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Subject : You Have Failed to Prove That Sodium Is Culpable for Hypertension

Karen,

Thank you for your detailed and informative response to my previous letter on salt. I appreciate the time you took to elaborate on your position and answer some of the questions I had. Still, I remain unconvinced by your “proof”. Most of your claims are baseless and have little significance to the underlying pathology and treatment of hypertension.

Let me ask you Karen, do you personally consume the recommended 1500 mg of sodium every day? Do you meticulously measure every milligram of salt you ingest? If not, it is disgraceful and hypocritical of you to expect the entire country to follow such restrictive and unnatural behaviours – especially since they’re not backed by any quality evidence.

The science is **NOT** settled. The totality of the evidence has **NOT** been considered. Your belief that excess sodium consumption causes hypertension is **WRONG**.

In this subsequent letter, I disprove many of the claims you present in your reply. I also explain in detail a possible causal factor for hypertension – one that has little to do with sodium intake. Once again, I ask you to keep an open mind when reading the following letter. If you truly care about the health of Canadians, please read on.

Hypertension : A Reminder

Hypertension, as defined by the [Public Health Agency of Canada](#), is a state in which “systolic blood pressure [is] at or above 140 mmHg or diastolic blood pressure [is] at or above 90 mmHg.” According to the American Heart Association, “essential hypertension (*essential* in a medical context [means idiopathic](#), that is, of unknown cause) accounts for [95% of all cases](#) of hypertension”.

You explain in your response that “[a high sodium intake,] physical inactivity, overweight and obesity, unhealthy diet, harmful use of alcohol, inadequate potassium intake and type 2 diabetes” are all factors that are *associated* with hypertension.

The “Cause” of Hypertension

As mentioned above, 95% of all cases of hypertension remain a mystery. Apparently though, you claim to know the cause of hypertension. “High blood pressure or hypertension occurs when blood pressure is too

high for long periods of time. ... It develops slowly overtime and without obvious symptoms.” ...This explains absolutely nothing. These appear to be nothing more than meaningless statements. The only purpose of this explanation is to prove how little you know about hypertension.

Furthermore, you claim that “too much [dietary sodium] can lead to high blood pressure” and that “it is estimated that over 30% of high blood pressure cases in Canada are due to high sodium intake.”

This is utterly false.

Upon reviewing [the study](#) that this statistic is based on, you grossly misinterpreted the conclusion made in the paper. The authors *actually* concluded that “reducing dietary sodium additives *may* decrease hypertension prevalence by 30%.”

The report does *not* mention anywhere that excess sodium intake was responsible for hypertension, simply that reducing sodium intake lowered blood pressure. How are you so certain that excessive alcohol intake, inadequate potassium intake, physical inactivity, obesity, type 2 diabetes, and/or other unknown factors weren't responsible for the the illness?

You *cannot* interpret studies whatever way you want – especially when public health is at stake. To present this interpretation as fact is misleading, unscientific and is a blatant lie.

Are you really basing dietary guidelines on “estimates”, “maybes” and faulty logic? Where is the quality research that supports your position on sodium restriction?

If this same logic used in creating Canada’s dietary guidelines, you may as well scrap the whole thing.

Salt In The Past Century

The prevalence of hypertension has almost *quadrupled* over the past 80 years. [[ref](#), [ref](#)]

Despite the drastic rise in hypertension, the consumption of salt actually went *down* over the past century. “Our current salt consumption (1.5 to 1.75 teaspoons per day, 8-9 grams) is about one *half* of the amount consumed between the War of 1812 and the end of World War II, which was about 3 to 3.3 teaspoons (16-17 grams) of salt per day.” [[ref](#)] Additionally, salt consumption [has remained relatively consistent](#) from 1957 to 2003 (mean, 3526 mg/day). How can sodium be blamed for the increasing rate of hypertension, when salt consumption has *decreased* over the past 2 centuries, and has remained unchanged for the past 50 years?

Thinking about it logically, it doesn’t make much sense that a nutrient that’s been in our environment for *hundreds of thousands of years* is suddenly responsible for the dramatic rise in hypertension seen in the past century. It much more likely that a new and recent change in our physical or food environment is linked to its etiology.

Problem Solve Like An Engineer

Before even suggesting a solution, engineers are taught to identify the root cause of a problem. This way, they are certain to address the real issue at hand, not just a symptom or a secondary effect.

For example : Ever since you've owned it, your hot tub has maintained a comfortable water temperature of 96°F. However, over the past few weeks, the temperature has been slowly and inexplicably creeping up, to the point where it's now almost unbearably hot. Your first instinct is to add a few trays of ice cubes to the tub. This immediately lowers the temperature and allows you to bathe comfortably again. This solution works for a little while, until the temperature creeps up yet again. This is what is called "treating a symptom".

Indeed, adding ice cubes to the tub will lower its temperature – it's a working solution. However, it's far from the most logical. Before even thinking about adding ice cubes to the tub, one should ask : *why* is the water getting hotter? Could it be that the hot tub is lacking ice cubes? Probably not, as it worked fine before. Or could it be that the temperature regulator is faulty? Ah, now we're using our brain. The temperature regulator is replaced, and the temperature returns to its comfortable 96°F.

What I'm trying to explain is that just because a solution works (in the short-term), does not mean that the solution was in any way related to the cause of the problem, or that the root issue was addressed. Lowering sodium intake may lower blood pressure (in the short-term), but it does not mean that excessive sodium intake is the root cause of hypertension. Like in my example – and as I'll explain further on – elevated blood pressure may be caused by a faulty regulator.

[This article](#) nicely explains *Hill's criteria* for determining causation in statistics.

Treating a Symptom

"Treating a symptom" is unfortunately extremely common in the field of health and medicine. Much of modern medicine is focused on addressing superficial symptoms without having a deep understanding of the underlying issues. With ever increasing specialization in health and medicine, it's becoming more and more difficult to piece together the sum of the evidence on a given problem. Each is working on their individual piece of the puzzle, yet no one puts the puzzle together. Few of these experts take the time – or are even able – to critically assess the root cause of the problem they're working on. It's not that they're unintelligent, but their specialization bubble prevents them from being able to piece together the totality of the evidence and put the puzzle together.

As an expert yourself, your contribution to the puzzle pertains to the effects of dietary sodium reduction on blood pressure. A somewhat useful piece of information. However, it's unlikely that you can solve the hypertension puzzle with just this one small piece.

Sodium & Hypertension : A Singled Out Victim

You seem to have forgotten that there are a number of other lifestyle and dietary factors that can also affect blood pressure. I understand that your department focuses primarily on food research, but you seem to focus all of your attention on sodium. I'm curious to know why your emphasis is on sodium restriction and why you essentially ignore the effects of potassium, sugar and alcohol consumption on blood pressure?

You might argue that it's because "even a modest reduction in dietary sodium can result in a significant decrease in blood pressure." Once again, this statement proves nothing more than your lack of understanding of the effects of sodium on blood pressure.

In [this study](#) – the one *you* referred to in your reply – “reducing dietary sodium additives by 1840 mg/day would result in a decrease of 5.06 mmHg (systolic) and 2.7 mmHg (diastolic) blood pressures.” In other words, if you are hypertensive (with blood pressure above 140/90 mmHg), cutting more than *half* of your sodium intake would only marginally decrease your blood pressure – and likely won’t resolve your hypertension.

In fact, some research suggests that even successfully lowering blood pressure may not even reduce the risk of cardiovascular events. “There were no significant differences in the rates of cardiovascular disease events during follow-up among those assigned vs not assigned to either sodium reduction or weight loss [despite reductions in blood pressure].” [\[ref\]](#) “In even successfully treated hypertensive patients, most CVD events that would have occurred without treatment *still* occur.” [\[ref\]](#)

Where’s The Proof?

Thus far, I have yet to come across *any* research that proves that excess sodium intake (in isolation) *causes* hypertension. There doesn’t seem to be a feasible pathological explanation as to why a prolonged, higher than recommended intake of sodium causes hypertension. There are many studies that demonstrate an association between excessive sodium intake and hypertension, but none that prove causality.

Although some studies show that reducing sodium intake reduces blood pressure, it does not imply that excess sodium was responsible for hypertension in the first place. From another point of view, one might think that excess alcohol consumption is the cause of hypertension, since [reducing alcohol intake](#) decreases blood pressure similarly to reducing sodium intake.

Research That You Neglected To Review

Although you say that the “totality of best evidence [supports] lowering sodium intake”, there appears to be high quality research that contradicts the current sodium guidelines. You have either neglected, ignored, or discarded these studies, since they don’t support your preexisting beliefs.

In [this Cochrane Review](#) (one of the most respected and trusted scientific reviews), the researchers conducted a meta-analysis of randomized controlled trials “to assess the long-term effects of advice and salt substitution, aimed at reducing dietary salt, on mortality and cardiovascular morbidity”. The authors of this review concluded that “there is *insufficient* power to confirm clinically important effects of dietary advice and salt substitution on cardiovascular mortality in nonsensitive or hypertensive populations.”

Yet, you claim that “there is evidence for a *causal* relationship between reductions in sodium intake and all-cause mortality, cardiovascular disease and hypertension”. I would love for you to provide me with this high quality evidence.

You mention that “a 2013 Institute of Medicine consensus report found that the methodological quality of studies linking lower sodium intake with adverse health outcomes was variable and this limited the ability to make comparisons or conclusions.” (With which I don’t disagree.) However, in the *exact same report*, the authors conclude that “evidence from studies on direct health outcomes is inconsistent and insufficient to conclude that lowering sodium intakes *below 2300 mg per day* either increases or decreases risk of CVD outcomes (including stroke and CVD mortality) or all-cause mortality.”

Along the same lines, the author of [this 2016 scientific paper](#) searched the scientific literature to find any quality research that justifies the current 2300mg/day sodium limit. He concluded that :

- 1 There are no randomized controlled trials (RCTs) allocating individuals to below 2,300 mg and measuring health outcomes;
- 2 RCTs allocating risk groups such as obese prehypertensive individuals and hypertensive individuals down to (but not below) 2,300 mg show no effect of sodium reduction on all-cause mortality;
- 3 RCTs allocating individuals to below 2,300 mg show a minimal effect on blood pressure in the healthy population (less than 1mm Hg) and significant increases in renin, aldosterone, noradrenalin, cholesterol, and triglyceride;
- 4 Observational studies show that sodium intakes below 2,645 mg and above 4,945 mg are associated with increased mortality.

The March 2019 report from the Academy of Sciences on Sodium and Potassium intake concludes that “there remains *insufficient evidence* to establish sodium [a dietary reference intake] for adequacy.”

I think I’ll stop here. If this isn’t proof that your guidelines are baseless, I don’t know what is.

The Wrong Suspect?

I’d like to remind you that “commercially processed foods account for 77% of [Canadian’s] sodium intake.” [\[ref\]](#) If those who reduce their sodium intake – likely by decreasing their consumption of processed foods – have lower blood pressure, who’s to say that it’s because of the reduction in sodium (a single nutrient), and not the reduction in processed foods? Perhaps the effect was due to the reduction of processed foods in the diet – notably refined carbohydrates – and had little to do with the reduction of sodium. (Replacing chips with carrots for example.)

Furthermore, you mention that hypertension “develops slowly overtime”. It seems more likely that factors that also develop slowly over time (such as obesity, type 2 diabetes, metabolic syndrome or insulin resistance) are more likely to be linked with the progression of hypertension.

Does sodium affect blood pressure? Yes. Is sodium causally responsible for hypertension? Not based on any research I’ve seen. Is there compelling evidence to support an alternative hypothesis for the cause of hypertension? Yes.

The Root Cause of Hypertension?

The cause of essential hypertension remains a mystery and may be multi-factorial. However, there is compelling evidence that insulin may explain its underlying pathology.

Insulin is an anabolic, fat storing hormone and aids in blood sugar regulation. It is well-known that hyperinsulinemia plays an important role in the pathology of obesity [\[ref, ref, ref, ref\]](#) and type 2 diabetes [\[ref, ref, ref\]](#). Unsurprisingly, there also appears to be an important relationship between elevated insulin levels and hypertension.

First, let’s understand how blood pressure is regulated.

[1] – The Renin-Angiotensin-Aldosterone System (RAAS)

If you are unfamiliar with this system, you should resign now. It is absurd that you are involved in making national dietary guidelines, yet know little to nothing about the diseases you're trying to prevent.

“The RAAS plays a major role in orchestrating the maintenance of normal blood pressures.” [ref] With help from the kidneys, it responds to blood pressure variations and aims to maintain regular blood pressure. This system respond very effectively to low or high blood pressures, and adjusts blood pressure accordingly by retaining or excreting sodium. *Pressure natriuresis* is the mechanism responsible for excreting excess sodium – along with extracellular fluid – in the urine to lower blood pressure.

“Alterations in the sympathetic nervous system and the renin-angiotensin-aldosterone system are key factors in the development and maintenance of hypertension.” [ref] Similar to my hot tub example, it appears that the root issue of hypertension lies in its regulation. Those with obesity tend to have an activated RAAS, which induces hypertension. [ref] Additionally, diabetics are twice as likely to have hypertension as compared to non-diabetics, due to “up-regulation of the renin-angiotensin-aldosterone system, oxidative stress, inflammation, and activation of the immune system.” [ref]

[2] – Insulin’s Role in Hypertension

Insulin plays an important role in blood pressure regulation (or deregulation) by promoting the activation of the RAAS. [ref] This is likely why type 2 diabetics – especially those taking exogenous insulin – are more susceptible to hypertension. The following studies serve to support this theory.

In two groups, matched for sex, age, body weight and body fat, “both plasma insulin (50 ± 6 vs. 30 ± 6 $\mu\text{U}/\text{mL}$) and plasma glucose concentrations (114 ± 8 vs. 85 ± 8 mg/dl) at two hours were significantly higher in the hypertensive than in the normotensive subjects.” “These results provide preliminary evidence that essential hypertension is an insulin-resistant state.” [ref]

Independent of obesity, hypertensives were significantly more insulin resistant. “There was a negative correlation between insulin sensitivity and ... systolic blood pressure.” The mean blood pressure and fasting insulin was :

- 125/74 mmHg & 4.7 mU/L for the normotensive group;
- 171/104 mmHg & 7.5 mU/L for the non-obese hypertensive group;
- 168/103 mmHg & 11.9 mU/L for the obese hypertensive group. [ref]

A random population of 2475 individuals were assessed for hypertension and glucose-intolerance. Of the hypertensives, “83.4% ... were either glucose-intolerant or obese – both established insulin-resistant conditions.” [ref]

Over a 6 to 12 year follow-up, “subjects with insulin values above the 75th percentile experienced three times more hypertension than did those below the 25th percentile.” [ref]

Metabolic syndrome – characterized primarily by insulin resistance – appears in “up to one-third of hypertensive patients.” “Visceral obesity, insulin resistance, oxidative stress, endothelial dysfunction, activated renin-angiotensin system, increased inflammatory mediators, and obstructive sleep apnea” all appear to play a role in the progression of hypertension in those with metabolic syndrome. [ref]

Many more scientific articles – supported by substantial evidence – further elaborate on this theory. Some researchers even claim that there is a *causal* and *independent* relationship between insulin and essential hypertension. [[ref](#), [ref](#), [ref](#)]

[3] – How Does Insulin Affect Blood Pressure?

Insulin inhibits renal sodium excretion and overrides the *pressure natriuresis* mechanism, which in turn increases blood pressure. [[ref](#), [ref](#), [ref](#), [ref](#), [ref](#)] So while excess sodium in the blood slightly raises blood pressure, the problem lies in that insulin prevents the kidneys from excreting the excess sodium, which keeps blood pressure elevated.

Yes, reducing dietary sodium intake will lower the concentration of sodium present in the blood and may marginally decrease blood pressure. Adding ice cubes to an overheated hot tub will cool it down. But this does *not* address the root issue. In healthy individuals with low levels of circulating insulin, any excess sodium – along with water – is simply excreted in the urine. (Hence why you get thirsty after eating something salty.) This is also why the 24-h urinary sodium excretion measurement is considered the gold standard method for measuring population sodium intake, as any excess sodium consumed is simply excreted in the urine.

[4] – Low Insulin & Hypertension

If insulin truly does play a role in the pathology of hypertension, you would expect that lowering insulin would lower blood pressure. Well, that's exactly what happens.

Hypertensive diabetics taking exogenous insulin were tested to see how lowering their insulin dosage would affect their blood pressure and sodium excretion. Their dosage was lowered by approximately 20 IU for 6 consecutive days. Prior to the intervention, the mean blood pressure was 171.3/97.3 mmHg, and urinary sodium excretion was 96.0 mEq/L. After 6 days on the intervention, the mean blood pressure fell drastically to 138.2/79.7 mmHg (almost non-hypertensive range for diabetics) and sodium excretion tapered down from 158 mEq/L on the first day, to 105.3 mEq/L on the sixth. (Once again, demonstrating that when insulin is lowered, the kidneys can release more sodium, thus reducing blood pressure, and vice versa.) The authors conclude that there may be a “causal relationship between insulin and blood pressure even in essential hypertension”. [[ref](#)]

In this dietary based study, a low-carbohydrate diet was tested against a low-fat diet for weight loss over 48 weeks. (Low-carbohydrate diets necessarily lower endogenous insulin secretion, since carbohydrates elicit a greater insulin response as compared to protein or fat.) The authors concluded that “the [low-carbohydrate diet (<20g carbohydrate/day)] had a more beneficial impact than [a low-fat diet] on systolic (–5.9 vs 1.5 mmHg) and diastolic (–4.5 vs 0.4 mmHg) blood pressures.” [[ref](#)]

[5] – Sugar's Effect On Blood Pressure

Refined carbohydrates – such as simple sugars and wheat flour – tend to illicit the greatest insulin response of any food. Unsurprisingly, such saccharides are found almost exclusively in processed foods (that coincidentally, may also be salty). As one would expect, they tend to increase blood pressure.

A systematic review of randomized controlled trials reviewed the effects of sugar on blood pressure. In trials over 8 weeks in duration – independent of increases in body weight – the mean systolic blood pressure increased by 6.9 mmHg, and diastolic blood pressure by 5.6 mmHg. [[ref](#)]

In this randomized controlled trial, consuming 200g of fructose daily for only 2 weeks lead to a mean increase in systolic blood pressure by 7 ± 2 mmHg, and diastolic blood pressure by 5 ± 2 mmHg. [ref]

This scientific article explains that “high-insulinemic food, typical of current “Western” diets, has the potential to cause hyperinsulinemia and insulin resistance, as well as an abnormally increased activation of the sympathetic nervous system and the [RAAS], alterations that play a pivotal role in the pathogenesis of ... hypertension.” [ref]

[6] – Additional Reading

If you are still unconvinced about the insulin hypothesis of hypertension, I’ve included 5 additional scientific articles below that go into much further detail on the topics of salt, sugar, insulin, hypertension and cardiovascular disease. [ref, ref, ref, ref, ref] If you have not read it, I highly recommend “The Salt Fix”, by Dr. James DiNicolantonio.

Conclusion

“Officials at Health Canada have determined that the scientific consensus and totality of best evidence continues to support the health benefits that can be achieved by lowering sodium intake.” As demonstrated in the present letter, there is **NOT** a scientific consensus. The totality of best evidence was **NOT** considered. There is **NO** meaningful research to support the health benefits from lowering sodium intake.

You’re targeting the wrong suspect. Insulin (not sodium) appears to play an important role – perhaps even a causal role – in the progression of hypertension. High-insulinemic foods – notably refined carbohydrates – should be the focus of dietary restriction for the prevention and treatment of hypertension, not sodium.

“The Department uses the best available evidence to support food and nutrition related policies and regulations. ... Our approach to sodium reduction is consistent with current international efforts of sodium reduction.” Prior to your reply, I trusted that Health Canada had a team of expert researchers that critically investigated important health matters. I was unaware, however, that Health Canada does not conduct any of its own research, and simply “copy-pastes” whatever large, international health institutions advise.

I am disappointed that you are not even a little bit intrigued by this alternative hypothesis to hypertension. Instead, you aim to dismiss my arguments and research, and back up your existing beliefs with illogical, inconclusive and unscientific “proof”. Science cannot progress if you don’t rethink your current beliefs.

If it’s true that “Health Canada continues to monitor emerging science to keep up to date with the latest evidence and adjust its policies accordingly”, I fully expect you to update the sodium guidelines based on this present letter and the research I’ve provided. If you do not personally have the authority to revise Canada’s Dietary Guidelines, please send this letter to those who do have the authority to reassess the guidelines.

I truly hope that we can come to an understanding and that you will take action on this matter, as I am prepared to take legal action if necessary.

The science is **NOT** settled. The totality of the evidence has **NOT** been considered. Your belief that excess sodium consumption causes hypertension is **WRONG**.

Remember that science has been wrong before. Starting in the 1920’s – and for over half a century – doctors were promoting the use of cigarettes, reassuring patients “that smoking was safe”. [ref]

Stop blindly trusting “the best available evidence” from large, international health institutions. They’re not infallible gods. They may even be as misguided and uninformed as you are. Start taking your job seriously and maybe someday you will have a real impact on Canadians’ health.

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P.S. I believe you meant “*over time*” and not “*overtime*”.