The Role of Microbes in Parkinson's disease

As in other neurological diseases, microbes may be implicated in Parkinson's disease (PD). A number of studies have looked at a correlation of the bacterium Helicobacter pylori (HP) which is responsible for stomach ulcers, and the development of Parkinson's disease. Some have suggested this microbe may play a role but others show no obvious correlation, although a recent meta-analysis (an analysis that looks at all the studies) felt overall, there probably was a link [1]. However, meta-analyses especially based on small studies, are not always reliable. In one centre, it was found in two separate studies, that 33% and 48% of patients with PD had evidence of infection with HP which is much higher than in the general population or other neurological diseases.

Once the infection was eradicated, their symptoms improved together with their quality of life. However, it was felt that the reason for this improvement was eradication of the bacteria led to better absorption of their medication used to treat the symptoms of PD [2].

Another study from Israel investigated whether there was an association between viral hepatitis B or C with the development of PD. Although there was a small increased with hepatitis C, the authors were unable to conclude that it was the virus responsible for the increased risk or the underlying liver disease. It is also known that liver cirrhosis can cause symptoms which are very similar to PD [3].

A study looking at the risk of developing PD depending on whether you had measles as a child, showed a protective effect. This suggests that the measles virus may prime the immune system in some way, against the agent responsible for PD [4].

Before symptoms affecting movement and rigidity occur, many but not all people with PD have a long history of a decrease in the sense of smell and constipation is a common symptom (28-80%), going back over the previous ten years [5, 6]. Their sleep is also affected and they are prone to bouts of depression.

The vagus nerve, which originates in the lower part of the brain, supplies and

receives messages from the stomach and intestine and the heart. New evidence suggests that PD may actually be caused by an agent that travels up the vagus nerve into the brainstem, and then affects that part of the brain, associated with Parkinsonian symptoms [7].

Historically, in the treatment of stomach ulcers before the advent of drugs to reduce acid production, the vagus nerve was cut surgically. In those who underwent this operation, the incidence of PD was reduced [8, 9]. The vagus nerve controls bowel movement. As constipation is a result of decreased bowel motility, it may be the vagus nerve is affected which is failing to stimulate the intestine.
The only other direct way (other than the circulatory system) is via the olfactory nerve, which originates in the nose and had direct access to the brain. It is therefore possible an agent responsible for PD gains access by this route [10].

People who smoke or drink a large amount of coffee have a decreased risk of developing PD.
It has been shown, that both of these affect the bacteria in the gut. Examination of the gut bacteria in those with PD, has shown that the quantity of a bacterial family called Prevotella is 78% lower than in controls, with five other types of bacteria being more common in those with PD [11-13].

What is unknown, is whether this altered gut bacteria are a cause, a risk factor or the result of changes in the intestine due to PD. One of the classical changes found in PD are called Lewy bodies. These are seen in the affected nerves but no one knows exactly what they are. They have been shown to contain a protein called a-synuclein but whether this is a cause or effect of PD is debated. It is known that a-synuclein can be produced by nerve cells but the question remains whether this is part of a defence system against some agent. Families in which Parkinson’s disease is passed on from generation to generation have a mutated form of this protein which suggests that the normal protein is in some way protective but why is it overproduced?

These Lewy bodies are also found in the nerves lining the gut suggesting a link between the gut and the brain. In addition, the way in which the Lewy bodies spread from nerve to nerve suggest the possibility of a transmissible agent [14].

Finally, a woman whose husband suffered from PD became aware that all Parkinsonian sufferers have a distinct smell. Her ability to smell people with PD

was confirmed in a blind trial.

People with PD have been shown to have increased seborrhoeic dermatitis which is a type of skin eczema.

This is another example of what is known as gut-brain-skin axis suggesting a link between all three.

Sebum as opposed to sweat is secreted in the middle of the top of the back and forehead but not in the armpits. Further research showed that it was the presence of at least two chemicals in the sebum of PD patients what this woman could smell (or contributed to the smell) which were not present in those without PD [15].

A type of fungus called Malassezia was found to be present on the skin, and has been shown to grow in excess in people with seborrhoeic dermatitis. It was also noted that some bacteria produce an anti-fungal agent eicosane, which was one of the chemicals found to be present in the sebum of PD patients.

The question is why do PD patients have increased sebum production in the first place and is the smell a chemical produced within the sebum, or secondary to an overgrowth of fungi on the skin surface?

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