LETTERS

The usefulness of ultrasound diagnosis specifically in taeniasis

In a recent issue of *Gut*, van Beurden *et al* showed the usefulness of ultrasound in the diagnosis of intestinal worms in a case of human taeniasis (*Gut* 2008;**57**:515 and 524). The examination was even more sensitive than the stool analysis, which remained negative. However, and specifically in the case of human taeniasis, ultrasound techniques might have a double diagnostic advantage: the detection of the intestinal adult tapeworm as well as the search of the presence of extraintestinal *Taenia* larval stages in the same patient.

Human taeniasis caused by pig (*Taenia solium*) or cattle (*T saginata*) tapeworms arises after eating pork or beef contaminated with cysticerci, the larval stage of these parasites. Due to the frequently asymptomatic and benign course of the intestinal parasitation, the main risk of these parasites is that taeniasis caused by *T solium* can lead to another dangerous disorder: cysticercosis. This disease is acquired through the ingestion of eggs shed by a carrier of the adult tapeworm. These seemingly asymptomatic patients place themselves, as well as other people (who accidentally ingest the eggs), at risk. The brain is most often affected (neurocysticercosis), and other common locations for the cysticerci include subcutaneous tissue, skeletal or heart muscle and the eye.

Up to 40% of patients harbouring intestinal *T solium* also show some type of cysticercosis, therefore it would be advisable to look for cysticerci in those patients who harbour *T solium*. Ultrasonographic techniques are particularly accurate in the diagnosis of cysticercosis.[1 2]

In the case reported by van Beurden *et al,* the species was identified as *T saginata*, which does not cause human cysticercosis. However, there is another pig tapeworm affecting humans, *T asiatica*, found in the majority of Asian countries, although its definitive geographical distribution is still unknown. The morphology of *T asiatica* overlaps with that of *T saginata*, so its specific diagnosis is only possible by using molecular methods.

The main uncertainty concerning *T asiatica* is whether the parasite is able to cause human cysticercosis, and this question should be addressed urgently in order to prevent this disorder.[3]

As no immunological method is available for the diagnosis of *T asiatica* cysticercosis, we believe that one important tool for the investigation of the presence of cysticerci, at least in patients harbouring the adult stage of *T asiatica,* is by using liver ultrasonography since *T asiatica* cysticercus shows a clear liver tropism in pigs, its natural intermediate host.[4]

We do not know if the patient treated by van Beurden *et al* had visited any Asian country, but due to his origin (Morocco), he would probably not eat pork, so he was likely to have harboured *T saginata*. But in any other case, and if no molecular technique is applied in the diagnostic process of the adult stage, it would be very useful to investigate the presence of cysticerci in the liver of all those patients harbouring *T saginata*-like adults.

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TRPV1-expressing sensory fibres and IBS: links with immune function

The paper by Akbar *et al* (*Gut* 2008;**57**:923–9) characterises the expression of the capsaicin receptor, transient receptor potential vanilloid type-1 (TRPV1), immune cell markers, and their relationship to symptoms in irritable bowel syndrome (IBS). They conclude that the increase in TRPV1-expressing nerve fibres may contribute to visceral hypersensitivity in IBS, and therefore provide a novel therapeutic target. An important parallel finding was that the overall density of innervation (protein gene product 9.5 (PGP9.5)- and substance P-positive) is increased along with TRPV1. Therefore, the upregulation of neuronal markers is not selective for TRPV1, and probably represents axonal sprouting. Most colonic sensory nerves normally express TRPV1,[1] indicating that it may be a useful general marker for them and their proximity to other cells.

Akbar *et al* also show a dramatic increase in the numbers of lymphocytes (CD3+) and mast cells (ckit+) in IBS, which are therefore more likely to release mediators in the vicinity of TRPV1+ sensory fibres. This corroborates evidence in the literature from several groups,[2–4] including ours, showing direct effects of mast cell degranulation on colonic sensory endings in a postinflammatory animal model.[5] Recently we also showed a strong correlation between increased release of proinflammatory cytokines by cultured peripheral blood mononuclear cells (PBMCs) from patients with postinfectious IBS and their symptoms.[6] These results indicate an activated immune system in these patients, with lymphocytes continuing to secrete proinflammatory mediators in the absence of overt pathological colonic damage. Whilst Akbar and colleagues have taken an anatomical approach to the correlation of sensory innervation with immune cell populations, we took a functional approach. We determined if mediators released by PBMCs (approximately 75% of which are CD3+) from patients with IBS and healthy controls are able to modify the function of colonic sensory nerves.

We took blood from 36 healthy controls (HCs) and 20 patients with postinfectious diarrhoea-predominant (PI/D) IBS consistent with ROME II criteria. PBMC supernatants were derived as described elsewhere[6] and pooled from each group. In vitro single fibre recordings were made from mouse colonic pelvic or lumbar splanchnic nerves.[1] Categorisation of afferent fibre properties was based on previously published classifications[1]: serosal and mesenteric afferents responded only to focal compression and high intensity stretch; mucosal afferents responded only to fine mucosal stroking; muscular afferents responded to low intensity stretch, but not mucosal stroking; and muscular-mucosal afferents responded to both stretch and mucosal stroking. Mechanosensitivity was determined before and after exposure to PBMC supernatant.

Sensitivity to mucosal stroking in pelvic muscular-mucosal afferents was reduced by HC supernatants. However, the sensitivity of pelvic serosal, muscular or mucosal afferents and lumbar splanchnic serosal afferents to their appropriate mechanical stimuli was unchanged following addition of HC supernatants (fig 1, and data not shown). In contrast, PBMC supernatants from patients with PI/D IBS increased mechanosensory responses of all afferent classes to the corresponding stimuli by up to 112% (fig 1).

Our data indicate immune sensitisation of all classes of pelvic and splanchnic afferent endings by PBMC supernatants from patients with PI/D IBS. This indicates that secretory factors from PBMCs act to sensitise colonic afferent endings irrespective of the layer of colon innervated and nature of mechanical stimuli transduced. An unexpected discovery was the inhibition of muscular-mucosal afferents by HC supernatants. PBMCs therefore normally secrete factors that act to dampen the mechanosensitivity of these afferent endings under healthy conditions. This adds another layer of complexity to the regulation of afferent signalling by interactions between neural and immune systems. Muscular-mucosal afferents were the most revealing of the change in neuroimmune interactions from