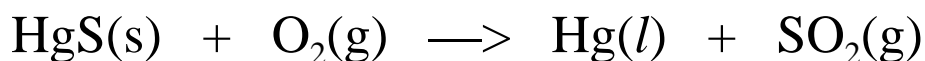


## Metals in the Environment

- Unlike organic contaminants, metallic components cannot be “destroyed”
- The speciation of the metal may significantly affect its toxicity, and hence its environmental impact.

### Issue #1: Mercury

- Mercury occurs as the element or as the sulfide (HgS, two forms: cinnabar and vermillion). Roasting in air gives the element directly



- Hg(*l*), b.p. 357°C, has a significant vapour pressure at ambient temperatures,  $2.4 \times 10^{-6}$  atm

*Calculate this in mg/m<sup>3</sup> at 298 K and compare with the TLV of 0.05 mg/m<sup>3</sup>*

- Toxicity of mercury: the metal and inorganic salts are much less toxic than alkylmercury compounds; arylmercury compounds (antifungal seed dressings) are not as toxic
- Methylmercury compounds are formed in the environment by microbial alkylation, involving cobalamin (Vitamin B<sub>12</sub>) which has a Co–CH<sub>3</sub> bond

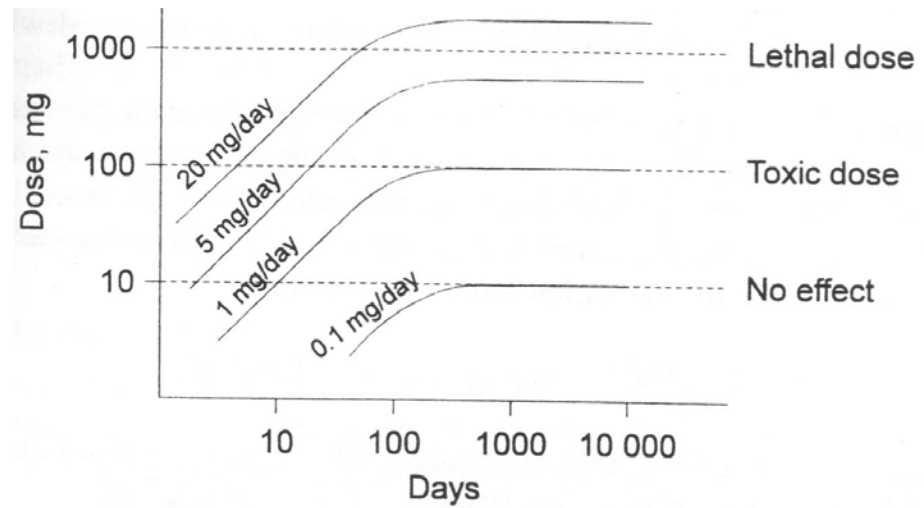
- C-Hg bonds are non-polar —> organomercury compounds are lipophilic and bioaccumulative
- Mercury and its compounds: neurotoxins and renal toxins

*Handwritten text in Italian, likely describing symptoms of mercury poisoning such as tremors and neurological effects.*

Handwriting of an Italian who worked in a mercury mine.

Renal toxicity due to strong Hg-S bonds: metallothioneins small proteins with a high percentage of -SH groups, Hg-metallothioneins deposited in kidney

- Mercury is a cumulative poison:



Accumulation curves for different levels of mercury in the diet.

### Exposure to mercury compounds:

- Historically, gilding mirrors, alchemy (Newton), hat-making (the Mad Hatter)
- Mining and refining mercury
- Gold mining (Amazon basin)
- Dental amalgams (dental offices a major source of mercury in Toronto's sewage)
- Scientific equipment and old science labs

### Mercury in the wider environment:

- Burning coal (mercury is volatile and not trapped as a flue dust) ... also volcanoes
- Garbage incineration: especially the use of Hg batteries for cameras, hearing aids (also landfills for the same reason)



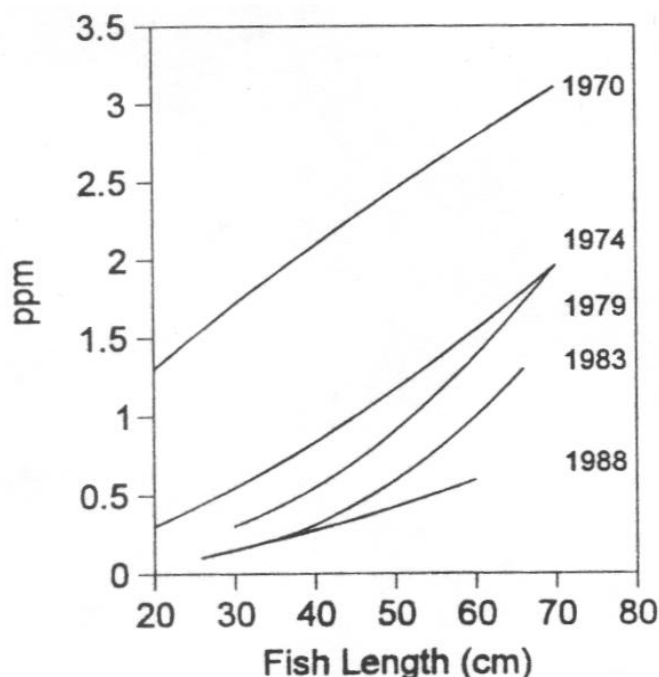
for which

$$\Delta G = \Delta G^\circ + RT \ln Q$$

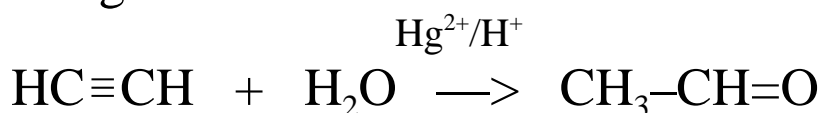
$Q = 1$  because all components are pure (s) or (l), hence  $E_{\text{cell}}$  does not change with time

- Use of organomercury fungicides (minor)
- The old flowing mercury cells for chloralkali process (1960s, losses of 200 g Hg per tonne of  $\text{Cl}_2$  produced: reduced to 0.15 g/tonne by 1980): Canadian example: Reed Paper Co in Northern Ontario: White Dog and Grassy Narrows Indian Reserves

- Mercury in fish: the limit of 0.5 ppm was set in the 1970s when Hg pollution was more of a problem *but* analytical methods were less advanced



- Use of mercury as an industrial catalyst: Minamata Disease (Japan): 1500 cases in the 1950s, 200 deaths; neurological symptoms; easily associated with shellfish but originally not clear whether this was a disease condition or a chemical poisoning



*Note the preceding two examples as historical only; these problems have been largely resolved*

Principal problem remaining today:

- air pollution due to coal burning and incineration

US EPA documents (1997) on mercury releases (1994/95 data):

<http://www.epa.gov/ttn/oarpg/t3/reports/>

Total of combustion sources ~140 t/yr: includes (major)

Coal-fired electric power plants ~ 50 t/yr

Coal and oil fired industrial boilers ~ 30 t/yr

MSW incinerators ~30 t/yr

Medical and hazardous waste incinerators ~ 30 t/yr

Clean Air Act Amendments expected to reduce emissions from incinerators

Total non-combustion sources ~ 20 t/yr

Chloralkali now ~ 5 t/yr

Cement production ~ 5 t/yr

Natural and anthropogenic releases of Hg are of similar orders of magnitude

volcanoes

flooding (including hydroelectric dams)

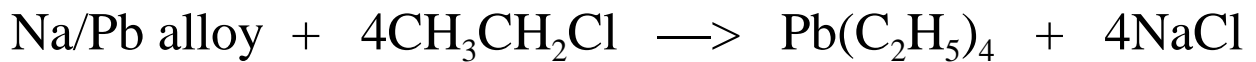
Recent article on Hg and cerebral palsy in Ontario: *Globe & Mail*, Saturday June 12, 2004.

## Issue #2: Lead

- Known since ancient times: used for plumbing systems (Roman Empire), roofing (medieval cathedrals)
- Modern uses:
  - solder: Pb-Sn alloy
  - paints (white lead,  $2\text{PbCO}_3 \cdot \text{Pb}(\text{OH})_2$ ; red lead,  $\text{Pb}_3\text{O}_4$ ; lead chromate  $\text{PbCrO}_4$ )
  - lead-acid batteries
  - gasoline anti-knock agents tetramethyl- and tetraethyl-lead [no longer used in Europe or North America]
  - crystal glass ( $\text{PbO} \rightarrow$  high refractive index)
  - lead shot (shooting ranges; waterfowl)
- Historical production of lead: see text, Figures 10.4 and 10.5
- Toxicity of lead:
  - $\text{Pb}^{2+}$  substitutes for  $\text{Ca}^{2+}$  in bone; depuration is very slow so that Pb accumulates with age
  - as with mercury, organoleads are more toxic than inorganic lead; even inorganic salts are partly covalent *e.g.*,  $\text{PbBr}_2$
  - hence Pb can cross blood-brain barrier  $\rightarrow$  neurotoxicity in adults and children; mental deficits in children (lead paint)
  - lead recycling (“secondary lead smelters”)  $\rightarrow$  air pollution since Pb has m.p.  $327^\circ\text{C}$ ; b.p.  $1740^\circ\text{C}$

## Lead in gasoline

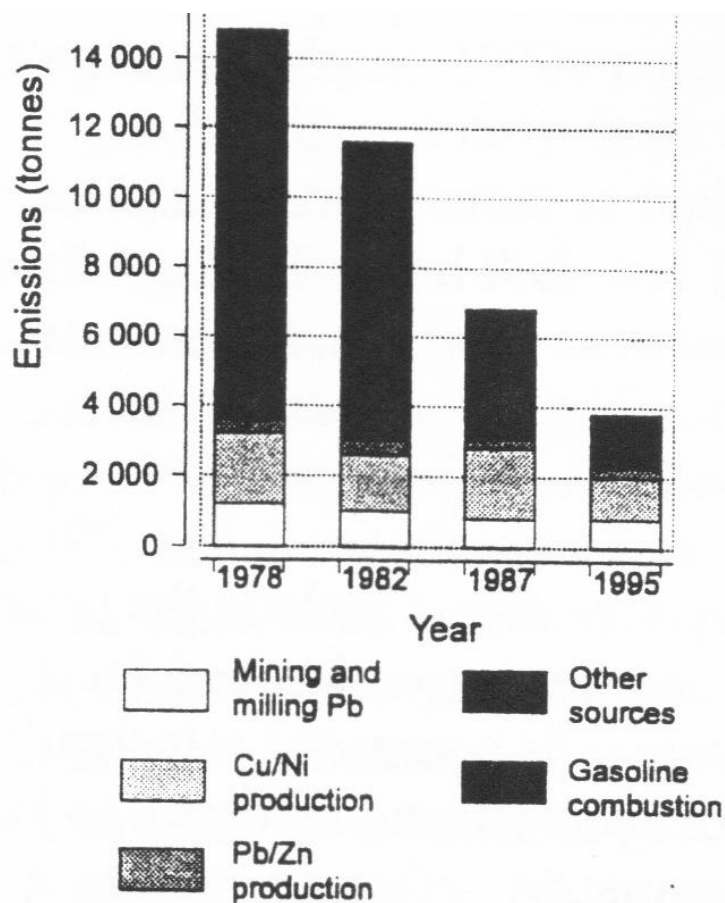
- petroleum is mostly straight chain alkanes
- branched chain alkanes burn more smoothly
- petroleum distillation —> “straight run” gasoline – a poor fuel with tendency to knocking/pinging (details in text)
- options to increase the **octane rating** are:
  - more complex refining processes —> more branched chain and aromatic hydrocarbons
  - addition of tetraalkyl lead compounds as “anti-knock agents”: Ethyl Corp, founded 1920s



in the engine:



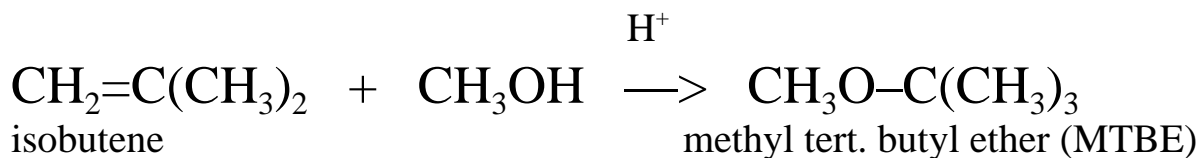
- roadside dusts are high in inorganic Pb compounds
- catalytic converters incompatible with leaded gasoline —> unleaded gasoline



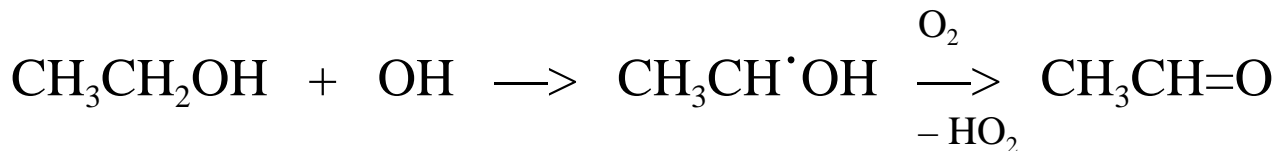
lead emissions in Canada, 1978-1995.

Related issue: oxygenates and MTBE

- US Clean Air Act mandated 2% by weight [O] from oxygenates to try to curb ozone in photochemical smog
- MTBE added, because it also has high octane rating ~110



- MTBE now found to be a ground water pollutant
  - recalcitrant
  - DNAPL source from leaking gas tanks
  - possible carcinogenicity
- alternative oxygenate = ethanol
  - from corn starch by fermentation and distillation
  - concern that ethanol increases aldehyde levels

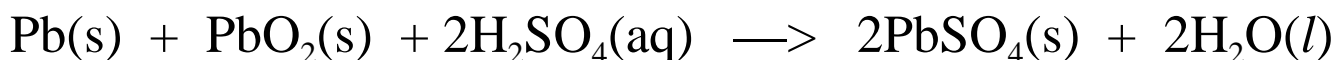


- recall that  $\text{CH}_3\text{CH}=\text{O}$  is a precursor of peroxyacetyl nitrate, PAN

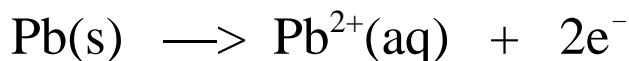
### Lead-acid battery (in cars)



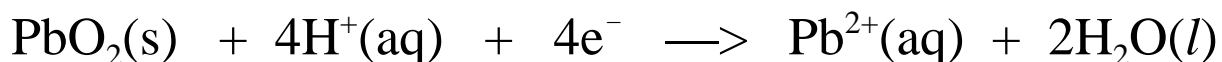
galvanic cell (when you start your car):



anode reaction:



cathode reaction:



electrolytic cell (when the battery charges):



For  $E_{\text{cell}}$  during discharge:

$$E = E^{\circ} - \frac{RT}{nF} \cdot \ln (1/[\text{H}_2\text{SO}_4])^2$$
$$\approx E^{\circ} + \frac{RT}{nF} \{2\ln [\text{H}^+] + 2\ln [\text{HSO}_4^-]\}$$

This is why the charge of a battery can be determined by measuring the *density* of the electrolyte

Problems with lead-acid batteries:

- technical problem of low energy density
- recycling – secondary lead smelters (Junction Triangle, Toronto)
- improper disposal:
  - landfill leachate
  - poisoning of cattle

### Issue #3: Arsenic

- 20<sup>th</sup> most abundant element in Earth's crust, but few uses
- toxic: essentially no known role as micronutrient
- unlike Hg and Pb, organoarsenicals are **less** toxic than inorganic arsenic
- seaweeds contain various arseno-sugars (little known about their toxicity)
  
- Shellfish contain arsenobetaine and the related arsenocholine; these are non-bioavailable, water-soluble, and hence readily excreted.



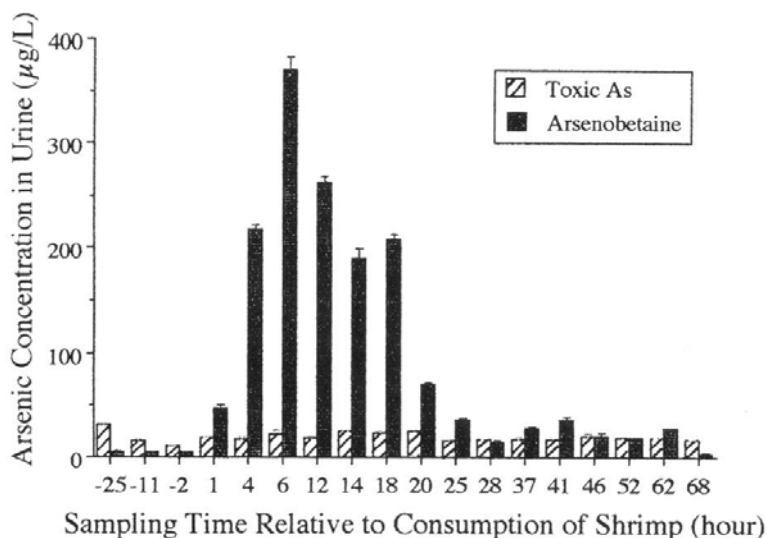
**Canadian exposure to arsenic** (*Canadian Chemical News*, September 1999, p. 18)

Food, usually < 0.04 mg/day in North America. With high seafood, up to 0.2 mg/day (mostly “non-toxic” forms).

<i>Food:</i>	fish	1700 ng/g
	meat, poultry, cereals, fats	20 ng/g
	shellfish	~20 μg/g

*e.g.*, 1 lobster meal could give ingestion of 5 mg of As, but principal form is the relatively non-toxic arsenobetaine

Arsenic is excreted rapidly:



*Water:* Maximum Canadian contaminant level 25 µg/L suggests < 50 µg/day

- “old” WHO guideline = North American standard/guideline = 50 µg/L
- “new” WHO guideline = 10 µg/L
- controversy in US over implementation of new standard because many DW sources (and many mine effluents) have  $10 < [As] < 50$  ppb.
- Removal of As to 10 ppb would be very expensive: what technology?? Refer back to Chapter 7: drinking water

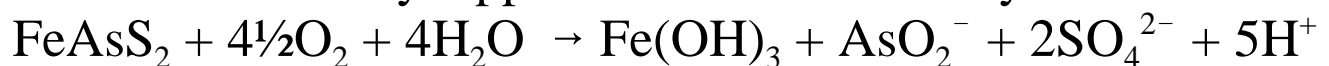
**Issues in many parts of Asia** (West Bengal, Bangladesh, Taiwan, Vietnam: *Chem. Eng. News*, December 6, 1999, pp. 128-131).

- 1970s: UNICEF program to provide new wells; success in reducing water-borne disease
- 1990s: thousands of cases of skin disorders (clinical sign of arsenicosis), fatigue/lethargy; cancers
- levels up to 1-5 mg/L; no good (cheap) methods for arsenic removal
- difficulty of analysis for As (no simple test)
  - occurs in water as arsenite (As(III) =  $\text{AsO}_2^-$ ) and arsenate (As(V) =  $\text{AsO}_4^{3-}$  and protonated species, like  $\text{PO}_4^{3-}$ )

### **Origin of arsenic in water**

- naturally occurring arsenopyrite ( $\text{FeAsS}_2$ ) is the usual source
- details of the solubilization chemistry are unclear and controversial

Oxidation chemistry: approximate stoichiometry



However,  $\text{Fe}(\text{OH})_3$  adsorbs inorganic arsenic quite strongly

Reduction chemistry, anaerobic conditions in sediments



Significance of this controversy (*New Scientist*, February 12, 2000, pp. 16-17):

- If oxidation chemistry predominates, dig the wells deeper
- If reduction chemistry predominates, deeper wells mean more anaerobic conditions

## Metabolites

Major metabolites from inorganic As in human urine:

- unchanged inorganic forms, 20-25%
  - monomethylated As,  $\text{CH}_3\text{AsO}(\text{OH})_2$ , MMA, 20%
  - dimethylarsinic acid  $(\text{CH}_3)_2\text{As}(\text{O})\text{OH}$ , DMA, 40-60%, also called cacodylic acid
- 
- For As compounds, methylation is a detoxification mechanism
- 
- Rat  $\text{LD}_{50}$ 's (*Canadian Chemical News*, Sept. 1999, p. 18)

$\text{AsO}_2^-$ (arsenite, As(III))	14 mg/kg
$\text{AsO}_4^{3-}$ (arsenate, As(V))	20
$\text{CH}_3\text{AsO}(\text{OH})_2$ (methylarsonic acid)	700-1800
$(\text{CH}_3)_2\text{AsO}(\text{OH})$ (dimethylarsinic acid)	700-2600
arsenocholine	6500
arsenobetaine	>10,000

## Uptake

- Efficient (95%) oral uptake of soluble inorganic As
- Low availability from insoluble salts (lead arsenate, arsenic sulfide), and from As in soil.
- Inorganic As: colourless, tasteless; a favourite potion for poisoners until the development of the “Marsh test” (1836) in which As compounds reduced to arsine ( $\text{AsH}_3$ ) which was decomposed in a capillary to (black) As metal. Length of black deposit  $\propto$  As concentration.

## Inhalation toxicology

- Dusts of  $\text{As}_2\text{O}_3$  are the commonest form in air
- Emphasis is on  $\text{As}_2\text{O}_3$  (metal smelters, since As minerals occur along with many metal ores, *e.g.*,  $\text{FeAsS}_2$ )
- Chronic animal studies and human epidemiological studies (workers at Chilean copper smelters, 1995)  $\rightarrow$  clear evidence of increased risk of lung cancer
- *Uptake*: Although exposure estimates have uncertainty, workers exposed to  $\text{As}_2\text{O}_3$  in smelters absorb 40-60% of the amount inhaled. Most (80%) of the  $\text{As}_2\text{O}_3$  deposited in the lung is then absorbed.
- Time weighted TLV =  $0.5 \text{ mg/m}^3$

### “Canadian content”

- Bankrupt Royal Oak Giant gold mine (Yellowknife): As sulfides  $\rightarrow$   $\text{As}_2\text{O}_3$ . About 200,000 tonnes  $\text{As}_2\text{O}_3$  presently buried in the mine + ~40,000 tonnes As in tailings ponds  
<http://www.carc.org/whatsnew/cleanup.htm>

## **Effects**

- Doses of 0.05-0.5 mg/kg/day for weeks or months —> GI upset. Symptoms disappear after exposure ceases
- Acute high doses → neurotoxin: encephalopathy; clinical signs confusion, impaired memory, emotional upset. Peripheral neuropathy from lower doses, numbness, pins and needles at first; later, loss of reflexes and weakness
- Dermal effects: hyperkeratosis, hyperpigmentation, warts and corns on the soles and palms which may lead to squamous cell carcinoma
- Long term oral exposure ~0.02 mg/kg/day in drinking water also —> skin cancer, also lung, bladder, liver. Not understood why pulmonary and oral cause cancer at different sites.

## **Metabolism and mechanism of action**

- Problem of finding suitable animal model for human toxicity
- Humans more susceptible to As intoxication than lab animals

## **Biomarkers for exposure**

- Analysis of hair and fingernails (Mee's lines = transverse white lines) allows estimation of past exposure, since fingernails grow ~0.3 cm/month. Important to avoid surface contamination
- Analysis of blood does not give useful data.
- No preferential accumulation in specific organs. Some deposition of As(V) in bone (analog of phosphate)

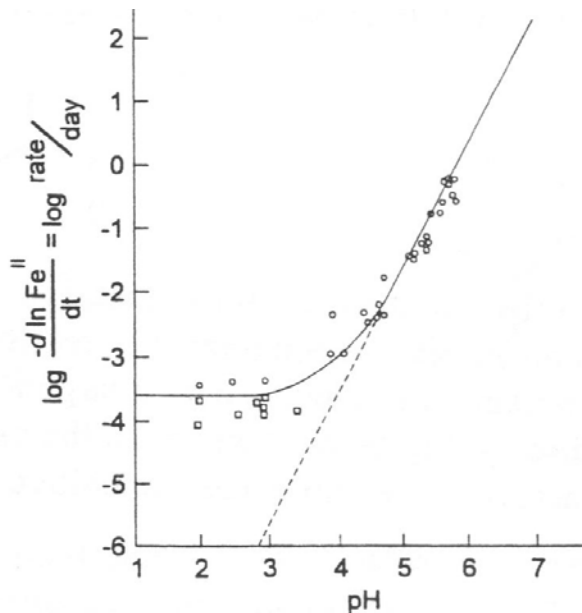
## Issue #4: Acid Mine Drainage (AMD)

- Major problem, Canada and elsewhere, (abandoned) mine sites – both metal ore mines and coal mines
- Biological origin of AMD – microbial oxidation of sulfides



- AMD problems are:
  - toxicity due to low pH
  - toxicity due to solubilization of metals Cu, Pb, Zn, Cd, Ni etc
  - deposition of  $\text{Fe}(\text{OH})_3$  downstream

$$-d[\text{Fe}^{2+}]/dt = k[\text{Fe}^{2+}][\text{OH}^-]^2p(\text{O}_2)$$



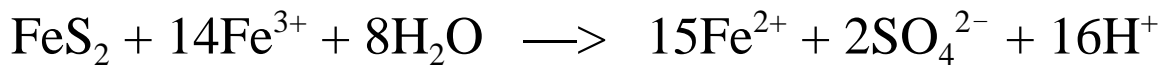
Oxidation of Fe(II) as a function of pH

## Dimensions of the AMD problem

- The Clark-Fork site, Montana (see text) 50,000 acres, contaminated with As, Cu, Cd, Pb, Zn from 100 years of mining
- Richmond Mine, Iron Mountain, California: in the sub-surface, extreme acidity pH to **-3.6!!!** and metal concentrations to **200g/L**,  $\text{SO}_4^{2-}$  to **760 g/L** (*Environ. Sci. Technol.*, 2000, **34**, 254) — the most acidic environmental water known

Proposed sequence of events:

- $\text{Fe}^{2+}$  and  $\text{Fe}^{3+}$  by conventional AMD formation and oxidation
- further reaction by exothermic process below



- heat released evaporates water and increases concentrations of all solutes

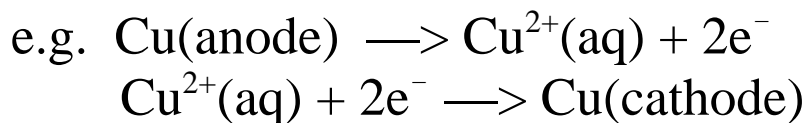
## AMD remediation

- Exclude air through:
  - submersion of tailings
  - cover the tailings with soil and grow grass
- Cathodic protection of the ore body using sacrificial Fe or Zn anode
- Remediate seepage via:
  - neutralization, usually with limestone: sludge handling problems, repeated treatment required
  - Neutralization: anoxic limestone drains (ALDs): how long will they last?
  - wetlands as filters: remove metals by precipitation and/or biomass incorporation; how long before they become choked?
  - phytoremediation using engineered wetlands: use of “hyperaccumulators”; what to do with the contaminated plants?
  - electrochemical technologies: finding suitable electrodes, current efficiencies; anode reaction?

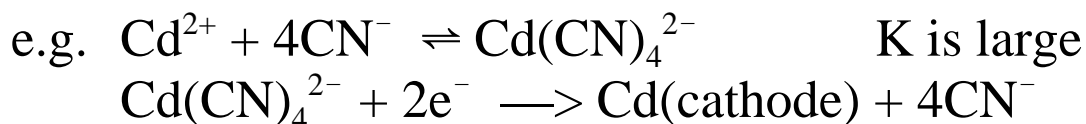


## Issue #5: Electroplating

- Few metals are deposited directly from acidic electroplating baths. Important examples: Ni and “bright” Cu, both of which are *electrorefined*



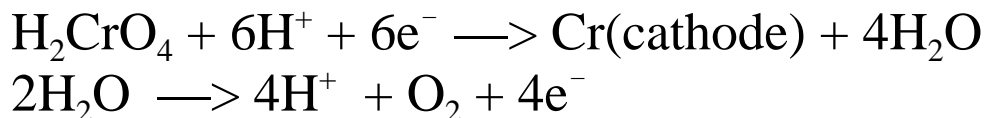
- Matte Cu, Cd, Ag, and Zn are plated from cyanide plating baths (typically 2 M  $\text{CN}^{-}$ ) because direct deposition gives an easily abraded surface. The metal serves as anode, as above. *Environmental issue: cyanide disposal*



- For Au, the bath is charged with  $\text{KAu}(\text{CN})_2$ . The cathode reaction is as above, and the anode reaction is oxidation of  $\text{OH}^{-}$ :



- For Cr, deposition occurs in acidic solution but using Cr(VI) as the source of chromium. The anode reaction is water oxidation. *Environmental issue: chromate carcinogenicity*



*The End*