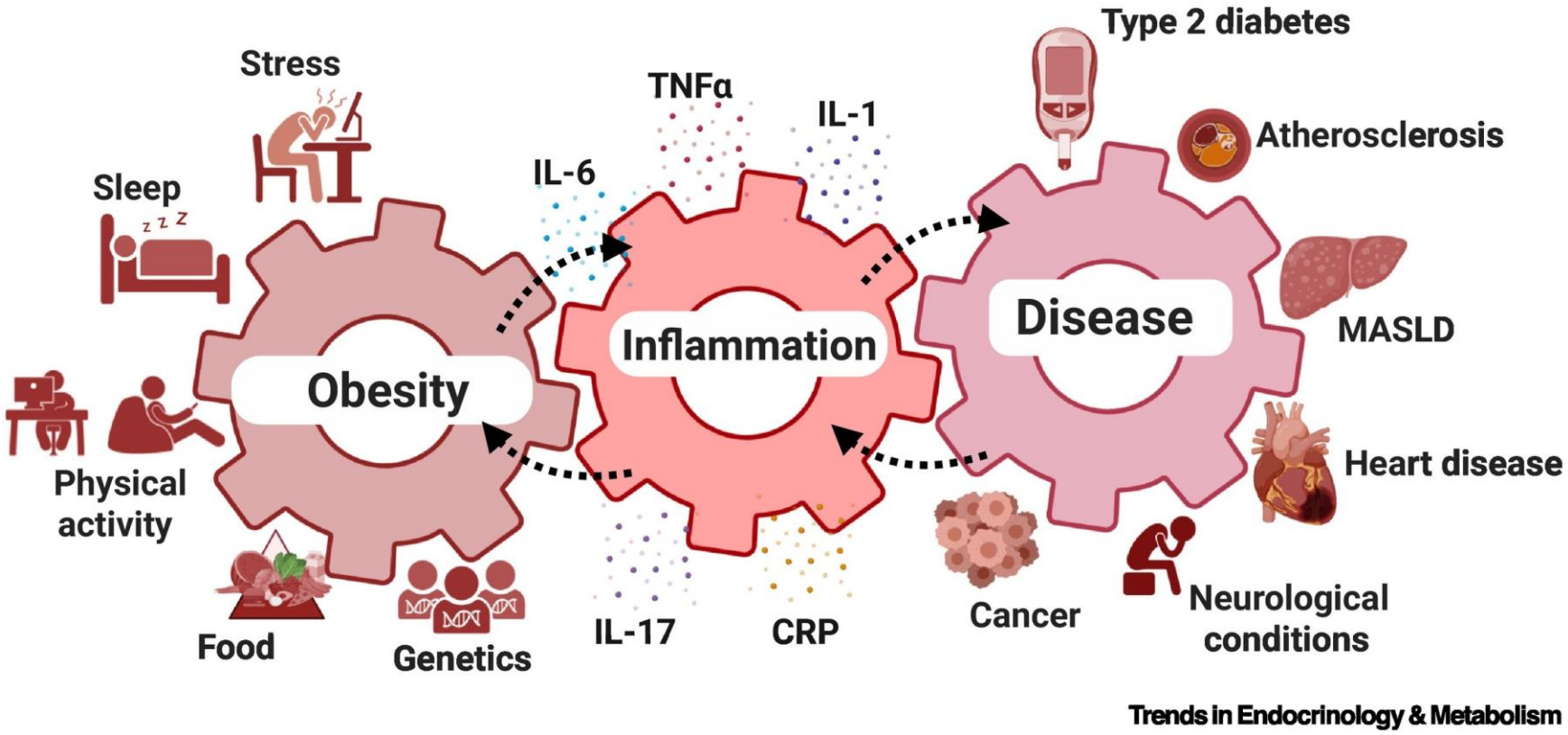


https://www.freepik.com/premium-vector/stopwatch-icon-45-minutes-timer-vector-graphics_63416291.htm

Education is not a singular inoculation



A change of science is a foot.....



…thus a change of GP LTC review <u>infrastructure</u> is a must

Hogan AE, Davis C, Jenkins BJ, Jones N, O'Shea D. Repurposing metabolic drugs as anti-inflammatory agents. Trends Endocrinol Metab. 2025 Aug 13:S1043-2760(25)00149-3

GENERAL PRACTICE CARDIOVASCULAR-KIDNEY - METABOLIC REVIEW

THE NEW LONG TERM CONDITION FRAMEWORK OF CARE FOR OUR HOUSES OF CARE

Dr Eimear Darcy

General Practitioner

15th January 2025

Dr Eimear Darcy

NHS GP Partner

MB BCH BAO DMH MRCGP (2017)

PG Cert Diabetes (Distinction)

SCOPE certified in Obesity Management

General Practitioner specialising in CKM



LEARNING OBJECTIVES

OFFICIALLY..

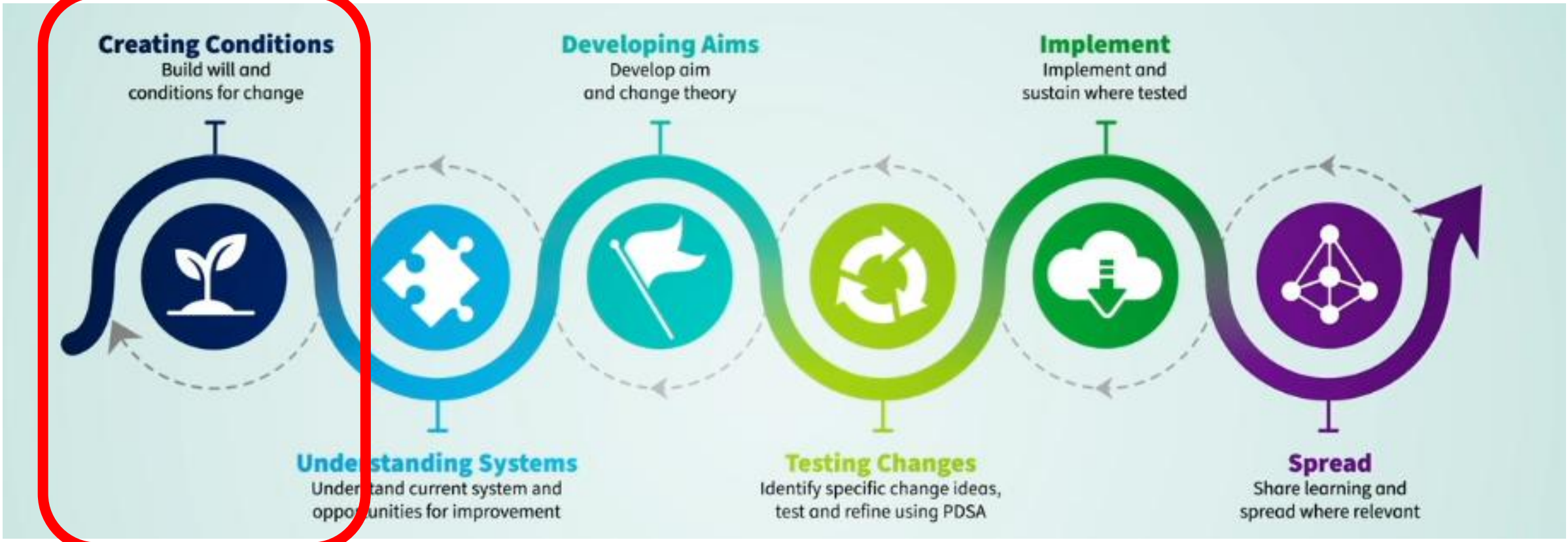
- Insights into modern management of General Practice Long Term Condition Care

- In the context of the disease(s) of Obesity and the broader Cardiovascular – Kidney – Metabolic picture

PERSONALLY…



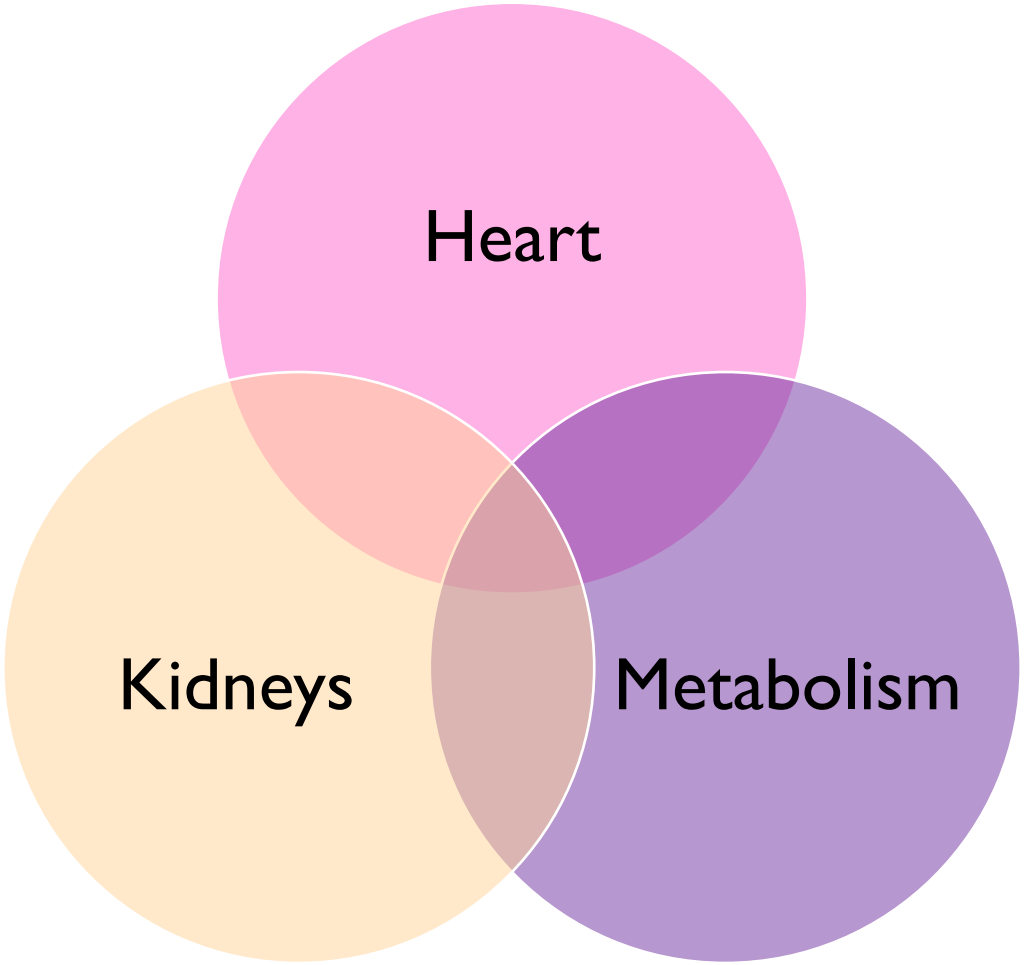
Lets build will and conditions for change





Images taken from TURAS

CVRM = CKM = CAREME/CRM



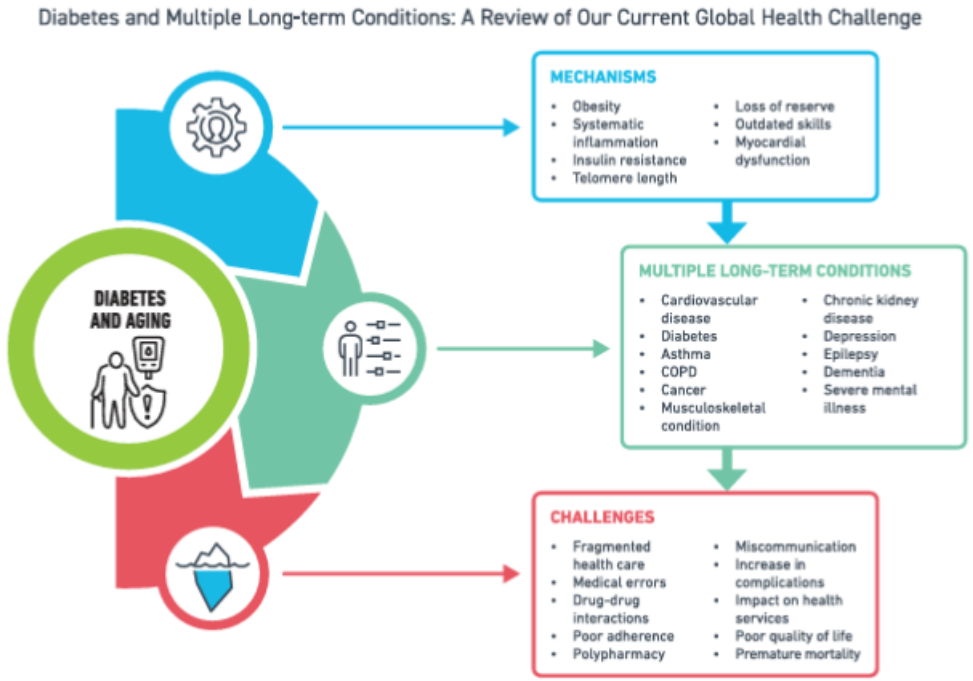
MULTIPLE LONG TERM CONDITIONS (MTLCS)

Diabetes Care
American Diabetes Association.

Diabetes and Multiple Long-term Conditions: A Review of Our Current Global Health Challenge

Kamlesh Khunti, Yogini V. Chudasama, Edward W. Gregg, Monika Kamkuemah, Shivani Misra, Jerry Suls, Nikhil S. Venkateshmurthy, and Jonathan Valabhji

Diabetes Care 2023;46(12):2092–2101 | https://doi.org/10.2337/dci23-0035



Received: 17 March 2020 | Revised: 15 April 2020 | Accepted: 29 April 2020
DOI: 10.1111/dom.14074

ORIGINAL ARTICLE

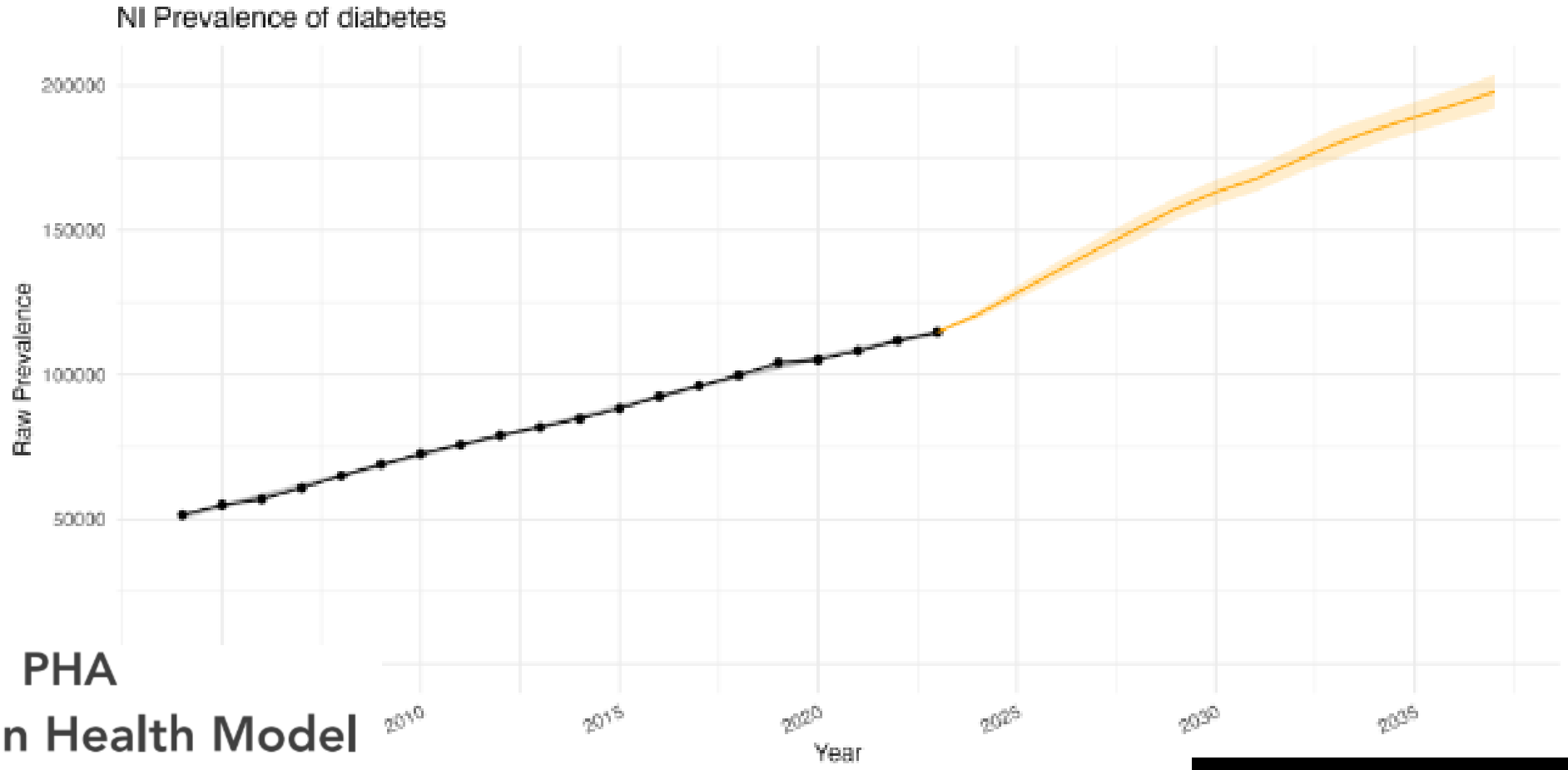
WILEY

Heart failure and chronic kidney disease manifestation and mortality risk associations in type 2 diabetes: A large multinational cohort study

Conclusion: In a large multinational study of >750 000 CVRD-free patients with T2D, HF and CKD were consistently the most frequent first cardiovascular disease manifestations and were also associated with increased mortality risks. These novel findings show these cardiorenal diseases to be important and serious complications requiring improved preventive strategies.

Khunti K, Chudasama YV, Gregg EW, Kamkuemah M, Misra S, Suls J, et al. Diabetes and multiple long-term conditions: A review of our current global health challenge. Diabetes Care. 2023;46(12):2092–101.
Birkeland KI, Bodegard J, Eriksson JW, Norhammar A, Haller H, Linssen GCM, Banerjee A, Thuresson M, Okami S, Garal-Pantaler E, Overbeek J, Mamza JB, Zhang R, Yajima T, Komuro I, Kadowaki T. Heart failure and chronic kidney disease manifestation and mortality risk associations in type 2 diabetes: A large multinational cohort study. Diabetes Obes Metab. 2020 Sep;22(9):1607-1618

PROJECTIONS OF NON COMMUNICABLE DISEASE PREVALENCE NI PRODUCED FOR SPPG JULY 2025

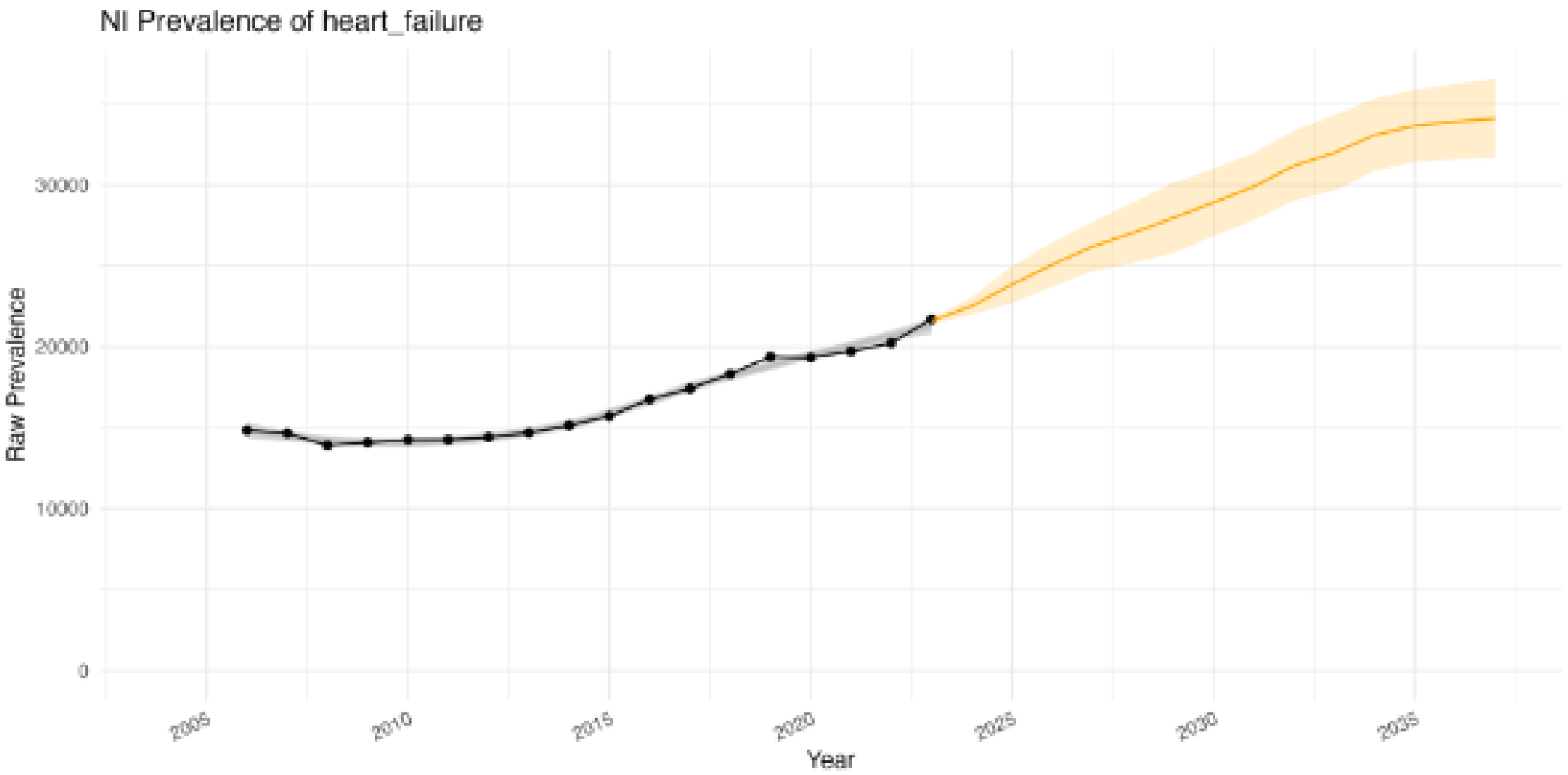


PHA

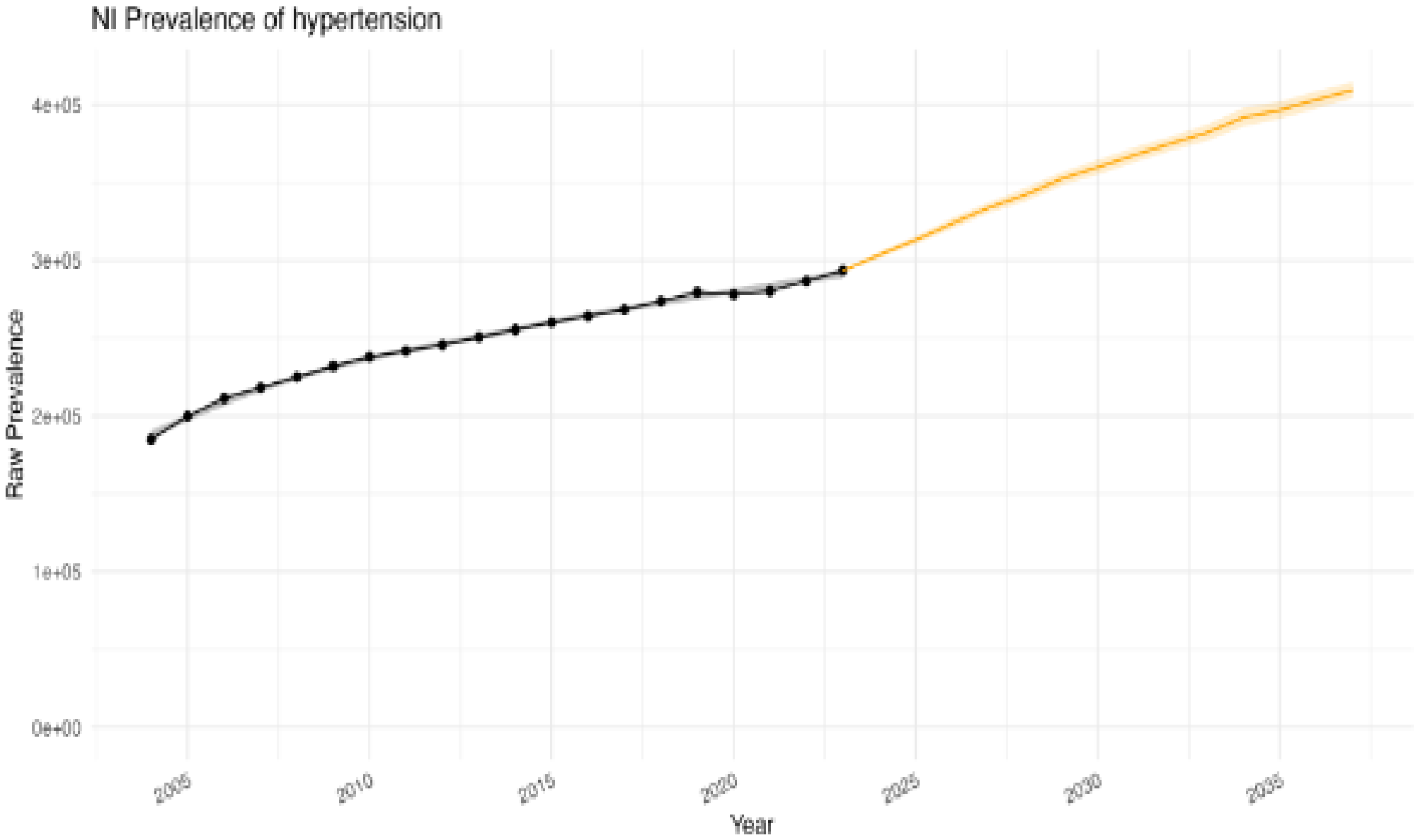
Population Health Model Projections

Produced for SPPG, July 2025

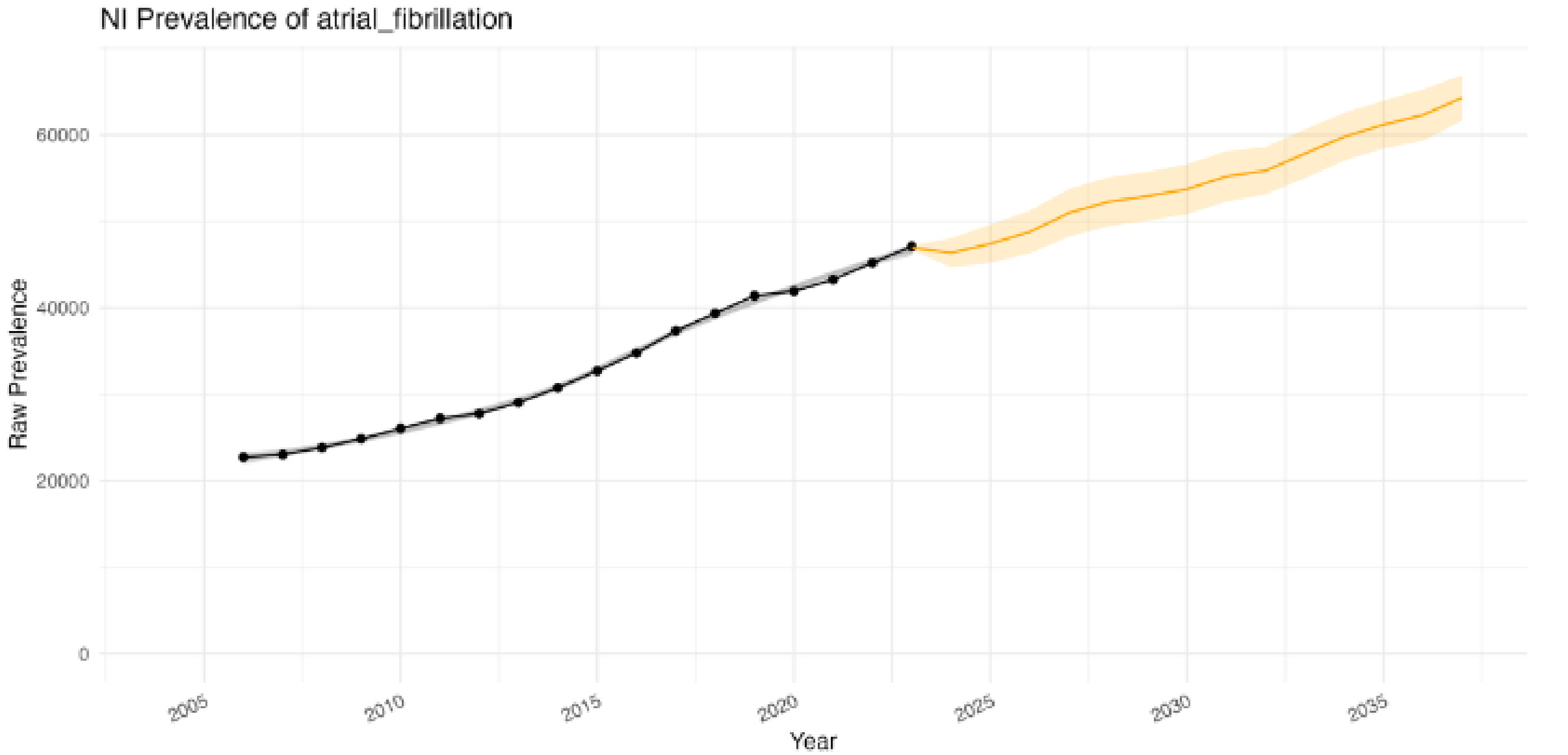
PHM Population Health Modelling
Northern Ireland Public Health Agency



PHM Population Health Modelling
Northern Ireland Public Health Agency

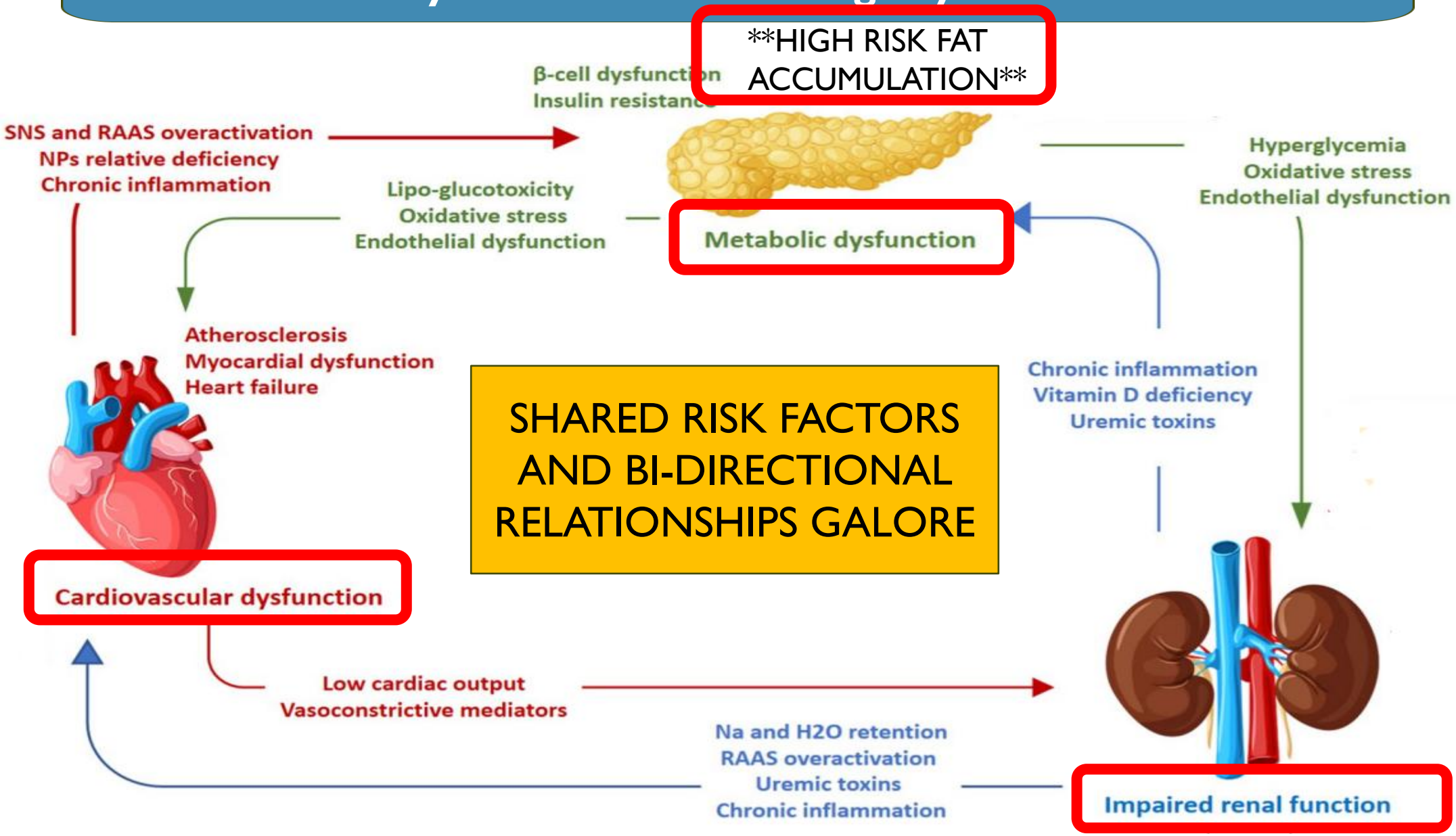


PHM Population Health Modelling
Northern Ireland Public Health Agency



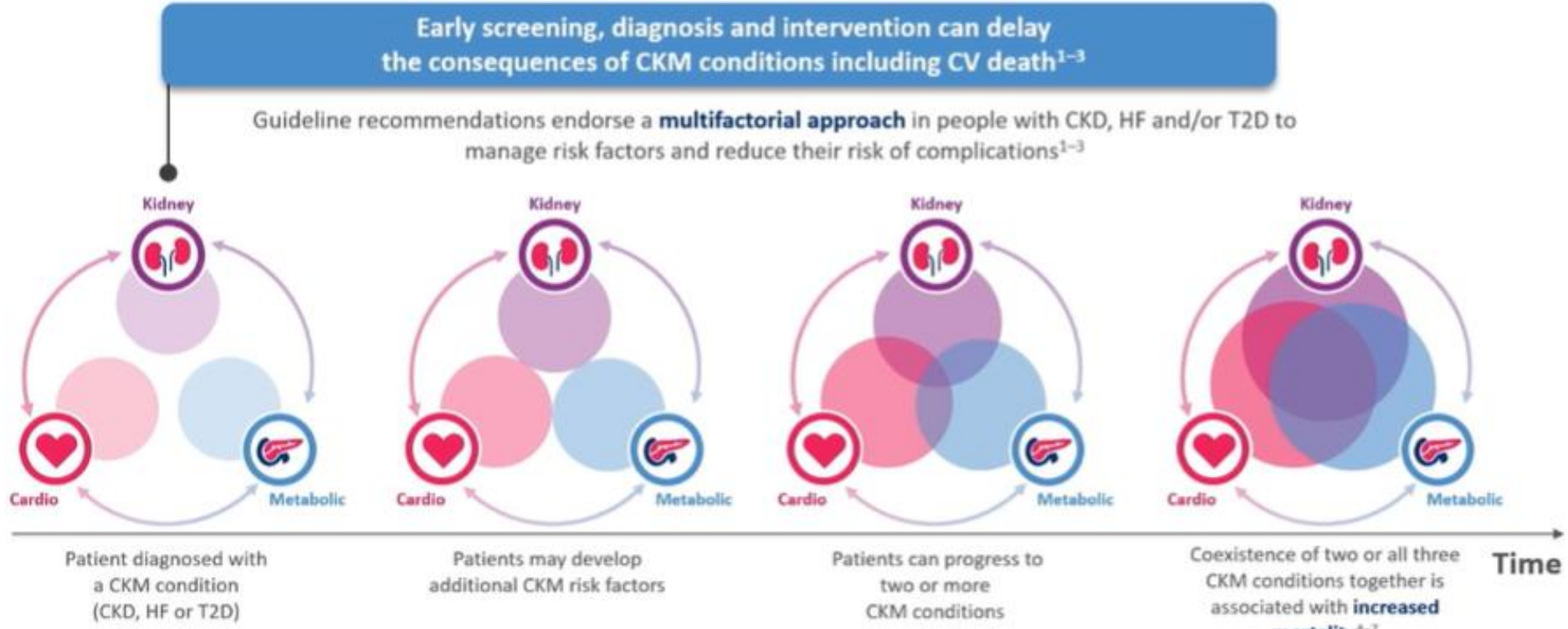
PHM Population Health Modelling
Northern Ireland Public Health Agency

CKM syndrome - Groundhog day for disease



Adapted from Marassi, M., Fadini, G.P. The cardio-renal-metabolic connection: a review of the evidence. *Cardiovasc Diabetol* **22**, 195 (2023) [accessed 2/9/25]

Early and multifactorial intervention is the best way to help protect by reducing the amplifying risks associated with interconnected CKM diseases



CKD, chronic kidney disease; CRM, cardiovascular, renal and metabolic; CV, cardiovascular; HF, heart failure; T2D, type 2 diabetes

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. *Kidney Int* 2022;102:S1; 2. Heidenreich PA *et al. J Am Coll Cardiol* 2022;79:e263; 3. American Diabetes Association. *Diabetes Care* 2023;46:S1;
4. Birkeland KI *et al. Diabetes Obes Metab* 2020;22:1607; 5. Ather S *et al. J Am Coll Cardiol* 2012;59:998; 6. Afkarian M *et al. J Am Soc Nephrol* 2013;24:302; 7. Lovre D et al. *Endocrinol Metab Clin North Am.* 2018;47:237

Slide ownership attributed to Dr Alice Cheung

CARDIOVASCULAR–KIDNEY–METABOLIC SYNDROME

Health disorder due to connections amount

- Heart disease
- Kidney Disease
- Type Two Diabetes
- Obesity

Leading to poor health outcomes

Circulation
Volume 148, Issue 20, 14 November 2023; Pages 1606-1635
https://doi.org/10.1161/CIR.0000000000001184

American Heart Association.

AHA PRESIDENTIAL ADVISORIES

Cardiovascular-Kidney-Metabolic Health: A Presidential Advisory From the American Heart Association

CKM syndrome – medical definition

'…a systemic disorder characterised by pathophysiological interactions among **metabolic** risk factors, **CKD** , and **CV** system leading to multi-organ dysfunction and a high rate of adverse CV outcomes'.

Includes individuals:

At risk for CVD disease due to metabolic risk factors.

Those with CVD

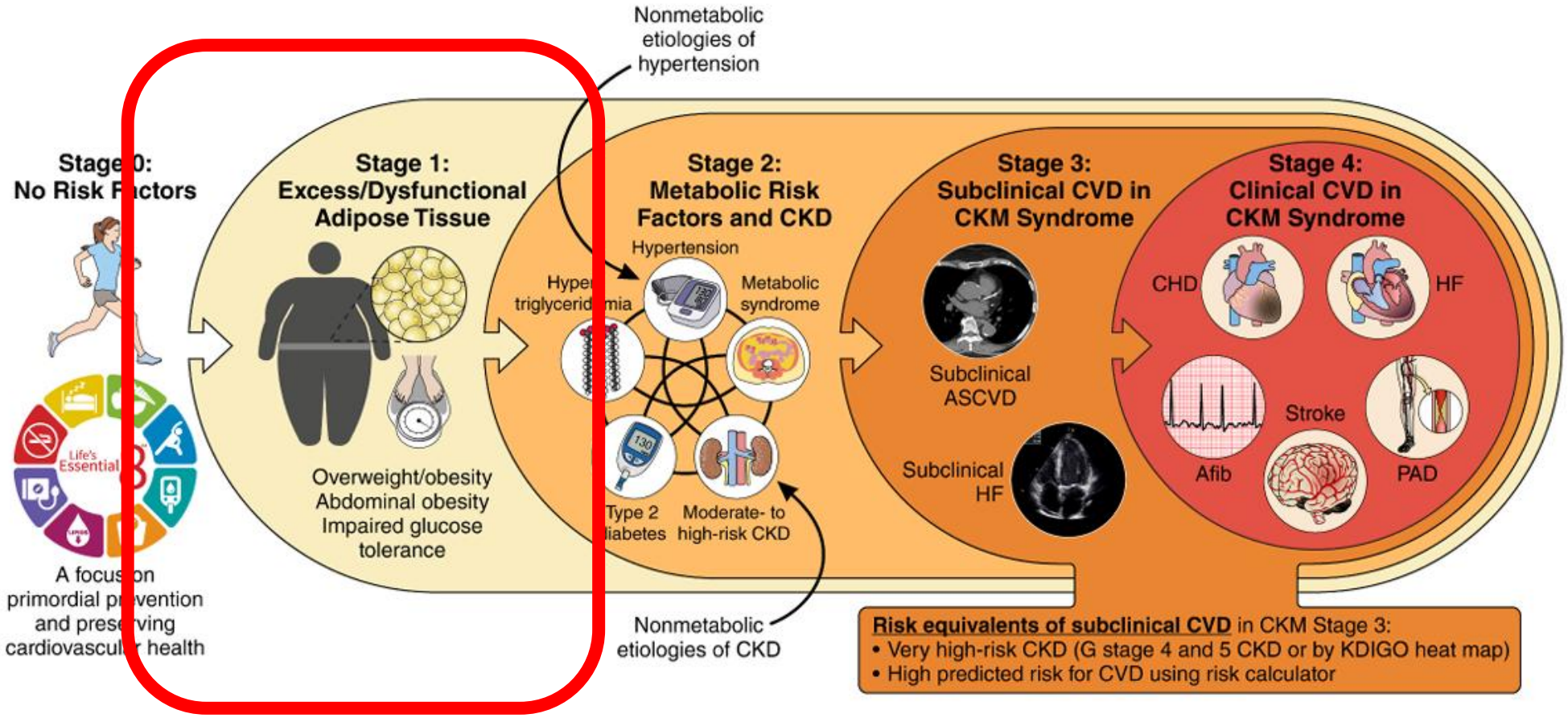
Those with CKD

Adapted from Ndumele CE, Neeland IJ, Tuttle KR, Chow SL, Mathew RO, Khan SS, et al. A synopsis of the evidence for the science and clinical management of cardiovascular-kidney-metabolic (CKM) syndrome: a scientific statement from the American Heart Association. Circulation. 2023;148(20):1636–64

General Practice CKM Review along the life journey

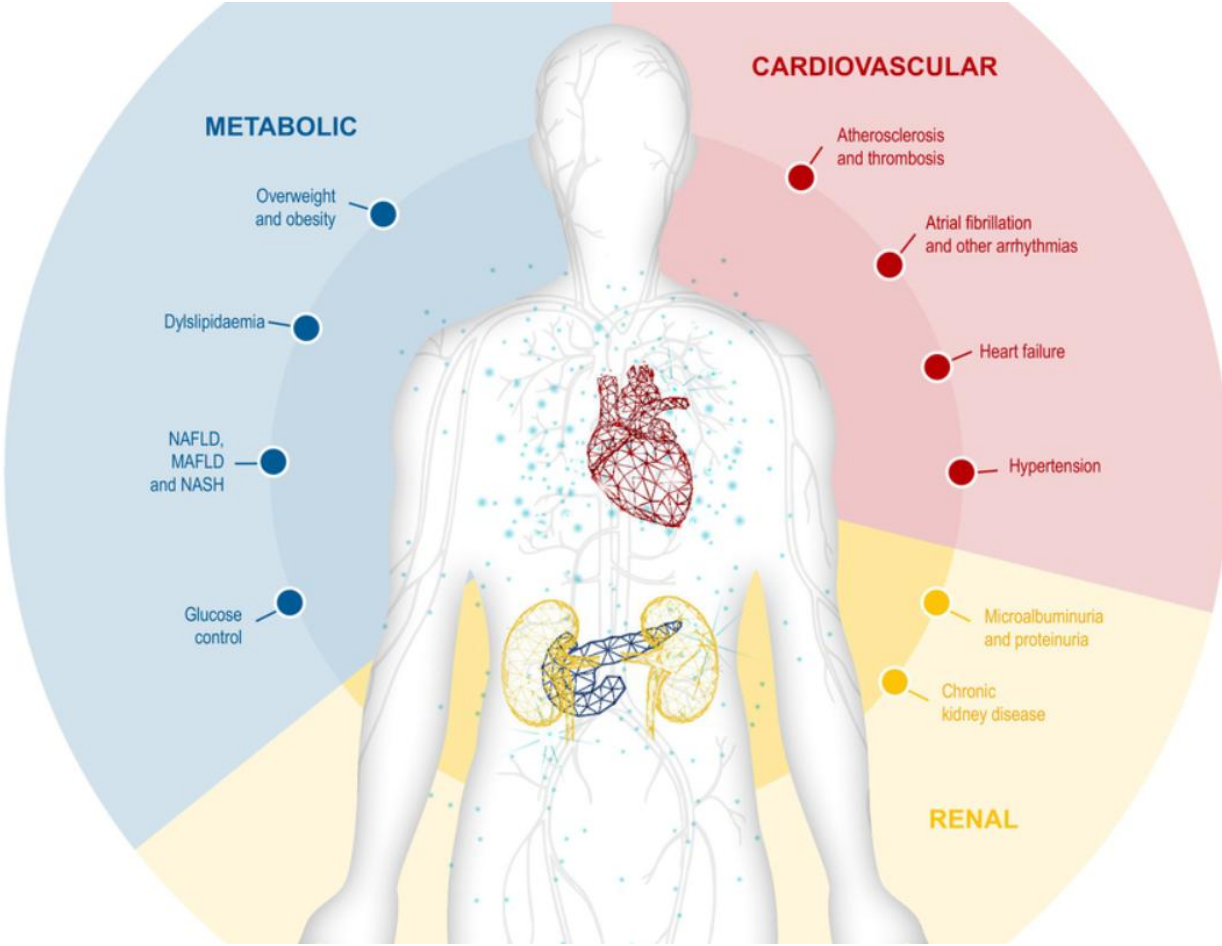


..AND IN THE CONTEXT OF THE PERSONS LIFE STORY



Every stage sits in General Practice

CKM – CARE OF PEOPLE , NOT JUST PATHOLOGY



Vora J, Cherney D, Kosiborod MN, Spaak J, Kanumilli N, Khunti K, et al. Inter-relationships between cardiovascular, renal and metabolic diseases: Underlying evidence and implications for integrated interdisciplinary care and management. Diabetes, Obesity and Metabolism. 2024;26(5):1567–81.

HOLISTIC IMPLEMENTATION

holistic

adjective

UK /həˈlɪs.tɪk/ US /hoʊlˈɪs.tɪk/

Add to word list

dealing with or treating the whole of something or someone and not just a part:

Cambridge Dictionary

ESC European Society of Cardiology
European Heart Journal (2025) 00, 1–29
https://doi.org/10.1093/eurheartj/ehaf314

Clinical staging to guide management of metabolic disorders and their sequelae: a European Atherosclerosis Society consensus statement

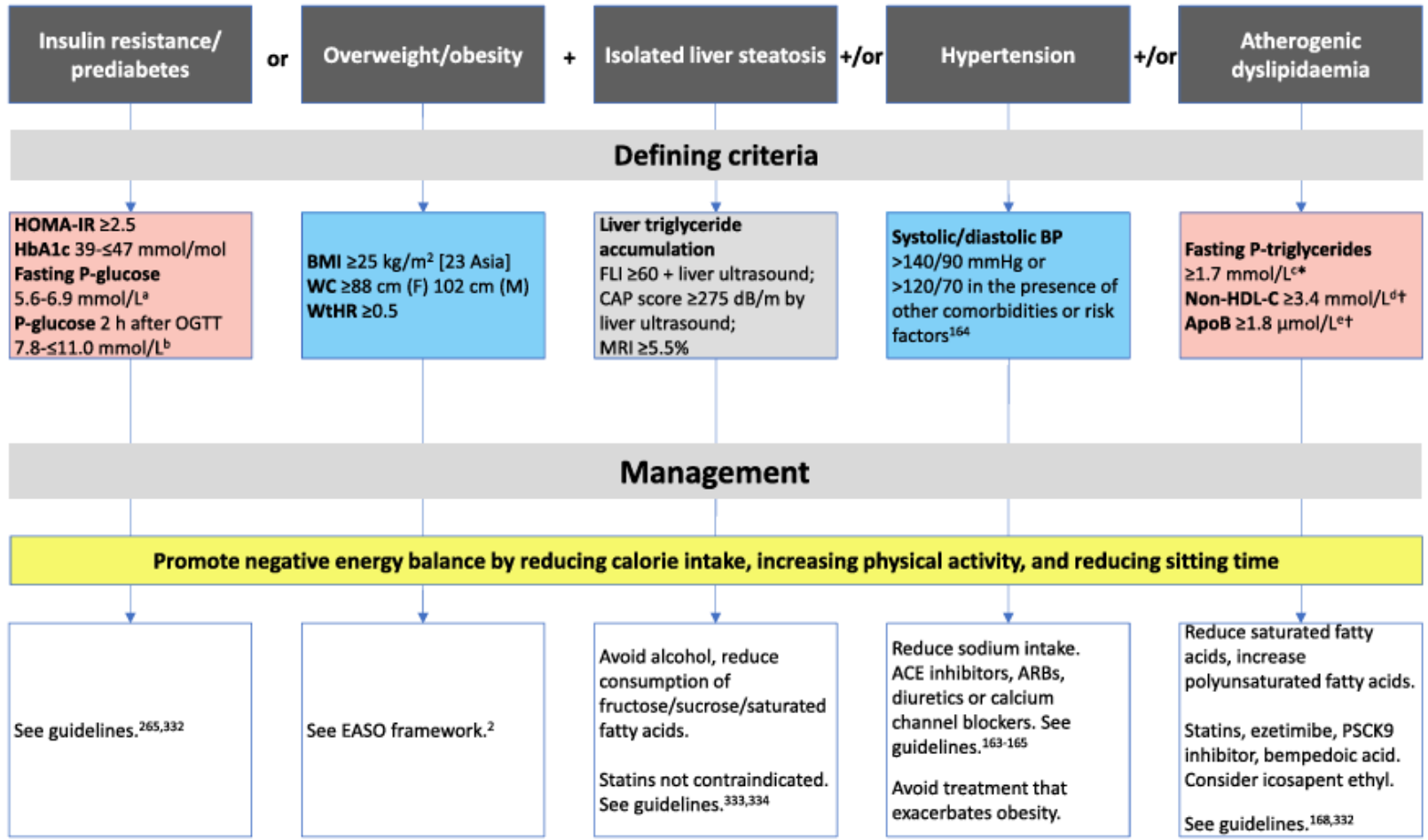


Figure 4 Defining criteria and potential management strategies for systemic metabolic disorder Stage 1. Conventional units: [a]101–124 mg/dL; [b]141 to ≤198 mg/dL; [c]150 mg/dL; [d]131 mg/dL: [e]100 mg/dL. *Non-fasting P-triglycerides ≥2.0 mmol/L (177 mg/dL). †Values shown for individuals at moderate risk of atherosclerotic cardiovascular disease. For those at high risk of atherosclerotic cardiovascular disease, lower thresholds apply: non–HDL-cholesterol ≥2.6 mmol/L (100 mg/dL); apolipoprotein B ≥1.5 µmol/L (≥80 mg/dL). P, plasma; WC, waist circumference; WtHR, waist-to-height ratio; EASO, European Association for the Study of Obesity; FLI, fatty liver index; CAP, controlled attenuation parameter; MRI, magnetic resonance imaging; BP, blood pressure; C, cholesterol; ACE, angiotensin-converting enzyme; ARB, angiotensin-II receptor blocker

communications medicine

Perspective

https://doi.org/10.1038/s43856-025-00742-9

Multiple long-term conditions as the next transition in the global diabetes epidemic

Check for updates

Edward W. Gregg[1,2], Naomi Holman[1,2,3], Marisa Sophiea[2], Shivani Misra[4,5], Jonathan Pearson-Stuttard[2,6], Jonathan Valabhji[3,5,7,8] & Kamlesh Khunti[8,9]

Several transitions, or new patterns and dynamics in the contributors and health outcomes, have altered the character and burden of the multi-decade, worldwide growth in prevalence of type 2 diabetes (T2DM). These changes have led to different needs for prevention and care. These dynamics have been driven by diverse demographic, socio-economic, behavioural, and health system response factors. In this Perspective, we describe these transitions and how their attributes have set the stage for multimorbidity, or multiple long-term conditions (MLTCs), to be the next major challenge in the diabetes epidemic. We also describe how the timing and character of these stages differ in high-, middle-, and low-income countries. These challenges call for innovation and a stronger focus on MLTCs across the spectrum of cause, effectiveness, and implementation studies to guide prevention and treatment priorities.

The global pandemic of type 2 diabetes (T2DM) is often viewed as a long-term by-product of the great epidemiologic transition, wherein in the previous century, human patterns of mortality and disease exchanged high rates of mortality due to infections and maternal and child mortality for lower mortality rates driven by chronic conditions such as cardiovascular disease (CVD), cancer, and T2DM[1,2]. However, there have been differing changes in incidence for specific chronic conditions. For example, deaths due to CVD decreased beginning in the 1970s and 1980s whereas T2DM, lacking effective policy or health care options for prevention, accelerated in the 1990s[2–5]. Although recent signs of a decrease in T2DM incidence in some countries have emerged[6,7], most countries of the world continue to experience unrelenting growth of the problem[7,8].

The T2DM pandemic has itself been complex and multi-phasic. Underlying the overall increases in prevalence, have been several transitions across distinct periods of the T2DM epidemic that impacted the burden and approaches to care, prevention, and research[9–12]. In this Perspective, we explore how these transitions have set the stage for multiple long-term conditions (MLTC, or multimorbidity) impacting morbidity, health services, and approaches to prevent T2DM.

Seeding and steady growth of the problem

The first phase of growth in the T2DM epidemic, evident in the 1970s and 1980s, consisted of steady, gradual, increases in prevalence in high income countries (HICs) and more rapid increases in selected low- and middle-income countries (LMICs) undergoing rapid urbanization, nutritional and other sociocultural changes associated with westernization[9,13–15] (Fig. 1). This was associated with a global nutrition transition, with dietary shifts toward refined carbohydrates, added sweeteners and edible oils, and away from legumes and fresh fruits and vegetables. Declining physical activity levels contributed further and collectively they are cited as driving an environment conducive to increases in obesity and T2DM[14,17]. However, where data were available, diagnosed diabetes prevalence rates were quite low, at around 2–4% in the US, UK, and Northern Europe[3,16,18] compared to recent global estimates, which have surpassed 10% of adults[1,8]. Further, this low diabetes prevalence contrasted with very high rates of severe complications, including a 20-fold increased rate of lower extremity amputation, 10 times the rate of end stage renal disease (ESRD), and three times the risk of CVD[19–26]. These high complication risks were attributed to suboptimal diabetes care, risk factors and self-management practices. For example, estimates from the some HICs in the mid-1990s showed that large segments of the population with diagnosed diabetes had HbA1c levels well over 9% (75 mmol/mol) and blood pressure levels > 140/80, levels considered to be a sign of inadequate management by current standards[27–29]. Thus, perception of the problem centred more on the complications accompanying T2DM rather than the incidence and potential prevention of T2DM.

[1]School of Population Health, RCSI University of Medicine and Health Sciences, Dublin, Ireland. [2]School of Public Health, Imperial College London, London, UK. [3]NHS England, Wellington House, London, UK. [4]Department of Diabetes and Endocrinology, St Mary's Hospital, Imperial College Healthcare NHS Trust, London, UK. [5]Division of Metabolism, Digestion & Reproduction, Faculty of Medicine, Imperial College London, London, UK. [6]Lane Clark & Peacock LLP, London, UK. [7]Chelsea & Westminster Hospital NHS Foundation Trust, London, UK. [8]Diabetes Research Centre, University of Leicester, Leicester, UK. [9]These authors contributed equally: Jonathan Valabhji, Kamlesh Khunti. e-mail: edwardgregg@rcsi.ie

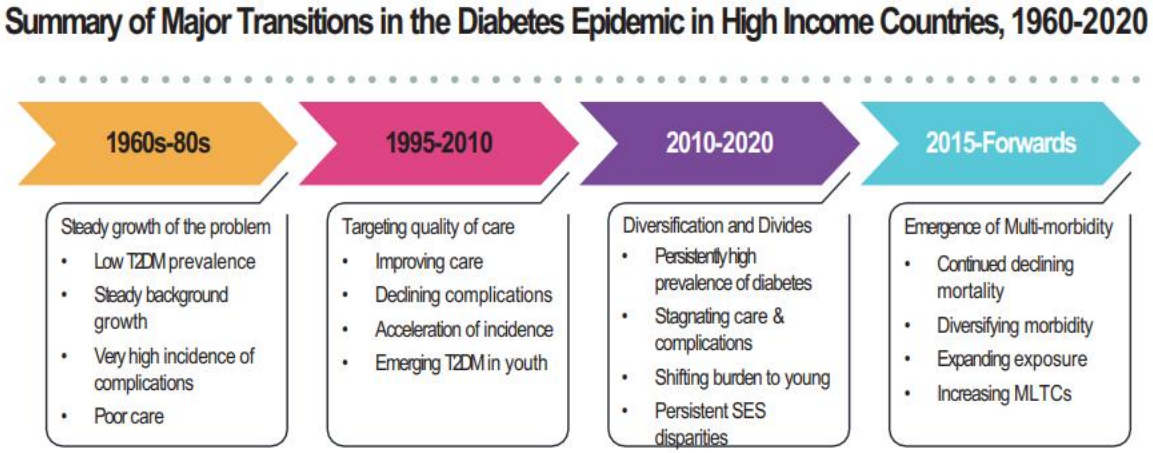


Fig. 1 | Summary of major transitions in the global diabetes epidemic, as experienced by high income countries, 1960–2020. The figure describes key patterns in prevalence, incidnece, complications, care, and socio-demographic factors affecting the T2DM pandemic. T2DM: Type 2 diabetes mellitus; DM: diabetes mellitus; SES: socio-economic status; MLTC: multiple long-term conditions

'if MLTCs are the next major transition in the diabetes epidemic, and the key drivers lie at both ends of the age spectrum as we suggest, the most practical priorities will lie in prevention, ongoing or routine care models and coordination,

i.e **GENERAL PRACTICE, THE WIDER PRIMARY CARE…..and secondary care ongoing care.**

WHY ARE GENERAL PRACTITIONERS NATURAL CKM LEADERS?

THIS REVIEW INVOLVES .....

- **Whole person medical care.**

RCGP FEEL, AND WE KNOW.....

- GPs have distinct expertise and experience in providing **whole person medical care.** (1)

(1) Standing up for general practice Joanne Reeve, Gail Allsopp and Michael Mulholland British Journal of General Practice 2025; 75 (750): 4-5. **DOI:** https://doi.org/10.3399/bjgp25X740373

A General Practitioner's complex interventions counts in CKM care

"A GP is a doctor who is a consultant in general practice. GPs have distinct expertise and experience in providing whole-person medical care while managing the complexity, uncertainty, and risk associated with the continuous care they provide."

Table 1. The complex intervention that is general practice — an advanced generalist perspective

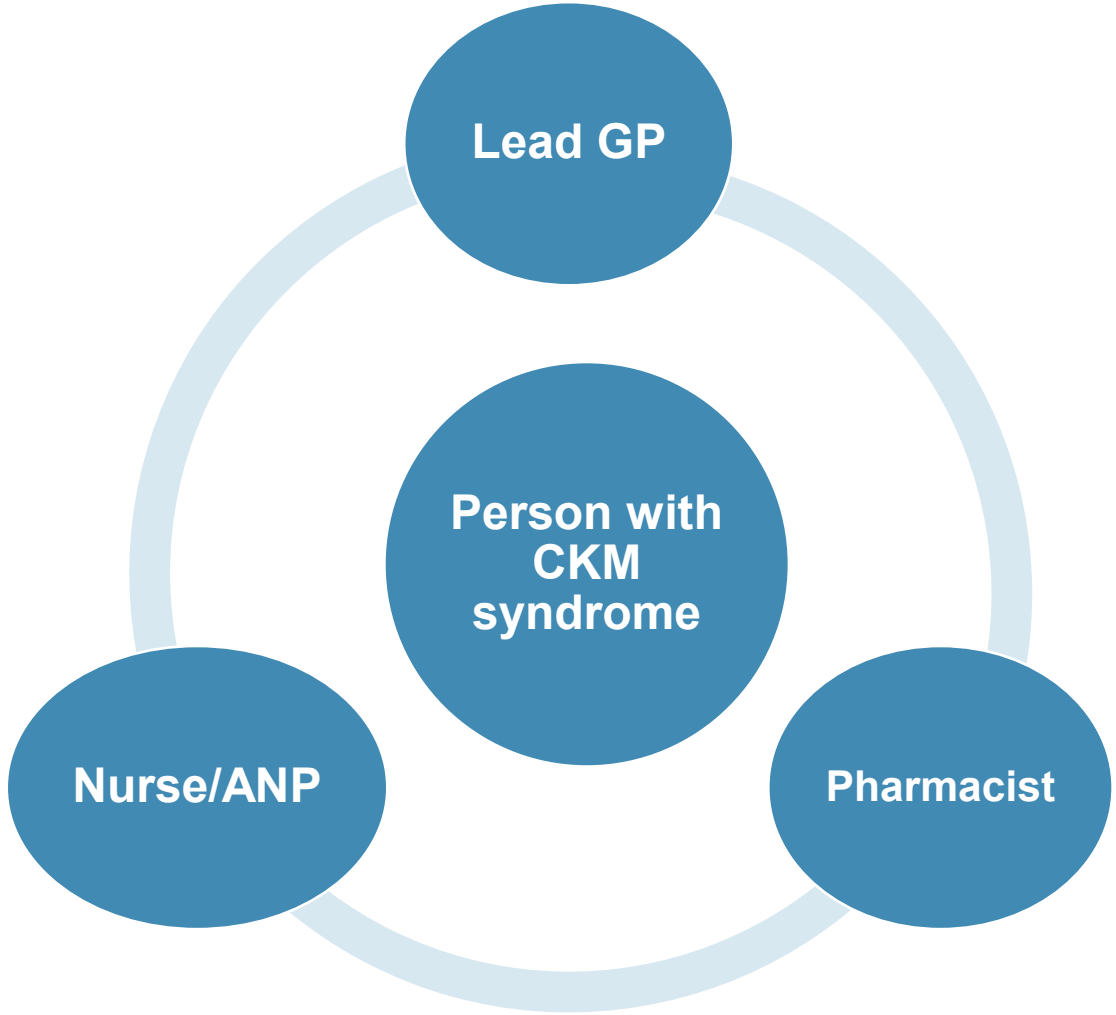
Complex intervention status	Elements identified by the RCGP definition of a GP	Expanded description[a]
Constant elements — always present	Community-based whole-person medicine-in-context	The expertise and experience to critically understand, manage, and evaluate whole-person medical care
Variable elements — adapts according to context	Knowing your patient(s), family, and carers	But recognising that GPs can deliver whole-person care from the first consultation
	Being accessible	Varies by location (such as in the surgery, at home) and method (in person, phone, digital). Beyond availability, instead recognising candidacy, concordance, and recursivity[7]
	Using trusted encounters	Trusted to do the best thing/right thing by me. Strengthened by consultation skills, empathy, relationships, partnerships. Built from the first consultation
	Knowing your community and context	Context always shapes the work done (constant component as above) What varies are the approaches we use to know — including experience (working in one place for a long time), conversations, analysis
	Working in multidisciplinary teams	Always working in teams, but the size and make-up of the team adapted to fit local needs
	Leading healthcare provision	Leadership as part of everyone's role. How we do that, and the proportion of our working week spent on this varies

[a]Developed from Reeve, 2023.[8] RCGP = Royal College of General Practitioners.

Standing up for general practice Joanne Reeve, Gail Allsopp and Michael Mulholland ; British Journal of General Practice 2025; 75 (750): 4-5. **DOI:** https://doi.org/10.3399/bjgp25X740373

#Teams based CKM care will be a future cornerstone of General Practice Long Term Condition Care.

Grange Family Practice CKM team

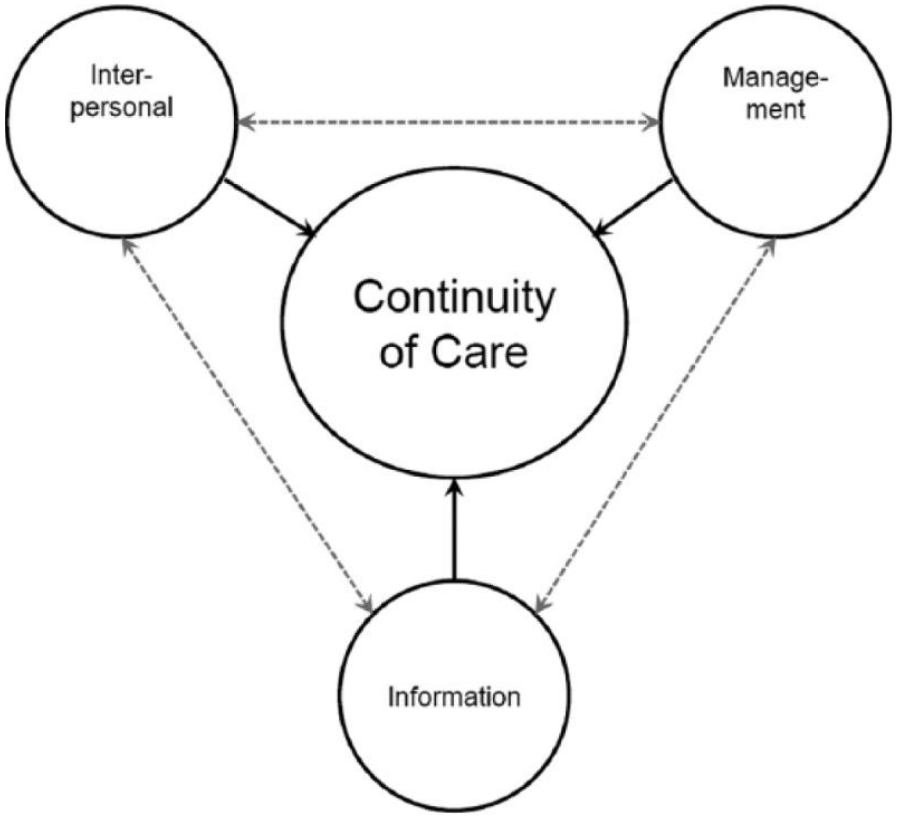


Supported by the solid foundations of Practice Managers and Admin Teams

> “the relationship between a patient and their GP is as important as a scalpel is to a surgeon.”

Benefits of relationship-based care

Benefits to patients	Benefits to GPs	Benefits to the NHS
• Improved experience and satisfaction	• Greater job satisfaction	• Fewer A&E attendances
• Better health outcomes	• Improved recruitment and retention	• Fewer unplanned admissions to hospital
• Lower mortality rates		• Better adherence to medical advice
• Increased engagement with medical advice		



Royal College of General Practitioners. The power of relationships: what is relationship-based care and why is it important. General practice COVID-19 recovery [download].accessed.https://www.rcgp.org.uk/-/media/Files/Policy/power-of-relationships-rcgp-2021.ashx. 2021

Beadles CA, Voils CI, Crowley MJ, Farley JF, Maciejewski ML. Continuity of medication management and continuity of care: Conceptual and operational considerations. SAGE open medicine. 2014;2:2050312114559261.

Global Effect of Cardiovascular Risk Factors on Lifetime Estimates

The Global Cardiovascular Risk Consortium

ABSTRACT

BACKGROUND

Five risk factors account for approximately 50% of the global burden of cardiovascular disease. How the presence or absence of classic risk factors affects lifetime estimates of cardiovascular disease and death from any cause remains unclear.

METHODS

We harmonized individual-level data from 2,078,948 participants across 133 cohorts, 39 countries, and 6 continents. Lifetime risk of cardiovascular disease and death from any cause was estimated up to 90 years of age according to the presence or absence of arterial hypertension, hyperlipidemia, underweight and overweight or obesity, diabetes, and smoking at 50 years of age. Differences in life span (in terms of additional life-years free of cardiovascular disease or death from any cause) according to the presence or absence of these risk factors were also estimated. Risk-factor trajectories were analyzed to predict lifetime differences according to risk-factor variation.

RESULTS

The lifetime risk of cardiovascular disease was 24% (95% confidence interval [CI], 21 to 30) among women and 38% (95% CI, 30 to 45) among men for whom all five risk factors were present. In the comparison between participants with none of the risk factors and those with all the risk factors, the estimated number of additional life-years free of cardiovascular disease was 13.3 (95% CI, 11.2 to 15.7) for women and 10.6 (95% CI, 9.2 to 12.9) for men; the estimated number of additional life-years free of death was 14.5 (95% CI, 9.1 to 15.3) for women and 11.8 (95% CI, 10.1 to 13.6) for men. As compared with no changes in the presence of all risk factors, modification of hypertension at an age of 55 to less than 60 years was associated with the most additional life-years free of cardiovascular disease, and modification of smoking at an age of 55 to less than 60 years was associated with the most additional life-years free of death.

CONCLUSIONS

The absence of five classic risk factors at 50 years of age was associated with more than a decade greater life expectancy than the presence of all five risk factors, in both sexes. Persons who modified hypertension and smoking in midlife had the most additional life-years free of cardiovascular disease and death from any cause, respectively. (Funded by the German Center for Cardiovascular Research [DZHK]; ClinicalTrials.gov number, NCT05466825.)

LOOK ALIVE – THINK FIVE!

- 5 **modifiable** risk factors account for ~50% global CVD burden……
- 50% of all CVD cases could potentially be **prevented** through **effective risk-factor management**.
- Fatal 5 : **Smoking ; Blood Pressure ; Cholesterol ; Diabetes ; obesity.**
- If your 50 and five –less….
- Women – CVD **13** yrs later ; death **14** yrs later
- Men – CVD **10** yrs later ; death **11** years later
- Behavioural change has impact!
  - HTN Mx & smoking cessation in midlife = longer life and more years CVD disease free.

Bottom line: Hope lies in our houses of care ; sustain life locally – look for and act on the five.

1. Magnussen C, Alegre-Diaz J, Al-Nasser LA, Amouyel P, Aviles-Santa L, Bakker SJ, et al. Global effect of cardiovascular risk factors on lifetime estimates. N Engl J Med. 2025.

BP threshold for treatment

confirm : BP ≥ 135/85 mmHg 7-day average HBPM or day-time average ABPM

start : pharmacological therapy on top of diet and lifestyle interventions irrespective of CVD risk.

On-treatment BP target

< 130/80 mmHg (measured by 7-day average HBPM or day-time average ABPM or office BP*)

or ALARA without causing unacceptable side-effects, and within 6-months of initiating treatment **for all adults.**

Exceptions:

frail and/or have limited life expectancy

use clinical judgement



CONSENSUS STATEMENT OPEN

Check for updates

Call to action: British and Irish hypertension society position statement on blood pressure treatment thresholds and targets

Luca Faconti[1], Nayanatara Tantirige[2], Neil R. Poulter[3], Jacob George[4], Vikas Kapil[5], Ajay Gupta[6], Pauline A. Swift[7], Anthony Heagerty[8], Eduard Shantsila[9], Sarah Partridge[10]✉ and Ian B. Wilkinson[2]

© The Author(s) 2025, corrected publication 2025

In this position statement the British and Irish Hypertension Society (BIHS) present a review of the current evidence for blood pressure (BP) treatment thresholds and targets. The BIHS recommend initiating pharmacological antihypertensive therapy, irrespective of cardiovascular disease risk, following a confirmed diagnosis of hypertension (sustained out-of-office BP ≥ 135/85 mmHg despite diet and lifestyle advice). The BIHS recommend an on-treatment BP target < 130/80 mmHg or as low as reasonably achievable without causing unacceptable side-effects, within 6-months of initiating treatment, for all adults. Possible subgroups to whom this may not apply are those who are frail and/or have limited life expectancy where higher targets may be appropriate based on clinical judgement and the individuals' tolerance to treatment. The BIHS believe that this simple 2-step approach will facilitate practitioners deliver evidence-based best practice, discourage therapeutic inertia around BP lowering and improve heath outcomes for all adults living with high BP.

Journal of Human Hypertension (2025) 39:537–540; https://doi.org/10.1038/s41371-025-01055-z

Faconti, L., Tantirige, N., Poulter, N.R. *et al.* Call to action: British and Irish hypertension society position statement on blood pressure treatment thresholds and targets. *J Hum Hypertens* **39**, 537–540 (2025). https://doi.org/10.1038/s41371-025-01055-z

MARY – TATT AND FLUSHING

- 50M - rare attender – keen for HRT
- Last seen 2021 (last bloods 2019).
- Nil reg meds.
- NKDA.
- Nil PMH bar Mirena coil last year

- Non smoker
- Occasional Social Alcohol
- Office worker
- Sedentary lifestyle but tries
- Married 2 kids

- PC: Non-specific TATT ? menopause

- HPC flushing , brain fog , vaginal dryness, more irritable, further weight gain from baseline raised BMI.
- HRT risk assessment done
- Full systematic questioning and examination yielded **nil TATT red flags**.

- Asked permission to discuss weight –agreed attend for review appt to do same – given STOPBANG to complete and bring to next assessment
- Agreed start HRT , TATT/CKM obesity bundle and review with results to discuss weight.

First do no harm

Be aware our weight bias and stigma as health care professionals is **harming** the patients we are trying to help.

Weight discrimination consequences (1) :

- Reduced health care use
- Avoidance of physical activity
- Low mood/reduced self esteem
- Increased binge eating

1. Puhl RM, Heuer CA. The stigma of obesity: a review and update. Obesity. 2009;17(5):941

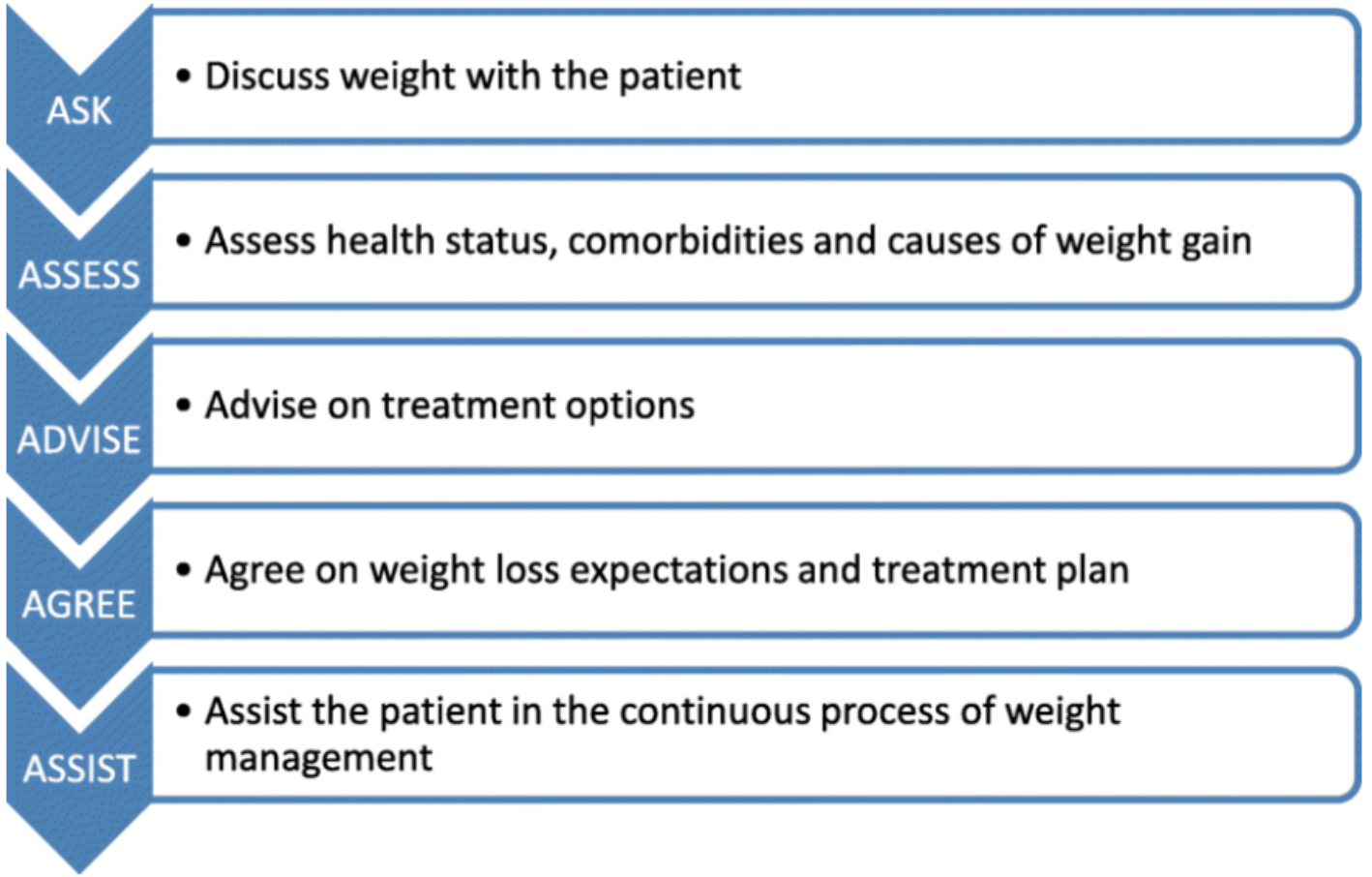
Kings Modified Obesity Staging and the 4 Ms Matter in Primary Care

	Criterion	Stage 0 Normal health	Stage 1 At risk of disease	Stage 2 Established disease	Stage 3 Advanced disease
A	Airway /Apnoea	**Normal** No snoring Neck circ < 43cm Epworth score <10	**Mild sleep apnoea** Mild snoring Epworth score ≥ 10 Mild OSA (dip rate<15/hr) Neck circ >43cm (size 18) Mild asthma	**Requires CPAP** Witnessed apnoea Dip rate >15/hr Uses CPAP (controlled) Severe asthma	Cor pulmonale Obesity hypoventilation syndrome Uncontrolled OSA
B	BMI	**<35 kg/m²**	**35-50 kg/m²**	**50-60 kg/m²**	**>60 kg/m²**
C	CVD risk	**<10%** CVD risk <10% over 10 years [JBS coronary risk prediction chart*]	**10-20%** CVD risk ≥10% over 10 years T2DM	**>20% or stable heart disease** Stable IHD CCF NYHA I-II, *or* >20% risk	Severe angina, *or* CCF NYHA III-IV
D	Diabetes	**Normal** Fasting or random glucose <5.7 mmol/L Normal HbA1c	**IFG** IFG / IGT, *or* previous GDM	**T2DM** Diet, insulin or OHA controlled HbA1c<9%	**Uncontrolled T2DM** HbA1c>9% Advanced microvascular disease
E	Economic complications	**Normal** Obesity has no financial impact	**Financial impact** Increased travel cost Increased clothes cost	**Workplace disadvantage** Earnings limited by obesity Receiving benefits due to obesity	Unemployed due to obesity Financial effect on 3rd party (e.g. carer required to reduce income)
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H	Health status (perceived)	**Normal** Good mental and physical well being	**Low mood/poor QoL**	Mild–moderate depression Takes treatment for depression	Severe depression Suicidal ideation Unmanaged substance abuse Active self harm
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K	Kidney	Normal	Proteinuria	eGFR <60	eGFR <30
L	Liver	Normal	Elevated LFTs, NAFLD on ultrasound	NASH	Liver failure

Four M´s	Characteristics included
Mental:	Mood, depression, eating disorders, anxiety, personality, cognition, attention deficit, sleep quality, potentials traumas, addictions.
Mechanical:	Musculoskeletal disorders, chronic pain, OSA, GERD, urinary incontinence, intertrigo, thrombosis, plantar fascitis, pseudotumour cerebri.
Metabolic:	T2D, hyperinsulinemia, dyslipidemia, hypertension, gout, cholelithiasis, MAFLD, PCOS, hypothyroidism, obesity-related cancers.
Monetary	Medical insurance, impact of obesity on income, education, employment, the ability to afford a healthy diet, access to weight loss programs

Dobbie, L.J., Coelho, C., Crane, J. *et al.* Clinical evaluation of patients living with obesity. *Intern Emerg Med* **18**, 1273–1285 (2023). https://doi.org/10.1007/s11739-023-03263-2
Reviews in Endocrine and Metabolic Disorders (2023) 24:795–807 https://doi.org/10.1007/s11154-023-09797-2

The 5 A's of Obesity Management



Welzel FD, Stein J, Pabst A, Luppa M, Kersting A, Blüher M, Luck-Sikorski C, König HH, Riedel-Heller SG. Five A's counseling in weight management of obese patients in primary care: a cluster-randomized controlled trial (INTERACT). BMC Fam Pract. 2018 Jun 23;19(1):97. doi: 10.1186/s12875-018-0785-7.

- **Goals of obesity care** – Worried about health , wants to be healthy for children and live to see them start their adult lives. Wants to be more active with kids. Doesn't want parents ill heath trajectory.

- **Weight Trajectory –** No childhood/teenage obesity. Move to University triggered weight gain , yo- yo dieting occurred through her 30's and 40's. Multiple diet and exercise regimes. Would always put the weight back on again. At current highest weight – pregnancies all escalated base line weight by ½ stone each time and now the broken sleep is causing night snacking. Stress from brain fog is causing her to comfort eat. Life triggers have been periods of stress and pregnancy and now menopause.

- **Food –** Busy working mum. Cooks 4 dinners a week but other nights has energy dense highly palatable quickly cooked meals.
-
- **Physical Activity –** 30mins of moderate intensity activity a week and 1 resistance based activity.

- **Stress** – menopause symptoms causing her extreme distress. No hx of any form of trauma (neglect/abuse etc)

- **Sleep** – 5hrs at most due to the flushing

- **Medication History** – No culprits.

- **Family History** – Mum – obesity ; Dad – obesity and T2D.

- **ASSESS**

- FBP/TFT/B12/coeliac/folate/iron stores/vitamin D all normal
- **Airways** -Snores but no other OSA symps Neck Circumference 41cm (at risk) STOP BANG 0 EPWORTH 0.
- **BMI** 36.8 wt104kg WC 141cm wt height ratio 0.66 (highest weight currently ; BMI>30 since at least 2006)
- **CVD:** LDL 3.6 QRISK 14% BP 150/92 at clinic. No S&S HF. HRSR.
- **Diabetes:** Hba1c 84 (repeat 82) ; recurrent thrush..
- **Economic:** weight not affecting work but menopause is.
- **Functional:** LBP long standing – no red flags – imaging NAD advised supportive mx previously 'loose weight'. Kids v active and cant do as much with them as she would like.
- **Gonadal** No Infertility/PCOS issues – 2 children
- **Health status.** Euthymic
- **Image** No binge eating. Comfort eats.
- **Gastro** functional dyspepsia HB N no nsaids/alcohol h plyori –ve no red flags on a PPI
- **Kidney** – GFR: >60 mL/min/1.73m^2 ACR: Normal
- **Liver** – LFTS N Fib 4 low risk 0.72



DIAGNOSIS FOUND

#CKM Diagnoses Found:

- Obesity
- T2D
- Dyslipidaemia
- Hypertension (24 hr BP cuff confirmation average day 143/92 mmHg) ECG: NSR, no LVH)

- **AND** functional dyspepsia / mechanical lower back ache / fungal infections/ relationship with family impacted.

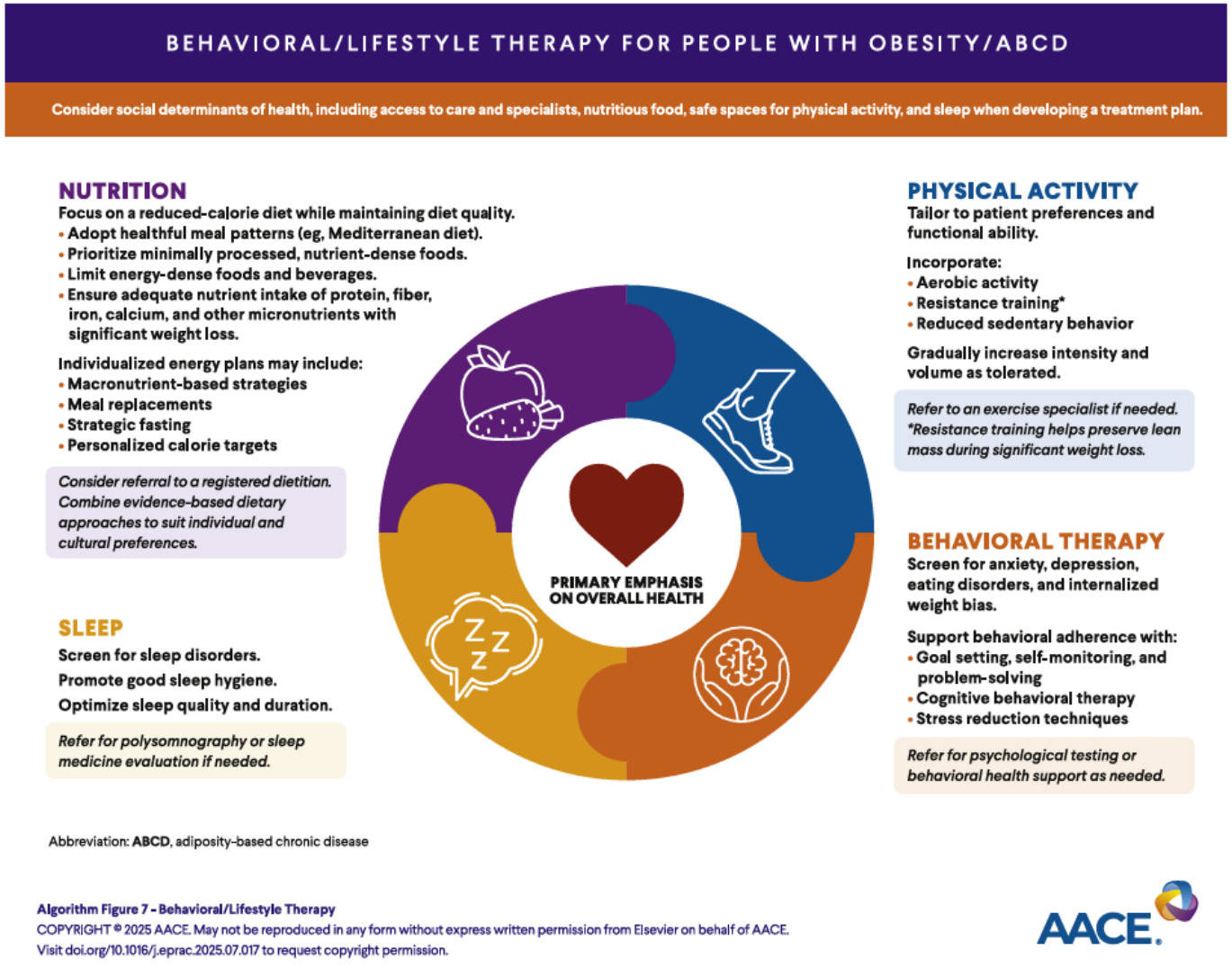
CKM care is comprehensive whole person care

SILO REVIEW CHECKERS → CKM REVIEW CHESS



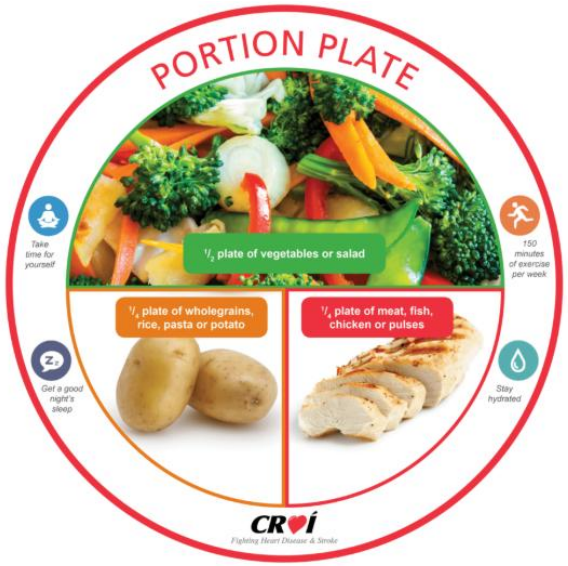


CHESS MOVES MADE



Algorithm 7. Behavioral/lifestyle therapy for people with obesity/adiposity-based chronic disease.

- Food Strategy
- Plate work
- Movement Strategy -5Ss
- Stress
- Sleep - CBTi
- Just start , think S.M.A.R.T



TYPE 2 DIABETES: PHYSICAL BEHAVIOUR TIPS

SITTING/BREAKING UP PROLONGED SITTING

Walking around or doing simple resistance exercises every 30 minutes breaks up prolonged sitting and improves glucose metabolism.

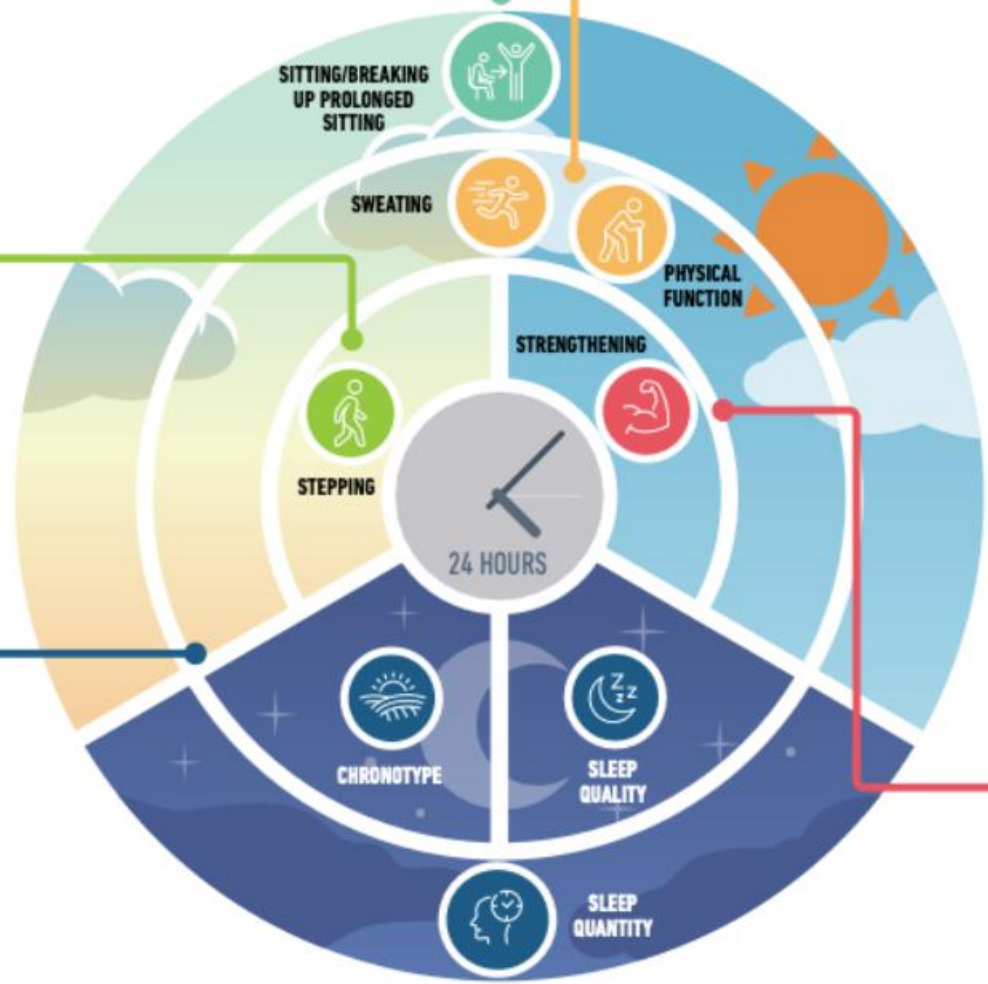
STEPPING

- An extra 500 steps a day can reduce risk of cardiovascular morbidity and all-cause mortality by 2-9%.
- Brisk walking for 5/6 minutes every day can add 4 years to life expectancy

SLEEP

Aim for consistent, uninterrupted sleep.

- More than 8 hours and less than 6 hours of sleep negatively impacts HbA1c
- Irregular sleep results in poorer glycaemic levels – this could be due to insomnia, obstructive sleep apnoea and restless leg syndrome
- 'Night owls' who go to bed late and get up late may be more susceptible to inactivity and poorer glycaemic levels compared to 'early birds' who go to bed early and get up early.



SWEATING

Aim for:

- 150 minutes or more of moderate-intensity exercise every week

OR

- 75 minutes or more of vigorous-intensity exercise spread over three days or more, every week.

Avoid no more than 2 consecutive days of inactivity and supplement with 2/3 resistance, flexibility and/or balance sessions.

The ability of people with type 2 diabetes to undertake simple exercises in middle-age is similar to that in people over a decade older.

Just 30 minutes of moderate-intensity physical activity a week improves metabolic profiles.

STRENGTHENING

Resistance exercise such as tai chi and yoga improves insulin sensitivity and glucose levels

leicesterdiabetescentre.org.uk
@LDC_Tweets
leicesterdiabetescentre

This image was adapted from Figure 2 of the new European Association for the Study of Diabetes (EASD) and American Diabetes Association (ADA) consensus report for management of hyperglycaemic in type 2 diabetes, published simultaneously in the journals Diabetes Care (Davies et al. Diabetes Care. 2022; Nov 1;45(11):2753–2786. doi: 10.2337/dci22-0034) and Diabetologia (Davies et al. Diabetologia. 2022; 65:1925–1966)

Leicester Diabetes Centre

BEHAVIOURAL THERAPY – WHY WE EAT

- **Increasing self monitoring –** daily records of intake, activity.
- **Setting Goals** – keeping it simple 'three changes'.
- **Social Support Mobilisation and Social Saboteur Assassination** – needed for modifying behaviours.
- **Relapse Prevention** – expecting set backs , be prepared, view them as temporary.
- **Stress Management.**

Engage in a spirit of **Motivational interviewing**

- Express empathy
- Develop discrepancy
- Avoid argumentation
- Roll with resistance.

CHESS MOVES MADE

<u>Non-pharmacological Moves:</u>

Everything discussed plus

DSMES / Dietician Referrals /Podiatry (low risk) /Regional eye screening (none present)

PARS offered

<u>Rapid Pharmacological Moves:</u>

Metformin 2g

SGLT2i

Losartan 50 mg (improved systolic to 132)

Atorvastatin 40 mg

Repeat Hba1c at 3 months was 60; **BMI unchanged**. Incretin initiated and titrated to maximum dose

CURRENT STATE OF PLAY

Hba1c 40

U&E N ACR N

LFTS N

Fib 4 Low risk

Systolic 128 (now off medication)

LDL 1.9

BMI 30.1

17 % body weight loss with incretin

11cms of waist circumference

HRT – CVD protection

Taking medications. On routine review.



What is she doing currently ? I do not know because she is busy flourishing in her life. I will find out at routine review or if she self represents with evolving clinical need.

OBESITY IS AN **INDEPENDENT** DISEASE STATE WITHIN THE CKM SYNDROME THAT DESERVES **INDEPENDENT** ATTENTION….IN **GENERAL PRACTICE**

OBESITY IS A CHRONIC, RELAPSING, MULTIFACTORIAL DISEASE CHARACTERIZED BY **NEUROENDOCRINE AND NEUROBEHAVIOURAL DYSREGULATION**, IN WHICH **EXCESS ADIPOSITY**—WITH OR WITHOUT **ABNORMAL DISTRIBUTION** OR **FUNCTION**—LEADS TO ADVERSE **METABOLIC, BIOMECHANICAL**, AND **PSYCHOSOCIAL** HEALTH CONSEQUENCES.

WHAT IS OUR NORMAL APPETITE BIOLOGY?

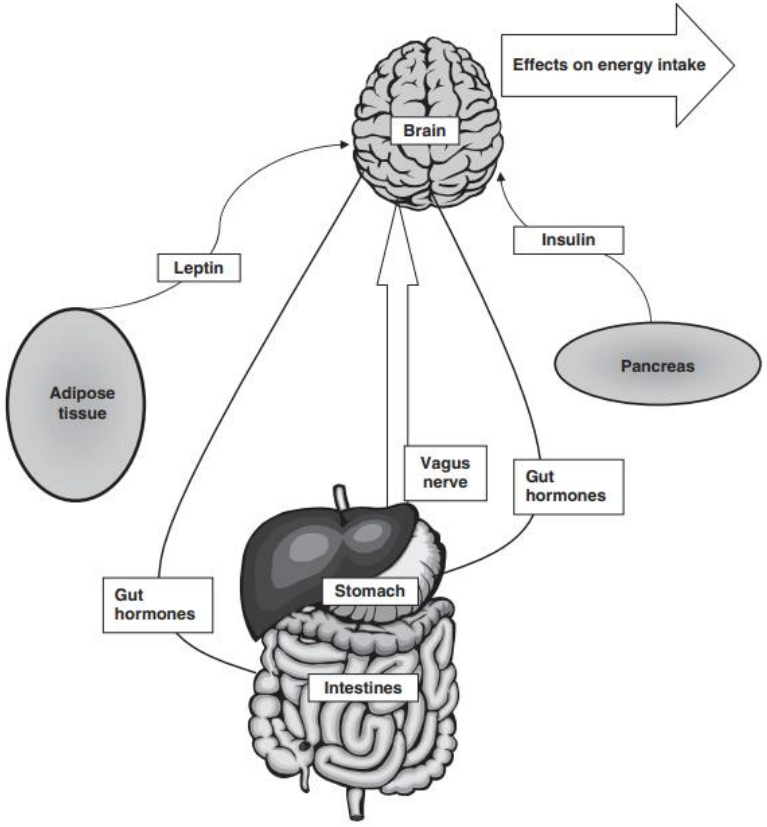


Figure 2 A schematic representation of physiological satiation and satiety signalling. When food is consumed, the gastric distension is communicated to the brain via the vagus nerve, which connects the gastrointestinal tract to the brain, initiating satiation. Gut hormones from the stomach and intestine are released when food is consumed and act on areas of the brain involved in appetite. Leptin from adipose tissue and insulin from the pancreas are related to the amount of fat stored in the body and act on the brain to modulate satiation and satiety in the longer term. All signals are integrated in the brain to affect energy intake and expenditure (Adapted from Wynne *et al.* 2005a).

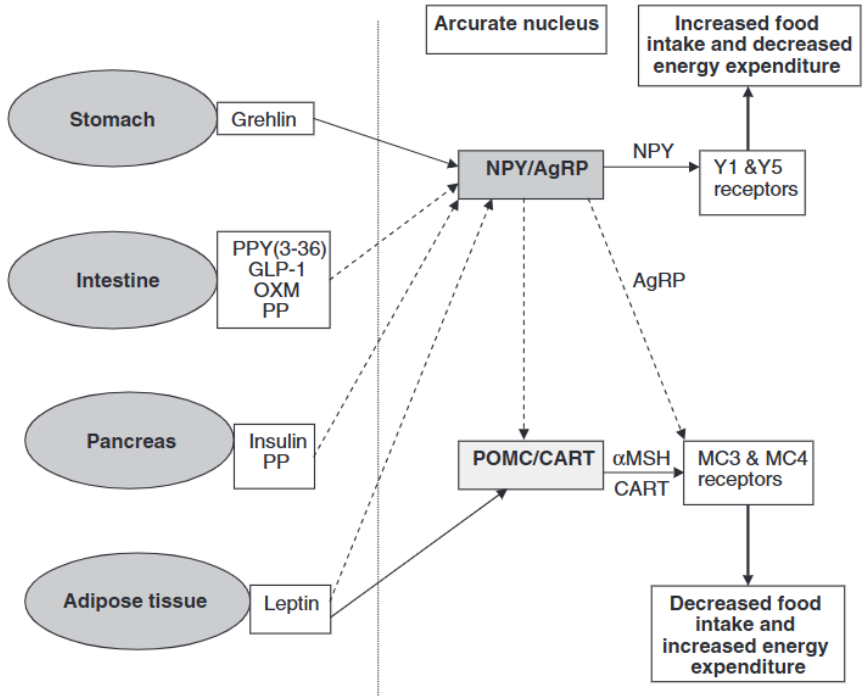
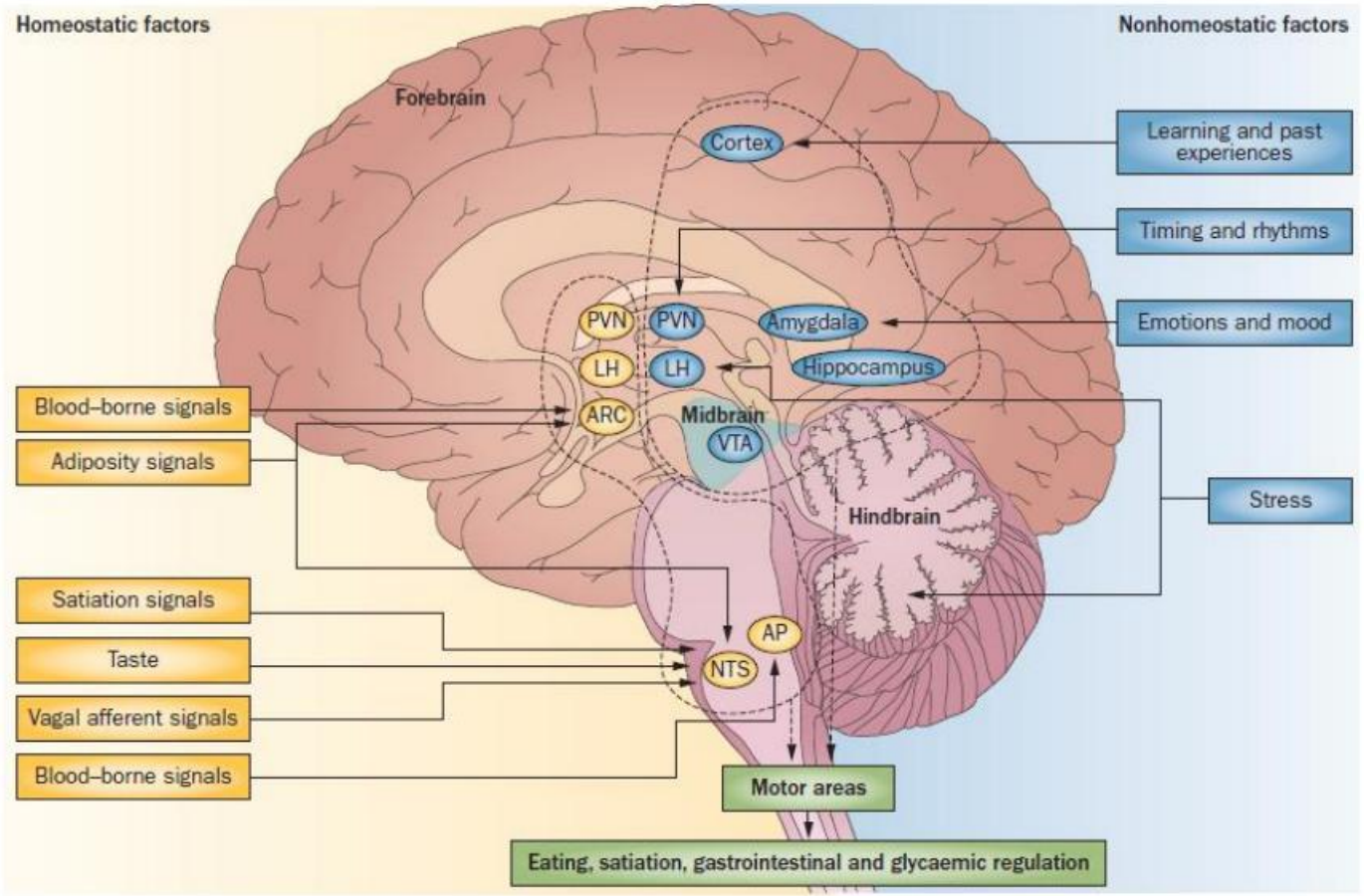


Figure 3 Circulating hormones influencing energy homeostasis via the arcuate nucleus (adapted from Murphy & Bloom 2004). Continuous lines indicate stimulatory effects, and dashed lines indicate inhibitory effects. AgRP, agouti-related peptide; CART, cocaine-and amphetamine-related transcript; GLP-1, glucagon-like-peptide 1; αMSH, alpha-melanocyte-stimulating hormone; NPY, neuropeptide Y, OXM, oxyntomodulin; POMC, pro-opiomelanocortin; PP, pancreatic polypeptide; PYY, peptide YY.

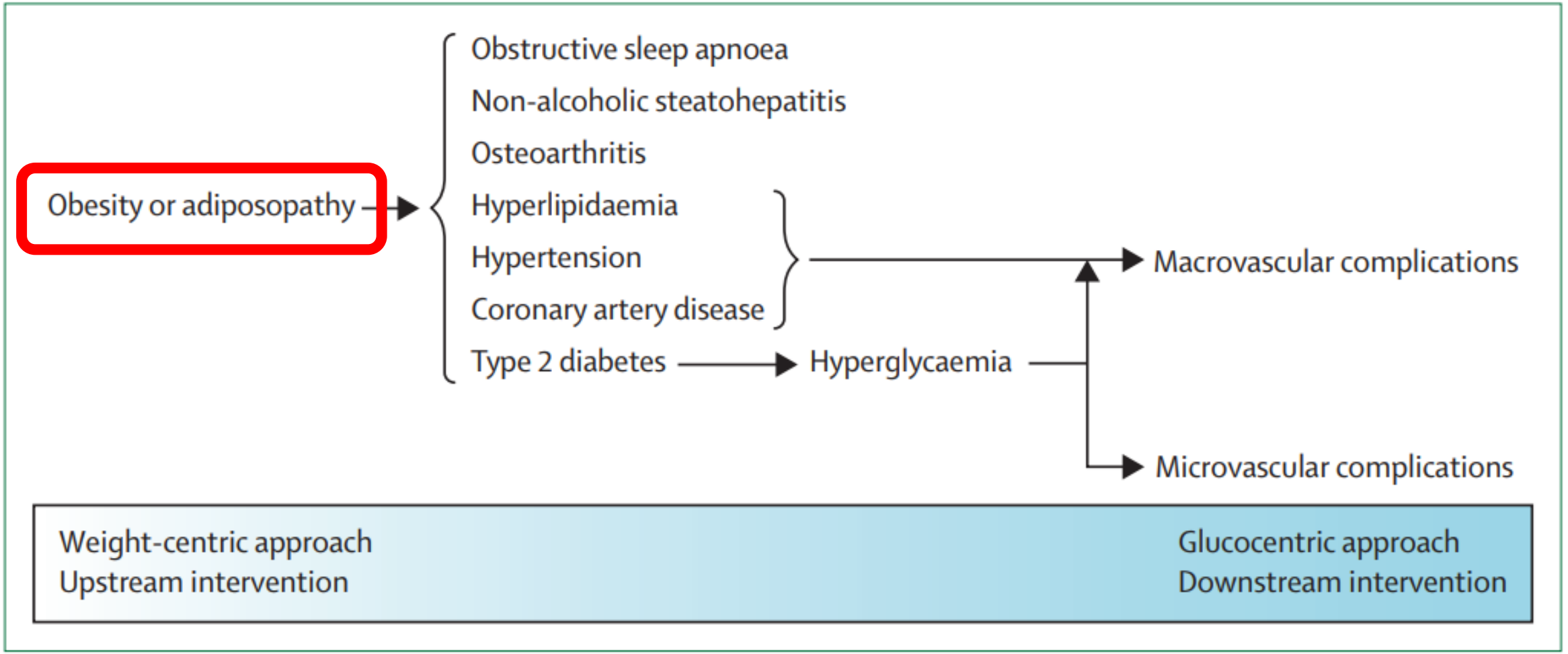
Benelam, B. (2009), Satiation, satiety and their effects on eating behaviour. Nutrition Bulletin, 34: 126-173. https://doi.org/10.1111/j.1467-3010.2009.01753.x

PEOPLE LIVING WITH OBESITY HAVE PATHOPHYSIOLOGY, **NOT** POOR WILL POWER.



- Hypothalamus – energy homeostasis
- Mesolimbic system – reward seeking behaviour
- Pre frontal cortex – executive functioning

Reframe our conversations, Reframe our review infrastructure



Lingvay I, Sumithran P, Cohen RV, le Roux CW. Obesity management as a primary treatment goal for type 2 diabetes: time to reframe the conversation. Lancet. 2022 Jan 22;399(10322):394-405. doi: 10.1016/S0140-6736(21)01919-X. Epub 2021 Sep 30. Erratum in: Lancet. 2022

Kings Modified Obesity Staging Model

Table 3 King's obesity staging criteria Adapted from Whyte et al. [78]

	Criterion	Stage 0 Normal health	Stage 1 At risk of disease	Stage 2 Established disease	Stage 3 Advanced disease
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K	Kidney	Normal	Proteinuria	eGFR <60	eGFR <30
L	Liver	Normal	Elevated LFTs, NAFLD on ultrasound	NASH	Liver failure

Stage 1 conditions: patients at risk of developing co-morbidities or with mild co-morbidities that may potentially justify bariatric surgery but do not need specialist opinion.

Stage 2 conditions: established disease and higher surgical risk. This may require medical review.

Stage 3 conditions: indicate a need for specialist opinion and may represent contraindications for surgery.

BMI Body Mass Index, *Neck circ* neck circumference, *CPAP* Continuous Positive Airway Pressure, *OSA* Obstructive Sleep Apnoea, *CVD* Cardiovascular Disease, *IFG* Impaired Fasting Glucose, *IGT* Impaired Glucose Tolerance, *GDM* Gestational Diabetes Mellitus, *T2DM* Type 2 Diabetes Mellitus, *GORD* Gastro-oesophageal Reflux Disease, *QoL* Quality of Life, *NAFLD* Non-alcoholic Fatty Liver Disease, *NASH* Non-alcoholic Steatohepatitis, *PPI* Proton Pump Inhibitor, *LFT* Liver Function Tests

Does this morning clinic look familiar?

9.00 am : **Airways** consult- OSA diagnosis and referral.
9.10 am : **BMI** consult– on incretin therapy – didn't disclose gallstones – biliary colic flare.
9.20 am : **CVD** – Heart Failure new presentation.
9.30 am : **Diabetes** foot ulcer review in treatment room.
9.40 am : **Economic** hardship consult with obesity implicated.
9.50 am : **Functional** MSK Lower back pain consult driven by obesity
10.00 am : Break
10.10 am**: Gonadal** Infertility due to PCOS referral
10.20 am :**Health perception** Depression Consultation – trauma.
10.30 am : **Image** – eating disorder consult.
10.40 am : **Gastro –** Functional dyspepsia
10.50 am: **Kidney** - ESRD JIC meds consult
11.00 am: **Liver** – MASLD consult.

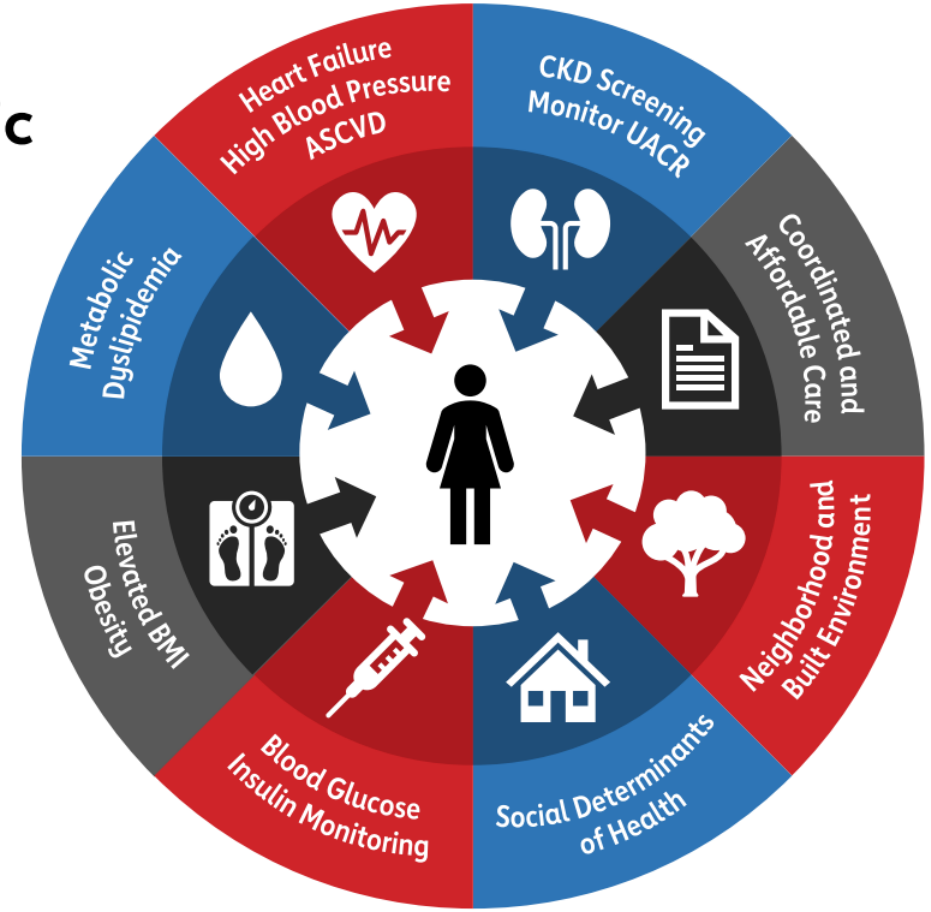


General Practice is enveloped by the consequences of obesity.... **yet our long term condition infrastructure has not made room for the root disease entity itself.**

CKM APPROACH ELIMINATES CARE FRAGMENTATION

Cardiovascular-Kidney-Metabolic Syndrome

Patient-Centered Implementation Focus



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; BMI, body mass index; CKD, chronic kidney disease; and UACR, urine albumin-creatinine ratio.

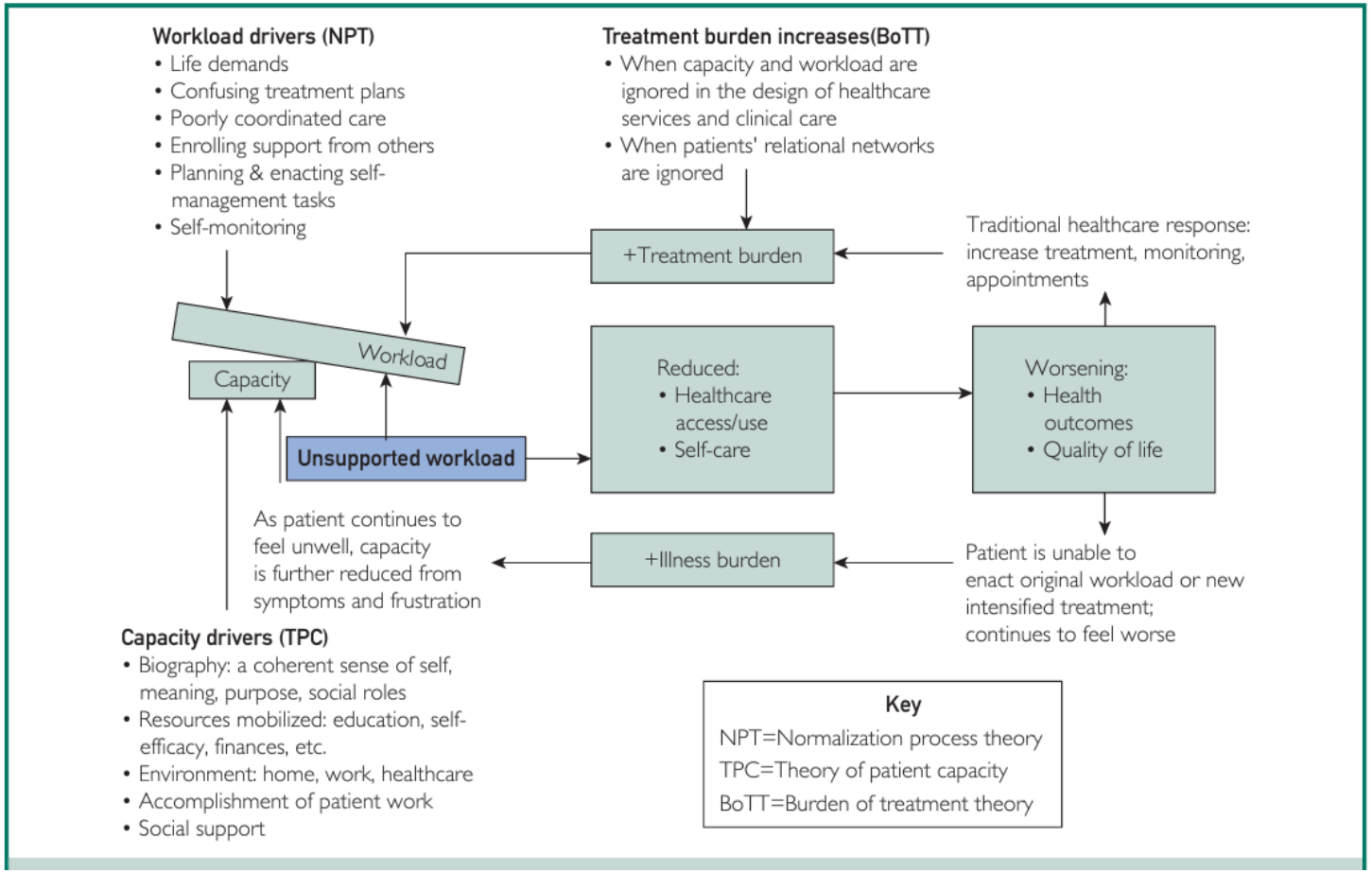
American Heart Association.

Ndumele, C.E. et al., Cardiovascular-Kidney-Metabolic Health: A Presidential Advisory From the American Heart Association. 2023. *Circulation*.

GENERAL PRACTICE CKM REVIEW IS MINIMALLY DISRUPTIVE MEDICINE

- Multi-organ impact
- Co-ordinated care
- ⬇ Appointment burden
- 1 review facilitating agendas across multiple specialties
- ⬇ Person workload

…Polypharmacy is at play



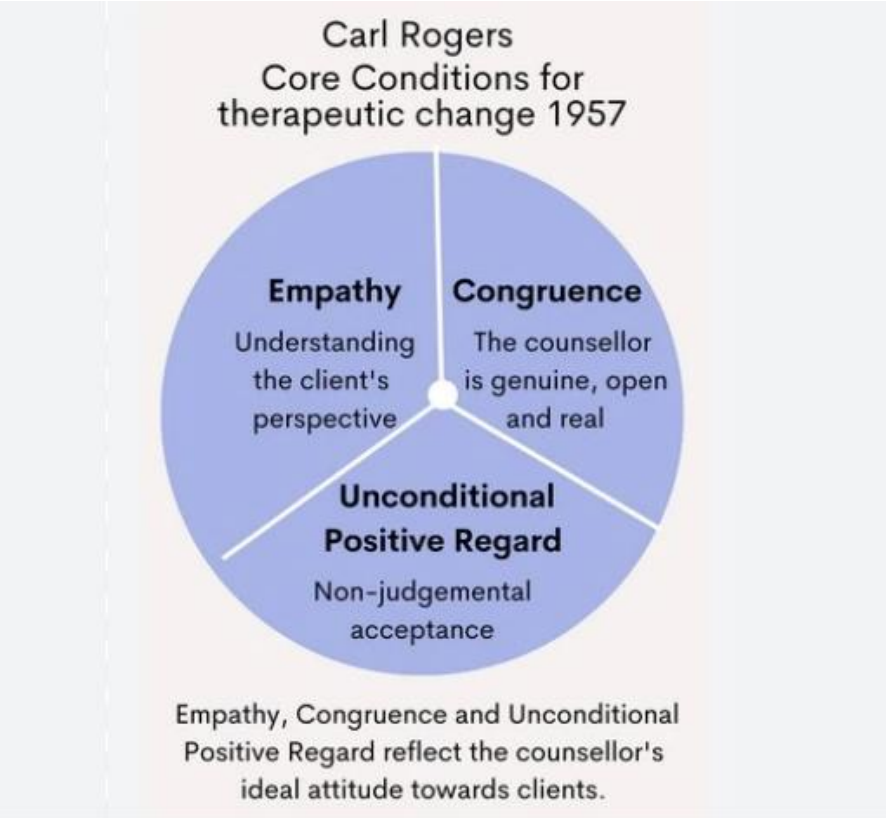
Boehmer KR, Gallacher KI, Lippiett KA, Mair FS, May CR, Montori VM. Minimally disruptive medicine: progress 10 years later. Mayo Clinic Proceedings; Elsevier; 2022

CKM PHILOSOPHY OF CARE

The professional conversation is a tool – use it.

Behavioural Science

Anchoring – Frame – Chunking & Checking –Cognitive ease – Choice architecture- Status Quo bias



Source : https://counsellingtutor.com/counselling-approaches/person-centred-approach-to-counselling/carl-rogers-core-conditions/

A compendium of CKM help for your GP Houses of Care.

Cardiovascular–Kidney–Metabolic Review Checklist

Medscape Primary Care Hacks

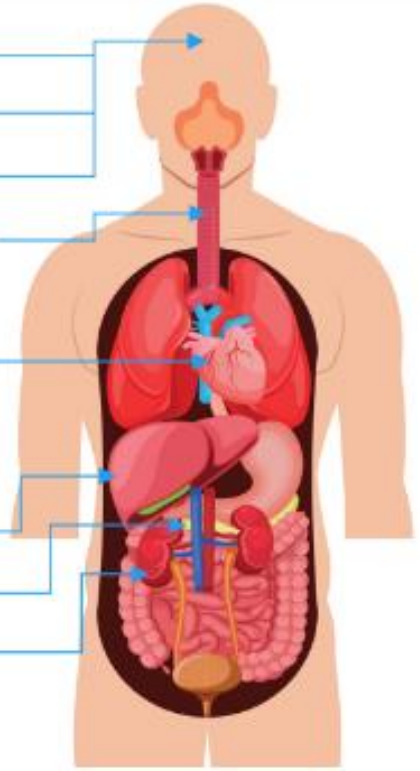
Authors: Dr Kevin Fernando, Portfolio GP, East Lothian and Content Advisor, Medscape (email: kfernando@webbed.net); Dr Eimear Darcy, GP Partner, Grange Family Practice Omagh

The cardiovascular, kidney, and metabolic systems are intrinsically interconnected, leading to the new description of the cardiovascular–kidney–metabolic (CKM) syndrome, also known as the CVRM syndrome.[1][2] The pleiotropic effects of many modern CKM therapies have demonstrated wide-ranging benefits in these overlapping organ systems.[1][3] However, even though the bidirectional relationship between these organ systems has long been recognised, many guidelines and clinical pathways still encourage a single-disease model of care.[1][3]

The delivery of healthcare and education needs to be reconfigured away from a disease-centred approach to a more holistic, person-centred model to improve both the quality and quantity of patients' lives and the sustainability of our healthcare systems.

This CKM checklist is a tool to facilitate an evidence-informed yet person-centred approach to patient care for all healthcare professionals.

The following areas should all be considered during shared decision-making.



- Severe Mental Illness
- Psychological Stress
- Migraine
- Sleep Health and Airways
- Heart:
  - ASCVD
  - Heart Failure
  - Atrial Fibrillation
- Blood Pressure
- Liver and MASLD
- Prediabetes and T2D
- Kidneys

- Frailty and Older People
- Overweight and Obesity
- Lipids
- Inflammatory Conditions
- Polycystic Ovary Syndrome
- Perimenopause and Menopause
- Erectile Dysfunction
- HIV
- Prescribing Considerations

Identification of Chronic Kidney Disease in Primary Care

Medscape UK X Guidelines
Primary Care Hacks

Authors: Dr Eimear Darcy, GP Partner, Grange Family Practice Omagh; **Dr William Hinchliffe**, Consultant in Renal and General Medicine, South Tyneside and Sunderland Foundation NHS Trust; **Dr Deepika Manoharan**, Registrar in Renal and General Medicine, South Tyneside and Sunderland Foundation NHS Trust; **Dr Kevin Fernando**, GP Partner, North Berwick Health Centre and Content Advisor, Medscape Global and UK (email: kfernando@webmd.net)

Prevention of ESRD matters, as it has lower survival rates than colorectal, prostate, and breast cancer.[1] Microalbuminuria and eGFR <60 ml/min/1.73 m² are independent and amplifying predictors of mortality risk.[2]

1. Identify Individuals at Risk of CKD[3–7]

Key Clinical Risk Factors:[3–6]
- Hypertension
- CVD
- Diabetes
- Obesity

Other Groups at Risk of CKD:[3–5]
- Previous history of AKI
- Family history of kidney failure
- Structural renal tract disease
- Recurrent nephrolithiasis
- Prostatic hypertrophy
- Gout
- Multisystem disease
- Hereditary kidney disease
- Long-term treatment with nephrotoxins (e.g. NSAIDs, lithium, CNIs)
- Older people

2. Test At-risk Adults for CKD[3,7]

Evaluate kidney function—eGFR (calculated based on sCr)
AND
Evaluate kidney damage—albuminuria (uACR[A])

If uACR ≥3 mg/mmol (≥30 mg/g) **OR** eGFR <60 ml/min/1.73 m² → Retest in 3 months[B]

If uACR <3 mg/mmol (<30 mg/g) **AND** eGFR ≥60 ml/min/1.73 m²

3. Diagnose CKD[3,7]

If low eGFR or high uACR is present for >3 months, **diagnose CKD**

Retest at least once per year

4. Classify and Code CKD[3–5,8]

CKD prognosis is depicted in the RAG/heatmap table (Figure 1); the recommended frequency for monitoring eGFR is represented by the numbers in the boxes (e.g. test once per year if uACR <3 mg/mmol and GFR ≥60 ml/min/1.73 m²)

Code CKD as priority 1, documenting eGFR + ACR + cause

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)

GFR categories (mL/min/1.73 m²) Description and range			Albuminuria categories Description and range: A1 Normal to mildly increased <30 mg/g <3 mg/mmol	A2 Moderately increased 30–299 mg/g 3–29 mg/mmol	A3 Severely increased ≥300 mg/g ≥30 mg/mmol
G1	Normal or high	≥90	Screen 1	Treat 1	Treat and refer 3
G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat and refer 3
G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat and refer 3
G3b	Moderately to severely decreased	30–44	Treat 2	Treat and refer 3	Treat and refer 3
G4	Severely decreased	15–29	Treat and refer* 3	Treat and refer* 3	Treat and refer 4+
G5	Kidney failure	<15	Treat and refer 4+	Treat and refer 4+	Treat and refer 4+

Low risk (if no other markers of kidney disease, no CKD)[C] · High risk · Moderately increased risk · Very high risk

Figure 1: Risk of CKD Progression, Frequency of Monitoring, and Referral to Nephrology According to GFR and Albuminuria[9]

5. Refer to Secondary Care Where Appropriate

Refer to Renal if:[3–5,9]
- **KFRE** (kidneyfailurerisk.co.uk): 5-year risk of ESRD of >5% in CKD G3 or 2-year risk exceeds 10% if eGFR <30 ml/min/1.73 m² (04)—it may be wise to calculate likely timing of clinical outcomes (ckdpcrisk.org/lowgfrevents) to contextualise life expectancy and probability of CVE within 2–4 years, as referral may impact QoL
- a sustained decrease in eGFR of ≥25% and a change in eGFR category within 12 months
- a sustained decrease in eGFR of ≥15 ml/min/1.73 m² per year
- uACR 70 mg/mmol, unless known to be secondary to diabetes
- uACR >30 mg/mmol **AND** haematuria
- poorly controlled hypertension, despite the use of **at least four** antihypertensives
- suspected genetic causation, e.g. PKD
- suspected renal artery stenosis
- recurrent nephrolithiasis
- persistent anaemia (Hb <10 g/dl) in G4, as EPO and intravenous iron may help QoL
- any previous episode where serum potassium has breached 6 mmol/l.

Urgent/immediate referral to/discussion with specialist services may be warranted if:[10]
- eGFR <15 ml/min/1.73 m²
- AKI
- nephrotic syndrome (heavy proteinuria with low serum albumin)
- hypertensive crisis: BP >180/120 mmHg
- suspicion that a systemic illness (e.g. sarcoidosis, myeloma) has renal involvement.

In patients with persistent NVH and no evidence of UTI, refer to Urology using a **suspected cancer pathway referral** if age ≥60 years (do not assume secondary to CKD).[F,11]

Footnotes
[A] A spot uACR is acceptable.[3,4]
[B] NICE suggests repeating the eGFR within 2 weeks if eGFR <60 ml/min/1.73 m²[3]—this is at the discretion of the ordering clinician, based on the acuity of the presenting cause. If there is persistent invisible haematuria, also consider referring for investigation for urinary tract malignancy.[3]
[C] Key markers of kidney disease include urine sediment abnormalities, tubular disorders, histology-proven abnormalities, imaging-proven structural abnormalities, and kidney transplantation.[3,5]

ACR=albumin to creatinine ratio; AKI=acute kidney injury; BP=blood pressure; CKD=chronic kidney disease; CNI=calcineurin inhibitor; CVD=cardiovascular disease; CVE=cardiovascular event; eGFR=estimated glomerular filtration rate; EPO=erythropoietin; ESRD=end-stage renal disease; GFR=glomerular filtration rate; KFRE=Kidney Failure Risk Equation; NSAID=nonsteroidal anti-inflammatory drug; NVH=non-visible haematuria; PKD=polycystic kidney disease; QoL=quality of life; RAG=red, amber, green; sCr=serum creatinine; uACR=urine albumin to creatinine ratio; UTI=urinary tract infection

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Interventions for Chronic Kidney Disease in Primary Care

Medscape UK X Guidelines
Primary Care Hacks

Authors: Dr Eimear Darcy, GP Partner, Grange Family Practice Omagh; **Dr William Hinchliffe**, Consultant in Renal and General Medicine, South Tyneside and Sunderland Foundation NHS Trust; **Dr Deepika Manoharan**, Registrar in Renal and General Medicine, South Tyneside and Sunderland Foundation NHS Trust; **Dr Kevin Fernando**, GP Partner, North Berwick Health Centre and Content Advisor, Medscape Global and UK (email: kfernando@webmd.net)

1. Lipids[3–5,15–18]
- For primary prevention, start **atorvastatin 20 mg OD**
- For secondary prevention, do not stop or attenuate dose if eGFR <30 ml/min/1.73 m²
- Offer **aspirin** for **secondary prevention** of CVD
- See also the Primary Care Hack on lipid management

2. RAASis and Blood Pressure[3–5,12,18–25]
- Aim for **SBP <130 mmHg**, or **as low as reasonably achievable**[A]
- Follow NG136[19] and NG203[20] when choosing medications
- When RAASi is first line, choose an **ARB**[21]
- Independent of BP:[18]
  - if uACR >30 mg/mmol: start **ARB** and titrate to maximum tolerated dose
  - if uACR >3 mg/mmol in people with diabetes: start **ARB** and titrate to maximum tolerated dose
    - additional agents may be required
- If eGFR <60 ml/min/1.73 m² when starting or increasing RAASi, check U&E within **28 days**; if eGFR ≥60 ml/min/1.73 m², there is no need to routinely recheck U&E
  - **if K+ ≥5.5 mmol/l or sCr rise ≥50% encountered, follow Figure 1**, otherwise, continue to optimise RAASi dose.

3. SGLT2 Inhibitors[18,20,21,26]
- Once RAASi has been titrated to maximum tolerated dose, start an **SGLT2i** in eligible groups as per Figure 2, unless contraindicated (e.g. in people living with T1DM)[18,21,23]
- Continue the SGLT2i until dialysis or transplant[18,23,24]
- See also the Primary Care Hack on extra-glycaemic indications of SGLT2is

4. Specific Considerations in T2DM
If T2DM, eGFR ≥25 ml/min/1.73 m², and uACR ≥3 mg/mmol (≥30 mg/g), consider adding:
- **finerenone**[21]—a nonsteroidal MRA that can be added as third line to an RAASi and an SGLT2i (or second line if SGLT2i is inappropriate or not tolerated) if serum potassium concentration is <5.0 mmol/l, to reduce the risk of adverse kidney and CV outcomes[18,21,22,27,28]—see bit.ly/3v8PX4f
- **semaglutide**[26]—the FLOW study demonstrated that weekly SC semaglutide 1 mg reduced the risk of adverse kidney and CV outcomes in T2DM and CKD[29]
  - consider if glycaemic and weight management goals are not met and/or K+ >5.0 mmol/l[18,19,21]
  - deprescribe any DPP4 inhibitor if initiating semaglutide[18]
  - do not switch to semaglutide if an existing GLP-1 RA with proven CV benefit is achieving goals (e.g. liraglutide or dulaglutide).

5. Holistic Care
- Opportunistically check FBC/HbA1c/lipids/LBTs/weight/WHHR/BP at the same time as checking U&E and ACR, to support holistic interventions (see the Primary Care Hacks T2DM CVRM checklist, as well as the Primary Care Hacks on lipid management and LBTs)
- Offer vaccinations according to the Green Book.[30,31]

Lifestyle and Dietary Modification
- Encourage **weight loss** and **smoking cessation**[3,4,32]
- Advise on **salt restriction** (ideally <2 g of sodium per day, equating to <5 g of sodium chloride)[3,4,6,32]
- If gastric protection is required, consider **H2RAs** over PPIs, as PPI use has been associated with increased risk of nephritis and progression of CKD[33,34]
- Avoid **NSAIDs**[3–6]
- Promote **exercise** of at least 150 minutes per week[3,4,32]
- Kidney Care UK has produced a helpful patient information leaflet to support people living with CKD.

Figure 1: Managing Acute Changes in Kidney Function Related to RAASis[3,4,21,35]

	Serum K+ (mmol/l) 5.5–6.1	6.2–6.4	≥6.5
Unwell and/or ≥50% sCr rise	Consider[1] hospital referral according to clinical circumstances, likely cause, and risk of deterioration **Suspend RAASi**[2,3]	**Urgent hospital referral** **Suspend RAASi**[3,5]	**Urgent hospital referral**
Well and <50% sCr rise	**Maintain RAASi**[2,3] **dose** **Repeat K+ within 14 days** If repeat K+ is: • ≤5.5: optimise RAASi[2,3] dose • 5.6–5.9: maintain RAASi[2,3] dose • 6.0–6.4: suspend RAASi[2,3] and discuss initiation of K+ binders with Renal team[3,4] • ≥6.5: **consider hospital referral**	**Halve RAASi**[3,5] **dose** **Repeat K+ within 7 days** If repeat K+ is: • ≤5.5: optimise RAASi[2,3] dose • 5.6–5.9: maintain RAASi[2,3] dose • 6.0–6.4: suspend RAASi[2,3] and discuss initiation of K+ binders with Renal team[3,4] • ≥6.5: **consider hospital referral**	**Consider hospital referral**

1. Note: RAASis include ACEIs, ARBs, potassium-sparing diuretics, and MRAs.
2. Suspend finerenone if K+ ≥5.5 mmol/l, restarting at 10 mg when K+ is <5.0 mmol/l.
3. NICE endorses potassium binders, as they enable RAASi use in those not on dialysis.
4. See NICE TA599 and TA621. Note: that K+ binders are contraindicated in people with a history of bowel obstruction, major GI surgery, or a swallowing disorder.

Table based on the authors' clinical experience and interpretation of clinical guidance.

Figure 2: SGLT2i Initiation in CKD

eGFR (ml/min/1.73 m²)	uACR (mg/mmol) <20	≥20
≥60	Suggested in T2DM	Recommended
45–60	Suggested in T2DM	Recommended
20–45	Recommended	Recommended
<20	Suggested[F]	Suggested[F]
Dialysis	Not recommended[G]	

Footnotes
[A] BP target can be relaxed and individualised if the patient cannot tolerate SBP <130 mmHg or because of other factors, e.g. frailty, reduced life expectancy, syncope.[3,4,12] NICE recommends targets of 120–139 mmHg (SBP) and <90 mmHg (DBP) if ACR <70 mg/mmol, and targets of 120–129 mmHg and <80 mmHg if ACR ≥70 mg/mmol.[3,4] However, in various studies, intense SBP control has not resulted in compromise.[18,22,23]
[B] Provide advice on SICK day rules and review the SADMANS mnemonic (to include finerenone and GLP-1 RAs). See bit.ly/4bRbejF.[24,32]
[C] Counsel patient on the main side effects of SGLT2i use, including risk of UTIs, mycotic genital infections, Fournier's gangrene, DKA, foot disease, and dehydration.[24,32] Provide SICK day guidance, including a leaflet (bit.ly/4bRbejF).[24,32] **DO NOT** routinely check renal function after commencing an SGLT2i.[21,24] Consider the patient's hydration status and adjust/reduce diuretic or anti-BP doses if high risk of hypovolaemia.[24] As eGFR drops below 45 ml/min/1.73 m², SGLT2is' glycaemic efficacy reduces.[24] However, SGLT2is can be continued if tolerated until eGFR 20 ml/min/1.73 m².[24,32,33]
[D] Relative contraindications to SGLT2i use include immunosuppression (e.g. kidney transplantation, lupus, vasculitis) and ADPKD.[24]
[E] Do not discontinue SGLT2i if renal function deteriorates (eGFR <20 ml/min/1.73 m²), as long as the SGLT2i was initiated prior to this.[24]
[F] Initiate in people with uACR ≥20 mg/mmol, or ≥3 mg/mmol in people with diabetes.[18,21,23]
[G] Initiate in people with uACR ≥20 mg/mmol, or ≥3 mg/mmol in people with diabetes, if they were established on an SGLT2i before dialysis.[18,21,23]

ACEI=angiotensin-converting enzyme inhibitor; ACR=albumin to creatinine ratio; ADPKD=autosomal dominant polycystic kidney disease; AKI=acute kidney injury; ARB=angiotensin II receptor blocker; BP=blood pressure; CKD=chronic kidney disease; CV=cardiovascular; CVD=cardiovascular disease; DBP=diastolic blood pressure; DKA=diabetic ketoacidosis; DPP4=dipeptidyl peptidase-4; eGFR=estimated glomerular filtration rate; FBC=full blood count; GI=gastrointestinal; GLP-1 RA=glucagon-like peptide-1 receptor agonist; H2RA=histamine-2 receptor antagonist; Hb=haemoglobin; HbA1c=haemoglobin A1c; HF=heart failure; KDIGO=Kidney Disease: Improving Global Outcomes; K+=potassium; LBT=liver blood test; MRA=mineralocorticoid receptor antagonist; NG=NICE Guideline; NSAID=nonsteroidal anti-inflammatory drug; OD=once daily; OOH=out of hours; PPI=proton-pump inhibitor; RAASi=renin–angiotensin–aldosterone system inhibitor; RRT=renal replacement therapy; SADMANS=euthyroid/uveitis, ACEIs, diuretics, metformin, ARBs, NSAIDs, SGLT2is; SBP=systolic blood pressure; SC=subcutaneous; sCr=serum creatinine; SGLT2=sodium–glucose co-transporter-2 inhibitor; SICK=sugar, insulin, carbohydrate, ketones; T1DM=type 1 diabetes mellitus; T2DM=type 2 diabetes mellitus; U&E=urea and electrolytes; uACR=urine albumin to creatinine ratio; UTI=urinary tract infection; WHtR=waist-to-height ratio

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Diagnosis and Management of Heart Failure with Reduced Ejection Fraction

Medscape Primary Care Hacks

Authors: Dr Patricia Campbell, Consultant Cardiologist and Clinical Lead for Heart Failure, Northern Ireland; **Dr Eimear Darcy**, GP Partner, Grange Family Practice, Omagh; **Dr Kevin Fernando**, Portfolio GP, East Lothian, and Content Advisor, Medscape (email: kfernando@webmd.net)

Diagnosis and Management of Heart Failure with Preserved Ejection Fraction

Medscape Primary Care Hacks

Authors: Dr Patricia Campbell, Consultant Cardiologist and Clinical Lead for Heart Failure, Northern Ireland; **Dr Eimear Darcy**, GP Partner, Grange Family Practice, Omagh; **Dr Kevin Fernando**, Portfolio GP, East Lothian, and Content Advisor, Medscape (email: kfernando@webmd.net)

While awaiting specialist assessment for suspected HF, consider managing congestion with diuresis (e.g. furosemide 40–80 mg or double
pre-existing dose) and **initiating an SGLT2i.**

YOUR GUIDE TO SGLT2 INHIBITORS

Western Health and Social Care Trust

WHAT ARE SGLT2 INHIBITORS?

- SGLT2 Inhibitors are drugs for Type 2 Diabetes, Heart Failure, and Chronic Kidney Disease.
- They help your heart pump better, which may stop hospital admissions and help you live longer.
- They protect your kidneys and may stop or delay the need for kidney dialysis.
- In people with Type 2 Diabetes, they help reduce high blood sugar levels.

WHY AM I TAKING THIS DRUG?

- ☐ TYPE 2 DIABETES
- ☐ HEART FAILURE
- ☐ CHRONIC KIDNEY DISEASE

WHICH SGLT2I INHIBITOR AM I TAKING?

- ☐ DAPAGLIFLOZIN (Forxiga)
- ☐ EMPAGLIFLOZIN (Jardiance)
- ☐ CANAGLIFLOZIN (Invokana)

YOU TAKE THESE DRUGS ONCE A DAY

POTENTIAL SIDE EFFECTS



THRUSH



Reduce risk by washing and drying genitals daily. Cotton underwear also helps. Make sure to change your underwear daily. Thrush is a more common side effect in people with Type Two Diabetes.

DEHYDRATION



You can expect to pass more urine because of the way this drug works. This side effect helps protect you and can improve over time. Make sure you stay hydrated. Avoid excess alcohol as this makes dehydration worse.

SKIN INFECTION



A very bad skin infection of your private parts may occur. Contact a doctor urgently if this area becomes red, tender or swollen. This is very rare.

DIABETIC KETOACIDOSIS (DKA)



SGLT2 inhibitors can rarely cause too much acid in your blood. Contact a doctor if you are taking this drug and have any of the following: tummy pain, nausea, vomiting,fast breathing, drowsiness, confusion or a sweet smelling breath.

The risk is higher if you have a long time with very little food or water , have too much alcohol or are sick. This is a rare side effect, but is serious and can happen even if you do not have diabetes.

SICK DAY GUIDANCE (WHAT TO DO WITH YOUR DRUG IF YOU BECOME UNWELL)
If you become unwell with any of the following vomiting, diarrhoea, fever, sweats, shaking or reduced fluid intake PAUSE this drug but RESTART once you are well again (after 24 - 48hrs of eating and drinking normally).

PREGNANCY



Do not take if pregnant. Contact your doctor, nurse or pharmacist if planning to get pregnant.

FOOD CONTENT



Do not start a very low calorie, low carbohydrate or ketogenic diet when taking this drug. Contact your doctor, nurse or pharmacist if you want to start one. They all involve reduced intake of bread, potatoes, pasta or rice.

FURTHER INFORMATION

To learn more about possible side effects, take a look at the Patient Information Leaflet for your SGLT2 Inhibitor. You'll find this leaflet in the box with your medicine. If anything worries you or if you have questions, don't be shy - talk to your doctor, nurse, or pharmacist. They're here to help you understand your medicine better.

EMPAGLIFLOZIN DAPAGLIFLOZIN CANAGLIFLOZIN

t·r·e·n·d™ DIABETES ILLNESS ADVICE

NOTES

_____

_____

_____

Date I started taking SGLT2 Inhibitor: _____

HEALTHCARE PROFESSIONAL CONTACT DETAILS

Name:

_____

Contact Number:

_____

Western Health and Social Care Trust

Use of Liraglutide, Semaglutide, and Tirzepatide for Adults Living With Overweight and Obesity

Medscape Primary Care Hacks

Authors: Dr Kevin Fernando, Portfolio GP, East Lothian and Content Advisor, Medscape (email: kfernando@webmd.net); Dr Eimear Darcy, GP Partner, Grange Family Practice Omagh

Incretin Therapy	Indication	Standard Dose Escalation Schedule (in Weeks)[A]									Further Considerations (see also *Prescribing Considerations and Special Precautions for Use*)
		1	2	3	4	5–8	9–12	13–16	17–20	21–24	
Liraglutide (Saxenda®)[3][4]	Weight management, as an adjunct to a reduced-calorie diet and increased physical activity in adults with an initial BMI of: • ≥30 kg/m², or • 27–30 kg/m² in the presence of ≥1 weight-related comorbidity.[B][C]	0.6 mg (od)[D]	1.2 mg (od)[D]	1.8 mg (od)[D]	2.4 mg (od)[D]	3.0 mg (od)[D]					• No dose adjustment is required according to age, but therapeutic experience is limited in patients aged ≥75 years and use is not recommended in these patients • No dose adjustment is required in mild/moderate renal impairment (CrCl ≥30 ml/min) or mild/moderate hepatic impairment • Avoid in severe renal impairment (CrCl <30 ml/min), including ESRD • Not recommended in patients with severe hepatic impairment; should be used cautiously in mild/moderate hepatic impairment.
Semaglutide (Wegovy®▼)[5–8]	Weight management, as an adjunct to a reduced-calorie diet and increased physical activity in adults with an initial BMI of: • ≥30 kg/m², or • 27–30 kg/m² in the presence of ≥1 weight-related comorbidity.[B][E] To reduce the risk of major adverse CV events in adults with established CVD and BMI ≥27 kg/m².[B] as an adjunct to a reduced-calorie diet and increased physical activity.	0.25 mg (once weekly)				0.5 mg (once weekly)	1.0 mg (once weekly)	1.7 mg (once weekly)	2.4 mg (once weekly)[F]		• No dose adjustment is required according to age, but there is limited therapeutic experience in patients aged ≥85 years • No dose adjustment is required in mild/moderate/severe renal impairment; avoid in ESRD (eGFR <15 ml/min/1.73 m²) • No dose adjustment is required in hepatic impairment; exercise caution when prescribing in severe hepatic impairment.
Tirzepatide (Mounjaro®▼)[9][10]	Weight management, as an adjunct to a reduced-calorie diet and increased physical activity in adults with an initial BMI of: • ≥30 kg/m², or • 27–30 kg/m² in the presence of ≥1 weight-related comorbidity.[B][G]	2.5 mg (once weekly)				5 mg (once weekly)	7.5 mg (once weekly)[H]	10 mg (once weekly)[H]	12.5 mg (once weekly)[H]	15 mg (once weekly)[H]	• No dose adjustment is required according to age, but there are limited data available for patients aged ≥85 years • No dose adjustment is required in renal impairment (including ESRD) • No dose adjustment is required in hepatic impairment; exercise caution when prescribing in severe hepatic impairment.

Footnotes

[A] **Slower dose titration can be a useful strategy to aid long-term adherence**

[B] **NICE recommends lower BMI thresholds (usually, reduced by 2.5 kg/m²) for people of South Asian, Chinese, other Asian, Middle Eastern, Black African, or African–Caribbean family backgrounds**[6][7][14]

[C] NICE TA664[6] recommends liraglutide for overweight or obesity if it is prescribed by a specialist, multidisciplinary, tier-3 weight-management service and is provided according to the commercial arrangement for the drug. NICE recommends provision in patients with all of the following: a BMI of ≥35 kg/m²; nondiabetic hyperglycaemia (HbA$_{1c}$ of 42–47 mmol/mol or fasting plasma glucose of 5.5–6.9 mmol/l); and high risk of CVD, based on risk factors.[6] After 12 weeks of treatment with the 3.0 mg/day dose, treatment should be discontinued if patients have not lost ≥5% of their initial bodyweight.[3]

[D] If escalation to the next dose is not tolerated for 2 weeks consecutively, consider discontinuing treatment.[3]

[E] NICE TA875[7] recommends semaglutide if it is used for a maximum of 2 years, is prescribed within a specialist weight-management service providing multidisciplinary management, and is provided according to the commercial arrangement for the drug; NICE recommends provision in patients with ≥1 weight-related comorbidity and either a BMI ≥35 kg/m² or a BMI of 30–34.9 kg/m² if the patient meets the criteria for referral to specialist weight-management services in NICE NG246.[7][15] If weight loss is <5% of initial weight after 6 months of treatment, consider stopping semaglutide,[7] as the SELECT trial has demonstrated CV benefits of semaglutide irrespective of weight loss, it may be worth considering continuation independent of weight loss when used for CV indications![8]

[F] If semaglutide is not tolerated at 2.4 mg, maintain at 1.7 mg for 4 more weeks then re-escalate afterwards.[8]

[G] NICE TA1026[10] recommends prescribing tirzepatide for adults with a BMI of ≥35 kg/m² and ≥1 weight-related comorbidity;[9] if weight loss is <5% of initial weight after 6 months of treatment, consider stopping tirzepatide

[H] Individualise tirzepatide above 5 mg depending on individual treatment goals, increasing dose by 2.5 mg after ≥4 weeks at current dose; 5 mg, 10 mg, and 15 mg are the recommen

This table is based on the authors' interpretation of summaries of product characteristics and relevant guidance. HCPs are asked to report all suspected adverse drug reactions to products with a Bl

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For references and to view this Primary Care Hack online, go to medscape-uk.co/Hack-incretin

Brand Names of Incretin Therapies for Different Indications

Drug	Brand Name (Maximum Dose) for Weight Management	Brand Name (Maximum Dose) for T2D	Notes
Liraglutide[3][12][13]	**Saxenda®** (up to 3.0 mg daily)	**Victoza®** (up to 1.8 mg daily)	• Liraglutide is now available as a generic in the UK (for T2D)[13]—e.g. as **Diavic®**.
Semaglutide[5][14]	**Wegovy®▼** (up to 2.4 mg weekly)	**Ozempic®** (up to 2.0 mg weekly)	—
Tirzepatide[9][15]	**Mounjaro®▼** (up to 15 mg weekly)		• In the UK, tirzepatide is currently only branded as Mounjaro®▼ • In the US, the FDA has approved Mounjaro®▼ for T2D and Zepbound® for weight management.[13]

Practical Considerations—Injection, Storage, Driving

- Incretin therapies are injected **subcutaneously** in the **abdomen, thigh, or upper arm**
- Needles must be prescribed separately for liraglutide and tirzepatide when used for weight management;[12][9] 4 mm needles will usually be suitable
- **Injection sites should be rotated**
  - if the individual also injects insulin, they should inject the incretin therapy into a different site[16][17]
- Do not forget to issue a **sharps bin**—a 1.8-litre bin is usually adequate
- Store incretin therapies in a refrigerator at 2–8°C, away from the cooling element; do not freeze incretin therapies[16][17]
  - **liraglutide:**[3] after first use, store at <30°C (preferably, at 2–8°C in a refrigerator); pens should be discarded after 30 days, even if they still contain medication
  - **semaglutide:**[5] after first use, store at <30°C (preferably, at 2–8°C in a refrigerator) for up to 6 weeks
  - **tirzepatide:**[9] may be stored unrefrigerated for ≤30 days at <30°C
- **Incretin therapies have a negligible impact on the ability to drive or use machines**[12][13][9]
  - however, if using incretin therapies alongside insulin/SUs, the usual advice and precautions should be given to avoid hypoglycaemia when driving/operating machinery. Ensure adherence with DVLA requirements.[12][18][19]

Behavioural Modifications and Interventions

- **Consider recommending behavioural modifications to all people with overweight or obesity**[11][14–16]
  - offer a brief intervention to people living with overweight or obesity, using **ASK, ASSESS, ADVISE, AGREE, and ASSIST**; see also PHE guidance
  - in these discussions, be aware of weight bias, stigma, and how language matters
  - use specific conversation techniques that have been shown to support brief, effective, and well-received conversations about weight loss
- Adequate support of behavioural modifications, as well as mental health care, needs to be considered **during** and **before** incretin therapy initiation[11][15][17][18]
- Consider **multicomponent interventions**, involving behaviour modification strategies and motivational interviewing;[11][17][20][21] key areas to support include:[14][17][22]
  - **nutrition** (including eating behaviours and diet content)
  - **increased physical activity** (including maintenance of muscle mass)—discuss the importance of resistance training to aid preservation of muscle mass and function
  - **stress management**
  - **sleep health**
- **Set personalised goals** that are realistic and achievable[11][23]
  - use a **SMART** goal-setting framework[20]
- Behavioural modifications and comprehensive obesity care in primary care should focus on **whole health gain**, not just weight loss, as this approach has been shown to improve long-term weight and behavioural outcomes![24][25]
- **Be aware that mental illness can impact obesity management efforts;** screen patients for potential mental illnesses that need to be addressed.[26]

Women's Health and Incretin Therapies

- **Incretin therapies are not recommended during breastfeeding and pregnancy**[12][13][9]
  - **women of childbearing potential should use contraception**[27]
- For women planning pregnancy:[12][13][9]
  - liraglutide: discontinue before attempting to conceive
  - semaglutide: discontinue ≥2 months before attempting to conceive
  - tirzepatide: discontinue ≥1 month before attempting to conceive
- **Specific OCP advice for tirzepatide:**[9]
  - **women with a normal BMI:** no dose adjustment of OCP is required

Side Effects

- The side effects of incretin therapies can lead to nonadherence and discontinuation—in one study of GLP-1 RA use, 21.2% of people had discontinued therapy by 12 months and only 48.6% were adherent![28]
- **The most common adverse effects (prevalence ≥10%) are mostly GI in nature.**[12][13][9] GI side effects mostly occur during dose escalation, usually fade with time, and are typically mild/moderate in severity
  - examples include nausea, vomiting, diarrhoea, constipation, abdominal pain, abdominal distension, dyspepsia, flatulence, and belching[12][13][9]
- Hair loss (likely due to telogen effluvium; usually transient and reversible), fatigue, headache, dizziness, and a small increase in resting HR (around 3 bpm on average, and not clinically significant) can also commonly occur.[12][13][9][30–32]

Managing GI Side Effects

- **Incretin therapies should be used with caution in people with severe GI disease**, e.g. severe gastroparesis![12][13][33]
- **GI side effects are dose-dependent, so consider slower dose escalation or drug holidays** (temporary cessation of incretin therapy) for those who are struggling with GI side effects in the early weeks of therapy![12][33]
  - a lower maintenance dose can be considered for individuals unable to tolerate the usual maintenance dose![12][13][33]
- Advise patients reporting GI side effects to adopt the following mitigating strategies:[12][33]
  - eat slowly, stop eating as soon as you start to feel full, and avoid eating when not feeling hungry
  - eat smaller portion sizes and eat more frequently during the day, but avoid eating late in the day
  - maintain good hydration, aiming for ≥2–3 litres of fluids daily (not including alcohol)
    - limit intake of alcohol and fizzy drinks, especially if experiencing nausea or dyspepsia
  - avoid eating high-fat, ultra-processed, and spicy foods
  - increase fibre and water intake if experiencing constipation
  - consider short-term use of PPIs, antiemetics, laxatives, and antidiarrhoeal medications for those with disabling side effects
- Consider alternative causes of GI symptoms if persistent despite mitigation strategies, or if red-flag features are present.[12][33]

Minimising Occurrence/Severity of GI Adverse Effects: General Guidance



Gorgojo-Martínez J, Mezquita-Raya P, Carretero-Gómez J et al. Clinical recommendations to manage gastrointestinal adverse events in patients treated with glp-1 receptor agonists: a multidisciplinary expert consensus. *J Clin Med* 2022; **12** (1): 145.

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Last updated: May 2025.

Advice for Missed Doses[3][5][9][34]

	Day of Usual Administration	Number of Days After Missed Dose						
		Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	
Semaglutide	Missed dose	Administer catch-up dose as soon as possible (within 5 days)						Skip dose and administer next dose on usual day
Tirzepatide	Missed dose	Administer catch-up dose as soon as possible (within 4 days)						Skip dose and administer next dose on usual day

After a dose of semaglutide or tirzepatide is missed (regardless of whether a catch-up dose is taken), individuals can then resume their regular once-weekly dosing schedule.

Note: the time between any two doses must always be ≥72 hours.

	0–12 hours after a missed dose	12–24 hours after a missed dose
Liraglutide	Administer catch-up dose as soon as possible	Skip dose and administer next dose on usual day

If >3 days have elapsed since the last dose, reinitiate liraglutide at 0.6 mg daily and follow the usual dose escalation schedule.[34]

Special Considerations for People With T2D and Overweight/Obesity

- Incretin therapies are not currently licensed for use in people with T1D and overweight/obesity![12][13][9][14]
- Deprescribe any DPP4 inhibitor if initiating an incretin therapy
- Based on the findings of a systematic review and meta-analysis, people **without** T2D may achieve significantly greater mean weight loss with GLP-1 RAs than people **with** T2D![43]
- **Risk of hypoglycaemia is low if the incretin therapy is not used alongside insulin or SUs**[12][13]
  - people with T2D taking insulin or SUs may need to lower the dosage of these medications initially when starting incretin therapies, to reduce the risk of hypoglycaemia![12][13][14]
  - SMBG is necessary when adjusting the dose of SU or insulin, and a stepwise approach to insulin reduction is recommended![11]
- **DKA risk**
  - the MHRA (2019)![41] warns of reports of DKA when insulin is rapidly reduced or discontinued alongside GLP-1 RAs
  - any dose reduction of insulin should be done in a stepwise manner, with careful SMBG![11]
- **Retinopathy—be aware that pre-existing DR may be worsened if HbA$_{1c}$ is rapidly lowered on initiation or escalation of incretin therapy**
  - use all incretin therapies with caution in patients who have DR requiring active ophthalmology follow up, suboptimal glycaemic control (HbA$_{1c}$ ≥88 mmol/mol), and are currently being treated with insulin![42–44]
  - ensure that all people living with T2D being considered for incretin therapies are up to date with retinal screening.[42–44]

Prescribing Considerations

- Incretin therapies can be administered at any time of the day, with or without meals![12][13][9]
  - injections of semaglutide and tirzepatide should be scheduled on the same day each week, but the time can be varied![35]
  - if a change of day is required for semaglutide or tirzepatide, **the time between the two doses during transition must be ≥3 days (≥72 hours)**![35]
- **All incretin therapies delay gastric emptying and therefore have the potential to impact the absorption of coadministered oral medications;** however, no dose adjustments are required for most oral medications![12][13][9]
  - if individuals are taking oral medications with a **narrow therapeutic index** (e.g. digoxin, lithium, warfarin), closer monitoring may be warranted according to clinical judgement![12][13]
  - **specific OCP advice is required for tirzepatide** (see *Women's Health and Incretin Therapies*)![9]
- **Sick day guidance** may be required![12][13][14] during any intercurrent dehydrating illness (e.g. diarrhoea or vomiting), a temporary pause of incretin therapy may be required to avoid worsening of any GI or other symptoms
  - the incretin therapy can be restarted when the patient is eating and drinking as normal and recovered from illness
- Incretin therapies can be used as adjunctive treatment after bariatric surgery for those with suboptimal weight loss or weight regain, offering a viable alternative to revision surgery![37] However, this approach should be discussed with a specialist in bariatric surgery and medicine
- **Contraindications:**
  - hypersensitivity to the active substance or any of the excipients present in the incretin therapy![12][13]
  - according to the US SPCs, all incretin therapies are contraindicated in individuals with MEN2 or with a personal or family history of MTC![14]
    - however, a 2023 systematic review and meta-analysis found that **semaglutide use in RCTs and real-world studies was not associated with an increased risk of any types of cancer (including pancreatic and thyroid cancer).**[38]

Special Precautions for Use

Adverse Effect	Frequency	Notes
Acute pancreatitis[12][13][9]	≤1% (uncommon)	• Use with caution in people with a history of pancreatitis • Discontinue if pancreatitis is suspected.
Acute gallbladder disease (cholelithiasis, cholecystitis)[12][13][9]	≤1% (uncommon)[A]	• Significant or rapid weight loss can increase the risk of gallstones![45] • If gallbladder disease is suspected, consider gallbladder imaging and appropriate clinical follow up as indicated.[45]
Pulmonary aspiration[12][13][9]	—	• Cases of pulmonary aspiration have been reported in people undergoing GA or deep sedation who are receiving incretin therapies • Before such procedures, the increased risk of residual gastric content (due to delayed gastric emptying) should be considered • UK societies have developed a consensus statement giving guidance on the perioperative management of incretin therapies: individuals should continue to take their GLP-1 and GIP RAs throughout the perioperative period.

[A] Cholelithiasis is listed as a common (≤10%) adverse effect of semaglutide[5] and liraglutide.[3]

Follow Up

- **Provide long-term, multicomponent, multimodal, multidisciplinary follow up to all people living with overweight or obesity**![11][17]
- **Set personalised goals that:**[11][17]
  - emphasise long-term, realistic, sustained weight loss
  - promote weight maintenance and prevention, improvement, and resolution of obesity-related diseases, disorders, and complications
- Consider agreeing a realistic 'best weight' (i.e. a weight that a person can achieve and maintain in the context of their life circumstances)![20]
- **Evaluation of response to incretin therapies is crucial:**
  - consider intensification of therapy or additional therapeutic options (e.g. metabolic surgery) if individualised goals are not achieved![11][17]
  - consider stopping incretin therapies if <5% of the initial weight has been lost after 6 months of the highest-tolerated dose of tirzepatide or semaglutide, or after 12 weeks of the highest tolerated dose of liraglutide![6][7][9]
  - ensure appropriate/optimal prescribing; consider deprescribing medications that may no longer be indicated due to the health benefits of weight loss (e.g. antihypertensives)
  - consider reassessing goals of therapy during treatment course![17]
  - long-term use of pharmacotherapy is recommended
- **Explain that regular physical activity is beneficial for weight maintenance** and improves cardiometabolic risk factors, health-related quality of life, and mood disorders![14]
  - in weight management interventions, aerobic and resistance exercise supports improvements in cardiorespiratory fitness, mobility, strength, and **muscle mass**; support strategies to minimise muscle loss![39]
  - resistance training in particular can promote weight maintenance and modest increases in muscle mass![40]
- **Set a defined timescale for follow up**
  - consider regular monitoring, as clinically indicated, to assess obesity and its related diseases, disorders, and complications (consider using the *Type 2 Diabetes CVRM Review Checklist*)![17][18]
  - remember that managing obesity-related diseases, disorders, and complications is part of obesity management![11]
- **Be aware of the risks of weight cycling on cardiometabolic health and adopt strategies that focus on sustained changes that maintain healthy habits over time**![11][14]
- Be aware that incretin therapies **may increase the risk of mental health disorders and suicidal behaviours.**[12] Assess mental health in all individuals on incretin therapies and manage as clinically indicated
  - a recent systematic review and meta-analysis found that, in individuals living with overweight/obesity and/or diabetes, GLP-1 RA treatment is not associated with an increased risk of psychiatric adverse events or worsening depressive symptoms compared to placebo and is associated with improvements in QoL, restrained eating, and emotional eating behaviour![26]

BMI=body mass index; **bpm**=beats per minute; **CrCl**=creatinine clearance; **CV**=cardiovascular; **CVD**=cardiovascular disease; **CVRM**=cardiovascular–renal–metabolic; **DKA**=diabetic ketoacidosis; **DPP4**=dipeptidyl peptidase-4; **DR**=diabetic retinopathy; **DVLA**=Driver & Vehicle Licensing Agency; **eGFR**=estimated glomerular filtration rate; **ESRD**=end-stage renal disease; **FDA**=Food and Drug Administration; **GA**=general anaesthesia; **GI**=gastrointestinal; **GIP**=glucose-dependent insulinotropic polypeptide; **GLP-1**=glucagon-like peptide-1; **HbA$_{1c}$**=glycated haemoglobin; **HCP**=healthcare professional; **HR**=heart rate; **HRT**=hormone-replacement therapy; **LTC**=long-term condition; **MEN2**=multiple endocrine neoplasia type-2; **MHRA**=Medicines and Healthcare products Regulatory Agency; **MTC**=medullary thyroid carcinoma; **NG**=NICE Guideline; **OCP**=oral contraceptive pill; **od**=once daily; **PHE**=Public Health England; **PPI**=proton pump inhibitor; **QoL**=quality of life; **RA**=receptor agonist; **RCT**=randomised controlled trial; **SMART**=Specific, Measurable, Achievable, Rewarding, Timely; **SMBG**=self-monitoring of blood glucose; **SPC**=summary of product characteristics; **SU**=sulfonylurea; **TA**=Technology Appraisal; **T1D**=type 1 diabetes; **T2D**=type 2 diabetes.

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For references and to view this Primary Care Hack online, go to medscape-uk.co/Hack-incretin

Last updated: May 2025.

Identification and Management of People with MASLD and MASH in Primary Care

Medscape UK X Guidelines
Primary Care Hacks

Author: Dr Kevin Fernando, GP Partner, North Berwick Health Centre; Content Advisor, Medscape Global and UK. Email: kfernando@webmd.net

What Is MASLD?[1–7]

- There has been recent international consensus to rename nonalcoholic fatty liver disease (NAFLD) to improve awareness and patient identification and reduce stigma;[1] using this terminology, the EASL, the EASD, and the EASO produced an updated guideline in 2024[2]
- NAFLD is now termed **metabolic dysfunction-associated steatotic liver disease (MASLD)**[1]
  - MASLD encompasses individuals who have **hepatic steatosis and at least one cardiometabolic risk factor**[2]
- **Metabolic dysfunction-associated steatohepatitis (MASH)** replaces nonalcoholic steatohepatitis (NASH).[1,3] MASH is defined by inflammation of hepatocytes and carries a risk of progression to fibrosis, cirrhosis, and HCC[3,4]
- **MetALD** describes individuals with MASLD who consume more than recommended amounts of alcohol per week (defined as 3.75–7.50 units/day [30–60 g/day] in men and 2.50–6.25 units/day [20–50 g/day] in women)[2]
  - for all adults in the UK, the recommended alcohol intake is ≤14 units/week (i.e. 112 g/week, or 2.0 units/day [16 g/day]), best spread evenly over ≥3 days[5]
- **MASLD is primarily a metabolic disease heavily influenced by lifestyle factors, and is the liver's manifestation of the MetS alongside hypertension, insulin resistance and dysglycaemia, dyslipidaemia, and obesity/increased waist circumference.**[6,7]

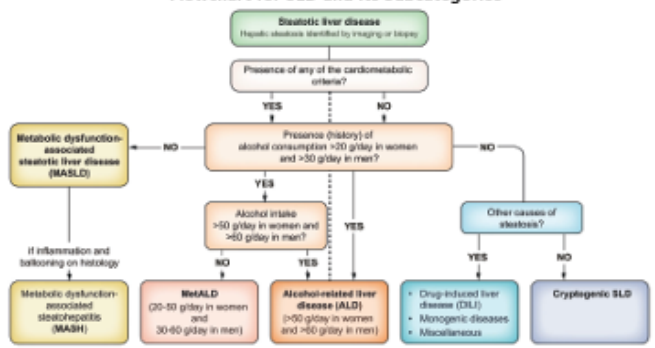
Cardiometabolic Risk Factors[2,8,9]

- BMI ≥25 kg/m² (23 kg/m² if high-risk ethnic minority) or waist circumference ≥94 cm (37.0 inches) for men (≥90 cm [35.4 inches] in men of South Asian or Chinese ethnicity, or ≥85 cm [33.5 inches] in men of Japanese ethnicity) or ≥80 cm (31.5 inches) for women of all ethnicities
- HbA$_1$ 39–47 mmol/mol, fasting plasma glucose of 5.6–6.9 mmol/l (100–125 mg/dl), or 2-hour plasma glucose during OGTT of 7.8–11 mmol/l (140–199 mg/dl), or established T2D
- BP ≥130/85 mmHg or antihypertensive drug treatment
- Plasma triglycerides ≥1.70 mmol/l or lipid-lowering treatment
- Plasma HDL-cholesterol ≤1.0 mmol/l in men, ≤1.30 mmol/l in women, or lipid-lowering treatment.

Secondary Causes of Hepatic Steatosis[2,8,10]

- Drug-induced liver injury, e.g. amiodarone, methotrexate, tamoxifen, and corticosteroids
- Endocrine disorders, such as hypothyroidism, PCOS, panhypopituitarism, or growth hormone deficiency
- Nutrient deficiency or malnutrition, such as from acute weight loss due to bariatric surgery or fasting, total parenteral nutrition, or small intestinal bacterial overgrowth
- Chronic hepatitis C virus infection.

Flowchart for SLD and Its Subcategories[2]



© European Association for the Study of the Liver, European Association for the Study of Diabetes, European Association for the Study of Obesity. EASL–EASD–EASO clinical practice guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *J Hepatol* 2024; **81** (3): 492–542. Reproduced with permission.

Note: 50 g of alcohol equates to 6.25 units, and 60 g equates to 7.5 units.

How Common and Serious Is MASLD?[2,11–13]

- MASLD is now the most common liver disorder in Western countries, and has been estimated to affect up to 30% of adults in the UK[11,12]
  - MASH has been estimated to affect up to 5% of the UK population[11]
- MASLD (specifically progressive MASH) is the fastest growing indication for liver transplantation in Western countries[13]
- MASLD is also associated with an increased prevalence and incidence of CVD[2]
  - CVD is a more common cause of death than liver disease in MASLD[2]
- MASLD is highly prevalent in people living with T2D.[2]

Screening for MASLD in Primary Care[2,14]

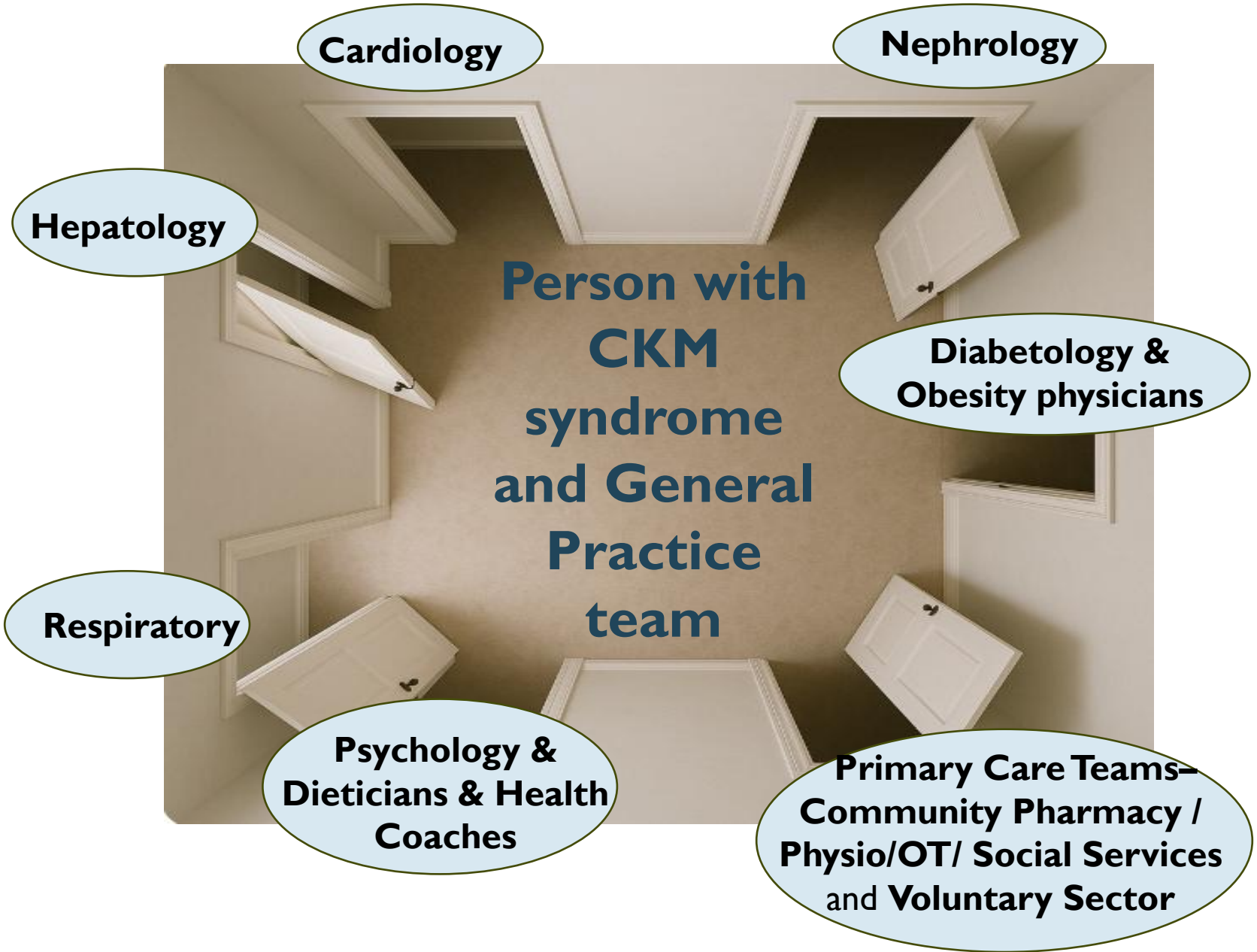
- The appearance of steatosis on abdominal USS is operator-dependent and a normal USS does not rule out MASLD[14]
- Consider case-finding strategies for MASLD with liver fibrosis in those who have abnormal liver enzymes, cardiometabolic risk factors, and/or incidental radiological signs of hepatic steatosis[2]
- The EASL, the EASD, and the EASO recommend looking for MASLD with liver fibrosis in individuals with one or more of the following:[2]
  - T2D
  - abdominal obesity and ≥1 additional metabolic risk factor
  - abnormal liver blood test results.

Useful Resources

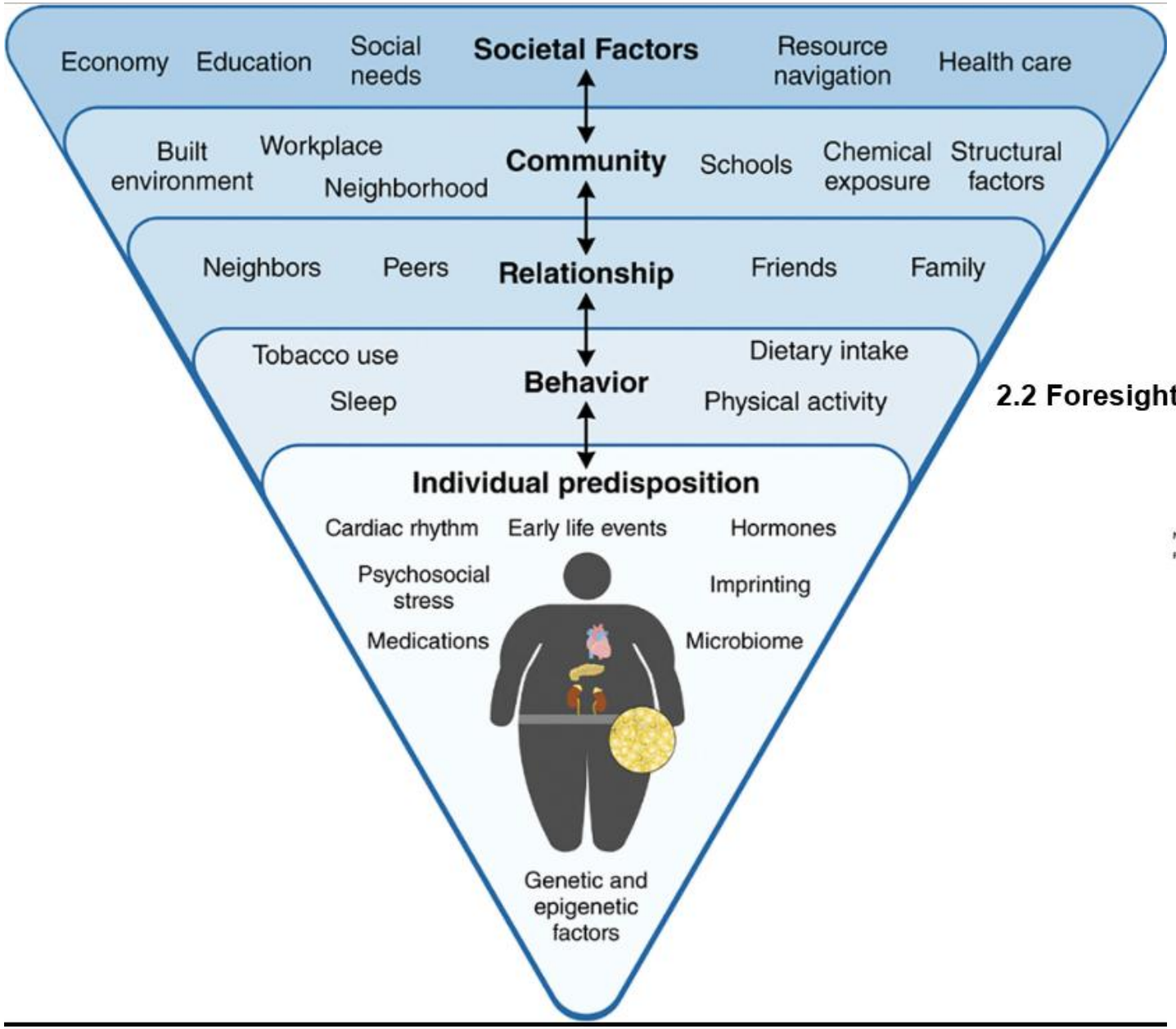
- The British Liver Trust:
  - *Coffee consumption and the liver—the potential health benefits*
  - *MASLD, NAFLD, and fatty liver disease*
- The EASL:
  - *Non-alcoholic fatty liver disease (NAFLD): how you can reduce the risk for your liver and for other health issues?*

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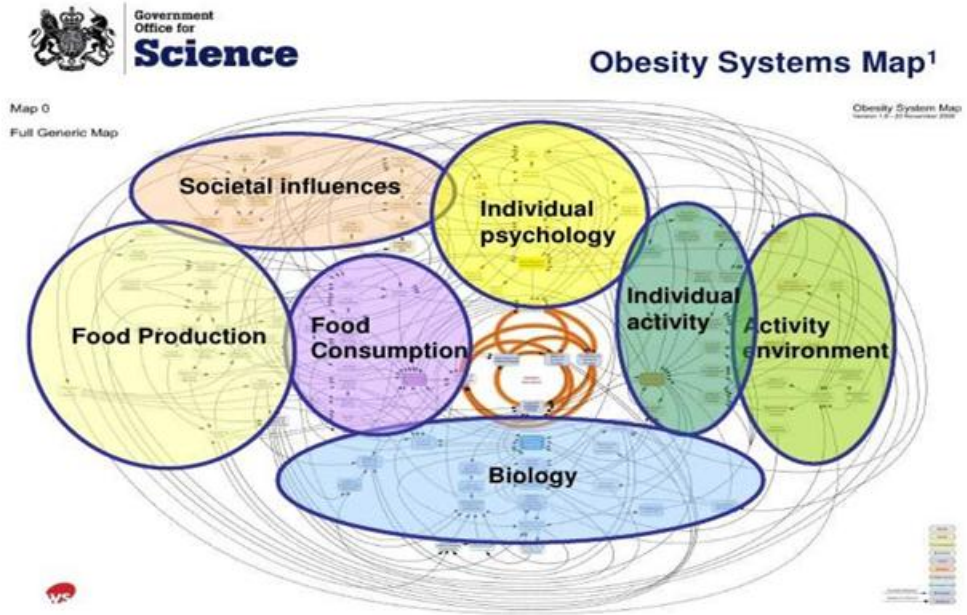
NHS General Practice CKM care



- **Whole person advanced generalist care :**
  - **Co-ordinated**
  - **Comprehensive**
  - **Continuous**
  - **Person centred**
- **Longitudinal life span care**
- **MDT care – stronger together**
- **Agile care –open doors of engagement with and back up from other teams when required.**



2.2 Foresight Obesity Map-Causes of Obesity



Ndumele CE, Neeland IJ, Tuttle KR, Chow SL, Mathew RO, Khan SS, et al. A Synopsis of the Evidence for the Science and Clinical Management of Cardiovascular-Kidney-Metabolic (CKM) Syndrome: A Scientific Statement From the American Heart Association. Circulation. 2023;148(20):1636–64.

Dr Darcys Instagram PRESCRIPTION FOR BETTER HEALTH

Patient Name: _____
Date: _____

℞ ADVICE — TAKE DAILY
☑ **Do not smoke.** Stop if you can. Cut down if you can't.
☑ **Eat a balanced diet.** Include plenty of fruit and vegetables.
☑ **Keep physically active.** Choose activity suited to your ability.
☑ **Manage stress.** Make time to relax and talk things through.
☑ **Drink alcohol in moderation**
☑ **Protect yourself from the sun** Cover up and avoid sunburn.
☑ **Practice safer sex**
☑ **Attend cancer screening when invited**
☑ **Be safe on the roads** Follow the Highway Code.
☑ **Learn First Aid** Airway • Breathing • Circulation

Dosage: Repeat: Daily — For Life
Warnings: No known side effects.
Benefits increase with regular use.

Prescriber:
Dr Darcy Health Coach and Sir Liam Donaldson
Chief Medical Officer for England (1999)
Signature: _____

From **Saving Lives: Our Healthier Nation** *(Department of Health, 1999*

Not wrong , continue to shout but be aware conveying advice unlikely to help those who have most to gain.

Dr Darcys NHS PRESCRIPTION FOR BETTER HEALTH

Patient Name: _____
Date: _____

℞ ADVICE — TAKE DAILY
☑ **Do not be poor.** If you can , stop. If you can't , try not to be poor for long.
☑ **Don't live in a deprived area.** If you do, move.
☑ **Don't be disabled or have a disabled child.**
☑ **Don't work in a stressful low-paid manual job.**
☑ **Don't live in damp, low quality housing or be homeless.**
☑ **Be able to afford to pay for social activities and annual holidays.** .
☑ **Don't be a lone parent.**
☑ **Claim all benefits to which you are entitled (even if you are illiterate).**
☑ **Be able to afford own a car.**
☑ **Use education as an opportunity to improve your socio – encomonic position.**

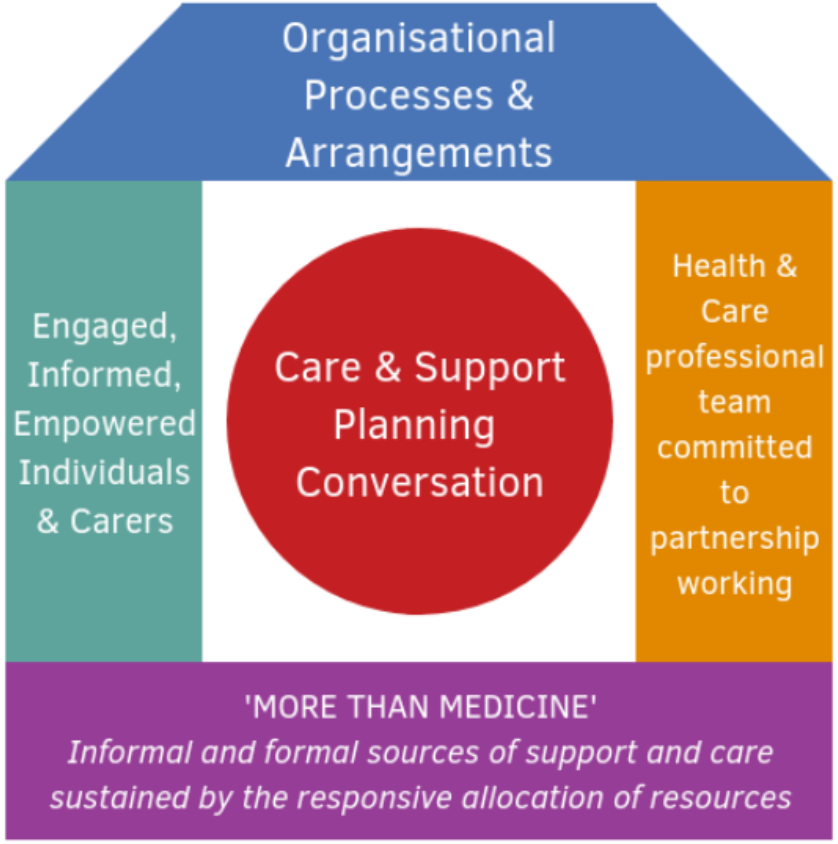
Dosage: Repeat: Daily — For Life
Warnings: No known side effects. Benefits increase with regular use.

Prescriber:
Dr Darcy and David Gordon , Universty of Bristol
Signature: _____

My Choices are constrained by context. We all meet our patients where they are and personalise care

1) *1999 by Sir Liam Donaldson*, then Chief Medical Officer of England, from the Department of Health's *Saving Lives: Our Healthier Nation* campaign. Source: DoH (1999) Saving Lives: Our Healthier Nation. London: The Stationery Office 2) https://www.bristol.ac.uk/poverty/healthinequalities.html

CKM WILL AFFECT OUR NHS HOUSES OF CARE, GENERAL PRACTITIONER AND GENERAL PRACTICE TEAM **LEADERSHIP** IS NEEDED



'The interdisciplinary care would involve **primary care physicians** working together with cardiovascular, nephrology, and endocrine colleagues to deliver optimal results.'

Our skills as General Practitioners and valued General Practice Team members in the narrative ahead is **indispensable.**

- https://www.alliance-scotland.org.uk/past-work-and-projects/scotlands-house-of-care/the-house-of-care-model/
- Farooq, W., & Kim, A. S. (2024). Cardiac, Renal, and Metabolic Overlap: A Public Health Crisis?. *Kidney News*, *16*(3), 25-25. Retrieved Feb 25, 2025, from https://www.kidneynews.org/view/journals/kidney-news/16/3/article-p25_15.xml

Take homes from a CKM/Obesity GP

- CKM syndrome - a **dynamic continuum** that **changes over time.**
- **Contextualise. Prioritise. Personalise.**
- Shared decision making - **with** the person , **not to** the person. **BEHAVIOURIAL SCIENCE IS KEY.**
- **Ordering** and **pacing** of interventions – a personalised marathon.
- **Life long treatments and interventions** ; **not** burst therapies or approaches.
- **Combination therapy and interventions =** organ and person **protection**.
- General Practice MLTC infrastructure **will evolve** from current set up to a **de-sioled format.**
- This will take a **team** – if you are doing this as a lone professional – **stop doing it alone.**

We are stronger together

https://www.alliance-scotland.org.uk/past-work-and-projects/scotlands-house-of-care/the-house-of-care-model/

CODE CODE CODE CODE
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General Practice Cardiovascular Kidney Metabolic Review

VALUE LIES IN A COMPREHENSIVE AND COMPREHENSIBLE APPROACH TO A COMPLEX PROBLEM

Larkin H. *JAMA*. 2023;330(21):2042–2043.

The Lightblub



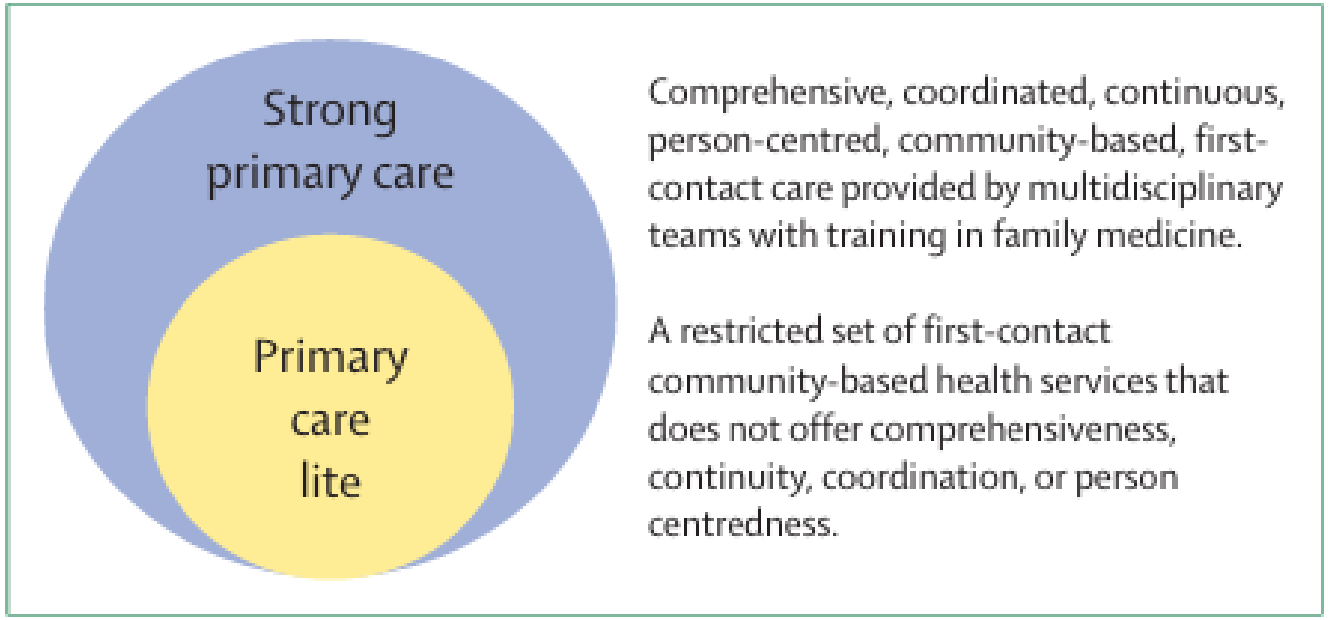


Figure: Strong and lite forms of primary care

THE LANCET
Primary Care

Models of global primary care post-2030

Luke N Allen, Kumanan Rasanathan, Robert Mash, Manuela Villar Uribe, Viviana Martinez-Bianchi, Michael Kidd

Lets get back to the basics, and get them right.

Journal of *Clinical Medicine*

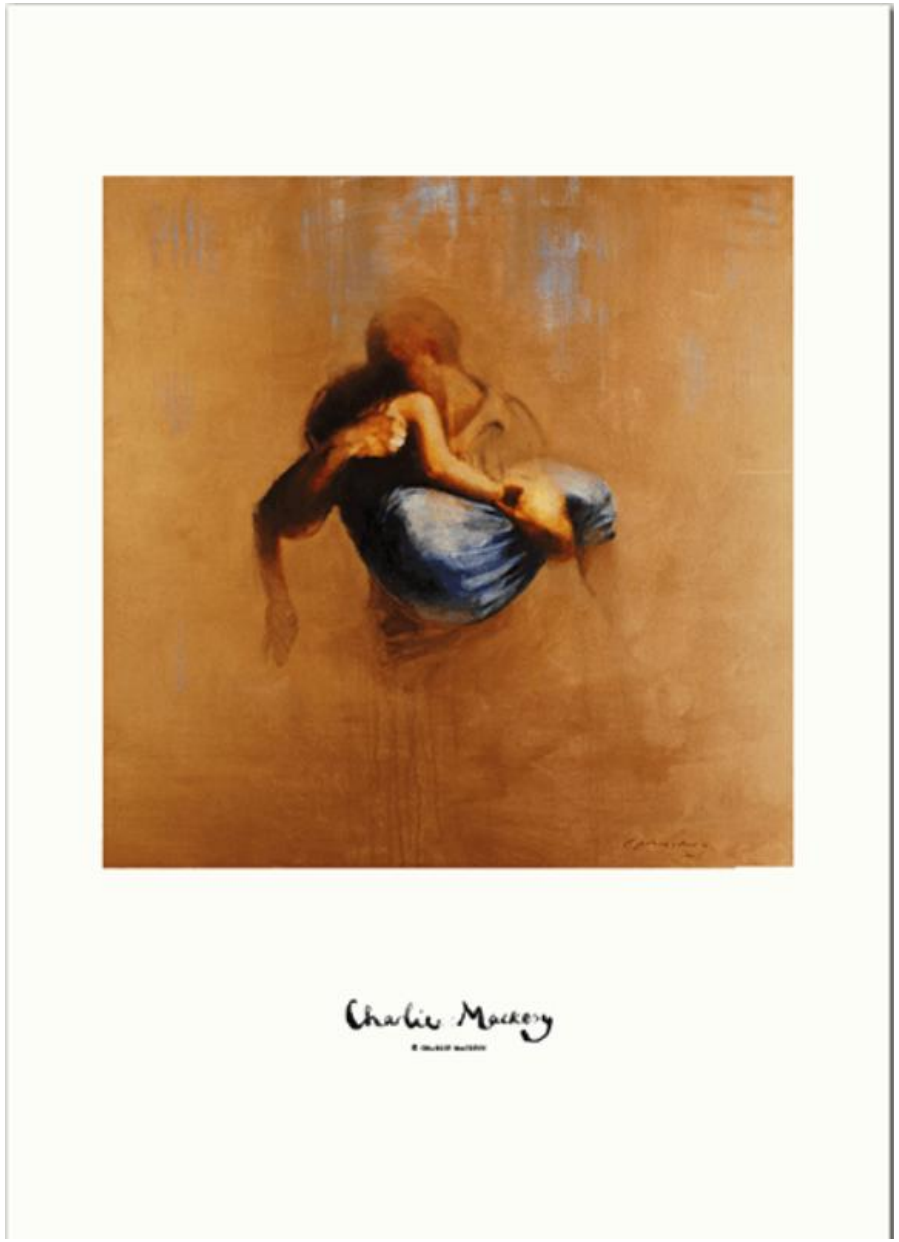
MDPI

Review

Cardiovascular–Kidney–Metabolic Syndrome: A New Paradigm in Clinical Medicine or Going Back to Basics?

Victoria Mutruc [1,2,*], Cristina Bologa [1,2], Victoriţa Şorodoc [1,2], Alexandr Ceasovschih [1,2], Bianca Codrina Morărașu [1,2], Laurenţiu Şorodoc [1,2], Oana Elena Catar [3] and Cătălina Lionte [1,2,*]

Mutruc, V.; Bologa, C.; S, orodoc, V.; Ceasovschih, A.; Morăras,u, B.C.; S, orodoc, L.; Catar, O.E.; Lionte, C. Cardiovascular– Kidney–Metabolic Syndrome: A New Paradigm in Clinical Medicine or Going Back to Basics? J. Clin. Med. 2025, 14, 2833. https://doi.org/ 10.3390/jcm14082833



General Practice #CKM care is the enactment of humanity.

Collectivism
Compassion
Care

The responsibility to curate it together lies with us.

#teamGP

Art – Charles Mackesy