

Who Amongst Us Will Be Selected?

IT IS EARLY WINTER OF 2011 AND INFLUENZA IS ENDEMIC IN Providence. Thirty-four youngsters attend a local first grade elementary school class; and five are made ill by this respiratory pathogen. Thus, 29 students continue to attend classes unaffected by influenza while five vulnerable ones are temporarily bedridden. The teacher might then wonder: “Do those five children represent a random sample of her class? Contrariwise, could *any* child in this small population of 34 have been attacked by influenza; or, alternatively, might some of her children, by biological nature or environmental circumstance, be more vulnerable than others to the ravages of this communicable disease?” And she might then reflect: “What, indeed, is the nature of vulnerability? Divinely determined or a reflection of very secular factors?”

Until such time in the distant future when effective vaccines to prevent all major communicable diseases will be available, an understanding of the epidemiological dynamics of communicable disease remains a vital part of public health policy. And so, the very existence of selective vulnerability—and its ramifications—remains a suitable subject of inquiry.

Let us assume a hypothetical population of 100 children all attending the same school class in some equally hypothetical city. Assume further that an airborne human virus has been introduced into the atmosphere of this classroom probably brought there by another child already incubating the disease. The vulnerability of these 100 children to a specific communicable disease may then be analyzed as a many-layered puzzle.

This airborne virus then takes root in some—but not all—of the children. Why? Immunologists will tell us that certain of these children, let’s guess at 13, were already immune to this specific virus strain either by having undergone a prior illness with it (thus rendering them immune) or by virtue of having been previously vaccinated against this specific strain of virus.

So now let us consider only those 83 remaining children with no prior “knowledge” of this virus. Of these, 77 will then develop clinical signs signifying that they have been duly infected. Again, a question. Somehow, six of those 83 children were allegedly exposed to the virus but were indifferent to it, did not come down with the disease. Exposure requires a physical intimacy with the virus in question; and in the case of an airborne virus, physical proximity to the carrier expelling the virus into the ambient air.

Is it possible, in this hypothetical cluster of children, that some youngsters are more gregarious than others? That some, by virtue of their personalities, make more physical contact, more breathing in each others’ faces, than do others? And, contrariwise, may not some be more shy, more physically withdrawn? When poliomyelitis had been rampant, some seven decades ago, public health physicians noted that when children were, by circumstance, more isolated and participated less in group athletics, they were noticeably less vulnerable to clinical polio. And during the height of the polio epidemics, bedridden children, for whatever reason, did not develop paralytic disease. Two infectious diseases—*influenza* and *polio*—demonstrate a similar pattern of

susceptibility: children less socially active (*influenza*) and children less physically active (*polio*) seem less vulnerable.

Verily, no two children are alike; but still these data show that the dynamics of human behavior may be instrumental in defining vulnerability to communicable disease.

Finally, let us consider those 77 children, of the original hypothetical group of 100, who went on to develop clinically apparent influenza. Would all 77 then demonstrate an equivalent degree of severity? Or, alternatively, might some have a more severe case of influenza than their sick classmates? Again, no two children are the same. In a recent retrospective study of Ohio children burdened by influenza, epidemiologists noted that those children with pre-existing diseases such as crooked spines (*scoliosis*) or asthma that might impair their capacity to breathe deeply were the children most severely affected by a respiratory disease such as influenza.

Life is not fair; nor is it a simple equation between good or evil, lucky or unlucky. In truth, our singular destinies are determined by countless secular variables. In centuries past, vulnerability or invulnerability to some infectious ailment, let us say bubonic plague, was hesitantly ascribed to ill-defined forces such as vindictive spells, one’s professed religion or divine fate. Today, we rely more on countless measurable factors—where and with whom we are, our inherited genomes, how we had conducted our lives and even random happenstance—to predict who amongst us develops a viral infection and who remains indifferent to its hazards.

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The author and his spouse/significant other have no financial interests to disclose.

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