effect. Notably the slowing of gastric emptying induced in the liraglutide group was more pronounced at 5 weeks than at 16 weeks, suggesting tachyphylaxis.

The main strength of this study is that, unlike previous studies, gastric functions were measured as rigorously as possible using currently available techniques. The clinical effect exerted on gastric emptying is important not only from a scientific viewpoint, but also for practical reasons, since measuring this gastric function after approximately 1 month of treatment could help to identify patients who would respond in the long term to liraglutide with significant weight loss, thus avoiding ineffective and expensive treatments in non-responders.

Certain criteria are required to determine the pathophysiological role of a gastrointestinal abnormality in the determinism of functional digestive disorders: the abnormality must be demonstrated in association with the pathological condition; a correlation must exist between level of impairment and severity of the associated condition; and effective treatment of the disease must congruently affect the underlying functional abnormality. Extrapolating these concepts to the field of obesity, the study by Halawi and colleagues links gastric dysfunction to the determinism of the disease. Nevertheless, the study leaves many open questions. Specifically, the observation that the delaying effect on gastric emptying decreases over time casts some shadows about the clinical effectiveness of the treatment: was this exclusively due to the tachyphylactic effect expected from drugs acting on cell receptors? Or rather to an autoimmune response developed by some predisposed individuals against injected proteins?

If an average of 5 kg were lost after 16 weeks, what is the expected duration of clinically meaningful treatment? Can a carryover effect be expected after suspension of the therapy? And, if so, for how long? Is it conceivable that repeated treatment periods would be both more effective in preventing tachyphylaxis and cheaper than continuous maintenance therapy? As physicians we need answers to these and other questions, but for the time being we have learned two important lessons. First, obesity is not “all in the head”; the periphery (ie, the stomach) plays a pivotal part in its determinism. Second, there are effective medical alternatives to surgery we can offer to our patients with obesity.

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Funding the elimination of viral hepatitis: donors needed

Chronic hepatitis B and C are life-threatening infectious diseases that cause serious liver damage, cancer, and premature death. More than 300 million people are infected with hepatitis B or hepatitis C and are at risk of developing escalating health problems. These infectious diseases cause 1·3 million deaths every year and are responsible for more than half of all new cases of liver cancer and one in every 12 cancer deaths. Whereas the burden of other major infectious diseases such as HIV, tuberculosis, and malaria is decreasing as a result of consistent large-scale global investment, viral hepatitis has been neglected, resulting in ever increasing numbers of people dying from hepatitis B and C. 2

Despite the size of the challenge and the years of inaction, in 2016 WHO member states signed up to the goal of eliminating hepatitis B and C as a public health threat by 2030, now just 13 years away. This critical target was agreed because well evidenced solutions for the prevention and treatment of hepatitis B and C exist. Vaccination provides highly effective protection against hepatitis B,3 safe blood and infection control should

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be the basis of any health system, and harm reduction works. Existing treatment can effectively manage hepatitis B and prevent liver cancer, and new treatments can cure hepatitis C in nearly all patients.\(^4\)

However, efforts to address viral hepatitis are only just beginning. Few countries have established national plans that make treatment and prevention available for all patients. A lack of national and international investments in viral hepatitis programmes, especially in low-income and middle-income countries, mean that even fewer countries have a national plan that is properly funded. As a result, the vast majority of patients remain undiagnosed—91% of those with hepatitis B and 80% of those with hepatitis C—and only 1% are able to access treatment.\(^1\) Even the birth dose hepatitis B vaccine, which can cost less than US$0·20, is not still used in many countries in Africa and the eastern Mediterranean region.\(^6\)

Part of the problem is that hepatitis B and C are silent epidemics with few obvious symptoms for many years and often hit children and marginalised populations the hardest. Hepatitis B continues to spread unnoticed from mother to child at birth, putting infants at high risk: 80–90% of babies infected with hepatitis B will develop chronic disease and one in four will die of liver-related causes during adulthood.\(^5\) Additionally, in low-income and middle-income countries, millions of people have been and continue to be infected, inadvertently and unnoticed, in health-care settings via the use of unsterilised equipment and unscreened blood transfusions.\(^3\) People living with HIV are also hard-hit by hepatitis B\(^6\) and hepatitis C,\(^7\) as are other stigmatised or marginalised groups with poor access to care, such as people who inject drugs, migrants, and indigenous populations.\(^1\)

The commitment to eliminate hepatitis B and hepatitis C by 2030 matters because it offers the opportunity to prevent approximately 36 million infections and save 10 million lives.\(^5,6\) Furthermore, as well as delivering on Sustainable Development Goal (SDG) 3.3 it offers broader progress towards other SDGs, particularly targets to end poverty, ensure good health and wellbeing, and reduce inequalities. Improving access to prevention and treatment services can help protect patients against catastrophic health-care costs and productivity losses. At the same time, many countries will realise cost savings from the elimination of hepatitis B and C. Elimination would help ensure healthy futures for children; protect, empower and reduce stigma among marginalised populations; and support and protect commitments to HIV-positive populations.

In an era where the emphasis is more on strengthening health systems than on vertical programmes, viral hepatitis elimination will require many interventions that are crucial for health system strength. Enhanced infection control services, including injection and blood safety and vaccination for health workers, will improve quality standards in health systems and reduce transmission of health-care-acquired illnesses for both patients and providers. Improved hepatitis B immunisation can help increase coverage of other childhood vaccinations and ensure that newborn babies receive appropriate postnatal care. Prevention of mother-to-child hepatitis B transmission can improve access to perinatal care, ensure healthy pregnancies, and promote institutional deliveries. Harm reduction efforts will help prevent the spread of blood-borne viruses such as HIV and syphilis.

Given the immensity of the task of eliminating hepatitis B and C—diagnosing more than 300 million people stands out as a particular challenge—donors are going to be essential. Domestic funding is unlikely to be able to deliver the results in the 13 years that remain till 2030. None of the big global donors shows any sign of committing to viral hepatitis. However, they are beginning to look at HIV/hepatitis C co-infection and both UNITAID and the UK’s Department for International Development (DFID) are supporting market-shaping programmes for hepatitis C. Continued advocacy with donors is needed to support prevention interventions, which almost all positively
Alcoholic hepatitis: calling time on an unhelpful diagnosis

Alcoholic hepatitis is a sudden and dangerous type of alcohol-related liver disease. Most clinicians with a passing familiarity with liver disease will recognise the clinical syndrome of a patient with alcoholic hepatitis: an unwell, heavily jaundiced, abuser of alcohol.1 The term alcoholic hepatitis has been commonplace in medical literature for over a century, but as a diagnostic term it is imprecise, with regard to both the psychiatric and physiological aspects of the condition. Recognising this, the diagnostic term alcoholic hepatitis should be replaced with a more precise and useful diagnosis that reflects the accumulated knowledge of this dangerous condition.

Use of the term alcoholic implies the presence of alcohol dependence, and as such the definition is inaccurate. Alcohol dependence is common but certainly not necessary for the development of alcoholic hepatitis. Indeed, data from the TREAT consortium2 in the USA found that patients with alcoholic hepatitis had fewer drinks per day and fewer binges than matched patients with other types of alcohol-related liver disease. A clear distinction between harmful use of alcohol and alcohol dependence exists that is not reflected by the blanket application of the term alcoholic. This term also has a strong stigma attached to it and carries many assumptions, for both the patient and the clinician, that can obstruct treatment.3 Achieving abstinence is the most important factor in predicting long-term survival in patients with alcoholic hepatitis. Accordingly, clinicians should remove every possible barrier to successful treatment of alcohol abuse or dependence. Abandoning inaccurate and pejorative terminology is an important step towards this goal.

Alcohol can undoubtedly cause hepatocyte necrosis, which can increase during acute illness. Nevertheless, concentrations of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are typically only moderately elevated in patients with alcoholic hepatitis. Therefore, patients who present to non-specialists with high aminotransferase concentrations—inconsistent with alcoholic hepatitis—can be misdiagnosed as having alcoholic hepatitis if they disclose hazardous alcohol use. Such misdiagnoses could contribute to the observation that non-specialist management of alcoholic liver disease is often sub-optimal.4