Koch's Postulates: why they hold importance

Origin of disease?

Miasma

Girolamo Fracastoro, 1546, proposes ideas about infectious agents

Agostino Bassi shows that a 'vegetable parasite' caused a disease in silkworms, 1813

Semmelweiss and mortality associated with childbirth, 1847

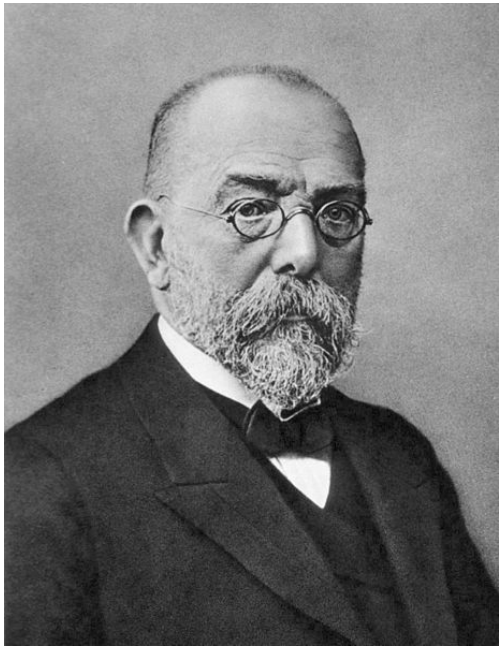
John Snow and Cholera, 1855 [Read, *the Ghost in the Well*]

Louis Pasteur, 1860-1864

Robert Koch, work with anthrax, 1893--Postulates

Louis Pasteur in his laboratory, painting by A. Edelfeldt in 1885

Koch's postulates are the following:



1. **The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.** Koch abandoned the universalist requirement of the first postulate altogether when he discovered asymptomatic carriers of cholera and, later, of typhoid fever.
2. **The microorganism must be isolated from a diseased organism and grown in pure culture.** The second postulate must also be suspended for certain microorganisms, all viruses, and entities that cannot (at the present time) be grown at all, such as prions responsible for Creutzfeldt–Jakob disease or Mad-cow disease (BSE).
3. **The cultured microorganism should cause disease when introduced into a healthy organism.** The third postulate specifies "should", not "must", because as Koch himself proved in regard to both tuberculosis and cholera, not all organisms exposed to an infectious agent will acquire the infection.
4. **The microorganism must be re-isolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.** In the case of HIV we would have to allow for a substantial number of mutations

Revised 21st Century 'molecular' rules to establish pathogenicity

1. A nucleic acid sequence belonging to a putative pathogen should be present in most cases of an infectious disease. Microbial nucleic acids should be found preferentially in those organs or gross anatomic sites known to be diseased, and not in those organs that lack pathology
2. Fewer, or no, copies of pathogen-associated nucleic acid sequences should occur in hosts or tissues without disease. This still suffers from the same problem that Koch had: many individuals carry an infectious agent, but do show not disease
3. With resolution of disease, the copy number of pathogen-associated nucleic acid sequences should decrease or become undetectable. With clinical relapse, the opposite should occur
4. When sequence detection predates disease, or sequence copy number correlates with severity of disease or pathology, the sequence-disease association is more likely to be a causal relationship
5. The nature of the microorganism inferred from the available sequence should be consistent with the known biological characteristics of that group of organisms
6. Tissue-sequence correlates should be sought at the cellular level: efforts should be made to demonstrate specific in situ hybridization of microbial sequence to areas of tissue pathology and to visible microorganisms or to areas where microorganisms are presumed to be located.
7. These sequence-based forms of evidence for microbial causation should be reproducible.

Fredericks DN, Relman DA (1996). "Sequence-based identification of microbial pathogens: a reconsideration of Koch's postulates". Clin Microbiol Rev 9 (1): 18–33.

Simplified more modern Koch's postulates are the following:

1. **Epidemiological association:** the suspected etiological agent must be strongly associated with the disease.
2. **Isolation:** the suspected pathogen can be isolated - and propagated - outside the host
3. **Transmission pathogenesis:** transfer of the suspected pathogen to an uninfected host, man or animal, produces the disease in that host

#1, Numerous studies from around the world show that virtually all AIDS patients are HIV-seropositive; that is they carry antibodies that indicate HIV infection

#2, Modern culture techniques have allowed the isolation of HIV in virtually all AIDS patients, as well as in almost all HIV-seropositive individuals. PCR and other sophisticated molecular techniques have enabled researchers to document the presence of HIV genes in virtually all patients at all stages of HIV disease

#3, Transfer of SIV to Rhesus macques causes AIDS. Accidental transfer of HIV to laboratory workers caused AIDS. Babies infected from i.v. drug using mothers died of AIDS. Hemophiliacs and surgical patients exposed to HIV-contaminated blood products died of AIDS

#3, Tragic incidents involving three laboratory workers with no other risk factors who have developed AIDS or severe immunosuppression after accidental exposure to concentrated, cloned HIV in the laboratory. In all three cases, HIV was isolated from the infected individual, sequenced and shown to be the infecting strain of virus

Transmission of HIV from a Florida dentist to six patients has been documented by genetic analyses of virus isolated from both the dentist and the patients. The dentist and three of the patients developed AIDS and died, and at least one of the other patients has developed AIDS. Five of the patients had no HIV risk factors other than multiple visits to the dentist for invasive procedures

CDC has received reports of 56 health care workers in the United States with documented, occupationally acquired HIV infection, of whom 25 have developed AIDS in the absence of other risk factors

The development of AIDS following HIV seroconversion has been repeatedly observed in:

pediatric and adult blood transfusion cases

in mother-to-child transmission

Hemophiliacs

injection-drug users

sexual transmission documented using serial blood samples

In a 10-year study in the Netherlands, researchers followed 11 children who had become infected with HIV as neonates by small aliquots of plasma from a single HIV-infected donor.
During the 10-year period, eight of the children died of AIDS. Of the remaining three children, all showed a progressive decline in cellular immunity, and two of the three had symptoms probably related to HIV infection
van den Berg *et al. Acta Paediatr* 1994;83:17).

Does HIV cause disease by direct inoculation?

- >12 strains of SIV cause a disease resembling AIDS in asian macaques: CD4 T cell depletion, generalized immune activation, opportunistic infections, weight loss, and wasting (species specific)
- Depending on the virus isolate and monkey species, SIV may or may not cause disease and may or may not cause death—rapid progression to death (SIVmac251 infection of newborn macaques), low-level viremia and not disease (avirulent isolates)
- Many attempts to infect chimpanzees with HIV caused infection but did not result in disease
- One such attempt resulted in AIDS 10 years after initial infection
- Virus from this chimp caused AIDS in a previously uninfected chimp
- The viruses re-isolated from these chimps showed extensive mutations in the *env* gene
- So within ethical limits one can say that purified immunodeficiency lenti viruses cause immunodeficiency disease in experimental infections
- HIV kills CD4 T cells in culture and in experimental mice given human lymphocytes

History of the epidemic as evidence for HIV as the cause of AIDS

Early suggestions that AIDS resulted from *behavior specific to the homosexual population* were not consistent with incidence of AIDS in distinctly different groups in the United States

- in male and female injection drug users
- recipients of blood products such as hemophiliacs *and* their partners
- blood transfusion recipients
- female sex partners of bisexual men
- infants born to mothers with AIDS or with a history of injection drug use
- HIV seroconversion in gay men occurred after 1978, but not before
- HIV seroconversion preceded AIDS by a few years in every part of the country
- no other infection or behavior has any predicted power for the appearance of AIDS
- 8000 people tested showed that HIV seropositive people were 1100x as likely to get AIDS
- 715 homosexual men followed for a median of 8.6 years. Every case of AIDS in this cohort occurred in individuals who were HIV-seropositive
- No AIDS-defining illnesses occurred in men who remained negative for HIV antibodies, despite the fact that these individuals had appreciable patterns of illicit drug use and receptive anal intercourse

References for the previous slide

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Prior to 1967 a total of **107** cases of pneumocystis had been described in the literature, all among individuals with an underlying immunodeficiency

By the end of 1999, there were **166,368** cases of HIV-infected people with definitive diagnoses of pneumocystis

Before HIV, KS was found in 0.2-0.6 cases per *MILLION* people

By the end of 1999, there were 46,684 HIV-infected people with definitive diagnoses of KS

The impact of HIV infection over five years in a rural population in the Masaka District of Uganda was studied: among 8,833 individuals tested, HIV+ people were 16x likely to die

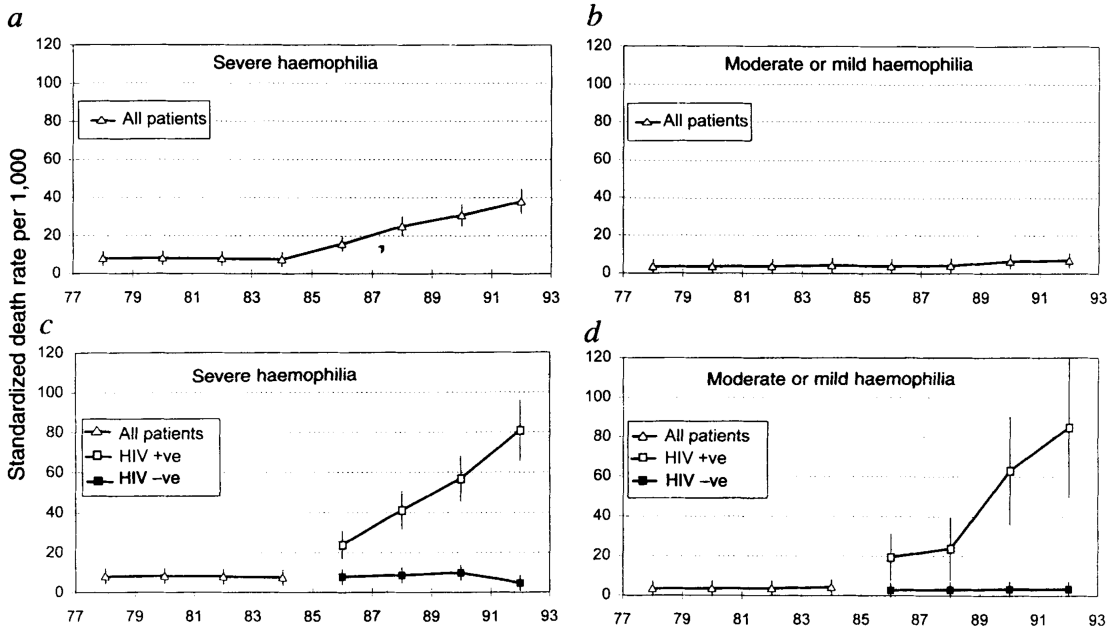
Similar finding in several other studies in Uganda, Tanzania, Malawi, Rwanda, Cote d'Ivoire, Zaire, and South Africa.

In Thai sex workers, death rate 52x higher for HIV+

HIV has been repeatedly isolated from the blood, semen and vaginal secretions of patients with AIDS, findings consistent with the epidemiologic data demonstrating AIDS transmission via sexual activity and contact with infected blood

Plasma RNA concentration Copies/mL of blood	Proportion of patients developing AIDS within 6 y
<500	5.4%
501-3,000	16.6%
3,001-10,000	31.7%
10,001-30,000	55.2%
>30,000	80.0%

analysis of 1,604 HIV-infected men in the Multicenter AIDS Cohort Study (MACS)



Mortality before and after HIV infection in the complete UK population of haemophiliacs

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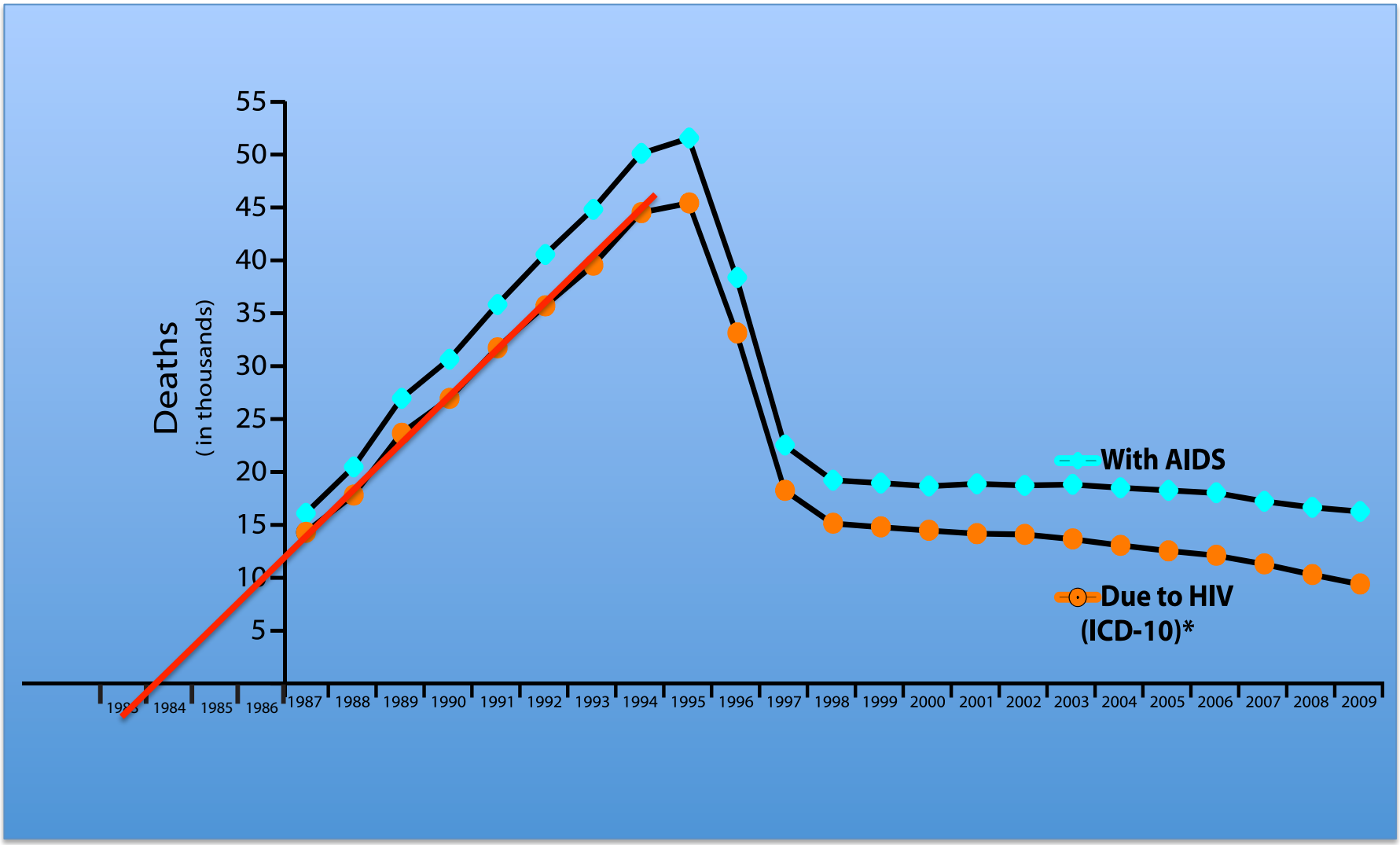
TABLE 3 Cause-specific mortality during 1985–92 by HIV status compared with national mortality

Certified cause of death (ICD-9 codes)	Tested seropositive for HIV: No*			Yes†		
	O‡	*E*§	*O/E*	*O*	*E*	*O/E*
(A) AIDS, HIV, etc. (279.1)	0	0.10	0.0	235	0.12	1,958.3***
(B) Causes significantly increased in severe haemophilia without HIV						
Hepatitis and liver disease (070, 570–573)	6	0.37	16.2***	11	0.30	37.0***
Liver cancer (155.0–155.1)	2	0.11	18.7*	1	0.07	15.1
Coagulation defects, etc. (280–289)	33	0.11	307.2***	72	0.06	1,155.7***
Intracranial haemorrhage (ICH, 430–432)	5	0.49	10.2***	1	0.37	2.7
Injury, poisoning and suicide (E800–999)	10	3.14	3.2**	8	3.68	2.2
All causes in category (B)	56	4.21	13.3***	93	4.47	20.8***
(C) Ischaemic heart disease (IHD, 410–414)	5	10.37	0.5	5	5.74	0.9
(D) Other causes						
Infections excl. hepatitis (001–139, excl. 070)	0	0.23	0.0	11‖	0.14	75.6***
Hodgkin's disease (201)	0	0.06	0.0	2¶	0.07	29.1**
Non-Hodgkin's lymphoma (200, 202)	0	0.29	0.0	12	0.21	57.1***
Other neoplasms excl. liver (140–239 excl. 155.0–.1 and 200–2)	9	9.38	1.0	7#	5.28	1.3
Endocrine disorders excl. AIDS, HIV, etc. (240–279 excl. 279.1)	1	0.50	2.0	1	0.30	3.3
Mental disorders (290–319)	0	0.35	0.0	2	0.25	8.1
Nervous system incl. dementia (320–389)	1	0.78	1.3	6	0.54	11.2***
Circulatory excl. IHD and ICH (390–459 excl. 410–4 and 430–2)	7	3.86	1.8	6	1.85	3.2*
Pneumonia (480–486)	0	0.63	0.0	12	0.31	38.6***
Other respiratory (rest of 460–519)	2	2.14	0.9	2	0.99	2.0
Digestive system excl. liver (520–579 excl. 570–573)	0	0.63	0.0	3	0.34	8.7*
Musculoskeletal and connective tissue (710–739)	1	0.11	9.0	2	0.06	33.8*
Other diseases (580–709, 740–799)	1	1.03	1.0	2☆	0.36	5.5
All causes in category (D)	22	20.00	1.1	68	10.70	6.4***
(E) Death certificate not located	1	—	—	2	—	—
All causes	84	34.68	2.4***	403	21.03	19.2***

‡ *O*, observed deaths.
§ *E*, expected deaths from national rates

The death-rate of HIV+ patients dropped dramatically with the introduction of HAART



Nearly everyone with AIDS has antibodies to HIV.
A survey of 230,179 AIDS patients in the United States revealed only 299 HIV-seronegative individuals. An evaluation of 172 of these 299 patients found 131 actually to be seropositive; an additional 34 died before their sero-status could be confirmed

The specific immunologic profile that typifies AIDS - a persistently low CD4+ T-cell count - is extraordinarily rare in the absence of HIV infection or other known cause of immunosuppression.
For example, in the NIAID-supported Multicenter AIDS Cohort Study (MACS), 22,643 CD4+ T-cell determinations in 2,713 HIV-seronegative homosexual and bisexual men revealed only one individual with a CD4+ T-cell count persistently lower than 300 cells/mm^3 of blood, and this individual was receiving immunosuppressive therapy.

Newborn infants have no behavioral risk factors for AIDS, yet many children born to HIV-infected mothers have developed AIDS and died.
Only newborns who become HIV-infected before or during birth, during breastfeeding, or (rarely) following exposure to HIV-tainted blood or blood products after birth, go on to develop the profound immunosuppression that leads to AIDS. Babies who are not HIV-infected do not develop AIDS. In the United States, 8,718 cases of AIDS among children younger than age 13 had been reported to the CDC as of December 31, 1999. Cumulative U.S. AIDS deaths among individuals younger than age 15 numbered 5,044 through December 31, 1999.

Peter Duesberg's arguments and responses by Blattner, Gallo, Temin (and me)

Human immunodeficiency virus (HIV) is not the cause of AIDS because it fails to meet the postulates of Koch and Henle, as well as six cardinal rules of virology.

1. HIV is in violation of Koch's first postulate because it is not possible to detect free virus, provirus, or viral RNA in all cases of AIDS.
2. In violation of Koch's second postulate, HIV cannot be isolated from 20 to 50% of AIDS cases. Moreover, "isolation" is very indirect. It depends on activating dormant provirus in millions of susceptible cells propagated in vitro away from the suppressive immune system of the host.

It was formerly true that evidence for the presence of HIV-1 could not be found in all AIDS patients. Improved molecular methods now show that HIV infection is present in essentially all AIDS patients.

3. In violation of Koch's third postulate, pure HIV does not reproduce AIDS when inoculated into chimpanzees or accidentally into healthy humans

HIV infects chimpanzees, but usually doesn't cause AIDS; however, after extensive mutation, one case of AIDS arose, and the virus from that chimp rapidly caused AIDS in a second. The reisolated virus from the second chimp was virtually identical to the virus isolated from the first. HIV accidently injected into humans most definitely does cause AIDS. Viruses are species specific, in terms of both infection and pathology. Herpes B, Yellow fever, and Dengue cause disease in humans, but not many species of monkey.

Peter Duesberg's arguments continued

4. In contrast to all pathogenic viruses that cause degenerative diseases, HIV is not biochemically active in the disease syndrome it is named for. It actively infects only 1 in 10^4 to > 10^5 T cells. Under these conditions, HIV cannot account for the loss of T cells, the hallmark of AIDS, even if all infected cells died. This is because during the 2 days it takes HIV to replicate, the body regenerates about 5% of its T cells, more than enough to compensate for losses due to HIV.

All these numbers come from just looking at peripheral blood lymphocytes. Looking at lymph node T cells, or intestinal CCR5$^+$ memory T cells, there is devastation. HIV doesn't have to complete replication in order to kill T cells.

5. It is paradoxical that HIV is said to cause AIDS only after the onset of antiviral immunity, detected by a positive "AIDS test," because all other viruses are most pathogenic before immunity. The immunity against HIV is so effective that free virus is undetectable, which is why HIV is so hard to transmit. The virus would be a plausible cause of AIDS if it were reactivated after an asymptomatic latency, like herpes viruses. However, HIV remains inactive during AIDS. Thus the "AIDS test" identifies effective natural vaccination, the ultimate protection against viral disease.

Antibody titers are not necessarily neutralization titers. Note in Figure 7 from the William Schief lecture (Richman DD, Wrin T, Little SJ, Petropoulos CJ. Proc Natl Acad Sci U S A. 2003 Apr 1;100(7):4144-9.). Although the antibody titers are impressive when tested against a control virus, ***they do not rise appreciably against the virus present at the time of testing.*** *Indeed HIV IS reactivated as the patient proceeds to AIDS. CD4 counts drop, viral titers increase. In addition, many viruses cause disease after immunity develops: HZV-shingles, HSV-local lesions, equine infect anemia, visna virus causes cns degeneration after appearance of neutralizing antibodies*

Peter Duesberg's arguments continued

6. The long and highly variable intervals between the onset of antiviral immunity and AIDS, averaging 8 years, are bizarre for a virus that replicates within 1 to 2 days in tissue culture and induces antiviral immunity within 1 to 2 months after an acute infection. Since all genes of HIV are active during replication, AIDS should occur early when HIV is active, not later when it is dormant. Indeed, HIV can cause a mononucleosis-like disease during the acute infection, perhaps its only pathogenic potential

This is confused in many ways. AIDS only occurs when CD4 T cell levels fall to low levels. HIV itself isn't an indication of immunodeficiency, it causes immunodeficiency by killing CD4 T cells either directly or indirectly. AIDS **does** *present when the viral titers rise.*

7. Retroviruses are typically not cytocidal. On the contrary, they often promote cell growth. Therefore, they were long considered the most plausible viral carcinogens. Yet HIV, a retrovirus, is said to behave like a cytocidal virus, causing degenerative disease killing billions of T cells. This is said even though T cells grown in culture, which produce much more virus than has ever been observed in AIDS patients, continue to divide

Retroviruses include 8 different virus subfamilies. No one would rationalize the behavior or pathogenesis of all family members from the example of one subfamily. Furthermore, HIV KILLS T CELLS IN CULTURE

8. It is paradoxical for a virus to have a country-specific host range and a risk group-specific pathology. In the United States, 92% of AIDS patients are male, but in Africa AIDS is equally distributed between the sexes, although the virus is thought to have existed in Africa not much longer than in the United States. In the United States, the virus is said to cause Kaposi's sarcoma only in homosexuals, mostly Pneumocystis pneumonia in hemophiliacs, and frequently cytomegalovirus disease in children. In Africa the same virus is thought to cause slim disease, fever, and diarrhea almost exclusively.

Agreed that these characteristics, although exaggerated here, are difficult to understand. Yet, that is not a good reason to deny the causal relationship of HIV to AIDS. Each of these subgroups has complicating factors: gay bowel syndrome for example. PCP is found in virtually all groups. CMV is found in all groups. Wasting or 'slim' is found in all groups. KS remains the most frequently reported cancer in some African countries, due to untreated HIV – e.g., Zimbabwe. It is caused by HSV8, largely in immunocompromised hosts.

The underlying pathology in AIDS is immune deficiency. The nature of the opportunistic agents that invade the susceptible host is a function of the agents that are most prominent in a particular population. For example, in the United States Pneumocystis is most prominent in affluent gay men, while human mycobacterial infections and toxoplasmosis are more frequent in socially disadvantaged Caribbean immigrants. Other agents, such as Cryptococcus, are more prominent in developing countries.

9. It is now claimed that at least two viruses, HIV-1 and HIV-2, are capable of causing AIDS, which allegedly first appeared on this planet only a few years ago. HIV-1 and HIV-2 differ about 60% in their nucleic acid sequences. Since viruses are products of gradual evolution, the proposition that within a few years two viruses capable of causing AIDS could have evolved is highly improbable

This shows a real lack of understanding. These two viruses clearly came from two different non-human primate species. They have been on earth for a long time, they probably entered the human species in the 20th century, but it wasn't until urbanization that they broke out as epidemics

Read for yourself
Duesberg on AIDS
www.duesberg.com/papers/ch2.html

MYTHs, can you debunk them?:

HIV antibody testing is unreliable

There is no AIDS in Africa. AIDS is nothing more than a new name for old diseases

HIV cannot be the cause of AIDS because researchers are unable to explain precisely how HIV destroys the immune system

AZT and other antiretroviral drugs, not HIV, cause AIDS

Behavioral factors such as recreational drug use and multiple sexual partners account for AIDS

AIDS among transfusion recipients is due to underlying diseases that necessitated the transfusion, rather than to HIV

High usage of clotting factor concentrate, not HIV, leads to CD4+ T-cell depletion and AIDS in hemophiliacs

The distribution of AIDS cases casts doubt on HIV as the cause. Viruses are not gender-specific, yet only a small proportion of AIDS cases are among women

HIV cannot be the cause of AIDS because the body develops a vigorous antibody response to the virus

Only a small number of CD4+ T cells are infected by HIV, not enough to damage the immune system

HIV is not the cause of AIDS because many individuals with HIV have not developed AIDS

Foo Fighters, HIV Deniers

Foo Fighters bassist Nate Mendel helped organize a sold-out concert in Hollywood to benefit **Alive and Well**, an "alternative AIDS information group". Foo fans were treated to a speech by **Alive and Well** founder Christine Maggiore, who believes AIDS may be caused by HIV-related medications, anal sex, stress, and drug use, and implies that people should not get tested for HIV nor take medications to counter the virus. Free copies of Maggiore's self-published book, "What If Everything You Thought You Knew About AIDS Was Wrong?," in which she declares "there is no proof that HIV causes AIDS," were also passed out to the concert-goers. This was February 2000

Christine Maggiore herself was diagnosed as HIV-positive in 1992. She was evangelized by Duesberg and took up a fight to deny HIV as the cause of AIDS. Christine Maggiore's 3-year-old daughter, Eliza Jane, was HIV positive and died in 2005. Her son was HIV-negative and remains healthy.

From Wikipedia:
In April 2005, Eliza Jane became ill with a runny nose. She was seen by two physicians, one of whom reportedly knew of Maggiore's HIV status. Eliza Jane was not tested for HIV, and was diagnosed with pneumonia.[4][10] When Eliza Jane failed to improve, Maggiore took her to see Philip Incao, a holistic practitioner and board member of Maggiore's AIDS-denialist organization Alive & Well AIDS Alternatives, who claimed Eliza Jane appeared to be only mildly ill,[2] and prescribed her amoxicillin for a presumed ear infection. On May 16, 2005, Eliza Jane collapsed and stopped breathing.[4] She was rushed to Valley Presbyterian Hospital in Van Nuys, California, where, after failed attempts to revive her, she was pronounced dead.[10]
An autopsy revealed that Eliza Jane was markedly underweight and under height, consistent with a chronic illness, exhibited a pronounced atrophy of her thymus and other lymphatic organs, and that her lungs were infected with *Pneumocystis jirovecii*, a common opportunistic pathogen in people with AIDS and the leading cause of pediatric AIDS deaths.[11] The post-mortem examination of Eliza Jane's brain showed changes consistent with HIV encephalitis; protein components of HIV itself were identified in Eliza Jane's brain tissue via immunohistochemistry.[10]

The coroner concluded unequivocally that Eliza Jane had died of Pneumocystis pneumonia in the setting of advanced AIDS.[2][10] Maggiore rejected the coroner's conclusion, ascribing it to political bias and attacking the personal credibility of the senior coroner, James Ribe.[12] Maggiore had the autopsy reviewed by AIDS denialist Mohammed Al-Bayati, who holds a Ph.D. in animal disease pathology, but is not a medical doctor, nor board-certified in human pathology.[13] Al-Bayati concluded Eliza Jane died from an allergic reaction to amoxicillin,[14] a conclusion Maggiore embraced.[2]
Al-Bayati's report has been dismissed as both biased and medically unsound[15][16] by independent pathology experts who agreed with the coroner's conclusion.[2][4]

Maggiore herself lived until 2008, when she died of pneumonia for which she had been treated for the previous 6 months. She was 52. Her supporters expressed shock over her death but were highly skeptical that is was caused by AIDS. They said it would not stop them from questioning mainstream thinking.

Christine Maggiore's death certificate states that the immediate cause or condition resulting in her death was disseminated herpes viral infection. Bilateral bronchial pneumonia is given as the underlying cause, and oral candidiasis is given as a significant condition contributing to death.
In HIV-infected people, all of the above conditions are AIDS-defining opportunistic infections, and in combination they are hauntingly typical of AIDS in the years before ARVs.
Many people who have died of AIDS shared the same constellation of opportunistic infections that killed Ms Maggiore. HIV-negative individuals never have all of these opportunistic infections simultaneously.
Maggiore's physician in the weeks prior to her death was Ilona Abraham, MD, of Encino, California. She signed the death certificate and states that Maggiore was in her care from December 16, 2008, to December 23, 2008, when she last saw her alive. Abraham graduated from Semmelweis Medical School in Budapest, Hungary, in 1967. She has an extensive history of malpractice cases and disciplinary actions, and no expertise in HIV. Abraham practices "anti-aging" medicine, and treats her patients with treatments such as chelation and homeopathy.

For more, go to aidstruth.org

MEDICAL EXAMINER
HEALTH AND MEDICINE EXPLAINED OCT. 8 2013 11:47 PM
Ken Sepkowitz

Tommy Morrison poses during a weigh-in in Tokyo on Nov. 2, 1996.

Tommy Morrison, once the heavyweight boxing champion of the world, died of AIDS last month. His case, however, was not typical for someone with HIV: He and his wife, Trisha, denied he had the infection to the bitter end. In fact, not only did they deny that Morrison was infected, they denied that HIV causes AIDS at all. And it's not even clear that they think the condition called AIDS actually exists

Peter Duesberg - professor at UC-Berkeley

Peter Duesberg

Read the article from AIDSTruth.org, “Peter Duesberg: Malignant Narcissism in the Cancer Lab”

http://www.aidstruth.org/features/2007/peter-duesberg-malignant-narcissism-cancer-lab

The deniers, what do they have in common? (edited from Kent Sepkowitz)

Anti-vaccination activists: At their core is a clutch of parents worried about their kids. Any parent knows that parenthood can be a rationality-free zone

Anti-global warming agnostics: They seem more like a front group for Big Oil or at least Chamber of Commerce boosterism than a group of wide-eyed (non)believers willing to take a life-and-death stand on principle (WSJ, Fox News, the US Chamber of Commerce)

Anti-evolution zealots are just expressing their deliberately blind faith in a view of life based on sacred texts

HIV/AIDS deniers are uniquely putting their lives on the line

HIV deniers are an oddball collection of prissy scientists, right-wing moralizers, standard big-government paranoids, and those still very spooked by the idea that men have sex with men. There are no TV stars in their midst, much less Big Money; they have neither conventions nor house organs—*even Fox News sits at a safe remove*. Indeed, they have no real political agenda except perhaps a limp suspicion that science is corrupt and scientists are even more corrupt and that all those taxpayer billions could be better spent on a real disease

They are true-blooded, bright-eyed nonbelievers…until they die a horrible death from multiple opportunistic infections

How are denialists so successful in getting a media audience for their agenda

Denialists, of whatever flavor, try to create the illusion of debate—where there is none.

Denialists appeal to our sense of skepticism

They try to appeal to openness by questioning the most basic established principles (age of the earth, distance of galaxies, evolution, atmospheric CO_2)

They enlist "experts" usually people without credentials in the field of interest

They change the standards as evidence accumulates

They use special pleading for each argument

- AIDS comes from recreational drug use
- No, AIDS comes from Factor VIII given hemophiliacs
- No, AIDS comes from AZT itself
- No, AIDS comes from multiple sex partners
- Actually, there is no AIDS, it is just old diseases repackaged

They appeal to our innate tendencies to believe in conspiracies

- Pharma companies sequester cancer cures to keep themselves in business
- George W. Bush orchestrated the demolition of the twin towers
- Iraq has weapons of mass destruction
- The entire scientific community of the world colluded to fake a moon landing

Top 10 ways to identify a misguided denier:

① Believes in a massive conspiracy
② Has an agenda (political, monetary, religious, etc)
③ Changes arguments with each new study
④ Claims to be an expert with no background in the field of interest
⑤ Claims most everyone else is misguided at best, but more likely stupid
⑥ Argues against a well supported principle on the basis that certain details are unclear
⑦ Misuses the term, theory
⑧ Is obsessed
⑨ Has a history of belief in mythology (Loch Ness, Astrology, etc.)
⑩ Listens to AM talk radio and watches Fox News