April 11, 2016

The Honorable Tom Vilsack
Secretary of Agriculture
U.S. Department of Agriculture
1400 Independence Avenue, S.W.
Washington, DC 20250

The Honorable Sylvia Burwell
Secretary of the Department of Health and Human Services
200 Independence Avenue, S.W.
Washington, DC 20201

Re: Requests for Withdrawal of Sodium Dietary Guideline Provisions, Transparent Rulemaking and Withdrawal of any Requests for Voluntary Salt Reductions in Food Products by the FDA.

Dear Secretary Vilsack and Secretary Burwell:

As demonstrated below, the provisions of the *2010 Dietary Guidelines for Americans* and the *2015-2020 Dietary Guidelines for Americans* related to sodium ("sodium provisions") both violate the National Nutrition Monitoring and Related Research Act, 7 U.S.C. §5301, et seq. As a result, we respectfully request the withdrawal of the sodium provisions in those *Guidelines* and request the initiation of an open and transparent rulemaking procedure, with public hearings, supported by current and reliable scientific and medical evidence. And we request that you resist the proposed call for voluntary salt reductions in food products, an action that would be imprudent and likely harmful in light of current scientific evidence. The federal government pushing for reformulation of almost every food product made in the U.S. is unprecedented in its audacity. Every citizen will feel the effect of this overreach. Consumers are already free to choose from many alternatives to suit their taste and salt is a necessary nutrient essential for life and good health.

The sodium provisions, jointly issued as part of the *Dietary Guidelines* on January 31, 2011, by the U.S. Department of Agriculture ("USDA") and the Department of Health and Human Services ("HHS"), were based on inadequate medical and scientific evidence, as admitted by their original author, the Institute of Medicine ("IOM"). IOM published "Dietary Recommended Intakes ("DRIs") in 2004, that were adopted as the *2010 Dietary Guidelines*, regardless of the IOM conclusion that: "...because of insufficient data from dose-response trials, an Estimated Average Requirement could not be established and thus a Recommended Dietary Allowance could not be derived."[1]

Regardless of this scientific conclusion, IOM's arbitrary, outdated, non-governmental guidelines, issued without adequate protections against bias and conflicts of interest, and without the protections of transparent rulemaking under the Administrative Procedures Act, were adopted by

[1] Institute of Medicine, *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, 269-423 (2004).

the *2010 Dietary Guidelines*,[2] improperly delegating the statutory role of the Departments and the Executive Branch, to an outside party, without regard to the statutory duties imposed on the Departments.

Your intervention is sought to assure both compliance with law and sound policy created by transparent rulemaking procedures that rely on current scientific and medical evidence, evaluated by the Departments.

The National Nutrition Monitoring and Related Research Act, 7 U.S.C. §5301 et seq., requires USDA and HHS to publish "nutritional and dietary information and guidelines for the general public" and to base the *Dietary Guidelines* on "*the preponderance of the scientific and medical knowledge which is current at the time the report is prepared*." 7 U.S.C. § 5341(a). Both the *2010* and *2015-2020* iterations of the *Dietary Guidelines* are inconsistent with the statutory mandate because their sodium provisions are arbitrary and capricious.

Alone, the flawed sodium provisions in these *Dietary Guidelines* cause significant harm to the public by distributing scientifically unsupportable information disparaging sodium, a mineral essential to human health, under the banner of the United States, and thereby increasing risks for consumers, adversely impacting the market for dietary salt, and causing concern for inappropriate regulatory and litigation initiatives. Furthermore, any call by the FDA for voluntary salt reductions in food products will ensure a greater risk to consumers with no promise of benefit.

The issuance of the *2015-2020 Dietary Guidelines for Americans* is clear proof of the unreliability of the process. We had previously warned both the U.S. Department of Agriculture and the Department of Health and Human Services that the sodium provisions of the *2010 Dietary Guidelines* were wrong and unsupported by the evidence, yet the Guidelines were published without reserve. **The *2015-2020 Dietary Guidelines* confirmed that the key recommendation of 1,500 mg sodium per day in the *2010 Dietary Guidelines* was unjustified and withdrew it**, but not before **consumers were misguided on salt recommendations for 5 years** – a situation that could have been totally avoided if the guidelines had been based on an unbiased review of the science.

Below, we explain further why the proposed sodium provisions in the *2010 Dietary Guidelines* and the current *2015-2020 Dietary Guidelines* should be withdrawn and we provide an assessment of current available scientific and medical evidence that was not adequately considered by the Departments. We trust that you will find this assessment helpful as you fulfill your duties to enhance the public health and welfare with sound dietary policies and standards consistent with medical and scientific evidence.

[2] Both the *Dietary Guidelines* and the *2005 Dietary Guidelines* contain the same sodium limit range of 1500-2300 mg/day. Because a Recommended Daily Allowance could not be determined, the IOM set DRIs that are the basis for the sodium limits in both the *2010 Dietary Guidelines* and the *2005 Dietary Guidelines*. *See* IOM, *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate* (2004).

## I. The Sodium Provisions of the *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* Violate the Statutory Mandate, were Contradicted by the Contemporary, Sound Scientific Evidence and Should have been Withdrawn.

The *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* are joint products of USDA and HHS. The *Dietary Guidelines* are reviewed, updated (if necessary), and published every five years. 7 U.S.C. § 5341(a)(1) ("At least every five years the Secretaries shall publish a report entitled 'Dietary Guidelines for Americans' [which]… shall contain nutritional and dietary information and guidelines for the general public, and shall be promoted by each Federal agency in carrying out any Federal food, nutrition, or health program").

The *Dietary Guidelines* must contain nutritional and dietary information for the general public and must be "based on the preponderance of the scientific and medical knowledge which is current at the time the report is prepared." 7 U.S.C. § 5341(a)(2). The process of generating each edition of the Dietary Guidelines is a joint effort of the USDA and HHS and has evolved to include at least three publicly disclosed stages. In the first stage, an external scientific Dietary Guidelines Advisory Committee ("DGAC") is appointed. During the second stage, the Agencies develop the Dietary Guidelines and consider comments provided in response to the DGAC's report. Finally, the two Agencies develop messages and material communicating the Dietary Guidelines to the general public.

For the *Dietary Guidelines*, the DGAC consists of nutrition and health experts who were appointed to conduct a rigorous and unbiased analysis of scientific information on diet and health and to prepare a report summarizing its findings. It is at this stage where the initial problems with the *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* arose. In the case of the *2010 Dietary Guidelines*, rather than independently assessing all of the scientific and medical data currently available, the DGAC merely adopted the conclusions of the DGAC that prepared the *2005 Dietary Guidelines* and apparently considered "subsequent evidence, especially regarding diet and blood pressure in children." *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010*, D6-2 (originally submitted June 14, 2010).

The DGAC in 2005 derived its sodium consumption recommendations by simply adopting the Dietary Recommended Intakes ("DRIs"), published in 2004 by the Institute of Medicine ("IOM").[3] This document clearly stated that for sodium, "…because of insufficient data from dose-response trials, an Estimated Average Requirement could not be established and thus a Recommended Dietary Allowance could not be derived." Despite acknowledging a lack of evidence, the document went on to make arbitrary recommendations that are followed to this day.[4] One common thread links the decision to adopt flawed recommendations in the first instance, and then base two separate sets of *Dietary Guidelines* on the flawed DRIs -- the chair of the 2010 DGAC's subcommittee on electrolytes served in the same capacity when the *2005*

[3] Institute of Medicine, *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, v.-xiii (2004).

[4] Both the *Dietary Guidelines* and the *2005 Dietary Guidelines* contain the same sodium limit range of 1500-2300 mg/day. Because a Recommended Daily Allowance could not be determined, the IOM set DRIs that are the basis for the sodium limits in both the *2010 Dietary Guidelines* and the *2005 Dietary Guidelines*. *See* IOM, *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate* (2004).

*Dietary Guidelines* were developed, and was the chair of the *Panel on Dietary Reference Intakes for Electrolytes and Water*, which was responsible for developing the DRI's. A rigorous analytical process cannot feature one and the same individual piloting the creation of standards and then being charged with evaluating his own recommendations, and then five years later, being tasked once again to evaluate his prior evaluation.

The *Dietary Guidelines* purport "to summarize and synthesize knowledge about individual nutrients and food components into an interrelated set of recommendations for healthy eating that can be adopted by the public[.]" The *Dietary Guidelines* also are relied upon by "policymakers in designing and carrying out nutrition-related programs, including Federal food, nutrition education, and information programs."

Rather than thoroughly assessing the current scientific and medical knowledge, the Agencies reached a conclusion in 2005 based on insufficient evidence and then repeated the error in 2010[5] and again in 2015.[6] To cure this defect, the Agencies should withdraw the flawed sodium provisions and subject the topic of appropriate sodium limits to rulemaking under the Administrative Procedures Act to ensure that all interested parties are permitted to participate in a public forum and that decision making is supported by sound and current scientific evidence.

As we described in numerous prior public comments,[7] USDA and HHS repeatedly failed to consider and account for strong, evidence-based data that contradicts their preconceived hypotheses related to sodium intake. Moreover, the latest and best scientific evidence contradicts the sodium provisions in the *2010 Dietary Guidelines*, emphasizes the critical role of sodium in health protection, and supports far higher levels of sodium intake than adopted by the Guidelines.

The most recent iteration of the *Dietary Guidelines (Dietary Guidelines for Americans: 2015-2020)*[6] has once again demonstrated that they are an inadequate means of conveying the latest science-based nutritional research to consumers. Although the latest edition of the *Dietary Guidelines* has withdrawn the strong recommendations in the *2010 Dietary Guidelines* to consume less than 1,500 mg sodium, because the latest research has shown this to be a risk to the general population,[8] [9] it continues to recommend an upper limit of 2,300 mg sodium, despite the overwhelming number of scientific research publications that have cautioned against it since the

---

[5] *See Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010*, D6-2 (originally submitted June 14, 2010).

[6] U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 – 2020 Dietary Guidelines for Americans. 8th Edition. December 2015. Available at http://health.gov/dietaryguidelines/2015/guidelines/.

[7] Dietary Guidelines for Americans, 2010 – Written Public Comments – (Posted Oct. 17, 2008 – July 30, 2010), Salt Institute, No. 00010 *Comments to Dietary Guidelines Committee*, (Oct. 21, 2008); No. 000248 *Statement 2: Comments to Dietary Guidelines Advisory Committee*, (Jan. 28, 2009); No. 000447 *Salt Institute Letter to the Dietary Guideline Advisory Committee* (Mar. 16, 2009); No. 000494 *Salt Institute Letter to the Dietary Guidelines Advisory Committee* (May 8, 2008); No. 000566, *Salt Institute Letter to the Dietary Guidelines Advisory Committee* (July 20, 2009); No. 000743 *Salt Institute Letter to the Dietary Guidelines Advisory Committee* (Oct. 28, 2009); No. 000744 *The Mediterranean Diet,* 4 (3) Salt & Health for Nutrition Policy Makers (2009); No. 000752 , *Salt Institute Letter to the Dietary Guidelines Advisory Committee* (Nov. 5, 2009).

[8] O'Donnell MJ, Mente A, Rangarajan S, et al. Urinary Sodium and Potassium Excretion, Mortality, and Cardiovascular Events. N Engl J Med. 2014; 371:612-623 August 14, 2014 DOI: 10.1056/NEJMoa1311889.

[9] Mente A, O'Donnell MJ, Rangarajan S, et al. Association of Urinary Sodium and Potassium Excretion with Blood Pressure. N Engl J Med. 2014; 371:601-611 August 14, 2014 DOI: 10.1056/NEJMoa1311989.

last *2010 Dietary Guidelines* were issued,[10] [11] [12] [13] [14] [15] including the landmark Institute of Medicine (IOM) publication,[16] which stated that there was no consistent evidence to support an association between sodium intake and any adverse effects on health outcomes. On the contrary, it indicated a negative association of reduced dietary sodium with cardiovascular disease outcomes and all-cause mortality. Reduced sodium has been linked to an increase in cardiovascular events and mortality. It is worth noting that the *2015-2020 Dietary Guidelines*, which was meant to be a full review of the latest evidence published since the *2010 Dietary Guidelines*, made several reference to old IOM reports, but totally neglected to mention the milestone 2013 IOM report that did not support salt reduction – public policy cannot be scripted by ignoring landmark reports and evidence that clearly contradicts the established dogma. The essence of scientifically derived evidence is that it is reproducible and cannot be circumvented by biased and unsubstantiated opinion.

The latest evidence persistently published during the last five years demonstrate that there is a safe "range" of salt consumption that results in a lower risk to the overall population. According to this research, the lower end of this safe range begins at 2,800 mg and extends up to 4,800 mg sodium.[17] [18] [19] [20] Americans consume about 3,400 mg sodium on average – at the lower end of this safe range. Notwithstanding this most recent evidence, the new *2015-2020 Dietary Guidelines* stubbornly clings to the invalid recommendation of 2,300 mg sodium – a figure outside the safe range because of obsolete "…evidence on blood pressure, a surrogate indicator of CVD risk."[6] Most physicians agree that surrogate measures have no place in effective public policy making.[21]

---

[10] Bayer R, Merritt Johns D, Galea S. Salt and Public Health: Contested Science and The Challenge of Evidence-Based Decision Making. Health Affairs. 2012; 31(12):2738-2746.

[11] Stolarz-Skrzypek K, Kuznetsova T, Thijs L, et al. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. JAMA. 2011 May 4;305(17):1777-85.

[12] DiNicolantonio JJ, Di Pasquale P, Taylor RS, Hackam DG. Low sodium versus normal sodium diets in systolic heart failure: systematic review and meta-analysis. Heart. 2013; Mar 12. doi: 10.1136/heartjnl-2012-302337.

[13] Todd AS, MacGinley RJ, Schollum JB, et al. Dietary sodium loading in normotensive healthy volunteers does not increase arterial vascular reactivity or blood pressure. Nephrology. 2012; 17: 249–256. doi: 10.1111/j.1440-1797.2011.01550.x

[14] Graudal NA, Hubeck-Graudal T, Jurgens J. Effects of Low-Sodium Diet vs High-Sodium Diet on Blood Pressure, Renin, Aldosterone, Catecholamines, Cholesterol and Triglyceride [Cochrane Review]. Am J Hypertension. 2012 Jan;25(1):1-15.

[15] Stolarz-Skrzypek K, Kuznetsova T, Thijs L, et al. Fatal and Nonfatal Outcomes, Incidence of Hypertension, and Blood Pressure Changes in Relation to Urinary Sodium Excretion. JAMA. 2011;305(17):1777-85.

[16] National Research Council: Institute of Medicine. Sodium Intake in Populations: Assessment of Evidence. Washington, DC: The National Academies Press, 2013.

[17] Asayama K, Stolarz-Skrzypek K, Persu A, Staessen JA. Systematic review of health outcomes in relation to salt intake highlights the widening divide between guidelines and the evidence. Am J Hypertens. 2014 Sep;27(9):1138-42.

[18] O'Donnell MJ, Yusuf S, Mente A, et al. Urinary sodium and potassium excretion and risk of cardiovascular even JAMA. 2011 Nov 23;306(20):2229-38.

[19] Graudal N, Jürgens G, Baslund B, Alderman MH. Compared with usual sodium intake, low- and excessive-sodium diets are associated with increased mortality: a meta-analysis. Am J Hypertens. 2014 Sep;27(9):1129-37.

[20] O'Donnell MJ, Mente A, Smyth A, Yusuf S. Salt intake and cardiovascular disease: why are the data inconsistent? Eur Heart J. 2013 Apr;34(14):1034-40.

[21] Furberg CD. Public health policies: no place for surrogates. Am J Hypertens. 2012 Jan;25(1):21.

However, the failure of USDA and HHS to follow their statutory mandate led to the issuance of these flawed *2015-2020 Dietary Guidelines*[22] that contain arbitrary, capricious, and potentially harmful findings.

## II. Both the Process Used to Derive the *Dietary Guidelines* and the Assessment of the Scientific and Medical Evidence Were Fundamentally Flawed

The processes used by USDA and HHS to develop both the *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* were systemically flawed. Rather than assessing all of the available scientific and medical evidence and using this analysis to draw valid conclusions, the DGAC began with a conclusion based on bias, and then justified its conclusion with selected evidence. By predetermining its conclusion, the DGAC was forced to undertake analytically suspect methods to justify its conclusion, including failing to consider the negative health impacts of sodium reduction in diets, failing to address the reality that there is a physiological sodium appetite, and failing to address conflicting and inconsistent evidence related to the impact of sodium on blood pressure and obesity.

### A. Key Members of the DGAC Appear to Have Injected Personal Bias into Both the 2010 and the 2015-2020 Processes

As we explained in Section I of this letter, we are concerned that the entire process that led to the development of the *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* was flawed. First, it appears that the DGAC began with a conclusion and then worked to justify its conclusion.[23] This is counter to its mandate to perform a rational and independent assessment of all currently available scientific and medical knowledge in order to arrive at its recommendations. In addition to being analytically unsound, an approach with a foregone conclusion evidences the biases of the members of the DGAC.

As we pointed out in our comments to the Agencies, at the first meeting of the 2010 DGAC, when invited to make an opening statement, the chair of the DGAC's subcommittee on electrolytes chose to use the platform to espouse his personal beliefs regarding the evidence. Rather than focusing the discussion on an assessment of all currently available scientific and medical evidence related to sodium, he revealed significant aspects of his own personal philosophy surrounding the issue of sodium intake and health, citing only the literature that supported his personal view.[24]

Further, as we described in Section I of this letter, the DGAC subcommittee chair was also the chair of the *Panel on Dietary Reference Intakes for Electrolytes and Water* – the group tasked

---

[22] Both the *2010 Dietary Guidelines* and the *2005 Dietary Guidelines* contain the same sodium limit range of 1500-2300 mg/day. Because a Recommended Daily Allowance could not be determined, the Institute of Medicine ("IOM") set Dietary Recommended Intakes that are the basis for the sodium limits in both the *2010 Dietary Guidelines* and the *2005 Dietary Guidelines*. *See* IOM, *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate* (2004).

[23] We also question whether the DGAC was constituted and operated in compliance with the Federal Advisory Committee Act. Public Law 92-463 (5 U.S.C. Appendix 2, the Federal Advisory Committee Act of 1972), as amended.

[24] Transcript of First Dietary Guidelines Advisory Committee Meeting, 197-203 (Oct. 30, 2008). Available at: http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/Meeting1/DGACMtg1-Day1transcript.pdf

with developing the flawed DRIs[25] upon which both the *2005* and *2010 Dietary Guidelines* are based. Rather than engaging in a fresh and objective analysis of all the scientific and medical evidence available to craft the *Dietary Guidelines*, the process that appears to have occurred was to put the same individual who oversaw the development of the flawed DRIs in the position of evaluating his own recommendations for the creation of the *2005 Dietary Guidelines*, and again for the creation of the *2010 Dietary Guidelines*. Rather than appearing neutral and unbiased, taken together with the DGACs failure to consider contrary evidence, the process used strongly suggests that the DGAC relied heavily on the predisposition of its subcommittee chair when drafting its recommendations**.** It is difficult to see how an objective review can be carried out when both the Chairperson of the sodium subcommittee and the DGAC abandoned an evidence-based approach in favor of preordained biased views.

In the case of the *2015-2020 Dietary Guidelines*, the sodium group was led by an individual who has publicly and repeatedly demonstrated a bias towards population-wide salt reduction. In fact, prior to the initial deliberations of the 2015-2020 DGAC, this individual was an author of the Presidential Advisory recommending maintenance of the previous Dietary Guidelines recommendations for salt reduction.[26] Considering that the leader of the sodium provisions section had publicly made the commitment to support the established sodium recommendations for salt reduction of the *2010 Dietary Guidelines* prior to the deliberations for the *2015-2020 Dietary Guidelines*, it should come as no surprise that the *2015-2020 Dietary Guidelines* process ignored the latest evidence and reiterated the 2,300 sodium upper limit, despite the fact that no new evidence to support it was presented**.** In fact, the real surprise was that an individual, publicly pre-committed to a one-sided view of the sodium issue, was selected to be on the DGAC, and then as head of the sodium group. It is difficult to imagine a more cynical view of the principle of an impartial scientific evaluation of evidence. How can this process not be considered flawed?

This disregard for carrying out a rigorous and balanced analysis of the scientific evidence on dietary salt and health outcomes was highlighted in a very recent research article entitled, "A metaknowledge analysis of the salt controversy,"[27] published in the International Journal of Epidemiology. Faced with such a disturbing case of bias in the interpretation of evidence, it would be nothing less than unethical to consider the sodium provisions of the Dietary Guidelines and any call for the reduction of salt in food products as legitimate.

---

[25] Institute of Medicine, *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, ix (2004) ("The group responsible for developing this report, the *Panel on Dietary Reference Intakes for Electrolytes and Water*, under the oversight and assistance of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes (the DRI Committee), has analyzed the evidence on risks and beneficial effects of nutrients included in this review.").

[26] Whelton PK, Appel LJ, Sacco RL, Anderson CA, et.al. *Sodium, blood pressure, and cardiovascular disease: further evidence supporting the American Heart Association sodium reduction recommendations*. Circulation. 2012 Dec 11;126(24):2880-9.

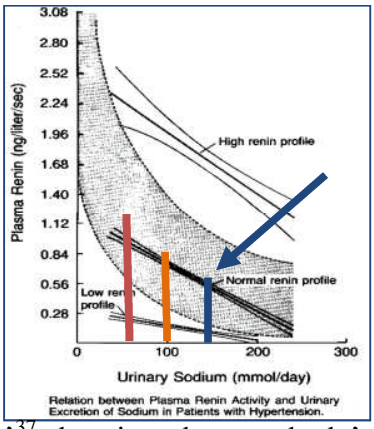
[27] Trinquart L, Johns DM, Galea S. Why do we think we know what we know? *A metaknowledge analysis of the salt controversy*. Int J Epidemiol. 2016 Feb;45(1):251-60. doi: 10.1093/ije/dyv184. Epub 2016 Feb 17.

## B. The DGAC Failed to Consider Evidence Related to Negative Impacts of Sodium Reduction

By committing immediately to the DRIs developed in 2004, the 2010 DGAC failed to properly consider evidence related to the impact of salt consumption on the production of plasma renin. The Renin-Angiotensin System ("RAS") is the physiological mechanism to make up for inadequate salt (sodium chloride) consumption. When any one of our body's sensory mechanisms detects that we're not consuming sufficient salt, the RAS is activated to signal the kidney conserve sodium and reabsorb it back into the circulatory system.[28] This complex neuro-hormonal chain reaction, perfected through biological evolution, is critical for maintaining balance in our circulatory system.

Unfortunately, although the RAS helps us make up for too little salt consumption, it does so at a heavy cost to our health. Elevated RAS levels cause metabolic syndrome,[29] insulin resistance,[30] cardiovascular disease,[31] and a host of other serious conditions.[32] [33] [34] There is no longer any doubt whatsoever that an elevated RAS is a very serious risk factor for overall health.

As can be seen from the diagram taken from Alderman,[35] as our sodium intake is reduced, the plasma renin increases dramatically – it is the body's natural response to salt reduction. The blue arrow shows that, once our sodium intake falls below 150 mmol sodium/day (3,450 mg), the body reacts by producing high levels of renin to activate the RAS chain reaction to conserve the available sodium. It is nature's way to make up for an inadequate salt consumption.



Relation between Plasma Renin Activity and Urinary Excretion of Sodium in Patients with Hypertension.

The level of 3,450 mg sodium per day comes to approximately 9 grams of salt, which is close to the average American consumption.[36] This is an example of the 'wisdom of the body,'[37] the view that our body's

[28] J.A. Schafer, H. Valtin, *Renal Function: Mechanisms Preserving Fluid and Solute Balance in Health,* (3d ed. 1995).
[29] C.H. Wang, F. Li, N. Takahashi, *The Renin Angiotensin System and the Metabolic Syndrome*. Open Hypertension. J. 2010;3:1-13.
[30] Z. Liu, *The Renin-Angiotensin System and Insulin Resistance*, 7 (1) Curr. Diab. Rep. 34-42 (Feb. 7, 2007).
[31] S. Verma, M. Gupta, D.T. Holmes, et al., *Plasma Renin Activity Predicts Cardiovascular Mortality in the Heart Outcomes Prevention Evaluation (HOPE) Study*. Eur. Heart J. (2011), (first published online Mar. 17, 2011, doi:10.1093/eurheartj/ehr066).
[32] J. Peti-Peterdi, J.J. Kang, I. Toma, *Activation of the Renal Renin–Angiotensin System in Diabetes—New Concepts*, 23(10) Nephrol. Dial. Transplant 3047-49 (2008).
[33] S. Inaba, M. Iwai, M. Furuno, et al., *Continuous Activation of Renin-Angiotensin System Impairs Cognitive Function in Renin/Angiotensinogen Transgenic Mice,* 52(2) Hypertension 356-62 (Feb. 2009). Epub 2008 Dec. 1.
[34] N. Takahashi, F.Li, K. Hua, et al., *Increased Energy Expenditure, Dietary Fat Wasting and Resistance to Diet-Induced Obesity in Mice Lacking Renin*, 6(6) Cell Metab. 506-12 (Dec. 2007).
[35] M.H. Alderman, S. Madhavan, W.L. Ooi, H. Cohen, J.E. Sealey, J.H. Laragh, *Association of the Renin-Sodium Profile With the Risk of Myocardial Infarction in Patients With Hypertension*, 324 N. Engl. J. Med. 1098–1104 (1991).
[36] A.M. Bernstein, W.C. Willett, *Trends in 24-h Urinary Sodium Excretion in the United States, 1957–2003: A Systematic Review*, 92 Am. J. Clin. Nutr. 1172-80 (2011).
[37] W.B Cannon, *The Wisdom of the Body* (1932).

physiology is the best authority on determining our personal needs. This average level of salt consumption is sufficient to prevent any spike in RAS activity. However, the *2010 Dietary Guidelines* recommend that we drop our consumption well below this, down to 2,300 mg sodium (100 mmol)/day. At this level, the orange line, the renin begins to rise rapidly. It is also abundantly clear that moving to the 1,500 mg sodium (65 mmol)/day level suggested in the *2010 Dietary Guidelines* for more than half the American population significantly increases the impact. At this red line level, renin levels spike up dramatically. Nature's response to reduced sodium has been deliberately downplayed and ultimately ignored to support the sodium provisions of both iterations of the *Dietary Guidelines*.[38]

While few would question the benefits of reduced blood pressure *per se*, salt reduction, the *Dietary Guidelines'* primary strategy to achieve this, is a very poor and dangerous choice. Other more effective lifestyle strategies to reduce blood pressure, such as more physical exercise or the adoption of a Mediterranean-type diet, have no negative side effects. But reducing salt to lower the risk of blood pressure in the general population will stimulate elevated RAS and increases the risk of other diseases.

In fact, a recent issue of *American Heart Journal*[39] makes it clear that the most important strategies to control cardiovascular disease involve blocking excess levels of renin and aldosterone, the principle components of the RAS. If blocking elevated RAS levels is so critical, then it's clear that consuming enough salt to prevent elevated RAS in the first place is essential to good health.

There have also been a string of recent meta-reviews making it clear that population-wide salt reduction will not provide any significant health benefits and may possibly result in harm to consumers. Three Cochrane Collaboration reviews[40] [41] [42] and a German Institute for Quality and Efficiency in Health Care meta-review[43] all conclude that there is insufficient evidence to warrant population-wide salt reduction.

There is also a significant body of scientific and medical evidence that illustrates other serious negative consequences of a low-salt diet. For example, a very recent study from Harvard Medical School demonstrated that when healthy people were placed on a low-salt diet, they developed insulin resistance within 7 days.[44] [45] Other recently derived evidence showing the

---

[38] IOM, *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, 282 (2004).
[39] G.C. Fonarow, C.W. Yancy, A.F. Hernandez, et al., *Potential impact of optimal implementation of evidence-based heart failure therapies on mortality*, 161 Am. Heart. J. 1024-30 (2011).
[40] L. Hooper, C. Bartlett, G. Davey Smith, S. Ebrahim, *Reduced Dietary Salt for Prevention of Cardiovascular Disease*, The Cochrane Library (2003), Issue 1.
[41] Jurgens G, Graudal NA. *Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglycerides*. Cochrane Database Syst Rev 2004; (1):CD004022.
[42] Taylor RS, Ashton KE, Moxham T, Hooper L, Ebrahim S. *Reduced dietary salt for the prevention of cardiovascular disease*. Cochrane Database Syst Rev 2011, Issue 7. Art. No.: CD009217. DOI: 10.1002/14651858.CD009217.
[43] Institute for Quality and Efficiency in Health Care. *Executive Summary of Report A05-21B, Benefit Assessment of Non-drug Treatment Strategies in Patients with Essential Hypertension: Reduction in Salt Intake (Nutzenbewertung Nichtmedikament? Ser Behandlungsstrategien Bei Patienten Mit Essenzieller Hypertonie: Kochsalzreduktion)*. Cologne, Germany 2009: Executive summary in English can be accessed at, http://www.iqwig.de/download/A05-21B_Executive_Summary_Nondrug_treatment_strategies_for_hypertension-reduction_in_salt_intake.pdf.
[44] Garg R., Williams G.H., Hurwitz S., Brown N.J., Hopkins P.N., Adler G.K., *Low-Salt Diet Increases Insulin Resistance in Healthy Subjects*, 60(7) Metabolism 965-68 (July 2010). Epub 2010 Oct 30.

grave negative consequences of a low-salt diet also does not appear to have been seriously considered in developing the *Dietary Guidelines*, including:

a) insulin resistance[46]
    i. This study demonstrates the insulin resistance induced by chronic dietary salt restriction.
b) metabolic syndrome[47]
    i. This study demonstrated that low-salt diets induced alterations in the plasma lipoproteins and in inflammatory markers that are common features of metabolic syndrome (precursor to heart attack, stroke, and diabetes) in healthy adults.
c) congestive heart failure[48]
    i. This randomized, controlled, double blind study demonstrated that low-salt diets result in much higher rates of mortality and hospital readmissions in patients with congestive heart failure compared to similar patients on a regular salt diet.
d) diabetes 2 (all-cause mortality)[49]
    i. In this study with type 2 diabetes patients, lower sodium was associated with increased all-cause and cardiovascular mortality.
e) cardiovascular events[50]
    i. This study was the third in a long series of NHANES-based analyses which showed higher mortality associated with lower sodium intake.
f) iodine deficiency diseases[51]
    i. This recent study demonstrated that more and more of the population is experiencing the potential for iodine deficiency diseases since the call for reduced salt consumption.
g) cognition loss[52]

---

[45] Garg R, Sun B, Williams J. *Effect of Low Salt Diet on Insulin Resistance in Salt Sensitive versus Salt Resistant Hypertension*. Hypertension. 2014 Dec; 64(6): 1384–1387.
[46] G.F. Ruivo, S.M. Leandro, C.A. do Nascimento, et al., *Insulin Resistance Due to Chronic Salt Restriction is Corrected by α and β Blockade and by l-arginine*, 88(4-5) Physiology and Behavior 364-70 (2006).
[47] E.R. Nakandakare, A.M. Charf, F.C. Santos, et al., *Dietary Salt Restriction Increases Plasma Lipoprotein and Inflammatory Marker Concentrations in Hypertensive Patients*, 200(2) Atherosclerosis 410-16 (2008).
[48] S. Paterna, G. Parrinello, S. Cannizzaro, et al., *Medium Term Effects of Different Dosage of Diuretic, Sodium, and Fluid Administration on Neurohormonal and Clinical Outcome in Patients With Recently Compensated Heart Failure,* 103(1) Am. J. of Cardiology 93-102 ( 2009).
[49] E.I. Ekinci, S. Clarke, M.C. Thomas, et al., *Dietary Salt Intake and Mortality in Patients With Type 2 Diabetes*, 34 Diabetes Care 703-09 (2011).
[50] H.W. Cohen, S.M. Hailpern, M.H. Alderman, *Sodium Intake and Mortality Follow-Up in the Third National Health and Nutrition Examination Survey (NHANES III)*. 23 (9) J. Gen. Intern. Med. 1297-302 (Sep. 2008). Epub 2008 May 9.
[51] M.P.J. Vanderpump, J.H. Lazarus, P.P. Smyth, et al., *Iodine Status of UK Schoolgirls: A Cross-Sectional Survey*, 377 (9782) Lancet 2007-12 (June 11, 2011).
[52] B. Renneboog, W. Musch,, X. Vandemergel, M.U. Manto, *Mild Chronic Hyponatremia is Associated with Falls, Unsteadiness, and Attention Deficits*, 119 Am. J. of Med. 71.el-71el 8 (2006).

i. This study demonstrated that mild, chronic hyponatremia in the elderly resulting from low-salt diets induce a high incidence of falls, possibly as the result of marked gait and attention impairments.

h) hyponatremia[53] [54]
   i. Mild hyponatremia even within the normal sodium range and hypernatremia are both associated with increased total mortality and major CVD events in older men without CVD which is not explained by known adverse CV risk factors.

i) death[55]
   i. This multi-year study on a very large cohort concluded that lower salt intakes resulted in higher morbidity and mortality.

As we have explained in prior comments, the evidence of the health outcomes of diets reduced in sodium show no benefit in terms of reduced mortality[56] and we now remind you that the single controlled trial of this hypothesis found that subjects in the salt-reduced group of the cohort had a considerably greater incidence of mortality and more frequent re-hospitalization.[57]

Because of the mistaken understanding that a reduction in salt intake will reduce blood pressure, which will in turn reduce cardiovascular events, the gold standard for dietary interventions for post-heart failure patients is a low sodium diet. However, the most recent evidence indicates that post-heart failure patients placed on low-sodium diets tend to die or are readmitted to hospital in far greater numbers than those that have not been placed on low-sodium diets.[58] [59] [60] Further,

---

[53] Wannamethee S, Shaper A, Lennon L, Papacosta O, Whincup P, *Mild hyponatremia, hypernatremia and incident cardiovascular disease and mortality in older men: a population-based cohort study*. Nutrition, Metabolism and Cardiovascular Diseases (2015), doi: 10.1016/j.numecd.2015.07.008.

[54] Sterns RH. *Disorders of Plasma Sodium — Causes, Consequences, and Correction*. N Engl J Med 2015; 372:55-65 DOI: 10.1056/NEJMra1404489.

[55] K. Stolarz-Skrzypek, T. Kuznetsova, L. Thijs, et al., *Fatal and Nonfatal Outcomes, Incidence of Hypertension, and Blood Pressure Changes in Relation to Urinary Sodium Excretion*, 305(17) JAMA. 1777-85 (May 4, 2011).

[56] M.H. Alderman, et al., *Dietary Sodium Intake and Mortality: The National Health and Nutrition Examination Survey (NHANES I)*, 351 Lancet 781-85 (1998); H. Cohen, et al., *Sodium Intake and Mortality in the NHANES II Follow-Up Study* 119 Am. J. of Med. 275 (2006); H.W. Cohen, S.M. Hailpern, and M.H. Alderman, *Sodium Intake and Mortality Follow-Up in the Third National Health and Nutrition Examination Survey (NHANES III)*, J. Gen. Intern. Med., (2008); DOI: 10.1007/s11606-008-0645-6 and Michael H. Alderman, *Presidential Address: 21st Scientific Meeting of the International Society of Hypertension: Dietary Sodium and Cardiovascular Disease: the 'J'-Shaped Relation*, 25(5) J. of Hypertension 903-907 (2007).

[57] S. Paterna; P. Gaspare; S. Fasullo; F.M. Sarullo; P. Di Pasquale, *Normal-sodium diet compared with low-sodium diet in compensated congestive heart failure: is sodium an old enemy or a new friend?, 114* Clinical Science 221-30 (London) (2008) (ISSN: 1470-8736); S. Paterna, et al., *Medium Term Effects of Different Dosage of Diuretic, Sodium, and Fluid Administration on Neurohormonal and Clinical Outcome in Patients With Recently Compensated Heart Failure,* Am. J. Cardiology 93–102 (2009).

[58] H.W. Cohen, S.M. Hailpern, and M.H. Alderman, *Sodium Intake and Mortality Follow-Up in the Third National Health and Nutrition Examination Survey (NHANES III)*, J. Gen. Intern. Med., DOI: 10.1007/s11606-008-0645-6, (May 18, 2008).

[59] S. Paterna, P. Gaspare, S. Fasullo, F.M. Sarullo, P. Di Pasquale, *Normal-Sodium Diet Compared With Low-Sodium Diet in Compensated Congestive Heart Failure: Is Sodium an Old Enemy or a New Friend?* , 114 Clinical Sci. 221-30 (London) (2008).

recent research indicates that there may indeed be very negative consequences if the diet limits sodium to the range of less than 2,300 mg Na/day as recommended in the *Dietary Guidelines*.[61] [62]

In addition to the compelling evidence related to the RAS and other negative impacts of a low-salt diet, the DGAC failed to address other studies that linked lowered salt intakes to a variety of health problems, including low-birth weights[63] and cognitive impairment[64] in children. Also ignored were peer-reviewed studies that demonstrated increased rate of falls[65] and fractures among the elderly,[66] another nutritionally susceptible segment of society. In assisted living facilities, where all residents are given low-salt diets, the rate of falls and fractures are three times as great as in the normal home environment.[67] We are left to surmise that the DGAC failed to consider this evidence because it did not fit within its justification for its predetermined conclusions.

Additional scientifically-derived clinical evidence continues to be published. In a recent study, published in the *Journal of the American Medical Association*, researchers studying 4,000 patients over 8 years found that lower sodium consumption was associated with an increased risk of cardiovascular mortality, while higher sodium consumption did not correspond with increased risk of hypertension or cardiovascular disease complications.[68]

### C. The DGAC Failed to Consider Evidence Related to the Reality that there is a Physiological Sodium Appetite

In furtherance of its salt reduction program, dating back to the first set of *Dietary Guidelines*, Americans have been cautioned, then warned, about alleged dangers in high salt intakes.[69]

---

[60] S. Paterna, G. Parrinello, S. Cannizzaro, S. Fasullo, D. Torres, F.M. Sarullo, and P. Di Pasquale, *Medium Term Effects of Different Dosage of Diuretic, Sodium, and Fluid Administration on Neurohormonal and Clinical Outcome in Patients With Recently Compensated Heart Failure*, 103 Am. J. Cardiology 93-102 (2009).

[61] Y. Shapiro, M. Boaz, Z. Matas, A. Fux, and M. Shargorodsky, *The Association Between the Renin-Angiotensin-Aldosterone System and Arterial Stiffness in Young Healthy Subjects*, 68(4) Clinical Endocrinology 510-12 (Apr. 2008).

[62] Edna R. Nakandakarea, Ana M. Charfa1, Flávia C. Santosa1, Valéria S. Nunesa, Katia Ortegab, Ana M.P. Lottenberga, Décio Mion Jr.b, Takamitsu Nakanoc, Katsuyuki Nakajimac, Elbio A. D'Amicod, Sergio Catanozia, Marisa Passarellia, Eder C.R. Quintãoa, *Dietary Salt Restriction Increases Plasma Lipoprotein and Inflammatory Marker Concentrations in Hypertensive Patients*, 200(2) Atherosclerosis 410-16 (Oct. 2008).

[63] A. Shirazki, Z. Weintraub, D. Reich, E. Gershon, M. Leshem, *Lowest Neonatal Serum Sodium Predicts Sodium Intake in Low Birth Weight Children*, 292(4) Am. J. Physiol. Regul. Integr. Comp. Physiol. R1683-89 (Apr. 2007). Epub 2006 Dec 14.

[64] J. Al-Dahhan, L. Jannoun, G.B. Haycock, *Effects of Salt Supplementation of Newborn Premature Infants on Neurodevelopmental Outcome at 10–13 Years of Age*, 86 Arch. Dis. Child Fetal Neonatal Ed. 120–123 (2002).

[65] B. Renneboog, W. Musch, X. Vandemergel, M.U. Manto, G. Decaux, *Mild Chronic Hyponatremia is Associated With Falls, Unsteadiness, and Attention Deficits*, 119(1) Am. J. Med. 71.el – 71.el8 (Jan. 2006).

[66] F. Gankam Kengne, C. Andres, L. Sattar, C. Melot, G. Decaux, *Mild Hyponatremia and Risk of Fracture in the Ambulatory Elderly*, 101(7) QJMed. 583-88 (2008).

[67] H.K. Kamel, *Preventing Falls in the Nursing Home. Annals of Long Term Care*, (Sept. 5, 2008). Available at: http://www.annalsoflongtermcare.com/article/6319?page=0,0.

[68] K. Stolarz-Skrzypek, T. Kuznetsova, L. Thijs, et al., *Fatal and Nonfatal Outcomes, Incidence of Hypertension, and Blood Pressure Changes in Relation to Urinary Sodium Excretion*, 305(7) JAMA. 177-85 (May 4, 2011); *See supra note 35*.

[69] Americans' salt intakes are exactly average in the world.

Americans have been convinced that salt intake should be minimized. Polls show that public education campaigns have been successful. Food companies have developed thousands of reduced-sodium foods to cater to this demand and those foods are consumed today in amounts far greater than in 1980. The "sodium density" of the American diet has been steadily decreasing resulting in less sodium intake per calorie. The result, however, has been an unchanged level of sodium intake and an increase in caloric intake leading to obesity.

**Although certainly not a primary cause, the continued promotion of salt reduction found in the recommendations in the *Dietary Guidelines* and the anticipated call by the FDA for voluntary salt reductions in food products will likely worsen, not improve, the ongoing obesity crisis because people will consume more calories just to satisfy their innate salt appetite.** Decades of animal feeding experience serve as a foundation for this statement. In addition, the most recent UK Food Standards Agency[70] survey demonstrated that despite the food industry reducing salt significantly (10-25%) in their processed food formulations, people still consume the same amount of salt, indicating they are voluntarily adding more with the shaker or are simply eating more food (and calories) to satisfy their need for sodium.

As we have reported in our comments, there is scientific evidence of a non-behavioral, neurally-mediated "salt appetite."[71] [72] Nowhere in the record created in support of the *Dietary Guidelines* were we able to find any serious consideration of this scientific evidence even though we shared it with the DGAC. Other recent studies support findings that there is a non-behavioral, neurally-mediated "salt appetite," including a recent study in *The Clinical Journal of the American Society of Nephrology* that indicates that physiology, not public policy, will determine a human's daily sodium intake. This research should have been considered by the DGAC because it undercuts the hypothesis that salt intake can be controlled by regulators rather than by nature. The study, *Can Dietary Sodium Intake be Modified by Public Policy?*,[73] analyzed existing research to determine whether sodium or salt intake follows a pattern consistent with a range set by the brain to protect normal functions of organs such as the heart and kidney. The analysis is based upon 19,151 subjects studies in 62 previously-published surveys and reflects the differing "food environments" of 33 countries. The data reported documents that humans have a habitual sodium intake in the range of 2800 to 4600 mg/day -- with an average intake of 3,600 mg/day. Currently, the U.S. citizens consume an average of about 3,400 mg/day of salt.[74]

Taken in combination, these two studies strongly suggest that salt/sodium intake is a neurally-determined salt appetite signaled unconsciously from the brain and not the product of taste, labeling, consumer education, nor of the availability of low-sodium alternative products. A

---

[70] Salt intakes remain static in Scotland - June 22, 2011 accessible at: http://www.food.gov.uk/news/newsarchive/2011/june/salt.

[71] J.C. Geerling, and A.D. Loewy, *Central Regulation of Sodium Appetite*, 93(2) Exp. Physiol. 177-209 (Feb 2008).

[72] Evans LC, Ivy JR, Wyrwoll C, et al. *Conditional Deletion of Hsd11b2 in the Brain Causes Salt Appetite and Hypertension*. Circulation. 2016 Apr 5;133(14):1360-70. doi: 10.1161/CIRCULATIONAHA.115.019341. Epub 2016 Mar 7.

[73] David A. McCarron, Joel C. Geerling, Alexandra G. Kazaks, and Judith S. Stern, *Can Dietary Sodium Intake be Modified by Public Policy?*, 4 Clinical J. of the Am. Soc of Nephrology 18788-82 (2009). Available at: http://cjasn.asnjournals.org/cgi/reprint/CJN.04660709v1.

[74] This is consistent with the conclusion of renowned Swedish researcher, Björn Fokow, who described a "hygienic safety range" for sodium of 2,300 mg/day to 4,600 mg/day – recognizing that it could be as high as 5,750 mg/day. *See* Bjorn Folkow, *News in Physiological Sciences* (1990).

needs-based salt appetite suggests that whatever the *Dietary Guidelines* may say about salt intake, physiology will prevail over the opinions of policy makers.

### D. The DGAC Failed to Address Conflicting and Inconsistent Evidence Related to the Impact of Sodium Intake on Blood Pressure

As we have previously explained to the DGAC, some recent research carried out on the issue of salt and health casts a significant shadow over the DGAC's predetermined conclusion that reducing salt intake to the degree prescribed necessarily leads to significant reductions in blood pressure. While we are in full agreement with the potential health benefits of reducing blood pressure for those in our population that require it, the means of doing so should have its intended consequence and not provoke the development of negative biomarkers or cause harm of any kind.

Reduction of sodium intake to the 2,300 mg/day level does not conform to those needs. In the first instance, the intended impact on the target population is not highly significant as stated in the 2003 Cochrane review[75] and restated once again in 2008.[76] While salt reduction may result in a minor reduction in blood pressure for some portion of the population, a reduction in salt intake increases the blood pressure of another significant portion of the population.[77] [78] [79] In light of these findings, if such a recommendation were to be made, surely the proviso must be given that a particular segment of the population will experience an increase in blood pressure. However, as with much of the other scientific and medical evidence provided in both our submitted comments and in this letter, it appears that no attention has been given to these studies by the DGAC in its dogged efforts to justify its predetermined conclusions.

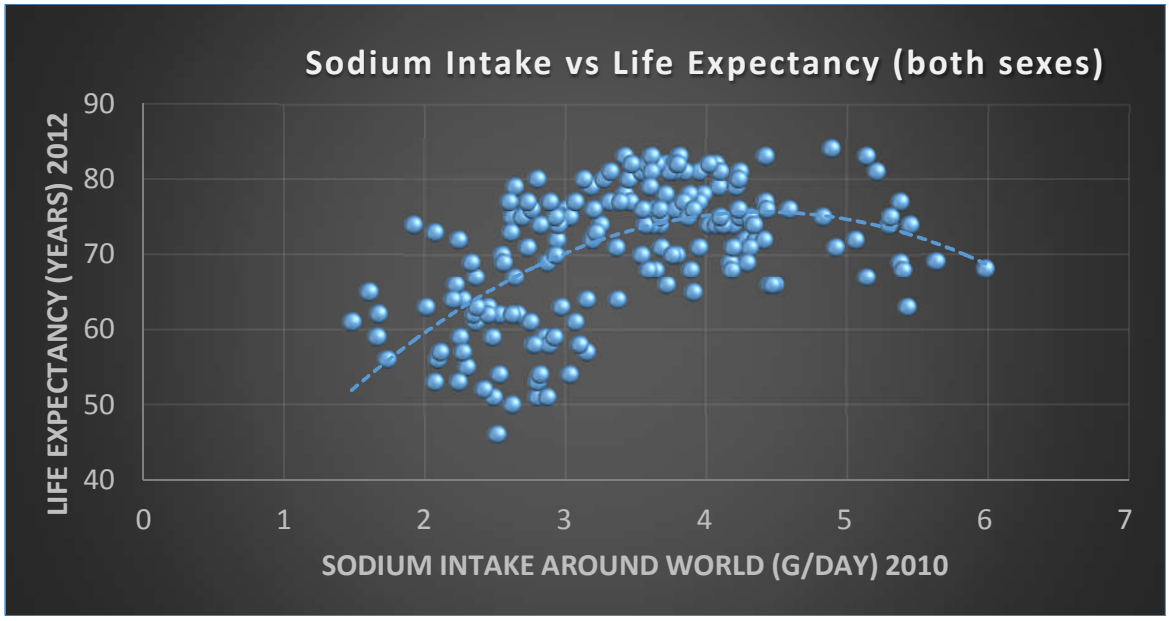
### E. Despite the Preponderance of Evidence that Population-Wide Sodium Reductions Are Not Warranted, the FDA Plans to Ask for Voluntary Salt Reductions in Food Products

In response to long-term pressure from the Center for Science in the Public Interest (CSPI), the FDA appears set to ask the food industry for voluntary salt reductions in food products. This is not a response to scientific evidence but an effort to appease CSPI. If the scientific evidence was there to support such an effort, the call for voluntary salt reductions in food products would have occurred years ago. As it happens, during the last 5-10 years, by far the preponderance of evidence has mitigated <u>against</u> population-wide salt reduction.

---

[75] L. Hooper, C. Bartlett, G. Davey Smith, S. Ebrahim, *Reduced Dietary Salt for Prevention of Cardiovascular Disease*, The Cochrane Library (2003), Issue 1.
[76] L. Hooper, C. Bartlett, G. Davey Smith, S. Ebrahim., "*Advice to Reduce Dietary Salt for Prevention of Cardiovascular Disease (Review)*," The Cochrane Library (2008), Issue 4.
[77] F.C. Luft, L.I. Rankin, R. Block R, et al., *Cardiovascular and Humoral Responses to Extremes of Sodium Intake in Normal Black and White Men*, 60 Circulation 697-706 (1979).
[78] J.Z. Miller, M.H. Weinberger, S.A. Daugherty, et al., *Heterogeneity of Blood Pressure Responses to Dietary Sodium Restriction in Normotensive Adults*, 40 J. Chronic. Dis. 245-50 (1987).
[79] F.C. Luft, D.A. McCarron, *Heterogeneity of Hypertension: The Diverse Role of Electrolyte Intake,* 42 Annu. Rev. Med. 347-55 (1991).

A call for voluntary salt reduction in food products holds clear dangers for consumers. In the first instance, consumers may not always be able to voluntarily make up for the salt removed from food product formulations. They will therefore be placed at a lower level of salt consumption, which has been shown (using World Health Organization data[80] [81]) to result in reduced life expectancy, reduced Health Adjusted Life Expectancy (HALE) and poorer health outcomes.




Reduced salt in food formulations may have a negative impact on the overall diet. Amongst the most important foods in a good diet are salads and vegetables. These items contained nutritious yet, somewhat bitter, phytonutrients. The bitterness in these products is naturally and routinely mitigated with salt or other foods containing an appropriate level of salt. Reducing the salt in food formulations may, at the same time, make complementary salads and vegetables less palatable. This would reduce the overall quality of the diet.

Reducing salt in food formulations cannot be done in isolation as food must be made acceptable to consumer tastes. Therefore, salt reduction must be accompanied by complex formulation changes that will satisfy the consumer's palate. While some of the larger multinational food companies have developed the complex technology to do so, smaller food companies will be placed at a disadvantage. Most importantly, consumers themselves will be at greater risk because, while their taste perception may be deceived, metabolic physiology will not. Reduced salt intake will result in increased plasma renin, angiotensin, aldosterone activity which will, in turn, result in a cascade of reduced health outcomes.

[80] Powles J, Fahimi S, Micha R, et al. *Global, regional and national sodium intakes in 1990 and 2010: a systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide*. BMJ Open 2013;3:e003733. doi:10.1136/bmjopen-2013-003733.
[81] World Health Organization Global Health Observatory. Accessible at http://www.who.int/gho/en/.

Finally, food products made in the US may be at a disadvantage to more traditional, imported foods, which are routinely preserved with salt, such as cheeses, hams and salamis. Many of these products have long established recipes and formal geographic designations, such as Parmesan and Gorgonzola cheeses from the Mediterranean Region, where cardiovascular health metrics are considered to be excellent. US products with reduced salt formulations may not be as competitive.

## III. Conclusion

It is troubling that the Agencies have, to this point, adopted a mentality of continuous justification of a preordained conclusion rather than doing their statutory duty and setting standards based upon a rigorous assessment of all available scientific and medical evidence. However, we encourage you to change this practice and abandon the sodium provisions in the *Dietary Guidelines* in favor of an open, transparent rulemaking proceeding. Continuing to build policy and regulation on a fatally flawed foundation is both bad government and does nothing to protect our citizenry.

We hope that you will agree that the portions of the *2010 Dietary Guidelines* and the *2015-2020 Dietary Guidelines* that pertain to sodium are fatally flawed and should be withdrawn because they are not based on a preponderance of the scientific and medical evidence. The Agencies must withdraw those portions of the *Dietary Guidelines* in order to meet their statutory mandate. We also suggest that the expected FDA efforts to have voluntary salt reductions in food products be abandoned. Failure to take this action would be against the interests of consumers given the admissions made regarding the flawed foundation of the sodium provisions of the *Dietary Guidelines*, the impropriety of the process used by the DGAC in justifying the sodium provisions in the *Dietary Guidelines*, and the lack of consideration of the current science and medical evidence, including the evidence of harm that will be caused by the sodium provisions in the *Dietary Guidelines*.

Thank you for your attention and consideration.

Sincerely,

Lori Roman

Lori Roman
President, Salt Institute