other words and broadly speaking, since misuse of antimicrobials (in whatever way) is the problem, a reduction in use or misuse would reduce or even reverse such resistance. All this is true. Unfortunately, however, the problem is more intractable.

Gilliver *et al.* (1999: 233-234; 2001: 30-38) and others (for example, *see* Mallon *et al.* 2002) report that antibiotic resistance is now prevalent in populations of some wild rodents, which as far as is known, have never been exposed to antimicrobials. This finding in wild populations has profound implications, as the solutions to the problem of drug resistance have so far been based entirely on 'over/under-use' assumptions. The authors suggest that the origin of resistance and the selection mechanisms responsible for its high prevalence, 'if indeed such mechanisms are even necessary' (Gilliver *et al.* 1999: 234), are unknown. This is confirmed by scientists such as Allan *et al.* (1995) and Eichorst and Tischler (1999). Animals 'in the wild' are of course not all confined to the wild, as many species migrate and travel from their original habitats. Omnivorous woodmice and badgers, for example, some carrying drug-resistant microorganisms, travel long distances in search of food and new territory (Gilliver *et al.* 1999).

To make matters worse, the trait of drug resistance is not confined only to a bacterial species. According to J. Raloff (1999:637), many viruses, such as the 'flu' virus, originate in migratory waterfowl. When 'culture' is added to 'nature,' culturally embedded practices such the Chinese custom of valuing ownership of a pig and a duck, often for 'auspicious' reasons tethering them together, may lead to the unwanted consequence of a rise in new infectious diseases. In China, the infected droppings of wildfowl affect mainly domesticated ducks. Pigs then acquire the infectious disease from the ducks because of their proximity. But it does not end there. Because of their particular genetic make up, pigs serve as a 'mixing vessel' allowing the bird virus to mutate into a form that can infect humans. It is postulated that this is the origin of the recent severe acute respiratory syndrome (SARS) epidemic (Garrett 2003). For microorganisms, drug-resistant or not, there are no real barriers.

Such examples serve to illustrate the complex relationships between all life forms and how these relationships contribute to the problem of global drug resistance. Further, microorganisms present a particular dilemma because for Eukaryotes,[25] such as humans, they provide both benefit (life) and cost (death). The very things that sustain the human species hold the potential to obliterate it.

Concluding Remarks

To summarise, in this chapter I have tried to show how human biological and cultural evolution and the development and spread of Western medicine, including its technological concepts and management of disease, have evolved together with other human forces such as power and domination. Furthermore, I have tried to show how health and disease are conceived largely as social constructs. This has profound ethical implications for the topic because diseases newly resistant to available drug therapies have been classified as EID. Infectious disease and antimicrobial drug resistance (henceforth ADR) cannot be conceived as exclusively linked to a measurable failing or aberration in the human body alone. They must be seen in a different way, as phenomena linked to measurable defects in the biotic community as a whole. Thus, an examination of some major influences on disease patterns uncovers a complex picture, and one increasingly recognised as important for understanding the pervasive influence of social, political, economic and environmental factors on the prevalence and rise of EID and ADR.

Predominately Western worldviews contributed to the process of the separation of humans from the environment and negated its complex web of interrelationships and interdependency. However, what must also be considered is our own biological evolutionary model. Large brains, opposable thumbs, bipedal, upright posture, language, the capability to think, imagine and reason are some unique human biological endowments. As a species, we are unquestionably the dominant force of life on earth today.[26] We often deceive ourselves into believing we are all-powerful and apart from the rest of nature. However, microorganisms prove this otherwise.

Viewed from a broad perspective, we can conclude that as we moved in or out of rhythm with Nature, microorganisms were moving according to their biological evolutionary models as well. The clash would be inevitable. I will develop these issues in subsequent chapters, turning now to the first side of the triangle contributing to global drug resistance: Microbial adaptation and change. The other two "legs" of the triangle, namely human social factors and environmental changes will respectively be discussed in Chapters 3 and 4.

Chapter 2: Microbial Adaptation and Change

Our mastery of the microbial world is less complete than we might imagine and more subject to chance interactions in the environment than we might care to admit. Zambon and Nicolson 2003: 670.

Introduction

Life is an exploration into a world where nothing is stagnant, either within or all around us. Many transformations and events are invisible to our unassisted sight. When faced with changes we tend to classify things and happenings simplistically as either good (safe, stable, understood) or bad (dangerous, erratic, ill understood). We embrace what we perceive as good warmly, but we are inclined to consider anything bad as something to conquer and obliterate. As Dubos (1959: 13) put it:

> *... we do this often blindly without reflection and indeed at great price, when we like the apprentices of sorcerers, have set in motion the very forces that are potentially devastating and may someday escape human domination.*

Little did Dubos realise that the potentially dangerous forces escaping human domination about which he wrote would so soon become a reality - for our historical approach to infectious disease, prior to understanding it has been to 'conquer' it.

We called antimicrobials 'magic bullets';[27] infectious diseases and complications from injuries were now outdated. With the discovery of antimicrobials, technology had once again provided the answer, this time to the rude confrontation of the microbial world.

To address the problem of ADR requires recognising it as a problem as well as taking action to prevent or control it. Thus, it has a direct human ethical implication. If people acted responsibly, then the problem of global drug resistance might at best be contained. However, as I have already mentioned, the problem of ADR becomes more complicated with increasing evidence confirming that drug-resistant microorganisms are more extensively distributed in the environment than was previously thought. This has grave implications because the reproductive potentials of microorganisms are so versatile that the trait may be acquired by more and more microorganisms. In addition, we do not know if – and if so – how the trait of drug resistance might affect the balance of larger ecological systems.

This does not diminish the implication of human moral responsibility for the onset and continuance of ADR. However, it does point directly to grave human errors based on our wants, desires, pretensions and greed. We have historically assumed that we could control the earth, *i.e.* viewed the earth's resources only as commodities to serve us. We have failed to consider that our actions or inactions exist within the boundaries of the earth's closed system.

This chapter will provide the reader with background 'technical' information on global ADR. The first section, *Darwin's Paradigm,* is an overview of Darwin's theory of natural selection, then in *Antimicrobials and Drug resistance* I will ground the idea of natural selection in the evolutionary-biological mechanism of microbial adaptation and change. In *Different Types of Drug resistance,* I will link Darwin's theory of natural selection to a simplified version of our current understanding of the biological-genetic evolutionary model of ADR.

In the section *Bacterial Genetic Exchange,* I will emphasise that in some cases the precise mechanism for the development and transfer of ADR remains unidentified. This is because we do not fully understand the complex ways in which environmental stress influences this model. I will highlight the significance of microbial numbers and diversity in comparison with the human model in the section *Versatile Numbers.*

Darwin's Paradigm

The shift from the belief in a static worldview, held from at least the time of Aristotle,[28] came about from a dramatic event on the 24th of November 1859. This was the publication of Charles Darwin's *On the Origin of the Species,* representing perhaps the greatest intellectual revolution experienced by humankind. It challenged not only the belief in the consistency and regency of the earth, but also the cause of the remarkable adaptation of organisms and, most shockingly, the uniqueness of humans in the living world. Darwin argued that punitive large-scale forces did not exist and that all evolution could be explained from small-scale forces known as natural selection. Further, he proposed an explanation for evolution that did not rely on any supernatural forces or powers[29] (Mayr 2001: 9-11). This rocked fundamental beliefs, for to understand Darwin's theory – the idea of evolutionary progression[30] – is to understand that humans, while highest on the phylogenic tree, are not necessarily 'better' than either their ancestors or other species. Fortified by Divine Creation theories David Lyell, a contemporary of Darwin (quoted in Gould 2002: 63), stated the central issue well:

> *... whether we are to believe that man is modified mud or modified monkey: the mud is a great comedown from the archangel ruined.*

And this was and is the problem, for the idea of evolution struck, and amongst some, still strikes an emotional blow. Some humans tended then, as they do now, to equate what is phylogenically the 'highest' (at least as far as we are currently aware) with what is evolutionarily 'the best,' 'the pinnacle,' the 'top of the heap,' as a natural permission for domination over life forms perceived as 'lesser'.

But what is this Darwinian concept? Darwin explained evolution naturally, that is by using phenomena and processes that could be observed in nature. He did not describe how life itself originated. During the 70 years following his publication, this remained an unanswered question. Darwin did tentatively hint that 'all the conditions for the first production of a living organism ... (could be met) ... in some warm little pond with all sorts of ammonia and phosphoric salts, light, heat, electricity, etc. present' (Darwin (1859) 1997: 67). We now know that life was in fact sparked off in very much this manner. Preceding it, and concurrently, there was (and is) constant change. What is actually involved in this continuing change of the organic world? The question was at first quite controversial, even though Darwin already knew the answer. Over the years, a consensus on evolutionary synthesis emerged: 'Evolution is change in the properties of populations of organisms over time'. In other words, the population is the 'unit,' the 'whole' of evolution. Genes, species, and individuals play an important role, but change in a population characterises organic evolution.

It is, of course, in the genes (specifically plasmids[31]) of an individual member of a species that adaptations occur. For example, an individual member of a species through a variety of mechanisms may develop a particular attribute – an adaptation – that enables it to compete better against other genetically successful organisms for a niche in its environment.

The adaptive trait or property of drug resistance is an example of this. Although a few naturally occurring drug-resistant microorganisms exist, the problem of global drug resistance came about because of the excessive and imprudent use of antimicrobials. When a microorganism has acquired the trait of drug resistance it can compete better in its environmental niche because it now has a better chance of surviving: it cannot be harmed by a particular antimicrobial (or, as the case may be, many antimicrobials).

However, it does not end there. To be successful, the new organism must carry reproductive capacity and the ability to contend with the environment long enough to ensure reproduction and the survival of offspring. Successful subsequent changes or adaptations are then reproduced in further generations and may be adopted as positive survival traits by other species only bounded by their reproductive capacities.

True to Darwin's theory, not all adaptations are successful. Some prove intrinsically faulty and some, when there are dramatic environmental shifts, for example in the human body or in parts of the earth's biosphere,[32] fail to survive. Thus, an entire species may be extirpated because it is unable to adapt 'naturally' or genetically to the new environment.

Antimicrobials and ADR

Infectious diseases are a problem for all life forms. Furthermore, recent evidence[33] indicates an inexorable rise in the prevalence of ADR among microorganisms. This upsurge runs parallel to increased antimicrobial drug use and misuse in all life spheres. Drug resistance refers to the ability of microorganisms to acquire resistance to the majority of (and possibly all) available antimicrobial agents; in other words, a pathogenic microorganism ceases to be killed or inhibited by a particular drug or drugs. The increasing prevalence of resistance to antimicrobial agents among microorganisms compromises the effective treatment of diseases in humans, animals and plants. ADR is a particular problem in the treatment of 'old' diseases and compounded by the advent of 'new' diseases, as well as those classified as 'emerging'.

Drugs such as the antibiotics have saved countless lives, altered science, and influenced the practice of medicine since the advent of antimicrobial (*i.e.* antibiotic) therapy. Over the last 50 years, the classic treatment of bacterial infectious diseases has been with antibiotic usage, the introduction of which immeasurably changed the relationship between microorganisms and humans. The antibiotic use of penicillin may serve as an example.

Penicillin was first discovered by a French medical student, Ernest Duchesne (Lewis 1995). In 1928, Alexander Fleming, while working on the same principle, noticed that some laboratory cultures of microorganisms died when they came into proximity with a particular fungus. He extracted 'penicillin' from *Penicillum notatum* that confirmed its bactericidal ability in laboratory and clinical situations.[34] The combination of sulphonamides (an antibiotic discovered earlier) and penicillin, it was believed, would make it possible to eliminate infections and the major infectious diseases present in the world. The sulphonamides proved to be less successful than penicillin. Penicillin, marketed in the 1940s on an industrial scale, represented the beginning of the now burgeoning pharmaceutical industry (*ibid*: 54).

Penicillin was first made widely available to troops during World War II and touted as the medical miracle. Battle wounds are particularly prone to postoperative infection. Penicillin diminished this threat, disabling the pathogenic microorganisms and rendering them innocuous even in the worst infected wounds. The genetic basis for penicillin's action against many types of human pathogenic bacteria follows a macroevolutionary[35] model in the form of 'evolutionary gradualness' (Mayr 2001: 190). Early in the history of penicillin, it almost immediately cured almost any infection caused by streptococci or spirochetes. But penicillin-resistant microorganisms were already evolving. In simple terms, this is how resistance occurs.

Bacteria are genetically variable, and when an antimicrobial drug (such as penicillin) is ingested the most susceptible ones die rapidly. To all effects thanks to the action of the drug, a large number are killed and the host recovers soon. However, a few members of this microbial population acquire mutant genes, are drug-resistant and survive longer. In addition, a number of bacteria survive after treatment is completed. In this way, the prevalence of strains that are resistant to varying extents gradually increases in populations, whether human, animal, or plant. At the same time, within the remaining microbial population(s), new mutations as well as gene transference occurs, providing even greater resistance, and so the process of inadvertent selection for greater resistance to an antimicrobial (such as penicillin) continues. Resistance persists even if larger or stronger drug doses are used, and the period of treatment is extended. Finally, a completely resistant strain (or strains) emerges. By gradual evolution, an almost completely susceptible species of bacteria has evolved into a completely resistant one (*ibid*: 10).

In 1947, just four years after mass production of penicillin began, microorganisms in clinical cases began to show penicillin resistance (particularly amongst the staphylococci). Fleming, a microbiologist, recognised both the benefits and potential harms of antimicrobials right from the start. He was gravely concerned about their indiscriminate prescription by doctors and that people would be able to purchase them without a doctor's prescription. He wrote (Fleming quoted in Zimmer 2001: 214):

> *The greatest possibility of evil in self-medication is the use of too-small doses, so that instead of clearing up the infection, the microbes are educated to resist penicillin and a host of penicillin-fast organisms is bred-out which can be passed on to other individuals and perhaps from there to others until they reach someone who gets a septicaemia or a pneumonia which penicillin cannot save. In such a case the thoughtless person playing with penicillin treatment is morally responsible for the death of the man who finally succumbs to infection with the penicillin-resistant organism. I hope this evil can be averted.*

His calls remained unheeded, for this was the beginning of the 'antibiotic age'. Technology would provide all the answers as humans 'fled from reality to an altogether more soothing world of techno-pastoral dreams' (Ehrenfeld 1981: 127).

Before the advent of antimicrobials, most commensal and pathogenic microorganisms causing disease in humans were susceptible to them. Over about two decades, because of the increased use of antimicrobials (importantly, not just in humans but in animals and agriculture as well), selection unprecedented in evolutionary terms has occurred (Levy 2002). The effects of antimicrobials (principally their growth and inhibitory effects) have reduced the numbers of susceptible strains, leading to the propagation of drug-resistant varieties. They in turn have evolved into prominent members of the microbial flora.

The antibiotic susceptibility of microorganisms, *i.e.* normal bacteria found on the human skin and in the environment, is today, according to Levy (*ibid*: 45),

> *... quite different from what it was prior to the discovery and use of antimicrobials and even ten years ago.*

Drug and multi drug resistance (MDR) is commonly found in microorganisms that cause infections, as well as in commensal organisms that colonise our digestive tract, skin and upper respiratory tract. These resistant microorganisms are the products of the ancient process of the survival of the fittest.

There is a tendency to believe that when an antibiotic is ingested its effect (or lack thereof) is confined to a particular living creature, and in one sense this is so. However, what is not commonly understood is that the antimicrobial treats more than just the individual, for it ultimately affects the greater environment. This is because microorganisms circulate everywhere and there is a continual interchange between various human, animal, plant and agricultural hosts. In other words, once they evolve in a microorganism, antimicrobial genes and their genetic vectors can spread through the earth's interconnecting commensal, environmental, and pathogenic bacterial populations to other types of bacteria anywhere in the world (O'Brien 2002: S78). An explanation of how they do this follows.

As Darwin explained, every 'germ' competes in its environment – its niche – for survival. A mutation in one of its thousands of enzymes might make it more successful, perhaps better able to tolerate something present in that particular niche. If it develops this mutation, as O'Brien (*ibid*: S79) notes, it will increase its ability to reproduce and thus pass on this beneficial adaptation to its progeny

Because antimicrobial exposure affects all niches, it makes strains of drug-resistant microorganisms a special case.

Upon administration of an antimicrobial, the drug-resistant strain has an advantage because its competitors in all the niches succumb, enabling that particular strain to disseminate through many or all of the niches exposed to the agent. The effectiveness of niche compartmentalisation, which would normally tend to contain the spread of the microorganism, is diminished. Thus, the use of an antimicrobial drug has the potential to affect not just a treated human, animal or portion of the environment, but all antimicrobial-treated hosts everywhere.

Because of natural selection, once a resistant strain of bacteria has evolved in one host, it is more likely to be among the strains that this host transfers to a second one (Donskey *et al.* 2000: 1929). Consistent with the mechanism of natural selection, repeated selections would enhance the chances of that particular strain becoming established and transmittable in a third host, to a fourth host, and so on. In this way, drug-resistant strains of microorganisms travel the earth 'selectively through networks of hosts being treated with antimicrobial drugs' (O'Brien 2002: S81). This 'network of hosts' is not confined to humans.

Use of an antimicrobial agent selects for overgrowth of a bacterial strain that has a gene expressing resistance to the agent. It also selects for the assembly and evolution of complex genetic vectors encoding, expressing, linking and spreading that and other resistant genes. Once evolved, a competitive construct of its genetic elements may spread widely throughout any or all of the world's bacterial populations. In this way, a bacterial isolate at any place may be resistant, not because nearby use of antimicrobials had amplified such a genetic construct locally, but because distant use had caused the construct or its components to evolve in the first place and spread there. The levels of resistance at any time and place may therefore reflect in part the total number of bacteria in the world exposed to antimicrobials up until then.

Types of Drug resistance

The basis of antibiotic resistance is genetic. However, there are different types of resistance. Some resistance is 'innate'. This means some organisms are inherently resistant to many antibiotics. This resistance probably evolved as a response to previous exposure.[36] For example, a streptomycete has a gene responsible for resistance to the antibiotic used against it, a Gram-negative bacterium has an outer membrane that establishes a permeability barrier against the antibiotic, an organism lacks a transport system for the antibiotic or it lacks the target or reaction that is hit by the antibiotic.

There is also acquired resistance. Bacteria previously sensitive to antibiotics can develop resistance by means such as mutational resistance and horizontal gene transfer, which change the bacterial genome. Acquired resistance is driven by two genetic processes in bacteria: (1) mutation and selection (sometimes referred to as vertical evolution); and (2) exchange of genes between strains and species (sometimes called horizontal resistance). Vertical evolution is strictly a matter of Darwinian evolution driven by principles of natural selection: a spontaneous mutation in the bacterial chromosome communicates resistance to a member of the bacterial population. In the discriminatory environment produced by the antibiotic, the wild types (non-mutants) are killed and the resistant mutant grows and thrives.

Finally, there is horizontal resistance. Horizontal resistance is important because it involves the acquisition of genes for resistance from another organism. For example, a streptomycete has a gene for resistance to streptomycin (the antibiotic used against it), but somehow that gene escapes and attaches to or invades another bacterium, *e.g. Escherichia coli* or *Shigella.* Or, more likely, a bacterium develops genetic resistance through the process of mutation and selection and then donates these genes to some other bacterium through one of several existing processes for genetic exchange.

Bacterial Genetic Exchange

Bacteria are able to exchange genes in nature by three processes: conjugation, transduction and transformation. Conjugation involves cell-to-cell contact as DNA crosses a sex pilus from donor to recipient. During transduction, a virus transfers the genes between mating bacteria. In transformation, DNA is acquired directly from the environment, having been released from another cell. Genetic recombination can follow the transfer of DNA from one cell to another, leading to the emergence of a new genotype (recombinant). It is common for DNA to be transferred as plasmids between mating bacteria. Since bacteria usually develop their genes for drug resistance on plasmids (called resistance transfer factors, or RTFs), they are able to spread ADR to other strains and species during genetic exchange.

The combined effects of fast growth rates, high concentrations of cells, genetic processes of mutation and selection, and the ability to exchange genes, account for the extraordinary rates of adaptation and evolution that can be observed in the bacteria, a point I will elaborate upon later. For now, it is sufficient to note that bacterial adaptation (resistance) to the antimicrobial environment takes place very rapidly in terms of evolutionary time.

New mechanisms of genetic plasticity of one microorganism or another are uncovered constantly. Spontaneous mutation may be just the beginning. The large populations of microorganisms such as haploids can immediately express genetic variations. This is because they have a wide range of repair mechanisms, themselves subject to genetic control. Some strains are highly mutable by not repairing their DNA, while others are relatively more stable. In responding to environmental stress, they are flexible. Mechanisms proliferate whereby bacteria as well as viruses exchange genetic material quite promiscuously. Plasmids spread throughout the bacterial world crossing boundaries with, for example, yeasts and bacteria. Bacterial species fluctuate greatly in their innate susceptibility or resistance to various antimicrobials. Further, there is also a range of vulnerability within any bacterial species, so some organisms are more susceptible than others are. Hundreds of resistances are known, but according to the UK Science and Technology Report (UKST: 1997-1998), all can be ascribed to one of the following five broad types of mechanisms:

1) If the bacteria can inactive the drug before it reaches the bacterial cell;
2) If the outer layers of the cell are impermeable;
3) If the drug enters the cell but is dispelled by the bacteria;
4) If the target is altered so that it is no longer recognised by the antibiotic; and
5) If the bacteria acquires an alternative metabolic pathway that renders the antibiotic's target redundant (called a 'by-pass').

Versatile Numbers

Life is cellular and cells have the capacity to evolve the nucleic acids involved in Darwinian evolution. Such properties include molecular variation and molecular selection (the basis for genetic variation and natural selection). This principle applies to all living things. However, not all living things are genetically equal; for example, there are vast differences between humans and microorganisms.

Microorganisms evolved, as have all life forms, and with the advent of microbial sex, about 1.1 billion years ago, additional species developed (Schopf 1993: 65). At some point in this distant past, a now long-lost oxygen-breathing bacterium gave rise to the ancestors of both *Rickettsia* and mitochondria. Both lineages were once free-living microorganisms feeding on nutrients that surrounded them. It worked in this way: Protomitochondria (the precursors of mitochondria) in all probability stayed close to early Eukaryotes, in order to feed on their waste. The Eukaryotes, which could not use oxygen for their metabolism, came to rely in turn on the wastes of these oxygen-breathing protomitochondria. Eventually the two species merged and the exchanges between them began to take place within a single cell.

As time passed, each lineage began to live inside other organisms. *Rickettsia* evolved as a parasite that could invade and ravage human and animal hosts. However, the bacteria that invaded our human ancestors ended up in a better relationship, certainly from the perspective of the Eukaryote. The primitive oxidative-capable bacterium (the mitochondrion) that invaded us enabled Eukaryotes such as the human species to receive nutrition from plant polysaccharides. This was a very beneficial development, as our own digestive enzymes are incapable of degrading these. More, microorganisms live as commensals on our skin, just as they have adapted for different beneficial functions in other nonhuman eukaryote species. Mitochondria[37] are present in every cell of our bodies.

As Lederberg (1997: 420) put it,

> *We are the re-synthesis of components of genetic development that diverged as far as the bacteria and then were reincorporated into the mitochondrial part of our overall genome.*

Without microorganisms, humans could not exist. Present in a largely unseen world, microorganisms helped to create the biosphere and continue to support life on earth. Microorganisms are ubiquitous, found in diverse localities such as hypothermal vents, water, soil, air and even soda lime lakes. In the natural environment, they are specialised, responsible for recycling nutrients and purifying physical sources such as water. Microorganisms are also used commercially in food production; the most common example is their use in fermentation. Contemporary wastewater treatment, biodegradation and biosynthesis are additional uses. Approximately 70% of antimicrobials currently in use are products of microbial fermentation. Most are harmless and do not cause disease in humans, animals, or plants.

True to the Darwinian thesis, humans continue to evolve biologically. Mutations regularly occur in our DNA and these are transmitted to our children. On the level of the genotype, these difference mutations, called polymorphisms, make each one of us unique. Further, an analysis of these differences identifies how closely we are related to each other at the level of the genotype. No generation has exactly the same genetic make-up as did the previous generation; chance alone is sufficient to guarantee this. However, and this is of critical importance, our species is a relatively slowly reproducing one compared with the world of microorganisms. Microorganisms outnumber humans.[38]

Lederberg (1997: 418-419) notes:

> *... the populations of microorganisms measure in exponents of 10^{12}, 10^{14}, 10^{16}, over a period of days.*

The problem of numbers is significant because microorganisms, with amazing diversity, continue to rapidly develop and change, adapt to new environments, reproduce, and survive. For example, over one billion trillion bacteria of diverse types live and compete in the earth's human and animal populations, as well as in the environment (O'Brien 2002: S78). The mutation rate for the majority of bacterial genes is approximately 10^8; however, some bacteria exceed this by rates of 10^{12}, 10^{14} and 10^{16} (Lederberg 1997: 419). It works in this way: if a bacterial population doubles from 10^8 cells to 2 x 10^8 cells, there is likely to be a mutant present for any given gene. Because bacteria grow to reach population densities far in excess of 10^8, such mutant cells could result from a single generation during only 15 minutes of growth. Hypothetically, one single bacterium could replicate every half hour to generate a billion progeny overnight. It is fortunate that any bacterium has only a fifty-fifty chance of replicating successfully (O'Brien 2002: S78).

There is a degree of mutual accommodation between humans and microorganisms that is to the benefit of both. Ideally, it would ensure the survival of both. However, the behaviour of microorganisms remains unpredictable when they are under stress, even though it remains under genetic control (Lederberg 1997: 420). The bottom line is that there is no possibility that humans, animals or plants can adapt genetically (in Darwinian or any other fashion) to meet the challenge of the numbers and potential threats that microorganisms present.

Concluding Remarks

In this Chapter, I have tried to show that Darwin's idea of natural selection is the basis on which our current knowledge of the mechanism of ADR as well as the different types of resistance is informed. There are two points in particular that I wish to emphasise. The first is that in some cases the precise mechanisms of development and transfer of ADR remains unknown. As I noted, this is because we do not fully understand the complex ways in which environmental stress influences the biological-evolutionary properties of microorganisms. With that knowledge forewarned, we should look upon this unseen world of microorganisms with a sense of caution when remedial measures of control or containment may be proposed. Secondly, the significance of microbial numbers and diversity in comparison with the human model is of critical concern to any control or containment strategy. Both of these points will gain relevance in further chapters.

I indicated that ADR is a serious problem affecting the biotic community because it has permeated the biosphere. Pragmatically, since the degrees and levels of ADR in the biosphere are only now being investigated (and this process will take years of political will and scientific perseverance to complete), the problem of ADR will take a very long time to contain, if this can be accomplished at all.

Finally, the observation was made that we as humans owe our existence to microorganisms; they sustain our lives and the life of our planet but paradoxically, because of human social factors, we have created the conditions conducive to greater evolution of pathogenic forms. This is the subject of the second leg of the triangle contributing to global drug resistance - the topic of the next chapter.

Chapter 3: Human Social Factors

... In serving the body the mind wreaks havoc on nature. And in doing so, it increasingly adds to our needs and desires, more dignified than those of the body but possessing an appetite equally as ravenous for the Earth's resources. This is evidenced by the physical prodigality of advanced cultures, which only increases the impact of an already excessively large population upon the shrinking resources of the natural world. Hans Jonas 1999: 53.

Introduction

The ways in which we contribute to EID and ADR cover a wide-range of human activities such as population pressures,[39] consumerist practices and misuse of antimicrobials in medicine and industry. In this Chapter, I will begin by first looking at *Human Population, Environment and Disease.* In the second section, *ADR and Industry*, I will show the variety of ways in which ADR is enhanced by the production and distribution of food to feed large populations. In the following section, *Misuse of Antimicrobials in the Agricultural/Food Animal Industry*, I will examine how ADR spreads because of indiscriminate and unnecessary use of antimicrobials. I will pay particular attention to the use of pesticides, showing how their indiscriminate use results in a shift in insect populations as insects become increasingly resistant to any pesticide in current use.

In the fourth section, *ADR and Medicine,* I will suggest that the borders between medicine, environment and the human sciences are complex and entwined. In the final section, *Misuse of Antimicrobials in Medicine,* I will introduce human and cultural factors in antimicrobial use and abuse, showing the different ways in which these factors influence continued ADR. Through an overview of pharmaceutical practices, I will show how through their marketing practices they contribute to ADR. Overall, I will show the complexity of addressing ADR.

Human Population, Environment and Disease

'Most if not all environmental problems in the world are caused directly or indirectly by human population pressure,' writes Coetzer (2002: 10), a sentiment echoed by many environmentalists, community health practitioners, public health officials, veterinarians and ecologists worldwide. The earth is overpopulated with humans. It is overpopulated now and there is little reason to believe that humans will act purposely to reverse this trend. Despite massive human mortality due to wars, epidemics and civil strife, the global human population is increasing at a tremendous rate.

The World Bank offers a sombre projection for the year 2050 of 11 to 14.5 billion humans living on the earth (Garrett 1994: 552). The USA population alone will double to 540 million during the next 70 years (Gelbard *et al.* 1999; PRB 1996; USBC 1999). Coetzer (2002: 8) provides local statistics:[40] 'The population growth in South Africa is 2.17% per annum. In 2022, there will be 79 million people – that represents an increase of one million per year.'

Social, environmental, and disease problems are already severe in urban areas of the world where the number of people continues to double particularly quickly, *i.e.* every 20-25 years.[41] According to projections by the World Resource Institute (WRI 1999), by 2010 more than half of the world's population will live in 'megacities' (defined as cities having more than 1 million residents). By 2025, two-thirds of the world's population will have settled in large urban areas.

Sustaining the human population places enormous pressure on the resources of the earth. While ecosystems have the capacity to recover after natural or human-made insults, 'this capacity is adversely affected by increasing stress or damage' (Coetzer 2002: 7). Increased environmental stress opens a variety of avenues for greater EID and ADR.

In Chapter 2, I pointed out that microorganisms vastly outnumber humans and have far superior adaptive properties, writing: 'The problem of numbers is significant because microorganisms, with amazing diversity, continue to rapidly develop and change, adapt to new environments, reproduce, and survive'. As an example, I used O'Brien's (2002: S78) findings that over one billion trillion bacteria of diverse types live and compete on the earth's human and animal populations, as well as in the environment.

While the problem of the numbers and adaptive properties of microorganisms is significant, it is rarely mentioned in relation to human population problems. It gains significance when held against the human model. This is because when increasing numbers of humans are combined with the far greater number of microorganisms, disease-carrying and drug-resistant microorganisms will have more hosts in which to continue their evolutionary process of adaptation and change, particularly when people live close together (the population density factor).[42] Disease and ADR will therefore affect not only increasing numbers of individuals but a greater proportion of the population.

The urban population of South Africa is projected to increase from 16.2 million in 1985 to over 25.7 million in 2004. Every year in this country over 750 000 people migrate from rural to urban areas (Coetzer 2002: 10). Optimally this would require over 3 000 residential units to be constructed every working day until 2004 (*ibid*). When increased population pressures are not managed optimally, squatter camps and other informal settlements arise, most without sufficient public health infrastructures. Such densely crowded habitats are conducive to the spread of disease and ADR.

It is known that once established in an organism or in non-organic life a drug-resistant strain can be transmitted in a variety of ways to affect, infect, and reinfect the biotic community. In addition, if the normal population regulation mechanisms (Density Independent Controls or Population Dependent Controls) are imbalanced in this community, additional avenues become available for the spread of disease and drug-resistant microorganisms.

The two major causes of increased antimicrobial resistance are found in the areas of industry and medicine. To illustrate this, in the following sections of this Chapter, *Drug resistance and Industry* and *Drug resistance and Medicine*, I will show how affluence and poverty, terms used in their broadest sense, both promote the growth and adaptation of disease and drug-resistant microorganisms.

Drug resistance and Industry

Industrialisation has both benefits and costs. Larger human populations require larger food supplies. Industry therefore has a vested stake in the successful production and marketing of food products and of antimicrobials for use in plants, animals and humans, for therapeutic purposes or not. In the broad field of 'agriculture,' antimicrobials are used for example in aquaculture and fruit tree production. All are important and linked, but the food animal industry remains the largest promoter of antimicrobial use and resultant ADR.

The emergence of resistance to antimicrobials has compromised control of many bacterial pathogens in human, veterinary, food-animal and agricultural practices, and its control is a mounting problem. Additionally, multiple drug resistance has emerged among many bacterial strains, including *Salmonella* species. The WHO (1997) reports:

> *The development of resistant pathogenic bacteria occurs from the human, animal and environmental uses of antimicrobials. Food animals are commonly exposed to antimicrobials for therapeutic indications, to improve feed efficiency, and weight gain.*

And this is true. But a short elaboration may be in order. Almost half the 50 million pounds of USA-produced antimicrobials marketed globally is used in food animals. (Production of such antimicrobials *per se* is not significant in developing countries.) Of this global market, the Union for Concerned Scientists (1999) reported that the percentage of antimicrobials used in the USA for various purposes was as follows:

> Therapeutic: 6% in livestock, 8% in human, and 15% in other.
>
> Non-therapeutic: 71% in livestock

'Non-therapeutic' use refers to antimicrobials given routinely to healthy animals, usually in feed. Antimicrobials are added to livestock and poultry feed for two reasons. The first is for 'growth promotion,' what the industry prefers to call 'digestive enhancers' according to the UK Science and Technology (UKST) Report of 1977-1978. The logic is that more meat will be obtained from a larger animal than from a smaller one, and more meat means greater profit. The second is for prophylactic purposes. The purpose of 'prophylaxis' as reported by Goldberg (2001: 2) is 'to compensate for crowded, stressful, unhealthy conditions of factory farms'.

Food animals themselves, because of the constant addition of antimicrobials to their foodstuffs, build up resistance to such drugs. When they become ill, different antimicrobials will be administered (which in turn feeds the drug industry). While there is only a moderate degree of overlap between the drugs used for cattle and chickens and those used for humans, as Goldberg (*ibid*) notes, 'fully 95% of the drugs used in swine production are also used in human medicine'. Thus, the intestinal flora of animals that have been exposed to human antimicrobial agents can serve as a reservoir of resistant bacteria. These organisms may be present in animal-derived food products (FDA 1999; FDA 2000). Sweden and Denmark have banned all non-therapeutic uses of antibiotics. Denmark has tracked the results quite carefully, and showed 'that total use of agricultural antibiotics has decreased 60% and has resulted in virtually no effect on animal health and productivity' (EEC 1999). The European Union has followed a similar initiative (*ibid*).

As the USA is the major manufacturer of antimicrobials marketed globally, they have an economic interest in continued promotion of antimicrobials. Although they lobbied against changes, under international pressure or perhaps in recognising the importance of increasing ADR, the USA Food and Drug Administration (FDA 2000) suggested new federal regulations concerning the use of antimicrobials in agriculture and the animal food industries. Concerning use in animals, these include:

1) that companies seeking to sell a new animal antibiotic would have to prove it is not expected to cause significant resistance;
2) that the government would test current levels of food-borne drug resistance and then set limits on how much resistance could increase before an implicated animal antibiotic would be restricted, even banned;

3) that the FDA would rank animal drugs, giving those most closely related to vital human antibodies extra scrutiny; and
4) that some companies might be ordered to conduct ongoing tests on farm animals to monitor drug resistance.

There are allowable levels of drug-resistant residues set by the FDA. Low levels of antimicrobial residues in agriculture and food animals were not traditionally viewed as a cause of resistance; however, new evidence suggests that even very low residues of certain antimicrobials may select for resistant bacteria in the human gastrointestinal tract (Coffman and Beran 1999: 5906).

A two-day meeting to deliberate the FDA's new proposal on the ban of antimicrobials in food animals highlighted the controversial nature of the issue. For example, Dr Brendan Fox, president of Elanco Animal Health, was quoted as saying, 'We believe the agency is overreacting'. Dr Frederick Angulo from the Center for Communicable Diseases (CDC) was quoted as responding, 'If there is no risk, you shouldn't be afraid of the FDA's rules' (Neergard 1999: 1). On one side, industry denies there is any serious risk to consumers, noting that no one has died from eating meat tainted with 'untreatable germs'. On the other side, public health experts say they must act to protect consumers from that ever happening[43] (*ibid*). Let us turn to some issues in the debate.

ADR extends to the larger community through various routes of exposure. Via humans, it may be furthered by way of foodstuffs themselves, be they animal or plant, exposed during slaughter or harvesting, in progressive handling, and finally through consumption. People working in the food industry contribute to the spread through handling of animals and crops (*i.e.* feed, fertilisers, manure) and thus transferring the resistance to themselves, their families and their working community (Levy *et al.* 1976 b: 586). Through contamination of soil and surface water, drug-resistant bacteria plus undigested antibiotics found in animal manure continues the cycle of contamination in humans and nonhuman life. Two trillion pounds of animal waste are produced in America per year (Goldberg 2001: 2). Such wastes are usually stored in large open containers but can overflow for example during rainstorms, continuing the cycle.

Levy reports there are five times as many food animals as people in the USA. He (2002: 620) writes:

> *... daily animal fecal excretion can be 5 to 400 times greater than that of humans. For example, the amount of feces excreted by a cow per day is 100 times that by a human. Hence, animals are contributing a large amount of resistant bacteria to the natural environment.*

He (*ibid*: 616) also refers to studies showing that flies caught on flypaper carried antibiotic-resistant organisms with the specific genetic markers of identical strains from nearby animals.

A USA geological survey study, published by Rue *et al.* (1998: 342-348), tested 139 streams in 30 states, 62 of them near agricultural facilities. Of 22 antimicrobials tested for, the researchers found 14 in stream samples. Of these five were used only in human medicine, one only in animal agriculture, and eight in both. Half the antimicrobials used in the USA to treat humans are also used to treat disease-infected domestic animals such as cats and dogs (ASM 1994; ASM 2001). The concurrent use of antibiotics for both humans and livestock enhances selection for drug-resistant microorganisms, further exacerbating the problem of ADR.

Pesticide Use

In a different although related way, pesticide use (generally including insecticides, herbicides and fungicides and mixed formulations) in crop production has been implicated as a major contributor to human and animal disease and environmental pollution. Use of pesticides has continued to increase in both developed and developing countries since its initial application after World War II (Praveen and Nakagoshi 2002: 1). In addition to industrial uses, pesticides are also used to control insects, particularly insect vectors of human diseases such as malaria (Coetzer 2002: 34; WHO 1990; WHO 1993).

Globally, approximately 1.3 billion kilograms of pesticides were used per year in the early 1990s. At that time, the number of human pesticide poisonings reached an estimated 500 000, with approximately 6 000 reported deaths (Labonte 1989). These figures increased dramatically in only a few years. In 1995, worldwide pesticide use was approximately 2.5 billion kilograms, and 2 years earlier approximately 3 million human pesticide poisonings were reported (Pimentel 1995: 55). In this same year, 220 000 people died of pesticide poisoning and about 750 000 were confirmed as having chronic pesticide-induced illnesses (WHO 1990). The majority of these cases are reported from developing countries where infrastructure and knowledge concerning the implications of pesticide use are lacking (Benbrook *et al.* 1996; Maddy *et al.* 1990).

In addition, in developing countries a vast range of pesticides is available, many of which have been banned in developed countries and subsequently 'dumped' on the market in developing countries.[44] According to Coetzer (2002: 208, 210) pesticide poisonings (human, animal and environmental) rise from a range of agricultural practices such as crop dusting, crop fumigation, seed coating, crop spraying, and application to plant roots in soluble form as vermicide. Pesticides are also used in the animal food industry for animal dipping and in canal irrigation systems. Pesticide use in developing countries is expanding, even though these countries already account for over 95% of serious poisonings and fatalities (Helliker 2001: 1). Some of the most acutely toxic chemicals are widely used, with no protection, by farmers and workers with no training and limited awareness of the hazards. USA data indicate that 18% of all pesticides and approximately 90% of all fungicides are carcinogenic and pose a hazard to human health (NAS 1987).

Developing countries do not use as much pesticide as developed ones do. As reported by Praveen and Nakagoshi (2002: 1), some Japanese farmers used protective clothing while applying pesticides, and stored them properly, because they knew of their harmful effects. However, they continued to use them to enhance the crop's cosmetic appearance and hence profitability. On the other hand, farmers in Bangladesh used lower doses of pesticides more frequently without knowledge of their harmful effects and therefore without any protective clothing or storage concerns. Thus both contribute, although in different ways, to the global problem of environmental destruction, disease and subsequent ADR. These examples illustrate that pathways are conducive to the spread of environmental decay, disease and antimicrobial resistance through our vibrant ecosystems.

In both developed and developing countries, the connection between use and abuse of pesticides and disease and ADR lies in disturbance of the ecological balance between pests and predators. Such disturbances include ground and surface water pollution, soil and food contamination, and subsequent impacts on wildlife and human health (PAHO 2002; Pimentel and Bashore 1998).

Perhaps the most widely read book on the results of pesticides is Rachel Carson's *Silent Spring* (1962). Carson painted a frightening picture – pesticides and other chemical compounds used by agriculture and industry were poisoning the land, animals and most shockingly the humans who were supposed to be reaping the benefits of chemical use. Her introductory Chapter 'A Fable for Tomorrow' describes a make-believe town, once thriving with diversity of plants, animals and prosperous farms, all of which fell victim to an insidious 'blight'. Sickness was more frequent amongst both adults and children; farm animals were having difficulty reproducing; and most significantly, a 'strange stillness' hung over the once vibrant community. Animal life, most prominently birds and insects, had vanished:

> *It was a spring without voices, on the mornings which once throbbed with the dawn chorus of robins, catbirds, doves, jays, wrens, and scores of other bird voices, there was no sound ... The apple trees were coming in bloom but no bees droned among the blossoms, so there was no pollination and there would be no fruit (ibid: 3).*

The description of the lifeless town, complete with a fallout of a 'white powder' on house roofs and lawns, in streams and fields, called to mind the image of a nuclear holocaust. But as Carson emphasises, 'no enemy nation had silenced the rebirth of new life in this stricken world. The people had done it to themselves' (*ibid*).

She proceeds to identify in detail the chemical composition, characteristics and apparent impacts of pesticide use on both human and animal life. The book and Carson personally were immediately the targets of intense criticism. She was portrayed as a 'hysterical' woman whose knowledge of science was suspect and whose emotional perspective obstructed any realistic and practical assessment of the situation.[45] However, her careful research, which inspired numerous other reports on the environmental impact of chemicals, pointed out that in the overuse of pesticides, humans were endangering themselves and all life around them. She identified this danger explicitly as the ways in which toxins enter the food chain at the lowest level and become more and more concentrated in each subsequent consumer until eventually lethal levels are reached. In addition, Carson uncovered evidence that pesticides often become ineffective over time as new generations of insects develop immunity to them.

In summary, we need to recognise not only that the use of pesticides disrupts the ecological balance of nature and contributes to human and animal disease, but that through adaptation and change insects build up resistances to the technological tools meant to produce their demise.

In concluding this section, we see that one of the major factors contributing to global ADR appears to lie in human needs and desires. For example, increasing human population pressures (such as the need for human habitat and greater amounts of foodstuffs) and global demographic population shifts result in greater population density. Such factors place untoward pressure on an already stressed environment and opportune the rise of infectious disease. The satisfaction of immediate desires as well as the confusion between needs and desired (achieved principally through globalisation of the 'good life') compounds the problem as the global human population is led to greater consumerist practices. Pesticide usage is directly linked to both. This is because they are used principally to protect the human population against disease vectors *e.g.* mosquitoes and in the food agriculture industry as crop protectors. Yet, it remains important to note that the mechanism of resistance is locked in the biological evolutionary model of microbial adaptation and change. So, be it in plants or animals, the acquisition of the trait of resistance remains a constant. ADR is also enhanced through the practice of medicine, the subject of the following section.

Drug resistance and Medicine

Platt (1996: 1) tells us that human infectious diseases are:

> *... a basic barometer of the environmental sustainability of human activity. Recent outbreaks result from a sharp imbalance between a human population growing by 88 million per year and a natural resource base that is under increasing stress. The dramatic resurgence of infectious diseases is telling us that we are approaching disease and economic development in a wrong way. Governments focus narrowly on individual cures and not on mass prevention; and we fail to understand that lifestyles can promote infectious diseases ... it is imperative that we bring health considerations in to the equation.*

What human factors contribute to the spread of infectious disease? Morse (1995 a: 14) provides a comprehensive assessment:

> *Responsible factors include ecological changes, such as those due to agricultural or economic development or to anomalies in the climate ... human population demographic changes and behaviour ... travel and commerce, technology and industry ... microbial adaptation and change ... and breakdown in public health measures.*

Similarly, the Institute of Medicine's (IM) 2000 report on emerging infections does not categorise microbial threats by type of agent but rather according to *factors held to be related to their emergence*, again representing a subtle shift in the conception of disease as not due to a single agent but to a more multifactoral approach.[46] Reconception of disease as multifactoral is not new in itself, but it remains a global tendency amongst medical practitioners is to ascribe the cause of a disease to a single agent that falls within medical 'control'. This may be understandable on one level as shifts beyond this are often fraught with uncertainties. For example, in the discipline of environmental health while emphasis is placed on the relationship between human health and environmental factors, it is often difficult to address problems because of uncertainty in both toxicology and epidemiologic practices. One reason is that to determine that an illness is environmentally provoked one is obliged to understand an individual's *past exposure* to a harmful agent. In this regard, traditional medical examinations do not include histories of past exposure to adverse environments (Harte *et al.* 1991). Past or even current residence in proximity to a paper-manufacturing industry, contact with asbestos, lead paint, petroleum products and insecticides are examples of adverse environments.

Another factor is the uncertainty of what is referred to as the *dose-response relationship* (*ibid*). In cases of acute poisoning, for example, clinicians are quite adept at assessing how a body will respond. However, the problem of long-term low-level exposures to adverse agents is not generally available (Kroll-Smith *et al.* 2000: 10). For example, the exposure to tobacco smoke, radon and asbestos are known to 'cause' cancers. However, how these carcinogens interact to cause cancer is currently not known (*ibid*).

It is also quite difficult for clinicians to recognise links with environmental factors and a specific disease. This Vyner (1988: 62) refers to as 'diagnostic uncertainty'. Another obstacle is that not all individuals living in the same adverse environment will present the same symptoms. This is because our particular genetic make-up predisposes us to particular diseases. In the context of infectious disease and drug resistance there is also a strong environmental cause-component.

These factors combined (exposure histories, dose-response predictions, synergistic effects, valid etiologic models and diagnostic capabilities) result in the fact that most likely many patients presenting with diseases that may well be environmentally induced are not recognised as such. Thus, there is a tendency to de-emphasise the environmental component in disease, placing importance on other less contentious medically or scientifically recognisable factors.

In spite of these obstacles, with subtle shifts in disease classifications, old ways of conceiving diseases are replaced by new ones asking such questions as, 'What is obscured in this way of conceptualising disease ?,' 'What is brought into relief ?'. As Farmer (1996: 267) says:

> *Historically every major retrospective study on diseases has shown us that what was not examined during an epidemic is often as important as what was.*

This concept is echoed by Eisenberg and Kleinman (1981: 56):

> *The key task for medicine is not to diminish the role of the sciences in the theory and practice of medicine but to supplement them with an equal application of ecology and social sciences in order to provide both a more comprehensive understanding of disease and better care of the patient. The problem is not 'too much science' but too narrow a view of the sciences relevant to medicine.*

It would appear then that the borders between medicine, environment and human sciences should not be demonstrated as clear sharp lines, but conceived as woven strands of long and perilously complicated elements of all three areas of study. The borders of medical practice then become broadened linking misuse of antimicrobials to broader social and environmental concerns.

Yet, misuse of antimicrobials in medicine is one of the major contributors to global ADR. The American Society of Microbiology (ASM 1994) states:

> *Rapid increase in ADR by disease organisms is caused by the widespread and overuse of more than 300 antibiotics by the medical profession.*

The burden of infectious diseases is critical in developing countries, in which diarrhoeal and respiratory diseases in particular influence morbidity and mortality among young children (Murray and Lopez 1997: 1269-1276). While headway has been made concerning improvement of health in developing countries (WHO 1995; WHO 2000 b; WRI 1996-1997), the possibility that rates of ADR would rise with concurrent slowing of the availability of new antimicrobials was not factored into health projections (Hart and Kariuki 1998: 647).

The emergence and spread of antimicrobial-resistant bacteria is augmented in settings in which treatment is inadequate because of socioeconomic constraints on large and growing populations often living in conditions of crowding and poor sanitation. Much of the information concerning antibiotic resistance in developing countries is anecdotal, no two countries are identical in their use of antimicrobials and patterns of use vary from one region of a country to another (Kunin 1987: 270-285). Understanding antimicrobial use or misuse requires at least consideration of cultural perceptions of health and disease in the populations, the availability of antimicrobials, and the overall characteristics of the system of medical care.

In developing countries, the use of antimicrobials is to some extent limited to those who are wealthy enough to afford them (Bledsoe and Goubaud 1988: 257). Generally, private medical practitioners over-prescribe antimicrobials more commonly than do doctors working in government hospital facilities. This is partly because more antimicrobials are available in the private sector, but it is also linked to financial gain (Donovan 1974: 375). Private practitioners charge higher fees for their services, while at the same time, as in developed countries, the demand for antimicrobials appears to be higher among private patients. In developed countries, there is a complex relationship between the availability of product information to the public and the demand for pharmaceutical products (Barclay 2003). Whether from fears of litigation or simply because it is easiest to give in to the demands of the patient, doctors will often prescribe unnecessary antibiotics (CDC 2001; Coffman and Beran 1999: 5906-5910). In this way, second- or even third-line drugs are unnecessarily utilised in great quantities by the public (ASM 1999).

In developing countries, access to different types of antimicrobials as well as their general availability is linked to financial factors (Hossain *et al.* 1982: 403). Tertiary or secondary hospitals usually have a greater supply of more types of antimicrobials than do clinics. Clinics therefore tend to dispense more first-line drugs. Nosocomial infections, though, remain a point of major concern in the spread of ADR. However, if first line drugs prove to be unsuccessful in treating a patient (because of factors such as non-compliance or sub-compliance), because of ADR, he or she may end up being admitted into a higher level in a health system with a more complicated illness as well as spreading his or her acquired first-line drug resistance.

In addition, even when laboratory facilities are available doctors tend to treat patients with antimicrobial combinations before results are obtained (Korte *et al.* 1996: 428-432). Hart and Kariuki (1998: 649), for example, report that in the Rajbari district of Bangladesh a survey of medical practitioners (barefoot doctors) showed that they each saw an average of 380 patients a month and prescribed antimicrobials to 60% of them based on their presenting symptoms. A study from Malaysia revealed that Malaysians are consuming far more antimicrobials than is needed. This type of study has been repeated in many developing nations (CDC 2000 b). The results of the Malaysian study indicated that the abuse of antimicrobials was simply due to doctors over-prescribing them. Similar studies indicated that over 90% of private hospital patients who received antimicrobials did not need them (CDC 1999).

There is a further connection with ADR in the problem of 'dispensing doctors'. In South Africa, until recently and currently challenged, doctors have been allowed by regulation to dispense pharmaceuticals in their surgeries. According to the South African Parliamentary Bulletin (1996):

> *A study by the South African Pharmacy Council found that dispensing doctors charged more than three times more than pharmacists for medicines to treat patients with similar conditions. Another study of medical claims of more than seven million medical aid members found that dispensing doctors wrote out an average of 3.71 scripts, compared with 2.01 scripts for prescribing (non-dispensing) doctors.*

While it is claimed that doctors dispense solely for the convenience of the patient, it is reasonable to ask whether they would continue to do so if the profit they make on dispensing were to be taken away (van Bogaert and Ogunbanjo 2001). The pharmacist-doctor dispensing debate has a long history,[47] and it should be noted that paying for a consultation is not accepted in all cultures. In India and Pakistan, for example, patients do not generally accept paying a consultation fee, and the commonest way doctors generate an income is by dispensing (selling) drugs (Kunin 1987: 285).

Drugs are not only a problem in the confines of the doctor's rooms or the pharmacy. In most developing countries, antimicrobials can be obtained without a prescription, even when this is not legal (Nwokolo and Parry 1989; PAHO 2000). Pharmaceuticals are readily available on demand from drug stores, pharmacies, hawkers, road stalls, informal vendors, and so forth, and some people travel to developing countries for just this reason. The scope of this casual selling of drugs, mainly antimicrobials, stretches from Mexico (Bojalil and Calva 1994: 147-156) to India (Dua *et al.* 1994: 717-724) to the former Soviet Union to the African continent (Alubo 1985: 96). There are two major reasons for this type of distribution, one being profit on the pharmaceuticals sold, and the other the reality that in many developing countries such drugs are often not available in local hospitals and clinics (Goel *et al.* 1996: 1155-1161).

Traditional healers are not innocent of involvement, with reports from China and Nigeria pointing to the addition of 'orthodox' antimicrobials in remedies marketed as 'traditional' (Hart and Kariuki 1998: 648). It is correct to assume that these pharmaceutical 'vendors' have little knowledge of the medicine they sell, whether in respect of storage, recommended doses, side-effects, or contraindications (Barnett *et al.* 1980: 479-499). From a patient's perspective, where cost is often a problem, the advantage is that smaller quantities can be purchased – which of course provides the perfect sub-inhibitory milieu predisposing to selection of resistant bacterial strains.

Common cultural beliefs about disease include the notion that there is a pill for every symptom and that injections are more 'powerful' than pills (Haak and Hardon 1996: 621; Birungi 1994: 125-135), and the idea that a 'good' doctor always prescribes a large amount of medicine and even better gives an injection as part of the 'treatment' (Alubo 1985: 89-113). In these and many other ways community perceptions of disease treatment become integrated into local cultures, and pharmaceuticals become, to add to the title of Haak and Hardon's article (1996: 620-621), 'widely demanded, often misused and greatly misunderstood'.

If these problems were not enough, the quality of many antimicrobials in developing countries is often below the standards of international formularies (Barnett *et al.* 1980). In some countries, many antimicrobial drugs are produced locally. In India and Ghana, for example, there are over 80 different brands of the fluoroquinolone ciprofloxacin (*ibid*: 482). Regulations to control the actual amount of antimicrobials present in a marketed product remain inadequate. In addition, Bledsoe and Goubaud (1988) report that some drugs sold in developing countries do not contain the concentration of active substances stated on their labels even at the time of manufacture. These counterfeit drugs flourish despite some regulatory efforts (Land 1992: 192; Silverman *et al.* 1992). Counterfeit antimalarial tablets have recently provoked an international response, according to Markles (2003). Approximately 65% of the 751 instances of counterfeit pharmaceutical products reported to WHO by Interpol from 28 countries in the last 15 years were produced in developing countries (Couper 1997: S155). Many of these had little or no active ingredients or contained substitutes with less expensive alternatives, the latter having led to many deaths (Alubo 1994: 97-103). Drugs that do not comply with minimum standards can produce a sub-inhibitory concentration *in vivo*, which fosters the selection of resistant strains.

Because laboratory facilities to diagnose the disease agent are often not readily available in developing countries, diagnosis of infectious disease is done empirically and these diseases are usually treated with antimicrobials. This applies to enteric (intestinal) pathogens as well. For example, during a cholera epidemic in Somalia there was a case fatality rate of 13%, because the initially sensitive *Vibrio cholerae* quickly acquired plasmid-encoded resistance to tetracycline, ampicillin, kanamycin, streptomycin and the sulphonamides (Coppo *et al.* 1995: 355). Pathogens associated with respiratory tract infections have also become resistant in this way. Tracked from its origin in Peru almost a decade earlier, a pneumococcus highly resistant to penicillin emerged in South Africa in 1977 (Applebaum *et al.* 1977: 995-997). Multidrug-resistant pneumococci and pneumococci resistant to penicillin have spread globally. Many penicillin-resistant pneumococci are also resistant to chloramphenicol and cephalosporins (cefuroxime and ceftriaxone), thus limiting treatment options.

Plague is a re-emerging zoonosis caused by *Yersinia pestis.* It is usually treated with streptomycin, chloramphenicol, kanamycin, tetracycline or gentamicin (WHO 1996 c). In a recent epidemic in Madagascar, a strain emerged resistant to all known antimicrobials. If the resistance becomes established, it could render plague untreatable (Hart and Kariuki 1998: 650). Suffice to say that there are vast numbers of infectious diseases that we are now unable to treat successfully, ranging from nosocomial to sexually transmitted, to other human disseminated infections.

To add to the conundrum, the WHO in 1996(a) reported:

> *... there is an inherent conflict of interest between the legitimate business goals of manufacturers and the social, medical, and economic needs of providers and the public to select and use drugs in the most rational way.*

When a successful formulation of a medication comes onto the market, other companies usually cash in on it by producing a 'me too' product to acquire a market share. That is why in India, for example, the drug market has 70 000 available human pharmaceutical products compared with the WHO's list of 250 essential drugs. (In the case of antimicrobials specifically, 20 are listed). In spite of overall profit reduction in world markets, the pharmaceutical industry topped profitability according to Fortune 500's 2001 report (*Public Citizen* 2002: 1). The global market for pharmaceuticals was worth 22 billion USA dollars in 1993, and in 2001 reached nearly 40 billion dollars (*ibid:* 2). With the increasing prevalence of antimicrobial resistance, because the first-line antimicrobial now has a greater chance of failure, a patient must consume second- or third-line drugs in order to obtain a cure. Moreover, the more the phenomenon of ADR continues, the more money pharmaceutical companies will have to invest in new research and development – recouped from the need to purchase newer drugs.

Concluding Remarks

Overall, in this Chapter I have tried to show how drug resistance is enhanced by many complex and diverse human social factors. Some of these factors include the enhancement of ADR because of human population and overpopulation through the stress it places on the environment, the production, consumption and distribution of foodstuffs necessary to feed human populations, the divergent ways in which the practice of medicine conduces greater prescribing and dispensing of antimicrobials, the problems of compliance and sub-optimal consumption of antimicrobials, and the application of the same principle of ADR in pesticide usage. In addition, an important concern lies in the role of the pharmaceutical industry and their marketing practices, seemingly pointing to the 'business of business' as only being one of profit. Such considerations raise important ethical concerns because in a global perspective, the control or containment of drug resistance is problematic because of such human social factors. Global imbalances such as the distribution of wealth, diversity of cultural practices (including medical practices), divergent cultural perceptions of what medical regimens should entail, ease of trade and travel, wars, strife, the role of corporate multinational pharmaceutical industries in the promulgation (including decisions as to the production of therapeutic regimes based not so much on global need, but on global profit) – are only a few of the problems that make the prospect of global containment of drug resistance massively problematic.

Moral responsibility for increased drug resistance lies in many and various human failings such as broad economic global differences that contribute to unavailability of drugs to demands for unnecessary treatment to food production and social or cultural perceptions of disease and the role of drugs. If these factors were not enough, the third leg of the triangle contributing to global drug resistance points to another major concern, that of environmental changes.

Chapter 4: Environmental Changes

Hurt not the earth, neither the sea, nor the trees Revelations 7: 3.

Introduction

This Chapter focuses on the third leg of the triangle contributing to global drug resistance, environmental changes. Used in its broadest sense, ecological change plays a major role in the rise and spread of emerging infectious diseases and drug resistance. What changes in the environment contribute to the rise in ADR and EID? While new diagnostic methods can reveal diseases or drug resistances not previously recognised, some general factors may be included. These include host habitat disruption, soil degradation, warmer climates, changes in the ways foodstuffs are acquired, shifts in dietary habits, changing vegetation, waning water sources and loss of biodiversity. Each of these factors is vast and in the context of this dissertation, I will only be able to identify some of them in relation to the topic.

In this 4th Chapter, I will show some of the ways in which the stress placed on the environment results in changes. Interactions such as microbial adaptation and change combined with pressure placed on the environment result in rising emerging infectious diseases and the opportunity for the rise and spread of drug-resistant microorganisms. As an example, I will look at the rise of some new viral diseases showing that environmental changes such as deforestation and El Niño/southern oscillation (ENSO) result in modifications of the natural environment of plant and animals. Changes, such as habitat shifts, I will show, trigger microbial transformations. At the same time I will point out that new or different cross-species disease transfer may result when human populations require additional habitat and expand into virgin territory. To put it simply: Genetic changes plus human societal factors plus environmental changes equal evolutionary opportunities for microorganisms.

Trends in our modern world are placing intense evolutionary pressures on microorganisms. At the same time, we are providing ever-greater opportunities for infectious agents to emerge locally and move globally. The combination of ecological change, travel and transport of organisms is nowhere more clearly identified than in ADR. The selective forces of widespread antimicrobial use have led to the emergence and movement of populations of resistant microorganisms carrying the genetic information enabling them to elude our therapeutic and prophylactic efforts. Sometimes the microorganisms travel only from one hospital bed to another, as in nosocomial transmissions, but other cases involve far greater distances. Even the penumococcus is now globally widely resistant to penicillin, denying us use of one of the best broad-spectrum antimicrobials ever developed for treatment of this common but life-threatening infectious agent. Meanwhile lying in wait, as it were, is a huge genetic pool of microorganisms that are not currently notable human pathogens but are subject to selection in human and animal reservoirs and may subsequently transfer their genetic material to other bacteria of more direct importance.

As seen in Chapter 2, selection plays an important part in the evolution of microorganisms. Many types of microorganisms exist in nature. Some, as has been discussed, are classified as bacteria because they are without definite nuclei and are single-celled. Others have defined nuclei and other intracellular organelles, for example yeasts such as *Candida* and the fungi. Only some of these are pathogenic to humans. Viruses, on the other hand, are much smaller than bacteria; tens of thousands can exist within a single cell, and they depend on the host cell to reproduce. The major thrust of all microorganisms is energy – to live and multiply.

Microorganisms and Environmental Stress

On a micro level, genes, as we saw in Chapter 2, are not new creations in evolutionary terms. We now know that they appeared in species of bacteria naturally present in the environment long before they entered the bacteria that cause human disease. Some data suggest that the genes conferring resistance evolved from genes found in soil bacteria, the very microorganisms from which most antimicrobial drugs are produced. We know a great deal about microorganisms such as the bacteria, but we may have just scraped the surface. We recognise that with their fluid ability to mutate and change and with the increasing amounts of antimicrobials in the environment, small numbers of bacteria are able to persist and multiply. So we are now faced with increasing numbers and types of strains, along with new combinations of resistances that have developed in the span of only two generations (Levy 2002: 114). However and importantly, microorganisms other than bacteria use these same mechanisms to protect themselves, adapt and increase.

The origins and roles of viruses in nature are unknown. Some biologists argue that they emerged from evolutionary events that produced agents capable of living on entire cells (*ibid*: 32). Others point out the evolutionary association of humans and viruses. For example, as reported by Martins (2003: 8), Professor John Coffin in the Bernard Fields Memorial Lecture opening a recent HIV conference 'offered insights into how the HIV retrovirus has encroached on the human genome describing pro-viral remnants of retroviruses (Human Evolutionary

RetroViruses - HERVs) as molecular fossils pointing to the frailty of humanity against the repeated onslaught of repeated integration of novel DNAs. 80 % of the human genome currently consists of HERVs. Professor Coffin asked delegates, the intriguing question as to whether these fossil remains could ever come back and cause human disease.' Nonetheless, however their evolution, these viruses (like other microorganisms) are a part of human life and contribute to the balance of nature. If a cell population increases, so do the viruses that kill those cells. Eventually the cell population will decrease, providing fewer hosts for the viruses. And without hosts the viral population is doomed – and the balance of nature is restored. As we shall see, like bacterial microorganisms, viruses are able to adapt their structure (particularly their genetic structure) readily when they are placed under stress.

Besides our 'fossil pro-viral remnants' some latent or chronic viruses such as the herpesvirus or polyomaviruses coevolved with humans, but they do not often cause serious disease. Other viruses transmitted in nature only affect humans incidentally. These form a pool of agents that can potentially adapt to inter-human transmission, as measles did centuries ago and HIV has done more recently (Fenner 1970; Gao *et al.* 1999: 436-441). For example, Influenza A[48] is an avian virus that periodically generates mutants and reassortants that can readily adapt to human transmission (Hay 2001: 102).

On a macro level, many EID and microorganisms, such as the Hanta pulmonary virus, are doing what they have always done. This virus has incidentally infected humans because of expansion of human populations into the territories of its natural hosts (rodents such as mice, rats and prairie dogs). Assisted by garbage dumps in which to forage for food, water supplies and new nesting spaces, and in combination with changing weather patterns (principally warmer temperatures), the rodent hosts thrived and their reproductive cycle increased. At the same time, the increased proximity of humans to the rodents carrying the virus enabled increased cross-species disease transfer. So far, the Hanta virus has not mutated into another form. This is not the case with the Nipah virus.

Important factors in the initial outbreak of Nipah virus appear to have been the proximity of the fruit bat or flying fox which had moved into more suburban areas because of loss of habitat, to a newly modernised and intensive pig-raising industry, where the animals lived in crowded conditions (Murray *et al*. 1998: 23). The life cycle of the virus had previously been confined to the flying foxes, but it is hypothesised that the monoculture of pigs created the setting necessary for the virus to mutate into its current human-to-human infectious form.

Many viruses do not readily spread from person to person, but the ability of some to do so may be increasing, an example being the filoviruses (*e.g.* Ebola and Marburg viruses). Nosocomial spread, intermediary nonhuman hosts and possible plant infective sources have been suggested as agents in the spread of Ebola virus (Clegg and Lloyd 1995: 227).[49] However, the primary concern is environmental because although the gene sequences of both Ebola and Marburg viruses have been mapped, their source in nature has not been identified – failure to do so, and to terminate the paths of transmission, could give the viruses the chance to adapt genetically to interhuman airborne spread. So it is important to identify this link, on the other hand is it less important to identify the conditions under which these and many other diseases have emerged?

As we are aware, bacteria were the first disease-causing microorganisms to yield to modern pharmacological interventions. However, the 'old' diseases such as tuberculosis (TB) and cholera continue to be a problem. Some have been identified as emerging or re-emerging due to the rise of antimicrobial-resistant forms, such as multi drug-resistant tuberculosis (MDR TB) and vancomycin-resistant enterococci (VRE) infections (*Enterococcus faecalis* or *E. facium),* the latter most often being nosocomial. Other microorganisms are defined as resurgent owing to changes in their geographical distribution or an increased incidence of infection, often following a decline in public health systems.[50]

From common human pathogens such as *Staphylococcus areus* to new strains of *Escherichia coli* (O157: H7 strain) and *Vibrio cholerae* (El Tor strain), the world of microorganisms is in constant flux. Yet environmental changes remain a major focus and there is perhaps no better example of how changes in the environment affect microbial life than the *V. cholerae* model.

Algal blooms, global warming, urban slums, ENSO and tetracycline appear to be unrelated phenomena. However, a growing body of evidence suggests that total convergence of these factors may be responsible for a deadly cholera epidemic that ravaged South America from 1991 to 1995, killing an estimated 11 000 people and causing disease in over a million more. This epidemic took health officials by surprise, as for over a century cholera had been absent from South America. At the same time, outbreaks in Africa[51] (1991) and in the former Soviet Union (1994) suggested to epidemiologists that a new pattern was emerging in cholera spread (Coppo *et al.* 1995: 351-359). Typically, cholera is a disease associated with unhygienic living conditions, often in direct association with poverty, because the poor often lack sanitation and water infrastructure (Speck 1993: 642-643).

Exclusively a human pathogen, the cholera bacterium is transmitted from one person to another, usually via food or drinking water contaminated with human faecal material. However, its optimal habitat is moderately salty waters of coastal estuaries, although it can live in pure seawater as well. Its survival in fresh water is dependent on the concentration of sewerage. The first outbreak in a coastal community did not surprise researchers, in view of the ecological preference of the pathogen. But it was not an isolated event. Almost simultaneously, coastal cities in Peru, Columbia and Chile – over 100 km apart – also experienced severe cholera outbreaks. That cholera had a potential to occur was not debated. For example, when the epidemic struck the Peruvian capital of Lima, 70% of the city's drinking water was drawn from the sewerage-choked River Rimac. The rapid urban population growth and the city's concurrent failure to keep pace with growing settlements (40% of the population had no access to clean water or sanitation facilities; this represented about 5 to 6 million people) as well as reliance by the many slum-dwellers on the river for food and water contributed to the potential. To add to all this, Peru's most popular foodstuff, *ceviche*, consisting of raw fish and onions marinated ('cooked') in lemon juice, played a further role in the cholera epidemic. The raw fish contained *V. cholerae* that if cooked by conventional (heat) methods, then it would have been rendered harmless.

Overcrowding and a lack or breakdown of public health infrastructures, basic knowledge and practice of hygiene create conditions ideal for a cholera outbreak, situations that can be duplicated in many parts of the world today and represent nothing particularly unusual. They characterise daily life for millions of our world's inhabitants – many living in areas free of cholera. So what additional factor or factors contributed to the dramatic re-emergence of the disease?

According to Colwell (1996: 2025-2031), in the late 1990s, it was found that certain species of marine zooplankton serve as hosts for *V. cholerae,* giving rise to speculation that the microbe may have adapted to lie dormant in coastal areas and suddenly emerge with algal blooms. Observations that cholera outbreaks in Bangladesh frequently coincide with algal blooms in the Bay of Bengal provide anecdotal support for this hypothesis. The cholera-plankton connection would also explain how the pathogen was able to travel long distances along a coastline or across oceans, spreading infection from Asia to South America to Africa simply by riding along inside planktonic cells drifting in ocean currents (and indeed perhaps assisted by ocean tankers carrying contaminated bilge water). Further, the heating of ocean surface waters, for example in the ENSO phenomenon, encourages massive phytoplankton blooms. (It is notable that the South American cholera epidemic occurred in tandem with the most prolonged El Niño episode on record.)

With warming waters, blooms of zooplankton quickly follow, especially in shallow coastal waters fertilised by the run-off of nutrient-rich sewerage. Contaminated water further feeds back into the biosphere because of absorption and source-destination flows, in itself contributing to global warming and ENSO's such as El Niño and thus results in increased algal blooms (Colwell 1996: 2026). These blooms, according to Colwell (*ibid*: 2029), 'seem to activate the *V. cholerae* 'hitchhikers' causing the potential pathogen to revert to its actual human-infectious state'. This represents a continuous circle of causes and effects and so if the cycle is not broken, we could be in for some unhealthy surprises.

The problem of cholera becomes still more complex when we consider its treatment. Lodging in the human intestinal tract, the microorganism produces a toxin that causes rapid onset of cramps, vomiting and severe watery diarrhoea. The treatment of choice for cholera is rehydration via oral or intravenous solutions to replace lost body fluids. In endemic areas, parenteral vaccination against cholera is not recommended because it offers only partial protection (Merck 1995: 111). The antibiotic of choice in cholera treatment is tetracycline or its derivative doxycycline.

Specific to humans, rapid spread of tetracycline-resistant *V. cholerae* strains has been identified (WHO 2000 a). Furthermore, a USA study by Raloff (1998 a) identified that bacteria naturally present in some rivers and coastal estuaries showed 30-40% resistance to tetracycline. In addition, more than 80% of these tetracycline-resistant bacteria were immune to one to six additional antibiotics (*ibid*). Rue *et al.* (1998) confirm these findings. Wild water birds harbour and transmit resistance to tetracycline as well as to other drugs (Eichorst and Tischler 1999; Ash *et al.* 1999; ASM 1999). Tainted water probably explains the resistant bacteria found in wild geese. Of course, wild geese are migratory and transmit their resistance to other rivers and estuaries, and eventually to humans. Domestic animals, however, are according to Novak (1998: 108) the major source of tetracycline resistance.

Farmers use animal feeds containing low dosages of antibiotics, particularly the tetracycline family, to improve growth. Inevitably, some of the bacteria in the animals, in manure-tainted fields and in water supplies evolve to coexist with tetracycline, as well as other drugs. When such animals are killed for food, the cycle potentially continues in the human host who has consumed the meat, as was shown in Chapter 3. Ultimately, an awareness of the problem of EID and ADR requires an understanding of the interrelatedness of life systems.

Directly implicated in the rise of EID is the environmental concern of global warming. As we have seen, it has a direct impact upon possible vectors, habitats, reproductive patterns, food supplies, transport, dissemination and so forth.

According to Epstein *et al.* (1994), conditions conducive to infectious disease transmission may actually be underestimated since they are based on changes in average temperatures, and global temperatures are warming.

But how do we know what to measure, how to measure and the importance of all the factors involved in disease assessment? Modern epidemiology studies diseases in human populations in terms of agents, exposures and risk factors. Generically these include microorganisms, chemicals or nutrients, and anthropometric, physiological and genetic features, as well as behaviours, mental states, race and socioeconomic status (Wing 1993: 222). But problems in interpretation of EID as well as exposure-related illnesses primarily focus on issues of measurement error, differences between groups being compared and questions concerning precisely what should be measured. The problem is that outcomes generally reflect experience with diagnosis rather than aetiological inquiry. For example, our lack of understanding about mechanisms of radiocarcinogenises means that the problems of measurement are secondary to the more fundamental problem of knowing what to measure (*ibid:* 219).

In a similar way to the problems of measurement and comparability, questions about the biological basis for models in disease and exposure are modern epidemiological concerns. This means that technological concerns – how can x be measured? – may lead epidemiologists to emphasise increasing control over measurement and extraneous factors that can distort the exposure-disease relation, while concerns over the issues in the exposure-disease association focus the discipline's theoretical attention on pathological processes of individual organisms. Because control over measurement and extraneous factors is hindered when investigations are entrenched in complex social and historical situations with their implied power structures, this combination of influences supports the movement of the practice of medicine, including epidemiology, away from engagement with issues of social theory, population biology and human ecology and towards more fundamental and traditional biomedical approaches.

Efforts to articulate alternative medical practice may be connected by attention not only to the historical contexts of disease phenomena but to the science of ecology, which must be included as a vital source of enquiry. This is naturally in direct opposition to the dominant assumptions that there are ahistorical and value-free independent exposure-disease and human health-environmental health associations. Environmental changes are critical to an understanding of the emergence of new infectious diseases and drug resistance. I will show the necessity of this in the next section with a few examples.

Some Impacts of System Destabilisation

Local as well as global disturbances in land use or agricultural practices may lead to changes in vector or reservoir circulation. Abuse of the soil and land resources is disrupting ecosystem sustainability in many parts of the world today. These actions influence the rise and dissemination of EID and ADR. It is hypothesised that resistance genes present in soil bacteria somehow escape and, after passage and evolution in other microorganisms, enter those that have direct contact with humans and animals (Levy 2002: 79). This theory is based on research showing that prior to the development and clinical use of antimicrobials, resistance genes were found in animal and human populations that had no contact with antimicrobials.

Two points are significant: the first is that while antimicrobial use or misuse does not create antimicrobial genes *per se*, such practices do contribute to the increase of the numbers of the trait of drug resistance that may be acquired by microorganisms. The second is that in introducing large amounts of antimicrobials into the environment we have altered the ecology of microorganisms. Because the trait of drug resistance is a positive adaptive trait, and because it is mutable, we offer microorganisms a selective advantage. The combination of resistance genes and transferable plasmids has enhanced the spread of ADR and EID. Together these present grave problems in clinical decisions about disease treatment and prophylaxis. But there are many things we do not know. We do not fully understand how for example, soil abuse contributes to the rise of EID and ADR. And we do not know how much drug resistance exists in nature. In the following, I will show the relation of soil degradation to EID and ADR.

The International Soil Reference Center estimates that 10% of the earth's vegetated surface is at least moderately degraded, while about 22 million acres are so severely degraded that their original biofunctions have been completely destroyed (WRI 1996-1997). In the foothills of the Himalayas 38% of the land has been abandoned because of topsoil erosion (Stutz 1993: 676). In central Chile and Nigeria, soil erosion is endangering any foreseeable increases in crop production, endangering half the country's peasant farmers (Faeth 1994: 312). In China, half or more of all arable land has already been lost to erosion and encroaching deserts (Real 1996: 89).

Soil is not a sterile homogeneous material. In its natural state, it is a living environment teeming with biological activity functioning in a complex ecosystem (Wilson 1995: 141). The types of life found in the soil are many, and the numbers of organisms are enormous. Nadakavukaren (2000: 144) estimates that a quarter teaspoon of a fertile soil (about one millilitre) contains 50 nematodes, 62 000 algae, 72 000 protozoa, 111 000 fungi, 2 920 000 actinomycetes, and 2 528 0000 bacteria. If that were not enough, a handful of such soil may also contain other forms of life such as earthworms, plant roots, insects and mites. Such organisms serve a variety of functions in the soil ecosystem. These include recycling of energy, carbon, and plant nutrients, water-nutrient absorption, soil recycling and nitrogen fixation, plus decomposition of chemical materials and other toxic substances.[52] Importantly, they also serve in disease transmission as well as disease control. Included in such processes are organisms such as bacteria in symbiosis with legume plants, cyanobacteria (blue-green algae), and some actinomycetes.

Through their digging, organisms that burrow through the soil mix it. Subsoil is brought up and mixed with topsoil, recycling the clays and minerals that are released from rooting zones. Microorganisms and organic matter produced by soil organisms are important in the formation of soil structure. Humus and exudates of roots and microorganisms act as a 'glue' holding soil particles together and fungal hyphae bind such particles. The surfaces of bacteria carry a net-negative charge, similar to clay, and have a similar effect on soil structure. Disruption in this process at any level results in disruption of the soil ecosystem. Any stress placed on the system results in conditions that will predispose to the microbial adaptation and change necessary for survival.

The most significant cause of soil degradation is overgrazing, the excessive pressure placed on the soil by cattle, goats, and sheep (WRI reports 1996-1997; 1998). For example, in India the condition of more than half of the land now regarded as wasteland can be attributed to grazing by goats prior to their slaughter. According to Tobias (1998: 75), there is no political will to control the goat population.[53] Under natural conditions, grasslands adapt well to a moderate level of grazing. For millennia herds of bison, antelope and so on have been the dominant animals in the grassland biome, evolving and adapting in conjunction with the native plants upon which they depend. However, when farmers or livestock managers introduce different types of grazing animals foreign to the native plant communities, the resulting pressure on that community destabilises the ecosystem. First, there is a decline in population of those plant species unable to adapt, and as they disappear, species able to tolerate heavy grazing are relieved of competition and expand to fill the vacated niche. At the same time, the loss of natural species results in an overall reduction in the biomass, height, and total grassland cover. When overgrazing persists, even the more resistant native plants will be unable to withstand the pressure and give way to invader species inferior in nutritional requirements for grazing animals. Eventually, because of their low appeal, invader plants such as weeds are disregarded by the grazing animals, trampled and reduced to sparse land coverage. At this stage, the effects of wind and water on the poorly protected soil results in barren mud flats or rocky hillsides devoid of any plant community (Wittaker 1975: 44).

This has relevance to our discussion, as soil degradation and changing wind patterns can mean that the microorganisms present in soil are more easily distributed into human populations. As an example, in the Kano district of Nigeria in 1996 an infamous research project fell under global ethical disapproval when a team of USA researchers tested an oral form of the drug Troval on about 200 children suffering from bacterial meningitis (Stephens 2000). The pathogen (*Neisseria meningitidis*) is commonly found in soil. Because of increasing population (the Kano district has about 5 million inhabitants) they required more food. Thus, this resulted in increased numbers of grazing animals, particularly goats. Poor grazing practices resulted in more desertification. The shift in global weather patters resulted in more sandstorms than usual in 1996. The degraded soil blew with the winds and spread the bacterium resulting in this particular bacterial meningitis epidemic. And it should be added that these factors contributed to other epidemics as well as different illnesses attributable to this bacterium (Kymail 2003). In this way we see that disruption of the micro and macro content of the soil ecosystem combined with other environmental changes advances greater microbial spread.

Another example is found in global warming for global warming matters significantly when we recall the marvellously versatile microbial adaptive capabilities:

> *The inhabitants of planet earth are quietly conducting a gigantic environmental experiment. So vast and sweeping will be the impacts of this experiment that, if it were brought before any responsible council for approval, it would be firmly rejected as having potentially dangerous consequences. Yet, the experiment goes on with no significant interferences from any jurisdiction or nation. The experiment in question is the release of carbon dioxide and other so-called greenhouse gases to the atmosphere.* (Broecker (1987) quoted in Nadakavukaren 2000: 424)

Broecker's admonition of the consequences of a human environmental 'experiment' stands in contrast to Shakespeare's description of glorious air:

> *... this most excellent canopy, the air, look you, this brave o'er hanging filament, this majestical roof fretted with golden fire ...*

What is the problem with the air and how might global warming be related to both it as well as influencing EDI and ADR? This 'excellent canopy,' 'brave o'er hanging filament,' 'majestical roof' has, in chemical properties, existed in roughly the same proportions over several hundred million years as nitrogen (N_2), oxygen

(O_2), argon (A_r), carbon dioxide (CO_2) and trace elements.[54] These, like microbial life, are in a continuous state of flux. They react with continents and oceans to form weather patterns in a constant process of renewal and recycling. The atmosphere on earth provides the major source of certain chemicals necessary for life on this planet. Atmosphere controls the earth's surface environment by regulating both the quality and quantity of solar radiation that enters and leaves the biosphere.

Early humans made no significant changes to this natural process. However, later our human technology resulted in atmospheric imbalance. The major factors attributed to contemporary human influences are (1) the release into the atmosphere of pollutant gases and particles not usually present there in significant amounts, and (2) changes in the concentration of natural atmospheric elements (Nadakavukaren 2000: 417). Because the earth is a closed system, every element that goes into the system, although it may circulate and change form, nonetheless remains within. The consequences of making the sky a convenient dumping ground for volatile wastes should be obvious. 'Vanishing into thin air' is a physical impossibility.

There are two important issues concerning human activities in relation to the earth-atmosphere system. The first is depletion of the ozone layer. Ozone, although a rare atmospheric gas, is vitally important in protecting life on earth from the ultraviolet rays of the sun. In the early 1970's atmospheric chemists identified that certain pollutant emissions into the atmosphere had the potential to disrupt the atmospheric chemical equilibrium and thus the integrity of the ozone layer. A second major concern is the rising levels of atmospheric carbon dioxide (CO_2). The prospect of global warming because of CO_2 induced enhancement of the greenhouse effect is one of the most pressing and politically charged global environmental issues.

Levels of atmospheric CO_2 have been rising since the dawn of the industrial revolution because of the consumption of fossil fuels to power society. When fossil fuels are burned, one of the primary combustion products released is carbon dioxide. Long ago, excess carbon dioxide, for example released through natural volcanic activity, was gradually absorbed by the oceans and eventually incorporated into carbonate rock, or photosynthesised by plants into a neutral element. The volume of CO_2 now released exceeds the capacity of earth to reabsorb it naturally. Since 1950, annual carbon emissions worldwide have risen four-fold, reaching a high of 6.3 billion tons in 1998 (Brown *et al.* 1998: 67).

The cumulative result is called the 'greenhouse effect'. In the same way as the glass in a greenhouse permits light to enter but prevents the escape of heat, thereby warming the air within, so the absorption of infrared ground radiation by CO_2 and its subsequent re-radiation back towards earth helps to maintain an average global temperature of 15^oC . Without CO_2 in the atmosphere, the earth's surface temperature would fall to about 0^oC, making life as we know it impossible. The process of global warming is an alteration in the global energy balance sufficient to cause a 2^o increase in the world's temperature, relative to its 1990 value, before the end of the 21^{st} century (Nadakavukaren 2000: 427). And if the problem of excess CO_2 was not enough, there are other gases contributing to the greenhouse effect such as methane (CH_4), nitrous oxide (N_2O), chlorofluorocarbons (CFCs), sulphur hexafluoride (SF_6), perfluorocarbons (PFCs) and hydroflurocarbons (HFCs) (Houghton and Jenkins 1996: 62).

The impact of the greenhouse effect is evidenced in, for example, the arrival of spring in the far North earlier than in the 1970s, the melting of mountain glaciers in Switzerland and other Alpine regions, the global rising of ocean temperatures, an increase in air temperature in the far North, sea-ice melting due to increases in air and water temperatures, and distributive patterns of plant and animals. There are already noticeable changes in the migration patterns of birds and butterflies, but more troubling is the spread of certain disease vectors into areas where they have never been previously identified. This is why the classification of EID includes 'old diseases occurring in new places'. For example, malaria-carrying mosquitoes have appeared in some mountain ranges in South America (Martens and Hall 2000: 105).

The cumulative result of global warming will include, but not be confined to, diminished crop yields, heat-induced declines in animal fertility, migration of insects to other regions, shifts in the balance of pest and predator species, loss of biodiversity, shifting weather patterns creating heavier monsoon seasons, for example in India, less precipitation in Northern America, and global rises in sea temperatures and levels. It is anticipated that Egypt may lose 15% of its arable land to encroaching seas by the end of the 21st century; in Bangladesh a sea level rise of even three feet will inundate one-sixth of the country's land area (Nadakavukaren 2000: 434). The warming effect of oceans will exacerbate hurricanes and storm surges, affecting three-quarters of the world's population living within 60 km of all coastlines (*ibid*). This disruption[55] will unsettle and displace human, animal and plant populations, opening avenues for greater microbial adaptation and change, and causing migration, war, civil strife – all conducive to disease and drug resistance.

In warmer climates, the vectors of arboviruses will flourish (McMichael 1993: 144; McMichael *et al.* 1998: 475-488). However, since there are many microorganisms that we still do not know of (to say nothing of the potential 'old' ones have for microbial adaptation and change), the further role of 'activation' by climate changes remains a worrying unknown. What is known is that climate has affected the timing and intensity of disease outbreaks throughout history. Yet modern technology, particularly the high volume burning of fossil fuels such as gas, oil and coal, is probably responsible for changes of historic importance in the global climate, and although this is common knowledge it appears that intervention is evaded by politicians whose scope of vision tend not to extend beyond the short-term economic interests of their own countries. Meanwhile, temperature increases expand all vector niches dependent on higher temperatures for breeding and substance. For example, in Sweden the tick population is spreading northwards, probably owing to higher temperatures in these regions, while concurrently resistance to doxycycline, the treatment of choice for tick-bite fever, has been reported (Witte 2000: 324).

From the 1980s, there has been a resurgence of malaria in Latin America and in central and east Africa, corresponding directly with global warming and resultant increases in insect populations (Coluzzi 1994: 223-227). A widespread and accelerating retreat of tropical summit glaciers, plant growth at higher altitudes, a diminution of the freezing zone by 150 metres and the acceleration of global warming since about the 1970s all bode ill for further climate change that will facilitate disease expansion[56] and new EID. We seem to have a theoretical grasp of the possible consequences of global warming and other specific environmental changes, but we do not know the full extent of ADR or its ramifications as far as the environment is concerned, the subject of the following section.

Of Woodmice and Badgers

I earlier reported the finding of drug resistance in coastal waterways and rivers as well as in some members of the rodent family living in the wild. These woodmice and badgers had not been exposed to ADR. The connection between water and animals is not difficult to find. Animals in the wild frequent water sources for a variety of reasons, and if water sources contain drug-resistant microorganisms, the animals will ingest them when they drink. The same principle applies to animals and birds that bathe or swim in such water (to say nothing of marine life), and to birds and animals that ingest drug-resistant microorganisms in grains and other foodstuffs.

Woodmice and badgers carrying drug-resistant microorganisms potentially serve as reservoirs for spreading them to other nonhuman creatures that may become exposed to their urine and faeces. They may of course also continue the cycle by transmitting the drug-resistant gene to their progeny. Rodents such as mice and rats live about a year. They therefore mature and reproduce rapidly, have short gestational periods, and have from four to twelve pups per litter, averaging four to seven litters a year (MacDonald and Barrett 1995: 154). We are already aware of the reproductive potential of microorganisms. So, since ADR has been identified in certain woodmice and badgers, and is a threat to human health, a programme of control or eradication might be indicated. This is a typical public health (if not human) response. However, all woodmice and badgers would have to be destroyed, as it would be impossible to identify only those carrying ADR. Confinement or quarantine would be impossible. But perhaps we should ask the question, would it matter if all woodmice and badgers were exterminated if they are a threat to human health? This is important because traditional medical or public health perspectives tend to dismiss the whole, particularly when a human population might be at risk.

Professor Graham Bell, publishing in *Science* (2001: 2413-2418), presents a hypothesis he calls 'the neutral theory of macroecology,' a term he uses to refer to a particular approach to biodiversity. In it, he makes the claim that since there are many biologically similar species in ecosystems the individual components of an ecosystem are not quite as necessary to the continuing health of the whole ecosystem as some ecologists argue. For example, the most common food source for birds such as owls, falcons and hawks is rodents, of which these birds need a constant supply to survive. Bell asks if it matters to owls, falcons, or hawks if their food source is only, say, the European wild rabbit, as opposed to the order *Rodentia* as a whole? The answer is, probably not. According to such a neutral theory of biodiversity, a case could be made for the extermination of woodmice and badgers. However, we would have to set aside the argument that they provide benefit to the biosphere by, for example, burrowing under fields and ventilating the soil, thus improving soil aeration and water-holding capacity. Other rodents could conceivably take over this function – or other species for that matter, for earthworms play a similar role. Something in his reasoning makes sense, but the problem is that we do not know the limits of natural biodiversity. At what point does biodiversity fail, if in fact it does?

Bell does not make the claim that his hypothesis is correct; he is only raising the issue to stimulate thought and further study. Indeed, he puts forward the best argument against his hypothesis, the argument of biological heterogeneity (versus homogeneity). For example, McCabe (2001: 3) explains how a team examined pond scum (bacteria) in a laboratory setting. Their overall aim was to gain an understanding of why there is more biodiversity in some places and less in others.

Initially the bacteria showed different genotypes, different properties and different functions, *e.g.* some clung to the sides of test tubes, others settled in the water. Then their environments (the type of water used) were changed. It was noted that in a heterogeneous environment (mimicking environmental diversity) the wide diversity of existing life forms flourished. However, in a homogeneous environment, a single type eliminated all others. In other words, greater biodiversity leads to greater productivity in plant communities, greater nutrient retention in ecosystems, and thus greater ecosystem stability.

What are the implications for woodmice and badgers carrying the ADR microorganisms? Holding to the theory of biodiversity, it would imply that they should not be exterminated.

Importantly though, the example points to an unanswered question concerning the extent of drug-resistant microorganisms existing in nature. To that question, we have no answers. What we do know is that while drug resistant microorganisms are present in the wild, they are not evenly distributed. In addition, ADR appears not to be readily discharged from the environment – as Levy (2002: 292) puts it, it is a 'slow environmental loss'. There is no doubt that its extent globally will never be known, because many countries do not have the infrastructure to measure possible source levels. The amount of drug resistance in nature matters qualitatively and quantitatively as a predictive factor for monitoring and surveillance. In another sense, it does not matter, *for if it is detected in nature anywhere, it has the potential to become present everywhere,* as I noted earlier on pages 68, 69 and 75 with reference to a report from O'Brien (2002).

Even if we hold this model to the idea of 'protecting only human health,' the fact that we have no answers is critical, and this is the danger. When the antimicrobials were first discovered to be beneficial to humans, the model of evolutionary microbial behaviour was, despite some warnings, not fully understood, internalised or applied. These drugs became 'quick fixes' in medicine and big business in industry before the mechanisms of resistance were even beginning to be unravelled. As I have shown, pathogens arise *de novo* very rarely, if at all. Like ADR, the microorganisms causing EDI are already present in the environment. To understand their emergence one must study not only the causative organism but also the social, economic, political and environmental factors influencing their ecological niches (Wilson 1995).

Concluding Remarks

Life systems connect. In examining EID and ADR in relation to environmental changes, there is no way to separate human health from that of the health of the environment. From the well-known practice of miners carrying canaries to test the air of mineshafts, to the writings of Engels, Orwell and Virchow, to black lung disease and asbestosis, to the millions of microorganisms living in the soil ecosystem, it is obvious that a direct relationship between all exists (Smith 1981: 343-349; Brodeur 1985).

It should be mentioned that aspects of environmental justice focus considerable attention on the intersection of medicine, techniques of environmental measurement, and social action (Kroll-Smith *et al.* 2000: xi). However, one of the problems in practice is that nowadays when epidemiologists study disease, a range of social, economic, and political norms are usually included in the final assessment to the exclusion of highlighting system-connectiveness. For example, in the recent SARS epidemic, public attention focused on the role of the Chinese public health system, its relationship to the type of government in place, the possible global consequences of wider spread, the loss of revenue from tourism and business, and so forth. That SARS was most likely a product of cross-species transfer was recorded and accepted without hesitation, and since it appears 'contained' it has receded from public interest. Questions as to why and how it may have arisen may be asked about it as well as numerous other EID (*e.g.* Ebola and Marburg) and ADR.

But it seems that we do not want to ask such questions, perhaps because the answers might yield more questions, uncomfortable questions about ourselves and our relationship with the environment. That questions are averted, perhaps deliberately, when medical issues such as biospheric chemical changes in disease inducement or the epidemiology of toxic waste is evaluated, is a little easier to identify. For social factors appear to be diluted if the claimed contaminant comes from commercial, industrial, or military organisations, implicit bases of power. It seems to me that a similar mode of presentation is given to ordinary people when it comes to EID and ADR. While governments and health care systems overall do tend to acknowledge some of the social causes of infectious disease, they either quite readily dismiss or fail to call to attention any environmental contaminants or environmental stress caused by human social, cultural, political and economic practices. This results in many infectious diseases being labelled as 'contested'. Even when some diseases have direct and proven links to environmental changes, it seems that because they are also linked to the production, distribution, and consumption of goods the very reasons for their existence are disputed or avoided.

We are indeed the 'Apprentices of Sorcerers' (Dubos 1959: 13). In the face of real or perceived danger from EDI or ADR, we tend to act on what we believe to be a single causative agent, trusting that its elimination alone can elicit a 'cure'.

In this Chapter, I have tried to show that links between the environment, environmental stress, emerging infectious disease and drug resistance remain, no matter how disputed or avoided; and that it is erroneous, even dangerous to believe otherwise.

Introduction to Part 2: Moral Responsibility and the Problem of Global Drug Resistance

In the context of infectious disease and drug resistance I began Part I with the idea of a triangle, each side representing one of the three major contributors to the rise in infectious disease and drug resistance microbial adaptation and change, human social factors and environmental change.

To understand their connections, I have tried to show that we must understand the evolutionary properties of microorganisms as fundamental to understanding the problem of global drug resistance. Importantly, I have shown that measured in centuries or millennia, pathogenic microorganisms evolve, they do not appear spontaneously. Usually, they have undergone some periods of development, or have evolved within a nonhuman host. For example, they may live symbiotically as non-host-threatening in a nonhuman host. The shift from nonhuman to human host is mainly due to environmental stress (Lederberg 1997: 422). Because of interactions between human populations and commensal microorganisms, the type and frequency of ADR in any particular organism may differ greatly between geographical locations. However, once antibiotic resistance is established in an organism, because of the ease of human travel, human interaction and environmental changes, resistant strains spread with greater or lesser speed worldwide (Garrett 1994: 588). The idea of the connectedness of such factors is a vital consideration.

As has been discussed, the scientific basis of ADR lies in the principle of a biological-evolutionary model. It is applicable in all circumstances, from human antimicrobials to plant pesticides. In the former, for example when health care workers give some antimicrobial treatment to many patients rather than optimal or full treatment to a few patients (EthicSA: 2001), drug resistance develops. Concerning the latter, the general biological phenomenon of drug resistance applies because resistance develops in all insect populations upon which the pesticide comes into contact.

Economic factors come into play in the phenomenon of ADR as well. For example, patients in poor countries may not be able to afford the cost of complete treatment regimens so they purchase and consume inadequate amounts. It is however, not confined to the world's poor but straddles patients from all class and educational strata. For example, those who demand drugs when there is no need for them (over-use), who take drugs inappropriately, who refuse compliance, who fail to complete regimens, all factor into the global problem.

Industrial practices are also implicated in the spread of ADR. In a shift from older marketing methods, pharmaceutical companies are now actively involved with the mass marketing of their new products beyond doctors and veterinarians to the public by way of the Internet, newspapers and magazines (Barclay 2003). Through advertising, pharmaceutical companies deliver information, claiming that their objective is to make the public more knowledgeable about a particular condition and its corresponding treatment (*ibid*). This results in greater demand, bringing about greater consumption (needed or not), and thus is a major contributing factor to global increase in antimicrobial demand and abuse.

Drug resistance will change the current practice of medicine. Some examples of this follow: In 1952, penicillin cured nearly all staphylococcal infections; by 1982, it could cure less than 10% of them (Brock 1990). Gram-positive bacteria have emerged as important causes of hospital- and community-acquired infections

(Fluckiger and Widmer 1999: 121-134). Health care professionals from South Africa planning to do locums in The Netherlands are now asked to submit on-site swabs from designated body areas to check for drug-resistant strains of *S. aureus*. In addition, as an example of the grave concern about spreading ADR, it is currently health care policy in The Netherlands that no patient may be admitted to any of their hospitals if the patient has been hospitalised outside the country, unless they are proven (by laboratory examination) not to be a carrier of methicillin-resistant *S. aureus* (van Bogaert personal communication 2003).

The Centers for Communicable Diseases (CDC 1997) suggest that full resistance of *S. aureus* to all available therapies may soon develop, thus limiting the usefulness of vancomycin, one of the latest 'front-line' drugs. Recent data from the National Nosocomial Infection Surveillance System identifies *S. aureus* as the most common cause of nosocomial pneumonia and the second most common cause of bloodstream infections in the United States (NNIS 1998). This trend is complicated by the increasing prevalence (from 2% in 1974 to as high as 64% in recent surveys) of methicillin-resistant *S. aureus* among nosocomial isolates (Maranan *et al.* 1997: 849). Increasing antimicrobial resistance is found even among entercoccus, the second most common nosocomial infection (Dickinson 2000). Doctors and other medical practitioners will be obliged to face many medical cases in which currently recommended drug regimens are ineffective. This has profound implications for the practice of medicine including societal perceptions of disease.

As previously mentioned, environmental changes, particularly global warming is strongly implicated in the rise of EID. For example, night and winter temperatures are, in general, key to the range and activity of mosquitoes and to the infectious agents they can transmit (such as West Nile virus and malaria).

However, since 1950, global warming has increased at twice the overall rate and faster still at high latitudes and high altitudes (Epstein 1995). One result is increased global flooding. Floods often precipitate 'clusters' of mosquito-, water-, and rodent-borne infectious disease outbreaks such as malaria, dengue fever, Rift Valley fever, cholera, cryptosporidiosis, rodent-borne leptospirosis and plague.

Genetically modified crops add to the conundrum, such as genetically modified cotton. This reduces a particular pest threat in the short term, but stresses the natural environment in the longer term because additional chemicals will be needed to control other newly evolving and thus competing insects (Levy 2002: 112). In addition, imprudent and over-use of chemicals and pesticides have direct implications for the well-being of humans, animals, plants and insect populations.

In developing countries, EID and ADR share a relationship with the degradation of the earth's resources, for example the burning of fields, fossil fuels, wood, coal and charcoal. Not only does this contribute to poor health by weakening human resistance to infectious diseases, but deforestation causes erosion, leading to drought, famine, and the introduction of disease into the human population because of habitat change.

Global movements of people, including populations displaced in war and civil strife, known and unknown vectors, the increased use of antibiotics in stock feeds, and changing climates are some examples of the factors contributing to the global spread of pathogenic microorganisms, viruses, fungi, and so on (WHO 1997).
Human population growth, density and consumer practices are strongly implicated in increased ADR. For example, as populations increase, the quest for additional resources causes movement into virgin habitats. This may result in cross-species disease transfer, resulting in new or emerging diseases that cannot

be cured because of drug resistance.[57] Increased population density gives microorganisms a greater population base in which to transfer, mutate and ultimately spread. And if drug therapy is used, even successfully, this therapy will influence the greater population and environment because of the evolutionary principles of microbial adaptation and change.

I have attempted to identify some ways in which the factors of microbial adaptation and change, human social factors and environmental change connect, for it is in the idea of the connectedness of these diverse factors that the problem of drug resistance finds relevance. Accordingly, the lines of this triangle are not crisp or straight but complex networks of relationships, full of surprises and packed with meaning – each influenced by and influencing another. So in the end while retaining its framework showing the primary connections, let the triangle conceptually expand to include the idea that one factor cannot be separated from another but are bound inexorably in the intra- and interactions of microbial adaptation and change, human social factors and environmental change.

In the first part of this dissertation, it was my intent to create the links between the 3 major factors contributing to EID and ADR. The basic principles of microbial adaptation and change remain as constant concerns, particularly in their numbers and potential for more adaptation and change. I showed in the context of our biological and cultural evolution, our humanness, how we contributed and continue to contribute to the rise in EDI and ADR. As I have tried to show, we were not entirely responsible in the sense that for a long period in our evolution we knew nothing about microorganisms, much less our connectedness to the biotic community. However now perhaps we can admit that in the expansion of science and technology, we have set aside warnings (such as Fleming's admonition concerning the unregulated use of antimicrobials) and knowledge (such as our innate biological foundation in nature) as ever-increasing power bases vie for global stakes.

In the quest for power, the idea of technology as a universal 'fix' was not only unalterably embedded but also mediasised in our consciousness. This resulted in, amongst other things, the creation of a public myth, the myth of technology as omnificent. This perpetuation created false assurances; whatever we might do to our environment, technical means would be found to remedy any ills. For example, the technological 'fix' of antimicrobial therapy told us that infectious disease was an unfortunate condition of the past now remedied by progressive application of our knowledge and wit. It is not surprising that the evolution of a global reliance on technology seemed to provide a soft cushion on which we rely, blissfully assuming that whatever happens, someone somewhere somehow will provide the technological means to propel us out of the quandary. However, as Vogel (1996: 6) points out, technology allows us to affect the natural world in ways both outside and within ourselves in ways that are 'cumulative, irreversible and planetary in scale', so our reliance on it appears to be ill grounded.

Concerning Nature, we progressively became selectively more arrogant and ignorant – arrogant in thinking that we could dominate her without cost, and ignorant because we have historically failed to appreciate that for every action or inaction concerning her, she responds consequentially. I have explored only a few of the many ways in which environmental changes impact on EID and ADR, from changes in soil and global warming contributing to greater EID to the unknown consequences of ADR remaining in the environment.

Now, I will grapple with the problem of global drug resistance in an ethical perspective. I will consider within environmental ethics some ways in which human-nature problems are conceived and addressed in: (Chapter 5) *Humans and Nature* and (Chapter 6) *Lessons from Environmental Ethics*. I conclude Part 2 with (Chapter 7) *Informing the Human-Nature Debate: Bryan Norton's Enlightened Anthropocentrism*.

Chapter 5: Humans and Nature

Eight times emerging from the flood
She mewed to every watery god,
Some speedy aid to send.
No dolphin came, no nereid stirred:
Nor cruel Tom or Susan heard.
A favourite has no friend.

And hence, ye beauties, undeceived,
Know, one false step is ne'er retrieved, And be with caution bold.
Not all that tempts your wandering eyes And heedless hearts is lawful prize; Nor all that glisters gold.

Thomas Gray (1747)

Introduction

In joining the three factors that led to the problem of global drug resistance: microbial adaptation and change, human social factors and environmental changes, I have set the stage for an ethical analysis of the problem of global drug resistance.

In this chapter, I will begin the inquiry into our moral responsibility and the problem of global drug resistance. The problem of drug resistance points to the dilemma of how to wed human concerns with those of the environment. Included in all environmental perspectives is the idea of a human-nature relationship. I will begin this chapter asking what nature is. This is because if we could arrive at a definition concerning what nature is, then we might be able to separate the unnatural from 'natural' nature. Our final answers to this question point us to different ways of seeing ourselves in / out / combined with / or separate from nature.

While accepting the argument that an environmental ethic must include components of nature (organisms, ecosystems and so forth) I will seek to separate drug-resistant microorganisms from the rest of nature's entities. I will do this by introducing the term *supra-natural*. In using this term, I refer to 'natural' as entities existing in or caused by nature' (Thompson 1995: 907). For example, species that have evolved over time in evolutionary processes may be said to be 'natural' entities. By 'supra', I mean that entities are beyond or more than natural; they are imposed upon nature. Antimicrobial drugs initiated the trait or property of drug resistance. The acquisition of this trait or property led to the development of species (sub-species, strains or isolates) of new microorganisms we now refer to as 'drug-resistant'. Such *supra-natural* entities do not follow the natural order of evolution and change over time.

I will argue that because we have inexorably altered nature, she is no longer natural. Moreover, I will assert that the changes in nature we *see* are not of such magnitude as to cause us to consider ways in which our practices alter her. This points to our general tendency to view nature as something removed from us. In addition, we lack information concerning these changes.

Using oil-eating microbes and drug-resistant microorganisms as examples, I will point to our common desires to seek immediate solutions to our human problems without consideration of technology's possibly harmful consequences. Then I will turn to a related question: should it make any *moral* difference if a living entity is *purposely* 'manufactured' by humans or if it comes into existence because of *accidental* human influence? I will use oil-eating microbes and drug-resistant microorganisms to illustrate this. I will suggest that the issue of imposing onto nature *supra-natural* entities does not seem to turn on the issue of human contrivance or manufacture. It appears to not morally matter as long as commonly held worldviews prioritise human wants or desires above all else. I will point to the conclusion that in both examples, they hold the potential to change nature adversely. I will then look to Kantianism and consequentialism asking if justification based on 'good will' or a weighing of the consequences is possible in the context of global drug resistance.

I will then argue that we bear the heavy moral responsibility for the existence of global drug resistance and that we are obliged to accept our moral responsibility for the 'production' of drug-resistant microorganisms. We are morally responsible for their *supra-natural* existence in nature. Their *supra-natural* existence in nature and their survival trait of drug resistance are sufficient properties by which they can be separated from other naturally occurring life forms. As for drug resistance, I will argue that we can do nothing except try to contain the process.

Then I will ask if, and if so, how we might manage, control or contain, emerging infectious diseases and drug resistance. I will identify prudential measures and problems in this regard. Moreover, I will argue that drug resistance shows that we cannot exclude nature from our lives and from our considerations. What is needed, I will argue, is a shift in our common worldview that excludes nature from our concerns.

Finally, I will assert that drug-resistant microorganisms *do exist* in nature, the survival trait of drug resistance *is mutable,* things in nature *are united* ecologically and we *continue* to devalue nature. Increasing global resistance to antimicrobial drug therapy is a reality; with increasing momentum, it will destroy the 'stability, integrity and beauty of the biotic community'. Thus, I will conclude if we hold to Leopold's maxim, then the existence of drug-resistant microorganisms is a moral wrong.

Humans and Nature

As the history of environmental ethics shows, shifts in conceptions about the relationships between humans and nature are not instantaneous. The prevailing deep anthropocentric worldview started to lose some pre-eminence because of the insights of Charles Darwin. Together with Thomas H. Huxley, Darwin initiated the dismantling of the human position on top of the *Scala naturae*. In the footsteps of Darwin and Huxley, Gifford Pinchot ([1947] 1987) and John Muir ([1916] 1981) also raised concerns about human mishandling of nature and of her resources. Pinchot's conservation philosophy was, however, welfarist and human-centred (Elliot 1998: 2). Inspired by anthropocentric and utilitarian values, he saw material resources as commodities to be used sparsely (Ehrenfeld 1981: 177). Similarly, Muir's philosophy, which inspired Aldo Leopold's *Land Ethic*, emphasised a 'wise use' of resources. These steps, albeit in the right direction, were nonetheless prudential. In other words, the best way to avoid the 'tragedy of the commons' (Hardin 1995: 330) was through urging enlightened self-interest (Callicott 1995 a: 160).

Through gradual development, we discern concepts of human responsibility to nature emerging. In this, human morality is obliged to reconsider its hitherto strictly anthropocentric and dichotomous axiology to incorporate newly found values into the traditional ethical framework. Connections established in the human-nature relationship are captured in Callicott's (1995 b: 274) words concerning the land ethic saying: *'... [it] does not cancel human morality, neither does it leave it unaffected'.*

Almost for the first time in contemporary moral philosophy, the specific question of the intrinsic value of nonhuman entities and the necessity to 'globalise' ethics in the context of humans and nature arises (Sosa 1996: 51). Like Callicott, Sosa admits that questioning the intrinsic value of human and nonhuman life does not mean that we can do entirely without any of the traditional anthropocentric ethics. But this could not happen without, and thus requires, a moving away from our traditional human chauvinism in a nonanthropocentric direction. In environmental ethics, the debate then appears to focus on how much nonanthropocentrism is justified and acceptable. The answers vary from *e.g.* weak or enlightened anthropocentrism, to biotic egalitarianism to Gaian ethics. The choice, says Sosa (ibid: 59), is

> *... either to limit intrinsic value to the survival of the human species, or to make nature the beneficiary of ethical competence.*

Related to this quote, authors Pierce and VanDeVeer (1995) chose 'The Elusive Broader View' as the title introducing their work. To look beyond our normal myopia (perceiving the world created for only human benefit) they invite us to consider that our actions, both as individuals and collectively, depend largely upon what we believe to be good, right, and permissible, in other words what we consider to be of value (*ibid*: 1). Mindful of this, I have tried to make the broad term 'global drug resistance' less elusive, and to meld the connections between microbial adaptation and change, human social factors and environmental changes into an explicit argument showing the connectedness of all life systems.

With some exceptions, the idea of us being connected and related to / in / of nature is not a concept that has been promulgated in Western moral philosophy. In addition, the influence of Western philosophy on Western culture (which influences most societies today) has over time served to enhance the separation of us from nature. One major problem of course is that our actions are defined by what we are taught to be of value or what we perceive as a good or good. In the perspective of wedding human health concerns with those of the environment, the attempt then becomes essentially one of how to instil in us the value of nonhuman nature concurrent with valuing the human other. It would appear that any prerequisite to an ethics concerning the environment must ultimately rest on the recognition of a relationship between humans and nature, from the perspective of the human and from, as best we can articulate, a nonhuman perspective.

In the search to define itself, environmental ethics, has been variously considered from a simple extension of prevailing social ethics, to redefining philosophy as a whole, to a reformulation of fundamental responsibilities to nature.

Broadly, we can say that environmental ethicists rally around the conviction that we are culpable in the continuing degradation of our planet. This is because our predominant worldview considers nature only as a commodity. Environmentalists seek to change this worldview. As Griffin (1996: xiii) writes, 'The human proclivity to evil in general, and to conflictual competition and ecological destruction in particular, can be greatly mitigated by a world order and its worldview'. Integrated then in all environmentalist perspectives is the idea of a human-nature relationship. Amongst other considerations, this involves assigning membership into the category of nature. Let us turn to the question:

What is Nature?

Conceptions as to what is nature and what features are necessary for an entity to be considered as nature remain blurred. Concerning the former, Passmore (1995: 129) notes the ambiguity of the term because in its various usages it '*faithfully* *reflects the hesitancies, the doubts and the uncertainties with which men have confronted the world around them'*. According to Brightman (1961: 346), *'at least for theistic personalists, absolutists and panpsychists everything including nature is mental: nature is a system of objects either in or for mind – God's mind, or the minds of monads, or both'*. Norton quotes McNamee (1991: 155) responding to the question: 'What is nature? *'... A complicated conception that 'springs from our culture's unconscious sense of life itself ...'* 'Nature' for Partridge (2001: 28), refers to amongst other things, that which *'supplies the environment that selects our genes and thus shaped our neurological and cognitive development'*. Hartshorne (1961: 443) writes, *'... we begin to see nature as consisting of organisms composed of organisms on many levels'*. G. E. Moore ([1903] 1987: 86) defined nature as *'including everything that has or ever will exist'*. Thus, we see the concept of nature as fluid and similar to its variety of portraits in the Arts, it is often expressed in terms of morality. Most likely this is because it is perceived as the stage upon which we conduct every aspect of our lives, and in our lives, whatever we do or do not do has ethical aspects.

The concept of nature would most likely hold considerations of, for instance, aesthetics, the biological and physical order (contrasted with rationality and intelligence), and for our purposes let us include a world of invisible agencies. Drawing from such conceptions, how would we define nature? It could be articulated from an environmentalist perspective as *all the stuff of which life is composed; the broad physical and spiritual environment that makes life possible.* In a more human-restricted sense, nature could be defined as *the entire physical environment in which human beings live and that constitutes the range of conditions for continued living.* And this is the muddle; from what perspective do we see nature? Are we in or of nature, or out, combined with or separated from her?

Using a variety of environmentalists' perspectives, I will later show ways in which we can consider nature and her entities beyond mere commodities. For now, since we are focusing on the miniscule things that run the world (as opposed to Wilson's 'The Little Things that Run the World'[58]), let us turn to the latter part of the question: what features are necessary for an entity to be considered as 'natural' in the sense of being a part of or existing in nature? While accepting the argument that an environmental ethic must include the components of nature (organisms, ecosystems and so forth) I seek to separate drug-resistant microorganisms from the rest of nature's entities.

Unnatural Natural Nature

For Aristotle, nature or the natural world was always as it had been, no change occurred.[59] Because of our relatively short lives, we tend to conceive nature in the same way at least in the sense of viewing it relative to what we see of it during our lives. I suggest this is a mistaken perspective.

Compared to the earth of 100 or even 50 years ago, most of what we call 'nature' is unnatural. As discussed in an earlier chapter, entities in the 'wild' are only in a sense 'wild'; 'nature' is not actually 'natural' nature. This is because of our encroachment and inescapable influence upon both nature's original state as well as her entities. From, for example, extinctions to percentages of carbon dioxide in the atmosphere, to acid rain to drug-resistant microorganisms, there is little doubt that we live in what McKibben (1990: 60) calls a 'postnatural' world. Concerning nature, the problem is compounded because we remain for the most part ignorant of such changes.

In an average human lifetime, it is most likely that we see some spontaneous changes in nature but they are generally not of drastic proportions and occur in what we wrongly perceive as seemingly separate realms. For example, we may notice that a forest is turned into a residential area or that a river close by seems to have changed colour over the years. Yet in our commonplace lives, we tend not to consider the implications or associate such changes. Commoner (1992: 188) highlights one aspect of this:

> *Since World War II there has been an unprecedented growth in biological research; yet we remain astonishingly ignorant of the profound changes that, during the same period, have occurred in our biological surroundings.*

We live in what we perceive to be a natural world but the way we imagine it, including its actual health, may be quite different from our perceptions. The changes in nature resultant from human interventions are often virtually unseen and rarely a part of common knowledge. This mixture of psychological perceptions and knowledge plays a part in the moral decisions we make about nature and her entities for good or bad, right or wrong. Such attitudes and actions about nature underlie our perceptions of what moral responsibility we as moral agents have or do not have towards nature.

But, distinctions between unnatural and natural nature in some ways carry an arbitrary tone. Since this is the case, which I believe it is, are we not looking at such distinctions in the wrong perspective? What would occur if we reversed the question and since most of nature is unnatural take as our rule the unnatural as the paradigm as opposed to the natural. In understanding the ways in which we have shaped the unnatural, we may better understand how our actions affect nature and vice versa.

It is easier to move from the unnatural as the norm because from the time when we first began (*e.g.* crop production) we have altered or influenced natural evolutionary processes in nature. Granted in the beginning of agricultural selection, we did not 'tinker with the genes' as our hybridisation was generally confined to interbreeding amongst similar biological species. Thus, while we 'contrived' nature, modifying and changing crops to meet human preference satisfactions we did not alter the genetic properties of the species. As long as we did not boldly try mixing a pomegranate with a dairy cow, we remained within 'natural' boundaries. Likewise, most people would consider the tomato a naturally occurring vegetable (actually, its classification is a fruit) despite the fact that it was bred from a poisonous plant. In one sense, because it was 'manufactured' it is an unnatural vegetable. But the consequences of any deleterious 'crop' hybridisation such as a tomato remaining poisonous were not necessarily disasters. They could be destroyed if for some reason it was found that they were unwanted, unnecessary or harmful to other entities, particularly those human. Unlike tomato plants, drug-resistant microorganisms move us beyond this model. Drug-resistant microorganisms exceed the unnatural norm.

Insofar as we are now able, we cannot alter the genes of, for example, methicillin-resistant *Staphylococcus areus*, thus we are unable to eradicate it from nature in the same manner as an unwanted crop. In addition, the penetration of drug-resistant microorganisms into greater nature (earth, air and water) follows a different method of dissemination than that of intra-species crop production. It then appears that one major difference between drug-resistant microorganisms and traditional (as opposed to genetically modified[60]) crop hybridisation seems to turn on the question of our imposition of it upon nature as *supra-natural* as opposed to a 'natural' rising from it.

Let us look to the genus *Staphylococcus* and species *areus* as an example.[61] *Staphylococcus areus* bacteria are naturally occurring microorganisms thus they are 'natural'. On the other hand, methicillin-resistant *Staphylococcus areus* are *supra-natural* microorganisms. This is because they would not have come into existence had it not been for antimicrobial therapy. We recognise their reality as existent life forms following their biological-evolutionary model over time. But morally we can separate them from other naturally evolving entities. The property of *supra-natural* (life forms imposed upon nature by human design) provides the means to sever certain entities from the inclusion of all naturally occurring life forms into the arena of moral considerability. I will return later to this important distinction.

I have argued that nature is no longer natural. True, nature as a natural phenomenon lives in us in our imaginations but is no longer the nature that nurtured us and secured our survival as a species. Now I turn to two related issues. Using oil-eating microbes and drug-resistant microorganisms as examples, I will look at our common societal orientation to seek immediate gratification without consideration of the possibly adverse consequences of technology upon nature. Our worldviews concerning nature, although shifting, still tend to place her outside our major concerns although simultaneously we recognise a balanced environment is necessary for our wellbeing (Therborn 1980). Then I will turn to a related question: should it make any *moral* difference if a living entity is *purposely* 'manufactured' by humans or if it comes into existence because of *accidental* human influence? The distinction is obviously one of intention.

Moral Motives

We seem to have no problems in accepting quite readily the overt manufacture of some microorganisms, for example those designed to 'eat' oil resulting from spills. But the equal acceptance of drug-resistant microorganisms is a different matter. The issue of bringing into nature *supra-natural* entities does not seem to turn on the issue of human contrivance or manufacture. Rather, it seems to turn on the immediate gratifications associated with the potential of antimicrobial therapy to alleviate diseases and the potential of oil-eating microorganisms to clean up our environmental oil-spill catastrophes. Many concerns raised in genetic engineering appear to be negated in this perception, in particular the objection based upon the unknown dynamics of genetic modifications becoming transferred to other living entities, then into ecosystems and the biosphere. Relevant to this is a quotation from Capron (1993: 490):

> *[The real threat] ... posed by modern genetics is to the collectivity, by the changes that genetics can bring about in values and the alterations it generates in our perceptions and understandings of the world, not merely because of its* ***discoveries*** *(as was true of Copernicus and Darwin) but also because of its* ***ability to modify*** *living things, including human beings.*

The 'ability to modify living things' appears to be downplayed in creating both oil-eating microbes and drug-resistant microorganisms. Only the immediate gratification of undoing either the results of oil spills and disease processes takes precedence.

Concerning oil-spills, the alleged purpose of oil-eating microorganisms is to have a mechanism readily available to avoid the catastrophes done to nature resulting from oil spills. It is well known that the use of one technology often may be applied for other purposes. Thus, oil-eating microbes, for example, could be used as a subtle weapon to threaten the oil production of some countries, a possible secondary and more sinister application. In the former case, we might view it as a good; after all, most people are dismayed when they see sea creatures, birds and other life forms killed or damaged because of oil contamination. As long as they fulfil a human need or desire, manufactured life forms may be both presented to and accepted by the public as a good. Yet if we dig deeper, we may identify that the public does not have the necessary information concerning any possible adverse effects. My point is that as long as our worldview is centred only on human interests we will not be able to see much less understand that our actions and inactions may directly affect nature. Concerning the latter, most people would be shocked to consider such a usage. But we must remember, as Rifkin (1999: 7) identifies, that no application of technology has only benign consequences.

My concern about the application of such technology is two-fold: the possibility of their genes moving into the broader environment and that their presence might serve to deflect attention from factors involved in oil spills in the first instance. It might be argued that such microorganisms will be constructed genetically without reproductive capacities. But even if that is the case, in their eventual organic breakdown we would have to know how any residual components might potentially interact with other organic life. In some perverse way, the intense research and development associated with the creation of oil-eating microorganisms may send a message that unsound vessels might be accepted just as long as any unfavourable consequences such as shipwreck and any resultant oil spills are controlled.

The same concerns apply to drug resistance. Living things are already being modified as the drug-resistant trait is acquired. Simultaneously we now seek new antimicrobial therapies based on genetic engineering. If, for example, such therapies are designed not to reproduce or to die after destroying their targets, it still does not answer questions concerning the possibility of such properties escaping into the larger biotic community. Movement in this direction, it may be argued is a good, after all, living in a world full of disease is not a desirable one. But the seemingly omnipresent reliance on technological fixes, as opposed to us evaluating ways in which we contributed to the problem of emerging infectious diseases and drug resistance in the first place, becomes cast in shadows.

I wish to emphasise that in ordinary life we tend to seek immediate gratification based on our interests and reliance on technological 'fixes' without considerations of broader consequences. All *supra-natural* organisms, here presented as oil-eating microbes and drug-resistant microorganisms, share a certain commonality. Both can change nature. The fabric of nature is changed because the trait of drug resistance is mutable and thus can be accepted by other microorganisms. If and when drug resistance becomes established in nature, it will necessarily change our world. Our world will be changed because we may have no means by which to change the course of infectious diseases in human, animal and plant life. I will return to this mechanism later.

Now let us turn to the question of *whether it matters morally if a living entity is created by purpose or if by accident.* Concerning this, I now turn to overview two philosophical perspectives, Kantianism and consequentialism.

From a Kantian perspective (as well as practical philosophy in general), concern is placed on what ought to happen as opposed to what does happen. Although

Wille belongs to a rational subject, it can also act in the world. Immanuel Kant asserts that actions can be based on principles, reason or other non-moral motives and he admits that even moral action may have bad consequences ([1785] 1996: 72). This is most likely grounded in the assertion that our actions take place in the phenomenal realm, or subject to external laws so we cannot be certain that our actions will succeed. However, our actions are still morally good if they reflect the well-intentioned will.

In this perspective, if we accept the premise that antimicrobial drug therapy was conceived in good intent, then although it resulted in the addition of new and potentially dangerous organisms in nature, the action remains a moral one.

I identified in Chapter 2 that it was known in 1928 that drug-resistant microorganisms would occur because of antimicrobial therapy. Alexander Fleming, one of the scientists credited with the discovery of penicillin, in 1947 voiced great concern about its control:

> *The greatest possibility of evil in self-medication is the use of too-small doses, so that instead of clearing up the infection, the microbes are educated to resist penicillin and a host of penicillin-fast organisms is bred-out which can be passed on to other individuals and perhaps from there to others until they reach someone who gets a septicaemia or a pneumonia which penicillin cannot save. In such a case the thoughtless person playing with penicillin treatment is morally responsible for the death of the man who finally succumbs to infection with the penicillin-resistant organism. I hope this evil can be averted* (Fleming quoted in Zimmer 2001: 214).

Fleming's concerns were not heeded. It may have been because of the time and circumstances that surrounded the discovery of penicillin, an effort principally born out of the need to manage the war wounds of Allied troops, and not based on a more global concern for the benefit of humankind. It is likely that (and this is conjecture), ordinary doctors did not know much about the adverse effects of microbial therapy. Moreover, as Levy (2002: 44) writes: 'Initially, penicillin was reserved exclusively to treat soldiers and a few lucky civilians. It more or less "belonged" to the military.' Eventually it was marketed in the USA as an over-the-counter product shortly to be discontinued with the rise of pharmaceutical companies about ten years later (Lewis 1995: 54). So we might ask if pharmaceutical companies (from 1940 onwards) became incorporated for goodwill or other less moral (read as profitable) motives. Did the pharmaceutical companies inform the medical community concerning the possibility of drug resistance? Were the consequences of misuse a part of public knowledge? Most likely not. Nonetheless, to assert unequivocal good will behind the act I argue remains tenuous at best.

Consequentialism considers that the moral value of actions is determined by the value of their relevant consequences. Therefore, the right action is that which produces or brings about the best consequences. This stands in opposition to Kant's perspective. In everyday life, consequences play an important part in understanding or characterising the moral weight of certain actions, and how they affect others as well as ourselves. To the extent that we feel responsible for the outcome of our actions, we are also responsible for the consequences of what we choose to do or actually do or do not do. Consequentialism focuses on the normative aspects of moral agency.

Disagreeing with Kant, John Stuart Mill ([1859] 1973) asserts that morality does not consist in motives: 'the motive has nothing to do with the morality of the action since it is governed by the end' (*ibid:* 419). For Mill, although duties are distinct from their end, duties also consist in the 'feeling of duty' (*ibid*: 434). This feeling is the pang of conscience which Mill claims is 'natural' at least in the secondary sense of human capacities. Duties are stated in the form of rules; rules are principles or formulas, which cover identical issues or cases, separated from time, space or circumstances. Mill states that there is one fundamental principle or law at the root of all morality 'the principle of utility or the greatest happiness principle': the greatest amount of happiness / pleasure for the greatest number of people (*ibid*: 403).

Can we say that antimicrobial drug therapy was wrong if evaluated in terms of its consequences (conditions instigating the 'birth' of drug-resistant microorganisms)? Keeping with the utilitarian account, we are obliged to weigh the consequences of both developing and monitoring antimicrobial therapy and not doing so. But we are hampered because whilst we know something concerning the consequences of drug resistance we do not know everything.[62]

From the advent of antimicrobial therapy, it is probably safe to say that from a strictly human perspective, the use of antimicrobial therapy has benefited more lives than it cost; lives were saved because of antimicrobial therapy. But in historical perspective, its usage represents a little over 55 years. The *global* problem of drug resistance is of a more recent vintage. In less than 2 generations, because of gross and imprudent antimicrobial usage, we now have documented reports of increasing numbers and types of drug-resistant microorganisms (Levy 2002: 114). This includes all pharmacological intervention in infectious disease and injuries, water purification, insect control and so on. As Levy (*ibid*) puts it:

> *We are presently witnessing a massive, unprecedented evolutionary change in bacteria.*

Combining this with all the factors we do not know *e.g.* how drug-resistant microorganisms will behave under environmental stress, how far the trait of drug resistance will spread and so forth, all we can say is that a weighing of the overall consequences, while leaning towards evolutionary disruption, remains difficult to determine.

In the end, perhaps the focus on 'accidental' or 'purposeful' intent is irrelevant to the discussion because from any moral viewpoint that such entities exist or will come to exist remains a fact. We bear the heavy moral responsibility for their existence. Likewise, we will bear heavy responsibility if other *supra-natural* entities are brought into existence. In so acting upon nature, we do not just *make an organism*. We interact with complex systems setting in process new interactions, the results of which may have unpredictable consequences. In the context of a relationship between human society and the environment, this reinforces the connectedness of life systems. It should give us pause to consider ways in which such creations may and do backfire not only on us, but also on other members of the biotic community.

Ecological Unity

We are ecologically united with nature and her entities. This is a scientific fact. Moreover, there appears to be consensus from most philosophical viewpoints that this is the case. Ecology focuses on relationships between and among things, the complex networks of relationships within the earth's closed system. An ecological ethics, according to Sosa (1996: 57), 'expresses the concern for the permanence of the human species in its moral form by proposing a transformation of our relationship with nature making nature the beneficiary of our ethical competence'.

Environmental ethics often emphasises ways in which we may extend moral status to nature and nonhuman others. And this is the muddle. As moral agents, we grant moral consideration to other humans. Because they are human, they are morally significant. Thus they are morally considerable. Something is morally considerable if it has a claim to be directly taken account of in our moral evaluations and judgments. Cahen (1995: 301) explains it this way:

> [... *moral considerability is ...] (A) the moral status x has if and only if they have interests (a good of their own), (B) it would be a prima facie wrong to frustrate the interests of x (to harm them) and (C) the wrongness of frustrating the interests of x is direct — that is, it does not depend on how the interests of any other being are affected.*

It is the concern with interests that often marks moral considerability from the other varieties of moral status upon which the preservationist intuition may possibly be grounded. I will expand upon some positions in environmental ethics in the following chapter but for now, I wish to point out that Cahen is not alone in emphasising interests. In addition to Bryan G. Norton (1986; 1991; 1995) and Paul W. Taylor (1986; 1995), whose views I will specifically discuss later, Goodpaster (1978) also restricts moral considerability to beings with interests. For him, 'life' grounds moral considerability. This is because he claims that living things have interests and such interests make them 'capable of being beneficiaries' (*ibid*: 323, 325). Singer (1995: 51-59) claims sentience, the capacity to feel pleasure and pain, as the criterion of an entity's moral considerability. For Regan (1976), being 'the subject of a life' denotes the acquisition of moral considerability in the form of rights.

For others, moral considerability is equated with intrinsic value. In this conception, both intrinsic value and interests are equally presupposed. The point is that in environmental ethics, attempts are made to say precisely which kinds of things are morally considerable and why. From a purely theoretical point of view, there are obstacles to the adoption of an extension of moral considerability and moral significance to all the biotic community. Even if one accepts the meta-ethical view that there are degrees of moral standing (Moore and Bruder 1996: 264), it remains unclear how differently one may treat entities according to the level of moral standing we humans allocate.

Agar (2001: 140) contends that a major obstacle remains in wholesale acceptance of all species into the biotic community. This is because no mode of identification is available within many theories for denoting the value of a particular species. To this end he (140) writes,

> *... such an account would presumably fix on a property possessed by a collection of organisms falling under the name species but not others. If such a property is identified then it would serve to identify which and why certain entities are morally considerable and which are not.*

My account is this. We are obliged to accept our moral responsibility for the creation of drug-resistant microorganisms. We are morally responsible for their *supra-natural* existence in nature. Their *supra-natural* existence in nature and their survival trait of drug resistance are sufficient properties by which they can be separated from other naturally occurring life forms. Thus, we have no moral duties to them nor are they morally considerable. Does this imply that we can harm them at will since we are dissolved of duties and obligations to them? Ironically, we cannot harm them even if we wanted to — they exist and they are spreading. But their properties present harm to all the biotic community.

To support this claim, I now turn to American conservationist Aldo Leopold, who influenced many environmental perspectives. Memorable is Leopold's oft-quoted maxim: 'A thing is right when it tends to preserve the stability, integrity and beauty of the biotic community. It is wrong when it tends otherwise' (1966: 32).

There is a force existing in nature as a survival trait or property. It is so mutable that it can be distributed amongst the foundational ecological building blocks of nature, in the genes of microorganisms. Without microorganisms, life as we know it on this planet would not exist. Now, imagine in our contemporary world that all the trillions and trillions of microorganisms living in and on all organic and inorganic life (*e.g.* skin, bark, organs, tissue, leaves, thermal vents, fins, feathers, earth, air and water) were to acquire this property and in an instant become resistant to all antimicrobials. In such a world, no effective medicines would be available for people, animals or plants needing relief from the scourge of infectious disease and injury. Because of population pressures and continued environmental degradation, we would experience a rise in new, old and emerging infectious disease. Medicinal drugs would no longer be effective. Pesticides would no longer be functional. Water supplies would be contaminated because chemical purification would no longer be successful. Factors such as these would feed into other life systems in a never-ending circle of misery. We would exist in a world of disease.

If this were to occur, it would endanger the 'stability, the beauty and the integrity of the biotic community'. But my scenario is hypothetical (or is it?). Drug-resistant microorganisms *do exist* in nature, the survival trait of drug resistance *is mutable,* things in nature *are united* ecologically and we *continue* to devalue nature. Increasing global resistance to antimicrobial drug therapy is a reality; with increasing momentum, it will destroy the 'stability, integrity and beauty of the biotic community'. The moral wrong is that through imprudent use of antimicrobials we imposed upon nature a *supra-natural* life form that confirms Leopold's maxim.

Lessons from Drug resistance

The lesson we learn from the problem of drug resistance is to consider that even seemingly benign applications of technology may carry global consequences. As for drug resistance, we can do nothing except try to contain the process. To contain the process above all requires a shift in our worldviews. This is because if we only consider human concerns then we miss the point that the additional stress we place on the environment (*e.g.* population pressures, environmental degradation) coupled with the penetration of drug-resistant microorganisms into the environment equals greater chances of drug resistance to spread. It is a circular process.

Can we gain insight on how to manage or control drug resistance? I have tried to show some of the intricate relationships involved in this phenomenon. It should be clear that moral responsibility for both lies squarely in human social behaviour (for microorganisms have no moral responsibility). However, it would seem in this complex situation that our personal options in assigning or accepting moral responsibility are not so clear-cut.

For example, do we tell a doctor in Bangladesh that he is immoral because he follows his cultural practice of selling drugs in lieu of a consultation fee? Do we prohibit poor patients from buying some pharmaceutical products unless they can afford a complete regimen? How do we convince often itinerant and illiterate tradespersons and hawkers to sell only complete regimens? By what means do we ensure that patients complete their drug regimens in spite of their (at least often claimed) adverse side effects?

Pressuring the animal-food industry to reduce drug use may be possible if there is common knowledge concerning drug resistance and if it is in some way organised. However, the interests of business and politics may impede action in this regard. For example, in 1986, the Fogarty International Center of the United States National Health Institute concluded a three-year project concerning the state of antimicrobial usage and worldwide resistance (Levy *et al.* 1987). This report identified that in 1987, current antimicrobial production was sufficient to meet the needs of citizens globally if all antimicrobials were effective and if resistance was not a factor. However, the distribution and the extent of drug resistance were found grossly unequal. To make the situation more difficult, they identified that in areas where antimicrobials were most needed, drug therapies met with the greatest amount of resistance.

Importantly, their study led to other findings such as although enough antimicrobials were being produced, most would not be useful in the face of large-scale resistance (*ibid*: 22). The extent of resistance, they found, varied with the particular drug type used and from country - to - country. Moreover, whilst the frequency of resistance to particular drugs varied with microorganisms overall, distant countries still shared the same kinds of resistant strains (*ibid*: 26).

Despite the commitment of the Fogarty Team, the report did not achieve its aim of public awareness. This is because, as Levy (2002: 304) asserts:

> *Political manoeuvrings and actions by pharmaceutical companies convinced U.S. officials and the National Institute of Health, which sponsored the project that the problem was being overstated and that support for the planned meeting and any future attempts should be withdrawn.*

Levy (*ibid*) refers to this as a 'sad commentary' on the world in which we live and wonders 'if this *avant-garde* initiative had been allowed to continue whether the global crisis of antibiotic resistance would be as grave as that which we face today'. As in environmental degradation and illnesses linked to environmental factors, the problem of drug resistance must negotiate with the interests of big businesses and political will if it is to be controlled. I must admit a sense of doom in this regard.

Do we rather focus on patients who, because of adverse side effects, do not complete drug regimens? Or do we develop a grading scale of condemnation assigning greater (or lesser) weight to those who claim for autonomous or other reasons they cannot complete the course? Prudentially, on one level, educating doctors and other medical practitioners is indicated. Should the focus be on expanding coverage of emerging infectious diseases and drug resistance in medical schools, specifically the ways in which they will increasingly alter the practice of medicine (HIV/AIDS and increasing drug resistance to therapies is a good example)?

As described in Chapter 3, doctors' lack of knowledge is apparent in their inadequate diagnosis of diseases, incorrect drug selection for treatment or prophylaxis of infections, and incorrect prescription of doses, duration and routes of antimicrobials. Other subjects worthy of study might include more quantitative ones, such as why doctors prescribe in response to patient pressures, why fears of litigation override obligations to prescribe correctly, why financial gain pre-empts proper patient care, and the influence of drug promotional pressures on practice. We should address how poor underlying health and high need influence drug choices. Issues inherent in the hazards of self-medication and problems innate in non-compliance should be investigated. Myths such as 'expensive is better' and 'more drugs are better' should be demystified, inappropriate beliefs discarded.

Governments should ensure that national drug and quarantine policies are appropriate and well communicated. Concerning quarantining humans who carry drug-resistant microorganisms, it would be an exercise in futility because of its prevalence in nature. Specific to drug resistance, essential drug lists must be established, updated and most of all communicated. Sales of drugs should be regulated, ideally in hospital or by private pharmacies, not left either to the informal sector or to medical practices unless they are far from any hospital clinic or other established pharmacy. Infection control strategies for disease control and drug resistance monitoring should become part of public debate. Hospitals should establish infection control committees to oversee their own drug resistance policies, and update and distribute such policies widely as more knowledge becomes available.

But doctors and other health care professionals are not the only people involved. From industrial misuse of antimicrobials as growth promoters to the creation of what are essentially monocultures of animals in which the potential for emerging infectious disease is enhanced, others too are implicated. The pharmaceutical industry is not immune to criticism, for example, in its focus on drug development for mainly developed countries and often-unethical practices of drug promotion. Moreover, the focus on genetically engineered microorganisms and drug therapies may have, as I will later suggest, untoward costs on the biotic community.

Prudential measures to contain global drug resistance requires at least changes in social, economic and political philosophies, enforcement of global infection controls, political will to sustain sustainable development, and limitations on human population and consumerist practices. These are examples of prudential measures that are aimed at the control of drug-resistant microorganisms.

But the reason we arrived at the situation in the first place, is because we considered ourselves to be above all else. We developed antimicrobial therapy for human benefit without considering its broader ramifications. It might be argued that at the time when antimicrobial therapy began we were not aware of possible side effects. Granted, some effects we did know, and some we did not anticipate. Yet, that is precisely where the problem lies for our tendency, then and now, is to place absolute faith in technology without consideration of any other factors other than ourselves. Drug resistance shows that we cannot exclude nature from our lives and from our considerations. What we do or do not has implications beyond ourselves.

I have shown that the consequences of global drug resistance affect beyond our families, our communities and us. On a prudential level, our moral responsibility is to curtail the creation of newly resistant strains which once selected, join the larger microbial population in the environment (for the existing drug-resistant strains, we can do nothing). Our prudential level in turn informs a higher moral level in which we are morally obliged to rethink our worldviews concerning nature as only a commodity and instead see our interconnectedness and interdependency with her. I will address this further in Chapter 7.

In closing, we can consider Rolston's (1988: 330) recommendation: 'an ethic of conflict has to become an ethic of complementarity: humans as completing and appreciatively residing in nature'. In the following chapter, I will explore other ways of viewing humans and nature.

Concluding Remarks

In this chapter, I have tried to show that within the young discipline of environmental ethics there have been gradual shifts in ways of conceiving human responsibility towards nature. Admittedly, environmental ethicists (or eco-philosophers) have not been the first to consider human-nature relationships but it is from this discipline that a deeper understanding of the contemporary need to reconceive our relationship with her has gained momentum. Indeed, any prerequisite to an ethics concerning the environment must ultimately rest on the recognition of a human-nature relationship.

I was particularly interested seeing the different ways in which nature is conceived; particularly what entities are considered 'in' nature, what entities are 'out'. It seemed overall that 'nature' somehow implies 'things natural'. This I believed could refer to entities that have evolved following the natural evolutionary mode of adaptation and change. They would be 'in' nature as 'natural entities'.

I then argued that nature and her entities do not exist anymore as 'natural' because of our human influence. I admit that while the concept of nature lives in our imaginations, it is no longer the 'natural nature' that sustained us throughout our evolutionary process. I proceeded to show how in our early cultural development we produced agricultural products within the boundaries of their same species.

Then I described how in application of antimicrobial therapy we initiated a new life form. I coined the term *supra-natural* to refer to a life-form that had been imposed upon nature by human-design. ('Supra' equals more than or beyond; 'natural' refers to entities that exist in or whose existence is brought about by natural evolutionary processes). I explained how the property or trait of drug resistance was brought about by the application of antimicrobials. In acquiring this property or trait, a new species (sub-species, strain or isolate) of microorganisms develops. I used the example of *Staphylococcus aureus* to show how this happens, namely: This species exists 'naturally'. Through the application of the drug methicillin, this organism acquires (through its function of adaptation) the trait or property of methicillin drug resistance. It is now a 'new' microorganism: methicillin-resistant *Staphylococcus aureus*. My purpose in making such distinctions was to find a way in which it would be possible to separate some life forms from others.

I then turned to our general tendencies not to consider consequences beyond our own needs and desires. This is a global problem and using the examples of oil-eating microbes and drug-resistant microorganisms, I proceeded to show that our perceptions of their value are often a form of moral myopia – we do not look beyond ourselves.

Wondering if it would make a moral difference concerning the reasons for the use of antimicrobial therapy, I turned to look at Kantianism and consequentialism. I argued that no unequivocal good will existed in the creation of antimicrobials. As to a judgment, of the rightness or wrongness of antimicrobial use based on consequences, I argued that we do not have enough information upon which to make a full judgment but that the leanings so far are factual in that antimicrobial use has influenced natural evolutionary processes. In doing so, it may bode ill for the biotic community. But then I observed that in some ways the moral motives do not matter. This is because drug-resistant microorganisms exist; nothing can be done to change that fact. We can only try to understand how our actions are interconnected with those of nature, and in the context of drug resistance, try as best we can to contain the process.

I evoked Leopold's (1966: 32) maxim 'A thing is right when it tends to preserve the stability, integrity and beauty of the biotic community. It is wrong when it tends otherwise' in a hypothetical narrative to illustrate how in extreme drug-resistant microorganisms might affect the biotic community.

Turning to the topic of ecological unity, I pointed to some objectives found in environmental ethics. One of these concerned ways in which we might extend moral considerability to nonhuman entities. I considered this relevant to my distinction of the *supra-natural* from natural.

In concluding the chapter, I turned to lessons we might learn from global drug resistance. I pointed out that one major problem is our reliance upon technological 'fixes' and another is that efforts to even understand the global impact of drug resistance is hamstrung by powerful economic interests. From inherent difficulties of managing drug resistance because of such factors as diverse cultures, social perceptions of antimicrobials and global economic disparities, containment of drug resistance will be most difficult to attain. However, I provided what I consider some prudential measures. In still holding that the problem of global drug resistance rests fundamentally on the ways in which we conceive the human-nature relationship, I now turn to Chapter 6: *Lessons from Environmental Ethics*.

Chapter 6: Lessons from Environmental Ethics

[The music] ... saddened me because it reminded me that in my century nothing is totally free of the taint of our arrogance. We have defiled everything, much of it forever, even the farthest jungles of the Amazon and the air above the mountains, even the everlasting sea which gave us birth. Ehrenfeld 1981:269.

Introduction

Keeping with the problems faced by global drug resistance as well affirming the interconnectedness of systems, in this chapter I seek to find direction in the human-nature debate from a variety of nonanthropocentric positions in environmental ethics.

According to Elliot (1998: 1-20), since the emergence of applied ethics as disciplines at the turn of the 1960s, three major steps[63] have been accomplished in a nonanthropocentric direction. As mentioned in the last chapter, as advocated mainly by Tom Regan and Peter Singer[64] moral concern was extended to the well-being of nonhuman animals. For Singer explicitly, moral concern was extended at least to those with the (assumed) neurophysiological ability to experience pain and pleasure. Thus, microorganisms were not part of the debate. Kenneth Goodpaster entered the conversation ascribing 'moral considerability' to all living biologically 'goal-directed' natural entities.[65] Then the work of Paul W. Taylor took the debate further in biocentric egalitarianism in his move to ascribe intrinsic value to all natural entities.

One may differ with Elliot's distinctions because there are many other philosophers who contributed in different perspectives. But the movement is clear: from sentience to rights to moral considerability to blanket intrinsic value to all living entities. In this chapter, I will overview some points raised in the arguments of Paul Taylor, J. Baird Callicott and Holmes Rolston III. All of these perspectives, albeit in different ways, raise particular interesting points to consider in the context of infectious diseases and drug resistance.

Paul Taylor's Strong Biocentrism

Taylor develops a theory according to which both animals and plants have a value in themselves independent of human valuation. He advocates a strong version of biocentrism, biocentric egalitarianism. For Taylor (1995: 126) this means that 'we have *prima facie* obligations that are owed to wild plants and animals themselves as members of the earth's biotic community'. This is because all that lives 'has a good of its own (enhancing or preserving its life and well-being) … the full development of its biological powers'. And this, he argues, means that 'all members of the earth's community of life deserve moral consideration on the grounds of intrinsic value' (*ibid*: 127). According to Taylor (1986: 75), all living things have inherent (intrinsic) value. To claim that an entity X has intrinsic value is to assert the following:

> *A state of affairs in which the good of X is realised is better than an otherwise similar state of affairs in which it is not realised (or not realised to the same degree), (a) independent of X's being valued, either intrinsically or instrumentally, by some human valuer, and (b) independently of X's being in fact useful in furthering the ends of a conscious being or in furthering the realisation of some other being's good, human or nonhuman, conscious or nonconsicous.*

According to Taylor, a living entity has inherent worth. This entails two moral judgements: that it deserves moral concern and consideration, and that all moral agents have a duty to promote the entity's good as an end in itself (*ibid*). Non-human life has the same inherent worth as human life does, so nonhuman life deserves equal concern and consideration (*ibid*: 261). Rejecting any value hierarchy in nature, he (*ibid*: 177-178) states:

> *We are ... required to respect (organisms') wild freedom by letting them alone. In this way we allow them as it were to fulfil their own destinies ... Of course some of them will lose out in their struggle with natural competitors and others will suffer harm from natural causes ... we remain strictly neutral between predator and prey, parasite and host, the disease causing and the disease.*

The fundamental value underlying all Taylor's propositions is that of 'life'; all living beings have an equal good. Therefore, they are entitled to equal moral considerability. We can agree that 'life' in the broad sense of its preservation and continuation is undoubtedly the fundamental biological value. And we could argue in a teleological sense that, for living entities, life has purpose. Adopting Taylor's line of thinking, it would follow that both methicillin-resistant *Staphylococcus aureus* and *Staphylococcus aureus* have an equal claim to existence and their claims are equal to our own. In keeping with Taylor, this requires neutrality on our part and indeed bodes ill for the practice of medicine be it human, animal or plant!

In consideration of my argument in the previous chapter, an 'informed' Taylor cannot grant equality to all life forms simply based upon their existence in nature. To this, Taylor may respond that I have missed the thrust of his argument as he (1995: 126) writes:

> *From the perspective of a life-centered theory, we have prima facie moral obligations that are owed to* ***wild*** *plants and animals themselves as members of the Earth's biotic community.* (*my emphasis added*).

The question then is would the context (*viz.* to plants and animals in the 'wild' as opposed to 'non-wild' entities) of granting equality of life alter my argument? I do not believe it does. Microorganisms, including those with the trait of drug resistant exist everywhere including 'wild' nature. It is, I suggest, reasonable to consider the distinction I made between *supra-natural* and 'natural' life forms as sufficient justification to contest Taylor's argument. For microorganisms, there is only one earth and it is devoid of any distinctions such as 'wild'.

When every living entity is equalised and all are morally considerable then we will inevitably face grave problems when conflicts of interests arise. We will see how Taylor argues this problem turning to his principle of self-defence. It may be seen somewhat odd to consider that while we are obliged to value all living entities equally that both sentient and non-sentient beings may be killed when doing so is essential to the continuation of our existence, albeit with limitations. He (*ibid*: 265) writes:

> *The principle of self-defence states that it is permissible for moral agents to protect themselves against dangerous and harmful organisms by destroying them ... This principle does not allow the use of just any means of self-protection, but only those means that will do the least possible harm to organisms consistent with the purpose of preserving the existence and functioning of moral agents.*[66]

Because Taylor insists all living things have equal inherent worth, it makes application of his theory very difficult in practice, in spite of his insistence that it is possible. In the case of drug-resistant microorganisms, this quote does not find too much relevance. The interesting twist is even if it is (and it is) for our own self-protection, we are not able to eradicate methicillin-resistant *Staphylococcus aureus* or any other existing drug-resistant microorganisms. Moreover, while it seems that Taylor gives us permission to destroy other dangerous and harmful organisms for self-protection, we are constrained because we can only do the least possible harm to them. In keeping, one may wonder just what means might be available for us to use when faced with methicillin-resistant *Staphylococcus aureus*. It is hard to see how that can be devised, at least at this time. However, within his egalitarian position, importantly, Taylor's aim is to show our obligation to respect life; to destroy life randomly and purposely is indeed a moral wrong. Following his ethic of 'respect for nature' then entails not harming the interests of other species and enhancing the conditions under which they can flourish.

Based on 'life' as a fundamental value, his egalitarian position obligates us to consider all life forms as equal. This then presents a challenge to my argument because assuredly, drug-resistant microorganisms are alive. I will argue for a way around this later. For now, let us simply note this concern as we see how he negotiates between moral agents and species-impartiality. Perhaps this will provide us with a way out. Taylor (*ibid*: 266) writes:

> *Despite what might at first appear to be a bias in favour of humans over other species, the principle of self-defence is actually consistent with the requirement of species-impartiality. It does not allow moral agents to further the interests of any organism because it belongs to one species rather than another. In particular humans are not given an advantage simply on the basis of their humanity.*

So as moral agents, we seem to be permitted to protect ourselves from harm (within certain limits), but we cannot be species-biased. In an environmentalist approach to drug resistance, we are obliged to consider it in a context beyond ourselves. So conceived, because drug resistance threatens the biotic community, we would be acting in defence of the whole. In this way, we move beyond human-only considerations. Yet at the same time, we are still acting against a particular species. The irony is that Taylor's argument in this regard does not apply to the problem of drug-resistant microorganisms (as a 'species'). This is because we have no medical means by which we can protect ourselves or other life forms from the trait of drug resistance that already has been or will be acquired and which has and will continue to spread globally.

Moreover, because 'life' is the fundamental value that grounds equality, how might this affect my argument that proposes to separate certain life forms from others? A way out might be to ask Taylor if the value of life, of 'merely being alive' is sufficient reason on which to ground equality? However, I believe my argument remains the best option.

Taylor frames all of his arguments in the context of the *natural*: 'natural environment', 'natural systems' and 'natural states' (1986, 1995). 'Life' in this regard is thus contextualised as life that naturally occurs as life forms, in systems and so forth. Thus, life may be seen as a natural value that exists in the products and processes of natural evolution — courses of events and evolvements occurring over space and time. My argument from the *supra-natural* delineates the *supra* from natural occurrences and natural entities. In doing so, certain species may be separated from others: the supra-from naturally occurring / evolving life forms. This then, while affirming the value of 'life' also restricts it to only natural or naturally evolving entities.

Taylor recognises there is conundrum concerning conflicts of interests. He (1995: 139) writes:

> *If we adopt the biocentric outlook and accordingly adopt the attitude of respect for nature as our ultimate moral attitude, how do we resolve conflicts that arise from our respect for persons in the domain of human ethics and our respect for nature in the domain of environmental ethics?*

In the context of drug resistance, I believe I have shown a possible solution to this problem. If we act to control drug resistance, then we act not only on our behalf but also for the biotic community as a whole. Delineating between naturally occurring entities and those imposed upon nature, affords us the way in which we can act without showing species-bias. Agreeing with Taylor, the fundamental value of life is maintained (both in and out of the 'wild') but narrowing the parameters, it is restricted to naturally occurring entities. The latter, I admit, is a weak point because I give the fundamental value of life only to 'naturally occurring' entities. This would exclude for example, human and nonhuman clones. Taylor, in his ascription of the fundamental value of life to all nature, retains his trump in this regard. But it is unworkable in the context of healthcare for, even if we admit the value of life, we need to find a way to delineate such value and do so in a non-chauvinistic way.

What Taylor importantly emphasises, is that if we agree we are members of a biotic community we are obliged to recognise that our optimal functioning is dependent on the optimal functioning of other entities. This point is well taken. Against Taylor, we see that if we hold strictly to biocentric egalitarianism it would follow that, for example, both methicillin-resistant *Staphylococcus aureus* and *Staphylococcus aureus* have an equal claim to existence and their claims are equal to our own. His arguments concerning self-defence in the context of drug resistance do not provide direction and, once again, the egalitarian requirement of neutrality on our part essentially destroys the practice of medicine.

Now let us turn to overview two other prominent philosophers in environmental ethics, J. Baird Callicott and Holmes Rolston III.

Ecocentrism

Generally, strong biocentric and strong ecocentric theories are quite controversial since they question one of the most fundamental moral assumptions prevalent in mainly Western countries: that humans have special moral standing. Strong ecocentrism is often referred to as environmental holism.[67] I will use insights from J. Baird Callicott to represent a strong ecocentric position. Then I will turn to overview the position of Holmes Rolston III representing the position from weak ecocentrism.

J. Baird Callicott's Strong Ecocentrism

I will limit this discussion of Callicott, reviewing some of his basic tenets and examining a few particular relevant points.

J. Baird Callicott, one of the most influential writers in environmental ethics, is a strong ecocentrist. His works are largely based on the writings of conservationist Aldo Leopold. 'One fundamental and novel feature of the Leopold land ethic', Callicott (1989: 15) writes '... is the extension of direct ethical considerability from human to nonhuman natural entities'. Leopold's view of ethics is based on ecology – on the concept of the 'biotic community'. It emphasises the contributing roles played by various species in the economy of nature and abandons the 'higher / lower' ontological schema in favour of a functional system of value. Callicott (*ibid*: 75-101) notes Leopold's arguments that all aspects of nature — plants, soil, waters, animals, humans — ought to be perceived as components of the biotic community. In this community, humans are simply a part of it as additional natural members to its whole.

As we will see later, opposing Norton, Callicott claims that a new environmental ethics is needed rather than a replication of older theories or an extension of traditional anthropocentric ethics. He (*ibid*: 3) argues for a 'shift in the locus of intrinsic value from individuals (whether individual human beings or individual higher animals) to terrestrial nature – the ecosystem as a whole'. Callicott affirms that a complicated web of relations is involved in any environment and this complexity determines the relations of organisms. He argues that, while an individual is continuous to the environment and is constituted by it, the role of the species as a whole outweighs the importance of one individual (*ibid*: 157-176).

For example, an individual may be killed off as prey, yet the species survives as a whole and continues to play its part in the larger ecosystem. Callicott wishes to stress the role of the species as a whole in order to emphasise holism as opposed to individualism. In supporting the 'land ethic', he places reliance on altruistic moral sentiments and intrinsic value projected to nonhuman life. The locus of value is extended to the biotic community. The duty derived from this approach is not to degrade the land as a whole: a principle of selection between elements of the land is clearly stated and unambiguous in all his works. He places much emphasis on preservation of 'wild' nature and her entities (*ibid*: 56). His arguments support a graduated scale of moral obligations based on degrees of intimacy which produces overall a nuanced ethic of relationships between humans and other entities.

Now I turn to his text (*ibid*: 71), believing it succinctly captures a major problem in the human-nature debate. Callicott discusses Passmore's contestation of the idea that, because a land or biotic community shares common interests with humans, ethical obligations on that basis alone may be derived. Passmore (1974) (quoted in Callicott *ibid*: 116) writes:

> *We sometimes now meet with the suggestion ... that animals do in fact form, with men, a single community to which we belong ...we may begin to use it with love and respect ... Ecologically, no doubt, men do form a community with plants, animals, soil in the sense that a particular life-cycle will involve all four of them. But it is essential to a community that the members of it have common interests. In the only sense in which belonging to a community generates ethical obligation, they do* ***not*** *belong to the same community. Bacteria and men do not recognise mutual obligations, nor have common interests. In the sense in which belonging to a community generates ethical obligation, they do not belong to the same community* (original emphasis).

Callicott identifies the construct of Passmore's claim as 1) the recognition of mutual obligations and 2) the sharing of common interests. The recognition of mutual obligations, Callicott claims, is not a necessary condition on which to make such an argument. In our society there are many individuals (such as the insane, mentally impaired, comatose, etc.) who, because of their mental states, are unable to recognise mutual obligations. Nonetheless, they are still considered legitimate members of society. So the inability of an entity to recognise mutual obligations is not a valid point on which to raise an objection.

Callicott (*ibid*) claims that both humans and bacteria meet the necessary condition of community membership:

> *Bacteria (whom Passmore must consider a very alien form of life) and people do have common interests. Indeed, in the case of some bacteria and all people the cooperative mutual dependency is so great that it could be called a biological symbiosis in the strictest sense. But all living things are united ecologically, and all share the common interest of life itself, the desire to live and to be let alone*

A few examples will show the mutually beneficial relationships between the environment and the microorganisms. Scientific data show that symbiosis exists in nature; the genetic matrix of early evolutionary life was and remains tied to microbial symbiosis.[68] This is shown in the cooperative relationships between certain legumes — peas, soybeans and alfalfa — and the soil bacteria 'fixing' or converting nitrogen from the air into a form that can be used by such plants. In this process, the rhizobia produce nitrogen for the plant and the plant returns the 'favour' by providing nutrients necessary for the growth and reproduction of the bacteria. From microbial components of rizospheres[69] to our own symbiotic reliance on certain microorganisms, to stratospheric constituency to water purification, microorganisms continue to support planetary life.

Specifically pertaining to Eukaryotes of which we are members, we would not be able to receive nutrition from plant polysaccharides because our own digestive enzymes are incapable of degrading these without the support of certain symbiotic microorganisms. Callicott's claim of mutual dependency gains strength in the observation that without microorganisms, particularly certain bacteria, life as we know it on this planet, including our own, would simply not exist.

In an evolutionary perspective, certain microorganisms and some of their bacterial-genetic components were necessary for us to evolve. Through the process of our evolutionary development, we acquired beneficially symbiotic bacteria. If these microorganisms should for some reason depart or be imbalanced to the extent that their function is impaired (*e.g.* as in the case when antimicrobial drugs are ingested because they initiate a reduction of normal bacteria in the gastrointestinal tract) then our species at worst would no longer exist or at best our function would be impaired. A similar analogy can be made to planetary sustainability.

However, while this enhances his claim reinforcing ecological / biological connections in the biotic community, the question of the moral still remains. This is the Humean is / ought dichotomy and G. E. Moore's *naturalistic fallacy* both of which appear to be examples of obstacles to a traditional philosophical basis for an environmental ethics.[70]

I have supported Callicott's argument concerning the ecological unity of the biotic community. Whilst I understand the need to preserve what he refers to as 'wild nature', as noted earlier, I do not consider any of nature as still 'wild'. He naturally refers to areas and inhabitants still *appearing* unspoiled by human encroachment. Pollution, acid rain, rivers, tides and currents, like drug-resistant microorganisms know no boundaries. Since drug-resistant microorganisms have been identified in gorillas in the supposed wild, then this also indicates human influence (Alexander 1999). Yet, his point is that we should preserve and protect what is possible of 'wild' nature, a valid concern. Against Passmore, the symbiotic relationship of humans and microorganisms identifies common interest if 'living' is construed as a bacterial 'interest'. In the sense of reaching a *telos* (as the *telos* of the acorn is to become an oak tree), we might concede that microorganisms exhibit the 'will' or the instinct to live and share a common interest in living. However, that they 'desire' to be left alone, as Callicott asserts is a contentious issue. One reason for this is that 'desire' is generally considered as a cognitive function, one beyond the capacity of microorganisms.[71]

Passmore's claim concerning only humans as capable of generating ethical obligations also deserves a turn. From an anthropocentric perspective in *Attitudes to Nature* (1995: 140), he asserts that we do not need a 'new' ethics to justify our degradation of the environment. He argues that we must reject our ill-conceived biases concerning our supreme value and adopt a new moral attitude towards nature and her entities (*ibid*: 141). However, one of the problems in this view is that unless we identify a way in which to value nature intrinsically, or find another approach to the problem, the idea of her as present for our instrumental use will remain. Claims such as Passmore's tend to close the conversation and, above all, fail to respond to the need for action generated by the current ecological crisis.

Holmes Rolston III's Weak Ecocentrism

Now I turn to some views from the environmental philosopher Holmes Rolston III.

In *Environmental Ethics: Duties to and Values in the Natural World* (1988), Rolston argues for the value of individuals, species and larger wholes such as ecosystems. He writes, 'The living individual, taken as 'point experience' in the web of interconnected life, is *per se* an intrinsic value' (*ibid*: 100). In this conception, the value of the whole – at least the whole of life – is greater than and prior to any of its parts, specific roles, niches and relations within the ecosystem. The value of the *whole* of life receives first priority and this value is attached to the life of *all* its individual members. Concurrently he argues equally for the value of species or kind. This is configured as first, value to the life system, then follow species, then individuals. In other words, the whole is greater than the sum of its parts. He (*ibid*: 187) writes: 'The system is a web where loci of intrinsic value are meshed in a network of instrumental value'.

In this conception, intrinsically valuable individuals become instruments from a systematic point of view. This is because the system itself is 'of value'; it is 'productive' because of the distinctive values within it as represented by species and individuals. Rolston (*ibid*: 101) tells us that each organism has 'a good-of-its-kind; it defends its own kind as a good kind'. He (*ibid*) explains this further writing:

> *In organisms, the distinction between having a good-of-its-kind and being a good kind vanishes, so far as any faulting of the organism is concerned. To this extent, everything with a good-of-its-kind is a good kind and thereby has value.*

Further, as Rolston implies, without much thought, we commonly label entities as simply 'good' or 'bad' relative to our human interests. However, as he (*ibid*) identifies, during the course of their normative expression, if an entity damages an ecosystem or causes widespread disease, then we can judge the entity as a 'bad kind'. Yet the entity it retains its status as a good-of-its-kind (*ibid*: 102).

Applying this to microorganisms carrying the trait of drug resistance then, whilst the normative *methicillin-resistant Staphylococcus areus* is a good-of-its-kind (because it is a spontaneously evaluative system), it would be in this conception a bad kind *instrumentally*. This is because of the role the mutable trait of drug resistance plays in damaging the ecosystem.

So far, we may see the outline of Rolston's argument as it applies to microorganisms carrying the trait of drug resistance and well we may concur. He (*ibid*) continues to argue that:

> *Even in "bad" cases there is value present in the offending organisms value which, although it clashes with ours, is morally significant merely because the organism is a spontaneously evaluative system.*

Rolston (*ibid*) reminds us that as good kinds, all of organic life is dependent on situated environmental fitness, on this we may agree. The parameters of this environmental fitness are bounded by an organism's particular genetic make-up as well as natural selection. He asks to reconsider the labelling of organic life forms as 'good' or 'bad' from a perspective beyond that of its 'victim or competitor' (*ibid*). (This is reminiscent of Dubos' assertion that I noted on page 60.) In this reasoning, the good of *methicillin-resistant Staphylococcus areus* would be judged not in the context of its possible harm (*e.g.* the unsuccessful management of a disease process due to methicillin resistance). Rather, we are asked to envision the overall good of the 'offending' organism upon the ecosystem (*ibid*).

One of the examples he uses to illustrate the overall good to an ecosystem of a supposedly 'bad' kind is the microorganism *Chlamydia*, implicated in a trachoma outbreak in bighorn sheep in Yellowstone National Park[72] during 1981-1982 (*ibid*: 53, 66, 102). His example concerns, in part, the rangers' decision not to interfere with what they perceived as 'wild' nature and in the context of drug resistance, I suggest, supports an argument for the prudential use of antimicrobials. Rolston (*ibid*: 103) writes:

> *The Chlamydia microbe is a bad kind from the perspective of the bighorns, but when one thing dies, something else lives. After the pinkeye outbreak, the golden eagle population in Yellowstone flourished as never*

observed in recent times, preying on the bighorn carcasses. For them Chlamydia is a good kind instrumentally ...

Responding to Rolston, I am unsure if this is entirely correct in light of new information. As we recall, Rolston published this work in 1988 and the epidemic took place 7 to 8 years earlier. Space does not permit a detailed explanation concerning the complex discussions — including new taxonomy and genetic findings - that surround the group *Chlamydiaceae*[73] of which *'Chlamydia'* exists as a species (Mayr and Provine 1998). Suffice to say that the etymology of the word *Chlamydia* was well chosen; *chlamys* is the Greek word for "cloak draped around the shoulder" as indeed, much of its life cycle and development appears shrouded.

The particular *'Chlamydia'* species in Rolston's reference *may* have been caused by the bacterium *Chlamydia trachomatis*, that, like others in the family it is a parasite, surviving only by growing inside other living cells. On the other hand, it may have been caused by another species or strain because although once assumed single-host microorganisms, the *Chlamydiaceae* now have evolved to infect multi-hosts and cross-species barriers.

It is now recognised that over 60 strains of it can infect most birds and mammals (including humans) (Everett 1999). In humans, one species is linked to eye infections that if unattended may lead to blindness (trachoma), sexually transmitted diseases and respiratory diseases. In animals, some species of *Chlamydia* cause respiratory disease, conjunctivitis, arthritis, enteritis and reproductive failure. In birds, some species produce a generalised infection resulting in lethargy and sometimes death (Bavoil *et al.* 2000). Mammals and birds can transmit members of *Chlamydiaceae* to humans, and vice versa. Certain species appear to infect sheep, goats and cattle causing chronic infection in reproductive tract resulting in spontaneous abortions. Yet, since cross-species transfer is now increasingly implicated, it may become difficult (unless sophisticated laboratory measures are used) to say exactly which species is implicated in a disease.

There are two points I wish to consider. First, within the confines of natural selection, genetic propensities and subject to environmental stress, when a sexually reproductive population (of anything) radiates into different habitats in which subpopulations (strains, sub-species, isolates) become relatively isolated, they follow unique pathways to eventually form new species. This is the basis of what is termed allopatric speciation. For example, it was thought that for every species of *Chlamydia*, there was a related and at least a more-or-less specific host (at least as far as the idea of a common ancestor is concerned). The common ancestor of *Chlamydiaceae* (it was believed) must have lived in an ancient mammal or bird because it was thought that they only could be found in warm-blood vertebrates. Now they have been isolated from lower vertebrates and even from invertebrates as multi-hosted (Moulder 1988; Everett 1999). The ways in which this occurs have relevance to the discussion.

One way of cross-species or multi-host change might be the chance infection of a new host, a subsequent adaptation to it and the emergence of an isolate or variant still showing characteristics of the parent species but maintaining a genetic preference to this new host (Woolhouse *et al.* 2001). Or the microorganism could select for traits that engender the crossing of species barriers without any specific host adaptations. As Bell (1997) reminds us, it is an axiomatic in evolutionary thought that specialists over generalists prevail.

Although microbiologists and geneticists are not sure of all the mechanisms involved, like the trait of drug resistance, the ability to adapt to many hosts is a positive evolutionary property. If, as I argue, this is the case, then it is plausible that selective pressure is placed on current single-host pathogenic microorganisms to evolve with the traits necessary for multi-host cross-species disease transmissions. A report by Bush and Everett (2001) concluded that virulence phenotype is more strongly influenced by specific adaptation to new habitats than by ancestry. This has obvious relevance to my overall argument that all human activities that result in adverse environmental changes bear impact not only locally, but affect the whole of life-systems.

Now, in Rolston's (1988: 53) account, the bighorn sheep had *Chlamydia* (we do not know the species). Following their demise, golden eagles fed on their carcasses and flourished (*ibid*: 102). In doing so, in the light of new information, it is possible that the golden eagles acquired *Chlamydia*. Amongst other facts concerning this versatile group is that all of the different species appear to lie dormant in their hosts for long periods. When conditions become stressful, they are 'activated'. This can be correlated with the human model. For example, *C. trachomatis*[74] was (and perhaps even now still is) generally considered a human disease that typically occurs and rapidly spreads under crowded and poor hygienic conditions. Questions arise. Was the *Chlamydia* outbreak in bighorn sheep per Rolston's account triggered by some stress within the sheep population? Perhaps climate changes triggered untoward population growth, perhaps the rain and wind patterns adversely changed facilitating a new environment for microorganisms to grow and flourish – these things we do not know, but we do know that we are responsible for most systemic changes. Knowing this, do such factors support an account that supposes that if left alone, nature 'takes care of herself'; natural predatation 'sorts things out'. Rolston (*ibid*: 183) argues this point writing:

> ... *Thus, Yellowstone bighorns should be "left on their own" to combat the Chlamydia microbe; San Clemente plants should be "left on their own" in their island ecosystems, even though this requires that they be protected against feral goats that humans introduced, disrupting the ecosystem'.*

Against Rolston, I suggest that a deeper exploration into microbial life informs the picture. If plants are protected against human-introduced feral goats, then why are bighorn sheep not protected against harmful microbes? Like the introduction of feral goats, we, in disregarding nature, have facilitated changes in microbial life; we have made favourable the conditions necessary for the rise of new organisms, new infectious diseases. Thus, I suggest, there are times when we should interfere with nature, at least on a case-by-case basis. I suggest it is not possible in the light of new information to make the claim that it was the right decision on the part of the ranger's not to treat the initial outbreak[75] (*ibid*: 53, 183).

From examples presented from the world of microorganisms and emphasising the connectedness of life systems I have previously argued that there is no natural or 'wild' nature – no matter how remote or pristine – which is not the same as saying that such areas should not be treasured and protected. Rolston's (*ibid*: 103) assertion that: 'Something many be a good kind intrinsically but a bad kind instrumentally in the system; these will be anomalous cases, however, and soon edited out' I suggest is a bit too hopeful. Microbial life is, like all life, subject to evolutionary 'misfits' that will be unable to compete in their niches and thus die. However, because of their far greater population in terms of numbers (as compared to that of human, animal and plant) the microbial 'misfits' that succumb represent a very small minority. The numbers and adaptive potentials of microorganisms place them in an advantageous position over all other life forms.

Delving into the world of the *Chlamydiaceae* adds support to my argument that it is right to interfere with nature at least on a case-by-case basis. In the 1980's the genome of the *Chlamydiaceae* was not mapped – this is still an on-going project – and it was not known then that species within the order had the ability to cross-species transfer or could be multi-hosted. So we may ask, when consuming the carrion of the bighorn sheep, did this opportune (whatever) *Chlamydia* species to acquire a new host and thus potentially evolve into a new species. Moreover, as a wild bird, the golden eagle following its natural behaviour, could have in flights and forages, shed in its droppings *Chlamydia* which conceivably could wait for another host. Did the carcasses of the bighorn sheep decay into the earth leaving in their detritus *Chlamydia* microorganisms to be acquired by for example, a passing rodent? To such questions, there are no immediate answers. Yet, armed with new knowledge, is it still right not to interfere with nature, as in not treating the Chlamydial infection of the bighorn sheep. I would argue 'No. Not now and perhaps not even then'.

Although there have been reports of resistance to antimicrobial-based ophthalmic ointment (Ayres 1998), my argument has never intended to infer that antimicrobials should not be used – only that they should be used prudentially and appropriately. I suggest that the risks involved in admitting another *Chlamydia* species to nature far outweigh the risks involved in the initial treatment of the outbreak (the possibility of drug-resistance). Yet, admittedly, this is a superficial response to a particular incident. The deeper problem remains – one concerning our attitudes towards nature and her creatures for it is we who have placed stress on natural evolutionary processes.

As I understand Rolston's argument, there is no absolute obligation to protect human life and there is no absolute obligation to protect species, particularly at the expense of the ecosystem. On this view, the system, while admittedly undergoing radical changes over the millennia, has resulted in more 'individuals and species that are filled into their communities' (*ibid*: 185). This we may accept. Furthermore, we may agree with Rolston that nature's system and her processes were / are faced with natural events such as 'at least five catastrophic extinctions'. 'Following each', as he (*ibid*) reminds us, 'they were followed by swift resurrections, often with novel and advanced forms (of life)'. He (*ibid*) then makes the claim that these phenomena indicate 'the rationality of the system, including trial and error'. And all of these were naturally occurring processes that resulted, as he has indicated, in the evolution of new life forms. However, such catastrophes were naturally occurring.

Drug-resistant microorganisms, as a human imposed catastrophe, affect the system, its products and its process. On the other hand, *Chlamydia* species are naturally occurring entities. *Chlamydiaceae,* at least based on current available knowledge, appears to be far more versatile in its evolutionary adaptation and change than assumed in the 1980's. In the sense of naturally occurring organisms, they still contribute value to the total value of the natural system. They may be harmful to other species members or instrumentally 'bad kinds' but as naturally evolved life forms, they are 'intrinsically good kinds' (*ibid*: 102). Mot likely we influenced the evolution of the *Chlamydiaceae* by giving them new opportunities to follow their natural genetic propensities because of the stress we place on other living entities and environmental systems. A point this comparison raises is that, be it from human design or human influence, because of our detrimental environmental actions, we profoundly affect natural systems.

Rolston may argue that the introduction of the trait of drug resistance to microbial life – although human imposed – does not change the value of the natural system. In the sense that natural selection is a naturally occurring process, he is correct as microorganisms may select for the trait of drug resistance because it is advantageous. The fine-line differentiation remains in that we forewarned, abused and misused antimicrobial drug therapies and in so doing in the space of two human generations, changed the evolutionary pattern of microbial life. The trait of drug resistance, while an intrinsic value to microbes, is instrumentally a bad kind to humans, plants and animals. It is the extent to which drug resistance may affect the overall well-being of living entities that remains conjecture. But the fact remains that the natural system is altered by the cataclysm of drug resistance, as an unnatural catastrophe. Therefore, it is no longer a *natural* system as the changes were not of natural design; it is ruptured. The natural system, including its 'rationality, including trial and error', if considered in the sense of entities following their natural evolutionary processes remains of value, but inexorably altered.

Concluding Remarks

In this chapter, I have presented some nonanthropocentric environmentalist perspectives from different philosophers. We might be reminded of why some feel that distinctly environmental ethics is needed. Sosa (1996) for one considers the anthropocentric stance insufficient. He points to greed as an example saying:

> *When greed directly harms other specific and proximate human beings, traditional ethical theories do consider it bad. Greed, however, may benefit human beings in the aggregate by creating jobs and stimulating economic growth while harming the environment (by causing, say the extinction of an economically worthless species) ... While traditional*

ethical theories may well condemn the immoral distribution of industrial goods, few if any will condemn the guiding principle of industrial civilisation. But today the consequences of that guiding principle are proving to be environmentally untoward ... The widespread impulse to extend the boundaries of our moral community beyond the scope of human interaction and grand moral status to nonhuman beings suggests that traditional ethical theories inadequately express the present, more fully involved condition of our moral sensibilities (*ibid*: 51).

As the exploration into the problem of drug resistance has shown, misguided worldviews concerning the value of nature have underlined our general tendencies to consider her as a never-ending commodity. We inflict harm upon nature and so change her. Concurrently we rely on technological 'fixes' to remove us from the very problems we have created in the first place. For example, one major factor in the rise in emerging infectious diseases is due to our need to develop new habitats because of our expanding population. To remedy the ills of newly infectious diseases, we rely on new antimicrobial therapies. So, the combination of disvaluing nature as well as our reliance on technological 'fixes' as remedies for the harms we inflict upon nature equal an untenable situation for us both. The bottom-line, I argue lies in an ill-conceived worldview. This calls not only for a shift in worldview in which we can value nature, but also for ways in which we can remedy the damages we have already inflicted upon her.

In the few examples I have shown, we can see that environmental ethics is multifaceted. One may be puzzled by the passionate and multidirectional arguments made by these positions in environmental ethics. Importantly, because of the relative newness of the discipline, the field remains fluid in the sense that as the debates grow they also attain maturity and clarity. While their differences may be large, what they have in common is their varying levels of distrust of anthropocentrism. Human chauvinism is their common enemy. For some, like biologist Ehrenfeld, the struggle is against humanism (in the sense of exclusive concern for only what is in narrow human-only interest). Others focus largely on ways in which intrinsic value can be theoretically grounded for nonhuman entities.

Yet, from a purely theoretical point of view, there are obstacles to the adoption of an extension of moral considerability and moral significance to the biotic community, as highlighted by Elliot (1998: 12):

> *(If) there is agreement about which kinds of things are morally considerable (having a claim to be taken into account in moral consideration) .... there is almost certain to be disagreement about their degree of moral significance (a degree of considerability).*

In other words, even if one accepts the meta-ethical view that there are degrees of moral standing (Moore and Bruder 1996: 264), it remains unclear how differently one may treat entities according to the level of moral standing we humans allocate.

Taylor's account provided good grounds in which to argue against the equal value of all living entities. 'Life', as his overriding principle of value, grants to all living things equality. In such an egalitarian perspective, we would not be able to treat diseases or attempt to control drug resistance since all entities are equal. In his account, the admission of moral agents into his discussion was not of much assistance as, although we as moral agents are given permission to destroy harmful entities, it is a constrained destruction.

I overviewed briefly Callicott's position principally in terms of his support for Aldo Leopold's land ethic. I looked at Callicott's argument in a different perspective, objecting to his notion of 'wild' nature while recognising that its usage is from the perspective of preserving what we consider as 'real' nature. In a text from Callicott presenting an argument from Passmore, I supported in part Callicott's claim of mutual ecological dependency. I objected however to his use of 'desire' in the claim that all living forms have interests. I find it conceptually difficult to accept that microorganisms have 'desires', which for me is subjective psychological labelling.

I discussed Rolston's example of *Chlamydia*, used to support his claims that we should not interfere with natural life processes as well as his use as an example of the intrinsic value of living entities as good-of-their-kinds intrinsically but viewed as 'bad' kinds instrumentally. I suggested that in light of new knowledge, that 'interfering' with nature remains on one level a problem of overall balancing risks and benefits. On a deeper level, the problem of our interfering with nature is related to our particular views about nature. By this, I mean that if we accorded value to nature in the first place, we would not face many of the environmental dilemmas we find today. Specific to Rolston's claim that value is generated by the system, I admit that if this is construed in the sense of evolutionary natural selection then the system retains value. However, as the trait of drug resistance represents an unnatural imposition, I suggested that the value of the system is altered.

If we were to assign a single word for each of the representative environmental positions I have discussed, the words 'respect', 'preservation', and 'value'[76] would immediately come to mind. In the final chapter, I will suggest that the enlightened anthropocentric approach presented by Bryan G. Norton provides a promising foundation upon which we can unite such concepts and address further moral responsibility and the problem of drug resistance.

Chapter 7: Informing the Human-Nature Debate: Bryan Norton's 'Enlightened' Anthropocentrism

That systems can be made ill, and that human causes of this illness raise moral issues are, after all, the core idea of the land ethic! (Norton 1991 b: 215).

Introduction

Anthropocentrism is the philosophical perspective asserting that human needs and interests are of the highest moral importance and for some strong anthropocentrists, even of exclusive value. In response to this, in the previous chapter, I presented some nonanthropocentric arguments in environmental ethics. As noted, the representative theories focused on various ways of extending intrinsic value to nature.

Now, I will turn to the argument of Bryan Norton (1995) in which he advocates for the acceptance of his 'weak' anthropocentric approach to environmental ethics. Here it should be noted that although Norton initially termed his approach 'weak anthropocentrism' (as in a weaker form of anthropocentrism), it has changed and is now commonly referred to as 'enlightened anthropocentrism' (Norton 1991 a; Brennan and Lo 2000; McKenna and Light 2004). This is because it more precisely captures his pragmatic philosophical position, the emphasis on 'enlightened self-interest, and avoids the somewhat debatable connotation of 'weak' anthropocentrism. I will however, use the term 'weak' as quoted in his texts.

First, I will overview his argument and then from it pull out some salient points in the context of global drug resistance. Using some examples, I will show aspects that I consider positive and negative in the application of his theory. Overall, I conclude that Norton's enlightened anthropocentric approach to environmental ethics provides so far the best means by which it is possible to unite both human concerns with that of the environment.

Then I will turn to restate the problem of global drug resistance and make an appeal to educators to include environmental concerns in all their curricula. I identify that in teaching biomedical ethics, some of Norton's conceptions make the inclusion of environmental concerns possible, but there are still some impasses to overcome.

Norton's Argument

In *Environmental Ethics and Weak Anthropocentrism* ([1984] 1995) Bryan Norton puts forth an argument saying that his enlightened anthropocentric approach fulfils the need to address the environmental degradation faced in our modern world. Norton is a philosopher, an environmental conservationist and preservationist. He is actively involved in environmental policy formation in North America, particularly the USA. An overview follows.

1. Norton's argument for an enlightened anthropocentric environmental ethics:

1.1. One needs not to allocate intrinsic value (*i.e.* independent of human value) to non-human entities to ground an adequate environmental ethics.

1.2. Enlightened anthropocentrism informs an adequate environmental ethics in the following ways:

i) One should eschew the problem of intrinsic value for non-humans for it is not needed to ground an adequate environmental ethics.

ii) One needs not to appeal to intrinsic value of non-human entities to formulate a worldview that is rationally defensible. It is not needed to allocate intrinsic value to hold to an 'ideal of maximum harmony with nature'. A rationally defensible worldview must be supported by science, metaphysics (*i.e.* the understanding that the value of non-human entities enlightens human values in terms of felt preferences, and the understanding of the distinction between felt and considered preferences), and aesthetic and moral ideals. A rationally supported worldview is consistent only with considered preferences, *i.e.* desires or needs expressed after 'careful deliberation'.

iii) One should emphasise the value of nature as a source of inspiration in value formation (*i.e.* values are formed and informed by contact with nature).

1.3. In this way, an enlightened anthropocentrism offers 'a *framework* for developing powerful reasons for *protecting nature'* (1995: 184) (my emphases added).

2. An adequate environmental ethics is distinctive by being non-individualistic (individualistic ethical theories focus on the

impermissibility to harm other individuals; they prohibit behaviours that have negative effects upon the present and future environment and on human or non-human individuals). A new enlightened anthropocentric (weakly anthropocentric) approach to environmental ethics is offered as a valid alternative.

2.1. Two characteristics of this ethics:

i) It retains all value in human loci: the focus is shifted from theoretical attempts in environmental ethics to assert or presuppose that nonhuman natural entities have value independent of human value to maintaining intrinsic value in humans based on human 'preferences'.

ii) It is non-individualistic: value is not restricted to the satisfactions of felt preferences of human individuals.

2.2. This ethics exhibits two levels:

i) The distributional level (distributive fairness guiding behaviours affecting other human beings' use of the environment); the felt preferences of individual humans have *prima facie* equality, therefore, one ought not to harm other human beings unjustifiably. Furthermore, individuals should be treated fairly regarding benefits derived from the environment and from other sources.

ii) The level of allocation: Current generations have the obligation to maintain a stable flow of resources necessary and available for us and for ongoing *human* life (and take steps to provide suitable substitutes for non-renewable resources). This should be achieved through the maintenance of the 'integrity and health of ongoing ecosystems as holistic entities' (*ibid*: 188). This does not imply that ecosystems should be kept stable, but rather entails a wise use of resources.

To avoid the possibility that i) (distributive fairness) could damage ii) (allocation), we have to consider the 'ideals' of the continuation of human life and of life 'in harmony with nature'. In other words, our felt preferences should be guided and redirected if needed by an enlightened rational worldview of ideals, values and principles regarding the human species' relationship to nature. Now I will turn to specific points in his thesis.

First, let us ask why Norton feels it necessary to differentiate between strong and weak anthropocentrism. As mentioned above, for him it is misguided to centre on attempts to attribute intrinsic value in or to nature. Rather than searching for intrinsic value in nature, he asserts we can work around this concern whilst maintaining a form of anthropocentrism. He (1987: 12) writes:

> *Weak anthropocentrism is a form of anthropocentrism because it attributes no intrinsic value to nonhuman species.*

Norton (1995: 183) in particular cites Callicott's nonanthropocentrism, in which he argues that the *source* of value is human. However, the *loci* of fundamental value may be found in the non-human.[77] Norton (*ibid*: 184) disagrees, writing:

> *... harmony [with nature] need not be attributable to the value of natural objects, nor need the prohibitions implied by it be justified with nonanthropocentric reasoning attributing intrinsic value to nonhuman*

> *natural objects. They can be justified as being implied by the ideal of harmony with nature. This ideal in turn can be justified either on religious grounds referring to human spiritual development or as being a fitting part of a rationally defensible worldview.*

Instead of attempting theoretical approaches based on intrinsic value, Norton considers that if the concept of an interest is developed then the need to identify intrinsic value in nature may be set aside. And he sets it aside without any further discussion!

I am unconvinced concerning his dismissal of attempts to identify ways in which intrinsic value can be found in nature. If his point is to commit us to action (as in practice) concerning environmental degradation and not to be obsessed with first finding a way to locate or source intrinsic value in nature (as in theory), then I take his concern. On the other hand, if environmental philosophy is to be considered a *legitimate* branch of moral philosophy, which I think some philosophers feel is desirable, then I can see no obstacle in simultaneously pursuing this goal. Nonetheless, Norton sets out to define what counts as human interests. When that is accomplished, he argues, we will be in a position to distinguish between a strongly anthropocentric value theory and one that is weakly anthropocentric (*ibid*). In doing so, it will become clear to us that 'nonanthropocentrism is not the only adequate basis for a truly environmental ethic' (*ibid*: 182).

Norton then distinguishes between the terms strong anthropocentrism and weak anthropocentrism. The former, he asserts, is based on the moral validity of the satisfaction of human 'felt preferences', broadly our wants and desires. He identifies that our personal experiences, interests, wants and desires influence the ways in which we perceive nature and the environment, often in unrecognised ways. Such personal *filters*, if you will, influence our worldviews. We could extend this and say that these personal filters are likewise influenced by collective filters. Collective filters define a shared way of viewing the world which in turn constitutes social paradigms. Such filters often come into play without our deliberation as felt preferences. They screen what we consider worthwhile, what is not, what is desirable, permissible and right and what is undesirable, unacceptable and wrong. In other words, such filters are shaped by uninformed human-centred values which also shape the more or less explicitly stated principles we use to define reality.

Weak anthropocentrism, Norton argues, asks us to recognise the moral validity of what he terms 'considered preferences' (*ibid*: 183). These are roughly our basic needs. Considered preferences, as the term applies, require a method of careful deliberation that relies on the adoption of a rational and enlightened worldview, thus it transforms into an 'enlightened anthropocentrism'.

This worldview is informed by three major factors: fully supported scientific theories, a metaphysical framework as means to interpret such theories (that is the understanding and acceptance of the distinction between felt and considered preferences), and a set of rationally supported aesthetic and moral ideals (which he does not specify) (*ibid*).

Norton believes that his enlightened anthropocentric approach to environmental ethics provides a framework in which we may criticise value systems that are purely exploitative of nature in a manner not available to a strong anthropocentric perspective. Strong anthropocentrism, he claims, has no means to check upon 'interests' (felt preferences) and concurrently no means by which to criticise environmentally detrimental actions (*ibid*: 184). He (*ibid*) asserts that an enlightened or weakly anthropocentric position makes criticism possible. This is because it recognises that our felt preferences can be either rational or irrational if judged in the context of an enlightened rational worldview. Value systems that exploit nature are thus open for criticism.

Norton (*ibid*) claims that in affording this view, two ethical resources become available to environmentalists. The first is dependent upon the case environmentalists can make concerning the positive and necessary relationship between humans and nature. Secondly, considerations of felt preferences are not negated in the process of value formation. In his construction, the process of value formation arbitrates between felt preferences and considered ones. If environmentalists are able to show that values are informed and formed by contact with nature, as opposed to nature being viewed as a commodity, then nature becomes an important source of teaching, of inspirational value (*ibid*).

To achieve these goals, Norton outlines the task of an environmental ethics writing:

> *The initial task of an environmental ethic is to state some principles from which rules can be derived proscribing the behaviours included in the set which virtually all environmentally sensitive individuals agree are environmentally destructive* (*ibid*: 182).

In doing so, he points unknowingly to the major problem in global drug resistance that as far as I am aware, I am the first to consider as an environmental problem. I have shown both the ways in which environmental problems contribute to the rise of emerging infectious disease and drug resistance and simultaneously how drug resistance threatens the stability of the global environment. The imprudent use of antimicrobials resulting in the introduction of massive amounts of drug resistant microorganisms into the environment is a new problem. Although it intersects with the conservation and preservation of nature, it has its own particular identity, one not encountered commonly as an 'environmentally destructive practice'.

Interestingly though, when we look at Norton's 'specific types of behaviours that do or would do damage to the environment' (*ibid*), we see that the problem of drug resistance shares the same specific behaviours he identifies as environmentally destructive, namely those found in 'industry, medicine and the environment' (*ibid*). He (*ibid*) also points to sub-behaviours that do or would do damage to the environment. Norton's examples include population pressures and environmental degradation, both of which I have shown to be contributing factors to emerging infectious disease and global drug resistance. Such behaviours, he claims must be agreed upon by environmentally sensitive individuals as destructive practices in order to proceed with the application of his thesis.

Now, to proceed with this analysis, I need to make two assumptions. The first is that I have shown that the problem of global drug resistance is an environmental problem.[78] It is important here to note that Norton (1991 a) classifies environmental problems as first, second and third generation. These types of problems are to be considered as progressive, intertwined and superimposed in and upon all others (*ibid*: 213). First generation environmental problems concern the use and abuse of natural resources including the wastefulness incurred. These types of problems are more focused and localised. For example, a first generation environmental problem would be a specific threat to a particular species such as the spotted grey owl or the snail darter fish. Second generation environmental problems are those which concern larger systems. The penetration of pesticides into larger systems as Carson (1966) noted, or the affects of pollutants are examples of second generation environmental problems. Third generation environmental problems as Norton (1991 a 213) writes:

> *... represent effects of changes in intermediate sub-systems, such as the atmosphere over cities and tropical forests, on yet larger systems, such as the global climate.*

Global drug resistance represents a third generation environmental problem. This is because, in applying Norton's thesis, it moves beyond simply a strongly anthropocentric concern. It is a globally interrelated problem affecting the entire biotic community; it 'plagues the beleaguered biosphere' (*ibid*: 214).

Second, I assume that the readers are environmentally sensitive persons who agree that drug resistant microorganisms themselves are at least potentially environmentally destructive. If this is the case, then, as he puts it, 'we can proceed to state some principles from which rules can be derived that will proscribe behaviours that we agree are environmentally destructive' (*ibid*).

Then Norton tells us that a further task is involved; we must refine an environmental ethic. This involves

> *... moving back and forth between the basic principles and the more or less controversial behaviours, adjusting the principles and /or rejecting intuitions until the best possible fit between principles and sets of prescribed behaviours is obtained for the whole environmental community* (*ibid*).

Let us now broadly enlighten the problem of global drug resistance. Although it was known that in the application of antimicrobial therapy some drug resistant microorganisms would necessarily evolve, no prudential measures were put into place concerning its control, uses and distribution. Our global felt preferences about antimicrobials concerned the alleviation of human infectious disease and other activities that supported human needs and desires. In this conception, the value of antimicrobials is determined solely by human-centred activities. Framed from a human-centred perspective, the uses of antimicrobials appear to be rational. So we might ask Norton why we would even need to criticise it.

In a strong anthropocentric perspective, human concerns are the only relevant concerns. Since the focus is only on human concerns, any considerations beyond this are excluded. Therefore, no criticism is possible for questioning activities that affect a wider world. This is unacceptable for Norton. In keeping, while maintaining considerations of our human needs and desires, he asserts we must broaden our perspectives looking at our actions and criticising them in light of new information and inspiration. This will lead us to broaden the parameters of our human-centred morality as we seek the ideal of an enlightened worldview which, by definition, includes concern for our environment. Thus, we are obliged to criticise our activities looking beyond ourselves to a 'more fully informed condition of our moral sensibilities' (Sosa 1995: 51). This is the essence of Norton's idea of such an ideal worldview.

Thus, returning to our example, our felt preferences (antimicrobial therapy as the means by which to experience relief from the effects of infectious diseases and other human-related needs and desires) determined their value. Now we are obliged to inform our felt preferences by a larger body of knowledge. So we must consider that whilst our felt preference remains as a rational choice (*e.g.* taking antimicrobial therapy when needed), we must consider its extended ramifications, such as our obligation to adhere to a prescribed regimen. This may extend to include other arguably less rational felt preferences (*e.g.* the uses of antimicrobials in the food and animal industry, effects of sub-optimal therapy, counterfeit trade in antimicrobials, and unnecessary demand for antimicrobials).

Now, importantly, environmentalists then would be obliged to inform the choices of antimicrobial usages showing how a felt preference when conjoined with adverse and unnecessary needs and desires skews the sustainability of the environment (*e.g.* imposing upon nature the uncontrollable survival trait of drug resistance held by *supra-natural* life forms which 'travel' and affect the environment in ways we do not fully understand). In admitting such criticisms (which could be extended further), we see how nature becomes a larger part of our world and, as such, we are obliged to see how our actions may adversely affect her. This is the major strength of accepting Norton's argument. Now, let me apply his theory to practice.

Putting Norton's Enlightened Anthropocentrism to the Test

If individuals do *not* have a bond with nature, be it from rational or spiritual sources, then it is easier to see the merits of Norton's approach. This is because through criticism, our range of understanding the human-nature bond is expanded. In keeping, making their case for connectedness with nature, it falls upon the environmentalists to present information and arguments in a manner that is comprehended by individuals.

In this section, I am interested in seeing how felt preferences might be mediated by considered preferences when people *already* have a worldview that includes a bond with nature serving as their moral guide. I use this type of construction rather than ones that do not have an existing bond because it identifies a weakness in Norton's argument. While he tells us our felt preferences are our moral guides, my objective is to show that individual contingencies can and do affect moral choices rather or more than enlightenment. I will show that depending on the internalisation, interpretation and creativity of the moral agent, such factors can lead to different moral decisions.

Norton, in the process of value formation, points to the attributes of nature – how they are necessary, vital and inspirational to our lives.[79] He (*ibid*: 189) claims for example that, 'experiences of nature can lead to a rejection of overly materialistic preferences'. If these are rejected then the ideal of humans living in harmony with nature can be established. So our further task would be to enlarge upon worldviews that promote this ideal.

Norton (*ibid*: 184) provides examples of well-developed worldviews found in Thoreau[80] and the Jains. Let me turn to the latter. He (*ibid*) writes: 'Hindus and Jains, in proscribing the killing of insects, show concern for their own spiritual development rather than for the actual lives of those insects'. Indeed, Schweitzer (1987: 109) affirms this writing:

> *... Indian piety recognises but a single world-principle. They are monistic and pantheistic. Their worldview has to solve the problem of how far we can recognise the original source of the world as ethical, and how far, correspondingly, we become ethical by the surrender to it of our will.*

The *unenlightened* Jain: "I live in India and I am a Jain. I have contracted an infectious disease. I experience the process of being ill. So my felt preference, my need and desire, is to experience wellness. The cost of a full treatment of antibiotics is 10 rupees but I have only 5 rupees. Nonetheless, I buy what I can in the hope of a cure."

Now let us expand this example using Norton's enlightened anthropocentric approach practically *enlightening* our Jain.

"I live in India and I am a Jain. I have contracted an infectious disease. My felt preference, my need and desire is to experience wellness. The cost of a full treatment of antibiotics is 10 rupees but I have only 5 rupees. As an 'enlightened' Jain, I am aware that certain microorganisms are the agents of my disease. I know that they are living entities. According to my worldview, I am proscribed from harming living entities. In the context of an informed worldview, I know that if I take a sub-optimal treatment I will also contribute to an increase in *supra-natural* life forms. These will ultimately adversely affect other life forms. Likewise, I know that if I take an optimal course of the therapy whilst I will still contribute to some *supra-natural* organisms into the environment, I will not contribute to as many as if I took a sub-optimal treatment. But in the ingestion of the drug, I will still be killing some life forms. So in considering my preferences, in the context of an enhanced worldview, I am obliged not to take the antibiotics and suffer the consequences of the infectious disease."

Our Jain had a felt preference: the need or desire to experience wellness. In keeping with Norton, we have informed and enlightened his felt preference inducing a considered preference. As per Norton (*ibid*: 183), necessary to this is the provision of information in the form of fully supported scientific theories, a metaphysical framework as means to interpret such theories (felt preferences as informed by considered preferences) and a worldview that includes a set of aesthetic and moral ideals. Our Jain already holds a worldview found in his belief system that includes moral ideals. The problem as I see it is that dependent on the force of a foundational worldview (in this case adherence to this Jain's spirituality) conflicts may arise. So conceived, felt preferences acting as moral guides may, when enhanced by an enlightened worldview, present a conflict with Norton's claim of maintaining the primacy of human life. Yet, if our Jain was to be ethical in the context of his spirituality, he had no choice but to surrender his will to that of his belief system. So we might ask Norton: when a worldview conflicts with information that would influence our considered preferences, is the worldview overriding?

Framed in the context of global drug resistance, my point is to show that the realisation of considered preferences spins off in different ways from person to person, context to context. In this example, the individual involved submitted his will to his greater informed spirituality. Would others do the same? Is this what is morally required? No. For Norton, the felt preferences of an individual are to be understood as its central value principles; this implies a *prima facie* equality of felt preferences (*ibid*: 189). On the other hand, considered preferences hold as their central value principle on-going human life and consciousness (*ibid*: 190).

In this example, we see a conflict between an informed worldview that is in keeping with environmental preservation but it is such that it conflicts with the very conception of its value: the continuance of on-going human life and consciousness.

There are two points I wish to consider. Although this could be equally applied to other situations in which circumstances largely influence choices, I believe my example identifies problems in informing felt preferences where, for example, drug availability is often contingent upon financial resources. No doubt, even if the adverse effects of sub-optimal treatment were known and one's felt preferences were moderated, the fact of illness combined with the fact of poverty (*viz*. the money to acquire a proper regimen) might well negate the value of considered preferences, no matter the enlightened worldview. Additionally, the extent to which an individual can internalise, rationalise and thus expand her worldview seems to be contingent upon not only knowledge but also the ability to apply creatively such knowledge to a particular situation. These I consider weaknesses in Norton's approach. However, these were hypothetical examples that led to one contrived conclusion, and now I will show how different decisions might be reached.

Let me clarify this by using the 'last man example'[81] in examples of the *unenlightened* and the *enlightened* Jain. (I will not use it in the sense of the attempt to separate anthropocentrists from nonanthropocentrists but rather to enlarge upon the idea of considered preferences.) I now turn to the *unenlightened* Jain.

Assume that a human being S, a Jain, is the last living member of *Homo sapiens sapiens*. S faces looming death caused by an infectious disease, one that because of cross-species transfer is also affecting other species. Assume that S has access to antimicrobials and knows that if he takes them his life will be saved. He knows that microorganisms are living entities and that if he ingests antimicrobials he will be killing a life form. His life experience, his worldview, consists in a single belief system: the injunction not to harm living things. Thus, he feels he has no option but to die from the infectious disease. He does not want to die, but he is bound by the feeling that there are no other options available.

In this illustration, our *unenlightened* Jain dies in order not to harm other living things. For him, his spirituality (worldview) is the same thing as his felt preferences. This we may contrast to the *enlightened* Jain:

Assume that a human being S, a Jain, is the last living member of *Homo sapiens sapiens*. S faces looming death caused by an infectious disease; one because of cross-species transfer, is also affecting other species. Assume that he has access to antimicrobials and knows that if he takes them his life will be saved. Assume that S has learned that, in addition to insects and other living entities, microorganisms are life forms. Assume that he also has been taught the consequences of drug resistance — each application increases the number of *supra-natural* drug resistant microorganisms into the environment.

Moreover, assume that in the environment the microorganisms' sensitivity to this drug is almost at break point. If he takes the drug, then he will contribute to a situation in which this particular infectious disease may no longer be controlled. This will not affect other humans — as he is the last man — but it may affect animals. Would he be wrong to take the antimicrobials? In a strict Jainian perspective, he cannot take the antimicrobials because it would be against his spiritual inclinations – it kills living things (the agents of his disease). In addition, if he ingests the antimicrobials he will add to the environment a number of *supra-natural* life forms which will threaten the effectiveness of the cure (and increase the number of drug resistant micro-organisms that will threaten other life-forms). Therefore, S holding to his spirituality, cannot ingest the therapy and thus he must die from the infectious disease.

However, as an *enlightened* Jain, S can as well consider the bounds of his spirituality. If he is spiritually bound not to harm living things then, he ponders, might he be bound to help them? If this is the case, then he must weigh two conflicting obligations of his spirituality within a larger worldview. He knows that if he takes the antimicrobials he will be killing life forms. And he knows that antimicrobials, if taken correctly, will naturally produce some *supra-natural* organisms. Because he is informed, he considers that a major contributing factor in global drug resistance was their imprudent use. Since he is the last man, and an environmentally responsible person, he knows no humans remain who will misuse antimicrobials and that he will not misuse them.

He also reasons that although he is the last man thus bereft of human companionship, he can find some solace in nature and her entities. He is bound to weigh the consequences of his actions within the framework of this information. This information may raise questions that shift, conflict, and enhance his current worldview. We could broadly say that his obligation not to harm could be construed as non-maleficence which conflicts with the obligation to help, or beneficence. In the end, our enlightened S decides that the obligation of non-maleficence is outweighed by beneficence. So he takes the drugs, and assumes his responsibilities within the context of his enlightened worldview.

Unenlightened S's felt preference was based on a single premise, the tenet of Jainism that obligates its followers not to harm other living entities. If narrowly perceived, the injunction not to harm would necessitate that S die of his infectious disease. On the other hand, our *enlightened* Jain deliberated and found within his belief system questions he had not previously considered. Thus he was able to mediate his initial response, a felt preference (albeit for him a disastrous one) by an informed, enlightened and deliberated considered preference. And this resulted in a totally different moral decision and outcome from the *unenlightened* Jain.

In these examples I have identified a weakness in Norton's enlightened anthropocentric approach to environmental ethics. Criticising our felt preferences in the context of an already informed worldview may lead to a difficult balancing of principles that we are obliged to pull out from that very enlightened worldview (which is not to say that an unenlightened view is devoid of this problem either). Moreover, in both illustrations, the ability to internalise, judge and weigh such principles remains highly contingent upon personal mental abilities and creativity. In addition, considered preferences may prove to be difficult to attain when faced with a world of dissimilar socio-economic and political circumstances.

Most of all, in a human context the problem of global drug resistance largely concerns health and disease. When the problem is one of human illness combined with the potential promise of antimicrobials to facilitate wellness, it will be difficult for the most persuasive environmentalist to deter an individual from any course of action that will impede her desire for relief. This includes whether the decisions are rational, irrational, unenlightened or enlightened, or if the therapy is appropriate, sub-optimal or unnecessarily demanded. In other words, the *fact of illness*, real or even perceived, presents a challenge to his enlightened anthropocentric approach conceived as it is as 'a framework for satisfying obligations to nature that goes beyond concern for satisfying human preferences' (*ibid*: 185).

Importantly though, he emphasises that attainment of an environmentally concerned worldview is an *ideal* towards which we should strive. Holding to this ideal — for to do nothing is hardly an option — where then should our moral responsibility lie? In the context of humans and antimicrobial therapy, we could say that the felt value of antimicrobial therapy is a human concern. This is validated in Norton's thesis. This is because he retains primacy of value in human loci – value is maintained in human life and consciousness. As we are able within his construction to justify rules of behaviour in this regard, then it is acceptable to use antimicrobial therapy. However, since these felt preferences are often irrational, then the future well-being of human life may be threatened.

Likewise our felt preferences may be rational (we may take antimicrobials wisely); however, we often take them unaware of their larger implications (*supra-natura*l microorganisms, effects on the environment and so forth). But in both instances, we must adjudicate felt preferences with enlightened ones informed by a rational worldview. Narrowly confined, this would include the human-centred merits of proper consumption of antimicrobials, in other words prudential use. In a broader sense, as an ideal, we should strive to expand our human concerns encompassing 'the ideals, values and principles that constitute a rational worldview regarding the human species' relationship to nature' (*ibid*: 190).

The necessary obligation of containing drug resistance does not imply that we should not take antimicrobials when they are necessary. It simply infers that we have a moral responsibility to use antimicrobials prudentially. This is because if we do not, our ability to treat successfully infectious diseases is in peril. Bryan Norton's environmental ethic maintains our value primacy yet it affords us a way in which we may critically consider how our actions may affect other living entities and the environment. Moreover, because we see that drug resistance also threatens our environment, the same obligation of prudential use equally applies. This is a strength in Norton's approach to environmental ethics.

Towards Achieving an Environmental Conscience

The Porto Alegre Declaration on University, Ethics and the Environment (1995: 221) includes these words:

The 21st century university ought both to bridge and to blend the sciences and humanities into an integrated whole. To speak effectively on environmental issues, the university should abandon the dogma that science deals with a domain of objective facts and the humanities with a domain of subjective values. Scientific inquiry is directed by our values and the revelations of science often inform, expand and transform our values in unexpected ways.

To meet such challenges, 'we have,' says Lutzenberger (1996: 43), 'to start an ethical revolution'. To achieve this, he claims, 'philosophers and theologians must learn a great deal about science and technology'. Likewise, we might add, scientists must learn a great deal about philosophy and theology. The point is that we are faced with a magnitude of environmental problems. The resolve to address them need not fall within a single discipline. Rather it should be multidisciplinary, yet voiced as a single consensus of like-minded environmentally sensitive individuals.

How do we accomplish expanding worldviews that now virtually consist in viewing nature as a commodity and where our wants and desires take precedence over rationally enlightened choices? First, it seems that there are problems in educational systems in this regard. Like Ehrenfeld, Rolston (1996: 163) accuses higher education of 'producing the knowledge to degradate the planet'. He (*ibid*: 189) further argues that 'a university education that is not environmental education is no education at all'. Moreover, he asserts:

Colleges and universities are supposed to defend the arts and the sciences, the wisdom of the human genius ... The knowledge accumulated in the universities ... is of great genius. Yet it has destabilised human life on our home planet ... But if this explosion of knowledge and its resulting *empowerment has introduced a planet in crisis, perhaps the genius of the university is not what we thought* (*ibid*: 135-136).

The least one could say is that Rolston makes a most damning condemnation of traditional academic education, which, in his view, promotes unethical rather than ethical behaviour. But how do we begin to address the challenge? Madsen (1996: 72) has a pragmatic approach. In his opinion, ideally, education should follow three steps. It should:

1. Promote environmental awareness;
2. Understand information concerning environmental problems; and
3. Commit to work towards protecting and restoring the environment.

His is not a lone voice. David Orr, author of a 1990 book titled *Ecological Literacy: Education and the Transition to a Postmodern World*, argues 'our modern age may soon end due to ecological collapse' and furthermore that 'in order to survive, humanity must go beyond the attitudes, values, and practices of the present age and develop an integrated scientific, ethical, aesthetic, and religious worldview'. (We can see a similar position *viz.*, informing our commonly held worldviews in Norton's enlightened anthropocentric approach.) In the context of education, the question Orr grapples with is: 'what is important to know and why?' According to Orr (*ibid*: 66), there is a general failure among educators to address this fundamental question. To remedy this, he contends that all education should aim at achieving sustainable development and environmental protection.

Let us add to this Norton's approach to environmental ethics in that it requires that people have the means by which to enlighten their felt preferences. Education is the key. Ideally, education should be followed or accompanied by personal and collective action and activism for the purpose of heightening the awareness of environmental problems in order to influence the political will to elicit positive change. As emphasised by Griffin (1996: ix-xiii), we need more constructive or revisionary thought to transcend, emancipate, and create a synthesis of modern and premodern truths and values. This implies we must expand our concerns beyond the strong anthropocentric axiology. Following Norton (1995: 191), this would be framed as an enlightened rational worldview of ideals in which felt preferences may be criticised and the values and principles regarding the human species' relationship to nature are established.

In the context of drug resistance, this points to a vital need in addressing the problem. This is because only scant information is available concerning it in the public domain. Information and education are crucial tools to achieve the environmental conscience needed to curb the problem of increasing drug resistance and its effects on the health of the biotic community. This may lead us to revisit Malone (1993: 14) who challenges conventional education that excludes environmental concerns in asking three fundamental questions: What kind of a world do we have? What kind of a world should we want? What must we do to get it?

I have shown the kind of a world we have, one facing the prospect of increasing disease and concurrently a challenged ability to treat them effectively because of mounting drug resistance.

What kind of world should we want? In the context of drug resistance, it is safe to say that we should want to live in a world that moves towards understanding that emerging infectious diseases and drug resistance are natural responses to a world out of balance, an unbalanced world of human making. In a world we want, we should realise that for every application of technology, there will be unknown consequences, be wary and work to inform any such *quick-fix* reliance. In a world we want, our human population in numbers and materialistically driven life-styles should be moderated. In the world we want, the environment would not be commodified but protected and respected. In the world we want, while never stopping to try to attain human rights, we should recognise that they are mainly aspirational, fragile and often unattainable. In the world we want, we should recognise that there is power behind the construction and mediasation of ideologies, be cautious and resist them.

To answer Malone's final question 'What must we do to get it?' requires a change in thinking about ourselves, a looking beyond ourselves to the biotic community as a whole, a reallocation and clarification of our felt preferences enlightened by a more environmentally sensitive worldview. To inform felt preferences requires a great amount of time and political will. Time, when faced with the numbers and potential of microorganisms, drug resistant or potentially so, is a grave problem. Political will is another impediment. Faced with a world of so many diverse competing claims for prioritisation of interests, it is difficult to see how the problem of drug resistance can be placed anywhere near the top, particularly in developing countries. Yet to do nothing is hardly a moral option.

In the context of expanding worldviews to consider the environment, I now turn to placing drug resistance in the perspective of biomedical ethics. I will argue that the traditional parameters of biomedical ethics are insufficient to accommodate environmental concerns. Moreover, I will show that it is possible in Norton's enlightened anthropocentric position to criticise and inform but need not necessarily rupture traditional approaches to teaching biomedical ethics.

Enlightening Traditional Biomedical Ethics Education

Barry Hoffmaster (1984: 2-3) argues that biomedical ethics should be re-conceived in a social context. He claims that moral justification in traditional biomedical ethics inherently includes the assumption 'that real life moral problems come sorted and labelled and ready for the manipulation of rules, principles or theories'. He (*ibid*) argues that in such assumptions, biomedical ethics,

> ... *disregards the extent to which moral concepts and norms derive their meaning and their force from the social and cultural surroundings in which they are embedded; neglects the ways in which moral problems are generated and framed by the practices, structures, and institutions within which they arise and ignores the means by which social and cultural ideologies, and the power relationships they entrench, can both perpetuate moral inertia and effect moral change.*

I will not address his particular concerns as I only wish to identify that there is movement within biomedical ethics to move beyond an individual-to-individual approach. I wish to extend interactions beyond the social to an environmental context. I will use a case study as an example to demonstrate this.

Dr. Jones, a general medical practitioner, has just prescribed an antimicrobial for his patient Mrs. Gama whom he diagnosed as having a chronic urinary tract infection. Were his actions morally justifiable?

An initial response to this case study might well be: what is the moral problem? A doctor examined a patient, reached a diagnosis and prescribed a drug therapy.

It would be difficult to reach a contrary position based on the information provided. But what if the example gave us additional details?

Dr. Jones, a general medical practitioner, has just prescribed an antimicrobial, amoxicillin, for his patient Mrs. Gama whom he diagnosed as having a chronic urinary tract infection.

Before we could make a moral assessment of this problem, we would have to know if Dr. Jones knew about the problem of global drug resistance. And this points to the first concern. As prudential measures in attempts to reduce the numbers of drug resistant microorganisms in the environment, medical schools should provide continuing education on current antimicrobial uses to prescribers. This could be accomplished as part of the requirements of continuing medical education as well as in lectures presented to medical, dental, veterinary and agricultural students. But to lecture on the relative problems separately, (*e.g.* because of a wide spectrum of resistance to amoxicillin, it is no longer an effective drug therapy in urinary tract infections; the Bonk-tick has now invaded South Africa; or because of widespread resistance, tetracycline is no longer recommended for use in the control of *Salmonella* in chickens) excludes considerations of the whole problem: an understanding of morality as a lived human-nature experience.

This would suggest then the acceptance of the primacy of the whole. In this conception, there is the idea that relationships are at least generally more fundamental than things; that wholes are primordial to their parts. Interrelatedness, interconnectedness exists; the world is interconnected. But this is an thought that is not promulgated in our current Western-type of orientation. We have a tendency to consider that parts in some ways exist independently and their interpretation is highly subjective. For example, dividing an individual in half does not make two individuals. An individual may be said to be composed of a head, or limbs; or of bones and muscles; or of hearts and lungs; or of nervous systems; or of cells; or of genes. What an individual is cannot be comprehended by simply looking at her parts.

Generally, we are accustomed to see parts as disconnected from the whole. In addition, in this context we look for solutions that will *fix problems*. We tend to consider problems as if they are external and can be resolved without *fixing* that which is within us which lead to their creation. Consequently, we are drawn into a spiral of superficial 'quick fixes' that in the long run can result in a worsening situation. For example, if we hold that the answer to the problem of drug resistance lies in the development of new genetic technologies (*e.g.* modifying the genes of pathogenic microorganisms) then its usage will most likely remain as it often is: reckless, indiscriminate, multi-purpose. However, through enlightening individuals concerning the primacy of the whole, such conceptions may serve to assist individuals to break the vicious cycle. As such, the ways in which drug resistance affects (and effects) the biotic community as a whole should be considered. Examples include the intersections of population pressures, consumerist practices, under-over consumption, global warming, environmental routes of transmission and so forth. So, to simply say, 'Beware, the Bonk-tick has invaded South Africa', 'Don't use amoxicillin for urinary tract infections' or 'Tetracycline is no longer the treatment of choice for chickens with *Salmonella*', does provide some necessary information, but does not enlighten the necessity of considering the whole.

Now let us say that Dr. Jones has been informed superficially about the problem of drug resistance. He knows that urinary tract infections are no longer responding to amoxicillin. Is he morally right in prescribing it?

The answer would be 'no'. This could be justified from many perspectives in biomedical ethics from moral theories, to principles, to virtues and values. Yet if he were enlightened beyond knowing the proscription against amoxicillin use in urinary tract infections as based on its drug resistance, he would ideally realise that drug resistance is an interconnected human-nature problem. Thus, he would see that each time he prescribes an antimicrobial he contributes to events that affect both humans and nature. So his concerns, while retaining proximate value (the doctor-patient relationship), gain depth and direction in consideration of the whole. This is consistent with Norton's approach to an enlightened anthropocentric ethic.

I have often pointed to the interconnectedness of systems in the problem of global drug resistance. In this conception, we are challenged to see the interrelatedness that exists with us and that surrounds us. Similar to the conception of parts as primordial wholes, we tend to see individuals as primordial to both the community and the environment in which she is embedded.

It is generally agreed that when we are asked to talk about ourselves, we relate stories, about our families, where we work and so forth. But where in this talk is our 'self'? The 'self' is not a thing. Kofman (2001: 26) says, 'a self is a point of view that unifies the flow of experience into a coherent narrative — a narrative striving to connect with other narratives'. Such narratives are of course, influenced by culture and culture is influenced or informed by our environment. Rolston (1988: 203) points to this writing

> ... *I find myself placed in a concentric field for valuing. The whole possibility is among natural events, including the openness of my apprising. John Dewey remarked that "experience is* ***of*** *as well as* ***in*** *nature".*[82] *We say that valuing is* ***in*** *as well as* ***of*** *nature.*

So conceived, we learn to construct stories based on and of culture and nature. And the stories we relate try to make sense of our experience, to give meaning to our thoughts and actions. When we fail to consider that the self has an inherent 'community character', then we tend to place a primordial value on the egoistic part of the self and see the community part (culture and nature) as secondary. We consider the secondary as nothing but a network supplementing our needs and desires (*e.g.* in materialistic-based preferences, economic exchanges). In this way, encounters with other individuals become transactions that can add to or subtract from the 'possessions' of the ego. Such a constricted understanding can result in incalculable loss – we are lost without a sense of place. In an enlightened perspective, seeing self as a community opens the door for powerful and beneficial changes in our underlying values. This applies to a broader perception of and value placed in communities framed in the larger circle of the environment.

I teach biomedical ethics, and I try to teach it in the larger context of the environment. I use global drug resistance as an example of the link between human health and that of the biotic community. Using Norton's (1995: 182) emphasis on interests as opposed to the ascription of intrinsic value to the biotic community, some theoretical impasses may be overcome.[83] Likewise, in maintaining value in human loci, (*ibid*: 187), the obligation not to harm other humans unjustifiably (*ibid*: 188), and in the value of ongoing human consciousness (*ibid*: 190) space is made available for the inclusion of human concerns within a larger and enlightened context.

Norton's rules of distributive fairness and allocation (*ibid*) can also be included as a perspective within the traditional biomedical teaching of distributive justice. Since the intrinsic value of human life is not questioned, it can critique human practices that are exploitative of nature. For example, it can ask if the human cost of acquiring a new infectious disease is worth the cost of clearing pristine land. His point is that the search to define ideals, values and principles that bind us to nature may be equated to the same search to find those which bind us to other humans (*ibid*). In this regard, most of our traditional appeals likewise serve as a means by which we evaluate right and wrong human actions.

Yet practical difficulties remain. The attainment of the ideal of an affinity with nature is necessary to enlighten worldviews. But the 'enigma of health' — if including the quest for wellness when one is ill – I suggest represents such a primary human need or desire that when faced with illness, all the rationally accepted ideals of an enlightened worldview fall apart.

Concluding Remarks

The weakness of strong anthropocentrism is in its inability to accommodate ethical concerns about the environment. Moreover, the weakness of strong non-anthropocentrism is that so far, efforts to allocate intrinsic value to non-human entities have failed to gain acceptance in traditional philosophical circles. The appeal of enlightened anthropocentrism resides in its ability to accommodate both human and environmental concerns while preserving human primacy. In the context of the subject matter of this dissertation, Norton's enlightened anthropocentrism provides us with a way to incorporate drug resistance into the scope of environmental ethics. Because drug resistance has two facets – it affects both human health and environmental health — I suggest a further expansion in terms of an awareness currently not within the scope of biomedical ethics. The gap should be bridged by widening the perspectives of the teaching of bioethics to encompass both human and environmental concerns.

The question then is: does Norton's enlightened anthropocentrism meet all the expectations and requirements to bridge the gap? Like all ethical theories, Norton's theory has strengths and weaknesses. He grounds his theory metaphysically on the distinction between felt and considered preferences. The former are typically anthropocentric. The latter widen our views on the moral consequences of our actions on other humans and on non-human entities. Considered preferences then are a tool to criticise and to redirect our felt preferences. This is a strength. However, when put to the test, weaknesses are discovered. The first *Gedankenexperiment* shows that the same worldview that guides moral decisions may lead to two diametrically opposed conclusions because of the differences in the capability to use information (enlightenment). The second *Gedankenexperiment* shows again that the implementation of Norton's concepts of felt and considered preferences may lead to diametrically opposed conclusions because of the lack of a weighting tool and because of the role played by life's contingencies.

Norton's theory retains the primacy of value in human life. In practice, however, felt preferences are often given primacy because our choices are irrational and selfish. We tend to disregard the consequences of our felt preferences for fellow human beings and on nature. We should introspect and involve enlightened considered preferences in our moral decisions. In this way, we will achieve a more encompassing realisation of our place in the biotic community. But this, says Norton, is an 'ideal' for which to strive. An ideal is something we only can strive for; it is never reached. On the other hand, if a goal is not set we do not know for what to strive. This should be the role of a more encompassing, an 'enlightened', if you will, teaching of ethics.

I began this work with a quote from Charles Babbage writing during the 19th century, which, I believe, is also a fitting ending:

... earth, air and ocean, are the eternal witnesses of the acts we have done ...
No motion impressed by natural causes, or by human agency, is ever obliterated.

Charles Babbage 1838.

Notes

Notes to Introduction to Part 1: Earth, Air, Fire and Water

[1] In his work *The Metaphysical Principles of Virtue* (1983), Immanuel Kant

argues that a 'good will' is one that acts for and from the sake of *duty*. This is because a morally good intention is a vital constituent of a morally good act. Morally good acts are not determined simply by or because of what one intends. In other words, it cannot be relativised to one's perceptions of their intentions of what is good or morally sincere. He (*ibid*: 51) writes:

It is not possible for a man to look so far into the depths of his own heart as ever to be entirely certain, even in one single action, of the purity of his moral purpose and the sincerity of his mental disposition, although he has no doubt at all about its legality.

[2] As I see it, the overall aim is to shift current worldviews concerning our environment. This then would involves many facets of human activity such as education, social changes, public policy shifts, and even global appeals however construed as moral duties such as obligations to future generations and to our fellow creatures with whom we share the planet.

[3] *See* American Society of Microbiology (ASM) 1999, 2001; World Resource Institute 1996-1997; Centers for Communicable Disease (CDC) 2000.

[4] *See* the World Health Organisation (WHO) 1992; 2000 b.

[5] The term 'emerging infectious diseases' generally is used to classify a variety of diseases(CDC 2000):
(1) a completely new disease or an old disease occurring in new places and new people;
(2) an old disease with new presentations;
(3) a disease newly resistant to available drug therapies.

The following are only examples of some infectious diseases classified under each heading (CDC 2001; CDC 2000 b): *New Diseases* include: HIV, Ebola, legionnaires, Nipah virus infection, and hantavirus. *Old Diseases in New Places* include hepatitis E in Haiti and West Nile Virus in North America. *Re-introduced Infectious Diseases* include: dengue fever in Texas, malaria in Korea, and plague in India. Malaria in USA Soldiers in Somalia, Marburg virus in lab workers in Germany, and cholera in Peruvian mountain villagers include infectious diseases classified under the heading of *New Populations*. Under *Increased Virulence* are found such infectious diseases as H5 N1 Influenza in Hong Kong, and *E. coli* 0157: H7 in the USA. Those *Drug-resistant* include *Campylobacter,* malaria and *Streptococcus aureus.*

[6] I will use the term animal(s) in its broadest sense to refer to nonhuman animals *e.g.* cats, dogs, pigs, antelopes, marine life, reptiles, amphibians, birds, etc.

[7] Prions are communicable proteins that do not meet the conventional definitions of microorganisms but are nonetheless infectious via human to human or nonhuman animal to person contact.

Notes to Chapter 1

[8] A biotic community as defined by Callicot (1989: 72 is:
'(biotic community) ... in its most expansive sense includes all living things ("plants and animals") and even non-living things ("soils and waters")'.

[9] Human technology and human actions contribute directly to the introduction and spread of many infectious diseases, and animals often serve as reservoirs for pathogens that cause human disease in cross-species transfer. While less researched, it should be mentioned that human pathogens can be transmitted to animals as well (Alexander 1999). Infectious diseases exist cross-species, interspecies and intraspecies.

[10] Palaeopathology is the study of ancient diseases. This perspective offers information concerning human evolution, environment and the relationships between human biology and culture and disease. An example of palaeopathology is the documentation of human disease and health patterns that changed during the transition from hunting and gathering to agriculture. Practically, palaeopathologists use a variety of technical tools such as bone analysis, DNA-RNA testing, imaging such as CAT scans, and re-saturation of dried human waste in their investigations. Paleoanthropology, on the other hand, focuses attention on the study of human origins, the study of fossils and the cultural remains of our extinct human ancestors.

[11] For example, the Black Death is attributed to *Yersinia pestis*, but it is unknown whether that particular *Y. pestis* is the same one that exists today, or has undergone mutation over the years (Lederberg 1997: 419).

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[12] Fire was controlled and used for cooking, warmth and protection against other predatory animals. Musical instruments, flaked stone tools, portable art and artefacts, implements and equipment were part of early hunter-gatherer societies.

[13] Altruism, it has been argued, is incompatible with evolution. Through analogous behaviour studied between various animal species it was discovered that altruism must be distinguished based on different kinds as well as different classes of recipients. The traditional definition is an act beneficial to a recipient but performed at cost to the altruist. According to Mayr (2001: 257-260) there are three different kinds of altruism: (1) Altruism for the benefit of an individual's own offspring, (2) Favoured treatment of close relatives or 'Kin Selection,' and (3) Altruism among members of the same social group.

[14] Polgar (1964) suggests that there are two lines of disease transmission, the first from microbes present in our pre-human ancestors that accompanied the evolutionary process to humans (such as head and body lice and pinworms) plus microbes similar to the type of protozoa found in the human body today (for example salmonella, typhi and staphylococci). Cockburn (1967) adds to the list, asserting that viral diseases, carried in nonhuman primates, had the potential by proximity, to also be transmitted to early humans. The second line of infectious disease transmission suggested by Polgar (1964) is classified as zoonotic. These microbes have animals as their primary hosts and only infect humans incidentally. Their mode of transmission is, for example, through insect bites, in preparation and consumption of contaminated flesh, and from wounds inflicted by animals. It is possible that diseases such as sleeping sickness (transmitted by the tsetse fly), tetanus, avian tuberculosis and other zoonotic diseases might have affected early hunters and gatherers, as there is evidence of their concurrent existence, and thus would be present in early agrarian societies as well. The problem now is that many of these new diseases are resistant to common antimicrobials.

[15] In his essay on *Air, Water and Places*, Hippocrates makes the claim that goats are not used in Greece and cites the problems of diseases in Asian countries using goats. Yet goats appear in Greek mythology.

[16] Archaeological evidence of domestic cattle, sheep and pig bones dates from before 8000 BCE in Greece and Southern Europe according the anthropological evidence. *See* Nielsen's report (Cited May 2001). Available at http: //fhss.byu.edu/anthro/faculty/Nielsen/110 Chap 10.htm.

[17] 'Medicalisation' refers to a shift in considering medical doctors as the first line of defence against disease as opposed to family or particular community members.

[18] Disease as perceived by many of my biomedical ethics students at the Medical University of Southern Africa suggests that the idea of disease as a 'sin' on the part of the patient, as well as retribution, divine or otherwise, is very common at least in local culture.

[19] Nearly all emergent disease incidences of the past 10 years have involved zoonotic agents (Murphy 2000: 2). Many different determinants contribute to the emergence of zoonotic disease agents, and they rarely act alone.

[20] The American Society of Microbiology (2001) provides examples of emerging infectious diseases in animals and plants: BCE (bovine spongiform encephalitis), considered as the agent for variant form of Creutzfeldt-Jacob disease in humans (*ibid* 6, 7); TB in cattle showing drug-resistant strains (*ibid*: 7); *Mycoplasma galliseptum* in captive and wild finches (ibid: 10); microsporidosis, an ancient animal parasite, now emerging in humans, particularly in immunocompromised (*ibid*: 8); an *M. galliseptum* species member infecting commercial poultry, different host-adaptive rabies viruses (*ibid*: 12); and 'whirling disease' in certain trout species (*ibid*: 14-16). The Geminivirus-whitefly complex (whiteflies transmit the Geminivirus) cause serious damage to cassava, tomatoes, mungbeans, peppers and cotton. Through clonal migrations, *Phytophthora infestans*, the cause of the 'Irish potato famine,' believed to have been eradicated over 150 years ago is, now becoming a scourge worldwide (*ibid*: 19).

[21] The UK House of Lords Science and Technology Report (1997-1998: 3) defines the term 'antibiotic': 'Antibiotic' is the term originally applied to a naturally occurring compound such as penicillin that attacked infecting bacteria without harming its host. 'Antibiotic' in current usage broadly refers to synthetic compounds as well as natural compounds and refers to antiviral as well as antibacterial drugs. 'Antimicrobial' is a generic term used to cover a broader field of microorganisms such as the bacteria, fungi, viruses, and other protozoa.

[22] Efavirenz is one of the newer drugs developed for HIV treatment. It is currently one of the drugs being used in combination with regimens of ddl/3TC (another newer drug) and standard TB DOTS (Directly Observed Tuberculosis Therapy) treatments in a Durban-based South African HIV study (Martins 2003: 16).

[23] The term 'nosocomial' refers to a hospital-acquired infection; not the patient's original condition.

[24] Hensley (2001) reports in the *Wall Street Journal* (October 9: A1) that purchases of antibiotics 'jump on fears of bioterrorism, although doctors see risks'.

[25] Eukaryotes include plants, fungi, and animals as well as single-cell protozoa such as amoebae and parasites (*e.g. Giardia*) that cause diseases such as malaria and dysentery. Humans are Eukaryotes. Eukaryotes originated about 2 700 million years ago and diversified so readily that scientists such as Margulis and Schwartz (1998) recognise no less than 36 phyla of these protists. They differ from prokaryotes by the possession of a nucleus surrounded by a membrane and contain individual chromosomes.

Notes to Chapter 2

[26] For example, we inhabit every continent (over-populate the earth), roam the seas (kill the oceans), create splendid cities (generate decaying slums), tame rivers (pollute water sources), bring water to the deserts (create new deserts), harness the atom (build weapons of planetary destruction), and tinker with genes (move non-consequentially in unknown parameters.

[27] Now a part of common speech when referring to antimicrobial drug therapy, the term 'magic bullets' gained popularity in 1940 following the release of the film, *Dr. Ehrlich's Magic Bullet*.

[28] The idea of 'natural tendency' was promoted in pre-Darwinian scientific and philosophical contemplation. Aristotle initiated the concept of 'natural tendency' in his philosophy believing that even the most trivial creature in nature was of interest if one only examined it honestly (Clark 1997: 31). Specifically, Aristotle's works, including his physics and biology, particularised the natural inclinations of kinds of objects, things, and the forces that impede that object reaching its intended state. All organisms are conceptualised in terms of a natural state model. Aristotle (1970: 415 a: 26) states: '(for) any living thing that has reached its normal development and which is un-mutilated, and whose mode of generation is not spontaneous, the most natural act is the production of another one like itself, an animal producing an animal, a plant a plant ...'.

[29] For an in-depth analysis of Darwin and Willem Paley (perhaps one of the most famous 'Evolutionist-Creationist') debates, see Gould 2002: 115-121; Lewin 1997: 33 also presents an account.

[30] Mayr (2001: 278) puts it this way: 'Evolution appears highly progressive when the lineage leading from microorganisms such as bacteria to cellular protists, higher plants and animals, primates and humans are considered. However, the earliest of these organisms, the bacteria, are just about the most successful of all organisms with a total biomass that may well exceed all other living organisms combined.' Progression works in this manner: The first protists incorporated symbiotic prokaryotes and this resulted in an immensely successful territory for the eukaryotes. Other progressive steps have also been cited: multicellularity, the development of highly specialised structures and organs, highly developed parental care, etc. The 'inventors' of each new progressive step were also highly successful. This contributed to their ecological dominance. The gist of every selective event is to favour individuals that have succeeded in finding a progressive answer to current problems. All these steps equal evolutionary progress. Primitive prokaryotes still survive more than 3 billion years after their first appearance on earth. Fish still dominate the oceans and except for humans, rodents are more successful in most environments than are primates. On the other hand, as shown by cave inhabitants and by parasites, evolution can also be retrogressive. But it is legitimate to refer to the series of steps from the prokaryotes to eukaryotes, vertebrates, mammals, primates, and humans as 'progressive'. This is because each step was the result of successful natural selection. The survivors of the selection process have been proven superior to those that were eliminated.

[31] Plasmids are genetic structures in cells that can reproduce independently of a cell's chromosomes. For example, circular DNA strands in a bacterium or protozoan.

[32] Coetzer (2002: 3) defines 'biosphere' as 'all plant and animal life on the earth'.

[33] See for example World Health Organization reports 1992, 1998, 2000 a, 2000 b, 2000 c; Centers for Disease Control reports 1997, 2000, 2001; United Kingdom Science and Technology Report 1997-1998.

[34] The history of antibiotic therapy dates back to the discovery of sulphamides. First identified by Ehrlich, then developed further by Domagk, the important observation that substances which stained certain bacteria under the microscope

must have chemical affinity to their membranes, was identified as a 'principle of affinity,' an affinity that could be utilised in medicine (Sournia 1992: 449).

[35] All macroevolutionary processes take place in populations and in the genotypes of individuals and are thus macroevolutionary processes.

[36] There are antibiotics present in the natural environment, such as pseudomonas and some species of enterococci.

[37] Mitochondria are microscopic organelles found in the cytoplasm of almost all eukaryotes. In humans, mitochondria supply almost all of the energy to the cell (90%). Without mitochondria, there is no life, because life requires energy. Cells with a high metabolic rate, *e.g.* heart muscle cells, may contain many thousands of mitochondria while some cells may contain only dozens.

[38] For example, a nautical group of bacteria known as *Prochlorococcus* removes carbon dioxide from air and fixes it into the carbon content of their own miniscule bodies. A quart of ocean water often contains 100 million *Prochlorococcus* cells. Its population in the global ocean could be as high as 10 trillion trillion. It is speculated that 'it may well be the most abundant organism on this planet' (Chisholm *et al.* 2000: 1).

Notes to Chapter 3

[39] In sorting out the complexities of the links between human overpopulation and the current health situation, a useful beginning is provided by Abdel Omran, an epidemiologist who first coined the term 'the epidemiological transition' (1971: 536). Omran contrasted the old-style 'Age of Pestilence and Famine' with the 'Age of Degenerative and Man-Made Disease' in which the annual mortality was well below 20 per 1 000 (*ibid*: 305-316). Following from this, between 1960 and 1994 in the world's economically advanced 'North,' life expectancy rose from the mid- to late 70s. Elsewhere, in all the world's poorest countries (mostly in sub-Saharan Africa), average life expectancy rose from around 46 to around 63 (SAIRR). This is before the HIV/AIDS pandemic, which has reduced the rates significantly – for example in South Africa, average life expectancy as of 2002 has been reduced by around 7 years to an estimated 37 years (BBC 2002). The pandemic is likely to affect life expectancy in other sub-Saharan countries to an even greater extent: 'Botswana, Malawi, Namibia, Swaziland, and Zambia could have expected life expectancies of between 56.8 years (Malawi) and 70.1 years

(Namibia) by 2010. With AIDS, not one will have a life expectancy of 40 years' (SAIRR 2001).

[40] It is not indicated if these figures are adjusted in light of the HIV/AIDS epidemic.

[41] Coetzer (2002: 9-10) lists some potential problems: 'Political violence, looting, pillaging, arson and bombings; water and sewerage reticulation unable to develop apace with population growth; slum formation with no city centres; frequent power failures; emergency services unable to cope with fires, accidents, and natural disasters; increasing incidence of crime; breakdown of nuclear family, increased suicide, divorce, spouse or child battering; alcoholism, drug abuse and prostitution; unemployment and poverty; street children and vagrancy; squatter camps and people living on the streets; malnutrition and famine; severe fuel burning appliances, industries and motor vehicles; noise pollution; labour unrest, strikes and demonstrations; corruption of armed forces, police brutality, extortion and graft; progressive household shortages; inadequate schools, hospitals, law courts, prisons, police and fire-fighters; surrounding deforestation, resulting in floods and soil erosion, lack of refuse disposal, and exposure to lead and other fuels from petrol combustion'.

[42] A Density Dependent Factor (DDF) is one where the effect of the factor on the size of the population depends upon the original density or size of the population. For example, if a population is dense and individuals live close together, each individual will have a higher probability of catching the disease than if the individuals had been living farther apart. Generally, density-dependent factors are biological factors, such as diseases, parasites, competition, and predation. Furthermore, in populations being controlled by density-dependent factors, growth rates are usually inversely proportional to population density. For example, if the population density is high, the growth rate is low. Conversely, if the density is low, the growth rate is high. Density Independent Control (DIC) refers to instances where the effect of the factor on the size of the population is independent of and does not depend upon the original density or size of the population. The effect of weather is an example of a density-independent factor. A severe storm and flood coming through an area can just as easily wipe out a large population as a small one. Another example would be a harmful pollutant put into the environment, *i.e.* in rivers and streams. The probability of that harmful substance at some concentration killing an individual would not change depending on the size of the population. In general, density-independent factors

are physical factors, such as weather factors, *i.e.* severe winters, or the presence of harmful chemicals (from Nadakavukaren 2000: 26-27; 62-63).

[43] An example from Harvard's Center for Health and the Environment (2002) serves as an example: Fluoroquinolones (FQs) are used in poultry production. This group includes ciprofloxacin, popularly known as 'Cipro'. In 1995, over the objections of the CDC, the FDA approved FQs to treat sick chickens and turkeys. The drug was administered flock-wide or to 30 000 chickens per barn via water. It was administered flock-wide as to treat a single fowl was not considered worth while. In humans FQs are key drugs in the treatment of food poisoning as well as a host of other illnesses, not least of which is anthrax. Since 1995, after its use in poultry was started, FQ resistance in *Campylobacter,* a germ found in and around poultry which can make humans extremely ill, rose dramatically. In October 2000, the FDA proposed to ban the use of FQs in poultry. The Bayer Corporation, which makes a variant of Cipro for poultry, is contesting this. Nonetheless, as reported by Goldberg (2001: 5), 'several major poultry producers claim they are voluntarily reducing use of antimicrobials, though these claims are not being independently verified. Fast Food restaurants such as McDonald's, Wendy's, and others announced that they will not buy FQ-treated chickens.'

[44] Coetzer (2002: 34-35) lists the factors resulting in increased incidences of pesticide poisonings in the developing world as: 'deliberate exportation of chemicals banned or severely restricted in the countries of origin for the reasons of *i.e.* extreme mammalian toxicity, and accumulation in the environment or poor selectivity; labels that are misleading or incomplete or not written in the vernacular or not intelligible because of illiteracy, poor storage practices with leakage and cross-contamination of foodstuffs; lack of training and safe application practices; lack of protective clothing; lack of properly designed and specific equipment; use of pesticide containers for food and water storage or regulation; lack of legislation regarding surveillance; use of pesticides for purposes beyond their intention, *i.e.* spraying crops with chlorinated hydrocarbons and poisoning animals with parathion; spraying in adverse weather; and unreasonable consumer demands for the "perfect" fruit or vegetable resulting in repeated and unnecessary applications.'

[45] *See* Kroll-Smith, Brown and Gunter (2000: 12) who point our that this is often the case when 'powerful corporations and government organisations' place the well-being of the organisation against the well-being of employees and citizens.

[46] For example, the Pan-American Health Organisation (2002) identifies the complex relation between cross-species transfer, population density, climate change and poor socioeconomic conditions, in the reporting of the rodent-borne hantavirus. Benign parasites for over 30 million years, the hantavirus is carried by native rodents in the Americas. Human population density caused increasingly large umber of people to expand their habitats. Greater human population growth shifted the niches of rodents and this greater density, compounded by poor sanitation and living conditions, resulted in habitats conducive to greater rodent presence. In 1993, climate changes caused by El Niño led to increased rodent breeding and a consequent greater spillover into areas of human habitation. People became seriously ill. However, the link between the rodent parasite and its subsequent transmission into a malignant form in the human host was not initially realised. Its primary mode of transfer is respiratory through the inhalation of infectious aerosols from rodent excreta. In later research it was found that in the Americas, more than 20 different viruses were responsible for and could cause a 'new' clinical disease, the identification of which is determined by epidemiological patterns dependent on host-species interaction and identification. Each particular virus, interestingly, is carried by a different rodent species, from the Norway roof rat to the prairie dog. Hantavirus has now been identified worldwide. In this, there exists no dispute about the causative *agent*, but the conception of disease is extended to go beyond the agent to include social and environmental factors.

Notes to Chapter 4

[47] In 1271 France, doctors and pharmacists were reported to have feuded over dispensing drugs (Bomack 1972: 35). This discussion has continued to this day in many countries, developed as well as developing. In 1240, the German Emperor Frederick II initiated the separation of the occupations performed by doctors and pharmacists. Doctors became the diagnostic and prescribing experts and pharmacists experts in drug dispensing and drug management. This development was made possible by urbanisation (*ibid*: 45). Doctors could generate an income from diagnosing and prescribing. Likewise, pharmacists were not prescribing but could sustain a living through the sale and dispensing of drugs. The fact that doctors could generate an income from diagnosing and prescribing, and pharmacists from dispensing alone, was fundamental to the separation of the two practices. This practice was adopted in Japan, the UK, in most American states and in many European countries (*ibid:* 66).

[48] The theoretical reasoning for yearly flu vaccination is based upon any virus's ability for mutant and reassortant adaptation and change.

[49] Reused injection syringes, as well as contamination by human body fluids were implicated in increased disease transmission. Such factors as these influenced social practices such as burying the dead, travel, trade and traditions (such as the eating of nonhuman primates). In such situations, existing public health systems will be unable to manage outbreaks without increased personnel and special training. Incomplete knowledge concerning use of Western medical equipment is also implicated - disposable syringes that were simply reused without sterilisation because this was believed unnecessary. These are some factors identified as contributing agents in the spread of Ebola in the 1995 Congo outbreak (Khan *et al.* 1995: 79).

[50] An example of the latter is the resurgence of diphtheria and whooping cough, both vaccine-preventable diseases, reported in some states in the former Soviet Union.

[51] In 1999, Africa accounted for 81% of the global number of reported cases of cholera, and with climate changes, the transmission mode of the disease is modifying (WHO 2000 a).

[52] The decomposition of organic matter releases stored energy and produces carbon dioxide, which can then be reused by plants through photosynthesis. Decomposing organic material reduces to available forms of chemicals for plant growth. Some organisms in the soil cause or transmit diseases while others, such as some fungi control disease organisms. Organisms such as fungi and actinomycetes form associations with plant roots and aid the plant in exploring the soil environment for water and nutrients. Some organisms take nitrogen from the atmosphere and transform it into organic forms that are then utilised by other organisms in the ecosystem.

[53] It may be of interest that the hills of ancient Greece at the time of Aristotle, before the advent of goats, were also once covered with shrubs and grasses. (I read this some time ago but am unable to find the source).

[54] Neon, helium, krypton, xenon, hydrogen, methane and nitrous oxide are the trace elements.

[55] In addition to creating new conditions in which microorganisms can evolve and adapt as shown in the cholera example, rising sea levels brought on by global warming have the potential to threaten populations in many major cities, reduce fertile agricultural land, jeopardise the purity of fresh water supplies, and even compromise the physical existence of some nations. This is not to mention the resultant acidity of the air, formation of acid rain, unbalancing of ecosystems, deterioration of buildings, death of forests, reduction of crop yields, disappearance of wetlands and lakes, extinction of species, and so on.

[56] In addition, extreme heat will affect mortality and morbidity rates. Decrease in the protection from the ozone layer will result in an increase in environmental carcinogens, due to the ultraviolet component of sunlight. Air pollution is involved in such conditions as aspiration pneumonia, asthma, chronic airway resistance and alveolar wall thickening of the bronchi. Reports indicate that in the United States, for example, a link between high levels of particulate air pollution and death due to lung cancer, respiratory disease and heart ailments is present even after controlling for individual risk factors (Dockery: 1996). And we must keep in mind that the ability to successfully treat these and many other diseases is compromised because of global ADR.

[57] For example, humans in need of new habitats have invaded pristine areas. At the same time, certain species of rodents increase in density, as they do following major meteorological events such as those induced by El Niño. In the El Niño phenomenon, long periods of drought reduce predator densities, thus permitting a concomitant increase in the numbers of rodents. A combination of factors – the presence of humans (from their wastes to agricultural practices), lack of predators, subsequent rainfall increasing the availability of grain and other food sources (as well as increasing the already high rodent densities) – all contribute to the potential for infections to move from nonhuman hosts to humans (Epstein *et al.* 1994; Bremner 1994). Hantavirus in the USA, transmitted by a deermouse species (Bennett and Haart 1994: 71-73), Dengue fever in Brazil, and bubonic plague in Mozambique (Murphy 2000) are examples of this cross-species transfer.

Notes to Part 2: Moral Responsibility and the Problem of Global Drug Resistance

Notes to Chapter 5

[58] *See* Wilson, E. 1995, The Little Things that Run the World.

[59] *See* note 27, Chapter 2.

[60] This has relevance both now and for future discussions. This is because the genome project in the manufacture of genetically modified microorganisms will inevitably raise questions concerning their 'natural' or 'unnatural' status. Genomics has evolved (at least in the general public domain) rather inconspicuously and of such alternations as noted by Commoner (1992), we remain essentially ignorant.

[61] According to binomial nomenclature, organisms have two names - a genus name and a species epithet or species name. Both are either underlined or italicised and the genus is always capitalised and the species is in lower case *e.g.* *Escherichia coli.* Used in this particular type of classification system (there are many), one does not refer to only a species name, (*e.g. coli*) but uses both in classifying an organism.

[62] For example, we know that their presence is growing and our ability to treat successfully injuries and infectious diseases is increasingly impaired. We know that they do interact with other naturally occurring microorganisms but we do not know the exact parameters of this interaction. We do not know how they will behave under greater environmental stress.

Notes to Chapter 6

[63] Such 'steps' should better be seen as gradual transitions rather than paradigm shifts. All of them are expressions of a gradual transition from deep anthropocentrism to an expansion of moral standing and of intrinsic value beyond humans.

[64] Tom Regan, who sees moral wrongness rather in the use of animals as resources or commodities, does not share the argument from sentience, promoted by Peter Singer.

[65] Goodpaster (1978) contends that we should extend minimal moral considerability to all living entities based on their common goal-directedness. But goal-directedness as a description of 'life' raises some difficulties. This is because living entities exhibit an immense range of ways of exhibiting life dependent upon their level of consciousness. Anything living has in some regard a direction of growth, often described as wishes, urges, impulses, and so forth. For example, in a laboratory situation a particular bacterium may move toward the contents of a specific agar plate. This may be perceived as a 'desire' to reach a goal but it could equally be regarded as a simple biological affinity. The claim

of granting moral considerability based on goal-directedness appears then to be somewhat subjective. In addition, to claim that a life form as merely being alive or having a 'good-of-their-own' automatically conduces to moral considerability is another matter. This is because Goodpaster does not address *how much* moral considerability should be allotted. Even if we were to accept the principle of extending moral considerability, it would be reasonable to want a way in which we could differentiate between living entities

[66] A similar position is found in Schweitzer's (1987) *Reverence for Life*.

[67] One interpretation of environmental holism is that the biosphere as an interconnected whole has moral standing (Pierce and VanDeVeer 1995: 111). Another claim is that holism views the biosphere as an interconnected whole. As individuals, we are part of a larger system and we should value the system of which we are a part. However, this is not the case: 'We value the system from the viewpoint of individuals rather than claiming the value of the system as the *source of independent value'* (Norton cited in *ibid*).

[68] On the early earth, Woese *et al.* (1990: 4576-4579) claim, there was no genealogy. Life had not yet separated into distinct lineages and thus no single species lies at the base of the tree of life. Our common ancestor was every microorganism that lived on the early earth, a fluid matrix of genes that covered the planet. However, according to this hypothesis, as more complex evolutionary systems developed it was inevitable that 'wandering' genes would find it more difficult to find a home in a new host. Over time and as gene systems became more specialised they were able to replicate DNA accurately. In this way genes through generations formed clear lines of descent. Earliest evolution points to three main branches of life, the Eukaryotes (of which we are members), archaea and bacteria (*ibid*). However, although they became distinct branches, all carry a mixture of genes as a remembrance of their fluid genetic matrix past.

[69] Rizospheres are the environment surrounding the root systems of all plants.

[70] The Naturalistic Fallacy and the Is /Ought Distinction
In 1903, Moore put forth the proposition that the word 'good' is indefinable and refers to an irreducible quality. He says, in essence, that 'good' cannot be identified with say, pleasure, adaptation or anything else without loosing some of its connotation. Any attempt in this direction commits the 'naturalistic fallacy' (*ibid:* 10) – that is, the identification of the meaning of goodness (an ethical concept) with other natural properties (natural concepts). It is fallacious to argue

that a thing is good because it is natural (*ibid*: 45). To avoid this identification, Moore argues that the meaning of good refers to its own meaning and cannot be identified with anything else. Whatever the intrinsic nature of an object may be, its good is not connected, not necessarily a part of its characteristics or properties. He, however, does not deny that good is a property of certain natural objects (*ibid*: 101-111); he rather argues that to define good in terms of natural properties would make it something it is not, a complex rather than a simple term. He uses as his example, the colour yellow. The meaning of yellow cannot be identified with physical waves but only with an irreducible quality we cannot define, yellow. In addition, he claims that if a thing to which the predicate good attaches is good when it is 'asserted' by something it means that its 'existence is really good' (*ibid*: 120). Goodness, he holds, could be intuited in good acts, much as we can observe yellow in marigolds.

Moore defines nature as including everything that has existed, exists or will exist in time *(ibid*: 40). Natural existence is existent 'in time', but good cannot exist 'by itself in time'; 'not goodness but only things or qualities, which are good, can exist in time ... (they) can be objects of perception' (*ibid*: 110-111). In other words, Moore tells us that natural objects do exist in time and that they can be good.

The 'so-called' naturalistic fallacy (Blackburn 1994) that has inaugurated a 'wild-goose chase' (Sellars 1961: 428) is still haunting the minds of environmental ethicists who are grappling with the nemesis to locate intrinsic value in the biotic community via any naturalistic theory and overcome the naturalistic fallacy (Callicott 1989: 118, 158-159). Because this hurdle, Callicott (*ibid*: 120) dismisses it by saying that non-conformity with Moore's belief about the nature of goodness 'is hardly a cause for alarm'. He further argues that it is not a logical fallacy proper 'since no argument or passage from premise to conclusion is involved'. Assuming that good cannot exist without bearers of the property of goodness, it follows that goodness does not stand alone like a Platonic Idea. But Moore claims that goodness cannot be instantiated into nature or reality. And this is in contradiction with the fact that he did find appreciation of the existence of natural beauty as a good as Rolston (1988: 41) identifies.

In his attempts to overcome the naturalistic fallacy, Callicott (1989: 147; 2002: 292) draws on Hume's moral sentiment theory to base the idea of a biotic community that has interests and the ensuing moral implications. He makes the claim that Hume's distinction of 'is' from 'ought' may serve to separate scientific judgement from bias. Callicott (1989: 147) argues that from a scientific view, nature is 'an orderly, objective, axiologically neutral domain' and that value is 'projected onto natural objects or events by the subjective feeling of observers'. He further supports Hume's claim that ethics is grounded in feeling, not in reason. Since moral sentiments are natural and as such universal, he argues, they are not relativistic. None the less, he concedes that theories of moral sentiments are not a promising basis upon which to build a theory of intrinsic value of the biotic community. This is because intrinsic value means 'that value inheres in natural objects as an inherent characteristic, that is, a part of the constitution of things' (*ibid*: 160). To move from this impasse, Callicott argues that the relation to the subject of an intrinsically valuable entity is based on a distinct type of subjectivity, an other-regarding subject bestowing an intrinsic value independent of the valuer (*ibid*: 133). He illustrates this with the example of newborn babies. We value them for themselves apart of their own ability to value. This, he claims, shows that, within conventional ethics, a precedent has been set. Value is projected or ascribed beyond the evaluator; the assignment of intrinsic value is 'intentional' and based on altruism.

The extension of moral sentiments among humans to animals and then to nature as a whole is another precedent that has been set, for example, by the relationship humans have with their pets. And this, he argues, is a form of social contract similar to the human-to-human social contract. It commits us to morality and obligations (*ibid*: 163). Morality, he claims, is 'prescriptive and normative'; morality prescribes limits on our behaviour ('ought') (*ibid*: 77).

Several queries come to mind. First, whereas careful observation is required in the sciences, the interpretation may be subjective and biased. Nature may be value-free but its interpretation is in the eye of the beholder. Second, if a conscious judgement is based on subjective feelings, then, how can it be of independent value? Would it not deny the possibility of an entity bearing value apart? Furthermore, having an interest, even strong feelings for other beings is not the same as assigning them equal or intrinsic value. Perhaps it would be wise to side with Rolston (1988: xii) writing, 'whether and in what sense such an ethic can be derived from nature, gaining an ought from an is, will have to be discovered along the way'.

To assign intrinsic value to and to enter a social contract with microorganisms (as beneficial and indispensable they may be) represents practical and psychological problems. When the microorganisms are drug-resistant (harmful and threatening the biotic community) the difficulty is even greater. The fact is that they 'are' and thus it seems repugnant to ascribe them intrinsic value.

[71] This is a debated issue in environmental ethics as some authors contend that desire may be reduced to 'biofunctional' properties (*See* McGinn 1989; Millikan 1989). I will set aside this debate as it is beyond the scope to this dissertation.

***Notes to Chapter** 7*

[72] An interesting report from the National Research Council Committee on the ecology of the Northern Range of Yellowstone National Park is available from the USA National Academy Press. Online at http://www. books.nap.esd/books/0309083451/htm.

[73] I will try to summarise the new classification trends based mainly from a report by Stephens (1998): This group was initially classified as containing only 2 genera, and only 3 species (*Chlamydia trachomatis, Chlamydia psittaci,* and *Chlamydiophila* [or *Chlamydophila*] *pneumoniae*). Recently, through phylogenetic analysis, it has been identified that the group is larger than originally thought. The order *Chlamydiales*, and its family *Chlamydiaceae,* now contain 3 species (*C. suis, C. muridarium* and *C. trachomatis*). The order *Chlamydiophila* now contains 6 recognised species and several 'isolates' (including what is now *Chalymdophila psittaci,* as well as *C. abortus, C. caviae, C. felis, C. pecorum,* and *C. pneumoniae*), and an additional 3 genera; *Neochlamydia* (one species - *N. hartmannellae*), *Parachlamydia* (one species – *Parachlamydia acanthamoebae* [a parasite of the amoeba *Acanthamoeba*]), *Simkania* (one species — *Simkania negevensis),* and *Waddlia* (one species — *Waddlia chondrophila).* There are also over 100 uncharacterised clonal isolates as well. All members of this family are obligate intracellular parasites that rely on their host cell for their continued existence.

All of the family members appear to have a life cycle that includes two phases. The first is an inert spore-like stage or an 'elementary body' (EB) that exists in the environment waiting to be phagocytised and infect a new host cell. The second is a metabolically active or 'reticulate body' (RB) that exists only intracellularly. An EB infects a cell and develops into an RB, which continues to divides until the cell is used-up, then the RB's develop into EB's and the cell lyses, thus releasing them to infect other cells.

There is a relationship between the highly diverse Chlamydial species infecting humans and animals, particularly the species *C. psittaci* and *C. pecorum* because they have been empirically proven multihost (Shewen 1991: 2-11). Transmission of an ornithosis strain (*C. pittaci*) from turkeys to sheep and then back to turkeys was reported by Pierce *et al.* (1964). Yet, even when we know the broad host ranges of some pathogenic microorganisms (such as the rabies virus, *C. pittaci,* and *C. trachoma*), the question remains as to *how* traits carried by some strains breach the species barrier, only that they do.

[74] *C. trachomatis* in humans causes a venereal disease that is easily spread since most infections are asymptomatic and untreated. Infection can lead to pelvic inflammatory disease (PID) in women, urethritis in men, and if untreated or re-infected repeatedly, can result in eventually sterility. While treatable (if clinically diagnosed) in humans, atypical forms of *C. trachomatis* have been identified and it is suggested that these abnormal forms might have contributed to their persistence and relative resistance to antibiotics (Ward 1999). Ocular infection of this same species may lead to conjunctivitis and blindness in humans primarily in developing countries. Aside from the physical properties of the microorganisms - their apparent ability to lie dormant for long periods and their often vague clinical symptoms – other factors that appear to contribute to 'activation' are reported as poor living conditions, stress and unhygienic practices (Ward *et al.* 1990). I am unable to find the reference now, but a report from Australia indicated that *C. trachomatis* also infects the koala, resulting in infertility that, along with habitat loss, is a serious threat to the survival of the species. In laboratory experiments, severely immuno-suppressed mice, suffer severe infection, with prolonged shedding of live *chlamydiae* and extension of the infection into deep tissues and organs. This is consistent with the theoretical model of the relationship between interferon, cell mediated immunity and chlamydial development (Laitinen *et al.* 1996; Philips *et al.* 1995; Wang *et al.* 1967). While treatable (if clinically diagnosed) in humans, atypical forms of *C. trachomatis* have been identified and it is suggested these abnormal forms might have contributed to their persistence and relative resistance to antibiotics (Ward 1999).

[75] This is reminiscent of Taylor's (1995: 177-178) argument to 'remain neutral between the disease and the disease-causing'.

[76] Rolston assuredly would include 'duty' as well as value.

[77] Callicott does not release consciousness as the source of value as it is 'institutionalised in the scientific worldview'. By the institutionalisation of the scientific worldview, he refers to the framework of Cartesianism or the subject and object involved in 'value-free' descriptions of the natural world. He (1989: 133) writes: 'I concede that from the point of view of scientific naturalism, the *source* of all value is human consciousness, but it by no means follows that the

locus of all value is consciousness itself or a mode of consciousness like reason, pleasure or knowledge'(*ibid*). In his conceptualisation, the locus of intrinsic value is distinct from its source. Thus in a relation, value flows from a source to a locus. In other words, we can phenomenologically judge something to have intrinsic value independently of ourselves.

[78] I am grateful to Professor J. Hattingh, Environmental philosopher at the University of Stellenbosch (personal communication) who rightfully identified that global drug resistance is a clear example of a third generation environmental problem.

[79] In the context of species preservation, Norton, in a later work (1987), further conceptualises types of value enlarging on these concepts. For example, he defines two types of instrumental value, 'transformative value' and 'demand value' (*ibid*: 11). The former he defines as 'an occasion for examining or altering a felt preference rather than simply satisfying it' (*ibid*: 10). The latter he considers as a means to 'provide satisfaction of some felt preference, either before or after it has been examined' (*ibid*).

[80] Henry David Thoreau (1817-1862) was an individualist who believed moral law trumped legal law. Thoreau considered sincerity a principal value in human relations. Within nature, Thoreau found great inspirational but not intrinsic value in nature as Norton (1995: 184) notes.

[81] Norton (1995: 183) relates an example of the 'last man' scenario: Assume that a human being S, is the last living member of *Homo sapiens* and that S faces imminent death. Would S do wrong to destroy wantonly some object X? A positive answer to this question with regard to nonhuman X is taken to entail nonanthropocentrism. If the variable X refers to some natural object, a species, ecosystem, a geological formation, etc. then it is thought that positions on such questions determine whether a person is an anthropocentrist or not, because the question cannot conceivably harm any human individual. If it is wrong to destroy X, the wrongness must derive from harm to X or to some other natural object. But one can harm something only if it is good in its own right in the sense of being a locus of fundamental value. However, Norton disagrees that the 'last man' scenario can be used to define anthropocentrists from nonanthropocentrists.

[82] *See* Dewey, J. cited in Rolston (1989: 367).

[83] What is not overcome is holistic versus individualistic approaches in ethics. In this regard, his thesis stands in opposition to traditional teachings in moral philosophy.

Bibliography

- Agar, N. 2001. *Life's Intrinsic Value.* New York: Columbia University Press.
- Aggleton, P. 1988. *Health*. London: Routledge.
- Alexander, A. 1999. Emerging infectious disease in Botswana's game reserves. Available at: http:// www.cdc.gov/ncidod/EID/vol5no6/letters.
- Allan, J. R., Kirby, J. S., Feare, C. J. 1995. The biology of Canada geese *Branta canadensis* in relation to the management of feral populations. *Wildlife Biology,* 1(3): 129-143.
- Alland Jr. A. 1970. *Adaptation in Cultural Evolution: An Approach to Medical Anthropology*. New York: Columbia University Press.
- Alubo, S. O. 1985. Drugging the people: pills, profits and underdevelopment in Nigeria. In: B. E. Jackson, A. Ugalde (eds.). *The Impact of Development and Modern Technologies in Third World Health.* Williamsburg, Virginia: College of William and Mary: 89-113.
- ______________1994. Death for sale: a study of drug poisoning and deaths in Nigeria. *Journal of Social Science and Medicine,* 38: 97-103.
- Annas, G. J. 1993. Control of tuberculosis: The law and the public health. *New England Journal of Medicine*, 328: 585-588.
- Annas, G. J., Grodin, M. A. 1992. *The Nazi Doctors and the Nuremberg Code.* Oxford: Oxford University Press.
- Applebaum P. C., Bhamjee, A., Scragg, J. N. e*t al.* 1977. *Streptococcus pneumoniae* resistant to penicillin and chloramphenicol. *Lancet,* 2: 995-997.
- (APPJ) 1993. Population Measures, *Asia-Pacific Population Journal,* 8(3): 47-59.

- Aristotle. 1970. T. McKeon (ed.). *The Basic Works of Aristotle*. New York: Random House Publishers.
- Ash, R. J., Morgan, H., De Hite, S. *et al.* 1999. Antibiotic-resistant bacteria in US rivers. In: *Abstracts of the 99th General Meeting of the American Society of Microbiology* (Abstract Q-383). Chicago, Illinois: The Society.
- (ASM) American Society of Microbiology. 1994. *Report of the ASM task force on antibiotic resistance.* Washington, D.C.: The Society.
- ________________________________ 1999. *Antimicrobial Resistance: An Ecological Perspective.* (cited August 2002). Available through http://biosafety.ihe.be/AR/ARmenu.html.
- ________________________________ 2001. *Emerging and Re-emerging Infectious Diseases of Animals and Plants.* New York: The Society's Public and Scientific Affairs Board Press.
- Axelrod, R., Hamilton, W. D. 1981. The evolution of cooperation. *Science,* 211: 1390-1396.
- Ayres, A. 1998. Micrologix advances development of antimicrobial peptides for the treatment of eye infections. [Cited December 2003]. Available at http:// www. Mbiotex.com/newsreleases/nr090998.htm.
- Babbage, C. [1838]. 1967. *The Ninth Bridgewater Treatise*. London: Frank Cass Publisher: Chapter 9.
- Barber, E. J. W., Peters, A. M. W. 1992. Ontology and phylogeny: What child language and archaeology have to say to each other. In: J. A. Hawkins, M. Gell-Maui (eds.). *The Evolution of Languages.* Redwood City, California: Adine-Wesley Publishers: 305-352.
- Barclay, L. 2003. *Direct-to-consumer drug ads: A newsmaker interview with Tom Abrams.* (cited January 2003). Available at MEDSCAPE: http://www.medscape.com/viewarticle/448048.

- Barnett, A., Creese, A. L., Ayivor, E. C. K. 1980. The economics of pharmaceutical policy in Ghana. *International Journal of Health Services*, 10(3): 479-499.
- Bavoil, P. M., Hsia, R.-C. and Ojcius, D. M. 2000. Closing in on *Chlamydia* and its bag of tricks. *Microbiology,* 146: 2723 - 2731.
- (BBC) British Broadcasting Company. 2002. BBC News. Life expectancy in Africa. (cited December 2002). Available at: http://www. bbc.co.uk/1/hi/world/Africa/1814609.htm.
- Beauchamp, T. L., Childress, J. F. 1994. *Principles of Biomedical Ethics.* New York and Oxford: Oxford University Press.
- Beauchamp, T. L. 2002. Biomedical ethics in the 21st century. *The Aventis Magazine,* 3: 2-4.
- Bell, G. 1997. *The Basics of Selection.* New York: Chapman and Hall Publishers.
- _______. 2001. Neutral macroecology. *Science,* 293: 2413-2418.
- Benbrook C. M., Groth, E., Hoaioran, J. M. *et al.* 1996. *Pest Management at the Crossroads.* Yonkers, New York: Consumers Union Publication.
- Bender, G. 1975. *Farming in Pre-history: from Hunter-Gatherer to Food Producer.* London: J. Baker Publishers.
- Bennett, M., Haart, C. A. 1994. Hantavirus infection (editorial). *Journal of Medical Microbiology,* 41(2): 71-73.
- Berger, P. L. 2002. *The Cultural Side of Globalisation.* Oxford: Oxford University Press.
- Berger, P. L., S. Huntington (eds.). 2000. *Many Globalisations: Cultural Diversity in the Contemporary World.* Oxford: Oxford University Press.
- Berger, P. L., Berger B., Kellner, H. 1973. *The Homeless Mind: Modernization and Consciousness.* New York: Random House.

- Bester, K., Hühnerfuss, H., Neudorf, B. *et al.* 1995. Atmospheric deposition of triazine herbicides in Northern Germany and the German bight (North Sea). *Chemosphere,* 30(9): 1639-1653.
- Birungi, H. 1994. Injections as household utilities: Injection practices in Busoga, Eastern Uganda. In: N. L. Etkin, M. L. Tan (eds.). *Medicines: Meanings and Contexts.* Quezon City, Philippines: HAIN Publishers: 125-135.
- Blackburn, S. (ed.). 1996. *Oxford Dictionary of Philosophy.* Oxford: Oxford University Press.
- Bledsoe, C. H., Goubaud, M. F. 1988. The Reinterpretation and distribution of Western pharmaceuticals: an example from the Mende in Sierra Leone. In: S. Van der Geest, S. R. Whyte (eds.). *The Context of Medicines in Developing Countries. Studies in Pharmaceutical Anthropology.* New York: Plentium.
- Bojalil, R., Calva, J. J. 1994. Antibiotic misuse in diarrhoea: a household survey in a Mexican community. *Journal of Clinical Epidemiology,* 47: 147-156.
- Bomack, A. C. 1972. *Medicine and Pharmaceutical Practice.* Boston: Little Brown and Company.
- Boyd, K. M., R. Higgs, A. J. Pinching (eds.), 1997. *The New Dictionary of Medical Ethics.* London: British Medical Association Publishing Group.
- Bremner, J. A. 1994. Hantavirus infections and outbreaks in 1993. *Community Disease Report (Review),* 4(1): 5-9.
- Brennan, A. 1996. Incontinence, self-deception, shallow analysis, myth-making, and rationality. In: J. Baird Callicott, F. J. R. da Rocha (eds.). *Earth Summit Ethics.* Albany, New York: State University New York Press: 93-114.

- Brennan, A., Lo, Y-S. 2000. The early development of environmental ethics. In: The Stanford Dictionary of Philosophy. [Cited February 2004]. Available at: http://setis.library.usyd.edu.au/stanford/entries/ethics-environmental/.
- Brightman, E. S. 1961. Personalism. In: V. Ferm (ed.). *History of Philosophical Systems.* New York: Littlefield, Adams and Company: 340-352.
- Britten N. 1994. Patients' ideas about medicines: a qualitative study in a general practice population. *British Journal of General Practice,* 44: 465-468.
- Brock, T. 1990. *Microorganisms from Smallpox to Lyme Disease.* New York: W. H. Freedman and Company.
- Brodeur, P. 1985. *Outrageous Misconduct: The Asbestos Industry on Trial.* New York: Pantheon Books.
- Broecker, W. 1987. Quoted in: Nadakavukaren, A. 2000. *Our Global Environment: A Health Perspective.* Prospect Heights, Illinois: Waveland Press: 424.
- Brown, L., Renner, M., Flaven, C. 1998. *Vital Signs.* Worldwatch Institute: W W. Norton and Company.
- Brown, P. 1992. Popular epidemiology and toxic waste contamination. *Journal of Health and Social Behaviour,* 33: 267-281.
- Brown, P., Mikkelson, E. 1990. *No Safe Place.* Berkeley: University of California Press.
- Brundtland, G. H. 2000. WHO/UATLD *Global Project on Anti-Drug resistance Surveillance 1997-2000.* WHO Report Number 2.(cited January 2001). Available at: http://www.who/cds/tb/2000.

- Burnet, F. M. 1962. *Natural History of Infectious Disease, 3rd Edition.* Cambridge: Cambridge University Press.
- Bush, R. M., Everett, K. D. E. 2001. Molecular evolution of the *Chlamydiaceae. International Journal of Evolutionary Bacteriology*, 51: 203-220.
- Cahen, H. 1995. Against the moral considerability of ecosystems. In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition*. Belmont, CA: Wadsworth Publishing Company: 300-312.
- Callicott, J. B. 1989. *In Defense of the Land Ethic: Essays in Environmental Ethics.* Albany: State University of New York Press.
- _____________. 1993. The search for an environmental ethic. In: T. Regan (ed.). *Matters of Life and Death.* New York: McGraw Hill: 322-376
- _____________. 1995 (a). The conceptual foundations of the land ethic In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition*. Belmont, CA: Wadsworth Publishing Company: 151-163.
- _____________. 1995 (b). Second thoughts on " A triangular affair". In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition*. Belmont, CA: Wadsworth Publishing Company: 173-175.
- _____________. 2002. My reply. In: W. Ouderkirk, J. Hill, (eds.). *Land, Value, Community.* Albany, New York. State University of New York Press: 289-330.
- Callicott, J. B., F. J. R. da Rocha. (eds.). 1996. *Earth Summit Ethics.* Albany: State University of New York Press.
- Capron, A. M. 1993. Which ills to bear? Revaluating the 'threat' of modern genetics. In: T. Shannon (ed.). *Bioethics.* New Jersey: Paulist Press: 488-516.
- Carson, R. 1962. *Silent Spring.* Boston, Massachusetts: Houghton Mifflin.

- Catton, W. R. Jr., Dunlap, R. E. 1980. A new ecological paradigm for post-exuberant sociology. *American Behavioral Scientist,* (14) 1: 15-47.
- ______________________________1997. Environmental sociology. In: *The International Handbook of Environmental Science.* New York: New York University Press: 426-478.
- (CDC) 1997. USA Centers for Disease Control and Prevention. 1997. Update: Staphylococcus aureus with reduced susceptibility to vancomycin in United States, *Morbidity and Mortality Weekly*, 6: 813-815.
- ______________________________. 1999. *Antibiotics and breakpoints.* 1999. (cited June 1999). Available at: http://www.cdc.gov/ncidod/dbmd/narms/annual/1999/html/tables/table_2.
- ______________________________.2000 (a). *History of quarantine.* 2000. (cited April 2001). Available at: http://www.cdc.gov/ncidod/dq/history.
- ______________________________. 2000 (b). *Antimicrobial resistance.* 2000. (cited January 2000). Available at: http://www.cdc.gov/drugresistance/.
- ______________________________. 2001. *Addressing emerging infectious disease threats: A Prevention Strategy for the United States.* 2001. Atlanta Georgia: USA Department of Health and Human Services.
- (CEQ) Council on Environmental Quality. 1996. *Environmental quality.* Washington DC: Council on Environmental Quality, Executive Office of the President Publication.
- ______________________________1997. *Lyme disease.* Washington DC: Council on Environmental Quality, Executive Office of the President Publication.

- Chambers, N., Simmons, C., Wachernagel, M. 2001. *Sharing Nature's interest: Ecological footprints as an indicator of sustainability*. London: Earthscan Publications.
- Chantrey, J., Meyer, H ., Baxby, D. *et al.* 1999. Cowpox: reservoir hosts and geographic range. *Epidemiology and Infection,* 122: 455-460.
- Chapin, C. 1921. State and municipal control of disease. In: M. Ravenel (ed.). *A Half Century of Public Health.* New York: American Public Health Association: 133-160.
- Cheng, S., Kalkstein, L. S., Focks, D. A. *et al.* 1998. New procedures to estimate water temperature and water depths for application in climate dengue modelling. *Journal of Medical Entomology,* 35:646-652.
- Childs, J. E., Krebs, J. W., Ksiazek, T. G. *et al.* 1995. A household-based, case-control study of environmental factors associated with hantavirus pulmonary syndrome in the southwestern United States. *American Journal of Tropical Medicine and Hygiene,* 52(5): 393-7.
- Chisholm, S. W., Rocap, G., Moore, L. 2000. *Prochlorococcus:* the smallest and most abundant photosynthetic microbe in the ocean. *Department of Education (USA) Human Genome Program Contractor-Grantee Workshop VIII February 27-March 2, 2000. Santa Fe, New Mexico. Abstract 125*: The Department of Education (USA) Publication.
- Christiani, D. C. 1993. Urban and trans-boundary air pollution: Human health consequences. In: E. Chivian, M. McCally, H. Hu, A. Haines (eds.). *Critical Condition: Human Health and the Environment.* Cambridge, Massachusetts: MIT Press: 13-30.
- Chua, K. B., Bellini, W. J., Rota, P.A. *et al.* 2000. Nipah virus: A recently emergent deadly paramyxovirus. *Science,* 228: 1432-1435.

- (CISET) Committee on Science, Engineering and Technology Committee Report. 1995. Infectious diseases a global health threat. In: *National Science and Technology Council Committee on International Science, Engineering, and Technology (CISET) Working Group on Emerging and Re-Emerging Infectious Diseases.* Washington, DC: World Health Organization Publication.
- Clark, S. R. L. 1997. Ancient philosophy. In: A. Kenny, (ed.). *The Oxford Illustrated History of Western Philosophy*. Oxford: Oxford University Press.
- Clegg J. C. S., Lloyd, G. 1995. Ebola haemorrhagic fever in Zaire '95: A perspective. *Current Opinion in Infectious Diseases*, 8: 225-228.
- Cockburn, T. A. 1967. *Infectious Diseases: Their Evolution and Eradication.* Springfield, Illinois: Charles C. Thomas.
- Coetzee, J. M. 1999. *Disgrace*. New York: Vintage Press.
- Coetzer, P. W. W. 2002. *The Ecology of Environmental Hygiene and Occupational Health.* Pretoria: Medical University of Southern Africa.
- Coffman, J. R., Beran, G. W. 1999. Trends in hospital antibiotic prescribing. *Journal of Antimicrobial Therapy,* 38: 5906-5910.
- Cohen, M. N., Armelagos, G. J. (eds.). 1984. *Palaeopathology and the Origin of Agriculture*. Orlando, Florida: Academic Press.
- Cohen, M. N. 1971. Infectious disease in ancient populations. *Current Anthropology,* 12(1): 45-62.
- ____________1977. *Food Crisis in Prehistory.* New Haven, Connecticut: Yale University Press.
- ____________1989. *Health and the Rise of Civilization.* New Haven, Connecticut: Yale University Press.

- Cohen, M. N., Armelagos G. J. (eds.). 1984. *Palaeopathology at the Origin of Agriculture.* Orlando, Florida: Academic Press.
- Coluzzi, M. 1994. Malaria and the afrotropical ecosystems: Impact of man-made environmental changes. *Parasitologia,* 36: 223-227.
- Colwell, R. R. 1996. Global climate change and infectious disease: the cholera paradigm. *Science,* 274: 2025-2031.
- Commoner, B. 1991. Rapid population growth and environmental stress. *International Journal of Health Services,* 91: 199-221.
- ____________1992. *Making Peace with the Planet.* New York: The New Press.
- Converse, K. C., Kidd, G. A. 2001. Duck plaque epizootics in the U S, 1967-1995. *Journal of Wildlife Diseases,* 37(2): 347-357.
- Cookson, S. T., Carballo, M., Nolan, C. M. *et al.* 2001. Migrating populations- A closer look at who, why, and so what. *Emerging Infectious Diseases,* 7 (Supplement): 551.
- Cooper, J. 1975. *Reason and Human Good in Aristotle.* Cambridge Massachusetts: Harvard University Press.
- Copleston, F. 1962. *A History of Philosophy.* Garden City New Jersey: Doubleday Image.
- Coppo, A., Colombo, M., Pazani, C. *et al.* 1995. Vibrio cholera in the horn of Africa: epidemiology, plasmids, tetracycline gene amplification and comparison between 01 and non-01 strains. *American Journal of Tropical Medicine and Hygiene,* 53: 351-359.
- Couper, M. R. 1997. Strategies for the rational use of antibiotics. *Clinical Infectious Disease,* 24 (Supplement 1): S154-S156.
- Coswell, J. P. 1967. *The History of Disease.* New York: Penguin Publishers.

- Cunha, B. A. 2000. Antibiotic resistance. *Medical Clinics of North America,* 84(6): 1407-1429.
- Cutler, J., Parker, G., Rosen, S. *et al*. 1986. Childhood leukemia in Wolburn Massachusetts. *Public Health Reports*, 101: 201-205.
- Cuttle, P. 2002. Safeguarding America's bounty: Plant protection and quarantine. (cited October 2002). Available at: http://www.ceris.perdue.edu/napis/a-facts/fsbounty.
- Cutter, S. (ed.). 1994. *Environmental Risks and Hazards.* Englewood Cliffs: Prentice-Hall.
- Darwin: C. 1859. On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life, First edition. (cited March 2000). Available at: http://www.literature.org/authors/darwin-charles/the origin of the species.
- De Laet, S. J. (ed.). 1994. *History of Humanity Volume 1: Prehistory and the Beginning of Civilization*. New York and London: Routledge.
- Diamond, J. 1996. Why women change. *Discover,* 6: 130-133.
- Dickens, P. 1992. *Society and Nature.* Philadelphia: Temple University Press.
- Dickinson, P. 2000. Management and avoidance of antibiotic resistance. *Program and abstract from the American Academy of Family Physicians 52nd Annual Scientific Assembly.* Dallas, Texas: The Association: 89.
- Dirckx, J. H. (ed.). 1997. *Stedman's Concise Medical and Allied Health Dictionary.* Baltimore: Williams and Wilkins Company.
- Dirks, R. 1999. Famine and disease. In: K. F. Kiple (ed.). *The Cambridge World History of Human Disease.* Cambridge: Cambridge University Press: 157-163.

- Dockery, D. 1996. An association between air pollution and mortality in six U.S. cities. *The New England Journal of Medicine*, 329(24): 347-353.
- Donovan, B. S. 1974. Improving drug use through education. *Journal of International Pharmaceutics,* 2: 375-377.
- Donskey, C. J., Chowdhry, T. K., Hecker, M. T. *et al.* 2000. Effect of antibiotic therapy on the density of vancomycin-resistant enterococci in the stool of colonized patients. *New England Journal of Medicine,* 343: 1925-1932.
- Douglas, M. 1970. *Natural Symbols.* London: Crescent Press.
- Dromigny, J., Rakoto-Alson, O., Miglaiani, R. *et al.* 2002. Emergence and rapid spread of tetracycline-resistant *Vibrio cholerae* strains. *Emerging Infectious Disease,* 8(3): 336-338.
- Dua, V., Kunin, C., White, L. V. 1994. The use of antimicrobial drugs in Nagpur India: A window on medical care in developing countries. *Journal of Social Science and Medicine,* 38: 717-724.
- Dubos, R. 1959. *Mirage of Health.* Garden City New York: Anchor Books.
- _________1965. *Man Adapting.* New Haven Connecticut: Yale University Press.
- Du Caju, M., Heinrichs, C., de Beaufort, C. *et al.* 2001. Sexual precocity after immigration from developing countries to Belgium: evidence of previous exposure to organochlorine pesticides. *Human Reproduction,* 16(5): 1020-1026.
- Duncan, R. C. 1993. The life-expectancy of industrial civilization: The decline to global equilibrium. *Population and Environment,* 14(4): 325-357.
- Dunlap, W., Catton, R. 1980. A new ecological paradigm for post-exuberant sociology. *American Behavoral Scientist,* 24: 791-805.

- (EC) 1999. European Commission. *The European Agency for the Evaluation of Medicinal Products Veterinary Medicines Evaluation Unit: Antibiotic Resistance in the European Union Associated with Therapeutic use of Veterinary Medicines* (cited July 1999). Available through http://biosafety.ihe.be/AR/ARmenu.
- (EC) 2001. European Commission of the European Union. *Strategies for the Control of Pollutants and Environmental Hazards*. (cited October 2002). Available at: http://europa.eu.int/comm/research/guide/en/a-14.
- (EEC) 1999. The European Economic Commission. *The European Commission Opinion of the Scientific Steering Committee on Antimicrobial Resistance.* (cited May 2000). Available through http://biosafety.ihe.be/AR/ARmenu.
- Ehrenfeld, D. 1981. *The Arrogance of Humanism. New* York and Oxford: Oxford University Press.
- Eichorst, S., Tischler, M. L. 1999. Antibiotic resistance among bacteria isolated from wild populations of resident Canada geese in a suburban setting. (Abstract Q-402: 610). In: *Abstracts of the 99th General Meeting of the American Society of Microbiology.* Chicago, Illinois: The American Society of Microbiology.
- Eisenberg, L., Kleinman A. 1981. Culture, illness, and care: Clinical lessons from anthropologic and cross-cultural research. *Annuals of Internal Medicine,* 88: 51-58.
- Ekins, P. 1999. *Economic Growth and Environmental Sustainability.* New York and International: Routledge.
- Elliott, C. 1999. *A Philosophical Disease.* New York and London: Routledge.

- Elliot, R. (ed.). 1998. *Environmental Ethics.* Oxford: Oxford University Press.
- Epstein, P. 1995. *Climate Change and Human Health.* USGCRP (USA Climate Change Science Program / USA Global Change Research Seminar) July 10, 1995. (cited August 2001). Available at:www.usgcrp.gov.
- Epstein, P., Ford, T., Puccia, C. *et al.* 1994. Marine ecosystem health: Implications for public health. In: Diseases in evolution, global changes and emergence of infectious diseases. *Annals of the New York Academy of Sciences,* 740: 13-23.
- Epstein, P., McMichael, A. 2001. Climate change and health; A response to the National Academy. *Health and the Global Environment,* 3: 236-243.
- Epstein, Y. M. 1981. Crowding stress and human behavior. *Journal of Social Issues,* 1:45-49.
- EthicSA. 2001. The Ethics Institute of South Africa: *Ethics Audit of Chris Hani Baragwanath Hospital.* Pretoria, South Africa: The Institute.
- Etzel, R. A. 20002. Mycotoxins. *Journal of the American Medical Association,* 287(4): 425-427.
- Eurosurveillance Weekly. 2001. R*apid reporting EU surveillance system for meningitis W135*: 2a: P1.2, 5 – update and summary. (cited April 2003). Available at: http://www.eurosurv.org/2001/010607.
- Evans, L. T. 1998. *Feeding the 10 Billion.* New York: Doubleday Books.
- Evans, R., Stoddart, G. 1990. Producing health, consuming health care. *Social Science and Medicine,* 31: 791-805.
- Everett, K. D. 1999. *Chlamydia* and *Chlamydiales*: more than meets the eye. *Veterinary Microbiology,* 75: 100-126.

- Ewald, P. W. 1994. *Evolution of Infectious Disease.* New York: Oxford University Press.
- Eyles, J. 1994. *Social Indicators, Social Justice and Social Well-being.* Ottawa: McMaster Centre for Health Policy Publication.
- Eyles, J., Donovan, J. 1990. *The Social Effects of Health Policy.* Aldershot: Avebury Press.
- Eyles, J., Woods, K. J. 1983. *The Social Geography of Medicine and Health.* New York: St. Martins Press.
- Faeth, P. 1994. Building a case for sustainable agriculture. *Environment,* 361: 312-319.
- (FAO). Food and Agricultural Organization of the United Nations 1993. *The Challenge of Sustainable Forest Management: What Future for the World's Forest?* Rome: FAO Publication.
- Farmer, P. 1996. Social inequalities and emerging infectious diseases. *Emerging Infectious Diseases,* 2: 259-269.
- (FASE) Foundation in Advancements in Science and Education. 1996. *Research Report 1-2 Antibiotic Misuse.* Foundation in Advancements in Science and Education. Washington, DC: The Foundation.
- Feare, C. J.,Sanders, M. F., Blasco, R. *et al.* 1999. Canada goose (*Branta canadensis*) droppings as a potential source of pathogenic bacteria. *Journal of the Royal Society of Health,* 119(3):146-155.
- (FDA) 1999. Food and Drug Administration of the United States. 1999. *Food Additives in Animal Production Report from the FDA 1999 BD2467-C.* Washington, D.C.: The Administration.
- ____. 2000. *FDA / Industry Meeting Presentation Anti-infectives for Resistant Bacteria*

Including Selective Spectrum Agents. (cited June 2000). Available through http://biosafety.ihe.be/AR/ARmenu.

- Fedoroff, N. V., Cohen, J. E. 1999. Plants and population: Is there time? *Proceedings of the National Academy of Sciences USA,* 96: 5903-5707. New York: The Academy.
- Fenlon, D. R. 1999. *Listeria monocytogenes* in the natural environment. In: E. T. Ryser, E. H. Marth (eds.). *Listeria, Listeriosis, and Food Safety.* New York: Marcel Dekker, Inc.: 21-37.
- Fenner, F. 1970. *The Impact of Civilisation on the Biology of Man.* Canberra: Australian National University Press.
- Ferguson, A. 1988. Commercial pharmaceutical medicine and medicalisation: A case study from El Salvador. In: S. Van der Geest, S. R. Whyte (eds.). *The Context of Medicines in Developing Countries. Studies in Pharmaceutical Anthropology.* Dordrecht, The Netherlands: Kluwer Academic Publishers: 19-46.
- Fidler D. P. 1997. Return of the fourth horseman: emerging infectious diseases and international law. *Minnesota Law Review,* 81: 771-868 Minnesota: The Law Commission.
- Fleming, A. quoted In: Zimmer, C. 2001. *Evolution-the Triumph of an Idea.* New York: Harper Collins: 214.
- Fluckiger, U., Widmer, A. F. 1999. Epidemiology of methicillin-resistant *Staphylococcus aureus*. *Chemotherapy,* 45: 121-134.
- Foucault, M. 1973. *Birth of the Clinic.* London: Tavistock.
- ___________1965 *Madness and Civilization*. New York: Vintage.
- Frankena, W. 1963. *Ethics.* Englewood Cliffs, New Jersey: Prentice Hall Publishers.
- Freidson, E. 1970. *Profession of Medicine*. New York: Dodds.

- Fridkin, S. K., Edwards, J. R., Pichette, S. C. *et al.* 1999. Determinants of vancomycin use in adult intensive care units in 41 United States hospitals. *Clinical Infectious Diseases,* 28: 1119-1125.
- Furbo. J. E., R. C. Rist, R. Sandal (eds.). 2002. *International Atlas of Evaluation Comparative Policy Series.* New York: Transation Publishers.
- Futuyama, D. J. 1986. *Evolutionary Biology.* Sunderland, Massachusetts: Sinauer Press.
- Galimand, M., Guiyoule, A., Gerbaud, G. *et al.* 1997. Multidrug resistance in *Yersinia pestis* mediated by a transferable plasmid. *New England Journal of Medicine,* (337):677-680.
- Gao, F., Bailes, E., Robertson, D. L. *et al.* 1999. Origin of HIV 1 in the chimpanzee Pan troglodytes troglodytes. *Nature,* 397: 436-441.
- Garforth, F. W. 1971. *The Scope of Philosophy.* London: Longman Group Publishers.
- Garrett, L. 1994. *The Coming Plague.* New York: Penguin Books.
- ________2003. Probing virus' roots. *Newsday* (cited April 27, 2003). Available at: http: //www.newsday.com/news/health/ny-hssars013200941apr01,0,1665890.story?coll=ny-health-headlines.
- Gelbard, A., Haub C., Kent, M. M. 1999. World Population and beyond 6 billion. Population Reference Bureau, Inc. (cited April 2002). Available at http:// www. prb.org/pubs/population_bulletin/bu54-1/54_1_intro.
- Georghiou, G. P. 1990. Overview of insecticide resistance. *American Chemical Society Symposium Series,* 421: 18-41.
- Gernhart, G. A. 1999. Forgotten enemy: PHS's fight against the 1918 influenza pandemic. *Public Health Report,* 114: 559-561.
- Giddens, A. 1984. *The Constitution of Society.* Cambridge: Polity Press.

- Gilliver, M., Bennett, M., Begon, M. *et al.* 1999. Antibiotic resistance found in wild rodents. *Nature,* 401: 233-234.
- ________________________________ 2001 Antibiotic resistance-How wild are wild mammals? Reply. *Nature,* 409: 30-38.
- Gillman, M. 1986. Early agriculture in Egypt. *Nutrition,* 21(4): 91-94.
- Gleick, P. H. 1993. *Water in Crisis.* New York: Oxford University Press.
- Godish, T. 1991. *Air Quality.* Chelsea Mississippi: Lewis Publishers.
- Goel, P., Ross-Degnan, D., Berman, P. *et al.* 1996. Retail pharmacies in developing countries. *Journal of Social Science and Medicine,* 42: 1155-1161.
- Goldberg, M. 2001. *Health and the Environment: Stress in Farm Animals.* Harvard Center for Health and the Environment. Harvard, Massachusetts: The Center: 1-3.
- Goodpaster, K. E. 1978. On being morally considerable. In, *Journal of Philosophy* 22: 308-325.
- Gopal-Rao, G. 1987. Injections in the Indian subcontinent. *British Medical Journal,* 295: 1281.
- Gostin, L. 1990. Controlling the resurgent tuberculosis epidemic: a 50-state survey of TB statutes and proposals for reform. *Journal of the American Medical Association,* 269: 20-36.
- _________ 2000. *Public Health Law: Power, Duty, Restraint.* New York and Berkeley, California: Milbank Memorial Fund and University of California Press.
- Gottlieb, S. 2003 CDC reports first case of vancomycin resistant *Staphylococcus aureus.* (cited June 2003). *British Medical Journal* on line at bmj.com.

- Gould, H. A. 1965. Modern medicine and folk cognition in rural India. *Human Organization,* 24: 201-208.
- Gould, S. J. 2002. *The Structure of Evolutionary Theory.* Harvard, Massachusetts: The Belnap Press of Harvard University Press.
- Govender, A. 1999. Tuberculosis South Africa: IPSF Scientific Subcommittee 1998/99. (cited December 1999). Available at : http://www.pharmweb.net/pwmirror/pw9/fip/ipsf/projects/comple_edu/tuberculosis.
- Grady, D. 1996. Quick-change pathogens gain an evolutionary edge. *Science,* 274: 1081.
- Gray, T. (1747) 1993. Ode on the death of a favourite cat. In: *Norton's Anthology of English Literature 6th Edition Volume 1.* New York: Johanna Lipking and W.W. Norton Publishing: 1247.
- Greenhalgh, T. 1987. Drug prescription and self-medication in India: an exploratory survey. *Social Science and Medicine,* 25(3): 307-318.
- Grey, W. 1993. Anthropocentrism and deep ecology. *Australasian Journal of Philosophy,* 7(4): 463-475.
- Griffin, D. R. 1996. Introduction to SUNY series in constructive postmodern thought. In: J.B. Callicott, and F.J.R. da Rocha (eds.). *Earth Summit Ethics.* Albany, New York: State University New York Press: ix-xiii.
- Grigg, D. B. 1993. *The World Food Problem.* Oxford, United Kingdom: Blackwell Publishers.
- Gross, P. R. (ed.). 1996. *The Flight from Science and Reason.* New York: The New York Academy of Sciences.

- Grimes, J. E., Owens, K. J., Singer, J. R. 1979. Experimental transmission of *Chlamydia psittaci* to turkeys from wild birds. *Avian Diseases,* 23(4):915-26.
- Guerrant R. L. 1997. *Cryptosporidiosis*: An emerging, highly infectious threat. *Emerging Infectious Diseases,* 3: 1-6.
- Guerrant, R. L., D. H. Walker, P. F. Wells (eds.). 1999. *Tropical Infectious Diseases.* Philadelphia, Pennsylvania: Churchill Livingstone.
- Gushalak, B. D., McPherson, D. W. 2000. Population mobility and infectious diseases. *Clinical Infectious Diseases,* 31: 776-780.
- Haak, H., Hardon, A. P. 1996. Antibiotic prophylaxis in developing countries, widely used, widely neglected. *Lancet,* 2: 620-621.
- Hardin, G. (1968) 1995. The Tragedy of the commons. In: C. Pierce and D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition.* New York: Wadsworth Publishers: 330-338.
- ________________ 2001. Memorandum. (cited June 2002). Available at: http://www.iapm.org/newsletters/june01.
- Hardin, G., Simon, J. 1982. Is the era of limits running out? A conversation with Garrett Hardin and Julian Simon. *Public Opinion,* 5 34-39. Reprinted in: Simon, J. 1990. *Population Matters.* New Brunswick, New Jersey: Transaction Publishers.
- Hart C. A., Kariuki, S. 1998. Antimicrobial resistance in developing countries. *British Medical Journal,* 317: 647-650.
- Harte, J., Holderen, C., Schneider, R. 1991. *Toxics A to Z.* Berkley: University of California Press.
- Hartshorne, C. 1961. Panpsychism. In: V. Ferm (ed.) *Philosophical Systems..* New York: Littlefield, Adams and Company.

- Harvard Center for Health and the Environment. Farming and environment. 2002. (cited December 2002). Available at: http:www:med.arvard.edu/chge/.
- Hatch, J. J. 1996. Threats to public health from gulls (*Laridae*). *International Journal of Environmental Health Research,* 6(1): 516.
- Hay, A. J. 2001. Potential of Influenza A viruses to cause pandemics. In, W. L. Irving, J. W. McCauley, D. J. Rolands, G. L. Smith (eds.). *New Challenges to Health: The Threat of Viral Infection, 60th Symposium of the Society for General Microbiology.* Cambridge: Cambridge University Press: 89-104.
- Hayward, T. 1997. Anthropocentrism: a misunderstood problem. *Environmental Values,* 6: 49-63.
- Helliker, B. G. 2001. Pesticide production. *Farm Industry News,* 3(1): 1-6.
- Helman, C. G. 2001. *Culture, Health, and Illness, 4th Edition.* New York: Arnold Publishers.
- Henderson, D. A., Inglesby, T. V., Bartlett, J. G. *et al.* 2000. Smallpox as a biological weapon: medical and public health management. *Journal of the American Medical Association,* 281: 2127-2137.
- Hensley S. 2001. Purchases of antibiotic jump on fears of bioterrorism, but doctors see risks. *Wall Street Journal.* October 9:A1.
- Herlihy, D. 1997. *The Black Death and the Transformation of the West.* Cambridge, Massachusetts: Harvard University Press.
- Hippocrates.(460-377 BCE). *Air, Water and Places.* The Electronic Library of World Literature. New York: Microsoft Corporation.
- Hoang, G. N. , Erickson, R. V. 1985. Cultural barriers to effective medical care among Indochinese patients. *Annual Review of Medicine,* 36: 229-39.

- Hoffman, F. L. 1908. The Mortality of Consumption in Dusty Trades. USA Bureau of Labor Bulletin no. 79. Washington, D.C. quoted in: Carmichael, A. Public Health and Sanitation in the West before 1700. In: K. Kiple, (ed.). *The Cambridge History of Human Disease.* 1993. Cambridge: Cambridge University Press: 196.
- Hoffman, W. M. 1993. Business and environmental ethics. In: T. White (ed.). *Business Ethics. A Philosophical Reader.* New Jersey: Prentice Hall: 830-834.
- Hoffmaster, B. 1984. *Bioethics in Social Context.* Philadelphia Pennsylvania: Temple University Press.
- Hossain, M., Glass, R., Khan, M. R. 1982. Antibiotic use in a rural community in Bangladesh. *International Journal of Epidemiology,* 11 402-405.
- Houghton, J. T., Jenkins, G. J. 1996. *Climate Change: The IPPC Scientific Assessment.* Cambridge: Cambridge University Press.
- Hu, H., Kotha, S., Brennan, T. 1995. The role of nutrition in mitigating environmental insults: Policy and ethical issues. *Environmental Health Perspectives*, 103(6): 186.
- Hudson, R. 1993. Concepts of disease in the West. In: K. Kiple (ed.). *The Cambridge World History of Human Disease.* Cambridge, United Kingdom: Cambridge University Press: 45-52.
- Hughes, J. M., Peters, C. J., Cohen, M. L. *et al.* 1993. Hantavirus pulmonary syndrome: an emerging infectious disease. *Science,* 262(5135): 850-851.
- Hüskes. R., Levsen, K. 1997. Pesticides in rain. *Chemosphere,* 35: 3013-3024.

- (IM) Institute of Medicine. 2000. *Emerging Infections: Microbial Threats to Health in the United States, 1992-2000.* Washington: National Academy Press.
- International Soil Reference Center. 1996. *Proceedings of the International Soil Reference Center Conference of the World Resource Institute.* Oxford: Oxford University Press.
- Isaäcson, M., Frean, J. A. 2001. African malaria vectors in European aircraft. *Lancet,* 357: 235
- Iseki, M. 1994. Effects of increases in the world's population and the globalization of human life on the incidence of parasitic diseases. *Japanese Journal of Parasitology*, 43: 448-452.
- Jackson M., Lynch, P. 1985. Isolation practices: a historical perspective. *American Journal of Infection Control,* 13: 21-31.
- Jewson 1976. The disappearance of the sick man from medical cosmology. *Sociology,* 10: 225-244.
- Jonas, H. 1984. *The Imperative of Responsibility, In Search of an Ethics for the Technological Age.* Chicago: University of Chicago Press.
- ________1999. *Mortality and Morality: A Search for the Good After Auschwitz.* L. Vogel (ed.). Evanston, Illinois: Northwestern University Press.
- Kam, K. M., Luey, K. Y., Fung, S. M. e*t al.* 1995. Emergence of multiple-antibiotic-resistant *Streptococcus pneumoniae* in Hong Kong. *Antimicrobial Agents and Chemotherapy,* 39: 2667-2670.
- Kamm F. 1992. Non-consequentialism , the person as an end in itself and the significance of status. *Philosophy and Public Affairs*, 21: 354-389.
- Kant, I. (1785) 1996. *The Metaphysics of Morals.* M. Gregor and R. J. Sullivan (eds.). Cambridge: Cambridge University Press.

- _________(1797) 1983. *The Metaphysical Principles of Virtue.* (James W. Ellington , trans.). Indianapolis, Indiana: Hackett Publishing Company.
- Khan, A. Q. 1967. Role of carriers in the intrafamilial spread of cholera. *Lancet,* 35: 245-246.
- Khan, A. S., Tshioko, F. K., Heymann, D. L. *et al.* 1995. The re-emergence of Ebola fever in the Democratic Republic of the Congo, *Journal of Infectious Diseases,* 179 (Supplement 1): 76-86.
- Khun, T. S. 1970. *The Structure of Scientific Revolutions.* Chicago: University of Chicago Press.
- Khuse, H., P. Singer. (eds.). 1998. *A Companion to Bioethics.* New York and London: Blackwell Publishers.
- King, L. S. 1958. *The Growth of Medical Thought.* Chicago: Haldane.
- King, M. H., Elliott, C. M. 1999. To the point of farce: a martian view of the Hardinian taboo-the silence that surrounds population control. *British Medical Journal,* 315: 141-143.
- Kiple, K. F. (ed.). 1993. *The Cambridge World History of Human Disease.* Cambridge: Cambridge University Press.
- Klein, R. 2000. Archaeology and the evolution of human behaviour. *Evolutionary Anthropology*, 9: 17-36.
- Kleinman, A. 1988. *The Illness Narratives.* New York: Basic Books.
- Kleinsmith, L. J., Kish, V. M. 1988. *Principles of Cell Biology.* Philadelphia, Pennsylvania: Harper and Row Publishers.
- Klugman, K. P. 1998. *Emerging infectious diseases-South Africa.* Emerging Infectious Diseases National Center for Infectious Diseases Centers for Disease Control and Prevention (cited April 1999). Available at http://www.ccd.gov. emerginginf/doc.4.3.

- Kofman, F. 2001. quoted in *The Fifth Discipline Fieldbook*. 2001. P. Senge, R. Ross, B. Smith, C. Roberts, A. Kleiner (eds.). London: Nicholas Brealey Publishers.
- Komesaroff, P. 1995. *Troubled Bodies*. Durham and London: Duke University Press.
- Korte, R., Rehle, T., Merkle, A. 1996. Strategies to maintain health in the third world. *Journal of Tropical Medicine and Parasitology,* 42: 428-432.
- Kristeller, P.O. 1961. Renaissance philosophy. In: V. Ferm (ed.) *History of Philosophical Systems*. New York: Littlefield, Adams and Company: 227-239.
- Kroll-Smith, S., P. Brown, V. Gunter (eds.). 2000. *Illness and the Environment: A Reader in Contested Medicine*. New York: New York University Press.
-
- Kunin, C. 1987. Social, behavioural, and practical factors affecting antibiotics use world wide. *Reviews of Infectious Diseases,* 9 (Supplement 3): 270-285.
- __________1993. Resistance to antimicrobial drugs a worldwide calamity. *Annals of Internal Medicine*, 118: 557-561.
- Kymail, G. 2003. *Rising Desertification in Nigeria*. Lagos: Nigerian Department of Health and Social Services Publication.
- Labonte, R. N. 1989. Pesticides and healthy public policy. *Canadian Journal of Public Health,* 80: 238-244.
- Land, T. 1992. Combating counterfeit drugs. *Nature,* 355: 192.
- Laitinen, K., Laurila, A. L., Leinonen, M., *et al.* 1996. Reactivation of *Chlamydia pneumoniae* infection in mice by cortisone treatment. *Infection and Immunity,* 64: 1488-1490.

- Last, J. 1987. *Public Health and Human Ecology.* East Norwalk: Appleton and Lang.
- Latouche, S. 1993. *In the Wake of the Affluent Society: An Exploration of Post-Development.* New York: Zed Books.
- ______________1996. (R. Morris, Translator). *The Westernisation of the World: The Significance Scope and Limits towards the Drive for Global Transformation.* New York: Polity Press.
- Lederberg, J. 1984. *Harnessing biomedical research to health needs.* Geneva: World Health Organisation Press: 60.
- ____________1986. Forty years of genetic recombination in bacteria. *Nature,* 324(6098): 627-628.
- ____________1993. Crowded at the Summit: emergent infections and the global food chain. *American Society of Microbiology News,* 45(4): 162-163.
- ____________1994. Emerging infections: Private concerns and public responses. *American Society of Microbiology News*, 60(5): 233-251.
- ____________1997. Infectious diseases as an evolutionary Paradigm. *Emerging Infectious Diseases,* 3(4): 417-423.
- Lederberg, J., Shope, R. E., Oaks, Jr. S. C. 1992. *Emerging Infections: Medical Threats to Health in the United States.* New York: Institute of Medicine National Academy Press.
- Lemons, J. 1996. Afterword. In: J. Baird Callicott, F. J. R. da Rocha (eds.). *Earth Summit Ethics.* New York: State University of New York Press: 193-220.
- Leopold, A. 1995. The Land Ethic. In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins and Plastic Trees 2nd Edition.* Belmont, California: Wadsworth Publishing Company: 142-151.

- Leopold, A. (1949) 1966. *A Sand Country Almanac*. New York: Ballantine Books.
- Lesho, M. E., Dorsey M. D., Bunner, D.1998. Feces, dead horses and fleas: Evolution of the hostile use of biologic agents. *Western Journal of Medicine*, 168: 512-516.
- Leslie, G. B., Haraprasad, V. 1993. Indoor air pollution from combustion sources in developing countries. *Indoor Environment,* 2: 4-13.
- Levin, H. M. 1993. Micronutrient deficiency disorders. In: D. T. Jamison (ed.). *Disease Control Priorities in Developing Countries.* New York: Oxford University Press: 424-439.
- Levy, M. A., Keohane, R. O., Has, P. M. 1992. Institutions for the earth: Promoting international environmental protection. *Environment,* 34(4): 12-17.
- Levy, S. B. 1986. Antibiotic use for growth promotion in animals: ecologic and public health consequences. *Journal of Food Products,* 50: 616-620.
- ________1993. Confronting antimicrobial resistance. *Journal of the American Medical Association,* 269: 1840-1842.
- ________1996. The challenge of antibiotic resistance. *Scientific American,* 278(3): 46-53.
- ________2002. *The Antibiotic Paradox: How Miracle Drugs are Destroying the Miracle, 2nd Edition.* Boston, Massachusetts: Persus Books.
- Levy, S. B., Fitzgerald, G. B., Macone, A. B. 1976 (a). Spread of antibiotic resistance plasmids from chicken to chicken and from chicken to man. *Nature,* 260: 40-42.
- ____1976 (b). Changes in intestinal flora of farm personnel after introduction of tetracycline-

supplemented feed on a farm. *New England Journal of Medicine,* 295: 583-588.

- Levy, S. B., J. Burke, E. Wallace (eds.). 1987. Antibiotic use and antibiotic resistance worldwide. *Infectious Disease Review,* 8: (Supplement 3).
- Lewn, R. 1997. *Complexity Life on the Edge of Chaos.* London: Phoenix.
- Lewis, M. W. 1996. Radical environmental philosophy and the assault on reason. In: P. R. Gross, M. W. Lewis (eds.). *The Flight from Science and Reason.* New York: The New York Academy of Sciences: 209-230.
- Lewis, R. 1995. The rise of antibiotic resistant infections. *Food and Drug Administration Consumer Magazine,* 9(1): 54-61.
- Li, J., Willke, R., Pinto, L. *et al.* 2001. Comparison of length of hospital stay for patients with known or suspected methicillin-resistant *staphylococcus* species infections treated with linezolid or vancomycin: a randomized, multicenter trial. *Pharmacotherapy,* 21: 263-267.
- Light, A. 2001. Taking environmental ethics public. In: D. Schmidtz and E. Willott (eds.). *Environmental Ethics. What Really Matters? What Really Works?* Oxford: Oxford University Press.
- Lindeboom, G. A. 1993. *Inleiding tot de Geschiedenis der Geneeskunde.* Rotterdam: Erasmus Publications.
- Locke, J. 1689 (1971) *Two Treatises of Government.* Cambridge: Cambridge University Press.
- Lomborg, B. 2001. *The Sceptical Environmentalist.* Cambridge: University of Cambridge Press.
- Loop, C. 2000. Testimony before the Subcommittee on livestock and horticulture regarding invasive species. (cited July 2000). Available at: http://www.fb.com/flfb/issues/invcom.

- Lovelock, J. 1979. *Gaia: A New Look at Life on Earth.* Oxford: Oxford University Press.
- Luke, T. 1991. Community and ecology. *Telos*, 88: 69-79.
- Luckin, B. 1984. States and Epidemic Threats. *Bulletin of the Social History of Medicine*, 3: 34-42.
- Lutzenberger, J. 1996. Science, technology, economics, ethics and environment. In: J. B. Callicott, and F. J. R. da Rocha, (eds.). *Earth Summit Ethics*. New York: State University of New York Press: 23-46.
- Lyell, D. (1830-1833). The Principles of Geology; or the Modern Changes of the Earth and its Inhabitants as Illustrative of Geology, 3 vol. London: Murray Publishers. Quoted in: Gould, R. J. 2002. *The Structure of Evolutionary Theory.* Cambridge, Massachusetts: Belnap Press of Harvard University: 63.
- Lynch, K. 1981. *Good City Form.* Cambridge: MIT Press.
- McKeown, T. 1976. *The Role of Medicine*. Oxford: Blackwell.
- McKibben, B. 1990. *The End of Nature.* New York: Anchor Books.
- MacDonald, D., Barrett, P. 1995. *Collin's Field Guide: Mammals in Britain and Europe.* London: Harper Collins Publishers.
- MacIntosh, R. P. 2002. Ecological science, philosophy, and ecological Ethics. In: W. Ouderkirk, J. Hill, (eds.). *Land, Value, Community: Callicott and Environmental Philosophy.* Albany, New York: State University of New York Press: 59-84.
- Maddy K. T., Edminston S., Richmond D. 1990. Illness, injuries, and deaths from pesticide exposure in California, 1949-1988. *Reviews of Environmental Contamination and Toxicology*, 114: 57-123.

- Madsen, P. 1996. What can universities and professional schools do to save the environment? In: J. B. Callicott, F.J.R. da Rocha (eds.). *Earth Summit Ethics*. Albany, New York: State University New York Press: 71-91.
- Mallon D. J. P., Salvern, D., Merchant, K. *et al.* 2002. Excretion of vancomycin-resistant *enterococci* by wild mammals. *Emerging Infectious Diseases,* 8: 636-638.
- Malone, T. F. 1993. Is there hope for humanity? Cited by Lemons, J. 1996. In: J. Baird Callicott, F. J. R. da Rocha (eds.). *Earth Summit Ethics.* New York: State University of New York Press: 208-209.
- Malthus, T. R. (1798) 1986. An Essay on the principle of population , as it affects the future improvement of society: with remarks on the speculations of Mr. Godwin M. Condorcet, and other writers. In: E.A. Wrigley, D. Souden (eds.). 1986. *The Works of Thomas Robert Malthus*. London: W. Pickering.
- Maranan, M. C., Moreira, B., Boyle-Vavra, S. *et al.* 1997. Antimicrobial resistance in staphylococci: epidemiology, molecular mechanisms, and clinical relevance. *Infectious Diseases Clinics of North America*, 11: 813-849.
- Marchese A., Schito, G. C. 2001. Role of global surveillance in combating bacterial resistance. *Drugs,* 61(2): 167-173.
- Margulis, L., Swartz, K. V. 1998. *Five Kingdoms*. New York: W.H. Freeman.
- Markel, H. 1995. Knocking out the cholera: cholera, class, and quarantines in New York City, 1892. *Bulletin of the History of Medicine,* 69: 420-457.
- Markles, D. 2003. Counterfeit Artesunate Tablets: Holographic images of Counterfeit Products. (cited April 2003). Available at shoklo-unit.com/fakeasz.pdf.

- Marris, R. 1994. *The Economic Theory of 'Managerial' Capitalism.* New York: The Free Press.
- Martel, K. P. 1995. Survey of antimicrobial resistance in bacterial isolates from diseased cattle in France. *Microbiology and Drug resistance,* 3: 273-283.
- Martell, L. 1994. *Ecology and Society.* New York: Blackwell Publishers.
- Martens, P., Hall, L. 2000. Malaria on the move: human population movements and malaria transmission. *Emerging Infectious Diseases,* 6: 103-109.
- Martinez B., Harris G. 2001. Doctors struggle to allay patients' fears as requests for anthrax antibiotic surge. *Wall Street Journal.* (October 15:B1).
- Martins, D. 2003. Considerations from the Boston conference. *The Southern African Journal of HIV Medicine*, May 2003: 8-16.
- Mayr, E. 1980. How I became a Darwinian. In: E. Mayr, W. B. Provine (eds.). *The Evolutionary Synthesis.* Cambridge, Massachusetts: Harvard University Press: 413-423.
- ________. 2001. *What Evolution Is.* New York: Basic Books.
- Mayer, E., Provine, W. B. 1998. *The Evolutionary Synthesis: Perspectives on the Unification of Biology.* Cambridge, Massachusetts: Harvard University Press.
- Mayer, J. 1979. Energy, food, and the consumer. *Technology Review,* 8: 42-46.
- McCabe, D. 2001. Upsetting the biodiversity applecart. The McGill Reporter. (cited April 2001). Available at: http://www.mcgill.ca/reporter/35/05/bell/.
- McGarry, F. R., D. Mara (eds.). 1977. *Weather, Wastes and Health in Hot Climates.* Chichester: John Wiley.

- McGinn, C. 1989. *Mental Content*. Oxford: Blackwell Publishers.
- McKenna, E., Light, A. 2004. *Animal Pragmatism: Rethinking Human-Nonhuman Relationships*. Bloomington, Indiana: Indiana University Press.
- McMichael A. J. 1993. *Planetary Overload: Global Environmental Change and the Health of the Human Species*. Cambridge, United Kingdom: Cambridge University Press.
- McMichael, A. J., Patz, J., Kovats, R. S. 1998. Impacts of global environmental change on future health and health care in tropical countries. *British Medical Bulletin,* 54: 475-488.
- McMichael, A. J., Guillebraud, J., King, M. 1999. Contrasting views on human population growth. *British Medical Journal,* 319: 931-932.
- McNamee, T. quoted in Norton, B. 1991. *Towards Unity Among Environmentalists*. New York: Oxford University Press: 155.
- McNeill, W. H. 1976. *Plagues and People.* Garden City, New Jersey: Anchor Doubleday.
- Mellors, M. D., Kuritzkes, D. 1998. Antiretroviral Therapy: Resistance. 8th Clinical Options in HIV Symposium. (cited 2000 Sept). Available at: http://www.meds.cape.com/meds.cape/HIV/journal/1998/v04.n02/hiv0402.03/hiv0402.03html.
- Melrose, D. 1982. *Bitter Pills: Medicines and the Third World Poor.* Oxford: OXFAM Publications.
- Mercer, A. 1993. *Death, disease and mortality in the demographic transition: epidemiological-demographic change in England since the eighteenth century as part of a global phenomenon.* Leicester, United Kingdom: Wiley and Sons.
- Merchant, C. 1980. *The Death of Nature: Women, Ecology and the Scientific Revolution.* San Francisco: Harper and Row: 44-45.

- *Merck Manual.* R. Berklow (ed.). 1995. Rathway, New Jersey: Merck Research Laboratories Publication.
- Messer, E. 1996. Hunger as a weapon of war. In: E. Messer, P. Uvin (eds.). *The Hunger Report.* Amsterdam: Gordon and Breach Science Publishers: 19-48.
- Mesthene, E. G. 1990. *Technological Change: Its Impact on Man and Society*. Harvard, Massachusetts: Harvard University Press.
- Michel, J. M. 1985. Why do people like medicines? A perspective from Africa. *The Lancet,* (i): 210-211.
- Midgley, M. 1995. The mixed community. In: C. Pierce and D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition.* New York: Wadsworth Publishers: 99-103.
- Milio, N. 1981. *Promoting Health through Public Policy.* Philadelphia: F. A. Davis.
- Mill, J. S. (1859; 1871) 1973. *On Liberty and Utilitarianism.* New York and Canada: Bantam.
- Miller, J. M., Boyd, H. A., Ostrowski, S. R. *et al.* 2000. Malaria, intestinal parasites and schistosomiasis among Barawan Somali refugees resettling to the United States. *American Journal of Tropical Medicine and Hygiene,* 65: 115-121.
- Millikan, R. 1989. In defence of proper functions. *Philosophy of Science,* 56: 288-302.
- Milton, K. 1993. Diet and primate evolution. *Scientific American,* 260: 86-93.
- Minteer, B., Manning, R. 1999. Pragmatism in environmental ethics: Democracy, pluralism and the management of nature. *Environmental Ethics,* 21: 191-207.

- Mithen, S. 1996. The early prehistory of human social behaviour: Issues of archaeological inference and cognitive evolution. In: W. C. Runciman, J., R. I. Maynard-Smith, M. Dunbar (eds.). *Evolution of Social Behaviour Patterns in Primates and Man: Proceedings of the British Academy.* London: The Academy.
- (MMWR) Morbidity and Mortality Weekly. 2001 Outbreak of Ebola hemorrhagic fever Uganda, August 2000-January 2001. *Morbidity and Mortality Weekly,* 50: 73-77.
- Mobley J. A. 1995. Biological warfare in the twentieth century: Lessons from the past, challenges for the future. *Milestones in Medicine,* 160: 547-553.
- Moulder, J. W. 1988. Characteristics of chlamydiae. In: A. L. Barron, (ed.). *Microbiology of Chlamydia*. Boca Raton, Florida: CRC Press: 21-46.
- Moore, B. N., Bruder, K. 1996. *Philosophy: The Power of Ideas.* Mountain View, CA: Mayfield Publishing Company.
- Moore, G. E. (1903) 1987. *Principa Ethica.* Cambridge: Cambridge University Press.
- Moore, P. S., Reeves, M. W., Schwartz, B. *et al.* 1989. Intercontinental spread of an endemic group A *Neisseria* meningitis strain. *Lancet,* 2: 260-263.
- Morse, S. S. 1993. Examining the origins of emerging viruses. In: S. S. Morse (ed.). *Emerging Viruses.* New York: Oxford University Press : 10-28.
- __________1995 (a). Factors in the emergence of infectious diseases. *Emerging Infectious Diseases,* 1: 7-14.
- __________1995 (b). Crossing over: the interspecies traffic of emerging infections. *Journal of Infectious Diseases,* 162: 17-14.

- ___________1997. The public health threat of emerging viral disease. *American Society for Nutritional Sciences,* 127 (Supplement): 951-957.
- Morticatti, L. J. 1969. *Western Philosophers.* London: Thames Press: 321.
- Muir, J. [1916]. 1981. *A Thousand Mile Walk to the Gulf.* Boston: Houghton Mifflin Company.
- Müller, A., Bialek, R., Kämper, A. *et al.* 2001. Detection of *microsporidia* in travelers with diarrhoea. *Journal of Clinical Microbiology,* 39: 1630-1632.
- Mumford, L. quoted in Rifkin, J. 1999. *The Biotech Century: Harnessing the Gene and Remaking the World.* New York: J. P. Tarcher Publishing: 7.
- Murphy, F. A. 2000. *A Perspective on Emerging Zoonotic Diseases: The Institute of Medicine Report.* Washington: The Institute.
- Murphy, R. 1994. *Rationality and Nature. A Sociological Inquiry into a Changing Relationship.* Boulder, Colorado: Westview Press.
- Murray, C. J. L., Lopez, A. D. 1997. *The Global Burden of Disease.* Geneva: World Health Organization Publication.
- Murray, K, Eaton, B., Hooper, P. *et al.* 1998. Flying foxes, horses and humans: A zoonosis caused by a new member of the *Paramyxoviridae.* In: W. M. Scheld, D. Armstrong, J. M. Hughes (eds.). *Emerging Infections.* Washington DC: American Society of Microbiology Press: 17-64.
- Murray, P. R., E. J. Baron, P. Faller, *et al.* (eds.). 1999. *Manual of Clinical Microbiology.* Washington, DC: American Society of Microbiology Press.
- Murry, B. E. What can we do about vancomycin-resistant *enterococci*? *Clinical Infectious Diseases,* 20:1134-1136.
- Myers, N. 1993. Environmental refugees in a globally warmed world. *BioScience,* 43: 752-761.

- Nadakavukaren, A. 2000. *Our Global Environment: A Health Perspective.* Prospect Heights, Illinois: Waveland Press.
- Naess, A. 1989. *Ecology, Community and Lifestyle: Outline of an Ecosophy,* (D. Rothenberg, Translator.). Cambridge: Cambridge University Press.
- (NAS) 1987. National Academy of Science (USA) *Antimicrobial Resistance a World-wide Concern.* New York: The Academy.
- (NAS) 2002. National Academy of Science (USA) *Evaluation of the Introduction of Biological Agents through Imports to the United States.* National Academy of Science Report. (cited June 2002). Available at: http://www.4.nas.edu/weber.nsf/ProjectScopeDisplay/BANR-O-00-12-A.
- Neergard, L. 1999. Food production and antimicrobial uses. *Animal Health Digest,* 2: 1-7.
- Neutra, R. 1991. Hypothesis to explain the higher symptom rates observed around hazardous waste sites. *Environmental Health Perspectives,* 94: 31-38.
- (NNIS) 1998. *National Nosocomial Infections Surveillance System report, data summary from October 1986 through April 1998.* Hospital Infections Program, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia: The Center.
- Nolan, C. M. 1994. A new era of TB resurgence demands updated laws and regulations. *Washington Public Health,* 12: 1-3.
- Norton, B. G. 1987. *Why Preserve Natural Variety?* New Jersey: Princeton University Press.
- ____________ 1991 (a). *Towards Unity Among Environmentalists.* New York and Oxford: Oxford University Press.

- _______________1991 (b). Thoreau's insect analogies: Or why environmentalists hate mainstream economists. *Environmental Ethics,* 13 (3): 203–221.
- _________________1995. Environmental ethics and weak anthropocentrism In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition.* Belmont, CA: Wadsworth Publishing Company: 182-192.
- Novak, D. 1998. Water Quality and Health. In: P. H. Gleick (ed.). *Water in Crisis.* Oxford: Oxford University Press: 103-128.
- Nussbaum, M., A. Sen (eds.). 1993. *The Quality of Life.* World Institute for Developing Economics Research Studies in Developmental Economics. Oxford: Oxford University Press.
- Nwokolo, N., Parry, E. H. O. 1989. Injections and health (Editorial). *Tropical Doctor*, 19(3): 97-98.
- O'Brien. T. F. 2002. Emergence, spread, and environmental effects of antimicrobial resistance: How use of an antimicrobial anywhere can increase resistance to any antimicrobial anywhere else. *Communicable Infectious Diseases,* 34 (Supplement 3): 78-81.
- Odum, E. P. 1959. *Fundamentals of Ecology.* New York: W B Saunders and Company.
- Omran, A. R. 1971. The Epidemiological Transition: A Theory of the Epidemiology of Population Change. *Milbank Memorial Fund Quarterly,* XLIX (4).
- Orr, D. 1996. noted in: Lemons, J. 1996. Afterword. In: J. Baird Callicott, F. J. R. da Rocha (eds.). *Earth Summit Ethics.* New York: State University of New York Press: 193-220.
- Ostroff, S. M., Kozarsky, P. 1998. Emerging infectious diseases and travel medicine. *Infectious Disease Clinics of North America*, 12: 231-241.

- Ouderkirk, W. 2002. *Land, Value, Community: Callicott and Environmental Philosophy.* W. Ouderkirk and J. Hill, (eds.). Albany, New York: State University of New York Press.
- (PAHO) Pan-American Health Organization. 2000. Antibiotic Resistance: Are We Killing the Cures? In: *Pan American Health Organisation Perspectives in Health*, 7(1). (cited July 2001). Available at: http://www.paho.org/english/DP/Number 13_article1_5.htm.
- ________________________________.2002. Epidemiological study of acute pesticide poisoning in the Central American Isthmus. *Epidemiological Bulletin,* 23(3). (cited November 2002. Available at: http://www. pao.org/.
- Partridge, E., 2001. Ecological morality and nonmoral sentiments. In: W. Ouderkirk and J. Hill (eds.). *Land, Value, Community: Callicott and Environmental Philosophy.* Albany, New York: State University of New York Press: 21-36.
- Parsons, T. 1951. *The Social System.* London: Routledge and Kegan Paul.
- Passmore, J. 1974. *Man's Responsibility for Nature.* New York: Charles Scribner's Sons: 116. Discussed in Callicott, J. B. 1989. *In Defense of the Land Ethic: Essays in Environmental Ethics.* Albany: State University of New York Press: 70-71, 75.
- __________1995. Attitudes to nature. In: R. Elliot (ed.). *Environmental Ethics.* Oxford: Oxford University Press: 129-141.
- Patz, J. A., Martens, W. J. M., Focks, T. A. *et al.* 2000. Dengue fever epidemic potential as projected by general circulation models of global climate change. *Environmental Health Perspectives,* 106: 147-153.
- Pearce, F. 1998. *Population bombshell.* (cited November 2001). Available at: http://www.dhushara.com/book/diversit/extra/popb/popb.

- Peeters C. 2002. *Riskante Humaniteit*. Vrij: The Nederland.
- Perry, R. 1954. *Realms of value, A Critique of Human Civilization.* Cambridge, Massachusetts: Harvard University Press.
- Phifer, P., Roebuck, P. 2001. The complexity of population growth: reply to Pletscher and Schwartz. *Conservation Biology,* 15(6): 1810-1811.
- Phillips, R. S. 2001. Current status of malaria and potential for control. *Clinical Microbiology Reviews,* 14: 208-226.
- Philips, H. L., Clarkson, M. J. 1995: Spontaneous change from overt to covert infection of *Chlamydia pecorum* in cycloheximide treated mouse McCoy cells. *Infection and Immunity,* 63: 3729-3730.
- Pierce, C., D. VanDe Veer (.eds.). *People, Penguins, and Plastic Trees.* New York and International: Wadsworth Publishing.
- Pierce, K. R., Carroll L. H., Moore, R. W. 1964. Experimental transmission of ornithosis from sheep to turkeys. *American Journal of Veterinary Research,* 25: 977-980.
- Pimentel, D. 1994. Natural resources and an optimum human population. *Population and Environment,* 1(1): 27-32.
- ____________1995. Protecting crops. In: W. C. Olsen (ed.). *The Literature of Crop Science.* Ithaca, New York: Cornell University Press: 49-66.
- ____________1995b. Environmental and economic costs of soil erosion. *Science,* 2(24): 48-53.
- Pimentel D., Bashore, T. 1998. Environmental and economic issues associated with pesticide use. *International Conference on Pesticide Use in Developing Countries: Impact on Health and Environment. Book of Abstracts: 8-14.* Costa Rica: San Jose Universidad National Printers.

- Pimentel, D., Pimentel, M. 1996. *Food, Energy and Society.* Niwot, Colorado: University Press of Colorado.
- Pindar, P. (518-438 BCE). 1974. *Pindar's Odes.* RA Swanson (ed.). Indianapolis, Indiana: Bobbs-Merril Company: 61.
- Pinchot, G. [1947]. 1981. *Breaking New Ground.* Washington DC: Island Press.
- Platt, A. 1996. *Infecting Ourselves: How Environmental and Social Disruptions Trigger Disease.* Worldwatch Paper, 129: Washington, DC: The Institute.
- Pletscher, D., Schwartz, M. 2000. The tyranny of population growth. *Conservation Biology,* 14: 1918-1919.
- Plotkin, H. C. 1986. Learning and evolution. In: H. C. Plotkin (ed.). *The Role of Behaviour in Evolution.* Cambridge, Massachusetts: MIT Press.
- Plowright, W. 1988. Viruses transmissible between wild and domestic animals In: G. R. Smith, J. P. Hearn (eds.). *Reproduction and Disease in Captive and Wild Animals.* Oxford, Oxfordshire and New York: Oxford University Press: 175-199.
- Plumwood, V. 1991. Ethics and instrumentalism: A response to Janna Thompson. *Environmental Ethics,* 13: 139-149.
- ______________1995. Nature, self, and gender. Feminism, environmental philosophy and the critique of rationalism. In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees. 2nd Edition.* Belmont, CA: Wadsworth Publishing Company: 197-213.
- Polgar, S. 1964. Evolution and the ills of mankind. In: S. Tax (ed.). *Horizons of Anthropology.* Chicago: Aldine: 76-89.
- Potter, V. R., Whitehouse, P J. 1998. Deep and global bioethics for a liveable third millennium. *The Scientist,* 12(1): 9.

- Potts, M. 1999. The population policy pendulum. *British Medical Journal,* 319: 932-933.
- Praveen, S., Nakagoshi, N. 2002. Impact of pesticides on the environment: Perceptions, knowledge and use practices of rice farmers of Japan and Bangladesh. (cited November 2002). Abstract available at http://eco-informa.ead.anl.gov/abstracts/54F_Praveen.pdf.
- (PRB) Population Reference Bureau. 1996. World Population Data Sheet. Washington (DC): Population Reference Bureau. (cited November 2002). Available at: http://www.worldpop.org/prbdata.
- ___________________________(PRB) Population Reference Bureau. 1999-2000. Population reference bureau statistics. (cited November 2002). Available at: http://www.worldpop.org/prbdata.
- Preston, S. H. 1996. The effect of population growth on environmental quality. *Population Research and Policy Review,* 15: 95-108.
- Priestly, J. J. 1968. Civilisation, water and wastes. *Chemistry and Industry*, 3: 254-258.
- Public Citizen. 2002. *Pharmaceutical industry ranks as most profitable industry-again*. (cited November 2002). Available at: http://www.citizen.org/pressroom/release.cfm?ID=1088.
- Rachels, J. 1976. Darwin, species and morality. In: T. Regan, P. Singer (eds.). *Animal Rights and Human Obligations.* Englewood Cliffs, New Jersey: Prentice Hall Publishers.
- __________ 1995. Created from animals. In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees 2nd Edition.* New York and International: Wadsworth Publishing: 62- 65.
- Raleigh, V. 1999. Population and health in transition. *British Medical Journal*, 319: 981-984.

- Raloff, M. 1999. Emerging antibiotic resistance. In: *World Resources 1998-1999.* World Resources Institute. Cambridge: Oxford University Press.
- Raloff, J. 1998 (a). Drugged waters. *Science News,* 153: 187.
- ________1998 (b). Livestock's role in antibiotic misuse. *Science News*, 154:159.
- _________1999. Waterways carry antibiotic resistance. *The Weekly Newsmagazine of Science*, 155(23): 636-638.
- Ravigiloane, M. 1999. *Global Tuberculosis Control.* Publication WHO/CD/TB/2002.275. Geneva: The World Health Organisation.
- Rawls, J. A. 1997. A theory of justice. In: H. La Follette (ed.). *Ethics in Practice*. Cambridge, Massachusetts: Blackwell Publishers: 519-532.
- Raz, J. 1984. On the nature of rights. *Mind,* 33. 194- 214.
- Real, L. A. 1996. Sustainability and the ecology of infectious diseases. *BioScience*, 46: 88-97.
- Reed, C. 2000. *One Child Policy and its Importance in Limiting China's Population*. Cleveland: Ohio State University Press.
- Regan, T. 1983. *The Case for Animal Rights.* Berkley, California: University of California Press.
- Regan, T., P. Singer (eds.). 1976. *Animal Rights and Human Obligations.* Englewood Cliffs, New Jersey: Prentice Hall Publishers.
- Reinhard, K. J. 1992. The impact of diet, and parasitism on anemia in the prehistoric West. In: P. Stuart-McAdam, S. Kent (eds.). *Diet, Demography and Disease: Changing Perspectives of Anemia.* New York: Aldine deGruyter: 219-258.
- Relman, D. 2000. Emerging Pathogens. 38th Meeting of the Infectious Diseases Society of America. Conference summaries. (cited April 2001). Available at:medscape.com/meds.cape/CNO/2000/ISDACS/ISDA-02.

- Rescher, N. 1969. *Introduction to Value Theory*. Englewood Cliffs, New Jersey: Prentice Hall Publishers.
- Richerson, L. M., Davis, M., Larouche, A. *et al.* 2001. Archaeology, climate and farming. *American Antiquities,* 66: 387.
- Richter, T. E., Ronald, P. C. 2000. The evolution of disease resistance genes. *Plant Molecular Biology,* 42: 195-204.
- Rifkin, J. 1999. *The Biotech Century: Harnessing the Gene and Remaking the World.* New York: J. P. Tarcher Publishing.
- ___________2001. *The Age of Access: The New Culture of Hypercapitalism Where All Life is a Paid-For Experience*. New York: J. P. Tarcher Publishing.
- Risse, G. B. 1993. Western medicine from Hippocrates to the germ theory. In: K. Kiple (ed.). *The Cambridge History of World Disease.* Cambridge: Cambridge University Press.
- ___________1992. A long pull, a strong pull, and all together: San Francisco and bubonic plague, 1907-1908. *Bulletin of the History of Medicine*, 66: 260-286.
- Rist, G. 1997. *The History of Development: Western Origins to Global Faith.* New York: Transation Publishers.
- Rogun, W. J. 1980. Pollutants in breast milk. *New England Journal of Medicine,* 302: 1450-1453.
- Rohe, J. F. 1998. *A Bicentennial Malthusian Essay: Conservation, Population and the Indifference to Limits*. Traverse City, Mississippi: Rhodes and Eastman.
- Rolston III, H. 1982. Are values in nature subjective or objective? *Environmental Ethics,* 4(2): 134.

- ______________1988. *Environmental Ethics, Duties to and Values in the Natural World.* Philadelphia: Temple University Press.
- ______________1996. Earth ethics: A challenge to liberal education. In: *Earth Summit Ethics.* J. B. Callicott, F. R. da Rocha, (eds.). New York: State University of New York Press: 161-192.
- ______________1997. Feeding People versus saving nature. In: R. S. Gottlieb (ed.). *The Ecological Community.* New York: Routledge.
- ______________1999. Ethics on the home planet. In: A. Weston (ed.). *An Invitation to Environmental Ethics.* New York & Oxford: Oxford University Press: 107-139.
- ______________2002. Naturalizing Callicott. In: W. Ouderkirk, J. Hill (eds.). *Land, Value, Community: Callicott and Environmental Philosophy.* Albany: State University of New York Press: 107-122.
- Ross, I. 1968. The influenza epidemic of 1918. *American History Illustrated*, 3:12-17.
- Rotberg, R. I. (ed.). 2000. *Health and Disease in Human History.* Cambridge, Massachusetts: MIT Press.
- Rue, L., Mercer, D. P., Valotte, H. *et al.* 1998. Tetracycline resistance in costal waterways. *Journal of Water Affairs,* 2(5): 342-348.
- Runciman, W. C., J. Maynard-Smith, R. I .M Dunbar (eds.). 1996. *Evolution of Social Behaviour Patterns in Primates and Man. Proceedings of the British Academy.* Cambridge: The Academy.
- Ruckelshaus, W. D. 1995. Toward a sustainable world. In: C. Pierce, D. VanDe Veer (eds.) *People, Penguins, and Plastic Trees, 2nd edition.* Belmont, CA, Wadsworth Publishing Company: 425-431.
- Ruse, M. 1995. *Evolutionary Naturalism.* London: Routledge: 242.

- Russell, J. C. 1972. Population in Europe. In: C. M. Cipolla (ed.). *The Fontana Economic History of Europe, Vol. I: The Middle Ages*. Glasgow: Collins/Fontana: 25-71.
- (SAIRR) South African Institute of Race Relations. 2001. Life Expectancy in Sub-Saharan Africa. (cited June 2002). Available at http:wwwsairr.org.za/wsc/pstories.htx?story id+210.
- Satin, M. 1976. *New Age Politics*. Vancouver, BC: Fairweather Press.
- Savitz, D. A., John, E. M., Kleckner, R. C. 1990. Magnetic Field exposure from electrical appliances and childhood cancer. *American Journal of Epidemiology,* 131: 763-771.
- Scambler, G. 1993. *Epilepsy.* London: Routledge.
- ___________2000. *Sociology and Medicine.* New York: W. B. Saunders.
-
- Schopf, L. 1993. Microfossils in the early Archean Apex chart: New evidence of the antiquity of life. *Science,* 260: 620-646.
- _________1999. *Cradle of Life: The Discovery of the Earth's Earliest Fossils.* Princeton: Princeton University Press.
- Schrader-Frechette, K. 1991. *Risk and Rationality.* Berkley: University of California Press.
- Schrader-Frechette, K., McCoy, E. 1993. *Method in Ecology.* Cambridge: University of California Press.
- Schultz, W. 2003. Amnesty International News Conference Human Rights Situation I. Washington DC. (cited June 2003). Available at: http www:clearwisdom_net.
- Schweitzer, A. 1987. *The Philosophy of Civilization.* Buffalo, N Y: Prometheus.

- ___________ 1976. Civilization and ethics. (J. Nash, Translator). Reprinted In: T. Regan, P. Singer (eds.). *Animal Rights and Human Obligations.* Englewood Cliffs, New Jersey: Prentice Hall.
- Scriven, M. 1962. Evaluation perspectives and procedures. In: J. W. Popham (ed.). *Evaluation in Education: Current Applications.* Berkley, California: Mc Cutchan Publishing Company.
- Sen, A. 1981. *Poverty and Famine.* Oxford: Clarendon Press.
- _______1994. *Sustainable Human Development: Concepts and Priorities, Occasional Paper 8 in Sustainable Human Development* (cited July 2000). Available at: http://www.undp.org/undp/hdro/oc8a.htm.
- Serba, J. 1997. Reconciling anthropocentric and nonanthropocentric environmental ethics. In: H. La Follette (ed.). *Ethics in Practice.* Cambridge, Massachusetts: Blackwell Publishers: 644-656.
- Serba, J. A. 1999. *Cradle of Life: The Discovery of the Earth's Earliest Fossils.* Princeton: Princeton University Press.
- Shakespeare, W. (1564-1616) *Hamlet.* Act ii, Scene 2. Full quotation available at http: www.bartleby.com/100/138.32.92.html.
- Shewen, P. E. 1991. Chlamydial infection in animals: A review. *Canadian Veterinary Journal*, 21: 2-11.
- Stern, W. (1897) quoted in: Schweitzer, A. 1987. *The Philosophy of Civilization*. Buffalo, New York: Prometheus: 259.
- Shilling, C. 1993. *The Body and Social Theory.* London: Sage Publishers.
- Shue. H 1996: *Basic Rights: Subsistence, Affluence and U.S. Foreign Policy.* Princeton: Princeton University Press.
- Siddique, A. K., Salam, A., Islam, M. S. *et al.* 1995. Why treatment centres failed to prevent cholera deaths among Rwandan refugees in Goma, Zaire. *Lancet,* 345 (8946): 359-361.

- Silverman, M., Lydecker, M., Lee, P. 1992. *Bad Medicine: The Prescription Drug History in the Third World.* Stanford, California: Stanford University Press.
- Simmons, I. 1993 *Interpreting Nature.* London: Routledge.
- Simon, J. L. 1981. *The Ultimate Resource.* Princeton, New Jersey: Princeton University Press.
- ____________1990. *Population Matters: People, Resources, Environment, and Immigration*. New Brunswick, New .Jersey: Transactions Press.
- Singer, P. 1995. Animal liberation. In: Pierce, C. D. VanDe Veer (eds.). *People, Penguins, and Plastic Trees*. New York and International: Wadsworth Publishing: 51-19.
- __________1990. *Animal Liberation, 2nd Edition.* New York: Review Press.
- Smil, V. 1991. Growth and nitrogen, exploring a critical balance. *Population and Development Review,* 17(4): 75-79.
- Smith, A., Jacobson, B. 1988. *The Nation's Health.* London: King's Fund.
- Smith, B. E. 1981. 'Black Lung' and the social production of disease. *International Journal of Health Services*, 11: 343-349.
- Smith, K. E., Hutchenson, L. J., Fernandez, P. B. *et al.* 1999. Quinolone-resistant *Campylobacter jejuni* infections in Minnesota, 1992-1998. *New England Journal of Medicine,* 340: 1525.
- Smith, K. R. 1997. Development, health and the environmental risk transition. In: G. S. Shahi (ed.). *International Perspectives on Environment, Development and Health.* New York: Springer Publishing Company: 51-62.
- Snaken, R. 1999. Control of influenza: Public health policies. *Vaccine,* 17 (supplement 3): 61-63.

- Snaken, R., Kendal, A. P., Wood, J. M. 1999. The next influenza pandemic: Lessons from Hong *Kong. Emerging Infectious Diseases,* 5: 195-203.
- Sober, E. R. 1980. Evolution, population thinking and essentialism. *Philosophy of Science,* 47: 350-383.
- ___________1984. *The Nature of Selection*. Cambridge, Massachusetts: MIT Press.
- ___________1992. The evolution of altruism: Correlation, cost, and benefit. *Biology and Philosophy,* 7: 177-188.
- ___________1995. Philosophical problems for environmentalism. In: R. Elliot (ed.). *Environmental Ethics*. Oxford: Oxford University Press: 226-247.
- Sober, E., Wilson, D. S. 1998. *Unto Others: The Evolution and Psychology of Unselfish Behaviour.* Cambridge, Massachusetts: Harvard University Press.
- Solomon, R. C. , Higgins, K. M. 1996. *A Short History of Philosophy.* New York and Oxford: Oxford University Press.
- Sosa, D. 1993. Consequences of consequentialism. *Mind*, 102: 405-431.
- Sosa, N. M. 1996. The ethics of dialogue and the environment: Solidarity as a foundation for environmental ethics. In: J. B. Callicott, F. J. R. da Rocha (eds.). *Earth Summit Ethics.* Albany, New York: State University of Albany Press: 47-70.
- Sournia, J.-C. 1992. T*he Illustrated History of Medicine.* London: Harold Starke Publishers.
- *South African Parliamentary Bulletin* 1996. (cited June 2000). Available at: http://www.anc.gov.parlim/doc.9684-3.html.

- Speck, R. 1993. Major human diseases past and present: Cholera. In: K. Kiple (ed.).*The Cambridge History of World Disease.* Cambridge: Cambridge University Press: 642-649.
- Star, C. G. 1973. *Early Man: Prehistory and the Civilizations of the Near East*. New York: Oxford University Press.
- Stephens, J. 2000. The body hunters: Testing amid an epidemic. *Washington Post* December 17, 2000 A1. (cited April 2000). Available at: wwwwashingtonpost.com/ac2/wp_dyn?articlecontenta=A11047.2000dec1 5.
- Stephens, R. S. (ed.). 1998. *Chlamydial Infections.* Proceedings of the ninth international symposium on human chlamydial infection. International Chlamydia Symposium, San Francisco, California.
- ________________. 1999. *Chlamydia. Intracellular biology, pathogenesis, and immunity*. Washington D. C.: American Society of Microbiology Press.
- Stevens, D. L., Herr, D., Lampiris, H. *et al.* 2002. Linezolid versus Vancomycin for the treatment of methicillin-resistant *Staphylococcus aureus*. *Clinical Infectious Diseases,* 34: 1481-1490.
- Stutz, B.1993. The landscape of hunger. *Audubon,* 95(2):676-683.
- Szanislo, M. 2002. Plane quarantined due to passenger's illness. *Boston Herald.* October 25, 2000: 2.
- Szasz, A. 1994. *Ecopopulism: Toxic Waste and the Movement for Environmental Justice.* Minnesota: University of Minnesota Press.
- Takahata, N., Satta, Y., Klien, J. 1995 Divergence time and population size in the linage leading to modern humans. *Theoretical Population Biology,* 48: 198-221.

- Taylor, P. W. 1986. *Respect for Nature*. Princeton, New Jersey: Princeton University Press.
- ________1995. The ethics of respect for nature. In: C. Pierce, D. VanDe Veer (eds). *People, Penguins, and Plastic Trees 2nd Edition*. Belmont, CA: Wadsworth Publishing Company: 125-139.
- Taylor, T. E., Strickland, G. T. 2000. Malaria. In: G. T. Strickland (ed.). *Hunter's Tropical Medicine and Emerging Infectious Diseases, 8th Edition*. Philadelphia: W. B. Saunders Company.
- Temkin, O. 1977. *The Double Face of Janus and Other Essays in the History of Medicine.* Baltimore: Witman Publishers.
- Tenover, F. C., Hughes, J. M. 1996. The challenges of emerging infectious diseases. Report of the Council on Scientific Affairs, American Medical Association. *Journal of the American Medical Association*, 281: 2330-2339.
- Tenover, F. C., Swenson, J. M., O'Hara, C. M. *et al.* 1995. Ability of commercial and reference antimicrobial susceptibility testing methods. *Journal of Clinical Microbiology,* 33: 1524-1527.
- Tertullian, Quintus (160-230 CE) cited in: Hardin: G. 1993. *Living within Limits: Ecology, Economics, and Population Taboos.* New York: Oxford University Press.
- Theriault, G. 1994. Cancer risks associated with exposure to magnetic fields. *American Journal of Epidemiology,* 139: 550-555.
- Therborn, G. 1980. *The Power of Ideology and the Ideology of Power.* London: Verso.
- Thompson, D. (ed.). 1995. *The Concise Oxford Dictionary, 5th Edition.* Oxford: Clarendon Press.

- Thompson, J. B. 1990. *Ideology and Modern Culture.* New York: Polity Press.
- Tobias, M. 1998. *World War III: Population and the Biosphere at the end of the Millennium.* New York: Continuum Press.
- Todd, E. C. D. 1996. Worldwide surveillance of food-borne disease. *Journal of Food Protection,* 1: 82-92.
- Todd, L. C. 1987. Analysis of kill-butchery bonebeds. and interpretation of Paleoindian hunting. In: M. H. Nitecki, D. V. Nitecki, (eds.). *The Evolution of Human Hunting.* New York: Plenum Press: 225-267.
- Toulmin, S. 1970. *An Examination of the Place of Reason in Ethics.* Cambridge: Cambridge University Press.
- ___________1982. How medicine saved the life of ethics. *Perspectives in Biology and Medicine,* 25: 740.
- Townsend, P., Davidson, N. 1982. *Inequalities in Health.* Harmondsworth UK: Penguin.
- Treiber, C. A., Taylor, D. E. 2002. Mutations in the 16S rRNA genes of *Helicobacter pylori* mediate resistance to tetracycline. *Journal of Bacteriology,* 184(8): 2131-2140.
- Turner, B. 1995. *Medical Power and Social Knowledge.* London: Sage.
- Tyler, P. 1994. Nature and economic boom devouring China's farmland. (*The New York Times*. March 27). Quoted in: A. Nadakavukaren. 2000. *Our Global Environment.* Prospect Heights, Illinois: Waveland Press: 178.
- Union for Concerned Scientists. 1999. *Report on Antimicrobial Uses and Abuse.* New York: The Union: 1-32.
- (UKST) United Kingdom Science and Technology Report of the U. K. House of Lords. Session 1997-1998. *Antibiotic Resistance* (cited May

1999). Available at: www.parliment.the-stationary–office.co.uk/pa/ld199798/Idselect/ldsctech/st0701.html.

- (UNDP) United Nations Development Programme. 1998. *United Nations: Analytical Tools for Human Development.* (cited April 2001). Available at: http://www. undp.org/hdro/anatools.htm.
- ____1999. *United Nations Development Report. Human Development Report 1997-1999.* (cited June 2001). Available at: http://www.undp.org.lb/programme/world ecret/2439.36.
- ____2000. *Position paper: Over prescribing Antibiotics.* (cited August 2002). Available at http://www.undp.org.lb/programme/governance/advocacy/nhdr/nhdr97/chp t3g.pdf.
- ____2001. *State of the World's Population. United Nations Report: Human Population.* (cited June 2002). Available at:http:wwwundp.org.lb./pop.chapt4pdf.
- ____2002. *Transition of China's Population: Regional Differentials and Implications.* (cited April 2002). Available at http www unescap. Org/ pop/journal/1990/v05/v5n4a2.pdf.
- (UNEP) *United Nations Environment Programme 1989. Conference of Plenipotentiaries on the Global Convention of the Control of Transboundary Movements of Hazardous Wastes: final act and text of Basel Convention.* Geneva: UNEP Publication.
- ____2001. *Conference of Plenipotentiaries on the Global Convention of the Control of*

Transboundary Movements of Hazardous Wastes: final act and text of Basel Convention, Revised. Geneva: UNEP Publication.

- (UNESCO) 2001. General Conference of Science on Bioethics international implications. 22 to 23 October 2001, Paris. (cited December 2001). Available at: UNESCOgeneralscience_ Bioethicsinternational implications.
- (UNFPA) United Nations Population Fund 1999. The State of the World Population 1999, 2000, 2001. (cited August 2002). Available at http://www.unfpa.org/publications/swp.
- Urey, H. C. 1952: On the early chemical history of the earth and the origin of life. *Proceedings from the National Academy of Sciences of the USA,* 38: 351-363.
- (USBC) United State Bureau of Census. Census 1999. (cited December 2000). statistics available from http://www.worldpop.org/prdata.htm.
- (USDD) United States Department of Defense. 2002. *Emerging Infectious Diseases Publication 5732002.* Washington, DC: The Department of Defense.
- van Andel, T. H., Shackelton, J. C. 1997. Later Paleolothic and Mesolithic coastlines in Greece and the Agean. *Journal of Field Archaeology,* 9: 445-454.
- van Andel, T. H., Zagger, E., Demitrack, A. 1990. Land use and soil erosion in prehistoric and historical Greece. *Journal of Field Archaeology,* 17, 379-396.
- van Bogaert, D. K., Ogubanjo, G. 2001. An ethical issue in family medicine: Limiting the practice of dispensing doctors. Oral presentation. *16th World Congress of Family Doctors, Durban, 13-17 May 2001. Abstract Book: AB75: 57.*

- van Niekerk, A. A. 2003. *Introduction to Medical Ethics.* Stellenbosch: University of Stellenbosch.
- van Staa, A., Hardon, A. 1998. *Injection practices in the developing world: Results and recommendations from field studies in Uganda and Indonesia.* Amsterdam: University of Amsterdam.
- Vernon, J. 1999. *Unity, Plurality, Politics*. New York: Palgrave Mc Millan Publishers.
- Vilcheck, M. M. 1991. The controls on the transfrontier movement of hazardous waste from developed to developing nations: the goal of a 'level playing field'. *Northwestern Journal of International Law and Business,* 11: 643- 674.
- Viscusi, W., J. Vernon, J. Harrington (eds.). *Economics of Regulation and Anti-Trust*. Cambridge Massachusetts, MIT Press.
- Vogel, L. (ed.). 1996. *Hans Jonas: Mortality and Morality A Search for the Good after Auschwitz.* Evanston, Illinois: Northwestern University Press..
- Vyner, H. 1988. *Invisible Trauma.* Lexington, Massachusetts: Lexington Books.
- Wackernagel, M., Onisto, L., Linares, A. C., *et al.* 1997. *Ecological footprints of nations: how much nature do they use? How much nature do they have?* Centre for Sustainable Development Veracruz, Mexico. Available at http://ecouncil.ac.cr/rio/focus/report/English/footprint/
- Waldron, J. 1989. Rights in Conflict. *Ethics,* 99: 503-519.
- Wallia, C. S. (ed.) 1970. Population and environment. In: C. S. Wallia (ed.). *Toward Century 2l, Technology, Society and Human Values.* New York: Basic Books: 53-63.
- Wassermann, U. 1981. Attempts at control over toxic waste. *Journal of World Trade Law,* 15: 410-430.

- Watts, S. 1997. *Epidemics and History.* New Haven Connecticut and London: Yale University Press.
- Wang G. 2001 *The White Paper: General Debate on National Experience in Population Matters: Population, Environment and Development. 34th Session of the United Nations Commission on Population and Development.* New York City, New York: UNDP Publication.
- Wang, S. A., Levine, R. B., Carson, L. A., *et al.* 1999. An outbreak of gram-negative *bacteremia* in hemodialysis patients traced to hemodialysis machine waste drain ports. *Journal of Infection Control and Hospital Epidemiology,* 20: 746-751.
- Wang, S. P., Grayston, J. T., Alexander, E. R. 1967. Trachoma vaccine studies in monkeys. *American Journal of Ophthalmology,* 63: 1615-1620.
- Ward, M. E., Bailey, R., Lesley, A *et al.* 1990. Persisting unapparent chlamydial infection in a trachoma endemic community in the Gambia. *Scandinavian Journal of Infectious Diseases,* (Supplement) 69: 137-148.
- Ward, M. E. 1999. Mechanisms of *Chlamydia*-induced disease.In: R. S. Stephens, (ed.). *Chlamydia. Intracellular biology, pathogenesis and immunity.* Washington, D. C.: American Society of Microbiology Press: 171-210.
- (WBDR) *World Bank World Development Report 1993 Investing in Health.* Washington, D. C: The World Bank Publication.
- Weinstein, R. A. 2000. Global Resistance Day: Unprecedented and Promising. *Proceedings: 40th Conference on Antimicrobial and Chemotherapy Day. Day one, September 17, 2000.* Toronto, Canada. (cited June 2001). Available at: http://www. meds.cape.com/meds.cape/cno/2000/IAC. :67.

- Wernsdorfer, W. H. 1991. The development and spread of drug-resistant malaria. *Parasitology Today,* 11: 297-303.
- (WHO) World Health Organization. 1990. *Public Health Impact of Pesticides in Agriculture.* Geneva: World Health Organization Publication.
- ________________________________. 1992. *Our planet, our health: Report of the WHO commission on health and environment.* Geneva: World Health Organization.
- ________________________________.1993. World malaria situation. *Weekly Epidemiological Record,* 68: 245-252.·
- ________________________________.1995. *Bridging the Gaps.* (cited June 2000). Available at http://www who.info.html.
- ________________________________.1996.a *Fighting disease, fostering development.* World Health Report Geneva: World Health Organization Publication.
- ________________________________.1996 b. AIDS: Global data. *Weekly Epidemiological Record,* 74: 205-2O7.
- ________________________________1996 c. Human plague. *Weekly Epidemiological Record,* 71: 165-168.
- ________________________________.1997. *The medical impact of the use of antimicrobials in food animals.* Geneva: Report of a WHO meeting, 1997 Oct 13-17. Geneva: World Health Organization Publication.
- ________________________________.1998. *MDR-TB report* . (cited June 1999). Available at: http://www who.int/gtb.
- ________________________________.2000 a. *Cholera fact sheet.* (cited June 2001). Available at http://www who.info.html.

- ______________________________.2000 b. *Preventative measures against infectious disease.* (cited April 15, 2003} Available at: http www who/int/ith/English/project.
- ______________________________.2000 c. Drug resistance Threatens to Reverse Medical Progress. Message from the Director-General. *Press Release,* WHO/41:12 June 2000. (cited May 2001). Available at: http://www.who.int/inf-pr-2000/en/pr2000-41.html.
- ______________________________.2003. World Health Organization. 2003. SARS Update. Available at Http www who don sars update 603_5.search ?html.
- Wilson, M. E. 1995. Travel and the emergence of infectious diseases. *Emerging Infectious Diseases,* 1: 39-46.
- Wilson, E. O. 1994. *The Diversity of Life.* New York: W.W. Norton.
- ___________1995. The little things that run the world. In: C. Pierce, D. VanDe Veer (eds.). *People, Penguins and Plastic Trees, 2nd Edition*. New York: Wadsworth International: 139-142.
- Windelband, K. 1980. *History and Natural Science.* J. Oakes, (transl.). New York: Harper and Brothers: xix.
- Wing, S. 1993. A review of recent findings on radiation and mortality at Oak Ridge National Laboratory. *Proceedings of the International Society of Radiological Protection.* Munich: Verlag: 217-228.
- Winslow, C. A. 1920. The untilled fields of public health. *Science,* 51: 21-28.
- Wise, R. 1999. The development of new antimicrobial agents. *British Medical Journal South African Edition,* 7: 157-159.
- Wittaker, R. H. 1975. *Communities and Ecosystems.* New York: Macmillan Publishers.

- Witte, W. 2000. Ecological impact of antibiotic use in animals on different complex microflora: environment. *International Journal of Antimicrobial Agents,* 14: 321-325.
- Woolhouse, M. E. J., Taylor, L. H., Hayden, D. T. 2001. Population biology of multihost pathogens. *Science,* 292: 1109-1112.
- (WRI). World Resources Institute 1996-1997. *The Urban Environment.* Oxford: Oxford University Press.
- ________________________________1999. *Environmental Change and Human Health*. Oxford: Oxford University Press.
- Wu, C. 1999. Beyond vancomycin. *Science News,* 155: 268.
- Young, J. E. 1993. *For the Love of Gold.* Washington and New York: World Watch Institute Publication.
- Zambon, M., Nicolson, K. G. 2003. Severe acute respiratory syndrome (editorial), *British Medical Journal,* 326: 669-670.
- Zimmer, C. 2001. *Evolution-the Triumph of an Idea.* New York: Harper Collins.
- Zohary, D. 1969. The progenitors of wheat and barley in relation to domesticated and agricultural dispersal in the old world. In: P. J. Ucko, G. W. Dimbley (eds.). *The Domestication and Exploration of Plants and Animals.* Chicago: Aldine Publishing Company: 44-67.
- Zubbay, G. 2000. *Origins of Life on Earth and in the Cosmos.* San Diego, California: Academic Press.
- Zucker, J. 1996. Changing patterns of autochthonous malaria transmission in the United States: A review of recent outbreaks. *Emerging Infectious Diseases*, 2: 37-43.