



Quelle consommation de sel idéale recommander dans une alimentation équilibrée?

Professeur JM Krzesinski

Néphrologie

CHU Liège



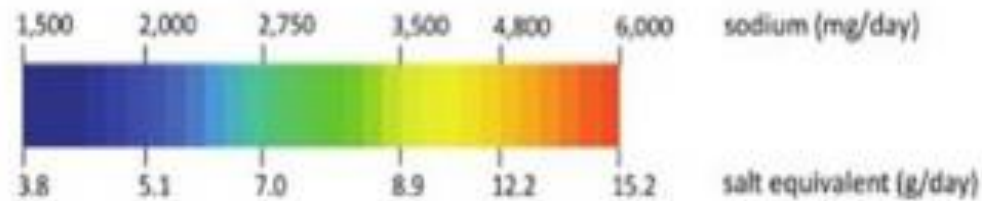
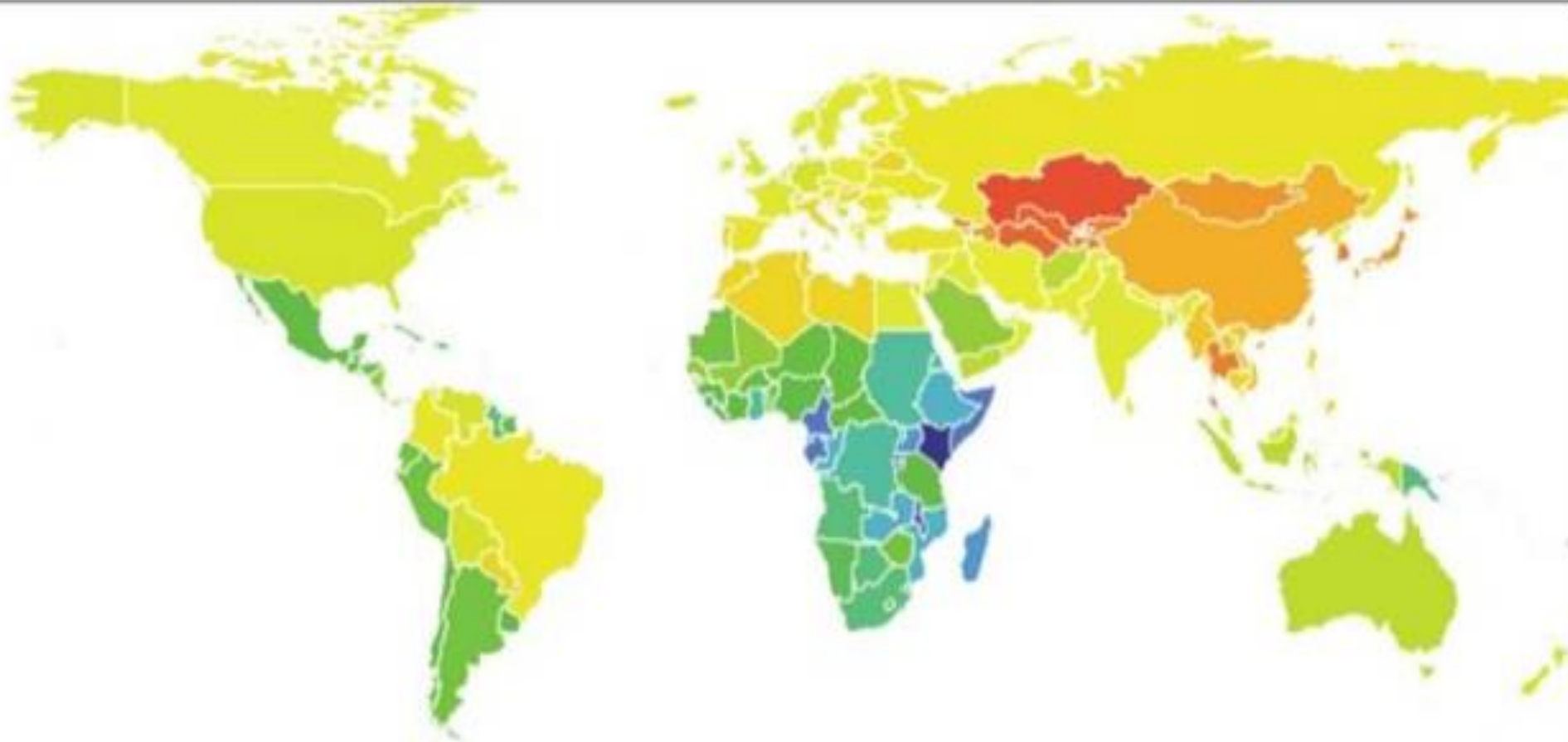
Salt and Your Health

Apport en sel, de quoi parle-t'on?

L'apport quotidien conseillé est de 5 g de sel (ou chlorure de sodium ou NaCl)

Soit 2 g ou 85 mmoles de sodium (Na^+) ou encore une cuillère à café de sel.

Consommation sodée selon les pays en 2010



Mean global sodium intakes in 2010, by country worldwide (from Powles et al., 2013).

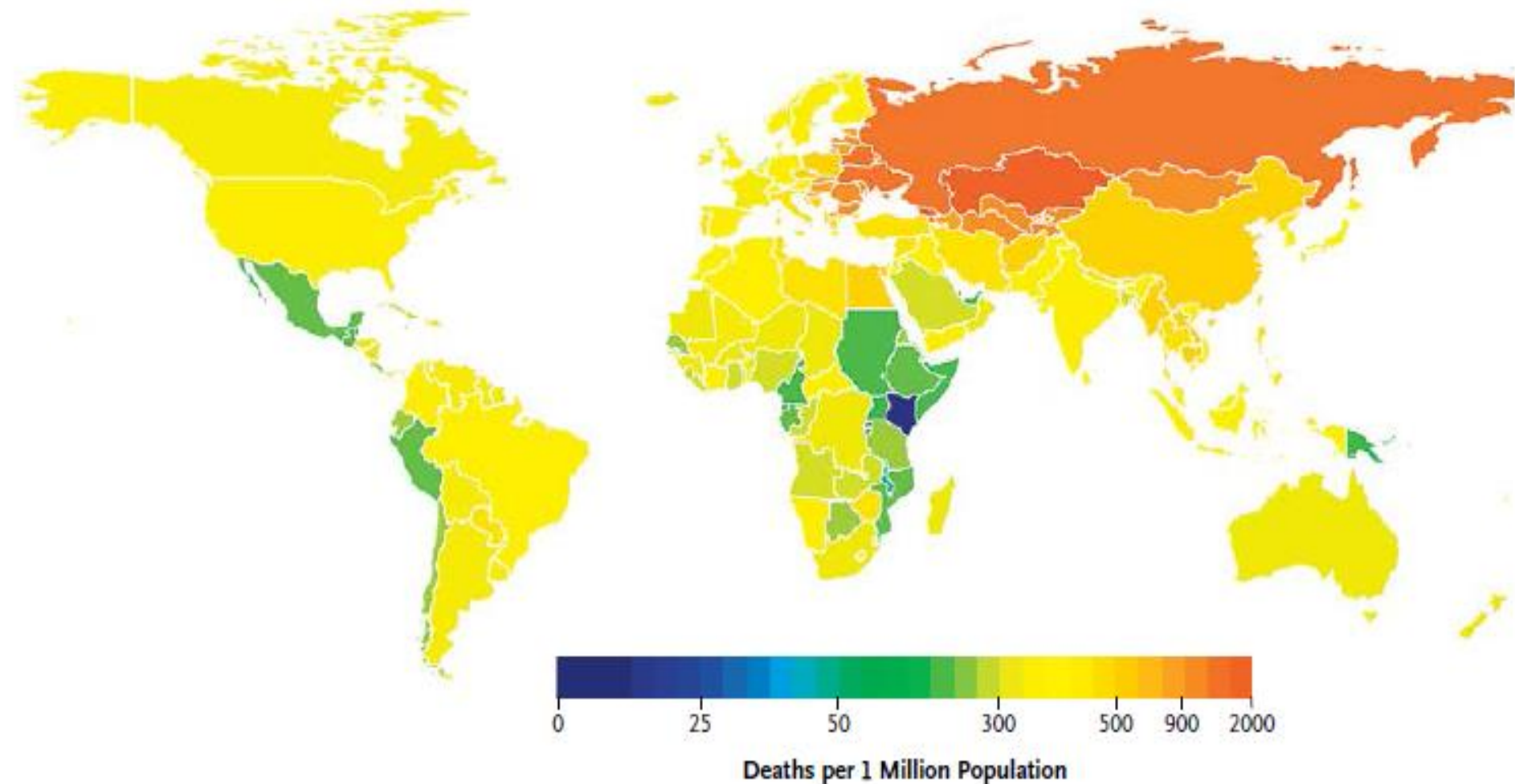
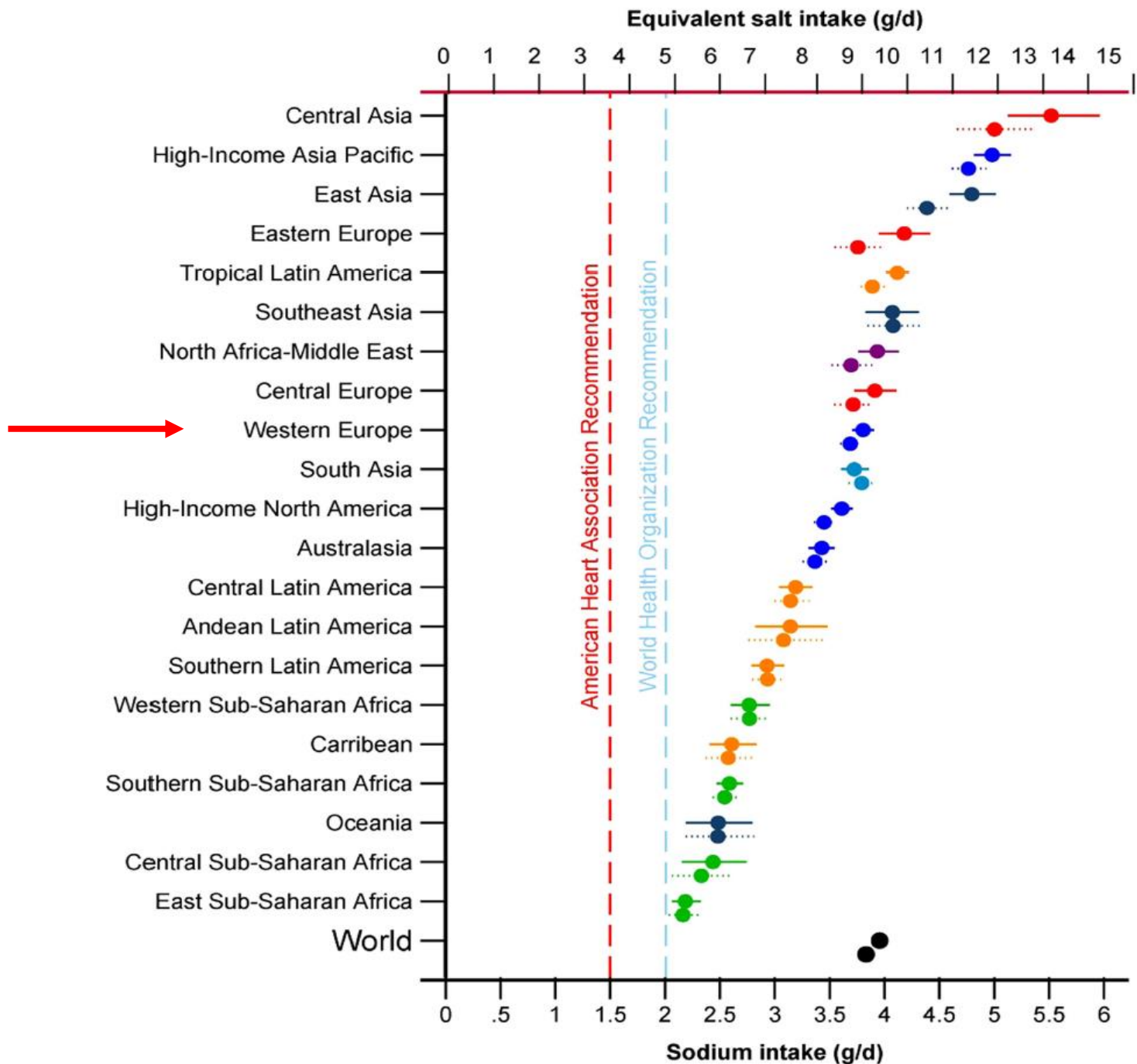


Figure 3. Absolute Cardiovascular Mortality Attributed to Sodium Consumption of More than 2.0 g per Day in 2010, According to Nation. The scale is based on the number of deaths from cardiovascular causes (per 1 million persons) in 2010 that were attributed to sodium consumption of more than 2.0 g per day.



Mean (95% uncertainty interval) age-standardised sodium intakes (g/day) in 1990 and 2010



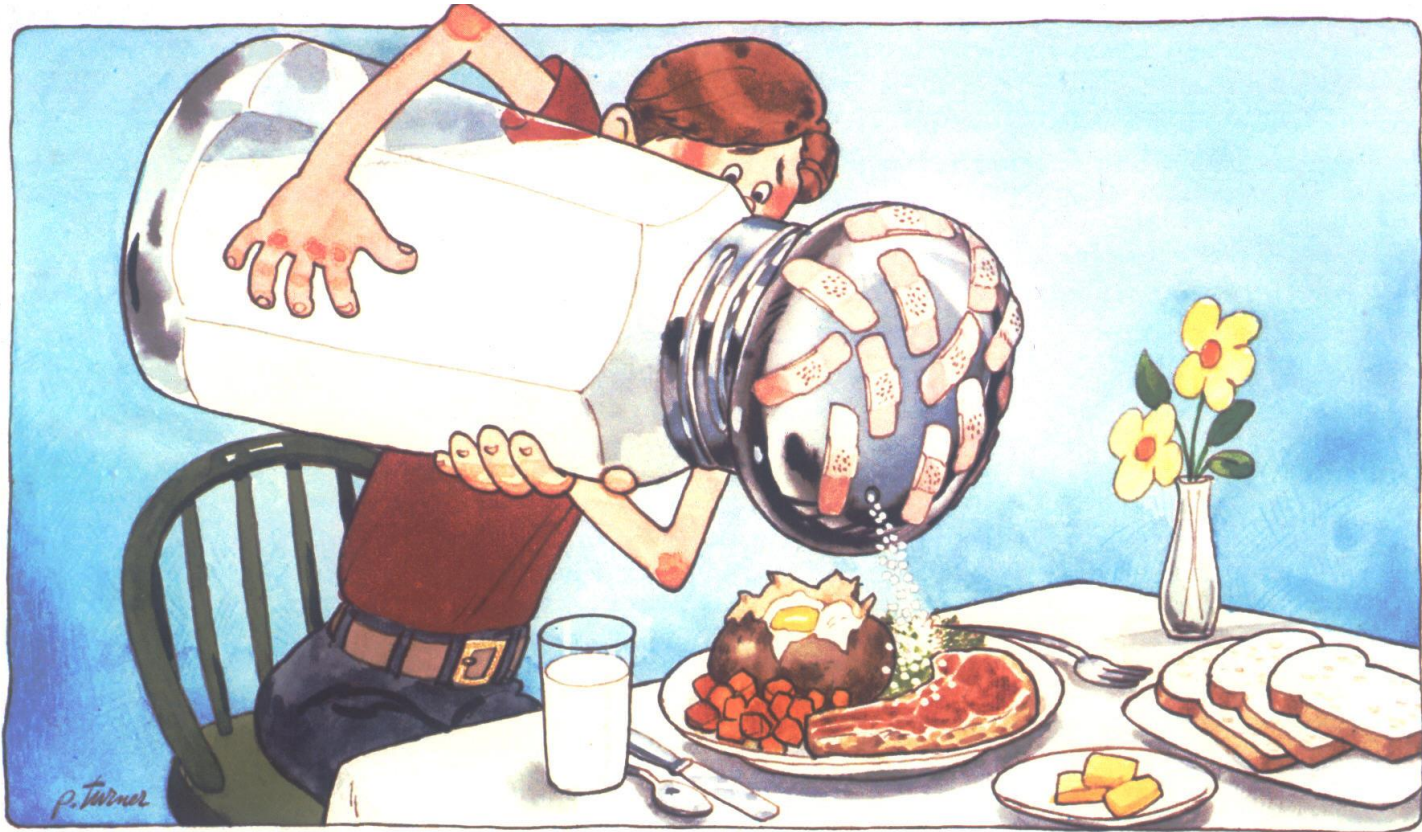
**Conseil
Supérieur de la Santé**

RECOMMANDATIONS ALIMENTAIRES POUR LA POPULATION BELGE ADULTE - 2019

Groupes alimentaires à limiter

1. Viandes rouges transformées et volailles transformées
 2. Viandes rouges, excepté les volailles
 3. Boissons avec sucres ajoutés
 4. Matières grasses riches en acides gras saturés athérogènes / acides gras trans
 5. Produits sucrés
 6. Produits salés
-

Quelle quantité de sel proposer dans une alimentation équilibrée?



Reduce your sodium intake, and you'll soon find you crave less salt.

Développement d'un intérêt pour le sel

- A l'âge de la pierre, consommation de NaCl: **700mg/j**
- Depuis 10000 ans, utilisation pour la conservation des aliments (élément stratégique)
- Développement d'un commerce pour le sel (or blanc), qui devient le symbole d'amitié, de loyauté, d'hospitalité
- A l'époque romaine, consommation de **25g/j** (préservation de la santé: salus, salubritas) et utilisation comme salaire (salarium, ration pour le sel)
- Au Moyen-Age, apparition en France d'une taxe royale sur le sel (Gabelle: 6% des revenus royaux) et apparition de la salière.



Figure 1. Salt cellar made of gold, enamel, and ebony by Benvenuto Cellini (1500 to 1571) and presented in 1543 as gift to Francois I, King of France (1494 to 1547). The salt cellar, valued at approximately US \$55 million, was stolen in May 2003 from the Kunsthistorisches Museum, Vienna, Austria. It was recovered undamaged by the Austrian police in January 2006. (Photograph obtained from unrestricted web page of FBI Top Ten Art crimes. Available at: <http://www.fbi.gov/hq/cid/arttheft/topten/cellini.htm>. Accessed April 11, 2007.)

**Au contraire de l'ère ancienne,
le sel ne fait pas du tout défaut
dans l'alimentation d'aujourd'hui!**



Régulation de la balance sodée

- L'apport quotidien actuel en NaCl avoisine en moyenne les 8-9 g/j en Belgique mais cela peut varier de 2 à 25 g de NaCl/j)
- La majorité du sodium est éliminée par les reins.
- La quantification de l'apport sodé s'effectue, le plus précisément, sur base de plusieurs récoltes d'urines de 24h!

Message is in the Measurement

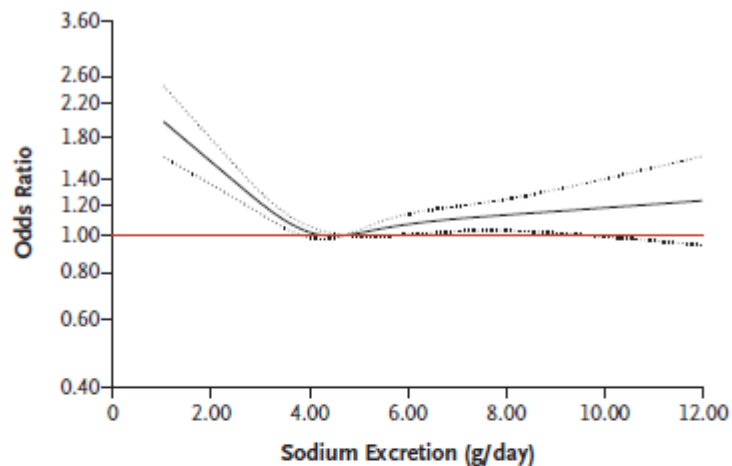
Hypertension. 2019;74:505-506.

90% of Sodium intake excreted in urine, but with variability by day, hour, posture;

Accuracy of urinary sodium measurement in individuals constrained by logistics, costs, need for large number of samples ⁵

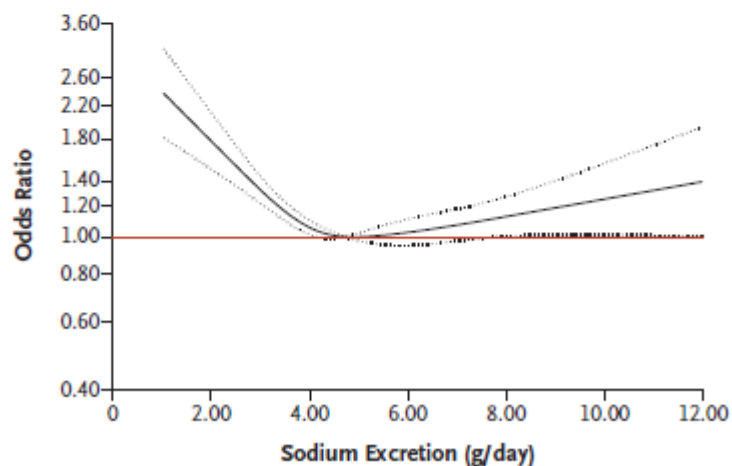
Sample Collection	Logistics	Costs	Accuracy	He et al. ⁴ analysis	Message	Conclusion
Spot urine 	Less complicated	low	Depends on formulas; formulas include factors which are themselves associated with mortality Validation: conflicted	Intrinsic bias and distortion of sodium uptake – mortality relationship	J- or U- shaped relationship of sodium intake to mortality with notable increase in mortality at estimated low dietary sodium intake	Potential harm with population-wide sodium reduction, especially at lowest levels of dietary sodium intake Public policy should recommend dietary sodium restriction only for populations at highest risk: persons with HBP, CVD, diabetes
24 hour urine 	Complicated	high	Depends on engagement of subjects and completion of collection; requires multiple measurements separated in time; highly accurate when properly performed	Linear relationship of sodium intake to mortality	Linear relationship between sodium intake and mortality, across wide range of dietary sodium intake	Dietary sodium restriction reduces cardiovascular morbidity across wide range of baseline dietary sodium intake Public policy should Recommend population-wide reduction in dietary sodium with ideal goal of about 1500 mg sodium

A Estimated Sodium Excretion and Risk of Death or Cardiovascular Events



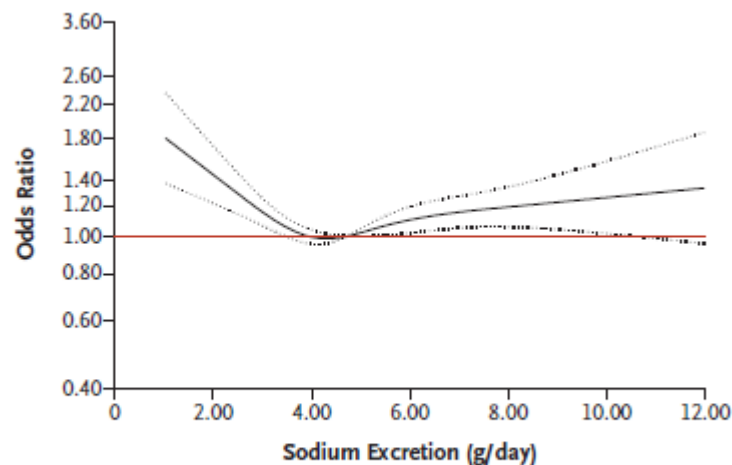
No. of Events	101	1,023	1,437	597	126	25
No. at Risk	1817	30,124	46,663	18,395	3885	756

B Estimated Sodium Excretion and Risk of Death from Any Cause



No. of Events	68	642	826	340	79	16
No. at Risk	1817	30,124	46,663	18,395	3885	756

C Estimated Sodium Excretion and Risk of Major Cardiovascular Events



No. of Events	57	602	869	369	75	13
No. at Risk	1817	30,124	46,663	18,395	3885	756

Figure 1. Association of Estimated 24-Hour Urinary Sodium Excretion with Risk of Death and Major Cardiovascular Events.

Assessing whether a spot urine specimen can predict 24-h urinary sodium excretion accurately: a validation study

TABLE 3. Comparison of estimated 24-h urinary sodium excretion from published equations^a

Equation	24-h USE (mean ± SD) (mg/day)	r_s	P value ^b	Mean difference ^c (95% CI) (mg/day)	RMS
Measured value	4558.25 ± 1908.75	Reference	Reference	Reference	Reference
Kawasaki	5326.52 ± 1556.29 ^d	0.332	0.002	768.26 (315.31–1221.22)	5.46
INTERSALT	3138.86 ± 688.00 ^d	0.352	0.001	–1419.39 (–1794.85 to –1043.93)	5.48
Tanaka	4460.33 ± 1154.00	0.525	<0.001	–97.92 (–469.19–273.34)	3.25
SunSMU	3604.62 ± 900.10 ^d	0.257	0.017	–953.63 (–1363.88 to –543.39)	4.94
SunPM	4344.09 ± 1041.70	0.407	<0.001	–214.02 (–605.70 to 177.66)	3.63

95% CI, 95% confidence interval; INTERSALT, International Cooperative Study on Salt, Other Factors, and Blood Pressure; r_s , Spearman correlation coefficient; RMS, residual mean square; SD, standard deviation; 24-h USE, 24-h urinary sodium excretion.

^aTwo participants did not void during afternoon periods, so the sample size of all equations was 85 except for the SunPM equation ($n = 83$).

^bThe significance test of correlation coefficient.

^cMean difference, estimated minus measured 24-h USE.

^dSignificantly different from measured 24-h USE assessed with Wilcoxon's Signed Rank test.

Conclusion: There is still no evidence to support that 24-h USE could be estimated accurately using the current equations, especially for the equations developed by casual spot urine specimens.

Table 1. Total Mortality in the Trials of Hypertension Prevention Cohorts Post-Trial Follow-Up by Categories of Measured 24-h UNa Excretion Among Individuals Not in a Sodium Reduction Intervention

Variable	Measured Sodium Excretion (mg/24 h)				P Trend	Hazard Ratios per 1000 mg/24 h	P Value
	<2300	2300–<3600	3600–<4800	≥4800			
Average measured 24-h UNa							
Deaths/total (%)	23/312 (7.4)	105/1182 (8.9)	93/979 (9.5)	51/538 (9.5)			
Hazard ratios (95% CI)	0.73 (0.45–1.20)	0.94 (0.70–1.26)	1 (reference)	1.09 (0.76–1.55)	0.204	1.12 (1.01–1.25)	0.032
First measured 24-h UNa							
Deaths/total (%)	44/440 (10.0)	71/811 (8.8)	57/693 (8.2)	68/716 (9.5)			
Hazard ratios (95% CI)	1.23 (0.81–1.86)	1.15 (0.80–1.64)	1 (reference)	1.20 (0.84–1.73)	0.850	0.99 (0.91–1.06)	0.703

From Cox proportional hazards regression models stratified by trial phase and adjusted for age, sex, race/ethnicity, clinic, treatment assignment, education status, baseline weight, alcohol use, smoking, exercise, and family history of cardiovascular disease. UNa indicates urinary sodium.

An accurate measurement of an individual's sodium intake is vital for epidemiological studies relating sodium intake to health outcome.³⁷ Twenty-four-hour UNa excretion is considered the gold standard method for assessing sodium intake. Despite this, 1 single 24-hour urine cannot be used to estimate an individual's usual intake because of the large day-to-day variations in sodium intake. To get a reasonably reliable estimate for an individual, a minimum of three 24-hour urine collections are needed.^{38–40} TOHP collected three to seven 24-hour urines during a period of 18 months to 4 years and demonstrated a direct linear relationship between sodium intake and mortality. However, replacing the accurately

Controverses quant à l'apport idéal en sel

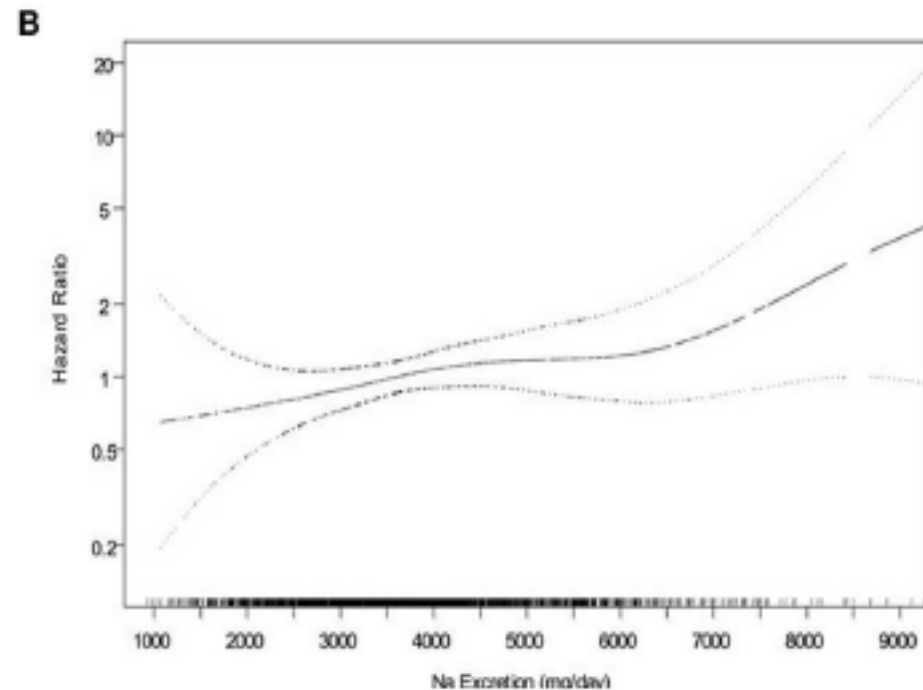
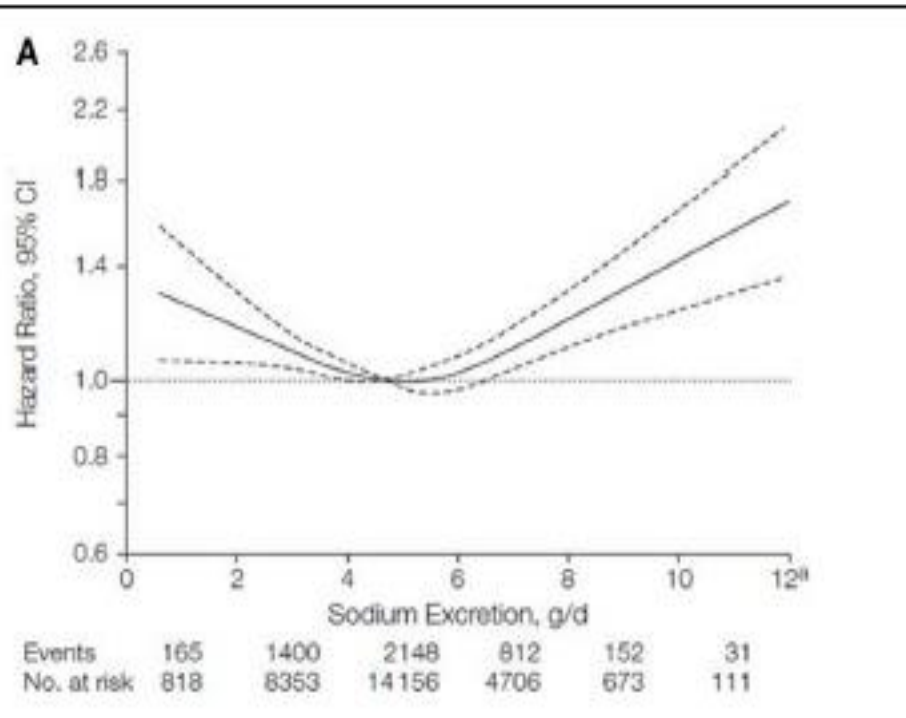
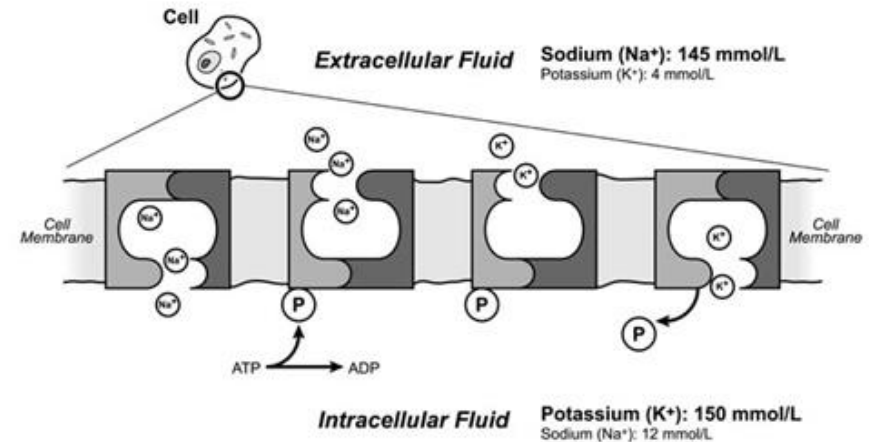


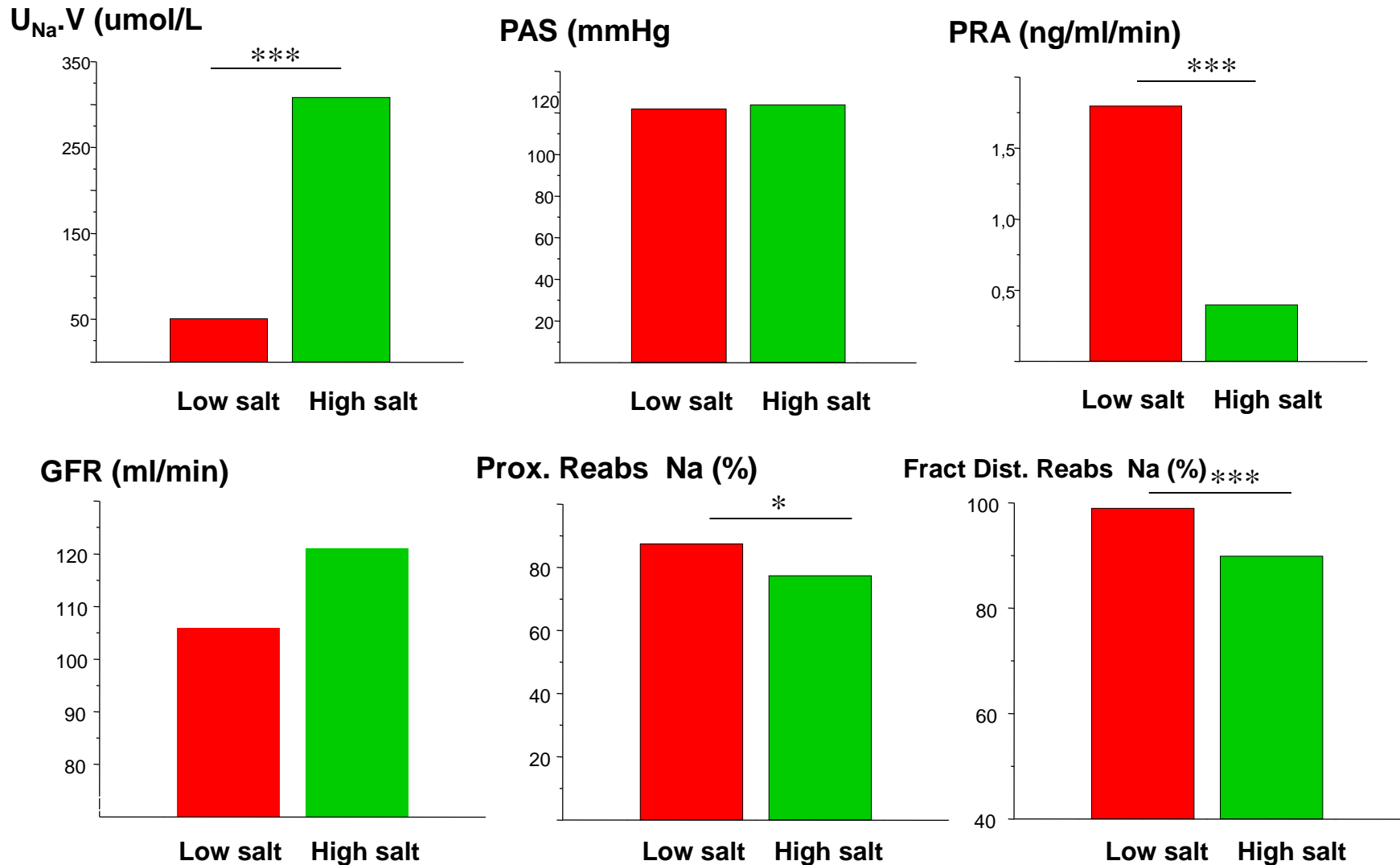
FIGURE 3 | Relationship between urinary salt excretion and the hazard ratio for cardiovascular events or death according to O'Donnell (A) suggesting a U-shape curve and according to Cook RN suggesting a linear relationship (B) (from O'Donnell et al., 2011; Cook et al., 2014).

Physiological role of sodium chloride

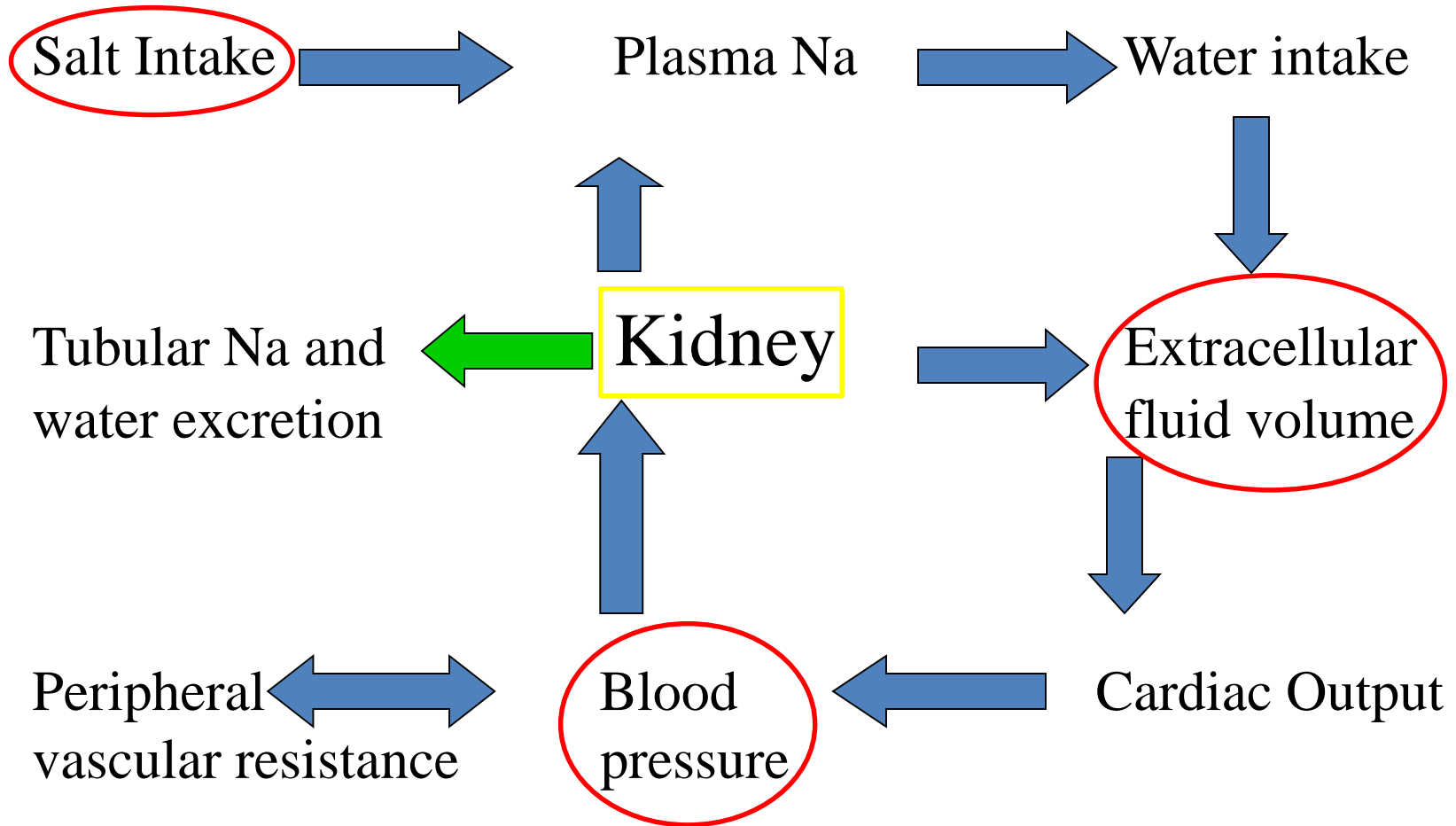
- 1. Maintenance of membrane potential
- 2. Nutrient absorption and transport
- 3. Maintenance of blood volume and blood pressure (importance of Chloride ion)



Normal renal response to a change in sodium intake



Role of the kidney in blood pressure control



Effets d'un excès alimentaire de sel

- Rôle sur la PA

Excess Salt Consumption and Essential Hypertension

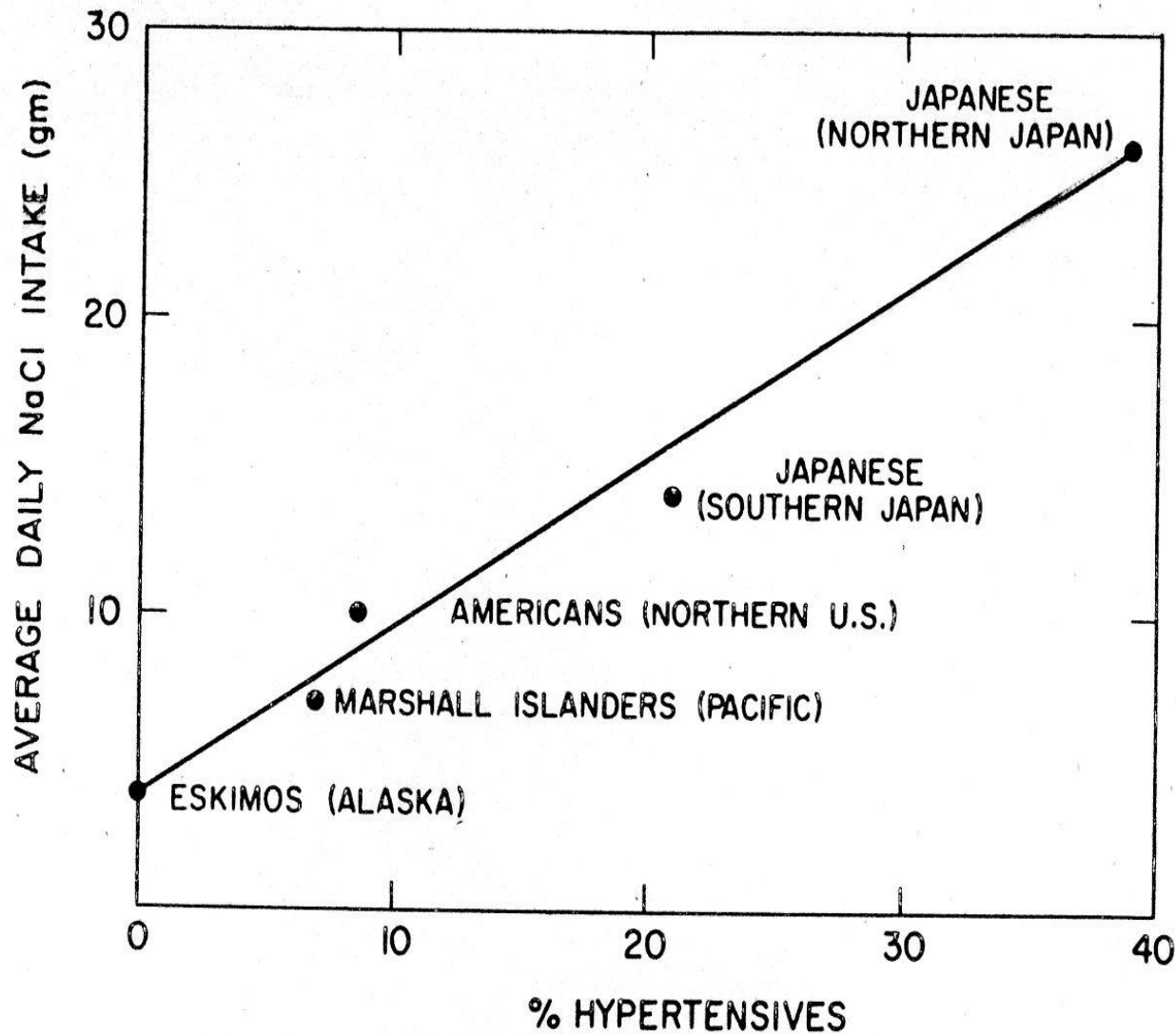


FIG. 1. Correlation of average daily salt (NaCl) intakes with prevalence of hypertension in different geographic areas and among different races. FROM: DAHL, L. K. In: Essential Hypertension. An International Symposium. Berlin, 1960. Springer-Verlag.

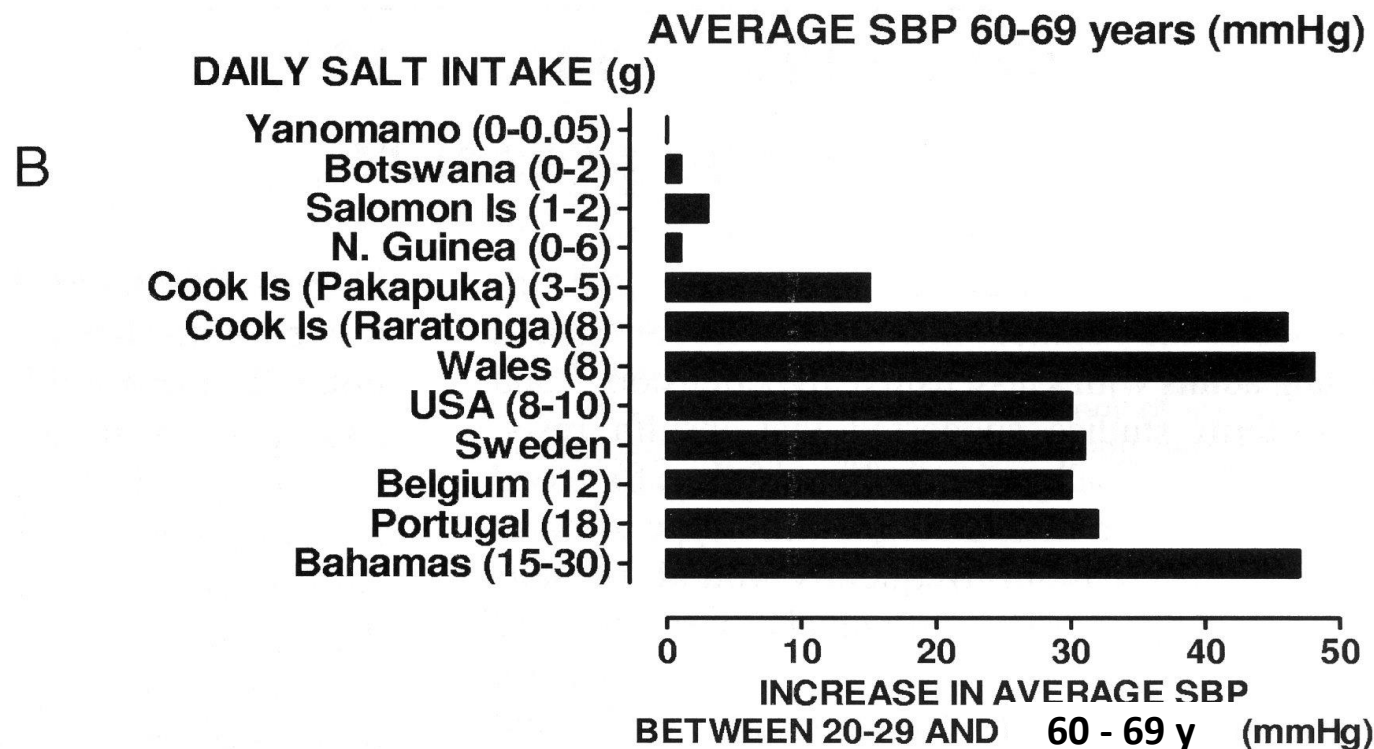
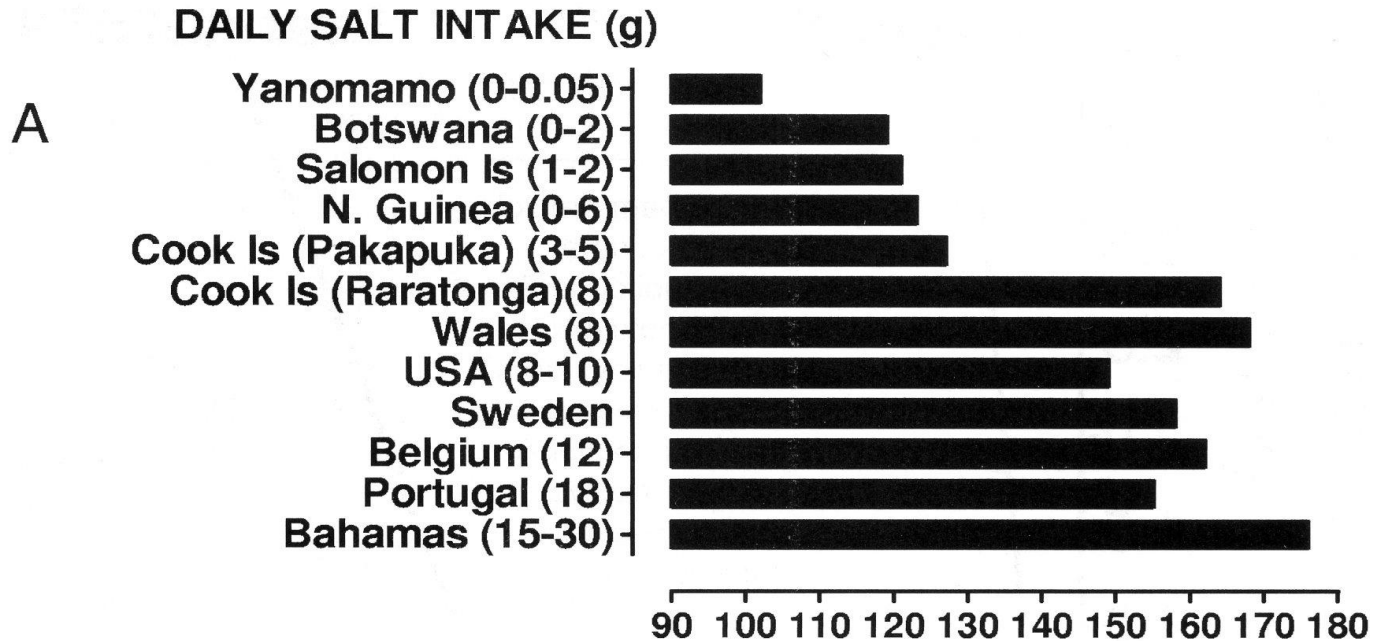
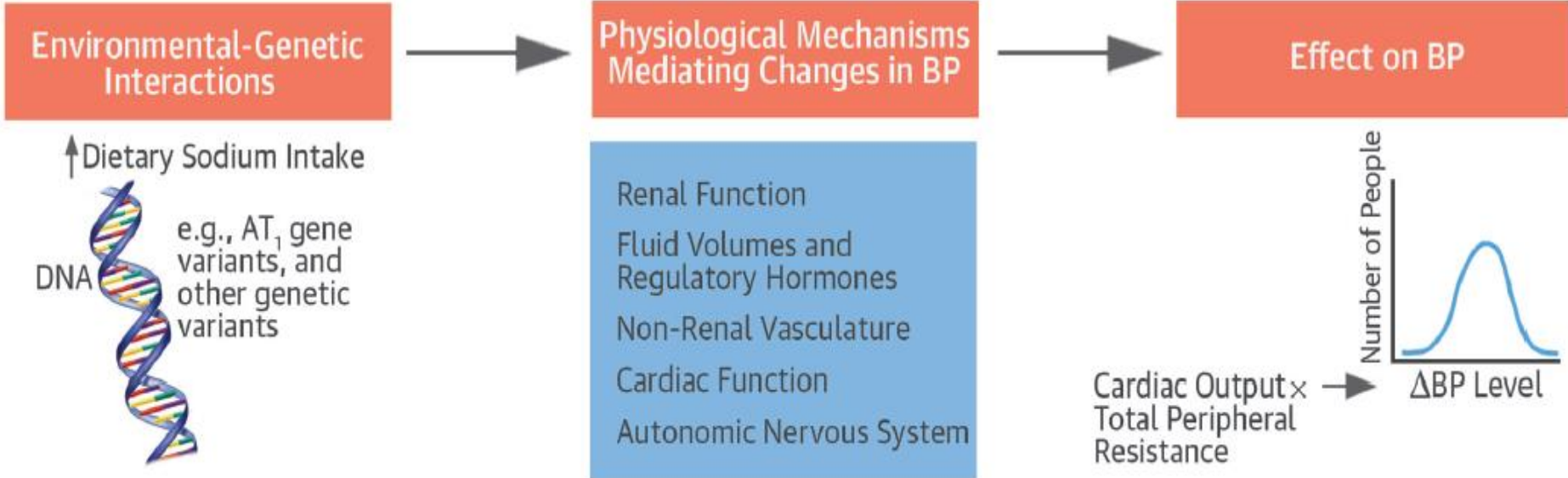


Figure 2. (A) Habitual daily salt intake and average systolic blood pressure (SBP) in selected populations 60 to 69 years old and (B) the difference in average SBPs at 20 to 29 years of age in the corresponding populations. More than 80% of hypertensive patients older than 60 years have salt sensitivity.⁴² (Data adapted from Meneton et al,¹¹ who quoted Joosens JV: Dietary salt restriction: The case in favor. R Soc Med Ser 26:243-250, 1980.)

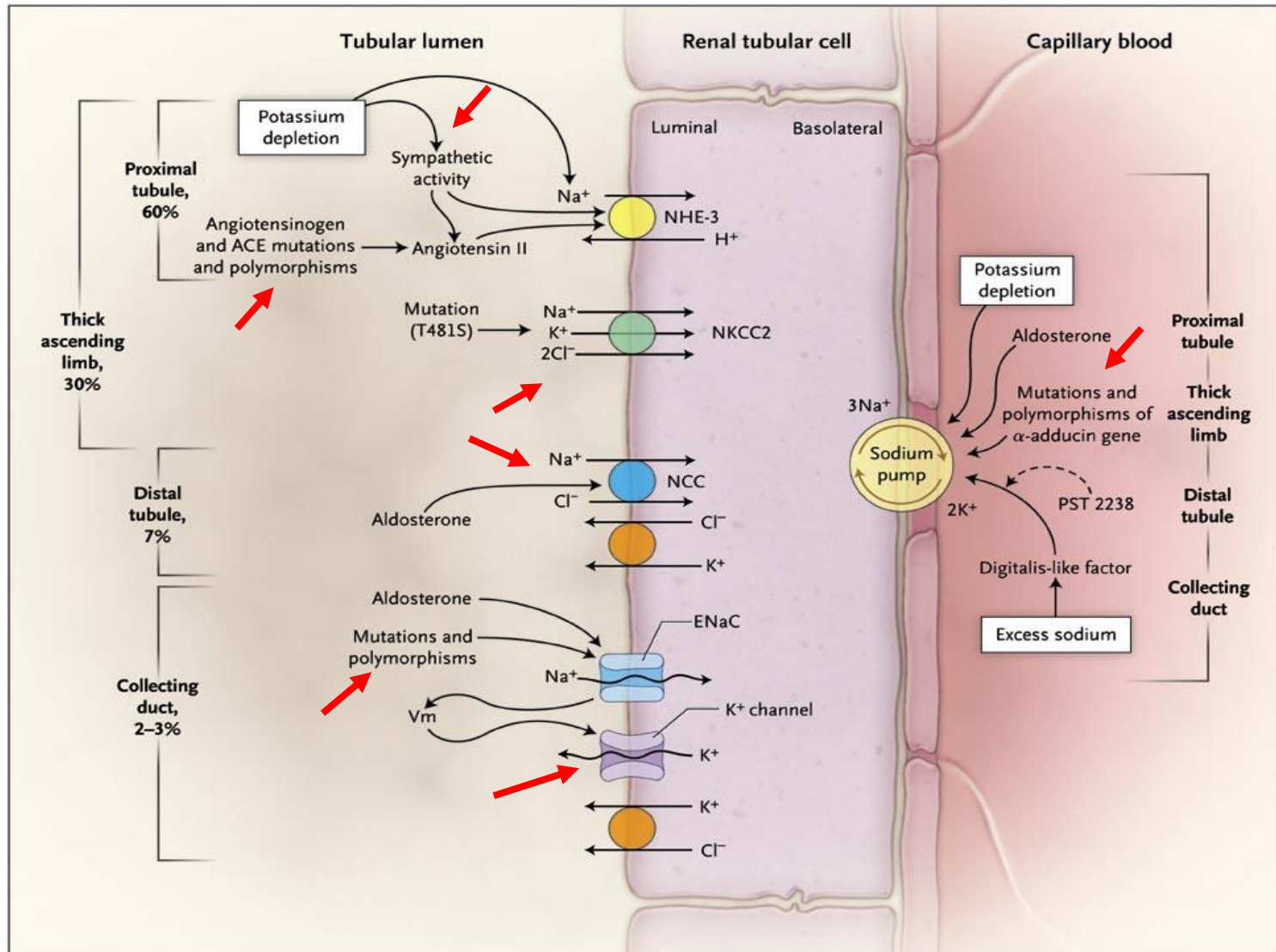
Mechanisms Mediating Dietary Sodium-Induced Alterations in BP



Farquhar, W.B. et al. J Am Coll Cardiol. 2015; 65(10):1042-50.

High dietary sodium can potentially exert its influence through various mechanisms to cause an increase in blood pressure (BP) through alterations in cardiac output and total peripheral resistance. The change (Δ) in BP varies considerably, even within a given population (as depicted in the distribution). AT₁ = angiotensin II receptor type 1.

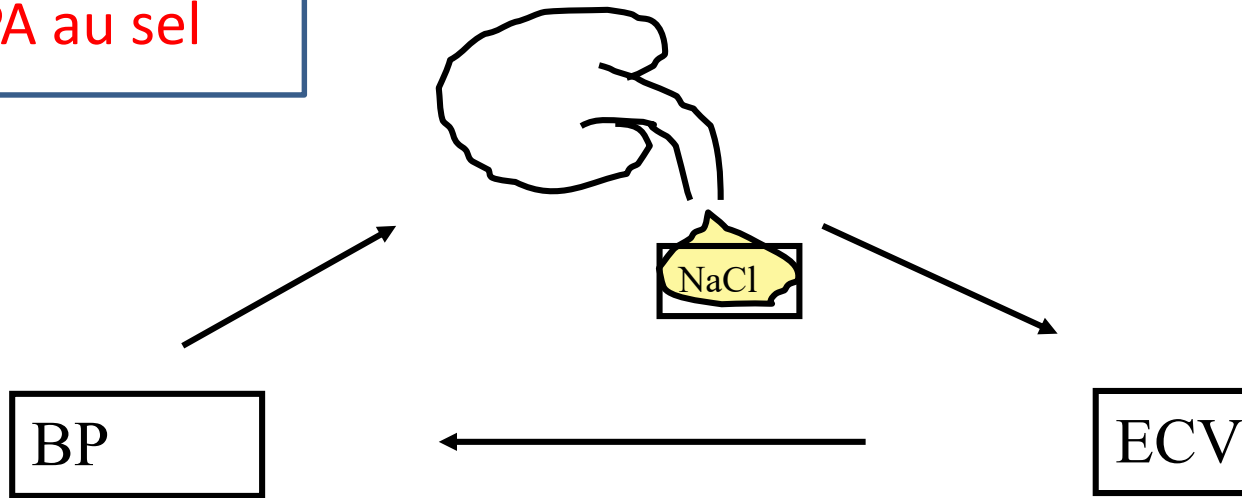
Mechanisms Implicated in the Retention of Sodium and Loss of Potassium by the Kidneys in Primary Hypertension



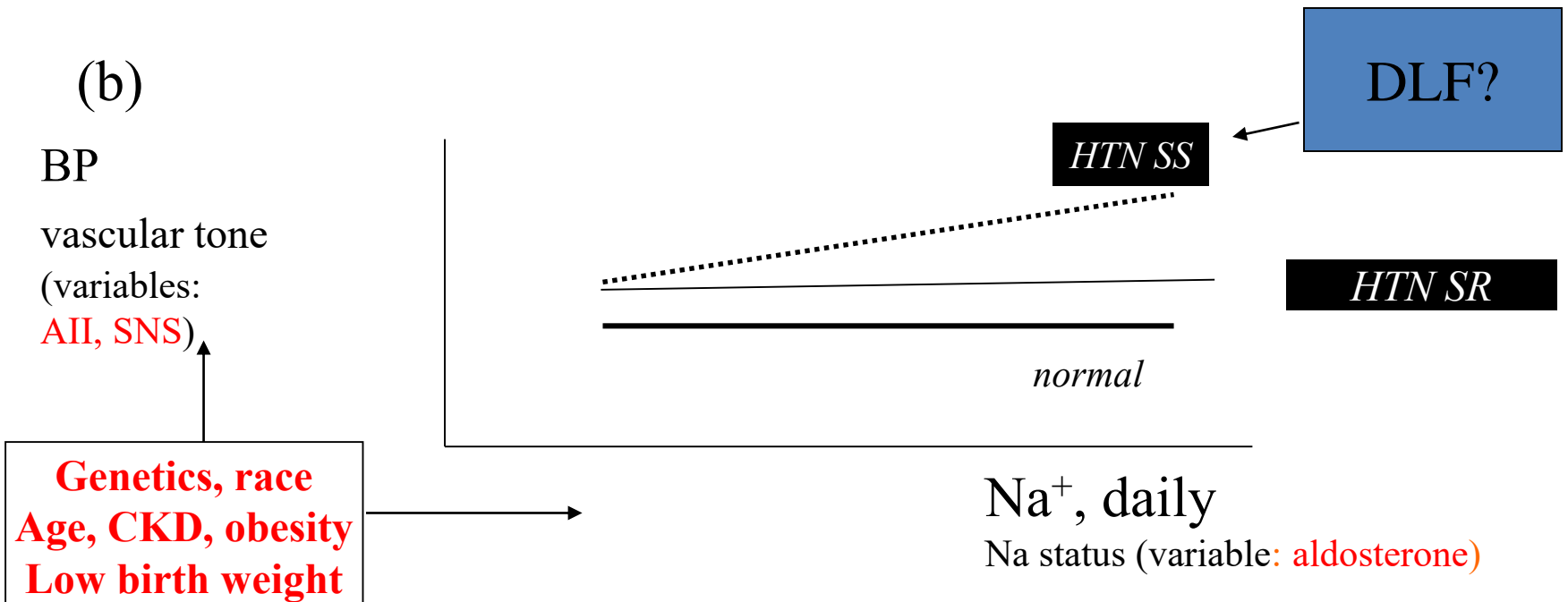
**Notion de sensibilité
de la PA au sel**

figure 1
Krzesinski&Cohen

(a)

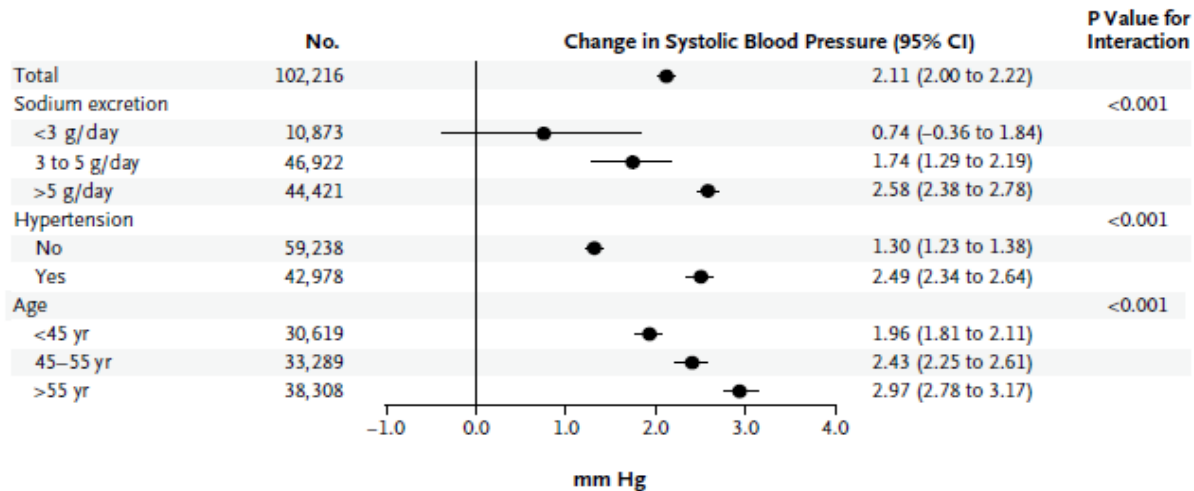


(b)



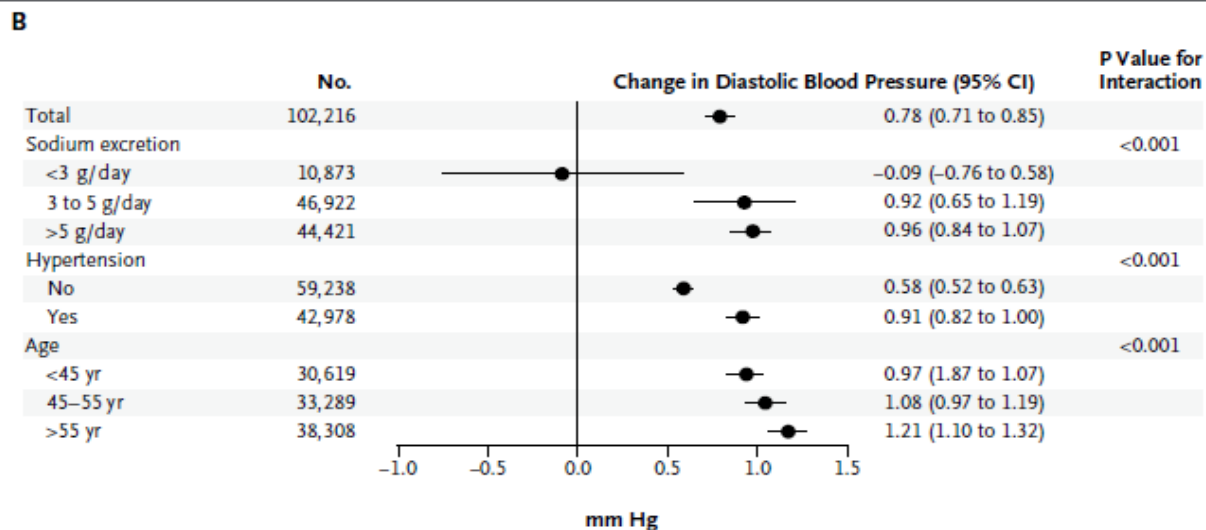
Association entre excrétion urinaire de sodium et PA

102000 adultes de 18 pays



Prospective Urban Rural Epidemiology (PURE) study is provided in the Supplementary Appendix, available at NEJM.org.

N Engl J Med 2014;371:601-11.

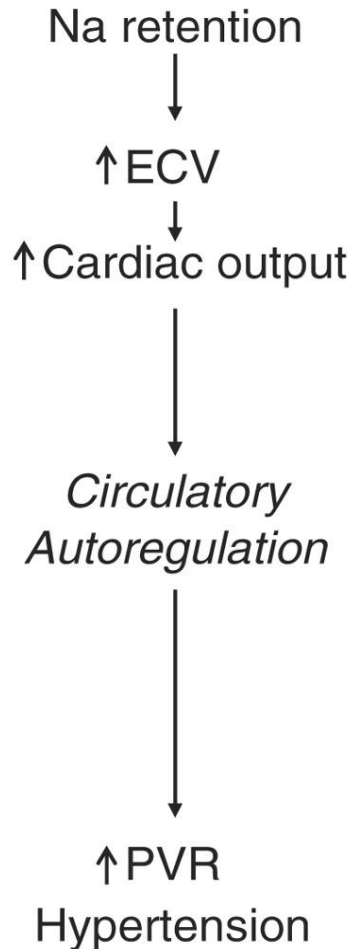


Sensibilité au sel:
Age, Sévérité HTA

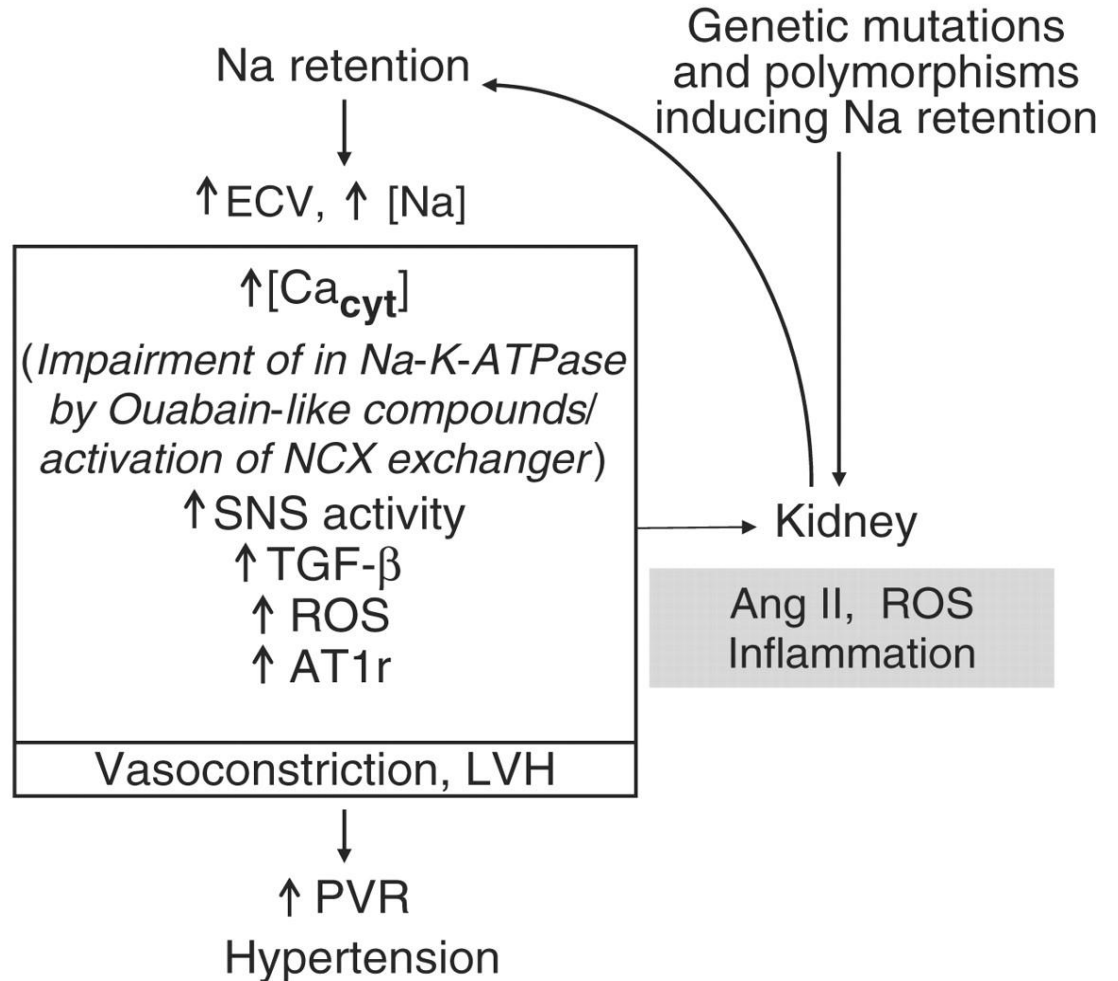
Figure 3. Forest Plots of Changes in Systolic and Diastolic Blood Pressure for Every 1-g Increase in Sodium Excretion. Data are based on multivariable linear regression models with adjustment for covariates and regression dilution bias.

Etiopathogenesis of salt-sensitive hypertension

Guyton, 1960–70's



Salt-sensitive hypertension 2000's



Disorders of Plasma Sodium — Causes, Consequences, and Correction

Richard H. Sterns, M.D.

N ENGL J MED 372:1 NEJM.ORG JANUARY 1, 2015

The NEW ENGLAND JOURNAL of MEDICINE

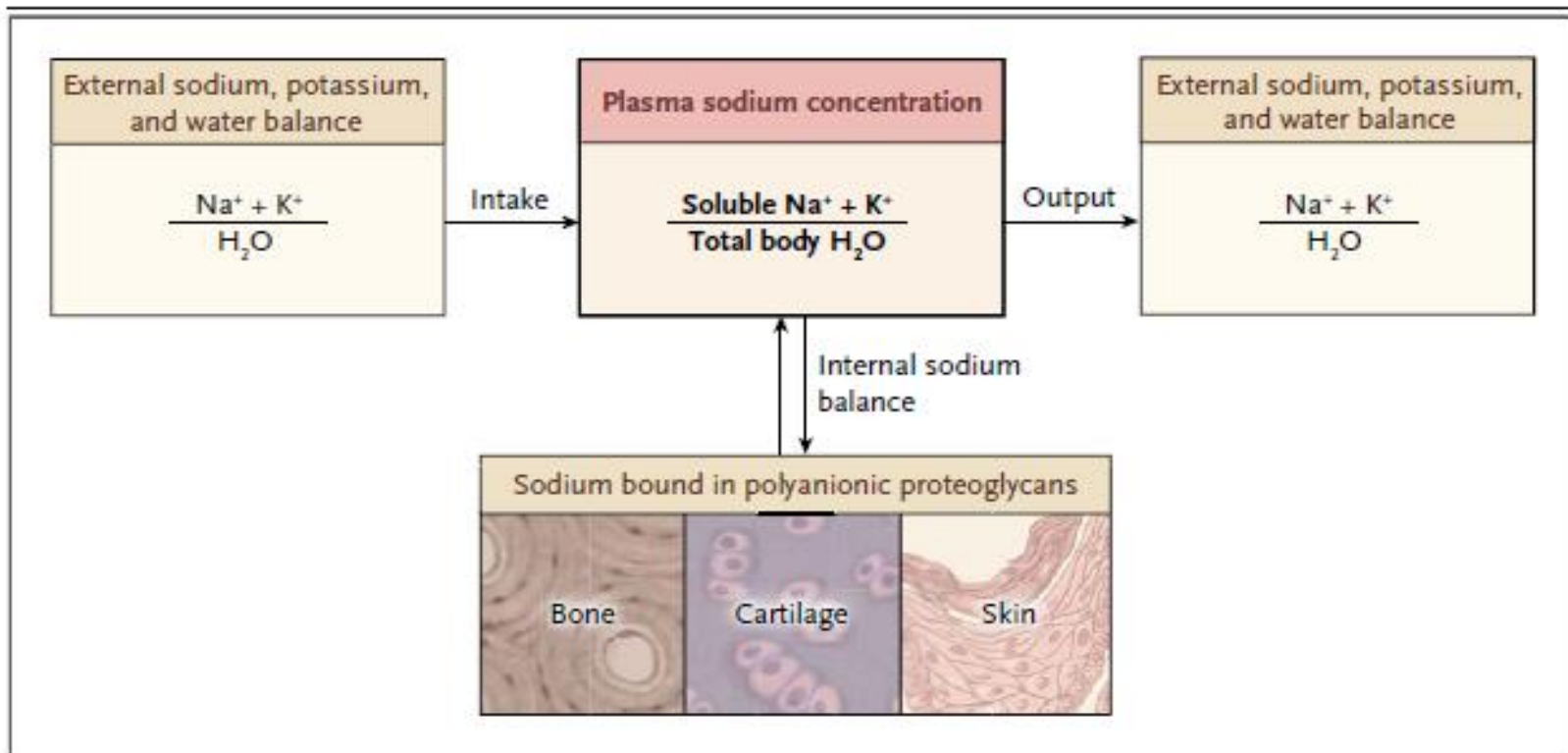
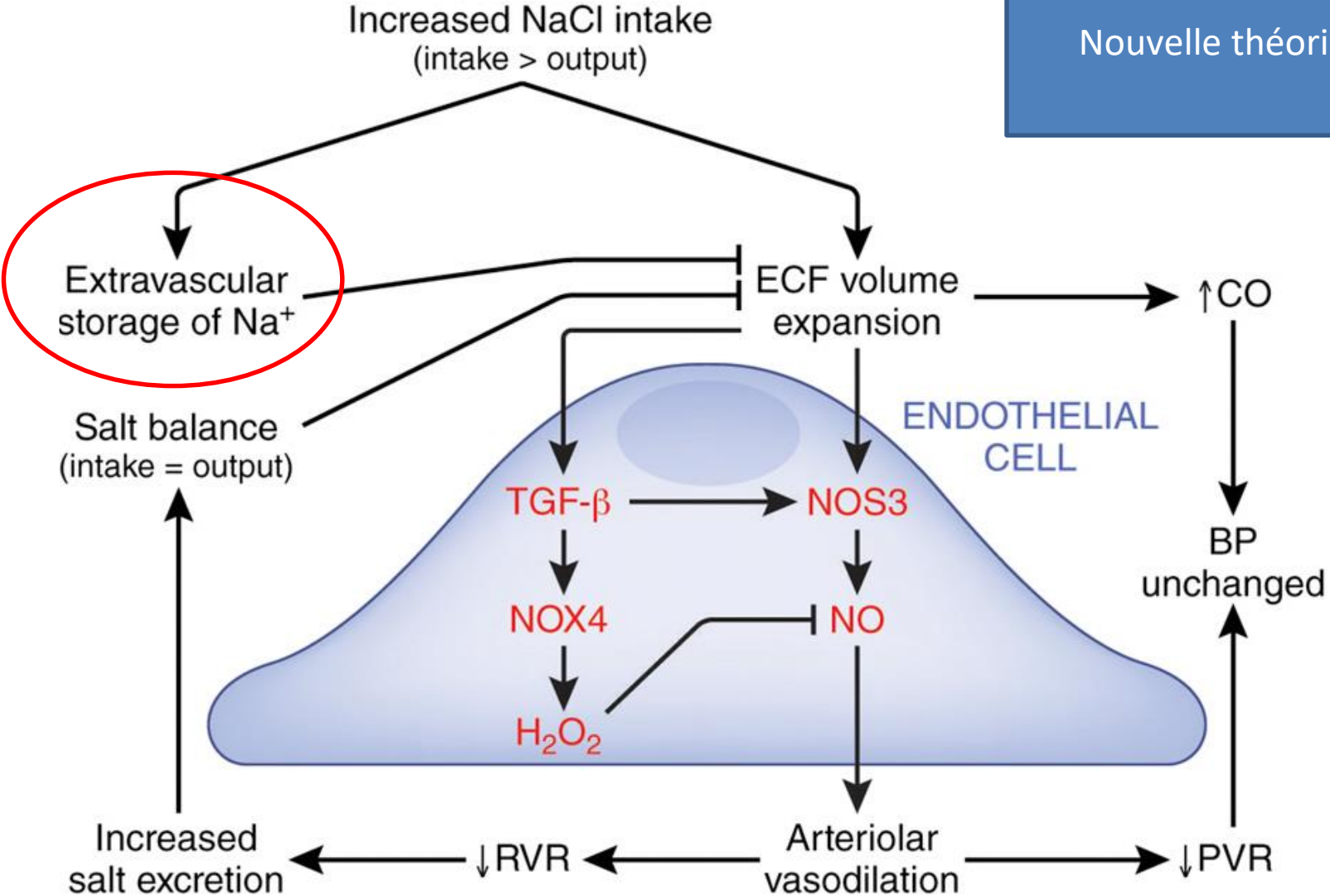


Figure 1. Internal and External Solute and Water Balance and the Plasma Sodium Concentration.

Nouvelle théorie



Novel Paradigms of Salt and Hypertension

Wenguang Feng,* Louis J. Dell'Italia,*† and Paul W. Sanders*†‡

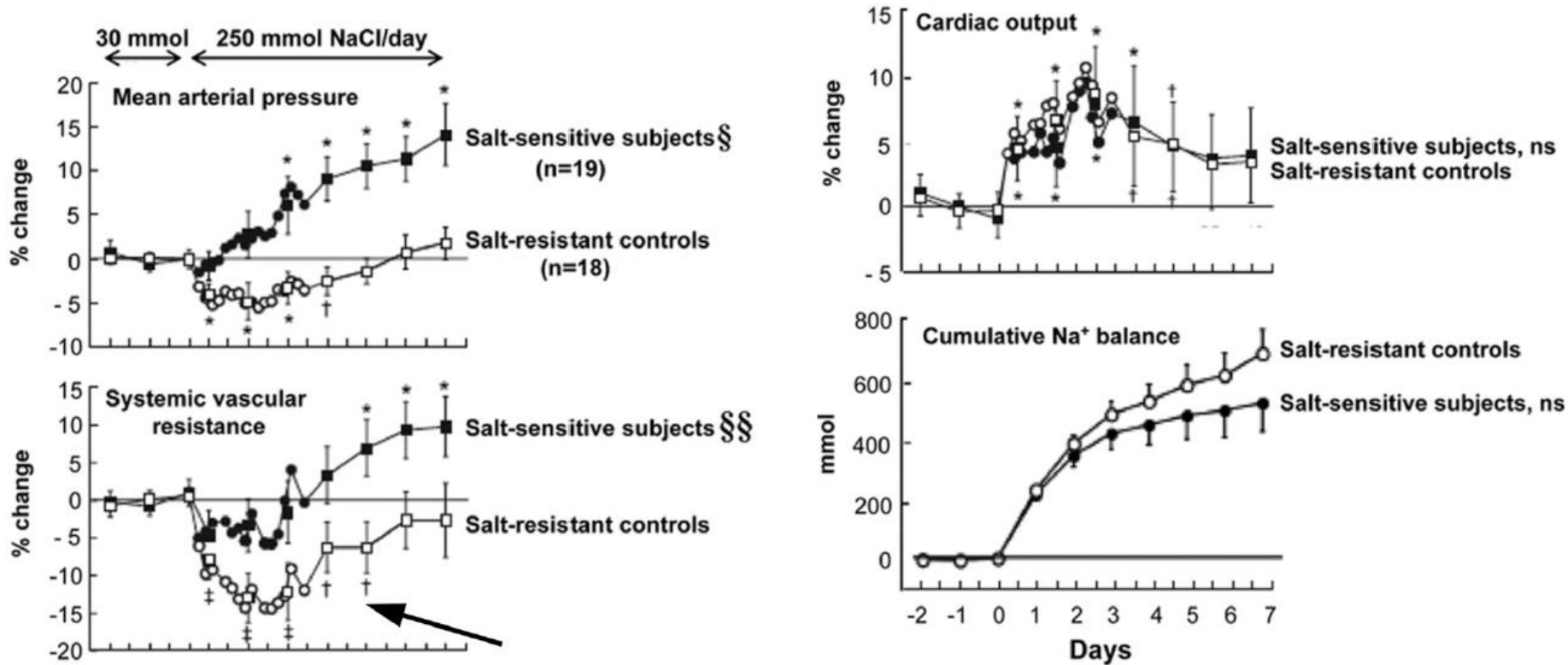


Figure 3. The hemodynamic effects of chronic high salt intake differed between SS and SR volunteers. Despite similar increases in CO (row 3) and cumulative sodium balance (row 4), SS but not SR patients manifested salt-induced increases in mean arterial pressure (row 1). The SR volunteers showed rapid reductions in calculated systemic vascular resistance (SVR; row 2), whereas SVR did not decline and actually increased over time in the SS patients. It is worth noting that SVR also increased with continued high salt ingestion in SR patients (arrow). * $P < 0.01$; † $P < 0.05$; §§ $P < 0.001$, compared with period of low salt intake. ns, not

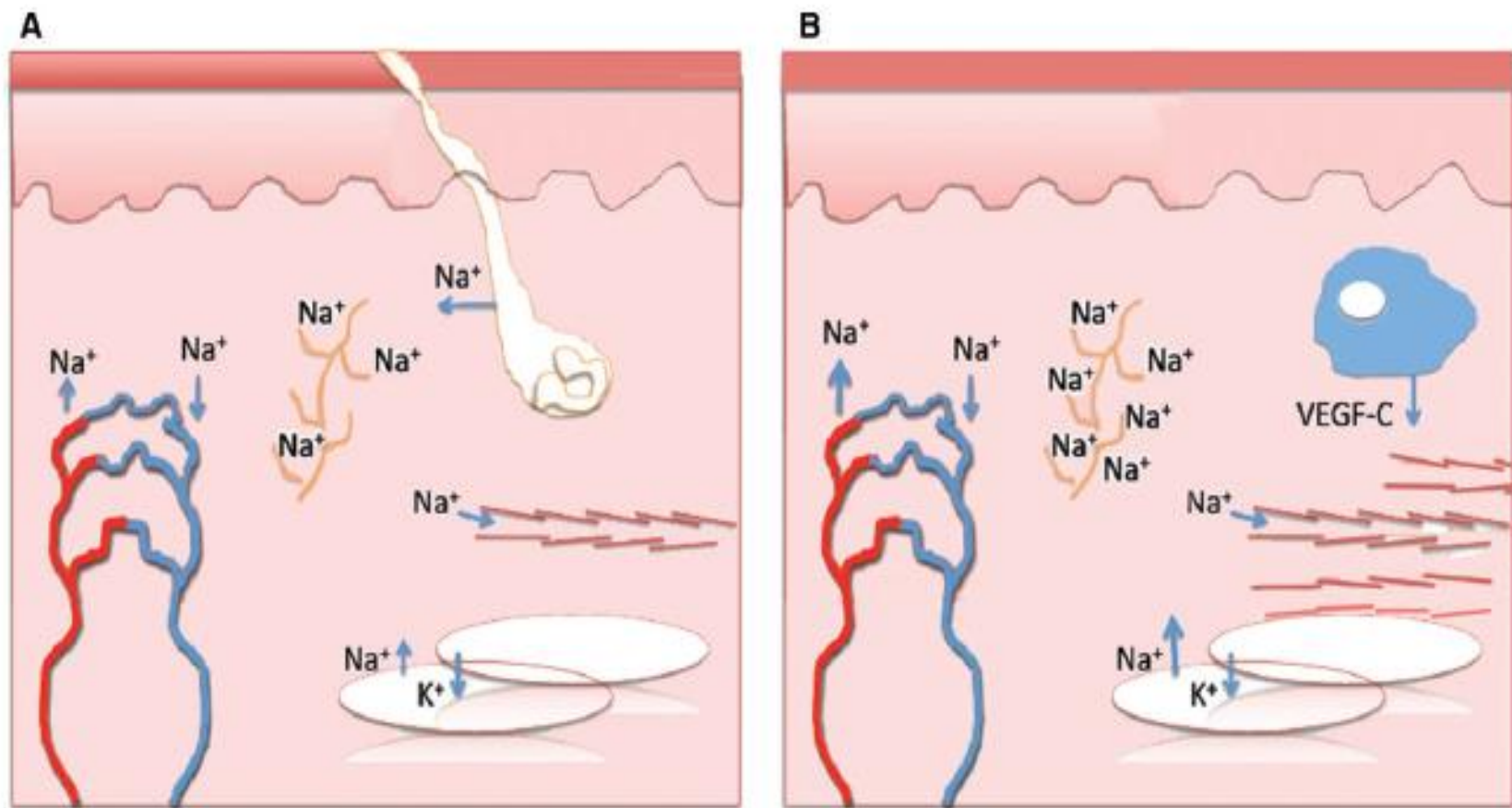


Fig. 1. Proposed new model of Na^+ balance in the subcutaneous compartment. **(A)** Normal physiology: Na^+ is filtrated into and reabsorbed from the interstitium at the level of the capillaries through the classical Starling equilibrium. In addition, Na^+ accumulates in the subcutaneous interstitium through reabsorption from sweat glands. Na^+/K^+ -ATPase on the membrane of cells keeps Na^+ in the extracellular compartment. Polyanionic matrix molecules in the interstitium can bind Na^+ without commensurate water. A small part of Na^+ and interstitial fluid is transported back to the circulation through the lymphatic system. **(B)** Salt loading: Na^+ binding capacity of the matrix molecules is increased. In addition, Na^+ is less efficiently kept in the extracellular compartment through inhibition of Na^+/K^+ -ATPase activity. However, when interstitial hypertonicity develops, despite these compensation mechanisms, influx of inflammatory cells can be observed that through an osmosensitive transcription factor release VEGF-C and induce lymphangiogenesis. The latter mechanism will increase Na^+ transport back into the circulation. 254 × 122 mm (300 × 300 DPI).

Risque lié à un apport excessif en sel

- Rôle sur la PA via la volémie
- Rôle sur le système immunitaire et indirectement sur la PA

Table. Impact of High-Salt Diet on Immune Cell Function

Macrophages
Promotes macrophage infiltration in diverse tissues ^{17,25,26,48}
Promotes proinflammatory M1 macrophage activation ^{27,29}
Depresses anti inflammatory M2 macrophage activation ³⁰
T cells
Increased T-cell infiltration, proliferation, and activation ⁴⁹
Promotes Th17 activation
Boosts the development of IL-17-producing CD4 ⁺ Th17 cells ^{18,19,24,39}
Promotes Th17 activation via p38 MAPK-, NFAT-, and SGK1-dependent signaling ¹⁸
Negative impact on Tregs
Impairs the function and development of regulatory forkhead box P3 ⁺ Tregs ^{24,38}
Induces SGK1-signal transduction and promotes interferon release from Tregs which abrogates their suppressive effects ¹⁹
Effects on other leukocytes
Splenic B cells ³²
Neutrophils ³³
Basophils ³⁴
Effects on dendritic cells
Increased sodium enters the dendritic cells via specific channels, and increased NADPH oxidase activity produces superoxide with subsequent formation of immunogenic isolevuglandin, promoting an autoimmune-like state leading to renal and vascular dysfunction and hypertension. ⁴²

Salt Intake and Immunity

Baris Afsar, Masanari Kuwabara, Alberto Ortiz, Aslihan Yerlikaya, Dimitrie Siriopol, Adrian Covic, Bernardo Rodriguez-Iturbe, Richard J. Johnson, Mehmet Kanbay

High Salt Diet

Hypertension

July 2018

↓
↑ Sodium concentration ↑ Osmolarity

Immune system

Modulation of T cell activity

T helper cells
(Th17)
M1 macrophages

>

Regulatory T cells
(Tregs)
M2 macrophages

Systemic inflammation
Autoimmunity

Endothelial dysfunction
Increased sympathetic nervous system activity
Impairment in pressure natriuresis

Hemodynamic effects

Stimulation of osmosensitive neurons
Cardiotonic steroids

↑ Sympathetic nervous system activity
↑ Cardiac output
↑ Peripheral vascular resistance

→ **Hypertension** ←

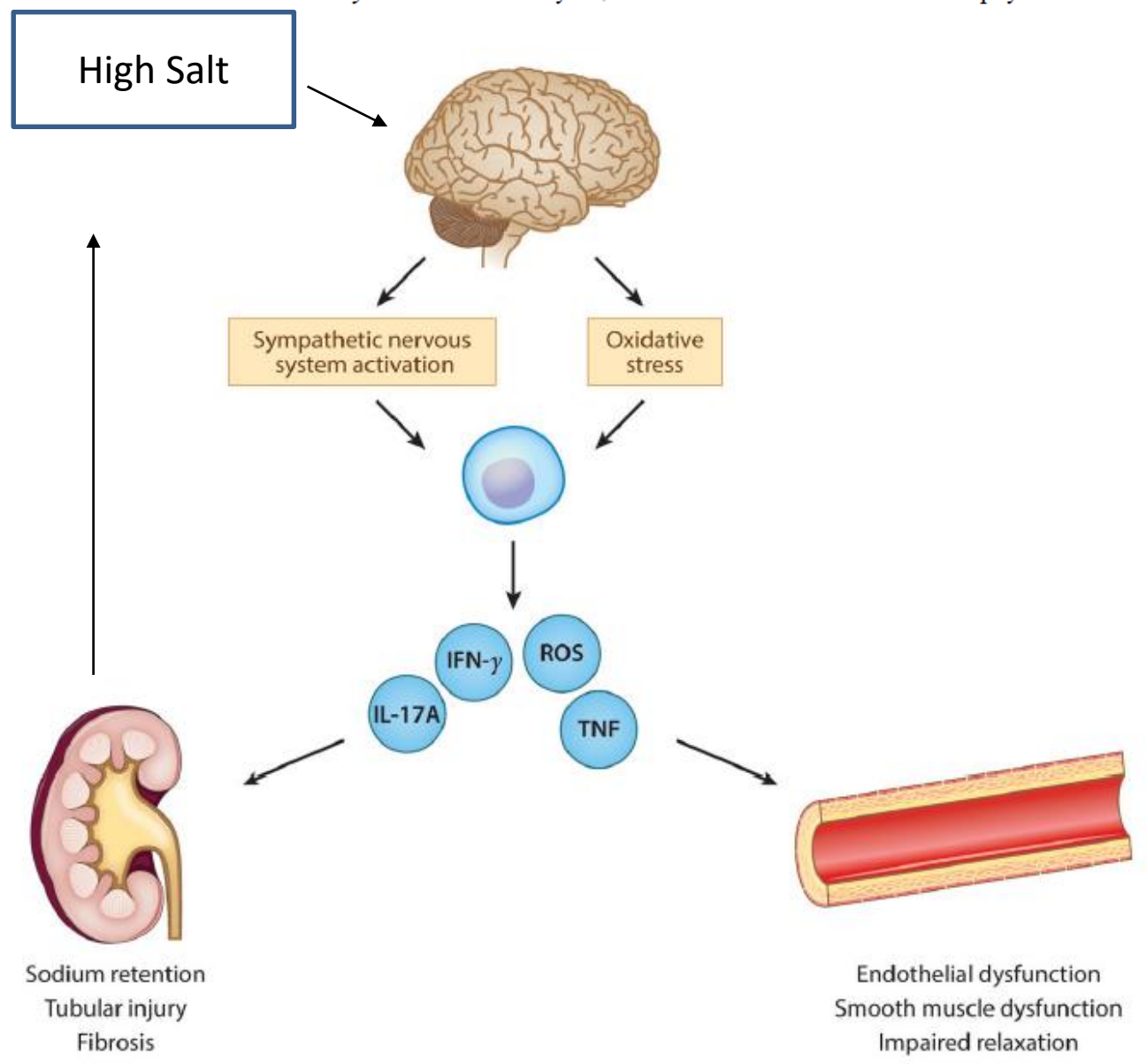
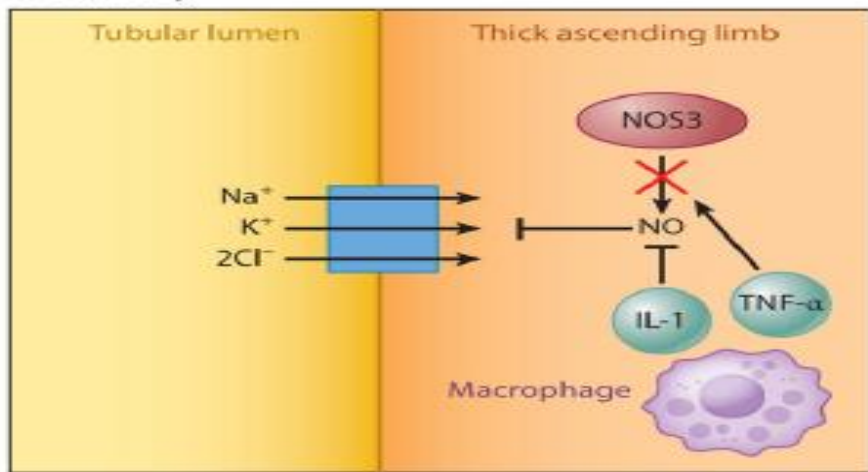


Figure 3. Proposed roles of T lymphocytes in hypertension and salt sensitivity. Sympathetic nervous

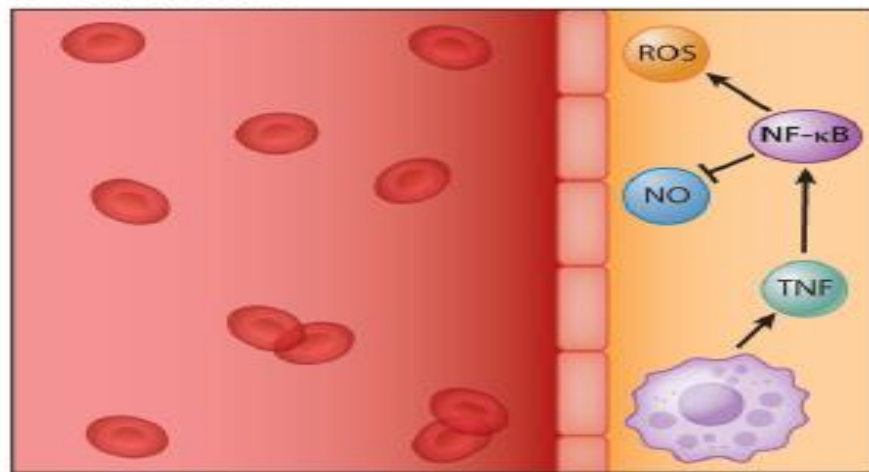
Salt, Hypertension, and Immunity

A. Justin Rucker^{1,2}, Nathan P. Rudemiller^{1,2}, and Steven D. Crowley^{1,2}

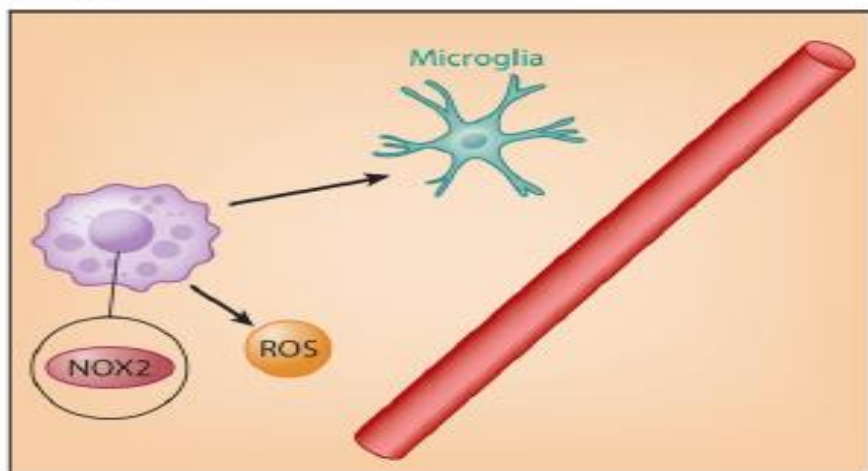
a Kidney



b Vasculature



c Brain



d Skin

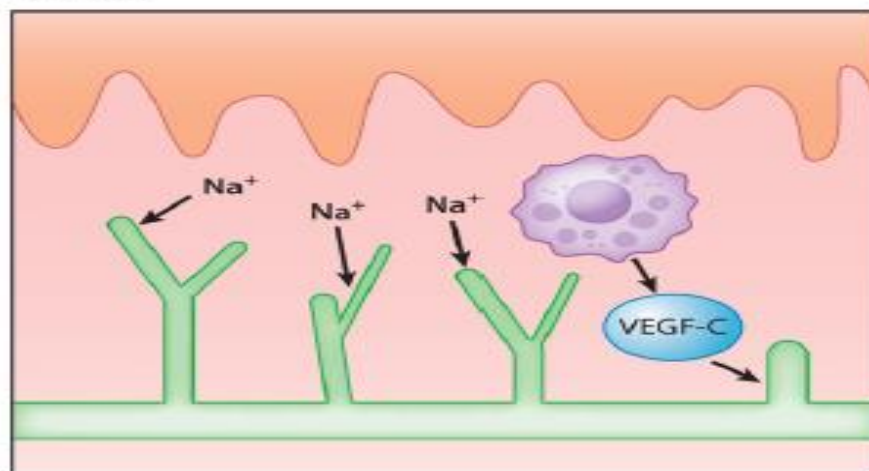
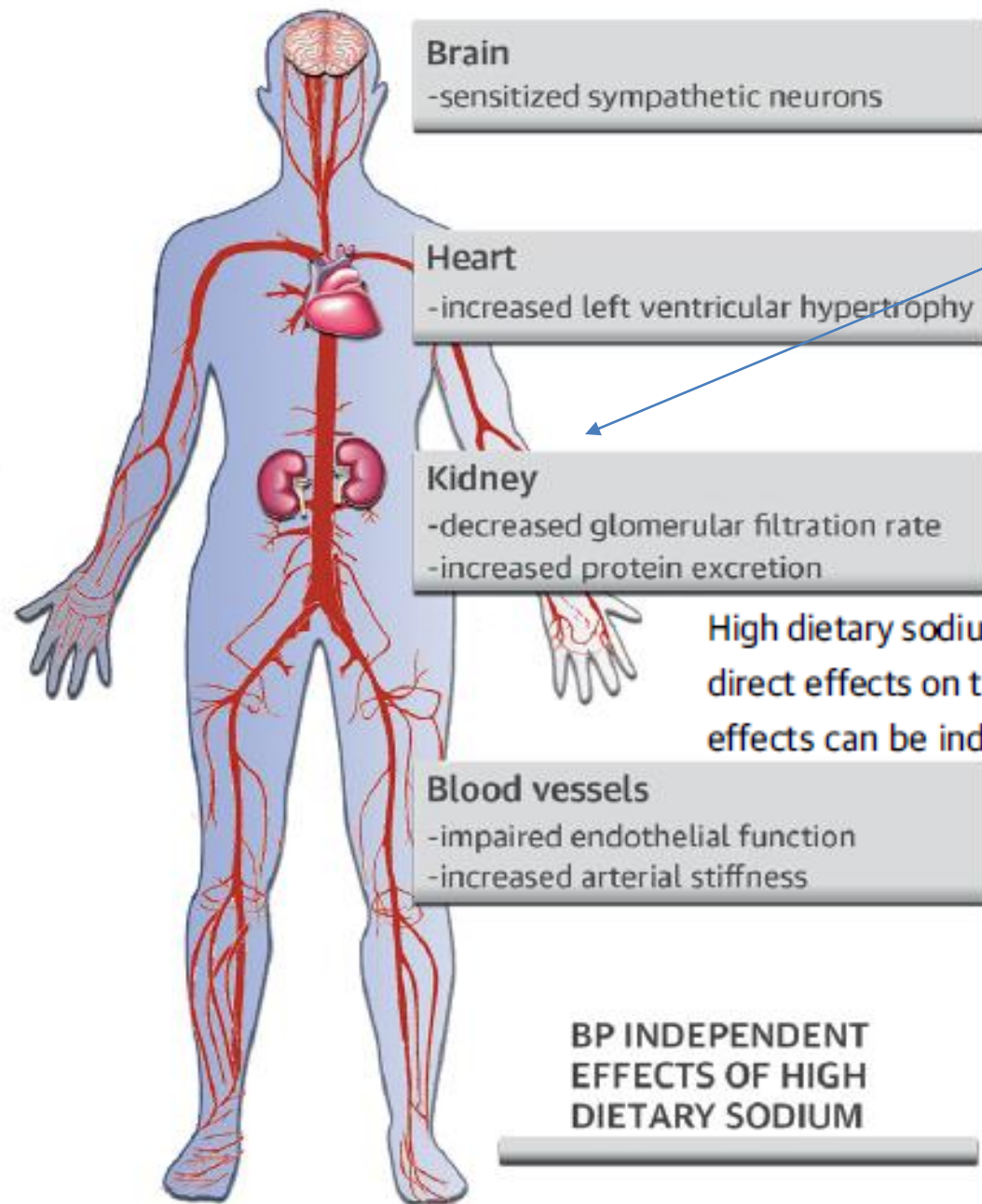


Illustration of the diverse roles for macrophages in salt-sensitive hypertension. Through generation of inflammatory cytokines and ROS, macrophages act in cardiovascular control centers to induce hypertension. (a) In the kidney, proinflammatory macrophage canonical

FIGURE 1 BP-Independent Effects of High Dietary Sodium



Lithiase calcique

High dietary sodium can cause target organ damage and may have direct effects on the brain, heart, kidneys, and vasculature. These effects can be independent of changes in blood pressure (BP).

Rôle délétère joué par le sel

- Rôle sur la PA
- Rôle via le système immunitaire
- Rôle via le microbiote intestinal

A High Salt Diet Modulates the Gut Microbiota and Short Chain Fatty Acids Production in a Salt-Sensitive Hypertension Rat Model

Nutrients 2018, 10, 1154; doi:10.3390/nu10091154

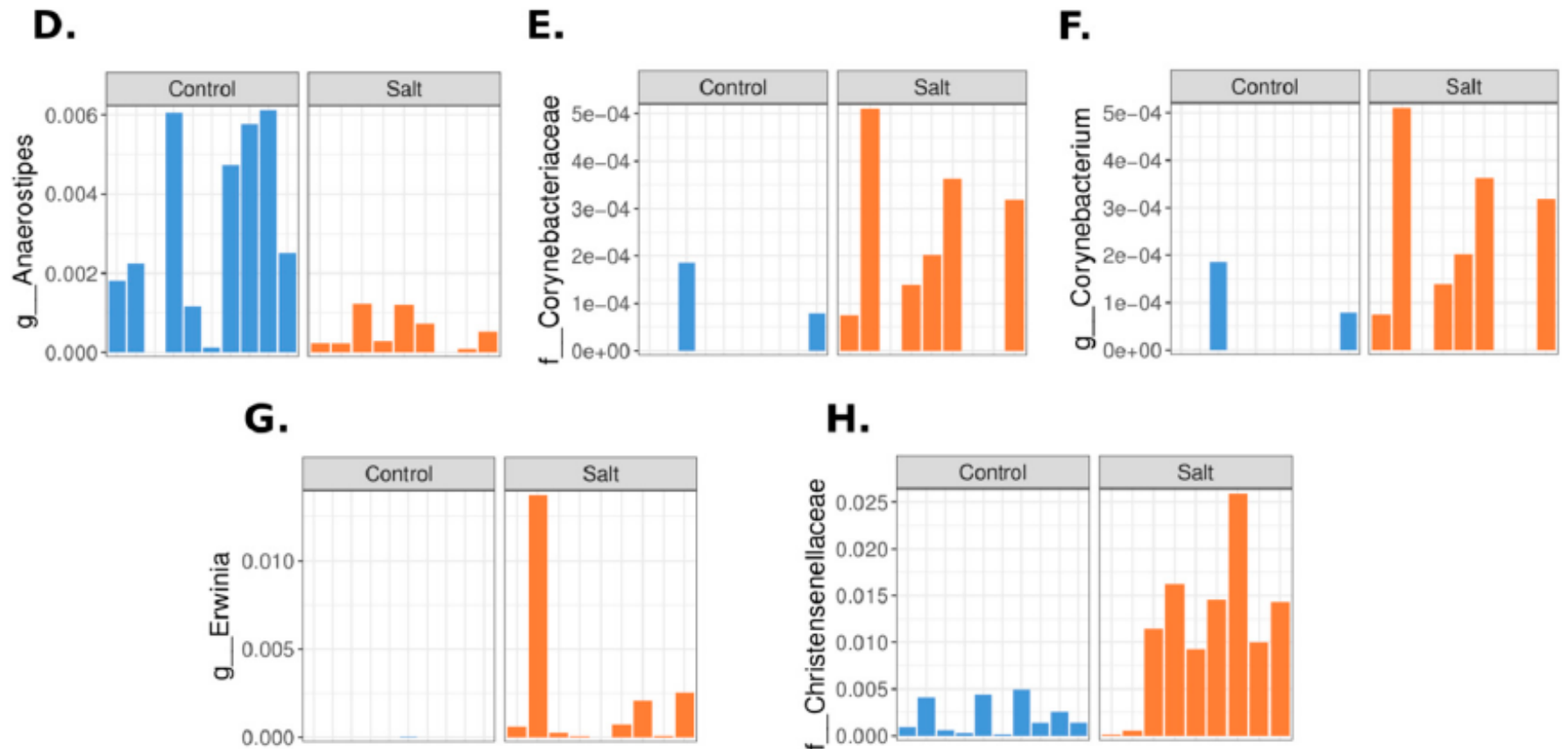


Figure 2. Microbial composition in high salt diet (HSD) differs from the control. Fecal pellets from HSD and control rats were analyzed using 16S rRNA-Amplicon Sequencing. Unweighted (A) and weighted

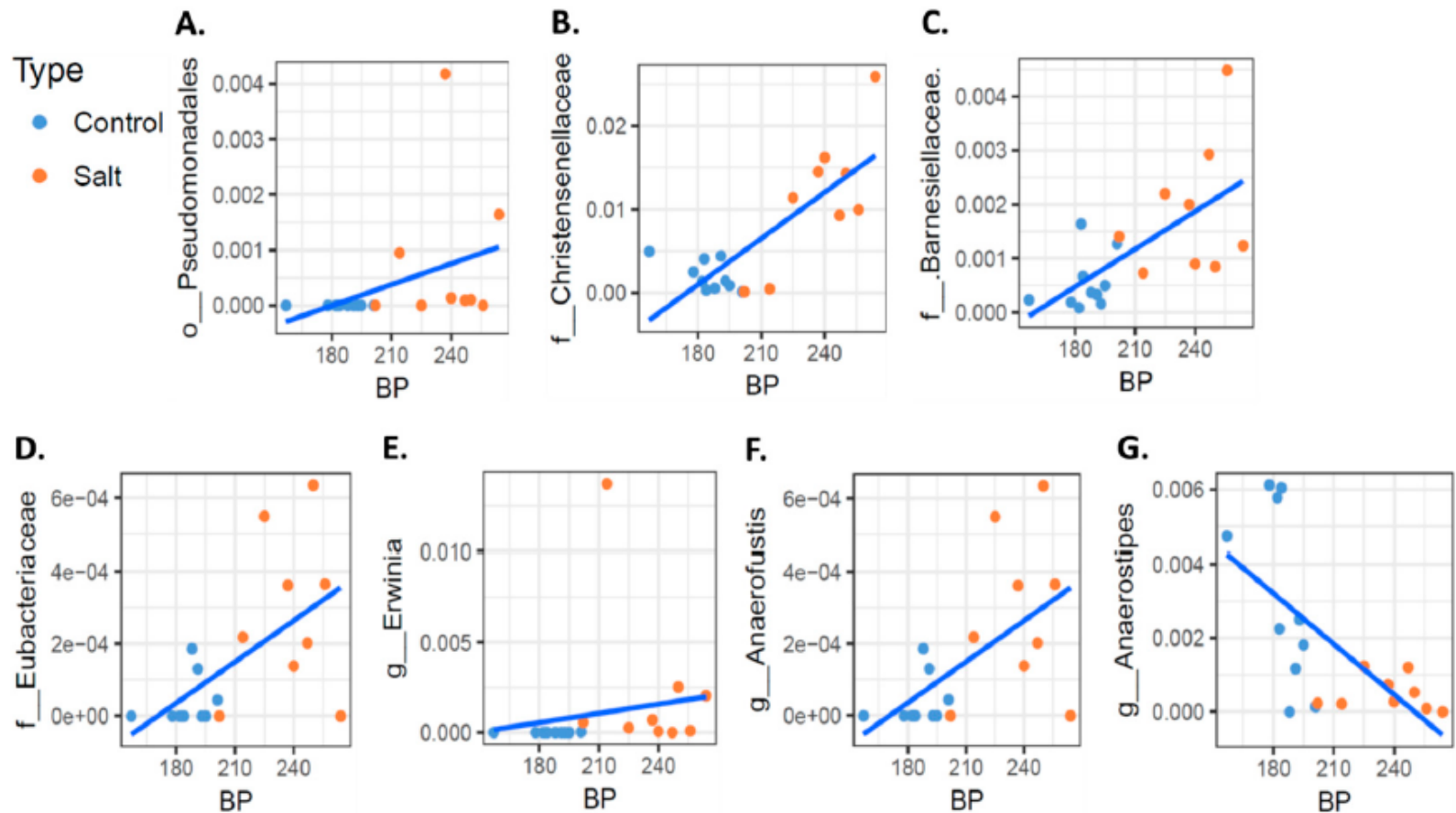


Figure 3. Correlation between blood pressure (BP) and bacterial taxa. Rat's BP values after 30 days were analyzed for their correlation with specific bacterial taxa; 7 microbial taxa displayed a significant correlation (q value ≤ 0.1) with BP (A–G) using the Multivariate Association with Linear Models (Maaslin) package. Blue dots and red dots depict fecal samples from the high salt diet and controls, respectively.

A High Salt Diet Modulates the Gut Microbiota and Short Chain Fatty Acids Production in a Salt-Sensitive Hypertension Rat Model

Nutrients 2018, 10, 1154; doi:10.3390/nu10091154

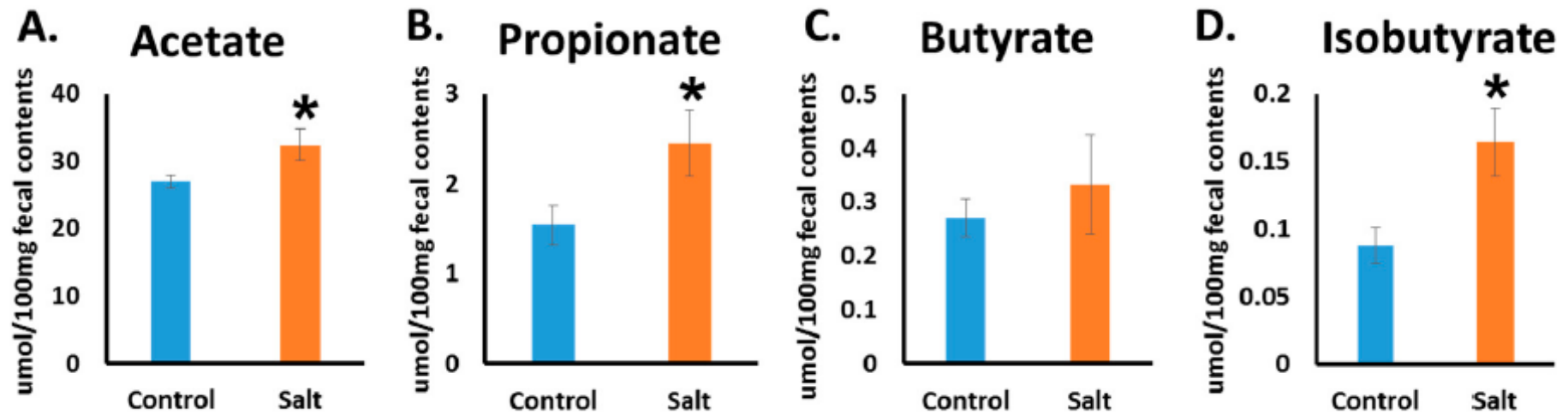


Figure 4. High salt diet elevated the SCFA level. The level of acetate (A); propionate (B); butyrate (C), and isobutyrate (D) were measured by GC-MS from fecal pellets at the end of the experiment. $n = 8-10$, * $p \leq 0.05$.

Risque lié au sel

- Rôle sur la PA par la volémie
- Rôle via le système immunitaire
- Rôle via le microbiote intestinal
- Rôle via la promotion de l'insulino-résistance

Insulin resistance and salt-sensitive hypertension in metabolic syndrome

Toshiro Fujita

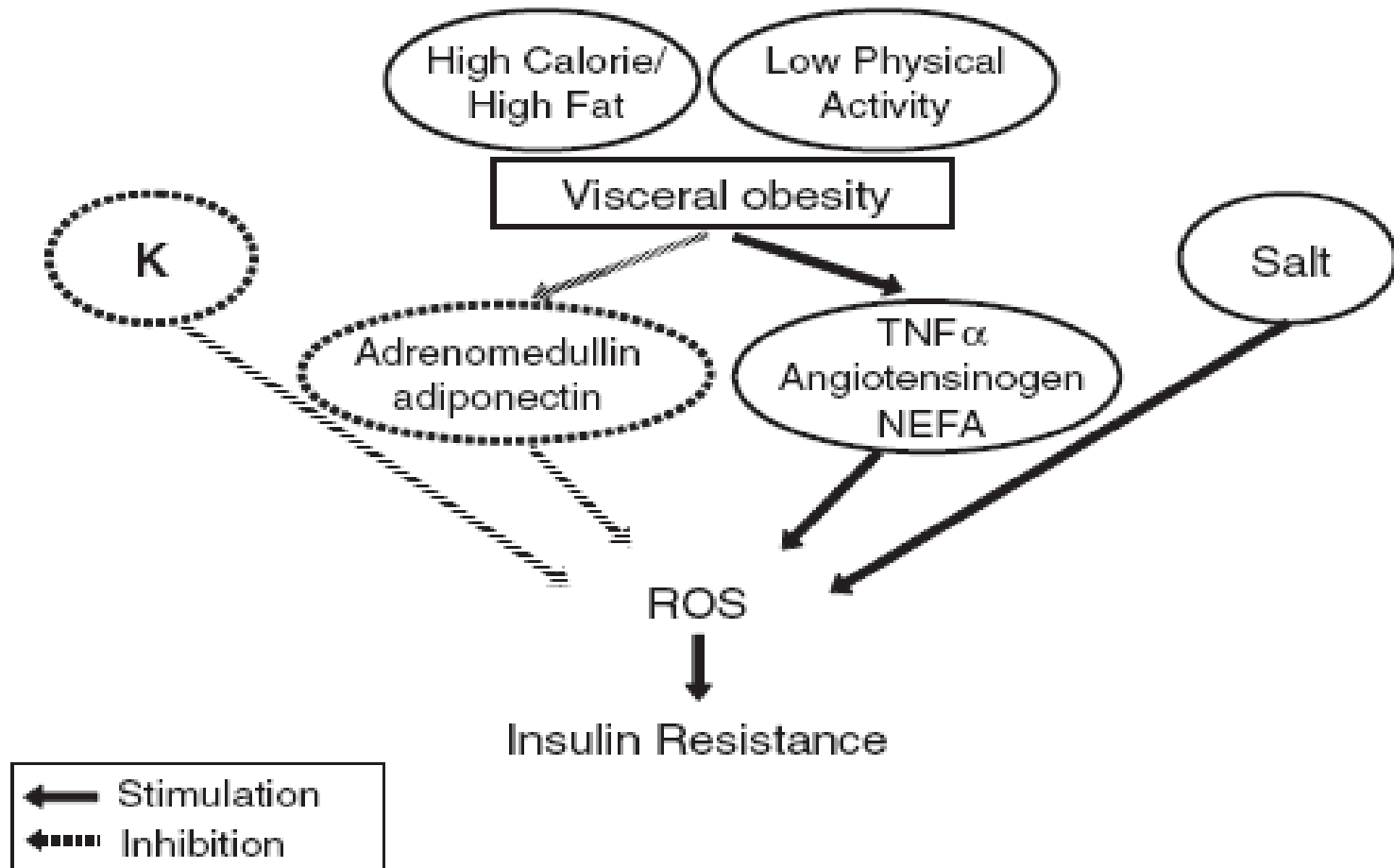


Fig. 1. Mechanism for insulin resistance in metabolic syndrome.

High Salt Intake

(Hypertension. 2015;66:843-849.

Independent Risk Factor for Obesity?

Yuan Ma, Feng J. He, Graham A. MacGregor

a direct association between salt intake and obesity independent of energy intake. We analyzed the data from the rolling cross-sectional study—the UK National Diet and Nutrition Survey 2008/2009 to 2011/2012. We included 458 children (52% boys; age, 10 ± 4 years) and 785 adults (47% men; age, 49 ± 17 years) who had complete 24-hour urine collections.

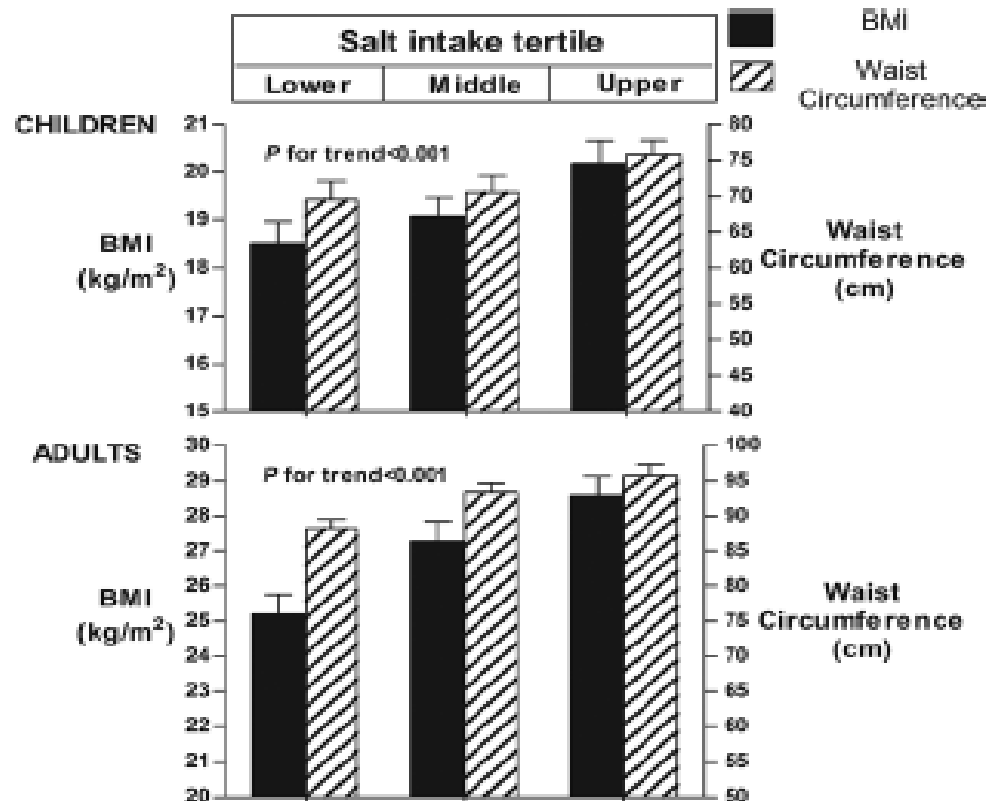


Figure. Adjusted mean body mass index (BMI) and waist circumference according to the tertiles of salt intakes measured by 24-hour urinary sodium excretion.

Le sodium est-il seulement le seul acteur alimentaire dans le contrôle de la PA et du risque CV?



Rôles joués par le sel

- Rôle sur la PA
- Rôle sur le système immunitaire
- Rôle sur le microbiote intestinal
- Rôle sur l'insulino-résistance et l'obésité
- Rôle atténué si alimentation riche en potassium

Daily potassium intake and sodium-to-potassium ratio in the reduction of blood pressure: a meta-analysis of randomized controlled trials

Aristea Binia^a, Jonathan Jaeger^b, Youyou Hu^b, Anurag Singh^b, and Diane Zimmermann^c

Paleolithic diet : K 150-290 mmol/d
Na 20-40 mmol/d

Modern diet : K 30-70 mmol/d
Na 80-250 mmol/d

So urinary Na/K around 3

Journal of Hypertension 2015, 33:1509–1520

Association of Urinary Sodium and Potassium Excretion with Blood Pressure

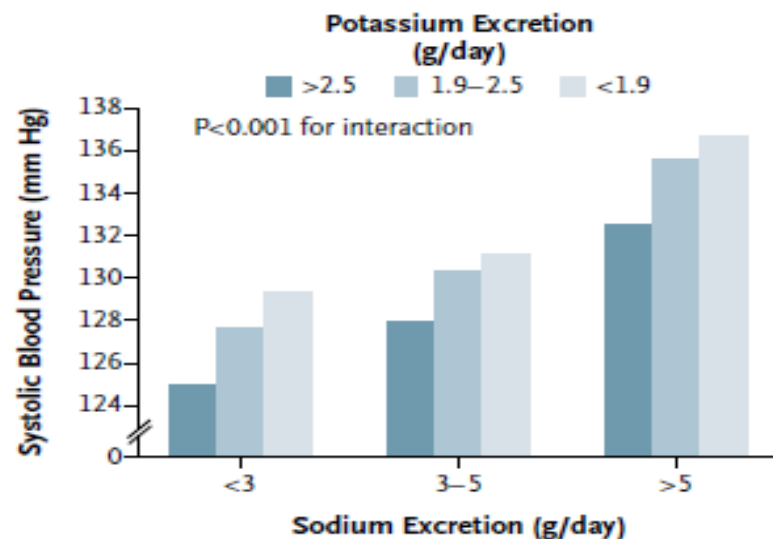
102000 adultes de 18 pays

Figure 4. Mean Systolic and Diastolic Blood Pressure According to Sodium and Potassium Excretion.

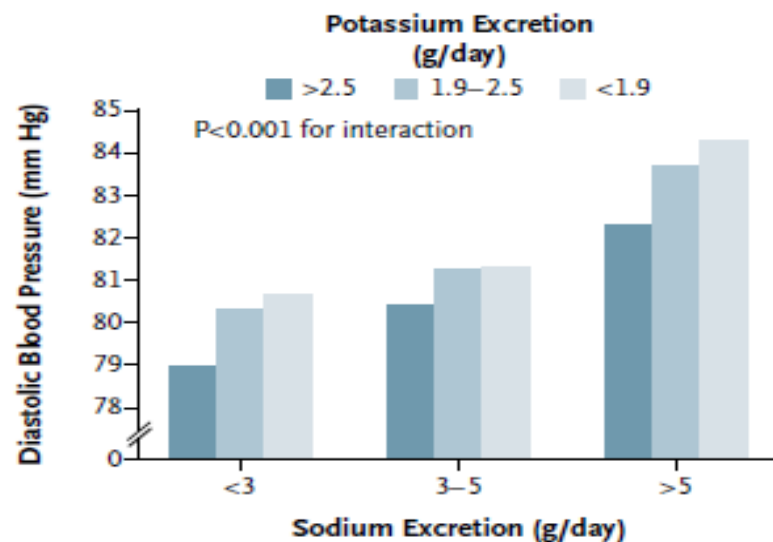
Adjustment was made for age, sex, geographic region, body-mass index, educational level, and alcohol intake. The P value for interaction is for testing the joint effect of the two electrolytes on blood pressure.

Prospective Urban Rural Epidemiology (PURE) study is provided in the Supplementary Appendix, available at NEJM.org.

N Engl J Med 2014;371:601-11.



B



Fruit and Vegetable Consumption and the Incidence of Hypertension in Three Prospective Cohort Studies

Hypertension. 2016;67:288-293.

Table 2. Pooled Hazard Ratios (95% Confidence Intervals) of Incident Hypertension for Combined Total Fruits and Total Vegetables Consumption in Nurses' Health Study, Nurses' Health Study II, and Health Professional Follow-up Study

	≤1 per Day	2–3 per Day	4–5 per Day	≥6 per Day	Linear <i>P</i> Trend
Total Fruits and Vegetables					
NHS*	1052/31 868	11 318/339 541	13 914/396 152	9091/266 861	
Adjusted hazard ratio†	1.00 (reference)	0.90 (0.84–0.95)	0.88 (0.83–0.94)	0.85 (0.80–0.91)	<0.001
NHS II‡	1427/79 840	10 019/528 610	8785/462 410	5015/273 616	
Adjusted hazard ratio†	1.00	0.97 (0.92–1.03)	0.95 (0.89–1.00)	0.94 (0.88–0.99)	0.02
HPFS§	666/22 176	5711/190 017	6085/201 161	4290/146 874	
Adjusted hazard ratio†	1.00	0.93 (0.85–1.01)	0.91 (0.83–0.98)	0.89 (0.81–0.97)	0.02
Pooled results 	1.00	0.94 (0.90–0.97)	0.91 (0.88–0.95)	0.89 (0.86–0.93)	<0.001

HPFS indicates Health Professionals Follow-up Study; and NHS, Nurses' Health Study.

*Follow-up in Nurses' Health Study was from 1984 to 2010 (cases/persons-years).

†Adjusted for age, race/ethnicity (white, African American, Asian, Hispanic, other), body mass index, current smoking status, physical activity, weight change per food frequency questionnaire cycle, menopausal status (NHS and NHS II), alcohol intake, current oral contraceptive use (NHS II), analgesic use (nonsteroidal antiinflammatory drugs, acetaminophen, aspirin), family history of hypertension, total energy intake, animal flesh intake (combination of processed and unprocessed red meat, poultry and seafood), whole grains, sugar-sweetened beverage intake, artificially sweetened diet beverage intake.

‡Follow-up in Nurses' Health Study II was from 1991 to 2011.

§Follow-up in Health Professionals Follow-up study was from 1986 to 2010.

||Pooled hazard ratios of the 3 cohorts using a fixed effects model.

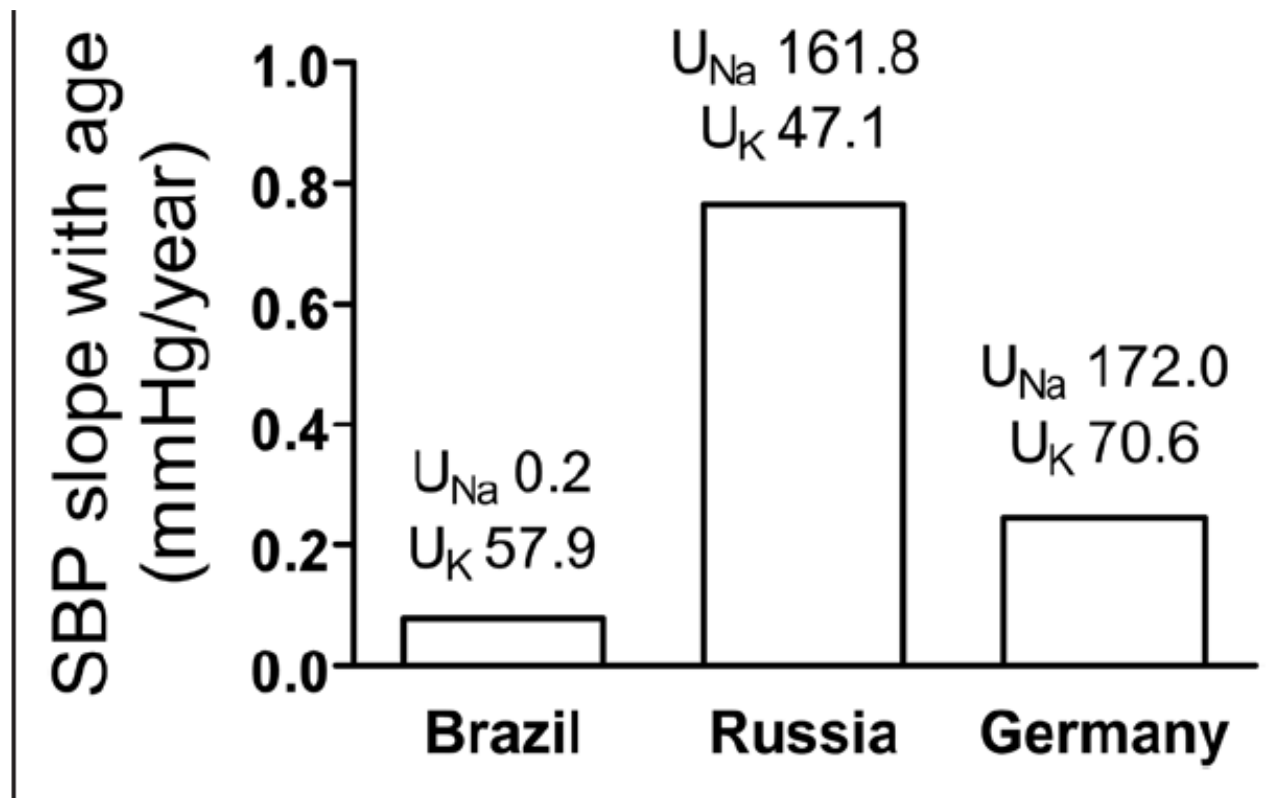
Gritter et al **K⁺ in BP Reduction and Cardiorenal Protection**

Figure 1. Comparison of blood pressure (BP) slopes (systolic BP [SBP] increase in mmHg/y) and urinary Na⁺ and K⁺ excretion (U_{Na}, U_K) of 3 selected cohorts from the Intersalt study.¹² The Brazilian cohort consists of the Yanomamo Indians whose diet primarily consists of K⁺ salts and who show almost no BP increase with aging. The Russian and German cohorts had similar U_{Na}, whereas U_K was 1.5 fold higher in the German cohort. This may have contributed to the remarkable difference in SBP slope between the people in these 2 countries.

Gritter et al K^+ in BP Reduction and Cardiorenal Protection

HTA 2019

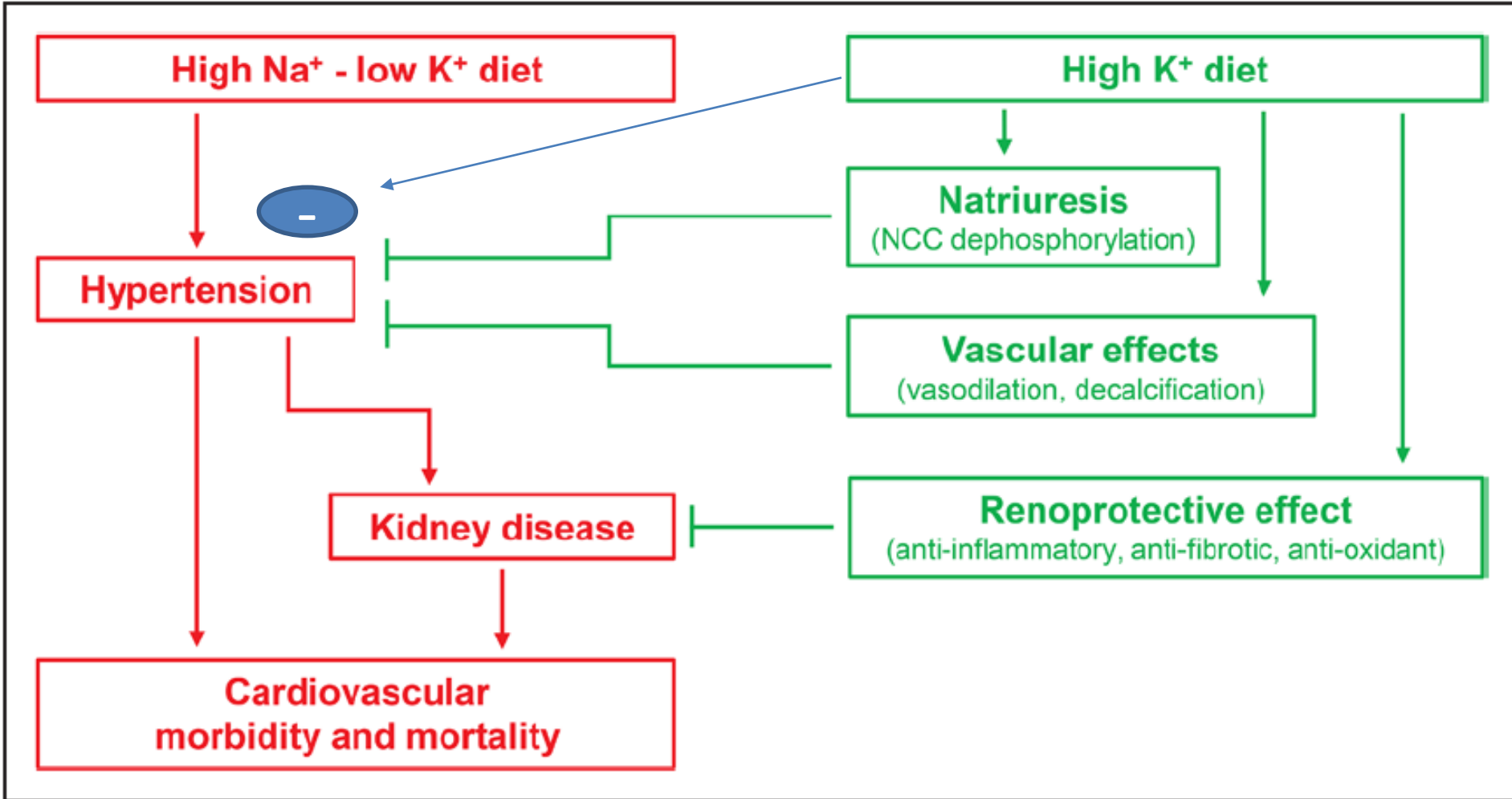


Figure 2. Proposed pathways through which high K⁺ can reduce blood pressure and provide cardiovascular and renal protection. The left

Dietary recommendations for Sodium and Potassium and BP

WHO recommendation (Weaver C Adv Nut 2013):

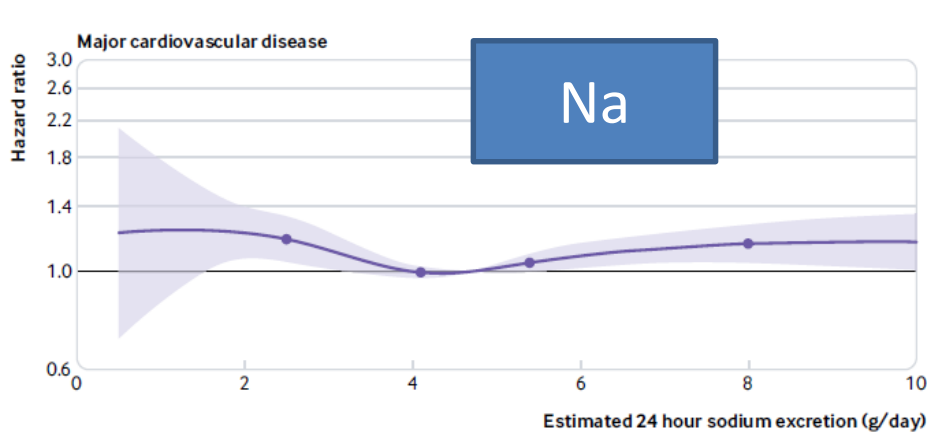
The ideal would be to lower sodium intake to < 100 mmol/d
and to increase potassium intake to > 90 mmol/d,
so the **urinary Na/K ratio should be near 1-1.5**

Joint association of urinary sodium and potassium excretion with cardiovascular events and mortality: prospective cohort study

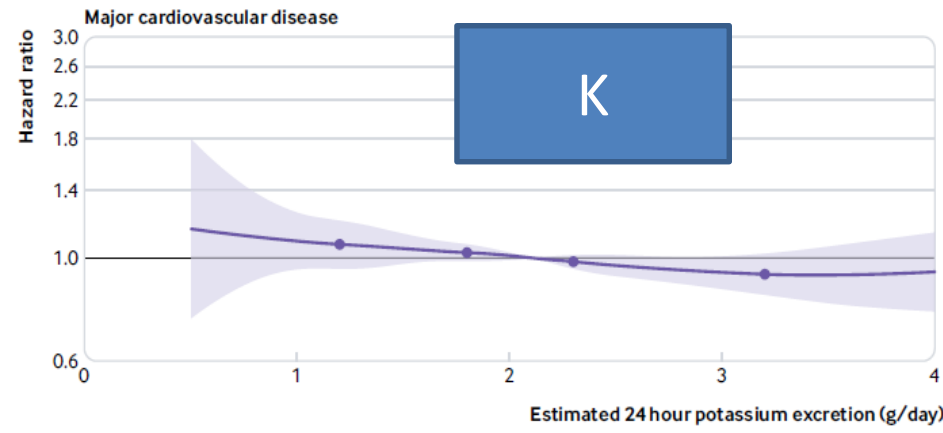
: *BMJ* 2019;364:l772

Dietary guidelines recommend sodium restriction but increased potassium intake (WHO recommends <2.0 g/day and >3.5 g/day, respectively)

Studies evaluation the association of sodium intake have reported inconsistent findings, but many report a J-shaped association, while those evaluating potassium intake generally report a linear reduction in mortality with increasing potassium intake



Range (g)	0-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10
Total	1534	8060	19092	23 414	19 022	11 326	5582	2470	1131
No of events	75	360	822	1002	937	594	331	150	68



Range (g)	0-1	1-2	2-3	3-4
Total	1749	39 814	43 836	6849
No of events	81	1907	2094	291

The combination of moderate sodium intake (3-5 g/day) with higher potassium intake was associated with the lowest risk of mortality and cardiovascular events

Nutrition and other Lifestyle Influences on Arterial Aging

Thomas J. LaRocca, Christopher R. Martens, and Douglas R. Seals

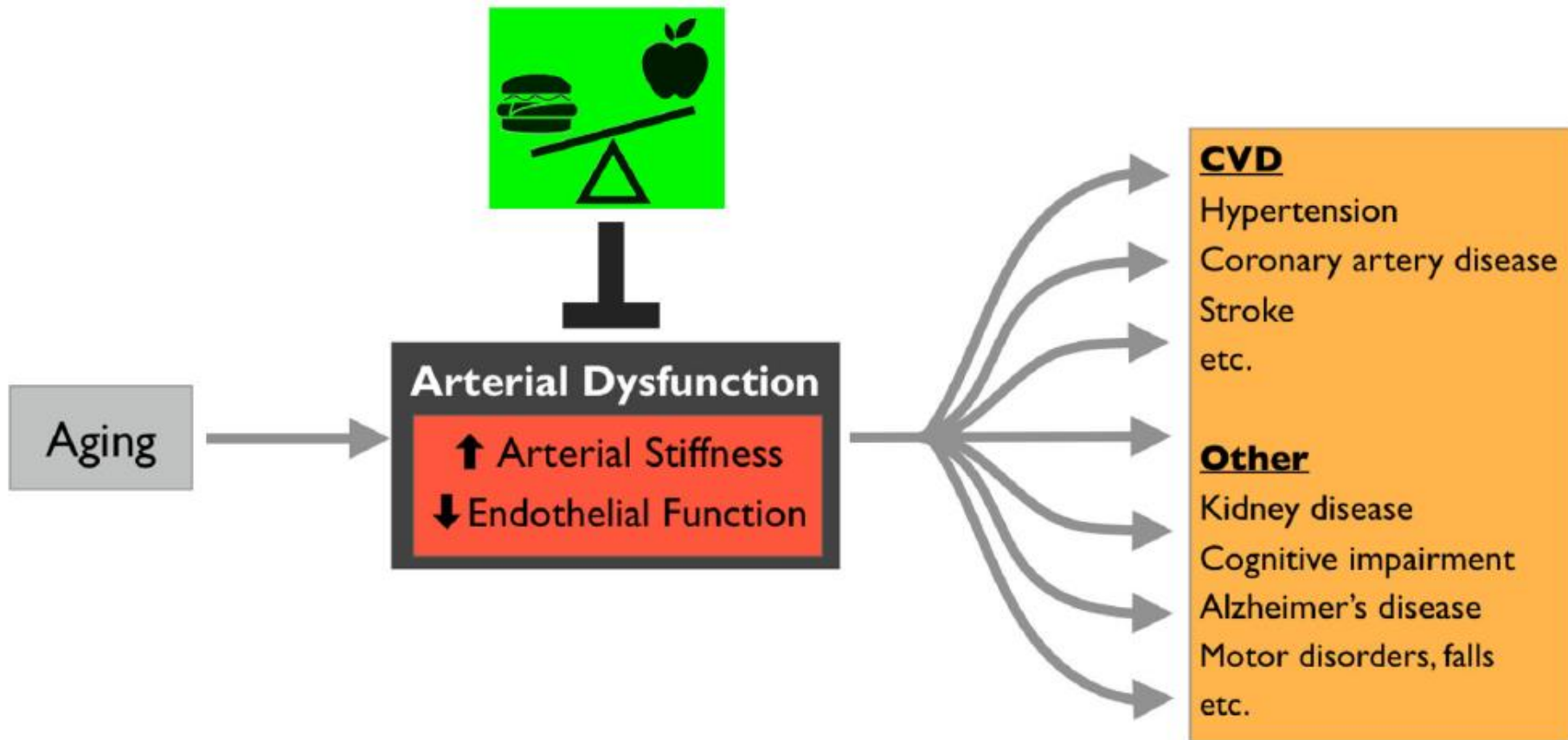
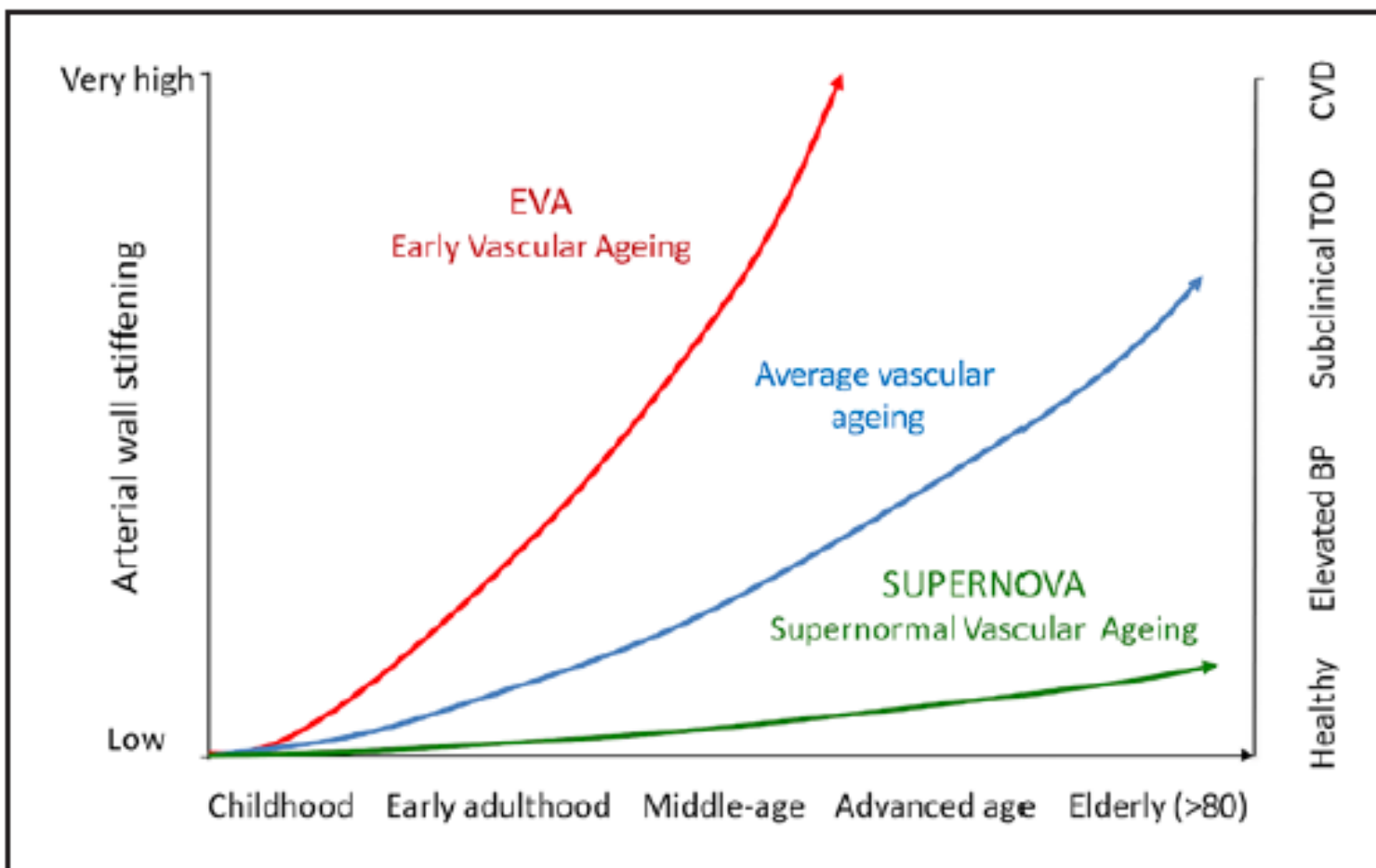


Figure 1.

Aging leads to arterial dysfunction (increased stiffness and reduced endothelial function) that predisposes us to cardiovascular diseases (CVD) and other chronic disorders. Diet/nutrition has the potential to modulate/prevent arterial dysfunction with aging.

Early, Normal, and Supernormal Vascular Aging



Place du sodium dans le processus de vieillissement vasculaire

Table. Putative Determinants of EVA and SUPERNOVA

Nonmodifiable Determinants	Determinants of EVA	Determinants of SUPERNOVA
Chronological age		
Ethnicity		
Sex		
Family history		
Prenatal fetal growth		
Genetics		
	Classical CV risk factors	
	High BP	Normal BP
	Hyperglycemia	Normal glycemia
	Insulin resistance	
	Diabetes mellitus	
	Obesity	Normal weight
	Abdominal fat	Low-calorie diet
	Metabolic syndrome	
	Dyslipidemia	Normal lipids
	Chronic kidney disease	
	High-salt diet	Low-salt diet
	Smoking	No smoking
	Lack of physical activity	Intense physical activity

	Additional CV risk factors	
	Oxidative stress	Insensitivity to oxidative stress
		Strong protective metabolic mechanisms
	Alcohol consumption	None of these risk factors
	Chronic low-grade inflammation	
	Gut microbiome composition	
	Social deprivation	
	High perceived stress	
	Abnormal sleep pattern	
	Thrombogenic factors	
	Hormonal status	
	...	

Hypertension. 2019;74:218-228.

Diet/factor	Effects	Evidence*
	Mediterranean, DASH, vegetarian(?) diets	↑ endothelial function ↓ arterial stiffness 
	Higher fruit/vegetable intake	↑ endothelial function ↓ arterial stiffness 
	Higher fish intake	↑ endothelial function ↓ arterial stiffness 
	High fat diet	↓ endothelial function ? arterial stiffness 
	Low fat diet	↑ endothelial function ? arterial stiffness 
	Specific electrolytes	↑ endothelial function ↓ arterial stiffness (some) 
	Sodium restriction	↑ endothelial function ↓ arterial stiffness 
	Nuts, tea, coffee, cocoa, whole grains, legumes, olive oil	↑ endothelial function ↓ arterial stiffness (some) 

Take home messages

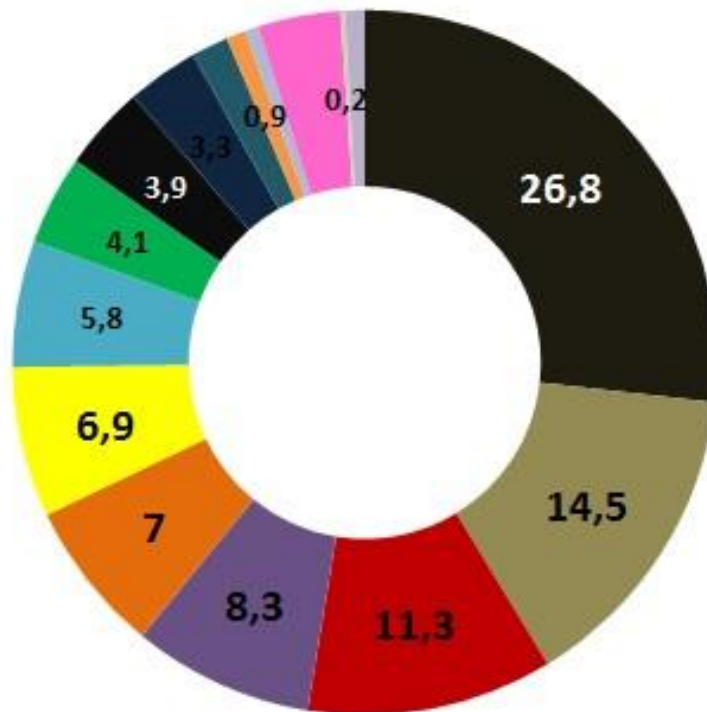
Evidence that salt is bad for you

www.areshumor.com



Contributions moyennes des groupes d'aliments (en %) aux apports en sodium chez les adultes

Quoidansmonassiette.fr
Etude INCA2



- Pain, céréales
- Condiments sauces, soupes, bouillons
- Charcuteries
- Plats composés
- Pizza, quiches, sandwiches
- Fromages

- Viennoiseries, biscuits, pâtisseries
- Légumes et légumes secs
- Viande, volaille, abat, œufs
- Lait, produits laitiers, crèmes desserts
- Boissons, eau
- Chocolat, sucre, café, glace
- Matières grasses
- Poissons, crustacés

$\frac{3}{4}$
apports

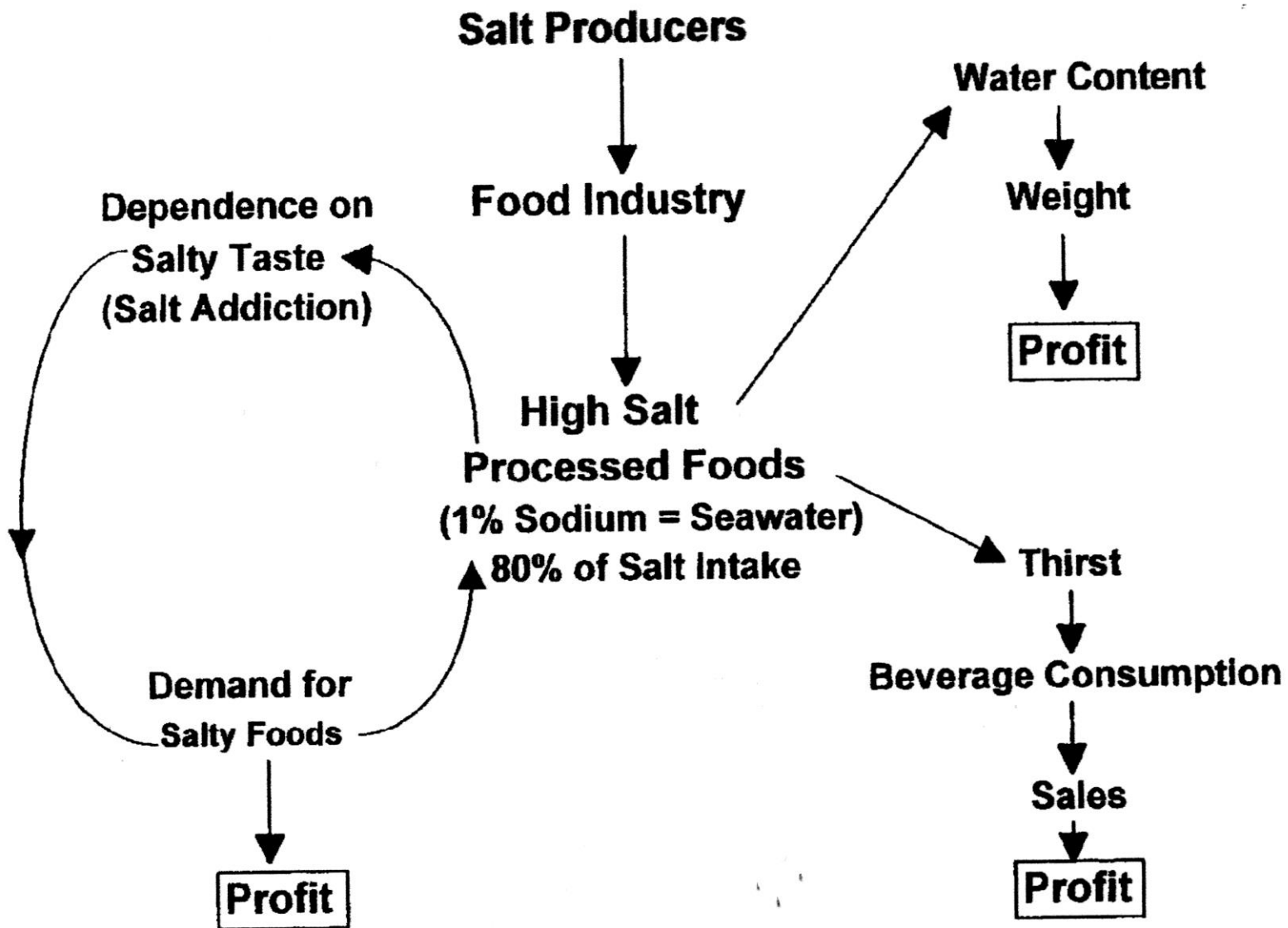


Fig. 5. The commercial importance of salt in processed food.

Table 1 | UK strategy for reducing salt

Salt intake			
Source	g/day	Reduction needed	Target intake (g/day)
Table/cooking (15%)	1.4 g	40% reduction	0.9 g
Natural (5%)	0.5 g	No reduction	0.5 g
Food industry (80%)	7.6 g	40% reduction	4.6 g
Total: 9.5 g			Target: 6.0 g

FDA Sodium Reduction Targets and the Food Industry: Are There Incentives to Reformulate? Microsimulation Cost-Effectiveness Analysis

The World Health Organization has recommended sodium reduction as a “best buy” to prevent cardiovascular disease (CVD). Despite this, Congress has temporarily blocked the US Food and Drug Administration (FDA) from implementing voluntary industry targets for sodium reduction in processed foods, the implementation of which could cost the industry around \$16 billion over 10 years.

The Milbank Quarterly, Vol. 00, No. 00, 2019 (pp. 1-23)

"WAITER! - THERE'S SOUP
IN MY SALT!"



National Salt
Awareness
Week

11th - 17th March 2013
www.actiononsalt.org.uk



Merci de votre attention