Polio Biology I
An Introduction to the Virus

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It may surprise you to know that we don't understand where viruses originally came from. Yet for every life form on the planet there is some virus somewhere that can penetrate and infect its cells. All viruses infect by following a similar process: they attach to a cell, penetrate it, reproduce, and propagate. The propagation step often involves bursting and killing the infected cell and the release of hundreds of new viruses. Polio does this to the cells it infects.

No virus will naturally infect a cell unless there is a receptor on the surface of the cell that the virus can "recognize". The receptor combines with the virus by attaching to it. This attachment is analogous to a hand slipping into a tight glove. Cells without the proper receptor are not naturally infected. Hence, polio only infects humans and closely related primates, and, only infects cells within those species that have receptors for polio virus. In humans, polio receptors can be found in the upper and lower throat, gut, brain, and on the motor nerve cells (polios-denoting gray matter) of the spinal cord.

Before the advent of the trivalent polio vaccines, polio virus was abundant within our environment. It passed from person to person by ingestion of extremely small bits of fecal material. This could happen through swimming, through food handlers who failed to wash hands properly, or through poor general sanitation. Polio virus is one of the smallest viruses and can be readily transmitted, in large numbers, easily. If the number of virus particles a person ingests is large enough (infectious dose), the virus strain infective enough (virulent), and the resistance of the person low enough (susceptible), the virus attaches to receptors and enters cells in the gut faster than the immune system can clear the virus from the body. Soon, as propagation proceeds, great numbers of virus particles are released into the blood. When virus is in the blood, the condition is called viremia. Eventually viruses find their way to the parts of the brain and spinal cord which have receptors for them. These cells then, unwittingly, take virus in. During propagation within the Central Nervous System, nerve cells (neurons) are killed and cannot divide to replace themselves. Within a couple of weeks the infectious stage ends but virus shedding from fecal material can continue for a few more weeks.

For most of us permanent damage resulted this way. Nevertheless, most of us recovered some function. Recovery involved a number of separate adaptations. For example, we substituted muscles that were less damaged (by nerve loss) for necessary tasks, we sprouted new end fibers from surviving nerves, confering on them a burden for which evolution had not prepared them. The ratio of muscle fiber types changed: the proportion of endurance fibers vs. strength fibers was altered in favor of the endurance type. These

adaptations served us well for a long time. Some are failing us now. This is the simplest and most understandable explanation for Post Polio Syndrome. Perhaps, because we are losing more endurance type muscle, manual strength tests on many of us fail to demonstrate the severity of muscle fatigue we experience. Anyway, these are the most likely explanations for the onset of PPS.

Questions do remain however. Why do some polio survivors and not others experience PPS? Is PPS a disease process which is a natural consequence of having polio? Can you have polio with the damage and repair adaptations and remain stable for life? If so, why? To complicate the legacy of our biological interaction with polio virus is the recent discovery of polio virus genes inside the spinal cords PPS patients. These genes are not present in polio survivors without PPS. This seems to be telling us something. And, the obvious question is "do these fragmments of virus cause PPS?"

It is possible to remove the outer protein shell from a polio virus by treating the virus with phenol. When this was done in the 1940's it was found that the genes inside were infectious without their outer coat. Virus genes could enter cells but were a thousand times less infective than whole virus. We can also assume polio's genes, in the cords of PPS patients, are defective. That is, they have mutated and are not propagating normally by bursting cells. Perhaps, as foreign viral parts, these genes can trigger inflammation, or interfere with nerve cells in another way. New research may resolve these questions. In the meantime polio virus biology remains ineluctably entwined with our own.

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