This data suggests that there may be a lack of education among Ugandan mothers surrounding these practices, and demonstrates a need for further inquiry.

The Nutrition and Early Childhood Development Project ran from 1998-2005 in about half of the country's districts and was successful in increasing awareness and promoting positive behavioural changes in complementary breastfeeding at six months, among other targets.[3] This was a national strategy involving all levels of government and the community; however, the local health offices believe the most effective way to create change in feeding practices and nutrition among mothers within a specific district is with peer mothers, community health workers, and midwives who can share their first-hand knowledge and evidence-based recommendations with mothers.[2]

The original goals of our UBC GHI project, which initiated our presence in Nakaseke Uganda, were in the field of health education, with a focus on sexual education, first aide, and life skills. We aimed to train the nursing aide students in Nakaseke District, Uganda, with the goal of having them translate this knowledge to secondary school students and the wider community. With the nursing aide school closing down in preparation for a new nursing program beginning in the next few years, our team will have the opportunity to connect with and train community health workers in the district on maternal and children's health. It is our hope that through focus groups with local mothers, insight may be gained to address our questions regarding the area's childhood malnutrition, improve maternal education, and to find a sustainable path towards improved childhood health in the Nakaseke region.

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Troubles with Diagnostic Tests: Observations from a Clinic in Tanzania

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In developed countries, diagnostic tests are a critical part of clinical medicine. This is because the available tests are generally highly accurate and can quickly confirm or exclude suspected diagnoses. As a result, physicians in developed countries have become increasingly reliant on such tests. In developing countries the same model is used, but the tests have limited accuracy. In these situations, physicians must rely more on clinical findings to obtain correct diagnoses. Drawing from a three-week shadowing experience at a clinic in Mwanza, Tanzania, I will provide a personal perspective on the challenges of diagnosing schistosomiasis and typhoid fever in a setting where resources are limited.

Schistosomiasis has a unique pathogenesis with multiple clinical stages. *Schistosoma haematobium* and *Schistosoma mansoni* are the blood flukes that cause urinary and intestinal schistosomiasis, respectively. In the population living on the shore of Lake Victoria, a region that includes Mwanza, the prevalence of *S. mansoni* ranges from 40-100%, while the prevalence of *S. haematobium* is the same if not greater.[1] Both species are found in contaminated water and infect humans by penetrating the surface of exposed skin. In the body, they travel hematogenously to veins of the bladder and intestine. Here, they imbed in the vasculature, reproduce, and shed their eggs, which then are excreted via the bowel or bladder. Patients with schistosomiasis can present with either acute or chronic symptoms. Acute symptoms, commonly called Katayama fever, are due to egg deposition in body tissue and include fever, myalgia, diarrhea, and hematuria (*S. haematobium*). Chronic symptoms are due to an inflammatory response to Schistosoma eggs and high worm burden. They include splenomegaly, hepatic and genitourinary fibrosis, and in later stages, bladder cancer.[2,3]

At the clinic, I saw many patients with symptoms resembling Katayama fever. These patients would have urine and stool samples taken for analysis because of the high clinical suspicion of microbial infection, and in particular schistosomiasis. However, it soon became evident that *Schistosoma* ova or parasites were only detected on rare occasions. In fact, when the clinic's laboratory records of 1000 stool and urine samples were examined from an unscreened population, only three documented cases of *Schistosoma* were found. Even for an unscreened population, the infection rate was surely higher in an endemic area.

The gold standard for schistosomiasis diagnosis remains

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microscopic examination of stool or urine, depending on the species. This examination is highly specific, but is problematic because many patients do not actively shed eggs.[2] In developed countries, serological testing with ELISA has largely replaced microscopy in diagnosis.[4] Seeing as these tests were not available at the Mwanza clinic, there was a high likelihood that schistosomiasis was underdiagnosed, possibly contributing to long-term complications such as fibrosis and cancer in several patients.

Like schistosomiasis, typhoid fever is also endemic in Mwanza, but, unlike the former, the effect of using poor diagnostic tests may be leading to overdiagnosis of this disease. While visiting another regional clinic in Mwanza, I saw a patient presenting with dyspnea. This man was wheelchair-bound from diabetic neuropathy that required amputation, and presented with elevated blood pressure, peripheral edema, and physical signs of fluid in his lungs. The clinic physicians agreed that this man likely had congestive heart failure and uncontrolled diabetes. He was prescribed medication for these conditions along with an antibiotic. The antibiotic was to cover *Salmonella*.

Salmonella enterica serotype typhi and paratyphi (henceforth referred to as *S. typhi* and *S. paratyphi*) are the major causes of tyhpoid fever in children and young adults in sub-Saharan Africa with an estimated 725 cases per 100,000 per year.[5] The bacteria are ingested into the small intestine where they invade the epithelial lining. The intestinal epithelium becomes damaged leading to macrophage recruitment. The macrophages come to the site of damage, phagocytose the bacteria, and become vectors for bacterial proliferation. Eventually the pathogens destroy their host macrophages and disseminate into the bloodstream causing fever and sepsis.[6] The patient with heart failure did not exhibit any of these symptoms but was still treated based on a high antibody titre on a Widal test.

The Widal test is a qualitative test for agglutinating antibodies to *S. typhi* and *paratyphi* antigens. This test was developed in the 19th century and is a serologic test that detects antibodies against flagellar H-antigens and somatic O-antigens present on *S. typhi* and *paratyphi*. Exact protocols vary, but generally a sample of the patient's blood is taken and diluted 1 in 40 into two samples. Twofold serial dilutions are then performed (1:80, 1:160, and 1:320) and then anti-H and anti-O antibodies are added: one to each sample. A positive agglutination test is defined as the presence of coagulation one minute after the addition of antibody. The most dilute sample (highest titre) that shows coagulation is recorded. At both clinics, a titre of 1:80 or greater, with either the H or O-antibody, was considered positive for typhoid fever.

The Widal test has been criticized for its poor specificity for typhoid fever. A recent study from the WHO has estimated the specificity to be around 50%.[7] Its use in endemic areas has also been questioned as the baseline antibody titre in these populations is unknown and likely already high. A study in Nigeria showed that 25% of malaria and *S. typhi* negative patients had a titre of 1:80 or greater.[8] Furthermore, to appropriately use this Widal test as a diagnostic tool, consecutive tests must be performed seven to ten days apart and they must show at least a four-fold rise in antibody titre. For many patients it is not feasible to wait this long because they must travel long distances at great expense to attend clinics. With no other diagnostic tests available, physicians in Mwanza commonly prescribe antibiotics to cover *S. typhi* based on a single abnormal test result. This likely results in an overdiagnosis of typhoid fever and contributes to possible misdiagnosis and antibiotic resistance.

Until better tests become available, the Mwanza clinic and others like it must continue to rely on tests that are available to them. To improve outcomes, these tests can be supplemented with clinical findings. For example, a study of Tanzanian schoolchildren showed that self-reported hematuria and schistosomiasis have similar efficacy to urine microscopy in determining *S. haematobium* infection.[9] Similarly, another Tanzanian study found that a history of fever lasting more than three days was more specific than the Widal test for typhoid fever. When the two were combined the specificity was 100%.[10] *S. typhi* is also a purely human pathogen and the likelihood of infection can be predicted from a patient's history, particularly if they have shared food and had close contact with anyone experiencing similar symptoms. Through careful history taking, physicians can supplement positive lab findings and avoid unnecessary testing in low risk patients.

When poor tests are used in endemic areas, problems with diagnosis become magnified. Low sensitivity tests create missed opportunities to treat diseases during their early stages before patients develop chronic complications, while low specificity tests cause inappropriate and excessive use of pharmaceuticals. In Canada we are fortunate to have better tests available to us, but we should remember that these are also not 100% accurate. For this reason, we should be aware of the accuracy of the tests we use, and always pair them with our clinical judgment.

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