

This is the suggested CellTrend M.E. Panel

Just a few words about why these – and one other

M.E. Panel

Beta1 adrenergic-receptor-ab

Beta2 adrenergic-receptor-ab

Muscarinic cholinergic M3-receptor-ab

Muscarinic cholinergic M4-receptor-ab

Alpha1 adrenergic-receptor-ab

Beta-1 adrenergic antibodies and their significance in M.E.

Beta-1 adrenergic receptors are the key receptors in cardiac cells. They augment heart rate and contractility. Antibodies to them are therefore prone to reduce the pumping capacity of the heart.¹

Beta-1 adrenergic receptors also support sodium retention in the kidneys. Antibodies to them can therefore contribute to loss of sodium. This hyponatremia means plasma decreases, because it needs salt as a solute, and this can eventually lead to lower blood volume (hypovolemia).²

1. Yoo B, Lemaire A, Mangmool S, Wolf MJ, Curcio A, Mao L, Rockman HA. Beta1-adrenergic receptors stimulate cardiac contractility and CaMKII activation in vivo and enhance cardiac dysfunction following myocardial infarction. *Am J Physiol Heart Circ Physiol*. 2009 Oct;297(4):H1377-86.

2. Singh, A., & Singh, K. (2024). *Pathology of the Renin-Angiotensin-Aldosterone System*. IntechOpen.

Beta-2 adrenergic antibodies and their significance in M.E.

Beta-2 adrenergic receptors normally facilitate vasodilation of the blood vessels in the muscles. Professors Scheibenbogen and Wirth have identified that this receptor is often prevented from acting normally in M.E./CFS for several reasons.¹ One of them is that autoantibodies to the beta-2 adrenergic receptors can activate vasoconstriction within the muscles' blood vessels instead of vasodilation. The result of this will be to create inadequate muscular blood perfusion. This can in turn cause hypovolemia.²

The beta-2 adrenergic cell receptor is also one of the main activators of the sodium-potassium pump,³ so autoantibodies to this receptor can suppress the action of this pump, negatively affecting the sodium balance both intracellularly and in the plasma, too, contributing to the global hypoperfusion.⁴

1. Gravelina S, Vilmane A, Svirskis S, Rasa-Dzelzkaleja S, Nora-Krukle Z, Vecvagare K, Krumina A, Leineman I, Shoenfeld Y, Murovska M. Biomarkers in the diagnostic algorithm of myalgic encephalomyelitis/chronic fatigue syndrome. *Front Immunol.* 2022 Oct 10;13:928945.

2. van Campen, Linda & Visser, Frans. (2018). Blood Volume Status in Patients with Chronic Fatigue Syndrome: Relation to Complaints. *International Journal of Clinical Medicine.* 09. 809-819. 10.4236/ijcm.2018.911067.

3. Abosamak NER, Patel P, Shahin MH. Beta2-Receptor Agonists and Antagonists. [Updated 2025 Dec 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing

4. Yu X, Stavakis S, Hill MA, Huang S, Reim S, Li H, Khan M, Hamlett S, Cunningham MW, Kem DC. Autoantibody activation of beta-adrenergic and muscarinic receptors contributes to an "autoimmune" orthostatic hypotension. *J Am Soc Hypertens.* 2012 Jan-Feb;6(1):40-7.

Muscarinic M3 receptor antibodies and their significance in M.E.

Muscarinic M3 acetylcholine receptors are responsible for many different functions in the body.¹ They play a role in general blood perfusion as they are responsible for vasodilation of the blood vessel wall via nitric oxide² – this is throughout the body and not just in the muscles and brain. They are found in both the endocrine and the exocrine glands, so they play a large role in the production of hormones. They modulate potassium channels, are additionally important in the activation of the parasympathetic branch of the nervous system. Autoantibodies against them can compromise all these functions, such as decreasing blood perfusion, disrupting endocrine and exocrine hormones, and impairing sympathetic/parasympathetic balance.³

1. <https://www.ncbi.nlm.nih.gov/gene/1131>

2. Guizoni DM, Vettorazzi JF, Carneiro EM, Davel AP. Modulation of endothelium-derived nitric oxide production and activity by taurine and taurine-conjugated bile acids. *Nitric Oxide*. 2020 Jan 1;94:48-53.

3. Loebel M, Grabowski P, Heidecke H, Bauer S, Hanitsch LG, Wittke K, Meisel C, Reinke P, Volk HD, Fluge Ø, Mella O, Scheibenbogen C. Antibodies to β adrenergic and muscarinic cholinergic receptors in patients with Chronic Fatigue Syndrome. *Brain Behav Immun*. 2016 Feb;52:32-39.

Muscarinic M4 receptor antibodies and their significance in M.E.

Muscarinic M4 acetylcholine receptors are vital for learning processes, impulse control, and maintaining dopamine balance.¹

Dopaminergic neurotransmission is highly dependent on M4 cholinergic muscarinic receptor function,² and antibodies to them can disregulate its release. “Antibodies against β 2, M3 and M4 receptors were significantly elevated in CFS patients compared to controls.”³ Their dysregulation “has been found to demonstrate sensitivity to the progression of ME/CFS.”^{4, 5}

Both M3 and M4 muscarinic receptor antibodies inhibit normal acetylcholine function - the parasympathetic nervous system’s key neurotransmitter³, which can disrupt normal involuntary functions and muscle movement.⁶

1. Klawonn AM et al. Muscarinic M4 Receptors on Cholinergic and Dopamine D1 Receptor-Expressing Neurons Have Opposing Functionality for Positive Reinforcement and Influence Impulsivity. *Front Mol Neurosci*. 2018 Apr 24;11:139.
2. Tzavara ET et al. M4 muscarinic receptors regulate the dynamics of cholinergic and dopaminergic neurotransmission: relevance to the pathophysiology and treatment of related CNS pathologies. *FASEB J*. 2004 Sep;18(12):1410-2.
3. Loebel M, Grabowski P, Heidecke H, Bauer S, Hanitsch LG, Wittke K, Meisel C, Reinke P, Volk HD, Fluge Ø, Mella O, Scheibenbogen C. Antibodies to β adrenergic and muscarinic cholinergic receptors in patients with Chronic Fatigue Syndrome. *Brain Behav Immun*. 2016 Feb;52:32-39.
4. Halpert G, Amital H, Shoenfeld Y. Dysregulation of G protein-coupled receptors of the autonomic nervous system, adrenergic and muscarinic acetylcholine receptors, in patients with autoimmune dysautonomic-related disorders. *Brain Behav Immun Health*. 2020 Mar 3;4:100056
5. Freitag H, Szklarski M, Lorenz S, Sotzny F, Bauer S, Philippe A, Kedor C, Grabowski P, Lange T, Riemekasten G, Heidecke H, Scheibenbogen C. Autoantibodies to Vasoregulative G-Protein-Coupled Receptors Correlate with Symptom Severity, Autonomic Dysfunction and Disability in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. *J Clin Med*. 2021 Aug 19;10(16):3675.
6. <https://www.osmosis.org/answers/anticholinergics>

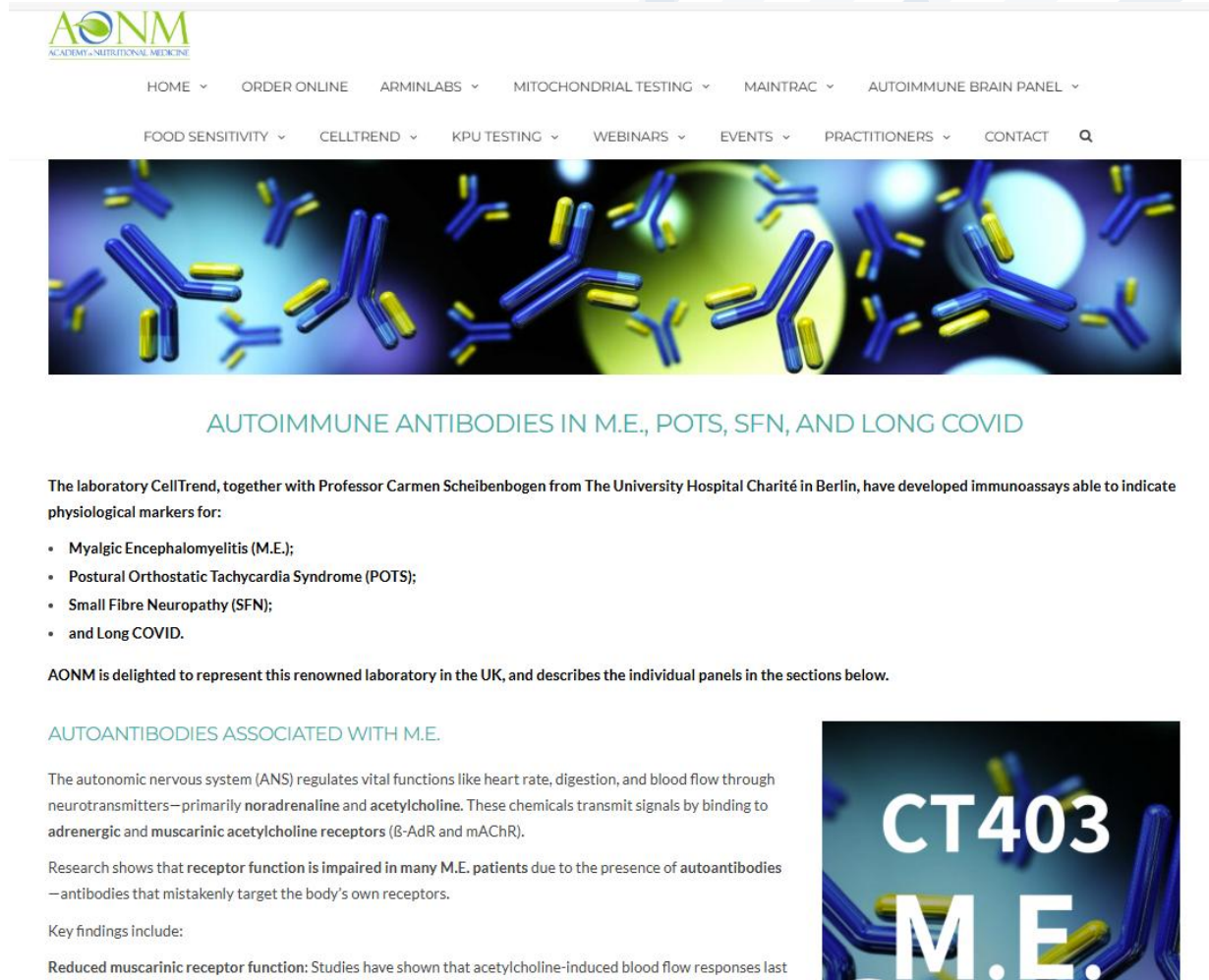
Alpha-1 adrenergic receptor antibodies

Alpha-1 adrenergic receptors regulate blood pressure.¹ They are very important for vasoconstriction of the small and large veins that return blood to the heart (especially when you stand up). Autoantibodies can impair this process. Instead veins may distend for various reasons. This can cause insufficient venous return to the heart, further contributing to the hypoperfusion we have already seen from the Beta 1 and Beta 2 adrenergic receptor antibodies. So this can lead to reduced cardiac output, and tissue perfusion, logically also contributing to POTS² and exercise intolerance.

1. Rudner XL, Berkowitz DE, Booth JV, Funk BL, Cozart KL, D'Amico EB, El-Moalem H, Page SO, Richardson CD, Winters B, Marucci L, Schwinn DA. Subtype specific regulation of human vascular alpha(1)-adrenergic receptors by vessel bed and age. *Circulation*. 1999 Dec 7;100(23):2336-43.

2. Gunning WT 3rd, Kvale H, Kramer PM, Karabin BL, Grubb BP. Postural Orthostatic Tachycardia Syndrome Is Associated With Elevated G-Protein Coupled Receptor Autoantibodies. *J Am Heart Assoc*. 2019 Sep 17;8(18):e013602.

CellTrend on AONM's website



The screenshot shows the AONM website header with the logo and navigation menu. The main content area features a large image of blue and yellow antibodies. Below the image is the section title "AUTOIMMUNE ANTIBODIES IN M.E., POTS, SFN, AND LONG COVID". The text describes the laboratory CellTrend and its collaboration with Professor Carmen Scheibenbogen. A list of conditions is provided: Myalgic Encephalomyelitis (M.E.), Postural Orthostatic Tachycardia Syndrome (POTS), Small Fibre Neuropathy (SFN), and Long COVID. A paragraph states that AONM represents this laboratory in the UK. The section is titled "AUTOANTIBODIES ASSOCIATED WITH M.E." and discusses the autonomic nervous system (ANS) and neurotransmitters. It notes that research shows receptor function is impaired in many M.E. patients. Key findings include reduced muscarinic receptor function. A small image on the right shows the text "CT403 M.E." over a background of antibodies.

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AUTOIMMUNE ANTIBODIES IN M.E., POTS, SFN, AND LONG COVID

The laboratory CellTrend, together with Professor Carmen Scheibenbogen from The University Hospital Charité in Berlin, have developed immunoassays able to indicate physiological markers for:

- Myalgic Encephalomyelitis (M.E.);
- Postural Orthostatic Tachycardia Syndrome (POTS);
- Small Fibre Neuropathy (SFN);
- and Long COVID.

AONM is delighted to represent this renowned laboratory in the UK, and describes the individual panels in the sections below.


AUTOANTIBODIES ASSOCIATED WITH M.E.

The autonomic nervous system (ANS) regulates vital functions like heart rate, digestion, and blood flow through neurotransmitters—primarily noradrenaline and acetylcholine. These chemicals transmit signals by binding to adrenergic and muscarinic acetylcholine receptors (β -AdR and mAChR).

Research shows that receptor function is impaired in many M.E. patients due to the presence of autoantibodies—antibodies that mistakenly target the body's own receptors.

Key findings include:

Reduced muscarinic receptor function: Studies have shown that acetylcholine-induced blood flow responses last



<https://aonm.org/celltrend/>

AONM Order form



TEST REQUISITION

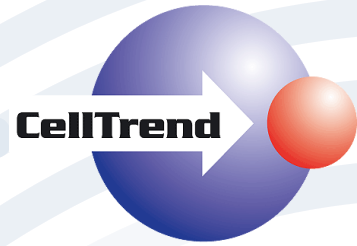
CellTrend



PATIENT INFORMATION		BARCODE (Lab use only)	Please send results to: <input type="checkbox"/> myself <input type="checkbox"/> my practitioner		
Patient FIRST NAME*: Patient SURNAME*: DATE OF BIRTH (DD/MM/YYYY)*: Sex* (please circle): male female			ORDERING DR/PRACTITIONER INFORMATION Dr. / Practitioner name: Clinic: Street Address: Postcode: City: County: Country: Tel no: Email*:		
Street Address: Postcode: City: Country: Country: Tel no: Email*:		Time of Blood Draw*: Date of blood draw (DD/MM)*: Material/Quantity <input type="checkbox"/> Serum (centrifuged)		AONM HELPLINE: +44 (0) 3331 210 305	

<input checked="" type="checkbox"/>	TEST NUMBER	NAME OF TEST	MATERIAL	PRICE
<input type="checkbox"/>	CT401	POTS Panel Angiotensin-II-receptor-1 AT1R-ab Endothelin-receptor-A ETAR-ab Alpha1 adrenergic-receptor-ab Alpha2 adrenergic-receptor-ab Beta1 adrenergic-receptor-ab Beta2 adrenergic-receptor-ab Muscarinic cholinergic M1-receptor-ab Muscarinic cholinergic M2-receptor-ab Muscarinic cholinergic M3-receptor-ab Muscarinic cholinergic M4-receptor-ab Muscarinic cholinergic M5-receptor-ab	Serum (centrifuged)	£496
<input type="checkbox"/>	CT402	Small fiber neuropathy (SFN) Panel FGFR3-ab TSHDS-ab	Serum (centrifuged)	£208
<input type="checkbox"/>	CT403	M.E. Panel Beta1 adrenergic-receptor-ab Beta2 adrenergic-receptor-ab Muscarinic cholinergic M3-receptor-ab Muscarinic cholinergic M4-receptor-ab	Serum (centrifuged)	£128
<input type="checkbox"/>		additional antibodies		
<input type="checkbox"/>	CT421	ACE2-ab	Serum (centrifuged)	£104
<input type="checkbox"/>	CT422	MAS1-receptor-ab		£104
<input type="checkbox"/>	CT423	PAR1-ab (Thrombinreceptor)		£104
<input type="checkbox"/>	CT424	CXCR3-receptor-ab		£104
<input type="checkbox"/>	CT425	Stab1-ab		£104

Test kits available from AONM



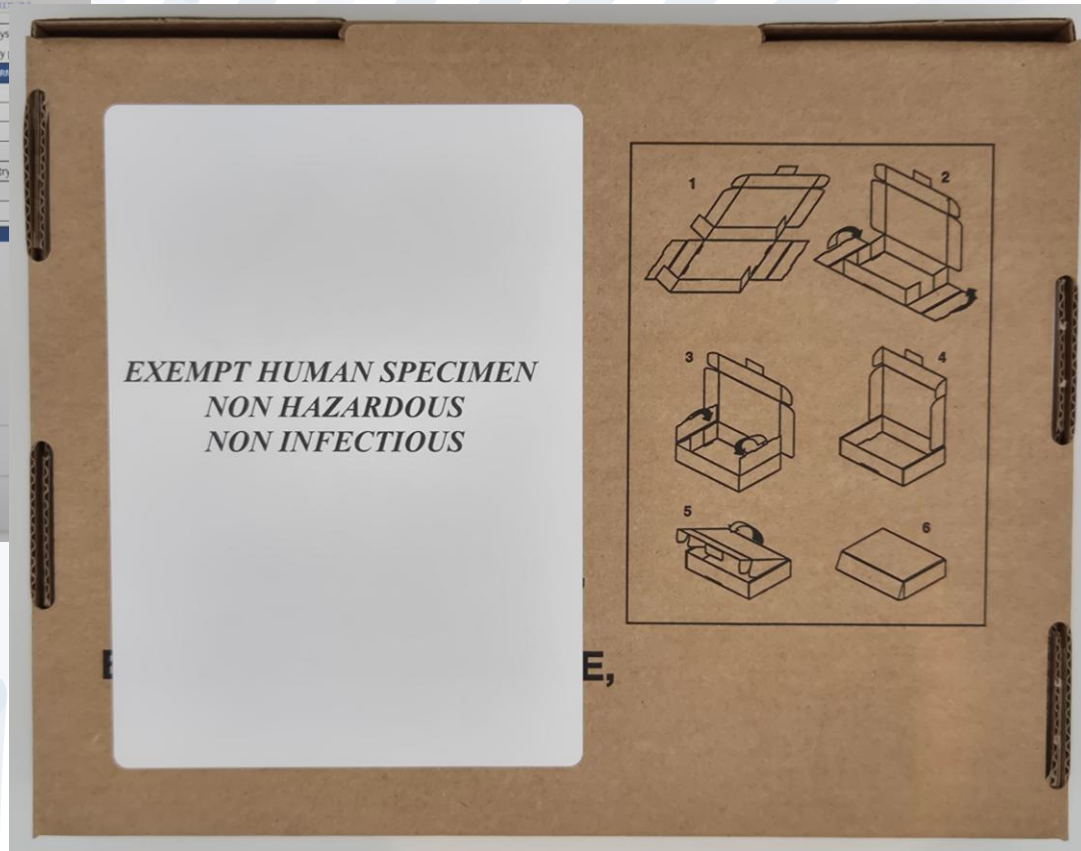
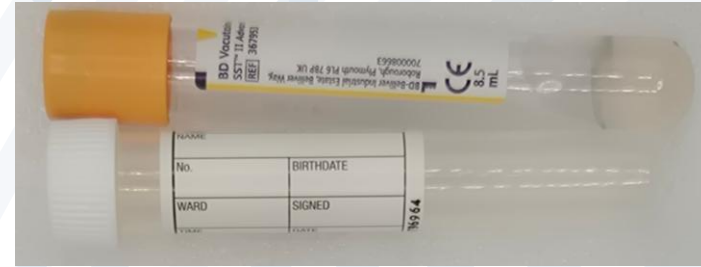
1 serum tube, centrifuged

INSTRUCTIONS for CellTrend Test Kit DHL
 Please read these instructions carefully before booking your blood draw

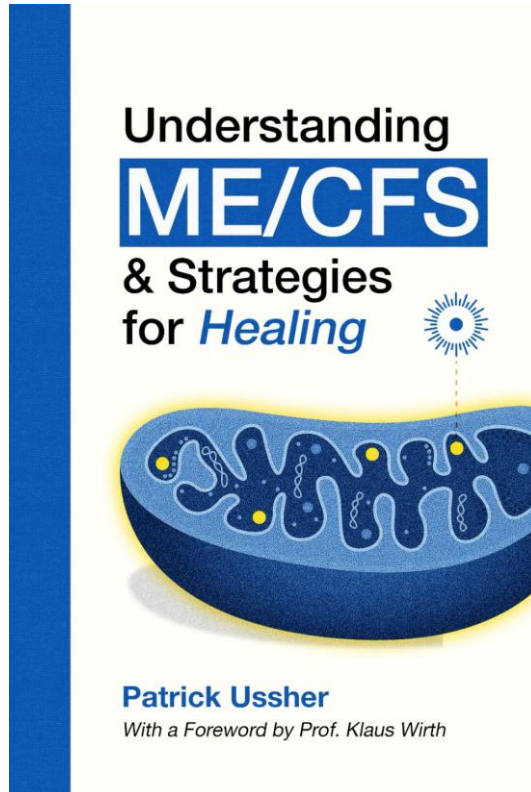
TEST REQUISITION
 CellTrend

PATIENT INFORMATION		ORDERING DR./PRACTITIONER INFORMATION	
Patient FIRST NAME:		Dr. / Practitioner name:	
Patient SURNAME:		Clinic:	
DATE OF BIRTH (DD/MM/YYYY):		Street Address:	
Sex* (please circle): male female		Postcode:	City:
Street Address:		County:	Country:
Postcode:	City:	Tel no:	
County:	Country:	Email:	
AONM HELPLINE: +44 (0) 3331 210 305			

#TEST NUMBER	NAME OF TEST	MATERIAL
<input type="checkbox"/> CT401	POTS Panel Angiotensin-II-receptor-1 AT1R-ab Endothelin-receptor-A ETAR-ab Alpha1 adrenergic-receptor-ab Alpha2 adrenergic-receptor-ab Beta1 adrenergic-receptor-ab Beta2 adrenergic-receptor-ab Muscarinic cholinergic M1-receptor-ab Muscarinic cholinergic M2-receptor-ab Muscarinic cholinergic M3-receptor-ab Muscarinic cholinergic M4-receptor-ab Muscarinic cholinergic M5-receptor-ab	Serum (centrifuged)
<input type="checkbox"/> CT402	Small fiber neuropathy (SFN) Panel FGFR3-ab TSHDS-ab	Serum (centrifuged)
<input type="checkbox"/> CT403	M.E. Panel Beta1 adrenergic-receptor-ab Beta2 adrenergic-receptor-ab	Serum (centrifuged)



Recent book of Patrick Ussher's, much of it on these topics



Understanding ME-CFS

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This channel is dedicated to explaining the research into ME/CFS in an accessible way as well as to discuss...more

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[Irish ME Trust Talk about Thirst in ME/CFS](#)

[Irish ME Trust Talk about Global Hypoperfusion in ME/CFS \(Research of Wirth and Scheibenbogen\)](#)

[Irish ME Trust talk about autoimmunity](#)

[Irish ME Trust talk about ancestral dieting](#)

[Irish ME Trust talk about the carnivore diet for ME/CFS](#)

[Irish ME Trust about finding meaning when you have ME/CFS](#)

<https://www.patrickussher.com/talks-and-media>

<https://www.youtube.com/watch?v=s2oOdHvIPEI>

Severe ME/CFS Explained: How Mitochondria Stop Producing Energy

Professor Klaus Wirth and Professor Carmen Scheibenbogen have written a “Unifying Hypothesis of M.E./CFS”



A COMPREHENSIVE DISEASE CONCEPT
BASED ON EXERCISE INTOLERANCE
AND A DERIVED THERAPEUTIC
STRATEGY

Klaus Wirth

International ME/CFS Conference Berlin 2025



Co-funded by
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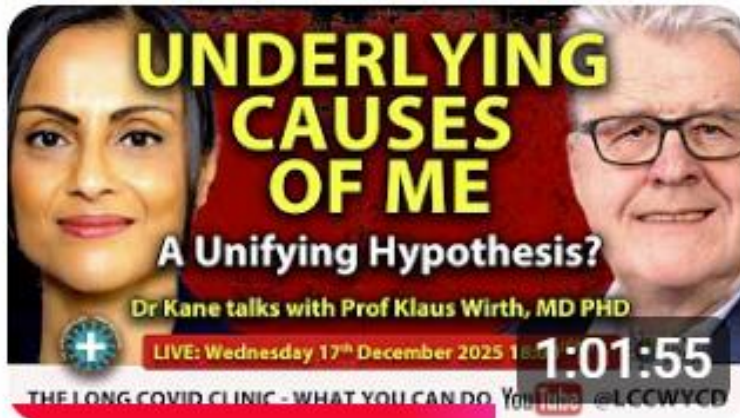
ME/CFS (Myalgic Encephalomyelitis/Chronic Fatigue Syndrome) is an acquired **mitochondrial myopathy** that also leads to vascular dysfunction via reactive oxygen species. Potential risk factors for the disease are autoantibodies, collagen diseases, and variants in mitochondrial, vascular, and muscle genes. Once fully developed, mitochondrial dysfunction, **caused by sodium-induced calcium overload in skeletal muscle**, reproduces itself with every post-exertional malaise (PEM) keeping ME/CFS patients captured in a vicious circle from which they cannot escape. MDC002 is being developed to break this vicious circle.

Interviews



New insights into CFS and M.E., interview with **Prof. Wirth**, by **Dr Sanjay Gupta and Patrick Ussher**

<https://www.youtube.com/watch?v=nMx38g1OguE>



Underlying causes of **#ME** - a unifying hypothesis?

<https://www.youtube.com/live/9Tv42q7oTms?t=1199s>

Prof. Wirth with Dr. Binita Kane, The Long Covid Clinic

Chronic Fatigue (ME/CFS), Blood Volume, & Excessive Thirst
Patrick Ussher with Dr. Eric Gordon, Gordon Medical

<https://gordonmedical.com/chronic-fatigue-blood-volume-excessive-thirst/>

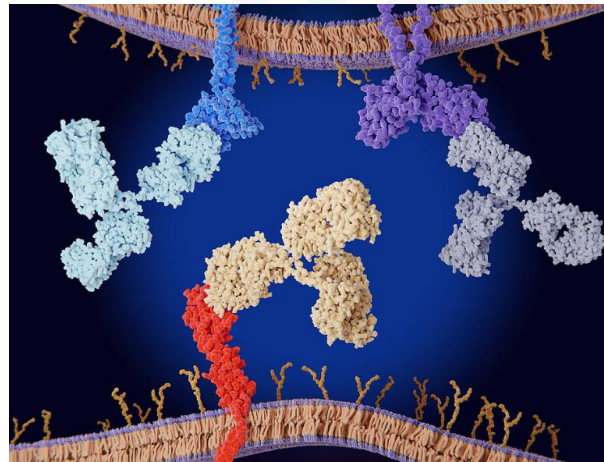
Treating the Root Cause of Fatigue & PEM in Long Covid & ME/CFS: Mitodicure
Prof. Klaus Wirth with Gez Medinger (three-part interview)

<https://www.youtube.com/watch?v=LzGGLHzcSLI>

The Man Trying to Cure ME/CFS & Long Covid: Prof. Klaus Wirth on Mitodicure

<https://www.youtube.com/watch?v=6FkenaYkz3c>

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



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Thank you! Q&A