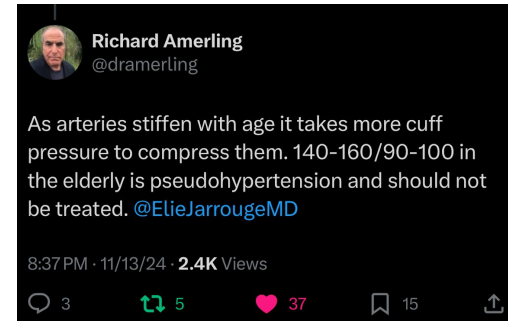




Twelve Medical Myths You Believe Pediatrics to Adults

Stefan Hartmann,
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[The Tamiflu fiasco and lessons learnt](#) (Gupta, Meenu, 2015)



1. Myth: “You need Antibiotics for ear infections”- parents/CYA medicine

A randomized, double-blind, placebo-controlled noninferiority trial of amoxicillin for clinically diagnosed acute otitis media in children 6 months to 5 years of age

Nicole Le Saux, Isabelle Gaboury, Marian Baird, Terry P Klassen, Johnna MacCormick.

Cmaj 172 (3), 335-341, 2005

Objectives: Debate continues with respect to a “watch and wait” approach versus immediate antibiotic treatment for the initial treatment of acute otitis media. In this double-blind noninferiority trial, we compared clinical improvement rates at 14 days for children (6 months to 5 years of age) with acute otitis media who were randomly assigned to receive

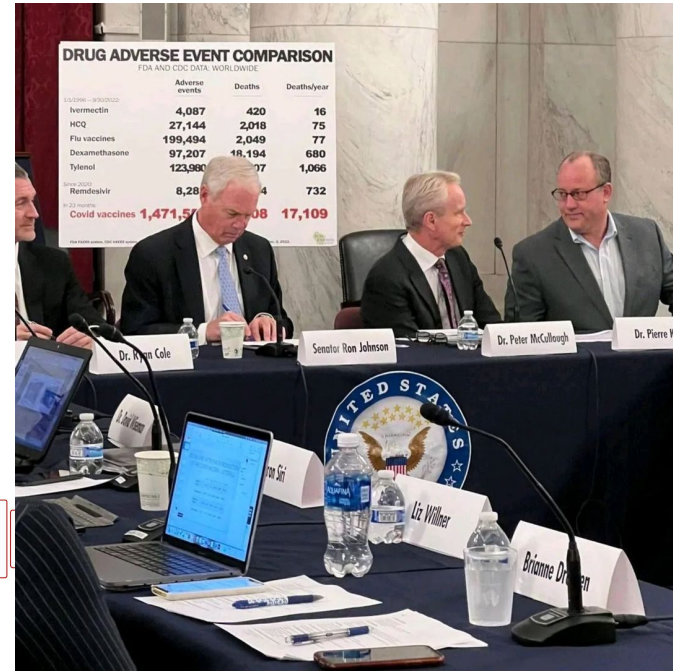
My mom never gave me antibiotics for "earache" as a child. Her solution was to **warm salt and place in a wash cloth which would then be placed over the ached ear. This is a Chinese Medicine trick to draw out the Yin energy (illness) from the ear with Yang salt. Her method is supported by the evidence. Placebo is noninferior to amoxicillin for otitis media.** Despite this well known fact, antibiotic overuse in children predominates medicine's fear-based culture of practice.

"Results: According to clinical scoring, 415 of the 512 children who could be evaluated had moderate disease. At 14 days 84.2% of the children receiving placebo and 92.8% of those receiving amoxicillin had clinical resolution of symptoms (absolute difference -8.6%, 95% confidence interval -14.4% to -3.0%). Children who received placebo had more pain and fever in the first 2 days. There were no statistical differences in adverse events between the 2 groups, nor were there any significant differences in recurrence rates or middle ear effusion at 1 and 3 months."
<https://pubmed.ncbi.nlm.nih.gov/15684116/>



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2. “Have to get fever down in kids because what about febrile seizure” - parents/CYA medicine



“Recent epidemiological observations suggest that acetaminophen (paracetamol) may contribute to asthma morbidity. Impaired endogenous antioxidant defences may have a role in the pathogenesis of a number of inflammatory pulmonary diseases, including asthma... clinically relevant concentrations of acetaminophen decreased: (i) intracellular Glutathione (GSH) in human pulmonary macrophages and type. These findings provide experimental plausibility to the challenging observations that frequent use of APAP may be a risk factor for asthma morbidity.” (Svetlana Dimova et al. Int J Biochem Cell Biol. 2005 Aug.)

“Treating fever” with Tylenol or Motrin is cheap but toxic. If looking to “treat viral illness” I would advise a comprehensive nutraceutical protocol.

Retrospective review of the management of simple febrile convulsions at a tertiary paediatric institution

Results: A total of 288 patients were identified. The patients were separated into two groups – those that were discharged from the ED and those that were admitted to the hospital...Of the 28 patients admitted to the hospital, 61% were diagnosed with a viral illness. (Dunlop, S. and Taitz, J. (2005), Retrospective review of the management of simple febrile convulsions at a tertiary paediatric institution. Journal of Paediatrics and Child Health, 41: 647-651.)

Assessment of febrile seizures in children

“There is no evidence of the effectiveness of antipyretics in preventing future FS. Prophylactic use of paracetamol, ibuprofen or a combination of both in FS, is thus a questionable practice. There is reason to believe that children who have experienced a simple FS are over-investigated and over-treated. This review aims to provide physicians with adequate knowledge to make rational assessments of children with febrile seizures.” (Fetveit, A. Assessment of febrile seizures in children. Eur J Pediatr 163: 17-27 (2008).)

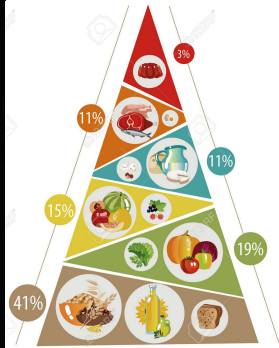
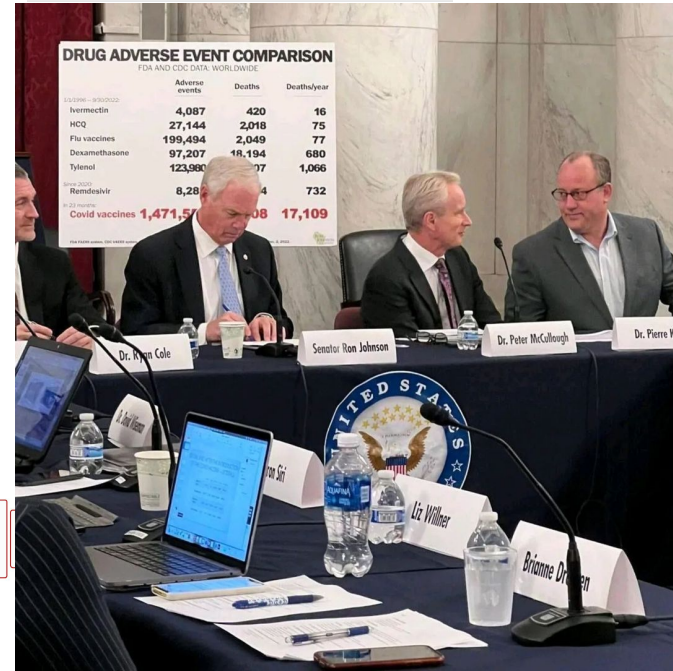
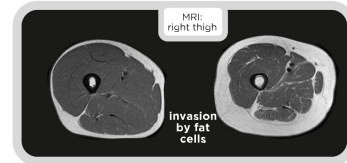
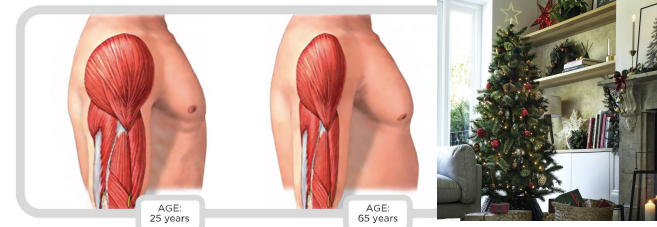
The Value of Early Postictal EEG in Children with Complex Febrile Seizures

Conclusions: The yield of abnormalities of an early postictal EEG in this population is low and similar to the reported rate of abnormalities in children with simple febrile seizures. The routine practice of obtaining an early EEG in neurologically normal children with complex febrile seizures is not justified. (Maytal, J., Steele, R., Eviatar, L. and Novak, G. (2000), The Value of Early Postictal EEG in Children with Complex Febrile Seizures. Epilepsia, 41: 219-221.)

Most fevers/cough are viral in children. For girls a UA might be necessary. Besides that, investigations are mostly unnecessary.

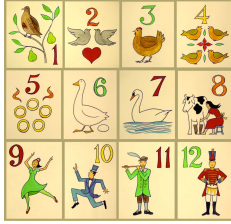


Twelve Medical Myths You Believe Pediatrics to Adults #3



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3. Myth: “ibuprofen is safe for athletic injury/recovery.”



NSAIDs interfere in every phase of the muscle healing process.

Duchesne, et al in an analysis of the pathophysiology of NSAID noted that “COX-2 inhibition does not only blunt the pro-inflammatory response but also inhibits the resolution of inflammation, which has a direct effect on muscle healing” (Duchesne, 2017)



Tscholl in the Swiss Journal of Sports & Exercise Medicine notes that up to 50% of elite athletes use NSAIDs and makes a few recommendations.

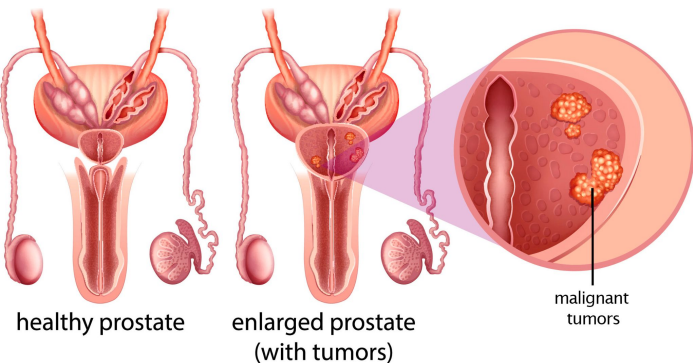
1. Impaired muscular and tendinous adaptation to exercise if NSAIDs are ingested chronically
2. Tscholl also agrees with what many orthopaedic surgeons have told me anecdotally is that there is the risk of delayed bone healing with NSAID use.

In a review article on the data of NSAID use, Lundburg in the Scandinavian Journal of Medicine and Sport Science advised “Both NSAIDs and paracetamol (tylenol) have been demonstrated to inhibit cyclooxygenase (COX) activity, which might explain the reduced anabolic response to acute exercise bouts. Consistent with this, NSAIDs have been reported to interfere with muscle hypertrophy and strength gains in response to chronic resistance training in young individuals” (Lundberg, 2018)

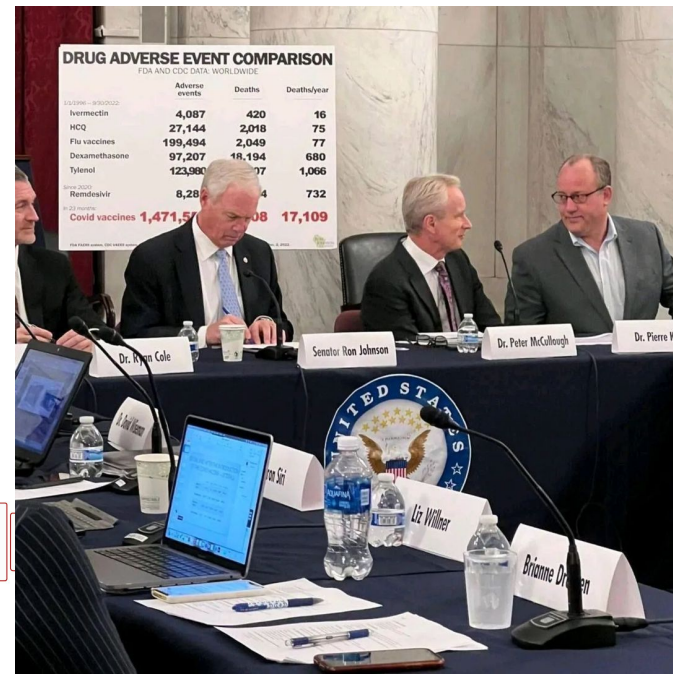


Twelve Medical Myths You Believe Pediatrics to Adults #4

Prostate Cancer



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4. Myth: “Vasectomy is perfectly safe.”



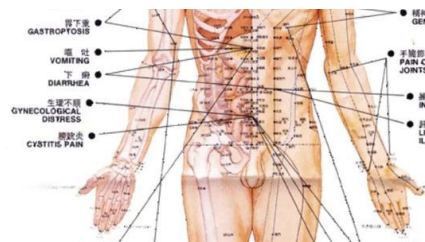
Review

Association between vasectomy and risk of prostate cancer: a meta-analysis

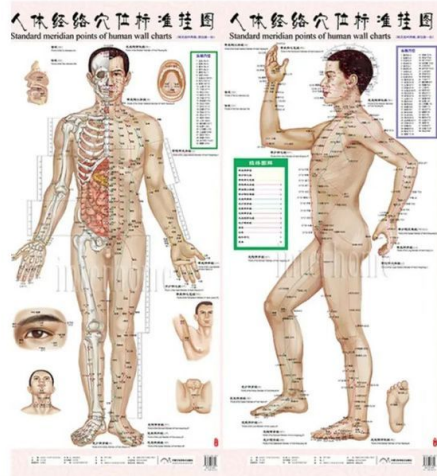
Yawei Xu et al. Prostate Cancer Prostatic Dis. 2021 Dec.

Results: A total of 58 studies involving 16,989,237 participants fulfilled inclusion criteria. There was significant association of vasectomy with risk of any prostate cancer (risk ratio, 1.18, 95% CI, 1.07-1.31). Association between vasectomy and advanced prostate cancer (risk ratio, 1.06, 95% CI, 1.01-1.12), low-grade prostate cancer (risk ratio, 1.06, 95% CI, 1.02-1.10), and intermediate-grade prostate cancer (risk ratio, 1.12, 95% CI, 1.03-1.22) were significant. There was no significant association between vasectomy and prostate cancer-specific mortality (risk ratio, 1.01, 95% CI, 0.93-1.10).

Conclusions: This study found that vasectomy was associated with the risk of any prostate cancer and advanced prostate cancer. From the current evidence, patients should be fully informed of the risk of prostate cancer before vasectomy.



Acupuncture chart



(Front)

(Side)

Blocking the flow of Qi (energy) through vasectomy has always been obviously wrong to myself who was raised as a boy to understand the principles of Chinese Medicine. This meta analysis raises red flag that blocking Qi energy may increase incidence of prostate cancer. However Western medicine has increasingly come to appreciate at least some Chinese medicine in the form of massage and acupuncture for the treatment of various ailments particularly in the chronic pain management arena. There is something deeply wrong about blocking vas deferens, a prominent source of Qi flow in Chinese Medicine.

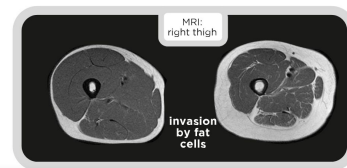
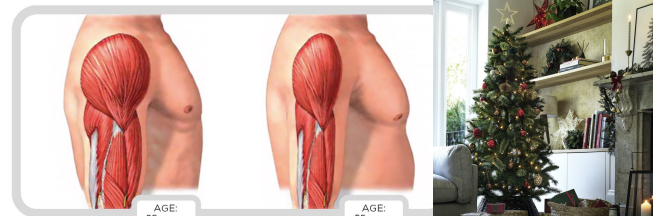
<https://pubmed.ncbi.nlm.nih.gov/33927357/>

Gift: 1 Chinese-English Instru

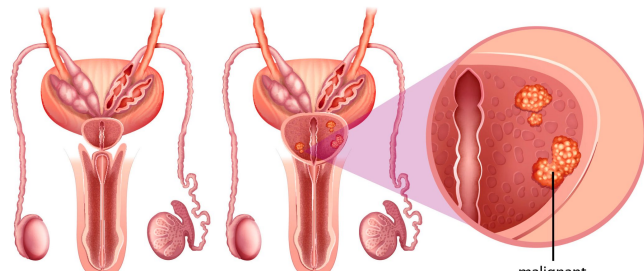
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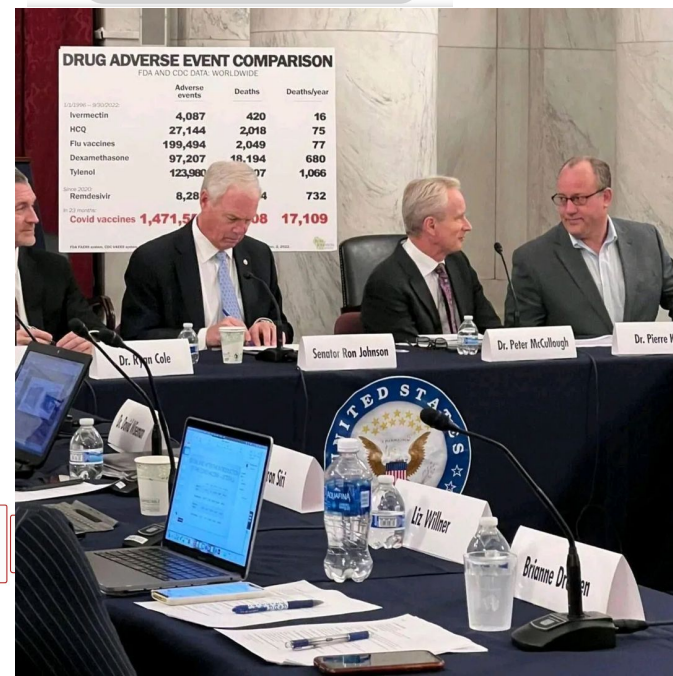
Twelve Medical Myths You Believe Pediatrics to Adults #5



Prostate Cancer



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	Adverse events	Deaths	Deaths/year
Ivermectin	4,087	420	16
HCQ	27,144	2,018	75
Flu vaccines	199,494	2,049	77
Dexamethasone	97,207	18,194	680
Tylenol	123,986	7	1,066
Remdesivir	8,284	4	732
Covid vaccines	1,471,508	108	17,109

Dr. Ryan Cole
Senator Ron Johnson
Dr. Peter McCullough
Dr. Pierre
Liz Wilber
Brianna D...



4. Myth: Testosterone isn't necessary to optimize.



DIABETES, OBESITY AND METABOLISM A JOURNAL OF PHARMACOLOGY AND THERAPEUTICS

ORIGINAL ARTICLE [Open Access](#)

Remission of type 2 diabetes following long-term treatment with injectable testosterone undecanoate in patients with hypogonadism and type 2 diabetes: 11-year data from a real-world registry study

Karim Sultan Haider MD, Ahmad Haider MD, Farid Saad DVM, Gheorghe Doros PhD,

James Cameron on making 'Titanic' and 'Avatar': I was 'a wild, testosterone-poisoned young man'

testosterone-poisoned young man. I always think of [testosterone] as a toxin that you have to slowly work out of your system."



TRT decreases mortality. All-cause mortality was significantly lower in the testosterone group as well (9.7% vs 64.3% among the small control group).

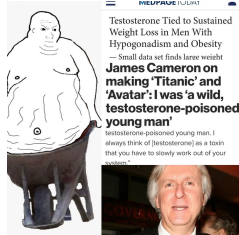
Testosterone Tied to Sustained Weight Loss in Men With Hypogonadism and Obesity

In registry data on 76 patients, those who opted to receive testosterone undecanoate experienced a 26% reduction in their body mass index (BMI) over close to a decade of follow-up, reported Farid Saad, DVM, PhD.

"Patients with hypogonadism and T2DM treated with testosterone had significant progressive and sustained reductions in fasting glucose, glycated haemoglobin (HbA1c) and fasting insulin over the treatment period. In the control group, fasting glucose, HbA1c and fasting insulin increased. Among the patients treated with testosterone 34.3% achieved remission of their diabetes and 46.6% of patients achieved normal glucose regulation. Of the testosterone-treated group, 83.1% reached the HbA1c target of 47.5 mmol/mol (6.5%) and 90% achieved the HbA1c target of 53.0 mmol/mol (7%). In contrast, no remission of diabetes or reductions in glucose or HbA1c levels were noted in the control group. There were fewer deaths, myocardial infarctions, strokes and diabetic complications in the testosterone-treated group." ([Haider et al 2020](#))

Yet you go to your PCP for diabetes and all you get is a higher dose of metformin or a sliding scale insulin.

TRT in Fat Men



Obese men with secondary hypogonadism achieved a **statistically significant reduction in fat mass (3.5 kg, p=0.03), increase in lean body mass (2.9 kg, p=0.03) and glycated haemoglobin (HbA1c) improvement (9 mmol/mol, p=0.03), associated with 52% improvement in beta-cell function with TRT.**⁷¹ Treatment also resulted in an overall improvement in metabolic state, including improvements in the following parameters: BMI, waist circumference, lipid profile, blood pressure, lipoprotein (a), haemoglobin, fasting glycaemia, insulin resistance and leptin resistance.^{72–78} TRT also reduced arterial stiffness, respiratory quotient, hepatic fat content and bone remodelling.^{79,80} TRT mediated and psychotropic effects included improved energy and motivation.⁸¹

Prediction of secondary testosterone deficiency using machine learning: A comparative analysis of ensemble and base classifiers, probability calibration, and sampling strategies in a slightly imbalanced dataset

Monique Tonani Novaes ^a, Osmar Luiz Ferreira de Carvalho ^b, Pedro Henrique Guimaraes Ferreira ^b, Taciana Leonel Nunes Tiraboschi ^a, Caroline Santos Silva ^a, Jean Carlos Zambrano ^a, Cristiano Mendes Gomes ^c, Eduardo de Paula Miranda ^d, Osmar Abilio de Carvalho Júnior ^e, José de Bessa Júnior ^f

Weight loss associated with TRT was almost exclusively due to loss of fat mass, whereas the weight loss due to dieting is from loss of both fat mass and lean mass. **Although TRT did not augment diet-induced loss of fat mass, it prevented diet-induced loss of muscle mass.**⁸² These favourable effects of TRT on body composition may be due to androgen receptor signalling in adipocytes and myocytes and/or increased physical activity seen in testosterone replaced men. However, successful visceral fat loss achieved by testosterone therapy is not maintained after cessation of TRT, as low testosterone (after TRT withdrawal) may lead to fatigue and inertia and less physical activity.^{83,84} This suggests that long term TRT is required to maintain the beneficial effects.

T on psychiatric symptoms [Treatment of Men for “Low Testosterone”: A Systematic Review \(Samantha Huo, Anthony R. Scialli, Sean McGarvey, 2016\)](#)

Twenty-nine of these studies focused on men without psychiatric disorders, and 16 on men with psychiatric disorders.

[58] found that testosterone treatment had no effect on the Hospital Anxiety Depression score (HADS) in men with testosterone ≤ 8.0 nM but improved the depression subset of the HADS in men with testosterone of 8.1–12 nM. Malkin et al.[1] found that 100 mg testosterone every 2 weeks improved the BDI score. This study had a Jadad score of 5.

The response of depression and dysthymia to testosterone was mixed and inconsistent. Among HIV-negative men, four studies (all with a Jadad score of 4 or 5) showed testosterone-associated improvements in standard scoring systems for depression and/or in the proportion of subjects who achieved remission of their psychiatric disorder.[37, 51, 125, 126] Four other studies (2 with a Jadad score of 4 or 5) showed no improvement in depression or dysthymia with testosterone compared to placebo.[36, 111, 127, 128] One study (Jadad score 4) showed a transient improvement in depression and melancholia after 3 months of treatment that was no longer apparent after 6 months of treatment.[]

The studies did not show consistent responses in subgroups of men who had low serum testosterone concentrations, depression resistant to standard therapy, or men characterized as middle-aged or elderly. In studies in which serum testosterone concentrations were measured on therapy (both with a Jadad score of 5), response of depression or dysthymia was not consistently associated with serum hormone concentration.

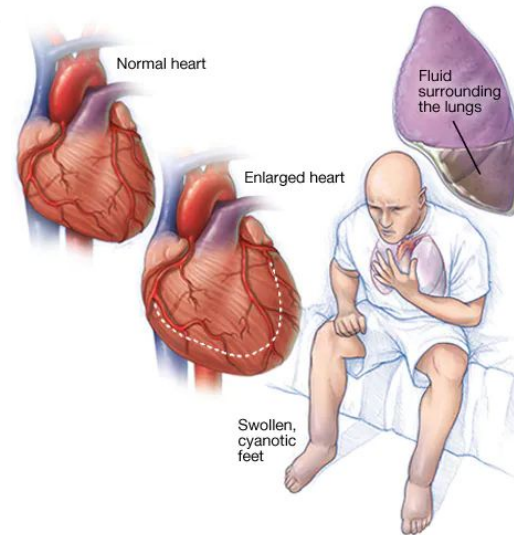
Testosterone and heart failure

The data shows that testosterone supplementation given in addition to optimal medical therapy improves functional capacity, large-muscle strength, and glucose metabolism in elderly patients with CHF. The increase in functional capacity and muscular strength is related to the increase in plasma levels of testosterone and not related to changes in left ventricular function.

Elie J. Chahla, Mireille El Hayek & John E. Morley (2011)

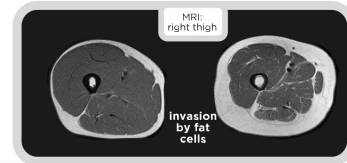
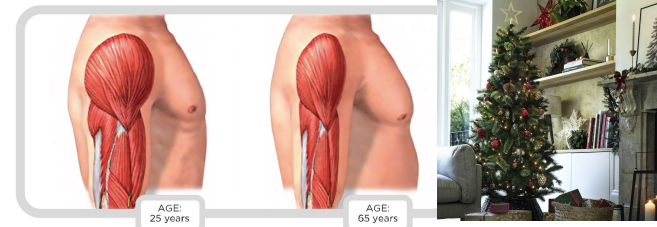
Testosterone replacement therapy and cardiovascular risk factors modification, *The Aging Male*, 14:2, 83-90, DOI:

[10.3109/13685538.2010.541538](https://doi.org/10.3109/13685538.2010.541538)

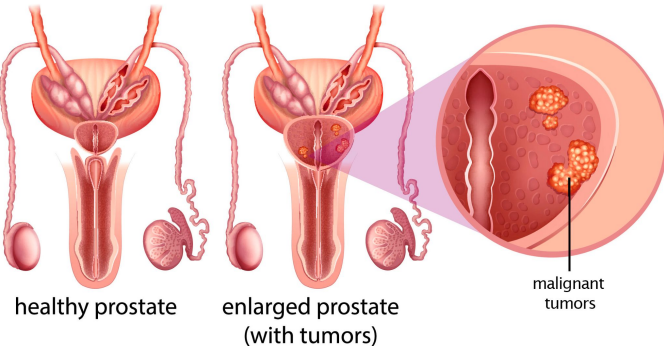




Twelve Medical Myths You Believe Pediatrics to Adults #6



Prostate Cancer



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	Adverse events	Deaths	Deaths/year
Ivermectin	4,087	420	16
HCQ	27,144	2,018	75
Flu vaccines	199,494	2,049	77
Dexamethasone	97,207	18,194	680
Tylenol	123,980	77	1,066
Remdesivir	8,280	4	732
Covid vaccines	1,471,500	108	17,109



Myth: “But doesn’t testosterone replacement cause prostate cancer?”



Low T is associated with worse cancer.

“It is becoming increasingly clear that testosterone therapy may have a role to play in controlling prostate cancer via optimisation of serum androgen concentrations; hypothesised by clinical observations showing low serum testosterone concentrations being associated with higher-grade prostate cancer [43,73,74]”

Is there a protective role of testosterone against high-grade prostate cancer? Incidence and severity of prostate cancer in 553 patients who underwent prostate biopsy: a prospective data register

Aksam Yassin , Mahmoud Salman, Riadh A. Talib & Dany-Jan Yassin
Pages 125-133 | Received 27 Sep 2016, Accepted 20 Feb 2017, Published online: 10 Mar 2017

The incidence of positive prostate biopsies was lowest in hypogonadal men receiving TRT, with significantly lower severity of PCa in terms of staging and grading in the same group. ([Aksam 2016](#))

Testosterone Therapy in Men with Biochemical Recurrence and Metastatic Prostate Cancer: Initial Observations

Abraham Morgentaler , Alejandro Abello , and Glenn Bubley

Published Online: 21 Jul 2021 | <https://doi.org/10.1089/andro.2021.0001>

Testosterone replacement in men with biochemical recurrence or metastatic prostate cancer and high-risk PCa was associated with symptomatic benefits and low rates of complications, although interpretation of these results must be tempered by small sample size and a heterogeneous population. Well-designed prospective studies are needed to provide better evidence for the potential use of TTh in similarly affected individuals. In the meantime, these results may provide clinicians with a framework to counsel patients who prioritize quality of life over longevity.

Review

Testosterone Therapy Among Prostate Cancer Survivors

Results Taylor M. Nguyen¹, Alexander W. Pastuszak MD, PhD^{2,3}✉

[Testosterone therapy ameliorates the symptoms of hypogonadism](#), decreases the risk for its negative sequelae, and can significantly improve quality of life. Recent studies do not support an increased risk for de novo prostate cancer, progression of the disease, or [biochemical recurrence](#) in hypogonadal men with a history of non-high-risk prostate cancer treated with testosterone therapy. Evidence supporting the use of testosterone in the setting of high-risk prostate cancer is less clear.

Conclusion

Despite the historical reluctance toward the use of testosterone therapy in men with a history of prostate cancer, modern evidence suggests that testosterone replacement is a safe and effective treatment option for hypogonadal men with non-high-risk prostate cancer. Additional work to definitively demonstrate the efficacy and safety of testosterone therapy in men with prostate cancer is needed, and persistent vigilance and surveillance of treated men remains necessary.

Investigation, Treatment and Monitoring of Late-onset Hypogonadism

Published Online: April 2nd 2012

European Endocrinology, 2006(2):84-87; DOI:<http://doi.org/10.17925/EE.2006.00.02.84>

Authors: Claude Schulman, Jean-Marc Kaufman

T replacement and Prostate Cancer

There is no evidence that administration of testosterone can induce the appearance of prostate cancer if it is non-existent. On the contrary, more recent studies have shown that hypogonadal men may have a higher incidence of prostate cancer and that low total or free testosterone may be associated with more aggressive (Gleason score >7) and more extensive disease. It has to be considered that keeping an appropriate level of testosterone might be protective for prostate cancer. It must be remembered that there is a clear inverse relationship between the decrease of testosterone with age and the opposite increase of prostate cancer incidence with age. Finally, androgen deprivation through surgical or chemical castration is only a palliative treatment of advanced disease and almost all men dying from advanced metastatic prostate cancer are castrated and have no testosterone.

Won't T replacement cause heart attacks?

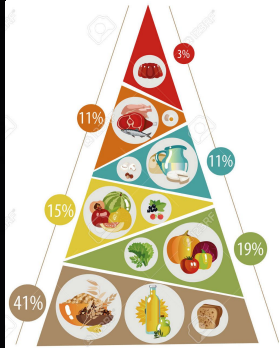
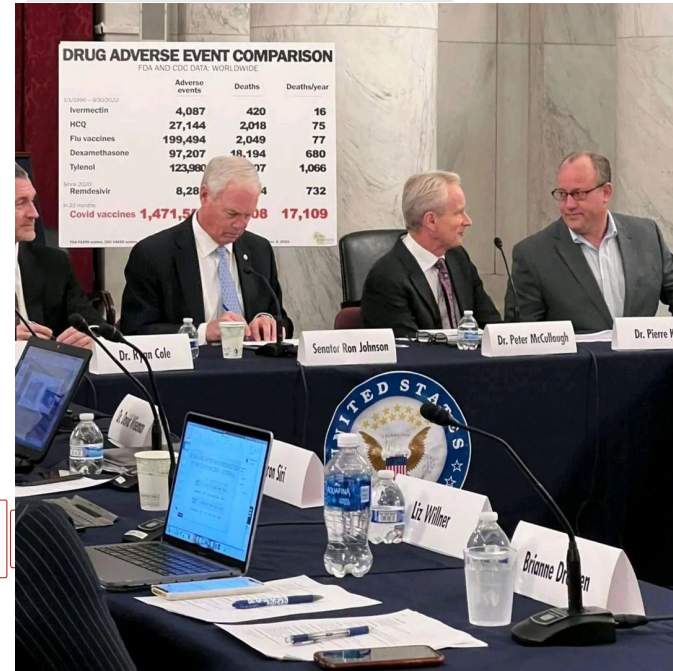
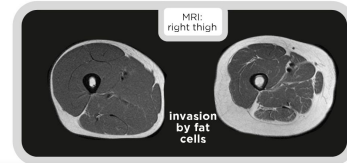
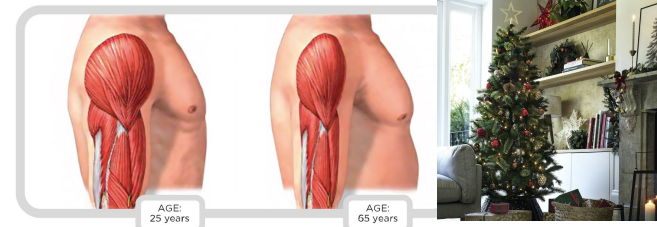
Due to conflicting evidences relating TRT to cardiovascular disease (CVD) and mortality, the US Food and Drug Administration has warned that TRT might increase heart attack and stroke. **A critical review of all the published systematic reviews found that two out of six systematic reviews and meta-analyses showed increased CVD risk with TRT.**⁹² However, recent reviews shows that there is a trend towards reduced major adverse cardiac events, all cause and cardiovascular mortality with TRT, with the greatest benefit seen in men at highest risk of CVD, such as those with T2DM and metabolic syndrome.⁹³ Moreover, greater benefits are observed among those treated to the target and for longer duration.⁹⁴ A recently published population-based observational study showed that longer duration TRT is associated with decreased mortality, CVD events and prostate cancer, whereas shorter duration TRT increased mortality and CVD events.⁹⁵

[A study we will look at in two slides on:](#) ‘Our results provide evidence that oral TU treatment has a beneficial effect on arterial stiffness in older men with testosterone at or less than the lower limit of normal range. In light of recent research showing that arterial stiffness was a predictor of cardiovascular outcomes,^{16, 17} our results may suggest that in the longer term, oral TU could potentially confer cardiovascular benefit through its effects on arterial stiffness”





Twelve Medical Myths You Believe Pediatrics to Adults #7



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Myth: “Saturated fat causes heart disease” - vegan cardiologists, your mother in law, medtwitter influencers

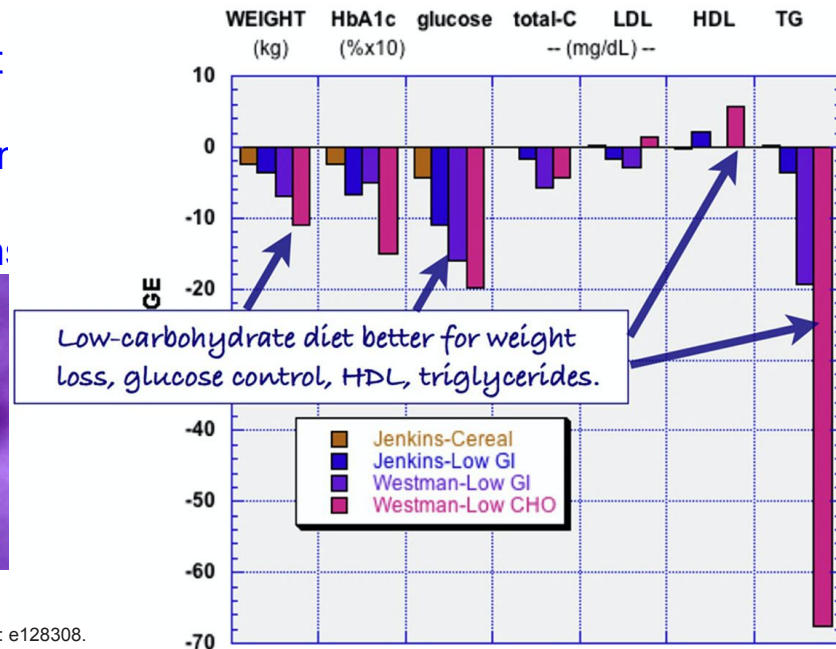
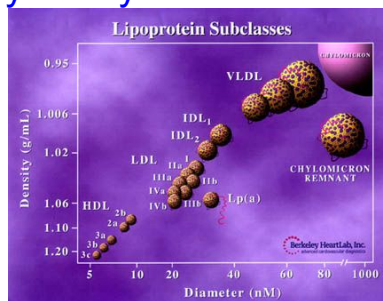


Advent
Calendar:
Twelve
Medical
Myths You
Believe
Pediatrics
to Adults

A paradigm shift from the American College of Cardiology. They state they have perhaps been wrong about saturated fats over the past 40 years and they are **not** linked to heart disease. They also suggest that focusing on the standard lipid panel is inadequate and small LDL size is more indicative of heart disease risk rather than just using standard LDL as a risk factor tool. [ACC](#)

The Brits are onto it as well. “Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition risk of which can be effectively reduced from healthy lifestyle intervention: (BMJ, 2017)

Dietary carbohydrate restriction improves metabolic syndrome independent of weight loss



“A LC diet increases LDL size and decreases small, dense LDL particles independent of LDL-C concentration.”

[JCI Insight](#). 2019 Jun 20; 4(12): e128308. Published online 2019 Jun 20. doi: [10.1172/jci.insight.128308](https://doi.org/10.1172/jci.insight.128308)

Protective LDL pattern A is found in the LC group and the pathogenic Phenotype B in the HC group.

Low carbohydrate diets are better than low-GI diets or high cereal diets for weight loss, HbA1c, triglycerides and HDL. Data from Westman, *et al* (2008) *Nutr Metab (Lond)*, 5 (36). and Jenkins, *et al* (2008), *JAMA* 300: 2742-2753.

The problem of cholesterol and atherosclerosis has been mentioned above. Wilber & Levine (1950) concluded that 'In the Alaskan Eskimos . . . there is a consistently high serum cholesterol on the one hand ; repeated clinical surveys, on the other, indicate an almost total absence of cardiovascular-renal diseases in the population' [their means are, however, 203 mg/100 ml. serum for males and 234 for females which, despite the ages not being stated, would hardly appear high from the data of Keys (1949)]. Others have commented on this absence in the Eskimo on his customary diet, but Rabinowitch (1936) believed he had 'definitely disproved the alleged incidence of arteriosclerosis in the Eskimo, at least in the Eastern Arctic'; his data seem to show that it was common in those Eskimos consuming our diet but there was no evidence of arteriosclerosis in the most northerly parts he visited where the true Eskimo dietary was practised. The same seems to be true of tuberculosis, diabetes mellitus, appendicitis, cancer and dental caries, although tuberculosis is becoming a severe problem (Lewis & Wherrett, 1947). Parasitism, including trichinosis, is extremely common (Brown, Green, Boag & Kuitunen-Ekbaum, 1950 ; Brown, Sinclair, Cronk & Clark with Kuitunen-Ekbaum, 1948 ; Brown, Cronk, deSinner, Green, Gibbons & Kuitunen-Ekbaum, 1949a ; Brown, Cronk, deSinner, Green, Gibbons & Kuitunen-Ekbaum, 1949b ; Hitchcock, 1950 ; Roth, 1949).

We may picture the true Eskimo, then, as a person who has adapted with extraordinary efficiency to subsisting in the Arctic as a typical carnivorous animal, and who is peaceful, extremely happy and healthy (except for occasional periods of starvation). But contact with white traders, trappers and missionaries is causing malnutrition and ill-health, as has happened extensively in the Indians. The contamination of these may now be considered.

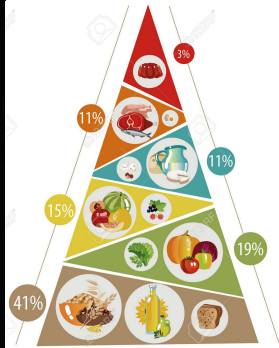
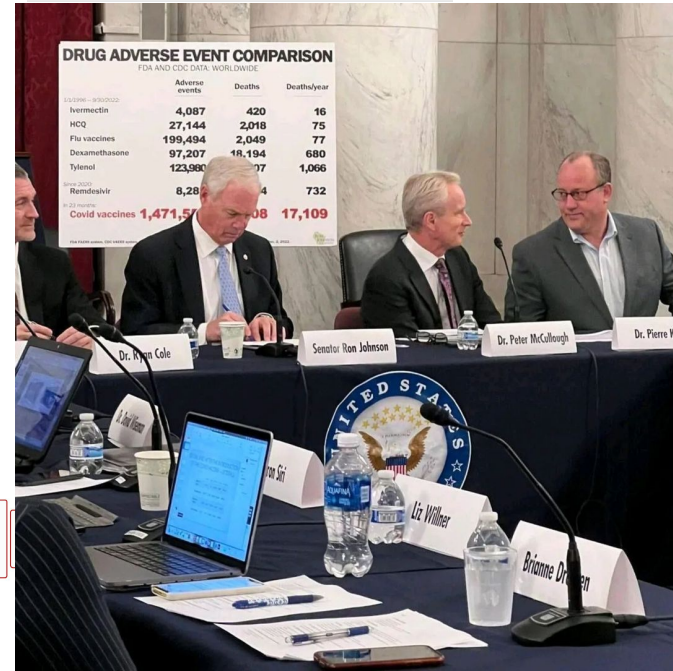
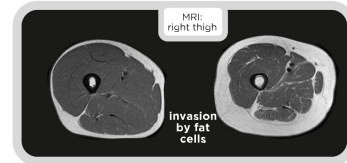
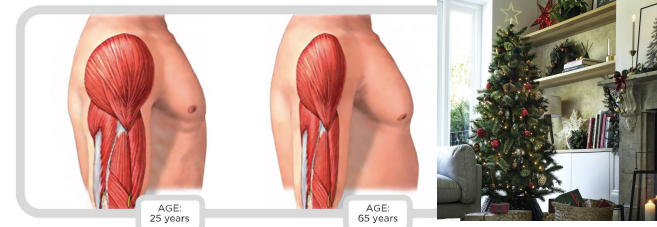
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These scientists in the arctic were forgotten to medicine and Ancel Keys who started the lipid hypothesis led to the next century of low-fat diets and high carb diets.





Twelve Medical Myths You Believe Pediatrics to Adults #8



Stefan Hartmann,
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Muscle Centric Medicine



Advent Calendar: Twelve Medical Myths You Believe Pediatrics to Adults



8. Myth: "Sunscreen is safe"



Double tap to zoom

Active Ingredients

Avobenzene 2.7%, Homosalate 9.0%,
Octisalate 4.5%, Octocrylene 6.0%

Inactive ingredients: Alcohol Denat., Isobutane, Isododecane,
Diisopropyl Adipate, VA/Butyl Maleate/Isobornyl Acrylate Copolymer,
Caprylyl Glycol, Butyloctyl Salicylate, C12-15 Alkyl Benzoate, Fragrance,
Tocopheryl Acetate.

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**Chemical Research
in Toxicology**

Get e-Alerts

Benzophenone Accumulates over Time from the Degradation of Octocrylene in Commercial Sunscreen Products

C. A. Downs*, Joseph C. DiNardo, Didier Stien,
Alice M. S. Rodrigues, and Philippe Lebaron

[Cite this:](#) *Chem. Res. Toxicol.* 2021, 34, 4, 1046–1054

Whatever you place on your skin goes into your bloodstream. Your skin is the largest organ in your body and is a sponge.

"Benzophenone is a mutagen, carcinogen, and endocrine disruptor. Its presence in food products or food packaging is banned in the United States. Under California Proposition 65, there is no safe harbor for benzophenone in any personal care products, including sunscreens, anti-aging creams, and moisturizers. The purpose of this study was to determine (1) if benzophenone was present in a wide variety of commercial sun protection factor (SPF)/sunscreen products, (2) whether benzophenone concentration in the product increased over time, and (3) if the degradation of octocrylene was the likely source for benzophenone contamination."

Results: "In vivo, up to 70% of the benzophenone in these sunscreen products may be absorbed through the skin...In the United States, there were 2999 SPF products containing octocrylene in 2019. The safety of octocrylene as a benzophenone generator in SPF or any consumer products should be expeditiously reviewed by regulatory agencies."

Chem. Res. Toxicol. 2021, 34, 4, 1046–1054

Publication Date: March 7, 2021

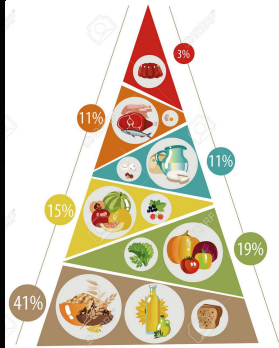
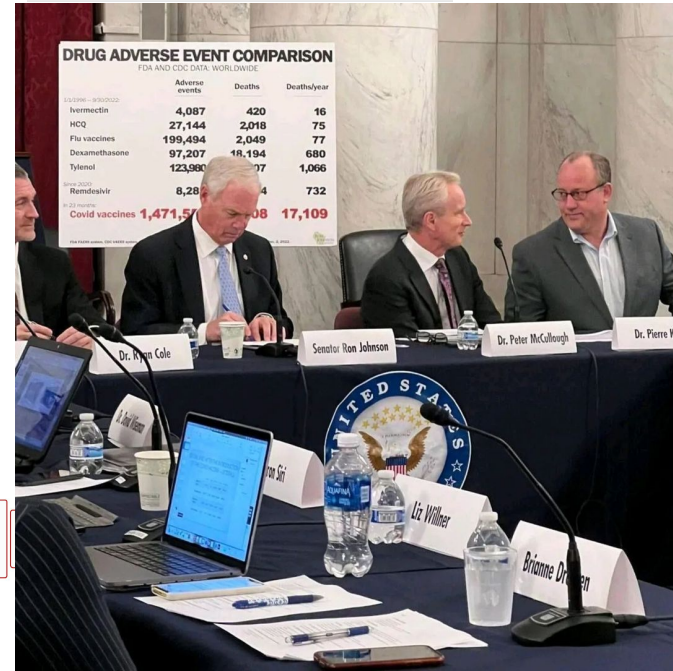
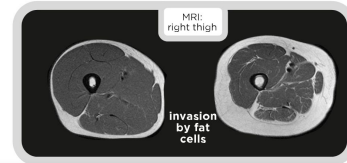
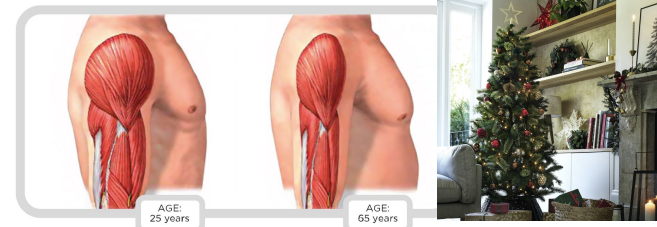
July 15, 2021 · 5:33 PM EDT
Last Updated a year ago

Healthcare & Pharmaceuticals

Pharmacies pull J&J sunscreens off shelves after carcinogen found in some sprays



Twelve Medical Myths You Believe Pediatrics to Adults #9



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Low salt diet interventions to reduce blood pressure and prevent cardiovascular mortality are misguided.



Salt and hypertension, what do we know? [DiNicolantonio, James J.; O'Keefe](#)

Recent findings

The recommendations for population-wide sodium restriction largely rely on one surrogate marker (blood pressure). However, recent evidence suggests that when looking beyond blood pressure (e.g. heart rate, aldosterone, renin, cholesterol, triglycerides, noradrenaline and adrenaline), the net effect of sodium restriction is likely harmful. Prospective studies support the notion that **those consuming the lowest amounts of salt are at the highest risk of cardiovascular events and premature death.**

Summary

There is no definitive proof that sodium restriction reduces cardiovascular events or death. It is time for the dietary guidelines to look at the totality of the evidence and reconsider the advice around population-wide sodium restriction.

[Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride](#), A Cochrane review of evidence.

Niels Albert Graudal ¹, Thorbjørn Hubeck-Graudal

Background: Recent cohort studies show that salt intake below 6 g is associated with increased mortality. These findings have not changed public recommendations to lower salt intake below 6 g, which are based on assumed blood pressure (BP) effects and no side-effects.

They looked at over 6,000 patients in about 90 different trials. The results were less than impressive for advocating salt reduction to treat hypertension.

Authors' conclusions: In white participants, sodium reduction in accordance with the public recommendations resulted in mean arterial pressure (MAP) **decrease of about 0.4 mmHg** in participants with normal blood pressure and a MAP **decrease of about 4 mmHg** in participants with hypertension. Weak evidence indicated that these effects may be a little greater in black and Asian participants. The effects of sodium reduction on **potential side effects (hormones and lipids) were more consistent than the effect on BP, especially in people with normal BP.**

Possibly the reason for the slight decrease in BP is that these hypertensives are consuming less processed carbs which incidentally also have high sodium. Let's look briefly at why this may be the reason for the widespread salt reduction guidelines.



The wrong white crystals: not salt but sugar as aetiological in hypertension and cardiometabolic disease (DiNicolantonio, Lucan)

- ▶ Sugar may be more meaningfully related to blood pressure than sodium, as suggested by the greater magnitude of effect with dietary manipulation.^{10 77}
- ▶ Reducing the amount of sodium in processed foods may lead to an increase in their consumption causing a greater prevalence of cardiometabolic disease (figure 1).²³
- ▶ Higher sugar intake significantly increases systolic (6.9mm Hg) and diastolic blood pressure (5.6 mm Hg) in trials of 8 weeks or more in duration.⁷⁷ This effect is increased to 7.6/6.1 mm Hg, when studies that received funding from the sugar industry are excluded.
- ▶ Ingesting one 24 ounce soft drink has been shown to cause an average maximum increase in blood pressure of 15/9 mm Hg and heart rate of 9 bpm.⁸¹
- ▶ Those who consume 25% or more calories from added sugar have an almost threefold increased risk of death due to cardiovascular disease.⁶²
- ▶ Fructose has been shown to stimulate sympathetic tone directly,²⁶ and indirectly by inciting insulin resistance and hyperinsulinaemia.^{27 45 46}

- ▶ An increase in sympathetic tone from the overconsumption of fructose is one likely mechanism for the sugar's ability to increase heart rate, cardiac output, renal sodium retention, and vascular resistance, all of which may interact to elevate blood pressure and increase myocardial oxygen demand.^{27 80 82}
- ▶ A high-fructose diet for just 2 weeks not only significantly increased 24 h ambulatory blood pressure (+7/5 mm Hg, $p < 0.004$ and $p = 0.007$, respectively) and increased pulse rate by 8% (4 bpm), but also increased triglycerides, fasting insulin, and homeostatic model assessment (HOMA) index (a measure of insulin resistance and β -cell function).⁸⁶ Excess fructose intake has also been shown to double the prevalence of the metabolic syndrome.⁸⁶
- ▶ Current US per capita intake of added sugars is approximately 2–8 times higher than current recommendations by the American Heart Association (AHA) and WHO.^{91 92} Considering adolescents specifically, current consumption might be as much as 6–16 times higher.⁹⁰
- ▶ Ingestion of sugars, including fructose, in their naturally occurring biological contexts (eg, as whole fruits) is not harmful and is likely beneficial.^{88 89}

While the potential benefits of sodium-reduction strategies are debatable, one fact about which there is little debate is that the predominant sources of sodium in the diet are industrially processed foods. Processed foods also happen to be generally high in added sugars, the consumption of which might be more strongly and directly associated with hypertension and cardiometabolic risk. Evidence from epidemiological studies and experimental trials in animals and humans suggests that added sugars, particularly fructose, may increase blood pressure and blood pressure variability, increase heart rate and myocardial oxygen demand, and contribute to inflammation, insulin resistance and broader metabolic dysfunction. Thus, while there is no argument that recommendations to reduce consumption of processed foods are highly appropriate and advisable, the arguments in this review are that the benefits of such recommendations might have less to do with sodium—minimally related to blood pressure and perhaps even inversely related to cardiovascular risk—and more to do with highly-refined carbohydrates. It is time for guideline committees to shift focus away from salt and focus greater attention to the likely more-consequential food additive: sugar

<https://pubmed.ncbi.nlm.nih.gov/33351135/>

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Systolic blood pressure (mmHg)	Relative Risk (95% CI)
160 to 169	0.65 [0.53 to 0.79]
150 to 159	0.75 [0.71 to 0.79] 0.75 [0.71 to 0.79] 0.63 [0.55 to 0.73]
140 to 149	0.76 [0.73 to 0.80] 0.73 [0.70 to 0.77] 0.68 [0.61 to 0.77]
130 to 139	0.85 [0.81 to 0.89] 0.95 [0.93 to 0.97] 0.77 [0.69 to 0.85]

Richard Amerling
@dramerling

As arteries stiffen with age it takes more cuff pressure to compress them. 140-160/90-100 in the elderly is pseudohypertension and should not be treated. @ElieJarrougeMD

8:37 PM · 11/13/24 · 2.4K Views

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Most relevant replies

R @raja_sekar_s · 2d
Dr. BMI Hedge said 40 years back 160 is normal BP.

PrarieFlower @SomeChickInKS · 6h
What's your thoughts in regard to this? [x.com/somechickinks/...](https://x.com/somechickinks/)

11:19

Post

P. D. Mangan Health & Freedom Max... · 3d

Blood pressure: lowest mortality risk in adults >75 years old was 140-160, i.e. "high" BP

Higher mortality if BP <130.

"In adults above 75 years with moderate to severe frailty and all above 85 years, there was no increased mortality risk with hypertension."

PMID: 32133525

Systolic blood pressure (mmHg)	Relative Risk (95% CI)
150 to 159	0.94 [0.92 to 0.97] 0.88 [0.87 to 0.92]
140 to 149	0.95 [0.93 to 0.98] 0.89 [0.85 to 0.93] 0.94 [0.87 to 1.01]
130 to 139	1.11 [1.07 to 1.15] 1.18 [1.12 to 1.24] 1.19 [1.07 to 1.32]
120 to 129	1.50 [1.42 to 1.59] 1.60 [1.58 to 1.73] 1.62 [1.46 to 1.79]
<120	0.83 [0.77 to 0.89] 0.94 [0.79 to 0.93] 0.61 [0.49 to 0.77]
>=180	0.75 [0.70 to 0.80] 0.75 [0.71 to 0.79] 0.65 [0.53 to 0.79]
170 to 179	0.76 [0.73 to 0.80] 0.68 [0.61 to 0.77]
160 to 169	0.85 [0.81 to 0.89] 0.95 [0.93 to 0.97] 0.77 [0.69 to 0.85]
150 to 159	0.76 [0.73 to 0.80] 0.73 [0.70 to 0.77] 0.68 [0.61 to 0.77]
140 to 149	0.85 [0.81 to 0.89] 0.95 [0.93 to 0.97] 0.77 [0.69 to 0.85]
130 to 139	Ref Ref Ref

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10:28

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Observational Study

Sodium intake, life expectancy, and all-cause mortality

Franz H Messerli et al. Eur Heart J. 2021.

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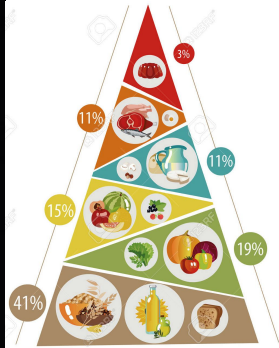
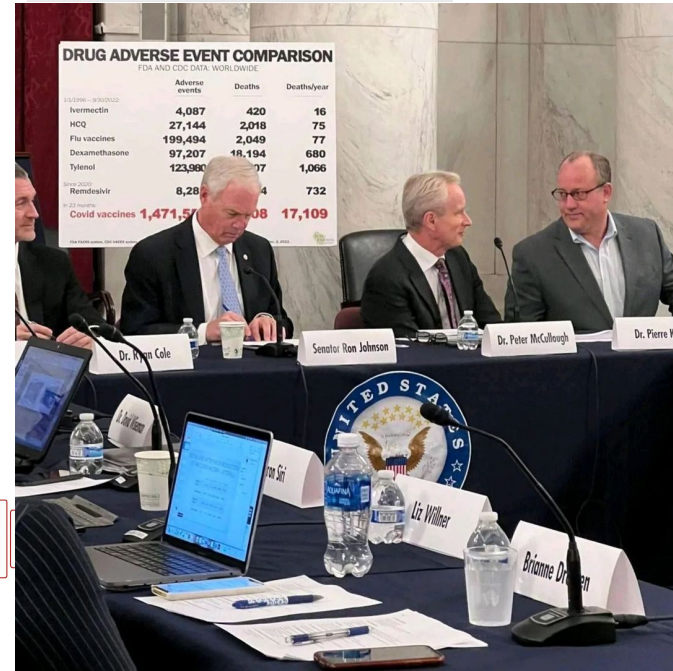
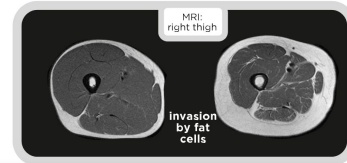
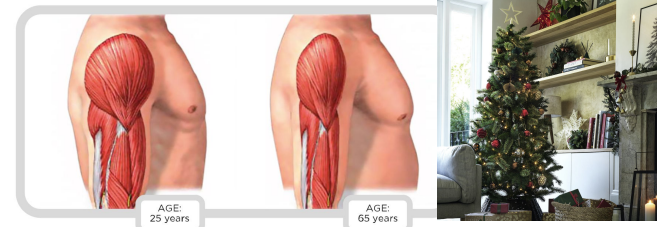
Abstract

Aims: Since dietary sodium intake has been identified as a risk factor for cardiovascular disease and premature death, a high sodium intake can be expected to curtail life span. We tested this hypothesis by analysing the relationship between sodium intake and life expectancy as well as survival in 181 countries worldwide.

Methods and results: We correlated age-standardized estimates of country-specific average sodium consumption with healthy life expectancy at birth and at age of 60 years, death due to non-communicable diseases and all-cause mortality for the year of 2010, after adjusting for potential confounders such as gross domestic product per capita and body mass index. We considered global health estimates as provided by World Health Organization. Among the 181 countries included in



Twelve Medical Myths You Believe Pediatrics to Adults #10



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#10. Myth: Generic “Increase fiber” advice

FIBER IN CONSTIPATION AND IRRITABLE BOWEL

SYNDROME. “Most physicians, including gastroenterologists and colorectal surgeons are quick to prescribe fiber supplements for constipation, citing inadequate fiber as the cause for constipation. Most patients complaining of constipation are likely to receive additional fiber from their doctors.” However, the evidence for this intervention is lacking.



1. A recent study from Brazil found that a low dietary fiber dosage was not associated with constipation[36]. **Several studies that looked at dietary fiber intake by people with chronic constipation did not find any difference in fiber intake compared to controls[37-40].**
2. Voderholzer et al[42] studied 149 patients with chronic constipation in Germany. The patients were treated with *Plantago ovata* seeds, 15-30 g/d for a period of 6 wk. They found that 80% of patients with slow transit and 63% of patients with a **disorder of defaecation did not improve with additional dietary fiber.**
3. There are also a number of reviews on the role of fiber in the treatment of irritable bowel syndrome. A review of **17 studies mostly using either ispaghula or wheat bran, found that fiber only conveyed marginal benefits on global irritable bowel syndrome symptoms and constipation, emphasizing that insoluble fiber may even worsen the clinical outcome[43].** A meta-analysis of the use of bulking agents in irritable bowel syndrome was performed in Switzerland[44]. After exclusion of low-quality trials, the odds ratio of symptomatic improvement with bulking agents did not reach statistical significance. **A Cochrane review also found that there was no clear evidence of benefit for bulking agents in irritable bowel syndrome[45].** A more recent **randomized-controlled trial in the UK also failed to show benefits of fiber over placebo[46].**
4. Muller-Lissner emphasized that a diet poor in fiber should not be assumed to be the cause of chronic constipation. In contrast, **they found that many patients with severe constipation deteriorated when dietary fiber intake was increased[35].**

FIBER IN CONSTIPATION AND IRRITABLE BOWEL SYNDROME. “Most physicians, including gastroenterologists and colorectal surgeons are quick to prescribe fiber supplements for constipation, citing inadequate fiber as the cause for constipation. Most patients complaining of constipation are likely to receive additional fiber from their doctors.” However, the evidence for this intervention is lacking. [World J Gastroenterol.](#) 2007 Aug 21; 13(31): 4161–4167. Published online 2007 Aug 21. doi: [10.3748/wjg.v13.i31.4161](#)

1. A study from Brazil found that a low dietary fiber dosage was not associated with constipation[[36](#)]. **Several studies that looked at dietary fiber intake by people with chronic constipation did not find any difference in fiber intake compared to controls**[[37-40](#)]. [Kok-Yang Tan](#) and [Francis Seow-Choen](#)
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Rees G, Davies J, Thompson R, Parker M, Liepins P. Randomised-controlled trial of a fibre supplement on the symptoms of irritable bowel syndrome. J R Soc Promot Health. 2005;125:30–34.
4. Muller-Lissner emphasized that a diet poor in fiber should not be assumed to be the cause of chronic constipation. In contrast, **they found that many patients with severe constipation deteriorated when dietary fiber intake was increased**[[35](#)].

HEMORRHOIDS, FISSURES,

“The need to evacuate large bulky stools frequently may also give rise to various anorectal disorders including haemorrhoids and anal fissures.” Since increased fiber causes increased bulk of stool it would likely be harmful to prescribe fiber for hemorrhoids or fissure treatment.

DIVERTICULOSIS- Traditionally thought to be caused by increased pressure in colon due to decreased fiber in Western Civilization.

Enter Asian studies whose population traditionally consume increased fiber: *Pattern and distribution of colonic diverticulosis: Analysis of 2877 barium enemas in Thailand*, ([Lohsiriwat](#), [Suthikeeree](#) 2013).

“Colonic diverticulosis... (has) widespread appearance in the Asian population... as early as adolescence[[20](#)] with peak prevalence at the age of 50-60 years[[10](#)].” This same kind of study done in 1991 in Singapore showed the same [Chia, Wilde, Ngoi, Tan and Choen](#) theorize that fiber could play a role in diverticular disease “We believe this is probably related to the massive gaseous build up associated with a high dietary fiber intake and that in fact fiber has an adverse effect on diverticular disease.”

FIBER AND COLORECTAL CANCER

Table 2

Case-control studies correlating dietary fiber with colorectal neoplasia

Longitudinal studies correlating dietary fiber with colorectal neoplasia

Reference	Location	Cohort	Follow-up period (yr)	Odds ratio of CRC comparing highest to lowest fiber intake groups	Dietary fiber protective	Reference	Country	Number of cases/controls	Odds ratio of CRC comparing highest to lowest fiber intake groups	Dietary fiber protective	
						Wakai et al 2006[15]	Japan	507/2535	0.65 ($P < 0.05$)	Yes (colon)	
Fuchs et al 1999[24]	US	88 757 (women)	16	0.95 (95% CI 0.73-1.25)	No	Levi et al 2001[16]	Switzerland	286/550	0.55 ($P < 0.05$)	Yes	
Mai et al 2003[25]	US	45 491 (women)	8.5	0.94 (95% CI 0.71-1.23)	No	Ghadirian et al 1997[17]	Canada	402/668	0.50 ($P < 0.01$)	Yes	
Lin et al 2005[26]	US	36 976 (women)	10	0.75 (95% CI 0.48-1.17)	No	Slattery et al 1997[18]	United States	1993/2410	0.70 (95% CI 0.5-1.0)	No	
Otani et al 2006[27]	Intervention studies correlating dietary fiber with colorectal neoplasia					No	Little et al 1993[19]	United Kingdom	147/329	0.60 (not significant after adjustment for energy intake)	No
Shin et al 2006[28]	Reference	Setting	n	Intervention	End point	Odds ratio of recurrence in intervention group	Dietary fiber protective				

Alberts et al 2000[30]	Postpolypectomy	2079	Counselling	Recurrent adenoma at 4 yr	0.88 (95% CI 0.70-1.11)	No
Schatzkin et al 2000[31]	Postpolypectomy	1429	Fiber supplement	Recurrent adenoma at 36 mo	1.00 (95% CI 0.90-1.12)	No
Ishikawa et al 2005[33]	Postpolypectomy	398	Fiber supplement	Recurrent adenoma at 4 yr	1.31 (95% CI 0.87-1.98)	No
Jacobs et al 2006[34]	Postpolypectomy	3209	Fiber supplement	Recurrent adenoma	0.91 (95% CI 0.78-1.06)	No
		Pooled from 2 studies				

“In summary, a strong recommendation cannot be made for a protective effect of dietary fiber against colorectal polyp or cancer. Despite a lack of evidence however, current recommendations are still to increase dietary fiber. In the latest position statement of the American Dietetic Association[3], increasing dietary fiber is still promoted to protect against colon cancer despite stating that there is no proof of efficacy in this regard.”

Fiber does not necessarily prevent constipation

“It is a misconception that dietary fiber and all isolated fibers provide a laxative effect in patients with Chronic Idiopathic Constipation. Our analysis suggests that treatment guidelines for CIC should make specific evidence-based recommendations as it pertains to fiber. To do otherwise takes the risk of perpetuating myth and misunderstanding and depriving patients of an effective therapy for CIC. A generic recommendation to “increase fiber intake” is akin to a recommendation to “increase pill intake” without regard to therapeutic or adverse effects,” (American Association of Nurse Practitioners, 2020)

A meta-analysis in 2012 also couldn't come to a definite conclusion. “In summary, our meta-analysis demonstrated that dietary fiber can obviously increase stool frequency in patients with constipation. The result also showed that dietary fiber did not obviously improve stool consistency, treatment success, laxative use and painful defecation. However, there were some possible influential factors such as small sample-sized studies, severity of constipation, assessment method for outcomes, *etc.* So further large trials examining the effect of dietary fiber in the treatment of constipation are needed, the possible influential factors should be taken into consideration, and more gastrointestinal symptoms and adverse events should be reported before dietary fiber was formally recommended.”

[Evidence From the National Health and Nutrition Examination Survey 2005-2010.](#)

“In conclusion, after adjusting for gender, age, ethnicity, education, marital status, income-poverty ratio, BMI, smoking, poor oral health, vitamin D deficiency, depression, diabetes, chronic diseases, milk, total fat, carbohydrates, protein, total saturated fatty acids, cholesterol, alcohol, moisture, and physical activity, multivariate logistic analysis results did not reveal a significant relationship between dietary fiber intake and constipation. However, for physically active participants, increasing dietary fiber intake was associated with stool consistency-related constipation while it was not strongly related to stool consistency-related constipation among non-active participants. In addition, increasing the intake of dietary fiber is not significantly associated with stool frequency in different physical activity groups.”

Treating GI disorders with fiber is not supported by science

Fiber and colorectal diseases: Separating fact from fiction

[Kok-Yang Tan](#) and [Francis Seow-Choen](#)

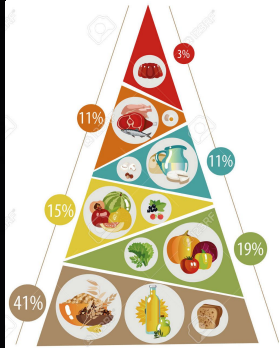
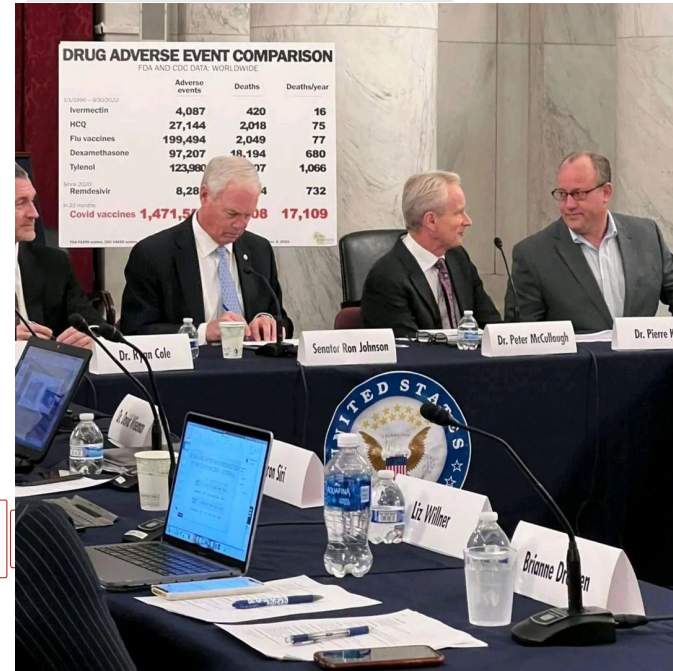
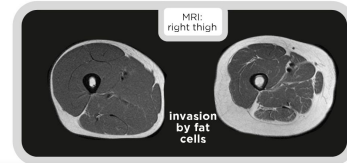
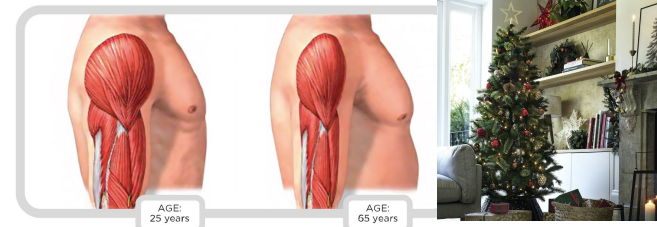
1. “The only benefits of fiber in the diet are in cases of diabetes mellitus and hyperlipidaemia[4], due to its anti-nutritive effect. Fiber is non-absorbable and therefore ingestion of large amounts leads to reduction in calorie intake. High natural fiber-diets rich in digestible fiber also produce gas, causing longer lasting satiety.”

However, fiber, can actually cause adverse effects on patients who should be instead following a low-FODMAPS diet (a diet that avoids fermentable foods to alleviate symptoms of IBS)

2. “Soluble fiber supplements should be considered when the desired effect is to delay gastric emptying and small bowel transit. The situations that thus will benefit from soluble fiber supplements are in cases of short gut syndrome or after a total colectomy”
3. Resistant starch is found in oats and cornflakes. They are resistant to α -amylase digestion. Modest (10 g/d) increases in resistant starch intake do not increase stool output suggesting that it may be completely fermented by colonic bacteria[4]. However, fermentation results in an increased production of colonic gas, leading to bloatedness and a distended abdomen.
4. One common but erroneous belief is that the moisture content of stool is increased when fiber intake is increased. The moisture content actually remains at 70% to 75% and does not change when more fiber is consumed. For most fiber substances, increase in quantity does not result in a more effective holding of water in the gut lumen[3,6].
5. A high fiber diet has also been shown to be associated with excessively long colons and a higher incidence of megacolon and volvulus[7] suggesting a negative effect of excessive fiber on colonic transit.
6. Fiber is fermented rapidly and may lead to a massive surge in microbial activity in the colon. Hydrogen, methane and carbon dioxide are then produced, causing cramps, bloatedness and distension[8].
7. The incidence of diverticulosis and complications of diverticular disease have been increasing in the West despite increase in dietary fiber intake[9]. This is probably related to the massive gaseous build up associated with a high dietary fiber intake.



Twelve Medical Myths You Believe Pediatrics to Adults #11



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#11. Myth: “Sore throat” requires antibiotics

Antibiotics for sore throat: Minimal benefit and massive
overuse **Diagnostic Methods, Clinical Guidelines, and Antibiotic
Treatment for Group A Streptococcal Pharyngitis: A Narrative Review**



Estimates of the rate of antibiotic prescription for American adults who seek treatment for pharyngitis vary: researchers have reported values of 75 (Neuner et al., 2003), 47 (Linder et al., 2006), 70 (Linder and Stafford, 2001), and 73% (Nakhoul and Hickner, 2013). A nationwide study also found that physicians prescribe antibiotics to children with sore throat 53% of the time (Linder et al., 2005). Overuse of antibiotics may be an even greater problem in low and middle-income countries. Analysis of physician practices at three hospitals in Egypt found that doctors prescribed antibiotics to 86% of patients with pharyngitis (Ahmed MH et al., 2015).

Symptom relief for GABHS is straightforward and can be achieved through the use of analgesic and antipyretic agents like acetaminophen (Shulman et al., 2012). **Appropriate antibiotics reduce the duration of illness by approximately one day** (Sheridan et al., 2007), with the greatest reduction in symptoms seen on the third day of treatment (Spinks et al., 2013). Improvement in symptoms may depend on the speed with which antibiotics are administered.

CYA medicine drives much of this overuse of Abx

A perspective prevalent in Europe is that GABHS is a self-limiting disease with low rates of complication. Therefore, antibiotics are unnecessary. Some physicians believe that antibiotics use should be restricted because their benefits are either nonexistent or modest ([Bisno, 2003](#); [Group et al., 2012](#)). Centor argues that empirical treatment of individuals with Centor scores of three or four is appropriate ([Centor, 2012](#)),

Rheumatic fever is the most common nonsuppurative complication of GABHS. However, the incidence of rheumatic fever is low in the United States and other high-income countries ([Snow et al., 2001](#); [Gerber et al., 2009](#)). Antibiotics may decrease the incidence of suppurative complications like peritonsillar abscesses ([Cooper et al., 2001](#); [Sheridan et al., 2007](#)). The benefits of antibiotics for preventing peritonsillar abscesses are limited when patients do not present until the complication has developed ([Cooper et al., 2001](#)). The rate of transmission for individuals infected with GABHS is approximately 35% ([Langlois and Andreae, 2011](#)). Antibiotics reduce the communicability of GABHS to 24 h, and aim to limit the spread of GABHS for high-risk patients ([Matthys et al., 2007](#)).

Urgent Cares who see patients as “clients” drive antibiotic overuse as well in my experience.

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Disagreements about whether and when to prescribe antibiotics for pharyngitis exist because there is no clearly superior management strategy. As such, it is not surprising that different treatment strategies are advocated by physicians and guidelines, even within the same medical institution (Singh et al., 2006). One opinion is that the question of the best method to diagnose and treat GABHS doesn't really matter. According to this view, all approaches besides empirical treatment have similar effectiveness and cost when the percentage of adults with pharyngitis who are GABHS-positive is approximately 10% (Neuner et al., 2003). Another reason for the lack of consensus on how best to diagnose and treat patients who may have GABHS are differences in how treatment goals are prioritized. Assumptions about the proportion of individuals with pharyngitis who are infected with GABHS also affect preferred management strategies (Singh et al., 2006).

Treatment for pharyngitis also depends on patient expectations (Cooper et al., 2001). Individuals with sore throat often have strong opinions about whether they should take an antibiotic (Neuner et al., 2003). Physicians may overestimate patients' desire for antibiotics, a misperception that contributes to excessive use of antibiotics (Kumar et al., 2003). What matters for many people in evaluating their treatment is whether their doctor seeks to understand their concerns (Cooper et al., 2001; Neuner et al., 2003). In many cases the relationship with a patient can be more effectively maintained by showing care than by writing a prescription for antibiotics (Kumar et al., 2003). Good communication skills are also important. Physicians need to be able to explain the benefits and risks of antibiotics to their patients in a clear manner (Butler and Francis, 2008; Tan et al., 2008). In sum, the quality of the doctor-patient relationship and interaction may be just as important as the particular diagnostic and treatment methods used for pharyngitis.

Corticosteroids for sore throat (might provide benefit however their use is not based on strong evidence)

[Corticosteroids for treatment of sore throat: systematic review and meta-analysis of randomised trials](#) (2017)

Results 10 eligible trials enrolled 1426 individuals. Patients who received single low dose corticosteroids (the most common intervention was oral dexamethasone with a maximum dose of 10 mg) were twice as likely to experience pain relief after 24 hours (relative risk 2.2, 95% confidence interval 1.2 to 4.3; risk difference 12.4%; moderate quality evidence) and 1.5 times more likely to have no pain at 48 hours (1.5, 1.3 to 1.8; risk difference 18.3%; high quality). The mean time to onset of pain relief in patients treated with corticosteroids was 4.8 hours earlier (95% confidence interval -1.9 to -7.8; moderate quality) and the mean time to complete resolution of pain was 11.1 hours earlier (-0.4 to -21.8; low quality) than in those treated with placebo. The absolute pain reduction at 24 hours (visual analogue scale 0-10) was greater in patients treated with corticosteroids (mean difference 1.3, 95% confidence interval 0.7 to 1.9; moderate quality). Nine of the 10 trials sought information regarding adverse events. Six studies reported no adverse effects, and three studies reported few adverse events, which were mostly complications related to disease, with a similar incidence in both groups.

Conclusion Single low dose corticosteroids can provide pain relief in patients with sore throat, with no increase in serious adverse effects. Included trials did not assess the potential risks of larger cumulative doses in patients with recurrent episodes of acute sore throat.

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(Published 20 September 2017)

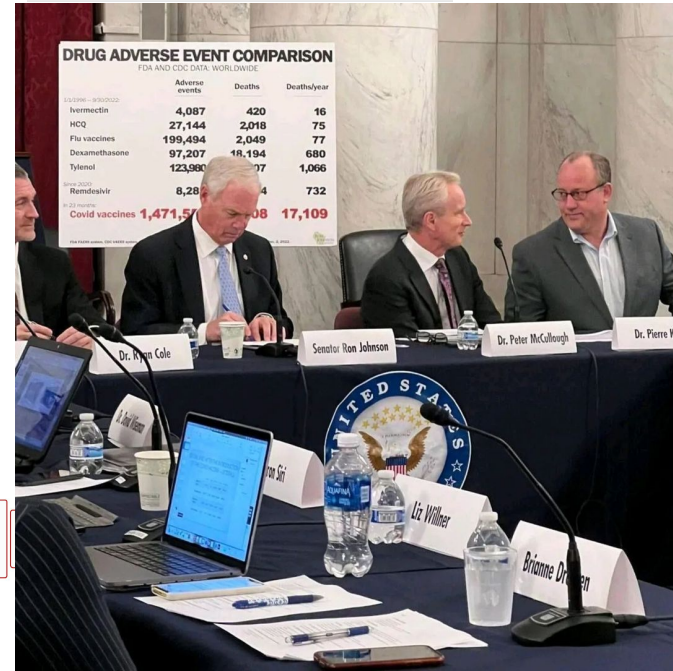
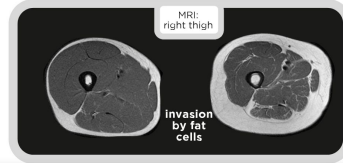
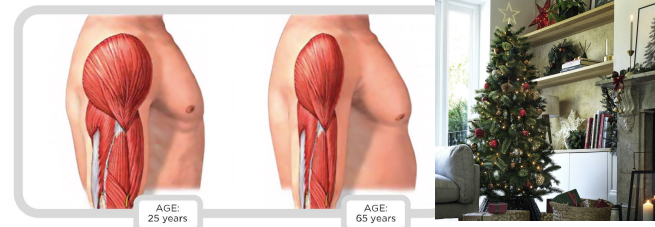
Cite this as: *BMJ* 2017;358:j3887

Corticosteroids as standalone or add-on treatment for sore throat (2020)

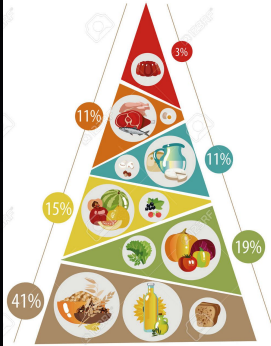
Oral or intramuscular corticosteroids, in addition to antibiotics, moderately increased the likelihood of both resolution and improvement of pain in participants with sore throat. Given the limited benefit, further research into the harms and benefits of short courses of steroids is needed to permit informed decision-making.



Twelve Medical Myths You Believe Pediatrics to Adults #12



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#12. Myth: “Tamiflu for influenza treatment”

The Tamiflu fiasco and lessons learned Gupta, Meenu, 2015)



Oseltamivir (Tamiflu), a neuraminidase inhibitor, was approved for seasonal flu by US Food and Drug Administration in 1999. The studies proving its efficacy “were funded by Roche, which also first marketed and promoted this drug. In 2005 and 2009, the looming fear of pandemic flu led to recommendation by prominent regulatory bodies such as **World Health Organization (WHO), Centers for Disease Control and Prevention...** etc for its use in treatment and prophylaxis of influenza, and it's stockpiling as a measure to tide over the crisis. Serious Adverse Events, especially neuropsychiatric events associated with Tamiflu started getting reported leading to a cascade of questions on clinical utility of this drug. The recommendations for stockpiling the said drug as **given by various international organizations viz WHO have also been put to scrutiny.**

Oseltamivir reduced the time to first alleviation of symptoms by 16.8 h (95% confidence interval 8.4–25.1 h $P < 0.001$) and by 29 h (95% confidence interval 12–47 h, $P < 0.001$) in healthy children.[23] The benefit of oseltamivir in asthmatic patients was insignificant. Pneumonia, hospitalization, and virus transmission were the major concerns with the disease for which benefits have been claimed in various studies. However, Cochrane review failed to establish any definitive benefit on these parameters.

“Approval by USFDA and EMA appears to be a judgment based medicine rather than evidence-based medicine. It appears that the regulatory authorities took their decision under the pressure of providing a pharmaceutical solution to a pandemic disease.”

Roche has benefitted with oseltamivir by more than 18 billion \$ since its launch in 1999. Its sales were increased by 84% by oseltamivir during US flu season in 2013. UK stockpiled the drug worth 710 million \$ for 40 million treatments. US spent 1.3 billion \$ on stockpiling oseltamivir for 65 million dosages.[39] India also increased its stock by 10 fold.[40]

Oseltamivir controversy "There is no evidence that oseltamivir reduces the likelihood of hospitalization, pneumonia or the combined outcome of pneumonia, otitis media and sinusitis." (Ebel, Call, 2013) Meta Analysis.

Methods.

We searched Medline without time or language restrictions, and trial registries maintained by the manufacturer. We included published and unpublished randomized double-blinded, placebo-controlled trials of oseltamivir in adults with suspected influenza that reported duration of symptoms, complications or hospitalizations. We abstracted data regarding study quality, the duration of symptoms and rates of complications and hospitalization.

Mark H Ebell, Marlene Call, JoAnna Shinholser

Family Practice, Volume 30, Issue 2, April 2013, Pages 125–133, <https://doi.org/10.1093/fampra/cms059>

Published: 21 September 2012

Results.

Three published and eight unpublished studies met our inclusion criteria. For the intention-to-treat (ITT) population, the mean reduction in the duration of symptoms was 20.7 hours [95% confidence interval (CI) 13.3 to 28.0 hours]. Two large unpublished studies in the elderly and in adults with chronic disease did not find a significant reduction in the symptom duration. There was no difference in the likelihood of hospitalization in the ITT population (33/2633 patients for oseltamivir versus 20/1694 for placebo). The rate of complications in the intention-to-treat infected (ITTI) population was reduced when acute bronchitis was included (-2.8%, 95% CI -0.6 to -4.9), but not when it was excluded. The risk of pneumonia was reduced in the ITTI population (-0.9%, 95% CI -0.1 to -1.7) but not in the ITT population.