<u>How Cells Really Work: The Ling Hypothesis</u>

<u>by Eric Armstrong</u>

Summary

When it comes to explaining how cell walls really work and how transfats cause disease the Ling Hypothesis has a lot going for it.

How do cells really work? In particular, how do oxygen and nutrients get past the cell walls and into the cell, where they can do some good. Surprisingly enough, science isn't really sure! The best science has been able to do, to date, is to put forth some educated guesses.

We know that the work of transporting nutrients through cell walls is performed by *phospholipids.* Biochemistry can explain that much. The first section summarizes the science. The question is how they arrange themselves to do what they do. The standard guess is known as the *BiLayer Theory*. The next two sections examine that theory briefly. The most recent and seemingly better guess is the *Ling Hypothesis*. The final section shows how that works.

Phospholipids

A *phospholipid* consists of a unsaturated fatty acid wedged between two saturated fatty acids, all joined to a phosphate molecule. (The word "phospholipid" means, "phosphate + fat (lipid)". Ain't science wonderful?)

Here is a diagram of a *phospholipid*:

```
   +---------
____|__  ____
   | \_/
   +---------
```

The two dashed lines at the top and bottom are the saturated fats. The one in the middle with the "kink" in it is an unsaturated fat. That little kink is where a hydrogen molecule s is missing, which causes the fat molecule to "bend" at that location.

At the left end is phosphate molecule. That's the "anchor" that the three lipids connect to. Like the saturated fats, which are inert, this part of the phospholipid is merely structural. It's the unsaturated fatty acid in the middle which does the real work.

The unsaturated fatty acid is the "chemically active" part, because the missing hydrogen means there is an unbound electron, so it just loves to lock on to a protein swinging by that is missing one. (With polyunsaturated fatty acids, several free electrons are present, so you actually get a little "electron cloud".)

Note:
Johanna Budwig addresses the quantum mechanics of fatty acids and proteins in her nearly unreadable little booklet, [*Flax Oil as a True Aid*](). In it, she points out that proteins are like the negative pole of a battery, while fatty acids are the positive pole. The free electrons in that tiny little "battery" are, in essence, the energy of life.

The free electron(s) in the phospholipid are responsible for chemical interactions which cause nutrients and even oxygen to be transported into the cell. It is here that insulin and other hormones bind with the cell in order to do their work.

The BiLayer Theory

According to the BiLayer Theory, there are two layers of phospholipids arranged into a wall. One layer faces out, ready to attach to nutrients and such, while the other faces inward.

Here's a diagram of that arrangement:

```
|>|        |>|        |>|        |>|        |>|
-+-        -+-        -+-        -+-        -+-
-+----+----+----+----+--...
-+-        -+-        -+-        -+-        -+-
|>|  |>|  |>|  |>|  |>|
```

Here, the other layer is facing up, and the inner layer is facing down. (In actuality, the phospholipids are crammed together. But the diagram shows them far apart so you can distinguish them from each other.)

According to the BiLayer Theory, nutrients that bind to phospholipids in the outer layer are somehow transferred to the inner layer. And from there, they move into the cell. That all sounds good when you read it quickly. But when you think it for a bit, several troubling questions arise:

- How did tiny little structures consisting of a few individual molecules ever decide to arrange themselves in this particular way? What keeps them doing so now, and keep doing so? (Since they have no volition of their own, you're pretty much forced to accept the proposition that God is directing all of the itty bitty molecules, one at a time, to do the right thing and act together).
- Biochemistry can explain how a nutrient binds to an outer phospholipid. But there is no explanation for how it migrates to the inner layer. One has to suppose that the outer phospholipid "switches places" with an inner one--real quick like, so the cell wall isn't broken.
- The nutrient is now bound to the inner phospholipid. How gets from there to the interior of the cell is unexplained. Does it suddenly break free and it's on its own? Doubtful. It defies basic biochemistry (at least, as far as I understand it, which is not a long trip by any stretch of the imagination. No need to pack a lunch.)
- If the nutrient needs to be escorted to the interior of the cell, then the inner phospholipid must suddenly "break free" to do so. Where then, does the

phospholipid come from to take its place? And how does that interchange occur without destabilizing the cell wall?

There is an alternative explanation that answers those questions rather well. It's called the *Ling Hypothesis*, after the scientist who developed it.

The Ling Hypothesis

According to the Ling Hypothesis, phospholipids form a sort of "cloud" at the outer edge of the cell. At the edge, they're very dense, crowding together to form a wall. Further towards the interior of the cell, they're less dense--so they're more fluid, rather than being held in place by their neighbors.

Here is a diagram that attempts to show that arrangement, where the outside of the cell wall is up, and each dot is a phospholipid:

The Ling Hypothesis has a lot of explanatory power. Occam's Razor says it's a better hypothesis to use, because it's requires fewer assumptions than the phospholipid BiLayer theory, and it explains more. For example:

- It explains where those extra phospholipids come from to shore up the cell wall when a nutrient comes in. The extras are right there all the time, milling around--trying, in effect, to get to the wall.
- It allows for one of two explanations for how the nutrient gets to the interior of the cell. It might be passed from one phospholipid to the next, in a kind of stochastic bucket brigade (which would be possible, given that there are so many of them, and which might even be logical, if the chemistry makes them prefer to face outward). On the other hand, the phospholipid might usher the nutrient to its final destination (probably by milling around until the nutrient is taken off its hands). That explanation, too, is accommodated by the Ling Hypothesis, without having to "break the cell wall", as would be required by the BiLayer Theory.
- It explains how a phospholipid can turn around or move inwards to transfer the nutrient it's bound to, because the phospholipid isn't "locked in place", the way the BiLayer Theory says it is.
- It explains why there isn’t a tiny hold in the cell wall for a short period of time, when the phospholipid turns or leaves. In the Ling Hypothesis, there *is* a small gap (which makes sense), but any substance that moves into the gap is met by another phospholipid in the cloud/crowd. It is then either recognized as a nutrient and bound, or if it isn't, it's blocked and eventually pushed out by the combined force of all the phospholipids behind it who are agitating to get to the outer layer.

Finally, while much work remains to be done, the Ling Hypothesis holds out the prospect for a sensible explanation for why cancers and other diseases caused by transfats occur seemingly at random--statistically, rather than uniformly.

If there were a single cell wall, for example, as suggested by the BiLayer theory then some cell would *immediately* be impaired when a transfat built into the cell will allow an allergen or carcinogen to penetrate. Under such a "clockwork" theory, one would expect the adverse effects of transfats to show up uniformly, in the same way that cyanide works uniformly on whomever is unfortunate enough to be exposed.

But we know that ingesting transfats is more a game of roulette. Like smoking cigarettes, diseases take a long time to develop. So long, in fact, that something else might kill you first, so their destructive effect shows up statistically, but not as a one-to-one cause-and-effect relationship that is immediately apparent.

The Ling Hypothesis provides an explanation for that statistical effect. If there are millions of phospholipids, then small levels of transfats would produce diseases only in very rare cases, larger levels more frequently, and so on.

Summary

All in all, the Ling Hypothesis of cellular function has a lot going for it. After examination, one is forced to conclude that it explains a lot more about the structure and operation of phospholipids than the BiLayer Theory.