Autoimmunity in Postural Orthostatic Tachycardia Syndrome (POTS)

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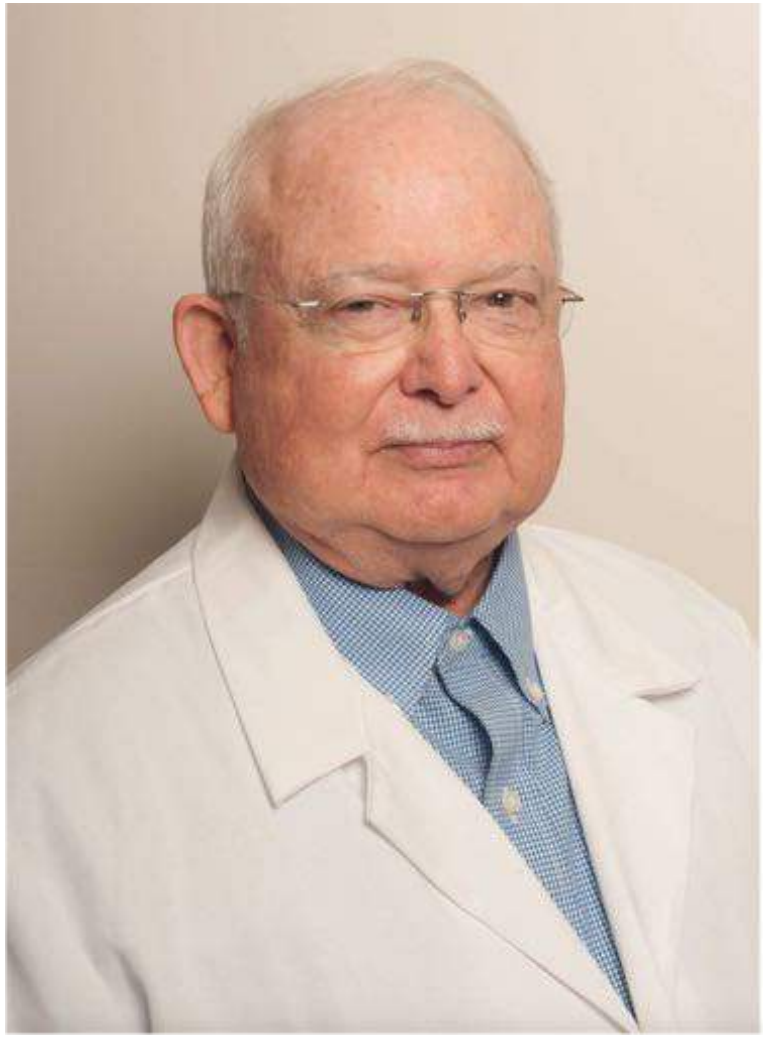
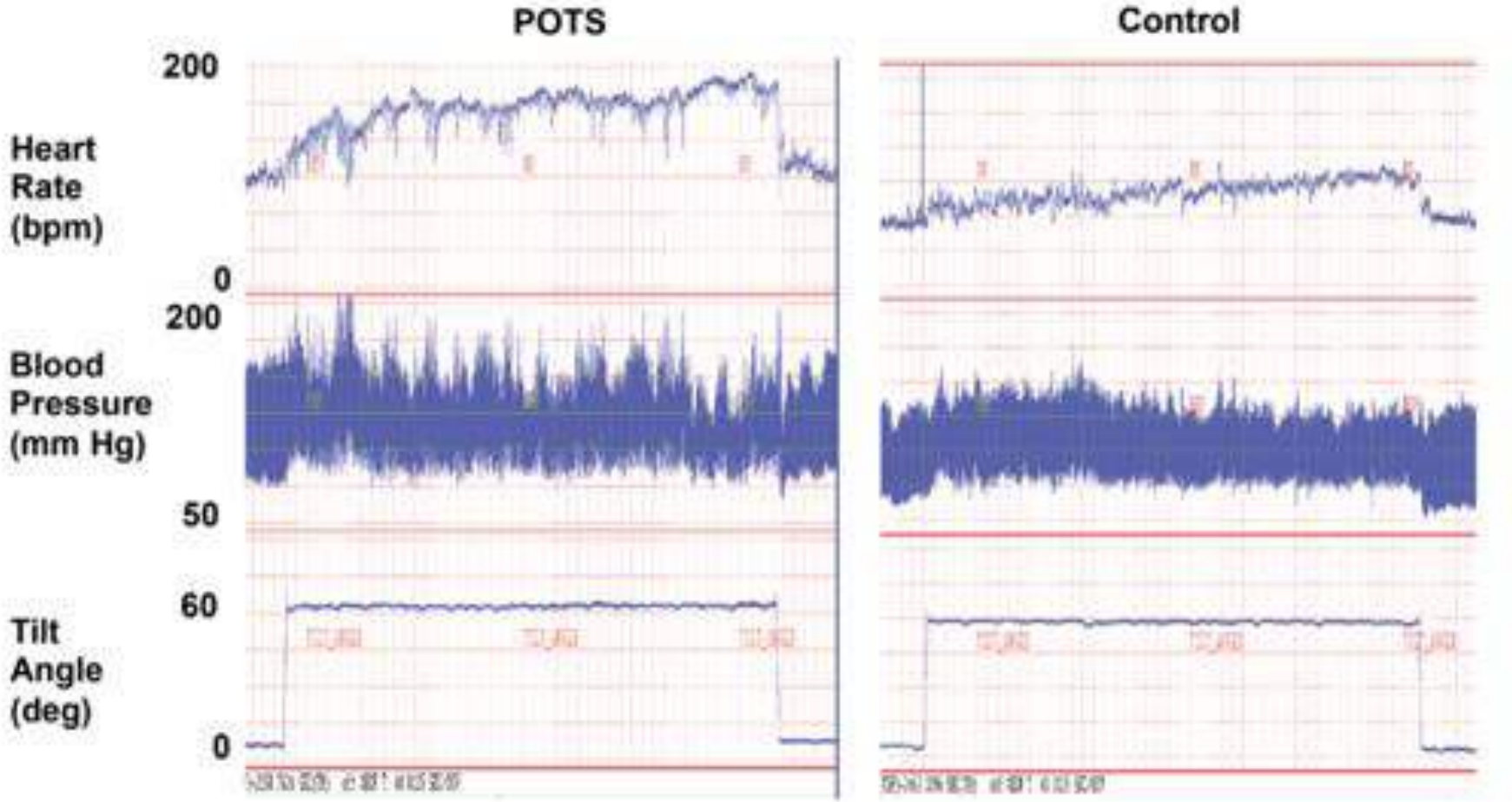
In memoriam: Dr. David Kem



http://www.dysautonomiainternational.org/blog/wordpress/in-memoriam-dr-david-kem/

Postural orthostatic tachycardia syndrome (POTS)



Jones PK, *et al*. *Pract Neurol* 2016;**16**:431–438. doi:10.1136/practneurol-2016-001405

Symptoms	Number	Total number*	% Total
Cardiovascular symptoms			
Lightheadedness	3992	4034	99
Tachycardia	3901	4032	97
Presyncope	3789	4032	94
Shortness of breath	3562	4032	88
Palpitations	3033	4031	87
Chest pain	3164	4032	79
Low blood pressure	2864	4033	71
Syncope	1452	4033	36
Gastrointestinal symptoms			
Nausea	3618	4032	90
Stomach pains	3357	4032	83
Bloating	3184	4031	79
Constipation	2845	4032	71
Diarrhoea	2783	4032	69
Neurological symptoms – head and brain			
Headache	3797	4032	94
Difficulty concentrating	3794	4032	94
Memory problems	3538	4032	87
Tremulousness	3124	4039	78
Neurological symptoms – eyes and ears			
Blurred vision	3015	4032	75
Dry mouth	2662	4031	66
Dry eyes	2383	4030	60
Neurological symptoms – extremities			
Muscle pains	3374	4029	84
Foot coldness	3377	4030	84
Muscle weakness	3344	4030	83
Hand coldness	3311	4029	82
Hand tingling	3060	4029	76
Foot tingling	2701	4028	67
Hand numbness	2627	4029	65
Foot numbness	2350	4029	58
Skin symptoms			
Skin flushing	2774	4029	69
Bladder symptoms			
Frequent urination	2733	4031	68

Immune triggers in POTS

- A recent history of suspected infection is reported in 20-50% of patients with acute triggers.

- Infectious agents linked to POTS include *Borrelia burgdorferi,* Epstein Barr virus (EBV), *Trypanosoma cruzi, Mycoplasma pneumoniae,* and recently SARS-CoV2 (30-60% of long-COVID syndrome).

- Of acute triggers: surgery (12%), pregnancy (9%), vaccine (6%), concussion (4%) which may have strong immune effects.

J Intern Med. 2019;286(438-448). *Mayo Clin Proc*. 1999;74(11):1106-1110; *Ann Clin Transl Neurol*. 2018;5(4):486-492; *Cardiol J*. 2011;18(1):63-66; *Ann Noninvasive Electrocardiol*. 2015;20(1):82—86; *Curr Sports Med Rep*. 15(1):41-45; *SAGE Open Med Case Rep*. 2020;8:2050313X20915413; *Klin Wochenschr*. 1980;58(23):1307-1311; *Infect Dis Clin Pract*. 2009;17(5); *Clin Auton Res*. 2020;30(5):449-451; *J Innov Card Rhythm Manag*. 2020;11(11):4302-4304; *Heart Rhythm*. December 2020

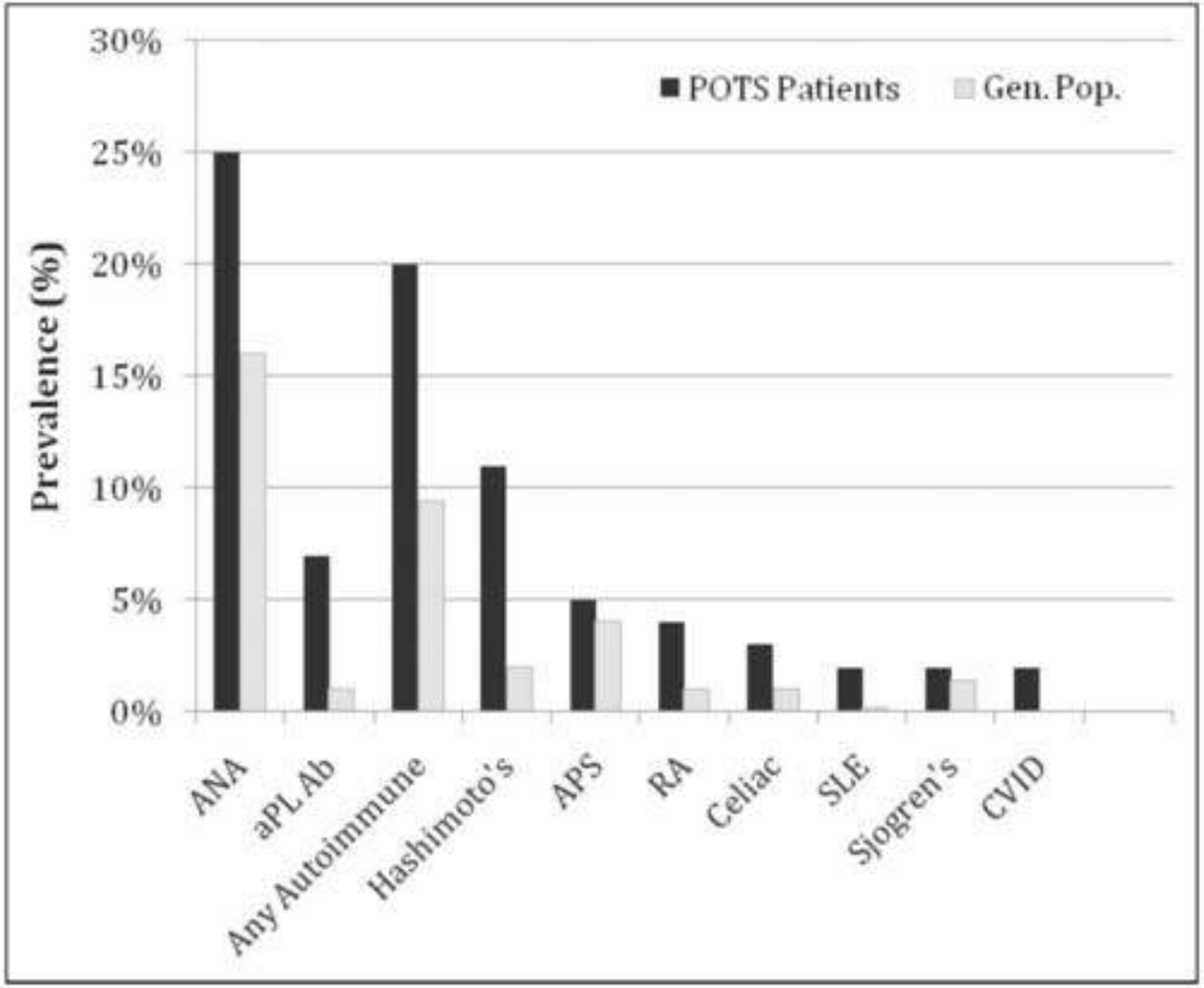
Table 2 *Common comorbidities in POTS patients*

Comorbidity	Number (%) (of 3933 respondents)
Migraine headaches	1557 (40%)
Irritable bowel syndrome	1192 (30%)
Ehlers–Danlos syndrome	994 (25%)
Chronic fatigue syndrome	809 (21%)
Asthma	798 (20%)
Fibromyalgia	786 (20%)
Raynaud's phenomena	610 (16%)
Iron deficiency anaemia	628 (16%)
Gastroparesis	548 (14%)
Vasovagal syncope	499 (13%)
Inappropriate sinus tachycardia	448 (11%)
Mast cell activation disorder	353 (9%)
Autoimmune disease	616 (16%)
Hashimoto's thyroiditis	228 (6%)
Coeliac disease	133 (3%)
Sjögren's syndrome	112 (3%)
Rheumatoid arthritis	93 (2%)
Lupus	81 (2%)
Other	160 (4%)

Patient reported

Journal of Internal Medicine, 2019, 286; 438–448

- Higher prevalence of autoimmune disorders in POTS patients and close relatives including: Hashimoto's thyroiditis, Sjögren's syndrome, celiac disease and systemic lupus erythematosus (SLE).



Auton Neurosci. 2018;215:78-82; *J Intern Med*. 2019;286(438-448); *Lupus*. 2015;24(13):1364-1369; *Cardiol Young*. 2020;30(3):388-394 ; *Eur J Gastroenterol Hepatol*. 2016;28(12):1383-1387; *Neurologist*. 2017;22(4):127-130; *Rheumatology (Oxford)*. 2004;43(5):609-614.

Autoimmune autonomic neuropathy

- Celiac disease and Sjogren's syndrome are common causes of autonomic neuropathy after diabetes.
- Dry eyes are common in POTS patients presenting to a neurology clinic but Sjogren's work up often not done.
- Antibody testing alone for Sjogren's often not enough (salivary biopsy).
- Small fiber neuropathy is present in Sjogren's, celiac, and other autoimmune causes that can co-exist with POTS.

Autonomic Neuroscience: Basic and Clinical 215 (2018) 78–82

Clinical Ophthalmology 2020:14

HLA association in POTS

	Phenotype frequency as no. (percentage)			Statistical analysis			
				POTS versus epilepsy controls		POTS versus healthy controls	
HLA allele or haplotype	POTS	Epilepsy controls	Healthy controls	OR (95% CI)	P_C[a]	OR (95% CI)	P_C[a]
Total (n=17)							
DQB1*06:09	7/17 (41%)	17/210 (8%)	36/485 (7%)	7.9 (2.7–23.5)	**8.9×10^{-3}**	8.7 (3.1–24.3)	**3.2×10^{-4}**
C*03:02	8/17 (47%)	32/210 (15%)	71/485 (15%)	4.9 (1.8–13.8)	0.075	5.2 (1.9–13.9)	**0.043**
DRB1*13:02	7/17 (41%)	28/210 (13%)	83/485 (17%)	4.6 (1.6–12.9)	0.23	3.4 (1.3–9.2)	0.65
B*58:01	7/17 (41%)	31/210 (15%)	59/485 (12%)	4 (1.4–11.4)	0.51	5.1 (1.9–13.8)	0.15
A*33:03	7/17 (41%)	56/210 (27%)	140/485 (29%)	1.9 (0.7–5.3)	>0.99	1.7 (0.6–4.6)	0.65
Haplotype#1*	7/17 (41%)	16/210 (8%)	32/485 (7%)	8.5 (2.8–25.3)	**2.6×10^{-3}**	9.9 (3.5–27.8)	**6.5×10^{-4}**
Haplotype#2*	6/17 (35%)	13/210 (6%)	29/485 (6%)	8.3 (2.6–25.9)	**6.4×10^{-3}**	8.5 (3.0–24.8)	**3.2×10^{-3}**
Patients with antibodies to both 2AR and 2AR (n=13)							
DQB1*06:09	7/13 (54%)	17/210 (8%)	36/485 (7%)	13.2 (4.0–43.9)	**1.2×10^{-3}**	14.6 (4.6–45.6)	**4.0×10^{-4}**
C*03:02	8/13 (62%)	32/210 (15%)	71/485 (15%)	8.9 (2.7–28.9)	**8.0×10^{-3}**	9.3 (3.0–29.3)	**4.2×10^{-3}**
DRB1*13:02	7/13 (54%)	28/210 (13%)	83/485 (17%)	7.6 (2.4–24.2)	**0.037**	5.7 (1.9–17.2)	0.11
B*58:01	7/13 (54%)	31/210 (15%)	59/485 (12%)	6.7 (2.1–21.4)	0.086	8.4 (2.7–25.9)	**0.021**
A*33:03	7/13 (54%)	56/210 (27%)	140/485 (29%)	3.2 (1.0–10.0)	>0.99	2.9 (0.9–8.7)	0.65
Haplotype#1*	7/13 (54%)	16/210 (8%)	32/485 (7%)	14.1 (4.2–47.1)	**3.4×10^{-4}**	16.5 (5.2–52.0)	**7.8×10^{-5}**
Haplotype#2*	6/13 (46%)	13/210 (6%)	29/485 (6%)	13.0 (3.8–44.3)	**1.2×10^{-3}**	13.5 (4.3–42.7)	**5.6×10^{-4}**

Annals of Clinical and Translational Neurology 2019; 6(5): 962–967

Autonomic Metabotrophic Receptors

Muscarinic receptors:

Receptor	G-protein	Messenger	Effect	Example Functions
M_1	G_q	Phospholipase C & IP_3 increased	elevated Ca^{++}	salivary gland & stomach secretion
M_2	G_i	cAMP decreased	decreased Ca^{++} influx & increased K^+ efflux	decreased heart rate and force
M_3	G_q	Phospholipase C & IP_3 increased	elevated Ca^{++}	constriction of vessels & bronchioles detrusor contraction (micturition)
M_4	G_i	cAMP decreased	decreased Ca^{++} influx & increased K^+ efflux	inhibitory effects
M_5	G_q	Phospholipase C & IP_3 increased	elevated Ca^{++}	present in CNS

Adrenergic receptors:

Receptor	G-protein	Messenger	Effect	Example Functions
α_1	G_q	Phospholipase C & IP_3 increased	elevated Ca^{++}	cutaneous & GI vasoconstriction; urethral sphincter contraction
α_2	G_i	cAMP decreased	decreased Ca^{++} influx	inhibition of neurotransmitter release
β_1	G_s	cAMP increased	elevated Ca^{++}	increased cardiac output (rate & force)
β_2	G_s G_i	cAMP increased cAMP decreased	elevated Ca^{++} decreased Ca^{++} influx & increased K^+ efflux	constriction of gut sphincters muscle vessel dilation; detrusor relaxation
β_3	G_s G_i	cAMP increased cAMP decreased	elevated Ca^{++} decreased Ca^{++} influx & increased K^+ efflux	adipose tissue lipolysis detrusor relaxation

http://vanat.cvm.umn.edu/ans/pages/Physiol.html

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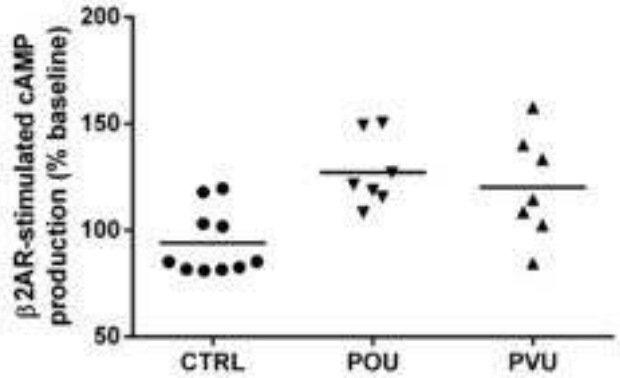
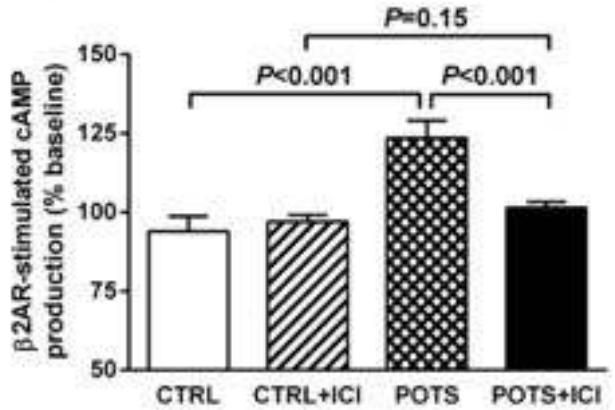
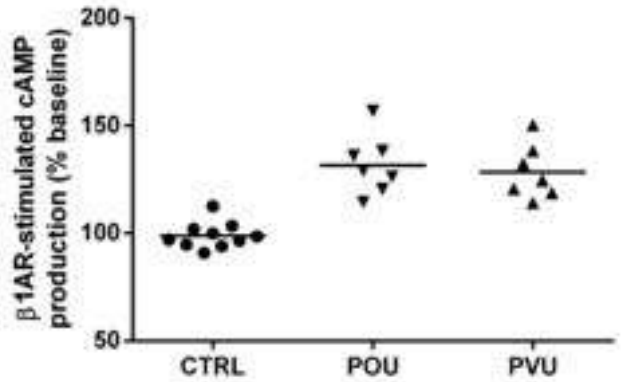
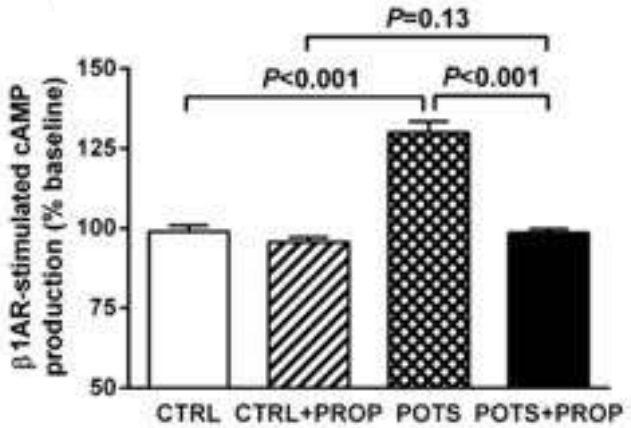
Autoimmune Basis for Postural Tachycardia Syndrome

Hongliang Li, MD, PhD; Xichun Yu, MD; Campbell Liles, BS; Muneer Khan, MD; Megan Vanderlinde-Wood, MD; Allison Galloway, MD; Caitlin Zillner, BS; Alexandria Benbrook, BS; Sean Reim, BS; Daniel Collier, BS; Michael A. Hill, PhD; Satish R. Raj, MD; Luis E. Okamoto, MD; Madeleine W. Cunningham, PhD; Christopher E. Aston, PhD; David C. Kem, MD









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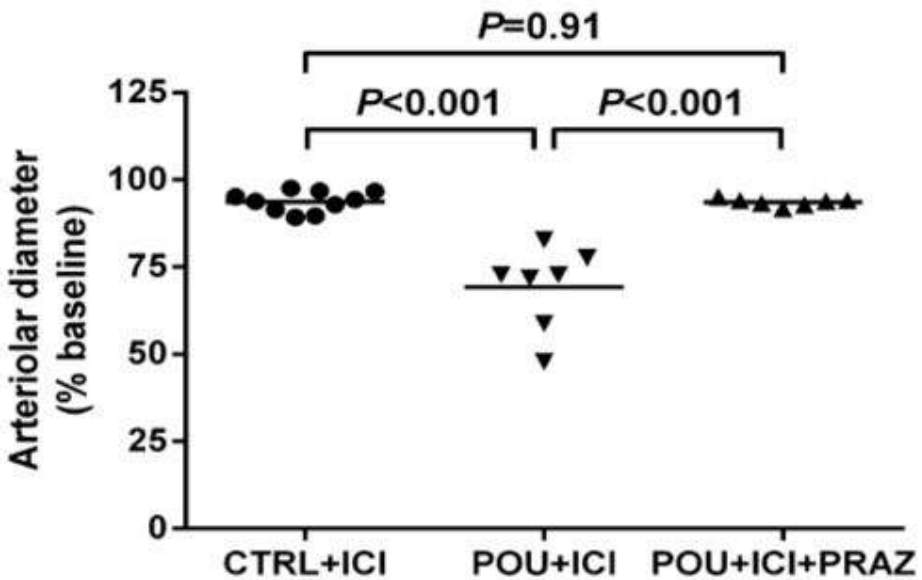
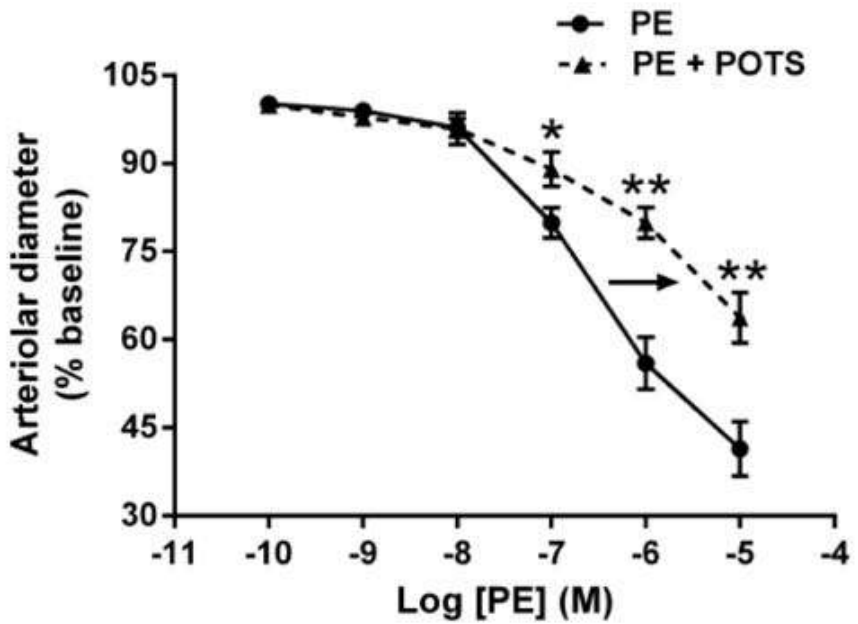
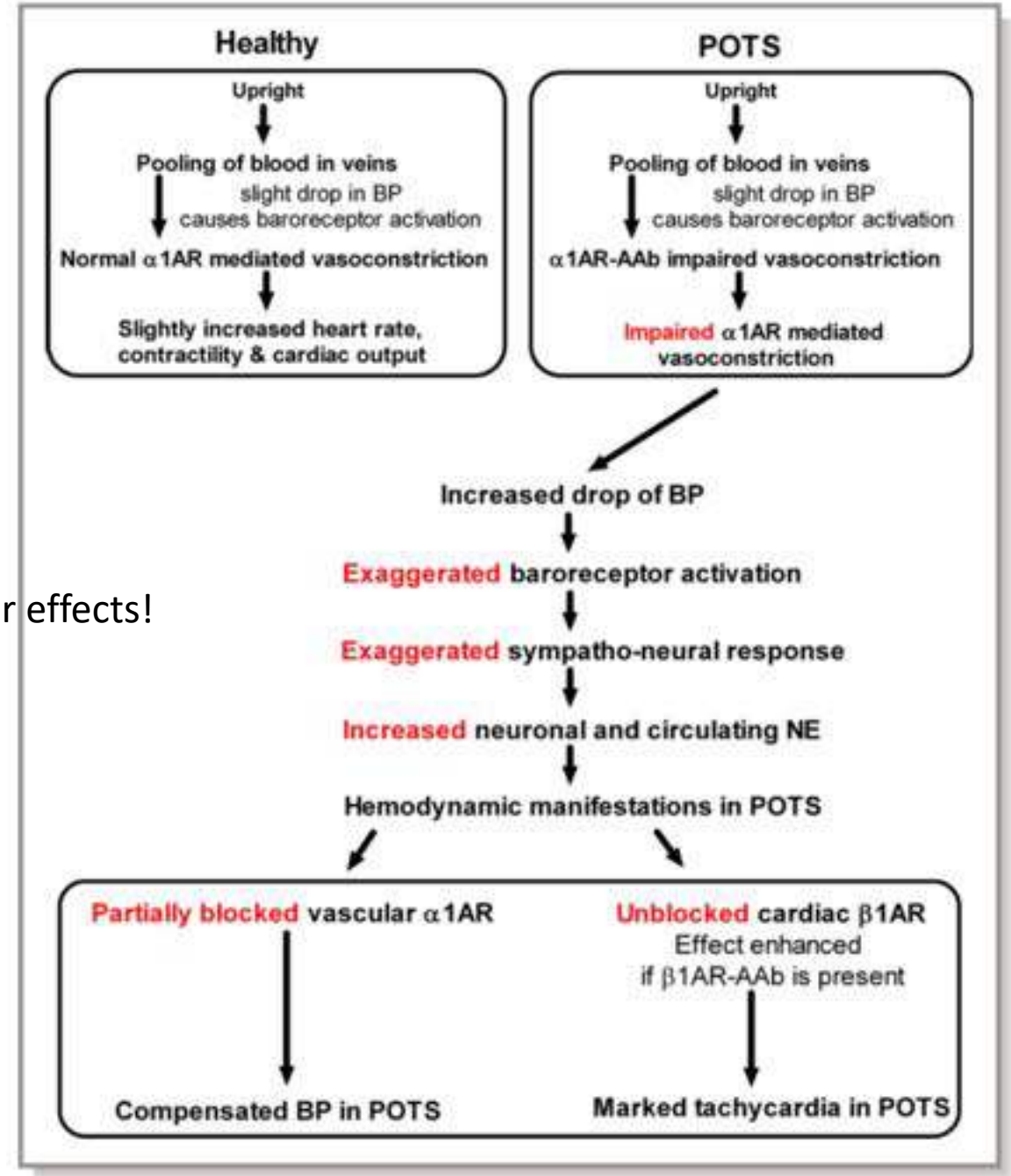




Not just vascular effects!



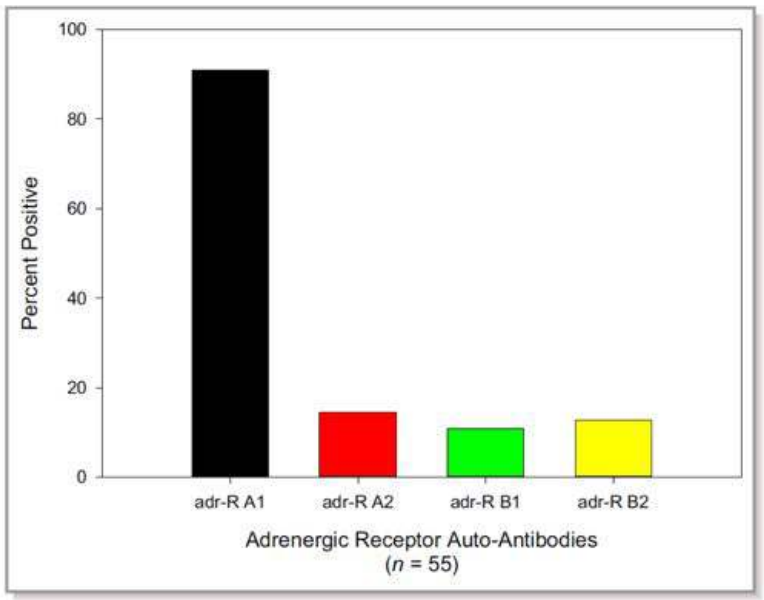
J Am Heart Assoc. 2014;3:e000755

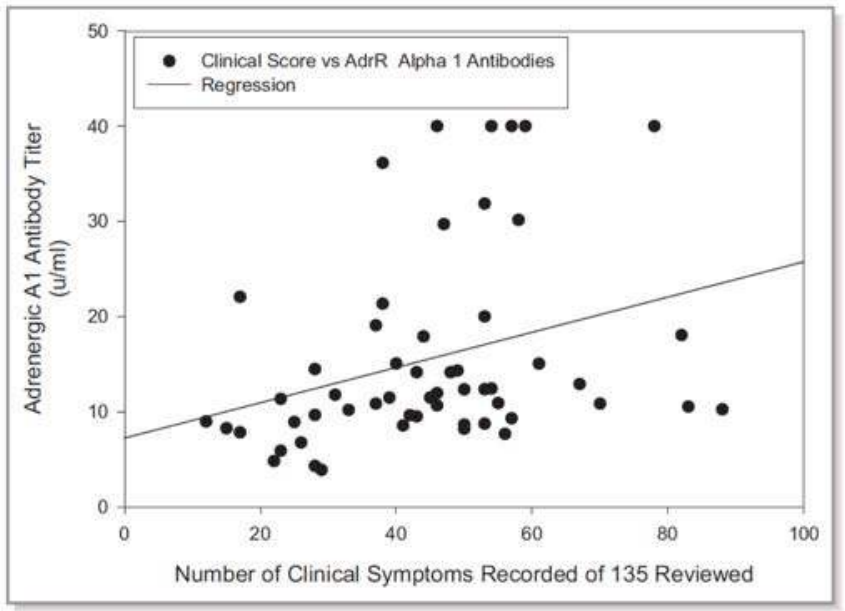
Table 2 Test positivity (direct-activating and/or ligand-modulating activity) among patients diagnosed with POTS

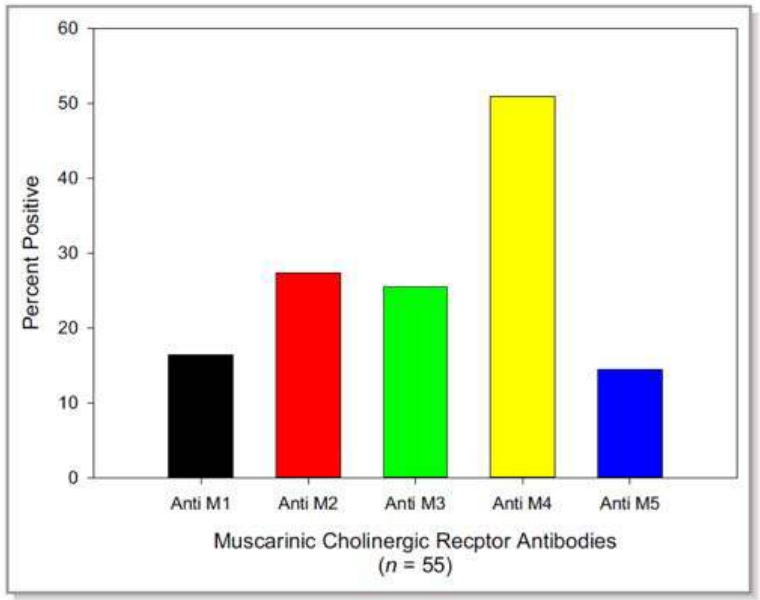
Patient no.	α1AR Ab		β1AR Ab		β2AR Ab
	Activating	Modulating	Activating	Modulating	Activating
1		×			×
2			×	×	
3		×	×	×	×
4	×		×	×	×
5	×		×	×	
6			×	×	×
7	×		×	×	
8	×		×	×	
9				×	×
10			×	×	×
11	×			×	×
12	×	×	×	×	×
13		×			×
14		×			×
15	×	×			
16		×	×	×	×
17	×	×	×	×	×
Total	8/17	8/17	11/17	13/17	12/17

Europace (2017) **19**, 1211–1219
doi:10.1093/europace/euw154

Adrenergic and Muscarinic Receptor antibodies by ELISA in POTS



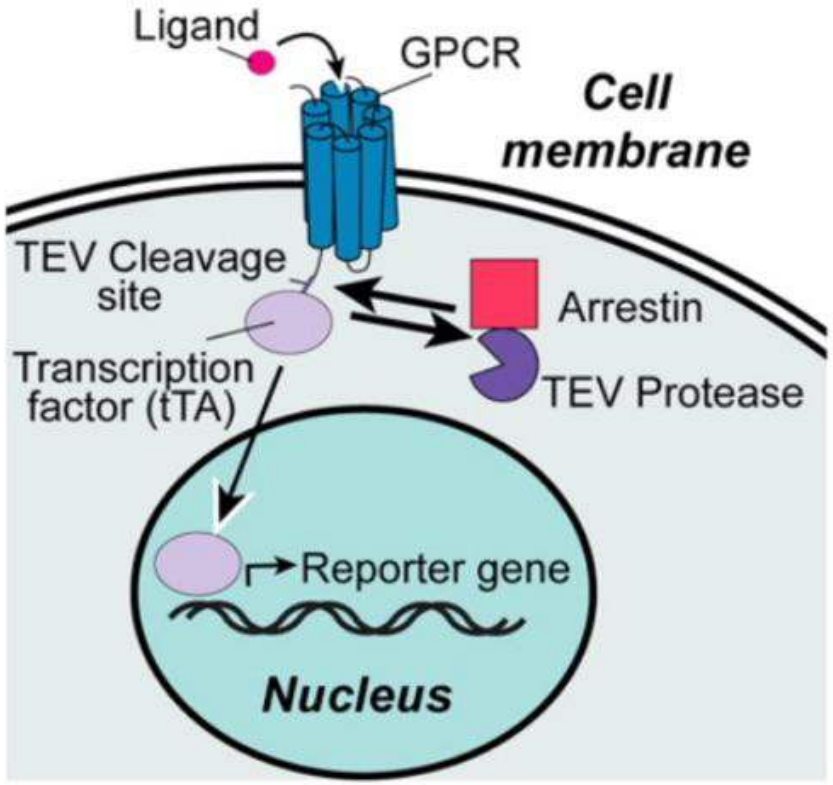




r=0.31, P=0.02

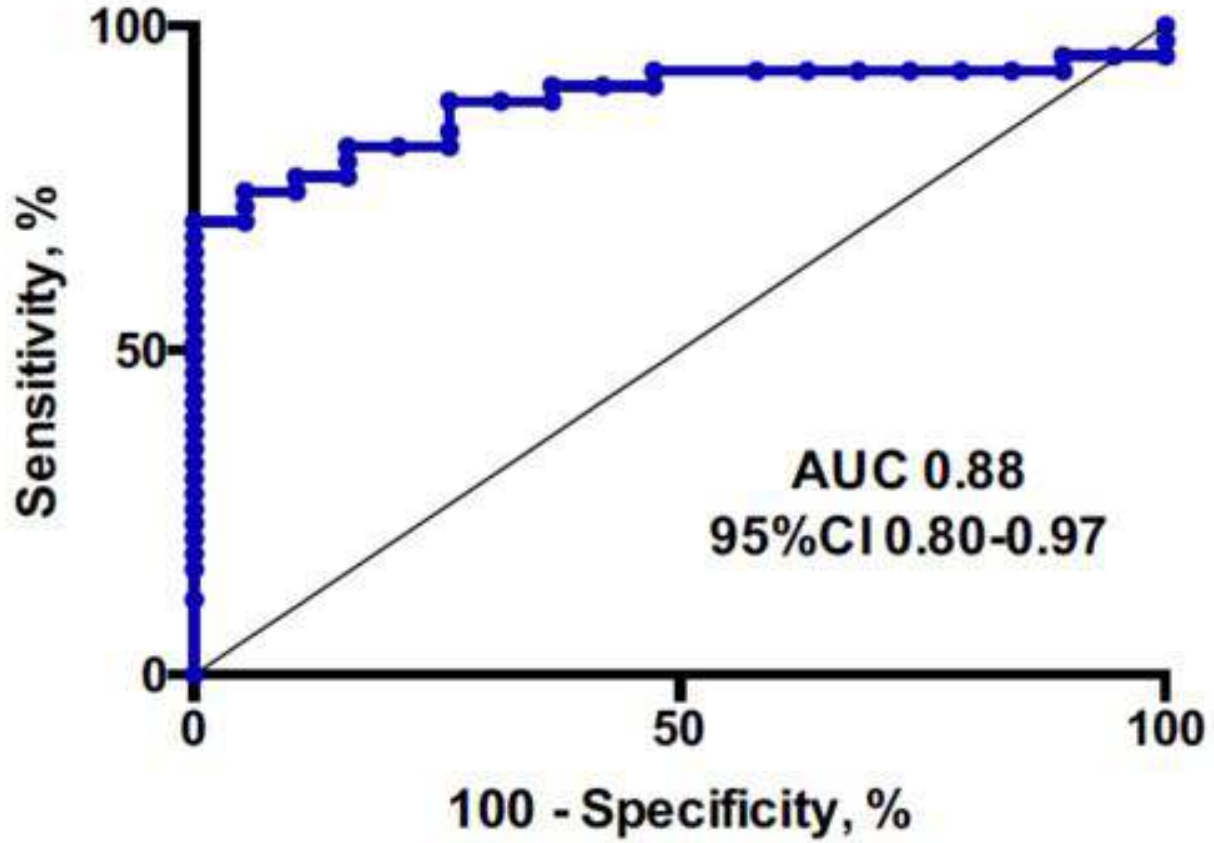
J Am Heart Assoc. 2019;8:e013602.

Novel assays for GPCR autoantibodies in POTS



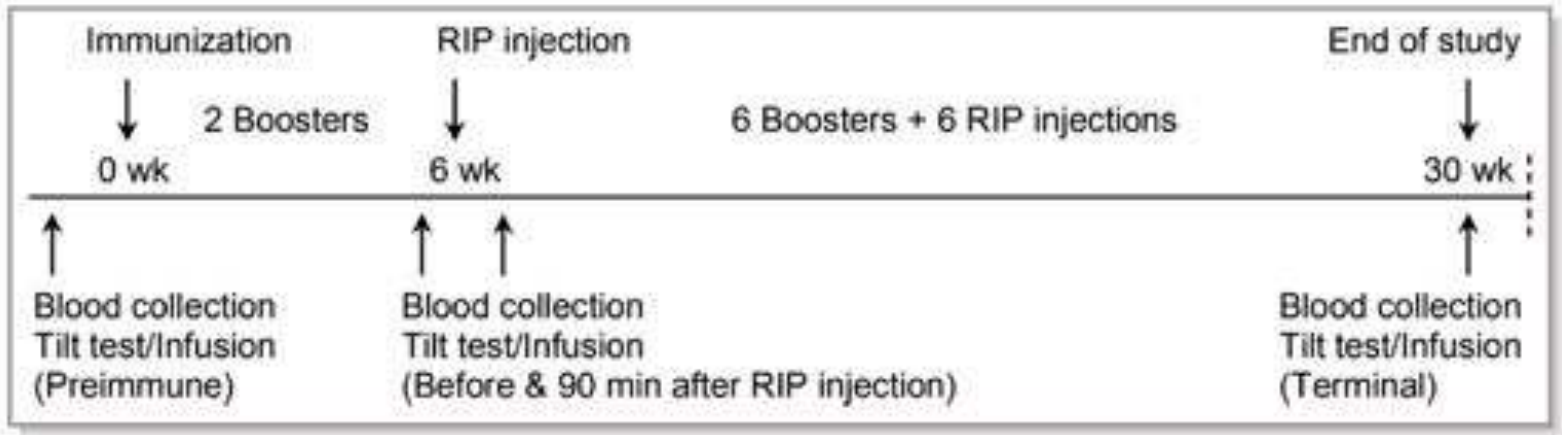
Tango FRET assay

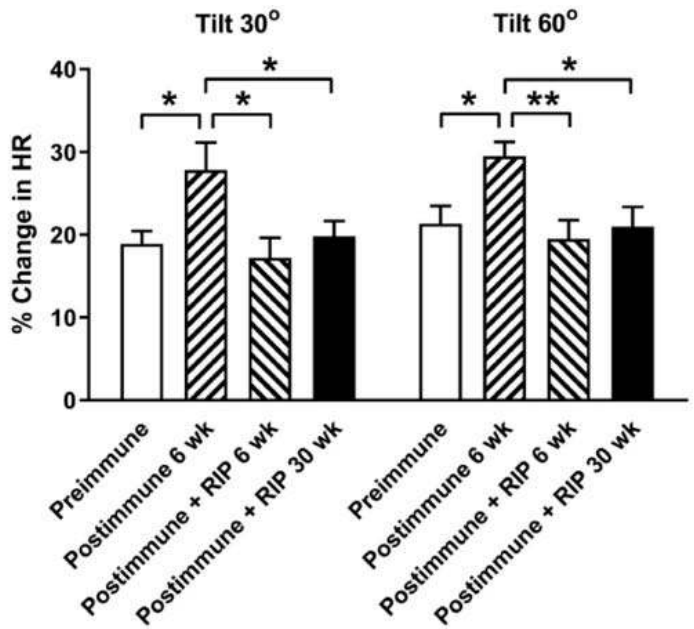
ROC: A1, B2, M2, opiod receptor-like 1

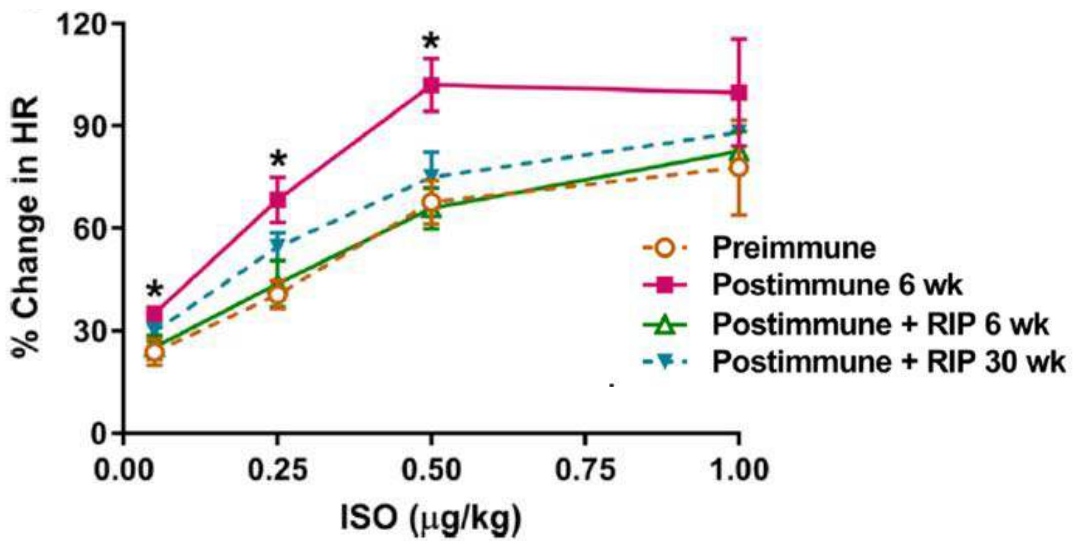


J Am Heart Assoc. 2020;9:e015989.

Adrenergic Autoantibody-Induced Postural Tachycardia Syndrome in Rabbits







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GPCR autoantibodies in other diseases

Disease	GPCR-AAB directed against ( ) - receptor	Activity
Idiopathic dilated cardiomyopathy	β1-adrenergic	agonistic
	muscarinic M2	agonistic
Peripartum cardiomyopathy	β1-adrenergic	agonistic
	muscarinic M2	agonistic
Chagas' cardiomyopathy	β1-adrenergic	agonistic
	muscarinic M2	agonistic
	β2-adrenergic	agonistic
Myocarditis	β1-adrenergic	agonistic
Electric cardiac abnormalities	β1-adrenergic	agonistic
	muscarinic M2	agonistic
	β2-adrenergic	agonistic
	serotoninergic 5HT4	n.d.
Refractory hypertension	α1-adrenergic	agonistic
Idiopathic pulmonary hypertension	α1-adrenergic	agonistic
	endothelin 1 ETA	agonistic
Malignant hypertension	angiotensin II AT1	agonistic
Preeclampsia	angiotensin II AT1	agonistic
	endothelin 1 ETA	agonistic
Orthostatic hypotension	β2-adrenergic	agonistic
	muscarinic M3	n.d.
Postural orthostatic tachycardia syndrome (POTS)	β1-adrenergic	agonistic
	β2-adrenergic	agonistic
	α1-adrenergic	agonistic
	muscarinic M2	agonistic
	angiotensin II AT1	agonistic
Diabetes mellitus type II	α1-adrenergic	agonistic
Vascular renal rejection	angiotensin II AT1	agonistic
Thromboangiitis obliterans	α1-adrenergic	agonistic
	endothelin 1 ETA	agonistic
	angiotensin II AT1	agonistic
Systemic lupus erythematosus	serotoninergic 5HT4	antagonistic
Allergic asthma	β2-Adrenergic	inhibitory
Open angle glaucoma	β2-Adrenergic	agonistic
Vascular dementia / Alzheimer's dementia	α1-adrenergic	agonistic
	β2-adrenergic	agonistic
	endothelin 1 ETA	agonistic
	angiotensin II AT1	n.d.
Benign prostate hyperplasia	endothelin 1 ETA	agonistic
Complex regional pain syndrome (CRPS)	muscarinic M2	agonistic
	β2-adrenergic	agonistic
Sjögren's syndrome	muscarinic M3	agonistic
Fatigue syndrome	β2-adrenergic	agonistic
	muscarinic M2	agonistic
	muscarinic M3	n.d.
	muscarinic M4	n.d.
Post cancer chemotherapy	α1-adrenergic	agonistic
	angiotensin 1–7 Mas	agonistic
Periodontitis	β1-adrenergic	agonistic

[Frontiers In Bioscience, Landmark, 24, 48-95, Jan 1, 2019]

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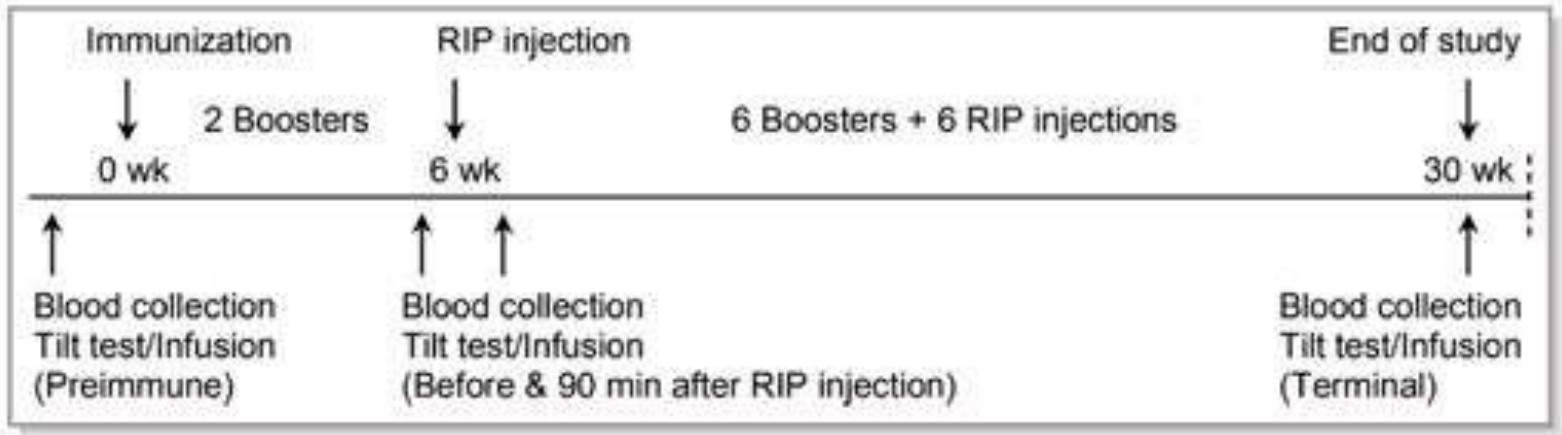

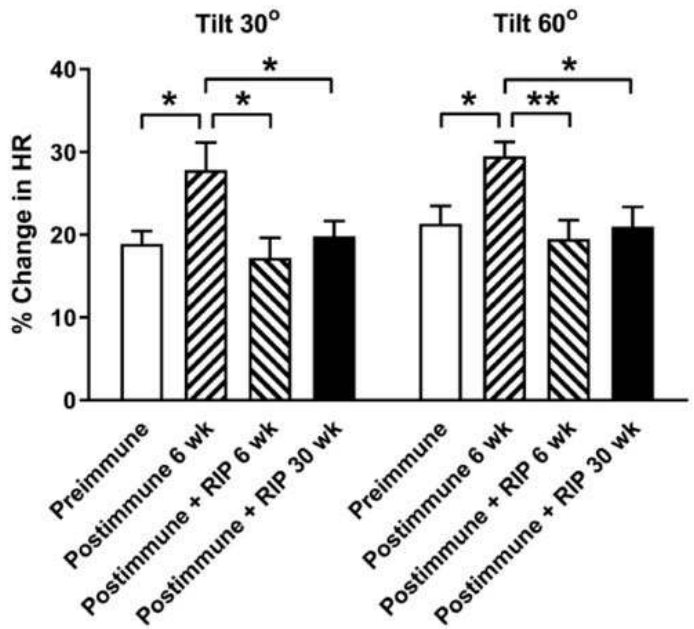

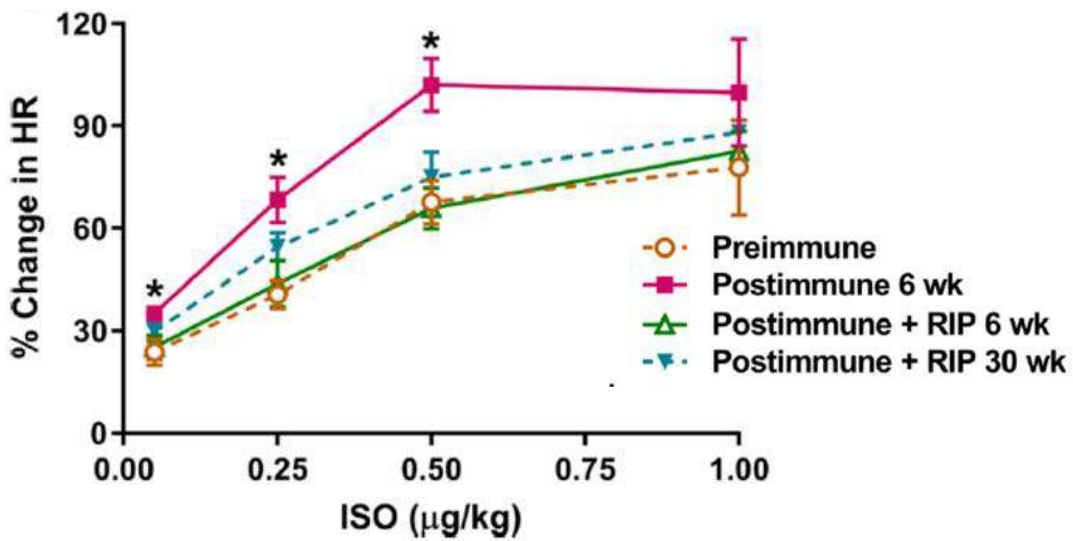

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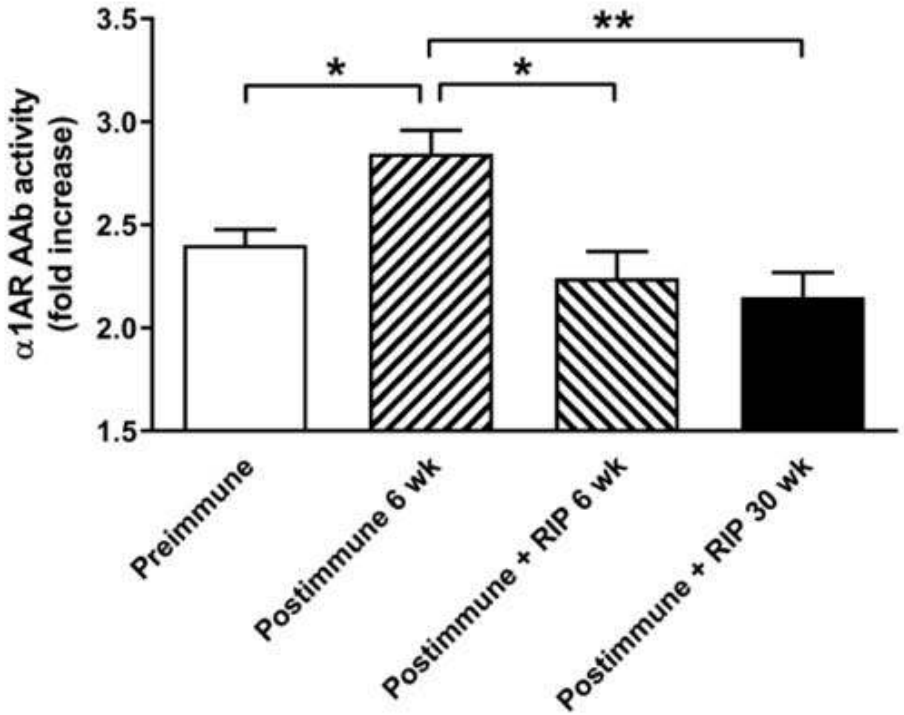

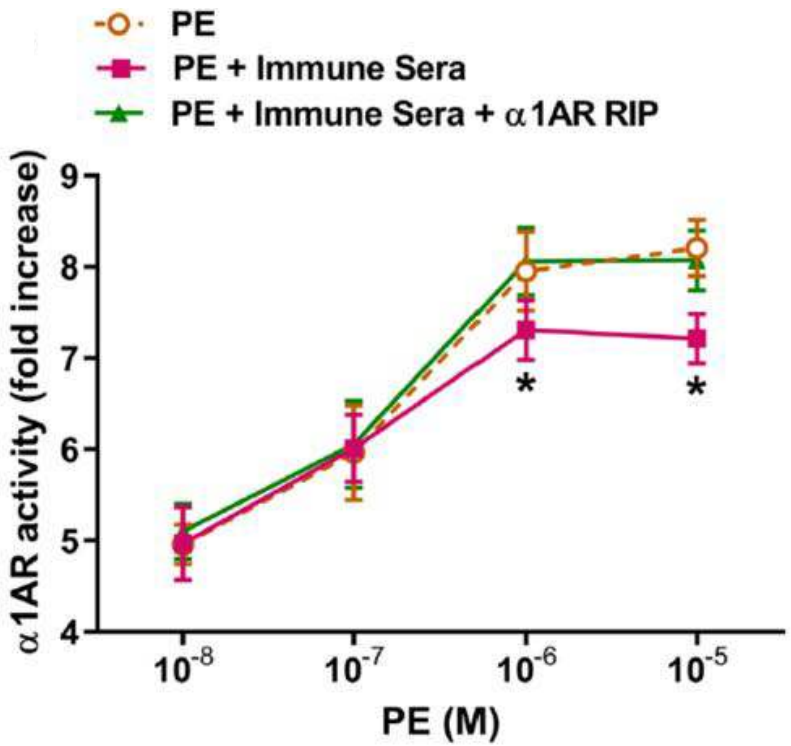

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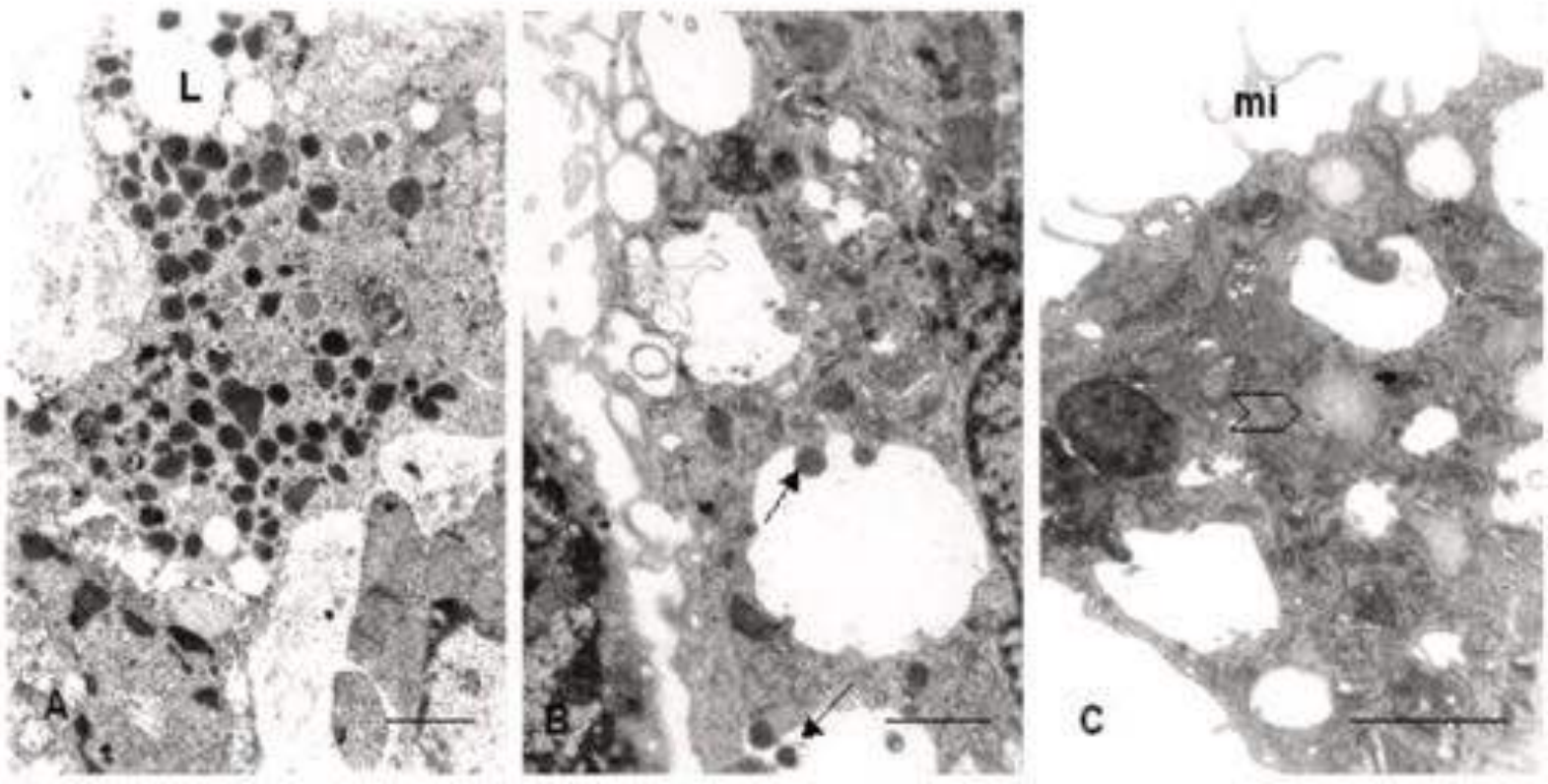
Whitebsky's postulates in autoimmunity

Witebsky's postulates (1957) [3••]
1. The direct demonstration of free circulating antibodies OR of cell bound antibodies that are active at body temperature
2. The recognition of the specific antigen against which this antibody is directed
3. The production of antibodies against the same antigen in experimental animals
4. The appearance of pathological changes in the corresponding tissues of an actively sensitized experimental animal that are basically similar to those in human disease

•• Witebsky E, Rose NR, Terplan K, Paine JR, Egan RW. Chronic thyroiditis and autoimmunization. *J Am Med Assoc* 1957;164:1439–

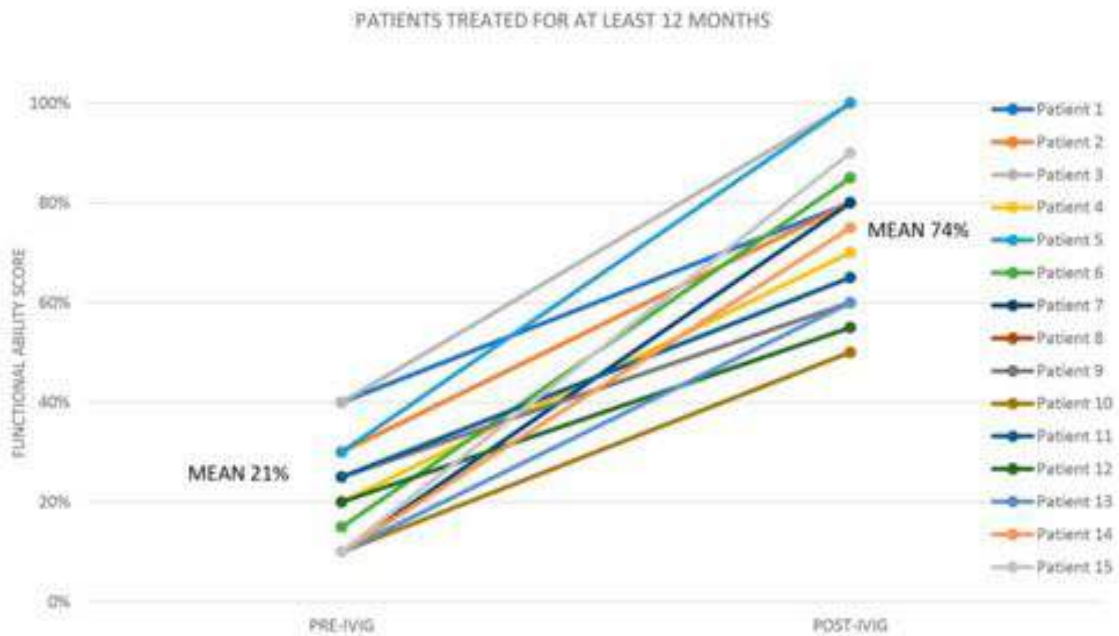
Autoantibodies against G-Protein-Coupled Receptors Modulate Heart Mast Cells

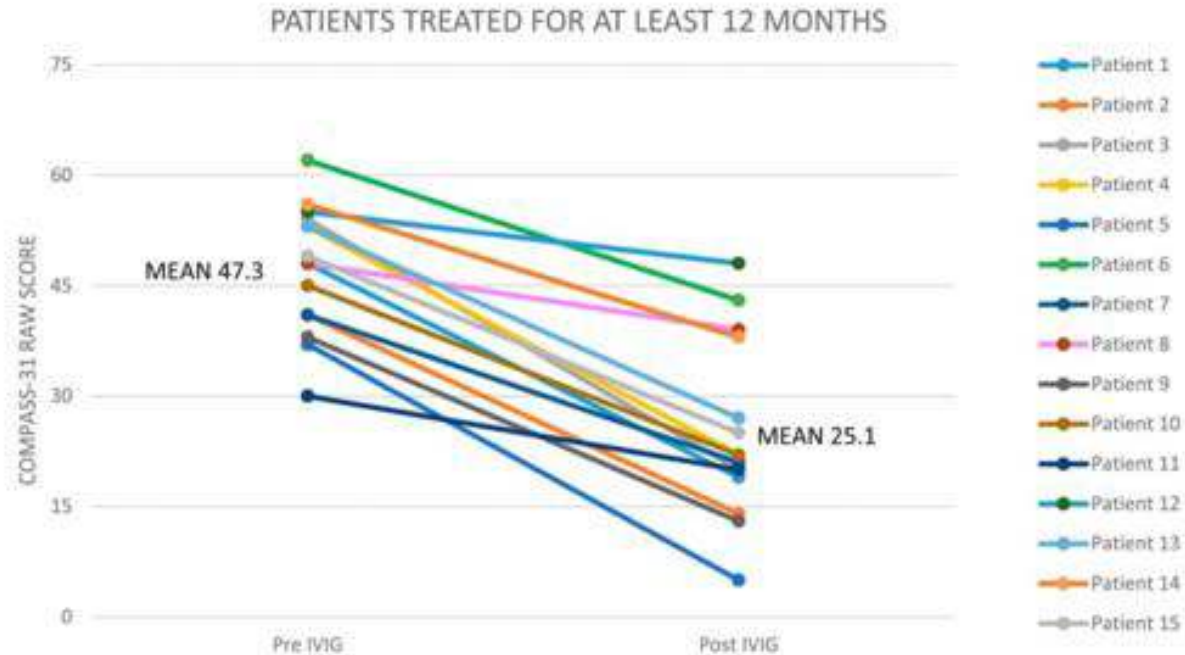


Mature mast cell Alpha-1R/Angiotensin 2R

Cellular & Molecular Immunology. 2007;4(2):127-133.

Intravenous Immunoglobulin Therapy in Refractory Autoimmune Dysautonomias: A Retrospective Analysis of 38 Patients

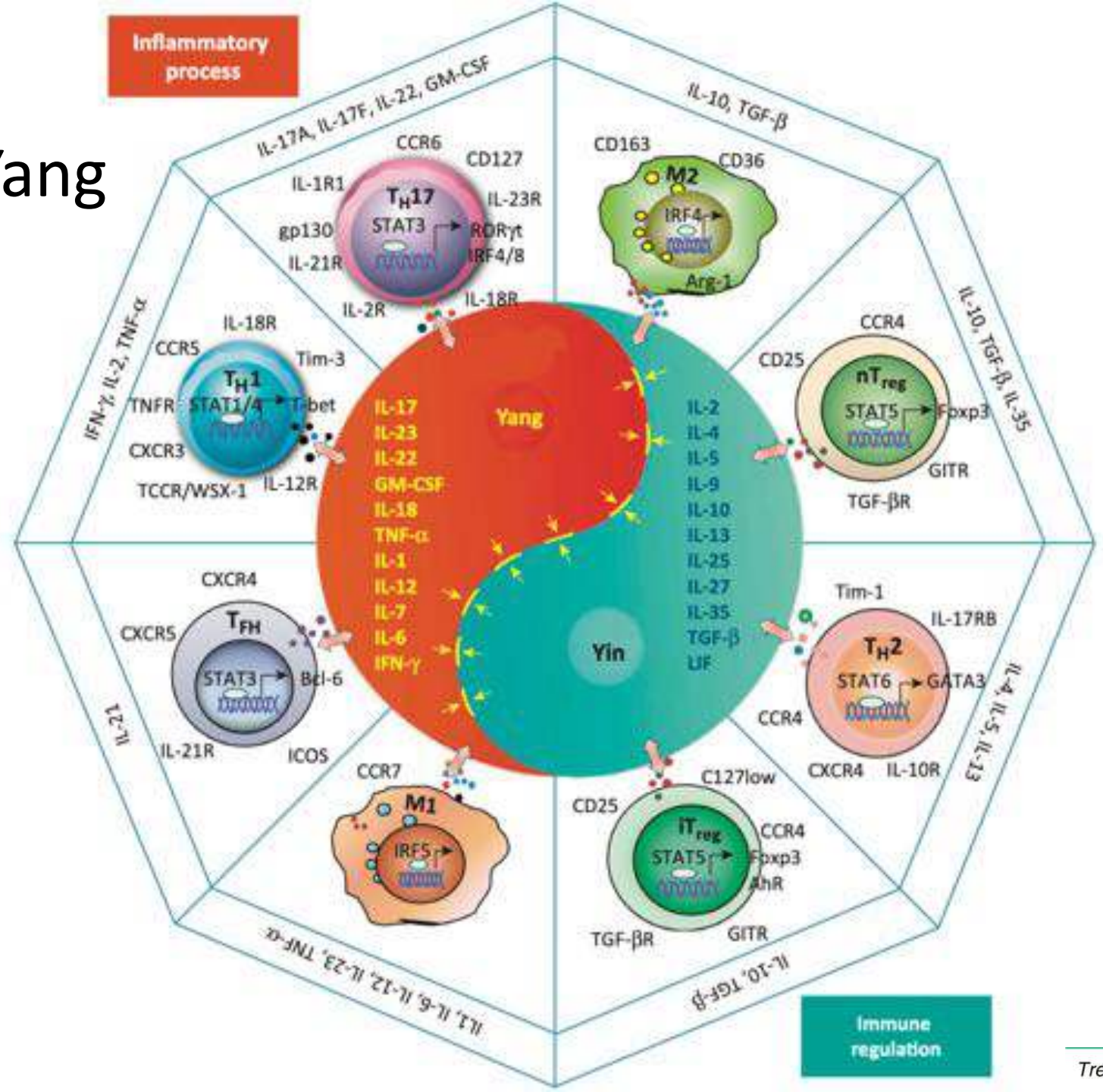




American Journal of Therapeutics 0, 1–13 (2018)

- Do the GPCR autoantibodies have anything to do with POTS pathophysiology?
- How do GPCR autoantibodies develop?
- Are we close to a reliable commercial assay to diagnose autoimmune POTS?
- What subset of POTS patients will respond (if at all) to immunotherapy?

The Yin and Yang Of Cytokines



Trends in Immunology March 2013, Vol. 34, No. 3

Inflammatory cytokine abnormalities in POTS

Cytokine/Chemokine	POTS Patients (n = 34) (pg/mL)	Normal (pg/mL)	Major Function
IL 1β	332 ± 100	<10	Regulates cell proliferation
IL 10	16 ± 3.6	<6	Inhibitory to T helper cells
IL 21	1918 ± 410	<200	Controls NK and T cells
TNFα	342 ± 78	<3	Regulates inflammation
INFγ	226 ± 62	<5	Antiviral
CD30	193 ± 59	<10	Regulates cell proliferation
CD40 L	119 ± 11	350–90	Recruits leukocytes
RANTES (CCL5)	995 ± 123	5000–6100	Chemotactic for T cells
P-Selectin	12,540 ± 1094	10,000–130,000	Recruits leukocytes
MCP-1	78 ± 5	65–1025	Recruits monocytes
AdR A1 antibodies	16.6 U/mL	<7 U/mL	Autoantibody
AChR M4 Abs	11.2 U/mL	<7 U/mL	Autoantibody

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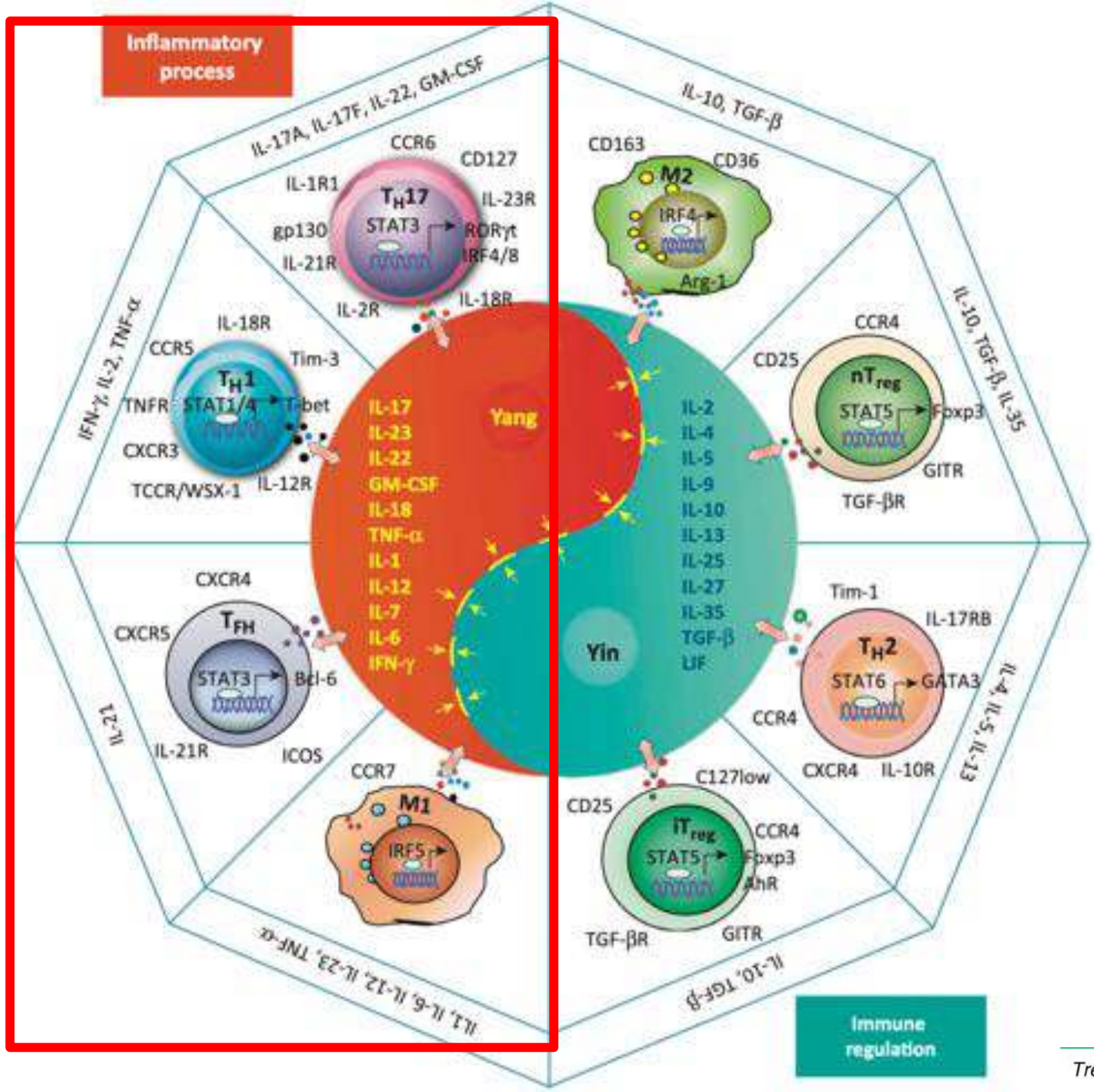
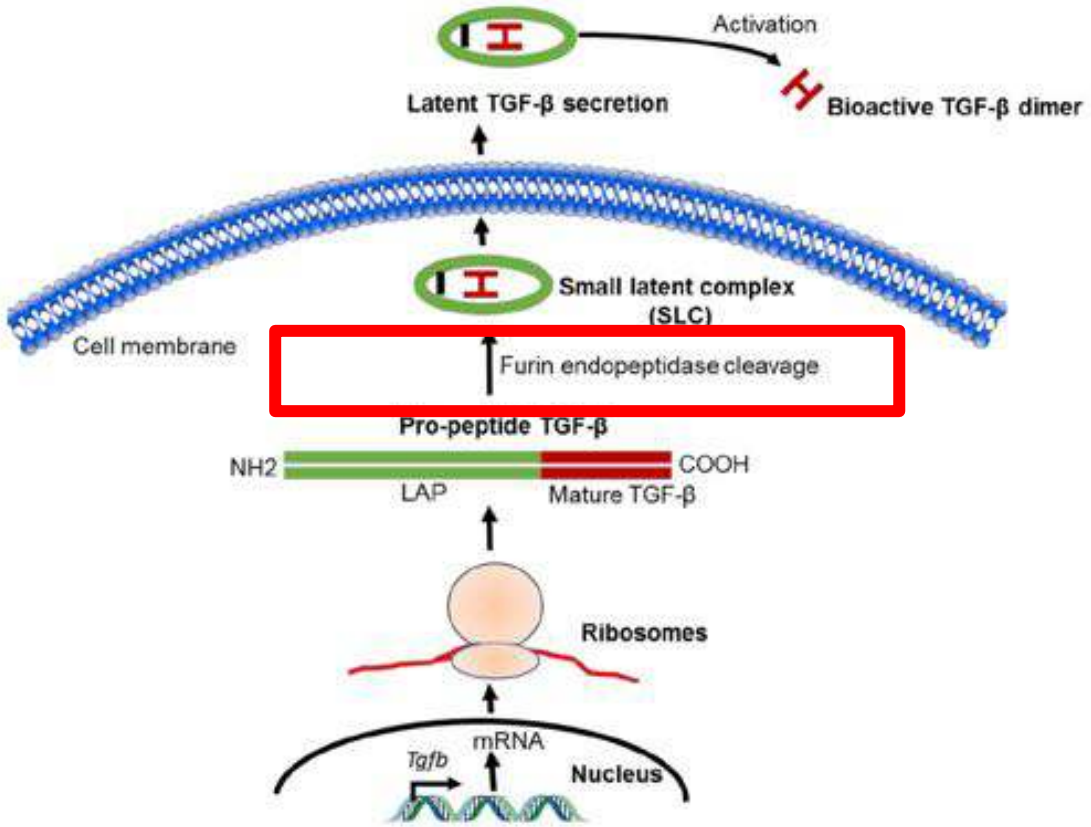
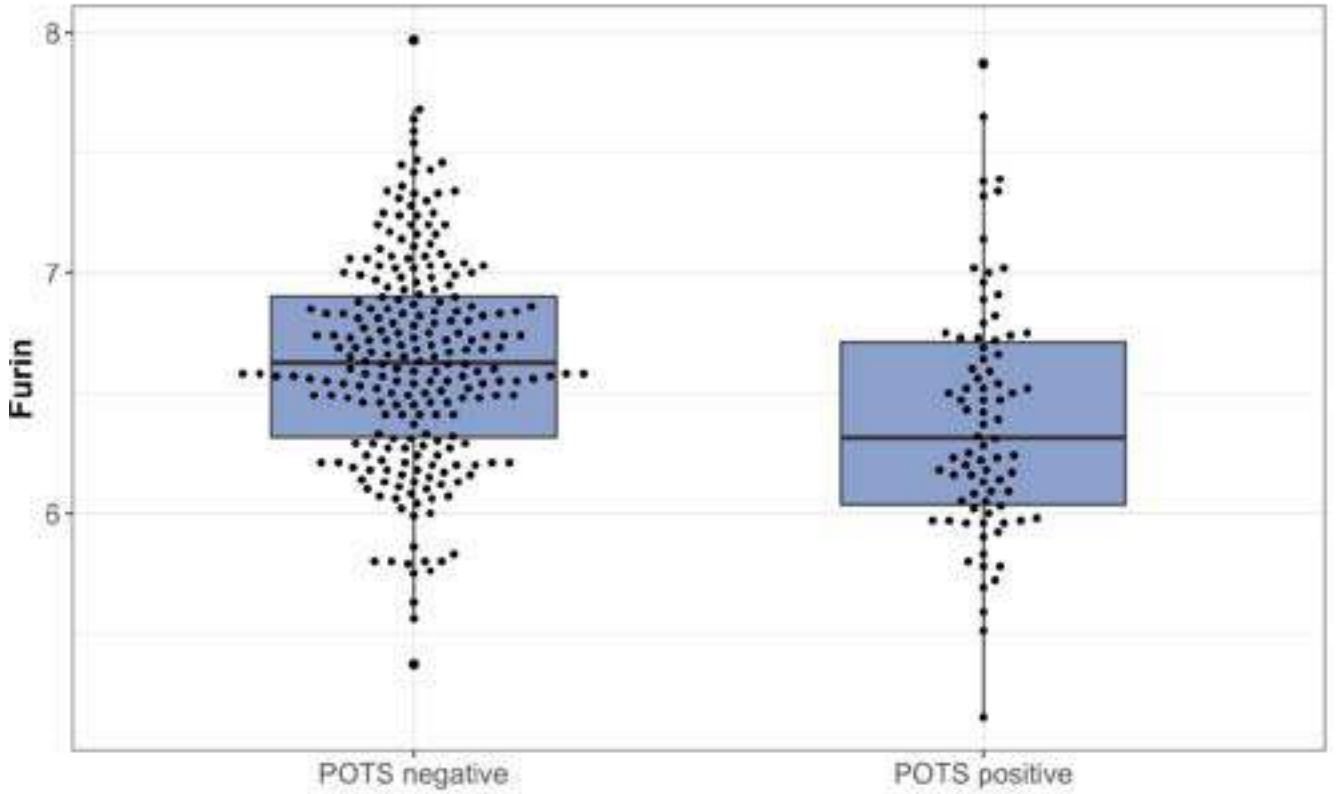
Cytokine/ Chemokine	POTS (n = 35) (pg/mL)	Non-POTS (n = 35) (pg/mL)	p Value	Source	Major Function
CD30	3638 ± 822	160 ± 12	$p < 0.0002$	Activated T and B	Regulates cell proliferation
CD40	340 ± 165	452 ± 171	ns	B cell, Mac	TLR7 PLT-neutrophil tethering
CD40 L (CD154)	31 ± 13	6.7 ± 0.8	ns	Platelets, Mono	Recruits neutrophils and monocytes
IL 1β	38 ± 8	4.4 ± 0.9	$p < 0.0001$	Mono/Mac, PLTs	Proinflammatory
IL-6	119 ± 18	58 ± 9	$p < 0.003$	Th Cells, Mac	Differentiates B cells to plasma cells
IL-8 (CXCL8)	145 ± 49	157 ± 25	ns	Mono, Neutro	Chemotaxis, proinflammatory
IL 10	24 ± 4	5.5 ± 1.0	$p < 0.0001$	T cell	Anti-inflammatory
IL-17	93 ± 20	4.2 ± 0.7	$p < 0.0001$	Th17	Proinflammatory
IL-18	207 ± 67	21 ± 9	$p < 0.009$	Mono	Proinflammatory, IL-1 family
IL 21	9025 ± 1875	2937 ± 517	$p < 0.003$	T cell	Controls NK and T cells
INFα	0.06 ± 0.04	223 ± 67	$p < 0.002$	Leukocytes	Anti-viral, phagocyte cell activation
INFβ	8219 ± 2230	6334 ± 3267	ns	Fibroblasts	Anti-viral, anti-proliferative
INFγ	8.5 ± 1.7	1.2 ± 0.2	$p < 0.0001$	NK, Th_1	Antiviral, increases Neut and Mono function
MCP1 (CCL2)	441 ± 102	13 ± 2	$p < 0.0002$	Endo, PLT	Recruits monocytes
RANTES (CCL5)	13706 ± 3022	517 ± 297	$p < 0.0001$	Platelet, NK, T	Chemotactic for T cells
TNFα	972 ± 250	506 ± 120	ns	Mono, NK	Proinflammatory

Elevations of cytokines/chemokines are in red font. Decreases in cytokines/chemokines in blue font.

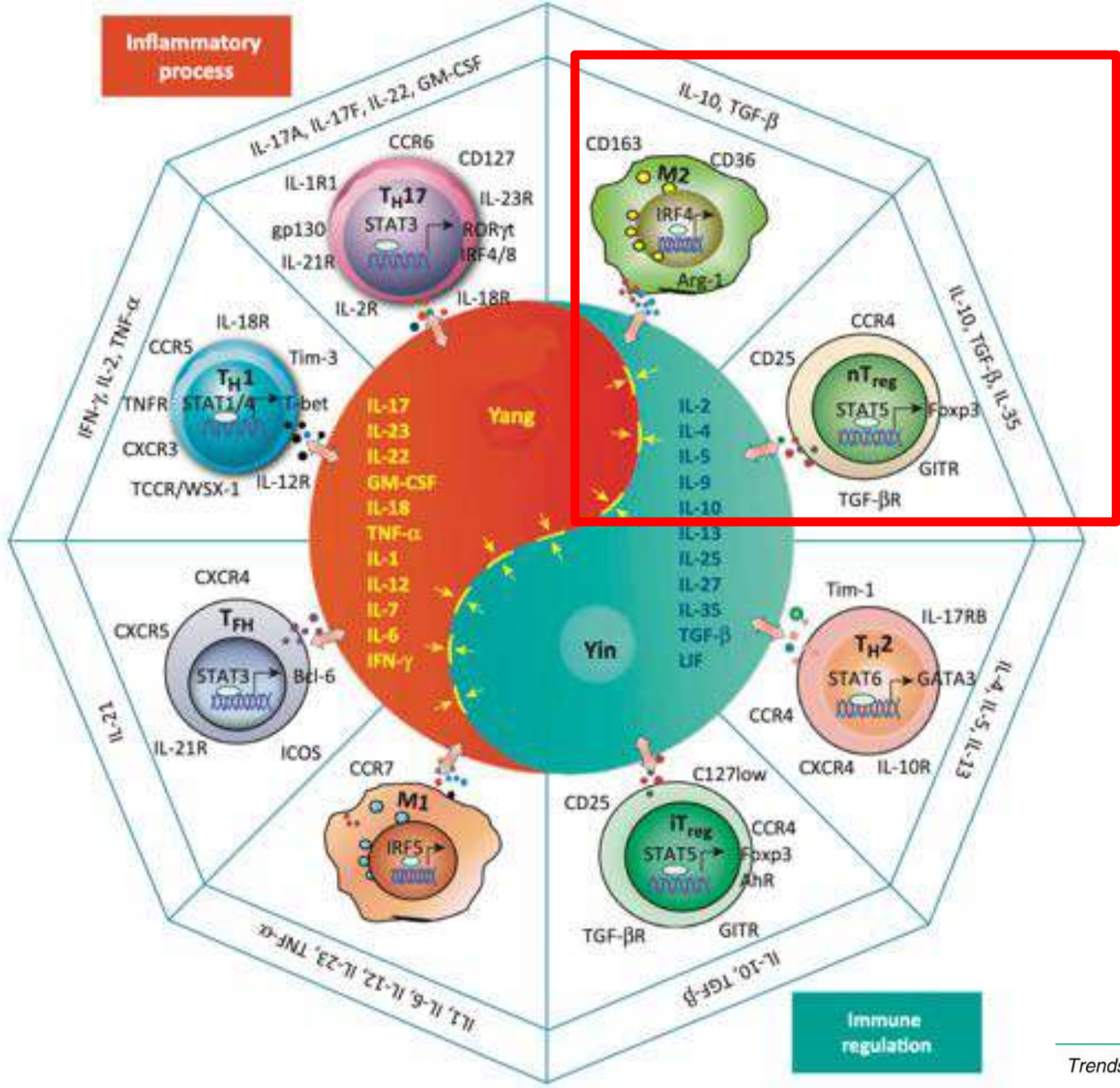

Inflammatory process
IL-17A, IL-17F, IL-22, GM-CSF
CCR6
CD127
IL-1R1
IL-23R
T_H17
STAT3
RORγt
IRF4/8
gp130
IL-21R
IL-2R
IL-18R
IL-18R
CCR5
Tim-3
T_H1
TNFR
STAT1/4
T-bet
CXCR3
TCCR/WSX-1
IL-12R
IFN-γ, IL-2, TNF-α
IL-17
IL-23
IL-22
GM-CSF
IL-18
TNF-α
IL-1
IL-12
IL-7
IL-6
IFN-γ
Yang
Yin
IL-2
IL-4
IL-5
IL-9
IL-10
IL-13
IL-25
IL-27
IL-35
TGF-β
LIF
CD163
CD36
M2
IRF4
Arg-1
IL-10, TGF-β
CCR4
CD25
nT_{reg}
STAT5
Foxp3
GITR
TGF-βR
IL-10, TGF-β, IL-35
CXCR4
T_{FH}
CXCR5
STAT3
Bcl-6
IL-21R
ICOS
IL-21
Tim-1
IL-17RB
T_H2
STAT6
GATA3
CCR4
CXCR4
IL-10R
IL-4, IL-5, IL-13
CCR7
M1
IRF5
IL-1, IL-6, IL-12, IL-23, TNF-α
C127low
CD25
iT_{reg}
STAT5
CCR4
Foxp3
AhR
TGF-βR
GITR
IL-10, TGF-β
Immune regulation

Furin levels and POTS





Immunol Res (2017) 65:987–994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Foxp3
AhR
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GITR
IL-1, IL-6, IL-12, IL-23, TNF-α
IL-10, TGF-β
Immune regulation

Summary: Immune dysregulation in POTS

- Infectious triggers are common
- Co-existent autoimmune or immune dysregulation is more common
- Autoantibodies are more common that include
  - Adrenergic receptors
  - Muscarinic receptors
  - Angiotensin 2 receptor
- Inflammatory cytokine abnormalities may be present in POTS
- No randomized clinical trials have been done for immunotherapy in POTS (underway)

UC San Diego Health

DYSAUTONOMIA INTERNATIONAL

AWARENESS ADVOCACY ADVANCEMENT

SCAN ME

POTS Study

Time-based eating intervention that may improve the health and quality of life of people with postural orthostatic tachycardia syndrome (POTS).

Long COVID Study

Therapy for patients with post-COVID-19 postural orthostatic tachycardia syndrome (POTS).

TO PARTICIPATE, CONTACT US FOR MORE INFO

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