FOOD CHOICE & VISION LOSS: IS AGE-RELATED MACULAR DEGENERATION (AMD) PREVENTABLE?

Chris A. Knobbe, MD

Associate Clinical Professor of Ophthalmology University of Texas Southwestern Medical Center – Dallas, Texas, USA

John R Cripps BSc., (biochemistry/physiology), MD FRCSC (Emeritus); Assistant Clinical Professor of Surgery, Northern Ontario School of Medicine (NOSM; retired) Certified American Board of Ophthalmology

Ballantrae Community Zoom presentation 7 Jan 2021

I wish to be very clear from the beginning of this presentation. My "**light-bulb moment**", regarding the etiology of age-related macular degeneration, or AMD, came about early in 2019 after reading the 1939 work of Weston Price – "**Nutrition and Physical Degeneration**". Subsequent discoveries led me to a book by ophthalmologist Chris Knobbe – "**Ancestral Dietary Strategy to Prevent and Treat Macular Degeneration** published in 2016". Because of my interest in nutritional biochemistry, I quickly went down the **diet** AMD "rabbit hole" following several conversations with Chris. If any ideas seem valuable, or slides are of interest during this talk, they are 100% attributed to Chris Knobbe's research over the previous 9 years. Like a Gary Taubes, Nina Teicholz or Tim Noakes the depth of research has convinced me of the validity of his hypothesis and the value to preventing blindness in millions of people worldwide. I declare NO CONFLICT OF INTEREST with the exception that I have followed a healthy lifestyle to improve my insulin resistance for > 2 years. (*LCHF*)

Today, ladies and gentlemen, you're going to hear a profound and revolutionary hypothesis, that age-related macular degeneration is not only entirely preventable, but may be reversible in the early stages, with diet. With an "Ancestral Diet".

Age-related macular degeneration, or AMD, is the leading cause of IRREVERSIBLE vision loss and blindness in developed nations over age 50.

To be clear, we're talking about IRREVERSIBLE VISION LOSS, here.



I am sure someone participating today is personally affected, or knows of a family member, friend, or colleague afflicted with AMD -- but are you aware of the lifetime risk of developing macular degeneration in the U.S. today? I mention the US data because it is the most extensive however best estimates reveal similar disease prevalence to Canada.



Nearly <u>1 of every 3 adults over the age of 75 currently affected with AMD (since 1992).</u>

Globally, in 2014, one of every 11 adults (8.69%) over age 50 has some degree of AMD

R Klein et al., Prevalence of Age-Related Maculopathy. **The Beaver Dam Eye Study** Ophthalmology. **1992**; 99: 933-943. Population study in **Wisconsin**, USA (**1/3>75 years**)

Mbiostat et al. 2014 –Global prevalence of AMD 8.69%



Like other ophthalmologists, medical colleagues, and optometrists, I was taught that AMD was "multifactorial" and caused mainly by genetics and aging.

Therefore, I would predict a similar burden of disease in 1900 compared to today. So, what was the AMD risk at that time in our history?



AMD Lifetime risk in US year 1900: Approximately Zero – to One in 1000's.



What is the prevalence of AMD today?

The number of Americans affected today is well in excess of 22 million.

In the year 2002, WHO data revealed > 2million blind with AMD

WHO World report on vision 2019 estimates: There were196 million people with agerelated macular degeneration in 2020 with 288 million projected by 2040

Of those 196 million AMD patients in 2020 best estimates suggest

~ 10.4 million with moderate-severe vision impairment/blindness that could have been prevented or has not yet been addressed

https://www.who.int/publications/i/item/world-report-on-vision: 195.6 million (95% CI 140–261) people aged 30 to 97 years with age-related macular degeneration in 2020



This graphic from the **WHO "World report on vision 2019" puts AMD into perspective for causes of visual loss**

Note that a large percentage of visual loss causes are reversible – for example by prescribing glasses – something we call correcting refractive errors. In contrast eye diseases like macular degeneration, diabetic retinopathy and glaucoma need to be picked up in the early stages to prevent permanent visual loss



WHO - **"World report on vision 2019"** – estimates at least ONE BILLION people with vision loss that <u>could have been prevented</u> or <u>has yet to be addressed</u>

The actual proportion of age-related macular degeneration that could have been prevented is unknown – it was estimated at 10.4 million .

Therefore, **AMD** is the 3rd leading cause of unaddressed vision loss – again notice that by correcting "refractive errors", with glasses, or removing cataracts – correction of vision impairment is possible



Here are the consequences of NOT addressing AMD

This means that, rather than having vision like that on the left, these people have vision like that on the right.

Huge central blind spots -- these people can't see to read, to drive, they can't see their children or grandchildren's faces, because of AMD.

Thank goodness we know the cause; or do we?



Now, because I'm an ophthalmologist my specialty has made some version of this statement, for many decades; "The etiology of age-related macular degeneration (AMD) is unknown." "Advanced age, northern European ancestry and genetics are risk factors."

I reviewed the latest 2019 wisdom from my own professional association in Canada – "The Canadian Ophthalmological Society" (COS), regarding AMD etiology - COS 2019 "Although the specific cause is unknown, AMD seems to be part of aging."

WE don't know what causes it. So, if we don't know what causes it, we CERTAINLY don't know how to prevent it.

And we really don't have much in the way of treatment -- but I will come back to that.



This is the reason your ophthalmologist, optometrist, or health care provider might believe AMD is a "natural part of aging" – who could blame them.

Overall, in 2020, worldwide AMD prevalence is estimated to increase from 4.2% in those aged 45-49 years, to 27.1% in those aged 80-85 years – that's a 6.5-fold increase

Observation: one day on a Sunday afternoon drive my wife made the following observation; most older males driving convertible sports cars are bald and have beer bellies (association/correlation)

Do sports cars cause baldness and beer bellies? CAUSATION

Observation: Most people die in bed (association/ correlation)

Are beds killing people? CAUSATION

Observation: Macular degeneration is more prevalent with age (association/correlation)

Is aging really the ROOT CAUSE of macular degeneration? CAUSATION

Let's briefly pause to review causation versus association (correlation) By reading the slide one can gain a better understanding of the difference between ASSOCIATION (correlation) and CAUSATION

Let's see if there is a reasonable explanation other than "multifactorial" or " aging and genetics" which clearly doesn't address the ROOT cause. We generally use terms in medicine, like multifactorial and idiopathic, when we don't understand the etiology.



The hypothesis Dr. Chris Knobbe has proposed, which I would like for you to consider, is this; The 'Displacing Foods of Modern Commerce' Are the Primary and Proximate Cause of AMD.

If you're not familiar with the term 'Displacing Foods of Modern Commerce,' just think of that as man-made, processed foods for now -- and we'll come back to that.

The reason for presenting this hypothesis for your consideration, is because 196 + million people are losing vision to this condition, and, like Chris Knobbe, I believe this is preventable.

Because many of you are interested in nutritional biochemistry, and overall good health, I want you to evaluate this hypothesis today.



So, I am going to show you what macular degeneration looks like And then I am going to walk you through three simple, interrelated stories, and you're going to see that macular degeneration **follows processed foods**, WHEREVER THEY GO.

So, here's the macula -- the central retina.

It is 6 mm across -- and accounts for the central 21 degrees of vision.

This is a healthy macula -- consistent with good vision – think 6/6 or 20/20 or better visual acuity

The retina, hands down, is the most metabolically active tissue in the human body

The retina, 10 layers thick, is akin to 9 layers of carpet lying on top of carpet backing; the extremely metabolically active cone cells are constantly shedding their outer segments and being renewed by the carpet backing; (the RPE – or retinal pigment epithelium). If the RPE gets overwhelmed, it says "**No Problem" it deposits** debris it can't handle under the carpet backing". We call that DRUSEN.

Standardised grading classifications with retinal photography:

- A. Wisconsin age-related maculopathy grading system
- B. International classification for age-related macular degeneration
- C. Rotterdam staging system



As drusen collect underneath the "Carpet backing" ,or RPE, the overlying retina begins to suffer the consequences of hypoxia (or lack of oxygen), and inflammation.

Classification Systems for AMD

Various classification systems for AMD lesions have been published during the past 30 years. All systems are based on color fundus photographs. In the early 1990s, the Wisconsin age-related maculopathy grading system set the tone for the diagnostic criteria of individual AMD lesions in a macular grid with subfields and standard circles to assess size and area. This was followed by an international consensus on AMD lesions by the International Age-Related Maculopathy Epidemiological Study Group. As these systems lacked a severity scale, a classification system addressing this was established by the

Rotterdam Study Group in 2001. The AREDS Study proposed multiple classification systems, of which the first system was published in 2001, classifying referable patients for AREDS supplements. Several years later, two systems were added by this study group for clinical and research purposes (i.e., a simplified scale and a 9-step severity scale, respectively).

A clinical classification was published by the Beckman initiative group in 2013.

Three Continent AMD Consortium (3CC)

The 3CC study in 2014, harmonized grading of the Rotterdam Study (Netherlands), the Beaver Dam Eye Study (Wisconsin, USA), the Los Angeles Latino Eye study, and the Blue Mountain Eye Study (Australia).

For screening and referrals, systems should be quick and easy to interpret. The AREDS simplified scale seems appropriate in this regard with limited and easily identifiable features (i.e., presence of large drusen and pigment changes). The Beckman classification is similar, but also classifies drusen according to size in small, intermediate, and large drusen, which may still be feasible for screening. This system, however, did not reveal high progression rates in any of the categories. For intervention studies focusing on early and intermediate AMD, systems should include categories with a high turnover rate to late AMD. Both the AREDS simplified scale and the Rotterdam Study had such categories.



Rapid central visual deterioration may ensue in the following scenario: abnormal new blood vessels (so-called neovascularization), may grow under or within the retina. LEAKAGE of red blood cells (hemorrhage), and PLASMA (containing lipids, proteins and other constituents) collect beneath the macula – the so-called "WET" form of AMD. This destroys the overlying cone cells responsible for your central vision. Although the DRY form of AMD accounts for approximately 90% of overall cases, WET AMD accounts for 90+% of blindness

HOW ABOUT TREATMENT FOR DRY AMD



I advised my patients to exercise, avoid smoking, eat dark leafy greens and consider eating oily fish, rich in ω -3 fatty acid 2X/week.

"The Fishy evidence" - based on weak observational studies; some suggest slowing AMD progression – not prevention of disease

I was less convinced regarding synthetic vitamins. I educated patients about AREDS supplements because it was "**standard of care**," — <u>my medical license could have been</u> <u>revoked if most of my colleagues prescribed these vitamins and I didn't</u> –NOTHING TO DO WITH SCIENCE WHATSOEVER

I also felt obliged to inform my patients about the 2012 Cochrane review on multivitamins; more on that in a minute

Fat Consumption and Its Association With Age-Related Macular Degeneration

<u>Elaine W.-T. Chong, MD, PhD, MEpi; Luibov D.</u> <u>Robman, PhD; Julie A. Simpson, PhD; et alAllison M.</u> <u>Hodge, PhD; Khin Zaw Aung, MD; Theresa K.</u> <u>Dolphin, BAppSci; Dallas R. English, PhD; Graham</u> <u>G. Giles, PhD; Robyn H. Guymer, MD, PhD</u>

Author Affiliations <u>Article Information</u> Arch Ophthalmol. 2009;127(5):674-680. doi:10.1001/archophthalmol.2009.60

Higher ω-3 fatty acid intake (highest quartile vs lowest quartile) was **inversely associated with early AMD** (odds ratio, 0.85; 95% confidence interval, 0.71-1.02; P = .03).

Late AMD - Chong et al., reviewed nine studies investigating the association of food, fish and supplement sourced omega-3 fatty acids with the progression of AMD. The pooled odds ratio (OR) from Chong et al. for **late AMD progression** with high omega-3 fatty acids intake compared with low intake was 0.62 (95% CI: 0.48–0.82; P < 0.001). The pooled OR for late AMD progression with high fish intake compared with low fish *intake was 0.67* (95% CI: 0.53–0.85; P < 0.001). Both these pooled ORs for omega-3 fatty acids intake and fish intake are in the range of the separate ORs reported by the studies included in this review, which ranged from 0.48 to 0.85.

Summary –NOT for primary prevention; > omega-3 intake = milder AMD; Greater omega-3 = less progression from intermediate to advanced AMD



In order to determine this let's examine the least biased database of systematic reviews; The Cochrane Collaboration. To summarize the slide there is no evidence to support taking vitamins or antioxidants to PREVENT AMD.

Antioxidant vitamin and mineral supplements for preventing age-related macular degeneration (Review) Evans JR, Lawrenson JG

Evans JR, Lawrenson JG. Antioxidant vitamin and mineral supplements for preventing age-related macular degeneration. **Cochrane Database of Systematic Reviews 2017, Issue 7.** Art. No.: CD000253. DOI: 10.1002/14651858.CD000253.pub4. www.cochranelibrary.com Antioxidant vitamin and mineral supplements for preventing age-related macular degeneration (Review) Copyright © 2017 The Cochrane Collaboration. Published by John Wiley & Sons, Ltd.

Authors' conclusions Taking vitamin E or beta-carotene supplements will not prevent or delay the onset of AMD. The same probably applies to vitamin C and the multivitamin (Centrum Silver) investigated in the one trial reported to date. There is no evidence with respect to other antioxidant supplements, such as lutein and zeaxanthin. Although generally regarded as safe, vitamin supplements may have harmful effects, and clear evidence of benefit is needed before they can be recommended. People with AMD should see the related Cochrane Review on antioxidant vitamin and mineral supplements for slowing the progression of AMD, written by the same review team. Implications for practice Taking vitamin E or beta-carotene supplements will not prevent or delay the onset of AMD. The same probably applies to vitamin C and the multivitamin (Centrum Silver) investigated in the one trial reported to date. There is no evidence with respect to other antioxidant supplements, such as lutein and zeaxanthin.

Do synthetic vitamins prevent or slow progression of existing AMD?

Cochrane Database of Systematic Reviews 2017¹ <u>19 studies</u> conducted in USA, Europe, China, and Australia

"People with AMD may experience some delay in progression of the disease with multivitamin antioxidant vitamin and mineral supplementation. This finding was largely drawn from one large trial, conducted in a relatively well-nourished American population. We do not know the generalizability of these findings to other populations. Although generally regarded as safe, vitamin supplements may have harmful effects. A systematic review of the evidence on harms of vitamin supplements is needed. Supplements containing lutein and zeaxanthin are heavily marketed for people with age-related macular degeneration, but our review shows they may have little or no effect on the progression of AMD".

PMID: 28756618

Do synthetic vitamins prevent or slow progression of existing AMD?

Let's again look at the gold standard - Randomized controlled clinical Trials and the Cochrane Collaboration Database. Unlike "observational trials" – e.g., where we <u>observed</u> that most people die in bed - randomized controlled clinical trials, so-called experimental trials, randomize patients to receive a treatment (drug or procedure) while the control group gets a placebo (no active drug or a SHAM procedure)

What did the authors find?

18 out of 19 studies showed NO benefit; a single US study showed some benefit in the selected population; don't waste your money on Lutein/Zeaxanthin supplements; **vitamin supplements may have harmful effects.**

Evans JR, Lawrenson JG. Antioxidant vitamin and mineral supplements for slowing the

progression of age-related macular degeneration. Cochrane Database of Systematic Reviews 2017, Issue 7. Art. No.: CD000254. DOI: 10.1002/14651858.CD000254.pub4.19 studies conducted in USA, Europe, China, and Australia



AREDS -- The only trial showing benefits to people with MODERATE AMD slowing progression (18 other trials showed NO benefit)

The AREDS trial - A Randomized, Placebo-Controlled, Clinical Trial of High-Dose Supplementation With Vitamins C and E, Beta Carotene, and Zinc for Age-Related Macular Degeneration and Vision Loss AREDS Report No. 8. Arch Ophthalmol. 2001 Oct; 119(10): 1417–1436. (Recruitment Nov 1992-Jan 1998) The AREDS TRIAL spun off some 35 papers with massive funding from the NIH (National Institute of Health) – you could not disagree or challenge the authors- it was the STATIN "drug" of ophthalmology HOW GOOD WAS IT-the authors reported a 25% reduction in progression from moderate to advanced AMD –GREAT –not so fast. This was the <u>relative risk reduction</u>

Over 5 years, patients taking the antioxidant and zinc supplement had a 20% chance of developing vision loss from advanced AMD compared to a 28% chance of developing vision loss from advanced AMD for patients taking a placebo pill. Therefore, the absolute risk reduction was only 8% over 5 years; or 1.6%/year.

The original AREDS (Age-Related Eye Disease Study), was a large, multicenter trial of patients with established AMD to evaluate the effect of a combination of 3 antioxidants (vitamin C, 500 mg; vitamin E, 400 IU; and vitamin A in the form of beta carotene, 25,000 IU), with zinc and copper. **Over 5 years, patients taking the antioxidant and zinc supplement had a 20% chance of developing vision loss from advanced AMD compared to a 28% chance of developing vision loss from advanced AMD for patients taking a placebo pill. This effect was statistically significant, but modest. Concerns were raised about the high dose of vitamin A, since beta carotene was known to cause harm at those levels, so another study was designed to evaluate a different combination that omitted the vitamin A and added other possibly beneficial components.**

The AREDS 2 trial - Mary E. Aronow, MD and Emily Y. Chew, MD. AREDS2: Perspectives, Recommendations, and Unanswered Questions. Curr Opin Ophthalmol. 2014 May; 25(3): 186–190.

In the new trial, there were four groups: (1) a control group got the original AREDS formula, and the other 3 got a formula that omitted the vitamin A and added (2) lutein and zeaxanthin, (3) the omega 3s DHA and EPA, and (4) both lutein/zeaxanthin and DHA/EPA.

There was no control group of patients not taking any supplement.

They concluded:

Addition of lutein + zeaxanthin, DHA + EPA, or both to the AREDS formulation in primary analyses did not further reduce risk of progression to advanced AMD. However, because of potential increased incidence of lung cancer in former smokers, lutein + zeaxanthin could be an appropriate carotenoid substitute in the AREDS formulation.

AREDS 2 had several strengths. It was large (4,203 subjects), it lasted 5 years during which 1940 eyes progressed to advanced AMD, the drop-out rate was low, it assessed adherence, and it measured blood levels of the study nutrients.

It also had some weaknesses. <u>They didn't use a no-supplement control group because they</u> <u>assumed that the original AREDS had proved the benefit of supplementation. This is a bit</u> <u>worrisome, since we know it is risky to rely on a single study</u>. The AREDS trial has not been replicated, and a **Cochrane systematic review** concluded:

People with AMD may experience delay in progression of the disease with antioxidant vitamin and mineral supplementation. This finding is drawn from One large trial conducted *in a relatively well-nourished American population*. The generalisability of these findings to other populations is not known. Although generally regarded as safe, vitamin supplements may have harmful effects. A systematic review of the evidence on harms of vitamin supplements is needed. –WELL Nourished American population???? In 2017 88% of the US population was "metabolically unhealthy."



Simple but important statistics: The **number needed to treat** is 100/absolute risk reduction=12.5(~13)

In the <u>AREDS trial</u>, only 8% of people, who took the AREDS supplement for 5 years, had a benefit. (<u>Absolute risk reduction 8%</u>) or **1.6%/year realized any benefit**

<u>Antioxidants (Vitamins E, C & beta-carotene 23%-ARR 5%); Zinc (22%)-ARR 6%.</u> That is, 20% of subjects progressed in the AREDS treatment group, 28% progressed in the control group. **Terrible progression in both groups**

This indicates that the **Number Needed to Treat (NNT) is 13,** that is, **13 people** (12.5%, actually) **must be treated with AREDS formula for 5 years, for 1 to benefit**.

Carl Awh's research found that at least 13%, and perhaps as many as ~30%, were worse, as a result of taking AREDS formula although this finding has been disputed by statisticians from the AREDS study group.

- The <u>original AREDS</u> (Age-Related Eye Disease Study), was a large, multicenter trial of patients with established AMD to evaluate the effect of a combination of 3 antioxidants (vitamin C, 500 mg; vitamin E, 400 IU; and vitamin A in the form of beta carotene, 25,000 IU), with 80mg zinc and 2mg of copper. While <u>a dose of 80 mg zinc was used in the original AREDS formulation</u> based on a prior trial suggesting efficacy, <u>there was also evidence to suggest that the maximal level absorbed was closer to 25 mg</u> Newsome DA, Swartz M, Leone NC, Elston RC, Miller E. Oral zinc in macular degeneration. Archives of ophthalmology. 1988;106(2):192–8. Epub 1988/02/01. [PubMed] [Google Scholar]; Hambidge M. Underwood Memorial Lecture: human zinc homeostasis: good but not perfect. The Journal of nutrition. 2003;133(5 Suppl 1):14385–42S. Epub 2003/05/06. [PubMed] [Google Scholar] In the AREDS1 trial, <u>7.5% of patients consuming the synthetic supplement had an increased risk of hospital admission for bladder and kidney complications from the high doses of zinc.</u>
- 2. The AREDS 2 trial

In the new trial, there were four groups: (1) a control group got the original AREDS formula, and the other 3 got a formula **that omitted the vitamin A and added** (2) <u>lutein and</u> <u>zeaxanthin</u>, (3) the <u>omega 3s DHA and EPA</u>, and (4) <u>both lutein/zeaxanthin and DHA/EPA</u>. There was no control group of patients not taking any supplement. They concluded:

- 1. Addition of lutein + zeaxanthin, DHA + EPA, or both to the AREDS formulation in primary analyses did not further reduce risk of progression to advanced AMD. However, because of potential increased incidence of lung cancer in former smokers, lutein + zeaxanthin could be an appropriate carotenoid substitute in the AREDS formulation.
- 2. AREDS 2 had several strengths. It was large (4,203 subjects), it lasted 5 years during which 1940 eyes progressed to advanced AMD, the drop-out rate was low, it assessed adherence, and it measured blood levels of the study nutrients.
- 3. It also had some weaknesses. They didn't use a no-supplement control group because they assumed that the original AREDS had proved the benefit of supplementation. This is a bit worrisome, since we know it is risky to rely on a single study. The AREDS trial has not been replicated, and a <u>Cochrane systematic review concluded</u>: People with AMD may experience delay in progression of the disease with antioxidant vitamin and mineral supplementation. This finding is drawn from one large trial conducted in a relatively well-nourished (?) American population. The generalisability of these findings to other populations is not known. Although generally regarded as safe, vitamin supplements may have harmful effects. A systematic review of the evidence on harms of vitamin supplements is needed.

	AGREE II Domains (%)						
Clinical Guideline	Scope and Purpose	Stakeholder Involvement	Rigour of Development	Clarity of Presentation	Applicability	Editorial Independence	
American Optical Association 2004	52.8	30.6	9.2	30.6	10.9	87.5	
International Council of Ophthalmology 2007	58.3	30.6	10.2	58.3	2.2	0.0	
Spanish Retina and Vitreous Society 2009	63.9	41.7	21.4	36.1	13.0	0.0	
Canadian Expert Consensus 2012	75.0	47.2	36.7	75.0	28.3	20.8	
German Ophthalmological Society 2014	30.6	25.0	19.4	52.8	0.0	0.0	
Eye Health Council of Ontario (Canada) 2015	61.1	22.2	14.3	41.7	13.0	20.8	
National Health Committee (New Zealand) 2015	75.0	13.9	20.4	55.6	30.4	0.0	
American Academy of Ophthalmology 2015	83.3	63.9	72.4	94.4	17.4	75.0	
Vitreo-Retina Society of the Philippines 2016	83.3	44.4	56.1	88.9	13.0	45.8	
NICE Clinical Knowledge Summary (CKS) 2016	88.9	83.3	68.4	91.7	39.1	87.5	
Clinical Advisory Committee (United States) 2017	52.8	22.2	9.2	44.4	4.3	0.0	
NICE Guideline (NG82) 2018	94.4	88.9	95.9	100.0	97.8	100.0	
Optometry Australia 2019	83.3	41.7	19.4	75.0	19.6	0.0	
Median (range)	75.0 (range 30.6 to 94.4%)	41.7 (range 13.9 to 88.9%)	20.4 (range 9.2 to 95.9)	58.3 (range 30.6 to 100%)	13.0 (range 2.2 to 97.8%)	20.8 (range 0 to 100%)	

In 2019, Lawrenson et al. published a study in the journal Nutrients. They identified Thirteen (13) National and International AMD clinical guidelines on diet and/or nutritional supplementation; QUALITY of evidence was evaluated using Appraisal of Guidelines for Research and Evaluation II (AGREE II) instrument, a standard research tool. Basically, lower scores are indicative of lack of applicability, bias, competing interests, poor use of scientific up-to-date systematic reviews, poorly written and target the wrong populations. '. Only four guidelines used evidence from systematic reviews to support their nutritional recommendations. The best evidence supported guidelines, with the least bias, were the National Institute for Health and Care Excellence (NICE) – UK Healthcare – like universal healthcare in Canada

So, what did these guidelines tell us?

A Critical Appraisal of National and International Clinical Practice Guidelines Reporting Nutritional Recommendations for Age-Related Macular Degeneration: Are Recommendations Evidence-Based? John G. Lawrenson, Jennifer R. Evans and Laura E. Downie Nutrients 2019, 11, 823

The aim of this study was to identify clinical guidelines with recommendations pertaining to dietary modification and/or nutritional supplementation for age-related macular degeneration (AMD), and to evaluate the overall quality of the guidelines using the <u>Appraisal</u> of <u>Guidelines for Research and Evaluation II (AGREE II)</u> instrument. We also mapped recommendations to existing systematic review evidence

We identified 13 national and international guidelines, developed or updated between 2004 and 2019. These varied substantially in quality. The lowest scoring AGREE II domains were for 'Rigour of Development', 'Applicability' (which measures implementation strategies to improve uptake of recommendations), and 'Editorial Independence'. <u>Only four guidelines</u> <u>used evidence from systematic reviews to support their nutritional recommendations</u>. In conclusion, there is significant scope for improving current Clinical Practice Guidelines for AMD, and <u>guideline developers should use evidence from existing high quality systematic</u> <u>reviews to inform clinical recommendations</u>.

Clinical Guideline	Dietary Advice	Use of Antioxidant or Mineral Supplements	Use of Omega-3 Fatty Acid Supplements	Contraindications or Side Effects of Supplements	Systematic Review Cited with Recommendation	
American Optical Association 2004	NR	√ 4	NR	~	None	
International Council of Ophthalmology 2007	NR	NR	NR	N/A	N/A	
Spanish Retina and Vitreous Society 2009	NR	NR	NR	N/A	N/A	
Canadian Expert Consensus 2012	NR	NR	NR	N/A	N/A	
German Ophthalmological Society 2014	✓ 1	√ 5	NR	N/A	Yes	
Eye Health Council of Ontario (Canada) 2015	✓ ²	√ ⁵	NR	N/A	None	
National Health Committee (New Zealand) 2015	NR	NR	NR	N/A	N/A	
American Academy of Ophthalmology 2015	NR	√ 5	NR	N/A	None	
Vitreo-Retinal Society of the Philippines 2016	NR	√ 5	NR	N/A	None	
NICE Clinical Knowledge Summary (CKS) 2016	NR	√ 5	NR	N/A	Yes	
Clinical Advisory Committee (United States) 2017	NR	10	NR	NR	None	
NICE Guideline (NG82) 2018	NR	NR	NR	N/A	N/A	
Optometry Australia 2019	√ 3	√7	NR	NR	None	
Abbreviations: \checkmark , recommendation include; INI high in green, leafy vegetables (rich in antioxida supplements (particularly tor nutritionally detici xanthophylls (lutein, zeaxanthin, meso-zeaxanthi	CE, National Institute ints and carotenoids) ient); [*] Evidence of n n) for 'sub-clinical' A	 for Health and Care Exceller ³ A diet high in macular ca benefit for antioxidant vitar MD. ⁷ Supplements not curre 	ce; NR, no recommendation. rotenoids (zeaxanthin and lu nin and/or mineral supplement ttly recommended for people	Guideline recommendation tein) and omega-3 fatty aci- ents for primary prevention with normal ageing change	s: ¹ Balanced diet; ² A diet ds; ⁴ Antioxidant nutrient ⁹ Supplement containing s.	

For primary prevention, most of the better guidelines, underlined in purple, agreed on EVIDENCE of **NO BENEFIT for antioxidant vitamin and/or mineral supplement**

SO, if you don't have macular degeneration taking a supplement won't prevent you from getting this disease

National and International Clinical Practice Nutritional Guidelines on AMD 30979051 Table 4. Nutritional recommendations for secondary prevention (i.e., slowing the progression) of age-related macular degeneration (AMD). Contraindications or Use of Antioxidant or Use of Omega-3 Fatty Systematic Review Cited **Clinical Guideline Dietary** Advice Side Effects of with a Recommendation **Mineral Supplements** Acid Supplements Supplements American Optical Association 2004 NR NR None ✓ 7a None International Council of Ophthalmology 2007 NR NR NR V1 √ 7a Spanish Retina and Vitreous Society 2009 NR 1 None √ 7a Canadian Expert Consensus 2012 12 NR 1 Yes √ 7a German Ophthalmological Society 2014 NR × 10 ~ Yes ✓ 7a 13 ✓ 10 Eye Health Council of Ontario (Canada) 2015 1 None ✓ 7a National Health Committee (New Zealand) 2015 NR NR NR None √ 7a NR American Academy of Ophthalmology 2015 NR ~ None √ 7a Vitreo-Retina Society of the Philippines 2016 NR NR None 13 √7a NICE Clinical Knowledge Summary (CKS) 2016 NR 1 Yes √ 7a Clinical Advisory Committee (United States) 2017 14 19 NR None ✓ 10 NICE Guideline (NG82) 2018 NR Yes √ 5 √ 7b Optometry Australia 2019 NR NR None Abbreviations: \checkmark , recommendation included; NICE, National Institute for Health and Care Excellence; NR, no recommendation. Guideline recommendations: ¹ Diet rich in carotenoids (lutein and zeaxanthin) and omega-3 fatty acids; ² Dietary intake of antioxidants, docosahexaenoic acid, and omega-3 fatty acids; ³ Diet high in fresh fruit and green, leafy vegetables (rich in antioxidants and carotenoids): ⁴ Consume only fish rich in DHA and follow healthire reating styles e.g., Mediterranean diet; ⁵ Å diet rich in green leafy vegetables, fish and antioxidants should be encouraged; ⁴ Antioxidant nutrient supplements; ²⁶ Age-Related Eye Disease Studies (AREDS) or AREDS2 supplement from the patient's general medical practitioner. ⁸ No evidence that benefit of AREDS supplement outweights the risk; ⁹ Omega-3 fatty acid supplements. No evidence that AREDS supplement outweighs the risk

What about secondary prevention? Recall that only 1 study (AREDS) in the US showed any benefit. 18 other studies failed to demonstrate beneficial results with supplements. Because of the influence of the American Academy of Ophthalmology AREDS and AREDS2 supplements are recommended by the professional organization practice guidelines shown in yellow

Note that the least biased and best guidelines, based on systematic reviews, underlined in red, is the UK NICE (National Institute for Health and Care Excellence). Because AREDS was the only trial to show limited benefit to vitamin and antioxidant/mineral treatment what did the latest 2018 NICE (*NG82*) guidance advise clinicians treating AMD patients? No evidence that AREDS supplement outweighs the risk

PMID:

20	12 Cochrane review - Antioxidant supplements for prevention of mortality in healthy participants and patients
wit Sev	ch various diseases ¹ Bjelakovic et al /enty-eight randomised trials with 296,707 participants
"W and be	e found no evidence to support antioxidant supplements for primary or secondary prevention. Beta-carotene d vitamin seem to increase mortality, and so may higher doses of vitamin A. Antioxidant supplements need to considered as medicinal products and should undergo sufficient evaluation before marketing."
Re	garding all-cause mortality you are worse off taking a multivitamin
Edi mi	torial: Ann Internal Med. 2013 Dec 17; 159(12):850-851. Enough is enough: stop wasting money on vitamin an neral supplements ²
	tudies find supplements do not help extend life or ward off heart disease and memory loss. "The probability of
3 s a n	neaningful effect is so small it's not worth doing study after study and spending research dollars on these
<mark>3 s</mark> a n qu	neaningful effect is so small it's not worth doing study after study and spending research dollars on these estions."
<mark>3 s</mark> a n qu	neaningful effect is so small it's not worth doing study after study and spending research dollars on these estions." PMID: 22419320

Eating nutrient-dense whole foods is best. Some people believe they can compensate for a nutrient deficient toxic diet with "supplements" – what is the best evidence for this. Let's once again look at the Cochrane Collaboration of systematic reviews:

"We found no evidence to support antioxidant supplements for primary or secondary prevention. Beta-carotene and vitamin seem to increase mortality, and so may higher doses of vitamin A. Antioxidant supplements need to be considered as medicinal products and should undergo sufficient evaluation before marketing." **In Summary, regarding all-cause mortality you are worse off taking a multivitamin.**


Medical Hypothesis (Chris Knobbe M.D.) "The 'Displacing Foods of Modern Commerce' Are the Primary and Proximate Cause of AMD"

So, Where did the term 'Displacing Foods of Modern Commerce' come from?



Weston A. Price. For those of you who don't know him, Weston A. Price was a highly accomplished scientist, researcher, and dentist, who -- in the 1930s -- spent a decade traveling the world, evaluating people on five different continents, 14 nations, 14 major populations, but hundreds of tribes and cultures, and thousands upon thousands of people, in attempt to determine what it was about diets that kept people's teeth healthy. **Many have referred to Weston Price as the "Isaac Newton of Nutrition**". Weston was born on a farm just east of Oshawa Ontario and emigrated to Michigan to begin his illustrative dental and science career.

And while dental health was Price's primary concern, his secondary area of interest was general physical and mental health and physical degeneration. Travelling the world was a necessity to find cultures, who were healthy, whilst eating their ancestral diets, because

dental decay and general health was rapidly deteriorating in the United States amid a population consuming nutrient deficient diets. **Truly genius reverse engineering thinking in nutrition research at the time.**

Price found that, no matter where he went — people who continued to eat their traditional ancestral diets were extraordinarily healthy.



People looked like this, with healthy, beautiful, straight teeth — about 99.7% cavity free — and they enjoyed immunity to degenerative diseases, like arthritis, cancer, heart disease, and infectious diseases, like Tuberculosis (TB), **if** they continued to consume their native traditional diets.

But when they began to consume what Price called 'The Displacing Foods of Modern Commerce', dental, physical and emotional health rapidly deteriorated.



The 'Displacing Foods of Modern Commerce,' which Price defined as white flour, sugar, canned goods, sweets, confectionary, and VEGETABLE OILS.



Consumption of 'Displacing Foods of Modern Commerce' instead of ancestral diets leads to:

Dental decay, as noted in these two Australian Aboriginal women on the left; followed by degenerative diseases -- like arthritis, cancer, and loss of immunity to infectious diseases.

The young lady on the far right was also an Australian Aboriginal woman, however, she had continued to consume her native, traditional diet enjoying excellent health

This was the scenario that played out in Price's research thousands upon thousands of times.

"Primitive Diets"

- Dozens... hundreds of diets?
- Macronutrient ratios didn't matter
- Nutrient-density critical
- All contained "Sacred" foods
- Diets of plant & animal origin

What was common among these primitive diets that kept these people so healthy and free of disease?

After all, there were literally dozens – perhaps hundreds of different diets or more.

Macronutrient ratios didn't matter – high fat, low fat, high-carb, low-carb, none of that made any difference.

What mattered was **nutrient density**.

All contained "**Sacred foods**" – which were very high in the fat-soluble micronutrients --vitamins A, D, and K2. These were foods like liver and fish eggs for example.

All these diets were of plant and animal origin (<u>OF INTEREST, PRICE DIDN'T FIND</u>, <u>A</u> <u>SINGLE HEALTHY POPULATION COSUMING A VEGAN ONLY DIET</u>)



Price sent back thousands upon thousands of samples of foods from these traditional living peoples to his labs, and what he found was that the **Ancestral Diets** had:

10 times as many fat-soluble vitamins, 4 times as many water-soluble vitamins and 1.5-60 times more minerals.

Now, we might think that we could just take synthetic multivitamins to replace what we're missing in food, right?



What about ALL-CAUSE MORTALITY?

Just a reminder about the Cochrane review by Bjelakovic et al. 2012 discussed earlier 78 Trials confirm higher death rate (1.04-fold) from all causes when consuming <u>synthetic</u> multivitamins.

Cochrane Database Syst Rev. 2012 Mar 14;(3):CD007176. Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases

Processed Foods → Diseases of Western Civilization Heart Disease Stroke Diabetes Cancer Metabolic Syndrome Osteoarthritis Alzheimer's Disease Obesity Autoimmune Disease (Lupus, MS, Rheum Arthr) Etc., etc., etc.

Following Price's research, investigators would eventually connect the dots between processed food consumption and a far greater risk and prevalence of the "Diseases of Western Civilization," which includes those conditions shown on the slide, any likely many more.

Therefore in 2013, Chris Knobbe considered the following question — Could macular degeneration be a disease that follows processed food consumption?



Could macular degeneration be a disease that follows processed food consumption? Well, lets try to find out.



So, the major risk factors for AMD include Aging & Genetics.

But we know that other risk factors for AMD include heart disease, type 2 diabetes, metabolic syndrome, obesity, lack of exercise, and smoking.

But what if AMD isn't caused by these diseases, it simply runs with them -- because they're all caused by the SAME Thing.

But back to Aging and Genetics -- If AMD is all about Aging and genetics. then the prevalence of AMD should have been the same 100 years ago, as it is today, would it not?

Part I: Prevalence of AMD

Was AMD always so prevalent?

When could ophthalmologists see the macula?



Ophthalmologists could see the macula in 1851, because of this man – Hermann von Helmholtz.

Von Helmholtz was a German physician and physicist who, in 1850, designed the ophthalmoscope – that we use to look in the back of the eye.

He published that design in 1851, and within a decade, use of the ophthalmoscope had spread around the world.



This is von Helmholtz' original ophthalmoscope.



And because of the ophthalmoscope, ophthalmologists were seeing the retina of the eye, and the macula.

And in fact, they were producing atlases of the retina, beginning in 1855 forward, that looked like this.

They were obviously seeing the macula.



However, it would be 23 YEARS, before this man, ophthalmologist Jonathan Hutchinson, London, England would describe macular degeneration for the first time – 1874.

He presented four cases in a scientific paper.



Another 11 years goes by, and this man, Otto Haab, German Ophthalmologist, discusses macular degeneration in a lecture – in 1885.

Another 10 years goes by, and Haab publishes a study in which he has evaluated 50,000 OPHTHALMIC MEDICAL RECORDS and **determines that macular degeneration is** about as **rare** as MYOPIC, or near-sighted MACULOPATHY and TRAUMATIC MACULOPATHY, caused by blunt trauma – both exceedingly rare conditions.

Chris Knobbe saw a handful of these conditions in 24 years of ophthalmology practice. Similarly, I have seen <10 cases of MYOPIC MACULOPATHY and between 20-25 cases of

TRAUMATIC MACULOPATHY during 3 decades of

solo private practice with 22,000+ active patients: this includes all ocular trauma from 5 hospital emergency departments in my catchment area.



1927, this man enters the picture: Sir Stewart Duke-Elder. British ophthalmologist, London, England.

Duke-Elder would become perhaps the most esteemed ophthalmologist - and dominant force in ophthalmology – for the next 40 to 50 years.

1927, he publishes his first comprehensive textbook of ophthalmology. 340 pages. Not a SINGLE WORD *relating to macular degeneration*. **76 years after Hermann Von Helmholtz invents the ophthalmoscope**

Yet, 13 years later – in 1940 – he publishes his 2nd textbook of ophthalmology. And in this one, he dedicates 13 pages of text to the condition of macular degeneration, 17 images, 6 of which are in full-color, and he calls macular degeneration **"a common cause of failure in central vision in old people."**



Many people are aware of the Framingham study in Massachusetts because of "cholesterol studies". AMD (macular degeneration) was also examined. Why did AMD prevaluce go from near ZERO, betweem 1851 and 1920, to 8.8% of the population over age 52 in 1975?

1920 – 4.9 Million Americans >65

If Prevalence of AMD in 1920 = 1990 (i.e., AMD = 22.8%)

Should have been 1.1 Million People with AMD in 1920 (if only considering those > 65)

1851 – 1920, Perhaps no more than about 50 cases of AMD documented worldwide

We know from Klein's work - The Beaver Dam Eye Population Study in Wisconsin, 1988-1990 – the Prevalence of AMD >age 65 was 22.8% in the USA. There were 4.9 million Americans>older than 65 in 1920; therefore, there should have been 1.1 million people affected (if only considering this age group). Yet WORLDWIDE between 1851 and 1920 perhaps 50 cases were described in the entire world literature.

R Klein et al., Prevalence of Age-Related Maculopathy. **The Beaver Dam Eye Study** Ophthalmology. **1992**; 99: 933-943. Population study in **Wisconsin**, USA (**1/3>75 years**)



Let's talk about the U.S. Diet – The Big Picture

4 Major Changes have occurred in the U.S., with the world following suit, three of which were introduced between 1880 and 1911.

These four things are nutrient-deficient, toxic, processed foods.



White flour – introduced in 1880. Up until 1880, all wheat was ground to whole-grain flour, because wheat was ground on stone mills.

In 1880, ROLLER MILL technology replaced stone mill technology.

Roller mill technology shears away the bran and the germ, leaving behind only the endosperm. This effectively removes most of the B-vitamins, E-vitamins, fiber, minerals, and omega-3 and omega-6 fats, thereby leaving behind a nutrient-deficient food.

Today, wheat is 20% of the world's diet on a caloric basis.

85.3% of wheat consumed is now "**highly processed refined grains**." – Loren Cordain's group has shown this.



Soybean Oil Corn Oil (Maize Germ Oil) Cottonseed Oil Rape/Rapeseed & Canola Oil Sunflower Oil Safflower Oil Rice Bran Oil Mustard Oil

Vegetable Oils – Polyunsaturated Fatty Acids — also known as PUFAs, introduced in 1866 after the American Civil War (cottonseed oil).

Manufacturers determined they could take cotton seeds – a waste product – crush them, heat them to HIGH temperatures multiple times, run them through a hydraulic press – and then take that substance and treat it to a petroleum derived hexane solvent bath, steam it, alkalinize it, bleach it, and finally, deodorize it – chemically – and declare "now we've got something they say is safe to eat".

These are extraordinarily dangerous products. They're highly oxidized by the time they hit the bottle. And then, when we cook with them and metabolize them, they oxidize even further.

I think these are the SINGLE MOST DANGEROUS substances in our food supply – because a poison is all about the dose – and we're <u>currently consuming 24% of our calories as PUFA</u> vegetable oils.



Here is total vegetable oil consumption in the U.S.

Chris Knobbe's colleague in this research, Marija Stojanoska, produced all these graphs So, look at vegetable oil consumption here. You can see we were near **zero grams a day in 1900**, climbing all the way to **80 grams a day by 2012**.

This is a staggering amount.



80 grams of oil = 720 calories accounting for 32% of US caloric intake of 2250 Calories/day in 2010 [1999; (NHANES data calorie consumption: 1999-2000)]

This change in Total vegetable oil consumption, 1.62g/person/day in 1900 to 80g/person/day in 2010, <u>is a 49.4-fold increase</u> (80/1.62=49.4) YES – an incredible 4,940% INCREASE



Consider this: In 1900, <u>99% of our ADDED FATS</u> came from <u>animal fats</u> – **butter, lard, and beef tallow.** By **2005**, <u>86%</u> of our added fats came <u>from vegetable oils</u>.

Now, let me remind you, there was essentially no macular degeneration in 1900, and virtually no heart disease.

Obesity was 1.2% prior to 1900; 13.3% by 1960 and <u>42.4%</u> in 2017-2018 (CDC National Center for Health Statistics (NCHS) from <u>NHANES data</u> Feb 2020; **the US is on track by 2030 for a 50% obesity rate**. (NHANES) National Health and Nutrition Examination Survey

Note: Added oil of 80 grams includes the vegetables oils added AND hidden in processed food (crackers, cakes, ice cream etc.)



Our THIRD nutrient-deficient, processed food, is trans fats – Partially hydrogenated and hydrogenated vegetable oils – CRISCO.

We all know these are extremely dangerous substances, and we've consumed billions of pounds of these since they were introduced in 1911.

The FDA removed these from GRAS status June 16, 2015, with 3 years for manufacturers to implement the removal, but they're not going away, because they're in vegetable oils.

Vegetable oils have up to 4.6% trans fats and average 1.1% trans fats.



Ten studies have found a higher risk of AMD or AMD progression with vegetable oil consumption and/or trans fat consumption.

This was before the research of Chris Knobbe and Marija Stojanoska.



This is from Chris Knobbe's research, showing sugar consumption from 1840 forwards.

Stephan Guyenet, University of Washington, showed us several years ago that the US consumed 6 pounds of sugar/person/year in 1822, which rose to about 108 pounds/person/year by 1999 – a **more than 17-fold increase in sugar** consumption in that time period.

Sugar is our 4th Nutrient-Deficient, Processed Food.



Put those four foods together, and as of 2009, you have 63% of the U.S. diet – made up of white flour, sugar, vegetable oils, and trans fats; 70% if alcohol is included.

Four processed foods – WITH VIRTUALLY NO MICRONUTRIENTS – Vitamins or Minerals at all, apart from a small amount of vitamin E in the vegetable oils.

This is a recipe for metabolic disaster!

And this is the same recipe for AMD. We'll see that next.



Now we're going to see that **AMD follows processed foods** – wherever they go.

Chris Knobbe used the two processed foods – sugar and vegetable oils -- as proxy markers of processed foods.

Sugar is a well-known proxy marker of processed foods, because it is in 74% of the 600,000 food items available in the U.S.. And **sugar** represents **21%** of U.S. food consumption.

Vegetable oils represent **24%** of U.S. food consumption and, therefore, represent even a bigger portion of processed food than does sugar.

The two together represent 45% of our calories (sugar 21% +PUFAs 24% =45%) but would likely track 90% of processed food.



When processed food was introduced into our diets: **SUGAR 1822**; **SEED OIL 1866** (Cottonseed) — Vegetable oils, more appropriately called "SEED OILS", began to come into greater use following the invention of the interrupted SCREW PRESS by Valerius Anderson in 1900; **REFINED WHEAT FLOUR 1880; TRANS-FATS 1911.**

Now remember to distinguish the non-harmful polyunsaturated vegetable oils, like Palm; Palm-Kernel; Coconut; Olive, Flaxseed, and Avocado from the Harmful vegetable oils as we review AMD prevalence around the world



Soybean Oil Corn Oil (Maize Germ Oil) Cottonseed Oil Rape/Rapeseed & Canola Oil Sunflower Oil Safflower Oil Rice Bran Oil Mustard Oil

To clarify – these are the "dangerous vegetable oils" or "SEED OILS"

1. I think these are the SINGLE MOST DANGEROUS substances in our food supply – because a poison is all about the dose – and we're <u>currently consuming 24% of our calories as PUFA vegetable oils</u>.



Although sugar and vegetable oil consumtion rose the greatest increase was "harmful vegetable oil" after the year 1900

- 1. We didn't have any AMD from 1850 to 1930. And for nearly 60 years of that, we had almost no vegetable oils.
- 2. We started developing AMD in the 1930s, just like the U.K.
- 3. And that's the point. We see in 22 nations that you must consume the processed foods for 30 to 50 years before AMD hits. That is the incubation period for this disease.
- 4. It's just like heart disease...
- Look at the green bars: the first from the Framingham study; (1973-75) 8.8% AMD (ages 52-85); 15 years later: <u>Beaver Dam Eye Study</u> Wisconsin (1988-1990); 20.9% (ages 43-86)
- Sugar consumption rose from 11g/person/day in 1840 to 194 g/person/day in 1998, dropping to 166g/person/day by 2011 (a 17.6-fold increase over 158 years; 1840-1998).
- Total vegetable oil consumption rose from 1.62 g/person/day in 1900 to 19 g/person/day in 1960 to 80 g/person/day in 2010 (a 49.4-fold increase over 110 years; 1900-2010)



- 1. Japan is the quintessential nation to illustrate this entire concept, because we see AMD develop in many of our own lifetimes. I was taught the Japanese were genetically protected!
- 2. Look at sugar rising from 1961; 49g/person/day to 77g/person/day by 2011 a 1.57-fold increase over 50 years
- 3. Polyunsaturated "harmful "vegetable oils rising from 9g/day in 1961 to 39 g/day by 2006 a 4.3-fold increase over 45 years
- 4. AMD rises from 0.2% prevalence in 1974 79 to 11.4% by 2007 a 57-FOLD INCREASE in AMD prevalence!
- 5. What changed? Vegetable oils and sugar, right?
- 6. Are you going to tell me a 57-fold increase in AMD prevalence, in less than 40 years, is due to aging? Or genetics?


- 1. In Nigeria, we see sugar consumption and polyunsaturated vegetable oil consumption are both very low.
- 2. And their AMD is also very low: 3.2% AMD prevalence in Onitscha, Nigeria, 2004. That is a metropolitan population of 1.1 Million people. These people have access to grocery stores processed foods, right?
- 3. Now look at the prevalence of AMD in 2007: 0.1% prevalence. This was in Southwestern rural Nigeria. Guess what they don't have access to? Grocery stores processed food.
- 4. They're consuming a native, traditional diet.
- 5. These are African people in Nigeria, of course.. So, keep this in mind as we look at Barbados....



- 1. Barbados is a 93% African population. (4% mixed)
- 2. First, this is a MECCA for processed food consumption.
- 3. Look at their sugar consumption 140 to 180 grams/day more than 4 times the World Health Organization's recommendations for sugar.
- 4. "Dangerous vegetable oil" consumption is more than 20 some grams/day
- 5. And to go with all this processed food consumption is a WORLD PROFILE of Metabolic disease: Obesity, type 2 diabetes, metabolic syndrome, and so on, of course.
- 6. Now look at their AMD 24.3%!!
- 7. They have **243 times the prevalence** of AMD than the Africans of southwestern **rural Nigeria**, <u>who can't get processed foods</u>!
- 8. Do you truly believe this is caused by genetics? Or aging?
- 9. Sugar consumption already HIGH: (1961) 142 g/person/day; 168 g/person/day by 2008 (1.2-fold increase over 47 years)
- **10. Harmful vegetable oil consumption** 0.05 g/person/day (1961); 31 g/person/day (2009) (<u>a 620-fold increase over 48 years</u>)



- 1. Here we have the Solomon Islands population of 630,000 people. (East of Papua New Guinea in Oceania (Australasia, Melanesia, Micronesia & Polynesia)
- 2. Look at their **sugar consumption extremely low**. 1961; (23 g/person/day); 2010 (36 g/person/day (1.5-fold increase over 49 years)
- 3. Now look at their "Harmful vegetable oils" in red 0 grams a day between 1961-2004; never > 1.73 g/person/day (2004-2010)
- 4. AMD prevalence over the last 10 years? 0.2%!
- 5. The <u>U.S. has at least 74 times more AMD</u> than they have and what's the difference? Sugar and vegetable oils.
- 6. The Solomon Islands has 3 ophthalmologists for a population of 630,000 people. Each one of those ophthalmologists sees about 1 AMD patient every 2 months! American ophthalmologists often see 5, 10, some of them even 20 AMD patients per day! I personally examined no less than 30 AMD patients daily prior to retiring.



- 1. Kiribati is an Island nation located 1200 miles directly south of Hawaii.
- 2. Population 113,000 people.
- 3. Look at their sugar consumption moderately high. (81-119 g/person/day)
- Harmful vegetable oil consumption ZERO between 1961-1995; 2.25g/person/day in 2009
- 5. Now look at their AMD 0.2% prevalence.
- 6. So, the U.S. has at least 74-fold more AMD than Kiribati and what's the difference here? Mostly vegetable oils.
- 7. Kiribati has one ophthalmologist for a population of 113,000 people Rabebe Takeroi. In 2016, he saw 2 patients with AMD. Two – for the year!
- 8. Genetics or age? Seriously? NON-smokers? At 51.4% Kiribati has the highest smoking rate among all adults in the world. Smoking is a well-described risk factor for AMD?



Approximately 50 cases worldwide until 1930

1970's epidemic

196 million with AMD in 2020

288 million by 2040

WHAT CHANGED?



The 'Displacing Foods of Modern Commerce' is what changed.

And we have evidence for proximate cause, because the processed foods always come **before** AMD – it's never the other way around.

Second, there is a temporal relationship – it's about 30 to 50 years. That's how long you must consume these foods to produce AMD.

Third, we see a dose-response relationship. More processed foods – more AMD.

Finally, if you look at all the data together, Chris Knobbe believes the relationship between processed food consumption and AMD is nearly a mathematical certainty. **I humbly agree**.



Of the processed foods, the **Polyunsaturated Vegetable Oil**s are THE GREATEST Contributor to AMD.

These are Biological Poisons.

Like Dr. Knobbe, I believe they are the Single Greatest Contributor to Irreversible Blindness.



So finally, I ask you: Do you believe this hypothesis may have validity?

If so, I am asking you to be ambassadors in this cause – and spread this message – because 196 + million people are losing vision.

I believe it is all preventable.



For more information, come to CureAMD.org

Dr. Knobbe has a book available, regarding dietary intervention and treatment of AMD, through Ancestral Dietary Strategy.



I declare no conflicts of interest