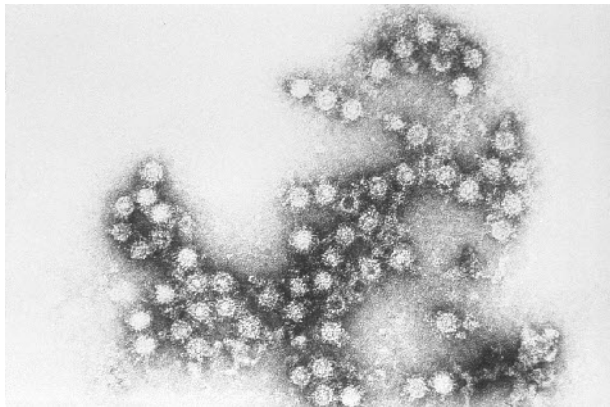


## Coxsackie – Doing damage to our very core: the energy delivery mechanisms of our heart?

By *Gilian Crowther, NT/ND, mBANT, mANP, mNNA, CNHC reg., AONM Director of Research*

Coxsackie (named after where it was first identified, in Coxsackie, New York) is an enterovirus that belongs to the family of Picornaviruses. These are small positive-strand RNA viruses without a lipid membrane. It falls under the same genera as polio, echovirus and other enteroviruses. Its transmission is oro-faecal, and its site of primary infection is the gut.



(Source: [https://en.wikipedia.org/wiki/Coxsackie\\_B\\_virus#/media/File:Coxsackie\\_B4\\_virus.JPG](https://en.wikipedia.org/wiki/Coxsackie_B_virus#/media/File:Coxsackie_B4_virus.JPG))

Coxsackie A virus is usually associated with fever, surface rashes, and can cause herpangia, as well as hand, foot and mouth disease(1). Type B typically causes internal symptoms: it can trigger illness ranging from gastrointestinal distress to full-fledged pericarditis and myocarditis (Coxsackievirus-induced cardiomyopathy)(2). Subtypes of both A and B can cause very severe symptoms, as can be seen from the table in the next column, such as meningitis and even paralysis.

Coxsackie B virus is very common in the UK, in the experience of Dr. Armin Schwarzbach of Arminlabs, who says “The level of infection in the UK that we are picking up in our testing exceeds that of any other country, by percentage of positive titres in those tested – especially IgA, suggesting current infection of the mucosal membranes. It is unclear why that is – we urgently need studies.” \*

Coxsackie virus B can trigger an autoimmune attack on translocator protein, also called the adenine

nucleotide translocator (ANT), in the heart(3). This appears to be because its VP (viral protein) capsid

	Poliovirus	Coxsackie A virus	Coxsackie B virus	Echovirus	Enterovirus (other)
Asymptomatic infection	yes	yes	yes	yes	yes
Meningitis	yes	yes	yes	yes	yes
Paralysis	yes	yes	yes	yes	?
Febrile exanthems	no	yes	yes	yes	yes
Acute respiratory disease	no	yes	yes	yes	yes
Myocarditis	no	yes	yes	yes	no
Orchitis	no	no	yes	yes	no

\* Enterovirus-D68 (EV-D68) can replicate in blood and may damage the central nervous system. It has been detected in cerebrospinal fluid of patients with acute flaccid paralysis.

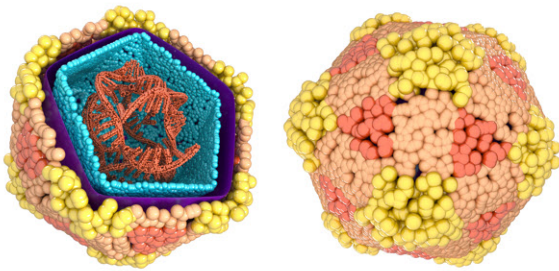
There have been reports of children hospitalized with muscle weakness or paralysis, usually in their arms and legs. They were tested for poliovirus, West Nile virus, and enteroviruses. About half of the children had EV-D68 in their nose secretions; usually, EV-D68 affects the respiratory system and it is not yet known if this respiratory infection is linked to their muscle weakness.

Source: <http://www.microbiologybook.org/virol/picorna.htm>

protein is cross-reactive to mitochondrial translocator protein(4). The ANT is the only transport system for ADP and ATP – it is the most important link of the body’s energy producing and consuming processes. Autoantibodies against the adenine nucleotide transporter were found in the sera of patients suffering from myocarditis and dilated cardiomyopathy that were capable of inhibiting nucleotide exchange activity(5). The mRNA pattern was found to be equivalent to the ANT isoform protein distribution in mitochondria(6). The importance of the virus in this process was shown by the fact that virus-positive patients had an approx. four times higher risk of isoform pattern alteration than those without enterovirus infection(7). This is a huge discovery, as mitochondria – the energy powerhouses in our cells – are rich in these translocator proteins (ANT). Cross-reactivity between the ANT protein and the calcium channel was also observed(8). ANT antibodies bind specifically at the calcium channel of the cell surface and change calcium influx into the cell(9). Raised intracellular calcium concentration causes intramitochondrial calcium overload and lowers mitochondrial transmembrane potential(10). This appears to be a frequent finding, too, in Acumen tests of patients with ME-type symptoms, and recent

research from Australia is also finding calcium channelopathies in the NK cells of ME/CFS patients(11). Could it be that they are suffering from blocked/lowered mitochondrial membrane potential – at least in some cases – due to Cocksackie B?

The group of doctors and scientists around Professor Malcolm Hooper (The John Richardson Research Group, including Dr. Irving Spurr, sadly just recently deceased) have long contended that Cocksackie B underlies many cases of so-called ME/CFS. The ME/CFS researcher Professor Peter Behan suggested that the ANT autoantibody might be the cause of many symptoms of the condition as far back as the 80's. One guinea pig study found that the measured energy output from hearts impacted by the ANT autoantibody was five times less than the energy output from healthy controls, and the hearts of the former animals produced more than twice the amount of lactate(12). The 2009 energy metabolism studies of Dr. Sarah Myhill, Professor Norman Booth (†) and Dr. John McLaren-Howard also found that ME/CFS patients have major disruption of their ANT protein – in both transport of ATP from the mitochondria into the cytosol and ADP from the cytosol into the mitochondria(13). Blocking of the translocator proteins is often found in Acumen tests by heavy metals, pesticides and other contaminants: that they have been disabled by the Cocksackie B virus certainly also appears another possibility in light of these studies.



Cocksackie virus

A 2010 mouse study found that in Cocksackie B myocarditis, a reduction in IL-17 reduced ANT autoantibodies(14). Th17 cells produce IL-17, and have been associated with the promotion of viral replication. N-acetyl-glucosamine has been shown to reduce Th17 and IL-17, as can several other agents. Interestingly, the article that found this is entitled “N-acetylglucosamine inhibits T-helper 1 (Th1)/T-helper 17 (Th17) cell responses and treats experimental autoimmune encephalomyelitis”.

This would appear to bring us round full circle to the PANS/PANDAS pathology that many specialists have said should really be renamed autoimmune encephalitis. Perhaps the underlying drivers of these merciless diseases are not so different after all.

**\*Dr. Schwarzbach suggests also testing for Echovirus where there is a suspicion of Cocksackie virus, as this is also frequently found. See the next newsletter for an item on the Echovirus (enteric cytopathic human orphan virus).**

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