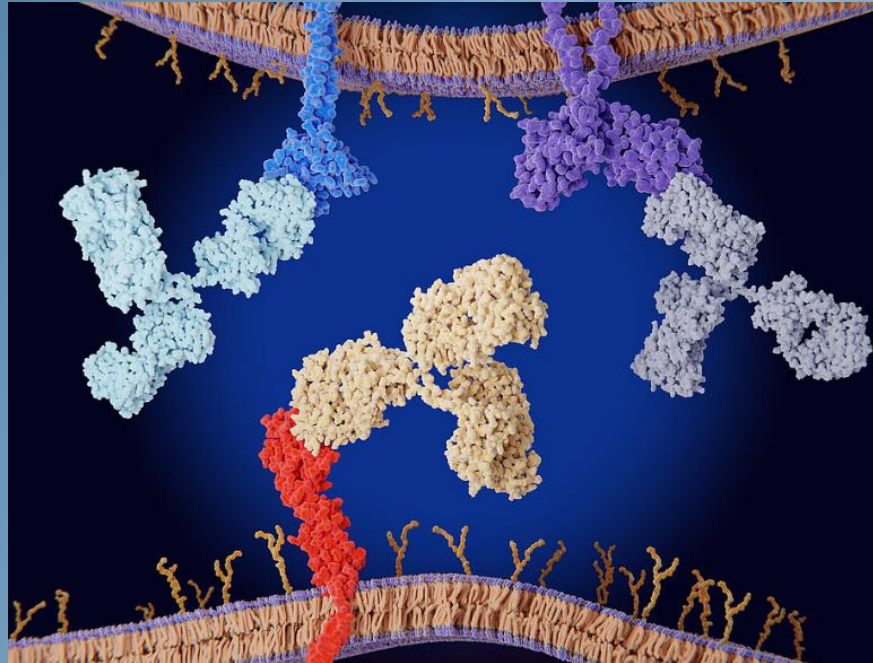


Autoimmune Antibodies: A Missing Piece In M.E., POTS And Long COVID?



Autoantibodies



Autoantibodies are well known and widely used in diagnostic assessments for e.g. Lupus, Rheumatoid Arthritis, Graves' Disease, Diabetes Type 1, ...

Have all the facts been told?

NO !

There is another class of autoantibody biomarkers that has not received enough clinical attention:

Autoantibodies against GPCRs (G* protein-coupled receptors)

* The "G" stands for **guanine**, referring to the [guanine nucleotides](#) GDP (guanosine diphosphate) and GTP (guanosine triphosphate) that these proteins bind and use as molecular switches to transmit signals inside the cell

Latest NEWS from today!



ME/CFS: Patienten packen aus

Ignoriert, stigmatisiert, für empfindlich erklärt: Betroffene schildern ihre Erfahrungen mit ME/CFS. Von ärztlichem Versagen und der Hoffnung auf gerechte Versorgung.



ME/CFS: Patients speak up

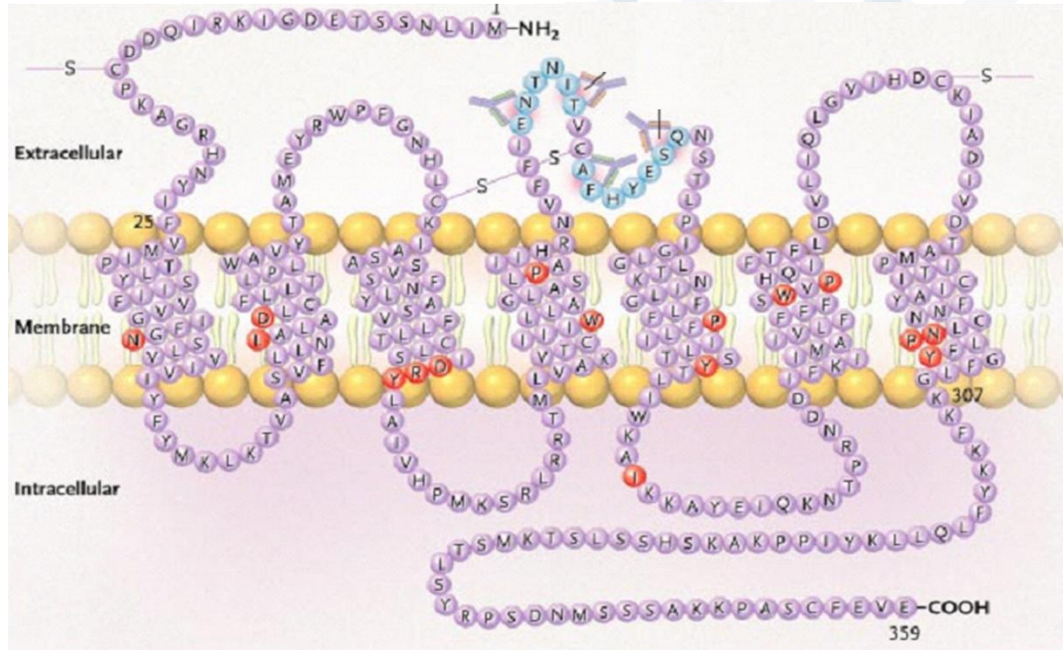
Ignored, stigmatised, declared sensitive: Patients affected describe their experiences with ME/CFS. From medical failure to the hope for fair care.

DocCheck: https://www.doccheck.com/de/detail/articles/52560-me-cfs-patienten-packen-aus?utm_source=DC-Newsletter&utm_medium=email&utm_campaign=DocCheck-News_2026-01-13&utm_content=asset&utm_term=article&dclid=cb52c196f2fd7b745b1e75a24480545e&sc_src=email_7242589&sc_lid=771191837&sc_uid=ZdDxkCAJzH&sc_llid=181853&sc_customer=130674

GPCR



Angiotensin-II Receptor-1 (AT1R)



Family of about 770 different receptors.

Ligands (binding partners of the receptor) ranging from small molecules to peptides to large proteins: odours, pheromones, hormones, and neurotransmitters.

About 30% of modern drugs interact with GPCRs: antihistamines, angiotensin receptor inhibitors, beta receptor blockers, dopamine agonists, neuroleptics, opioids.

Nobel Prize for their discovery only awarded just over a decade ago



A Brief History of G Protein Coupled Receptors

Nobel Lecture, December 8, 2012

by Robert J. Lefkowitz

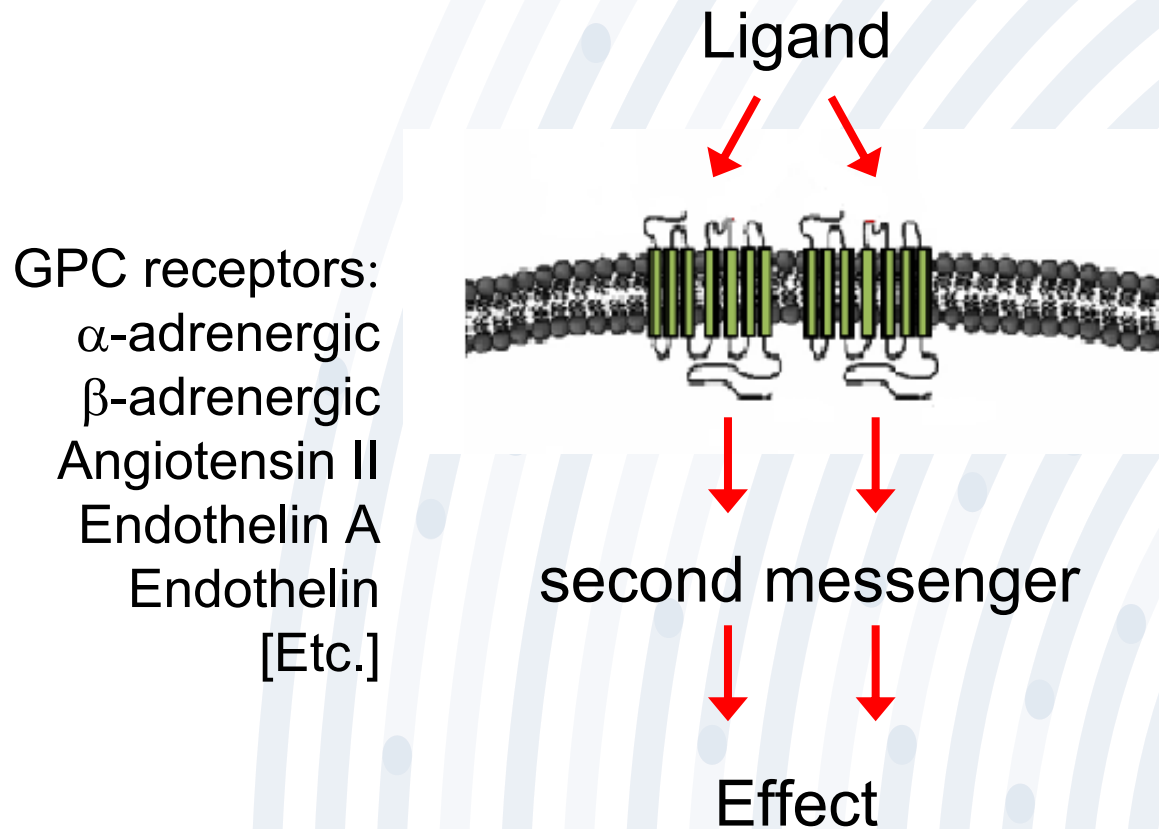
Howard Hughes Medical Institute, Duke University Medical Institute, Durham, North Carolina 27710 USA.

“Today we know that the G protein coupled receptors, also known as seven transmembrane receptors, represent by far the largest, most versatile and most ubiquitous of the several families of plasma membrane receptors. They comprise almost a thousand genes which **regulate virtually all known physiological processes in humans** including the sensory modalities of vision, taste and smell.”

GPCR* Action



Signal Transduction

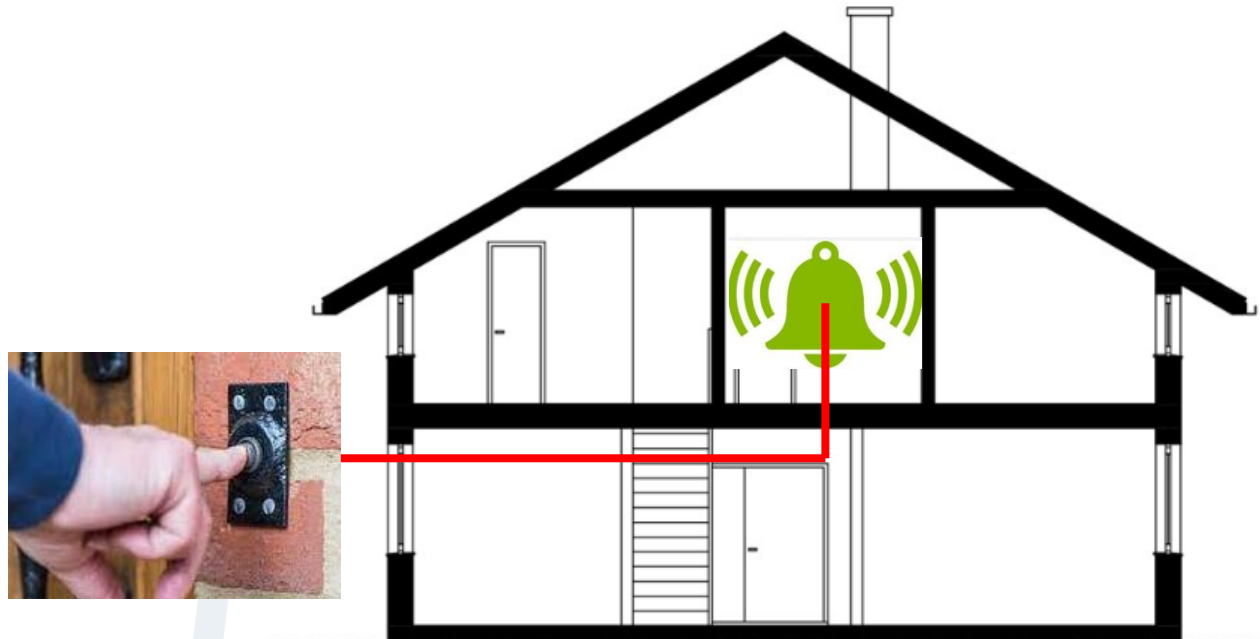


* G protein-coupled receptor

GPCR Action



GPCRs transmit a signal/information from outside to the inside of a cell
like a doorbell



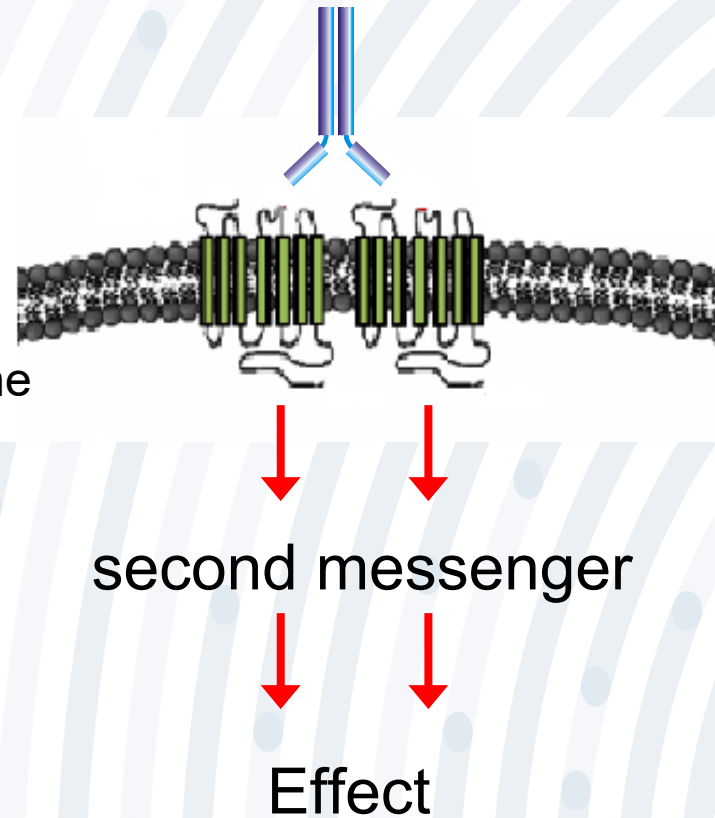
GPCR Action



Signal Transduction

Autoimmunity can generate autoantibodies that bind GPCRs

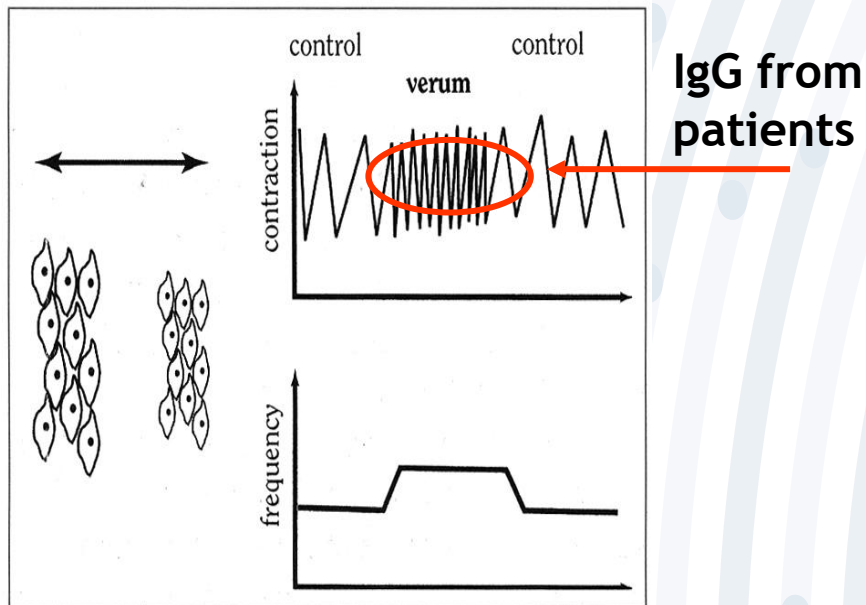
They activate the receptor like the natural ligand



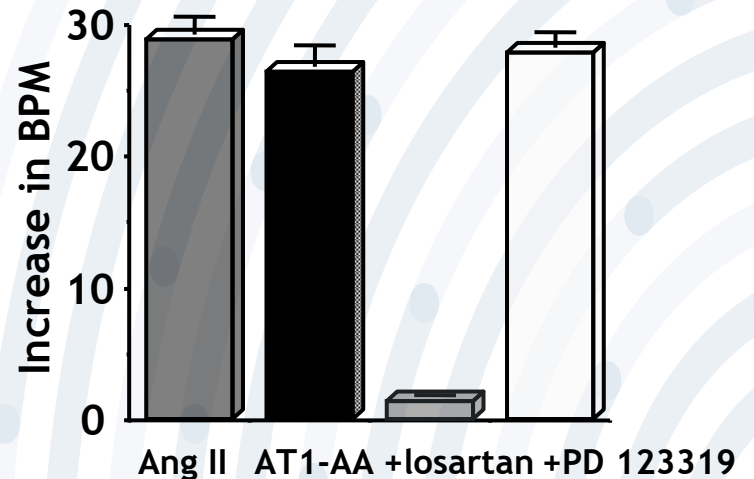
Discovery of agonistic GPCR autoantibodies using a bioassay with rat cardiomyocytes

Preeclampsia: AT1R / DCM: $\beta 1$

A bioassay for agonistic receptor activity



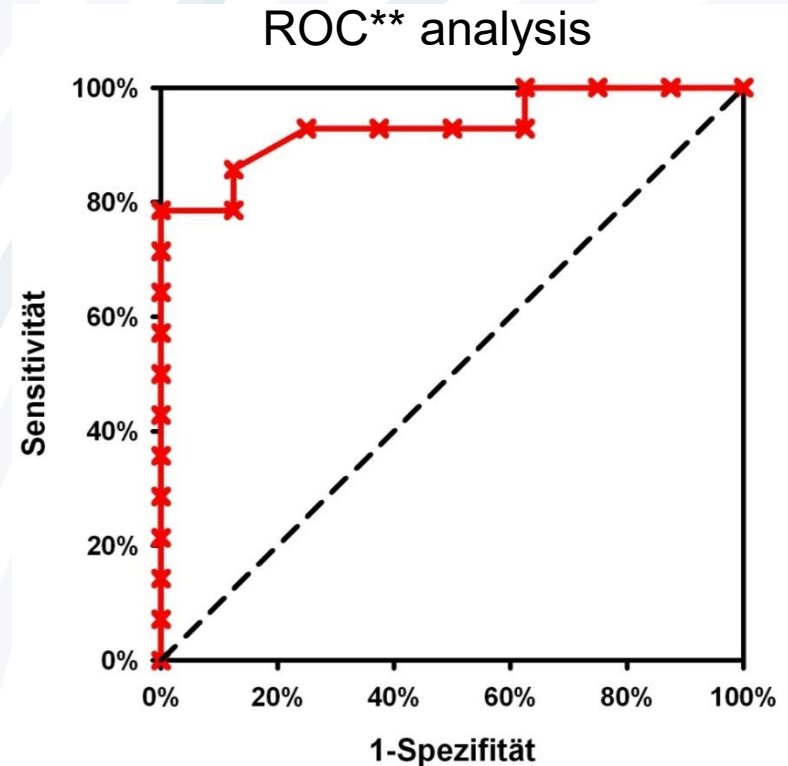
IgG agonistic response is AT1R specific



Bioassay



	Bioassay	EIA*
Primary Cells	Yes	No
High Throughput	No	Yes
Effort	High	Low
Specificity	Medium	High

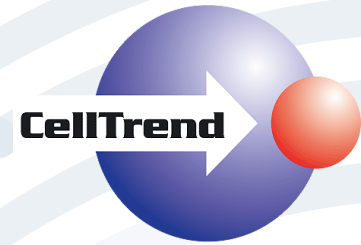


Sensitivity 93%
Specificity 75%
**...as compared to a
cardiomyocyte bioassay**

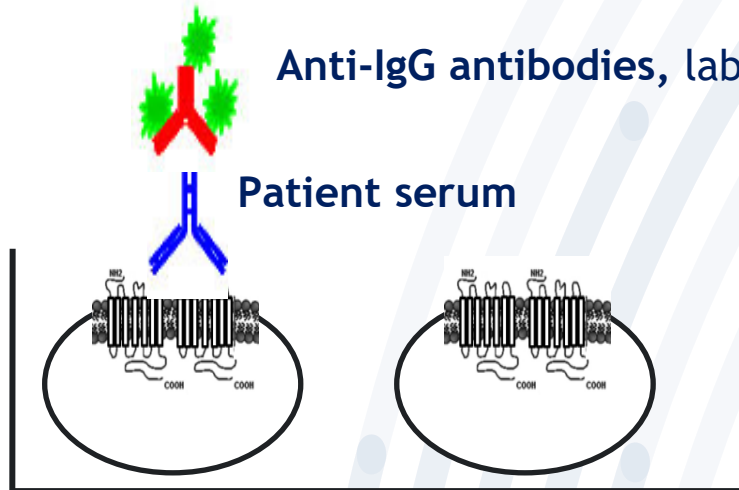
* Enzyme Immunoassay

** Receiver Operating Characteristic

Enzyme Immunoassay



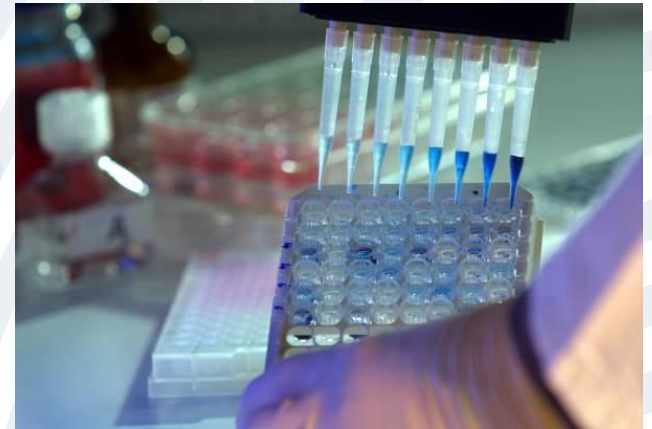
TMB* (colorless) → TMB (oxidized, blue)



Anti-IgG antibodies, labelled with Horseradish Peroxidase

Patient serum

Complete GPCRs
native receptor conformation



Platform Technology

* TMB = Tetramethylbenzidine

Diagnostic Marker



The first validated diagnostic kit detected
Angiotensin II-receptor subtype 1 autoantibodies
AT1R

Intended use:
for non-HLA antibody-mediated kidney rejection

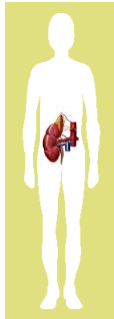
AMR Associated with High Levels of AT₁R Specific Antibody in Patients with no Donor HLA Specific or MICA Antibodies

Acute Rejection	High anti- AT₁R (>17 units) antibodies	Low anti- AT₁R (<17 units) antibodies
AMR	6	1
CMR	0	9

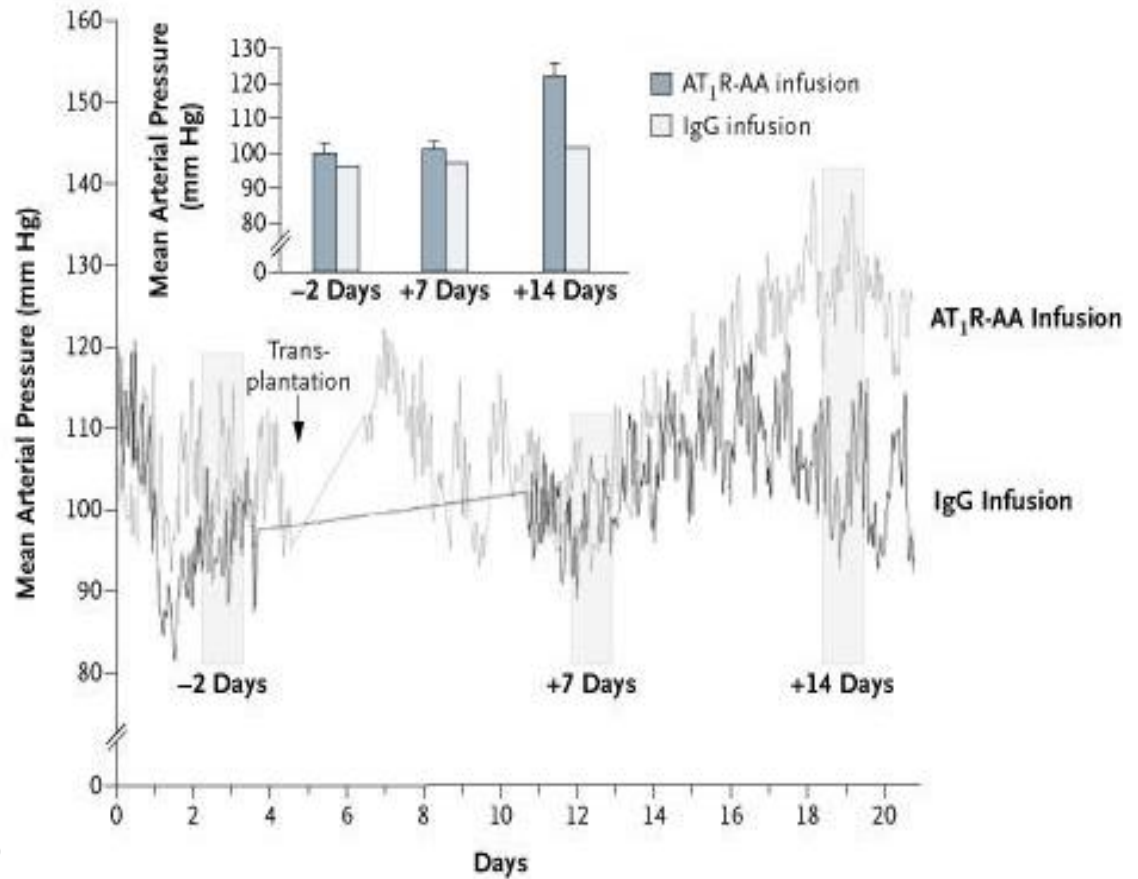
**Total number of patients with no donor-specific antibody to HLA or MICA = 63
P =0.0009, Fisher's Exact Test developed 16 rejection episodes (ACR=9,
AMR=7)**

**AMR = Antibody Mediated Rejection
CMR = Cell Mediated Rejection
AT₁R = Angiotensin Type 1 Receptor
DHSA = Donor HLA Specific Antibodies**

Passive human AT₁R-Ab transfer induces vascular rejection and hypertension in rat-KTx model



AT₁R-Ab pos.
human IgG

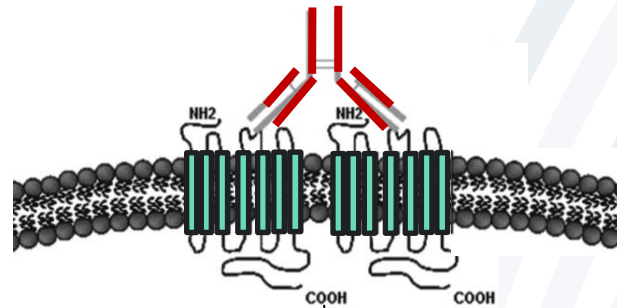


GPCRs often trigger the symptoms (1/3)

CellTrend

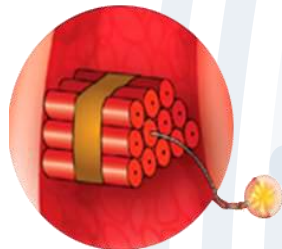


Functional antibodies act independently from natural GPCR ligands and define high-risk patients (“fast progressors“)



sustained signaling

fast onset of organ damage



Vasculopathy

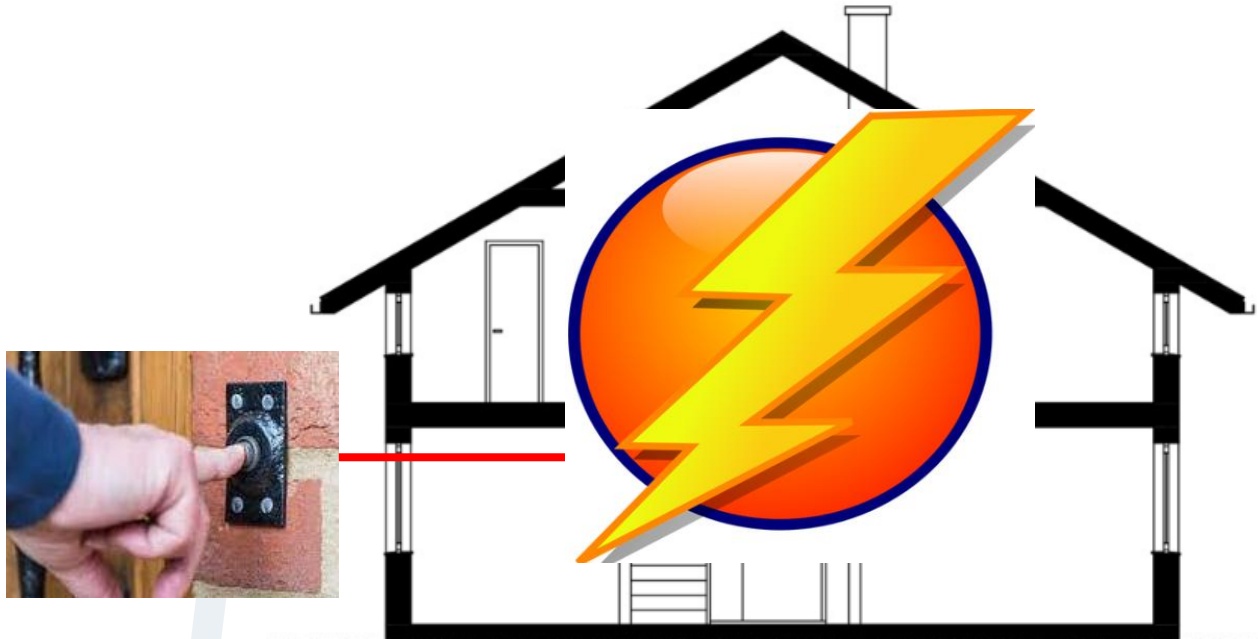
GPCRs often trigger the symptoms (2/3)

CellTrend



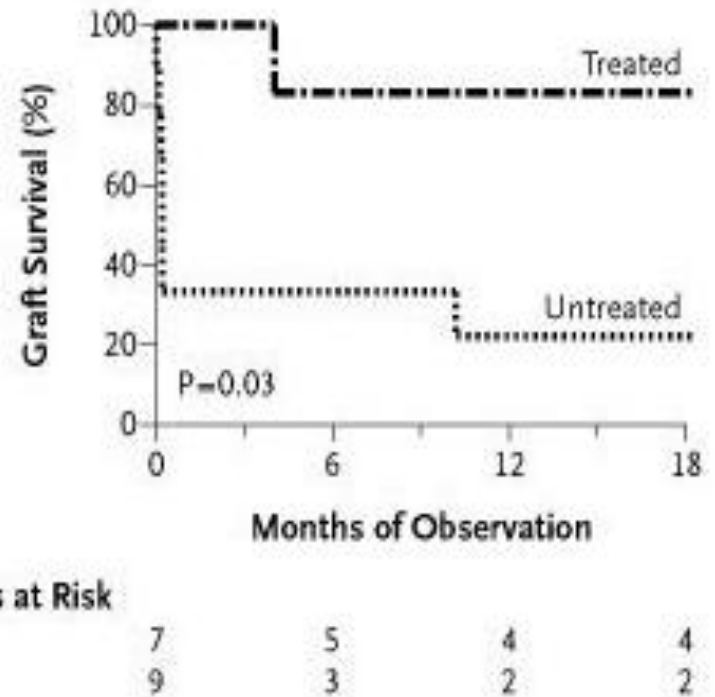
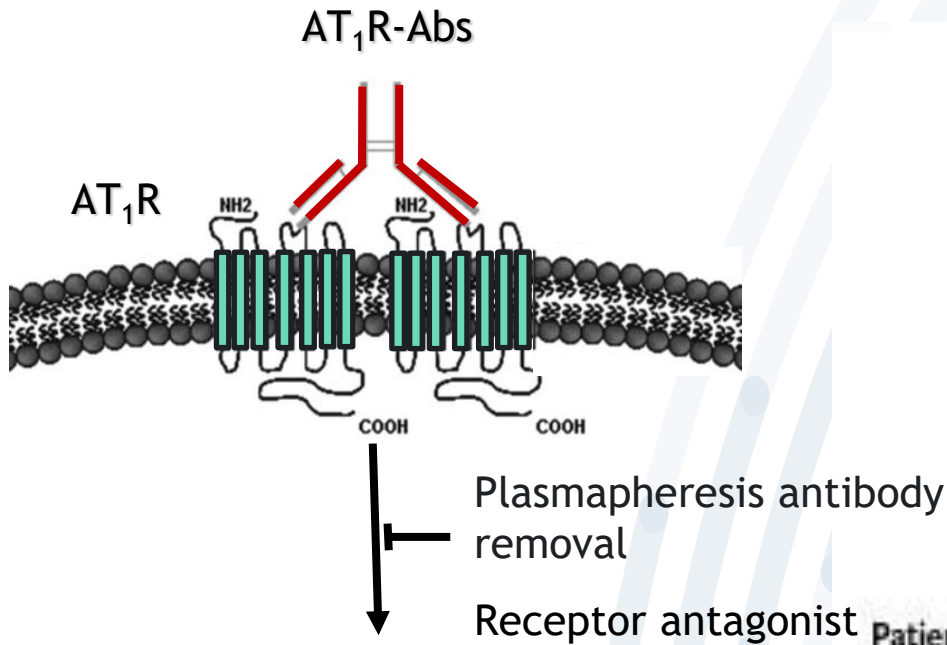
Physiological ligand: present for only a short time

GPCR autoantibodies: persist in the blood for long time
causing sustained signalling and activation



GPCRs often trigger the symptoms (3/3)

Translation: back from bench to bedside



Therapeutic “proof of concept”

Therapeutic approaches



In some disorders elevated GPCR autoantibody levels are detected and identified as a pathophysiological agents

Examples of new treatment options targeting autoantibodies (all under evaluation):

1. Removal of autoantibodies
(Plasmapheresis, Immunoabsorption, INUSpheresis)
2. Receptor blockers
3. IVIG (intravenous immunoglobulins)
4. Anti-CD20 (Rituximab)

Myalgic Encephalomyelitis



Myalgic encephalomyelitis (M.E.) is a complex, debilitating, long-term medical condition. The root cause/s of the syndrome is/are unknown and the mechanisms not fully understood, but it is often triggered by viral infections.





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journal homepage: www.elsevier.com/locate/ybrbi



Antibodies to β adrenergic and muscarinic cholinergic receptors in patients with Chronic Fatigue Syndrome



Madlen Loebel^{a,*}, Patricia Grabowski^a, Harald Heidecke^b, Sandra Bauer^a, Leif G. Hanitsch^a, Kirsten Wittke^a, Christian Meisel^{a,c}, Petra Reinke^{d,e}, Hans-Dieter Volk^{a,e}, Øystein Fluge^f, Olav Mella^{f,g}, Carmen Scheibenbogen^{a,e}

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^f Department of Oncology and Medical Physics, Haukeland University Hospital, Bergen, Norway

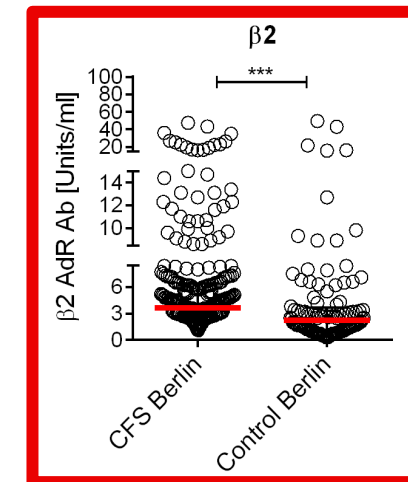
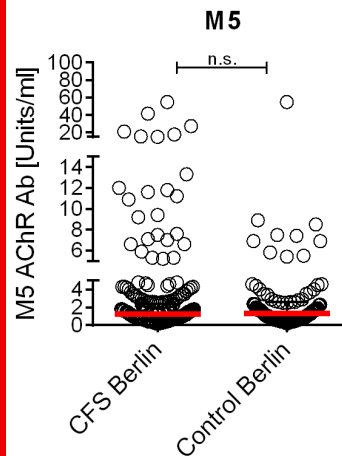
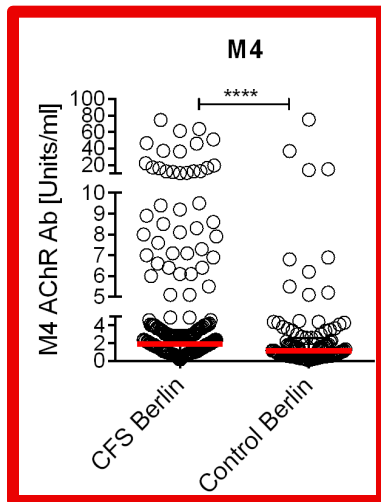
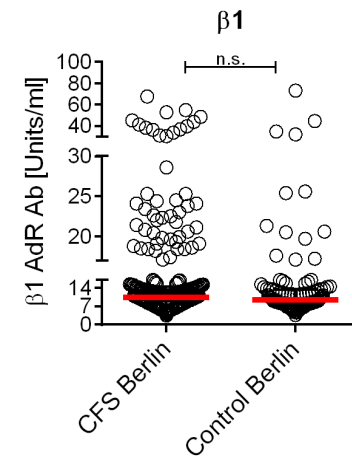
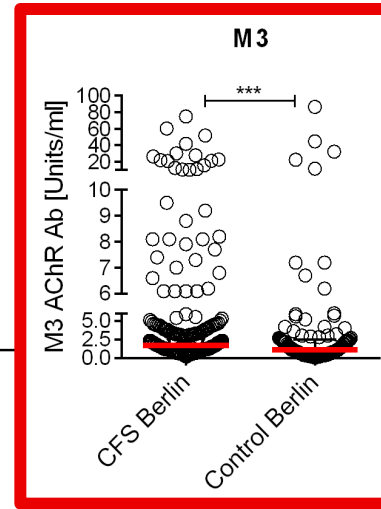
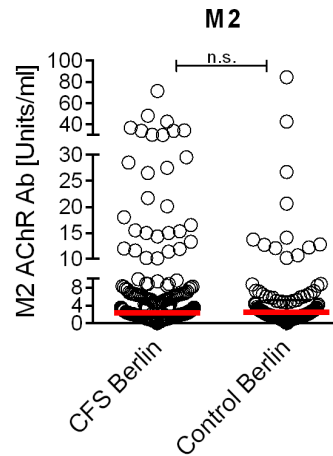
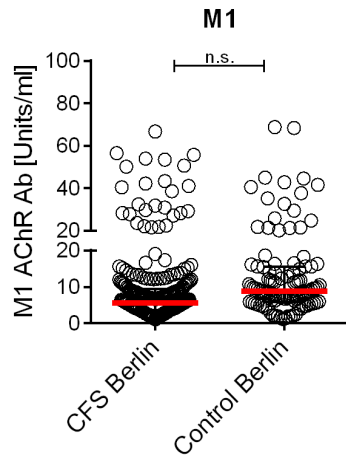
^g Department of Clinical Science, University of Bergen, Bergen, Norway

ME/CFS Pre-COVID



Muscarinic Cholinergic Receptor AABs

β -adrenergic Receptor AABs



ME/CFS



About 30% of ME/CFS patients were positive in one or more of the neurotransmitter GPCR autoantibodies.

No significant differences in the case of α adrenergic, dopamine, serotonin, angiotensin, and endothelin receptors.

ME/CFS cohort Berlin n=268
Healthy control cohort n=108

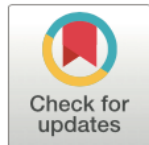
RESEARCH ARTICLE

Immunoabsorption to remove β 2 adrenergic receptor antibodies in Chronic Fatigue Syndrome CFS/ME


Carmen Scheibenbogen^{1,2*}, Madlen Loebel¹, Helma Freitag¹, Anne Krueger³, Sandra Bauer¹, Michaela Antelmann¹, Wolfram Doehner⁴, Nadja Scherbakov⁴, Harald Heidecke⁵, Petra Reinke^{2,3}, Hans-Dieter Volk^{1,2}, Patricia Grabowski¹

1 Institute for Medical Immunology, Charité—Universitätsmedizin Berlin, Berlin, Germany, **2** Berlin-Brandenburg Center for Regenerative Therapies (BCRT), Berlin, Germany, **3** Department of Nephrology, Charité—Universitätsmedizin Berlin, Berlin, Germany, **4** Center for Stroke Research Berlin, Charité—Universitätsmedizin Berlin, Berlin, Germany, **5** CellTrend GmbH, Luckenwalde, Germany

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Abstract

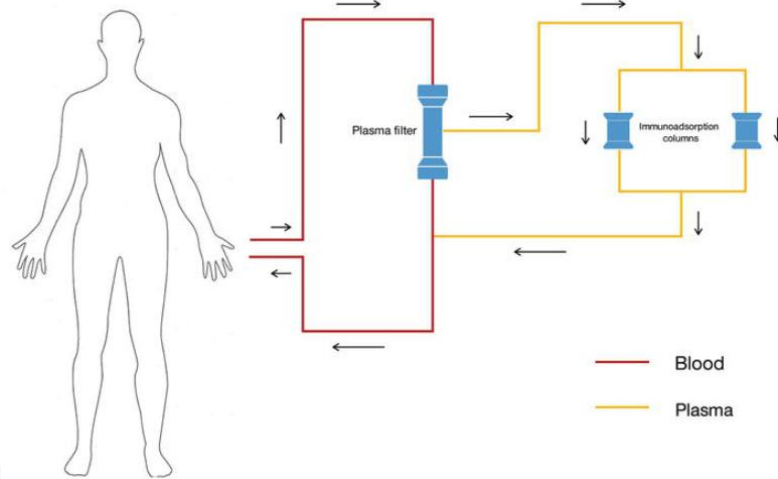
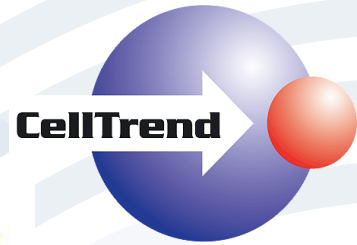
 OPEN ACCESS

Citation: Scheibenbogen C, Loebel M, Freitag H, Krueger A, Bauer S, Antelmann M, et al. (2018) Immunoabsorption to remove β 2 adrenergic receptor antibodies in Chronic Fatigue Syndrome CFS/ME. PLoS ONE 13(3): e0193672. <https://doi.org/10.1371/journal.pone.0193672>

Introduction

Infection-triggered disease onset, chronic immune activation and autonomic dysregulation in Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME) point to an autoimmune disease directed against neurotransmitter receptors. We had observed elevated autoanti-

Immunoadsorption



Immunoadsorption (IA) is an extracorporeal (outside the body) medical treatment that selectively removes harmful antibodies, autoantibodies, and immune complexes from a patient's blood plasma, acting like a specialised filter for the immune system. Unlike plasma exchange (PE) that removes most plasma proteins, IA uses special adsorption columns with specific ligands (like protein A) to bind target immune molecules, preserving essential proteins like albumin, often preferred for autoimmune diseases and prevention of transplant rejection.

ME/CFS



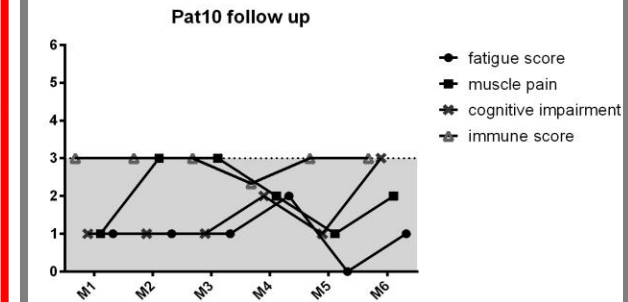
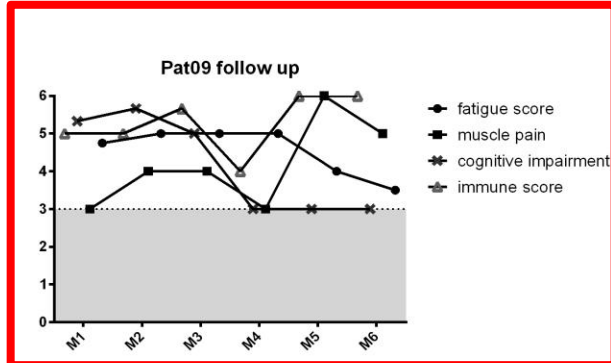
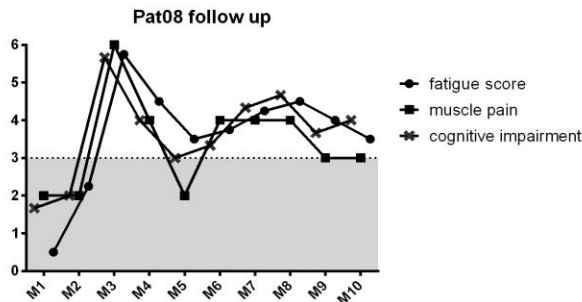
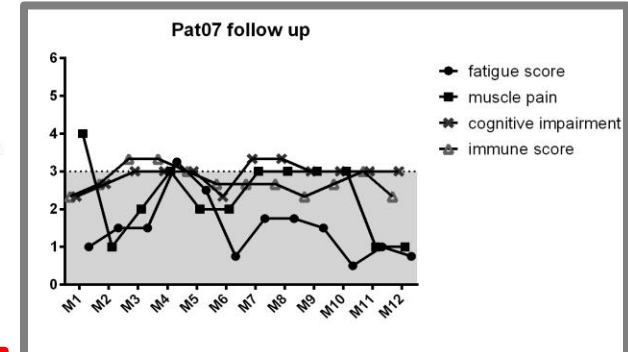
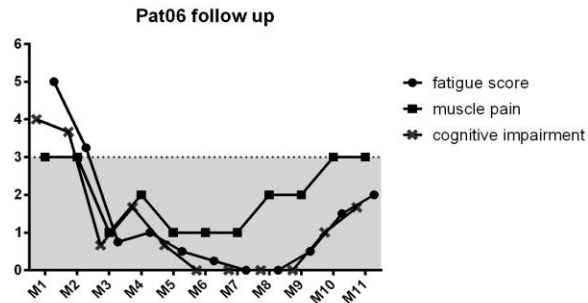
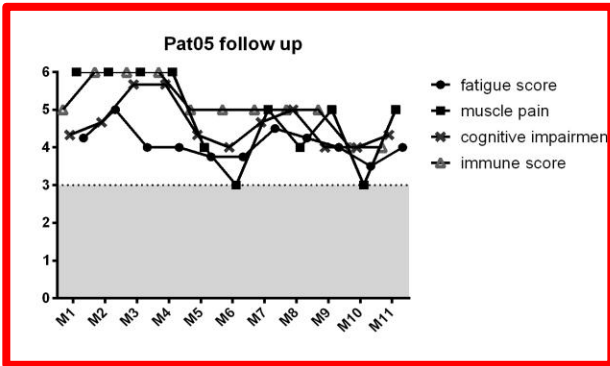
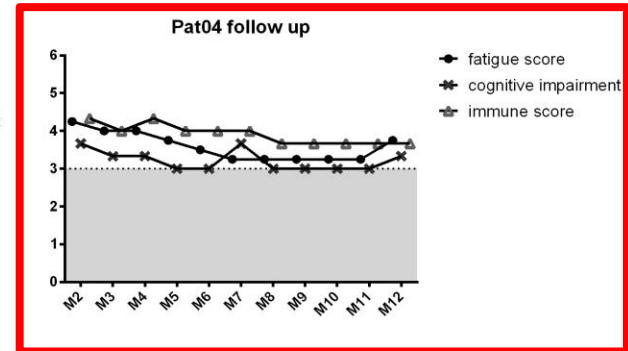
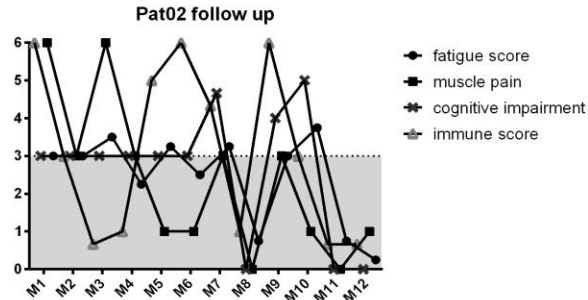
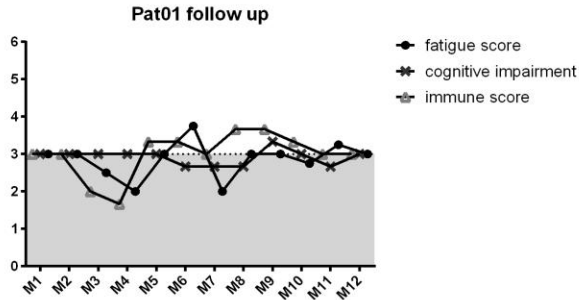
The therapy was conducted in 5 cycles on days 1–3 and 6–7 with 2 to 2.5-fold plasma volume filtered. After the 5th cycle all patients received 25 g of IVIG (Octagam, Octapharma).

SUMMARY Immunoadsorption led to

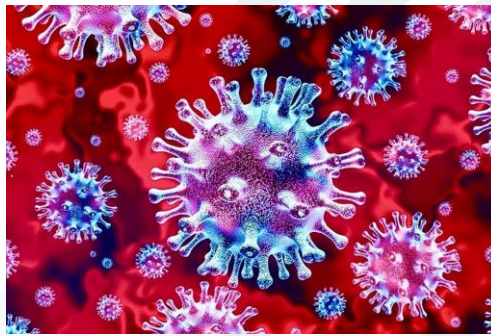
- a rapid decrease of autoantibodies in 9 out of 10 ME/CFS patients
- β 1 and β 2 autoantibodies were significantly lower after 6 months
- diminished memory and enhanced plasma cells after Immunoadsorption
- a rapid symptom improvement in 7 of the patients
- a sustained symptom improvement in 3 of these 7 patients

Evidence that some forms of ME/CFS are antibody-mediated?

Symptom improvement months 1 -12 – improvement > 6 months



Post-Covid Syndrome



CellTrend

Post COVID-19 Syndrome (PCS, PASC), commonly known as long COVID, can affect anyone exposed to SARS-CoV-2, regardless of age or severity of original symptoms.

Definition

It is defined as the continuation or development of new symptoms 3 months after the initial SARS-CoV-2 infection, with these symptoms lasting for at least 2 months with no other explanation.

Symptoms

While common symptoms of long COVID can include fatigue, shortness of breath and cognitive dysfunction over 200 different symptoms have been reported that can have an impact on everyday functioning.

PCS and GPCRs



CellTrend

2022: Multi-Center Study:

- 80 patients with Long Covid (40 with ME/CFS)
- 40 healthy controls after COVID infection
- 38 healthy controls without COVID infection

 frontiers | Frontiers in Immunology

TYPE Original Research
PUBLISHED 27 September 2022
DOI 10.3389/fimmu.2022.981532

 Check for updates

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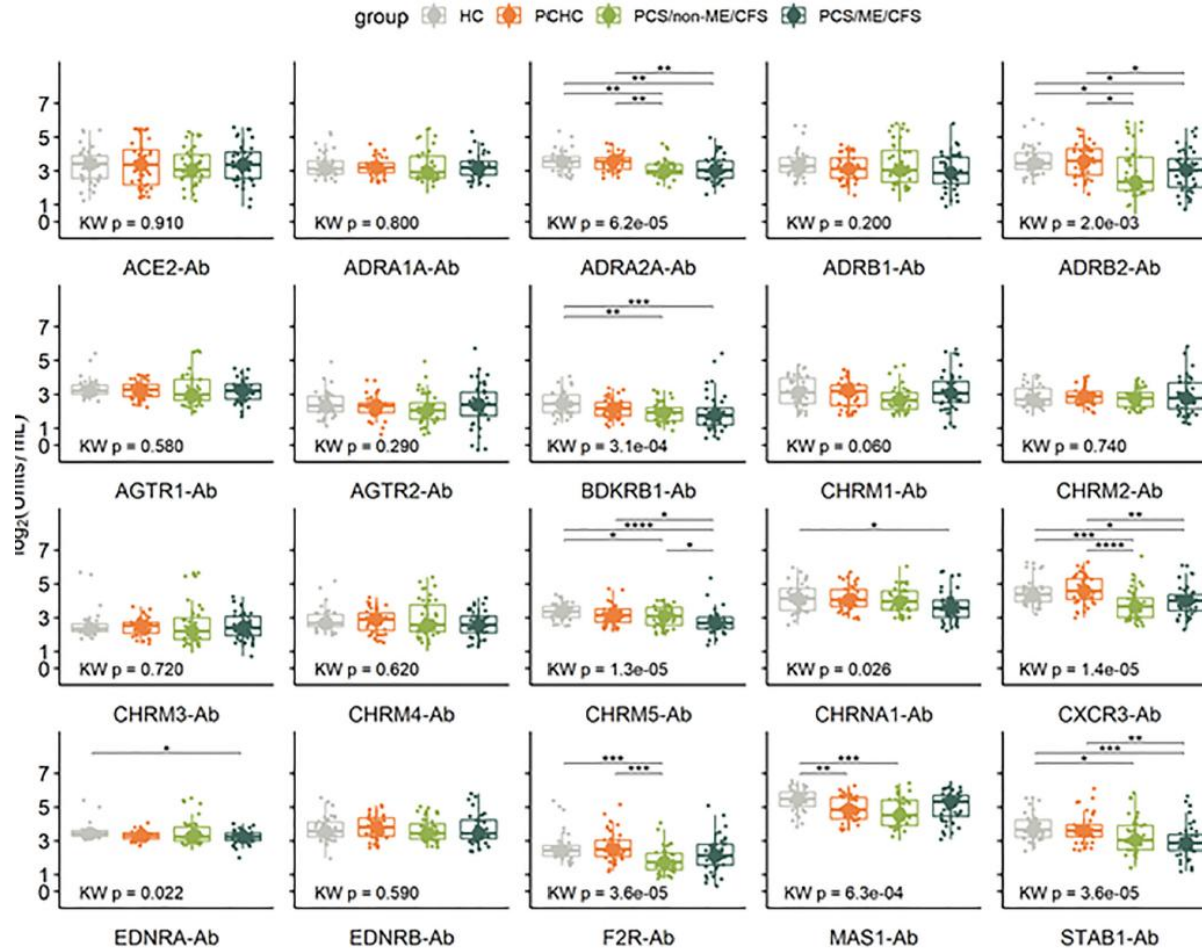
Dysregulated autoantibodies targeting vaso- and immunoregulatory receptors in Post COVID Syndrome correlate with symptom severity

Franziska Sotzny^{1*}, Igor Salerno Filgueiras^{2*}, Claudia Kedor¹, Helma Freitag¹, Kirsten Wittke¹, Sandra Bauer¹, Nuno Sepúlveda^{3,4}, Dennyson Leandro Mathias da Fonseca⁵, Gabriela Crispim Baiocchi², Alexandre H. C. Marques², Myungjin Kim⁶, Tanja Lange⁷, Desirée Rodrigues Praça⁸, Finn Luebber^{7,9}, Frieder M. Paulus⁹, Roberta De Vito¹⁰, Igor Jurisica^{11,12,13,14,15}, Kai Schulze-Forster¹⁶, Friedemann Paul^{17,18,19,20}, Judith Bellmann-Strobl^{1,17,18,19}, Rebekka Rust^{1,17,18,19}, Uta Hoppmann^{1,17,18,19}, Yehuda Shoenfeld^{21,22}, Gabriela Riemekasten⁷, Harald Heidecke¹⁶, Otavio Cabral-Marques^{2,5,8,23,24*} and Carmen Scheibenbogen^{1*}

¹Institute for Medical Immunology, Charité – Universitätsmedizin Berlin, corporate member of Freie

PCS and GPCRs

20 markers investigated



PCS and GPCRs

The logo for CellTrend, featuring a blue sphere with a white arrow pointing right, and a red sphere to its right.

CellTrend

Result:

- Clear differences between the groups.
- About 77% of the patients showed different GPCR autoantibodies compared with healthy controls
- Correlation with symptoms
- Therapeutic implications: YES, as with M.E.

Increased protease-activated receptor 1 autoantibodies are associated with severe COVID-19

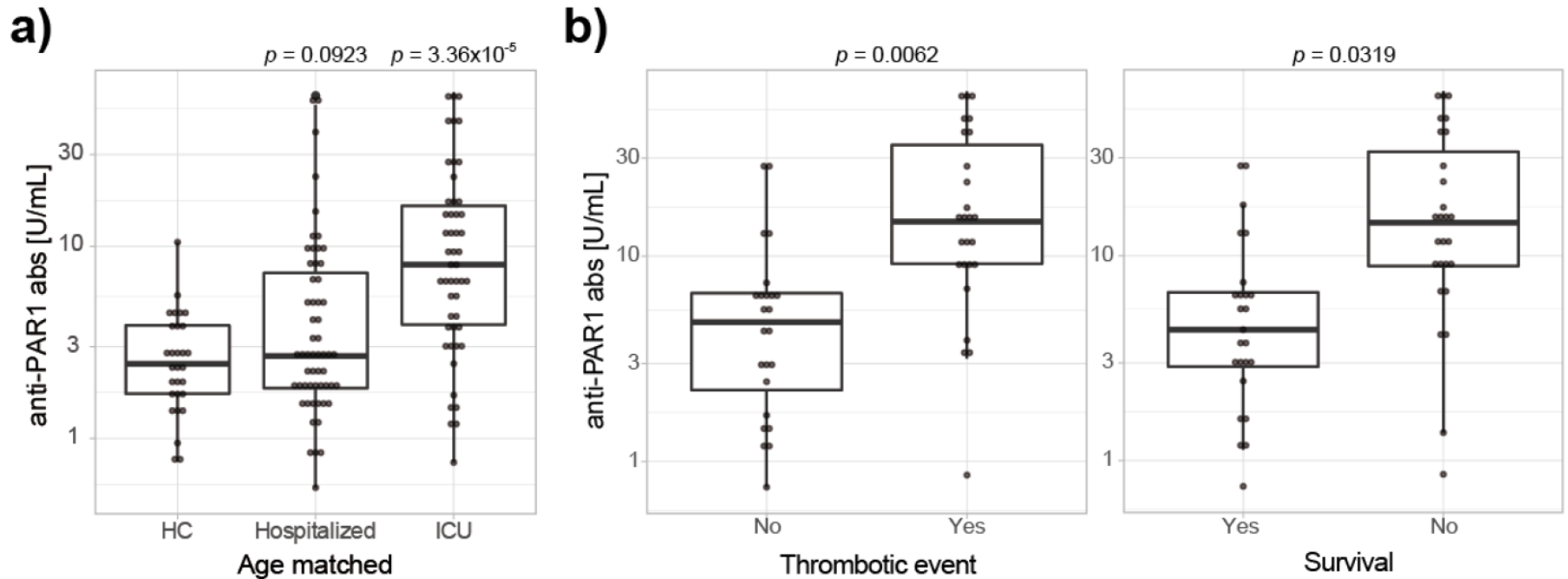
Florian Tran^{1,2}, Danielle MM Harris^{1,3}, Alena Scharmacher¹, Hanna Graßhoff⁴, Kristina Sterner⁴, Susanne Schinke⁴, Nadja Käding⁵, Jens Y Humrich⁴, Otávio Cabral-Marques^{6,7,8}, Joana P Bernardes¹, Neha Mishra¹, Thomas Bahmer^{2,9}, Jeanette Franzenburg^{1,10}, Bimba F Hoyer¹¹, Andreas Glück², Martina Guggeis^{1,2}, Alexander Ossysek¹, Andre Küller¹², Derk Frank^{12,13}, Christoph Lange^{14a,b,c,d}, Jan Rupp⁵, Jan Heyckendorf^{2,14a,b,e}, Karoline I Gaede^{14a,b,e,f}, Howard Amital¹⁵, Philip Rosenstiel¹, Yehuda Shoenfeld^{15,16,17}, Gilad Halpert¹⁵, Avi Z Rosenberg¹⁸, Kai Schulze-Forster¹⁹, Harald Heidecke¹⁹, Gabriela Riemekasten^{3,20,21} & Stefan Schreiber^{1,2,21}

Plain language summary

Antibodies targeting a receptor on platelets (protease-activated receptor 1, PAR1) were increased in patients with severe/fatal COVID-19 disease course. PAR1 antibody levels correlate with blood markers for blood clotting (D-dimers), a hallmark of COVID-19. These results suggest a role for antibodies against PAR1 in the development of blood clotting observed in the setting of COVID-19.

High correlation of PAR1 with severity

CellTrend



Efficacy of repeated immunoadsorption in patients with post-COVID myalgic encephalomyelitis/chronic fatigue syndrome and elevated β 2-adrenergic receptor autoantibodies: a prospective cohort study

Elisa Stein,^a Comelia Heindrich,^a Kirsten Wittke,^a Claudia Kedor,^a Rebekka Rust,^{a,c} Helma Freitag,^a Franziska Sotzny,^a Anne Krüger,^b Markus Tölle,^b Patricia Grabowski,^a Carmen Scheibenbogen,^a and Laura Kim^{a,*}

Findings The treatment was generally well tolerated, reducing total immunoglobulin G by 79% (CI: 73–84%) and β 2 AR-AB by 77% (CI: 58–95%). Patients demonstrated a mean increase in the SF36 PF of 17.75 points (CI: 13.41–26.16), with the greatest improvement occurring between months two and three, and significant gains maintained through month six. 14/20 (70%) patients were categorized as responders with an increase in the SF36 PF of \geq ten points. Further lasting improvements were reported in fatigue, post-exertional malaise, pain, cognitive, autonomic, and immunological symptoms. Female patients had increased repeat handgrip strength at month six.

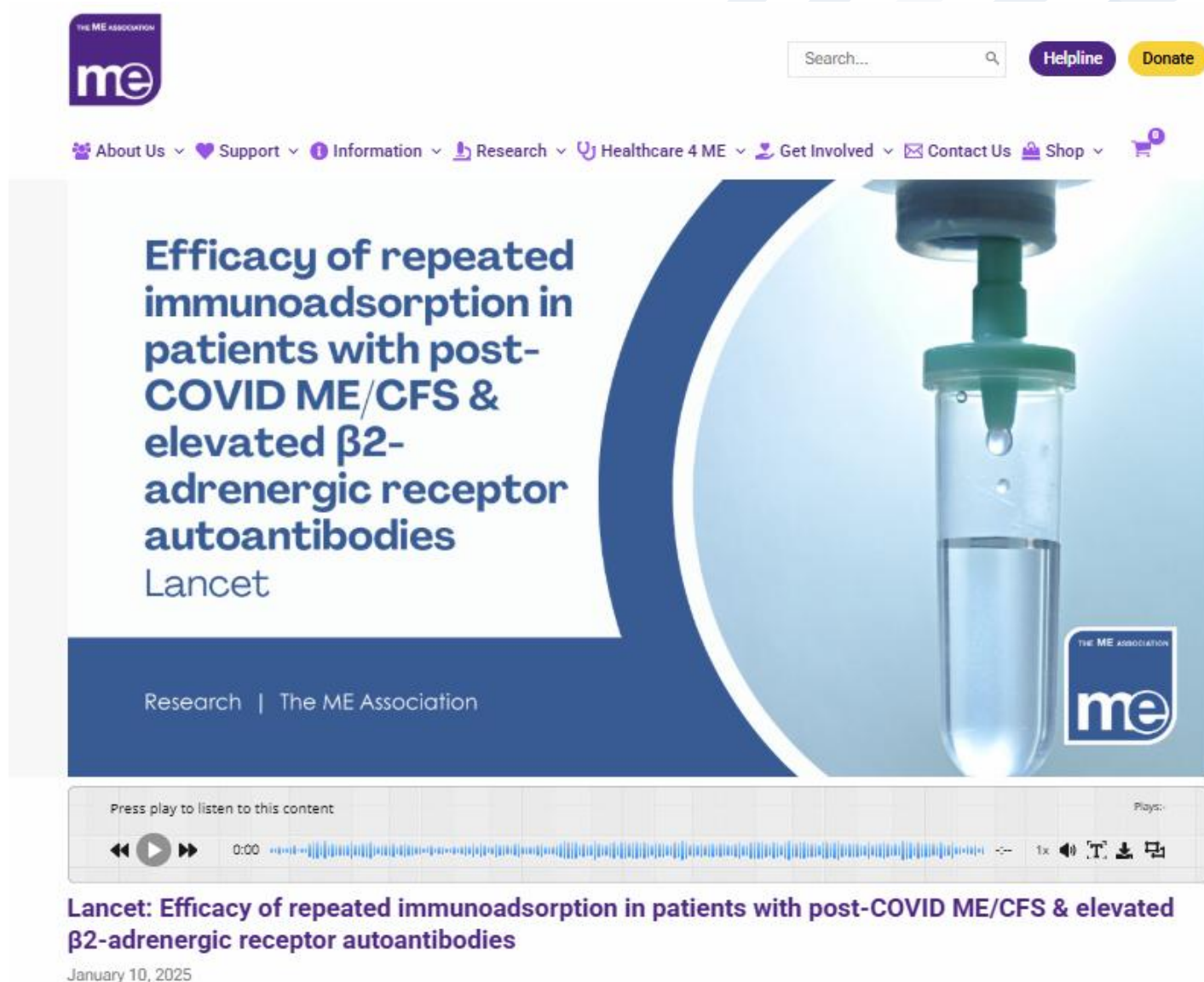
Interpretation Immunoadsorption may improve symptoms in post-COVID ME/CFS patients. The beneficial effects of IgG depletion suggest a significant role for autoantibodies and disturbed B-cell function in the condition's pathophysiology.

Patients' health-related quality of life was assessed using the 36-Item Short-Form Survey (SF-36), with scores ranging from 0 to 100, with 100 indicating no limitations.

The Lancet Regional
Health - Europe
2025;49: 101161

Published Online xxx
<https://doi.org/10.1016/j.lanepe.2024.101161>

Also covered by the M.E. Association in the UK



Efficacy of repeated immunoadsorption in patients with post-COVID ME/CFS & elevated β 2-adrenergic receptor autoantibodies
Lancet

Research | The ME Association

Press play to listen to this content

Lancet: Efficacy of repeated immunoadsorption in patients with post-COVID ME/CFS & elevated β 2-adrenergic receptor autoantibodies

January 10, 2025

<https://meassociation.org.uk/2025/01/lancet-efficacy-of-repeated-immunoadsorption-in-patients-with-post-covid-me-cfs-elevated-%CE%B2-adrenergic-receptor-autoantibodies/>

Proof of Concept



CellTrend

Transfer into animal model possible for ME/CSF
2 recent publications

Science




Antibodies from Long Covid patients prompt symptoms in mice

Two new studies suggest dysfunctional immune system attacking a patient's own tissues might drive the challenging condition

21 JUN 2024 · 6:20 PM ET · BY JENNIFER COUZIN-FRANKEL

Two studies had found that mice receiving IgG preparations containing autoantibodies from M.E. patients showed signs of similar symptoms to what those patients experienced.

Immunomodulatory treatment in postural tachycardia syndrome: A case series

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Abstract

Background and purpose: Postural tachycardia syndrome (POTS) is a form of autonomic dysfunction characterized by symptoms of orthostatic intolerance, often accompanied by sudomotor dysfunction and gastrointestinal dysmotility. Recently, evidence has accumulated that in a subset of patients, the pathogenesis of dysautonomia may be immune-mediated. The aim of the current report was to evaluate the use of intravenous immunoglobulin (IVIg) treatment in patients with progressive and/or refractory immune-mediated POTS.

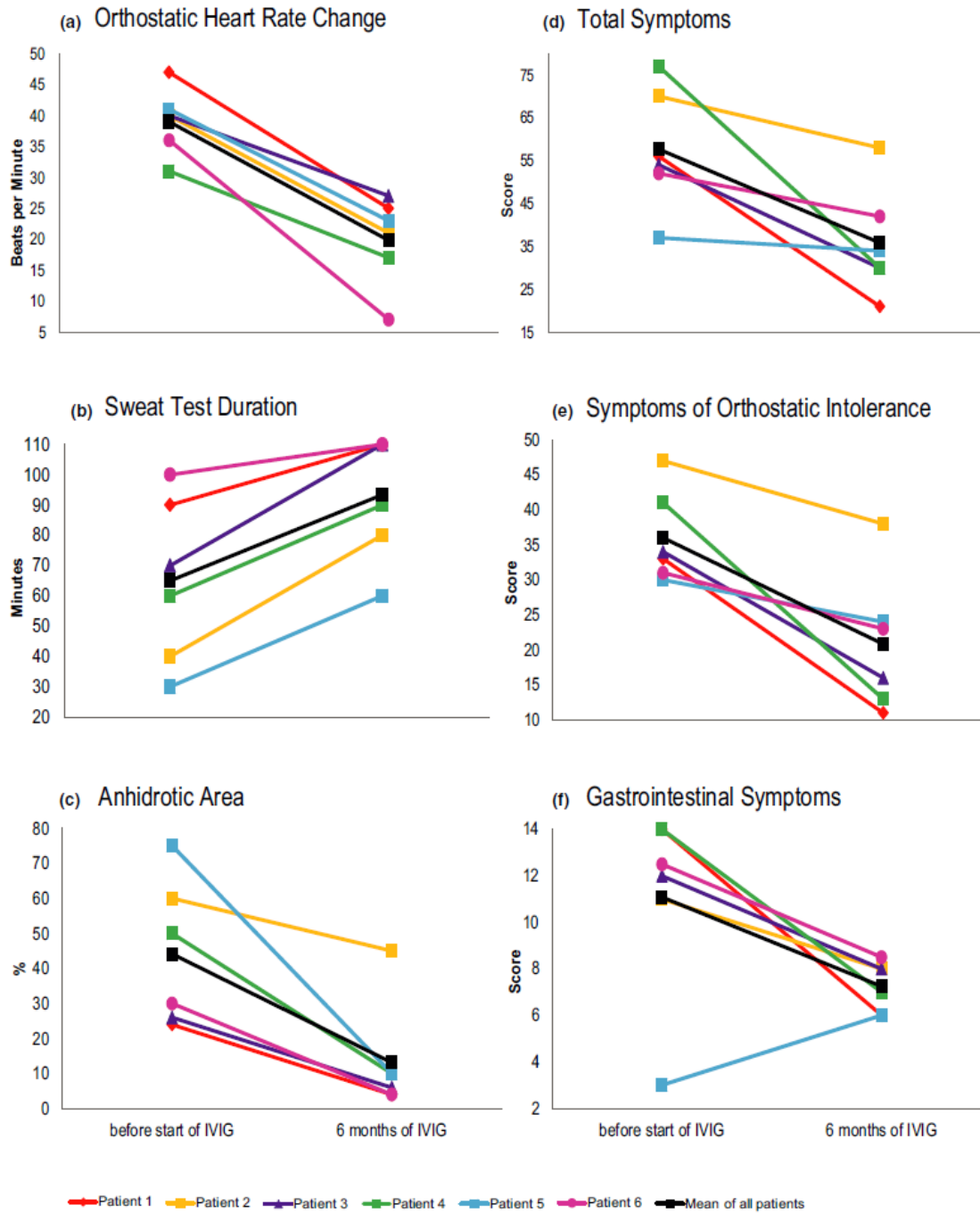
Methods: We retroactively assessed the effect and tolerance of monthly administered IVIg in six patients using autonomic function testing, standardized symptom questionnaires, and patients' symptom diaries both before and 6 months into IVIg treatment.

Conclusions: Using subjective but also standardized objective measures, the case series describes promising effects of IVIg treatment in POTS patients with immune-mediated dysautonomia. By reducing the infusion rate, pretreatment with steroids, and intravenous hydration, tolerance could be improved, and no patient had to discontinue the treatment.

AAb assays used

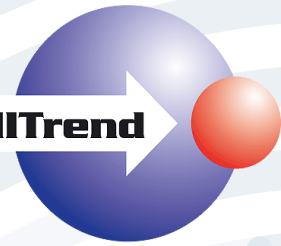
To identify AAb in our POTS patients, all patients are routinely investigated using a commercially available enzyme-linked immunosorbent assay to detect the following antibodies (IgG): adrenergic ($\alpha 1$ and $\alpha 2$) and muscarinic cholinergic (M1–5). Analysis was performed by CellTrend.

POTS



More markers are on their way for

CellTrend



- Cardiovascular conditions (CXCR3 ...)
- Cancer
- Alzheimer's
- Etc.

Our Network



Prof. Yehuda Shoenfeld



Prof. Gabriela Riemekasten



Prof. Carmen Scheibenbogen



Prof. Stefan Bornstein

CellTrend History



- Founded in 1998
- Certified according to ISO 13485:2016 (medical devices/IVD)
- CellTrend strictly adheres to the rules of GMP (Good Manufacturing Practice)
- 4 private owners, no venture capital
- 10 employees
- Located in Luckenwalde (near Berlin)
- CellTrend is the only manufacturer worldwide for these parameters



Panels - examples



CellTrend GmbH

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Name
 Address
 City

sample: Name
 date of birth: dd.mm.yyyy
 sample date: dd.mm.yyyy
 date of receiving: dd.mm.yyyy
 ordered by: Dr. XYZ
 method: ELISA

RESULT REPORT

Parameter	Cut off	Units/ml
anti β-1-adrenergic Receptor Antibodies	<15.0 U/ml: negative > 15.0 U/ml: positive	57,8
anti β-2-adrenergic Receptor Antibodies	<8.0 U/ml: negative 8.0 – 14.0 U/ml: at risk > 14.0 U/ml: positive	70,4
anti-Muscarinic Cholinergic Receptor-3-Antibodies	<6.0 U/ml: negative 6.0 – 10.0 U/ml: at risk > 10.0 U/ml: positive	40,7
anti-Muscarinic Cholinergic Receptor-4-Antibodies	< 10.7 U/ml: negative > 10.7 U/ml: positive	39,5



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Name
 Address
 City

sample: Name
 sample date: dd.mm.yyyy
 date of receiving: dd.mm.yyyy
 ordered by: clinic
 method: ELISA

RESULT REPORT

Parameter	Cut off	Units/ml
anti AT1R Antibodies	<10.0 U/ml: negative 10.0 – 17.0 U/ml: at risk > 17.0 U/ml: positive	11,8
anti ETAR Antibodies	<10.0 U/ml: negative 10.0 – 17.0 U/ml: at risk > 17.0 U/ml: positive	16,3
anti α-1-adrenergic Receptor Antibodies	<7.0 U/ml: negative 7.0 – 11.0 U/ml: at risk > 11.0 U/ml: positive	14,1
anti α-2-adrenergic Receptor Antibodies	<15.0 U/ml: negative > 15.0 U/ml: positive	29,2
anti β-1-adrenergic Receptor Antibodies	<15.0 U/ml: negative > 15.0 U/ml: positive	40,2
anti β-2-adrenergic Receptor Antibodies	<8.0 U/ml: negative 8.0 – 14.0 U/ml: at risk > 14.0 U/ml: positive	41,4
anti-Muscarinic Cholinergic Receptor-1-Antibodies	<9.0 U/ml: negative > 9.0 U/ml: positive	15,9
anti-Muscarinic Cholinergic Receptor-2-Antibodies	<9.0 U/ml: negative > 9.0 U/ml: positive	29,7
anti-Muscarinic Cholinergic Receptor-3-Antibodies	<6.0 U/ml: negative 6.0 – 10.0 U/ml: at risk > 10.0 U/ml: positive	10,5
anti-Muscarinic Cholinergic Receptor-4-Antibodies	< 10.7 U/ml: negative > 10.7 U/ml: positive	11,2
anti-Muscarinic Cholinergic Receptor-5-Antibodies	<14.2 U/ml: negative > 14.2 U/ml: positive	24,8

Thank You for Your Attention !

