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International journal of Advanced Biological and Biomedical Research

Volume 1, Issue 8, 2013: 899-903

The effects of psychological stress and other environmental factors on incidence of diseases

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ABSTRACT

The main purpose of this paper has been to discuss the effects of psychological stress and other environmental factors on incidence of diseases. We were specifically concerned with stress that increased susceptibility of uninfected hosts when exposed to infection. If such stresses also reduced resources, fecundity and/or survivorship, there was a reduction in the host carrying capacity. In addition, stress that increased parasite mortality decreased disease. The opposing effects of stress on disease dynamics made it difficult to predict the response of disease to environmental stress. Stress can simultaneously increase parasite mortality and impair host vital rates, making it more likely that parasites will be extirpated by stress. Stress may have multiple effects on hosts and parasites such as increasing susceptibility to disease while decreasing host vital rates, such as reproduction. The knowledge about stresses help clarify predictions about the interaction between environmental stress and disease in natural populations.

Key words: Disease, Infectious, Psychological Stress, Infertility

INTRODUCTION

In our rapidly changing world, a variety of stresses may affect the functioning of organisms, ranging from toxins to the vicissitudes of climate change. The ultimate impact of such stress, however, depends upon the dynamic behavior of the stressed system. One arena in which this issue is particularly important is in evaluating the joint impacts of environmental stress and infectious diseases. There is growing evidence that infectious diseases play a major role in the dynamics of many natural populations and communities (Holmes, 1996). One can predict that increasing stress either aggravates or moderates impacts of infectious disease, depending upon what aspect of the system one emphasizes. Hence, quite generally, stressed individuals should be more susceptible to infection (Scott, 1988; Holmes, 1996), although the exact impact of any stressor is likely to vary by genotype within a species (Blanford et al. 2003). This line

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of thought suggests that infectious disease should be aggravated by environmental stress. An opposing prediction emerges from considering the population dynamic context of infection. A key component of infectious disease models is that the likelihood and impact of an epidemic increases with host density (Anderson & May, 1986). Outside stressors that depress population density, therefore, should reduce the chance of an epidemic, or even the ability of a parasite to persist at all, because factors that reduce host density also reduce contact rates between infected and uninfected individuals. This dependence of infection upon density is one of the key intersections of epidemiology and conservation biology. Threats to biodiversity, which are generally mediated through reductions in abundance, should normally indirectly reduce risk to host-specific infectious diseases (Lafferty & Gerber, 2002). Direct reduction of host density should reduce disease. Culling of seal populations reduces intestinal nematode parasites by reducing hostdensity below transmission thresholds (Des Clers &Wootten, 1990). Fishing can similarly reduce parasites in fish populations (Arneberg et al., 1998). A swim bladder nematode was apparently extirpated from native trout in the Great Lakes after a variety of stressors reduced trout populations to very low levels (Black, 1983). Alternatively, some stressors may increase parasitism by increasing host density. The stress most commonly observed to be associated with increased parasitism in fishes and invertebrates is eutrophication (Lafferty, 1997). The addition of nutrients to aquatic systems increases primary productivity that indirectly increases some grazers and predators. Such increases in host density could facilitate disease invasion. It is also possible that nutrients or associated pollutants could increase individual susceptibility. Stress may have multiple effects on hosts and parasites such as increasing susceptibility to disease while decreasing host vital rates, such as reproduction. As top predators, marine mammals bioaccumulate lipophillic toxins that can affect the immune system (Swart et al. 1994); for example, harbour seals fed fish from polluted areas have lower killer cell activity, decreased responses to T and B cell mitogens and depressed antibody responses (DeStewart et al. 1996). Such immunosuppression may be a cofactor in mass mortalities associated with morbillivirus (Van Loveren et al. 2000) and there is some correlative evidence for the hypothesis that marine contaminants may increase sea otter susceptibility and exposure to infectious diseases (Lafferty & Gerber 2002; Miller et al. 2002). Along with effects on susceptibility to disease, sea lions suffer lower reproductive success when subject to high contaminant concentrations and this may have limited sea lion abundance in some areas (O'Shea & Brownell, 1998). In these cases, it is difficult to predict the overall effect of stress on disease. Stress can simultaneously increase parasite mortality and impair host vital rates, making it more likely that parasites will be extirpated by stress. Blooms of diatoms containing domoic acid (a neurotoxin that passes up the food chain) have been implicated in mass mortalities of marine mammals (Lefebvre et al. 1999). But these toxins may affect some parasites as well. In small doses, domoic acid (from macroscopic red algae) has been traditionally used in Japan to cure roundworm (nematode) infections in humans (Daigo, 1959). Dolphins that died of domoic acid poisoning in California in 2002 were remarkably free of intestinal parasites. Because both parasites and domoic acid transmit through the food chain (even in the same food items), there may be important interactions between harmful algal blooms and helminth infections in marine mammals.

The effects of stress on reproduction performance

It is well recognized that stress and infertility are linked. However, the causal or consequential nature of this link remains unclear. In a review, Edelmann and Connolly (1986) were unable to confirm that there were psychological causes of infertility. They concluded that infertility clearly has psychological consequences for some couples, although the underlying mechanisms are poorly understood. Golombok (1992) felt that the impact of fertility on psychological function was complex and subject to a variety of factors. The interaction between man and environment is continuous and certainly has influenced the

process of evolution of the species. This interaction is in certain cases beneficial but in many it is hostile. Indeed, mankind has introduced elements into the environment that either pollute or modify environmental conditions with resulting negative effects on human health. Conversely, adverse environmental conditions not controlled or influenced by man can also affect human health and behavior. This is a continuously evolving process, with some elements that remain fairly constant over a relatively long period of time (decades or centuries) and others that can rapidly progress or change in a much shorter time frame (environmental disasters). Before attempting to evaluate environmental influences on adult reproductive functions in the human male and female, it is necessary to provide some definitions to focus the terms and the scope of the problem. The definition of reproductive health given by the World Health Organization also has a broad scope, suggesting that multifactorial environmental inputs may introduce disorders of an organic, functional, or psychological nature. How is reproductive dysfunction evaluated, then? This is one of the complex aspects of the problem, and it has certainly contributed to the present state of confusion in the field. If one is going to evaluate the fertility potential of a couple, outcome (pregnancy) is obviously the gold standard. Other parameters, such as semen quality, ovulation, etc., although important markers for potential dysfunction, are not reliable measures of outcome. Evaluation of conception rates is obviously a desired parameter to ascertain the impact of a given environmental factor. Yet, if one is to analyze the impact of that same factor on an individual man or woman, less precise or reliable parameters (other than pregnancy) have to be used to quantitate and characterize the effect. Even pregnancy itself is a somewhat misleading parameter when applied to an individual couple, since subfertility induced by an environmental agent may still exist although pregnancy is achieved at a given time. It appears from the above that well-controlled, large population studies can provide reliable data on the influence of specific environmental factors on infertility. However, these studies are not without problems because levels of exposure, length of exposure, and preexisting or coexisting conditions may clearly affect the degree of damage to the reproductive capacity of the couple. In the case of environmental disasters, many of the above conditions could be more closely controlled, and, thus, lessons can be learned from those occurrences. In most other instances, however, analysis of the impact of an environmental agent or toxin needs to be conducted using different indexes of reproductive performance in addition to pregnancy. The immune system reacts and responds to antigenic exposures and, in turn, can modulate or adversely affect reproductive events. The respiratory and gastrointestinal systems as well as the skin act as routes of entry/exit for many environmental factors and biologically active agents. The ultimate target of environmental factors are the receptors, enzymes, and second messenger systems as well as the genes that are involved in the regulation of cellular development, differentiation, and function in reproductive tissues. These targets also include enzyme systems responsible for the metabolism, inactivation, and detoxification of any biologically active substance or toxin. These concepts clearly indicate that reproductive health can be impaired not only by agents or toxins that directly affect reproductive tissues but also by substances or factors that affect a number of other tissues or systems which indirectly regulate or support reproductive functions. In the area of stress and infertility, our knowledge of the basic mechanisms mediating stress responses has progressed substantially, and this will allow interested researchers to formulate well-designed paradigms to evaluate the impact of stressful agents or situations on reproductive performance. The combination of psychological tests to measure stress levels with biochemical parameters to quantitate stress hormone responses should provide a good framework in which to address specific questions on fertility parameters. A more challenging problem still remains in trying to isolate confounding variables because stress situations as discussed above are often associated with other types of disorders. A fruitful area of research is emerging in the interplay between the neuroendocrine and the immune systems, particularly to ascertain how chronic stress modifies immune system function and what contribution this has to infertility. In general, environmental factors are often invoked as contributing to many cases of otherwise

unexplained infertility. However, the direct causal relationship between those factors and the ensuing infertility of couples is seldom well established and remains largely anecdotal. Several problems contribute to maintain this relatively confusing state of affairs: a) the multifactorial nature of the contributing factors; b) the poor design of many of the studies; c) the diversity of parameters evaluated and whether they measure outcome (i.e., pregnancy rates) or intermediate events (semen values, ovulation, etc.) and d) the difficulty in monitoring exposure both in terms of time and degree of intensity. Until unified criteria are applied consistently and systematically to evaluate environmental influences on human reproductive health, many of the cases of female or male infertility will remain unexplained.

Acknowledgments

The authors would like to have a special thank to Dr. Rozita Jamili Oskouei for criticism reading and editing of the manuscript.

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