Water stress – induced stomatal limitation to photosynthesis

1. Stomatal resistance in the context of the entire CO2 pathway
2. How to measure stomatal resistance to CO2 uptake.

Stomatal limitation of photosynthesis

-Stomatal closure prevents CO2 from diffusing to sites of carboxylation.

-Soil, OR atmospheric water stress may cause stomata to close.

-Atmospheric humidity: some species show '**feed-forward**', some show "**feedback**" stomatal responses

-Soil moisture stress: a couple of mechanisms…

-Soil moisture stress:

-1. Can cause cavitation, inducing stomatal closure

2. Can reduce leaf water potential, inducing stomatal closure

We will stop at this level of detail for now, and come back to stomatal function and hydraulics later on. Let's get back to the link to photosynthesis now.

-If stomata close due to water stress, how does this limit photosynthesis?

-We need to consider the pathway of CO2 into the leaf and how stomata limit it when they close.

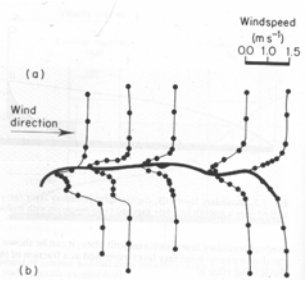
Four resistances to CO2 reaching sites of carboxylation: three gas phase and one liquid phase.

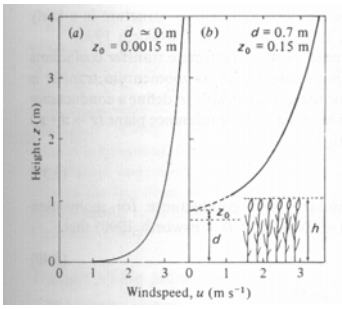
1. Leaf boundary layer (gas phase) – related to leaf size, shape, and wind speed.
2. Stomatal resistance (gas phase) – physical barrier to CO2 diffusion from outside to inside the leaf.
3. Inter-cellular resistance (gas phase) – tortuous gas phase path to mesophyll cells.
4. Mesophyll resistance – liquid CO2 diffusion to chlorplasts.

Boundary layers: nested, hierarchical, fractal(?) (leaf, branch, crown, canopy)

leaf

canopy





Very commonly, the stomatal resistance is much, much larger than any of the other three resistances.

Ballpark numbers:

May be 10-100X boundary layer resistance in tall trees.

Intercellular resistance – may only cause a drop of 5ppm CO2 (compare to perhaps 300 ppm inside guard cells.

Mesophyll resistance – typically much less than stomatal, due in large part to the high total surface area exposed to the gas phase. Some recent workers have questioned this though, especially in sclerephylous plants or trees…

How can we quantify the level of stomatal restriction to CO2 supply for Photosynthesis?

"Stomatal Resistance" – often labelled "r_s"

Photosynthesis directly depends on stomatal resistance:

$A = (ca – ci)/Pr_s$,

where A = photosynthesis (umol/m2s),

Ca is atmospheric CO2 level (partial pressure – 380ppm = 380 ubar)

Ci is intercellular partial pressure, and P is total atmospheric pressure (1 bar = 1atm = 0.1MPa)

We can easily measure A (gas exchange machine), P, and Ca, but can't directly measure Ci. So this presents a problem for determining rs. (= (ca-ci)/PA)

But we can use the intimate connection between water loss and carbon gain through stomata to get around this problem.

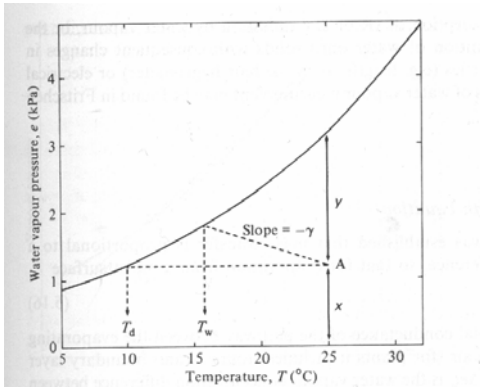
The crux of the solution:

-Exploit $E = (ea-ei)/Pr_s$ instead – the r_s for H2O is directly proportional to the r_s for CO2

Why shift to water vapor to solve for CO2 uptake?

- E, ea, P are as simple to measure as A, ca, P, and ei is much simpler to infer than ci.

We assume that the humidity is saturating (100% RH) inside the leaf, so that ei is simply a function of leaf temperature. If we know leaf (canopy) temperature, we simply compute ei, and solve for rs.



Clausius –clapeyron relationship

So now we can solve

$Ci = Ca - APr_s$

There is only 1 more little thing to consider:

Rs for CO2 is 1.6 times Rs for water.

Molecule per molecule, CO2 is intrinsically 1.6 times 'harder' to get into a leaf than H2O gets out.

We simply need to use this factor of 1.6.

Where does the 1.6 arise?

Graham's law of Diffusion:

Diffusion of a species is inversely proportional to the square root of its mass. Let's work through this…

1. What is the kinetic energy of a billiard ball?

2. Air has bigger/heavier billiard balls (CO2), smaller/lighter billiard balls (H20), but a property of gases is that all constituents have the same kinetic energy.

3. $½ mV^2(h2o) = ½ mV^2 (co2)$

4. $V_{CO2}/V_{H2O} = \text{sqrt}(m_{h2o}/m_{co2}) = \text{sqrt}(18 \text{ g/mol} / 38 \text{ g/mol}) = 1.5$

4. Why 1.5 and not 1.6??

Outline for today:

Environmental impacts on Photosynthesis

1. From h20 to co2 limitations on photosynthesis
2. temperature
3. Light
4. nutrients

1. From H_20 to CO_2 limitations on photosynthesis

To summarize from last time:

We figured out how to get stomatal resistance(conductance) to CO2 transfer by solving the more tractable stomatal conductance to water vapor (how more tractable?)

We can now compute how stomatal restrictions due to water stress limit photosynthesis:

$$A = (ca - ci)/Pr_{s,co2}$$

$A = (ca - ci)/Pr_{s,co2}$ tells us how stomata controls the supply of CO2 for photosynthesis, and ultimately how water limitations limit photosynthesis.

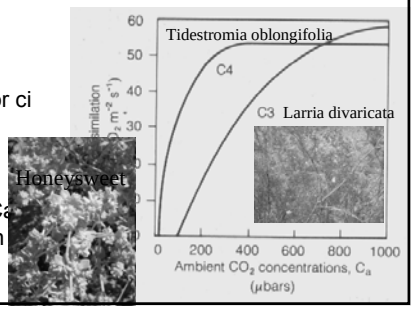
This equation also seems to close the book on how CO2 impacts photosynthesis. For example, with all else fixed, higher ca should mean higher A.

It is far from this simple –

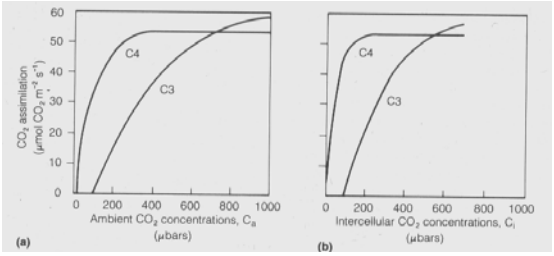
-A is not linear with Ca nor ci

-C3 may do better in high Ca world

-A saturates at levels of Ca that humans may reach in the next century!

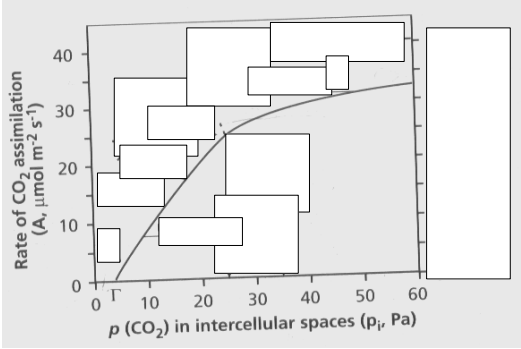


From the A-ca curve, and rs, we can easily generate the A-ci curve. This gives the intrinsic limitation of co2 supply on photosynthesis – a more fundamental biochemical relationship.

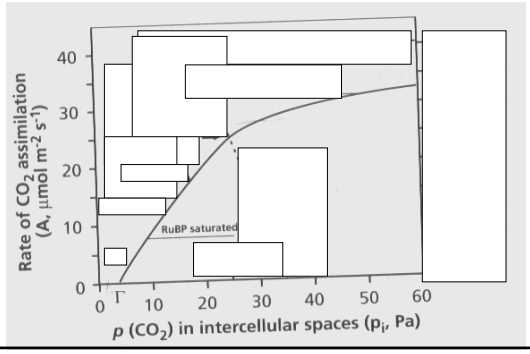


What gives rise to the shape of this curve?

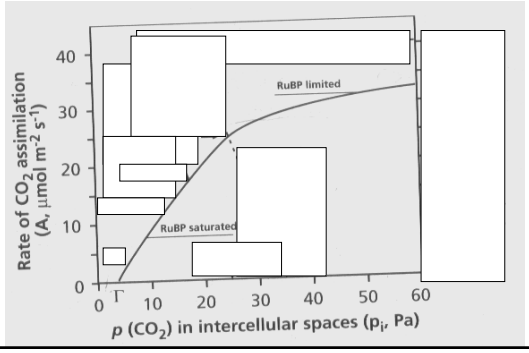
The A-ci curve: Not a single curve, but a composite curve representing two key processes.



A roughly 'linear phase' at lower ci: CO2 limited region (more RuBP around than CO2 – Calvin Cycle 'starved' of CO2). Nearly proportional impact of increased CO2 on A.

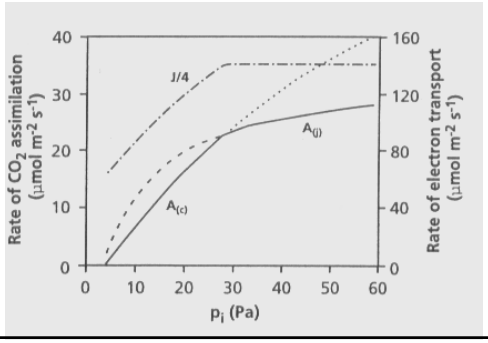


A saturating phase: limited by the regeneration of RuBP (plenty of CO2 around). RuBP limitation is in turn limited by ATP, NADPH, which are limited by electron transport in the light reactions.



The A-ci curve is the minimum of these two curves. (solid line below is minimum of two dashed lines)

1. Origin of the lower left-hand portion (labeled A(c))



1. Origin of the lower portion (labeled A(c))

Determined by reaction kinetics involving RuBP, CO2, Rubisco.

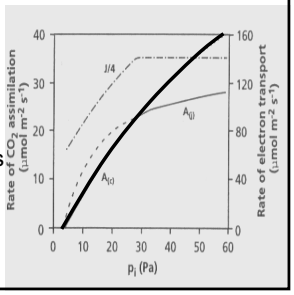
A© = A_{max} *c_i/(c_i + K), where K is a michaelis-menton constant

This is a hyperbolic equation

At high ci, (ci >> K), A© = Amax

At low ci, (ci < K), A© = Amax/K * ci

Next, let's see how the A(j) curve clips this hyperbole.



2. Origin of the upper portion (labeled A(c))

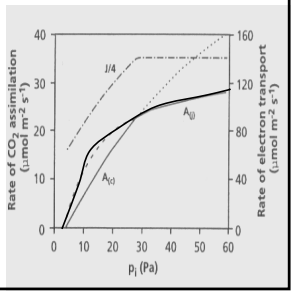
Determined by how fast the Calvin cycle can produce RuBP. This in turn depends on how fast light reactions can provide ATP and NADPH.

Light harvesting (and hence electron transport) saturates because of finite concentration and capacity of light harvesting complexes

A(j) = J(ci-Γ)/(4 [ci + 2Γ]) -respiration

where Γ is photo-respiration (O2 fixation competing with CO2)

This ends up having a similar form as that given by Michaelis-menton enzyme kinetics.



Why is the following figure plotted as J/4?

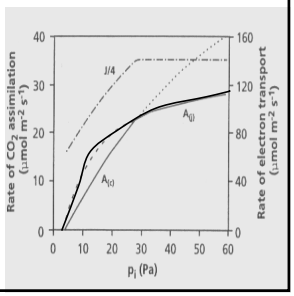
1. CO2 + H2O -> CH2O + O2 is the basic stoichiometry of photosynthesis
2. One molecule of O2 requires 4 electrons:

$2H_2O$ + light -> $4H^+ + O_2 + 4e^-$

3. Note J/4 maxes out at more than 4x the A(j) curve (here shown as ~140 vs. ~25 umol/m2s

This is due to the loss of electron transport due to photo and 'dark' respiration.

4. Why does A(j) continue to rise after J/4 flat-lines?



Taking stock:

- Stomata limit CO2 uptake, but there is also an independent, intrinsic capacity for the photosynthetic machinery to fix the Ci.

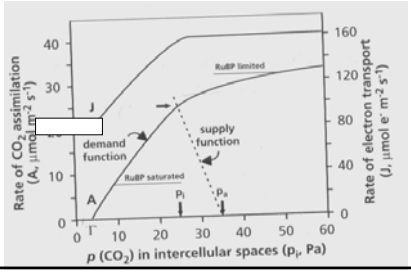
- Two processes shape the overall A-ci response:
  - CO2 limiting the CO2 + RuBP + Rubisco reaction
  - RuBP limiting the CO2 + RuBP + Rubisco reaction

The A-ci curve also allows us to consider how stomata and photochemistry both co-limit Photosynthesis. Let's see how…

The A-ci curve is also called the 'demand' function, because it describes the strength of the photo/biochemical sink for CO2.

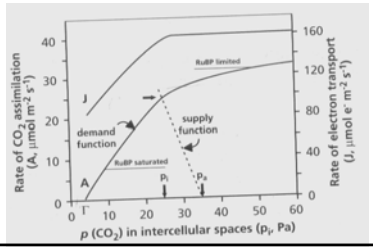
We can also consider a "supply" function, which is the rate at which stomata allow CO2 into the leaf. The slope of the supply function is: ?

$$g_s = 1/r_{sco2} = AP/(ca-ci)$$



Important point: Optimality of integrated leaf photosynthetic function.

Under a given A-ci curve (which can change depending on stress – Tezara), stomata tend to operate such that the intersection of the supply function with the demand function is at the point where the CO2 limitation and RuBP regeneration curves intersect – I.e. co-limit photosynthesis. Typically, this is at about ci/ca = 0.7. Neat!



Summary: CO2 impacts on Photosynthesis:

Stomata and biochemistry both directly limit – and usually co-limit photosynthetic fixation of CO2.

There are important indirect impacts of CO2 on photosynthesis that we have not yet considered – e.g. stomata may have a direct closing response to Ca!

The general shape of A-ca and A-ci curves tell us that vegetation on earth is approaching a limit to how it can make use of elevated CO2.

Will C3 plants outcompete C4 plants? A-ci curves tend to support this idea.

Outline for today:

Environmental impacts on Photosynthesis

1. From h20 to co2 limitations on photosynthesis
2. **temperature**
3. Light
4. nutrients

Temperature:

1. Photosynthesis shows optimal temperature ranges that differ by species and even population.

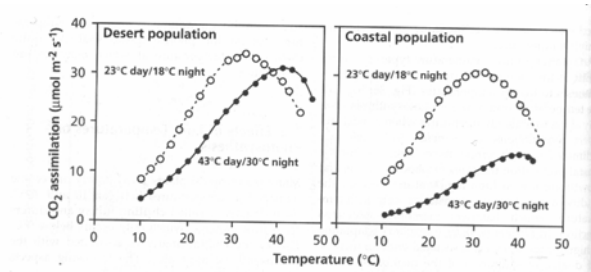


FIGURE 36. Photosynthesis in response to temperature of two populations of the C_4 species *Atriplex lentiformis* (Pear 1977, as cited in Berry & Raison 1981).

What causes this?

1. The thermodynamic behavior of enzymes (Rubisco): less active at low temperatures, more active at higher temperatures. At super-optimal temperatures, Rubisco shows greater intrinsic affinity for O2 than CO2.

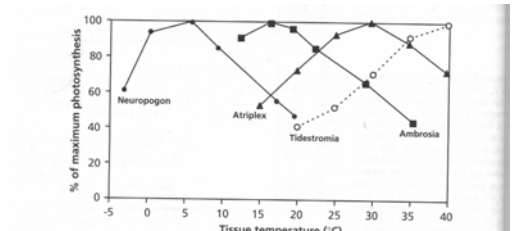
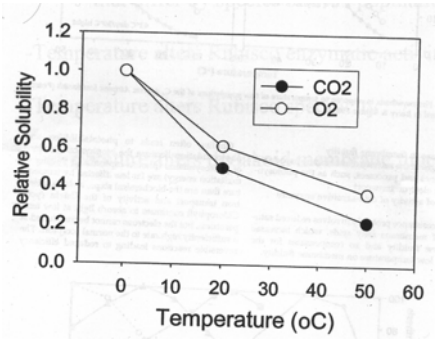


FIGURE 37. Photosynthetic response to temperature in plants from contrasting temperature regimes. Curves from left to right are for *Neuropogon acromelanus*, an antarctic lichen, *Ambrosia chamissonis*, a cool coastal dune plant, *Atriplex hymenelytra*, an evergreen desert shrub, and *Tidestromia oblongifolia*, a summer-active desert perennial (Mooney 1986). Copyright Blackwell Science Ltd.

2. The solubility of CO2 declines faster than the solubility of O2 as temperature warms – so Rubisco fixes O2 relatively more than CO2.



3. Temperature impacts thylakoid membrane function:

- At high temperatures, membranes become too fluid, membrane-associated function is affected (e.g anchoring of membrane bound proteins)

- At low temperatures, membranes become too rigid, embedded proteins lose function.

- Membrane fluidity and temperature sensitivity depends on the degree to which the lipid tails are saturated vs. unsaturated with hydrogens. Like olive oil vs. butter. Membrane degree of saturation differs by species, and can differ within a species or even individual (chilling resistance).

4. The rate of dark respiration (which we have not yet talked about!) – $CH_2O + O_2 \rightarrow CO_2 + H_2O$ – increases dramatically and non-linearly with temperature.

So Net photosynthesis, in addition to Gross Photosynthesis declines at high temperatures.

Temperature and photosynthesis: summary

1. Rubisco activity changes with temperature
2. O2 and CO2 solubility change with temperature, and so does photorespiration.
3. Membrane-associated function changes with temperature
4. Dark respiration changes with temperature

The amount of photosynthate consumed in respiration varies with tissue type and with environmental conditions.

When nutrients are limiting, respiration rates in roots increase dramatically.

Item	Utilization of photosynthates % of C fixed: Free nutrient availability	Limiting nutrient supply
Shoot growth	40*–57	15–27*
Root growth	17–18*	33*–35
Shoot respiration	17–24*	19–20*
Root respiration	8–19*	38*–52
• growth	3.5–4.6*	6*–9
• maintenance	0.6–2.6*	?
• ion acquisition	4–13*	?
Volatile losses	0–8	0–8
Exudation	<5	<23
N_2-fixation	negligible	5–24
Mycorrhiza	negligible	7–20

Source: Van der Werf et al. 1994.
*, inherently slow-growing species; ?, no information for nutrient-limited conditions.

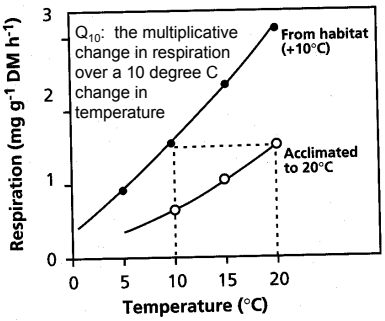


FIGURE 17. Temperature response of respiration of *Vaccinium myrtillus* (bilberry) shoots of populations acclimated to 10°C and 20°C. The dashed line shows the respiration rate of each plant at its acclimation temperature (Körner & Larcher 1988). Copyright The Company of Biologists.

Mitochondrial Respiration (like photorespiration) increases rapidly with temperature. Can this lead to reduced growth at high temperatures?

Maybe, but most likely only in extreme cases. Respiration "generally" acclimates to changes in temperature.

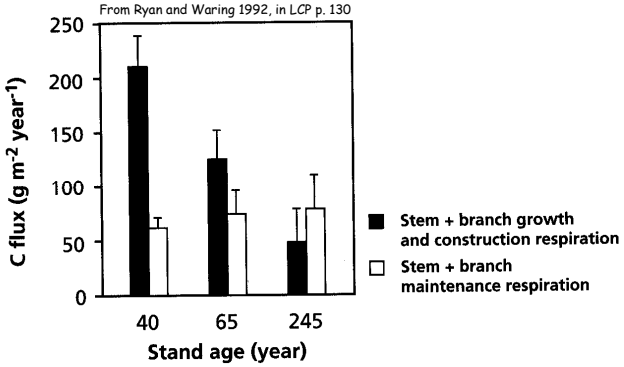
Respiration is often subdivided into Growth, Maintenance and Transport costs

Growth respiration: (a.k.a. "construction respiration") – a "fixed cost" that depends on the tissues or biochemicals that are synthesized. Often described in terms of "glucose equivalents"

Maintenance respiration: The cost of maintaining existing tissues and functions, (Protein turnover is the largest cost of maintenance respiration)

Do high maintenance "costs" reduce growth of large trees?

From Ryan and Waring 1992, in LCP p. 130



Evidence appears unlikely, even though most textbooks cite respiration as the cause of growth decline in trees/forests