Scientific Support for Chapter 3

How to Determine the NNT

The number needed to treat (NNT) is equal to the reciprocal of the absolute risk. The reciprocal is the amount obtained when dividing the number one by another quantity. In an experiment, the reciprocal is obtained by dividing the number "1" by the difference in effectiveness. In the case of statins, the NNT is 100, obtained by dividing the number "1" by 1%, i.e. 1/.01 = 100.

What is a "p-value?"

Statistics is mathematical and therefore tends to be extremely detailed and difficult. Fortunately, the essential concepts describing clinical trials and experiments are relatively simple:

1. The first value looked at by physicians and others (and mistakenly too often assumed to be the only important value in a clinical trial) is the "p-value " or 1 – (p-value), meaning the probability (p) that a particular intervention's experimental result occurred by chance alone, i.e., **the drug doesn't really work but appears to work**.

If the p-value were set at a 95% confidence level, then it would mean that if 100 trials were done by a different experimenter, 95 of them would include the same variance between the means (averages) in each trial as those achieved by the original experimenter. You could expect the same type of results 95 out of 100 times—**5% of the time you'd accept results implying the intervention worked when it didn't work**. That's the price paid for a 95% confidence level. If you increased the confidence level to 99% (by increasing sample size and/or the required number

of successes in the intervention arm), the accuracy is greatly increased but the cost of the trial would likely go up too. (That is, unless the intervention worked extremely well, such as in the IOWA experiment, discussed later in this Scientific Support.)

When the study or experiment is repeated many times using the same general group of people, this same 5% "**successful result**" will occur **when the experiment is actually a failure**, but, again, it is entirely due to chance alone. *The item of interest (drug or nutraceutical) may not have worked at all*, but we may be led to think that it did work based on the false positives. **That's why the more studies performed = the greater likelihood of false results being accepted as true**.

Never forget, the experimental **failures** are much more telling than "successes." It is not merely judicial "preponderance of the evidence."

2. Typically, the p-value is set to 0.95 (at a 95% confidence level you get an inherent 5% possible error rate allowed) for the medical intervention to be considered "statistically significant." If p = 0.95 then the study would be termed a 95% confidence level study (although a bit more information is still required). A 0.99 (1% error rate) or 0.995, p-value (0.5% error rate) would be even better because there would be much less of a random chance effect behaving as though the drug worked when it really didn't, thereby fooling both the physician and patient. Never forget that with p=95%, even if the drug didn't work, there is still a 5% chance that you would get these pseudo-positive results 5% of the time, making it *appear like the drug did work*.

Never forget: This 5% error rate means 1 out of 20 times you can be FOOLED into thinking FAILURE is SUCCESS.

This means that IF 15,000 "studies" of fish oil showed success (which isn't true), 750 of the studies that actually failed would be wrongly deemed successes—*fooling both physicians along with their patients*. Numerous fish oil studies do fail and we are hearing more and more about them, but due to the huge number of continual studies, many supposed "successes" are true failures. Physicians need to understand this fact.

Once again, a 95% p-value means that if this experiment were carried out in the same patient population sample 100 separate times, every time showing the drug being tested didn't work, then this same result would be included at least 95% of the time; however, a *false-positive result* **would occur entirely randomly 5% of the time,** *although the drug was actually a complete FAILURE***—*5 of those failed trials would appear to be successes*.**

It's easy to mislead those who don't understand statistics—almost everyone. All a company has to do is to conduct many studies and then purposely select only those that randomly show a "positive" result. Don't mention the failures, and, presto, you have a "successful" drug! All you need is lots and lots of money.

The p-value is NOT a measure of the size or magnitude of the effect of the drug. That is a completely different issue and has to do with the means (difference of the averages between both groups). Many physicians and patients don't understand this critical fact and mistakenly think that a p-value alone is all that is needed. Wrong, wrong, wrong—it is only part of the picture.

It is true that the MINIMUM p-value should be at least 95%; however, even IF the study has a "significant" effect with the intervention, then one must ask this next critical question:

How Strong is the Effect? A Little, or a Lot?

You need to ask "*What is the magnitude of the positive effect*?" A positive effect can range from a very small negligible effect to a tremendous effect. It may work on everyone, but with very little positive effect.

What is considered a significant amount or a significant effect?

If more than 51% (the majority) of a group doesn't respond **in absolute numbers** (NOT relative measures discounting sample size) to the drug, then I am not impressed, and you shouldn't be, either. **If something works, it should work on nearly every patient**—the majority being, as a minimum, greater than 51% of all patients.

Typically today, if just 20% of the treated group—the degree considered "clinically effective"—obtains any positive effect (regardless of how little that effect), it is considered a huge success. **This really means 80% FAILURE**.

Is the Item Measured Significant, or a Worthless "Surrogate" (Association)?

Low NNT is a necessary, but not an entirely sufficient condition, to be able to claim victory. **Is there a DIRECT cause/effect relationship**? This is absolutely required because if it isn't, you are being misled. Stains decrease low-density cholesterol

(LDL-C) with an NNT =1: a superb job. However, this doesn't significantly translate into stopping and reversing CVD.

A Worthless Surrogate—NOT the Specific Desired Result—is Often Used

Even though statins lower LDL-cholesterol, CVD is not significantly reduced.

The tragic truth was only recently accepted in **2012**. This still hasn't stopped the pharmaceutical companies and physicians who rely on those drugs from saying that lowered LDL cholesterol is all that counts in preventing cardiovascular disease. This has been proven incorrect, and patients are paying for this mistake with their health.

Therefore, one **cannot** assume the "disease" is solved when a worthless "surrogate" (association) is used **instead** of measuring the result itself, such as *how many heart attacks occur with and without statins* (the answer is nearly the same amount). This means that statins are ineffective at stopping heart disease.

A recent example: The JUPITER (Justification for the Use of Statins in Primary Prevention) Failure Hailed as a Success

Of course, from the above, it goes without saying that there must first be a direct cause/effect relationship to the disease. If you treat 100 patients with a drug and all 100 improve, the drug's number needed to treat (NNT) is 1 (100 patients/100 successes). If you treat 100 patients and only 1 patient responds positively, the NNT would be 100 (100 patients treated/1 positive response). This is an awful result— a 99% failure rate.

The 2008 JUPITER study obfuscated the fact that numerous attempts had been made to prove the "cholesterol theory" (the lower the patient's LDL-C, the greater the prevention of CVD), by attempting to make the case that the real mode of action of statin drugs was C-reactive protein (CRP) reduction from the statin. However, there is one tragic flaw in this argument: CRP—the protein that shows up in elevated levels in response to inflammation—is not a reliable prognostic indicator of cardiovascular events; there are better markers. An article entitled *Largest-Ever Meta-Analysis Finds CRP Is Unlikely to Be Causal for CVD* reports that scientists of the Cambridge-based Emerging Risk Factors Collaboration (ERFC) found:

> "[A]lthough CRP concentration was linearly associated with CHD (coronary heart disease), stroke, and vascular mortality, as well as nonvascular mortality, statistical adjustment for conventional cardiovascular risk factors resulted in considerable weakening of associations."

An Example of Misleading Statistics

In the JUPITER Study, **the NNT was 240 for statins[1] in preventing any stroke.** ***This is a 99.58% failure rate.*** **The "relative risk" statistics were used instead and disguised as a hazard ratio—essentially a time-valued relative risk—of 0.52 (52%)); p-value was 0.002). The NNT in this study was not stated**.

This means that the JUPITER Study had an **undisclosed** NNT of 240 (**99.6% FAILURE**) for preventing any stroke— instead, a

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1 Peskin, Brian Scott, "The Failure of Statins: A New Physiologic Solution to Cardiovascular Disease, Medical Therapeutics," *American Academy of Anti-Aging Medicine*, 2010, chapter 230, pages 259–273.

hazard ratio of 0.52 (appearing as a 52% success) was published, thus **making the trial appear immensely more successful than it actually was**.

What *appears* more impressive—a 0.4 success rate (**99.6% FAILURE** rate) or a 52% success rate (48% FAILURE rate)? Physicians are deceived along with their patients.

An Example of Modern-Day Low NNTs and High Effectiveness: IOWA Experiment

(*See* brianpeskin.com/BP.com/experiments IOWA-Experiment-Results.pdf (for entire screening information.)

There is a non-interventional way to screen subjects for arterial flexibility. It is called photoplethysmography with digital pulse analysis. This particular experiment was called the IOWA experiment—**I**nvestigating **O**ils **W**ith respect to **A**rterial health. The details will be described later but here were the results so you can see how both NNTs and p-values can be low, a high degree of significant effectiveness.

Long-term Use in Subjects with PEO Formulation Screened with Photoplethysmography

Significant differences (p-value=0.0015) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 8.8 years lower than their actual physical age.

Notice two points: People taking PEOs long-term had arterial flexibility 8.8 years lower—a younger cardiovascular "biological

age" than expected. Of the 34 subjects who were screened, 25 subjects improved. This is a very significant effectiveness measured either by absolute or relative measures.

> On average, the "biological age" difference was significant, almost a decade!
>
> The probability that this was a random chance occurrence was < 0.0015. You can take this result "to the bank."
>
> Note: The typical clinical study uses a 5% cutoff. This is 30 times more confident!
>
> The overall effectiveness was that 73% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 1.4.

Short-term Improvement in Subjects with PEO Formulation Screened with Photoplethysmography

> **Significant differences (p-value=0.0099) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 7.2 years lower than their actual physical age.**

> On average, the "biological age" difference was significant—more than seven years "biologically younger" than expected.

The probability that this was a random chance occurrence was < 0.0099. You can take this result "to the bank." Note: This is 5 times more confident than a 5% cutoff!

The overall effectiveness in the short-term was that 44% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 2.3. Of 16 subjects, 7 subjects rapidly improved. I like to see 80+% improvement effectiveness in screening for interventions but the timeframe was short to impact the cardiovascular system so significantly—less than a year.

PEOs Versus Fish Oil Subjects Who Discontinued Fish Oil Supplementation, Replacing it with PEO Formulation Screened with Photoplethysmography

Significant differences (p-value=0.0001) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 11.1 years lower than their actual physical age.

On average, the "biological age" difference was significant: more than 11 years "biologically younger" than expected.

The probability that this was a random chance occurrence was < 0.0001. You can take this result "to the bank." Note: This is 500 times more confident than a 5% cutoff!

> The overall effectiveness was that 87% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 1.2. Of 15 subjects, 13 subjects improved. This translates to a 87% effectiveness—in just 3.5 months with PEO use (on average). Because this effectiveness is about double the screening for just PEOs alone—44%—the conclusion is that **SIMPLY STOPPING FISH OIL gave nearly everyone a 4-year improvement in increased arterial health**!

These general PEO results and PEOs versus fish oil results—in screening for arterial flexibility—are incredible and predictable, as you will soon discover in later chapters.

Always ask for the SAMPLE SIZE, since without it you cannot draw any meaningful conclusions.

Always ask for the ABSOLUTE RISK DIFFERENCE BETWEEN BOTH GROUPS (NNTs), since without it you cannot draw any meaningful conclusions.

Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction

Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction, Hannia Campos, H., Ana Baylin, A., and Walter C. Willett, W.C., "Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction," Circulation, 2008; 118:339-345.

- "Greater alpha-linolenic acid [**parent omega-3**] … associated with **lower risk of myocardial infarction.**
- "Similarly, **low intakes of alpha-linolenic acid** can be found in developing countries where **cardiovascular disease is on the rise**.
- "**Fish intake was similar in cases and controls**, and the *variation within each group was large*….
- **Fish** or eicosapentaenoic acid [**EPA**] and docosahexaenoic acid [**DHA**] intake at the levels found in this population **did not modify the observed association**."
- "***Conclusions***—Consumption of vegetable oils rich in alpha-linolenic acid [**parent omega-3**] could confer **important cardiovascular protection**. The apparent protective effect of alpha-linolenic acid is **most evident among subjects with low intakes**.
- "In summary, consumption of vegetable oils rich in alpha-linolenic acid [***parent omega-3***] could confer ***important cardiovascular protection***."

[Important Note: This result is independent of the level of fish consumption. Given all of fish oils supposed miraculous claims, didn't these researchers wonder why? However, the researchers understand that the parent omega-3 did something the derivatives didn't do.]