On Climate Change and Infectious Disease: Implications for Political Destabilization and Conflict

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Mr. Chairman and Members of the Science and Technology Committee, thank you for inviting me here today to share with you my views regarding the impact of Global Climate Change on Infectious Disease, its implications for economic and political instability, and for U.S. national security. I am the Director of the Project on Health, Environment, and Global Affairs, which is an inter-university research initiative between Colorado College and the University of Colorado, Colorado Springs, and Senior Advisor to the Center for Homeland Security at the University of Colorado. I serve as Assistant Professor of Political Science at Colorado College, and have held previous appointments at Columbia University and the University of South Florida. Over the years I have served as consultant or advisor to the US Department of Energy, and Department of Defense, the World Bank, the United Nations Development Program, and the Council on Foreign Relations.

On Etiology and Emergence

In the twenty first century, novel pathogens are currently 'emerging' at the rate of approximately one new agent per annum. Emerging diseases often are the result of 'emergent properties' wherein antecedent variables (e.g. population density, speed of transport) combine in unusual and unforeseen ways that facilitate the emergence of a

given pathogen which then becomes endogenized within the human ecology. The classic modern example of such emergent properties leading to viral proliferation is the SARS coronavirus which appeared in Guangzhou, China in late 2002, and subsequently spread throughout the Pacific Rim nations. In that particular case, this virulent coronavirus spread from its natural reservoir in east Asian bat populations, into palm civets. The variant of the virus that infected civets was transmissible among humans, amplified by elements of the human ecology such as the 'wet markets' of East Asia, the closed environments of modern hospitals which amplified degrees of infection, and modern jet airplane technology that facilitated the rapid spread of the virus throughout the Pacific theatre. Individually these disparate variables would not predict the emergence of epidemic disease, however, when combined together the SARS contagion of 02-03 resulted.

The dynamics of contagion frequently exhibit such emergent properties,[1] and the relations between pathogen, human host, and vectors of transmission (e.g. mosquitoes) are central to both the transmissibility and lethality of any given manifestation of contagion. Furthermore, epidemics and pandemics exhibit non-linearities and threshold dynamics. For example, pathogens may simmer in a given population for some time, but once the rate of transmission passes from <1 to >1, the proliferation of the pathogen may then increase on an exponential scale. Diseases also exhibit high levels of interactivity, and the capacity for co-infection. The classic example is HIV which destroys the host's immune system, and thereby facilitates colonization by other pathogens (e.g. tuberculosis) that ultimately kill the host. What then is the relationship between climate

[1] For an in-depth discussion see Andrew Price-Smith, Contagion and Chaos, MIT Press, forthcoming 2008.

change, infectious disease, prosperity, and political stability and security? The complexity of such interactions is enormous, and so we begin with the relations between climate and disease, focusing on malaria in particular.

(Insert Figure 1 here)

Data provided by the IPCC regarding changes in precipitation from 1900-2000 indicate enormous variance on a global scale. Certain regions, such as the arctic and sub-arctic regions of the northern hemisphere, the northeastern sector of south Asia, and Eastern Australia are clearly enjoying increased levels of precipitation. Certain vectors of disease, (such as mosquitoes and snails) thrive in wet environments. Consequently, increases in precipitation will induce the proliferation of vectors, and thereby increase the transmission rates of certain pathogens such as malaria and schistosomiasis.

(Insert Figure 2)

Pathogens and their vectors of transmission are often highly sensitive to changes in temperature as well. IPCC data from 1976-2000 clearly indicate increasing temperatures for much of the surface of the planet, with the greatest increases evident in the temperate to polar regions. As isotherms shift toward the polar regions, this will expand the latitudinal range of the vectors in question (i.e. anopheles mosquitoes) and thereby permit the expansion of malaria in previously non-malarious zones. Similarly, increasing surface temperatures permit the movement of malaria in higher altitudes than before. For

example, Nairobi has historically been non-malarial due to its altitude, but in recent years increases in temperature have seen the pathogen moving into the region. The temperature-induced expansion of malaria is problematic because it exposes novel populations, who often lack any genetic or acquired immunity to the pathogen. Thus, the mortality and morbidity in such regions may be much higher than in zones where malaria is endemic.

Increasing temperatures also affect the biting rate of vectors. As temperatures rise, the vectors (mosquitoes) feed with greater frequency, and therefore increase the transmission rate of the *plasmodium* (the parasite) into human populations. Furthermore, increasing temperatures also affect the extrinsic incubation rate of the pathogen, such that it replicates within the gut of the vector at a greatly augmented rate. Thus, under conditions of higher temperatures, there are greater numbers of plasmodium within the vector, and the vector bites with much greater frequency.[2] On a macro level, all of this means that as temperatures increase, the burden of disease (e.g. malaria) is likely to increase to a significant degree. Precipitation and Sea Surface Temperatures (SST's) are strong predictors of malarial incidence.[3]

In the case of cholera, increasing SST's are highly correlated with the growth of algal blooms. The blooms move across oceans courtesy of dominant currents and winds, and function as vectors of transmission of the *vibrio*. Thus, we see a long-term empirical association between SST and the incidence of cholera. In the case of cholera we have also seen that incidence is responsive to the modulation of the El Nino Southern Oscillation

[2] See Reiter 2001, Kovats et al 2001, Hunter 2003, van Lieshout 2004, Patz et al 2005, McMichael 2006.
[3] M.C. Thompson et al., 2005

(ENSO), with preliminary evidence from case studies carried out in Bangladesh (Rodo, 2002). There is also considerable evidence of thresholds and non-linearities, such that warming temperatures may produce minor and linear increases in vibrio incidence until a threshold point is reached, after which the numbers of the pathogen increase at an exponential scale.[4]

Schistosomiasis is a frequently lethal disease induced by parasitic blood flukes, and it is prevalent in tropical and temperate zones. The vector of the parasite is the snail (*oncomelania*) which thrives under conditions of increased precipitation, and within the temperature range of 15.3 degrees C to an optimal temperature of 30 degrees C. The balance of available evidence suggests that global climate change (GCC) will shift the distribution of the vectors into new regions, and thereby afflict previously uninfected populations. A caveat however, the IPCC data clearly indicate that certain regions (e.g. West Africa) are becoming increasingly arid, which is inimical to the vector. Consequently, those zones that witness declining precipitation levels will see a decline in the incidence of schistosomiasis in their respective populations. In those regions that exhibit both increasing precipitation, coupled with increasing temperature, we are likely to witness augmented geographic zones of transmission, and increased frequency of transmission within those regions. Thus, GCC will result in winners and losers, dependent upon the particular pathogen in question, and its sensitivity to aridity and temperature.[5]

[4] See Xavier Rodo et al, 2002, J. Patz, 2002
[5] Nagasaki 1960, Zhou et al, 2002, Yang et al 2005, Steinmann et al 2006, Guo-Jing Yang et al 2007.

Economic Outcomes

The economic historian Robert Fogel won the Nobel Prize in economics in 1994 for his analysis of the hypothesis that population health was the central driver of economic productivity (NBER 1994). If health promotes prosperity, then disease erodes productivity and wealth. At the microeconomic level disease erodes productivity through mechanisms such as the debilitation of workers, increased absenteeism, increased medical costs, reduced savings and investment, and the premature death of breadwinners. At the sectoral level, disease imposes a particular burden upon those sectors of the economy that are labor-intensive, such as agriculture, and resource-extraction, and thereby imposes a relatively greater effect upon the economies of the developing world.

The impact of malaria is illustrative at the macroeconomic level. Sachs and Malaney estimate that for those countries where malaria is endemic, the pathogen generates a 1.3 percent drag on their GDP growth rate, per capita/per annum. Further, Gallup and Sachs estimated that a 10 percent decline in malaria incidence resulted in a 0.3 percent increase in the growth rate of GDP per capita/per annum. McCarthy estimated that malaria imposed a drag on the GDP growth rate of affected nations, at the level of 0.25 to 0.55 percent per annum.[6] In case studies of individual nations, malaria control has resulted in greater prosperity for the polity in question. For example, malaria control measures in Zambia resulted in a $7.1 billion increase to that nation's economy.[7]

[6] D. McCarthy et al., NBER paper 7541, 2000
[7] Utzinger et al., 2002

The burden of infectious disease falls primarily upon the poor and middle classes, and therefore as the burden of disease increases in certain regions it will likely exacerbate both the perceived and real level of economic inequities between socio-economic strata. Historically, such perceptions of inequity have led to periods of social and political destabilization.[8] On a global scale, GCC-induced increases in the burden of disease will exert a drag on the global economy, and the perpetuation of poverty within the LDCs.

Assessments of the economic burden of a given illness (e.g. malaria) are complicated by the lack of adequate surveillance infrastructure throughout much of the developing world where the disease is endemic.[9] Moerover, the complexity of measuring the economic impact of GCC-induced infectious diseases is augmented by the interactivity of various pathogens in a given population. For example, the population of country X may be increasingly beset by increased incidence of malaria, dengue fever, and schistosomiasis, and certain individuals may exhibit co-infection with one or more pathogens.

Pathogens may also erode the functionality and efficacy of the state as well. For example, disease-induced economic stagnation (or contraction) of the macro economy will consequently reduce tax-based revenues available to the state. Diminished revenues will in turn impede the state's capacity to provide public goods and services (e.g. education, law enforcement) to its population. This may in turn reduce the populace's perceptions of the legitimacy of the state. In the domain of human capital, disease may further erode

[8] Price-Smith, Contagion and Chaos, MIT Press, 2008, forthcoming
[9] Worral et al., 2004, 2005

state capacity by debilitating and/or killing trained and skilled personnel, thereby reducing institutional resilience and efficacy.[10]

On Poverty, Instability and Conflict

The association between poverty, political destabilization, and outright conflict is complex. In particular, there is an endogeneity issue regarding the direction of causality. However, we can make some preliminary observations at this point. First, various iterations of the State Failure Task Force conducted empirical investigations and determined that infant mortality (as a measure) is a strong empirical predictor of state failure.[11] Ted Gurr argued that increasing levels of poverty induced a psychological state of deprivation (perceived injustice) that often led to intra-state conflict.[12] This hypothesis that conditions of deprivation (both real and perceived) led to civil strife was supported by Deininger (2003), and low levels of the Human Development Index are associated with conflict in Indonesia (Malapit et al 2003). Other political scientists have found that poverty combines with ethnic fragmentation to produce intra-state conflict (Easterly and Levine 1997, Wilkinson 2004, Korf 2005). Charles Tilly has argued that inequities are directly associated with intrastate conflict (Tilly 1998).[13] Further, there is empirical evidence that social polarization leads to conflict (Esteban and Ray 1994, 199, Boix 2004), and that conflict may function as a 'coping strategy' for those populations confronted with extreme levels of economic deprivation (Humphreys and Wienstein

[10] An expanded analysis of the pernicious effects of disease on the state can be found in Andrew Price-Smith, The Health of Nations, MIT Press, 2002.
[11] D. Esty et al, State Failure Task Force I and II
[12] Gurr 1970.
[13] Also see Stewart 2000, Langer 2004, Mancini 2005

2004, Verwimp, 2005). Convincing arguments take the form of the state weakness hypothesis wherein deprivation combines with a weakened state to offer both the motive and the opportunity for political violence, with evidence from numerous case studies (see Kahl 2006, and Homer Dixon 1999). Political scientists (Singer 2002) have also hypothesized that increased levels of infectious disease may lead to conflict between sovereign states. Although there is evidence that contagion leads to political acrimony and trade disputes between nations, there is no evidence that infectious disease results in war between nations (Price-Smith, 2008). Despite the proliferation of literature to support the hypothesis that economic deprivation generates political violence at the intra-state level, additional cross-national empirical analysis, using time-series data, is required. That said, the balance of existing evidence supports the hypothesis.

Conclusions

Pathogens function as *stressors* that impose burdens on both populations (i.e. society), and upon the structures of the state itself. Historical analysis of the stresses generated by epidemic disease demonstrate that pathogens have exacerbated pre-existing conflicts between socio-economic classes, between ethnicities, between those of different religious affiliations, and frequently induced conflicts between state and society.[14] Thus, the GCC-induced proliferation of disease may facilitate socio-political destabilization, particularly in the weak states and impoverished populations of the developing world. However, such destabilization is contingent upon several factors, it is pathogen-specific, and it depends

[14] See Friedrich Prinzing 1916, David Baldwin 2004, Richard Evans 2005, Alfred Crosby 1986, William McNeill 1976, Charles Rosenberg 1987, Sheldon Watts 1999, Terence Ranger and Paul Slack 1996, and J.N. Hays 1998.

upon existing socio-economic and political cleavages within the polity in question. Areas at risk of such disease-induced destabilization include the sub-tropical to temperate zones, as tropical pathogens and their attendant vectors expand into these contiguous zones to affect immunologically naïve populations. Thus, we should be concerned about nations in South Asia, Central and East Asia, Southern Africa, and South America. Typically the effects of disease-induced destabilization upon the security of the United States will be *indirect*, however, in the post 9-11 era we now recognize that weak and failed states in the developing world may generate externalities (such as terrorism and political radicalization) that threaten the material interests of the dominant powers of the international system, including the United States.

In conclusion, further research is required to flesh out the complex chain of possible causation that I have detailed above. This will require the formation of interdisciplinary teams of both social and natural scientists who will then model the impacts of climate change upon disease, and the consequent effects upon the economic and political domains. This might involve the compilation of a time-series dataset across a representative sample of countries. One obvious problem involves modeling the long-term processes of climate change, however we might use the ENSO effect to model how short-term changes in climate induce variance in disease incidence, and then observe the resulting economic and political impacts over the very short-term.

Thank you, Mr. Chairman, for providing me this opportunity to appear before you. I'm happy to respond to members' questions.

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