Identifying stealth infections that may underlie ME, fibromyalgia and other syndromes
Tips on test selection and live Q&A

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Agenda

General overview

- The difficulties of using antibody assays for testing Lyme Disease
- The advantages of the "EliSpot" LTT (Interferon Gamma Release Assay- lymphocyte transformation test)
- Why the CD57+ test is also useful
- Selecting the most relevant tests

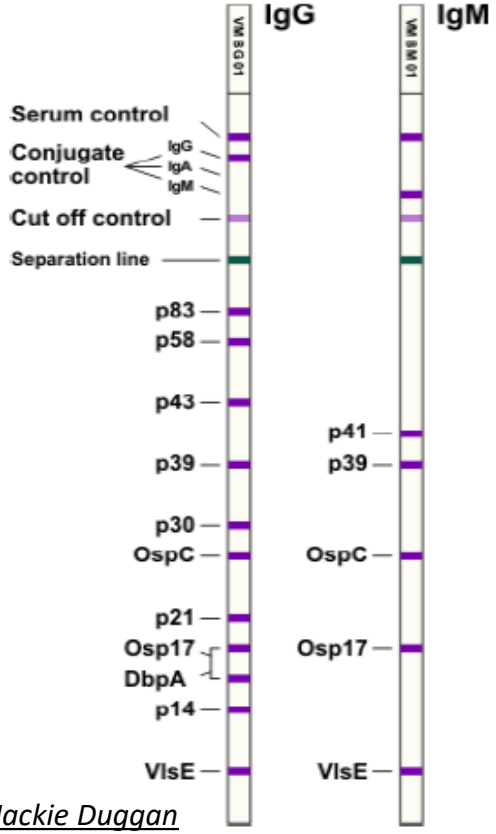
Examples of bacterial and viral infections that may underlie unexplained conditions

- Fibromyalgia
- ME
- Dementia/Alzheimer‘s
- Neuropsychiatric syndromes: OCD and Tourette‘s

NHS Lyme testing – Rare and Imported Pathogens Laboratory

Official testing in the UK consists of a two-tiered serodiagnostic algorithm: an initial enzyme-linked immunosorbent assay – "ELISA" – is followed, if positive or equivocal, by an IgG and/or IgM "Western blot" (also called "Immunoblot")

- C6 ELISA (Immunetics)
- IgM and IgG Lineblots (Viramed Virastripe system)
- PCR (introduced from Southampton)



Source: Public Health England, Lyme Disease Conference, October 9, 2013, presentation by Dr. Jackie Duggan

However, antibody assays have insufficient sensitivity

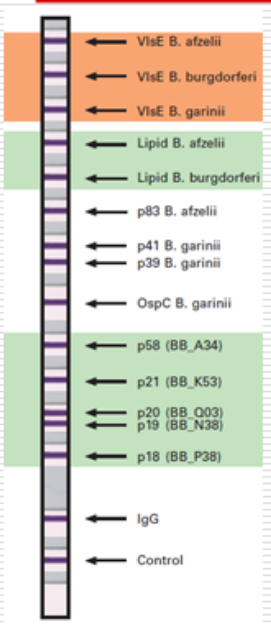
Sensitivity of the ELISA test in chronic Lyme Disease is only 32 - 42%

Dr. Armin Schwarzbach, Tick Talk Dublin 2012

The specificity of the Western Blot (Immunoblot) is high, but its sensitivity is only ~ 60%

Dr. Armin Schwarzbach, Lifting the Veil I, London 8 March 2015

Antibodies by Immunoblot: EUROLINE-RN-AT



Antigen	Prevalence*	Specificity*
VlsE Ba	66%	99%
VlsE Bb	89%	
VlsE Bg	68%	
Lipid Ba	25%	
Lipid Bb		0%
p83		95%
p39		99%
		96%
	21%	98%
	9%	99%
	7%	100%
(BB_N38)	9%	99%
p18 (BB_P38)	22%	99%

→High specificity: 95-100%
→But: Low sensitivity: 60 %

Many factors account for this serodiagnostic ambiguity

Reasons Lyme serology tests may be false-negative (extract):

- Spirochetes are encapsulated (e.g. by lymphocytic cell walls) or deep inside host tissue (fibroblasts, neurons, etc.)
- No spirochetes in body fluid on day of test
- Genetic heterogeneity:hundreds of strains; most controls use only a few strains as reference point
- Surface antigens change with temperature
- Spirochetes in dormancy phase (L-form) with no cell walls
- Recent antibiotic treatment
- Concomitant infection with Babesia may cause immunosuppression
- Other causes of immunosuppression
- Lab tests not standardized for late stage disease
- Lab tests labeled "for investigational use only"
- Encapsulated by glycoprotein "S-layer" which impairs immune recognition
- Immune deficiency , e.g., downregulation by cytokines
- Revised Western Blot criteria fails to include most significant antigens

Source: Multiple references - see e.g. following slides

Specificity ("false positive") and sensitivity ("false negative") of Borrelia antibody tests

Year	Author/Literature	Specificity/Sensitivity
(1993)	Schmitz et al. Eur J Clin Microbiol Infect Dis 12,419.424	100% / 66%
(1995)	Engstrom SM, Shoop E et al. J Clin Microbiol 33, 419–27.	96% / 55%
(1996)	Ledue TB, Collins MF, Craig WY J Clin Microbiol 34, 2343–50.	100% / 44%
(1999)	Trevejo RT, Krause PJ et al. J Infect Dis 179, 931-8.	100% / 29%
(2001)	Nowakiwski et al. Clin Infect Dis 33, 2023-2027	99% / 66%
(2003)	Bacon RM, Biggerstaff BJ et al. J Infect Dis 187, 1187-99.	99% / 67%
(2005)	Coulter P, Lema C et al. J Clin Microbiol. 43(10), 5080–5084.	- / 25%
(2008)	Steere AC, McHugh G et al. Clin Infect Dis 47,188-95.	99% / 18%
(2008)	Binnicker MJ, Jespersen DJ et al. J Clin Microbiol 46, 2216–21.	100% / 49%
(2009)	Klemann W, Huismans BD. Umwelt-Medizin-Gesellschaft; 22(2) 132-138	- / 60%
(2010)	Schwarzbach A. (unpublished)	92% / 60% Blot - /32-42% ELISA

- **Average** **~99% / ~43%**

Borrelia antibodies by ELISA and Immunoblot: evidence-based literature on "false seronegativity"

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Borrelia antibodies by ELISA and Immunoblot: evidence-based literature on "false seronegativity"

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A T-cell test is a much better alternative: an "Elispot" lymphocyte transformation test

This tests the cellular immune response towards specific antigens; it measures the current degree of disease activity using isolated lymphocytes from a blood sample

Elispot®-LTT: FDA and CDC approved LTT technique in U.S.
Actual T-cellular activity in the blood against Borrelia burgdorferi, Chlamydia pneumoniae/trachomatis, Ehrlichia/Anaplasma

In May 2011 the U.S. Food and Drug Administration (FDA) and the Centers for Disease Control and Prevention (CDC) have approved the **Elispot®-LTT (T-Spot)** technique beneath the QuantiFERON® TB Gold In-Tube test. Both tests represent Interferon-Gamma Release Assays (IGRAs) in form of Lymphocyte Transformation Tests (LTT).
No other laboratory T-cell tests have been approved (MELISA® or ITT® techniques are not approved!) in the field of all Lymphocyte Transformation Tests (LTT) by the FDA/CDC yet.

In the paper of the CDC regarding Interferon-Gamma Release Assays (IGRAs) from May 2011 the CDC says:
"... A positive result suggests that an infection is likely, a negative result that an infection is unlikely..."
"...Results can be available within 24 hours..."

Source: Center for Disease Control and Prevention. Updated Guidelines for Using Interferon Gamma Release Assays to Detect Mycobacterium tuberculosis Infection, United States. MMWR 2010; 59 (No.RR-5) http://www.cdc.gov/mmwr/pdf/rr/rr5905.pdf

ELISPOT:
T-Cell test was a game-changer for Lyme Disease

… The sensitivity of the ELISPOT is estimated at 84%, and the specificity is 94%...

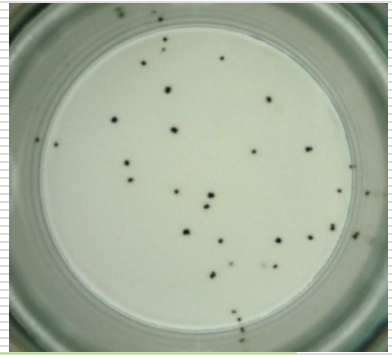
… ELISPOT assays provide robust, highly reproducible data…

… the tests in the two-assay system (ELISPOT + CD57 cell count) complement each other in the quest to understand T cell-mediated immunity in vivo….

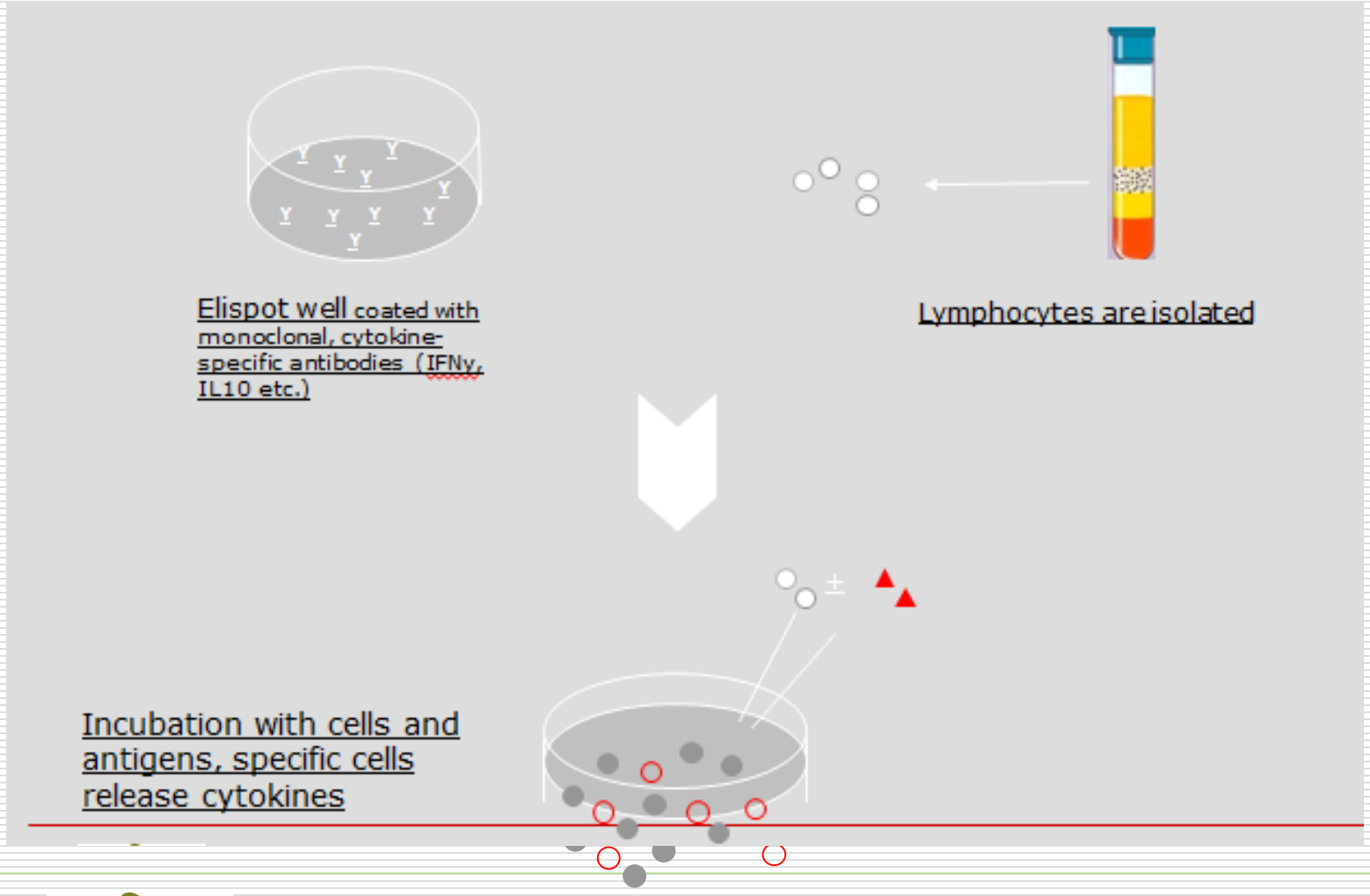
Lehman PV et al.: Unique Strengths of ELISPOT for T Cell Diagnostics in: Kalyuzhny AE. Handbook of ELISPOT: Methods and Protocols, Methods in Molecular Biology, Vol. 792. 2nd Ed: Springer; 2012: 3-23

94 % **Specificity of Borrelia Elispot-LTT**

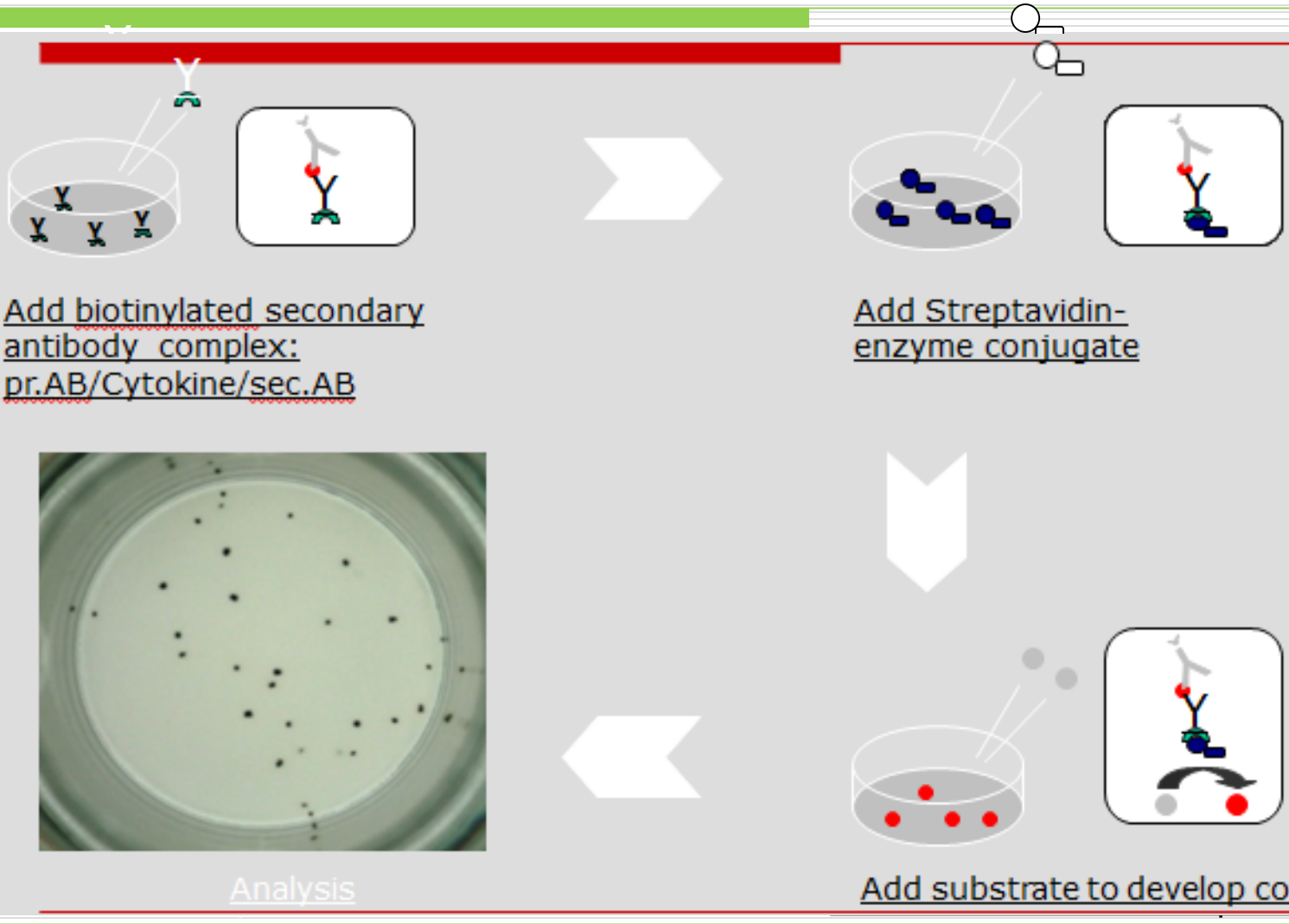
84 % **Sensitivity of Borrelia Elispot-LTT**



Elispot LTT: The principle (I)



Elispot LTT: The principle (II)



Borrelia antigens in the Borrelia EliSpot LTT

- Borrelia burgdorferi full antigen: Borrelia burgdorferi B31 reference strain (Borrelia burgdorferi sensu stricto)
- Borrelia burgorferi peptide mix: OspA from Borrelia b. sensu stricto, Borrelia afzelii, Borrelia garinii + OspC native + DbpA recombinant
- Borrelia burgdorferi LFA-1 (Lymphocyte Function Antigen 1): Own body protein + Borrelia burgdorferi sensu stricto (shared epitope). Often associated with autoimmune diseases: collagenosis, Rheumatoid Arthritis, vasculitis (ANA, CCP antibodies, ANCA)

Explanation: Native = cultured antigens; Recombinant: produced using genetic technology

Currently the EliSpot is available for:

- Borrelia burgdorferi (3 subspecies: B.b. sensu stricto + B.b. garinii + B.b. afzelii)
- Borrelia myamotoi
- **Bartonella henselae (new)**
- **Babesia microti (new)**
- Chlamydia pneumoniae
- Chlamydia trachomatis
- **Mycoplasma pneumoniae (new)**
- Ehrlichia
- Yersinia species
- Epstein Barr Virus (EBV)
- Cytomegalovirus (CMV)
- Herpes Simplex Virus 1 / 2
- **Varicella Zoster Virus (VZV) (new)**

Also new:
Candida
Aspergillus niger

Comparing Lyme Testing

Key terms:

ELISA – Enzyme Linked Immuno Sorbent Assay

Specificity - True negative rate

Sensitivity - True positive rate

Borrelia Testing Method	Summary	Testing accuracy	Clinical application
ELISA IgG / IgM	Tests B-cell imunmune response against Borrelia	Poor sensitivity Poor specificity	Screening for Borrelia antibodies
ELISA C6	Tests part of B-cell immune response against Borrelia	Poor sensitivity Poor specificity	Alternative partly screening for Borrelia antibodies
IgG/IgM Seraspot	Tests B-cell immune response (modern Westernblot)	Poor sensitivity High specificity	Confirmation test for Borrelia antibodies (modern Westernblot)
Tickplex Basic	**Tests B-cell immune response including "roundbodies"**	**High sensitivity High specificity**	**Screening for Borrelia antibodies including "roundbodies"**
Western blot	Tests B-cell immune response	Poor sensitivity High specificity	Confirmation test for Borrelia antibodies
PCR	Assesses presence of DNA of Borrelia in blood	Poor sensitivity High specificity	Reflects current presence of Borrelia
Elispot	**Tests T-cell activity against Borrelia**	**High sensitivity High specificity**	**Reflects current activity last 6-8 weeks**
Borrelia culture	Asseses presence of Borrelia in blood	Poor sensitivity High specifictiy	Reflects current presence of Borrelia

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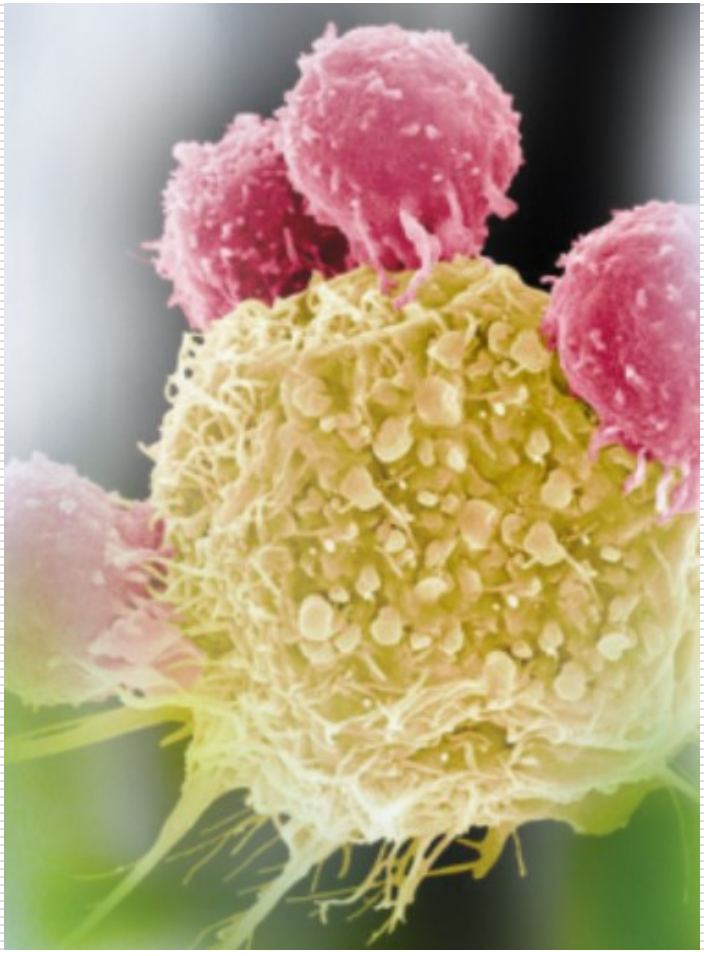
CD57+ Natural Killer cells (NK cells): CD57 flow cytometry



CD3-/CD57+ T-lymphocytes

1. Subpopulation of the CD56+ NK cells
2. Reduction may indicate **chronic** Lyme disease (symptoms > 1 year)
3. Reduction in untreated and inadequately treated Lyme disease
4. Not highly specific: Also low in other bacterial infections, esp. Chlamydia pneumoniae and Mycoplasma pneumoniae

Reference range

Lyme patient:	< 130 /ul
Healthy:	> 130 /ul

The CD57+ and chronic immune suppression

CD3-/CD57+ Cells

5 CD3-/CD56+ Flow Cytometry					
5 T cells CD3+ (%)	**+**	**82,18 %**	**62,00 - 80,00**	[.....	*>
5 T cells CD3+ (absolute)		1225 /ul	900 - 1900	[..*.....	]
5 NK cells CD56+ CD3- (%)	**-**	**4,75 %**	**6,00 - 29,00**	<*	]
5 NK cells CD56+ CD3- (absolute)		71 /ul	60 - 700	[*.....	]
5 CD57+ NK-cells (%)		18,27 %	2,00 - 77,00	[.*.....	]
5 CD57+ NK-cells (absolute)	**-**	**13 /ul**	**100 - 360**	<*	]

The result of the CD57-cell count indicates chronic immune-suppression, which can be caused by Borrelia burgdorferi or other bacteria like Chlamydia pneumoniae or Mycoplasma pneumoniae.

ELSEVIER

Immunology Letters

Volume 76, Issue 1, 1 February 2001, Pages 43-48

Decreased CD57 lymphocyte subset in patients with chronic Lyme disease

Raphael B. Stricker [a], Edward E. Winger [b]

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https://doi.org/10.1016/S0165-2478(00)00316-3

Abstract

Background: Chronic Lyme disease (LD) is a debilitating illness caused by tickborne infection with the spirochete *Borrelia burgdorferi*. Although immunologic abnormalities appear to play a role in this disease, specific immunologic markers of chronic LD have not

"All 31 chronic LD patients who were tested prior to antibiotic treatment had significantly decreased CD57 lymphocyte counts (mean, 30±16 cells per μl; normal, 60–360 cells per μl, *P*<0.001)."

Lyme symptom checklist

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Short Symptom Checklist for Lyme Borreliosis

Name, first name ..... Date: .....

►	Actual and former symptoms: Please mark with a cross	X
1	Former or recent tick bite	☐
2	Former or recent bull´s eye rash	☐
3	Summer flu after tick bite	☐
4	Fatique/Malaise/Lethargy	☐
5	Loss of physical/mental capacity, general weakness	☐
6	Neck-pain, neck stiffness	☐
7	Headache	☐
8	Painful joints, swollen joints	☐
9	General aches and pains, tendon problems	☐
10	Muscle pain, muscle weakness	☐
11	Fever, feverish feeling, shivering	☐
12	Ears: intermittent red, swollen earlap	☐
13	Heart problems, disturbance of cardiac rhythm	☐
14	Cough, expectoration, breathlessness	☐
15	Night sweat	☐
16	Sleeplessness, waking up around ..... p.m.	☐
17	Tinnitus	☐
18	Swollen lymph nodes	☐
19	Numbness of the skin	☐
20	"Burning" or "pins and needles" skin sensations, painful sole or foot	☐
21	Back pain, back stiffness	☐
22	Muscle pain, muscle weakness	☐
23	Shivering, chill	☐
24	Blurred, foggy, cloudy, flickering, double vision	☐
25	Aggressiveness, drowsiness, panic attacks, anxiety, mood swings	☐
26	Concentration problems, short-term memory loss, forgetfulness	☐
27	Skin partly thin, paper-like, transparent, dry	☐
	Total number of symptoms for Lyme Borreliosis	

Antibiotics? When? Which one(s)? How long? Please send the form to info@aonm.org or call 03331 210 305

Coinfections checklist

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Coinfections-Checklist

Name, first name ..... Date (DD/MM/YYYY) .....

	Actual and former symptoms Please mark with a cross	X	Score-Points (filled in by physician/naturopath)	Ranking
1	Stomach ache, gut problems	☐	Ehrlichia:	
2	Anaemia	☐	Babesia:	
3	Diarhoea intermittent	☐	Rickettsia:	
4	Fever or feverish feeling	☐	Bartonella:	
5	Lack of concentration, memory disturbance, forgetfulness	☐	Chl.pneumoniae:	
6	Encephalitis/Inflammation of the brain (NMR)	☐	Chl.trachomatis:	
7	Yellowish colour of the skin/eyes	☐	Yersinia:	
8	Painful joints, swollen joints	☐	Mycoplasma:	
9	General aches and pains, tendon problems	☐	Coxsackie-Virus:	
10	Flu-like symptoms intermittent	☐	EBV/CMV/HSV:	
11	Rash(es)	☐		
12	Small red/purple spots of the skin	☐		
13	Heart problems, disturbance of cardiac rhythm	☐		
14	Cough, expectoration	☐		
15	Headache	☐		
16	Impaired liver function/ liver laboratory values	☐		
17	Pneumonia, bronchitis	☐		
18	Swollen lymph nodes	☐		
19	Tonsilitis	☐		
20	Enlargement of the spleen	☐		
21	Fatigue / exhaustion, intermittent or chronic CFS	☐		
22	Muscle pain, muscle weakness	☐		
23	Shivering, chill	☐		
24	Blurred, foggy, cloudy, flickering, double vision	☐		
25	Nausea, vomiting	☐		
26	Dark urine	☐		
27	Itching or pain when urinating	☐		

Please send the form to info@aonm.org or call our helpline on 03331 210 305

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Electronic version fills automatically

Coinfections-Checklist

Name, first name ..... Date (DD/MM/YYYY) .....

	Actual and former symptoms Please mark with a cross	X	Score-Points (filled in by physician/naturopath)	Ranking
1	Stomach ache, gut problems	☒	Ehrlichia: 7	3
2	Anaemia	☐	Babesia: 4	6
3	Diarhoea intermittent	☐	Rickettsia: 6	4
4	Fever or feverish feeling	☒	Bartonella: 8	2
5	Lack of concentration, memory disturbance, forgetfulness	☒	Chl.pneumoniae: 9	1
6	Encephalitis/Inflammation of the brain (NMR)	☐	Chl.trachomatis: 5	5
7	Yellowish colour of the skin/eyes	☐	Yersinia: 5	5
8	Painful joints, swollen joints	☒	Mycoplasma: 7	3
9	General aches and pains, tendon problems	☒	Coxsackie-Virus: 9	1
10	Flu-like symptoms intermittent	☒	EBV/CMV/HSV: 7	3
11	Rash(es)	☒		
12	Small red/purple spots of the skin	☐		
13	Heart problems, disturbance of cardiac rhythm	☒		
14	Cough, expectoration	☐		
15	Headache	☒		
16	Impaired liver function/ liver laboratory values	☐		
17	Pneumonia, bronchitis	☐		
18	Swollen lymph nodes	☒		
19	Tonsilitis	☐		
20	Enlargement of the spleen	☐		
21	Fatigue / exhaustion, intermittent or chronic CFS	☒		
22	Muscle pain, muscle weakness	☒		
23	Shivering, chill	☐		
24	Blurred, foggy, cloudy, flickering, double vision	☒		
25	Nausea, vomiting	☒		
26	Dark urine	☐		
27	Itching or pain when urinating	☐		

Ranked in order of priority – draw for first place here: Chlamydia pneumoniae (CPN) and Coxsackie

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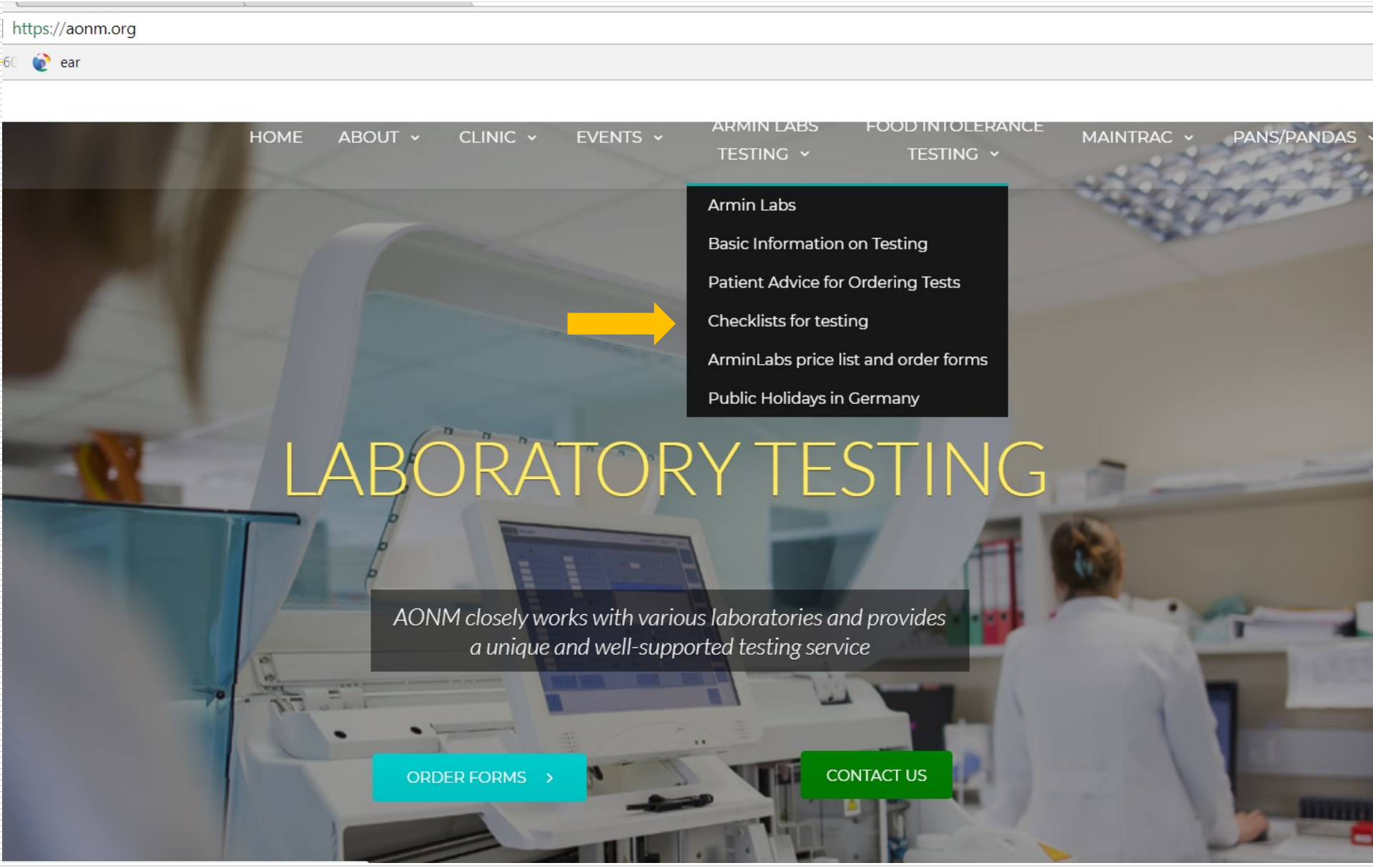
DIAGNOSING TICK-BORNE DISEASES

Where to find the checklists:

www.aonm.org – ArminLabs tab



Testing Panels in Syndromes

Syndromes that may have infectious drivers: just a few examples

- **Fibromyalgia/Rheumatoid Arthritis**
  - Correlations with Borrelia and coinfections
  - Testing suggestions
- **Myalgic Encephalomyelitis (ME/CFS)**
  - Correlations with Borrelia and coinfections
  - Testing suggestions
- **Dementia/Alzheimer's**
  - Correlations with Borrelia and coinfections
  - Testing suggestions
- **Neuropsychiatric syndromes, e.g. OCD/Tourette's**
  - Correlations with Borrelia and coinfections
  - Testing suggestions

Fibromyalgia symptoms and Borrelia – clear associations

Richard Horowitz MD
Why Can't I Get Better?

Are Your Fibromyalgia Symptoms Due to Lyme Disease?

Tick-borne disorders often mimic chronic pain syndromes

Like 6.9K

Posted Dec 15, 2013

SHARE TWEET EMAIL MORE

Lyme disease is the number one vector borne spreading epidemic worldwide, and mimics common diseases such as Fibromyalgia (FM), Chronic Fatigue Syndrome (myalgic encephalomyelitis), autoimmune diseases like rheumatoid arthritis and MS, as well as psychiatric conditions such as depression and anxiety. The CDC recently released new statistics showing that ten times more individuals have been affected with Lyme than previously suspected. Since the blood tests for diagnosing Lyme disease have been shown to be unreliable, we would expect that a certain percentage of those diagnosed with FM are in fact suffering from Lyme disease. This has been my personal experience. In the last 26 years, I have seen over 12,000 chronically ill individuals with Lyme and associated tick-borne disorders, many of whom have been to 10-20 doctors looking for answers for their chronic fatigue and musculoskeletal pain. Lyme and associated tick-borne infections were often one of the underlying causes of their problem.

?xai=AKAOjssWB9XgJzyqUXBsmsREpY3DTN2e6rV5yN7vuVDae9JXyrGCo-_DJZUoh26OH81Nu_NFyE6NKDGHMjEStHAhvpXjKJeonoK(

"Most fibromyalgia patients are Lyme positive." *(Rheum Dis Clin North Am. 1998 May;24 (2):323-51 & report of Lida Mattman,M.D.)*

Source: https://www.psychologytoday.com/blog/why-can-t-i-get-better/201312/are-your-fibromyalgia-symptoms-due-lyme-disease

Numerous scientific references substantiate this

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Staud R. *Autonomic dysfunction in fibromyalgia syndrome: postural orthostatic tachycardia.* Curr Rheumatol Rep. 2008 Dec; Vol 10 (6), pp 463-6

Fibromyalgia symptoms and Chlamydia pneumoniae (CPN)

Musculoskeletal Pain and Inflammation ■ Soft tissue infection by Chlamydia pneumoniae and subsequent inflammation. ■ Fibromyalgia Syndrome often starts after injury/accident. In the normal response to tissue repair, injured and inflamed areas attract macrophages. Chlamydia pneumoniae infected macrophages can leave Chlamydia pneumoniae behind in injured/inflamed area. Infection then becomes progressive gradually spreading from that area. As generalized inflammation increases (from free circulating cytokines) these sites are further infected by parasitized macrophages drawn to increasingly inflamed sites, etc. See http://www.cpnhelp.org/how_chlamydia_pneumoniae_ ■ Porphyrins blocking GABA receptors will also lower pain tolerance. ■ Generalized cytokine load

Chlamydial Endotoxins. Chlamydia pneumoniae contains a number of endotoxins in its structure, such as LPSi and HSPi-60. These endotoxins cause widespread inflammation (cytokine cascades) and a host of other metabolic disturbances. These are released chronically in small amounts in Chlamydia pneumoniae infection and in large amounts when Cpn cells are killed.

Source: http://www.prohealth.com/library/showarticle.cfm?libid=12763

Fibromyalgia and Mycoplasma

Diagnosis and Treatment of Chronic Mycoplasmal Infections in Fibromyalgia and Chronic Fatigue Syndromes: Relationship to Gulf War Illness

Garth L. Nicolson, Marwan Nasralla, Joerg Haier and Nancy L. Nicolson

The Institute for Molecular Medicine, 15162 Triton Lane
Huntington Beach, CA 92649

Summary

Mycoplasmal infections are associated with several acute and chronic illnesses, including Pneumonia, Asthma, Rheumatoid Arthritis, Immunosuppression Diseases such as AIDS, Genitourinary Infections and Gulf War Illness (GWI). Using forensic Polymerase Chain Reaction blood samples from 132 Chronic Fatigue Syndrome (CFS) (Myalgic Encephalomyelitis) and/or Fibromyalgia Syndrome (FMS) patients were investigated for the presence of mycoplasmal infections in blood leukocytes. CFS and FMS patients had completely overlapping signs and symptoms and were grouped for purposes of analysis. There was a significant difference between symptomatic CFS/ mycoplasmal infections (~63%) and healthy positive controls (~9%) (P incidence of *Mycoplasma fermentans* infections in these CFS/FMS pati (0%)(P<0.001). The prevalence of mycoplasmal infections in female an similar. Similar to GWI patients with mycoplasmal infections (~50%) a symptoms, mycoplasma-positive CFS/FMS patients respond to 6-week doxycycline, minocycline, ciprofloxacin, azithromycin and clarithromyci antibiotics plus nutritional support appear to be necessary for recovery.

"The identification of mycoplasma infections in the leukocyte blood fractions of a rather large subset of CFS, FMS and arthritis patients suggests that mycoplasmas, and probably other chronic infections as well, may be an important source of morbidity in these patients."

Fibromyalgia and Epstein Barr Virus/CMV

Journal of Neurology and Neuroscience

Evaluation of Antiviral Antibodies against Epstein-Barr Virus and Neurotransmitters in Patients with Fibromyalgia

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Citation: Reshkova V, Kalinova D, Milanov I. Evaluation of Antiviral Antibodies against Epstein-Barr Virus and Neurotransmitte Patients with Fibromyalgia. J Neurol Neurosci. 2016, 6:3. doi: 10.21767/2171-6625.100035

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Abstract

Fibromyalgia (FM) is characterized by chronic widespread pain lasting for a minimum of three months, and pain at mechanical pressure in at least 11 of the 18 tender points. The cause of fibromyalgia is unknown. Several hypotheses have been developed including "central sensitization". This theory proposes that fibromyalgia patients have a lower threshold for pain because of increased reactivity of painsensitive neurons in the spinal cord or brain. Some researchers supposed that different

"The obtained results revealed that high EBV IgG concentrations in the serum of patients with FM correlated with pain intensity and associated clinical symptoms. This is consistent with the fact that FM is connected to the immune response to certain infectious agents (e.g. EBV, CMV)."

Consider Borrelia in the differential diagnosis of Rheumatoid Arthritis

Clinical and Vaccine Immunology
CVI Article | Journal Info. | Authors | Reviewers | Permissions | Journals.ASM.org

Clin Vaccine Immunol. 2007 Nov; 14(11): 1437–1441. PMCID: PMC2168181
Published online 2007 Sep 19. doi: 10.1128/CVI.00151-07

Serum Reactivity against *Borrelia burgdorferi* OspA in Patients with Rheumatoid Arthritis▽

Yu-Fan Hsieh,[1] Han-Wen Liu,[1] Tsai-Ching Hsu,[1] James C.-C. Wei,[2] Chien-Ming Shih,[3] Peter J. Krause,[4] and Gregory J. Tsay[1,2,*]

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ABSTRACT

Go to:

Lyme arthritis and rheumatoid arthritis share common clinical features and synovial histology. It is unclear whether they also share similar pathogenesis. Previous studies have shown that the severity and duration of Lyme arthritis correlate directly with serum concentrations of antibody against outer surface protein A (OspA) of the causative pathogen *Borrelia burgdorferi*. We tested the sera of 68 subjects with rheumatoid arthritis, 147 subjects with other autoimmune diseases, and 44 healthy subjects who had never had Lyme

Detection of Mycoplasmal Infections in Blood of Patients with Rheumatoid Arthritis

Jörg Haier[1], Marwan Nasralla[1], A. Robert Franco[2], and Garth L. Nicolson[1,3]

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SUMMARY

Objectives: Mycoplasmal infections are associated with several acute and chronic illnesses. Some mycoplasmas can enter a variety of tissues and cells and cause system-wide or systemic signs and symptoms.

Methods: Patients (14 female, 14 male) diagnosed with Rheumatoid Arthritis (RA) were investigated for mycoplasmal infections in their blood leukocytes using a forensic Polymerase Chain Reaction (PCR) procedure. Amplification was performed with genus- and species-specific primers, and a specific radio-labeled internal probe was used for Southern hybridization with the PCR product. Patients were investigated for presence of *Mycoplasma spp.*, and positive cases were further tested for infections with the following species: *M. fermentans*, *M. hominis*, *M. pneumoniae* and *M*

Results: The *Mycoplasma spp.* sequence, which is not entirely specific for mycoplasmas, from the peripheral blood of 15/28 patients (53.6 %), and specific PCR products could not 13 patients (46.4 %). Significant differences ($p<0.001$) were found between patients and p controls in the genus-test (3/32) and in the specific tests (0/32). Moreover, the incidence o infections was similar in female and male patients. Using species-specific primers, we were infections of *M. fermentans* (8/28), *M. pneumoniae* (5/28), *M. hominis* (6/28) and *M. pene* RA patients. In 36% of the patients we observed more than one mycoplasma specie leukocytes. All multiple infections occurred as combinations of *M. fermentans* with other spe

Conclusions: The results suggest that a high percentage of RA patients have systemic mycoplasmal infections. Systemic mycoplasmal infections may be an important cofactor in the pathogenesis of RA, and their role needs to be further explored.

"The results suggest that a high percentage of RA patients have systemic mycoplasmal infections."

Fibromyalgia/Rheumatoid Arthritis: possible lab tests

(but use the checklist to tailor this to the patient)

1. Borrelia SeraSpot + Borrelia EliSpot + CD57-cells
2. Chl. pneumoniae IgG/IgA antibodies + Chl. pneumoniae EliSpot
3. Chl. trachomatis IgG/IgA antibodies + Chl. trachomatis EliSpot
4. Myco. pneumoniae IgG/IgA antibodies + Myco. pneumoniae Elispot
5. Ehrlichia/Anaplasma IgG/IgM antibodies + Ehrlichia/Anaplasma EliSpot
6. Rickettsia IgG/IgM antibodies
7. Bartonella IgG/IgM antibodies + Bartonella EliSpot
8. Coxsackie Virus IgG/IgA antibodies
9. EBV antibodies including Early Antigen + EBV EliSpot
10. CMV IgG/IgM antibodies + CMV EliSpot
11. VZV IgG/IgA/IgM antibodies + VZV EliSpot
12. HSV1/2 IgG/IgA/IgM antibodies + HSV1/2 EliSpot
13. Yersinia IgG/IgA antibodies + Yersinia EliSpot
14. HHV6 IgG/IgM antibodies
15. ANA (antinuclear antibodies) + CCP (cyclic citrullinated peptide) antibodies

ME and Lyme Borreliosis: connection recognized two decades ago

Chronic Fatigue Syndrome in Patients with Lyme Borreliosis

Johannes Treib Markus T. Grauer Anton Haass Jürgen Langenbach
Gerhard Holzer Ralph Woessner

Department of Neurology, University Hospital of the Saarland, Homburg, Germany

Key Words
Lyme disease · Neuroborreliosis · Chronic fatigue syndrome

Abstract
Several authors have reported a chronic fatigue-like syndrome in patients that have suffered from Lyme borreliosis in the past. To further investigate this suspicion of an association without sample bias, we carried out a prospective, double-blind study and tested 1,156 healthy young males for Borrelia antibodies. Seropositive subjects who had never suffered from clinically manifest Lyme borreliosis or neuroborreliosis showed significantly more often chronic fatigue ($p = 0.02$) and malaise ($p = 0.01$) than seronegative recruits. Therefore we believe it is worth examining whether an antibiotic therapy should be considered in patients with chronic fatigue syndrome and positive Borrelia serology.

Introduction

Lyme borreliosis is the most common vector-borne infection in the northern hemisphere with an annual incidence of 69 cases per 100,000 inhabitants [1]. Although the clinical course of borreliosis has been well described, data supporting the existence of a syndrome of chronic fatigue following infection is only just emerging [2]. Symptoms of the postinfectious chronic fatigue syndrome include persistent headaches, neuropsychological deficits and general malaise which can occur months or even years after infection and successful treatment of borreliosis [3–5]. This has been reported by Shadick et al. [5] in a population-based, retrospective cohort study showing a significantly higher incidence of fatigue (26 vs. 9%; $p = 0.04$) and impaired ability to concentrate (16 vs. 2%; $p = 0.03$) in patients that had been diagnosed with Lyme borreliosis when compared with an uninfected control population. In addition, Benke et al. [3] studied patients several years after they had been treated for Lyme borreliosis and compared their scores on various neuropsychological tests against an age- and education-matched control group. These patients showed deficits in memory, mental flexibility and articulatory and phonematic skills that were limited to a few memory functions and did not appear to be linked to a general mental decline.

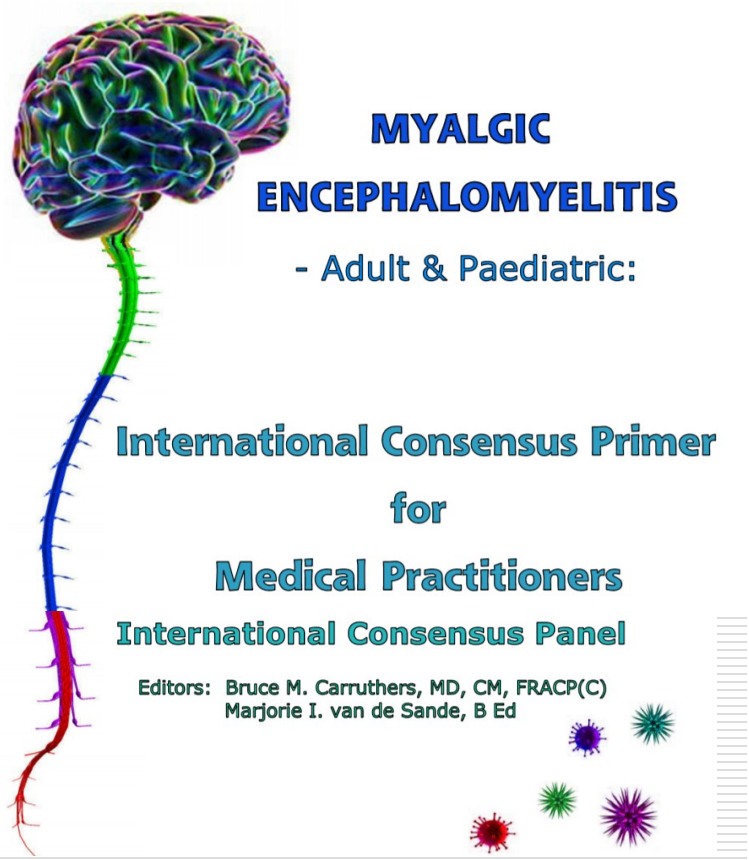
In a recent clinical and serological follow-up study, Treib et al. [6] observed a significant reduction of neurological deficits 4.2 years after antibiotic treatment. However, more than half of the patients reported unspecific complaints such as headache as well as memory and concentration problems, similar to a chronic fatigue syndrome.

The association between Lyme borreliosis and chronic fatigue remains uncertain, however. It is also unclear whether an infection with Borrelia can lead to isolated neuropsychological deficits that resemble a chronic fatigue syndrome without the patient manifesting any other clinical signs of borreliosis or neuroborreliosis.

This was almost two decades ago – in 2000: a large-cohort study, with 1,156 subjects. Conclusion:

"When encountering a patient with chronic fatigue syndrome one should consider Borreliosis as a possible cause"

The ME International Consensus Primer recommends considering Borrelia burgdorferi



Source: http://www.lymefiles.com/ME%20adult%20and%2

Immune: Tender lymphadenopathy: ☐ cervical, ☐ axillary, ☐ inguinal regions (more prominent in acute phase), ☐ flares with exertion; ☐ crimson crescents in the tonsillar fossa: ☐ demarcated along margins of both anterior and pharyngeal pillars, ☐ if patient has no tonsils, they assume a posterior position in the oropharynx; ☐ splenomegaly

GI: ☐ increased bowel sounds, ☐ abdominal bloating, ☐ abdominal tenderness: epigastrium (stomach), right lower quadrant (terminal ileum) and left lower quadrant (sigmoid colon) – *most patients have tenderness in 2-3/3 areas*

Cardiovascular & respiratory: ☐ arrhythmias: ☐ BP as above; ☐ mottling of extremities, ☐ extreme pallor, ☐ Raynaud's phenomenon, ☐ receded moons of finger nails (chronic phase) _____

Laboratory/Investigative Protocol: **Diagnose by criteria. Confirm by laboratory and other investigations.** A broad panel of tests provides a more robust basis to identify symptom patterns, abnormalities and orient treatment.

Routine laboratory investigation: ☐ CBC, ☐ ESR, ☐ CA, ☐ P, ☐ RBC Mg, ☐ vitamin D3, ☐ B12 & folate, ☐ ferritin, ☐ zinc, ☐ FBS, ☐ PC, ☐ Hb A1C, ☐ serum electrolytes, ☐ TSH, ☐ protein electrophoresis screen, ☐ CRP, ☐ creatinine, ☐ ECG (U+ T wave notching), ☐ CPK and liver function, ☐ rheumatoid factor, ☐ antinuclear antibodies, ☐ urinalysis, ☐ essential fatty acids, ☐ CoEnzyme Q10, ☐ immunoglobulins, ☐ diurnal cortisol levels, ☐ TTG, ☐ serotonin

Additional laboratory investigation: *(as indicated by symptoms, history, clinical evaluation, lab findings, risk factors)* ☐ 24 hour urine free cortisol, ☐ DHEA sulphate, ☐ ACTH, ☐ chest x-ray, ☐ hormones including free testosterone ☐ panoramic x-ray of dental roots, ☐ amino acid profile, ☐ abdominal ultra sound, ☐ lactose/fructose breath test

Further testing with specificity to ME, *if and as indicated*. *Some tests are in the research stage but can identify abnormalities and focus treatment. Viral tests should be interpreted by a physician experienced in these infections.*

Pathogen	*Tests*	*Pathogen*	*Tests*
☐ Enterovirus	RT-PCR, serology, stomach biopsy	☐ mycoplasma	DNA-PCR, serology
☐ EBV, ☐ CMV, ☐ HHV-6	DNA-PCR, serology, antigenemia	☐ Borrelia burgdorferi	DNA-PCR, serology, Western Blot
☐ Clamydia pneumonia	DNA PCR, serology	☐ Parvovirus B19	DNA-PCR, IgG, IgM,

Immune system profiles: ☐ ***↓NK cell function & ↑ cytotoxicity**; ☐ B & T-cell function: ☐ IgG, ☐ IgG subclasses 1-4; ☐ IgA, ☐ IgM (shift from T1 to T2), ☐ cytokine/chemokine profile panel (94% accuracy): IL-8, IL-13, MIP-1β, MCP-1, IL4, ☐ flow cytometry for ↑ lymphocyte activity, ☐ ↑ 37 kDa 2-5A RNase L immunoassay – defect/ratio & bioactivity, ☐ food sensitivity panel, ☐ chemical sensitivities, ☐ stool for WCB - D-lactic acid bacteria balance, ova & parasites, ☐ autoimmune profile, **Intestinal dysbiosis:** ☐ IgA & IgM for intestinal aerobic bacteria in serum, ☐↑ leukocyte elastase activity in PBMCs, ☐ IgG food intolerance test, ☐ toxoplasmosis

Neurological & static testing: ☐ ***SPECT scan with contrast - ↓** cortical/cerebellar region cerebral blood flow (rCBF) in the frontal, parietal, temporal and occipital & brain stem regions - *more brain involvement indicates increased illness severity*, ☐ **MRI of brain –** *(increased T2-weighted images in high white matter tracts & loss of GM volume) & rule out MS,* ☐ **MRI of spine** *(dynamic disc bulges/herniation , stenosis),* ☐ **sleep study** *(↓ stage 4 sleep, sleep pattern & rule out treatable sleep dysfunctions – upper airway resistance syndrome, sleep apnea, etc.)*

PENE: *A 2 consecutive day comprehensive 8-12 minute cardiopulmonary exercise stress test (measuring heart, lung, and metabolic function) - only ME patients have significantly worse scores the second day & abnormal recovery from exertion.*

* **Exercise tolerance test with expired gas exchange** - (2 consecutive days) – *measure cardiovascular, pulmonary &*

Editors: Carruthers & van de Sande 11

T & DIAGNOSTIC WORKSHEET

References for the Lyme Disease/ME connection (1/2)

Undiagnosed Lyme disease often called ME or CFS

Gustaw K. Chronic fatigue syndrome following tick-borne diseases.Neurol Neurochir Pol.2003;37:1211–21. 71% of ME/CFS cases had Lyme infection.

Chronic Fatigue Syndrome Patients Subsequently Diagnosed with Lyme Disease Borrelia burgdorferi: Evidence for Mycoplasma species Co-Infections Garth L. Nicolson, PhD, Nancy L. Nicolson, PhD and Joerg Haier, MD, PD, Journal of Chronic Fatigue Syndrome 2008; 14(4):5-17

Several Bacteria and ME / CFS scientific research papers at http://www.immed.org/fatigue_illness_research.html

RETROSPECTIVE ANALYSIS OF A COHORT OF INTERNATIONALLY CASE DEFINED CHRONIC FATIGUE SYNDROME PATIENTS IN A LYME ENDEMIC AREA.. Samuel Shor, MD, FACP

Treib, J., Grauer, M.T., Haas, A., Langenbach, J., Holzer, G.,Woessner, R. Chronic Fatigue Syndrome in patients with Lyme borreliosis Eur Neurol. 2000; 43(2): 107 - 9

Nicolson GL, and Nicolson NL. Chronic infections as a common etiology for many patients with chronic fatigue syndrome, fibromyalgia, and Gulf War Illness. Intern J Med. 1998; 1:42-6.

Taylor, S. Lyme disease (borreliosis). A plague of ignorance regarding the ignorance of a plague. http://www.autoimmunityresearch.org/lyme-disease/

In a presentation to the Edinburgh ME group in September 2005, Professor John Gow of Glasgow University stated that gene expression regulation in those with ME is identical to that seen in Lyme patients.

Relationship between Lyme Disease and CFS. Critical Needs and Gaps in Understanding Prevention, Amelioration, and Resolution of Lyme and Other Tick-Borne Diseases: The Short-Term and Long-Term Outcomes - Workshop Report. Released: April 20, 2011

Donta, S. Lyme disease as a model of Chronic Fatigue Syndrome. CFS Res Rev. 2002; 3(2): 1 - 4

Source: http://www.me-ireland.com/scientific/8.htm

References for the Lyme Disease/ME connection (2/2)

Goldenberg, D.L. Fibromyalgia, chronic fatigue syndrome, and myofascial pain syndrome. Curr Opin Rheumatol. 1995; 7(2): 127 - 35

Reponse to Dr. Ho-Yen in Scotland regarding Lyme and ME / CFS

News article at Lyme disease — a ticking timebomb that health authorities say does not exist

Evidence from Dr Samuel Shor, USA

See paper at RETROSPECTIVE ANALYSIS OF A COHORT OF INTERNATIONALLY CASE DEFINED CHRONIC FATIGUE SYNDROME PATIENTS IN A LYME ENDEMIC AREA

Results: Of the total 210 included in the analysis, 209 or 99% were felt to represent a high likelihood of "seronegative Lyme disease." Initiating various antimicrobial regimen, involved at least a 50% improvement in clinical status in 130 or 62%. Another 55 patients subjectively identified a beneficial clinical response to antimicrobials, representing a total of 188 or 88% of the total identified as having a high potential for seronegative Lyme disease.

Conclusions: A potentially substantial proportion of patients with what would otherwise be consistent with internationally case defined CFS in a Lyme endemic environment actually have a perpetuation of their symptoms driven by a persistent infection by Borrelia burgdorferi. By treating this cohort with appropriately directed antimicrobials, we have the ability to improve outcomes.

This is verified in another paper Lyme Disease Presenting as Chronic Fatigue Syndrome

Evidence from Dr Kenny de Meirleir, Belgium. The following evidence concerning the relationship between ME, CFS and undiagnosed lyme disease was presented by Dr. Kenny De Meirleir to the Belgian Senate in 2014 - ME, CFS, Lyme Presentation

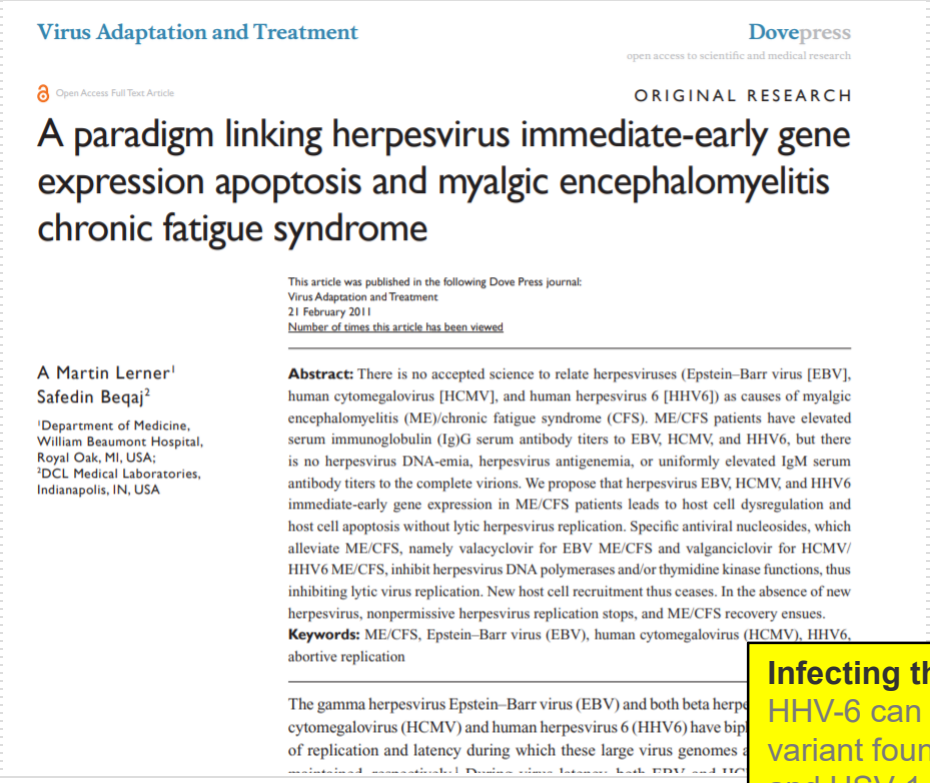
A news report of this conference was provided on http://nelelijnen.be/index.php/lyme/300round-tafel-23-april-2014

Evidence presented by Dr. Richard Horowitz to the Belgian Parliament: The following presentation was made to the Belgian Parliament in June 2014 by Dr. Richard Horowitz, who has treated over 12,000 patients with Chronic Lyme, ME, and other infectious diseases

https://www.youtube.com/watch?v=JSx3KdFaupA&t=8m40s

Source: http://www.me-ireland.com/scientific/8.htm

In patients with a central nervous system component, think herpes viruses (EBV, HSV, CMV, VZV, HHV6)



Virus Adaptation and Treatment

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ORIGINAL RESEARCH

A paradigm linking herpesvirus immediate-early gene expression apoptosis and myalgic encephalomyelitis chronic fatigue syndrome

This article was published in the following Dove Press journal:
Virus Adaptation and Treatment
21 February 2011
Number of times this article has been viewed

A Martin Lerner[1]
Safedin Beqaj[2]

[1]Department of Medicine, William Beaumont Hospital, Royal Oak, MI, USA; [2]DCL Medical Laboratories, Indianapolis, IN, USA

Abstract: There is no accepted science to relate herpesviruses (Epstein–Barr virus [EBV], human cytomegalovirus [HCMV], and human herpesvirus 6 [HHV6]) as causes of myalgic encephalomyelitis (ME)/chronic fatigue syndrome (CFS). ME/CFS patients have elevated serum immunoglobulin (Ig)G serum antibody titers to EBV, HCMV, and HHV6, but there is no herpesvirus DNA-emia, herpesvirus antigenemia, or uniformly elevated IgM serum antibody titers to the complete virions. We propose that herpesvirus EBV, HCMV, and HHV6 immediate-early gene expression in ME/CFS patients leads to host cell dysregulation and host cell apoptosis without lytic herpesvirus replication. Specific antiviral nucleosides, which alleviate ME/CFS, namely valacyclovir for EBV ME/CFS and valganciclovir for HCMV/ HHV6 ME/CFS, inhibit herpesvirus DNA polymerases and/or thymidine kinase functions, thus inhibiting lytic virus replication. New host cell recruitment thus ceases. In the absence of new herpesvirus, nonpermissive herpesvirus replication stops, and ME/CFS recovery ensues.

Keywords: ME/CFS, Epstein–Barr virus (EBV), human cytomegalovirus (HCMV), HHV6, abortive replication

The gamma herpesvirus Epstein–Barr virus (EBV) and both beta herpe
cytomegalovirus (HCMV) and human herpesvirus 6 (HHV6) have bip
of replication and latency during which these large virus genomes a

"We propose that herpesvirus EBV, HCMV, and HHV6 immediate-early gene expression in ME/CFS patients leads to host cell dysregulation and host cell apoptosis without lytic herpesvirus replication. Specific antiviral nucleosides, which alleviate ME/CFS, namely valacyclovir for EBV ME/CFS and valganciclovir for HCMV/ HHV6 ME/CFS, inhibit herpesvirus DNA polymerases and/or thymidine kinase functions, thus inhibiting lytic virus replication. New host cell recruitment thus ceases. In the absence of new herpesvirus, nonpermissive herpesvirus replication stops, and ME/CFS recovery ensues."

Infecting the brain via the Olfactory Nerve, Limbic Encephalitis

HHV-6 can travel to the brain through the nose, and is also the dominant variant found in the sensory ganglia (Hufner 2007). Like HHV-6, measles and HSV-1 tend to affect the limbic system as well as the hippocampus (Harberts 2011). There have been a number of abnormalities found in CFS patients in the hippocampus: reduced concentration of N-acetylaspartate, (Brooks 2000), hippocampal atrophy and 5-HT1A receptor binding in the hippocampus (Cleare 2005).[1]

Source: 1. https://hhv-6foundation.org/associated-conditions/hhv-6-and-chronic-fatigue-syndrome

Coxsackie frequently found in ME – especially B1

"L. Postviral Fatigue Syndrome - also known as myalgic encephalomyelitis (ME), it occurs as both sporadic and epidemic cases. It is a poorly characterized illness, the cardinal feature being excess fatiguability of the skeletal muscles. Other symptoms that may be present include muscle pain, headache, inability to concentrate, paraesthesiae, impairment of short term memory and poor visual accommodation. Focal neurological signs are rare. Evidence of myopericarditis may be present occasionally. There may be a history of a nonspecific viral illness and some lymphadenopathy may be present. Routine laboratory investigations are usually normal. Recovery usually takes place within a few weeks or months but the illness may persists in some patients with periods of remission and relapse.

The aetiology is uncertain but it is thought that there is a substantial functional component as well as a viral component in many cases. ME occasionally follows confirmed virus infections such as varicella/zoster, influenza A and IM. It may follow some bacterial infections such as toxoplasma gondii and leptospira. In the majority of cases though, the initiating infection cannot be diagnosed specifically. **There is now substantial evidence for a persistent enterovirus infection, particularly coxsackie B viruses in many cases of ME. Patients with ME appears to have a higher prevalence of antibodies against coxsackie B viruses than matched controls. Furthermore, coxsackie B viruses may occasionally be isolated from the faeces as well as skeletal muscle biopsies in patients with ME."**

Source: http://virology-online.com/viruses/Enteroviruses5.htm

ME (Myalgic Encephalomyelitis), often called CFS in the UK *(but do the checklist to tailor this)*

1. Borrelia SeraSpot + Borrelia EliSpot + CD57 cells
2. Chlamydia pneumoniae IgG/IgA antibodies + Chlamydia pneumoniae EliSpot
3. Mycoplasma pneumoniae IgG/IgA antibodies
4. Bartonella IgG/IgM antibodies
5. Parvovirus B19 IgG/IgM antibodies
6. Echovirus IgG/IgM antibodies
7. Coxsackie Virus IgG/IgA antibodies
8. EBV EliSpot
9. CMV EliSpot
10. Herpes Simplex Virus 1/2 IgG/IgA/IgM antibodies + Herpes simplex Virus EliSpot
11. HHV 6-IgG/IgM antibodies

2016 editorial, Journal of Alzheimer's Disease, 31 authors: "incontrovertible evidence of a microbial component"

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doi: 10.3233/JAD-160152
PMCID: P
NIHMSID: NI

Microbes and Alzheimer's Disease

Ruth F. Itzhaki,[a,*] Richard Lathe,[b,*] Brian J. Balin,[c] Melvyn J. Ball,[d] Elaine L. Bearer,[e] Heiko Braak,[f] Maria J. Chris Carter,[h] Mario Clerici,[i] S. Louise Cosby,[j] Kelly Del Tredici,[f] Hugh Field,[k] Tamas Fulop,[l] Claudio Grassi, Griffin,[n] Jürgen Haas,[b] Alan P. Hudson,[o] Angela R. Kamer,[p] Douglas B. Kell,[q] Federico Licastro,[r] Luc Letenr Lövheim,[t] Roberta Mancuso,[u] Judith Miklossy,[v] Carola Otth,[w] Anna Teresa Palamara,[x] George Perry,[y] Chris Preston,[z] Etheresia Pretorius,[aa] Timo Strandberg,[bb] Naji Tabet,[cc] Simon D. Taylor-Robinson,[dd] and Judith A. Hudson[ee]

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We are researchers and clinicians working on Alzheimer's disease (AD) or related topics, and we write express our concern that one particular aspect of the disease has been neglected, even though treatment based on it might slow or arrest AD progression. We refer to the many studies, mainly on humans, implicating specific microbes in the elderly brain, notably herpes simplex virus type 1 (HSV1), *Chlam pneumoniae*, and several types of spirochaete, in the etiology of AD [1–4]. Fungal infection of AD bra 6] has also been described, as well as abnormal microbiota in AD patient blood [7]. The first observati of HSV1 in AD brain were reported almost three decades ago [8]. The ever-increasing number of these studies (now about 100 on HSV1 alone) warrants re-evaluation of the infection and AD concept.

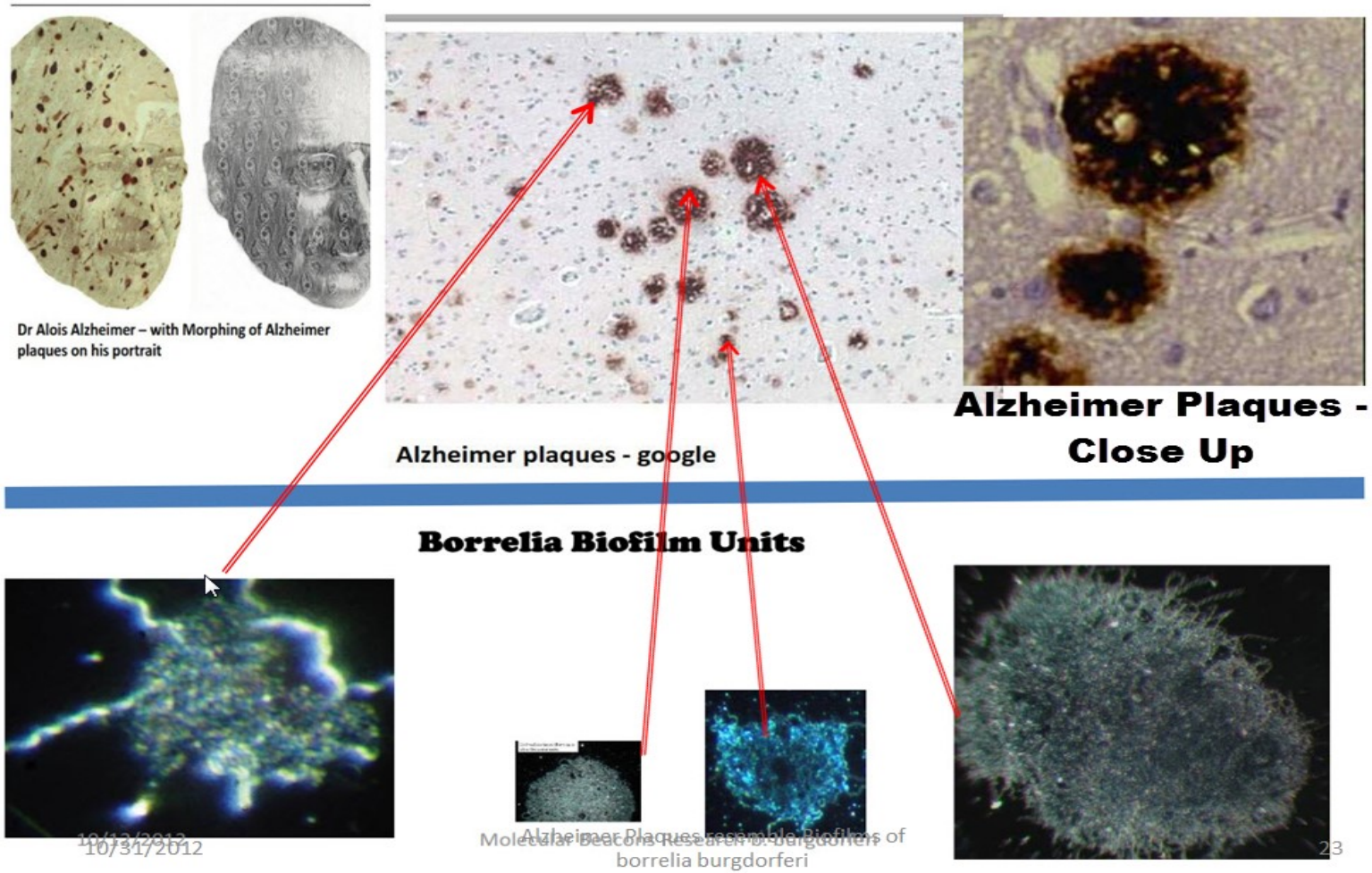
AD is associated with neuronal loss and progressive synaptic dysfunction, accompanied by the deposition

"There is incontrovertible evidence that Alzheimer's Disease has a dormant microbial component. We can't keep ignoring all of the evidence"

Professor Douglas Kell, Manchester University

"We refer to the many studies, mainly on humans, implicating specific microbes in the elderly brain, **notably herpes simplex virus type 1 (HSV1), *Chlamydia pneumoniae*, and several types of spirochaete**, in the etiology of AD [1–4]."

Alzheimer Plaques can be Borrelia biofilms – also confirmed by Dr. Dale Bredesen now



Amyloid plaques in Alzheimer's Disease: Protection against microbial infection?

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Amyloid-β peptide protects against microbial infection in mouse and worm models of Alzheimer's disease

Deepak Kumar Vijaya Kumar[1,*], Se Hoon Choi[1,*], Kevin J. Washicosky[1,*], William A. Eimer[1], Stephanie Tucker[1], Jessica Ghofrani[1], Aaron Lefkowitz[1], Gawain McColl[2], Lee E. Goldstein[3], Rudolph E. Tanzi[1,†] and Robert D. Moir[1,†]

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* These authors contributed equally to this work.

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Rehabilitation of a β-amyloid bad boy

A protein called Aβ is thought to cause neuronal death in Alzheimer's disease (AD). Aβ

Science Translational Medicine

Vol 8, Issue 340
25 May 2016

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"Our findings raise the intriguing possibility that Alzheimer's pathology may arise when the brain perceives itself to be under attack from invading pathogens"

Alzheimers / Dementia

1. Borrelia SeraSpot + Borrelia-EliSpot + CD57 cells
2. Chlamydia pneumoniae IgG/IgA antibodies + Chlamydia pneumoniae EliSpot
3. Mycoplasma pneumoniae Elispot and IgG/IgA antibodies
4. Coxsackie Virus IgG/IgA antibodies
5. Herpes simplex virus 1 / 2 IgG/IgA/IgM antibodies + Herpes simplex virus EliSpot
6. EBV EliSpot
7. CMV EliSpot

Vast number of microbes associated with psychiatric symptoms (1/2) ….

Box 1. Some microbes associated with mental symptoms & mental illness.

Spirochetes

- *Borrelia afzelii* (Lyme disease in the UK and the rest of Europe)
- *Borrelia burgdorferi sensu stricto* (Lyme disease in the USA, UK and rest of Europe)
- *Borrelia garinii* (Lyme disease in the UK and rest of Europe)
- *Borrelia hermsii* (relapsing fever)
- *Borrelia turicatae* (relapsing fever)
- *Leptospira* (Leptospirosis)
- *Treponema pallidum pallidum* (syphilis)

Bacteria

- *Anaplasmas phagocytophilum* (human granulocytic ehrlichiosis)
- *Bartonella henselae* (cat scratch fever)
- *Bartonella quintana* (trench fever)
- *Bartonella rochalimae* (bartonellosis)
- *Chlamydophilia pneumoniae* (chlamydia)
- *Chlamydophila psittaci* (chlamydia)
- *Coxiella burnetti* (Q-fever and post-Q fever fatigue syndrome)
- *Ehrlichia chaffeensis* (human monocytic ehrlichiosis)
- *Francisella tularensis* (rabit fever or tularemia)
- *Haemophilus influenzae* (haemophilus)
- *Listeria*
- *Meningococcus* (meningococcal meningitis)
- *Mycoplasma fermentans*
- *Mycoplasma pneumoniae*
- *Mycobacterium tuberculosis* (tuberculosis)
- *Rickettsia akari* (rickettsialpox)
- *Rickettsia rickettsii* (rocky mountain spotted fever)
- *Rickettsia* species (eastern tick-borne rickettsiosis)
- *Shigella* (shigellosis)
- *Streptococcus pneumoniae* or pneumococcus (pneumonia)
- *Streptococcus* (pediatric autoimmune diseases associated with *Streptococcus*, Sydenham's chorea and St Vitus dance)

Yeast

- *Candida albicans* (candidiasis)
- *Candida dubliniensis*

Source: Bransfield RC. Preventable cases of autism: relationship between chronic infectious diseases and neurological outcome. Ped Health. 2009;3:125–40.

… Microbes associated with neuropsychiatric syndromes (2/2)

Box 1. Some microbes associated with mental symptoms & mental illness (cont.).

Prion

- Variant Creutzfeldt–Jakob

Viruses

- Borna virus
- *Coltivirus* (Colorado tick fever)
- *Coxsackievirus*
- *Cytomegalovirus*
- *Enterovirus*
- *Flaviviridae* virus (Japanese B encephalitis)
- Hepatitis C virus
- Herpes virus family
- Human endogenous retroviruses
- *Human herpesvirus 4* or Epstein–Barr virus
- HIV
- Influenza A virus subtype H3N2 (Hong Kong flu)
- Influenza virus
- Pandemic influenza of 1918
- *Papopavirus*
- *Paramyxovirus* (measles virus)
- *Parvo B19*
- *Poliovirus*
- Rabies virus
- Rubella
- Toga virus
- Varicella zoster virus (chicken pox)
- Viral meningitis
- West Nile virus

Protozoa

- *Plasmodium* (malaria)
- *Babesia microti* (babesiosis)
- *Babesia duncani* (babesiosis)
- Other *Babesia* species (babesiosis)
- *Toxoplasma gondii* (toxoplasmosis)

Parasites

- *Blastocystis* (blastocystosis)
- *Taenia solium* (neurocysticercosis or cysticercosis)

Fungal

- *Cryptococcoccus*
- Coccidiomycosis
- Histomycosis

Source: Bransfield RC. Preventable cases of autism: relationship between chronic infectious diseases and neurological outcome. Ped Health. 2009;3:125–40.

"Greater frequency of Lyme disease symptoms and disease-related impairment was related to greater OCD"

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Obsessive-compulsive symptoms in adults with Lyme disease

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ARTICLE INFO

Keywords:
Lyme disease
Obsessive compulsive disorder
OCD
Lyme

ABSTRACT

Objective: This study examined the phenomenology and clinical characteristics of obsessive compulsive symptoms (OCS) in adults diagnosed with Lyme disease.

Method: Participants were 147 adults aged 18–82 years (M = 43.81, SD = 12.98) who reported having been diagnosed with Lyme disease. Participants were recruited from online support groups for individuals with Lyme disease, and completed an online questionnaire about their experience of OCS, Lyme disease characteristics, and the temporal relationship between these symptoms.

Results: OCS were common, with 84% endorsing clini
symptoms onset during the six months following their L
symptoms were temporally related. Despite the common
identified these symptoms as problematic. Greater freq
impairment was related to greater OCS. In the majority of
to psychological and pharmacological treatment. Around
provement in OCS following antibiotic treatment.

"… greater OCS occurred in the group who experienced the symptoms, in comparison to the group that did not experience that symptom."

Source: Johnco C, Kugler BB, Murphy TK, Storch EA. Obsessive-compulsive symptoms in adults with Lyme disease. Gen Hosp Psychiatry. 2018;51:85-89.

Presence of Toxoplasma gondii more frequent in OCD patients than in controls *(2017 study, 7471 subjects)*

Eur Psychiatry. 2017 Feb;40:82-87. doi: 10.1016/j.eurpsy.2016.09.001. Epub 2016 Dec 16.

Toxoplasma-infected subjects report an Obsessive-Compulsive Diso and score higher in Obsessive-Compulsive Inventory.

Flegr J[1], Horáček J[2].

Author information

Abstract

BACKGROUND: Latent toxoplasmosis, the life-long presence of dormant stages of Toxoplasma in in anamnestic IgG antibodies in blood, affects about 30% of humans. Infected subjects have an increas including schizophrenia. Several studies, as well as the character of toxoplasmosis-associated distu toxoplasmosis could also play an etiological role in Obsessive-Compulsive Disorder (OCD).

METHODS: The aim of the present cross-sectional study performed on a population of 7471 volunte between toxoplasmosis and OCD, and toxoplasmosis and psychological symptoms of OCD estimate Inventory-Revised (OCI-R).

RESULTS: Incidence of OCD was 2.18% (n=39) in men and 2.28% (n=83) in women. Subjects with toxoplasmosis had about a 2.5 times higher odds of OCD and about a 2.7 times higher odds of learning disabilities. The incidence of 18 other neuropsychiatric disorders did not differ between Toxoplasma-infected and Toxoplasma-free subjects. The infected subjects, even the OCD-free subjects, scored higher on the OCI-R.

LIMITATIONS: Examined subjects provided the information about their toxoplasmosis and OCD statuses themselves, which could result in underrating the strength of observed associations.

CONCLUSIONS: The results confirmed earlier reports of the association between toxoplasmosis and OCD. They also support recent claims that latent toxoplasmosis is in fact a serious disease with many impacts on quality of life of patients.

"…. the presence of anti-Toxoplasma gondii IgG in serum was more frequent in OCD patients than in controls …

In a 1991 study, Strittmatter and colleagues showed that the CNS areas most affected by T. gondii were the cerebral hemispheres (91%) and the basal ganglia (78%) which are implicated in OCD neurobiology [146]."

Source: Flegr, J.; Horacek, J. Toxoplasma-infected subjects report an obsessive-compulsive disorder diagnosis more often and score higher in obsessive-compulsive inventory. Eur. Psychiatry 2017, 40, 82–87.

Mycoplasma also associated with OCD and Tourette's

Open Neurol J. 2012; 6: 124–128.
Published online 2012 Nov 16. doi: [10.2174/1874205X01206010124]
Suppl 1

PMCID: PMC3514747
PMID: 23230453

The Relationship between Tourette's Syndrome and Infections

Daniela L Krause* and Norbert Müller

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"*Mycoplasma* has also been associated with OCD, Tourette's syndrome, parkinsonism, and dystonia.137–139"[1]

Abstract

Increasing evidence shows that infections and an activated immune status might be involved in the pathogene-sis of tic disorders. Studies discuss the influence of neurotrophic bacteria and viruses on different psychiatric disorders. In addition, signs of inflammation and immunological abnormalities have been described especially in schizophrenia and Tourette's syndrome (tic disorder). Neuroimaging studies revealed increased microglial activation in psychiatric diseases; indicating an inflammatory state of the CNS.

However, it still remains unclear what the underlying mechanism is of how infectious agents could contribute to tic symp-toms. One hypothesis is that not only one particular infectious agent causes directly to the disease; instead different (chronic) infections influence the immune balance and are therefore involved in the pathology. In tic disorders, infections with group A streptococci, *Borrelia burgdorferi* or *Mycoplasma pneumoniae* seem to be associated with symptoms of the disease. Studies have shown that immunologic treatment improves and prevents the re-occurrence of clinical symptoms in Tourette's syndrome. Also post-infectious events by cross-reactive antibodies(against M-protein) and an altered dopamine rgic(noradrenergic) neurotransmission as well as inflammatory/immunological dysregulations were considered as possible mechanisms to cause symptoms. Another contributing factor to the pathogenesis of these diseases could be an activation of the tryptophan catabolism through infectious

Source: 1. Rhee H, Cameron DJ. Lyme disease and pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS): an overview. International Journal of General Medicine. 2012;5:163-174; 2. Muller N, Riedel M, Forderreuther S, Blendinger C, Abele-Horn M. Tourette's syndrome and Mycoplasma pneumoniae infection. Am J Psychiatry. 2000;157(3):481–482

OCD/Tourette's Syndrome

1. Borrelia SeraSpot + Borrelia-EliSpot + Tickplex Basic + CD57-cells
2. Toxoplasma IgG/IgM
3. Mycoplasma pneumoniae IgG/IgA antibodies + Mycoplasma EliSpot
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□5	Borrelia DNA PCR	EDTA	£134
□6	Ehrlichia/Anaplasma Elispot	CPDA	£76
□7A	Anaplasma IgM/IgG antibodies	Serum	£80
□7B	Ehrlichia IgM/IgG antibodies	Serum	£80
□8	Ehrlichia/Anaplasma DNA PCR	2 x EDTA	£157
□9	Bartonella IgG antibodies (henselae + quintana)	Serum	£80
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□12A	Babesia microti Elispot	CPDA	£76
□13	Babesia DNA PCR	EDTA	£157
□14	Babesia DNA FISH	2 x EDTA	£236
□15	Chlamydia pneumoniae Elispot	CPDA	£76
□16	Chlamydia pneumoniae IgG/IgA antibodies	Serum	£55
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□46	Total protein, protein electrophoresis	Serum	£18
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□49	Thyroid antibodies (TPO abs, TG Abs, TSH receptor Abs)	Serum	£114
□50	Parvovirus B19 IgG/IgM antibodies	Serum	£46
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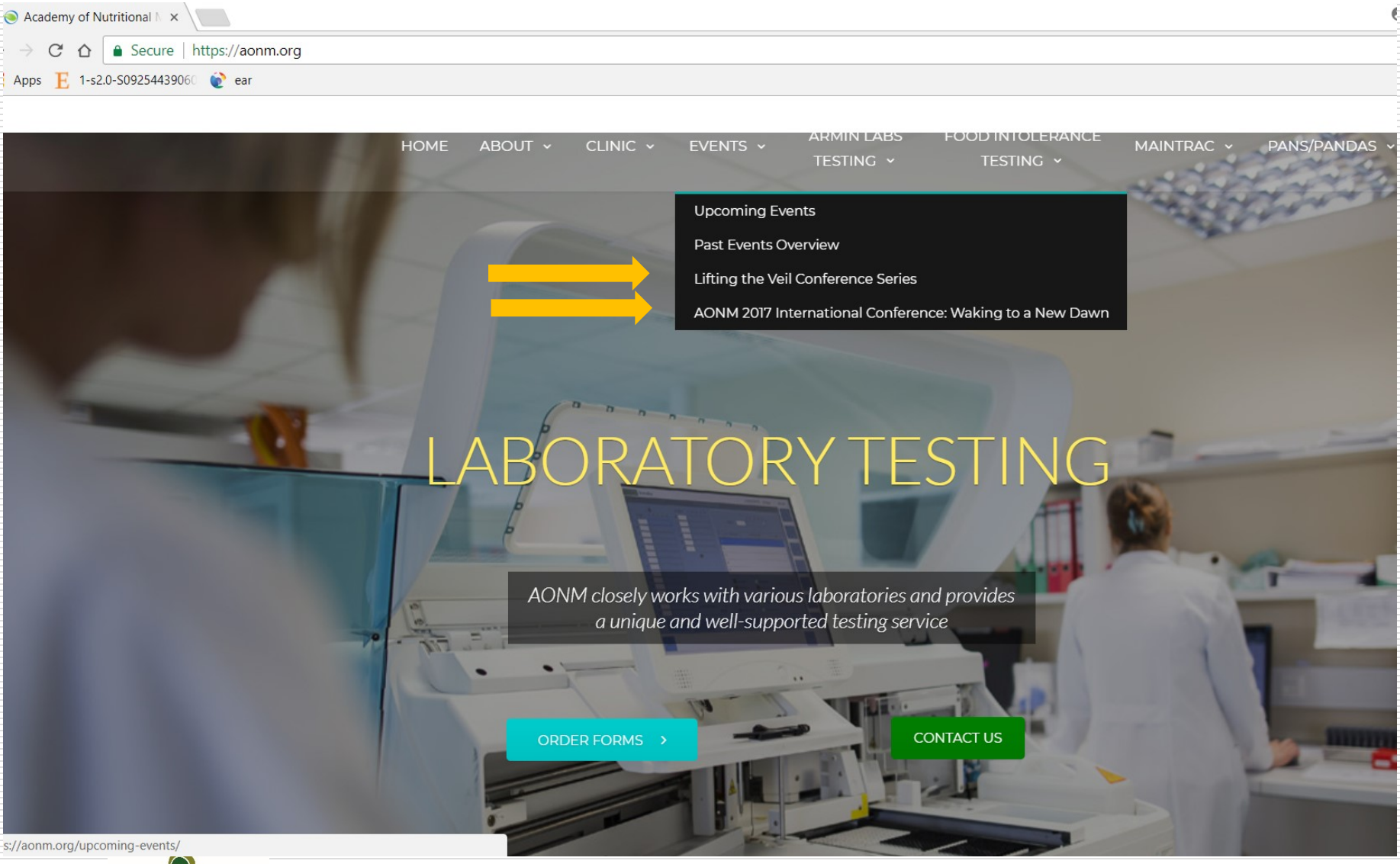

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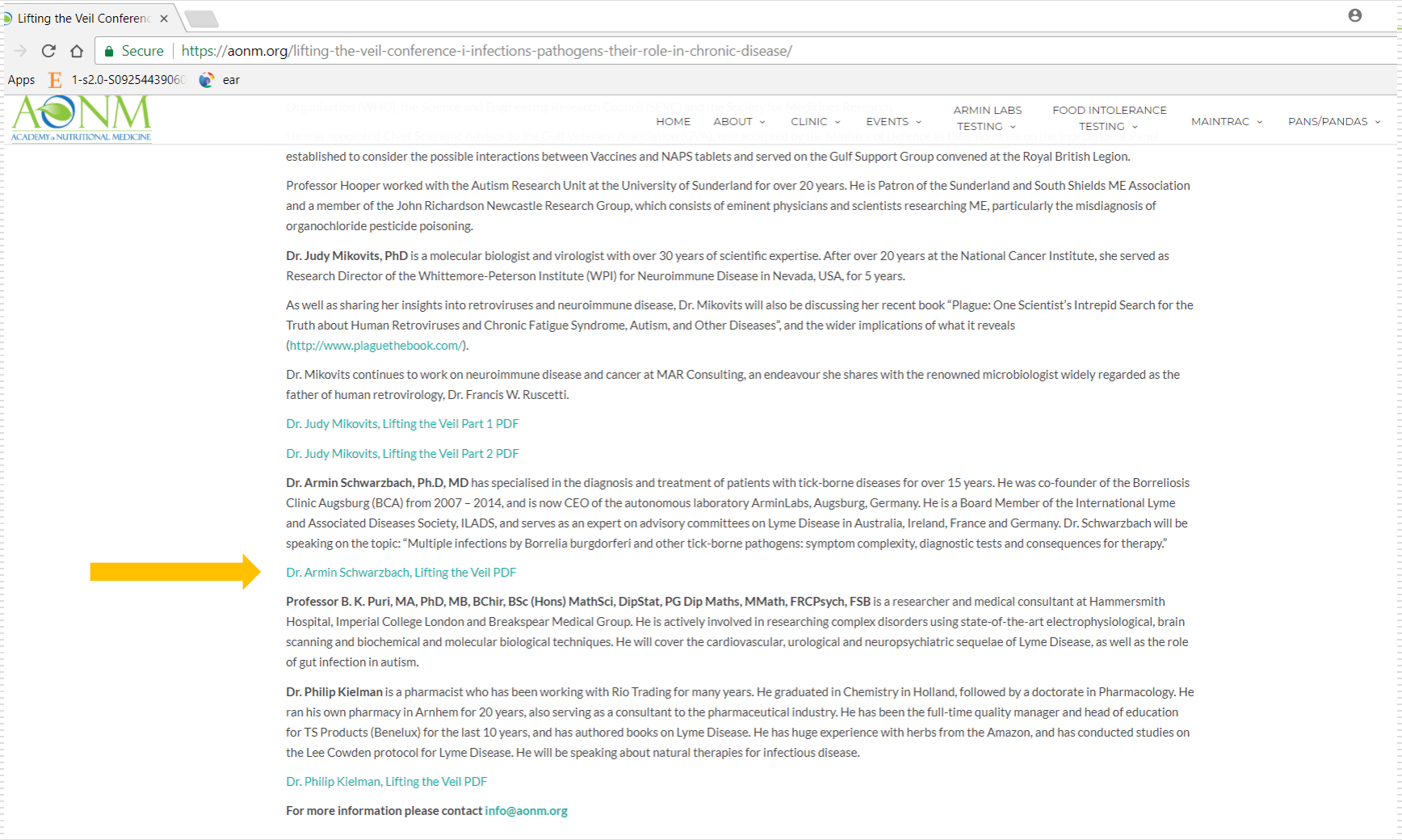

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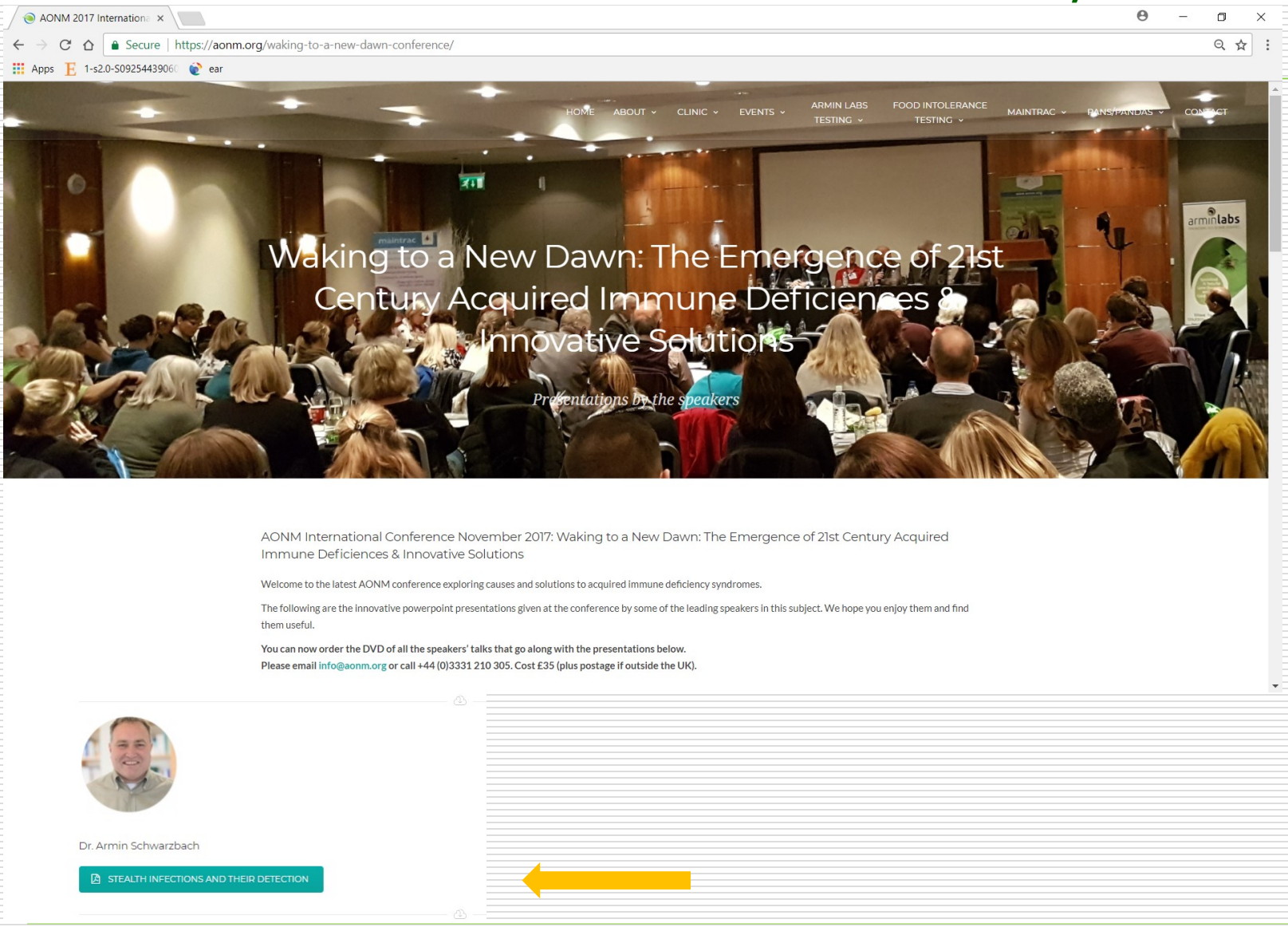

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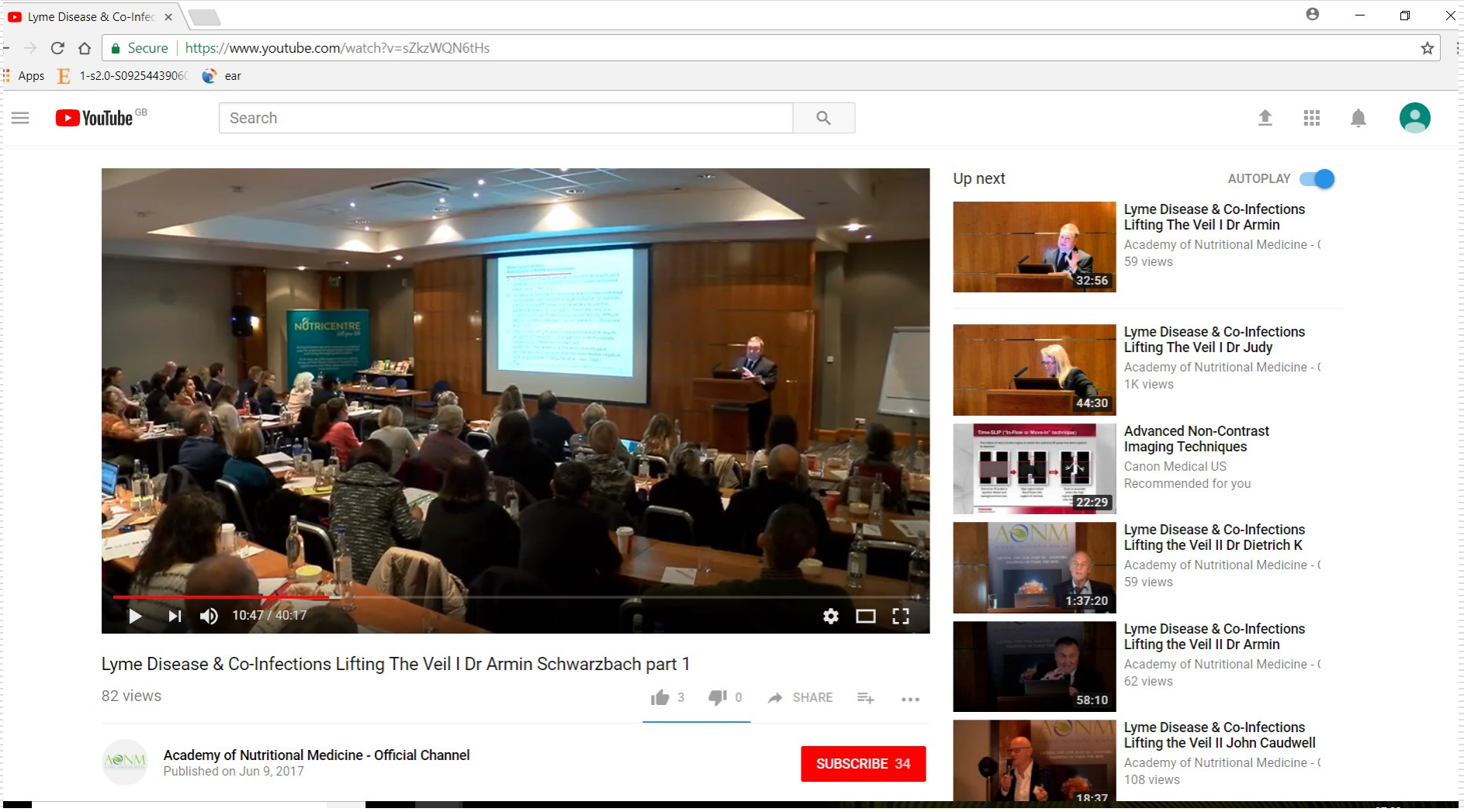

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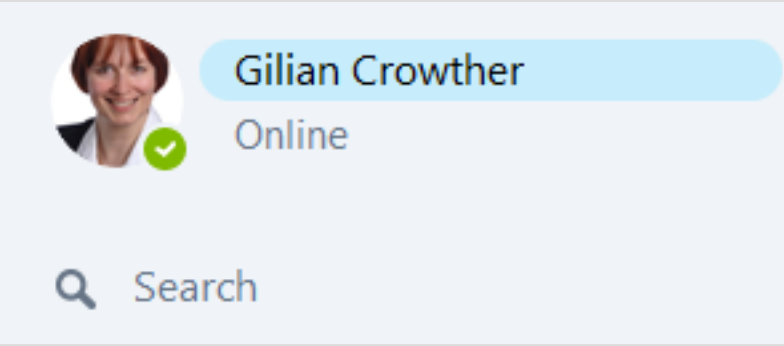

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