Persistent Enterovirus Infection

In ME research, the idea of persistent enterovirus infection, especially Coxsackie B, is one hypothesis used to explain why symptoms can continue long after an initial viral illness. It's not proven as the sole cause of ME, but it is biologically plausible and has some supporting findings.

1. Initial infection (acute phase)

A person catches an enterovirus such as Coxsackie B. During this phase, the virus can infect:

*muscle tissue (including cardiac muscle)

*gut lining

*nervous system cells

*immune cells

Symptoms can include:

*flu-like illness

*sore throat, fever

*chest pain or palpitations

*extreme fatigue etc

*neurological symptoms (headache, dizziness)

In most people, the immune system clears the infection.

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2. In some cases: incomplete viral clearance

The hypothesis is that in a subset of people:

*the immune system does not fully eliminate the virus

*instead, the virus may enter a low-replication or "defective" state

This means:

*it is not causing a full acute infection

*it is not always detectable in blood

but it may still be present in tissues

Evidence for this comes from studies showing:

*enteroviral RNA fragments in muscle biopsies

*viral protein detected in gastric tissue (gut biopsies) in ME/CFS patients (notably work by Dr John Chia and others)

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3. Persistent infection in tissues (not bloodstream)

Unlike acute infections, enteroviruses in this model are thought to “hide” in:

*skeletal muscle

*heart muscle (myocardium)

*autonomic nervous system pathways

*gut tissue

*possibly brainstem or glial cells (theoretical)

Instead of destroying cells rapidly, they may:

*replicate at very low levels

*produce incomplete viral particles

This can keep the immune system in a constant low-grade activation state.

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4. Immune dysfunction and chronic inflammation

A key idea in ME research is that persistence leads to:

*chronic cytokine signaling (immune activation)

*oxidative stress

*impaired antiviral response (especially interferon pathways)

*immune exhaustion or dysregulation

Importantly, this does not always look like a standard infection in blood tests.

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5. How this connects to ME symptoms

A. Post-exertional neuroimmune exhaustion (PENE)

If muscle or mitochondrial energy systems are already compromised:

*physical or cognitive exertion increases energy demand

*damaged or stressed cells produce more inflammatory signals

*the immune system responds disproportionately

Result:

*delayed “crash” after exertion

*worsening fatigue, pain, cognitive dysfunction

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B. Autonomic dysfunction (POTS-like symptoms)

If autonomic nerves or brainstem circuits are affected:

*heart rate and blood pressure regulation become unstable

*blood flow regulation becomes inefficient

*standing or activity triggers symptoms

Possible link:

*viral or immune-mediated irritation of autonomic control centers

C. Mitochondrial and energy metabolism effects

Enteroviruses (especially Coxsackie B have been studied for their ability to:

*disrupt cellular energy production

*alter mitochondrial function indirectly through inflammation

*increase oxidative stress

This may contribute to:

*low energy tolerance

*muscle weakness or heaviness

*cognitive fatigue (“brain fog”)

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D. Neuroinflammation

If nervous system tissue is involved:

*brain signaling becomes less efficient

*sensory processing can become hypersensitive

*cognitive fatigue and “wired but exhausted” states can occur

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6. Why triggers matter (why symptoms can flare later)

In this model, ME symptoms can worsen when something increases system stress, such as:

*another viral infection

*physical overexertion

*emotional or physiological stress

*surgery or trauma

*sleep disruption

These don't necessarily "reactivate" a classic virus flare like herpes - rather they may:

*increase immune activation

*worsen energy deficits

*amplify existing inflammatory signaling

7. Where science stands today

*Persistent enterovirus in ME is supported by some tissue studies and clinical observations

(However:

*not all ME patients show evidence of enterovirus

*findings are not consistent across all studies

it is one of several competing/overlapping hypotheses (alongside immune, autonomic, metabolic, and post-infectious models)

References

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