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Nutritional Supplements

Biogenetix

Disclaimer

* *Biogenetix products are not intended to diagnose, treat, reverse, cure, or prevent any disease. They are intended to be used as dietary supplements for the sole purpose of supporting patients. The following statements have not been evaluated by the FDA.*

* *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Why DM2/1.5?

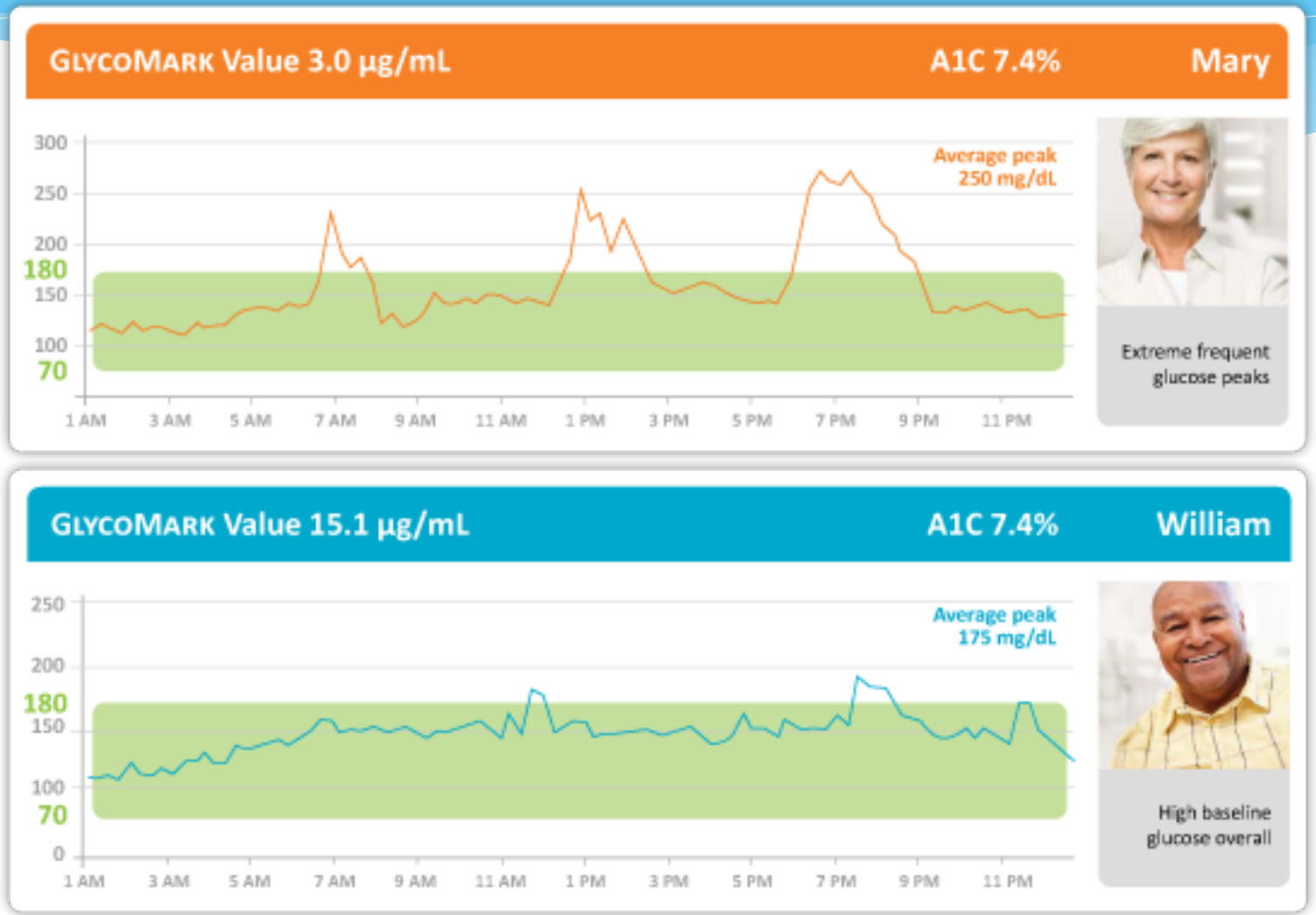
CHRONIC DISEASE…

* 117 Million Americans with Chronic Disease
* 7/10 Deaths.
* Number one cause of Death and Disability in the US.

*****YOU are their answer.**

New Standards in Assessment

* Glycomark
* Hemoglobin A1c
* C-Peptide
* Fasting Insulin*



Assessment 1 - Glycomark

If Glycomark is <15 µg/mL:

1. Focus on diet – quality of food (macronutrient ratio) and quantity of food
2. GLP-1 stimulators (pre-meal)
    1. Fish oil
    2. Pea protein, glutamine
    3. Quercitin
    4. Bile acid support
    5. MUFA
    6. Chew food thoroughly
    7. Olive leaf extract

Diabetes Care 1

Glucose Variability in a 26-Week Randomized Comparison of Mealtime Treatment With Rapid-Acting Insulin Versus GLP-1 Agonist in Participants With Type 2 Diabetes at High Cardiovascular Risk

DOI: 10.2337/dc15-2782

The FLAT-SUGAR Trial Investigators*

CLIN CARE/EDUCATION/NUTRITION/PSYCHOSOCIAL

OBJECTIVE

A1C is associated with diabetes complications but does not reflect glycemic variability (GV), which may worsen outcomes by inducing inflammation, oxidative stress, and cardiac arrhythmias. We tested whether a glucagon-like peptide 1 agonist-based regimen can reduce GV and cardiometabolic risk markers while maintaining similar A1C levels in people with insulin-requiring type 2 diabetes and high cardiovascular risk.

RESEARCH DESIGN AND METHODS

After run-in on metformin and basal-bolus insulin (BBI), 102 participants continued metformin and basal insulin and were randomized to exenatide dosing before the two largest meals glucacon-like peptide-1 receptor agonist and insulin (GLIPULIN group) or continuation of rapid-acting insulin analogs (BBI group). Indices of GV by continuous glucose monitoring (CGM), hypoglycemia, weight, risk markers, and cardiac arrhythmias were assessed. The primary end point was change in glucose coefficients of variation (CV) by CGM from baseline to 26 weeks.

RESULTS

At randomization, the median A1C was 7.3% (57 mmol/mol) for GLIPULIN and 7.4% (56.3 mmol/mol) for BBI, and glucose CVs were 30.3 for BBI and 31.9 for GLIPULIN. At 26 weeks, A1C levels were similar (7.1% [54 mmol/mol] vs. 7.2% [55 mmol/mol]), whereas mean CV improved with GLIPULIN (−2.4 vs. 0.4, P = 0.047). Other GV indices followed similar nonsignificant patterns of improvement with GLIPULIN. There were no differences in hypoglycemic events during CGM or arrhythmias during electrocardiographic monitoring. On-trial changes in body weight (−4.8 kg vs. +0.7 kg, $P < 0.001$), alanine aminotransferase (P = 0.0002), and serum amyloid A (P = 0.023) favored GLIPULIN.

CONCLUSIONS

GLIPULIN reduced GV, weight, and some cardiometabolic risk markers while maintaining equivalent A1C levels versus BBI and might improve clinical outcomes in a larger trial.

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Received 22 December 2015 and accepted 23 March 2016.

Clinical trial reg. no. NCT01524705, clinicaltrials.gov.

This article contains Supplementary Data online at http://care.diabetesjournals.org/lookup/suppl/doi:10.2337/dc15-2782/-/DC1.

*The members of the Writing Committee of the FLAT-SUGAR Trial are listed in the APPENDIX.

Diabetes Care Publish Ahead of Print, published online April 19, 2016

Assessment 2 – Hemoglobin A1C

If HbA1C is above 5.3%:

1. Exercise – skeletal muscle contraction + steady state cardio
2. Diet – macronutrient ratio, quantity of food
   * Paleo-Mediterranean
3. Supplementation - Same supplements as Glycomark protocol
   * Berberine
   * Gymnema
   * Banaba leaf
   * Bitter melon
   * Alpha-lipoic acid
   * Chromium
   * Vanadium

Assessment 2 – Hemoglobin A1C

If HbAIC is above 5.7%:

1. Exercise – skeletal muscle contraction + steady state cardio
2. Diet – macronutrient ratio, quantity of food
   * Paleo-Mediterranean
3. Supplementation - Same supplements as Glycomark protocol
   * Lipotropic factors – choline, methionine, TMG

Assessment 3 – C-Peptide

If C-peptide is above 2.5:

1. Mitochondrial support
   * Glutathione
   * NAC
   * ALA
   * Carnitine

If C-peptide is below 1.1 + elevated A1C:

1. GABA

Application 1: Suppress Glucagon

* Gamma amino butyric acid (GABA)
  * With meals

*****CLINICAL KEY**

Intra-islet insulin suppresses glucagon release via GABA-GABA$_A$ receptor system

Elaine Xu,[1,2,7] Mohan Kumar,[1,2,7] Yi Zhang,[1,2] William Ju,[1,2] Toshiyuki Obata,[3] Nina Zhang,[2] Shiying Liu,[2] Anna Wendt,[4] Shaoping Deng,[5] Yousuke Ebina,[3] Michael B. Wheeler,[1] Matthias Braun,[4,6] and Qinghua Wang[1,2,*]

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The intra-islet action of insulin is essential to exert the effect of glucose on the alpha cells since, in the absence of insulin, glucose is not able to suppress glucagon release in vivo. However, the precise mechanism by which insulin suppresses glucagon secretion from alpha cells is unknown.

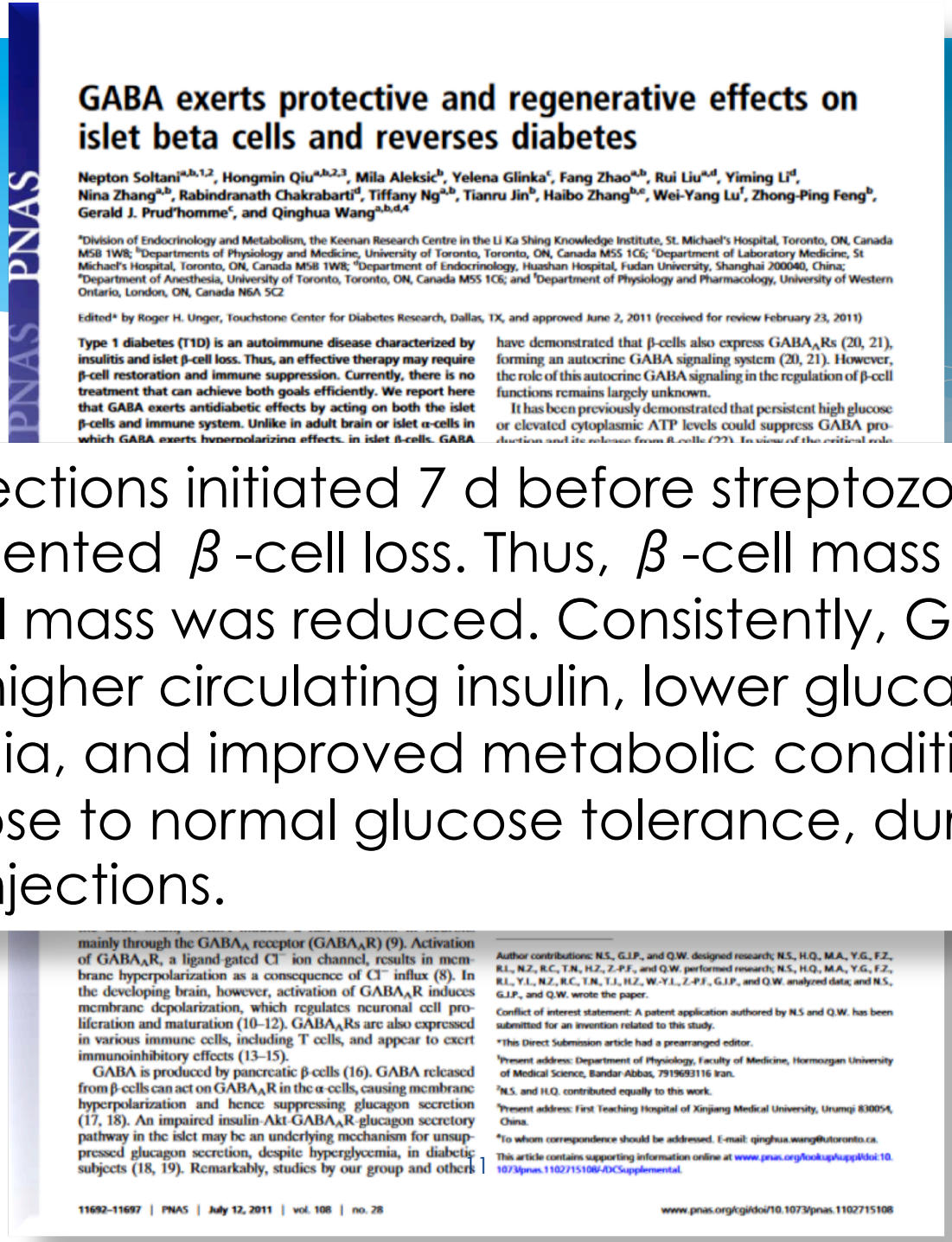
In this study, we show that insulin induces activation of GABAA receptors in the alpha cells by receptor translocation via an Akt kinase-dependent pathway. This leads to membrane hyperpolarization in the alpha cells and, ultimately, suppression of glucagon secretion. We propose that defects in this pathway(s) contribute to diabetic hyperglycemia.

quate understanding of the mechanisms underlying suppression of glucagon by insulin in response to hyperglycemia.

Secretion of glucagon from α cells is regulated by various factors, including glucose, zinc, and the chemical transmitter γ-aminobutyric acid (GABA) (Pipeleers et al., 1985; Ishihara

(Rorsman et al., 1989). The failure to detect an increase in GABA release does not exclude the possibility that there is an increase in the responsiveness of GABA$_A$Rs on α cells upon hyperglycemia; however a clear-cut mechanism has not been delineated.

CELL METABOLISM 3, 47–58, JANUARY 2006 ©2006 ELSEVIER INC. DOI 10.1016/j.cmet.2005.11.015 47



GABA exerts protective and regenerative effects on islet beta cells and reverses diabetes

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Edited* by Roger H. Unger, Touchstone Center for Diabetes Research, Dallas, TX, and approved June 2, 2011 (received for review February 23, 2011)

Type 1 diabetes (T1D) is an autoimmune disease characterized by insulitis and islet β-cell loss. Thus, an effective therapy may require β-cell restoration and immune suppression. Currently, there is no treatment that can achieve both goals efficiently. We report here that GABA exerts antidiabetic effects by acting on both the islet β-cells and immune system. Unlike in adult brain or islet α-cells in which GABA exerts hyperpolarizing effects, in islet β-cells, GABA

have demonstrated that β-cells also express $GABA_AR$s (20, 21), forming an autocrine GABA signaling system (20, 21). However, the role of this autocrine GABA signaling in the regulation of β-cell functions remains largely unknown.

It has been previously demonstrated that persistent high glucose or elevated cytoplasmic ATP levels could suppress GABA pro-

Daily GABA injections initiated 7 d before streptozotocin (STZ) treatment prevented β-cell loss. Thus, β-cell mass was preserved, whereas α-cell mass was reduced. Consistently, GABA-treated mice showed higher circulating insulin, lower glucagon, nearly normal glycemia, and improved metabolic conditions, and maintained close to normal glucose tolerance, during a period of 53 d after STZ injections.

mainly through the $GABA_A$ receptor ($GABA_AR$) (9). Activation of $GABA_AR$, a ligand-gated Cl^- ion channel, results in membrane hyperpolarization as a consequence of Cl^- influx (8). In the developing brain, however, activation of $GABA_AR$ induces membrane depolarization, which regulates neuronal cell proliferation and maturation (10–12). $GABA_AR$s are also expressed in various immune cells, including T cells, and appear to exert immunoinhibitory effects (13–15).

GABA is produced by pancreatic β-cells (16). GABA released from β-cells can act on $GABA_AR$ in the α-cells, causing membrane hyperpolarization and hence suppressing glucagon secretion (17, 18). An impaired insulin-Akt-$GABA_AR$-glucagon secretory pathway in the islet may be an underlying mechanism for unsuppressed glucagon secretion, despite hyperglycemia, in diabetic subjects (18, 19). Remarkably, studies by our group and others

Author contributions: N.S., G.J.P., and Q.W. designed research; N.S., H.Q., M.A., Y.G., F.Z., R.L., N.Z., R.C., T.N., H.Z., Z.-P.F., and Q.W. performed research; N.S., H.Q., M.A., Y.G., F.Z., R.L., Y.L., N.Z., R.C., T.N., T.J., H.Z., W.-Y.L., Z.-P.F., G.J.P., and Q.W. analyzed data; and N.S., G.J.P., and Q.W. wrote the paper.

Conflict of interest statement: A patent application authored by N.S and Q.W. has been submitted for an invention related to this study.

*This Direct Submission article had a prearranged editor.

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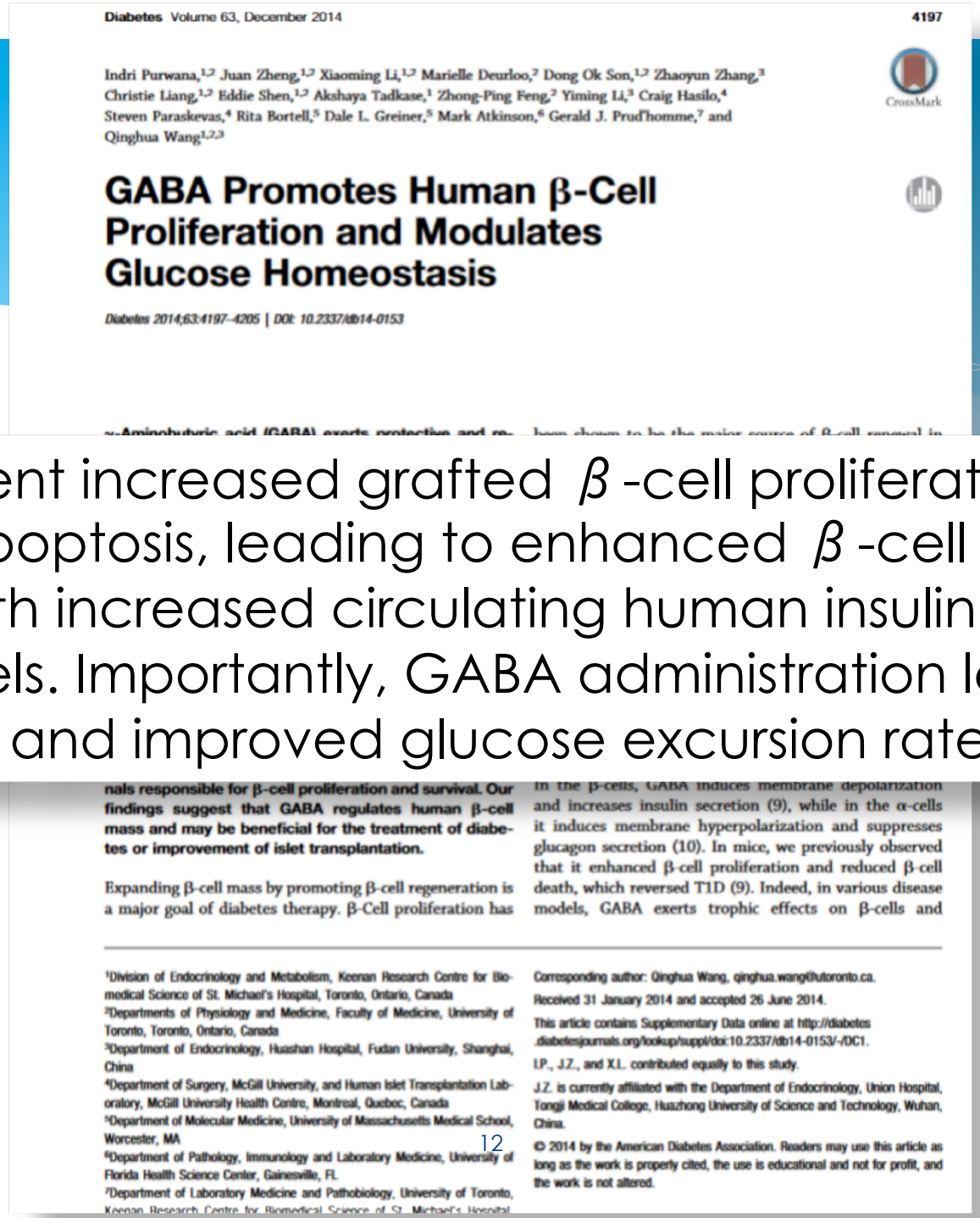
This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1102715108/-/DCSupplemental.



Diabetes Volume 63, December 2014 4197

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GABA Promotes Human β-Cell Proliferation and Modulates Glucose Homeostasis

Diabetes 2014;63:4197–4205 | DOI: 10.2337/db14-0153

γ-Aminobutyric acid (GABA) exerts protective and re- ... been shown to be the major source of β-cell renewal in

nals responsible for β-cell proliferation and survival. Our findings suggest that GABA regulates human β-cell mass and may be beneficial for the treatment of diabetes or improvement of islet transplantation.

Expanding β-cell mass by promoting β-cell regeneration is a major goal of diabetes therapy. β-Cell proliferation has

In the β-cells, GABA induces membrane depolarization and increases insulin secretion (9), while in the α-cells it induces membrane hyperpolarization and suppresses glucagon secretion (10). In mice, we previously observed that it enhanced β-cell proliferation and reduced β-cell death, which reversed T1D (9). Indeed, in various disease models, GABA exerts trophic effects on β-cells and

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Received 31 January 2014 and accepted 26 June 2014.

This article contains Supplementary Data online at http://diabetes.diabetesjournals.org/lookup/suppl/doi:10.2337/db14-0153/-/DC1.

I.P., J.Z., and X.L. contributed equally to this study.

J.Z. is currently affiliated with the Department of Endocrinology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China.

GABA treatment increased grafted β-cell proliferation, while decreasing apoptosis, leading to enhanced β-cell mass. This was associated with increased circulating human insulin and reduced glucagon levels. Importantly, GABA administration lowered blood glucose levels and improved glucose excursion rates.

Application 2: Antioxidant Support

* Comprehensive Antioxidant Protocol
  1. BioGmax Series Antioxidants (GSH, C c/RLA, Resveratrol)
  2. Glucostatic Balance
  3. Effecsulin

*****CLINICAL KEY**

Insulin resistance is a cellular antioxidant defense mechanism

Kyle L. Hoehn[a,1,2,3], Adam B. Salmon[b,1], Cordula Hohnen-Behrens[a], Nigel Turner[a], Andrew J. Hoy[a], Ghassan J. Maghzal[c], Roland Stocker[c], Holly Van Remmen[b], Edward W. Kraegen[a], Greg J. Cooney[a], Arlan R. Richardson[b], and David E. James[a,2]

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Edited by Michael Karin, University of California, San Diego School of Medicine, La Jolla, CA, and approved August 28, 2009 (received for review March 4, 2009)

Mitochondrial $O_2^{\bullet -}$ has previously been linked to hyperglycemia-induced metabolic dysfunction in endothelial cell systems and in inflammation in adipocytes.

A major advance of the present study is the observation that mitochondrial $O_2^{\bullet -}$ is upstream of IR in skeletal muscle and adipose tissue.

The origin of IR has been difficult to elucidate in part due to the diverse set of risk factors linked to this condition including overnutrition, physical inactivity, pregnancy, Hepatitis C, polycystic ovarian syndrome, HIV protease inhibitor therapy, and antiinflammatory corticosteroids. Do such factors converge at a common intermediate in the insulin action pathway or does IR represent a collection of distinct cellular disorders? For example, endoplasmic reticulum (ER) stress, proinflammatory responses, oxidative stress, intracellular ceramide accumulation, or the activation of JNK, IKK, or PKC are all currently implicated in the development of IR in overnourished or obese rodents (2, 3). In such models, correcting any one of these intracellular stresses is sufficient to improve IR leading to the possibility that these factors are somehow interconnected. One view is that insulin receptor substrate 1 (IRS1) represents a common convergence point for many defects contributing to IR (4). However, this view has been challenged in that the ability of IRS1-independent receptor tyrosine kinases to activate

described a reproducible system for studying IR in myotubes and adipocytes in culture relying on the translocation of the facilitative glucose transporter GLUT4 to the plasma membrane (5). This

Author contributions: K.L.H., A.B.S., and D.E.J. designed research; K.L.H., A.B.S., C.H.-B., N.T., A.J.H., and G.J.M. performed research; K.L.H., A.B.S., R.S., H.V.R., E.W.K., G.J.C., and A.R.R. contributed new reagents/analytic tools; K.L.H., A.B.S., and C.H.-B. analyzed data; and K.L.H. and D.E.J. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at www.pnas.org/cgi/content/full/0902380106/DCSupplemental.

www.pnas.org/cgi/doi/10.1073/pnas.0902380106 PNAS | October 20, 2009 | vol. 106 | no. 42 | 17787–17792

CELL BIOLOGY

Insulin resistance is a cellular antioxidant defense mechanism

Kyle L. Hoehn[a,1,2,3], Adam B. Salmon[b,1], Cordula Hohnen-Behrens[a], Nigel Turner[a], Andrew J. Hoy[a], Ghassan J. Maghzal[c], Roland Stocker[c], Holly Van Remmen[b], Edward W. Kraegen[a], Greg J. Cooney[a], Arlan R. Richardson[b], and David E. James[a,2]

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Edited by Michael Karin, University of California, San Diego School of Medicine, La Jolla, CA, and approved August 28, 2009 (received for review March 4, 2009)

This hypothesis is exciting for a number of reasons. For example, it suggests that IR may be a protective mechanism, in which case we should perhaps reconsider using therapeutic strategies to overcome unless they also eliminate the primary defect. Moreover, it suggests that cells have evolved sophisticated mechanisms to not only guard against nutrient lack, such as the AMPK pathway, but also nutrient excess.

The origin of IR has been difficult to elucidate in part due to the diverse set of risk factors linked to this condition including overnutrition, physical inactivity, pregnancy, Hepatitis C, polycystic ovarian syndrome, HIV protease inhibitor therapy, and antiinflammatory corticosteroids. Do such factors converge at a common intermediate in the insulin action pathway or does IR represent a collection of distinct cellular disorders? For example, endoplasmic reticulum (ER) stress, proinflammatory responses, oxidative stress, intracellular ceramide accumulation, or the activation of JNK, IKK, or PKC are all currently implicated in the development of IR in overnourished or obese rodents (2, 3). In such models, correcting any one of these intracellular stresses is sufficient to improve IR leading to the possibility that these factors are somehow interconnected. One view is that insulin receptor substrate 1 (IRS1) represents a common convergence point for many defects contributing to IR (4). However, this view has been challenged in that the ability of IRS1-independent receptor tyrosine kinases to activate

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The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at www.pnas.org/cgi/content/full/0902380106/DCSupplemental.

www.pnas.org/cgi/doi/10.1073/pnas.0902380106 PNAS | October 20, 2009 | vol. 106 | no. 42 | 17787–17792

CELL BIOLOGY

Diabetes Volume 64, March 2015 673

Christopher J. Nolan,[1] Neil B. Ruderman,[2] Steven E. Kahn,[3] Oluf Pedersen,[4] and Marc Prentki[5]

Insulin Resistance as a Physiological Defense Against Metabolic Stress: Implications for the Management of Subsets of Type 2 Diabetes

The β-cell is particularly vulnerable to glucolipotoxicity. Other tissues, such as heart and skeletal muscle, that express the insulin-regulated glucose transporter GLUT4 have the capacity to protect themselves from glucolipotoxicity by developing IR, which restrains glucose entry into cells and therefore the glucose arm of this potentially damaging process.

T2D. Potential molecular mechanisms underlying these concepts; their clinical implications for stratification of T2D management, particularly in overweight and obese patients with difficult glycemic control; and future research requirements are discussed.

high-dose insulin therapy) will cause them harm. We believe that the concept of "insulin-induced metabolic stress" provides a plausible explanation for many of the unexpected outcomes of major T2D clinical trials. The important implications of this concept for ongoing

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[4]Novo Nordisk Foundation Center for Basic Metabolic Research, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark
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Received 1 May 2014 and accepted 9 October 2014.

	Hypoglycemia	Early IR	Insulin Resistance	Late IR	Type II Diabetes
Glucose	↓	Normal	↑	↑↑	↑↑↑
C-Peptide	↑/N	↑	↑↑	Normal	↓
HA1C	Normal/Low	Normal	↑	↑↑	↑↑↑
Cholesterol/ Triglyceride	Normal	Normal	Less than 2:1	Less than 2:1	Less than 2:1
LDH	↓	NA	NA	NA	NA

Way Back When…

* Insulin Resistance, Reverse it, organize it, focus on it, DO WHATEVER YOU CAN!
* Research as changed the paradigm.
* OUT FRONT - clinician vs technician.

How about 1.5?

At first, LADA can be managed by controlling your blood sugar with diet, weight reduction if appropriate, exercise and, possibly, oral medications. But as your body gradually loses its ability to produce insulin, insulin shots will eventually be needed. - Mayo Clinic

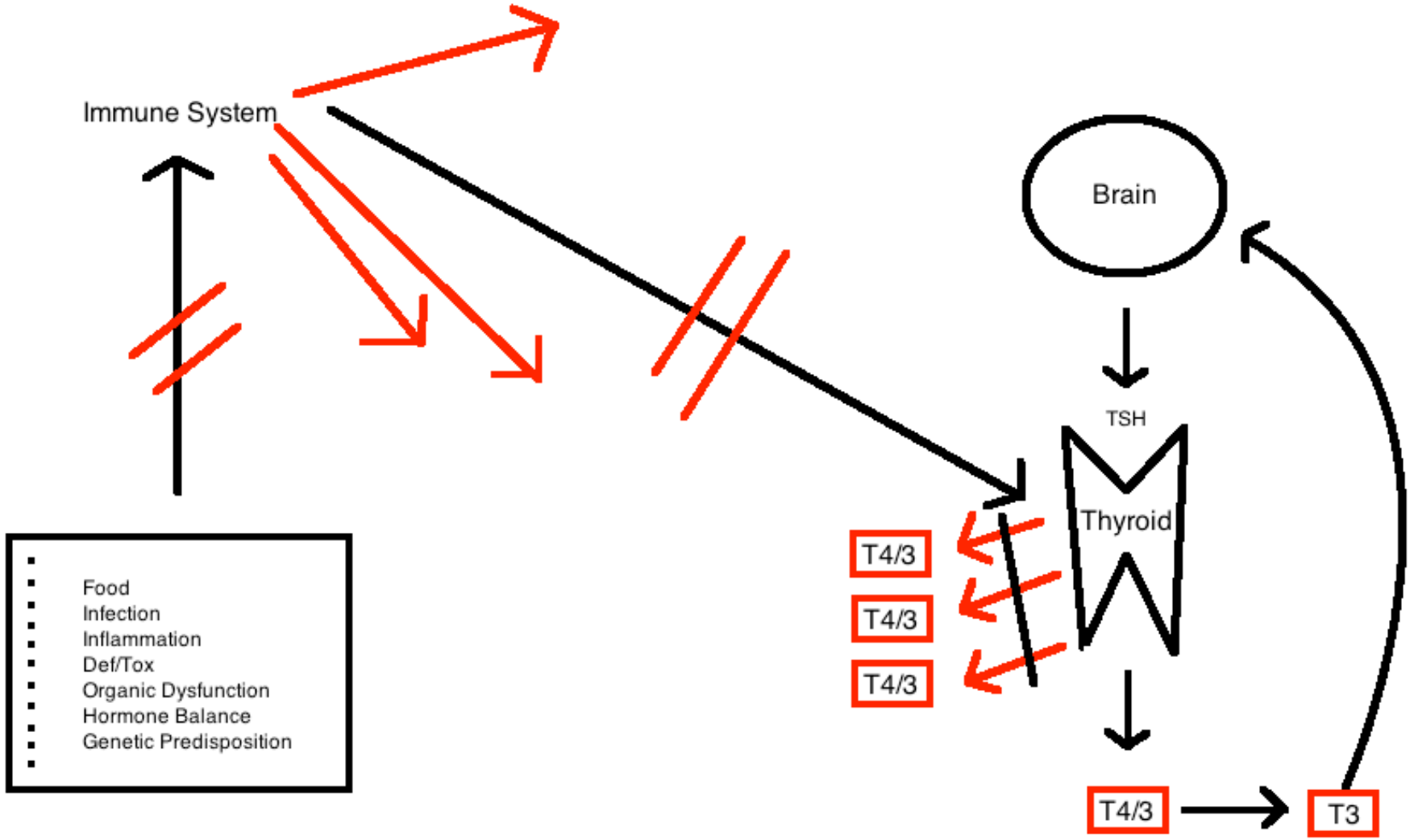
Antibodies

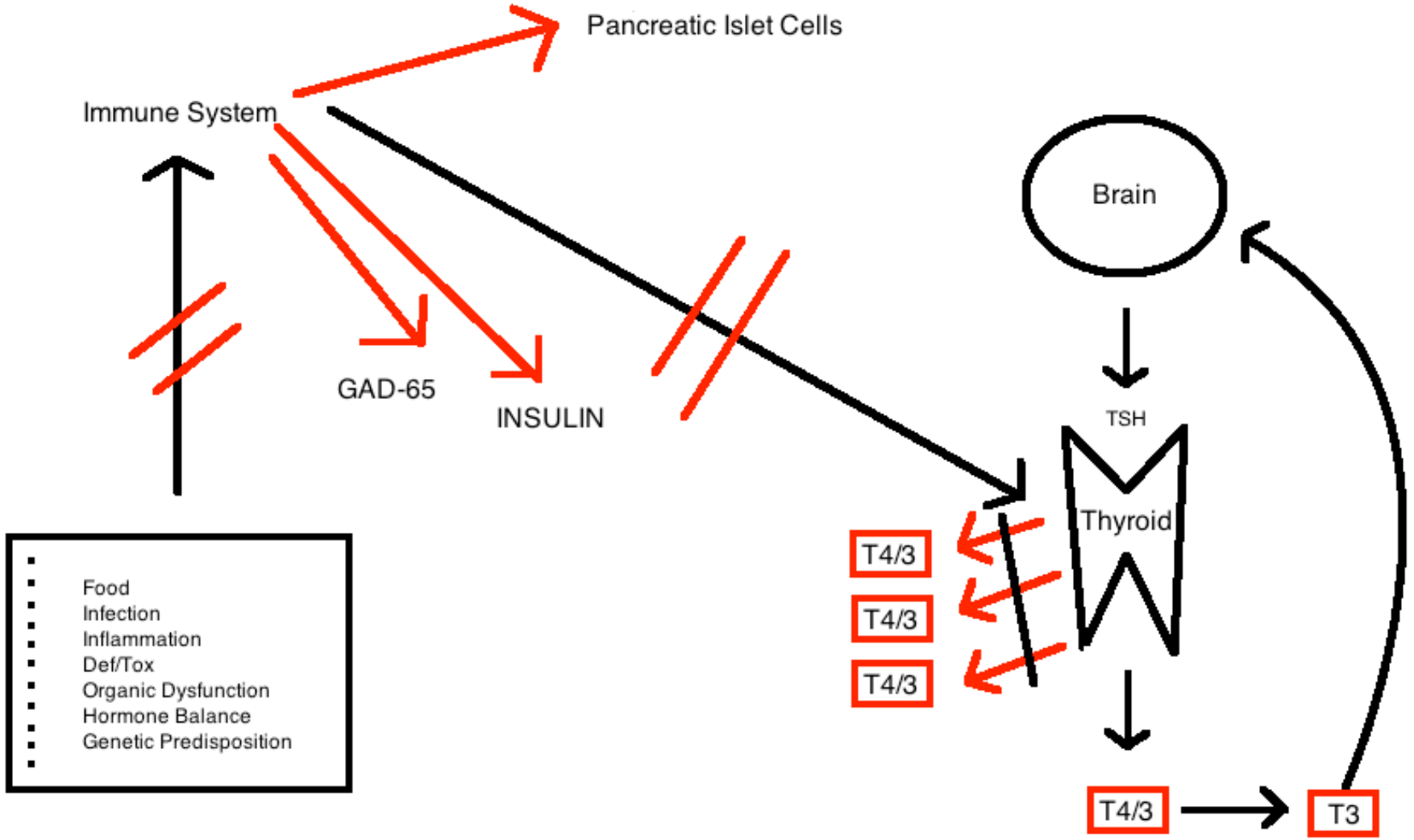
* Glutamic Acid Decarboxylase - 65 Ab (GAD-65 Ab)
* Insulin Ab
* Pancreatic Islet Cell Ab

Journal of Diabetic Medicine:

1.Latent autoimmune diabetes of the adult (LADA) is an autoimmune diabetes defined by adult-onset, presence of diabetes associated autoantibodies, and no insulin treatment requirement for a period after diagnosis.
2. Immunologically, glutamic acid decarboxylase 65 autoantibodies are by far the most common autoantibody in adult-onset diabetes.
3. LADA is the most prevalent form of adult-onset autoimmune diabetes and probably the most prevalent form of autoimmune diabetes in general.
4. LADA shares genetic features with both type 1 and type 2 diabetes.
Phenotypically, LADA patients are often misdiagnosed as having type 2 diabetes.
5. LADA patients generally have worse HbA1c levels than type 2 diabetes patients.
Clinically, LADA patients tend to have a lower mean age at diabetes onset, lower body mass index and more frequent need for insulin treatment than patients with type 2 diabetes.
6. Management of LADA **may require a dedicated strategy**, yet currently there is a paucity of randomized controlled trial data.

Diabet Med. 2015 Jul; 32(7): 843–852.
Published online 2015 Feb 7. doi: 10.1111/dme.12700


Immune System
Brain
TSH
Thyroid
T4/3
T4/3
T4/3
T4/3
T3
Food
Infection
Inflammation
Def/Tox
Organic Dysfunction
Hormone Balance
Genetic Predisposition


Pancreatic Islet Cells
Immune System
GAD-65
INSULIN
Brain
TSH
Thyroid
T4/3
T4/3
T4/3
T4/3
T3
Food
Infection
Inflammation
Def/Tox
Organic Dysfunction
Hormone Balance
Genetic Predisposition

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