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Some Potential Roles of Fibrin Amyloid ('Fibrinaloid') Microclots in Fibromyalgia Syndrome

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Abstract:

Fibromyalgia syndrome (FMS) is a complex multisystem disorder that, like many other chronic diseases with an infectious origin, is commonly accompanied by inflammation, oxidative stress, autoimmunity (not least antiphospholipid antibodies), endothelial dysfunction, pain, fatigue and other symptoms that can vary significantly between individuals, and that can interact in multiple and complex ways. As well as 'primary' fibromyalgia, such disease comorbidities include rheumatoid arthritis, myalgic encephalomyelitis/ chronic fatigue syndrome (ME/CFS) and long COVID. Such disorders are also commonly accompanied by microclots in blood plasma, that we refer to as fibrinaloid microclots since they both contain fibrin and stain with amyloid stains, and also by platelet activation. Following an original proposal by Berg in 1999, and our own findings of the fibrinaloid microclots in such diseases, we here develop the idea in a prospective review (with much evidence) that these fibrinaloid microclots may in fact be a substantial contributor to the aetiology of fibromyalgia. This would open up a number of useful and valuable therapeutic approaches to the treatment of FMS, including the use of anticoagulants, antiplatelet therapy, fibrinolytic enzymes, and/or other natural products.

Keywords: Fibrinaloid Microclots; Clotting; Fibromyalgia Syndrome; Inflammation.

1. Introduction

"There is an epoch in the growth of a science during which facts accumulate faster than theories can accommodate them." Medawar, P. (1982) in Pluto's Republic. Oxford University Press, Oxford, p. 29.

Fibromyalgia, or fibromyalgia syndrome (hereafter FMS), is a devastating multisystem disorder of seemingly uncertain origin. Its worldwide prevalence, depending on the criteria employed (Jones et al., 2015), may be as much as 2-3% (Berwick et al., 2022; Jurado-Priego et al., 2024) or more (Branco et al., 2010; Heidari et al., 2017; Marques et al., 2017), and it represents a substantial economic (D'Onghia et al., 2022) as well as personal (Bennett, 1996) burden. It has been widely reviewed (e.g. (Al

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Sharie et al., 2024; Berwick et al., 2022; Clauw, 2014; Giorgi et al., 2023; Gyorfi et al., 2022; Häuser et al., 2015; Inanici & Yunus, 2004; Jahan et al., 2012; Jay & Barkin, 2015; Jurado-Priego et al., 2024; Kocyigit & Akyol, 2022; Macfarlane et al., 2017; Mascarenhas et al., 2021; Sarzi-Puttini et al., 2021; Siracusa et al., 2021)). As with many other such disorders it affects predominantly women (as much as 9:1 vs. males (Rus et al., 2023; Staud, 2009; Sumpton & Moulin, 2014; Weir et al., 2006)). The main manifestations (symptoms) (Barhorst et al., 2022; Fitzcharles et al., 2021; Sumpton & Moulin, 2014; Wolfe et al., 2010) are a widespread pain, often accompanied by fatigue, although there is considerable variation between individuals (Maurel et al., 2023) and (whether in truth or more because of social/cultural reasons around diagnosis and reporting) between ethnicities (e.g. (Jones et al., 2015)). There are a variety of comorbidities and disease similarities (e.g. (D'Amuri et al., 2024; Fitzcharles et al., 2018)), some of which are collected in Table 1. We note explicitly that several of these (AIDS, Hepatitis C, and as most recently recognised (Bjornevik et al., 2023) multiple sclerosis, caused by Epstein-Barr virus (Buchwald et al., 1987)) are clearly viral in origin. Other infectious agents implicated include *Giardia* and *Borrelia burgdorferi* (the agent of Lyme disease).

Table 1. Some Comorbidities and Co-Occurrences of Fibromyalgia Syndrome.

Disease, property, or syndrome	**Selected references**
Acquired Immuno Deficiency Syndrome (AIDS)	(Demirdal et al., 2019)
Alzheimer's disease / Dementia	(Nagakura et al., 2023; Tzeng et al., 2018)
Atrial fibrillation	(Akkaya et al., 2020; Sarifakioglu et al., 2014)
Behçet's syndrome	(Jobanputra et al., 2017; Plant et al., 2023)
BMI	(Ghizal et al., 2016)
Coeliac disease	(Kılıçoğlu et al., 2024)
Ear and hearing problems	(Skare et al., 2024)
Ehlers–Danlos Syndrome	(Fairweather et al., 2023; Gagnon et al., 2023; Gullapalli & Javed, 2023; Molander et al., 2020; Zhang et al., 2019)
Giardia infection	(Hunskar et al., 2022)
Hepatitis C (chronic)	(Afifi et al., 2022; Mohammad et al., 2012)
Hereditary haemochromatosis	(Mohammad et al., 2013)
Human herpes (virus)	(De Maio & Caterina Turco, 2024; Duffy et al., 2022; Ievina et al., 2024; Krumina et al., 2019; Löhr, 2022)
Hypertension	(Brusco et al., 2021; Ketenci et al., 2024; Wolfe et al., 2020)
Inflammation	(Bains et al., 2023; Paroli et al., 2024; Wåhlén et al., 2020)
Inflammatory bowel disease and irritable bowel syndrome	(Erdrich et al., 2020; Hampannavar et al., 2024; Hunskar et al., 2022; Palm et al., 2001; Slim et al., 2015; Tarar et al., 2023)
Ischaemic heart disease	(Mansour et al., 2024)
Long COVID	(Calabrese & Mease, 2023; Clauw & Calabrese, 2023; Fialho et al., 2023; Goldman, 2022; Hackshaw et al., 2023; Jennifer et al., 2023; Mariette, 2024; Silva-Passadouro et al., 2024; Simone Turner et al., 2023; Ursini et al., 2021; Zulbaran-Rojas et al., 2024)
Lower back pain	(Couëpel et al., 2024)
Lyme disease	(Dinerman & Steere, 1992 ; Kobayashi et al., 2022; Radolf et al., 2021)
Migraine	(Creed, 2022; Lee et al., 2024; Lin et al., 2022; Onder et al., 2019 ; Penn et al., 2019 ; Whealy et al., 2018)
Multiple Sclerosis	(Clemenzi et al., 2014 ; Marrie et al., 2012; Thomas et al., 2023 ; Ulusoy, 2020)
Myalgic encephalomyelitis/ chronic fatigue syndrome (ME/CFS)	(Barhorst et al., 2022; Blitshteyn et al., 2018; Blitshteyn & Chopra, 2018; Borchers & Gershwin, 2015; Collin et al., 2017; Eccles et al., 2021; Falaguera-Vera et al., 2020; Hunskar et al., 2022; Martin et al., 2023 ; McKay et al., 2021; Mohabbat et al., 2020; Nunes et al., 2023; Ramírez-Morales et al., 2022 ; Ryabkova et al., 2023; Tsamou et al., 2024; van Eeden et al., 2023)
Nonalcoholic steatohepatitis (NASH)	(Moon et al., 2021; Rogal et al., 2015)
Obesity	(D'Onghia et al., 2021; Gota et al., 2015; Rivera et al., 2024)
Parkinson's disease	(Abuhasira et al., 2019; Toda & Harada, 2010) (see also (Wood et al., 2007))

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Postural Orthostatic Tachycardia Syndrome (POTS)	(Contreras-Merino et al., 2022; Staud, 2008)
Pregnancy and pregnancy disorders	(Atasever et al., 2017; Genç et al., 2017; Koné et al., 2022; Mucci et al., 2023)
Psoriasis	(D'Onghia et al., 2024; Thune, 2005)
Psoriatic arthritis	(Mease et al., 2024; Navarini et al., 2024)
Rheumatoid arthritis	(Choy & Mease, 2009; Doss et al., 2017; Fitzcharles et al., 2016; Goldenberg, 2023; Lee et al., 2011; Minhas et al., 2023; Wolfe et al., 2020; Zhao et al., 2019)
Sexual dysfunction	(Deggerone et al., 2024)
Sjögren's syndrome	(Applbaum & Lichtbroun, 2019; Gau et al., 2021; Ostuni et al., 2002; Pego-Reigosa et al., 2021)
Small fibre (poly)neuropathy	(Grayston et al., 2019; Martínez-Lavín, 2018; Nissan et al., 2023; Oaklander & Nolano, 2019; Petropoulos et al., 2021)
Systemic lupus erythematosus (lupus)	(Bennett, 1997; Buskila et al., 2003; Mistry et al., 2024; Staud, 2006; Torrente-Segarra et al., 2016)
Tension-type headaches	(Lenaerts & Gill, 2006)

Other accompaniments of fibromyalgia include (the related) inflammation (e.g. (Bäckryd et al., 2017; Conti et al., 2020; Coskun Benlidayi, 2019; Fineschi et al., 2022; Kaplan et al., 2020; Littlejohn & Guymer, 2018; Paroli et al., 2024; Sánchez-Domínguez et al., 2015; Yao et al., 2023)), iron dysregulation (Kucuk et al., 2021; Okan et al., 2019; Ortancil et al., 2010) and oxidative stress (e.g. (Assavarittirong et al., 2022; Beyaztas et al., 2023; Fatima et al., 2013; Meeus et al., 2013; Ozgocmen et al., 2006; Sánchez-Domínguez et al., 2015; Shukla et al., 2021; Zhang et al., 2022)). Note, however, that low levels of serum ferritin, astonishingly referred to as 'iron deficiency anaemia' (Yao et al., 2021), should not be interpreted as such since ferritin is an intracellular molecule and has no business appearing in plasma except via cell death (Kell & Pretorius, 2014), where it can release free iron and thereby cause immense trouble (Kell, 2009; Kell, 2010; Douglas B. Kell & Etheresia Pretorius, 2018). Notably, because of the likely heterogeneity and lack of understanding of the causative mechanisms (Creed, 2020), there are no really effective treatments (Atzeni et al., 2017; Sarzi-Puttini et al., 2020; Serio & Tovoli, 2019), making a better understanding of the aetiology of FMS a high priority (Goebel et al., 2023; Treister-Goltzman & Peleg, 2023).

Importantly, much evidence (Aloisi & Casini, 2025), including from nailfold capillaroscopy (Choi & Kim, 2015; Coşkun Benlidayi et al., 2021; Jeschonneck et al., 2000; Morf et al., 2005) and other methods (Guedj et al., 2008) points to a role for issues with blood supply in the microcirculation (also known as 'blood stasis' (Kell et al., 2025)) leading to hypoxia, as we shall detail below. A preprint has been posted (Douglas B. Kell & Etheresia Pretorius, 2024). We begin with a brief rehearsal of the nature and properties of the fibrinaloid microclots that we argue can underpin FMS.

2. What Are Fibrinaloid Microclots?

Blood clotting occurs via a well-established cascade in which a major part involves the removal of two small 'fibrinopeptides' from the abundant molecular complex fibrinogen (Litvinov et al., 2021; Undas, 2024; Wolberg, 2023). This leads to the self-assembly of fibrinogen into fibrin strands, which in the electron microscope have the appearance of nicely cooked spaghetti, and which form the basis of a clot. More than 10 years ago, one of us (EP) showed that under certain circumstances the clots can look very different, however, forming a rather unpleasant mess resembling parboiled spaghetti strands that have stuck together (Lipinski et al., 2012), that was referred to at the time (Pretorius & Lipinski, 2013; Pretorius et al., 2013) and subsequently (Nielsen & Pretorius, 2014; Page et al., 2018; Pretorius et al., 2015; Soma & Pretorius, 2015; Swanepoel et al., 2016) as 'dense matter deposits'.

While it is not at all new that proteins can form distinct and stable conformational macrostates, it was probably the discovery of prions (Prusiner, 1982, 1991, 1998, 2012, 2013) – proteins that can adopt a highly stable and non-standard conformation with what we now know is a crossed-β-sheet morphology (e.g. (Chen et al., 2017; Nguyen et al., 2021; Ren et al., 2019; Serpell et al., 2007; Serpell et al., 2000; Tycko, 2014; Zhang et al., 2021) and with no change in primary sequence – that made this especially clear. These structures are referred to as amyloid structures, and can be stained with a variety of fluorogenic stains including thioflavin T (Freire et al., 2014; Gade Malmos et al., 2017; Griffin et al., 2010; Kell et al., 2022; Kuwana et al., 2023; LeVine, 1999; Robbins et al., 2012; Sulatskaya et al., 2018; Xue et al., 2017).

What we discovered in 2016 (Pretorius et al., 2016) was that the anomalous clots seen in the electron microscope were indeed also amyloid in character, and could also be stained with thioflavin T. In the original paper (Pretorius et al., 2016) the (highly

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potent) catalyst was bacterial lipopolysaccharide (active at one molecule per 100 million fibrinogen molecules). In later work we have used bacterial lipoteichoic acid (Pretorius, Page, et al., 2018) and SARS-CoV-2 spike protein (Grobbelaar et al., 2021). As with prions and related proteins (Aguzzi & Lakkaraju, 2016; Scheckel & Aguzzi, 2018), there is no thermodynamic problem; it is simply due to kinetic pathways and the fact that one anomalously folded (amyloid) monomer can catalyse the folding of other monomers into the amyloid form (Kell & Pretorius, 2017, 2023) (Figure 1). Importantly, the anomalous forms are far more resistant to proteolysis (indeed, in the case of prions this forms the basis for their detection (Petrotchenko et al., 2012; Silva et al., 2015; Wang et al., 2018)). In the case of fibrin these forms are intrinsically significantly more resistant to the normal pathways of fibrinolysis (Grobbelaar et al., 2021; Kell & Pretorius, 2015), and in addition the clots can entrap molecules such as α2-antiplasmin (Kruger et al., 2022; Etheresia Pretorius et al., 2021) that make them even more so. Indeed the substantial differences in the non-fibrin molecules they entrap, relative to those associated with normal clots, reflect their amyloidogenicity and mean that in the case of the fibrinaloid microclots such proteins are likely incorporated into the bodies of the growing amyloid-like fibrils (Douglas B. Kell & Etheresia Pretorius, 2024).

Fairly obviously (e.g. (Kell et al., 2022)), a consequence of the blockage of capillaries and microcapillaries with microclots is that blood cannot flow properly, and O_2 transport to tissues is thereby inhibited, leading to low oxygen saturation, precisely as observed in FMS (Rubio-Zarapuz et al., 2025).

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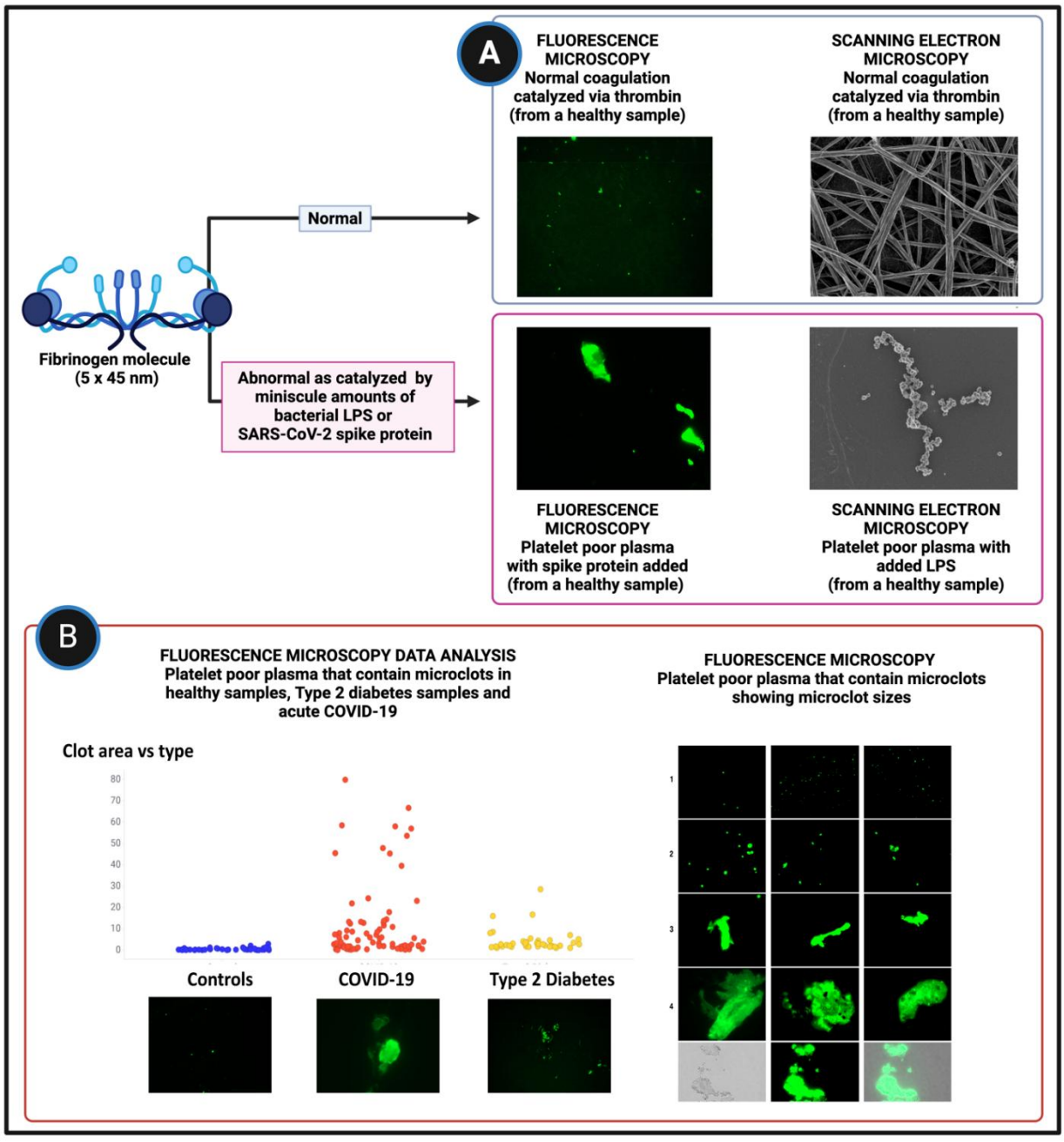

Figure 1. In the presence of certain catalysts or triggers, blood clotting can occur into a highly anomalous form, including into microclots that are much more resistant to fibrinolysis than normal clots and that we refer to as fibrinaloid microclots. These can cause all kinds of pathological effects, as rehearsed in the text. A) Fluorescence and scanning electron microscopy micrographs of healthy platelet poor plasma with and without thrombin and with and without spike protein or LPS (raw data from (Grobbelaar et al., 2021)). The green signal is from Thioflavin T and shows fibrinaloid microclots. B) Microclot counts (found in platelet poor plasma in controls, type 2 diabetes and acute COVID-19 (without addition of thrombin), as well as size ranges of microclots (raw data from (Pretorius et al., 2020)). [Created with BioRender.com.]

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Because these clots are basically blood clots since they are dominated by fibrin, and because the term 'amyloid' can have rather negative connotations, we have referred to them as 'fibrinaloid microclots' (Justine M. Grixti et al., 2024; Justine M. Grixti et al., 2024; Douglas B. Kell et al., 2024; D. B. Kell, M. A. Khan, et al., 2024; Kell et al., 2022; D. B. Kell, G. Y. H. Lip, et al., 2024; Kell & Pretorius, 2023; Douglas B. Kell & Etheresia Pretorius, 2024; Nunes et al., 2022; Pretorius & Kell, 2023; Pretorius & Kell, 2024; Pretorius, Nunes, et al., 2024; Pretorius, Thierry, et al., 2024; Simone Turner et al., 2023; S. Turner et al., 2023). As previously trailed (e.g. (Kell, 2009; Douglas B. Kell & Etheresia Pretorius, 2018; Kell & Pretorius, 2022)), many of these chronic diseases are known to share a variety of properties, including an infectious origin (not least in sepsis (Douglas B. Kell & Etheresia Pretorius, 2018; Schofield et al., 2024)), inflammation, iron dysregulation, oxidative stress, and the presence in the individuals' plasma of these microclots that can be stained by amyloid stains such as thioflavin T (Kell & Pretorius, 2017; Pretorius et al., 2016). Since these diseases include ME/CFS (Nunes et al., 2023; Jean M. Nunes et al., 2024; Nunes et al., 2022; Tsamou et al., 2024) and Long COVID (as well as others such as Parkinson's (B. Adams et al., 2019; de Waal et al., 2018; van Vuuren et al., 2021), Alzheimer's dementia (Grobler et al., 2023; Pretorius, Bester, et al., 2018), migraine (de Villiers et al., 2019), and type 2 diabetes (Pretorius et al., 2020) that we have studied), it occurred to us that these fibrinaloid microclots might also be an accompaniment, and possibly a contributory cause, to fibromyalgia. In the case of Long COVID the microclots can account for a variety of the otherwise multitudinous symptoms (Kell et al., 2022), including post-exertional malaise (post-exertional system exacerbation) (Kell & Pretorius, 2022), the genesis of certain kinds of autoantibodies (Kell & Pretorius, 2023), postural orthostatic tachycardia syndrome (POTS) (Douglas B. Kell et al., 2024), and even atrial fibrillation (Douglas B. Kell et al., 2024).

In summary then:

- Fibrinaloid microclots form using the normal clotting machinery involving fibrinogen and thrombin.
- As with amyloids and prions they have no necessary change in primary sequence; they simply represent thermodynamically more stable macrostates of the relevant proteins that are normally kinetically inaccessible.
- Their production can be catalysed by minuscule amounts of certain effectors, such as bacterial LPS ('endotoxin') or spike protein.
- Their production can also be autocatalytic once a molecule of fibrinaloid has been formed.
- They are significantly more resistant to fibrinolysis than normal clots.
- They can aggregate to form larger clots that can block capillaries and blood vessels of the relevant size.
- They could in principle be deposited anywhere where fibrinogen is found.

The aim of the present article is thus to develop the idea that fibrinaloid microclots are also part of the aetiology of FMS. An overview of the chief elements of our present thinking is given in Figure 2.

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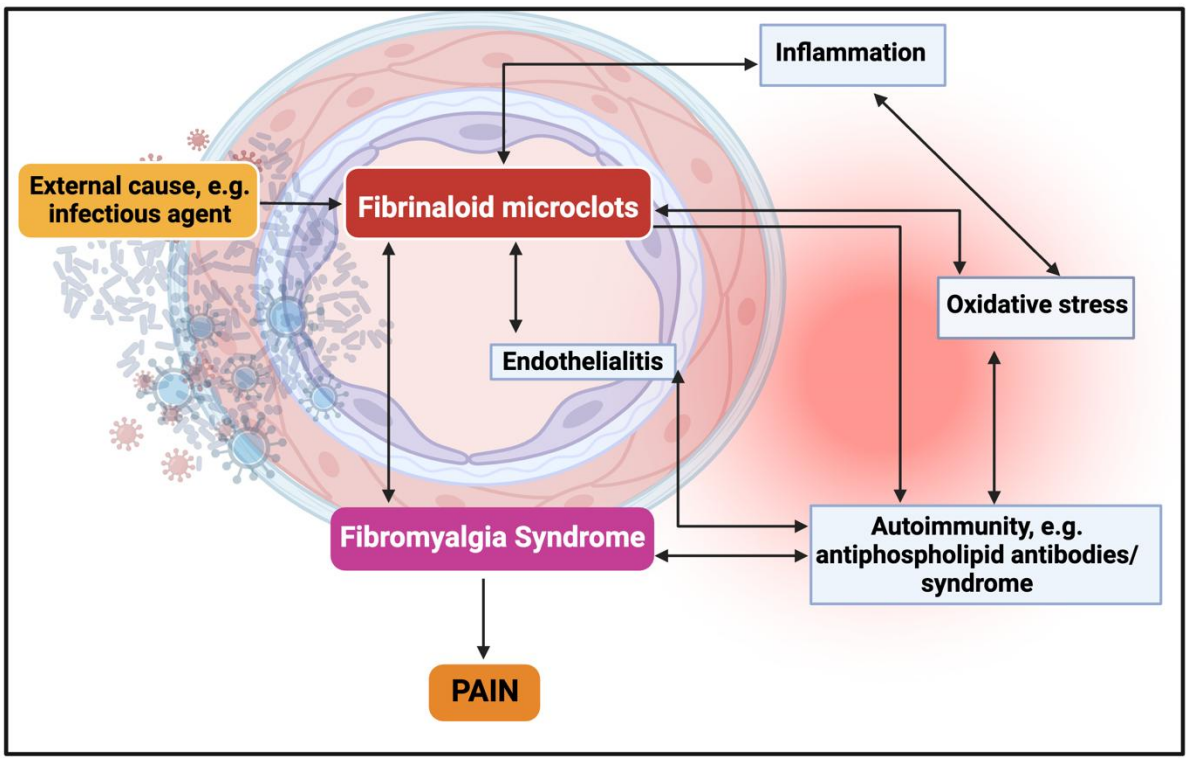

Figure 2. An overview of the chief elements that we consider to be as part of the fibrinaloid micro-clot-mediated genesis of fibromyalgia syndrome. Other interactions also exist but are omitted for clarity. [Created with BioRender.com.]

3. Present Theories of Fibromyalgia Syndrome

The overriding symptom of FMS is a widespread pain (Hu et al., 2024; Pacheco-Barrios et al., 2024; Vatvani et al., 2024), often referred to as nociplastic pain (Bułdyś et al., 2023; Fernández-de-las-Peñas et al., 2022; Galli, 2023; Goebel, 2024; Murphy et al., 2023; Nijs et al., 2021; Nijs et al., 2023) when seen as central, whether in 'primary' fibromyalgia or as a 'secondary' accompaniment to the kinds of diseases mentioned above as comorbidities. By and large, present thinking focuses on a role for both CNS and peripheral (Caxaria et al., 2023; Clauw, 2015; de Tommaso et al., 2022; Doppler et al., 2015; Maixner et al., 2016; Marshall et al., 2024; Martínez-Lavín, 2018; Navarro, 2009; Yunus, 2012, 2015) pain processing defects (Fitzcharles et al., 2021; Minhas et al., 2023) as a mediator of FMS. The relative importance of each are uncertain (Bailly et al., 2020; Martínez-Lavín, 2022) (and, one would assume (Williams, 1956), variable between individuals). Other measurands that accompany FMS (Berwick et al., 2022) include inflammation (measured as cytokine differences (Wallace et al., 2015) or the FM/a test (Straub & Mounsey, 2021)), and in particular autoimmunity (Clauw et al., 2023; Mountford et al., 2024). An especially striking observation is the sensitisation of nociceptive neurons by IgG from FMS patients, leading to sensory hypersensitivity (Goebel et al., 2022; Goebel et al., 2023; Goebel et al., 2021), though the antigens of these IgG are presently unknown. This said, as with any systems biology analysis, it is the causes and chief mediators that we really seek here (Douglas B. Kell & Etheresia Pretorius, 2018), and the finger of suspicion begins to fall on antiphospholipid antibodies.

As to current treatments, presently only three drugs are FDA-approved for the treatment of fibromyalgia. These are duloxetine, pregabalin, and milnacipran (Tzadok & Ablin, 2020). Duloxetine is a serotonin and noradrenaline reuptake inhibitor (SNRI) with antidepressant activity (Lunn et al., 2014) and shows some limited efficacy (Arnold et al., 2004; Migliorini et al., 2023). Pregabalin is a ligand of the α2-δ calcium channel, and has also shown some efficacy in subsets of the patient population (Arnold et al., 2018). The same is true of milnacipran (Arnold et al., 2013) (also an SNRI, but not approved for major depressive disorders in the US). Overall, present treatments are less than satisfactory in a substantial fraction of the population.

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4. Origins and Occurrence of Antiphospholipid Antibodies

Antiphospholipid antibody syndrome (APS) (Ambati et al., 2023; Barbhaiya et al., 2023; Chaturvedi & McCrae, 2017a, 2017b; Depietri et al., 2023; Garcia & Erkan, 2018; Keeling et al., 2012; Knight et al., 2023; Lim et al., 2006; Meroni et al., 2014; Tektonidou et al., 2019; Tincani et al., 2023; Tripodi et al., 2011) is among the most severe forms of autoimmune diseases (including in pregnancy disorders, in particular pre-eclampsia (Abrahams et al., 2017; Ament, 1994; Chighizola et al., 2015; Chighizola et al., 2014; De Carolis et al., 2010; do Prado et al., 2010; Hadi & Treadwell, 1990a, 1990b; Kell & Kenny, 2016; Kenny & Kell, 2018; Lockshin, 2013a, 2013b; Martínez-Zamora et al., 2010; Tong et al., 2021; Tong et al., 2015)), and can be externally triggered (Martirosyan et al., 2019). The diseases in which it occurs are all essentially autoimmune or auto-inflammatory (Sciascia et al., 2012) diseases such as rheumatoid arthritis (Giacomelli et al., 2017; Gladd & Olech, 2009) and especially Systemic Lupus Erythematosus (lupus) (Anderson & Belmont, 2022; Chock et al., 2019; Domingues et al., 2022; El Hasbani et al., 2021; Li et al., 2024; Nagy et al., 2024; Zuily et al., 2017), as well as viral diseases such as (acute) COVID-19 (Blickstein et al., 2023; Butt et al., 2022; Capozzi et al., 2023; Devreese et al., 2020; Foret et al., 2021; Haghighipour et al., 2023; Nosrati et al., 2022; Serrano et al., 2022; Taha & Samavati, 2021; Uslu, 2023; Xiao et al., 2020; Zhang et al., 2020; Zuo et al., 2020a).

Table 2. The three main known targets of 'antiphospholipid antibodies' (of which some may interact with more than one target). Individuals with each kind of antibody ('triple positive') are at particular risk of clinical events (Tripodi et al., 2023).

Target	Comments	Selected references
β_2-glycoprotein I	Main target for 'antiphospholipid antibodies'; can interact with toll-like receptors and activate endothelia and monocytes. Inflammagenic. Possesses multiple conformational macrostates	(Bai, 2017; Benagiano et al., 2017; Meijide et al., 2013; Ninivaggi et al., 2012)
Cardiolipin	'anticardiolipin antibodies'	(Ament, 1994; Galli et al., 2003; Islam et al., 2017; Meijide et al., 2013; Pastori et al., 2019)
Lupus anticoagulant	Common in Lupus as its name suggests, but also in other disorders including pregnancy loss	(Galli et al., 2003; Meijide et al., 2013; Owaidah et al., 2003)
	Direct oral anticoagulants can actually interfere with Lupus Anticoagulant measurement	(De Kesel & Devreese, 2020; Tripodi et al., 2023)

5. Role of Complement and Platelet Activation in Antiphospholipid Syndrome

Complement activation is another well-established feature of antiphospholipid syndrome (Avalos & Tsokos, 2009; Bećarević, 2017; Chaturvedi et al., 2019; Cole et al., 2023; Girardi et al., 2003; Kello et al., 2019; Lim, 2011; O'Neil, 2007; Oku et al., 2016; Salmon et al., 2003) that contributes to the procoagulant state, especially via endothelial damage (see below). The same is true for platelet activation (Breen et al., 2015; Capozzi et al., 2023; Chayoua et al., 2021; de Mesquita et al., 2013; Joseph et al., 1998; Shi et al., 2021; Tang et al., 2023; Tomer, 2002; Warkentin et al., 2003).

6. Antiphospholipid Antibodies in Acute COVID

Antiphospholipid antibodies are clearly associated with a prothrombotic state (Amory et al., 2015; Cohen et al., 2021; García-Grimshaw et al., 2022; Garcia et al., 2013; Romay-Penabad et al., 2009; Turiel et al., 2005; Zuily et al., 2020), including in acute COVID-19 (Arcani et al., 2023; Benjamin et al., 2021; Bertin et al., 2022; Butt et al., 2022; Devreese et al., 2020; Foret et al., 2021; Garcia-Arellano et al., 2023; Hossri et al., 2020; Kahlon et al., 2022; Mendel et al., 2023; Nosrati et al., 2022; Oba et al., 2023; Serrano et al., 2022; Shah et al., 2022; Taha & Samavati, 2021; Talotta & Robertson, 2021; Trahtemberg et al., 2021; Zeng et al., 2022; Zhang et al., 2020; Zuo et al., 2020b), and this may be mediated by a variety of mechanisms (Foret et al., 2022) including a lowered rate of fibrinolysis (Forastiero & Martinuzzo, 2008). Anticoagulation seems effective in lowering the incidence of thrombotic events in individuals with APS (Ott et al., 2023; Uslu, 2023), while low APS antibody levels are found in those who recover from acute COVID (Blickstein et al., 2023; Favaloro et al., 2022).

7. Antiphospholipid Antibodies in Long COVID

Given the frequency of APS antibodies in acute COVID, it is unsurprising that they are also detected in the plasma or serum of individuals with Long COVID (Bertin et al., 2021; Pisareva et al., 2022; Simone Turner et al., 2023). As discussed, (Simone Turner et al., 2023), antiphospholipid antibodies are known to promote thrombosis by stimulating neutrophils to release

neutrophil extracellular traps (NETs) (Zaiema et al., 2024) and by activating endothelial cells and platelets (Chen et al., 2021; Knight et al., 2021).

8. Relationship between Antiphospholipid Antibodies and Endothelialitis

It has been known for many years (Ruíz-Argüelles et al., 1991) that antiphospholipid antibodies can cause endothelial damage (Blum & Simsolo, 2004; Corban et al., 2017; D'Ippolito et al., 2007; Gharavi et al., 2003; Pierangeli & Harris, 2003; Velásquez et al., 2018). This provides an important aetiological link, since endothelial function is now well established as a major contributor to both Long COVID (e.g. (Durstenfeld et al., 2024; Golzardi et al., 2024; Simone Turner et al., 2023; Turner et al., 2024; Xu et al., 2023)) and ME/CFS (Nunes et al., 2022; J. Massimo Nunes et al., 2024).

9. Relationship Between COVID-19 And Endothelialitis

The role of endothelialitis in both acute (Ackermann et al., 2020; Mentzer et al., 2022; Vrints et al., 2021; Xu et al., 2023) and Long COVID (Ahamed & Laurence, 2022; Charfeddine et al., 2021; Crook et al., 2021; Kruger et al., 2022; Laubscher et al., 2021; Proal & VanElzakker, 2021; Simone Turner et al., 2023; S. Turner et al., 2023) is well established by now, and we simply cite a variety of papers and reviews that rehearse it.

10. Berg's Largely Forgotten Theory of the Role of Thrombotic Dysfunction and Antiphospholipid Antibody in Fibromyalgia

In 1999, David Berg and his colleagues presented the idea (Berg et al., 1999) that FMS was actually linked to, and potentially caused by, a pro-thrombotic state, a view only occasionally echoed by others (e.g. (Han et al., 2020; Molina et al., 2019; Ramírez-Tejero et al., 2018)). Their analyses (Berg et al., 1999) showed low level coagulation activation from immunoglobulins (IgGs) as demonstrated by Anti-β_2Glycoprotein PI (Anti-β_2GPI) antibodies, which they considered implied the classification of these diseases as a type of antiphospholipid antibody syndrome (APS). We can only echo our view that the prescience of this analysis has not remotely been matched by the attention it has received – something that we hope may now change.

Although we are not aware of studies of APS in individuals initially diagnosed with FMS, one study (Costa et al., 2011) did look explicitly at the converse, finding that individuals with primary antiphospholipid (Hughes) syndrome were five times as likely to show fibromyalgia (Costa et al., 2011). This is a large effect.

11. Effects of Amyloid Proteins on Membranes

Many proteins not necessarily considered amyloid or amyloidogenic (i.e. not seen as involved in classical amyloidoses (Alraawi et al., 2022; Blancas-Mejía & Ramirez-Alvarado, 2013; Chiti & Dobson, 2017; Farrugia et al., 2020; Jamroziak & Puła, 2020; Nevone et al., 2020; Picken et al., 2012; Wechalekar et al., 2016)) may in fact be so (e.g. (Balistreri et al., 2020; Burdukiewicz et al., 2017; Kell & Pretorius, 2017; Louros et al., 2020; Szulc et al., 2021; Varadi et al., 2018)). A clear example is provided by the SARS-CoV-2 spike protein, that is both amyloidogenic itself (Nyström & Hammarström, 2022) and capable of seeding amyloidogenesis among other proteins such as fibrin(ogen) (Grobbelaar et al., 2021; Kell & Pretorius, 2023; Larsson et al., 2023; Pretorius & Kell, 2023). Note too that protein misfolding can be catalysed by lipids (Gursky, 2015).

The main purpose of this paragraph is thus simply to point out the widespread and known cytotoxicity of all kinds of amyloid proteins, mainly as an initial step via the exposure of hydrophobic and amphipathic elements (Alraawi et al., 2022) effecting membrane damage (e.g. (Brender et al., 2012; Butterfield & Lashuel, 2010; Camilleri et al., 2013; Drolle et al., 2014; Evangelisti et al., 2016; Gonzalez-Garcia et al., 2021; Harris, 2012; Lee et al., 2017; Limbocker et al., 2023; Ma et al., 2023; Marinko et al., 2019; Raleigh et al., 2017; Rawat et al., 2018; Relini et al., 2013, 2014; Salahuddin et al., 2021; Sciacca et al., 2021; Sciacca et al., 2013; Tempra et al., 2022; Valincius et al., 2008; Viles, 2023; Wiatrak et al., 2021; Yang et al., 2022)). Such cytotoxicity in cells such as endothelia (Cho et al., 2011; Ghoneim et al., 2021) explains many of the sequelae e.g. in Long COVID, and in particular the pain that accompanies the fibromyalgia.

An important recent development is the discovery of amyloid deposits in the skeletal muscle of Long COVID patients (Appelman et al., 2024); although their proteomic constitution is not yet established, it seems plausible that these also represent fibrinaloid microclots, and that their location also reflects the breakdown of endothelial barriers that accompany (and may be a major mediator of) both acute (Ackermann et al., 2020; Volod et al., 2022) and Long COVID (Astin et al., 2023; Buonsenso et al., 2022; Davis et al., 2023; Grobbelaar et al., 2021; Nicolai et al., 2023; Tziolos et al., 2023; Yan et al., 2023). Another important recent development (Schofield et al., 2024) is the discovery of fibrinaloid microclots in sepsis and, in particular, in disseminated intravascular coagulation (with an Odds Ratio of 51, which must be close to a record).

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12. Significance of APS and Fibrin Amyloid Microclots for Fibromyalgia

We note the endothelial cell damage caused by autoantibodies in APS (George et al., 1998) and lupus (Dhillon et al., 2016), that is also a hallmark of COVID-19, as rehearsed in more detail above. Given the often massive elevation in fibrinaloid microclots that we consider a significant aetiological part of these many chronic, inflammatory diseases, as well as their ability to cause oxidative stress (Kell et al., 2022), post-exertional malaise (Kell & Pretorius, 2022), and autoantibodies (Kell & Pretorius, 2023) (all part of fibromyalgia syndrome), we would point out that although the evidence for amyloid deposition in fibromyalgia is as yet relatively modest it is quite significant as it was largely unsought (Table 3). Consequently, we see a potentially major role for their deposition in the tissues that experience the greatest symptoms of fibromyalgia in any individual, clearly implying that either anticoagulation or increasing fibrinolysis may be of benefit.

Table 3. Some Examples of Amyloid Deposition Related to Fibromyalgia.

System	Summary	Reference
ME/CFS, Gulf War Syndrome and fibromyalgia CSF proteome	Significant increase in amyloid β (A4) precursor like protein 1 (APLP1)	(Baraniuk et al., 2005) (see also (Hannan et al., 2000) for coagulation)
Detailed review of Egyptian subjects	Huge increase in serum amyloid A	(Hamouda, 2018)
Hereditary Transthyretin Amyloidosis (hATTR) Polyneuropathy	Said to be misdiagnosed as fibromyalgia	(D. Adams et al., 2019; Luigetti et al., 2020)
Role of HSV in fibromyalgia and Alzheimer's dementia	Review, plus evidence of the benefit of famciclovir + celecoxib in fibromyalgia treatment	(Itzhaki & Lathe, 2018) citing (Pridgen et al., 2017)
Brain imaging *in vivo*	Significant increase ($P<0.003$) in Ab_{1-42} peptide leading to neural damage	(Lo et al., 2022)
Corneal Confocal Microscopy used to Image Small Nerve Fibre Degeneration	Relationships between amyloid neuropathy and fibre neuropathy	(Petropoulos et al., 2021)
Measurement of amyloids in serum	Significant increases in serum total-tau protein and $A\beta_{1-42}$ peptide; these were related to sleep disturbances	(Thi Nguy et al., 2022)

13. Consequences for the Treatment of Fibromyalgia

Knowing the causal pathway(s) of a disease makes it much more plausible to develop a principled treatment for it. Thus, heparin or other anticoagulant use in APS is widespread (Bakow et al., 2023), and can be monitored successfully (Cohen et al., 2021; Ryan et al., 2023), for instance with Thromboelastography (TEG) (Nguyen et al., 2019); by contrast, despite Berg's proposal, anticoagulant use in FMS seems close to non-existent and we have found no published review.

There are however, some therapeutic successes against amyloidosis (Nevone et al., 2020), including the famciclovir + celecoxib mentioned above (Pridgen et al., 2017). Clearly, lowering to an appropriate level the amyloids capable of seeding or cross-seeding amyloidogenesis is one strategy, and has been shown to work with amyloid A (Lachmann et al., 2007). It is not so clear how effective this may be with fibrinogen, which is both highly abundant and of course necessary for normal clotting.

In general terms, if fibrin amyloid microclots are an issue their continuing formation may be prevented via the so-called triple treatment (Laubscher et al., 2023; E. Pretorius et al., 2021) (provided bleeding is monitored and this therapy is under the clear

direction of a qualified physician). We have also pointed up the value of enteric-coated fibrinolytic enzymes such as nattokinase, serrapeptase and lumbrokinase (references in (Kell et al., 2022; Kell & Pretorius, 2022, 2023)). They are known to pass through the intestine so are orally available (Chen et al., 2018; Dabbagh et al., 2014; Ero et al., 2013; Fujita et al., 1995; Kapoor et al., 2015; Sumi et al., 1990; Sumi et al., 2004; Zhou et al., 2021), and generally recognised as safe (Bresson et al., 2016; Gallelli et al., 2021; Wu et al., 2019). Many other nutraceuticals (de la Cruz Cazorla et al., 2024; Mishra et al., 2022) were previously covered and include substances such as cannabis-derived molecules (Lopera et al., 2024), iron chelators (Kell, 2009; Kell, 2010; Visentin et al., 2017), kynurenic acid (Alves et al., 2024; Athnaiel et al., 2022), antioxidants (Bjørklund et al., 2018; Giorgi et al., 2023) such as polyphenols (Camilleri et al., 2013; Del Bo' et al., 2019; Neveu et al., 2010; Perron & Brumaghim, 2009; Scalbert et al., 2005), ergothioneine (Borodina et al., 2020; Cheah & Halliwell, 2021; Halliwell et al., 2023) and melatonin (Andrade et al., 2023; Dai et al., 2021; Nopparat et al., 2023), a Mediterranean diet (Proietti et al., 2024) and appropriate antioxidants (Fernández-Araque et al., 2022; Giorgi et al., 2023; Kadayifci et al., 2022) have indeed been found beneficial in fibromyalgia. Traditional Eastern Medicines, especially those involving the use of herbal cocktails, have been found of value in diseases of blood stasis (Kell et al., 2025), and this is also true of fibromyalgia (Cao et al., 2010; Chen et al., 2010; Li et al., 2023). While 'everyone is different' (Williams, 1956), especially when it comes to complex diseases, we would consider there is more than enough biochemical evidence, as summarized, to look into some of these treatments. We also note one study (Gezici et al., 2014) in which the eradication of *Helicobacter pylori* had a very substantial effect in decreasing the number of tender points.

14. Concluding Remarks

Due to the state of scientific knowledge, this has necessarily and purposely been a prospective review. It does, however, lead to some clear predictions, beyond those for which evidence already exists, and in particular that (i) amyloid deposition should be readily observed in the muscles (and likely the plasma/serum) of individuals with FMS, and (ii) that suitable anticoagulant treatments, not least heparin variants such as sulodexide (e.g. (Charfeddine et al., 2022; Huang et al., 2023; Lauver et al., 2005)) or enoxaparin (Barco et al., 2023; Saithong et al., 2022; Voci et al., 2023; Wright et al., 2025), and fibrinolytic enzymes such as nattokinase (Justine M Grixti et al., 2024), serrapeptase (Kumar & Sabu, 2019) and lumbricase/lumbrokinase (Wang et al., 2013) as mentioned above, should have therapeutic benefits. This seems a strategy well worth pursuing.

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Conflicts of Interest: E.P. is a named inventor on a patent application covering the use of fluorescence methods for microclot detection in Long COVID.

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