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CME: Diabetes

# Diabetes and the liver ☆

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ABSTRACT

Type 2 diabetes mellitus (T2D) and metabolic dysfunction-associated steatotic liver disease (MASLD), formerly termed non-alcoholic fatty liver disease (NAFLD), are increasingly prevalent conditions that are closely associated. The shared pathophysiology of insulin resistance, chronic inflammation and increased lipid deposition means that there is a bidirectional relationship between the two conditions. MASLD is now a leading cause of cirrhosis and hepatocellular carcinoma (HCC) and has a high prevalence in patients with T2D. This review explores the synergy between MASLD and T2D, while also outlining the diagnostic approach and management of T2D in the context of MASLD. Management involves lifestyle intervention, optimisation of cardiometabolic risk factors and multidisciplinary team involvement. We evaluate the use of antidiabetic medication such as SGLT-2 inhibitors and GLP-1 receptor agonists. Early identification of MASLD can help guide treatment and reduce the risk of progression to liver cirrhosis in high-risk patients.

## Introduction

In the UK, an estimated 5.8 million people live with diabetes mellitus (DM); 90% have type 2 diabetes (T2D).[1] The co-existence with liver disease is a well-recognised association that is not always prioritised in clinical practice. The formerly termed non-alcoholic fatty liver disease (NAFLD) is now called metabolic dysfunction-associated steatotic liver disease (MASLD).

As defined by the European Association for the Study of the Liver (EASL),[2] MASLD is the presence of excess triglyceride storage in the liver with at least one cardiometabolic risk factor. MASLD and T2D are closely associated, both raising the risk of cardiovascular disease.[2] There is a bidirectional relationship between DM and liver disease: type 1 diabetes (T1D) can lead to excess hepatic fat while on insulin therapy;[3] T2D increases hepatic lipid production and the risk of atherosclerotic disease; similarly, steatotic liver disease increases hyperglycaemia,[3] increasing the risk of developing T2D.[4] This review explores how T2D is a cause and consequence of liver dysfunction.

## Liver disease and diabetes

The association with liver disease and diabetes primarily concerns T2D and MASLD. T2D has the hallmark of insulin resistance (IR) and it is acquired via genetic and environmental factors, resulting in hyperglycaemia, subsequent lipogenesis and increased fatty acid deposition.[3] Fig. 1 describes the relationship between T2D pathophysiology, lipid metabolism and the liver.

## MASLD

MASLD is a spectrum of conditions that includes steatosis (MASL), steatohepatitis (MASH) and liver fibrosis[2,4] (Fig. 2).

In T2D there is an increased hepatic uptake of FFA and *de novo* liponeogenesis[5] (Fig. 1). Adipokines such as leptin and tumour necrosis factor-alpha (TNF-a) are produced in excess and promote inflammation, cellular necrosis and ultimately fibrosis.

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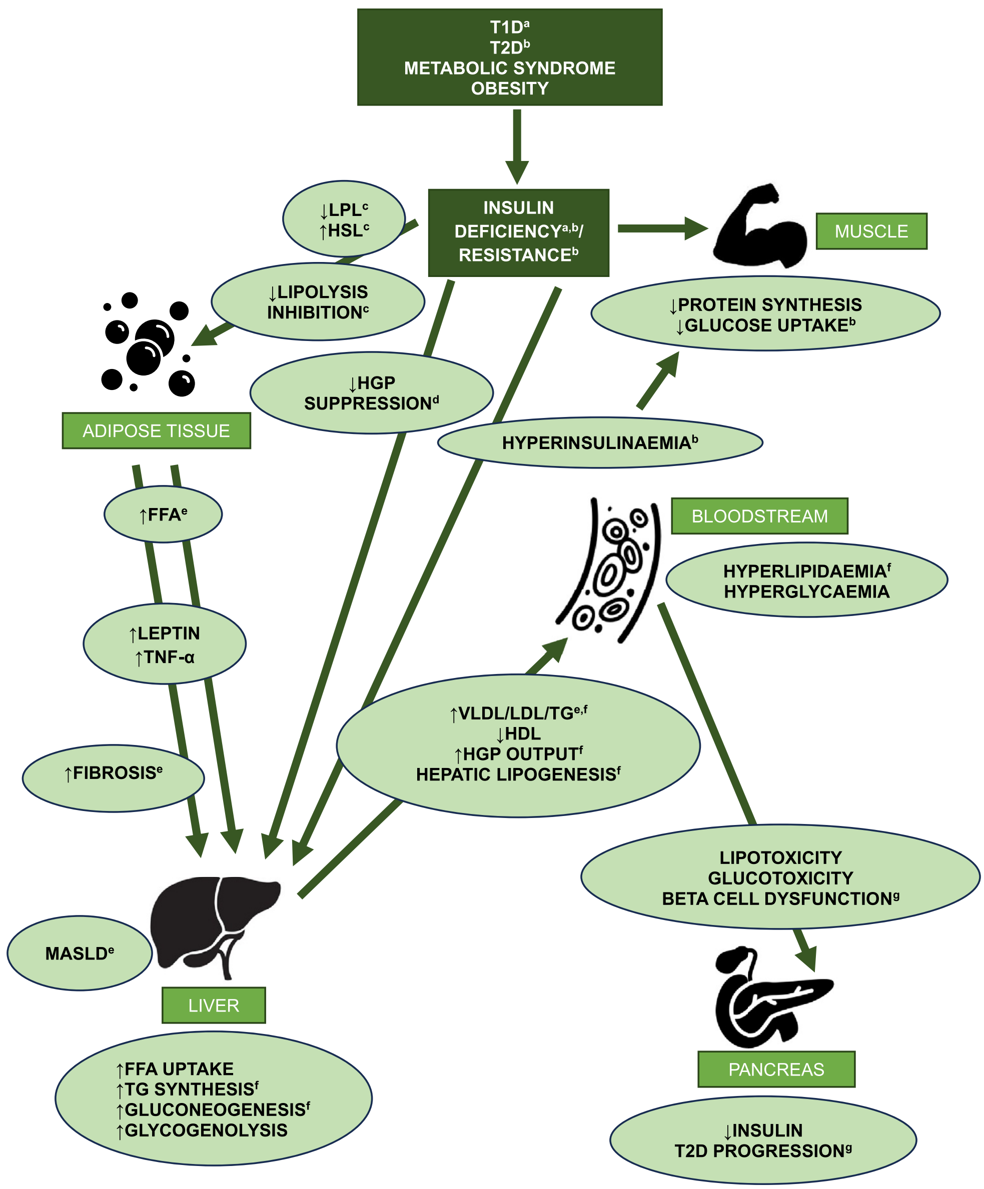


**Fig. 1.** Schematic diagram illustrating the connection between insulin resistance, lipid metabolism and glucose metabolism.[5]
[a]T1D is characterised by autoimmune destruction of pancreatic beta-cells. [b]In T2D, obesity and metabolic syndrome, insulin has a reduced effect on sensitive tissues and there is reduced glucose uptake from peripheral tissues. There is a compensatory hyperinsulinaemia. In both circumstances, there is insufficient insulin action. [c]Insulin is required to suppress lipolysis. Without sufficient insulin, there is reduced lipolysis inhibition via reduced stimulation of LPL and reduced inhibition of HSL as well as [d]reduced suppression of HGP. [e]Increased lipolysis causes a higher hepatic output of lipids (vLDL, TG), progression to steatohepatitis (MASH/MASLD) and fibrosis and increased HGP. [f]Ultimately, insulin resistance and hyperinsulinaemia result in hyperlipidaemia, adipose hypertrophy and hepatic *de novo* lipogenesis as well as hyperglycaemia. [g]Chronic hyperlipidaemia impairs pancreatic beta-cell function and subsequently causes a progression of T2D.
Abbreviations: FFA, free fatty acids; HGP, hepatic glucose production; HSL, hormone sensitive lipase; LDL, low-density lipoprotein; LPL, lipoprotein lipase; MASLD, metabolic dysfunction-associated steatotic liver disease; MASH, metabolic dysfunction-associated steatohepatitis; TG, triglycerides; T1D, type 1 diabetes mellitus; T2D, type 2 diabetes mellitus; TNF-α, tumour necrosis factor alpha; VLDL, very low-density lipoprotein.

The current estimated global prevalence of MASLD in adults is 38% and is projected to be more than 55% by 2040.[6] This is accompanied by a rise in obesity, with the World Obesity Federation estimating a doubling of rates by 2035.[6] MASLD is now the leading cause of cirrhosis, surpassing alcohol-related liver disease.[6,7]

Metabolic syndrome is a cluster of conditions which include obesity, hypertension, dyslipidaemia and IR. The parallel rise of both obesity and MASLD is indicative of the synergistic relationship between T2D, IR and liver disease.[8] The global prevalence of MASLD in T2D is estimated to be up to 69%.[6] There is increased prevalence in North African, Middle Eastern, Latin American and Asian populations.[4] The overlap includes obesity (though not always), genetic predisposition and visceral adiposity.[7–9] 'Lean MASLD' is an increasingly prevalent phenotype most observed in South and East Asian populations (BMI <23kg/m$^2$ and <25kg/m$^2$ in other populations). This subgroup is 10–20% of the MASLD cases globally and carries similar hepatic and cardiometabolic risks.[2,6]

MASLD is a multisystem disease which includes extra-hepatic associations such as atherosclerotic cardiovascular disease, chronic kidney disease and reduced bone density.[2,9] The most severe form of MASLD is irreversible liver cirrhosis (Fig. 2), occurring in up to 25% of patients with MASH.[6] Cirrhosis is typically clinically silent until decompensation, including complications such as portal hypertension, variceal bleeding and ascites.[2,6] Hepatocellular carcinoma (HCC) is a complication with poor prognosis and a 5-year mortality of 74.6%.[6] Patients with T2D are up to three times more likely to develop HCC than those without T2D.[10]

*Diagnostic approach*

Though annual DM reviews do not mandate routine testing for liver disease[11], UK guidelines (NICE CKS, EASL) recommend screening for MASLD in higher-risk groups, including patients with T2D[2,12,13] (Box 1). Caution is required in the presence of anaemia, often seen in advanced liver disease, when interpreting HbA1C.[14] Similarly, the oral glucose tolerance test has diagnostic limitations in compensated cirrhosis, where glucose levels may not be elevated.[15]

Mildly deranged liver function tests (LFTs) are commonly seen in patients with T2D.[16] This is likely due to excess FFA that are toxic to hepatocytes. Coupled with increased oxidative stress, hepatocellular injury occurs.[16] Gamma-glutamyl transferase (GGT) is a non-specific marker

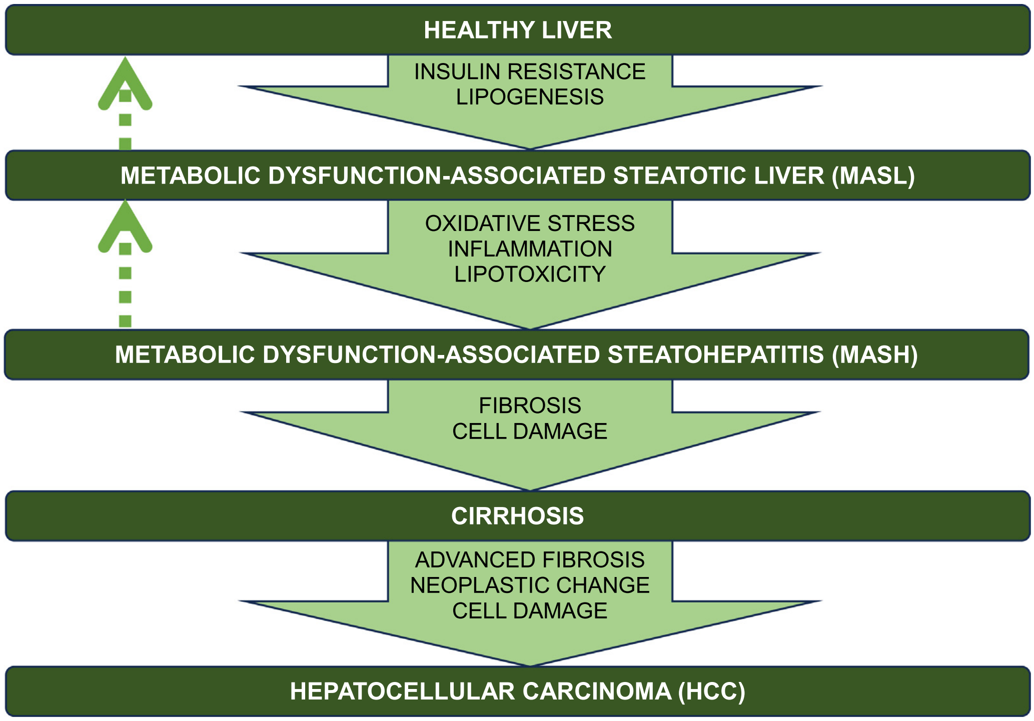


**Fig. 2.** Flowchart illustrating the progression of MASLD.[2]
MASLD encompasses a spectrum of conditions and there is typically, but not always, a sequential nature of liver injury. Hepatic triglyceride accumulation (fat in >5% of hepatocytes) without a secondary cause would be defined as the first benign presentation of MASL (hepatic steatosis). Progression to MASH occurs through further inflammation from adipokines. Ongoing cellular injury, fibrogenesis and architectural destruction leads to cirrhosis. Regression is possible with effective management until the cirrhotic stage. At this point, only liver transplantation would be able to treat liver cirrhosis. Chronic metabolic dysfunction and fibrosis carries a high risk of neoplastic transformation into the malignant HCC.

**BOX 1**
Guidelines for annual diabetes monitoring.[14,15]

- HbA1C
- Review of blood glucose patterns (CGM or CBG data)
- Medication review
- Urinary albumin : creatinine ratio (ACR)
- Renal function
  - serum creatinine
  - eGFR
- Foot health
- Retinal screening
- Metabolic health
  - Weight and BMI
  - Liver function tests (LFTs) – ALT, AST, GGT, ALP, bilirubin, albumin
  - Lipid profile
  - Screen for MASLD if deranged LFTs
- Mental health
- Blood pressure
- Smoking status
- Hypoglycaemia frequency and awareness
- Driving and occupational hazard

Abbreviations: ALP, alkaline phosphatase, ALT, alanine aminotransferase, AST, aspartate aminotransferase, BMI, body mass index, CBG, capillary glucose monitoring, CGM, continuous glucose monitoring, GGT, gamma-glutamyl transferase, eGFR, estimated glomerular filtration rate, HbA1C, haemoglobin A1C (glycated), MASLD, metabolic dysfunction-associated steatotic liver disease.

that can also be raised and has a positive association with alcohol intake, coronary artery disease and high BMI.[16]

Radiological steatosis or abnormal LFTs can be the first marker of MASLD. As few as 3.1% of people at risk of MASLD are aware of the diagnosis.[8] British Society of Gastroenterology (BSG)[13] guidance suggests that chronically raised LFTs for more than 6 months warrant a screen for treatable causes of chronic liver disease, including MASLD, with a liver ultrasound in the first instance. Baseline LFT monitoring is recommended for pioglitazone and it should be avoided if ALT is more than 2.5 times the upper limit of normal.[11]

### *Risk stratification*

FIB-4 score is an index that highlights the risk of liver disease (Box 2), with the caveat that it is not validated for those <35 years old.[17] There are more false positives in T2D and older age due to altered ALT and platelet profiles.[2] Enhanced liver fibrosis (ELF) score is another non-invasive blood test that assesses the level of liver scarring (Box 2). Both these scores help to stratify the need for specialist input and further imaging. Those with T2D and evidence of steatosis on ultrasound should be screened with the FIB-4 or ELF. Additional non-invasive markers include the NAFLD fibrosis score (NFS) and AST : platelet ratio index (APRI).

FibroScan is a type of transient elastography measuring liver stiffness. NICE and EASL-EASD-EASO guidelines[2,12] suggest that individuals with indeterminate or high-risk non-invasive scores (Box 2) should undergo a FibroScan. Results can be combined with serological markers for scores (Box 2). Should the FibroScan results be discordant with the blood test scores, a liver biopsy is recommended.[2]

### *Screening*

Screening should be done annually in DM and, once diagnosed with MASLD, testing for advanced fibrosis should be every 3 years. 6-monthly surveillance ultrasound scans are offered to those at higher risk of developing HCC.

**BOX 2**

Recommended diagnostic approach for MASLD.[17,18]

- Identification of high-risk groups, eg patients with pre-diabetes/T2D, obesity, hepatic steatosis, PCOS
- History including comorbidities
  - Liver disease
  - Metabolic syndrome-related issues, eg OSA, hypertension, dyslipidaemia
  - Alcohol use
  - Medication, eg corticosteroids, methotrexate, ARVs
- LFTs, HbA1C, fasting lipids
- Exclude secondary causes of steatohepatitis (eg viral hepatitis, starvation and refeeding, inherited disorders, autoimmunity)
- FIB-4 score (https://www.mdcalc.com/calc/2200/fibrosis-4-fib-4-index-liver-fibrosis)
  - [Age (years) x AST (U/L)] ÷ [platelet count ($10^9$/L) x $\sqrt{\text{ALT (U/L)}}$]
    - ≥1.30 (aged 36–64 years) or ≥2.0 (aged >65 years) require further assessment; >2.67 is high risk
- ELF score – 2nd line test
  - Hyaluronic acid (HA)
  - Procollagen III amino-terminal peptide (PIINP)
  - Tissue inhibitor of metalloproteinaise 1 (TIMP-1)
    - < 9.8 – lower risk of disease progression, ≥9.8 requires specialist referral
- Other scores: NFS, APRI, FAST, MAST, MEFIB index
- Imaging
  - Liver ultrasound
  - FibroScan/elastography
    - LSM ≥8 kPa is intermediate-high risk of liver fibrosis in MASLD and warrants specialist input
  - MRI liver or liver biopsy if unclear aetiology

Abbreviations: ALT, alanine aminotransferase; APRI, Aspartate aminotransferase: Platelet ratio index; ARV, antiretrovirals; AST, aspartate aminotransferase; ELF, enhanced liver fibrosis; FAST, FIB-4 + AST; FIB-4, fibrosis-4 score; HbA1C, haemoglobin A1C (glycated); LFT, liver function test; LSM, liver stiffness measure, MAST, MRI + AST; MEFIB index, magnetic resonance elastography + FIB-4; MRI, magnetic resonance imaging; NFS, NAFLD fibrosis score; OSA, obstructive sleep apnoea; PCOS, polycystic ovaries syndrome; T2D, type 2 diabetes mellitus.

### *Management of T2D in the context of MASLD*

Multidisciplinary primary and secondary care is required for effective management. Referral to specialists while monitoring for diabetic and hepatic complications such as nephropathy, microangiopathy and HCC provides holistic care. The move towards multidisciplinary MASLD clinics is on the horizon, though this would be a resource-rich development. Managing contributing cardiometabolic risk factors, such as hypertension, T2D and obesity, is essential.

### *Lifestyle modification*

Weight loss is a hallmark of management and with 5–10% loss, there is a reduction in liver stiffness, hepatic steatosis, insulin sensitivity as well as normalisation of liver function tests.[18,19] Dietician input can help instil specific nutritional strategies and a hypocaloric diet. This includes a high protein intake (1.5 g per kg body weight), alcohol abstinence (especially with significant fibrosis), minimal saturated fats and reduced refined carbohydrates. Sports and exercise specialists can introduce manageable physical activity. Both changes reduce visceral fat and hyperglycaemia, leading to a resolution of steatohepatitis.[18,19]

### *Metformin*

Metformin increases insulin sensitivity and is gold-standard first-line T2D therapy. There is scarce evidence to show direct histological improvement of MASLD, so it is not used as a targeted therapy.[18,19]

### *Thiazolidinediones*

Pioglitazone is an effective treatment for MASLD, with strong evidence in reduction of histological steatosis and improvement of IR, especially when combined with a hypocaloric diet. Pioglitazone can cause fluid retention and weight gain, so caution is required in patients with co-existing heart failure.[18,19]

### *Sodium glucose cotransporter-2 inhibitors (SGLT-2i)*

A modest reduction of hepatic steatosis (monitored with MRI) has been observed with dapagliflozin (EFFECT-II) and empagliflozin (E-LIFT).[19,20] Dapagliflozin monotherapy reduces hepatocyte injury and liver biomarkers, independent of changes in HbA1c and weight.[19,20] The common risks of SGLT-2i include euglycaemic diabetic ketoacidosis and urinary tract infections,[18] due to the increased renal excretion of glucose.

### *Glucagon-like peptide-1 receptor agonists (GLP-1 RA)*

These incretin mimetics enhance insulin action, have potent anorectic effects and reduce weight. Semaglutide (Ozempic/Wegovy/Rybelsus) also improves steatohepatitis (without worsening in fibrosis); however, there is limited evidence to suggest an improvement in fibrosis.[21,22] The ESSENCE trial (2025) showed an improvement in fibrosis in 36.8% of patients taking weekly semaglutide injections (mostly with T2D) vs 22.4% on placebo.[22] This has led to Food and Drug Administration (FDA) approval of using semaglutide in adults with biopsy-proven MASH and stage F2/F3 fibrosis.[22]

The emergence of dual GLP-1 and glucose-dependent insulinotrophic polypeptide (GIP) receptor agonist, tirzepatide (Mounjaro), has shown an improvement in MASLD-related biomarkers and hepatic steatosis (SURPASS-3).[23] Recent trials (SYNERGY-NASH) showed improvement in histology, with no fibrosis worsening and with MASH resolution in up to 62% of individuals taking 15 mg, the highest dose. This is compared to 10% of patients taking placebo.[24]

There is scope for GLP-1 RAs to manage co-existing T2D and MASLD, targeting a reduction in weight, glycaemic control and IR[21,22,25] as well as possible reduction of HCC incidence.[26] Insulin is an effective diabetes medication that does not offer the same specific improvements on liver disease.[20,23] There is hope to further explore the potential of GLP-1 RAs and consider triple receptor agonist therapy (GLP-1, GIP, glucagon) to counteract undesired effects.[26] Another combination therapy, cagrilintide–semaglutide (CagriSema), uses the incretin effect. This combines an amylin analogue and GLP-1 RA. Early studies show promising improvements with weight loss superior to monotherapy and a greater reduction in HbA1C compared to cagrilintide alone.[27] Ongoing studies will need to look at hepatic disease endpoints.

*Bariatric surgery*

Bariatric surgery can resolve MASH when >10% weight loss is achieved.[18,23] Meta-analyses show improved liver histology, MASH resolution (85% of patients) and reduced fibrosis; 12% of patients had worsening fibrosis.[28] Surgery can induce diabetes remission and is typically a sustainable intervention for weight loss. Sleeve gastrectomy is recommended over a gastric bypass with patients with cirrhosis due to the risk of decompensation.[19]

*Antifibrotic agents*

Resmetirom, an FDA-approved, selective thyroid hormone receptor beta agonist, has demonstrated fibrosis improvement and histological MASH resolution in phase three trials (MAESTRO-NASH).[29] Emerging therapies include fibroblast growth factor (FGF) 21 analogues (eg efruxifermin and pegozafermin), which have demonstrated histological liver fibrosis improvement without worsening of MASH and with reduction in serum ALT and ELF score (HARMONY trial).[30] The focus of these studies has been regarding MASLD treatment and not specific to patients with T2D.

## Consequences of liver disease on diabetes management

Cirrhosis is the irreversible state of advanced liver disease (Fig. 2). The complexity in management includes nutritional challenges, variable glycaemic control and altered drug metabolism.[14] Reduced insulin clearance is commonly seen in patients with cirrhosis and there can be a higher rate of hypoglycaemia, mortality and cardiovascular events compared with patients who do not use insulin.[14]

It is a challenge to manage suboptimal T2D, due to reduced hepatic counterregulatory mechanisms in glucose metabolism as well as the hepatic metabolisation of standard medications. Metformin can contribute to lactic acidosis and sulfonylureas can cause hypoglycaemia. They should both be avoided in decompensated cirrhosis, due to reduced lactate clearance and a reduced ability to counteract hypoglycaemia.[11,14,16] SGLT-2 inhibitors, dipeptidyl peptidase-4 (DPP-4) inhibitors and GLP-1 RAs are generally considered safe in compensated cirrhosis, as they involve limited hepatic metabolism.[14] Dietary restrictions can worsen sarcopenia seen in patients with cirrhosis; however, a high protein intake is of value.

## Conclusion

Metabolic syndrome, T2D and MASLD are closely linked. Early identification of the hepatic manifestation of IR is important for the management of MASLD. Clear communication with patients and specialists can lead to targeted therapy, which improves outcomes including glycaemic control, weight, cardiovascular risk factors and HCC risk. Management of DM is a key part of reducing the risk for developing advanced liver disease and there is hope for novel pharmaceutical agents to manage co-existing disease.

## Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

J. Makaronidis reports financial support was provided by the National Institute for Health and Care Research (NIHR). J. Makaronidis reports financial support was provided by the Society for Endocrinology. J. Makaronidis reports institutional financial support was provided by Novo Nordisk. J. Makaronidis reports institutional financial support was provided by Rhythm Pharmaceuticals Inc. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## CRediT authorship contribution statement

**Ebo Dadey:** Writing – review & editing, Writing – original draft. **Tiong Yeng Lim:** Writing – review & editing. **Janine Makaronidis:** Writing – review & editing.

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