

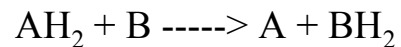
Review for Final:

Study old exams - I will reuse/rewrite some of those questions

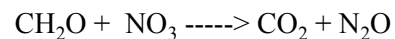
Review big themes - see the rest of this lecture and last lecture

Anaerobic respiration....

Same thing but with a different e⁻ acceptor



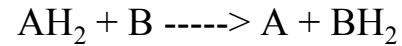
For anaerobic chemoheterotrophs (anaerobic respiration):



See Fig. 6.8

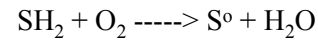
Remember from lec. 2....

Kluyver and van Niel proposed that all respiratory reactions (aerobic and anaerobic) could be summarized using the simple formula:

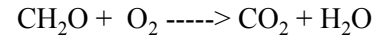


Aerobic Resp.

For *Beggiatoa* (example of aerobic respiration)



For aerobic chemoheterotrophs (example of aerobic respiration):



All catabolic reactions are oxidation/reduction reactions - it's all about movement of electrons.....

An extreme illustration of this is *Geobacter sulfurreducens* (Delta-Proteobacteria) - it can use metal electrodes as electron acceptors for anaerobic respiration. It is now being tested in microbial fuel cells to generate electricity. It can use acetate as its e⁻ donor, thus electrical current production is dependent upon oxidation of acetate to carbon dioxide.

Acetate + metal surface ----> electrical current (flows to + side of the "battery").

Please read the abstract to this paper..... Electricity Production by *Geobacter sulfurreducens* Attached to Electrodes. Appl. Environ. Microbiol. 69: 1548-1555, (D.R. Bond & D.R. Lovley) (Full paper is on class web site)

Electricity Production by *Geobacter sulfurreducens* Attached to Electrodes. Appl. Environ. Microbiol. 69: 1548-1555, (D.R. Bond & D.R. Lovley)

Previous studies have suggested that members of *Geobacter* can use electrodes as electron acceptors for anaerobic respiration. In order to better understand this electron transfer process for energy production, *G. sulfurreducens* was inoculated into chambers in which a **graphite electrode served as the sole electron acceptor and acetate or hydrogen was the electron donor.** The electron-accepting electrodes were maintained at oxidizing potentials by connecting them to similar electrodes in oxygenated medium (fuel cells) or to potentiostats that poised electrodes at +0.2 V versus an Ag/AgCl reference electrode (poised potential). When a small inoculum of *G. sulfurreducens* was introduced into electrode-containing chambers, **electrical current production was dependent upon oxidation of acetate to carbon dioxide** and increased exponentially, indicating for the first time that electrode reduction supported the growth of this organism. When the medium was replaced with an anaerobic buffer lacking nutrients required for growth, acetate-dependent electrical current production was unaffected and cells attached to these electrodes continued to generate electrical current for weeks. This represents the first report of microbial electricity production solely by cells attached to an electrode. Electrode-attached cells completely oxidized acetate to levels below detection (<10 μ M), and hydrogen was metabolized to a threshold of 3 Pa. The rates of electron transfer to electrodes (0.21 to 1.2 μ mol of electrons/mg of protein/min) were similar to those observed for respiration with Fe(III) citrate as the electron acceptor ($E_o' = +0.37$ V). The production of current in microbial fuel cell (65 mA/m² of electrode surface) or poised-potential (163 to 1,143 mA/m²) mode was greater than what has been reported for other microbial systems, even those that employed higher cell densities and electron-shuttling compounds. Since acetate was completely oxidized, the efficiency of conversion of organic electron donor to electricity was significantly higher than in previously described microbial fuel cells. These results suggest that the effectiveness of microbial fuel cells can be increased with organisms such as *G. sulfurreducens* that can attach to electrodes and remain viable for long periods of time while completely oxidizing organic substrates with quantitative transfer of electrons to an electrode.

As an illustration - Rabbits in Australia.

Rabbits were first introduced to Australia from England in 1859. Because they had **no natural enemies** they soon became major pests. Finally in 1950 a scientist isolated a Myxoma virus that caused a not-very-serious disease in a species of Brazilian rabbits. When this **virus was brought to Australia** (it was a new disease to those English rabbits) it was extremely virulent to them. Within a year, **99.9% of all the rabbits in Australia died.** Over the following 20 years, scientists closely monitored the co-evolution of this new disease and the rabbits of Australia. The main finding of these studies was that **the virulence of the virus decreased** each year and the **resistance of the rabbits increased.** Finally after about ten years the number of rabbits in Australia increased back up to about 20% of their pre-virus levels.

Evolution of diseases:

In many cases the longer a population is exposed to a pathogen, the less susceptible that population is to the disease. Over the short term (months to years) this is due to the fact that more hosts become immune. Over the long term, this is due to natural selection which tends to **weed out extremely virulent pathogens**, because if they kill all of their hosts, then they too will go extinct. Simultaneously hosts that are more resistant to a prevalent pathogen have more offspring. Thus, through evolutionary time specific pathogens tend to become less virulent to their hosts become more resistant to their pathogens. It is thought that some non-pathogenic relationships evolved from former antagonistic ones via a progression like this...

Pathogenic ----> Commensalistic --?--> Mutualistic

Two important points about the above story.

- 1) The long-term change in the rabbits was genetic, i.e. more resistant rabbits were selected for.
- 2) It took about ten rabbit generations (assuming 1 generation per year) for the rabbits and virus to reach an equilibrium in which the rabbit was quite resistant and the virus was much less virulent.

Let's quickly review that with an overhead.

Of course not all diseases evolve exactly like the rabbit/myxoma case. Co-evolution is a back and forth process that can lead to a balance, as in the rabbit/myxoma case, or to either the pathogen or the host gaining advantage at any point in time. And evolution can lead to re-emergence of diseases like the flu, plague and other diseases we have discussed.....

Emerging Infectious Diseases

The rabbit story is pertinent to how and why new diseases keep cropping up (see Lecture 23.....)

From Lec. 23.....Emerging Diseases

“New, reemerging, or drug-resistant infections whose incidence in humans has increased within the past two decades or whose incidence threatens to increase in the near future.”

From Lec. 23 Fig. 20.10. Some emerging infectious diseases. Most are old diseases that are re-emerging or diseases that jump from animals to humans (HIV, Ebola,)

Some reasons for emerging diseases:

- **climate change**
- **Over use of antibiotics**
- **human encroachment on wilderness / use of wild animals as food**
- **increased international travel - faster dispersal of new pathogens**

Know reservoirs, vectors and pathogens (including where on the big tree they fall) for diseases we have discussed....

Also try to understand why they are on the increase or emerging.....

Emerging diseases:

AIDS (jumped from animals)

Anthrax (biological warfare/terrorism)

Cholera (disruptions in public sanitation, due to poverty, tsunamis etc.)

Tuberculosis (antibiotic resistance, AIDS, breakdown in public health)

West Nile Virus (international travel)

Whooping Cough/Pertussis (lax immunization programs)

Yellow fever (global warming?)

Flu (jumps from animals every year)

Plague (could re-emerge due to large reservoirs and lack of public awareness)

Prion diseases (poor practice in feed lots and burger industry)