

Summary of Feedback on Wild Rice / Sulfate Hypotheses

1A Wild rice is only negatively affected by elevated sulfate while it is actively growing (April through August).

1B From September through March sufficient sulfate diffuses into sediment, or adjacent soils, so that wild rice growth is harmed.

8A The negative effects of elevated sulfate do not persist into the wild rice growing season.

8B Sulfate does not have negative effects during some months, due to lower temperature or other reason.

3A Sulfide produced in the sediment is toxic to wild rice seeds, reducing germination.

3B Sulfide produced in the sediment is toxic to the roots of wild rice. Roots release O_2 to oxidize sulfide in the rhizosphere.

4A High-iron sediments reduce toxicity of sulfide (because iron precipitates sulfide as iron sulfide, reducing the toxicity). Dutch researchers have extensively investigated the role of sulfate reduction in wetlands and have documented that iron can reduce the toxicity of sulfide (van der Welle et al. 2006).

4B High-nitrate systems reduce sulfide toxicity (because sulfide is oxidized back to sulfate by nitrate). Research has shown that there are many microbial pathways for nitrate, including the oxidation of sulfide to sulfate (Haaijer et al. 2006, Burgin and Hamilton, 2008).

5A Relatively high nitrate availability inhibits sulfide production. Research in the Netherlands has demonstrated that nitrate can inhibit the production of sulfide in wetlands, even when sulfate concentrations are elevated (Lucassen et al. 2004).

5B Relatively high oxidized manganese in the sediment inhibits sulfide production. In principle, oxidized manganese (and oxidized iron) will be utilized as an electron acceptor before sulfate is respired (Kirk 2004).

5C Relatively high oxidized iron in the sediment inhibits sulfide production. In saturated soils, oxidized iron can be the dominant electron acceptor for respiration of organic matter (e.g., Frenzel et al. 1999).

6A Wild rice roots develop a barrier in reaction to sulfide, which inhibits uptake of essential nutrient(s) such as iron or nitrogen.

6B Sulfide precipitates essential nutrient metal(s) such as iron, copper, or zinc, making them less bioavailable (Kirk 2004).

Priority B hypotheses. These hypotheses potentially occur in the Minnesota environment, but are significantly less likely to be causative than A's.

2A Elevated calcium, magnesium, potassium, or sodium correlated with sulfate is the actual toxic agent.

5D Water movement carries away organic matter, limiting sulfide production.

7A Reduced water movement (e.g., via impoundment) increased organic matter, increasing sulfide production (e.g. residual wild rice straw).

6C Sulfide production enhances P bioavailability, increasing production of organic matter, which increases sulfide production (a positive feedback loop).

6D Sulfate, through sulfide precipitation of iron, increases P bioavailability, increasing growth of perennial plants that exclude wild rice through shading, root competition, and/or allelopathy.

7B Longer ice-free period, elevated temperatures have increased bacterial activity and sulfide. If sulfide production is limited by the temperature of surface sediment, recent increases in ambient temperature could be responsible for increased sulfide production.

7C External loading of phosphorus and/or nitrogen has increased production of organic matter, increasing sulfide production. If organic production has increased due to external nutrient loading, then sulfide production could have increased—independent of any change in sulfate loading.

Priority C hypotheses. These hypotheses concern potentially significant impacts of sulfate in the Minnesota environment, but they do not address the wild rice issue.

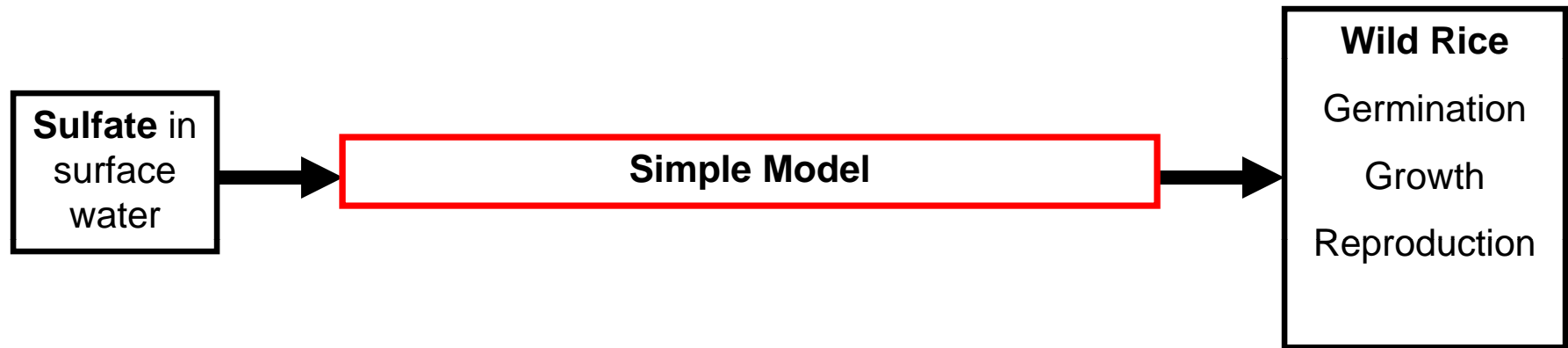
9A Increased sulfate enhances methylation of mercury.

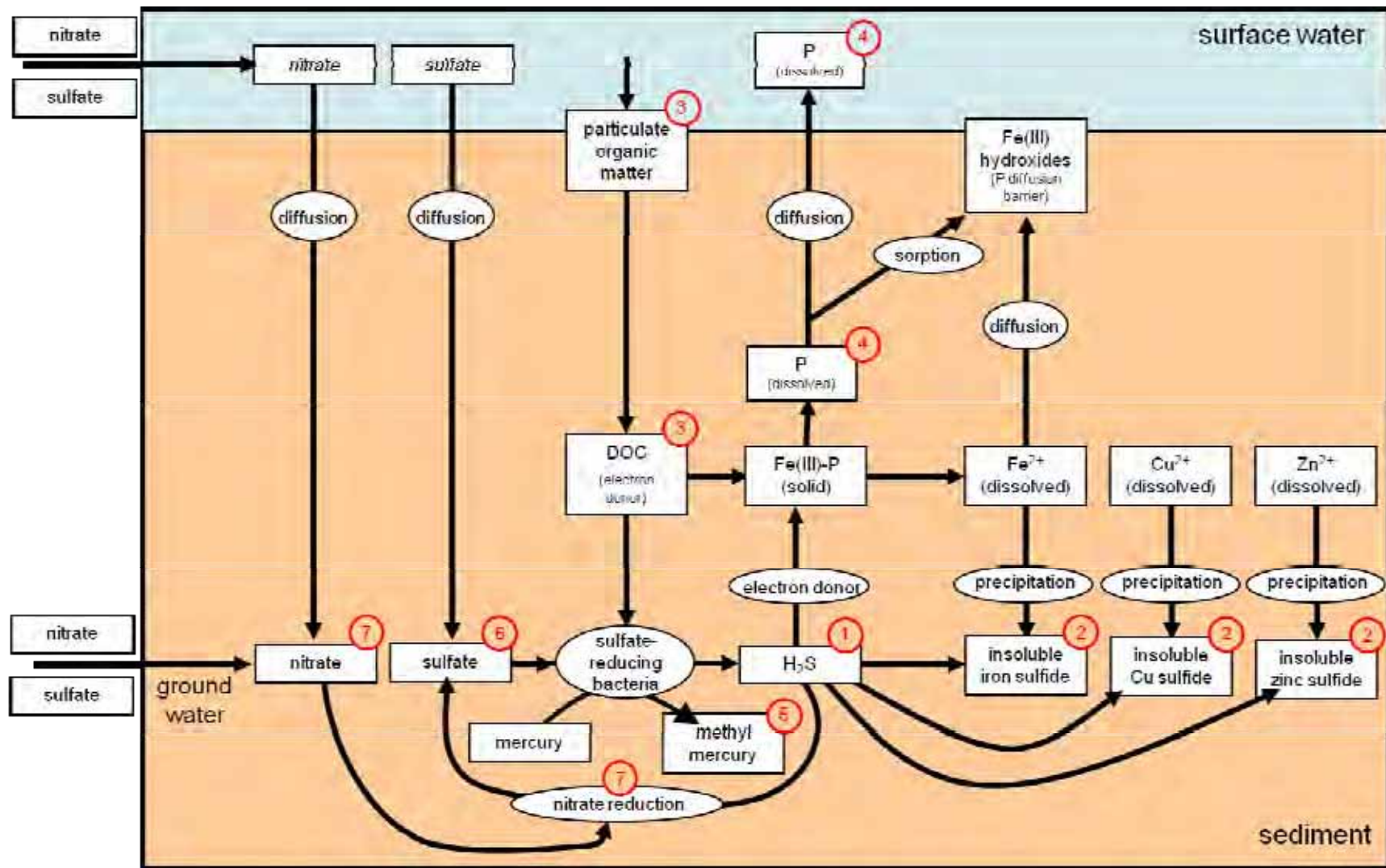
9B Very high sulfate concentrations inhibit methylation of mercury.

9C Factors that remove sulfide from pore water (hypotheses 4A and 4B) increase the production of methyl mercury.

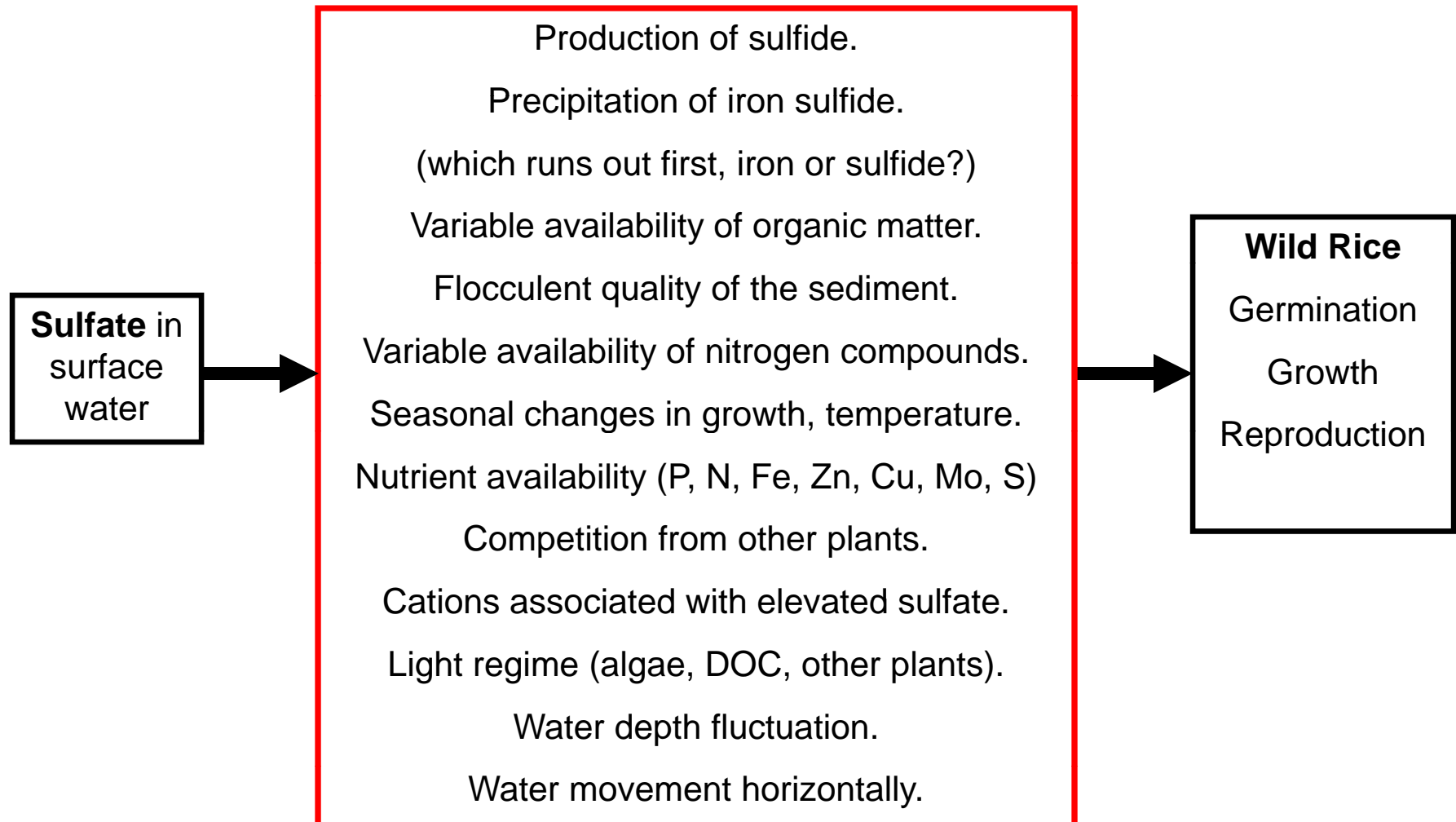
9D Factors that reduce the production of sulfide (hypotheses 5A and 5D) reduce the production of methyl mercury

**A model would be ideal...
to relate sulfate to wild rice**

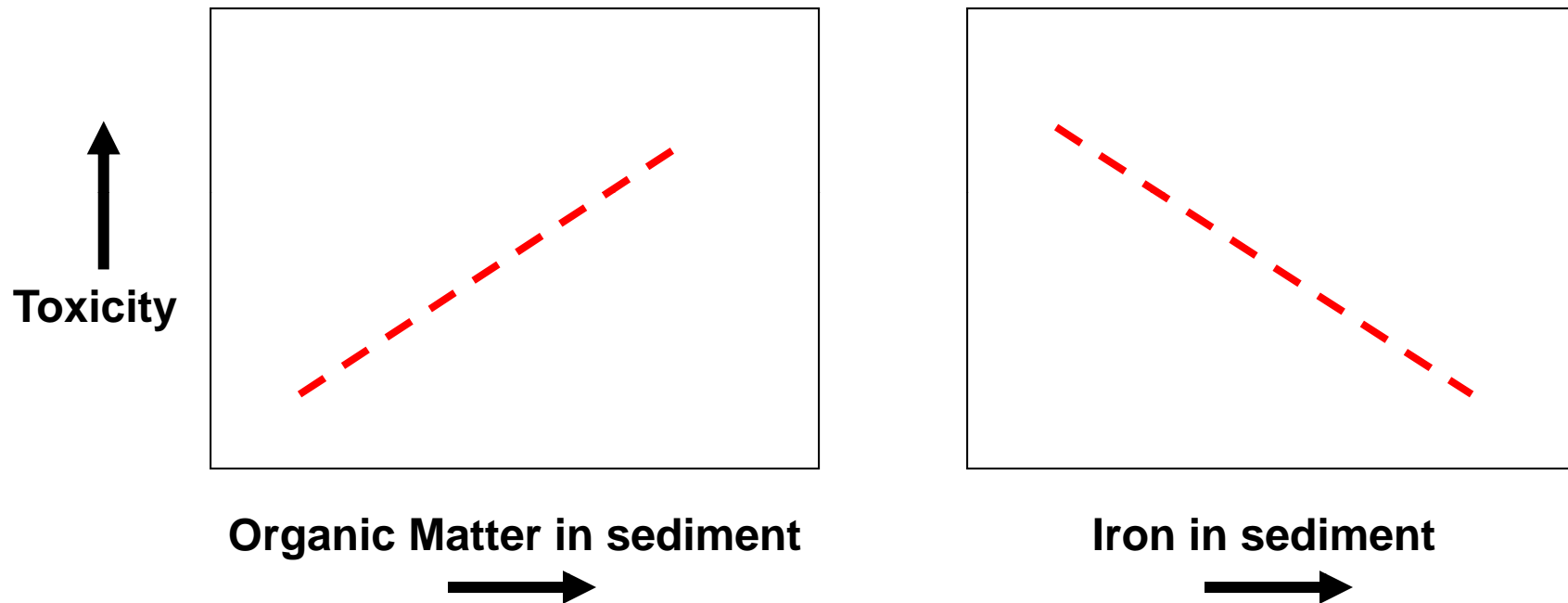




A model would be ideal... to relate sulfate to wild rice



The toxicity of a given concentration of sulfate could depend on environmental conditions, for instance:



Perhaps an empirical model would be adequate

- We don't need to understand all the mechanisms.
- But different background conditions may produce different results.
- So we need to identify the potential controlling variables for designing data collection and interpreting data:
 - Iron, manganese, copper, zinc
 - Organic Matter
 - Nitrogen....and so on.
- What is the most efficient design for this study?

Hypotheses are not the same as “proposed data collection”

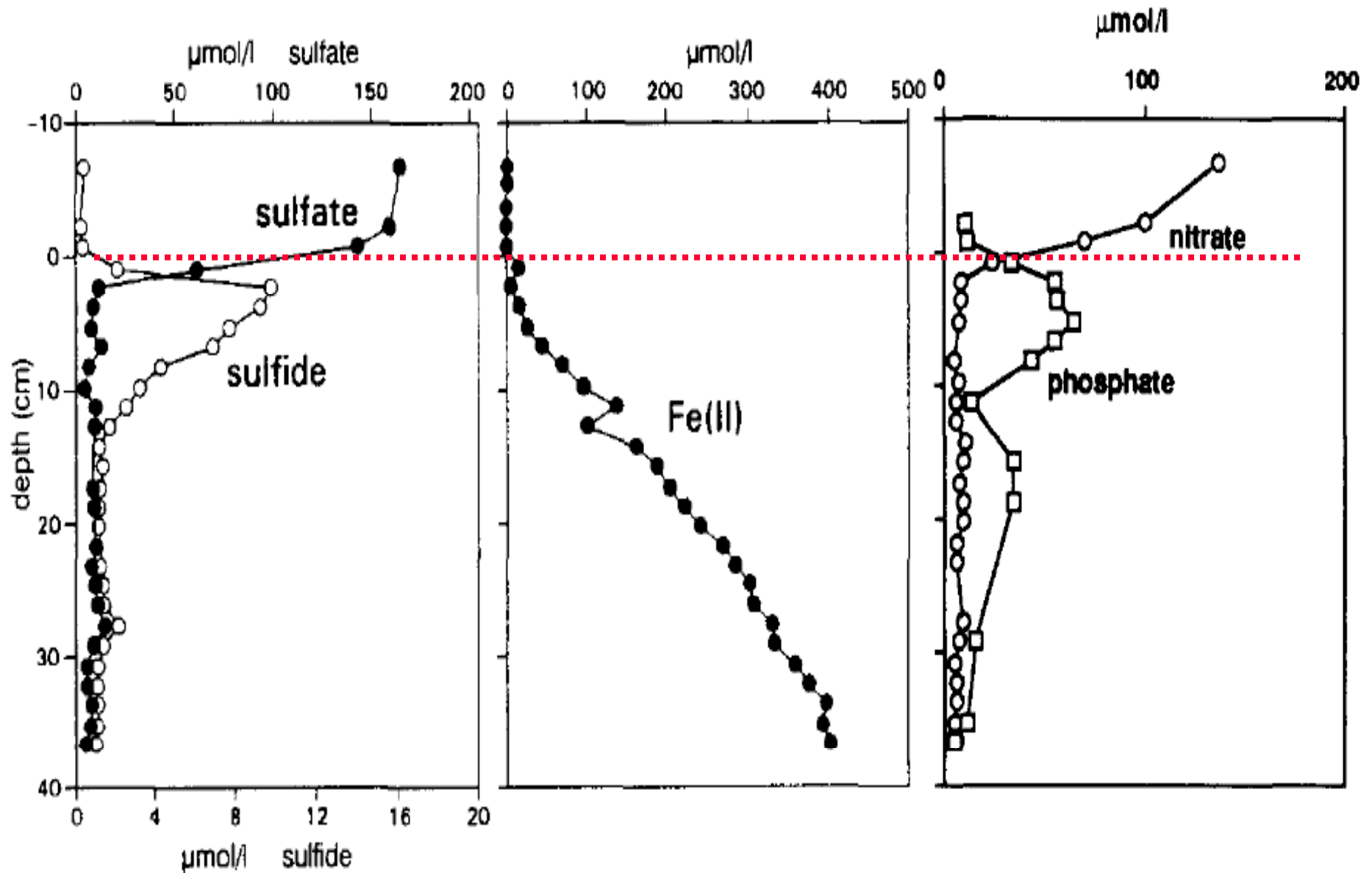
- What is the most efficient way to test as many high-priority hypotheses as possible?
- We probably have two field seasons (after a preliminary field survey in 2011).
- What should we do over those two field seasons?

One Idea

- A. Test tolerance to varying sulfate levels (hydroponic; keep sulfate as sulfate).
Germination through seed production. Could test multiple cations (Mg, Na, K, Ca).
- B. Parallel container studies with organic matter to produce reducing conditions. Vary: Fe, N sources, P, Mn, Zn, Cu (and organic matter).
- C. Repeat B in second year, reflecting lessons learned.
- D. In-situ mesocosm experiments to test findings of B and C.







**Bottom water & porewater chemistry of Lake Greifen
(Wersin, Hohener, Giovanoli, & Stumm 1991)**

Afternoon Breakout Session:

Discussion of a Straw Proposal