Update on prediction, prevention and clinical trials in type 1 diabetes

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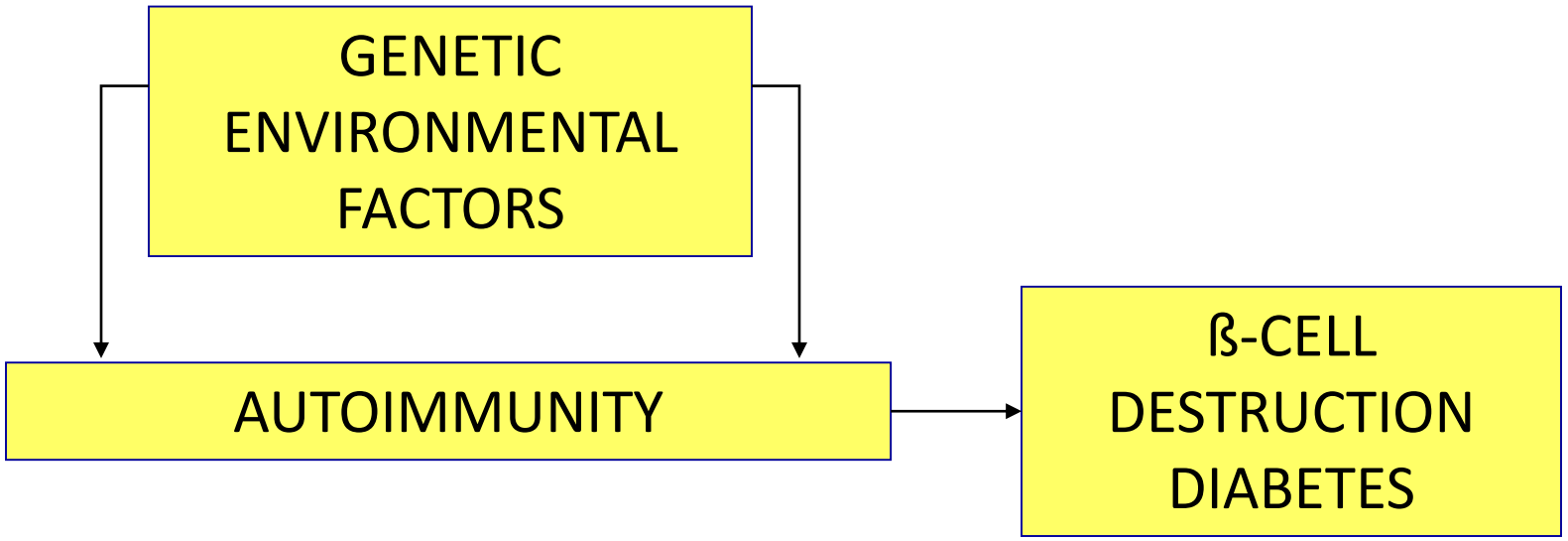
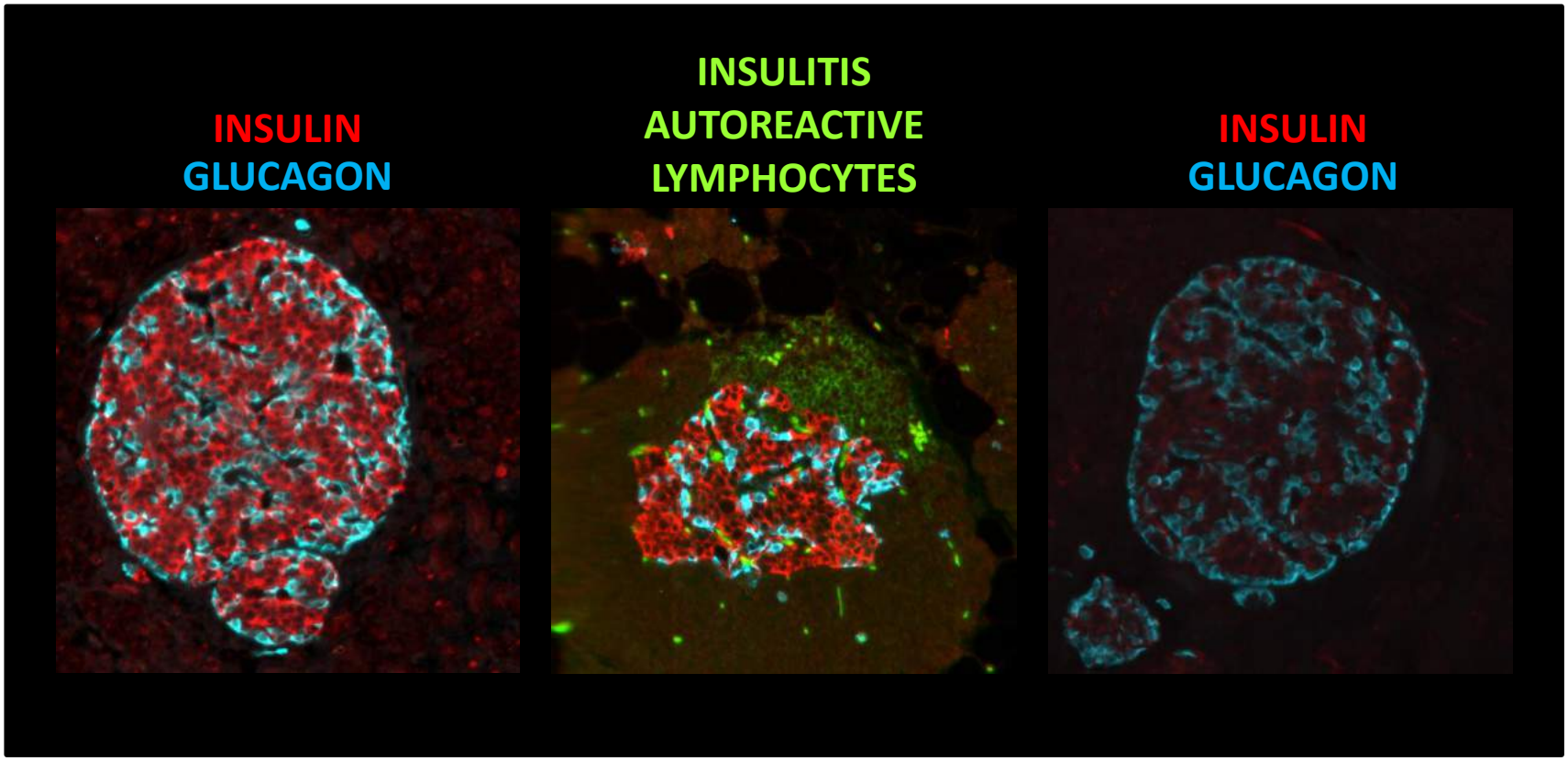
I do not have any financial relationships relative to the content of this program.

Alberto Pugliese

Type 1 Diabetes (T1D)

- Multifactorial, organ-specific, autoimmune disease
- Leads to the destruction of insulin-producing ß-cells in the pancreas, followed by hyperglycemia and virtually complete insulin deficiency
- Typical onset in childhood/young adulthood, but can occur at any age
- Relatively common: 1/300; ~ 15,000 new cases each year with ~1 million people affected in the US, but incidence varies among races and ethnic groups
- Incidence rising in many countries; non-linear rise recently reported; becoming more frequent in younger children

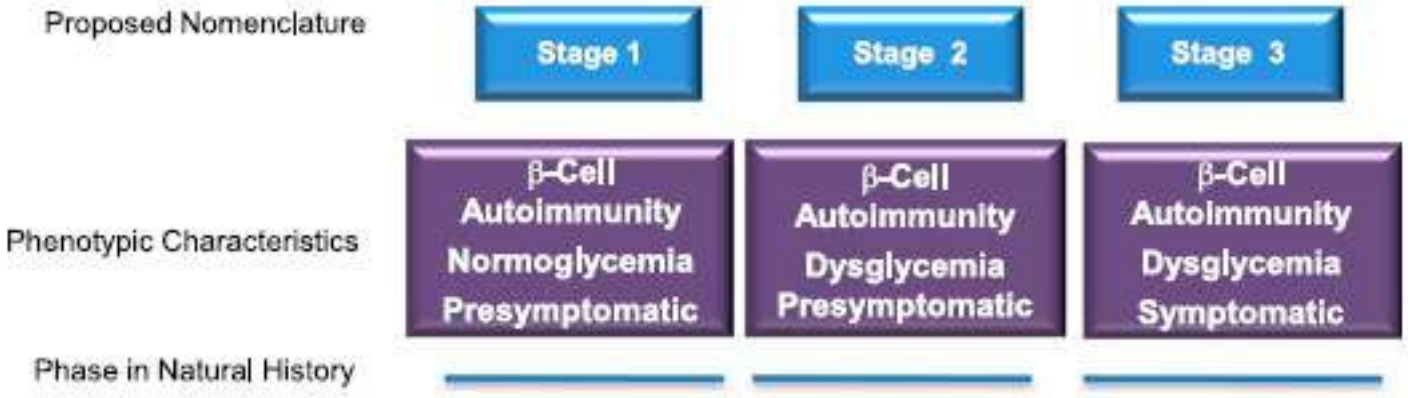


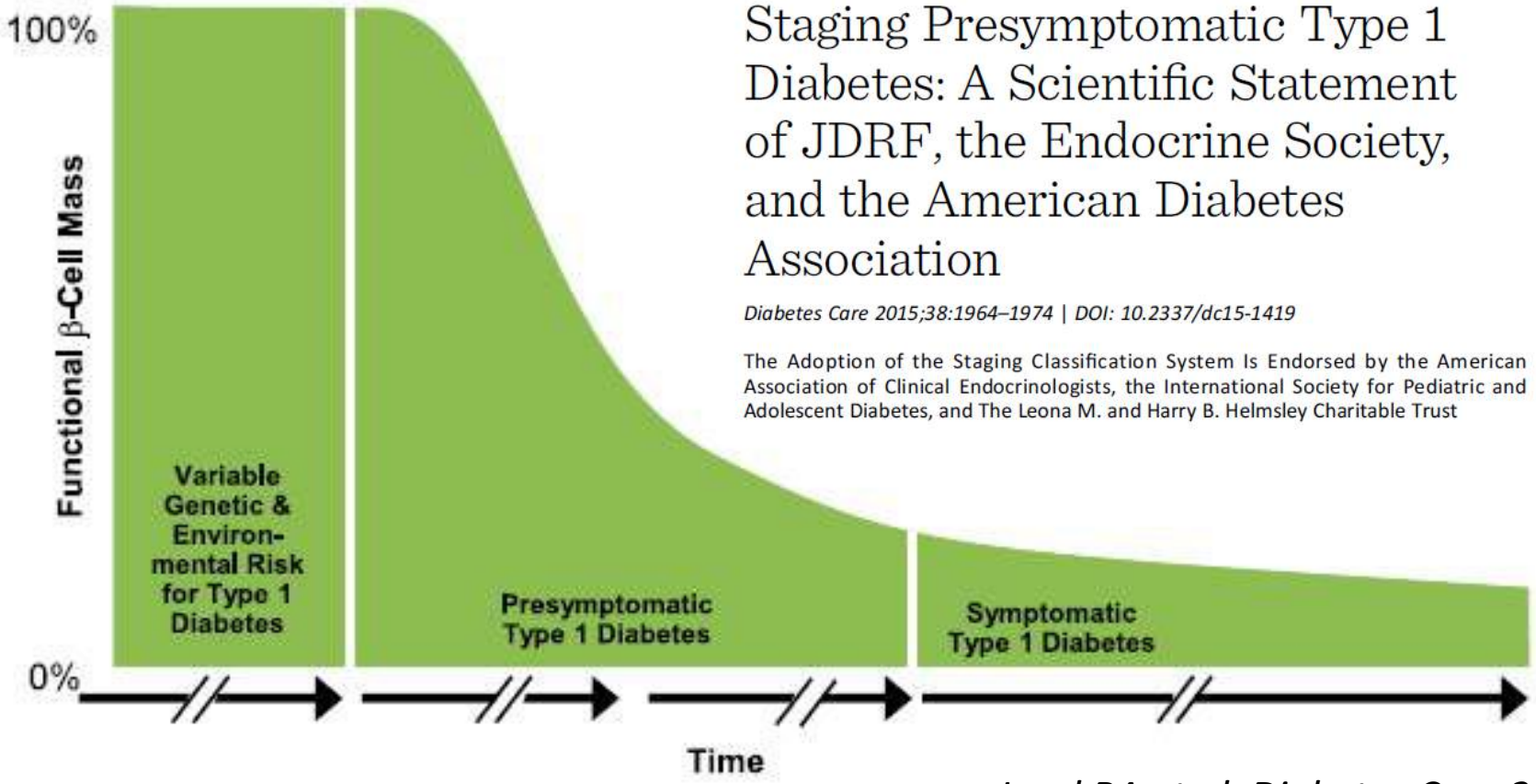


NORMAL

PREDIABETES & RECENT ONSET

ESTABLISHED DIABETES





Staging Presymptomatic Type 1 Diabetes: A Scientific Statement of JDRF, the Endocrine Society, and the American Diabetes Association

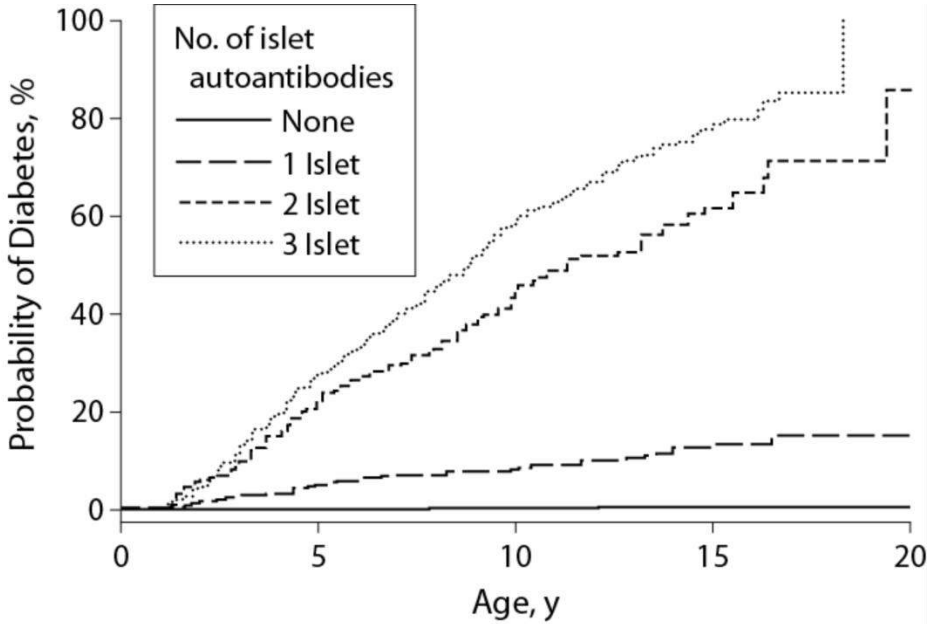
Diabetes Care 2015;38:1964–1974 | DOI: 10.2337/dc15-1419

The Adoption of the Staging Classification System Is Endorsed by the American Association of Clinical Endocrinologists, the International Society for Pediatric and Adolescent Diabetes, and The Leona M. and Harry B. Helmsley Charitable Trust

Insel RA et al. Diabetes Care 2015

From: **Seroconversion to Multiple Islet Autoantibodies and Risk of Progression to Diabetes in Children**

JAMA. 2013;309(23):2473-2479. doi:10.1001/jama.2013.6285



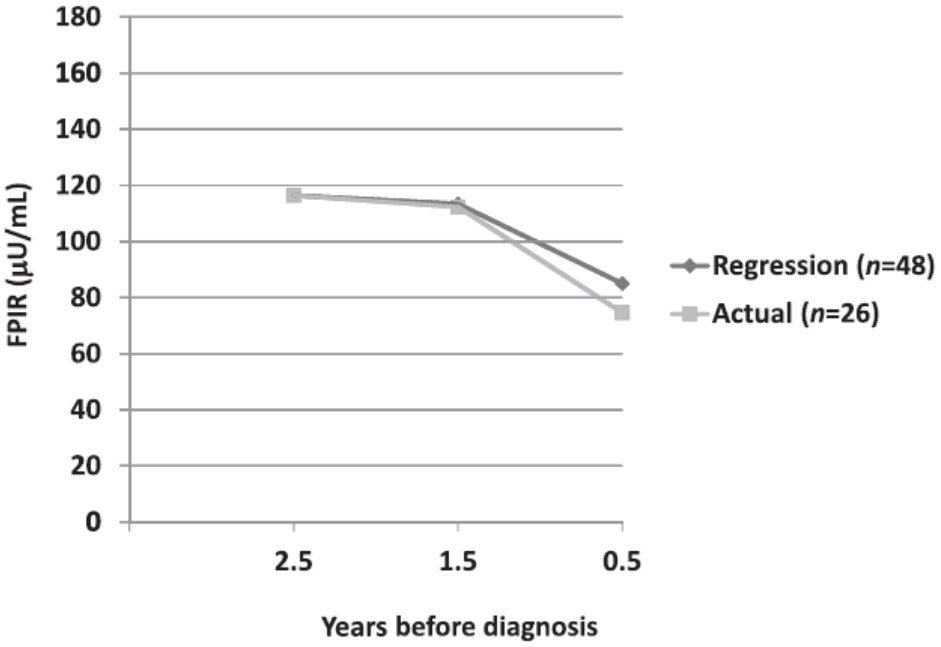
No. at risk Islet autoantibodies, No.					
3 Islet	358	250	112	20	
2 Islet	227	168	82	19	1
1 Islet	474	430	272	118	9
None	12318	8875	5253	1161	44

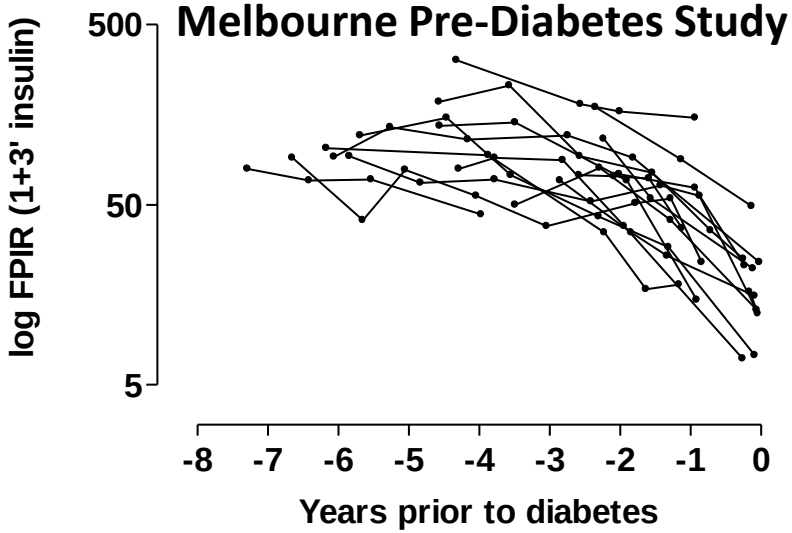
Figure Legend:

The numbers at risk represent the children receiving follow-up at age 0, 5, 10, 15, and 20 years.

Acceleration of the Loss of the First-Phase Insulin Response During the Progression to Type 1 Diabetes in Diabetes Prevention Trial–Type 1 Participants

Jay M. Sosenko,[1] Jay S. Skyler,[1] Craig A. Beam,[2] Jeffrey P. Krischer,[2] Carla J. Greenbaum,[3] Jeffrey Mahon,[4] Lisa E. Rafkin,[1] Della Matheson,[1] Kevan C. Herold,[5] Jerry P. Palmer,[6] and the Type 1 Diabetes TrialNet and Diabetes Prevention Trial–Type 1 Study Groups*





Diabetes, 2013

The Metabolic Progression to Type 1 Diabetes as Indicated by Serial Oral Glucose Tolerance Testing in the Diabetes Prevention Trial–Type 1

Jay M. Sosenko,[1] Jay S. Skyler,[1] Kevan C. Herold,[2] Jerry P. Palmer,[3] and the Type 1 Diabetes TrialNet and Diabetes Prevention Trial–Type 1 Study Groups

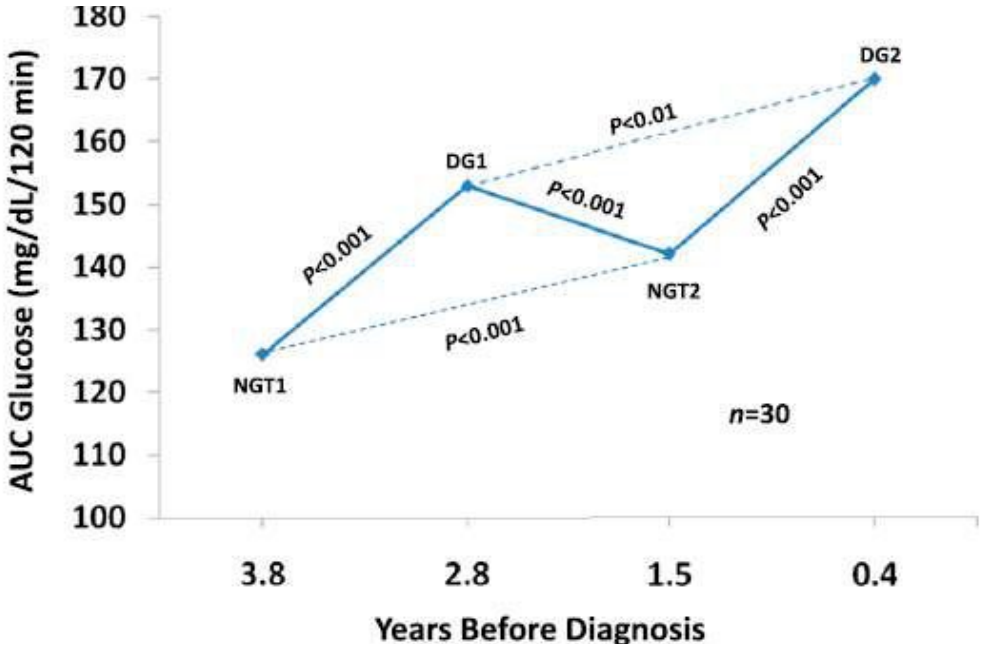
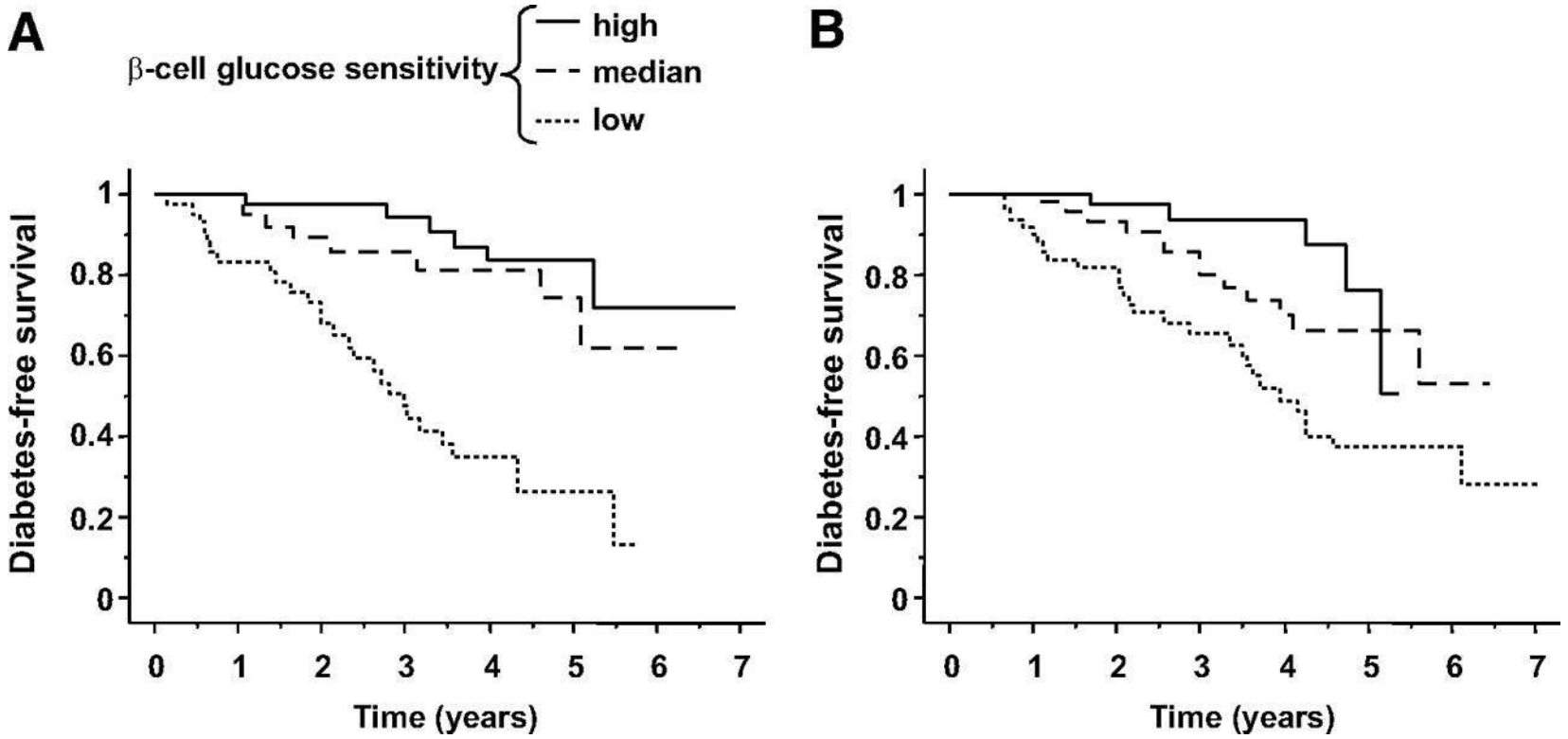


FIG. 4. Shown are excursions between normal glucose tolerance (NGT) and dysglycemic (DG) states in 30 progressors to T1D. Each point represents the mean AUC glucose from the OGTTs. The mean time before diagnosis is shown for the OGTTs. There were significant increases in the AUC glucose from each of the normal OGTTs to their subsequent respective dysglycemic OGTTs. There were also significant increases from the first normal OGTT to the second normal OGTT, and from the first dysglycemic OGTT to the second dysglycemic OGTT. (The dashed lines indicate differences in glucose levels after a return to the same state of glycemia.) (A high-quality color representation of this figure is available in the online issue.)

Diabetes, 2013

A reduction in Beta Cell glucose sensitivity precedes diagnosis and predicts progression to T1D



Kaplan-Meier plots of diabetes-free survival in 280 subjects with normal glucose tolerance at baseline according to tertile of baseline β-cell glucose sensitivity (log-rank $\chi 2 = 25.5$, $P < 0.0001$, and 13.2, $P = 0.0003$, in female [A] and male [B] subjects, respectively).

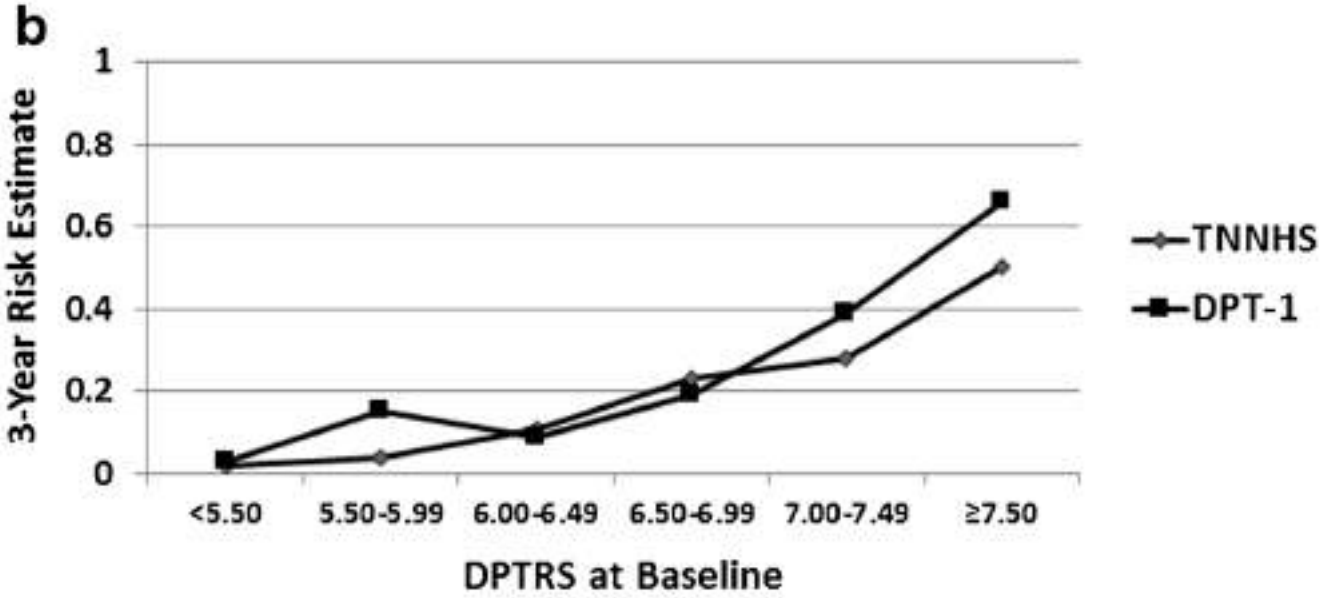
Ele Ferrannini et al. Diabetes 2010;59:679-685

American Diabetes Association.

Curr Diab Rep (2015) 15: 49
DOI 10.1007/s11892-015-0626-1

The Development, Validation, and Utility of the Diabetes Prevention Trial-Type 1 Risk Score (DPTRS)

Jay M. Sosenko[1] · Jay S. Skyler[1] · Jerry P. Palmer[2] ·
The Diabetes Type 1 TrialNet and Diabetes Prevention Trial-Type 1 Study Groups

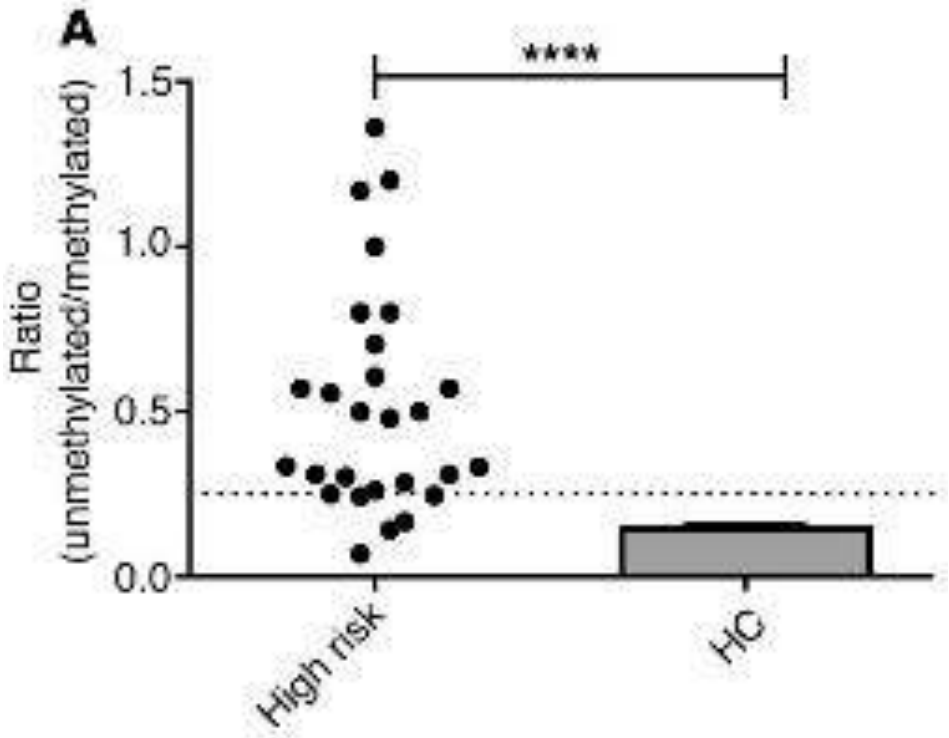


n for TNNHS	290	172	185	153	82	109
n for DPT-1	78	86	132	148	100	126

The Journal of Clinical Investigation 2015

β Cell death and dysfunction during type 1 diabetes development in at-risk individuals

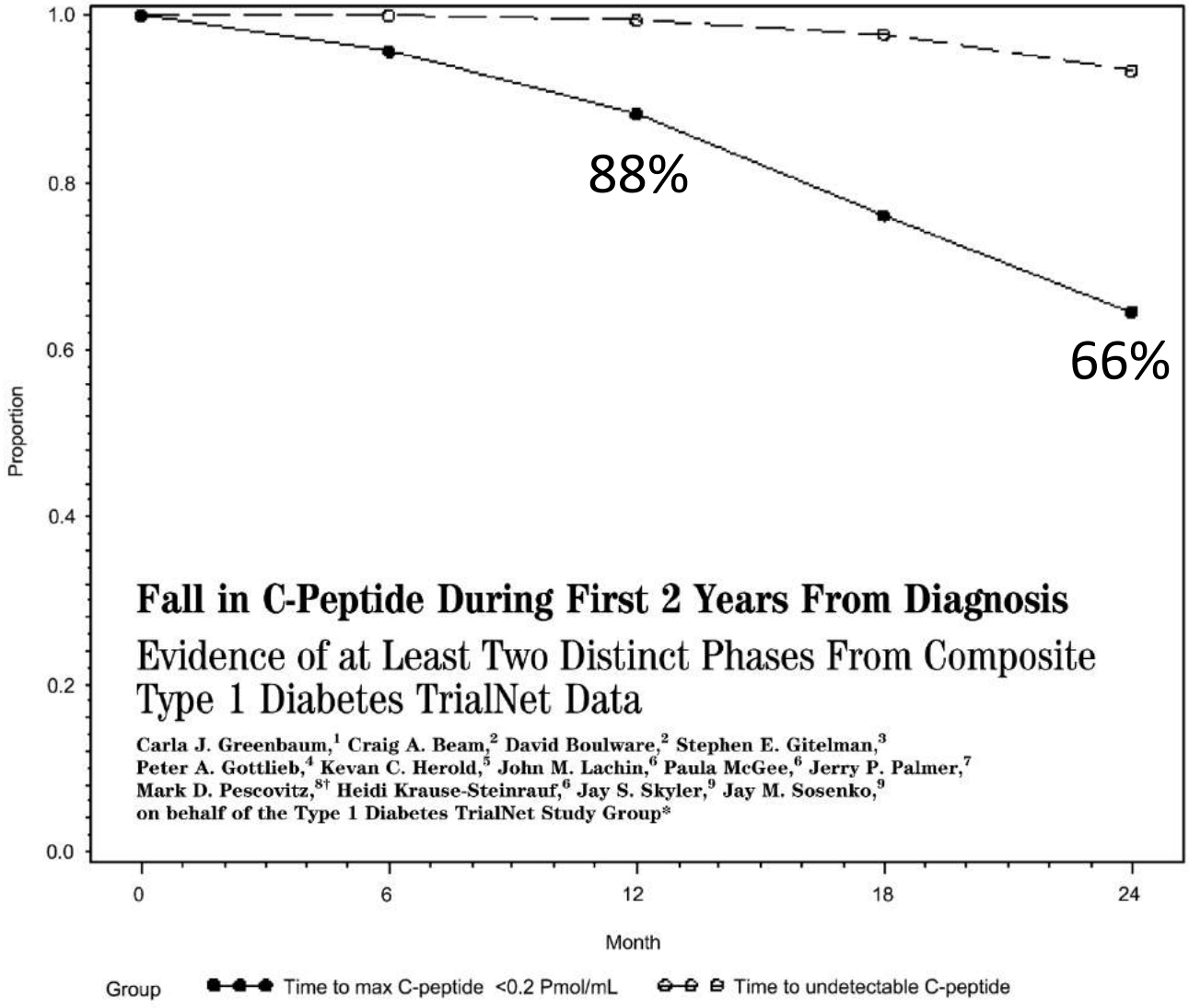
Kevan C. Herold,[1] Sahar Usmani-Brown,[2] Tara Ghazi,[1] Jasmin Lebastchi,[1] Craig A. Beam,[3] Melena D. Bellin,[4] Michel Ledizet,[2] Jay M. Sosenko,[5] Jeffrey P. Krischer,[6] Jerry P. Palmer,[7] and the Type 1 Diabetes TrialNet Study Group



Proportions of patients with peak stimulated C-peptide ≥0.2 pmol/mL during the first 2 years after diagnosis

Undetectable C-peptide
1 year 1%
2 years 7%

C-peptide ≥0.2 pmol/mL
1 year 88%
2 years 66%



Fall in C-Peptide During First 2 Years From Diagnosis

Evidence of at Least Two Distinct Phases From Composite Type 1 Diabetes TrialNet Data

Carla J. Greenbaum,[1] Craig A. Beam,[2] David Boulware,[2] Stephen E. Gitelman,[3] Peter A. Gottlieb,[4] Kevan C. Herold,[5] John M. Lachin,[6] Paula McGee,[6] Jerry P. Palmer,[7] Mark D. Pescovitz,[8†] Heidi Krause-Steinrauf,[6] Jay S. Skyler,[9] Jay M. Sosenko,[9] on behalf of the Type 1 Diabetes TrialNet Study Group*

FIG. 1. Percent of individuals with detectable C-peptide and C-peptide ≥0.2 pmol/mL over time.

Diabetes 2012

The majority of patients with long-duration type 1 diabetes are insulin microsecretors and have functioning beta cells

Richard A. Oram · Angus G. Jones · Rachel E. J. Besser · Bridget A. Knight · Beverley M. Shields · Richard J. Brown · Andrew T. Hattersley · Timothy J. McDonald

Diabetologia 2014

Red line marks the level above which patients are eligible for clinical trials at onset

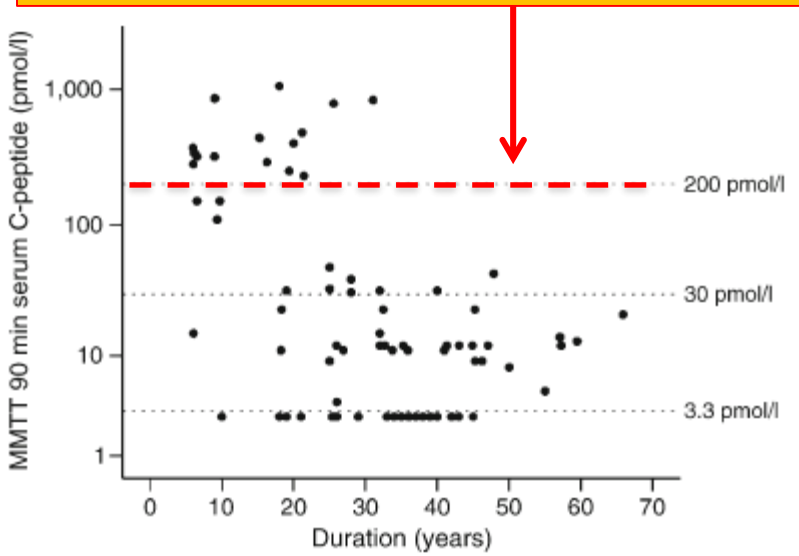


Fig. 1 Scatterplot of serum C-peptide at 90 min after a mixed meal ($\log_{10}$ scale) against duration of diabetes. Dotted reference lines indicate 200, 30 and 3.3 pmol/l. MMTT, mixed-meal tolerance test

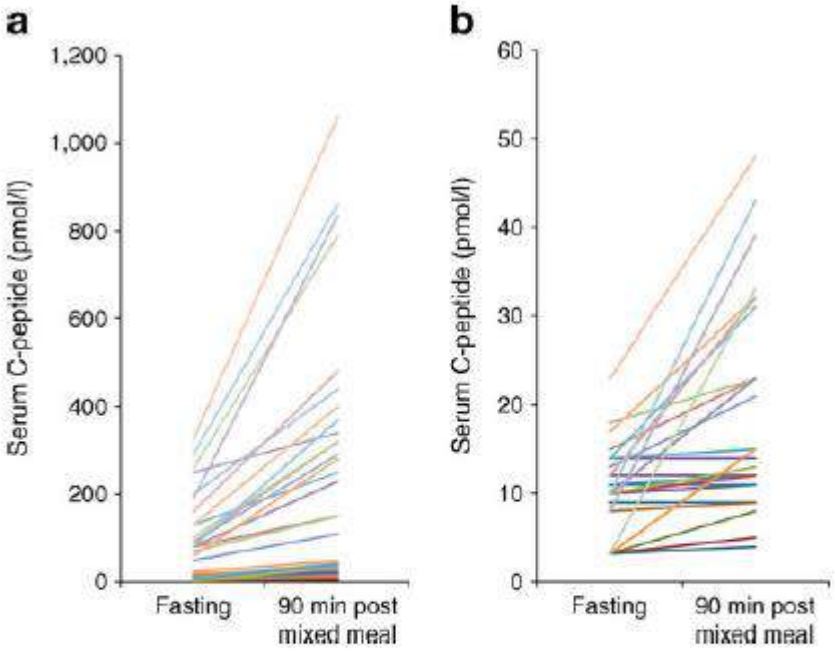
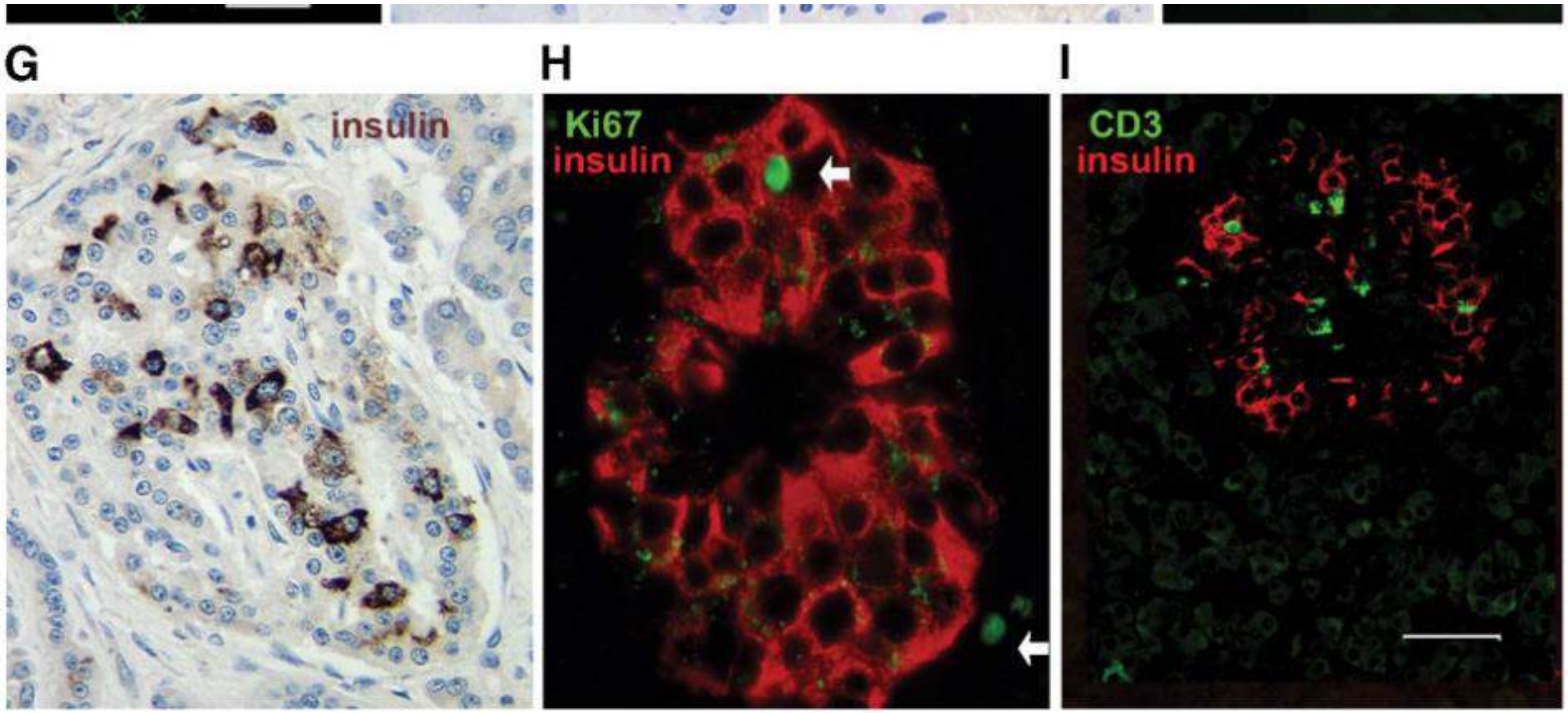


Fig. 2 The effect of a meal stimulus on serum C-peptide levels in participants with detectable insulin ($n=54$). (**a**) Paired fasting and mixed meal results for all patients with detectable C-peptide. Each line represents an individual patient. (**b**) Results for all patients with fasting C-peptide below 30 pmol/l ($n=36$). Of 54 patients, 34 (80%) had a serum C-peptide value that rose after the mixed meal. None had a fall in the C-peptide value after the meal

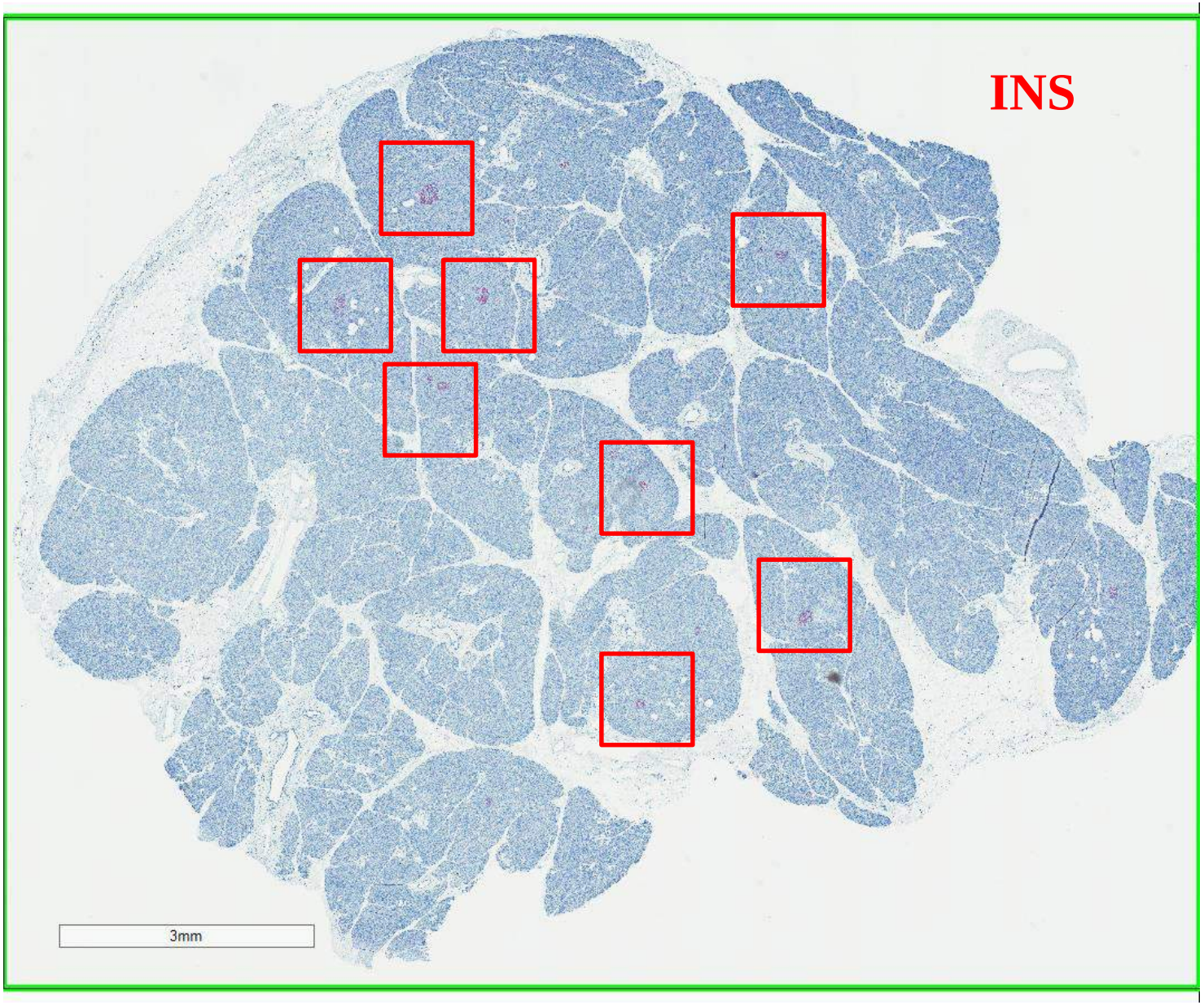
Residual Insulin Production and Pancreatic β-Cell Turnover After 50 Years of Diabetes: Joslin Medalist Study

Hillary A. Keenan,[1,2] Jennifer K. Sun,[1,3,4] Jared Levine,[1,2] Alessandro Doria,[1,2] Lloyd P. Aiello,[1,3,4] George Eisenbarth,[5] Susan Bonner-Weir,[1,2] and George L. King[1,2]

Diabetes 2010



Longer Duration T1D – 8 years



Longer Duration T1D

6046
18 years old (**8 year duration**) Caucasian Female

AutoAb: IA2A+ZnT8+

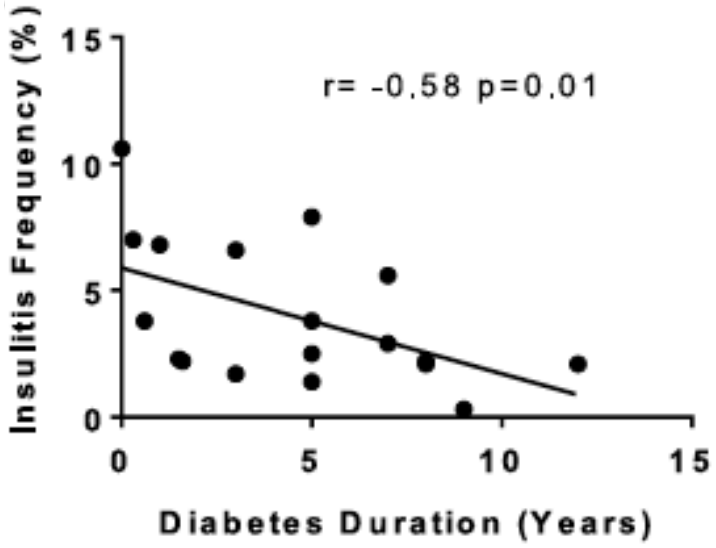
C peptide: <0.05 ng/ml
BMI: 25.2

Histopathology : Ins+ islets in some lobules, other lobules/entire blocks ins-/gluc+ islets. Insulitis +. CD3+ or CD45+ used. Also infiltrates are mainly acinar/extra-acinar. Mild acinar atrophy and adipose infiltration.

HLA: A*0201/0301
B*1501/3901
DRB1*0101/0401
DQA1*0101/0301
DQB1*0302/0501

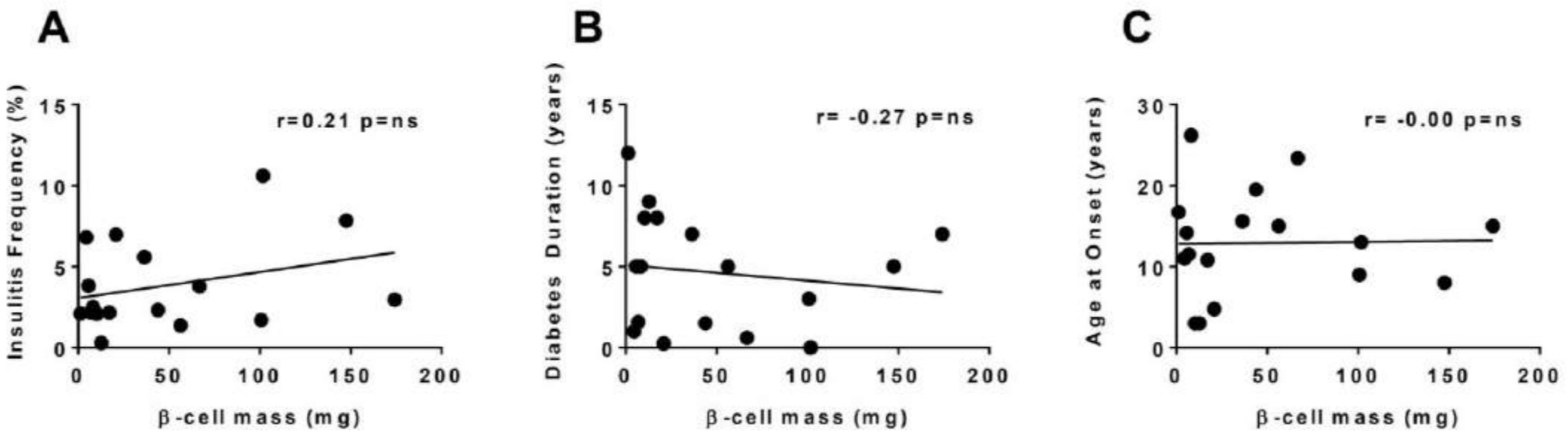
Low frequency of insulitis and modest correlation with disease duration

(T1D nPOD donors with insulitis; n=18)



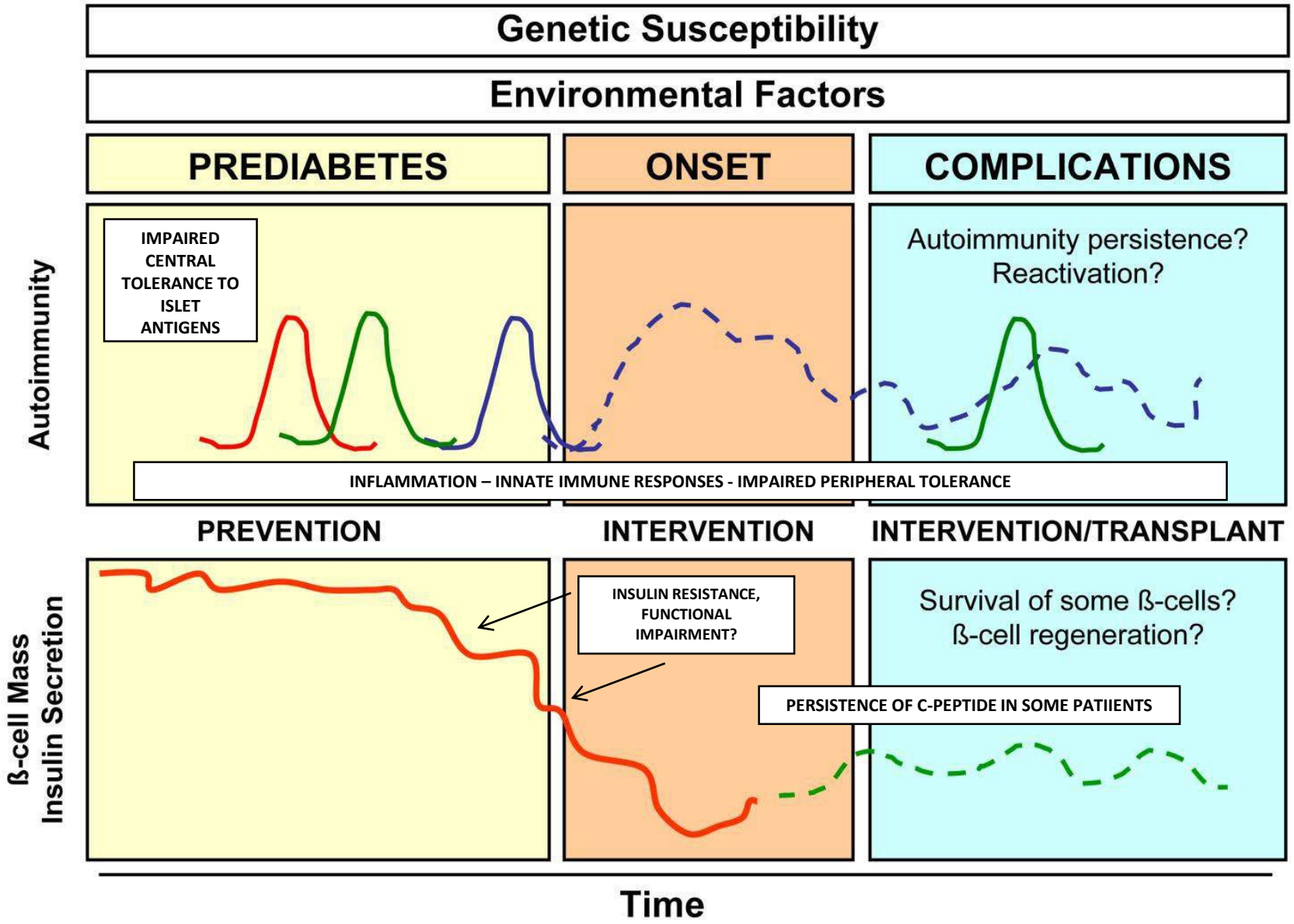
Campbell-Thompson M. et al., Diabetes, 2016

Frequency of insulitis, T1D duration, and age of onset show no significant correlation with beta cell mass



Campbell-Thompson M. et al., Diabetes, 2016

Type 1 Diabetes: a Chronic Autoimmune Disease



Patient - SPK-3601

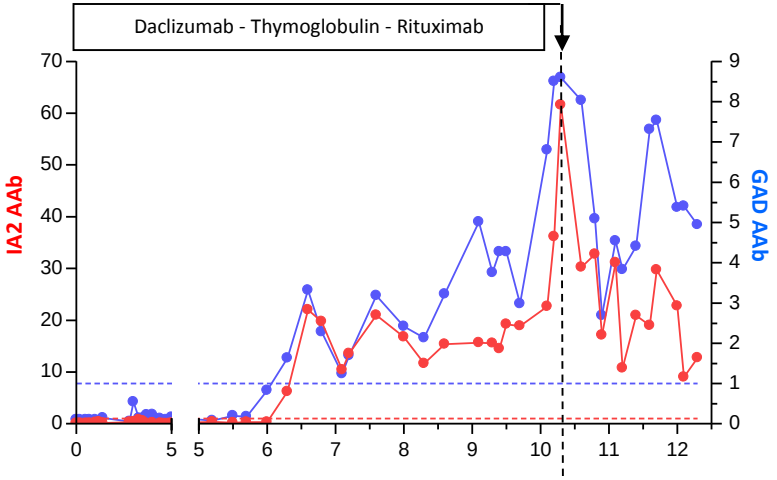
Diabetes 59:947–957, 2010

ORIGINAL ARTICLE

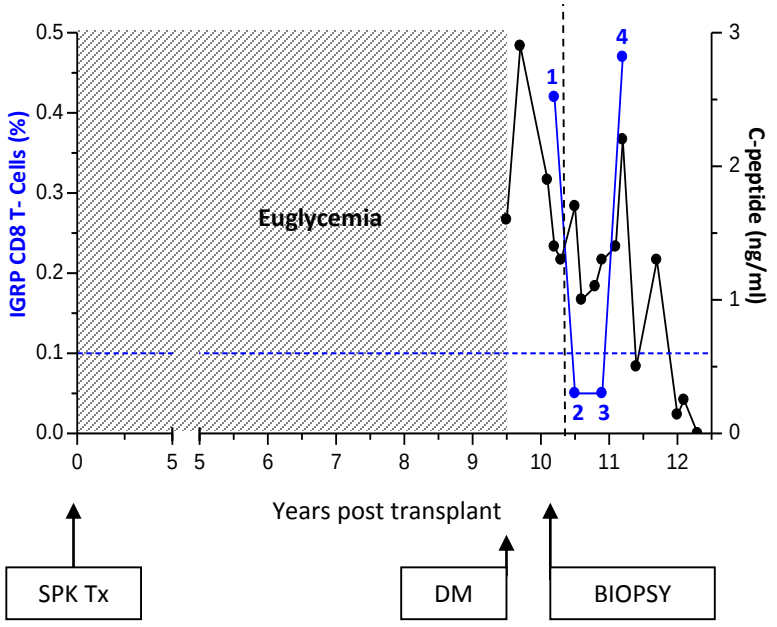
Recurrence of Type 1 Diabetes After Simultaneous Pancreas-Kidney Transplantation, Despite Immunosuppression, Is Associated With Autoantibodies and Pathogenic Autoreactive CD4 T-Cells

Francesco Vendrame,[1] Antonello Pileggi,[1,2] Elsa Laughlin,[3] Gloria Allende,[1] Ainhoa Martin-Pagola,[1] R. Damaris Molano,[1] Stavros Diamantopoulos,[1] Nathan Standifer,[3,4] Kelly Geubtner,[3] Ben A. Falk,[3] Hirohito Ichii,[1,2] Hidenori Takahashi,[2] Isaac Snowhite,[1] Zhibin Chen,[5] Armando Mendez,[1,6] Linda Chen,[2] Junichiro Sageshima,[2] Phillip Ruiz,[2] Gaetano Ciancio,[2] Camillo Ricordi,[1,2,5,6] Helena Reijonen,[3] Gerald T. Nepom,[3] George W. Burke III,[1,2] and Alberto Pugliese[1,5,6]

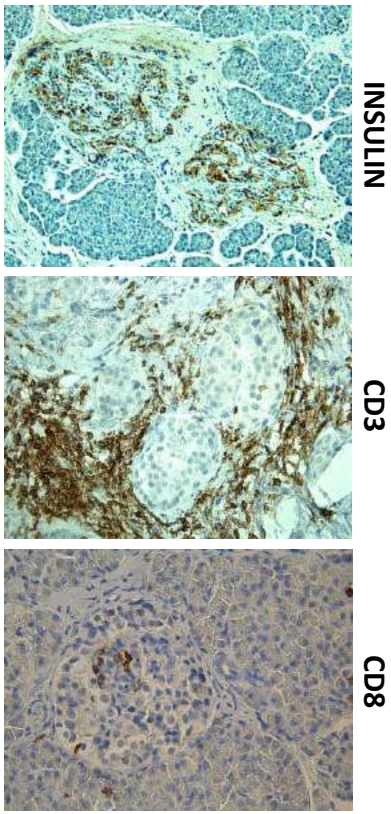
A. AUTOANTIBODIES



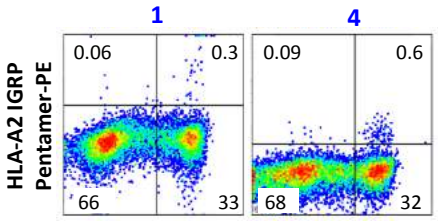
B. C-PEPTIDE AND AUTOREACTIVE T CELLS



C. PANCREAS TRANSPLANT BIOPSY



D. AUTOREACTIVE T CELLS



BLOOD

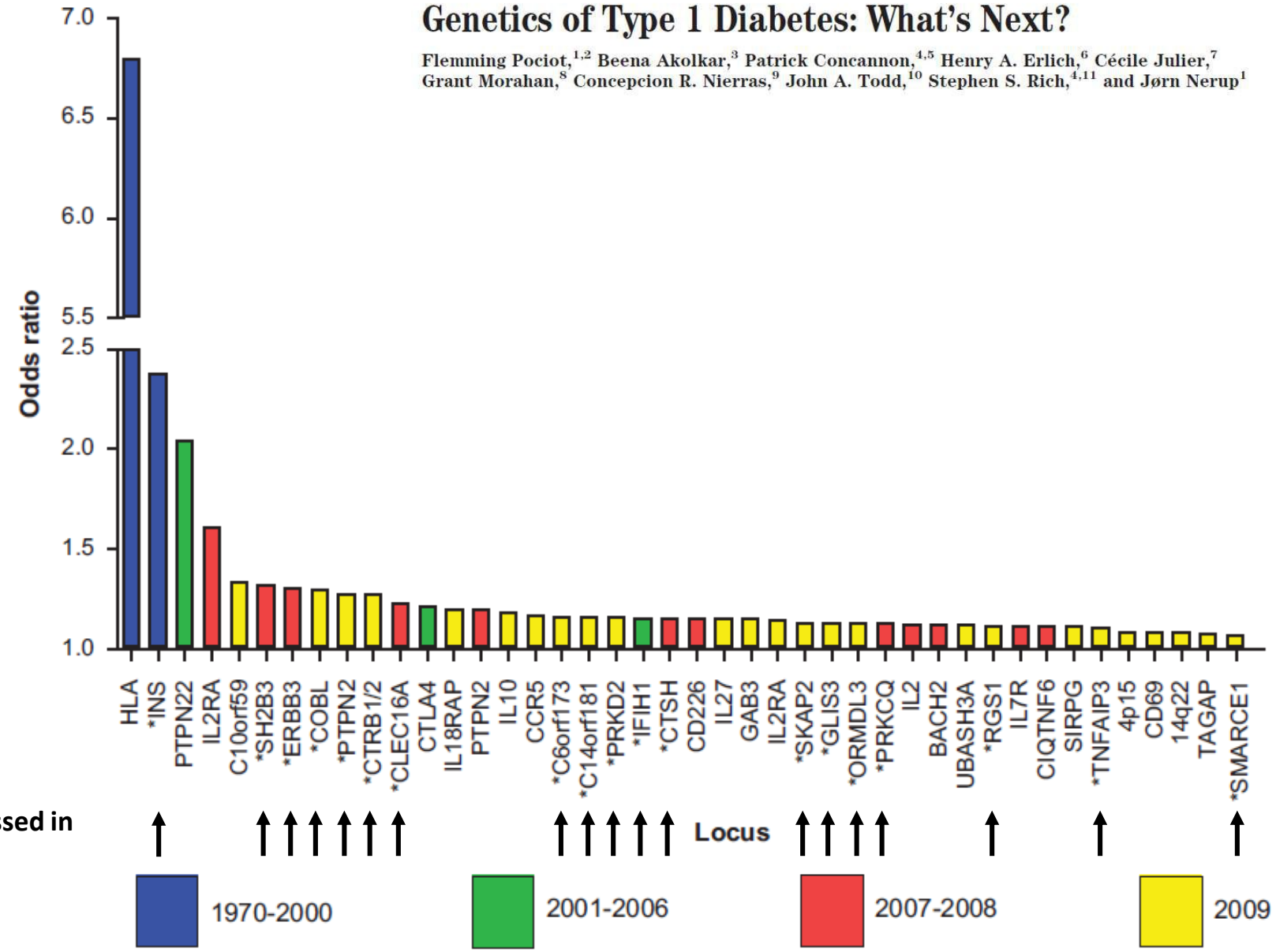
Genetics

- Most (85-90%) patients lack affected family members
- Approximately 10-15% of cases have an affected relative
- Yet there is familial clustering as shown by the increased risk of T1D in family members

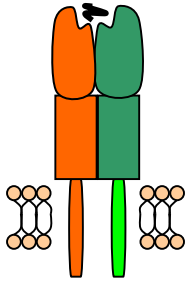
LIFETIME RISK OF T1D

– Monozygotic Twins	30-50%, up to 70%
– Siblings	~6% (1-20%)
– Offspring of diabetic father	6-9%
– Offspring of diabetic mother	1-4%
– Parents of diabetic child	3%
– General population	0.4%

Over 50 genetic regions linked to T1D

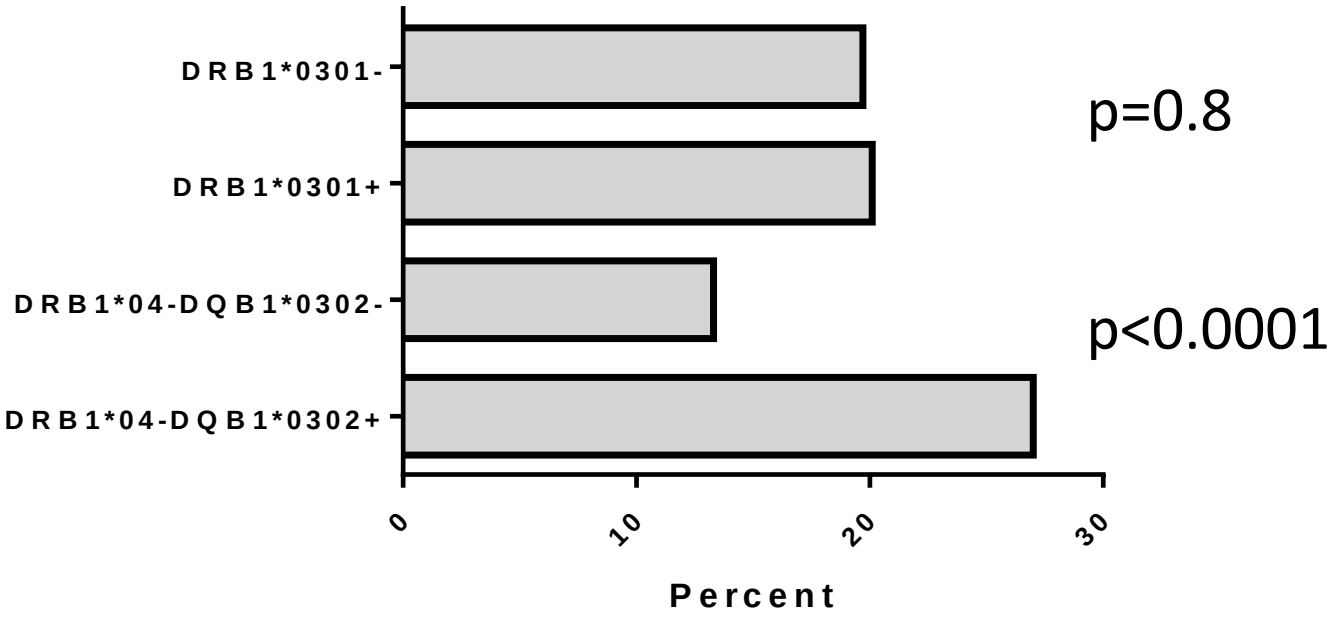


Predisposition and Protection From the HLA-DR and HLA-DQ Genes



DRB1	DQA1	DQB1	
04 (DR4)	0301	0302	(DQ8) - Predisposing
04 (DR4)	0301	0301	Neutral
0301 (DR3)	0501	0201	(DQ2) - Predisposing
1501 (DR2)	0102	0602	(DQ6) - Protective

High-risk DRB1*04-DQB1*0302 haplotypes are associated with increased risk of conversion to multiple autoantibodies and T1D among relatives

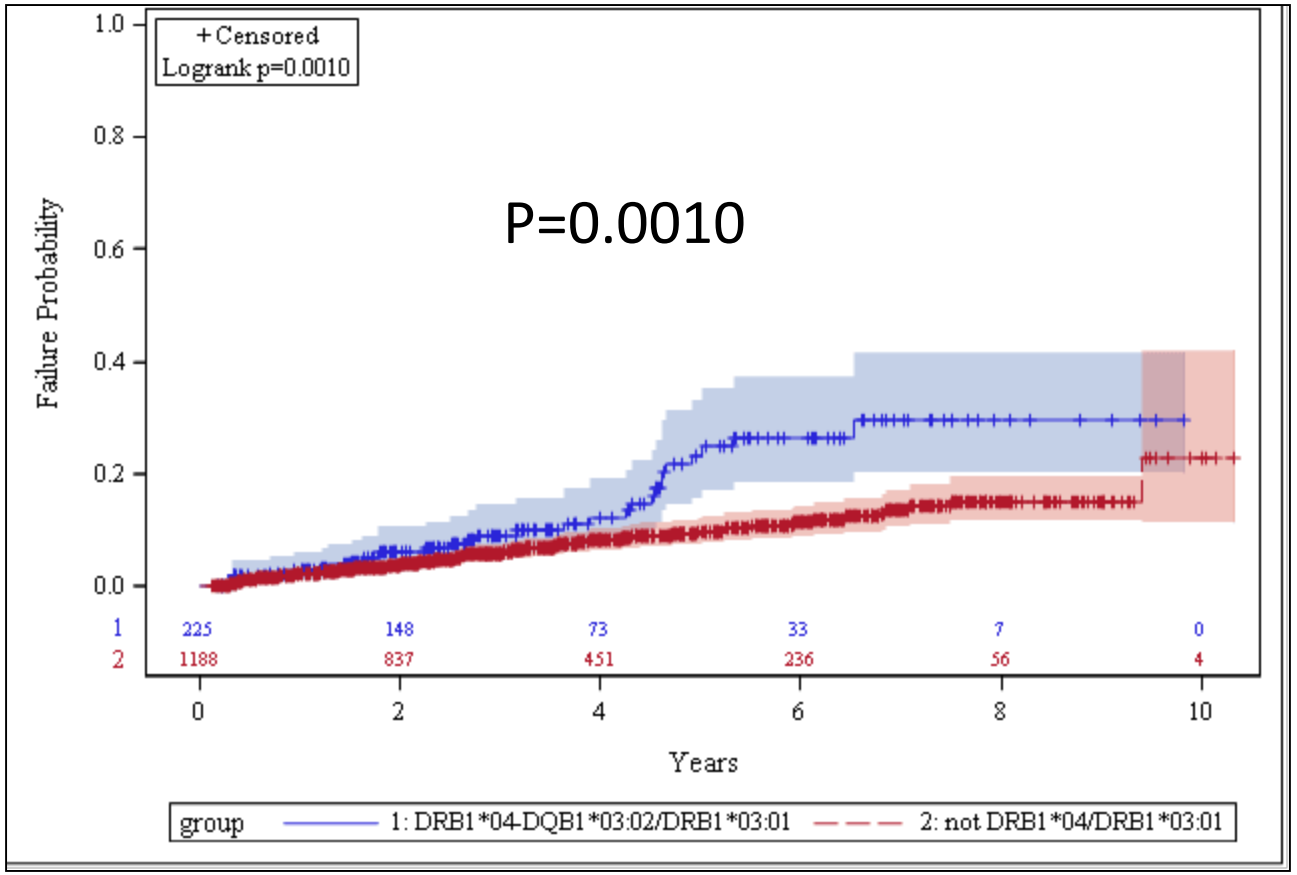


Pugliese and the TrialNet Study Group, unpublished

Type 1 Diabetes TrialNet

Cumulative incidence of T1D in relative with a single autoantibody at screening

DRB1*04-DQB1*03:02/DRB1*03:01 vs not DRB1*04/DRB1*03:01

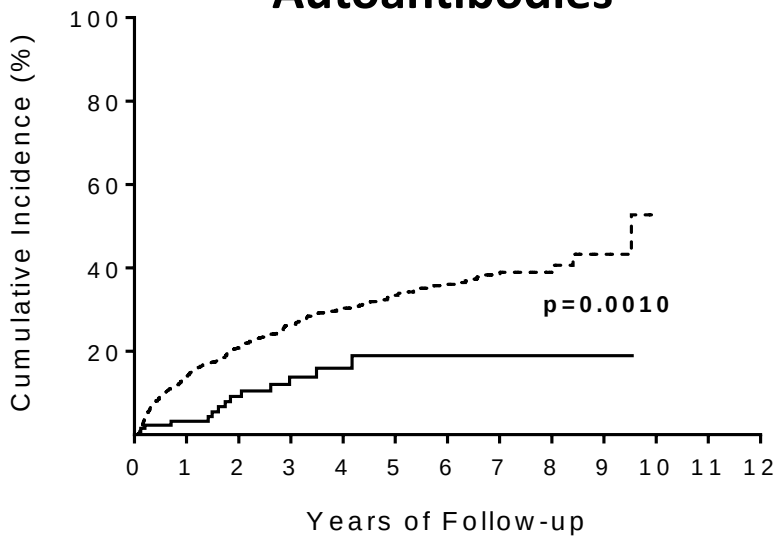


Pugliese and the TrialNet Study Group, unpublished

Type 1 Diabetes TrialNet

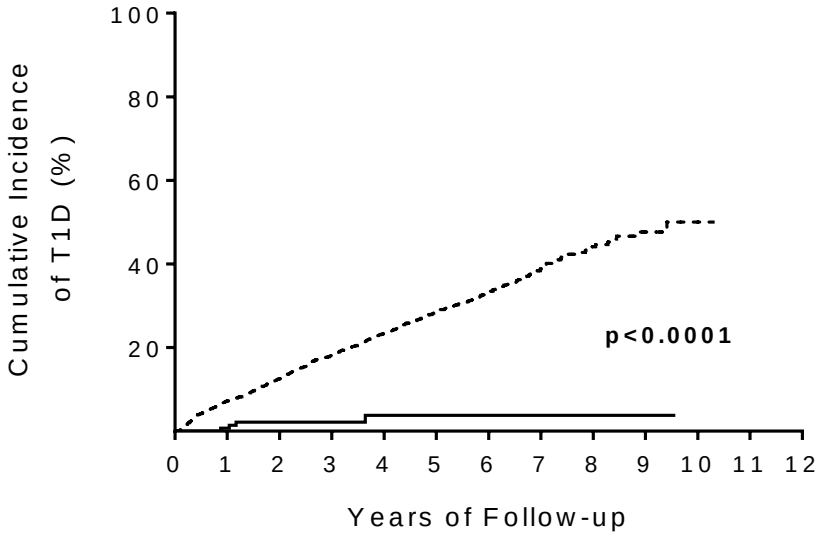
Genetic Protection in Autoantibody-Positive Relatives By HLA DRB1*1501-DQA1*0102-DQB1*0602

Conversion from Single to Multiple Autoantibodies



0602-	n=1890	n=251	n=1
0602+	n=131	n=18	n=0

Cumulative Incidence of T1D



0602-	n=3194	n=653	n=5
0602+	n=155	n=35	n=0

Pugliese and the TrialNet Study Group, Diabetes 2016

Type 1 Diabetes TrialNet

DPT-1 Oral Insulin Study
Time to Diabetes - By Treatment
Subset: IAA Confirmed ≥ 80 nU/ml

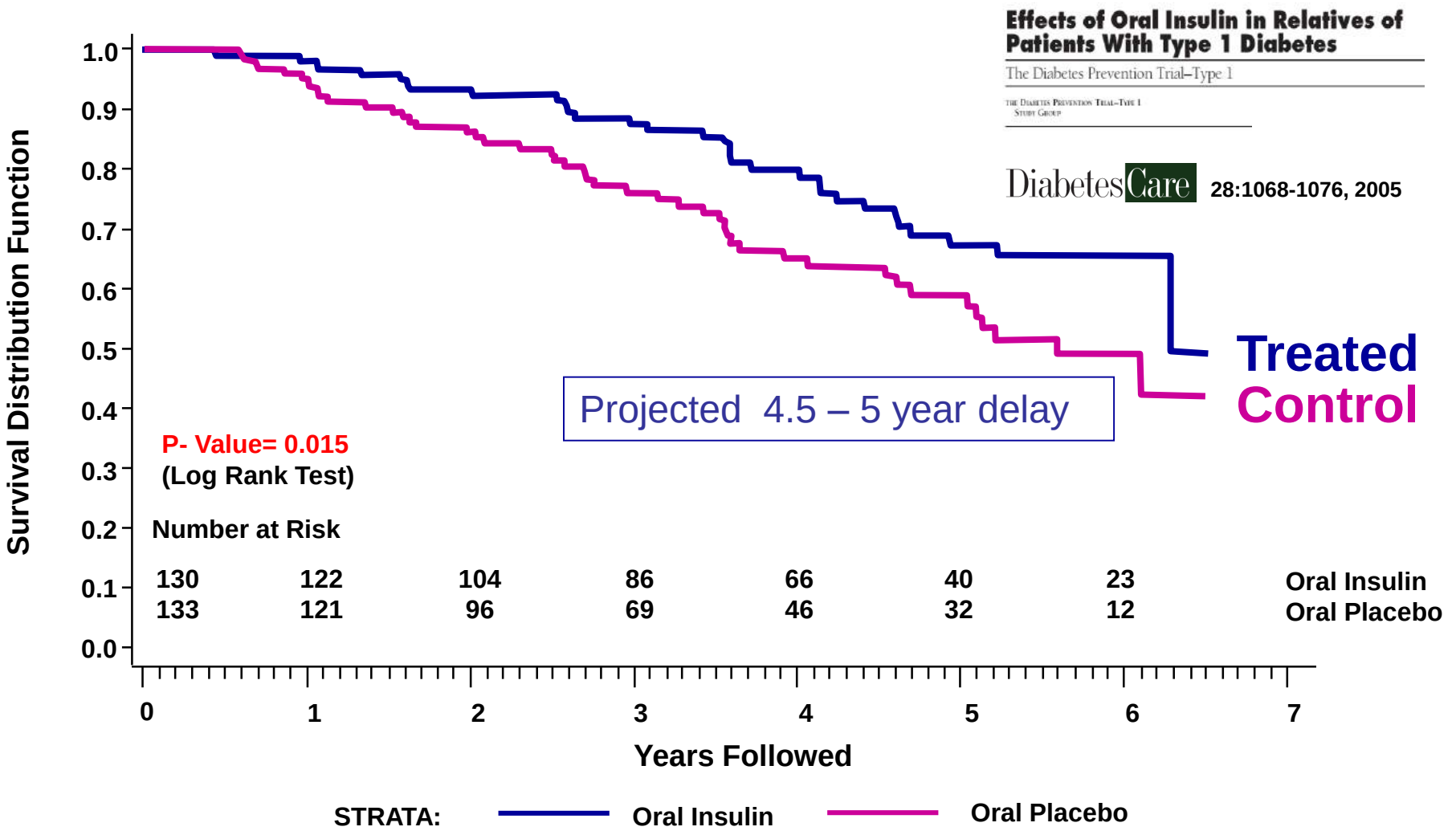


Table 1—Type 1 diabetes risk stratification by family history and genetic susceptibility

Population	Risk of type 1 diabetes (%)	Frequency in population (%)
Low risk (<1%)		
Newborns: European/U.S. population	0.4–1	100
Newborns with HLA protective genotypes (124)	<0.05	75
FDR with HLA protective genotypes (124)	0.3	0.3
FDR with low gene risk score* (HLA and non-HLA risk genes) (23)	<1	0.1
Intermediate risk (1–12%)		
Newborns with HLA high-risk genotypes (37)	4	4–5
Newborns with high gene risk score** (HLA and non-HLA risk genes) (23)	12	1
Newborn first-degree relatives of people with type 1 diabetes	5	0.5–1
High risk (12–25%)		
FDR plus HLA high-risk genotypes (125)	10–20	0.1
FDR plus high gene risk score*** (HLA and non-HLA risk genes) (23)	40	0.1
Multiple affected FDRs (126)	20–25	<<0.1
Very high risk (>25%)		
Identical twin of a patient with type 1 diabetes (28,29)	30–70	<<0.1
Multiple affected FDRs plus HLA risk genotypes (126)	50	<<0.1
Sibling affected plus HLA risk genes, identical by descent (30)	30–70	<<0.1

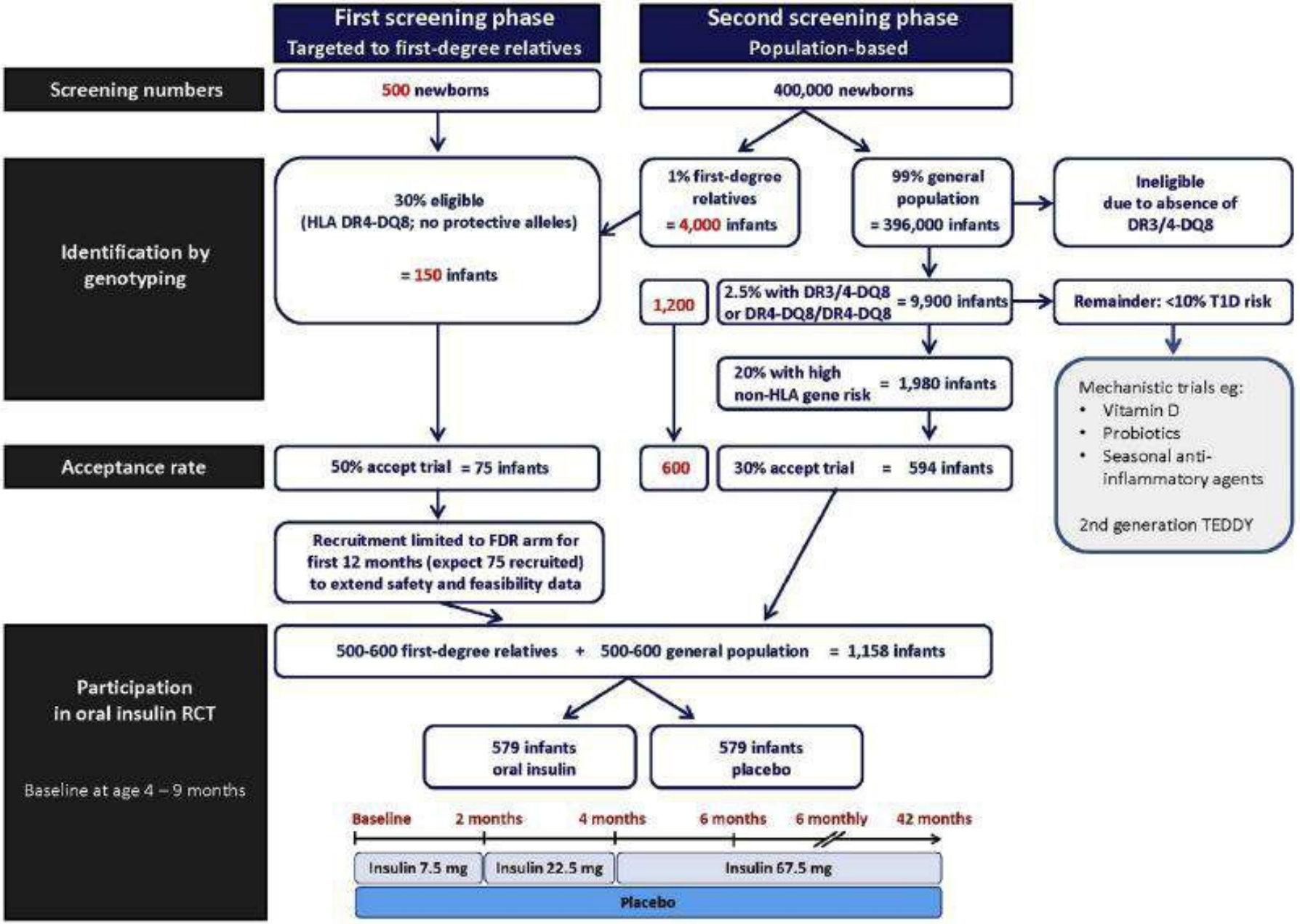
Insel RA et al. Diabetes Care 2015

Primary prevention of beta-cell autoimmunity and type 1 diabetes — The Global Platform for the Prevention of Autoimmune Diabetes (GPPAD) perspectives

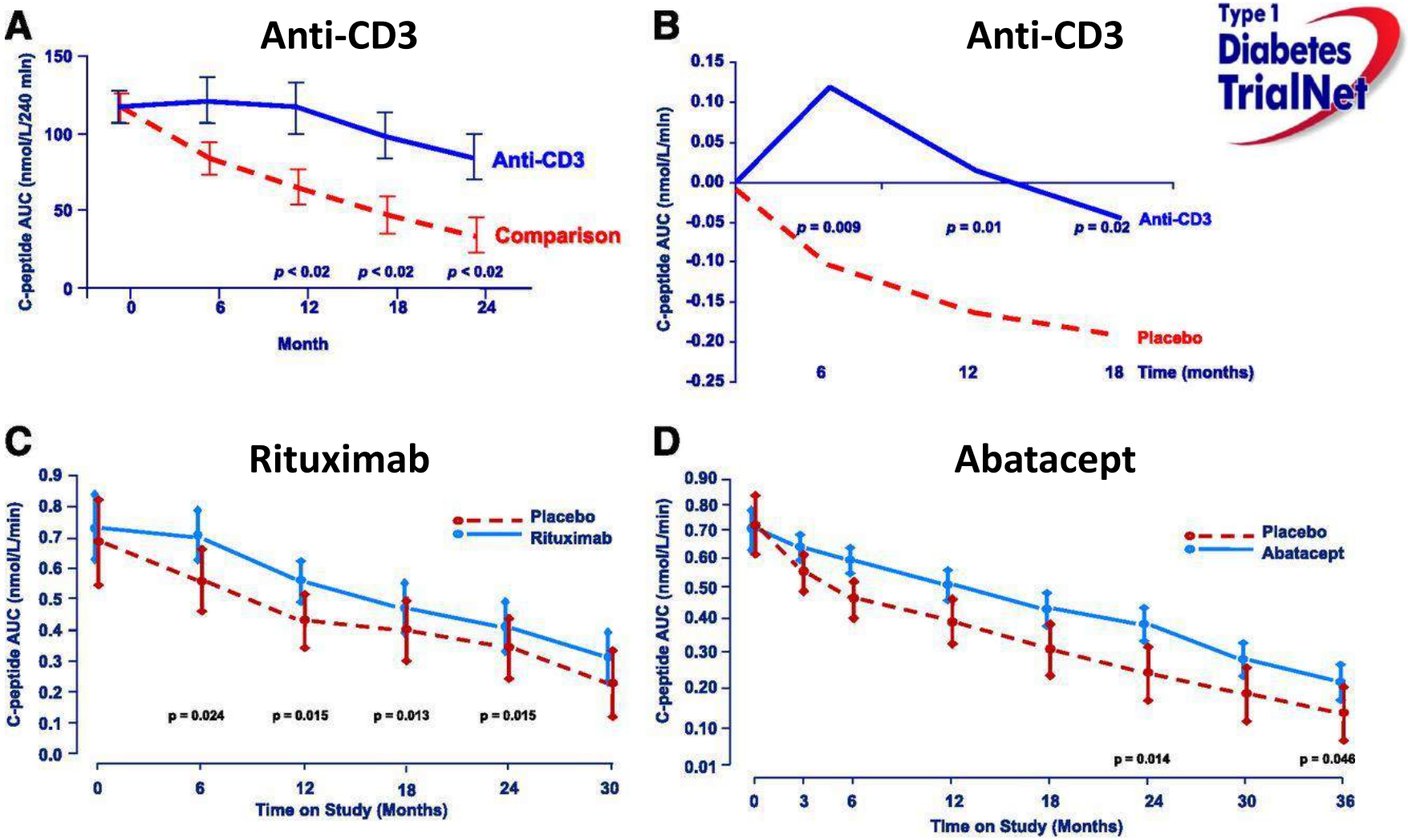
MOLECULAR METABOLISM 2016

A.G. Ziegler[1,2,*], T. Danne[3], D.B. Dunger[4], R. Berner[5], R. Puff[1], W. Kiess[6], G. Agiostratidou[7], J.A. Todd[8], E. Bonifacio[9]

- Authors modeled recruitment into a randomized controlled trial (RCT) for infants with and without a first-degree family history of T1D based on genetic risk testing.
- HLA genotyping and, for the general population, genotyping at additional T1D risk genes, will identify children with around 10% risk of beta-cell autoimmunity.
- Thus, testing of ~500,000 newborns or infants would be needed to identify 1,160 infants for randomization.
- The proposed RCT would have 80% power to detect a 50% reduction in the development of multiple beta-cell autoantibodies by age 4 years.
- It is timely and feasible to establish a platform for primary prevention trials for type 1 diabetes in Europe.


First screening phase
Targeted to first-degree relatives
Second screening phase
Population-based
Screening numbers
500 newborns
400,000 newborns
Identification by genotyping
30% eligible
(HLA DR4-DQ8; no protective alleles)
= 150 infants
1% first-degree relatives
= 4,000 infants
99% general population
= 396,000 infants
Ineligible due to absence of DR3/4-DQ8
1,200
2.5% with DR3/4-DQ8 or DR4-DQ8/DR4-DQ8
= 9,900 infants
Remainder: <10% T1D risk
20% with high non-HLA gene risk
= 1,980 infants
Mechanistic trials eg:
• Vitamin D
• Probiotics
• Seasonal anti-inflammatory agents
2nd generation TEDDY
Acceptance rate
50% accept trial = 75 infants
600
30% accept trial = 594 infants
Recruitment limited to FDR arm for first 12 months (expect 75 recruited) to extend safety and feasibility data
Participation in oral insulin RCT
Baseline at age 4 – 9 months
500-600 first-degree relatives + 500-600 general population = 1,158 infants
579 infants oral insulin
579 infants placebo
Baseline
2 months
4 months
6 months
6 monthly
42 months
Insulin 7.5 mg
Insulin 22.5 mg
Insulin 67.5 mg
Placebo

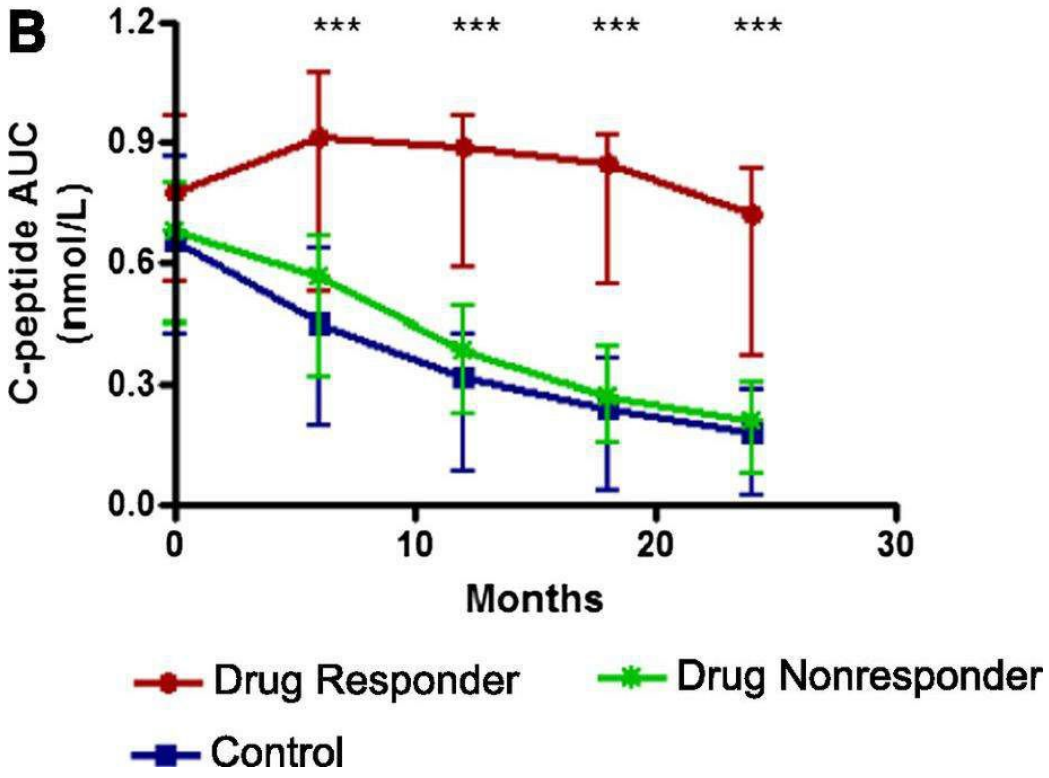
Recent trials show some efficacy in slowing decline of beta cell function after diagnosis



Jay S. Skyler Diabetes Care 2015;38:997-1007

Teplizumab (Anti-CD3 mAb) Treatment Preserves C-Peptide Responses in Patients With New-Onset Type 1 Diabetes in a Randomized Controlled Trial

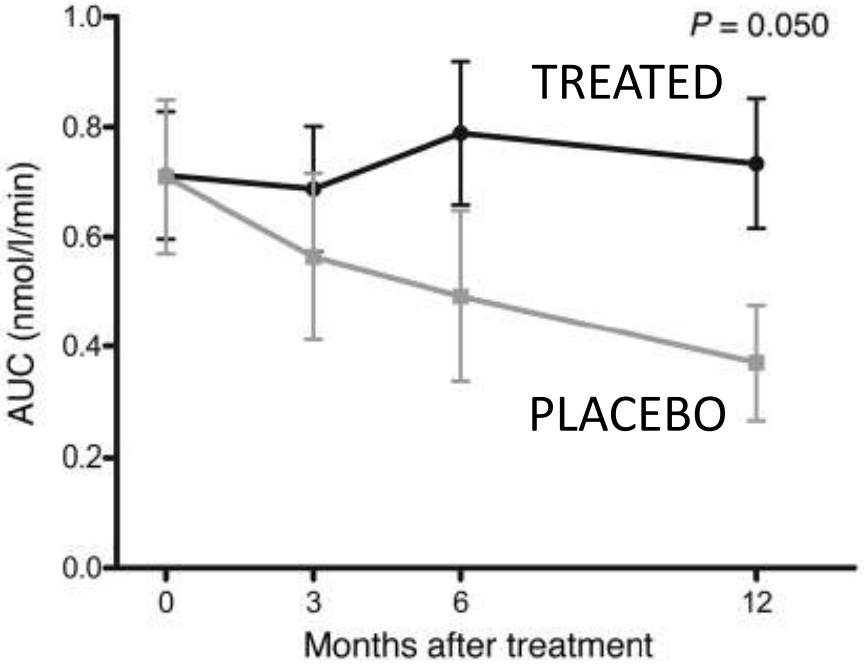
Metabolic and Immunologic Features at Baseline Identify a Subgroup of Responders



Kevan C. Herold et al. Diabetes 2013;62:3766-3774

Anti-thymocyte globulin/G-CSF treatment preserves β cell function in patients with established type 1 diabetes

Michael J. Haller,[1] Stephen E. Gitelman,[2] Peter A. Gottlieb,[3] Aaron W. Michels,[3] Stephen M. Rosenthal,[2] Jonathan J. Shuster,[4] Baiming Zou,[4] Todd M. Brusko,[5] Maigan A. Hulme,[5] Clive H. Wasserfall,[5] Clayton E. Mathews,[5] Mark A. Atkinson,[5] and Desmond A. Schatz[1]



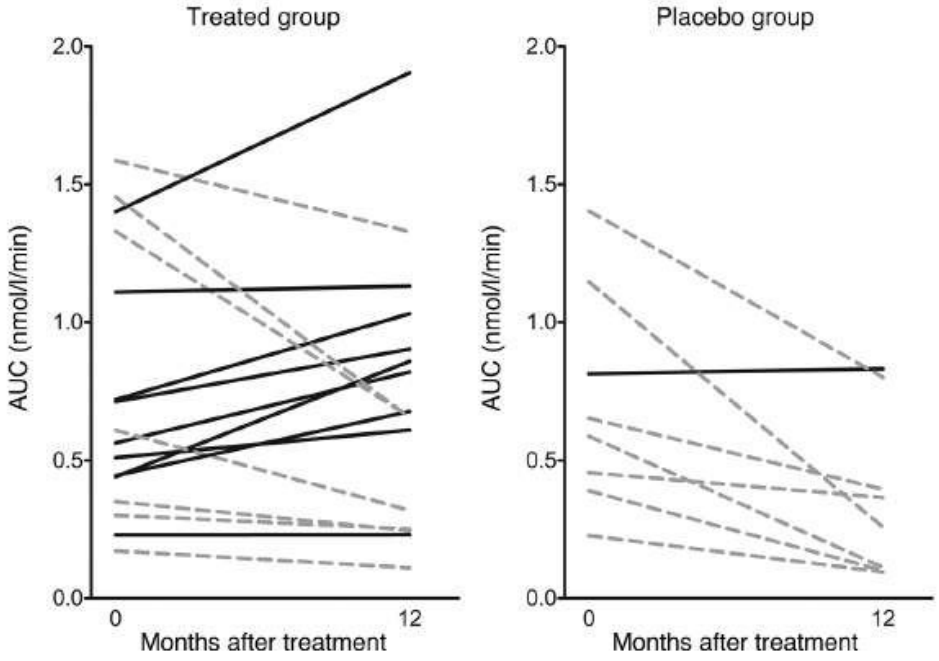
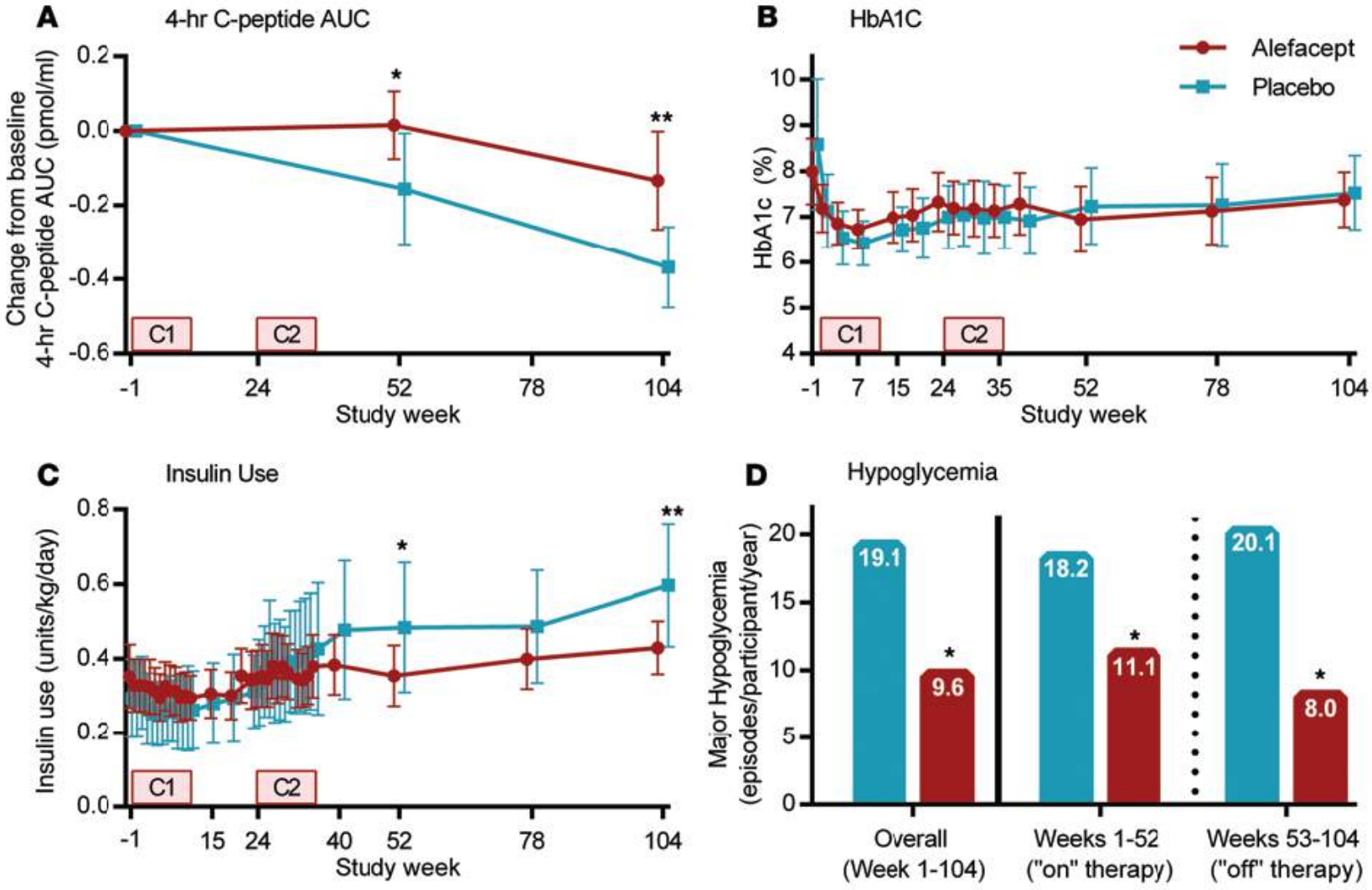


Figure 3. AUC C-peptide at baseline and 1 year following ATG/G-CSF compared with placebo. Data from each subject is shown. Subjects are separated by study drug assignment. Subjects depicted by solid black lines had sustained or increased AUC C-peptide over 1 year. Subjects depicted by dashed gray lines had a reduction in AUC C-peptide over 1 year. AUC C-peptide is shown as the AUC divided by 120 minutes.

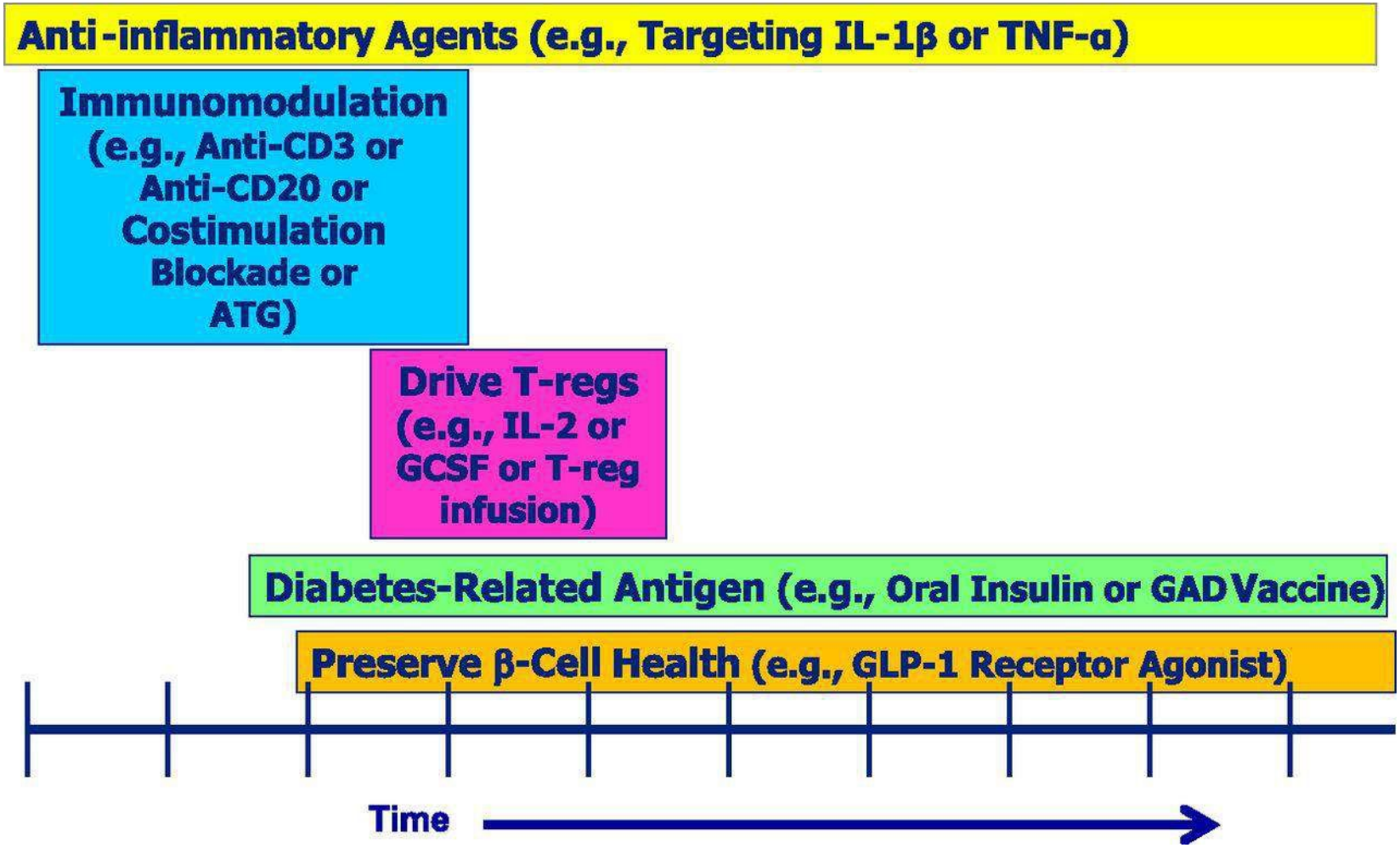
Journal of Clinical Investigation, 2015

Targeting effector memory T cells with Alefacept in new onset T1D

Rigby et al. Lancet Endocrinology 2013 and JCI 2015



Combinatorial Therapies in T1D



Jay S. Skyler Diabetes Care 2015;38:997-1007

American Diabetes Association.

Key questions for immunotherapy in T1D

- Can short course therapies be successful?
- For how long?
- Is chronic treatment required?
- If so, wouldn't chronic immunosuppression be difficult to implement (safety concerns)
- Can we promote immune regulation without immunosuppression?

Low-dose IL-2 as a therapeutic agent for tolerance induction

"...the requirement for IL-2 by Tregs raises the possibility that IL-2 may serve as a biologic to promote T-cell tolerance."

KEYWORDS: IL-2 ▪ tolerance ▪ T regulatory cells ▪ Type 1 diabetes

IL-2 is produced primarily by recently activated naive and memory T cells after antigen stimulation of the T-cell receptor. The duration of IL-2 secretion is short lived as production of this cytokine is under stringent transcriptional and post-transcription regulation. IL-2 mediates its biological activity by binding to a high affinity receptor consisting of three subunits, IL-2Rα (CD25), IL-2Rβ (CD122) and γc (CD132). Expression of the high affinity IL-2R is also under stringent transcriptional regulation that is positively linked to T-cell receptor and IL-2 stimulation, leading to IL-2R expression mainly on recently activated T effector (Teff) cells and Tregs. Under physiological levels of IL-2, T-cell immunity is enhanced by increasing expansion and effector activity of antigen-specific T cells and by promoting memory cell development. IL-2 also promotes T-cell tolerance by providing essential signals for thymic development and peripheral homeostasis of Tregs. More detailed information concerning that received IL-2 therapy and/or IL-2 expanded lymphocytes [2]. This work was followed by some initial success in patients suffering from renal cell carcinoma and melanoma [3]. However, as many more patients underwent IL-2 therapy, it became apparent that the response rate is low [4]. Nevertheless, IL-2 is still sometimes used as a therapy for renal cell carcinoma and melanoma cancers owing to their poor prognosis and lack of more efficacious treatment. IL-2 was also heavily tested to boost immunity in HIV/AIDS patients, but this approach was proven to be ineffective [5].

There are two fundamental reasons that are likely to account for these poor outcomes when using IL-2 to boost immunity. First, sufficient antigen-specific T cells may not be mobilized by IL-2 therapy because the expression of the high affinity IL-2R does not persist on antigen-specific T cells. IL-2 has a very short half-life in the circulation (~30 min) after infusion *in vivo*.

Thomas R Malek

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tmalek@med.miami.edu

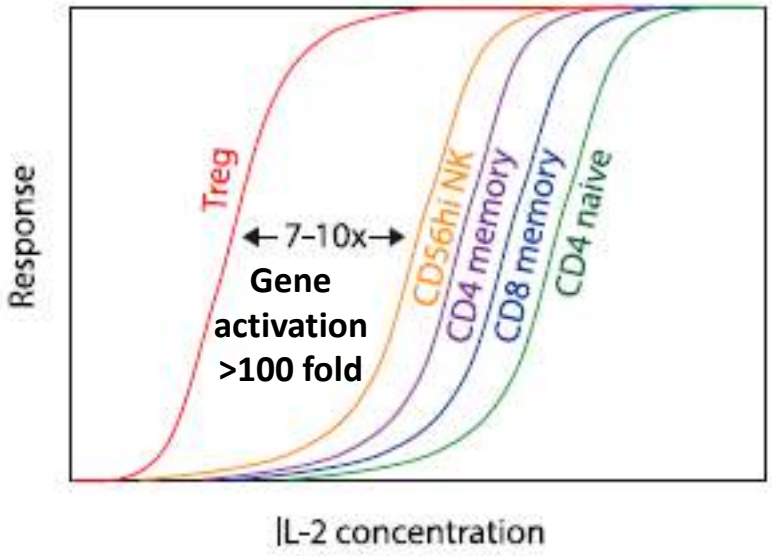
Alberto Pugliese

Department of Medicine, Division of Endocrinology, Diabetes & Metabolism

Selective IL-2 Responsiveness of Regulatory T Cells Through Multiple Intrinsic Mechanisms Supports the Use of Low-Dose IL-2 Therapy in Type 1 Diabetes

Diabetes 2015;64:2172–2183 | DOI: 10.2337/db14-1322

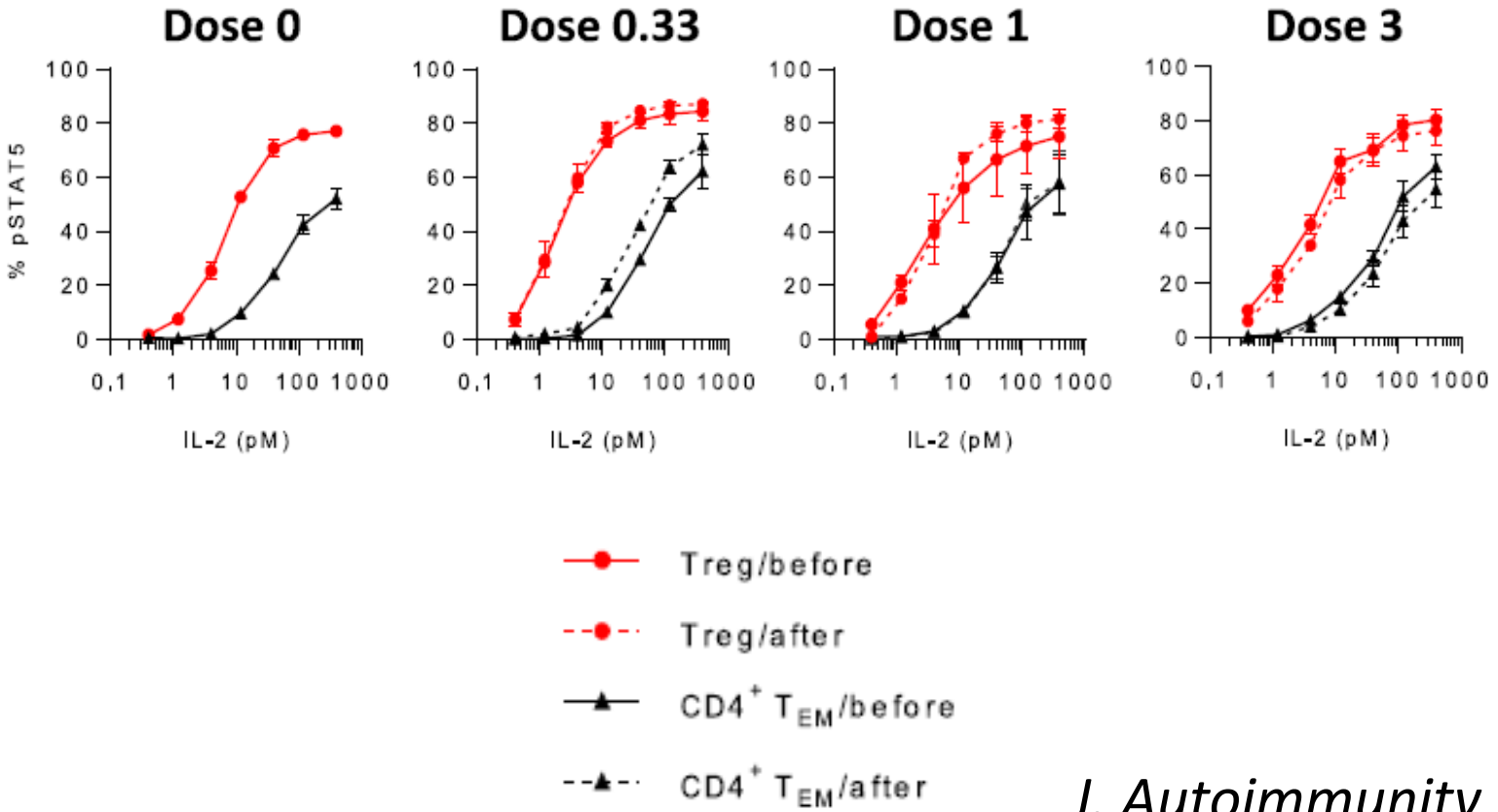
Aixin Yu,[1] Isaac Snowhite,[2] Francesco Vendrame,[2] Michelle Rosenzwajg,[3,4,5] David Klatzmann,[3,4,5] Alberto Pugliese,[1,2,6] and Thomas R. Malek[1,2]



Qizhi Tang Diabetes 2015;64:1912-1913

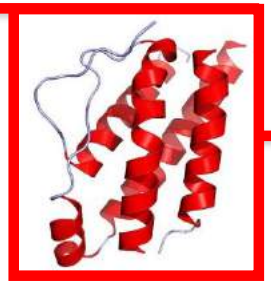
Low-dose interleukin-2 fosters a dose-dependent regulatory T cell tuned milieu in T1D patients

Michelle Rosenzwajg [a, b, c], Guillaume Churlaud [a, b, c], Roberto Mallone [d, e, f, g], Adrien Six [a, b, c], Nicolas Dérian [a, b, c], Wahiba Chaara [a, b, c], Roberta Lorenzon [a, b, c], S. Alice Long [h], Jane H. Buckner [h], Georgia Afonso [d, e, f, g], Hang-Phuong Pham [i], Agnès Hartemann [j], Aixin Yu [k, m], Alberto Pugliese [k, l, m], Thomas R. Malek [k, m], David Klatzmann [a, b, c, *]



J. Autoimmunity 2015

Low Dose IL-2



in Established Type 1 Diabetes

A Clinical Trial

Study Objectives: Conduct a clinical intervention study to assess, in patients with established T1D:

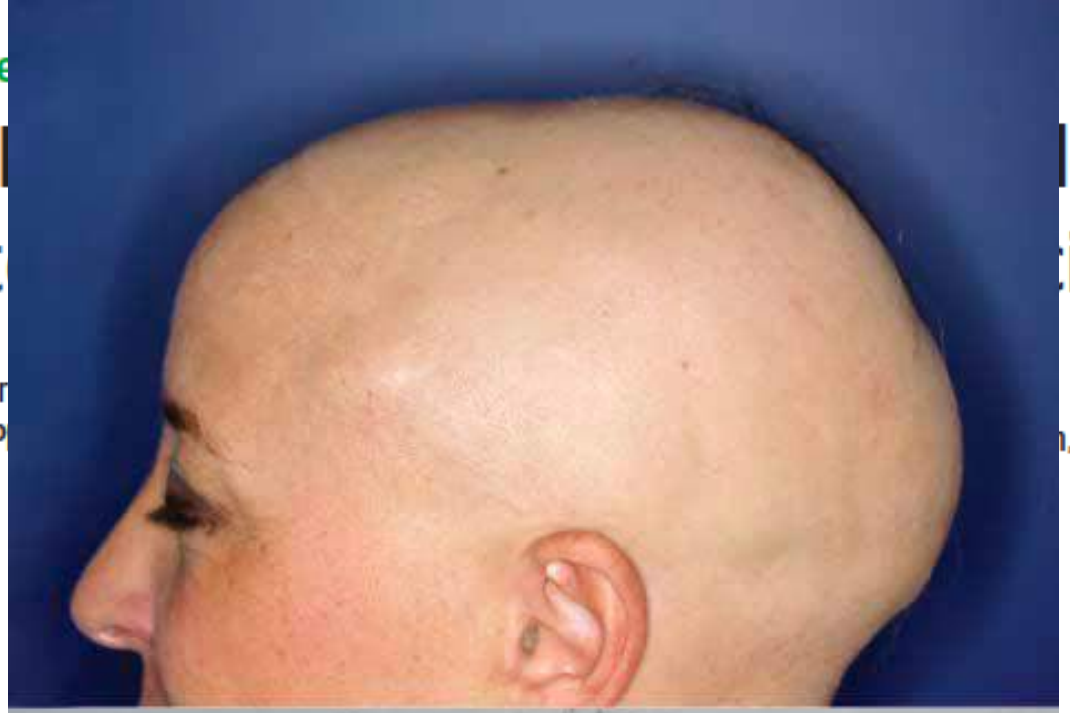
1. the safety of low dose IL-2
2. the effects of low dose IL-2 on stimulated insulin secretion
3. the immunological effects of low-dose IL-2

Study Design: Double-blinded, randomized, controlled, multicenter, 2 arms, phase I/II clinical trial

Case Report/Case Se

Effects of I leukin 2 to Promot tia Areata

Emeline Castela, MD; Flor
Paul Hofman, MD, PhD; P , MD, PhD



JAMA Dermatology, 2014

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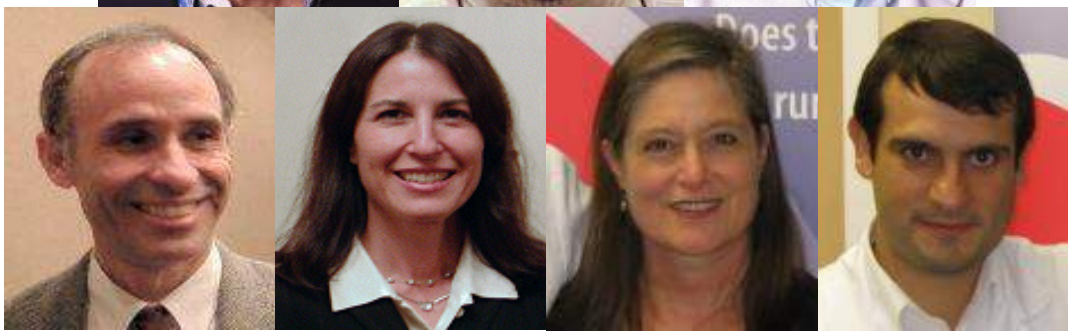


Type 1 Diabetes TrialNet

Della Matheson
dmatheso@med.miami.edu
Carlos Blaschke
cblaschke@med.miami.edu

Phone: 305-243-3781

Which website provide information about type 1 diabetes and clinical trials?

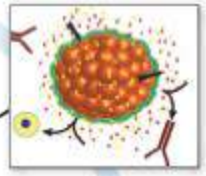
1. **Type 1 diabetes TrialNet** www.diabetestrialnet.org
2. **Immune Tolerance Network** www.immunetolerance.org
3. **JDRF** www.JDRF.org
4. www.clinicaltrials.gov
5. **Diabetes Research Institute** www.diabetesresearch.org

DRI BioHub

- *Site*
- *Sustainability*
- *Supply*

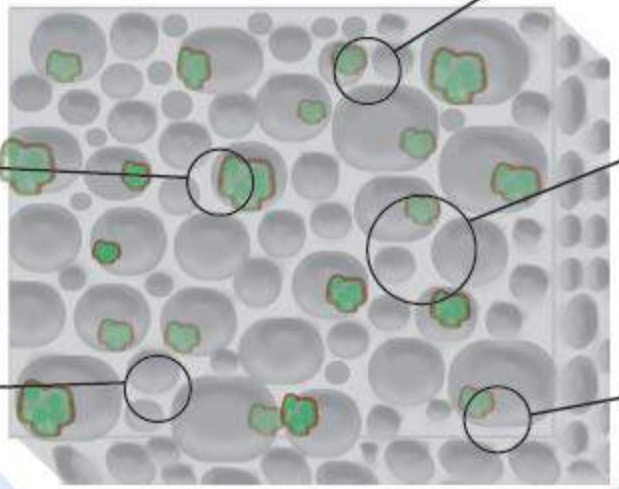
Re-educating the Immune System

Increasing Cell Supply



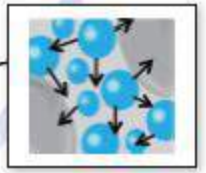
Encapsulation

Co-delivery of Helper Cells



Structural Housing

Localized Drug Delivery



Oxygen Delivery

Clinical Trials

DIABETES RESEARCH INSTITUTE
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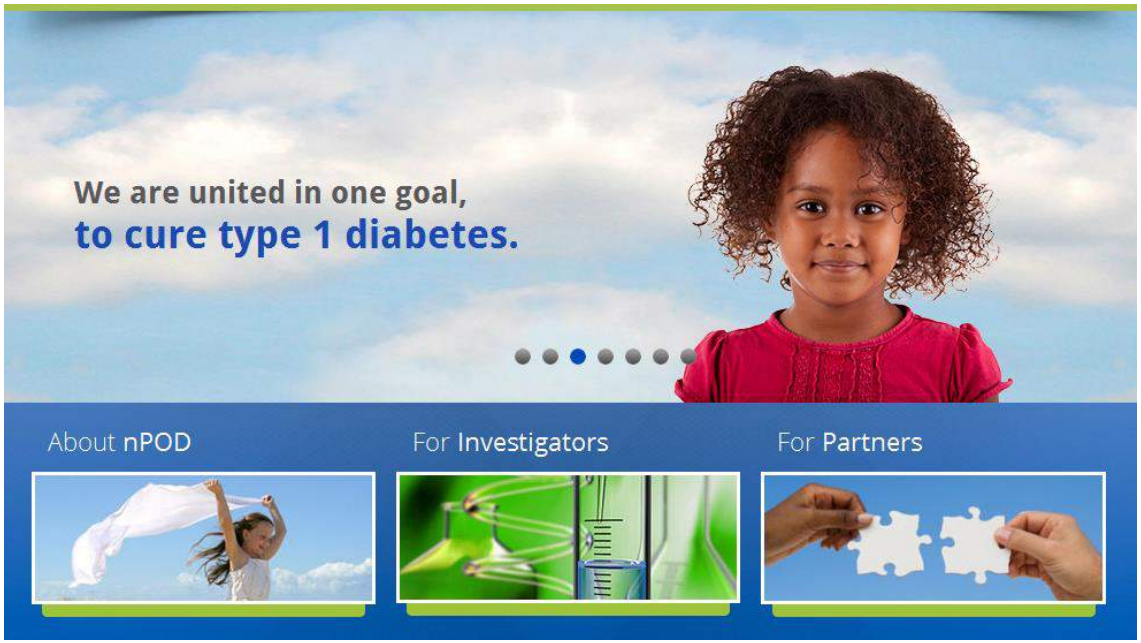
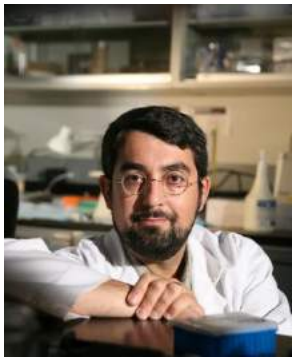
http://www.diabetesresearch.org/

The Network for Pancreatic Organ Donors with Diabetes
www.jdrfnpod.org



Mark Atkinson, PhD
University of Florida
Executive Director



Alberto Pugliese, MD
University of Miami
Executive Co-Director

- Obtain tissues from organ donors with T1D (diagnosed or sub-clinical)
- Distribute tissues to approved research projects (~140 since 2007)
- Promote tissue and data sharing, collaboration, manage project interactions
- Promote a comprehensive understanding of human T1D, identify new therapeutic targets

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