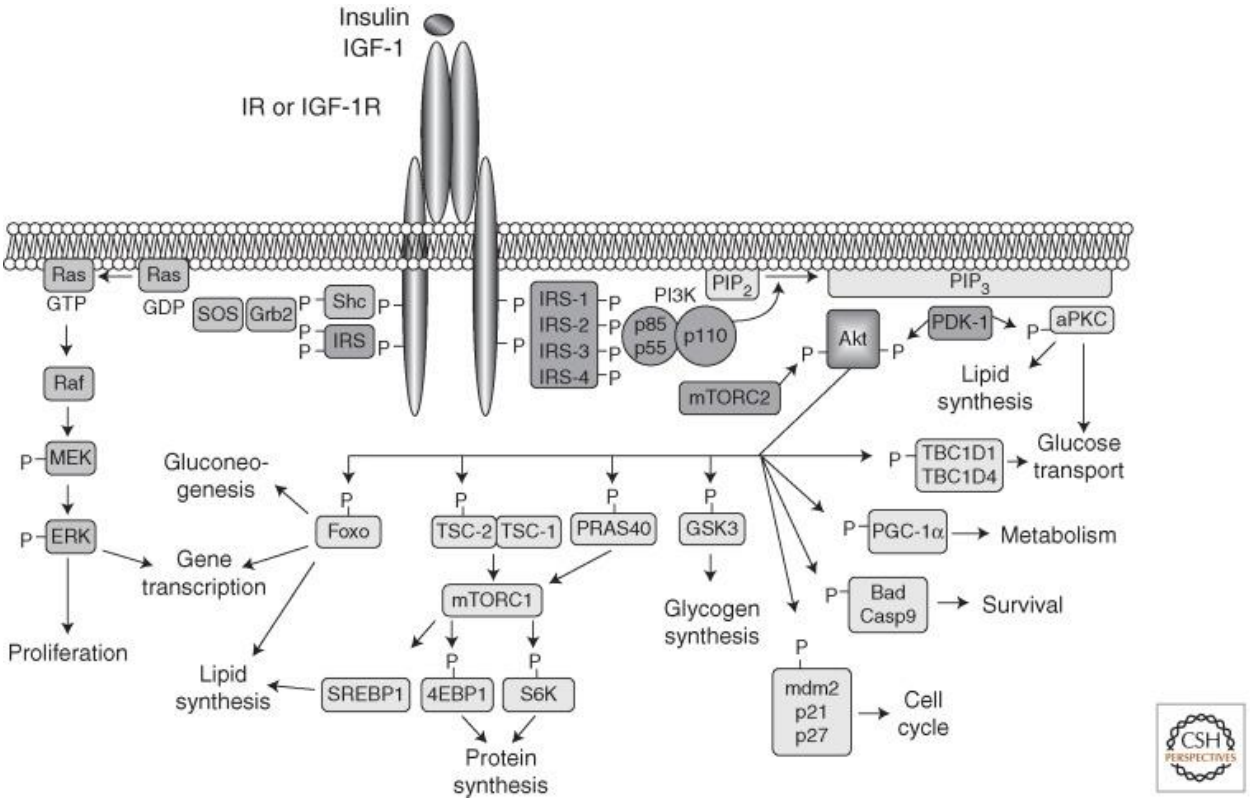
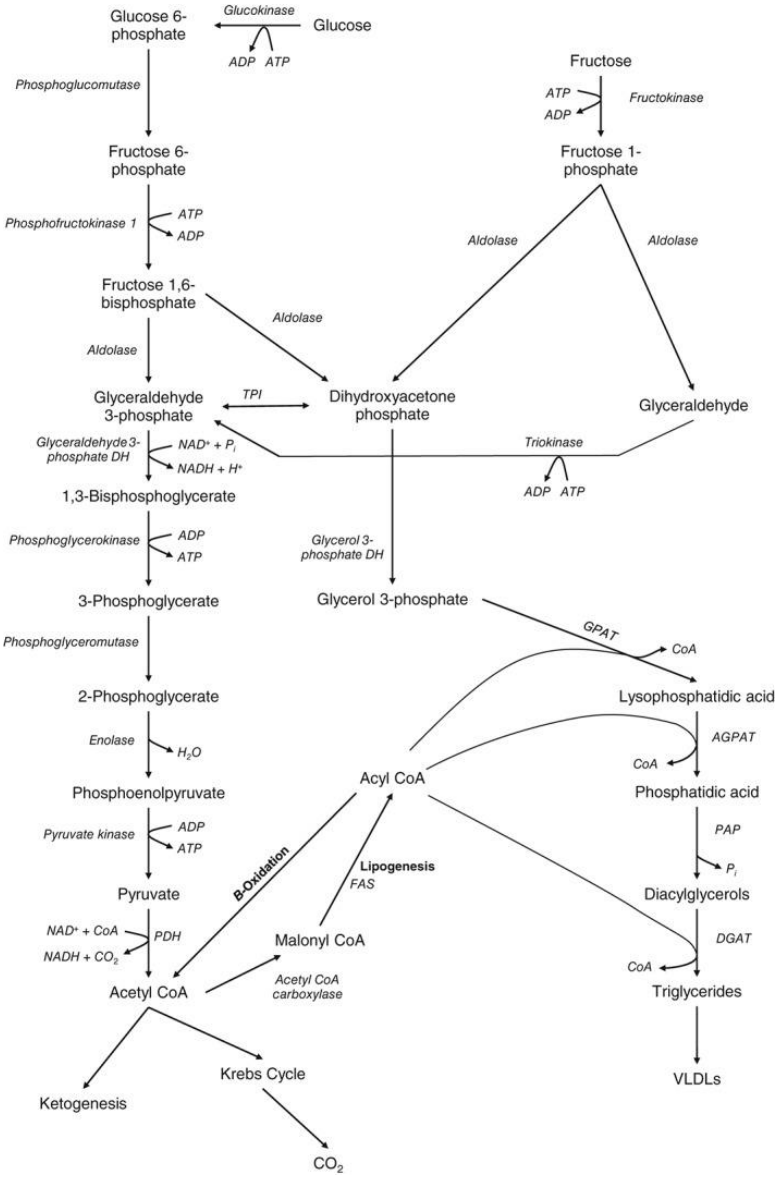


- **Supplementary figure 1: Insulin- and IGF-1-signaling pathways.**
- Activation of insulin and IGF-1 receptors by their ligands initiates a cascade of phosphorylation events. A conformational change and autophosphorylation of the receptors occur at the time of ligand binding, leading to the recruitment and phosphorylation of receptor substrates such as IRS and Shc proteins. Shc activates the Ras-MAPK pathway, whereas IRS proteins mostly activate the PI3K-Akt pathway by recruiting and activating PI3K, leading to the generation of second messenger PIP_3. Membrane-bound PIP_3 recruits and activates PDK-1, which phosphorylates and activates Akt and atypical PKCs. Akt mediates most of insulin's metabolic effects, regulating glucose transport, lipid synthesis, gluconeogenesis, and glycogen synthesis. Akt also plays a role in the control of cell cycle and survival. The Shc-Grb2-Sos-Ras-Raf-MAPK pathway controls cellular proliferation and gene transcription.

Adapted with permission from Boucher J, Kleinridders A, Kahn CR. Insulin receptor signaling in normal and insulin-resistant states. Cold Spring Harb Perspect Biol. 2014 Jan 1;6(1): a009191. doi: 10.1101/cshperspect.a009191. PMID: 24384568; PMCID: PMC3941218.

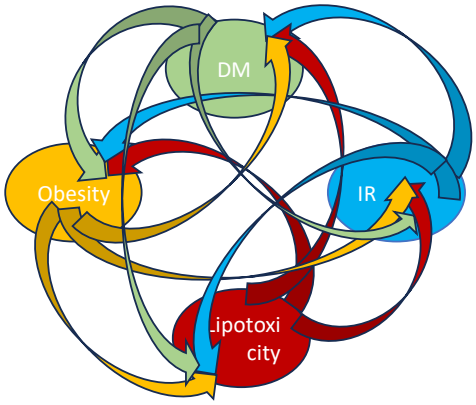


- **Supplementary figure 2: Hepatic glycolysis, fructolysis and de novo lipogenesis (DNL).**

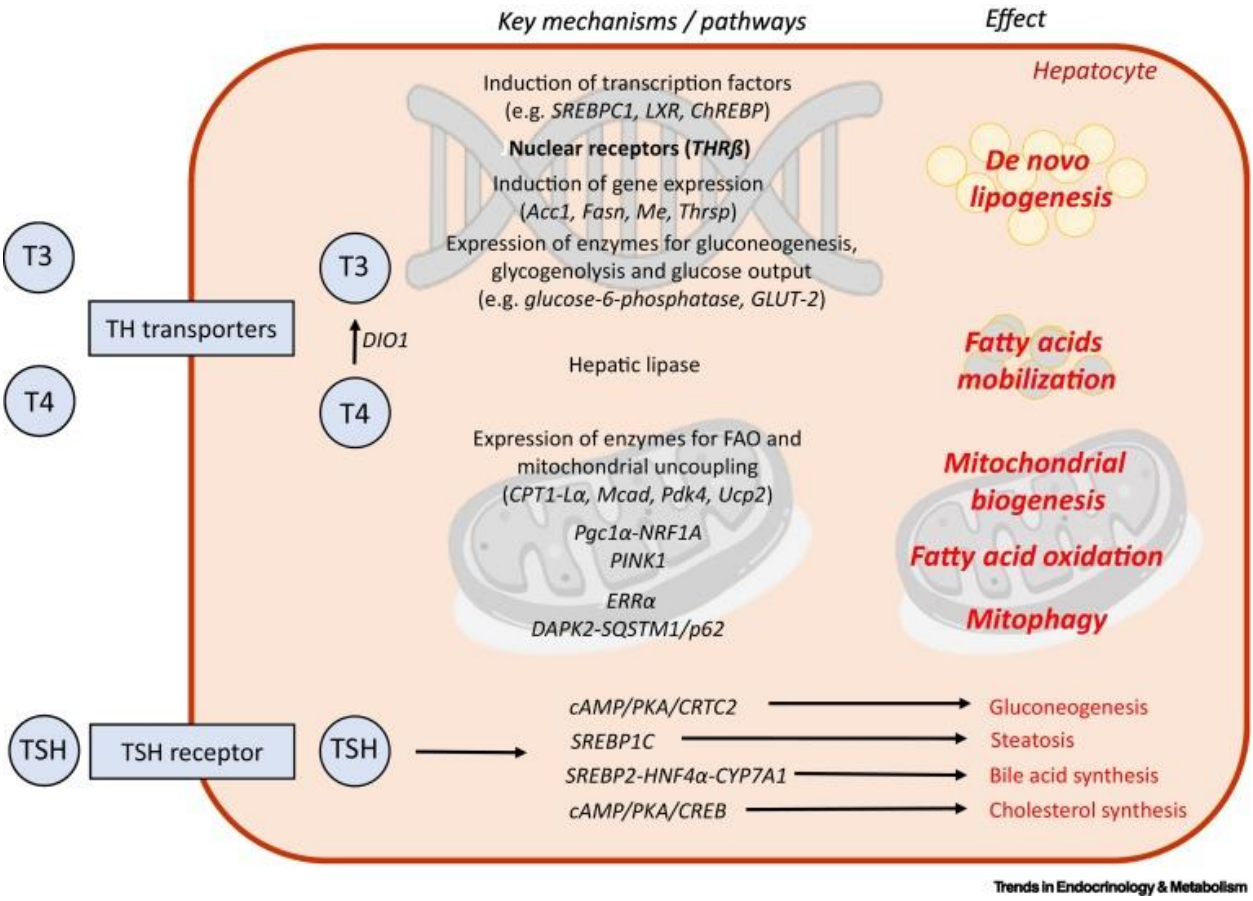
- Glucose undergoes the multistep process of glycolysis to generate pyruvate which is subsequently utilized for a range of cellular metabolic processes. This includes complete oxidative catabolism to carbon dioxide, lipogenesis under the catalysis of fatty acid synthase (FAS), and ketogenesis to generate ketone bodies for oxidative metabolic fuel. The fructolytic pathway feeds into the glycolytic pathway at the level of the trioses: dihydroxyacetone phosphate and

glyceraldehyde 3-phosphate. This thus bypasses the crucial regulatory steps in liver glycolytic metabolism, whereby glucokinase and phosphofructokinase-1 are controlled by the metabolic milieu in coordination with hormonal effectors. This allows for the production of acetyl-CoA from fructose to continue without regulation by insulin, and hence promote lipogenesis, and subsequent triglyceride (TG) synthesis from the acyl-coenzyme A (CoA) product. This involves catalysis of progressive acylation of a glycerol-phosphate backbone by glycerol-phosphate acyl transferase (GPAT), acylglycerol-phosphate acyl transferase (AGPAT). Dephosphorylation of the glycerol backbone then occurs under the catalysis of phosphatidic acid phosphorylase (PAP), followed by a further acylation of the diacylglycerol by diacylglycerol acyl transferase (DGAT). The TGs produced are packed into very low-density lipoproteins (VLDLs) for export from the liver or are stored within hepatocytes. DH, dehydrogenase; NAD^+, nicotine adenine dinucleotide; NADH, reduced form of NAD^+; PDH, pyruvate dehydrogenase; TPI, triose phosphate isomerase.

Adapted with permission from Sanders FW, Griffin JL. De novo lipogenesis in the liver in health and disease: more than just a shunting yard for glucose. Biol Rev Camb Philos Soc. 2016 May;91(2):452-68. doi: 10.1111/brv.12178. Epub 2015 Mar 4. PMID: 25740151; PMCID: PMC4832395.



Supplementary figure 3: Inter-relationship between risk factors and MASLD



Supplementary figure 4: Molecular actions of thyroid hormones in the hepatocyte.

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