WITH DR. THOMAS O'BRYAN

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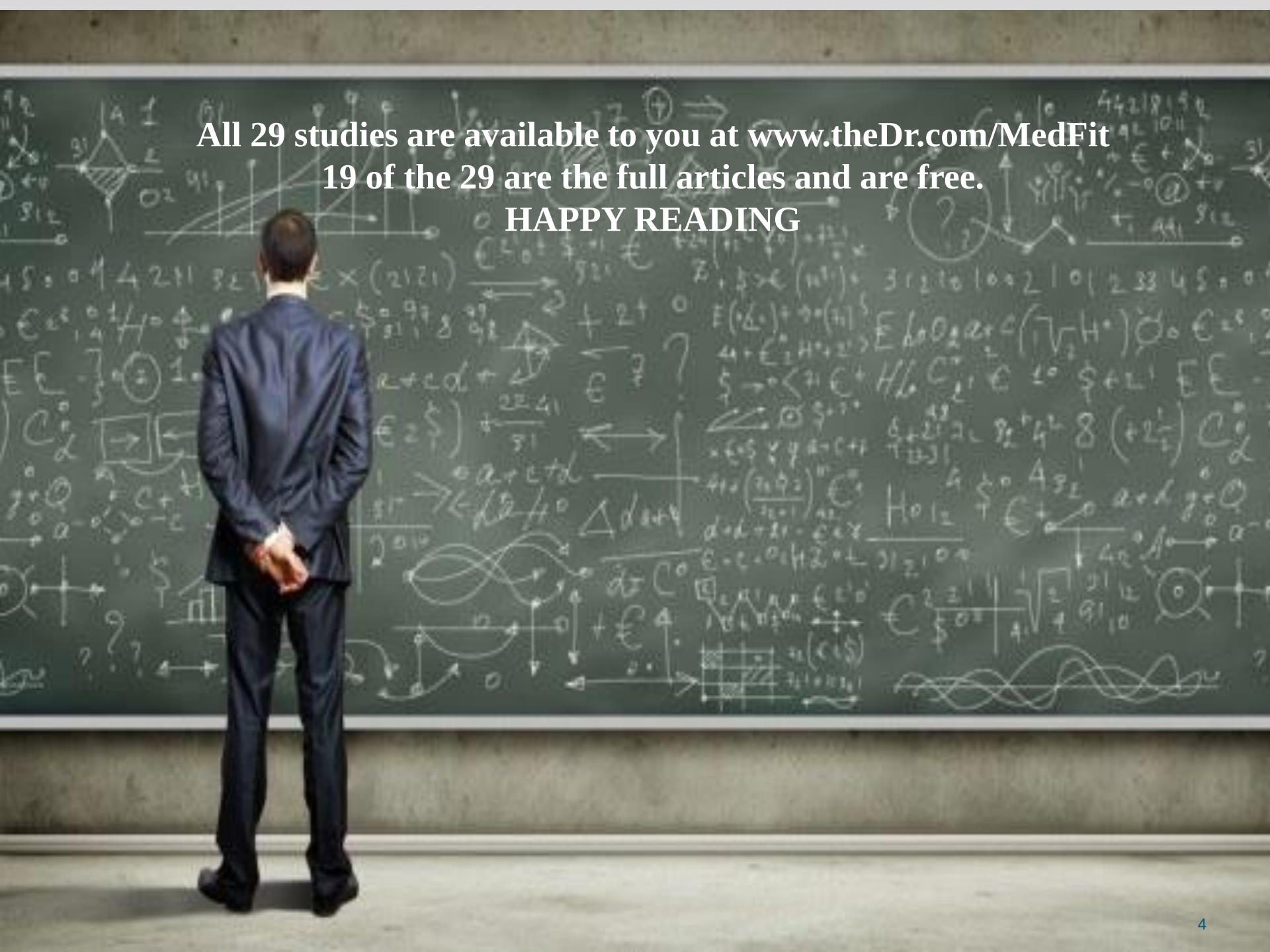
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HAPPY READING

Premise #1

What is the Basic Platform Of all Chronic Diseases

Detective Adrian Monk

Professor Alessio Fasano

- Professor of Pediatrics at Harvard Medical School
- Professor of Nutrition at Harvard T.H. Chan School of Public Health
- Chief of Pediatric Gastroenterology, Mass General Hospital
- Director, Mucosal Immunology Center, Harvard
- Director, Center for Celiac Research and Treatment
- Director, Mucosal Immunology and Biology Research Center;
- Associate Chief for Basic, Clinical and Translational Research
- 213 publications on pubmed.gov
- Identified zonulin as the protein activated in intestinal permeability

F1000Research F1000Research 2020, 9(F1000 Faculty Rev):69 Last updated: 31 JAN 2020

Check for updates

REVIEW

All disease begins in the (leaky) gut: role of zonulin-mediated gut permeability in the pathogenesis of some chronic inflammatory diseases [version 1; peer review: 3 approved]

Alessio Fasano [1,2]

[1]Mucosal Immunology and Biology Research Center, Center for Celiac Research and Treatment and Division of Pediatric Gastroenterology and Nutrition, Massachusetts General Hospital for Children, Boston, Massachusetts, USA
[2]European Biomedical Research Institute of Salerno, Salerno, Italy

v1 **First published:** 31 Jan 2020, **9**(F1000 Faculty Rev):69 (https://doi.org/10.12688/f1000research.20510.1)
Latest published: 31 Jan 2020, **9**(F1000 Faculty Rev):69 (https://doi.org/10.12688/f1000research.20510.1)

Abstract

Improved hygiene leading to reduced exposure to microorganisms has been implicated as one possible cause for the recent "epidemic" of chronic inflammatory diseases (CIDs) in industrialized countries. That is the essence of the hygiene hypothesis that argues that rising incidence of CIDs may be, at least in part, the result of lifestyle and environmental changes that have made us too "clean" for our own good, so causing changes in our microbiota. Apart from genetic makeup and exposure to environmental triggers, inappropriate increase in intestinal permeability (which may be influenced by the composition of the gut microbiota), a "hyper-belligerent" immune system responsible for the tolerance–immune response balance, and the composition of gut microbiome and its epigenetic influence on the host genomic expression have been identified as three additional elements in causing CIDs. During the past decade, a growing number of publications have focused on human genetics, the gut microbiome, and proteomics, suggesting that loss of mucosal barrier function, particularly in the gastrointestinal tract, may substantially affect antigen trafficking, ultimately influencing the close bidirectional interaction between gut microbiome and our immune system. This cross-talk is highly influential in shaping the host gut immune system function and ultimately shifting genetic predisposition to clinical outcome. This observation led to a re-visitation of the possible causes of CIDs epidemics, suggesting a key pathogenic role of gut permeability. Pre-clinical and clinical studies have shown that the zonulin family, a group of proteins modulating gut permeability, is implicated in a variety of CIDs, including autoimmune, infective, metabolic, and tumoral diseases. These data offer novel therapeutic targets for a variety of CIDs in which the zonulin pathway is implicated in their pathogenesis.

Keywords

Chronic inflammatory diseases, Gut permeability, microbiome, zonulin

Open Peer Review

Reviewer Status ✔ ✔ ✔

	Invited Reviewers		
	1	**2**	**3**
version 1 31 Jan 2020	✔	✔	✔

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1 **Xin M. Luo**, Virginia Tech, Blacksburg, USA

2 **Michael Maes**, Chulalongkorn University, Bangkok, Thailand

3 **Arul Jayaraman**, Texas A&M Health Science Center, Bryan, USA

Any comments on the article can be found at the end of the article.

Page 1 of 12

F1000Research F1000Research 2020, 9(F1000 Faculty Rev):69 Last updated: 31 JAN 2020

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There is growing evidence that the elements of gut permeability, immune system response, and gut microbiome—together with genetic predisposition and exposure to environmental triggers— make the *"perfect storm"* for Chronic Inflammatory Disease development.

our immune system. This cross-talk is highly influential in shaping the host gut immune system function and ultimately shifting genetic predisposition to clinical outcome. This observation led to a re-visitation of the possible causes of CIDs epidemics, suggesting a key pathogenic role of gut permeability. Pre-clinical and clinical studies have shown that the zonulin family, a group of proteins modulating gut permeability, is implicated in a variety of CIDs, including autoimmune, infective, metabolic, and tumoral diseases. These data offer novel therapeutic targets for a variety of CIDs in which the zonulin pathway is implicated in their pathogenesis.

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the *"perfect storm"* for Chronic Inflammatory Disease development.

- Genetic Vulnerability
- Environmental triggers
- Altered Microbiome (Dysbiosis)
- Intestial Permeability
- Systemic Immune Response (Innate and Adaptive = Inflammation)

Where to start in reducing environmental triggers?

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REVIEW

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Open Peer Review

Among the several potential intestinal luminal stimuli that can stimulate zonulin release (thus Intestinal Permeability), small exposure to large amounts of bacteria (and its exhaust LPS) and gluten, have been identified as the two most powerful triggers

gastrointestinal tract, may substantially affect antigen trafficking, ultimately influencing the close bidirectional interaction between gut microbiome and our immune system. This cross-talk is highly influential in shaping the host gut immune system function and ultimately shifting genetic predisposition to clinical outcome. This observation led to a re-visitation of the possible causes of CIDs epidemics, suggesting a key pathogenic role of gut permeability. Pre-clinical and clinical studies have shown that the zonulin family, a group of proteins modulating gut permeability, is implicated in a variety of CIDs, including autoimmune, infective, metabolic, and tumoral diseases. These data offer novel therapeutic targets for a variety of CIDs in which the zonulin pathway is implicated in their pathogenesis.

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Abstract

Improved hygiene leading to reduced exposure to microorganisms has been implicated as one possible cause for the recent "epidemic" of chronic

Gluten is misinterpreted by the zonulin pathway as a potential harmful component of a microorganism. (Bystander Activation)

have focused on human genetics, the gut microbiome, and proteomics, suggesting that loss of mucosal barrier function, particularly in the gastrointestinal tract, may substantially affect antigen trafficking, ultimately influencing the close bidirectional interaction between gut microbiome and our immune system. This cross-talk is highly influential in shaping the host gut immune system function and ultimately shifting genetic predisposition to clinical outcome. This observation led to a re-visitation of the possible causes of CIDs epidemics, suggesting a key pathogenic role of gut permeability. Pre-clinical and clinical studies have shown that the zonulin family, a group of proteins modulating gut permeability, is implicated in a variety of CIDs, including autoimmune, infective, metabolic, and tumoral diseases. These data offer novel therapeutic targets for a variety of CIDs in which the zonulin pathway is implicated in their pathogenesis.

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JAMA | Review

Celiac Disease and Nonceliac Gluten Sensitivity
A Review

Maureen M. Leonard, MD, MMSc; Anna Sapone, MD, PhD; Carlo Catassi, MD, MPH; Alessio Fasano, MD

IMPORTANCE The prevalence of gluten-related disorders is rising, and increasing numbers of individuals are empirically trying a gluten-free diet for a variety of signs and symptoms. This review aims to present current evidence regarding screening, diagnosis, and treatment for celiac disease and nonceliac gluten sensitivity.

OBSERVATIONS Celiac disease is a gluten-induced immune-mediated enteropathy characterized by a specific genetic genotype (*HLA-DQ2* and *HLA-DQ8* genes) and autoantibodies (antitissue transglutaminase and antiendomysial). Although the inflammatory process specifically targets the intestinal mucosa, patients may present with gastrointestinal signs or symptoms, extraintestinal signs or symptoms, or both, suggesting that celiac disease is a systemic disease. Nonceliac gluten sensitivity is diagnosed in individuals who do not have celiac disease or wheat allergy but who have intestinal symptoms, extraintestinal symptoms, or both, related to ingestion

celiac disease should be followed up closely for dietary adherence, nutritional deficiencies, and the development of possible comorbidities.

JAMA. 2017;318(7):647-656. doi:10.1001/jama.2017.9730

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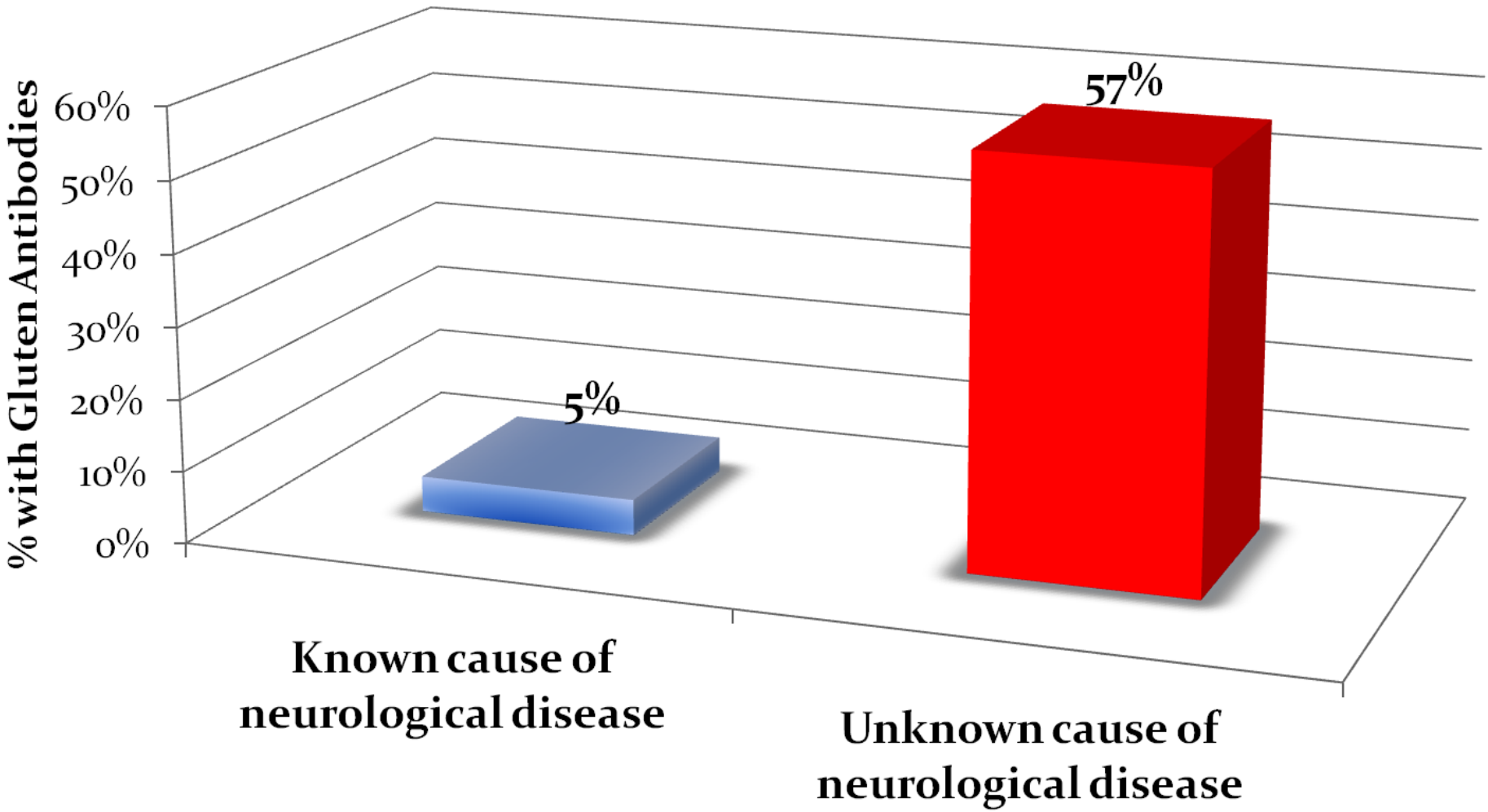
Section Editors: Edward Livingston, MD, Deputy Editor, and Mary McGrae McDermott, MD, Senior Editor.

Previous studies have shown that gliadin (in wheat) can cause an immediate and transient increase in gut permeability. This process takes place in all individuals who ingest gluten.

Celiac disease is a chronic, small-intestinal immune-mediated enteropathy initiated by exposure to dietary gluten in genetically predisposed individuals and characterized by specific autoantibodies against tissue transglutaminase 2 (anti-tTG2), endomysium, and/or deamidated gliadin peptide.[1] Although up to 40% of the population carries the genotype *HLA-DQ2* or *HLA-DQ8*, which is required for the development of celiac disease, only 2% to 3% of *HLA-DQ2* or *HLA-DQ8* carriers subsequently develop celiac disease.[2] Celiac disease, once considered a relatively rare gastrointestinal condition affecting almost exclusively young white children, can develop at any age and can affect almost any race. Celiac disease was first described by Samuel Gee in 1887. Wheat was hypothesized as the possible offending agent by William Dicke in 1941.[3]

The epidemiology, clinical presentation, pathophysiology, and management of the disease have changed since its initial description. There is strong evidence that celiac disease is an autoimmune disease triggered by the ingestion of gluten present in wheat, barley, and rye in genetically predisposed individuals. The prevalence of celiac disease in the general population is 1%, with regional differences (Table 1).[4] Celiac disease can affect any human organ or tissue (Table 1 and Table 2).[6]

Nonceliac gluten sensitivity is a term used to describe individuals who have intestinal signs or symptoms, extraintestinal signs or symptoms, or both, related to ingestion of gluten-containing grains (Table 2), with improvement when these are removed from a patient's diet. The frequency of nonceliac gluten sensitivity is unknown owing to the lack of validated biomarkers, but it is thought to be more common than celiac disease. Wheat allergy, the third gluten-related disorder, which will not be addressed in this review, is defined as an adverse type-2 helper T-cell immunologic reaction to wheat proteins and typically presents soon after wheat



J Neurol Neurosurg Psychiatry **2002 72: 560-563**

Gluten sensitivity

Gluten sensitivity as a neurological illness

M Hadjivassiliou, R A Grünewald, G A B Davies-Jones

From gut to brain

It has taken nearly 2000 years to appreciate that a common dietary protein introduced to the human diet relatively late in evolutionary terms (some 10 000 years ago), can produce human disease not only of the gut but also the skin and the nervous system. The protean neurological manifestations of gluten sensitivity can occur without gut involvement and neurologists must

this disease was the gut. The first report of neurological manifestations associated with CD was by Carnegie Brown in 1908.[3] In his book entitled *Sprue and its treatment* he mentioned two of his patients who developed "peripheral neuritis". Elders reported the association between "sprue" and ataxia in 1925.[4] The validity of these and other such reports before 1960 remains doubtful given that

may not be the sole protagonist in this disease.

THE NEUROLOGY OF COELIAC DISEASE

In 1966 Cooke published a landmark paper on 16 patients with neurological disorders associated with adult CD.[8] This was the first systematic review of the subject after the introduction of diagnostic criteria for CD. Ten of these patients had a severe progressive neuropathy. All patients had gait ataxia and some had limb ataxia. Neuropathological data from postmortem examinations showed extensive perivascular inflammatory changes affecting both the central and peripheral nervous systems. A striking feature was the loss of Purkinje cells with atrophy and gliosis of the cerebellum. All 16 patients had evidence of severe malabsorption as evidenced by anaemia and vitamin deficiencies as well as profound weight loss.

When the cause of a neurological disease is known, the percentage of those patients with elevated antibodies to gluten is 5%. When the cause of a neurological disease is unknown, the percentage of those patients with elevated antibodies to gluten is 57%.

This extract is from the book on chronic diseases by Aretaeus the Cappadocian, one of the most distinguished ancient Greek doctors of the first century AD. This chapter, entitled "on the coeliac diathesis", was the first description of coeliac disease (from the greek word κοιλιακη meaning abdominal). Aretaeus' books were first published in Latin in 1500 and the new Latin word coeliac was used to translate κοιλιακη. Coeliac disease (CD) remained obscure until 1887 when Samuel Gee gave a lecture entitled *On the coeliac affection*[2] at the Hospital for Sick Children, Great Ormond Street, London. In it he acknowledged Areteaus' contribution and went on to give an accurate description of CD based on his own clinical observations.

With clinical manifestations primarily confined to the gastrointestinal tract or attributable to malabsorption, it was logical to assume that the target organ and hence the key to the pathogenesis of

been the lack of satisfactory demonstration of antibodies to the protein concerned". He went on to demonstrate the presence of circulating antibodies against gliadin (antigliadin antibodies), the protein responsible for CD. This provided further evidence that CD was immunologically mediated and that the immune response is not confined to the mucosa of the small bowel. Antigliadin antibodies became a useful screening tool for the diagnosis of CD.

In 1966, Marks *et al* demonstrated an enteropathy in nine of 12 patients with dermatitis herpetiformis,[7] an itchy vesicular skin rash mainly occurring over the extensor aspect of the elbows and knees. The enteropathy had a striking similarity to that seen in CD. It was later shown that the enteropathy and the skin rash were gluten dependent but skin involvement could occur even without histological evidence of gut involvement. This was the first evidence that the gut

Peripheral neuropathy	29
Myopathy	13
Ataxia with myoclonus	9
Myelopathy	4
Dementia (usually with additional features)	6

A review of all such reports (with biopsy proved CD) from 1964 to date shows that ataxia and peripheral neuropathy are the commonest neurological manifestations seen in patients with established CD (table 1). Less common manifestations include inflammatory myopathies[10] and myoclonic ataxia.[11] Isolated dementia is uncommon and most cases tend to have additional neurological features (for example, ataxia or neuropathy). Patients with epilepsy associated with occipital calcifications on CT and CD have been described,[12] mainly in Italy. Most present with epilepsy in

Case Study #1
A 14 year old girl misdiagnosed with psychosis

Nutrients 2015, 7, 5532-5539

Nutrients **2015**, *7*, 5532-5539; doi:10.3390/nu7075235

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Case Report

Gluten Psychosis: Confirmation of a New Clinical Entity

A 14-year-old girl came to our outpatient clinic for psychotic symptoms that were apparently associated with gluten consumption.

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In May 2012, after a fever, she became increasingly irritable and reported daily headache and concentration difficulties. One month after, her symptoms worsened presenting with severe headache, sleep problems, and behavior alterations, with several unmotivated crying spells and apathy.

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Case Report

Gluten Psychosis: Confirmation of a New Clinical Entity

Her school performance deteriorated, as reported by her teachers. The mother noted severe bad breath, never suffered before. The patient was referred to a local neuropsychiatric outpatient clinic, where a conversion somatic disorder was diagnosed and a benzodiazepine treatment (*i.e.*, bromazepam) was started.

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Case Report

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In June 2012, during the final school examinations, psychiatric symptoms, occurring sporadically in the previous two months, worsened. Indeed, she began to have complex hallucinations. The types of these hallucinations varied and were reported as indistinguishable from reality (she saw people coming off the television to follow and scare her).

MassGeneral Hospital for Children, 55 Fruit Street, Boston, MA 02114, USA

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Case Report

Gluten Psychosis: Confirmation of a New Clinical Entity

She also presented weight loss (about 5% of her weight) and gastrointestinal symptoms such as abdominal distension and severe constipation. She was admitted to a psychiatric ward.

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Case Report

A CT scan of the brain and a blood pressure holter were also performed and resulted normal. EEG showed mild nonspecific abnormalities and slow-wave activity. Due to the abnormal autoimmune parameters and the recurrence of psychotic symptoms, autoimmune encephalitis was suspected, and steroid treatment was initiated.

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Case Report

In September 2012, shortly after eating pasta, she presented crying spells, relevant confusion, ataxia, severe anxiety and paranoid delirium. Then she was again referred to the psychiatric unit. A relapse of autoimmune encephalitis was suspected and treatment with endovenous steroid and immunoglobulins was started. During the following months, several hospitalizations were done, for recurrence of psychotic symptoms.

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Case Report

Cerebral and spinal cord magnetic resonance imaging, lumbar puncture, and fundus oculi examination did not show any pathological signs. Several EEG were performed confirming bilateral slow activity. The laboratory tests showed only mild microcytic anemia with reduced levels of ferritin and a slight increase in fecal calprotectin values.
All markers for CD were negative.

[4] The Division of Paediatric Gastroenterology and Nutrition and Center for Celiac Research, MassGeneral Hospital for Children, 55 Fruit Street, Boston, MA 02114, USA

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Case Report

In September 2013, she presented with severe abdominal pain, associated with asthenia, slowed speech, depression, distorted and paranoid thinking and suicidal ideation up to a state of pre-coma. The clinical suspicion was moving towards a fluctuating psychotic disorder. Treatment with a second-generation anti-psychotic (*i.e.*, olanzapine) was started, but psychotic symptoms persisted.

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Case Report

November 2013, due to gastro-intestinal symptoms and further weight loss (about 15% of her weight in the last year), a nutritionist was consulted, and a gluten-free diet (GFD) was recommended for symptomatic treatment of the intestinal complaints; unexpectedly, within a week of gluten-free diet, the symptoms (both gastro-intestinal and psychiatric) dramatically improved, and the GFD was continued.

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Case Report

Due to parental choice, the girl did not continue assuming gluten and she started a gluten-free diet with a complete regression of all symptoms within a week. Her mother finally recalled that she was returned a "normal girl". Nine months after definitely starting the GFD, she is still symptoms-free.

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Case Report

Until a few years ago, the spectrum of gluten-related disorders included only CD and wheat allergy, therefore our patient would be turned back home as a "psychotic patient" and receive lifelong treatment with anti-psychotic drugs.

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A patient can present with a calm exterior, *“I feel fine”*

But inside can be a raging fire of destruction

Premise #2

The Brain is Your ‘Yellow Canary in the Coal Mine’

Detective Adrian Monk

What are the current numbers of frequency of brain dysfunction at both ends of life?

The Prevalence of Parent-Reported Autism Spectrum Disorder Among US Children

Michael D. Kogan, PhD,[a] Catherine J. Vladutiu, PhD, MPH,[a] Laura A. Schieve, PhD,[b] Reem M. Ghandour, DrPH,[a] Stephen J. Blumberg, PhD,[c] Benjamin Zablotsky, PhD,[c] James M. Perrin, MD,[d] Paul Shattuck, PhD,[e] Karen A. Kuhlthau, PhD,[d] Robin L. Harwood, PhD,[a] Michael C. Lu, MD, MPH[f]

abstract

OBJECTIVES: To estimate the national prevalence of parent-reported autism spectrum disorder (ASD) diagnosis among US children aged 3 to 17 years as well as their treatment and health care experiences using the 2016 National Survey of Children's Health (NSCH).

METHODS: The 2016 NSCH is a nationally representative survey of 50 212 children focused on the health and well-being of children aged 0 to 17 years. The NSCH collected parent-

characteristics and co-occurring conditions.

CONCLUSIONS: The estimated prevalence of US children with a parent-reported ASD diagnosis is now 1 in 40, with rates of ASD-specific treatment usage varying by children's sociodemographic and co-occurring conditions.

WHAT THIS STUDY ADDS:

The estimated prevalence of US children with parent-reported diagnosis of ASD is now 1 in 40.

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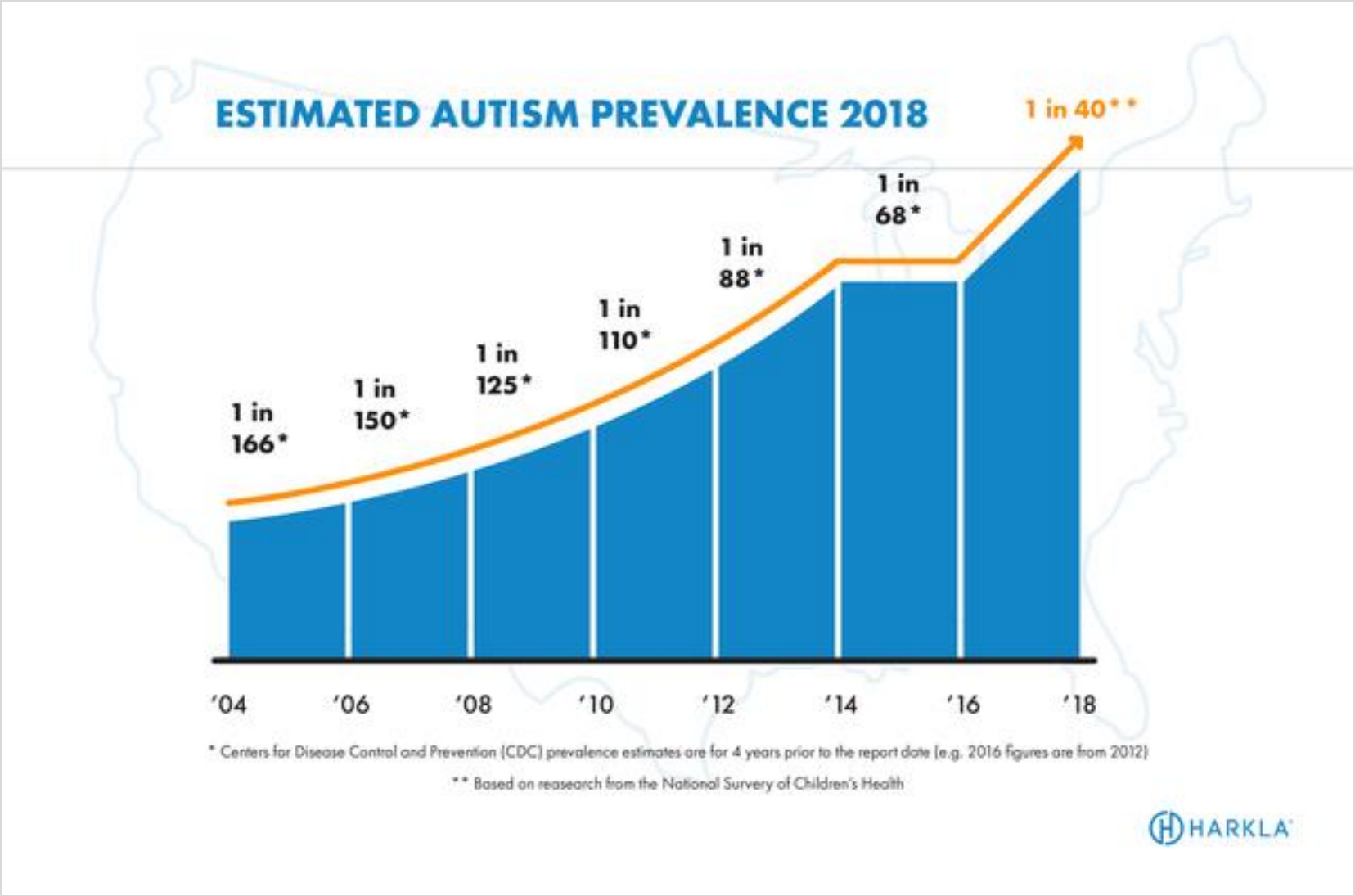
Dr Kogan conceptualized and designed the study and drafted most of the initial manuscript; Dr Vladutiu conducted the data analyses and assisted with drafting of the initial manuscript; Dr Schieve assisted with drafting of the initial manuscript and provided critical review of subsequent manuscript drafts; Drs Ghandour, Blumberg, Zablotsky, Perrin, Shattuck, Kuhlthau, Harwood, and Lu provided critical reviews on all manuscript drafts; and all authors approved the final manuscript as submitted.

WHAT'S KNOWN ON THIS SUBJECT: Previous studies over the last 20 years have shown an increasing prevalence of autism spectrum disorder (ASD) among US children. Moreover, families of children with ASD have reported greater health care needs and challenges compared with children with other emotional or behavioral conditions.

WHAT THIS STUDY ADDS: In this study, we present new nationally representative data on the prevalence of ASD, reported health care challenges, and estimates on ASD-specific behavioral and medication treatments. The estimated prevalence of US children with parent-reported diagnosis of ASD is now 1 in 40.

To cite: Kogan MD, Vladutiu CJ, Schieve LA, et al. The Prevalence of Parent-Reported Autism Spectrum Disorder Among US Children. *Pediatrics*. 2018;142(6):e20174161

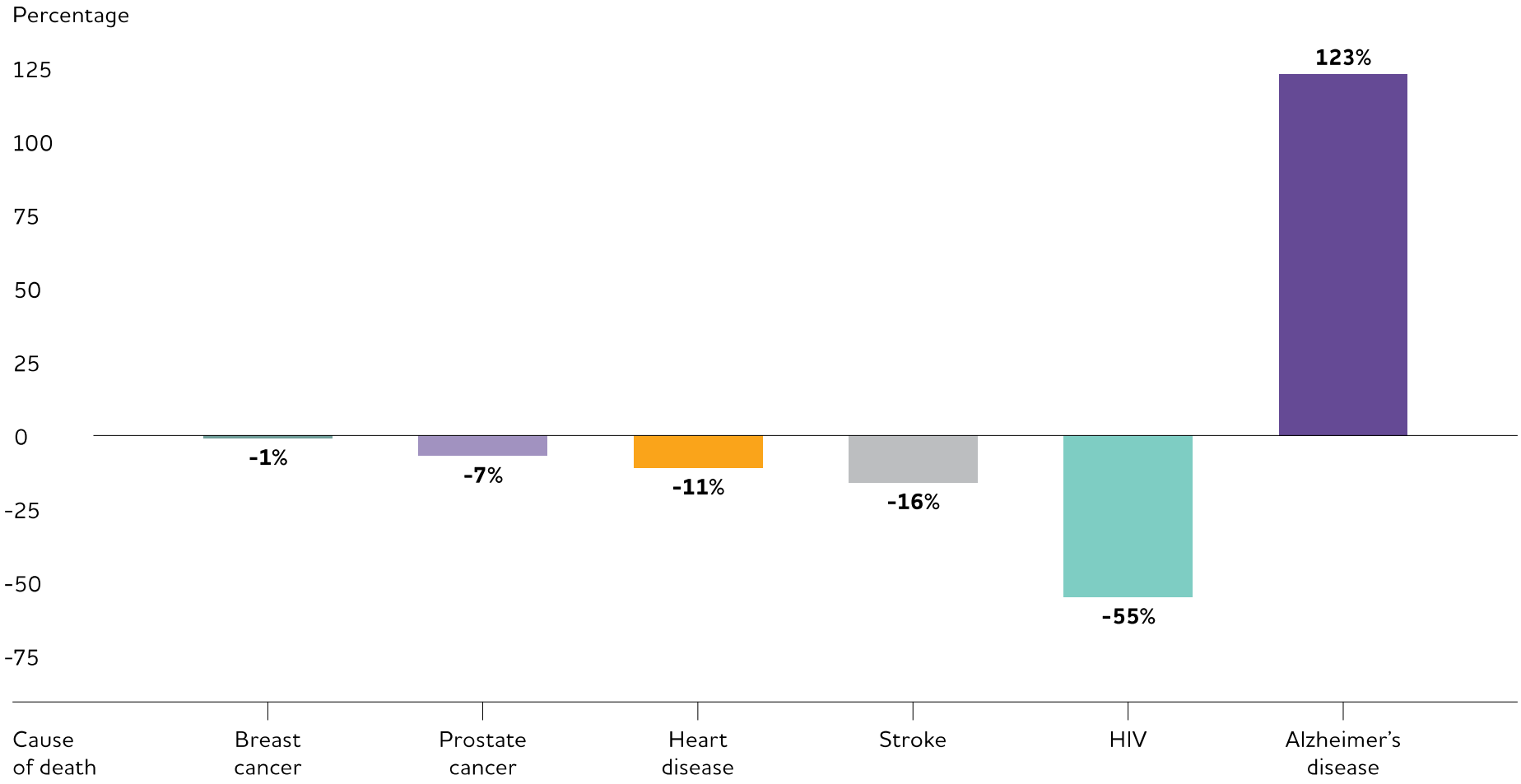
Pediatrics. 2018;142(6):e20174161


ESTIMATED AUTISM PREVALENCE 2018
1 in 40**
1 in 166*
1 in 150*
1 in 125*
1 in 110*
1 in 88*
1 in 68*
'04
'06
'08
'10
'12
'14
'16
'18
* Centers for Disease Control and Prevention (CDC) prevalence estimates are for 4 years prior to the report date (e.g. 2016 figures are from 2012)
** Based on reasearch from the National Survery of Children's Health
HARKLA

MORTALITY AND MORBIDITY

FIGURE 5

Percentage Changes in Selected Causes of Death (All Ages) Between 2000 and 2015

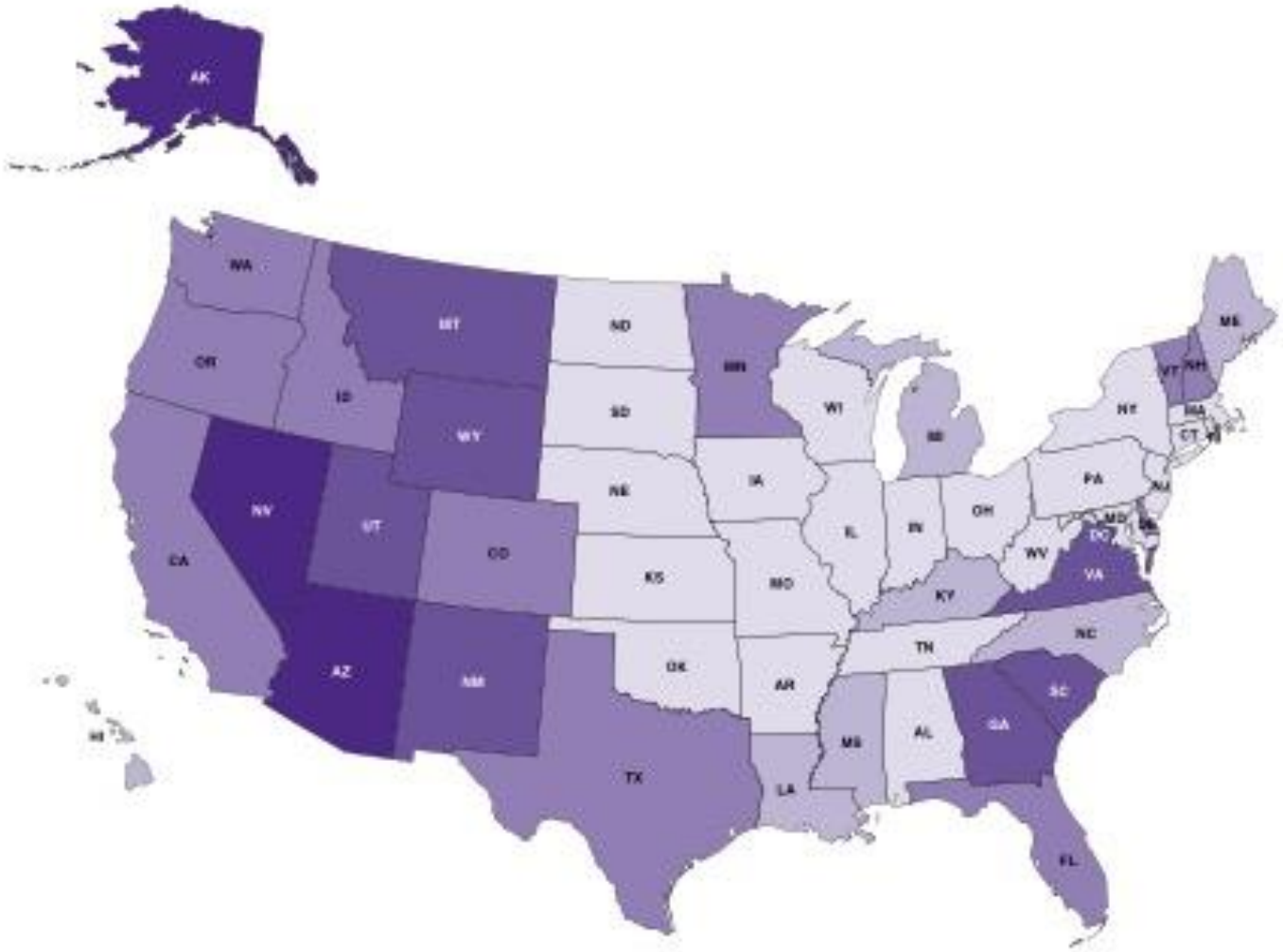


rom data from the National Center for Health Statistics.[232,243]

FIGURE 2

Projected Increases Between 2018 and 2025 in Alzheimer's Dementia Prevalence by State

13.6% - 20.2% | 20.3% - 26.8% | 26.9% - 33.5% | 33.6% - 40.1% | 40.2% - 46.7%



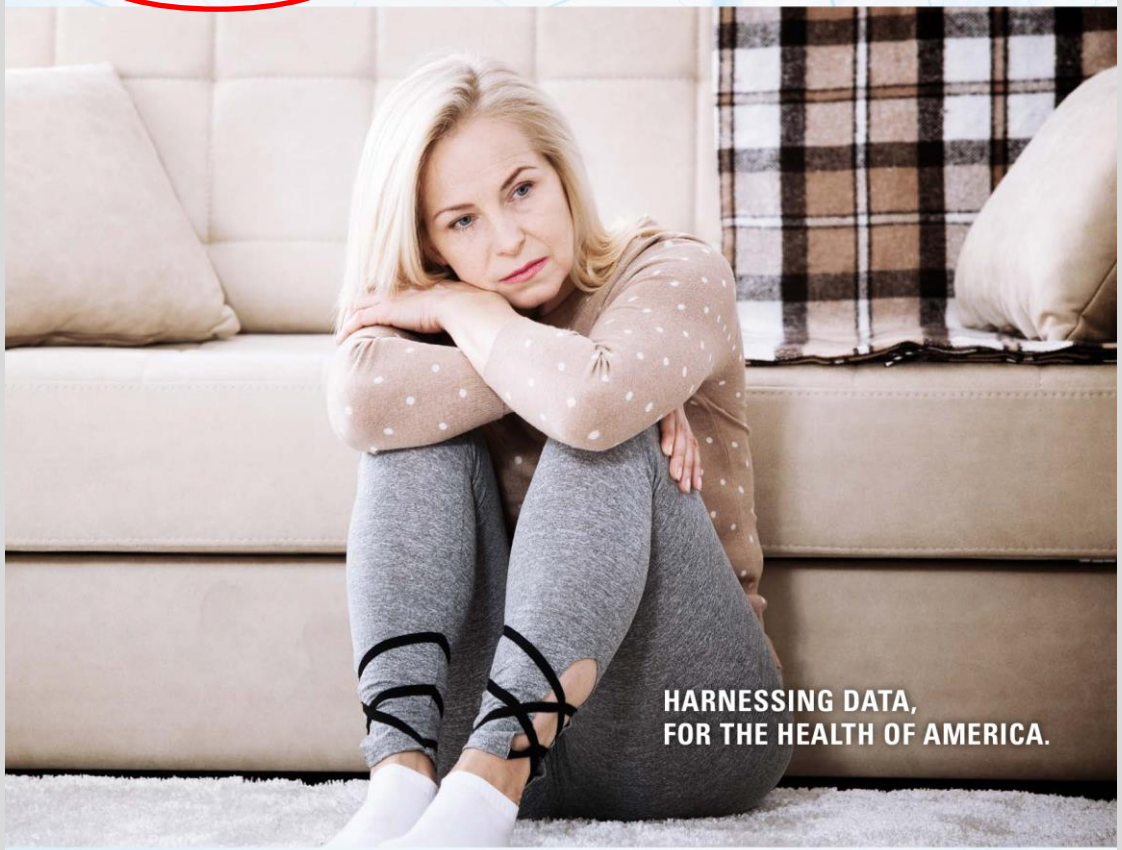
Change from 2018 to 2025 for Washington, D.C.: 1.1%

Created from data provided to the Alzheimer's Association by Weuve et al.[47,108]

BlueCross BlueShield
THE HEALTH OF AMERICA℠

EARLY-ONSET DEMENTIA AND ALZHEIMER'S RATES GROW FOR YOUNGER AMERICANS

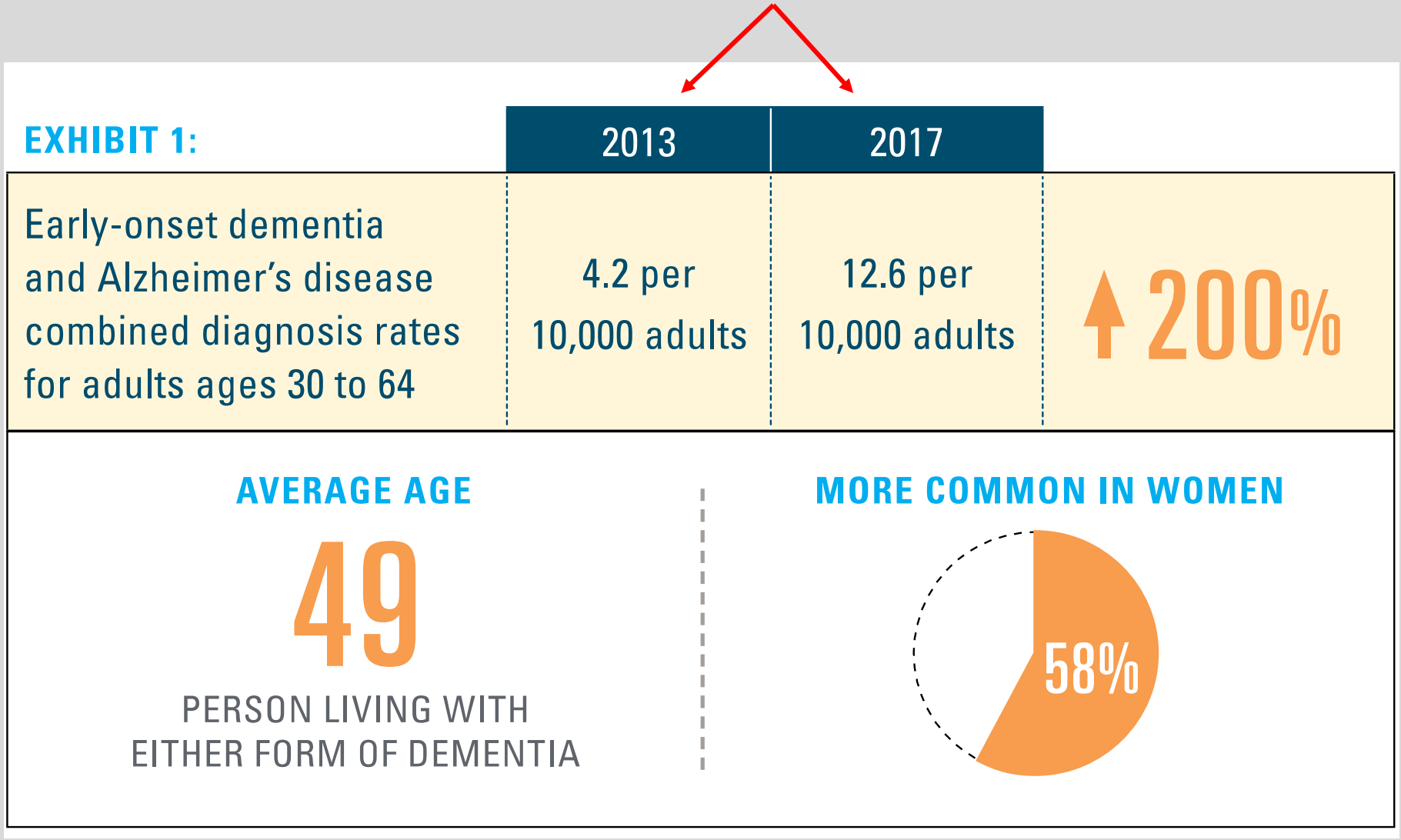
February 27, 2020



bhi Blue Health Intelligence®

https://www.bcbs.com/the-health-of-america/reports/early-onset-dementia-alzheimers-disease-affecting-younger-american-adults

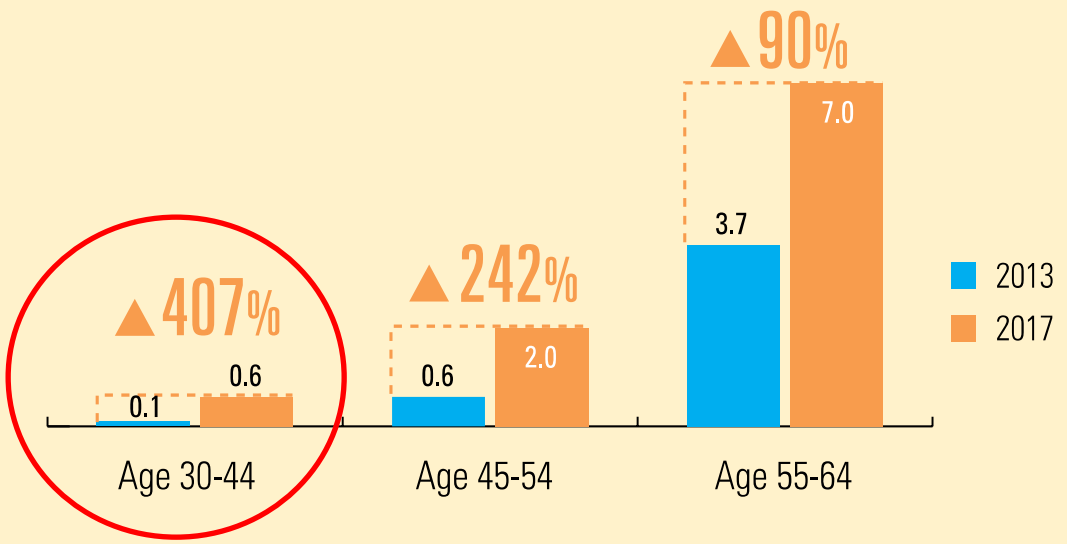
4 Years



https://www.bcbs.com/the-health-of-america/reports/early-onset-dementia-alzheimers-disease-affecting-younger-american-adults

Overall diagnosis of the condition is small, but from 2013 to 2017, there were large increases in early-onset Alzheimer's disease among people ages 30 to 54. (See Exhibit 5.)

EXHIBIT 5: DIAGNOSIS RATES OF EARLY-ONSET ALZHEIMER'S DISEASE BY AGE, PER 10,000 PEOPLE (2013 vs. 2017)



https://www.bcbs.com/the-health-of-america/reports/early-onset-dementia-alzheimers-disease-affecting-younger-american-adults

So what is the takeaway from these startling statistics as to what is happening to our Brains?

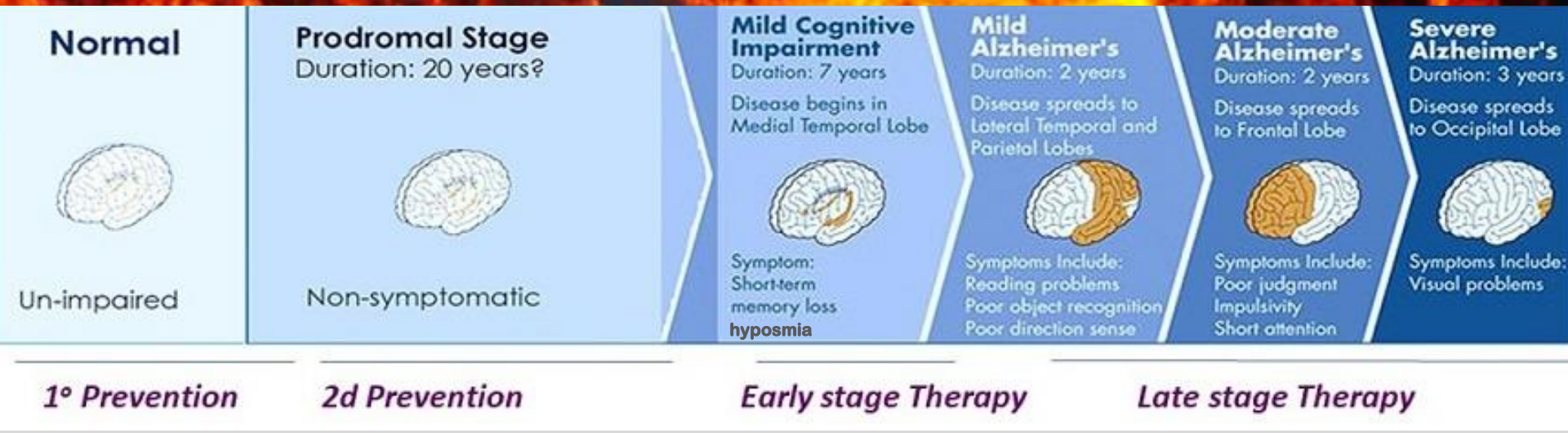
The Brain, our *'yellow canary in the coal mine'*, is under constant assault from conception to elderly

and We're Losing the Battle

How Do We Begin to Understand Where This De[illegible]From?

First and foremost is the understanding that Alzheimer's is a decades-long process of deterioration

Normal levels of brain antibodies (cellular regeneration, apoptosis,…)



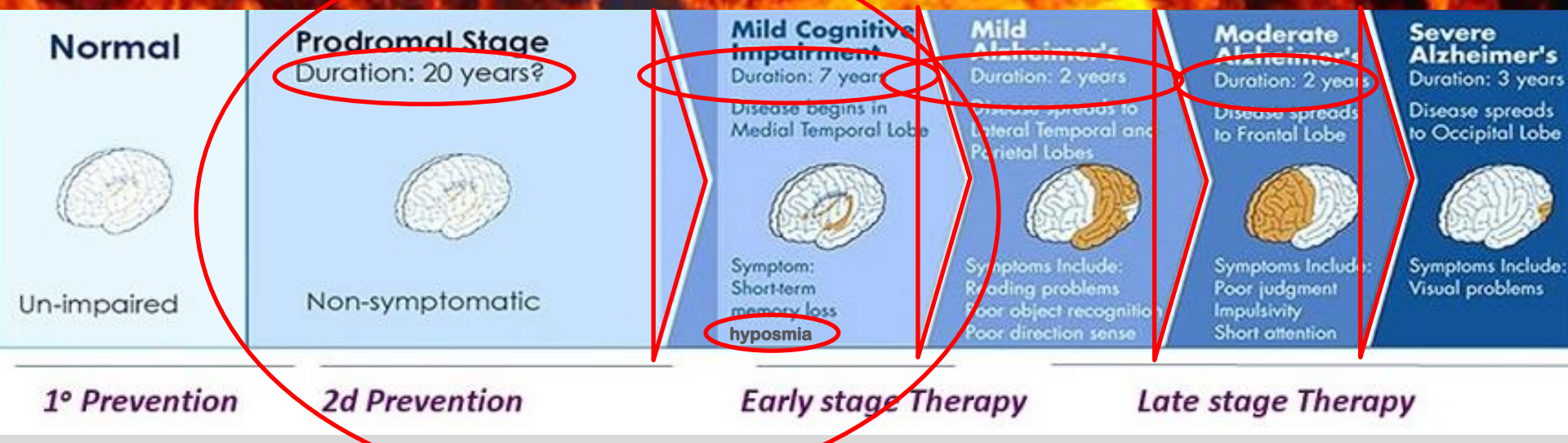
Food sensitivities (loss of oral tolerance)
Infections (bacterial, viral, LPS, … loss of tolerance)
Environmental exposure loss of tolerance
Electromagnetic pollution loss of tolerance
Emotional stress loss of tolerance
Structural loss of tolerance

https://aspe.hhs.gov/advisory-council-july-2019-meeting-presentation-adrd-therapeutic-approaches

Elevated levels of brain antibodies

(molecular mimicry, self-antigen modification, bystander activation, and immune reactivity modulation)

Creates a Breach of the Blood Brain Barrier(B4)



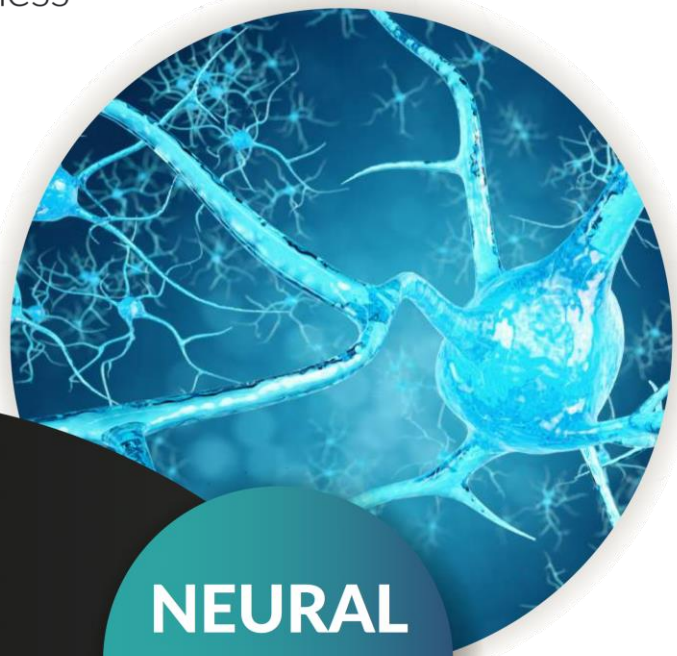
Food sensitivities (loss of oral tolerance)
Infections (bacterial, viral, LPS, … loss of tolerance)
Environmental exposure loss of tolerance
Electromagnetic pollution loss of tolerance
Emotional stress loss of tolerance
Structural loss of tolerance


© AP

It is of critical clinical value to have valid tests identifying if the brain is suffering from chronic inflammatory conditions that eventually accumulates damage into become a disease.

Test, Don't Guess

VibrantWellness



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Peptide level identification of neural sensitivity

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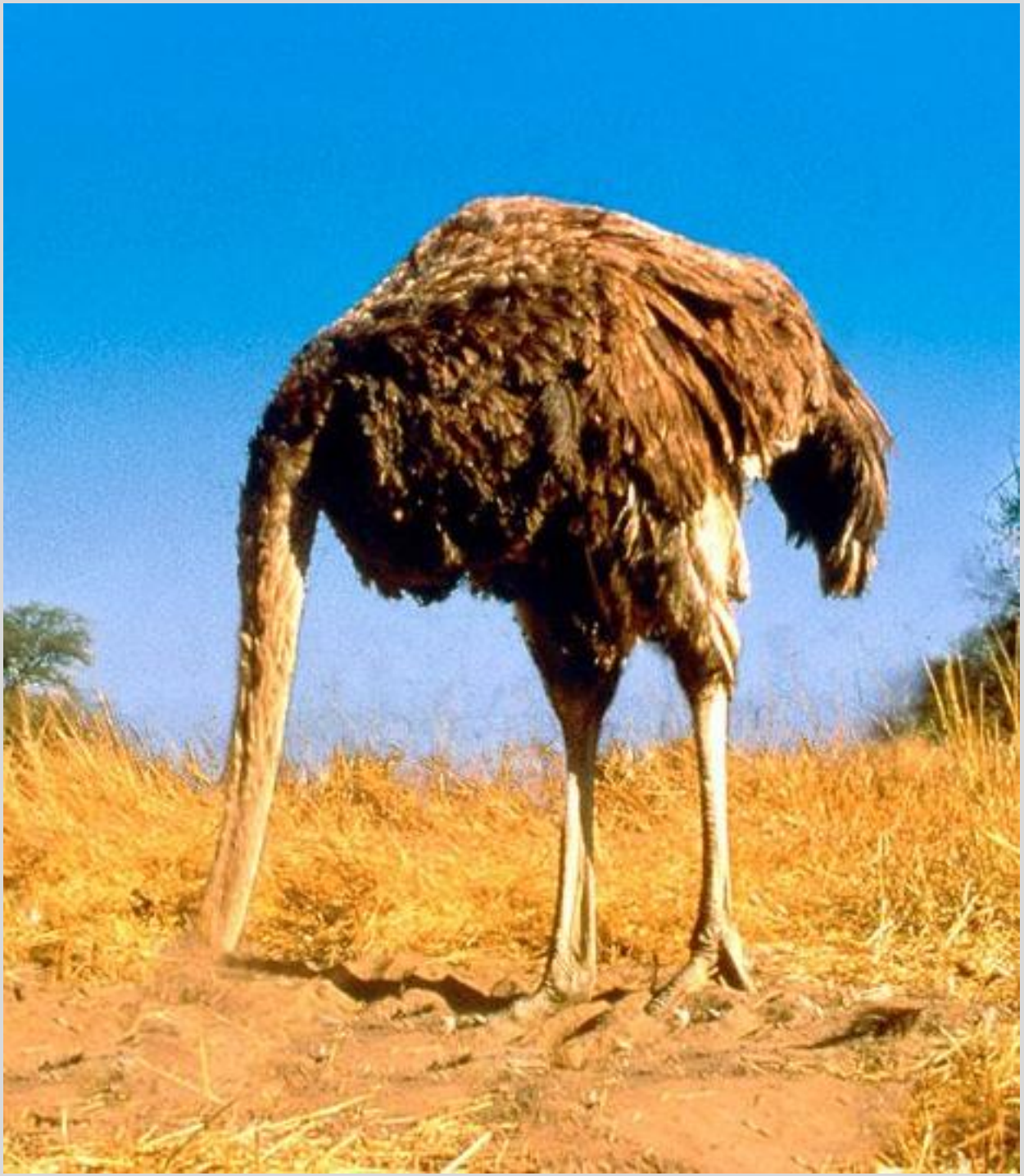
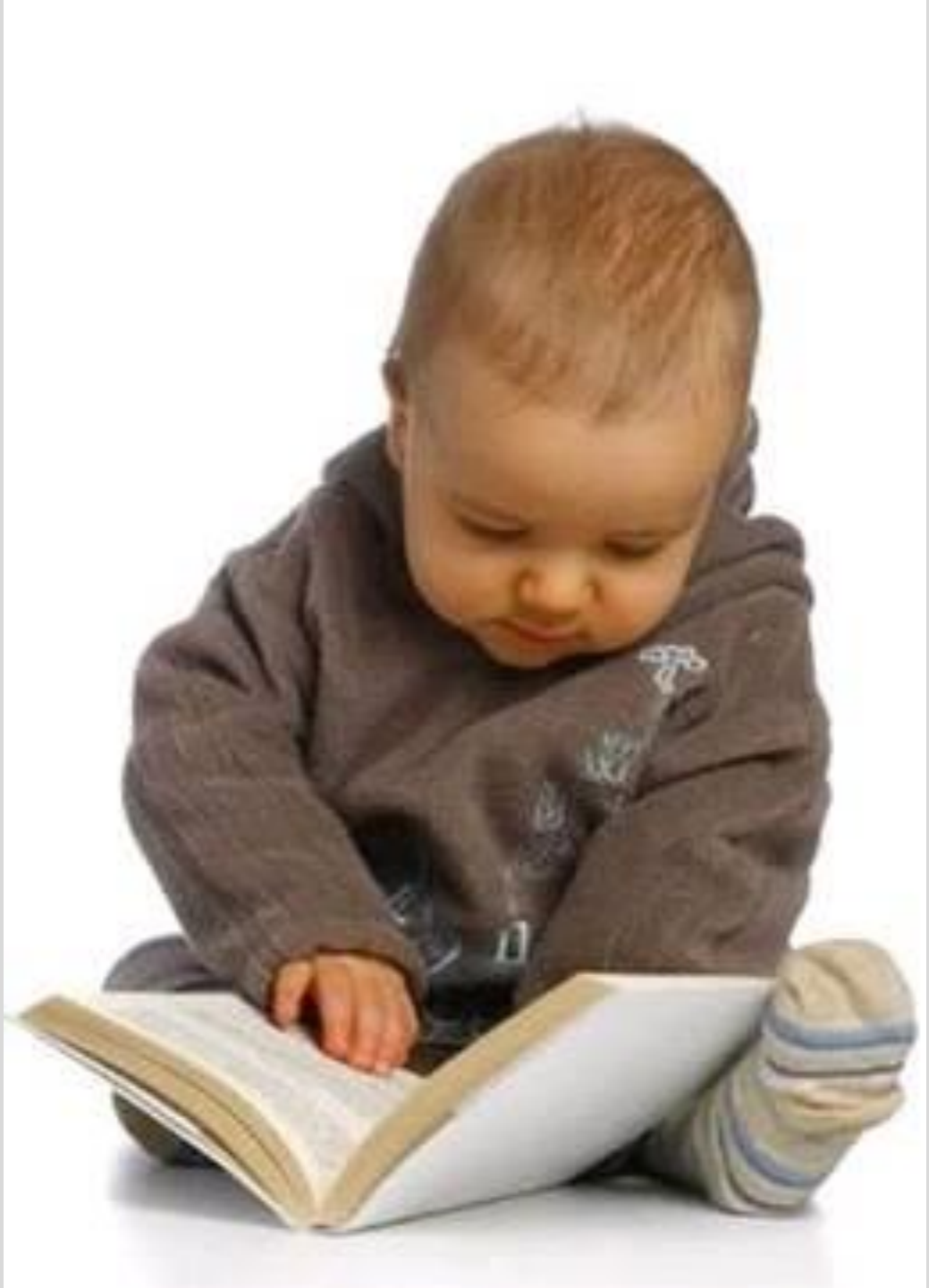
Testing for Brain Inflammation

- Antibodies to Brain Tissue
- Antibodies identifying a Breach of the Blood Brain Barrier
- Antibodies to optical and ANS disorders
- Antibodies to systemic Brain autoimmunity
- Antibodies to bacteria in the Brain causing inflammation
- Antibodies to viruses in the Brain causing inflammation
- Antibodies creating demyelination
- Antibodies creating peripheral neuropathies

How are we supposed to understand a test like this?





English

Russian

German

Turkish

Portuguese (Brazil)

Korean

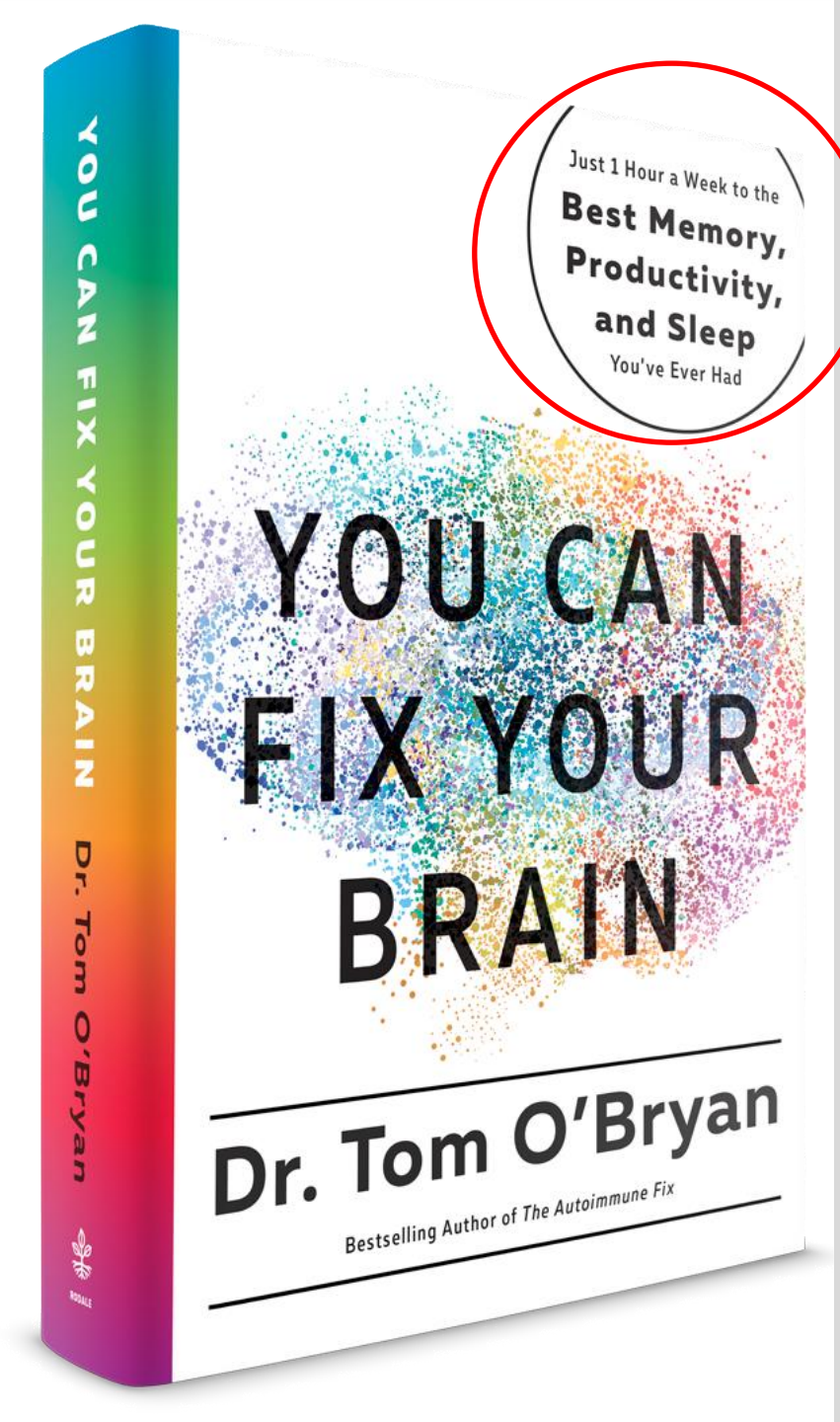
Chinese

Hungarian

Thai

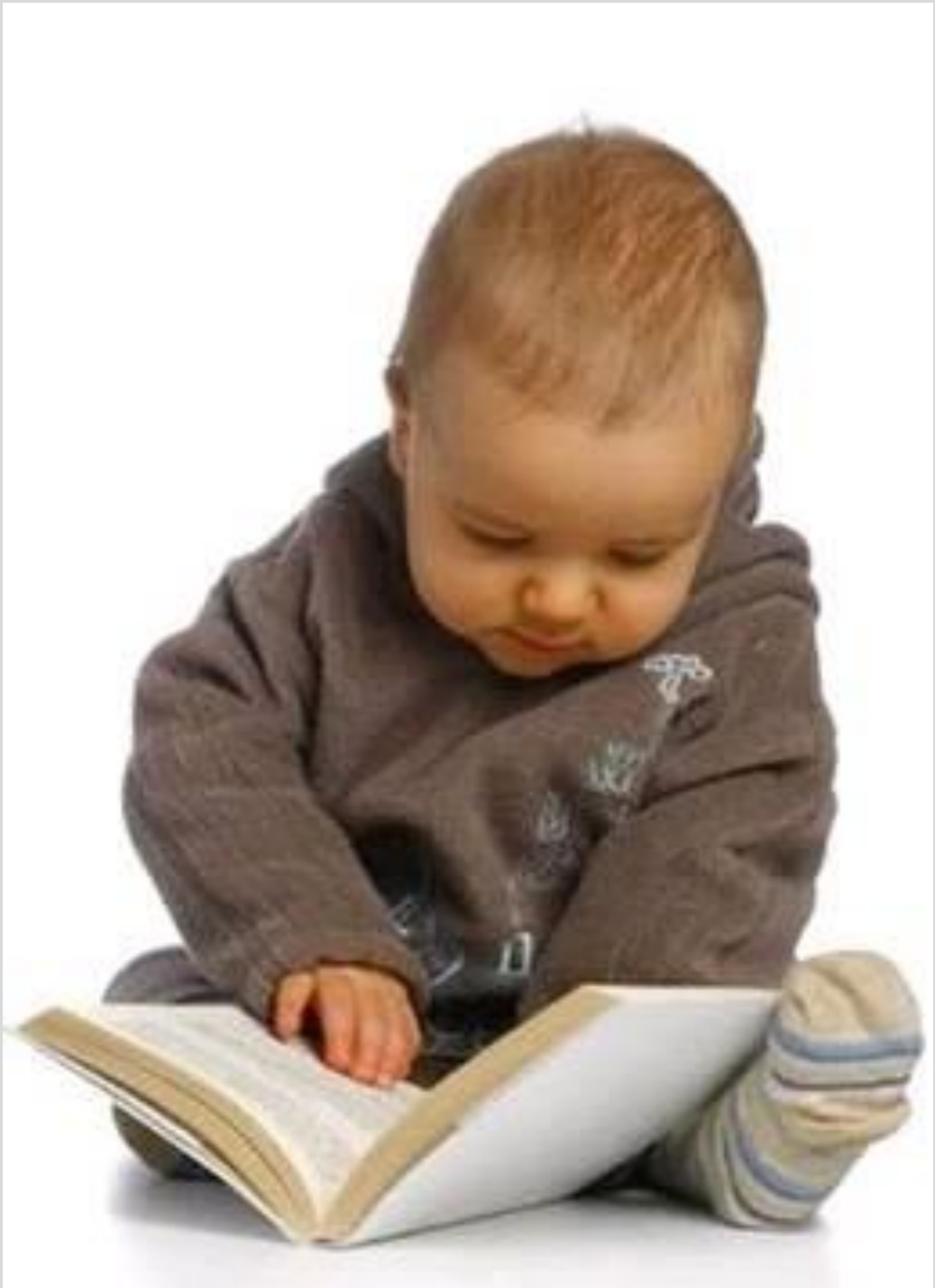
Spanish

Polish



Case Study

An Autoimmune Brain Mechanism Secondary to Bacterial Infection and Molecular Mimicry



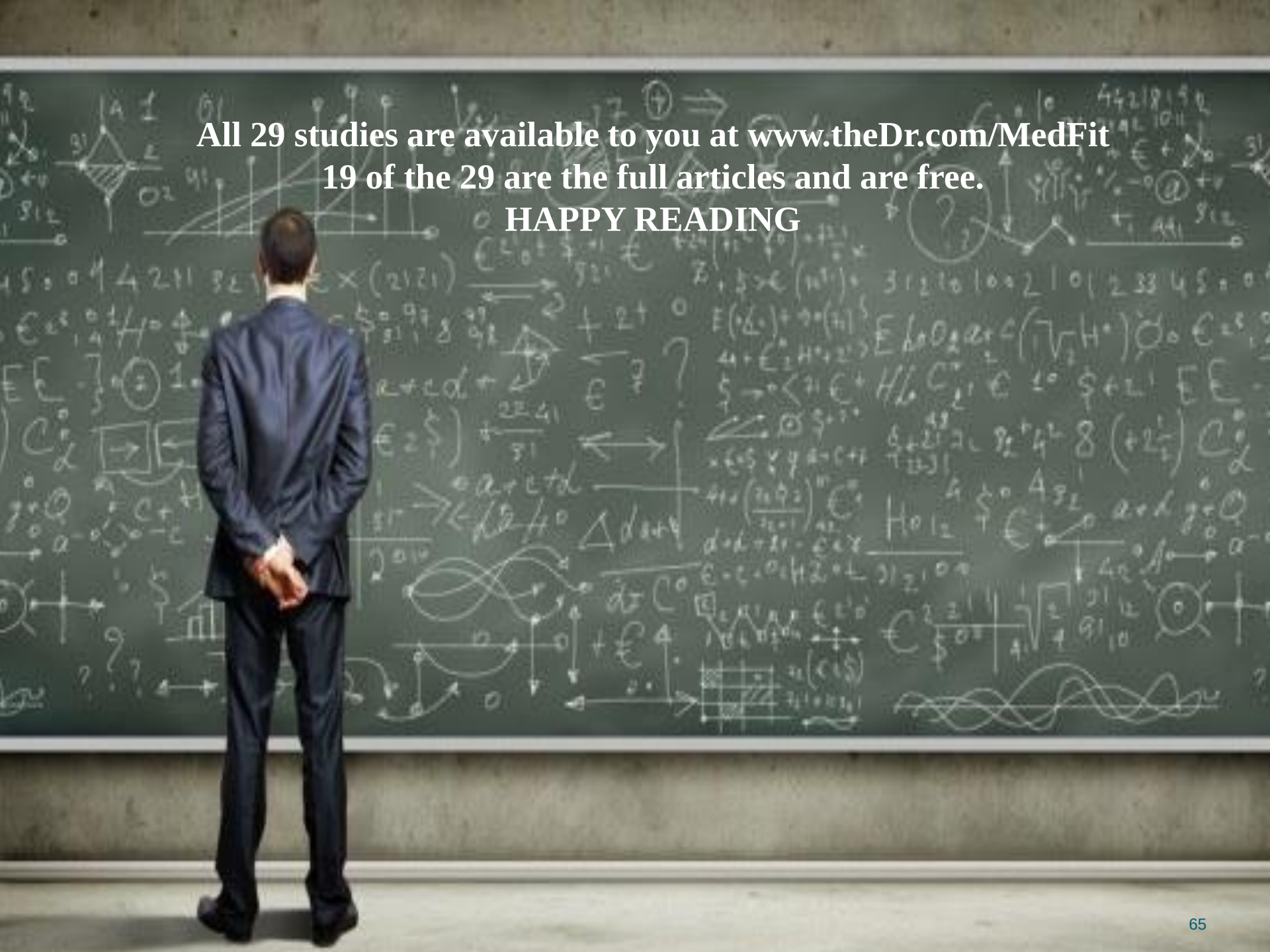
the *"perfect storm"* for Chronic Inflammatory Disease development.

- Genetic Vulnerability
- Environmental triggers
- Altered Microbiome (Dysbiosis)
- Intestial Permeability
- Systemic Immune Response (Innate and Adaptive = Inflammation)

The Brain is your
‘Yellow Canary in the Coal Mine’

Detective Adrian Monk

SUMMARY


All 29 studies are available to you at www.theDr.com/MedFit
19 of the 29 are the full articles and are free.
HAPPY READING

Take Care of Yourself

Make Sure to Tell those Important to You How Much You Love them


“Thank You for Your Kind Attention”