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QFMCRC: Qatar Family Medicine Clinical Reasoning Cycle: An Integrated Clinical Reasoning Cycle for Family Medicine: Bridging Diagnosis, Management, and Reflection

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Editorial

Integrating Innovation, Humanity, and Health Across the Lifespan

The current issue of the journal brings together five diverse yet remarkably interconnected contributions that collectively illuminate one of the defining challenges of contemporary healthcare: how to harness scientific and technological progress while preserving the human dimensions of medicine. The papers span clinical reasoning, women's health, dementia, digital culture and identity, and artificial intelligence. Although they originate from different disciplines, they share a common concern—the need to balance innovation with person-centered care, ethical responsibility, and societal well-being.

The issue opens with an important contribution by Salem and colleagues, who propose the Qatar Family Medicine Clinical Reasoning Cycle (QFMCRC), an integrated framework designed to strengthen clinical decision-making in family medicine. Recognizing that clinical reasoning extends beyond diagnosis to encompass management planning, follow-up, and reflective practice, the authors present a practical cyclical model that mirrors the realities of primary care. Their work highlights the increasing need for structured reasoning frameworks capable of supporting clinicians facing complex, undifferentiated presentations within modern healthcare systems. At a time when healthcare is becoming increasingly data-rich and technologically driven, the QFMCRC reminds us that effective clinical reasoning remains the cornerstone of safe and compassionate patient care.

The second article shifts attention to an equally important but often underappreciated stage of life. In their comprehensive review of Lifestyle Medicine as a Therapeutic Approach to Menopause, Ahmed Abdulrahman emphasizes that menopause should not merely be viewed as an endocrine transition but as a critical opportunity for preventive healthcare and healthy aging. By examining the six pillars of lifestyle medicine—including nutrition, physical activity, restorative sleep, stress management, avoidance of harmful substances, and social connection—the review presents a compelling argument for holistic, patient-centered interventions that extend beyond pharmacological treatment. The paper reinforces the growing recognition that lifestyle medicine has become an essential component of modern clinical practice and public health strategy.

Aging and cognitive health remain among the most pressing healthcare challenges worldwide. In this issue, Abyad provides a comprehensive review of other dementias, including normal pressure hydrocephalus, Creutzfeldt–Jakob disease, Huntington's disease, HIV-associated dementia, and toxin- and infection-related cognitive disorders. The review highlights an important clinical message: not all dementias are alike, and some are potentially reversible if recognized early. The paper illustrates how advances in neuroimaging, molecular diagnostics, biomarkers, genetics, and precision medicine are transforming diagnostic pathways while simultaneously reinforcing the enduring importance of careful clinical evaluation. Particularly relevant is the emphasis on treatable causes of cognitive decline, a reminder that diagnostic vigilance remains essential even as technological capabilities expand.

The next contribution moves beyond traditional clinical medicine into the broader social determinants of health and well-being. Elghblawi's thought-provoking exploration of beauty, identity, and the psychology of perfection in the age of digital influence examines the profound effects of social media, cosmetic culture, and influencer-driven ideals on self-image and mental health. The paper raises critical concerns regarding body dysmorphic disorder, unrealistic beauty standards, repeated aesthetic interventions, and the ethical responsibilities of healthcare practitioners. Its relevance extends beyond dermatology and cosmetic medicine; it touches fundamental questions about identity, authenticity, psychological resilience, and the impact of digital environments on human behavior. As healthcare professionals increasingly encounter patients shaped by online narratives and social comparison, understanding these dynamics becomes essential.

The issue concludes with a timely discussion of one of the most transformative developments of our era: artificial intelligence. The paper on AI, copyright, intellectual property, and human knowledge explores both the extraordinary potential and the substantial risks associated with emerging AI technologies. It addresses concerns regarding ownership of knowledge, protection of intellectual property, privacy, employment, child welfare, and the ethical use of technological innovation. Importantly, the paper challenges readers to consider whether society is developing appropriate safeguards to ensure that AI serves human advancement rather than undermining human creativity, autonomy, and social structures. These questions are particularly relevant for healthcare, where AI is

increasingly influencing diagnosis, research, education, and clinical decision-making.

Viewed collectively, the papers in this issue reveal several unifying themes. First, they underscore the continuing importance of human judgment, whether in clinical reasoning, lifestyle counseling, dementia diagnosis, psychological assessment, or AI governance. Second, they emphasize the value of holistic approaches that recognize the complex interplay between biological, psychological, social, and technological influences on health. Third, they remind us that scientific progress must always be accompanied by ethical reflection and a commitment to preserving human dignity. As medicine enters an era characterized by unprecedented technological advancement, increasing longevity, and rapidly evolving social environments, the challenge facing clinicians, researchers, and policymakers is not merely to innovate but to innovate wisely. The contributions presented in this issue offer valuable insights into how that balance may be achieved. Together, they encourage a vision of healthcare that is evidence-based yet compassionate, technologically advanced yet ethically grounded, and scientifically rigorous yet profoundly human.

We commend the authors for their thoughtful and diverse contributions and trust that readers will find in these pages both practical guidance and intellectual inspiration for navigating the opportunities and challenges of modern healthcare.

The issue also includes a timely review by Al Shammari and Sait on hypertension in women with obesity, a growing global health concern with significant cardiovascular implications. The authors examine the complex interplay between obesity, hormonal influences, metabolic dysfunction, and hypertension across the female lifespan. By addressing female-specific risk factors such as menopause, polycystic ovary syndrome, and pregnancy-related hypertensive disorders, the review highlights the importance of individualized, sex-specific approaches to prevention and management. Equally important, the paper emphasizes the role of lifestyle interventions, pharmacological therapies, emerging metabolic treatments, and public health strategies in reducing long-term cardiovascular risk. As obesity and hypertension continue to rise worldwide, this contribution provides a valuable framework for clinicians and policymakers seeking to improve outcomes for women across diverse populations.

Warm regards,
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QFMCRC: Qatar Family Medicine Clinical Reasoning Cycle: An Integrated Clinical Reasoning Cycle for Family Medicine: Bridging Diagnosis, Management, and Reflection

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Abstract

Clinical reasoning is the intellectual foundation of family medicine. This paper reviews the evolution of clinical reasoning models and proposes an integrated Clinical Reasoning Cycle that unifies diagnostic reasoning, management planning, and reflective practice. The model emphasizes iterative thinking, contextual understanding, and patient-centered decision-making which are essential characteristics of Family Medicine. This integrated framework aims to enhance clinical education, strengthen physician decision making, and improve patient care outcomes.

Keywords: Clinical reasoning, family medicine, diagnostic reasoning, reflective practice, medical education

Introduction

Clinical reasoning is the central competency in family medicine, essential for managing undifferentiated clinical presentations, guiding safe and effective management, and delivering person centered and longitudinal management. Existing clinical reasoning models are often fragmented and do not fully capture the complex, iterative, and context dependent nature of primary care practice. Within Qatar's rapidly advancing healthcare system, Family Physicians require an integrated clinical reasoning approach that combines diagnostic thinking, management planning, and reflective practice. The Qatar Family Medicine Clinical Reasoning Cycle (QFMCRC) is proposed as a unified and practical framework designed to strengthen clinical decision making, enhance residency education, and improve the quality and safety of patient care.

Models of Clinical Reasoning in Family Medicine

Several theoretical models describe the cognitive processes underlying clinical reasoning. Each highlights different aspects of how clinicians gather information, generate hypotheses, and make clinical decisions.

1. Diagnostic Reasoning Models

- **Hypothetical–Deductive Model:** Clinicians generate early diagnostic hypothesis and test them through targeted data collection (1)
- **Pattern Recognition:** Experienced physicians rapidly identify familiar clinical patterns based on previous exposure (2)
- **Illness Script Theory:** Clinicians use structured mental representations that link risk factors, pathophysiology, and clinical features to guide diagnosis (3)
- **Dual-Process Theory:** Combines intuitive, fast reasoning (System 1) with slower, analytical reasoning (System 2) (4)

2. Contextual and Narrative Reasoning

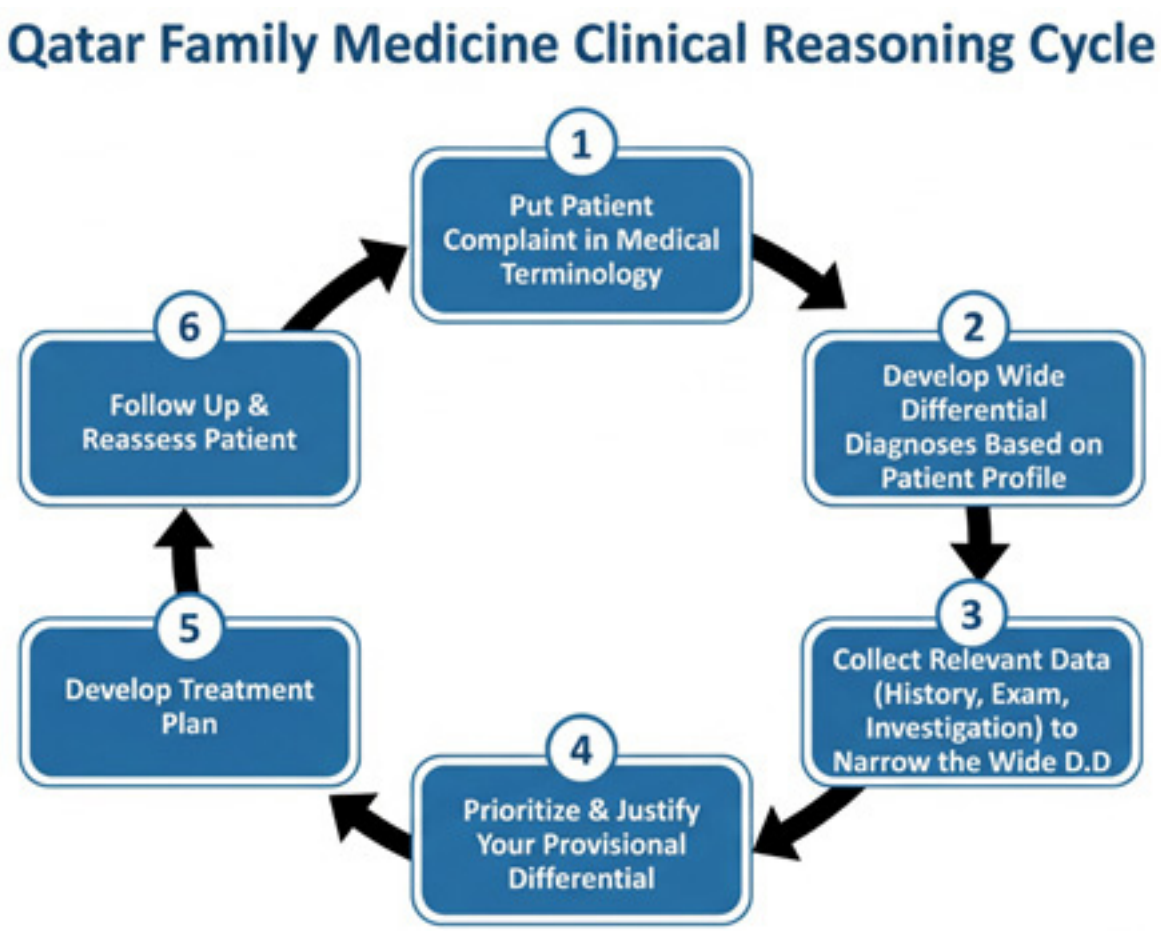
- This approach emphasizes understanding the patient's story, beliefs, cultural background, and psychosocial context, which is fundamental to person centered Family Medicine practice (5)

3. Clinical Judgement Models

- **Tanner's Model:** Describes clinical judgement as a process of noticing, interpreting, responding, and reflecting (6)
- **Levett-Jones' Clinical Reasoning Cycle:** An eight phase, cyclical model that mirrors continuous assessment and follow up in clinical care (7)

4. Educational and Teaching Models

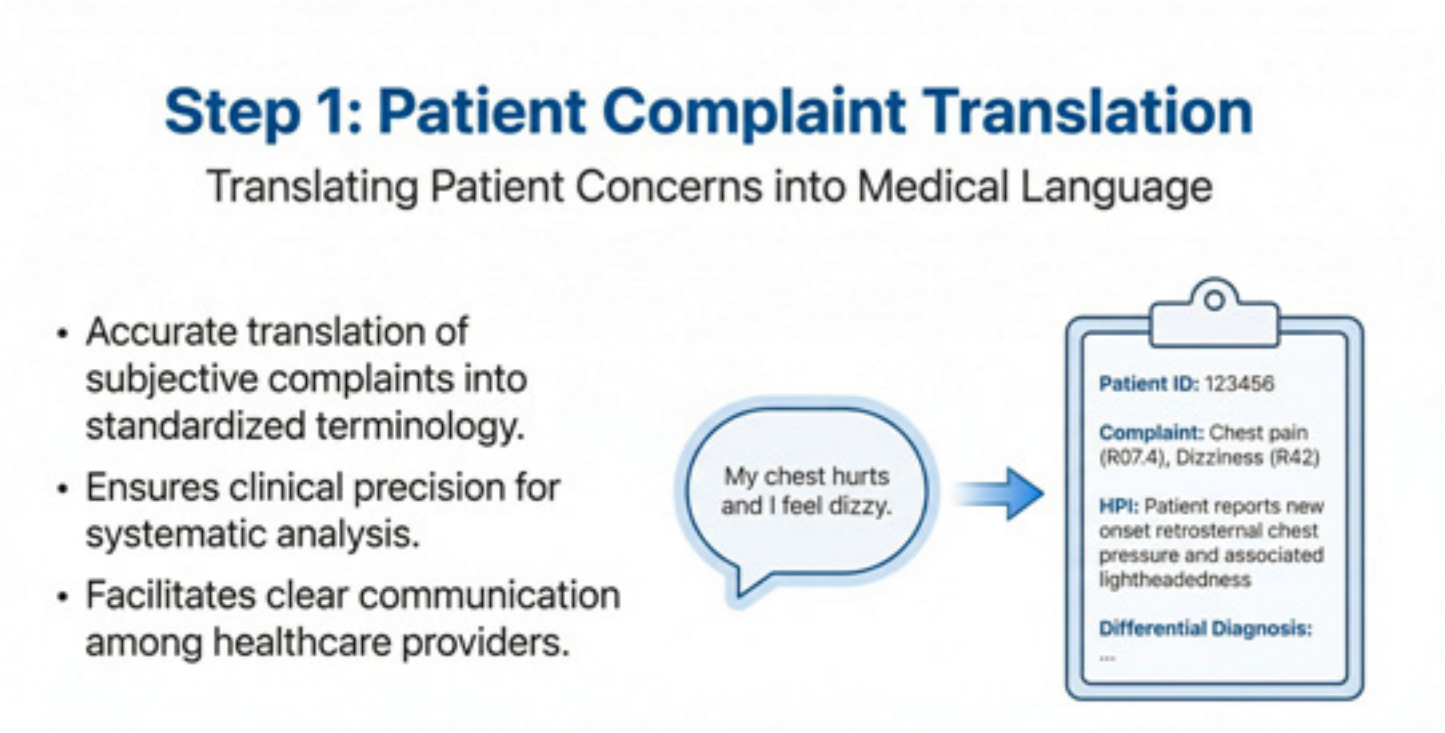
- **SNAPPS:** A learner centered framework that encourages trainees to analyze differential diagnoses, express uncertainty, and develop management plans (8)



The Qatar Family Medicine Clinical Reasoning Cycle
It is an integrated, practical framework that reflects the real flow of a family medicine consultation. It unifies diagnostic reasoning, management planning, and reflective practice into one continuous, iterative process. The cycle includes six key steps:

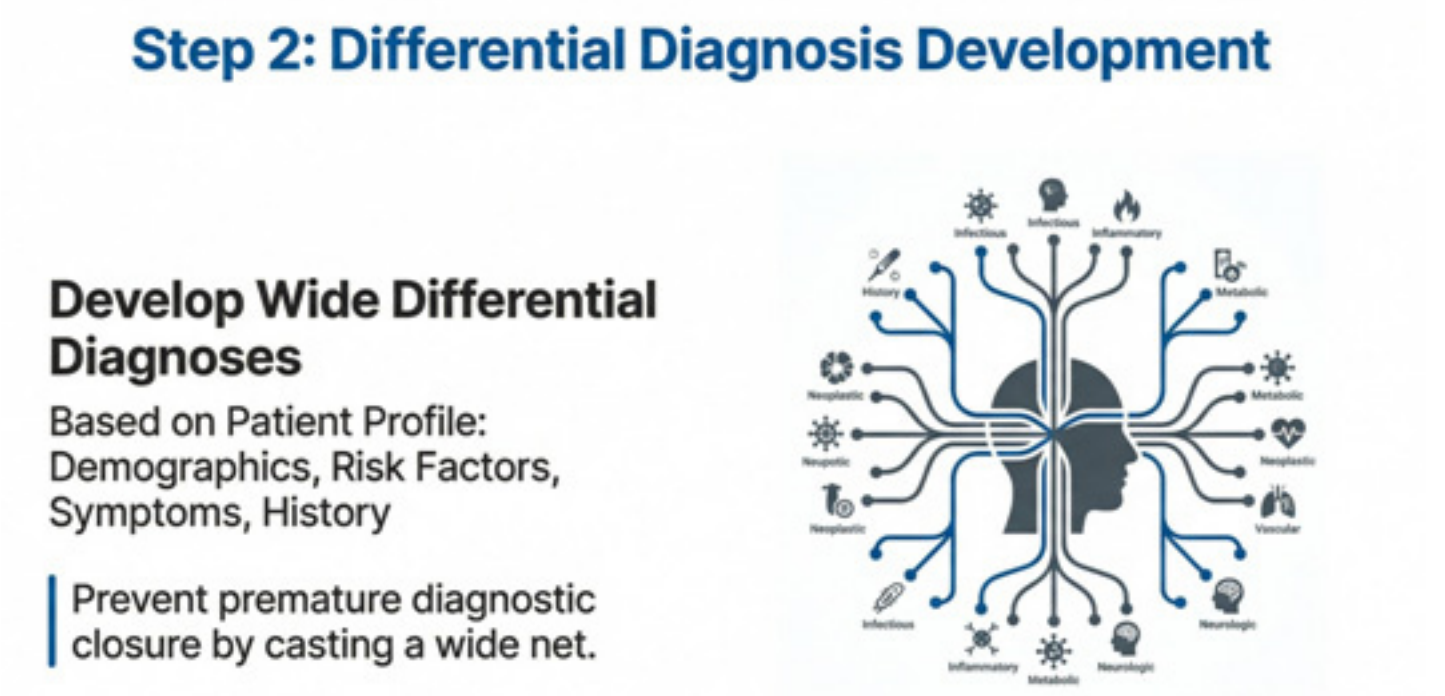
Step 1: Patient Complaint Translation

Translating Patient Complaints into Medical Language. The clinical reasoning process begins with accurately translating the patient's concerns into appropriate medical terminology. This step ensures that subjective experiences are captured with clinical precision, facilitating effective communication among healthcare professionals and enabling structured clinical analysis.

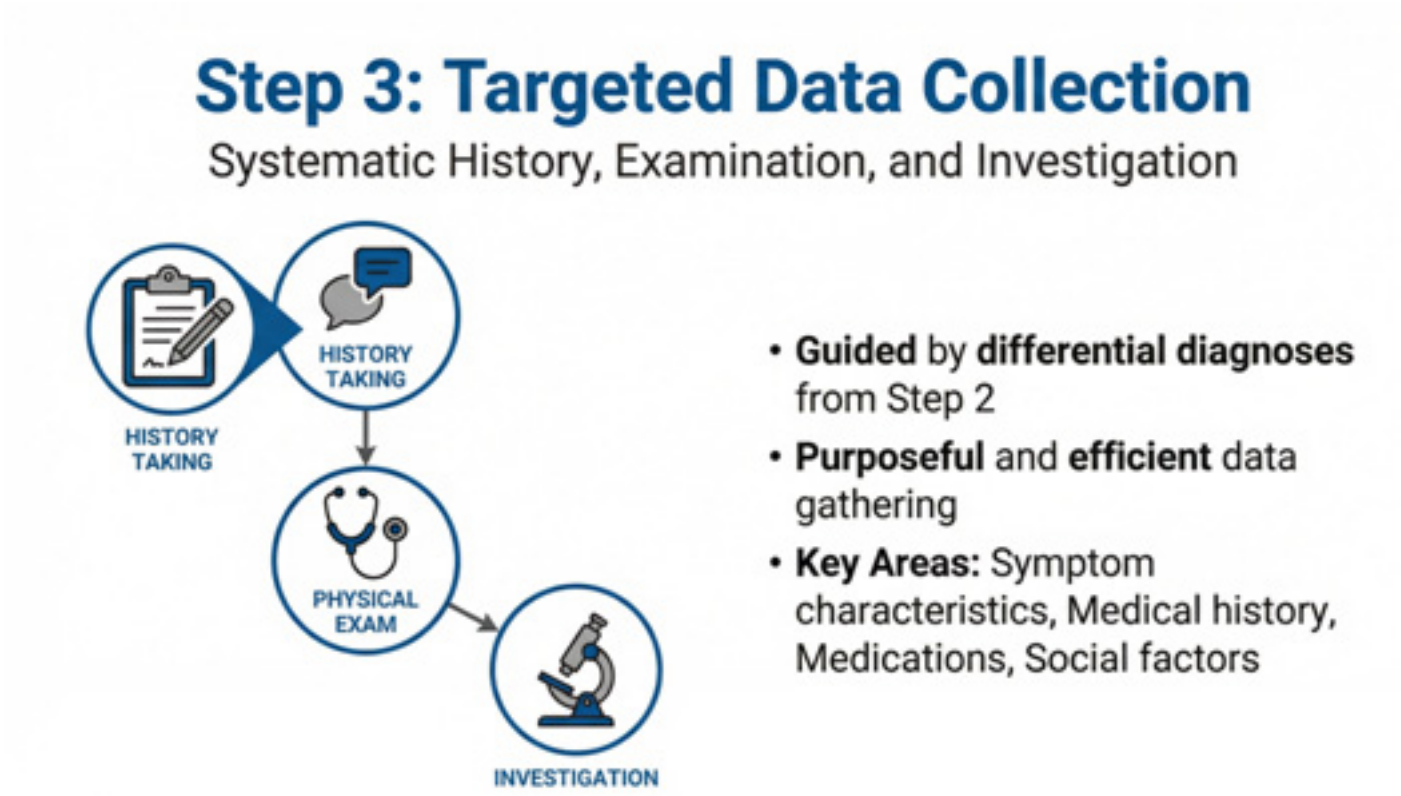


Step 2: Developing a Broad Differential Diagnosis

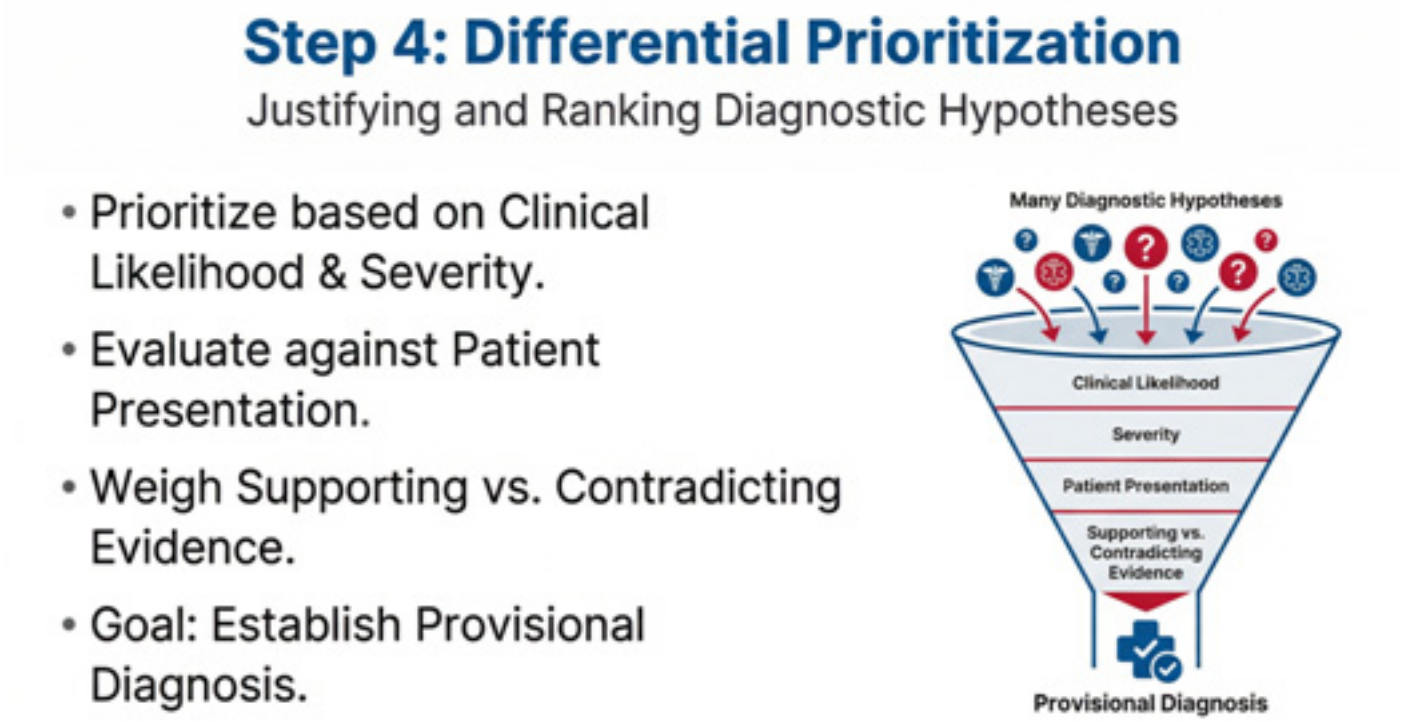
Developing a broad differential diagnosis based on the patient's demographics, risk factors, presenting symptoms, and relevant medical history, clinicians generate a comprehensive list of possible diagnoses. This approach reduces the risk of premature diagnostic closure and ensures that clinically significant conditions are considered.



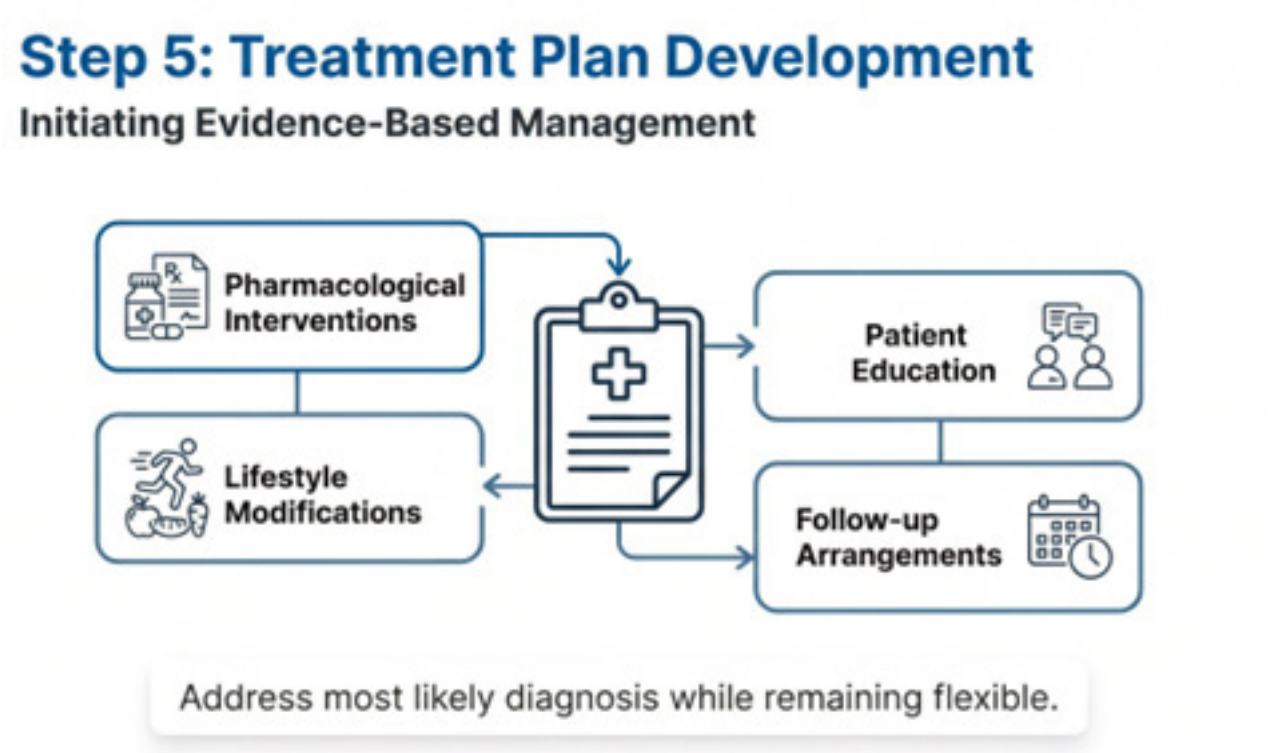
Step 3 - Targeted Data Collection
Clinicians gather relevant information through focused history taking, physical examination, and appropriate investigations. Data collection is guided by the differential diagnosis, ensuring efficiency and clinical relevance.



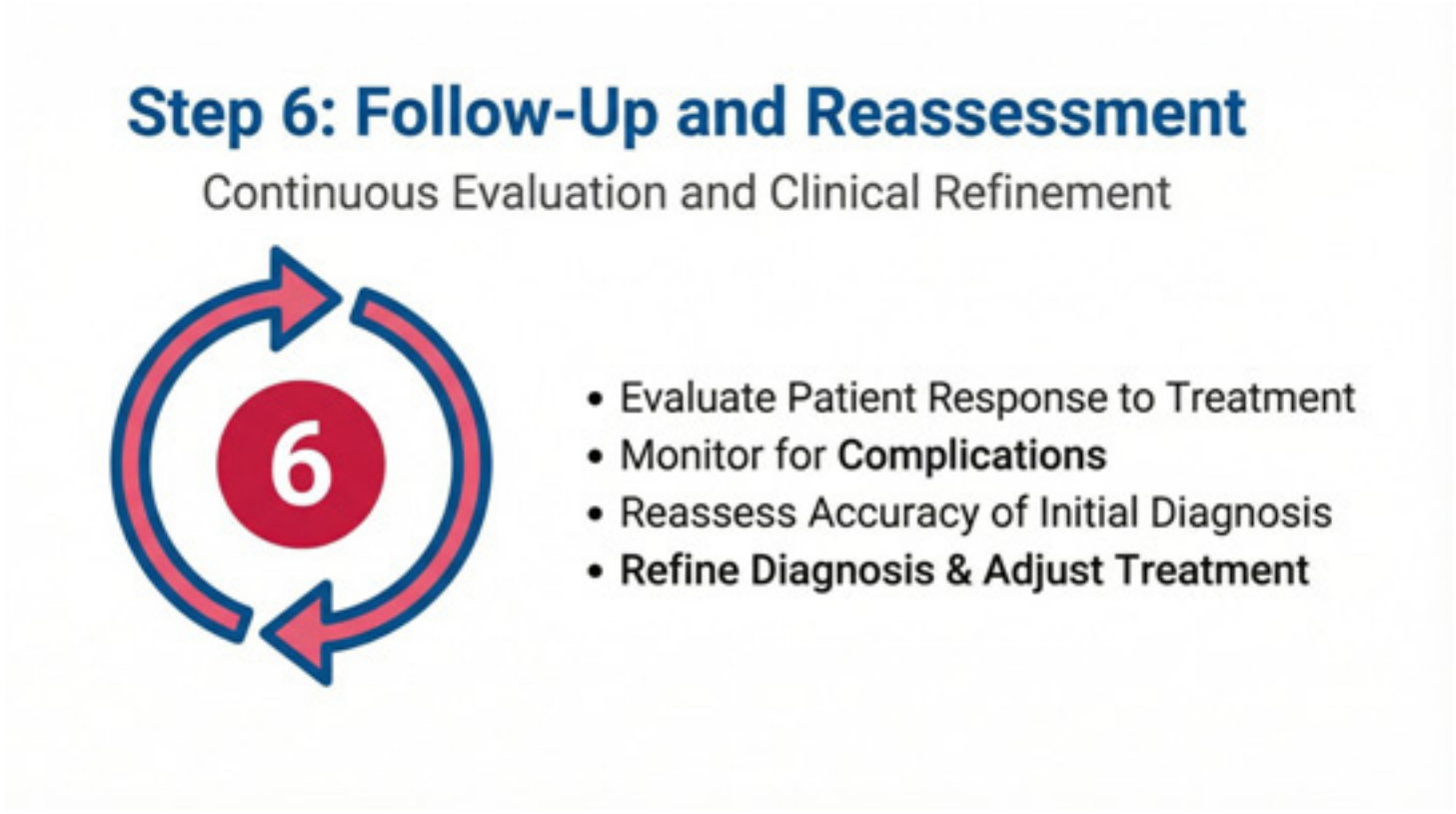
Step 4: Prioritizing and Justifying the Differential Diagnosis
Using the collected data, clinicians prioritize diagnostic possibilities based on likelihood, severity, and supporting evidence. Each diagnosis is evaluated against clinical findings to establish a provisional working diagnosis.



Step 5: Developing a Treatment and Management Plan
Initiating Evidence-Based Management based on the prioritized differential diagnosis, clinicians develop a comprehensive treatment plan. This plan addresses the most likely diagnosis while remaining flexible to accommodate alternative diagnoses if the patient does not respond as expected. The treatment plan integrates pharmacological interventions, lifestyle modifications, patient education, and follow-up arrangements to optimize patient outcomes.



Step 6 - Follow-Up and Reassessment
Ongoing follow up allows clinicians to evaluate treatment response, reassess diagnostic accuracy, and refine management plans. This step reinforces the cyclical nature of clinical reasoning and supports patient safety and continuity of care.



The Cyclical Nature of Clinical Reasoning
The QFMCRC is inherently cyclical rather than linear. New information obtained during follow up may prompt clinicians to revisit earlier steps, refine diagnostic hypotheses, and adjust management strategies. This iterative process reflects the dynamic reality of Family Medicine practice and promotes continuous professional development.

Core Purpose:
This model strengthens decision-making, supports resident training, and aligns with the holistic, contextual, and longitudinal nature of family medicine.

The proposed Qatar Family Medicine Clinical Reasoning Cycle aligns closely with established reasoning theories yet offers unique advantages that make it particularly suited to the realities of family medicine. Traditional models provide strong foundations for understanding how clinicians think, but many describe reasoning in fragmented components or focus primarily on diagnostic processes. In contrast, the proposed cycle integrates diagnostic reasoning, management planning, and reflective practice into one coherent, iterative framework.

Comparison with Established Models
The Qatar Family Medicine Clinical Reasoning Cycle (QFMCRC) aligns closely with several well-established clinical reasoning theories while offering a more integrated and practice-oriented structure. Many traditional models provide valuable insights into specific cognitive processes but often address diagnostic reasoning, management planning, and reflection as separate or loosely connected components.

The QFMCRC differs by explicitly integrating these elements into a single, continuous cycle that mirrors the real-world workflow of Family Medicine. It translates complex theoretical constructs into clearly defined, actionable steps that are easily understood by clinicians and trainees. This clarity makes the model particularly suitable for both daily clinical use and structured educational settings.

Clinical Reasoning Step	Corresponding Concepts in Established Models
1. Put patient complaints in medical terminology	Problem Representation (Integrative Model), Cue Acquisition (Hypothetico-Deductive), Noticing (Tanner's Model), Consider the patient situation (Levett-Jones Cycle)
2. Develop wide differential diagnoses list	Hypothesis Generation (Hypothetico-Deductive), Interpreting (Tanner's Model), Identify problems/issues (Levett-Jones Cycle)
3. Collect relevant data (history, exam, investigation)	Cue Interpretation (Hypothetico-Deductive), Information Gathering (Integrative Model), Collect cues/information (Levett-Jones Cycle)
4. Prioritize and justify provisional differential	Hypothesis Evaluation (Hypothetico-Deductive), Analyze Differentials (SNAPPS), Process information (Levett-Jones Cycle)
5. Develop treatment plan	Plan Management (SNAPPS), Take Action (Levett-Jones Cycle), Responding (Tanner's Model)
6. Follow up and reassess the patient	Evaluate Outcomes & Reflect (Levett-Jones Cycle), Reflection (Tanner's Model)

Clarity, Practicality, and Cyclical Design

A key strength of the QFMCRC is its clarity and direct applicability. While some models are primarily descriptive and focus on explaining how clinicians think, this cycle provides a prescriptive guide outlining what clinicians should do at each stage of patient care. Although the steps may appear linear, the model is inherently cyclical. Follow-up and reassessment generate new clinical information that prompts reconsideration of earlier steps, reinforcing reflective practice and continuous learning.

Unlike dual-process theory, the QFMCRC does not explicitly label intuitive and analytical reasoning. Instead, it accommodates both modes naturally within the workflow. Experienced clinicians may rely on pattern recognition to move quickly toward a provisional diagnosis, while trainees may engage more deliberately in analytical reasoning. This flexibility allows the model to support clinicians at different levels of experience.

Diagnostic Framework for Identifying Physician Competency Gaps

Beyond guiding clinical care, the QFMCRC can function as a diagnostic framework for identifying gaps in physician competence. Failure at specific stages of the cycle may reflect distinct educational or performance deficiencies, allowing for targeted feedback and remediation.

Failure to accurately translate patient complaints into medical terminology may indicate difficulties in communication skills, problem representation, or empathic engagement. An inability to generate a broad differential diagnosis may suggest limited medical knowledge, cognitive bias such as premature closure, or overreliance on intuitive reasoning. Inadequate data collection can reflect weaknesses in history-taking, physical examination skills, or hypothesis-driven information gathering.

Difficulty prioritizing and justifying differential diagnoses often points to challenges in analytical reasoning, data synthesis, or probabilistic thinking. Deficiencies in management planning may indicate gaps in therapeutic knowledge, limited application of evidence-based medicine, or insufficient shared decision-making skills. Failure to conduct appropriate follow-up and reassessment suggests weaknesses in reflective practice, clinical ownership, or systems-based care.

By mapping observed performance issues to specific steps in the cycle, educators can provide focused feedback and design individualized learning plans that address underlying reasoning deficits rather than isolated errors.

Step in clinical reasoning Cycle	Potential Physician Defect if Not Done Properly	Supporting Rationale from Clinical Reasoning Literature
1. Put complaint in medical terminology	Defect: Poor communication and interpersonal skills; weak problem representation; lack of empathy.	This initial step is about creating an accurate problem representation, a critical phase in the Integrative Model (9). Failure here suggests the physician cannot effectively translate the patient's narrative into a clinically meaningful context, a process Tanner's model calls Noticing and which requires engagement with the patient's concerns (6).
2. Develop wide differential diagnoses list	Defect: Deficient medical knowledge base; cognitive biases (e.g., premature closure, availability heuristic); over-reliance on non-analytical reasoning.	This is the Hypothesis Generation phase from the Hypothetico-Deductive model (1). A narrow list points to a limited knowledge of disease scripts or being unduly influenced by recent or memorable cases. It indicates a failure to engage System 2 (analytical) thinking to consider a broad range of possibilities (4).
3. Collect relevant data	Defect: Poor clinical skills (history-taking, physical exam); inefficient data gathering; inability to test hypotheses.	This step involves Cue Interpretation and information gathering to test the generated hypotheses (9,1). A physician who collects irrelevant data or performs a poor examination lacks the procedural skills and the strategic thinking needed to narrow the differential diagnosis efficiently.
4. Prioritize and justify provisional differential	Defect: Flawed analytical and critical thinking skills; poor synthesis of clinical data; difficulty with probabilistic reasoning.	This is the core of Hypothesis Evaluation (1). The inability to weigh evidence, consider disease prevalence, and justify a leading diagnosis indicates failure in higher-order analytical reasoning. The physician may have the data but cannot synthesize it into a coherent and defensible conclusion.
5. Develop treatment plan	Defect: Lack of knowledge of therapeutic guidelines; inability to apply evidence-based medicine; poor shared decision-making.	This step, corresponding to Responding or Taking Action (6), requires more than just a diagnosis. It involves translating the diagnosis into a practical, evidence-based plan that considers the patient's context and preferences. Failure here can indicate a gap between knowing *what* the patient has and knowing *what to do* about it.
6. Follow up and reassess the patient	Defect: Lack of metacognitive skills (reflection); poor sense of responsibility and ownership; systems-based practice issues.	This final step is crucial for learning and safety. It aligns with Reflection in Tanner's model and the Levett-Jones cycle (6). A failure to follow up and learn from outcomes indicates a breakdown in metacognition, the ability to think about one's own thinking, which is critical for continuous professional development and error prevention.

Educational Implications

Integrating the QFMCRC into Family Medicine residency training has the potential to strengthen clinical reasoning skills across all levels of learners. The model can be used as a framework for case-based discussions, structured clinical encounters, reflective writing, simulation exercises, and longitudinal patient follow-up. It also offers faculty a shared language for teaching and assessing reasoning processes.

Assessment strategies aligned with the QFMCRC should emphasize reasoning pathways, adaptability, and reflection rather than diagnostic accuracy alone. Tools such as case-based discussions, Mini-CEX, direct observation, and reflective portfolios can be mapped to specific steps of the cycle, enhancing both formative and summative assessment.

A risk in all of these efforts is that we come to believe we are teaching "clinical reasoning" as a generalizable skill that can be applied to any clinical problem. Unfortunately, this fond hope has little empirical support. From the earliest studies of medical problem solving to the present, the most reproducible result is that clinical reasoning performance is highly content (and context) specific (10,1).

Obtaining a good grasp of clinical reasoning processes and principles in medical school is important to safeguard patients after graduation. There is an emerging stance that clinical reasoning should be taught more explicitly in medical education (11).

In fact, it is recognized that most clinical reasoning errors are not merely attributed to physician incompetence or lack of knowledge, but rather the 'frailty of human thinking under conditions of complexity, uncertainty, and pressure of time' (12).

The cultivation of clinical reasoning in medical training is achieved through deliberate practice and dialectical instruction in naturalistic settings but can be supported with simulated learning (13). Broadly, clinical clerkship attachments are most effective when trainees are immersed in an iterative process of direct observation, deliberate practice and receiving feedback, with further consolidation of learning achieved through dialectical methods of instruction such as Socratic/clinical questioning (13), chart review or chart-stimulated recall (14) and mini-CEX (clinical evaluation exercise) assessments (15).

Summary

The ability to articulate a patient's complaint using appropriate medical terminology may vary depending on whether the resident is a native English speaker and their level of cognitive functioning. A structured assessment cycle can help identify specific deficiencies at each stage of clinical evaluation:

- **History taking:** Difficulty in generating a broad differential diagnosis may indicate a gap in foundational medical knowledge. Challenges in data collection could stem from language barriers, poor communication skills, or an inability to formulate appropriate questions.
- **Physical examination:** Deficiencies may be observed in either technique (a skill-based issue) or interpretation (a knowledge-based issue).
- **Investigations**: Ordering appropriate tests reflects clinical reasoning and judgement. Errors here may point to deficits in medical knowledge or critical thinking.
- **Diagnostic reasoning:** Difficulty in narrowing down the differential diagnosis often suggests a flaw in the resident's analytical thinking process.
- **Management planning:** Inability to formulate a suitable treatment plan may again highlight gaps in medical knowledge.

Conclusion

Clinical reasoning in Family Medicine is a multidimensional process that integrates analytical thinking, intuition, contextual awareness, and reflection over time. The Qatar Family Medicine Clinical Reasoning Cycle offers a unified, practical framework that aligns closely with the philosophy and clinical realities of Family Medicine practice. By integrating diagnostic reasoning, management planning, and reflective practice into a single cyclical process, this model has the potential to enhance clinical education, support faculty development, and improve patient-centered care. Future work should focus on implementation, validation, and evaluation of the model's impact on learner development and clinical outcomes.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process
During the preparation of this work the author(s) used [Chat GPT and Copilot] in order to [generate data]. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

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Lifestyle Medicine as a Therapeutic Approach to Menopause: A Narrative Review

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Abstract

Background: Menopause is a normal change characterized by the end of ovarian function and lower oestrogen. It causes vasomotor, psychological, metabolic, musculoskeletal, and genitourinary symptoms, which may affect quality of life and health. As life expectancy rises, women spend more years postmenopausal. This highlights the need for effective management strategies. Hormone therapy is important, but many women seek non-pharmacological approaches for symptom relief and health promotion.

Objective: To evaluate lifestyle medicine as a primary, holistic approach for menopause symptom management and healthy aging.

Methods:
We conducted a narrative review of observational studies, randomized trials, systematic reviews, meta-analyses, and clinical guidelines. The review focused on six pillars of lifestyle medicine: nutrition, physical activity, restorative sleep, stress management, avoidance of harmful substances, and social connection.

Results: Evidence suggests lifestyle interventions improve menopausal symptoms like hot flushes, sleep issues, mood changes, and weight gain. Lifestyle changes also promote cardiovascular and metabolic health, preserve bone and muscle mass, boost psychological well-being, and lower the chance of chronic disease. Nutrition, regular exercise, stress reduction, and healthy sleep provide added benefits. These not only relieve symptoms but also promote long-term health and function.

Conclusion: Lifestyle medicine is a safe, evidence-based, patient-focused approach to menopause. Adding lifestyle changes to routine care may improve symptoms, quality of life, and healthy aging, and reduce the chance of chronic disease. More high-quality research is needed to build evidence and improve clinical practice.

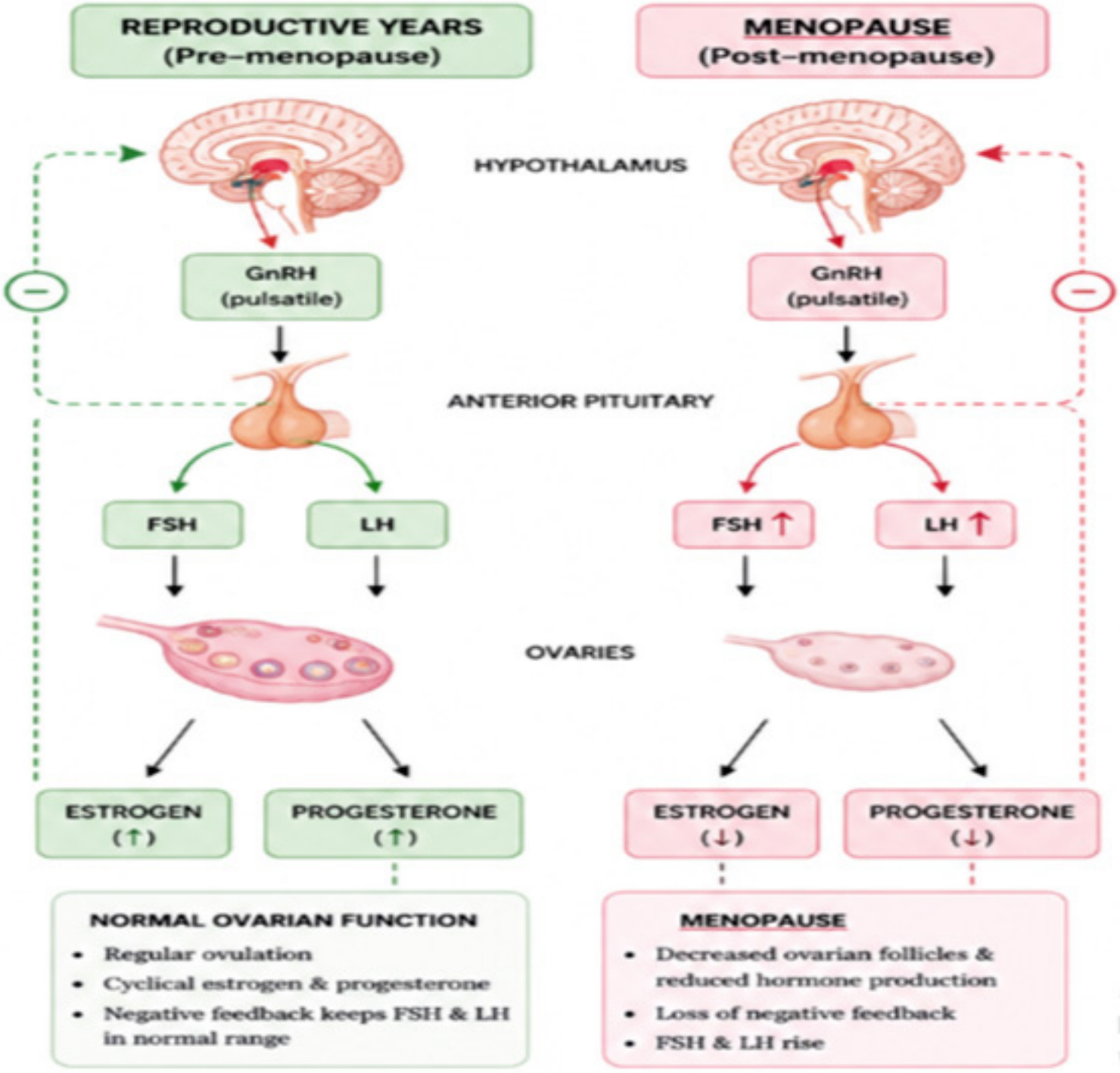
Keywords:
Menopause; Lifestyle Medicine; Healthy Aging; Women's Health; Menopausal Symptoms.

Introduction

Menopause is a life stage defined by the permanent end of menstruation (1). This happens when ovarian follicular activity is depleted (2). Diagnosis is made after 12 consecutive months of missed periods, provided there are no other causes (1). On average, women reach menopause at about 51 years of age, but this age may vary depending on geography, ethnicity, and socioeconomic status (1). As women are living longer, they spend more years after menopause. This makes menopause and its health effects an important public health issue.

The menopausal transition brings major endocrine changes, especially drops in oestrogen and progesterone (Figure 1) (4-7).

Figure 1. Hormonal changes during menopause. Adapted from Hall (4), Burger et al. (3), Davis (9).



Such hormonal shifts lead to many symptoms, such as hot flashes, sleep problems, mood changes, sexual issues, muscle and joint pains, weight gain, and lower quality of life (Table 1) (8-9).

Table 1: Role of oestrogen and progesterone deficiency in menopause

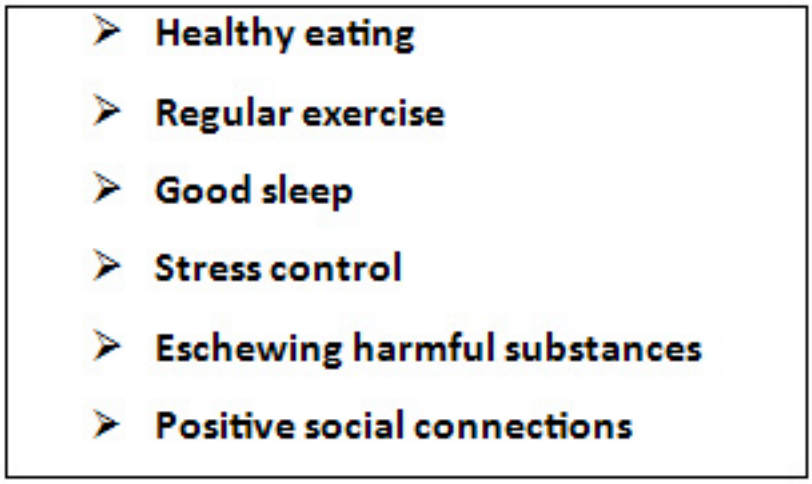
Feature	Oestrogen Deficiency	Progesterone Deficiency
Timing	Predominantly in late perimenopausal and postmenopausal women	Typically arises in early perimenopause owing to anovulatory ovarian function
Primary cause	Reduced ovarian follicular activity and depletion of ovarian reserve	Decreased ovulation resulting in reduced corpus luteum formation
Menstrual Changes	Amenorrhoea and eventual cessation of menstruation	Irregular cycles, shortened cycles, heavy or unpredictable bleeding
Vasomotor Symptoms	Hot flushes, night sweats, temperature dysregulation	May contribute indirectly but not a primary cause
Genitourinary Symptoms	Vaginal dryness, dyspareunia, urinary frequency, recurrent urinary tract infections	Minimal direct effect
Bone health	Accelerated bone loss and increased osteoporosis risk	Limited direct effect on bone metabolism
Cardiovascular Effects	Adverse changes in lipid profile, endothelial dysfunction, increased cardiovascular risk	Less clearly established
Psychological Symptoms	Mood changes, anxiety, depression, cognitive complaints	Mood instability, irritability, anxiety, premenstrual-type symptoms
Sleep Disturbance	Secondary to vasomotor symptoms and neuroendocrine changes	May contribute to insomnia because progesterone has sedative and anxiolytic properties
Body Composition	Increased visceral adiposity and reduced lean muscle mass	Less pronounced effect
Sexual Function	Reduced libido, vaginal discomfort, impaired arousal	May contribute to reduced sexual wellbeing through mood and sleep disturbances

Menopause also raises the risk of problems like osteoporosis, heart disease, cognitive decline, and metabolic conditions (10-12).

Menopausal hormone therapy (MHT) is still the most effective way to treat many symptoms (13). However, some women cannot use it, and many choose non-drug options. Interest is growing in evidence-based methods that help with both symptoms and long-term health (13)

Lifestyle medicine is a science-based field that aims to prevent, treat, and sometimes reverse chronic diseases through lasting behaviour changes (14). It has six key areas (Table 2): healthy eating, regular exercise, good sleep, stress control, eschewing harmful substances, and positive social connections (14).

Table 2: Pillars of lifestyle medicine

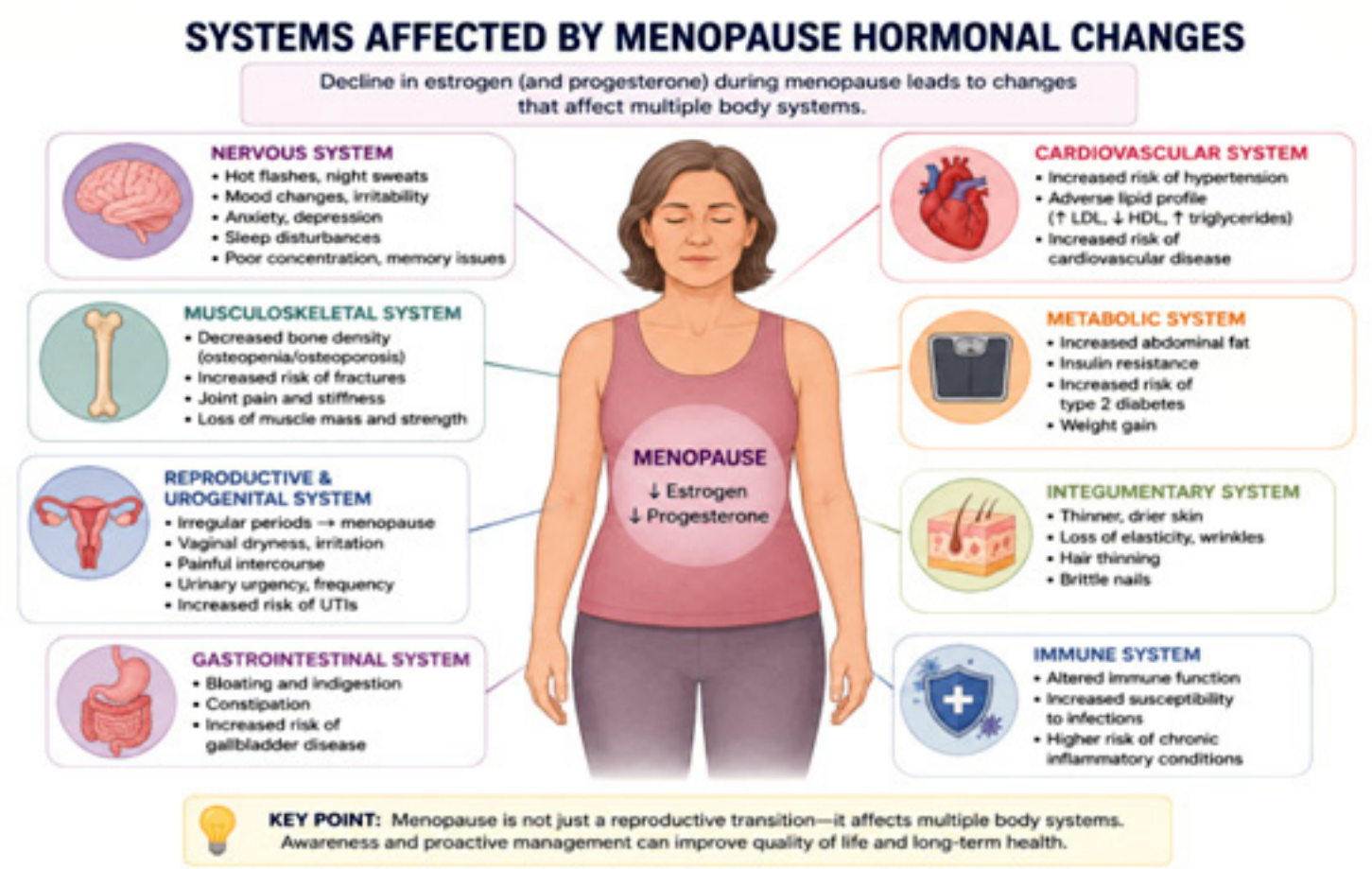


These form a strong base to support women during menopause. Research shows that such lifestyle changes can ease menopausal symptoms, improve quality of life, and lower the chance of chronic diseases linked to menopause (15-16).
This review examines how lifestyle medicine can support the management of menopause. It focuses on what the evidence says, ways to apply it in practice, and where future research needs to go.

Menopause: Physiological and Clinical Considerations

Menopause represents the final stage of reproductive aging. It is characterized by substantial endocrine and metabolic alterations (Figure 1) (3) (6). The progressive depletion of ovarian follicles results in a decline in ovarian oestrogen and inhibin production (3)(6). This leads to compensatory elevations in circulating follicle-stimulating hormone (FSH) and luteinizing hormone (LH) concentrations (3)(6). Such hormonal changes exert widespread effects on multiple physiological systems (Figure 2) (3)(6). They contribute to the multiple clinical manifestations observed during the menopausal transition.

Figure 2: Systems Affected by Menopause Hormonal Changes
Adapted from Hall et al (4), Burger et al (3), Davis (9)



The menopausal transition is conventionally categorized into three stages (3)(6):

Premenopause:
Premenopause (2) is the reproductive phase before the menopausal transition. During this time, ovarian hormone function is mostly preserved, and menstrual cycles are usually regular. Fertility is maintained, but minor hormonal changes can occur. Women in this stage usually have few menopausal symptoms and stable hormonal function.

Perimenopause:
Perimenopause (2) is the transition phase leading to menopause. It includes the time up to 12 months after the final menstrual period. During this stage, the number of ovarian follicles drops. Estrogen and progesterone levels fluctuate. Menstrual cycles become irregular. Symptoms such as hot flushes, mood changes, sleep problems, and metabolic changes may appear.

Postmenopause:
Postmenopause (2) starts 12 months after the final menstrual period and continues after that. The ovaries stop producing eggs and oestrogen levels stay low. This stage brings changes that raise the risk of osteoporosis, heart disease, genitourinary issues, and metabolic problems.

Beyond symptom burden, oestrogen deficiency is associated with several chronic health effects. These include accelerated bone loss and increased risk of osteoporosis (3). Oestrogen deficiency also leads to adverse alterations in body composition, with increased central adiposity (3). It is linked to insulin resistance, dyslipidaemia, and an elevated risk of cardiovascular disease (3). Emerging evidence also points to possible associations between menopause and cognitive decline in susceptible individuals (3).

Consequently, menopause should be regarded not only as a reproductive milestone but also as an important period for preventive healthcare and risk-factor modification (3). In this context, lifestyle medicine offers an all-encompassing framework for tackling both the immediate symptoms of menopause and the long-term health risks associated with hormonal aging. This approach supports healthy aging and improves quality of life in midlife and older women.

Pillars of Lifestyle Medicine (Table 2)
Lifestyle medicine uses lifestyle interventions to prevent, treat, and manage chronic diseases (14). It targets behavioural and environmental health factors by facilitating sustainable changes in daily habits (14).

Lifestyle medicine is built on six related pillars (14):
1. Whole-Food, Plant-Predominant Nutrition
Consume mostly whole, minimally processed plant-based foods; limit ultra-processed items and saturated fats.
2. Consistent Physical Exercise
Regularly perform a mix of aerobic, resistance, flexibility, and balance exercises.
3. Reparative Sleep
Adequate quality sleep supports brain, hormone, and immune health.
4. Stress Management
Use mindfulness, relaxation techniques, cognitive strategies, or support to reduce chronic stress.
5. Avoidance of Risky Substances
Avoid tobacco, excess alcohol, and other harmful substances for health.
6. Positive Social Connections

Building supportive relationships and engaging socially enhances well-being and mental strength.
These pillars address many biological, behavioural, and psychosocial factors in menopausal symptoms and the higher chance of chronic disease during menopause and after.

Nutrition and Menopause

Role of Nutrition in the Management of Menopausal Symptoms
Nutrition is a core part of lifestyle medicine. It is one of the most accessible, cost-effective, and sustainable interventions for women during menopause. Dietary habits affect many systems influenced by menopause (17). These include endocrine regulation, body composition, cardiovascular function, bone metabolism, inflammatory pathways, and psychological health (18).

More evidence suggests that diets high in fruits, vegetables, whole grains, legumes, nuts, and unsaturated fats improve health in menopausal and postmenopausal women (19). These foods are rich in fibre, antioxidants, vitamins, minerals, and phytoestrogens (19). These nutrients may help lower vasomotor symptoms, maintain bone mineral density, improve cardiometabolic health, and reduce cardiovascular risk (19). Following nutrient-dense diets is also linked to reduced inflammation and improved quality of life.

Optimizing nutrition is a main part of lifestyle medicine for menopause (19). It helps relieve symptoms and lowers the risk of problems such as osteoporosis, cardiovascular disease, obesity, and metabolic dysfunction (19).

Mediterranean Dietary Pattern (20)

The Mediterranean diet is often studied in relation to menopause. It focuses on eating (Figure 3):
- Fruits and vegetables
- Whole grains
- Legumes
- Nuts and seeds
- Olive oil as the principal source of dietary fat
- Fish and seafood
- Moderate consumption of dairy products

Figure 3: A new Mediterranean diet pyramid. Adapted from Sofi et al (87)

Studies show that sticking to the Mediterranean diet brings many health benefits for menopausal women (20). These include:

- Reduction in the frequency and severity of vasomotor symptoms
- Improved cardiovascular risk profiles
- Enhanced glycaemic control and metabolic health
- Lower concentrations of inflammatory biomarkers
- Improved overall quality of life and psychological wellbeing

The Mediterranean diet's benefits may come from its anti-inflammatory, antioxidant, and heart-protective properties. It also helps control weight and supports metabolic function (21). As a result, this diet is seen as a proven way to support health during menopause and beyond (21).

Phytoestrogens

Phytoestrogens are naturally occurring plant-derived bioactive compounds that display weak oestrogenic and anti-oestrogenic properties through their interaction with oestrogen receptors (22). Among these compounds, isoflavones have been the most extensively investigated for menopausal symptom management. Rich dietary sources of isoflavones include soy-based foods such as soybeans, tofu, tempeh, edamame, and soy milk (23). Soy isoflavones are promising for managing menopausal symptoms but are not as potent as hormone therapy (23).

Phytoestrogens in menopausal women with positive oestrogen tumours:
Phytoestrogens are plant-derived compounds structurally like oestradiol that bind oestrogen receptors, with relative selectivity for ER-β (88-91). In postmenopausal women, they are primarily obtained from soy isoflavones, flaxseed, and legumes and are used to relieve vasomotor and metabolic symptoms. Their safety in women with oestrogen receptor–positive (ER+) tumours is still an area under ongoing investigation (88-91). Evidence from randomized controlled trials of soy isoflavone supplementation in postmenopausal women and breast cancer survivors, together with prospective cohort data such as the Shanghai Breast Cancer Survival Study and subsequent meta-analyses, demonstrates no significant increase in recurrence or mortality (88-91). Overall findings are heterogeneous but generally reassuring, supporting cautious individualized clinical use pending further long-term randomized evidence.

Transitioning from phytoestrogens, it is important to also consider lifestyle elements that impact menopausal health, such as weight management.

Weight Management
Weight gain is a common feature of the menopausal transition and results from a complex interplay among hormonal alterations, aging-related metabolic changes, declining physical activity, and changes in body composition (24). Menopause is associated with an increase in visceral adiposity, a reduction in lean skeletal muscle mass, and a decline in resting metabolic rate, all of which lead to progressive weight accumulation (24).

In addition to food choices, maintaining a healthy weight confers multiple health benefits during menopause. Excess adiposity during midlife is associated with adverse health outcomes, including increased vasomotor symptom burden, heightened cardiovascular risk, development of type 2 diabetes mellitus, osteoarthritis, and impaired quality of life (25). As a result, weight management is a key element of comprehensive menopausal care (25). Lifestyle interventions focusing on dietary modification and regular physical exercise represent the foundation of weight management strategies. Clinical evidence shows that even a modest weight reduction of 5–10% of initial body weight can improve menopausal symptoms, metabolic parameters, and general health outcomes (26).

Nutritional Considerations for Bone Health
The postmenopausal period is characterized by accelerated bone loss secondary to oestrogen deficiency, resulting in an increased risk of osteopenia, osteoporosis, and fragility fractures (27-28). Optimizing nutritional status is therefore essential for the preservation of skeletal health.

Key nutrients involved in bone metabolism include calcium, vitamin D, magnesium, vitamin K, and dietary protein. Adequate intake of these nutrients aids bone mineralization, maintenance of bone mass, and musculoskeletal function (29). When combined with regular weight-bearing and resistance-based exercise, appropriate nutritional strategies help substantially prevent osteoporosis and reduce fracture risk in postmenopausal women (29).

Physical Activity and Menopause (Figure 4)

Role of Physical Activity in Menopausal Health
Physical activity is an effective non-pharmacological intervention for menopausal health (30). Regular exercise benefits cardiovascular function, metabolic health, musculoskeletal integrity, psychological well-being, and quality of life (30). Structured activity reduces symptom severity and may help prevent long-term diseases often seen in postmenopausal women (31).

International guidelines recommend adults get 150 minutes of moderate-intensity aerobic activity per week (32). People should also do muscle-strengthening exercises at least two days each week (32). Balance and flexibility training are also encouraged to increase function and reduce fall risk (32).

Aerobic Exercise
Aerobic exercise includes walking, cycling, swimming, jogging, and dancing. Regular participation improves cardiorespiratory fitness and insulin sensitivity (33-35). It also lowers blood pressure, aids weight management, and boosts psychological well-being (33-35). Studies show active menopausal women have fewer symptoms, including better mood, fewer vasomotor symptoms, and overall quality of life (33-35).

Resistance Training

Resistance training is vital during menopause. Declining oestrogen increases loss of skeletal muscle and strength (36-37). Regular strength training helps preserve lean mass, raise metabolic rate, improve glucose metabolism, and maintain bone density (38). It also supports functional performance and independence and reduces the risk of frailty and disability later in life (38).

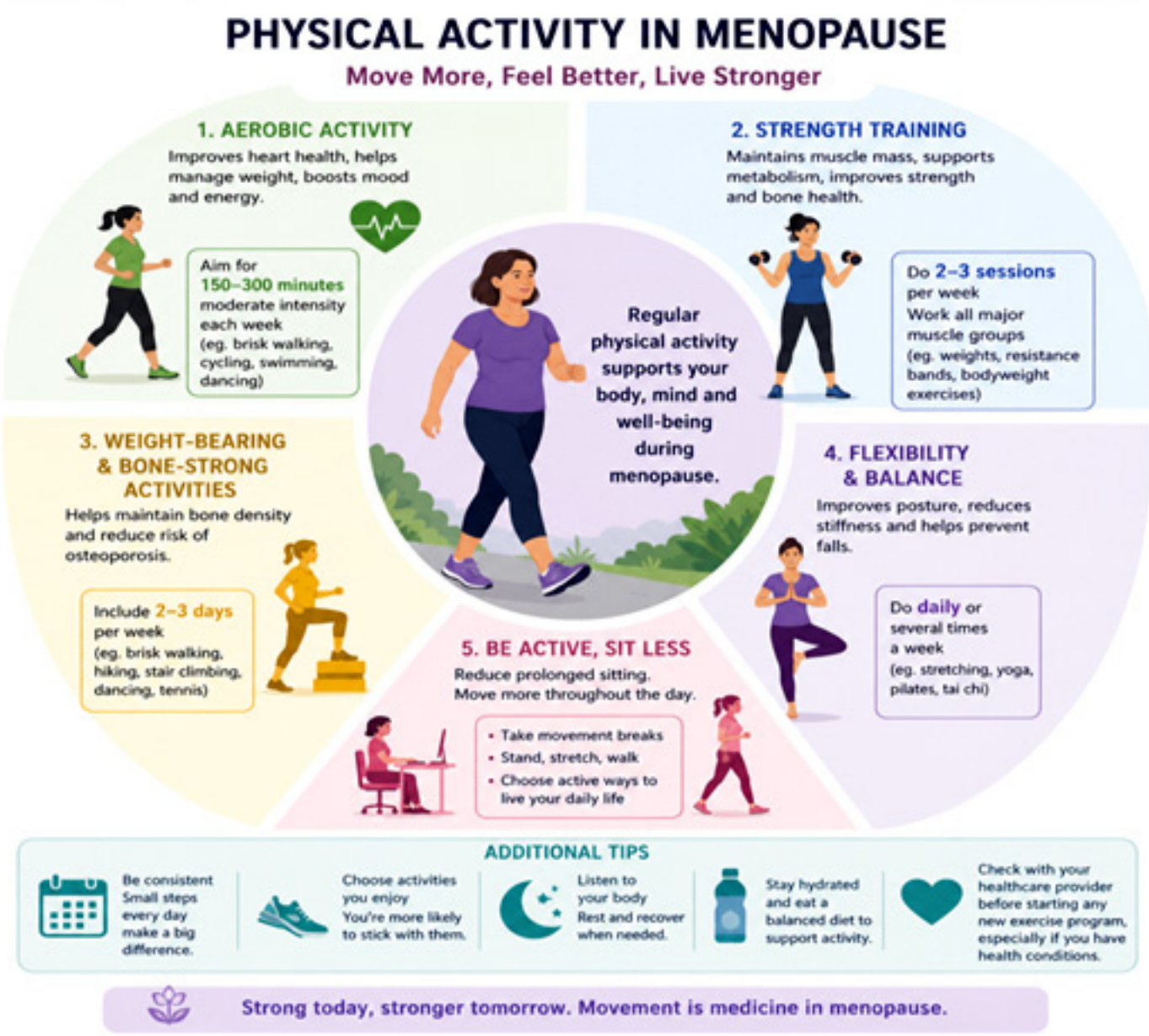
Exercise and Skeletal Health

Weight-bearing and resistance exercises maintain bone health during and after menopause (39-40). Walking, stair climbing, resistance training, dancing, and hiking stimulate bone formation and help slow bone loss linked to oestrogen deficiency. Programs that combine resistance training with impact activities are especially effective at preserving bone density and decreasing the risk of osteoporosis and fractures (39-40).

Exercise and Psychological Wellbeing

Physical activity offers many psychological benefits. These include endorphin release, better sleep, reduced inflammation, and higher self-efficacy (41). Evidence shows regular exercise reduces depression, anxiety, stress, and distress in menopausal women (33-35). Therefore, physical activity is key in comprehensive menopausal care for boosted mental and physical health.

Figure 4: Physical activity in women with menopause



Sleep and Menopause

Sleep Disturbances during Menopause (Figure 5)
Sleep disturbances are among the most prevalent and burdensome symptoms during the menopausal transition (42-47). These issues affect up to 60% of women (42-47). Common complaints include difficulty falling asleep, frequent nighttime awakenings, early-morning awakenings, and non-restorative sleep (42-47). The causes of sleep dysfunction during menopause are multifactorial. They may include vasomotor symptoms, especially nocturnal hot flushes and night sweats, mood disorders, changes in circadian rhythm, obstructive sleep apnoea, and psychosocial stressors (42-47).

Poor sleep quality has major consequences for overall health and wellbeing. It contributes to declined cognitive performance, emotional dysregulation, reduced physical functioning, increased cardiometabolic risk, and lower quality of life (42-47).

Lifestyle-Based Strategies for Improving Sleep
Several lifestyle changes, supported by research (42-47), have been shown to help women in menopause sleep better.

- ***Sleep Hygiene***

Optimizing sleep hygiene is a key part of sleep management. Recommended practices include keeping steady sleep and wake times and creating a comfortable, quiet sleep environment (42-47). Other suggestions are limiting caffeine and other stimulants, avoiding large meals near bedtime, and limiting evening screen exposure (42-47).

- ***Physical Activity***

Regular physical exercise consistently improves sleep quality and capability (34-35). It also reduces insomnia symptoms. Both aerobic and resistance exercise benefit sleep outcomes in midlife women (34-35).

Stress Reduction
Stress-management interventions, such as mindfulness and relaxation techniques, may reduce hyper-arousal, thereby supporting sleep initiation and maintenance (48-50).

Weight Management
Maintaining a healthy weight is important given the rise in obesity and sleep-disordered breathing in midlife. Weight loss improves sleep and reduces the risk and severity of obstructive sleep apnoea (51).

Apart from physical health considerations, it is also important to address stress management and mental health, particularly during the menopausal transition.

Menopause often occurs alongside major life events and stressors, such as job changes, caregiving, aging parents, and shifting relationships. Hormonal changes can also affect mood and mental health (52).

Common psychological symptoms during menopause are anxiety, depression, irritability, mood swings, and lower self-confidence. These may impair daily life and quality of life (52).

Mindfulness-Based Interventions
Mindfulness entails maintaining present-moment awareness with a non-judgemental attitude (48-50). Evidence suggests mindfulness-based interventions can decrease perceived stress, anxiety, and depressive symptoms, while also improving sleep quality plus distress from vasomotor symptoms (48-50).

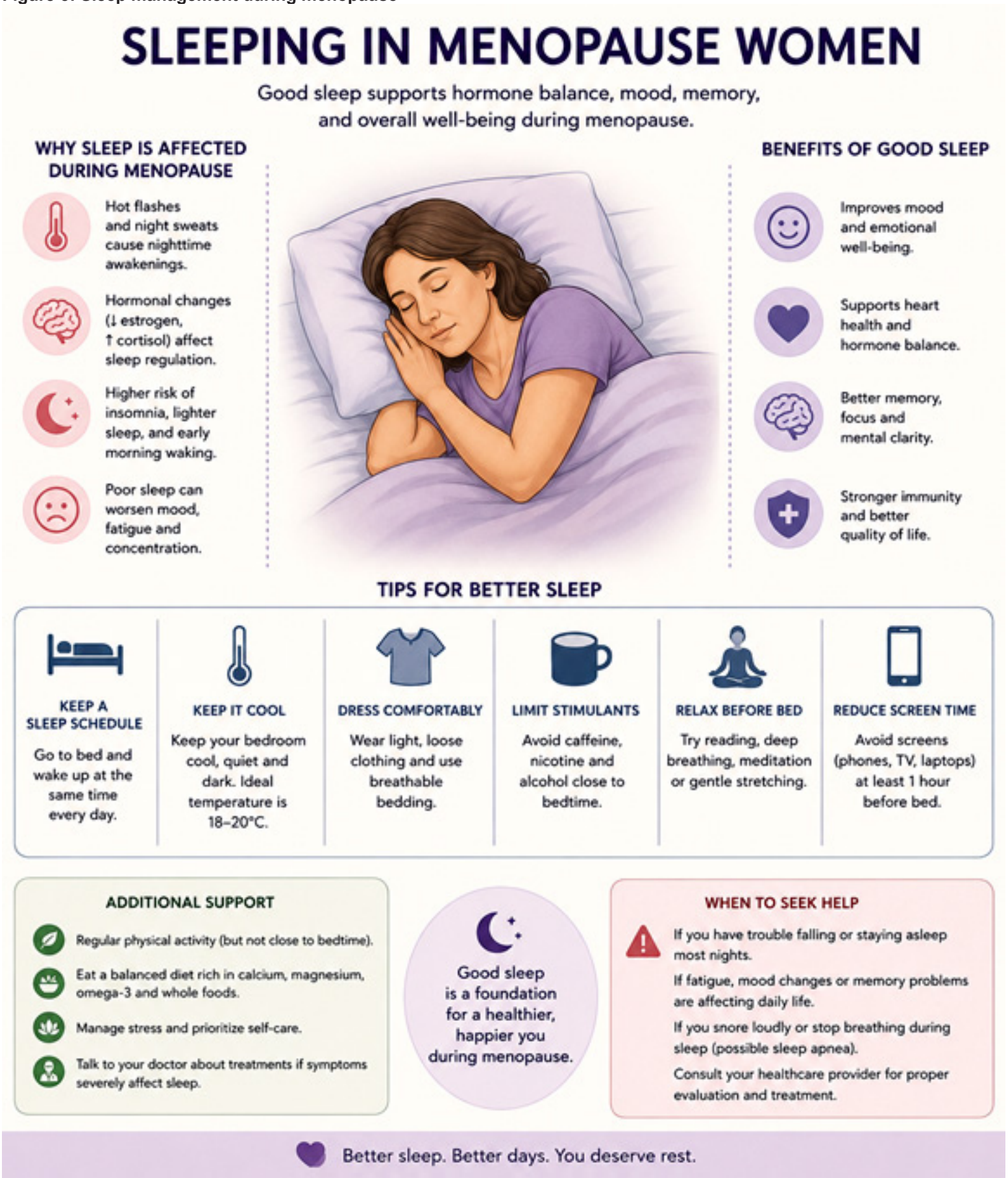
While mindfulness may not reduce the frequency of hot flushes, it can enhance symptom acceptance, coping, and fortitude (48-50).

Cognitive Behavioural Therapy
Cognitive behavioural therapy (CBT) is a well-studied, effective non-pharmacological option for menopausal symptom management. Evidence supports its role in reducing insomnia, anxiety, depressive symptoms, and vasomotor symptom burden, while improving quality of life (48).

CBT helps build coping strategies, supports behaviour change, and identifies and changes unhelpful thought patterns (48).

Relaxation Techniques
Other evidence-backed techniques for mental health include deep breathing, muscle relaxation, yoga, meditation, and tai chi (53-55). These help regulate the nervous system, reduce stress, and build resilience throughout menopause (53-55).

Figure 5: Sleep management during menopause



Social Relationships, Social Support, and Menopausal Health

Social connectedness is an important but often overlooked determinant of health during menopause (56-57). Evidence shows that strong social support networks improve psychological well-being (56-57). They help with coping strategies, promote adherence to therapy, and increase quality of life (56-57). Women with supportive relationships, family, friends, or community, report less menopausal distress and more ability in handling symptoms (56-57).

Group-based lifestyle medicine interventions may offer added benefits (58). They foster social engagement, peer support, and accountability (58). This helps facilitate sustained behavioural change and supports wellbeing.

Avoidance of Dangerous Substances

Smoking
Tobacco use leads to many negative health outcomes during menopause. Smoking is linked to earlier menopause, worse vasomotor symptoms, higher cardiovascular risk, faster bone loss, and poorer health overall (59). Quitting smoking should be a priority for women going through menopause.

Alcohol Consumption
Excessive alcohol use can worsen many menopausal symptoms. These include sleep problems, vasomotor symptoms, and mood complaints (60). Alcohol use also raises the chance of chronic diseases like breast cancer, hypertension, and liver disease (60). Alcohol guidance should be customized to each woman's risk. Promote moderation or abstinence as appropriate.

Cardiovascular Health and Menopause

Cardiovascular disease is the leading cause of death for women worldwide (61). As women approach menopause, their bodies produce less oestrogen. This change leads to negative effects on heart health (62). Women may see higher LDL cholesterol, lower HDL cholesterol, endothelial dysfunction, more abdominal fat, and insulin resistance (62).

Lifestyle medicine is important for lowering the risk of heart disease during and after menopause (63-64). Studies show (63) that a better diet, regular exercise, maintaining a healthy weight, controlling blood pressure, and keeping blood sugar stable all help reduce the risk of heart disease. The time around menopause is key for assessing heart risks, offering prevention advice, and starting healthy lifestyle changes to protect long-term health (64).

Metabolic Health and Diabetes Prevention

Menopause increases the risk of metabolic syndrome and type 2 diabetes (65). This happens because of hormonal and physical changes. The main causes include increased belly fat, lowered muscle mass, greater insulin resistance, and lowered physical activity (65). Together, these issues raise metabolic and heart health risks.

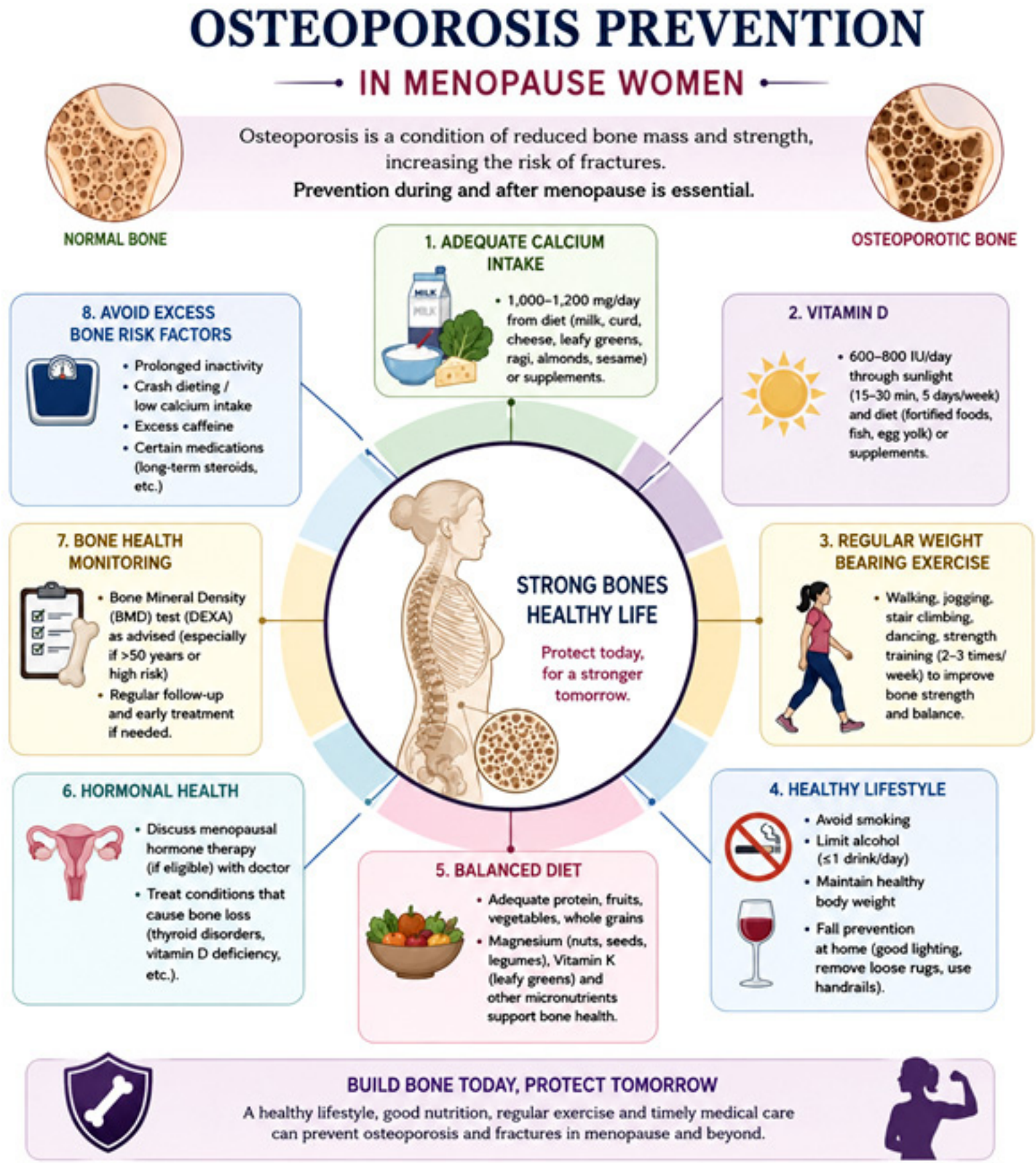
Lifestyle changes are key for preventing diabetes and staying healthy during midlife. Research shows a better diet, regular exercise, and weight control can prevent prediabetes from turning into diabetes (66). These changes also improve blood sugar, insulin sensitivity, body composition, and other health measures.

Osteoporosis Prevention through Lifestyle Medicine

Osteoporosis is a major cause of illness and reduced quality of life for postmenopausal women (40). Lower oestrogen levels accelerate bone loss and reduce bone mineral density, increasing the risk of fractures (36-37).

Lifestyle-based strategies are important for maintaining bone health (40). These include regular resistance training, weight-bearing activity, adequate intake of calcium and vitamin D, quitting smoking, and limiting alcohol intake. Starting these approaches early in menopause may lower fracture risk and support musculoskeletal health in the long term (Figure 6).

Figure 6: Bone health in menopausal women



Sexual Health and Intimate Relationships

Menopause can harm sexual health and relationships in several ways (67-68). Common causes include vaginal dryness, pain during sex, less desire, and psychological difficulties such as anxiety, depression, or low self-esteem (68).

Lifestyle changes can improve sexual wellbeing (69-71). They boost cardiovascular health, enhance body image, lower stress, and sustain mental health. A thorough approach should also include open partner communication, relationship support, and healthcare teams addressing sexual dysfunction during menopause (69-71).

Lifestyle Medicine and Menopausal Hormone Therapy

Use lifestyle medicine alongside standard menopausal care. Do not use it as a replacement for medical therapies. In women on menopausal hormone therapy, lifestyle change can improve overall health and lessen disease risk.

If women cannot or do not want hormone therapy, lifestyle medicine offers useful non-drug ways to control symptoms and prevent disease. Merging medical treatment with lifestyle changes is likely to yield the best results for symptoms, function, and long-term disease prevention (72-73).

Future Directions

State-of-the-art research is revealing new possibilities toward personalized lifestyle medicine in menopausal health. Exciting frontiers comprise precision nutrition, digital health coaching, wearable health-monitoring technologies, microbiome-targeted interventions, and individualized exercise prescription.

Prioritize evaluating long-term clinical outcomes, building effective implementation strategies, and addressing health equity in future research. Take concrete steps to ensure wider access and optimize lifestyle medicine interventions for different populations of menopausal women.

Clinical Trial Evidence Supporting Lifestyle Medicine in Menopause

The body of evidence for lifestyle medicine interventions in menopausal women has grown significantly over the past two decades. Menopausal hormone therapy is still the most effective treatment for vasomotor symptoms. However, a growing number of randomized controlled trials, prospective cohort studies, and systematic reviews show that lifestyle interventions improve menopausal symptoms, cardiometabolic health, psychological well-being, skeletal health, and quality of life. Importantly, lifestyle medicine addresses more than just symptom management (Table 3). It helps prevent chronic diseases that become more common after menopause.

One influential study examining lifestyle modification and menopausal health was the Women's Health Initiative Dietary Modification Trial (74). The study enrolled more than 48,000 postmenopausal women. It tested a low-fat diet that emphasized fruits, vegetables, and whole grains. The main outcomes focused on heart disease and cancer prevention. Secondary analyses showed that women with significant weight loss saw significant decreases in vasomotor symptoms. Those who lost at least 10% of their starting body weight were most likely to report the complete resolution of hot flushes and night sweats. These results provided early evidence that diet and weight management can help control menopausal symptoms.

Further support for behavioural interventions came from the MsFLASH research network (75-76). This multicentre program ran several RCTs on non-hormonal treatments for menopausal symptoms. One trial studied aerobic exercise, yoga, and omega-3 fatty acids in peri- and post-menopausal women with vasomotor symptoms (75-76). Exercise was not always effective in reducing hot flushes. However, researchers observed considerable improvements in sleep quality, physical function, mood, and health-related quality of life. The data show that exercise has clinical benefits beyond direct reduction of vasomotor symptoms.

Psychological interventions have similarly demonstrated substantial efficacy. The MENOS 1 and MENOS 2 randomized controlled trials (77-78) evaluated cognitive behavioural therapy (CBT) in women experiencing bothersome vasomotor symptoms. Both studies (77-78) found significant reductions in hot flush problem ratings, insomnia severity, anxiety, and depressive symptoms. Importantly, these improvements were sustained during long-term follow-up, supporting the durability of CBT-based interventions. Evidence suggests that CBT primarily enhances symptom appraisal, coping strategies, and psychological adjustment rather than directly reducing the frequency of vasomotor symptoms, illustrating the importance of behavioural and cognitive factors in menopause management.

Further evidence is provided by the Women's Healthy Lifestyle Project (WHLP) (79), a longitudinal randomized trial assessing the effects of dietary counselling, structured physical activity, and behavioural support initiated before menopause and continued throughout the menopausal transition. Women assigned to the intervention group experienced significantly less weight gain, smaller increases in waist circumference, and more favourable lipid profiles compared with control participants. This evidence indicates that early adoption of healthy lifestyle behaviours may mitigate many of the adverse metabolic changes associated with menopause.

Exercise-based interventions have regularly demonstrated benefits for cardiometabolic health and functional capacity. The Sedentary Women Exercise Adherence Trial (SMART) (80) evaluated structured aerobic exercise programs among midlife women and found significant improvements in cardiorespiratory fitness, body composition, insulin sensitivity, and mental health. Although the effects on vasomotor symptoms were variable, participants experienced considerable improvements in physical functioning and overall quality of life. These outcomes support current clinical recommendations that advocate regular physical exercise as an essential element of menopause management.

Similarly, the SHAPE-2 trial (81) compared exercise-induced weight loss with dietary-induced weight loss among postmenopausal women. Both approaches produced significant improvements in body composition and metabolic health indicators. However, exercise-induced weight loss was associated with greater preservation of lean muscle mass and superior improvements in physical fitness. These findings highlight the value of integrating physical activity with dietary modification to optimize health outcomes during and after the menopausal transition.

The potential role of nutritional interventions has also been examined through randomized controlled trials investigating phytoestrogen supplementation. Numerous studies evaluating soy isoflavones (82-83) have demonstrated modest reductions in the frequency and severity of vasomotor symptoms. Meta-analyses generally report reductions in hot flush frequency of approximately 20–30% relative to placebo (84). Although the magnitude of benefit is less than that achieved with menopausal hormone therapy, soy-based interventions (84) may represent a useful non-hormonal option for women seeking alternative approaches to symptom management. Variability in treatment response may be attributable to differences in gut microbiota composition, isoflavone metabolism, dosage, and baseline symptom severity.

Collectively, these clinical trials provide robust evidence supporting the integration of lifestyle medicine into comprehensive menopause care. While lifestyle interventions may not consistently achieve the degree of symptom relief observed with hormone therapy, they offer broad-ranging benefits across multiple health domains, including metabolic health, cardiovascular risk reduction, psychological wellbeing, physical function, and long-term disease prevention. Consequently, lifestyle medicine should be considered a foundational component of evidence-based menopause management.

Bone health constitutes a critical target for lifestyle medicine interventions during the postmenopausal period. Evidence from the LIFTMOR (Lifting Intervention for Training Muscle and Osteoporosis Rehabilitation) trial (85) demonstrated that supervised high-intensity resistance and impact-loading exercise significantly increased lumbar spine bone mineral density while enhancing functional performance in postmenopausal women with low bone mass. Importantly, the intervention was shown to be both safe and efficacious when delivered under appropriate professional supervision. These findings support the incorporation of resistance and impact-loading exercise as fundamental components of osteoporosis prevention and musculoskeletal health promotion strategies in postmenopausal populations.

Mindfulness-based interventions have appeared as promising non-pharmacological approaches for menopausal symptom management. Several randomized controlled trials of mindfulness-based stress reduction (MBSR) programs have shown significant improvements in perceived stress, anxiety, sleep quality, and menopause-specific quality of life (86). These interventions appear to have limited effects on the frequency of vasomotor symptoms but are associated with reductions in symptom-related distress and enhanced psychological strength (86). These findings stress the importance of handling emotional, cognitive, and psychosocial dimensions of menopause, as well as its physical symptoms (86).

Table 3: Evidence base trials on lifestyle medicine and menopause

Author(s)	Trial	Intervention	Outcome(s)
Howard et al (92)	the Women's Health Initiative Dietary Modification Trial	Tested a low-fat diet	Weight loss and dietary interventions may help reduce vasomotor symptoms and improve menopausal symptom control.
Cohen et al (75) Newton et al (76)	MsFLASH research network	3×2 factorial: Aerobic exercise and yoga vs. usual activity, plus omega-3 supplementation vs. placebo capsule	Yoga, exercise, and omega-3 supplementation were not associated with significant improvements in vasomotor symptoms.
Ayres et al (78) Mann et al (77)	MENOS 1 and MENOS 2	cognitive behavioural therapy (CBT) in women experiencing bothersome vasomotor symptoms	Both studies showed sustained improvements in hot flush symptoms, insomnia, anxiety, and depression, supporting the long-term effectiveness of CBT-based interventions.
Simkin-Silverman et al (79)	the Women's Healthy Lifestyle Project	Lifestyle interventions including dietary counselling, exercise, and behavioural support during the menopausal transition	Healthy lifestyle behaviours may help reduce metabolic changes associated with menopause, including weight gain, central fat accumulation, and unfavourable lipid profile changes.
Cox et al (80)	The Sedentary Women Exercise Adherence Trial	structured aerobic exercise programs among midlife women	Exercise interventions improved cardiometabolic health, physical functioning, mental well-being, and quality of life, despite inconsistent effects on vasomotor symptoms.
van Gemert et al (81)	SHAPE-2 trial	compared exercise-induced weight loss with dietary-induced weight loss among postmenopausal women	Both dietary and exercise-induced weight loss improved body composition and metabolic health, although exercise provided greater preservation of lean mass and improved physical fitness
Taku et al (84)	Extracted or synthesized soybean isoflavones reduce menopausal hot flush frequency and severity; systematic review and meta-analysis of randomized controlled trials	Efficacy of extracted or synthesized soybean isoflavones in the alleviation of hot flushes in perimenopausal and postmenopausal women	Soy isoflavone supplements, derived by extraction or chemical synthesis, are significantly more effective than placebo in reducing the frequency and severity of hot flushes
Harding et al (85)	LIFTMOR trial	High-intensity resistance and impact exercise improved lumbar spine bone density and physical function in postmenopausal women with low bone mass.	The intervention was shown to be both safe and efficacious when delivered under appropriate professional supervision
Wong et al (86) Aliabadi et al (93)	randomized controlled trials of mindfulness-based stress reduction (MBSR) programs	This study aimed to assess the effectiveness of Mindfulness-Based Stress Reduction (MBSR) in alleviating menopause-related symptoms, compared with an active control condition, the Menopause Education Control (MEC)	MBSR show a greater reduction of psychological symptoms of depression and anxiety above active controls but do not reduce other somatic, urogenital and vasomotor symptoms.

A broad body of evidence from systematic reviews and meta-analyses supports the assimilation of lifestyle medicine into menopause care. Cochrane reviews of exercise interventions have shown improvements in physical functioning, cardiovascular fitness, and health-related quality of life. Meta-analyses of cognitive behavioural therapy show significant reductions in hot flush-related distress, insomnia severity, anxiety, and depressive symptoms. Reviews of Mediterranean diets have found favourable effects on cardiovascular risk profiles, inflammatory biomarkers, body composition, and general health outcomes. There is also emerging evidence that mindfulness-based interventions promote psychological wellbeing and improve sleep quality.

Key recommendations for clinical practice include routinely prioritizing lifestyle medicine as a core component of menopausal care; combining evidence-based lifestyle interventions with pharmacologic therapy when symptoms warrant; regularly evaluating cardiovascular, metabolic, skeletal, psychological, and quality of life outcomes; and individualizing care within a patient-centred model to optimize healthy aging and long-term function. This integrated strategy maximizes health benefits for menopausal women.

Conclusion

Menopause is a major physiological transition. It is characterized by a range of acute symptoms and chronic health consequences that may substantially affect quality of life and general health outcomes. Menopausal hormone therapy remains the most effective treatment for many menopausal symptoms, particularly vasomotor symptoms. However, lifestyle medicine affords a comprehensive, evidence-based approach to supporting health and wellbeing throughout the menopausal transition and beyond.

The six core pillars of lifestyle medicine are healthy nutrition, routine physical activity, refreshing sleep, effective stress management, avoidance of toxic substances, and positive social connections. These pillars address the complex biological, psychological, and social determinants that influence menopausal health. Emerging evidence indicates that these interventions can reduce the burden of vasomotor symptoms. They can also improve psychological well-being and sleep quality, preserve bone and musculoskeletal health, optimize body weight and metabolic function, reduce cardiovascular risk, and increase overall quality of life.

Women now spend a substantial proportion of their lives in the postmenopausal period. Integrating lifestyle medicine into routine menopause care is a sustainable and effective strategy for promoting healthy aging and sustained well-being. A multidisciplinary, patient-centred model combines evidence-based lifestyle interventions with appropriate medical therapies. This combination is likely to provide the most comprehensive and effective approach to menopause management.

Acknowledgment:
This review is dedicated to women undergoing the menopausal transition. Their experiences and fortitude continue to inform and advance research in women's health. This review intends to synthesize current, evidence-based knowledge on lifestyle interventions that support physical, psychological, and metabolic wellness during menopause. It integrates recent findings on nutrition, physical activity, stress management, and behavioural strategies. The goal is to provide clinically relevant insights to help optimize health results and quality of life during this important life stage.

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Beauty, Identity, and the Psychology of Perfection in the Age of Digital Influence

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Abstract

Social media, influencers, and commercial marketing have transformed beauty into a relentless pursuit of perfection, increasing pressure on women and, increasingly, men to undergo cosmetic procedures. This paper examines the rapidly changing landscape of beauty standards and cosmetic enhancement in modern society and explores how these influences shape self-image, redefine identity, and impact mental well-being, while encouraging repeated aesthetic enhancement. It also considers the role of appearance-related psychological conditions, including Body Dysmorphic Disorder, in motivating cosmetic intervention for some individuals. The paper advocates for ethical, patient-centred practice that incorporates psychological awareness, informed consent, and realistic expectations, prioritising mental well-being, authenticity, and patient safety over commercial interests and unrealistic beauty ideals.

Keywords: cosmetic procedures, body dysmorphia, influencers, and social media

Synopsis

What was once subtle self-expression has evolved into a highly visible and often extreme culture of bodily modification. Procedures ranging from permanent makeup, fillers, and botulinum toxin to thread lifts, fat transfer, and body sculpting are now widely promoted as quick, normal, and empowering solutions. However, beneath this surface of empowerment lies a complex interplay of psychological vulnerability, social pressure, and ethical concerns that significantly impact mental health, particularly among women, while increasingly affecting men as well.

At the centre of this transformation is the powerful role of social media. Platforms such as Instagram, TikTok, and Snapchat which amplify idealised and often heavily filtered images of beauty. Influencers and marketing campaigns promote "flawless skin," "glow-ups," and "quick fixes," often masking and overlooking the reality of medical risk and long-term consequences. These complicated narratives create unrealistic expectations and normalise constant aesthetic alteration, and call for it pliably. Individuals, particularly younger audiences, are repeatedly exposed to curated perfection, leading to constant comparison, dissatisfaction, and internalised pressure to conform. By then, souls lose their compass, scope, and hearts forget themselves, and only start the haunting process of seeking perfection, which will keep looping like a non-stop cycle.

This competitive bombarding environment contributes significantly to body image disturbances, distortion, and the impending mental health challenges. One of the most important conditions linked to this phenomenon is Body Dysmorphic Disorder (BDD). BDD is a psychiatric condition, often underrecognized, and is defined in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders as a preoccupation with a perceived defect or flaw in one's physical appearance when, in fact, they appear normal or invisible, and which are often minor or not observable to others. BDD is a prevalent psychiatric condition characterized by an all-consuming focus on perceived physical imperfections, leading to distressing repetitive actions and, at times, suicidal behaviour and ideation. Individuals with BDD often seek unnecessary surgical interventions (1). Usually, the affected patients are engaged in repetitive behaviours like excessive mirror checking, excessive grooming, excessive camouflage, and comparing their own appearance to others, seeking attention and validation from the exterior, which is time-consuming, uncontrollable, frustrating, and ultimately distressing. Such patients are mostly dermatology and plastic surgeons' regular visitors for any flaws, imperfections, and demand repair and correction, even if it is extreme, excessive, or too much, for their perceived defects and flaws.

I once heard of a man who underwent many extensive and expensive transformative surgeries and yet was still not content or satisfied, and declared openly on social media that he would go big and proud to be doing so.

BDD has a complex aetiology because multiple biological, psychological, and socio-environmental factors have been projected as having a part in the development of BDD (1).

The history of BDD goes back in time, when Morselli, Janet, and Freud described body dysmorphia, causing obsessive appearance distress, leading to shame and distress due to their perceived ugliness (2).

Individuals with BDD may experience severe anxiety, depression, social withdrawal, and compulsive behaviours such as repeated cosmetic procedures in pursuit of an unattainable ideal. Even after multiple interventions, satisfaction is rarely achieved because the underlying psychological distress remains unaddressed. Some have gone so far as to request procedures to replicate and mimic their idol celebrities' looks and idolize them. I recall the most heavily copied figures included Angelia Jolie's face, with great emphasis on her jawline and an alluring, seductive, curvy, pillowy volume, distinctive, luxuriously thick voluptuous beauty lip shape; Kim Kardashian's buzzy exaggerated hour glass figure, ultra-curvaceous enhanced backside with curvy butt lift and pronounced hips paired with extremely narrowed cinched waist, slim thick proportions and contours; Kylie Jenner for exaggerated hour glass contours and curved hips; and Megan Fox for siren eyes and a sculpted face, and tailored bones and defined curves. Some have asked for a cat-eye operation. All of this causes them to lose their natural uniqueness and identity, creating a homogenized 'clone' look, like copy-and-paste with the same, more or less, outcome (2).

Those cosmetic procedures are often marketed as solutions to improve confidence and boost self-esteem. However, in many cases, they fail to address the psychological drivers behind the desire for change, the transformation, and the mutation. Instead, some individuals enter cycles of repeated procedures, chasing temporary gratification, to numb those internal urges and calls for perfection and flawlessness. This cycle can deepen emotional distress, reinforce self-criticism, and intensify feelings of inadequacy and insufficiency. In extreme cases, individuals may lose connection with their original identity, forget their original physical look, and constantly seek external validation and approval through physical transformation and makeover.

This brings up a vital concern about the role of medical and aesthetic practitioners within this system. While many clinicians prioritise patient safety and ethics, there is growing concern that some providers focus primarily on profitability rather than psychological assessment and informed consent. Patients are often not adequately questioned about the underlying drive and motivation for seeking those gross procedures. There is insufficient exploration of whether the desire for change is driven by social media pressure to portray perfection, ignore underlying trauma, low self-esteem, or body dysmorphia. Without this thorough understanding, procedures may be performed on vulnerable individuals without addressing the root cause of their distress.

In fact, the hurried commercialisation of aesthetics has led to the rapid expansion of cosmetic industries into nearly every aspect of appearance, and the cycle loops on forever, endlessly, from one aspect to another, nonstop. Hair, nails, skin, body shape, and facial features are all subject to modification and alteration irreversibly. Some even promote a whole package to attract more clients under the so-called alluring sales and savings, which some cannot ignore and will be tempted to have a go. The aesthetic extended to every part of the body, and just to name a few. For instance, eyelash extensions, which involve using glue and or more recently evolving by using magnetism to adhere, forgetting its implications in the long run for eyelashes thinning and falling, besides triggering allergy and sensitivity from the glue's ingredients, microblading and the synthetic sharp intense bold dark eyebrows look where all women nowadays have that dark thick drawn like lines and strokes that mimic the appearance of bird feather and as the face is overly made up, nail sculpting, nail damage from UV-cured gels and glues, skin irritation from adhesives, also long acrylic nails of various forms and recently the trend seems to be with pointed nails ending like a spike with various ornaments placed on the nail surface by gluing, where the wearer can hardly type or pick up things, or just type with the tip of their extended acrylic nail as I have seen some do and this will act as a mechanical leverage from the repetitive trauma and will cause vibration to the nail bed, causing onycholysis (separation), joint strain and weakening of the nail's natural structure, hair extensions which involve clipping and traction leading to traction alopecia, hair entangling and matting from plaiting and braiding. Tattooed makeup is widely accepted, but they are not risk-free either. The long-term dependence on aesthetic maintenance is increasingly observed (3).

Facial procedures such as dermal fillers and botulinum toxin injections have become especially prominent and pronounced. While they are often presented as non-invasive and reversible, repeated use can lead to complications such as tissue distortion, loss of natural facial expression, and long-term structural changes, creating plastic creatures, extra-terrestrials, and 'aliens'. Some individuals develop an overfilled or "puffy" appearance, often referred to in clinical settings as filler-related facial distortion, or overdone faces. In certain cases, permanent fillers or poorly administered products can cause irreversible changes, infections (biofilm), or nodular formations that are difficult or impossible to fully correct, as some harbour certain bacteria, difficult to eradicate (Figure 1).

Mental health consequences across this spectrum are significant. Anxiety, depression, nonstop social comparison stress, and identity disturbance and distortion are commonly observed in individuals heavily exposed to aesthetic pressures. Repeated cosmetic interventions may also reinforce avoidance behaviours, where individuals rely on external modification rather than psychological healing. Over time, this can contribute to emotional dependence on aesthetic procedures as a coping mechanism for deeper psychological distress (4). The question is, do those procedures bring a sense of feeling good and content, or ask for more?

Ethically, the paper raises concerns about informed consent and patient-centred care. True consent requires not only explanation of physical risks but also exploration of psychological readiness and expectations. Without addressing the emotional drivers behind cosmetic desire, medical interventions risk becoming superficial solutions to complex mental health issues. Clinicians are encouraged to prioritise safety, honesty, and psychological screening, ensuring that procedures are not driven solely by commercial demand.

In conclusion, the evolution of beauty has created a complex intersection between culture, commerce, and mental health issues. While cosmetic enhancement can offer confidence and personal satisfaction for some, it also carries significant psychological and physical risks when driven by external pressure or unresolved emotional distress. Thus, a balanced, ethical approach is required, one that prioritises mental health, informed decision-making, and authenticity over profit and idealised perfection.

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Figure 1: Overdone ® by E. Elghblawi

Plastic Mirrors
Frozen borrowed smiles,
Filled lips, curves chase hollow beauty
True selves blur

Other Dementias: A Comprehensive Review of Normal Pressure Hydrocephalus, Creutzfeldt–Jakob Disease, Huntington's Disease, HIV-Associated Dementia, and Toxin- and Infection-Related Cognitive Disorders

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Abstract

Background. Although Alzheimer's disease and vascular dementia account for the majority of dementia cases worldwide, several less common disorders collectively contribute substantially to cognitive impairment, disability, caregiver burden, and healthcare utilization. These conditions include normal pressure hydrocephalus (NPH), Creutzfeldt–Jakob disease (CJD), Huntington's disease (HD), HIV-associated dementia (HAD), and toxin- and infection-related dementias. Unlike many common neurodegenerative dementias, several of these disorders are potentially reversible or amenable to disease-specific interventions, making early recognition clinically important.

Objective. To provide a comprehensive review of the epidemiology, pathophysiology, clinical manifestations, diagnosis, management, prognosis, and future directions of major non-Alzheimer dementias encountered in neurological and geriatric practice.

Methods. A narrative review approach was employed using contemporary evidence from peer-reviewed literature, international guidelines, and major neurology, infectious disease, and dementia references. Emphasis was placed on diagnostic advances, biomarker development, neuroimaging findings, and emerging therapeutic strategies.

Results. NPH remains one of the most treatable causes of cognitive decline, with cerebrospinal fluid diversion producing meaningful improvement in appropriately selected patients. CJD represents a rapidly progressive prion disease characterized by severe neurological deterioration and poor survival. HD is an autosomal dominant neurodegenerative disorder caused by CAG repeat expansion within the huntingtin gene and serves as a model for gene-targeted therapeutic development. HIV-associated dementia has become less common in the era of combination antiretroviral therapy but continues to contribute significantly to morbidity. Toxin- and infection-related dementias encompass a heterogeneous group of disorders in which early diagnosis may prevent irreversible neurological injury.

Conclusions. The biological diversity of these disorders highlights the need for individualized diagnostic and therapeutic approaches. Advances in biomarkers, neuroimaging, genetics, artificial intelligence, and precision medicine are expected to transform the evaluation and management of these conditions. Improved recognition of potentially reversible causes of dementia remains essential for optimizing patient outcomes.

Keywords: Dementia; Normal Pressure Hydrocephalus; Creutzfeldt–Jakob Disease; Huntington's Disease; HIV-Associated Dementia; Neurocognitive Disorders; Prion Disease; Cognitive Impairment; Biomarkers; Precision Medicine.

Overview

Introduction

Dementia is a clinical syndrome characterized by acquired cognitive decline sufficient to interfere with independence in daily functioning. According to the World Health Organization, more than 55 million individuals worldwide are currently living with dementia, a number expected to increase substantially as populations age (World Health Organization [WHO], 2023). Although Alzheimer's disease and vascular dementia account for most cases, a variety of less common neurological, infectious, genetic, and toxic disorders contribute significantly to the global burden of cognitive impairment.

The term "other dementias" encompasses a heterogeneous group of disorders that differ markedly in etiology, pathophysiology, clinical course, and treatment responsiveness. Some conditions are progressive neurodegenerative disorders associated with irreversible neuronal loss, whereas others may stabilize or improve if diagnosed and treated early. Recognition of these distinctions is essential because therapeutic opportunities and prognostic implications vary considerably among disease categories.

This review focuses on five major groups of non-Alzheimer dementias: normal pressure hydrocephalus, Creutzfeldt–Jakob disease, Huntington's disease, HIV-associated dementia, and toxin- or infection-related dementias. Together, these conditions illustrate the diverse biological mechanisms capable of producing cognitive decline.

Normal Pressure Hydrocephalus

Normal pressure hydrocephalus is a potentially reversible neurological disorder characterized by enlargement of the cerebral ventricles despite normal or intermittently elevated cerebrospinal fluid (CSF) pressure. The classic clinical triad consists of gait disturbance, cognitive impairment, and urinary dysfunction (Nakajima et al., 2021). Among these manifestations, gait impairment is typically the earliest and most prominent symptom.

NPH is particularly important because it represents one of the few treatable causes of dementia. Appropriate patient selection for ventriculoperitoneal shunting may result in significant improvement in mobility, functional status, and quality of life. However, diagnosis remains challenging because symptoms often overlap with those of Alzheimer's disease, vascular dementia, and Parkinsonian syndromes.

Creutzfeldt–Jakob Disease

Creutzfeldt–Jakob disease is a rare but devastating prion disorder characterized by rapidly progressive cognitive decline, motor dysfunction, and death. The disease results from abnormal folding of the cellular prion protein into a pathogenic isoform capable of inducing further protein misfolding and neurodegeneration (Hermann et al., 2021).

Unlike most dementias, which progress over years, CJD often evolves over weeks to months. Clinical manifestations commonly include dementia, myoclonus, cerebellar dysfunction, visual disturbances, pyramidal signs, and akinetic mutism during advanced stages. The introduction of real-time quaking-induced conversion (RT-QuIC) assays has substantially improved diagnostic accuracy and facilitated earlier recognition of prion disease.

Huntington's Disease

Huntington's disease is an autosomal dominant neurodegenerative disorder caused by expansion of CAG trinucleotide repeats within the huntingtin (HTT) gene on chromosome 4. The disease is characterized by a combination of motor, cognitive, and psychiatric manifestations (McColgan & Tabrizi, 2018).

Although chorea is the most recognizable clinical feature, cognitive impairment and behavioural disturbances often contribute more substantially to long-term disability. Progressive degeneration of the striatum and associated neural networks ultimately results in severe functional dependence. Importantly, Huntington's disease has become a major focus of precision medicine research because its genetic basis is clearly defined, creating opportunities for targeted therapeutic interventions.

HIV-Associated Dementia

HIV-associated dementia represents the most severe end of the spectrum of HIV-associated neurocognitive disorders (HAND). Prior to the widespread availability of combination antiretroviral therapy (ART), HAD was a common neurological complication of advanced HIV infection. Although its incidence has declined significantly, milder forms of neurocognitive impairment remain prevalent among people living with HIV (Nightingale et al., 2023).

The pathogenesis of HAD involves chronic neuroinflammation, immune activation, viral persistence within the central nervous system, and indirect neuronal injury. Clinical manifestations typically include psychomotor slowing, executive dysfunction, impaired attention, and behavioural changes. Early diagnosis and optimization of antiretroviral therapy remain essential components of management.

Toxin- and Infection-Related Dementias

A variety of environmental toxins and infectious agents can produce cognitive impairment and dementia. Toxic causes include chronic alcohol misuse, heavy metal exposure, carbon monoxide poisoning, and certain occupational or environmental neurotoxins. Infectious causes include neurosyphilis, tuberculosis, cryptococcosis, progressive multifocal leukoencephalopathy, Whipple disease, and other central nervous system infections.

These conditions are clinically important because many are partially or completely reversible when recognized early. Consequently, evaluation of potentially treatable causes remains a fundamental component of every dementia assessment. Failure to identify such disorders may result in preventable neurological disability and unnecessary progression of cognitive decline.

Comparative Features of Major Other Dementias

Table 1. Major Characteristics of Selected Other Dementias

Disorder	Primary Mechanism	Typical Progression	Potential Reversibility
Normal Pressure Hydrocephalus	CSF circulation dysfunction	Months–years	Moderate to high
Creutzfeldt–Jakob Disease	Prion protein misfolding	Weeks–months	None
Huntington's Disease	HTT gene mutation	Years–decades	None
HIV-Associated Dementia	Neuroinflammation and viral injury	Variable	Partial
Toxin-Related Dementias	Neurotoxic exposure	Variable	Often partial
Infection-Related Dementias	CNS infection	Variable	Often substantial

Significance of Early Recognition

Despite their relative rarity compared with Alzheimer's disease, these disorders hold disproportionate clinical importance because several are potentially treatable, require specific management strategies, or have unique implications for patients and families. Early diagnosis can facilitate timely intervention, improve functional outcomes, guide prognostic counseling, and enable access to disease-specific therapies.

Advances in neuroimaging, molecular diagnostics, genetic testing, and biomarker research are increasingly allowing clinicians to identify these disorders at earlier stages than previously possible. As a result, the distinction between neurodegenerative, infectious, toxic, and potentially reversible causes of dementia has become increasingly important in contemporary neurological practice.

Etiology and Epidemiology

Etiology
The disorders discussed in this review represent diverse pathogenic mechanisms leading to cognitive impairment and dementia. Unlike Alzheimer's disease, which is primarily characterized by amyloid-β and tau pathology, these conditions arise from disturbances in cerebrospinal fluid dynamics, protein misfolding, genetic mutations, chronic viral infection, toxic exposures, or direct central nervous system (CNS) infections. Understanding their etiological basis is essential because treatment options, prognosis, and opportunities for prevention vary considerably among disorders.

Normal Pressure Hydrocephalus
Normal pressure hydrocephalus (NPH) results from impaired cerebrospinal fluid (CSF) circulation and absorption, leading to progressive ventricular enlargement despite normal or intermittently elevated CSF pressure (Nakajima et al., 2021). The condition is traditionally classified as either idiopathic or secondary.
Idiopathic normal pressure hydrocephalus (iNPH) accounts for most cases and occurs without an identifiable precipitating event. Although the exact pathogenesis remains incompletely understood, current evidence suggests dysfunction of CSF resorption pathways, altered intracranial compliance, impaired glymphatic clearance, and age-related vascular changes contribute to disease development.

Secondary NPH occurs following conditions that disrupt normal CSF dynamics, including:
- Subarachnoid hemorrhage
- Traumatic brain injury
- Meningitis
- Intracranial surgery
- Other inflammatory CNS disorders

The resulting impairment of CSF circulation leads to ventricular dilation and secondary disruption of periventricular white matter pathways involved in gait, cognition, and bladder control.

Creutzfeldt–Jakob Disease
Creutzfeldt–Jakob disease (CJD) is a transmissible spongiform encephalopathy caused by the conformational conversion of the normal cellular prion protein (PrP^C) into the pathogenic isoform (PrP^Sc), resulting in the accumulation of protease-resistant prion aggregates and progressive neurodegeneration. (Hermann et al., 2021).

Four major forms of CJD are recognized:

Sporadic CJD
Sporadic CJD accounts for approximately 85–90% of cases and arises without an identifiable cause. The initiating event remains uncertain but is believed to involve spontaneous conformational alteration of normal prion protein.

Genetic CJD
Approximately 10–15% of cases are associated with pathogenic mutations in the prion protein gene (PRNP). More than 50 disease-associated mutations have been identified, producing variable clinical phenotypes and disease durations.

Iatrogenic CJD
Rare cases have occurred following exposure to contaminated biological materials, including:
- Human growth hormone preparations
- Dura mater grafts
- Corneal transplantation
- Neurosurgical instruments

Variant CJD
Variant CJD is associated with exposure to bovine spongiform encephalopathy ("mad cow disease") and demonstrates distinct clinicopathological characteristics.

Huntington's Disease
Huntington's disease (HD) is an autosomal dominant neurodegenerative disorder caused by expansion of a CAG trinucleotide repeat within exon 1 of the huntingtin (HTT) gene located on chromosome 4p16.3 (McColgan & Tabrizi, 2018).

Normal individuals typically possess fewer than 27 CAG repeats, whereas pathogenic expansions usually contain 40 or more repeats. Expanded repeats produce an abnormal huntingtin protein that undergoes aggregation, disrupts intracellular signaling pathways, impairs mitochondrial function, and ultimately causes neuronal death, particularly within the striatum.

A characteristic feature of HD is genetic anticipation, whereby larger repeat expansions are transmitted to subsequent generations, often resulting in earlier disease onset and more severe clinical manifestations.

HIV-Associated Dementia
HIV-associated dementia (HAD) represents the most severe manifestation of HIV-associated neurocognitive disorders (HAND). The disorder results not from direct neuronal infection but from indirect neuronal injury mediated by infected macrophages, microglia, chronic immune activation, and persistent neuroinflammation (Nightingale et al., 2023).

Mechanisms contributing to cognitive impairment include:
- Chronic neuroinflammatory activation
- Release of neurotoxic cytokines
- Synaptic dysfunction
- White matter injury
- Blood-brain barrier disruption
- Persistent viral reservoirs within the CNS

The widespread implementation of combination antiretroviral therapy (ART) has significantly altered disease epidemiology and reduced the incidence of severe dementia.

Toxin-Related Dementias
Numerous environmental and occupational toxins have been implicated in cognitive impairment. The severity of neurological injury depends upon exposure duration, cumulative dose, patient susceptibility, and timeliness of intervention.

Major causes include:
Alcohol
Chronic alcohol misuse may result in:
- Alcohol-related dementia
- Wernicke encephalopathy
- Korsakoff syndrome

through direct neurotoxicity, nutritional deficiency, and disruption of neuronal metabolism (Zis et al., 2021).

Heavy Metals
Neurotoxic metals associated with cognitive decline include:
- Lead
- Mercury
- Arsenic
- Manganese

These substances promote oxidative stress, mitochondrial dysfunction, and neuronal injury.

Carbon Monoxide
Carbon monoxide poisoning produces cerebral hypoxia and delayed neuropsychiatric sequelae, which may include persistent cognitive impairment and executive dysfunction.

Infection-Related Dementias
A wide range of infectious diseases can affect cognitive function either directly through CNS invasion or indirectly through inflammatory and vascular mechanisms.

Important causes include:
Neurosyphilis
Untreated infection with Treponema pallidum may produce progressive cognitive decline, personality change, psychiatric symptoms, and dementia.

Tuberculosis
Tuberculous meningitis may cause chronic cognitive impairment through meningeal inflammation, hydrocephalus, cerebral infarction, and parenchymal injury.

Cryptococcal Infection
Cryptococcal meningoencephalitis remains an important cause of cognitive dysfunction among immunocompromised individuals.

Progressive Multifocal Leukoencephalopathy
Progressive multifocal leukoencephalopathy (PML) results from reactivation of the John Cunningham (JC) virus and causes demyelination of cerebral white matter.

Whipple Disease
Although rare, Whipple disease may produce cognitive decline, psychiatric symptoms, and movement disorders.

Epidemiology
The epidemiological characteristics of these disorders differ substantially. Some conditions are relatively common among older adults, whereas others remain rare but clinically significant because of their severity, treatability, or unique diagnostic implications.

Normal Pressure Hydrocephalus
NPH is increasingly recognized as an important cause of potentially reversible cognitive decline in older adults. Population-based studies estimate prevalence rates ranging from 0.2% to 2.9% among individuals over 65 years of age, with prevalence increasing markedly in advanced age groups (Nakajima et al., 2021).

Many experts believe NPH remains underdiagnosed because symptoms overlap with more common neurodegenerative disorders. Improved neuroimaging and greater awareness have contributed to increasing recognition worldwide.

Creutzfeldt–Jakob Disease
CJD is rare but occurs globally. The annual incidence remains remarkably consistent across populations at approximately 1–2 cases per million persons per year (Hermann et al., 2021).

Sporadic CJD typically presents between 60 and 70 years of age. Variant CJD remains exceedingly uncommon following implementation of public health measures designed to control bovine spongiform encephalopathy.

Huntington's Disease
The prevalence of HD varies considerably by geographic region and ancestry. Rates are highest in populations of European descent, where prevalence generally ranges from 5 to 14 cases per 100,000 individuals (McColgan & Tabrizi, 2018).

Lower prevalence rates have historically been reported in Asian and African populations, although improved genetic testing has increased recognition globally.

HIV-Associated Dementia
Before the introduction of effective ART, HAD affected approximately 15–30% of individuals with advanced HIV infection. Although severe dementia has become less common, milder forms of HIV-associated neurocognitive impairment continue to affect a substantial proportion of patients living with HIV (Nightingale et al., 2023).
The burden is greatest in regions with high HIV prevalence and limited healthcare resources.

Toxin- and Infection-Related Dementias
Reliable epidemiological estimates are challenging because these conditions encompass numerous etiologies and frequently remain under-recognized.

The prevalence of toxin-related cognitive impairment varies according to:
- Occupational exposures
- Environmental regulations
- Alcohol consumption patterns
- Public health infrastructure

Similarly, infection-related dementias demonstrate considerable geographic variability. Neurosyphilis remains more common in regions with limited access to screening and treatment, whereas tuberculosis-related cognitive impairment is concentrated in countries with high tuberculosis prevalence.

Comparative Epidemiology

Table 2. Global Epidemiological Characteristics of Major Other Dementias

Disorder	Estimated Prevalence/Incidence	Typical Age at Onset
Normal Pressure Hydrocephalus	0.2–2.9% of adults >65 years	>60 years
Creutzfeldt–Jakob Disease	1–2 cases per million/year	60–70 years
Huntington's Disease	5–14 per 100,000 (Western populations)	30–50 years
HIV-Associated Dementia	Variable; reduced in ART era	Any age with HIV
Toxin-Related Dementias	Variable	Exposure dependent
Infection-Related Dementias	Variable	Infection dependent

Clinical Implications
The epidemiological diversity of these disorders highlights the importance of maintaining a broad differential diagnosis when evaluating cognitive impairment. While rare diseases such as CJD and HD account for a small proportion of dementia cases, their diagnostic and prognostic implications are substantial. Conversely, disorders such as NPH, HIV-associated dementia, and several toxin- or infection-related conditions may be partially reversible, emphasizing the value of early recognition and targeted intervention.

Collectively, these conditions illustrate the need for a comprehensive diagnostic approach that incorporates clinical assessment, neuroimaging, laboratory testing, genetic evaluation, and consideration of potentially treatable causes of cognitive decline.

Genetic Factors and Pathophysiology

The dementias reviewed in this article arise through diverse biological mechanisms, ranging from inherited genetic mutations and protein misfolding disorders to chronic infection, neuroinflammation, toxic injury, and disturbances in cerebrospinal fluid (CSF) dynamics. Despite these differences, many ultimately converge on common pathways involving synaptic dysfunction, neuronal loss, network disconnection, and progressive cognitive impairment.

Genetic Factors
Genetic contributions vary substantially among these disorders. Huntington's disease (HD) is the clearest example of a monogenic dementia syndrome and results from expansion of a CAG trinucleotide repeat within the huntingtin (HTT) gene. Repeat length correlates inversely with age at onset and influences disease progression, making HD one of the best-characterized neurogenetic disorders (McColgan & Tabrizi, 2018).

Genetic factors also contribute to prion diseases. Approximately 10–15% of Creutzfeldt–Jakob disease (CJD) cases are associated with pathogenic mutations in the prion protein gene (PRNP). In addition, polymorphisms at codon 129 of PRNP influence disease susceptibility and phenotype (Mead, 2022).

In contrast, normal pressure hydrocephalus (NPH), HIV-associated dementia (HAD), and most toxin- or infection-related dementias are not primarily inherited disorders. Nevertheless, genetic variability may influence susceptibility to neurodegeneration, inflammatory responses, and treatment outcomes.

Table 3. Major Genetic Factors in Other Dementias

Disorder	Principal Genetic Factor	Clinical Significance
Huntington's disease	*HTT* CAG repeat expansion	Direct disease cause
Genetic prion diseases	*PRNP* mutations	Familial disease forms
Sporadic CJD	Codon 129 polymorphism	Modifies susceptibility and phenotype
NPH	No established causative gene	Limited genetic contribution
HIV-associated dementia	Host genetic modifiers	Variable susceptibility
Toxic/infectious dementias	No specific causative genes	Environmental predominance

Pathophysiology

Cerebrospinal Fluid Dysfunction in NPH
The pathophysiology of NPH is characterized by impaired CSF circulation and reduced clearance of interstitial metabolites. Progressive ventricular enlargement exerts mechanical stress on periventricular white matter tracts involved in gait, cognition, and bladder control. Emerging evidence also implicates dysfunction of the glymphatic system and age-related vascular changes in disease development (Nakajima et al., 2021).

Protein Misfolding in CJD
CJD represents the prototypical protein misfolding disorder. Conversion of normal cellular prion protein (PrP^C) into a pathological isoform PrP^Sc) initiates a self-propagating cascade of aggregation, neuronal injury, and spongiform degeneration. Unlike conventional infectious agents, prions lack nucleic acids and propagate through conformational templating (Hermann et al., 2021).

Mutant Huntingtin Toxicity in HD
In HD, expanded polyglutamine tracts within mutant huntingtin protein promote intracellular aggregation and disrupt multiple cellular processes, including transcriptional regulation, axonal transport, mitochondrial function, and proteostasis. Progressive degeneration is particularly prominent within the caudate nucleus and putamen, leading to motor, cognitive, and psychiatric manifestations (McColgan & Tabrizi, 2018).

Neuroinflammation in HIV-Associated Dementia
HIV does not directly infect neurons. Instead, infected macrophages and microglia trigger chronic neuroinflammation, cytokine release, excitotoxicity, and synaptic injury. Persistent CNS viral reservoirs may sustain inflammation even in individuals receiving effective antiretroviral therapy (Nightingale et al., 2023).

Toxic and Infectious Injury
Toxin-related dementias arise through diverse mechanisms, including oxidative stress, mitochondrial dysfunction, hypoxic injury, and disruption of neurotransmitter systems. Similarly, infection-related dementias may result from direct microbial invasion, chronic inflammation, vasculitis, hydrocephalus, demyelination, or secondary neuronal loss.

Shared Mechanisms of Cognitive Decline
Despite their distinct etiologies, several biological pathways are common across these disorders:

- Neuroinflammation
- Synaptic dysfunction
- Oxidative stress
- White matter injury
- Network disconnection
- Progressive neuronal loss

These convergent mechanisms help explain why clinically diverse diseases ultimately produce similar cognitive and functional impairments.

Table 4: Comparative Pathophysiological Mechanisms

Disorder	Dominant Mechanism
NPH	CSF circulation and glymphatic dysfunction
CJD	Prion protein misfolding
HD	Mutant huntingtin toxicity
HIV-associated dementia	Chronic neuroinflammation
Toxic dementias	Neurotoxic injury
Infectious dementias	Infection-induced CNS damage

Key Points

Genetic factors are central to Huntington's disease and inherited prion disorders but play a more limited role in NPH, HIV-associated dementia, and most toxin- or infection-related dementias. The underlying pathophysiological mechanisms differ substantially; however, all ultimately lead to neuronal dysfunction and cognitive decline through convergent pathways involving inflammation, cellular injury, and network disruption. Understanding these mechanisms has become increasingly important as biomarker-guided diagnostics and targeted therapies continue to evolve.

Clinical Presentation and Variants

The clinical manifestations of these dementias reflect the underlying anatomical and pathological processes involved. Although cognitive impairment is a shared feature, the pattern of deficits, associated neurological findings, rate of progression, and systemic manifestations often provide important diagnostic clues. Recognition of characteristic clinical syndromes remains essential for early diagnosis and appropriate management.

Clinical Presentation

Normal pressure hydrocephalus (NPH) classically presents with the triad of gait disturbance, cognitive impairment, and urinary dysfunction. Gait impairment is usually the earliest and most prominent feature, often described as a broad-based, slow, "magnetic" gait. Cognitive deficits primarily involve attention, executive function, and psychomotor speed rather than the episodic memory impairment typical of Alzheimer's disease (Nakajima et al., 2021).

Creutzfeldt–Jakob disease (CJD) typically presents as a rapidly progressive dementia developing over weeks to months. Cognitive decline is accompanied by neurological manifestations including myoclonus, cerebellar dysfunction, visual disturbances, pyramidal signs, extrapyramidal symptoms, and eventually akinetic mutism. The unusually rapid progression remains one of the most important diagnostic clues (Hermann et al., 2021).

Huntington's disease (HD) is characterized by a triad of motor, cognitive, and psychiatric symptoms. Chorea is the most recognizable motor feature, although dystonia, bradykinesia, and gait abnormalities may also occur. Cognitive impairment predominantly affects executive function, processing speed, and attention. Depression, irritability, anxiety, apathy, and behavioural dysregulation are common and may precede motor manifestations (McColgan & Tabrizi, 2018).

HIV-associated dementia (HAD) usually presents with a subcortical pattern of impairment characterized by psychomotor slowing, executive dysfunction, impaired attention, and reduced information-processing speed. Behavioural symptoms may include apathy, social withdrawal, and mood disturbance. Motor slowing and gait instability may develop in advanced disease (Nightingale et al., 2023).

Toxin- and infection-related dementias demonstrate considerable clinical variability depending upon the underlying cause. Common manifestations include memory impairment, executive dysfunction, personality change, gait abnormalities, psychiatric symptoms, and fluctuating cognition. Associated systemic features may provide important diagnostic clues.

Major Clinical Features

Table 5. Comparative Clinical Presentation of Other Dementias

Feature	NPH	CJD	HD	HIV Dementia	Toxic/ Infectious
Cognitive Decline	Gradual	Rapid	Progressive	Variable	Variable
Executive Dysfunction	Common	Common	Common	Common	Common
Memory Impairment	Mild–Moderate	Common	Moderate	Mild–Moderate	Variable
Gait Disturbance	Early hallmark	Common	Common	Possible	Variable
Urinary Symptoms	Common	Rare	Rare	Rare	Variable
Myoclonus	Rare	Characteristic	Rare	Rare	Rare
Chorea	Absent	Rare	Characteristic	Absent	Absent
Psychiatric Symptoms	Mild	Possible	Very common	Common	Common
Progression	Months–Years	Weeks–Months	Years–Decades	Variable	Variable

Clinical Variants

Normal Pressure Hydrocephalus
NPH is generally categorized into:
- Idiopathic NPH
- Secondary NPH

Secondary forms occur following conditions such as subarachnoid hemorrhage, meningitis, traumatic brain injury, or neurosurgical procedures. Clinical manifestations are often similar, although secondary NPH may occur at a younger age and have a more clearly identifiable precipitating event.

Creutzfeldt–Jakob Disease
Several clinicopathological variants of CJD have been described. Sporadic CJD is the most common form, whereas genetic, iatrogenic, and variant CJD are less frequent. Certain phenotypic variants may initially present with visual symptoms, cerebellar dysfunction, or psychiatric manifestations, potentially delaying diagnosis.

Huntington's Disease
Adult-onset HD is the most common phenotype. Juvenile Huntington's disease, typically associated with very large CAG repeat expansions, often presents with rigidity, bradykinesia, seizures, and cognitive decline rather than prominent chorea. This variant generally follows a more aggressive clinical course.

HIV-Associated Neurocognitive Disorders
Current classification recognizes a spectrum of disease severity:
- Asymptomatic Neurocognitive Impairment (ANI)
- Mild Neurocognitive Disorder (MND)
- HIV-Associated Dementia (HAD)

This framework reflects the shift from severe dementia toward milder forms of cognitive impairment in the era of effective antiretroviral therapy.

Infection-Related Variants
Clinical phenotypes vary according to the causative pathogen. Neurosyphilis may present with personality change and cognitive decline, tuberculosis may be associated with hydrocephalus and focal deficits, while progressive multifocal leukoencephalopathy often produces cognitive impairment accompanied by focal neurological abnormalities.

Red Flags Suggesting Specific Diagnoses
Certain clinical features strongly favour particular diagnoses:

Table 6. Diagnostic Clinical Red Flags

Clinical Feature	Diagnostic Consideration
Rapid decline over weeks	CJD
Magnetic gait with ventriculomegaly	NPH
Chorea with family history	HD
HIV infection with psychomotor slowing	HAND/HAD
History of toxin exposure	Toxic dementia
Cognitive decline with CNS infection	Infection-related dementia

Clinical Implications
Careful characterization of symptom onset, progression, associated neurological findings, family history, infectious risk factors, and exposure history remains critical in the evaluation of dementia. While substantial overlap exists among syndromes, characteristic clinical patterns frequently provide the first indication of the underlying diagnosis and guide subsequent laboratory, imaging, and genetic investigations.

Diagnosis

Accurate diagnosis of uncommon dementias requires integration of clinical assessment, laboratory testing, neuroimaging, biomarker analysis, and, in selected cases, genetic evaluation. Because several of these disorders are potentially treatable or have important prognostic implications, early identification is essential. The diagnostic approach should focus on distinguishing reversible causes from irreversible neurodegenerative conditions while simultaneously identifying disease-specific features.

Differential Diagnosis
The differential diagnosis varies according to symptom profile and disease tempo. Rapidly progressive cognitive decline raises concern for prion disease, autoimmune encephalitis, central nervous system infection, metabolic disorders, or toxic exposure. In contrast, slowly progressive syndromes are more consistent with NPH, HD, or chronic HIV-associated neurocognitive impairment.

Several clinical features can assist differentiation:
- Early gait dysfunction and urinary symptoms suggest NPH.
- Rapid progression with myoclonus favours CJD.
- Chorea and positive family history strongly suggest HD.
- Psychomotor slowing in an individual with HIV infection supports HAND.
- Exposure history may indicate toxic etiologies.
- Fever, meningismus, or immunosuppression should prompt investigation for infectious causes.

Because symptom overlap is common, clinical evaluation alone is rarely sufficient.

Diagnostic Approach
A structured diagnostic framework is recommended.

Clinical Assessment
The evaluation should include:
- Detailed history of symptom onset and progression
- Functional assessment
- Medication review
- Exposure history
- Family history
- Neurological examination

Special attention should be given to gait abnormalities, movement disorders, psychiatric symptoms, and focal neurological deficits.

Cognitive Assessment
Commonly used instruments include:
- Mini-Mental State Examination (MMSE)
- Montreal Cognitive Assessment (MoCA)
- Addenbrooke's Cognitive Examination III (ACE-III)
- Frontal Assessment Battery (FAB)

The pattern of deficits may provide diagnostic clues. Executive dysfunction is particularly prominent in NPH, HD, and HIV-associated dementia, whereas rapidly progressive global impairment is more characteristic of CJD.

Laboratory Evaluation
Routine laboratory studies remain essential to exclude reversible causes of cognitive decline.

Recommended investigations include:
- Complete blood count
- Electrolytes
- Renal and liver function tests
- Thyroid function tests
- Vitamin B12 and folate levels
- Inflammatory markers
- Syphilis serology
- HIV testing when indicated

Additional investigations should be guided by clinical suspicion.

Disease-Specific Testing
For suspected infectious etiologies:
- Cerebrospinal fluid (CSF) analysis
- Microbiological studies
- Molecular pathogen detection

For suspected toxic disorders:
- Heavy metal screening
- Toxicology testing
- Occupational exposure assessment

For suspected HD:
- HTT genetic testing

For suspected prion disease:
- RT-QuIC testing
- CSF tau
- CSF 14-3-3 protein

(Hermann et al., 2021).

Neuroimaging
Neuroimaging is a central component of diagnosis and frequently provides disease-specific findings.

Magnetic Resonance Imaging
MRI is generally the preferred imaging modality because of its superior sensitivity and anatomical detail.

Characteristic findings include:

Normal Pressure Hydrocephalus
- Ventriculomegaly
- Disproportionately enlarged subarachnoid-space hydrocephalus (DESH)
- Narrow high-convexity sulci
- Reduced callosal angle

(Nakajima et al., 2021).

Creutzfeldt–Jakob Disease
- Cortical ribboning on diffusion-weighted imaging
- Basal ganglia hyperintensity
- Thalamic involvement in selected variants

Huntington's Disease
- Caudate nucleus atrophy
- Striatal volume loss
- Progressive cortical atrophy in advanced disease

HIV-Associated Dementia
- White matter abnormalities
- Cerebral atrophy
- Subcortical changes

Infection-Related Disorders
Findings vary according to pathogen but may include:
- Meningeal enhancement
- Hydrocephalus
- Focal lesions
- Demyelination

Electroencephalography
EEG has limited utility in most dementias but remains valuable in suspected CJD.

Characteristic findings include:
- Periodic sharp-wave complexes
- Diffuse slowing

Although not specific, these findings can support the diagnosis when interpreted alongside clinical and biomarker data.

Biomarkers
Biomarkers have transformed the diagnosis of several neurological disorders and are increasingly incorporated into routine clinical practice.

Cerebrospinal Fluid Biomarkers
Prion Disease
Important markers include:
- RT-QuIC
- Total tau
- 14-3-3 protein

RT-QuIC currently represents the most specific biomarker for CJD and has significantly improved diagnostic accuracy (Hermann et al., 2021).

HIV-Associated Dementia
Potential biomarkers include:
- HIV RNA levels
- Inflammatory mediators
- Neurofilament light chain (NfL)

Although primarily research tools, these markers may assist disease monitoring.

Blood-Based Biomarkers
Growing interest exists in blood biomarkers because they are less invasive and more practical for longitudinal monitoring.

Among the most promising is neurofilament light chain (NfL), which reflects axonal injury and may correlate with disease activity across multiple neurological disorders (Bridel et al., 2019).

Comparative Diagnostic Features

Table 7. Key Diagnostic Findings in Other Dementias

Disorder	Characteristic Diagnostic Features
NPH	Ventriculomegaly, DESH pattern, positive CSF tap test
CJD	Rapid progression, RT-QuIC positivity, cortical ribboning
HD	HTT mutation, caudate atrophy, chorea
HIV Dementia	HIV infection, executive dysfunction, white matter changes
Toxic Dementias	Exposure history, toxicological evidence
Infection-Related Dementias	Microbiological confirmation, inflammatory findings

Diagnostic Strategy
A practical approach begins with clinical assessment and cognitive testing, followed by targeted laboratory investigations and MRI. Genetic testing is reserved for suspected hereditary disorders, while advanced biomarker studies are most useful in prion disease and selected research settings.

Importantly, clinicians should actively search for potentially reversible causes, particularly NPH, HIV-associated cognitive disorders, toxin exposure, and treatable CNS infections. Early diagnosis not only improves management options but may significantly alter long-term outcomes and quality of life.

Management, Prognosis, and Future Directions

Management strategies for other dementias vary considerably because the underlying mechanisms range from potentially reversible disturbances of cerebrospinal fluid dynamics to irreversible neurodegenerative and prion disorders. Nevertheless, several common principles apply across conditions, including early diagnosis, multidisciplinary care, symptom management, rehabilitation, caregiver support, and optimization of quality of life.

Management

Normal Pressure Hydrocephalus

Normal pressure hydrocephalus represents one of the few potentially reversible causes of dementia. The primary treatment is cerebrospinal fluid diversion, most commonly through ventriculoperitoneal shunt placement (Nakajima et al., 2021).

Appropriate patient selection is critical and is generally based on:

- Compatible clinical presentation
- Characteristic neuroimaging findings
- Positive response to CSF drainage testing

Gait dysfunction demonstrates the greatest likelihood of improvement following surgery, whereas cognitive and urinary symptoms often show more variable responses. Postoperative rehabilitation may further enhance functional outcomes.

Creutzfeldt–Jakob Disease

No curative therapy currently exists for CJD. Management is therefore primarily supportive and focuses on symptom control, patient comfort, and palliative care (Mead, 2022).

Common interventions include:

- Treatment of myoclonus with clonazepam or valproate
- Nutritional support
- Prevention of complications related to immobility
- Management of behavioural symptoms
- Advance care planning

Because disease progression is usually rapid, early discussions regarding goals of care are particularly important.

Huntington's Disease

Management of HD requires a multidisciplinary approach involving neurology, psychiatry, rehabilitation medicine, nutrition, and social services.

Pharmacological therapy focuses on symptom control:

Motor Symptoms

- Tetrabenazine
- Deutetrabenazine
- Selected antipsychotic agents

Psychiatric Symptoms

- Antidepressants
- Antipsychotics
- Mood stabilizers

Rehabilitation interventions including physical, occupational, and speech therapy play an important role throughout the disease course (Saft et al., 2023).

HIV-Associated Dementia

The cornerstone of treatment is effective antiretroviral therapy (ART). Viral suppression has dramatically reduced the incidence of severe HIV-associated dementia and remains the most effective intervention for preserving cognitive function (Nightingale et al., 2023).

Additional management strategies include:

- Optimization of ART adherence
- Treatment of psychiatric comorbidities
- Cognitive rehabilitation
- Management of cardiovascular risk factors

Toxin- and Infection-Related Dementias
Treatment depends upon the underlying etiology.

Toxin-Related Disorders
Management may include:
- Removal of toxic exposure
- Alcohol abstinence
- Nutritional supplementation
- Chelation therapy when indicated
- Treatment of complications

Infection-Related Disorders
Therapy is directed toward the causative pathogen and may involve:
- Antibiotics
- Antifungal agents
- Antiviral therapies
- Management of hydrocephalus and other neurological complications

Because many infectious and toxic causes are potentially reversible, early intervention is crucial.

Comparative Treatment Overview

Table 8. Principal Management Strategies

Disorder	Primary Treatment
NPH	Ventriculoperitoneal shunting
CJD	Supportive and palliative care
HD	Symptomatic pharmacotherapy and rehabilitation
HIV Dementia	Antiretroviral therapy
Toxic Dementias	Exposure removal and supportive treatment
Infection-Related Dementias	Pathogen-specific antimicrobial therapy

Prognosis
Prognosis varies substantially among these disorders and depends on disease mechanism, stage at diagnosis, treatment availability, and patient-specific factors.

Normal Pressure Hydrocephalus
Among the conditions reviewed, NPH offers one of the most favourable prognostic profiles because meaningful improvement may occur following shunt surgery. Early diagnosis, shorter symptom duration, and positive CSF drainage testing are associated with better outcomes (Nakajima et al., 2021).

Creutzfeldt–Jakob Disease
CJD carries the poorest prognosis. Median survival is generally measured in months, and most patients experience relentless neurological deterioration despite supportive care (Hermann et al., 2021).

Huntington's Disease
HD follows a chronic progressive course. Survival typically ranges from 15 to 25 years following symptom onset, although considerable variability exists. Disease progression is influenced by CAG repeat length, age at onset, and comorbidity burden (McColgan & Tabrizi, 2018).

HIV-Associated Dementia
The prognosis of HIV-associated dementia has improved dramatically in the era of combination ART. Many patients achieve stabilization or partial improvement, although residual cognitive deficits frequently persist (Nightingale et al., 2023).

Toxic and Infectious Dementias
Outcomes depend largely on the speed of diagnosis and treatment. Early intervention may result in substantial recovery, whereas delayed recognition can lead to permanent neurological injury.

Table 9. Comparative Prognosis

Disorder	Typical Clinical Course	Potential Reversibility
NPH	Often improved with treatment	Moderate–High
CJD	Rapidly progressive and fatal	None
HD	Slowly progressive	None
HIV Dementia	Variable, improved with ART	Partial
Toxic Dementias	Variable	Variable
Infection-Related Dementias	Variable	Often significant

Future directions

Recent advances in neuroscience, genetics, biomarker development, and computational medicine are reshaping the diagnosis and management of dementia.

Biomarker Development
Blood-based biomarkers such as neurofilament light chain (NfL) and glial fibrillary acidic protein (GFAP) may facilitate earlier diagnosis, disease monitoring, and prognostic assessment (Hansson, 2024).

Precision Medicine
Precision medicine approaches aim to integrate:
- Genetic information
- Biomarker profiles
- Neuroimaging findings
- Clinical phenotypes

to guide individualized treatment strategies.

Gene-Targeted Therapies
HD has emerged as a leading target for gene-based interventions, including:
- Antisense oligonucleotides
- RNA-targeted therapies
- Gene-silencing approaches

Although still investigational, these strategies represent one of the most promising developments in neurodegenerative disease research (Tabrizi et al., 2024).

Artificial Intelligence
Artificial intelligence and machine-learning technologies may improve:
- Neuroimaging interpretation
- Diagnostic accuracy
- Prognostic prediction
- Clinical decision support

These tools are increasingly being incorporated into dementia research and clinical practice.

Prevention
Future public health strategies should emphasize:
- HIV prevention and treatment
- Occupational safety
- Environmental toxin reduction
- Infection control
- Early identification of treatable neurological disorders

Prevention remains particularly important because several causes of cognitive decline discussed in this review are potentially avoidable or reversible.

Conclusion

Other dementias comprise a diverse group of neurological disorders that differ markedly in etiology, pathophysiology, clinical presentation, treatment options, and prognosis. Normal pressure hydrocephalus, Creutzfeldt–Jakob disease, Huntington's disease, HIV-associated dementia, and toxin- and infection-related dementias illustrate the broad range of mechanisms capable of producing cognitive impairment.

Recognition of these disorders is clinically important because several are potentially treatable and require disease-specific management. Advances in neuroimaging, molecular diagnostics, biomarkers, genetics, and precision medicine are improving diagnostic accuracy and expanding therapeutic possibilities. Continued research into disease mechanisms and targeted interventions offers the prospect of earlier diagnosis, improved outcomes, and more individualized care for patients affected by these challenging conditions.

Table 10. Key Clinical Messages

Key Message	Clinical Importance
NPH remains one of the most treatable causes of dementia	Early recognition can improve outcomes
CJD should be considered in rapidly progressive dementia	Prompt diagnosis facilitates appropriate management
HD requires genetic confirmation and multidisciplinary care	Essential for patient and family counseling
HIV-associated cognitive disorders remain clinically relevant	ART optimization improves outcomes
Toxic and infectious causes must always be excluded	Many are reversible if detected early
Biomarkers and precision medicine are transforming dementia care	Likely to influence future clinical practice

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AI, IP, Copyright, and human knowledge and traditions

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Abstract

While AI at this stage of development mainly encompasses a world of promises it has the potential for preparing a new world of opportunity and mutual advancement. Many 'AI' offerings at the moment however can be defined as hyped up search engines, useful software tools, and even malicious programs that facilitate, for example, child abuse, cybercrime, mental health disorders in children and which could and do steal personal identity, IP and Copyrighted material.

While several of the main players started out altruistically the main entities are competing commercially for users and profit. Several are also demanding access to our personal communications and our data storage.

Others are deliberately exploiting human endeavour and putting our (human) systems and personal data at great risk.

We are also told AI might steal jobs. We need to decide if and where AI is indeed preferable to human contact and do we have a moral obligation to keep humans employed in a world where one must earn one's keep to survive. Humans also need stimulating work for good mental health.

One of the arguments against mental health disorders in children brought about by insidious social media is the sidelining of human childhood traditions of play and socialisation with friends. Social media prefers to keep children in plain sight and keep feeding them with warped messaging. Social media is not good for child mental health and the acquiring of people skills that interaction with other children facilitates. Most countries have now brought in legislation to protect children. Are we applying the same cautions to AI?

While AI can provide exceptional benefits to human life it can be a hugely destructive force if not regulated and applied morally and carefully.

Some AI is also claiming ownership of all knowledge built to date; knowledge which has been the result of millennia of human endeavour. Add to that, currently the world's knowledge is housed and judiciously taught by our respected global academic institutions where it is subject to copyright and is acknowledged intellectual property. Are the commercial AI purveyors safeguarding our human tradition or using it for commercial advantage?

This paper looks at these issues and at what safeguards are being applied to AI development and use globally to protect Intellectual Property and Copyrighted material.

Keywords: artificial intelligence, intellectual property, copyright, human knowledge, human tradition.

The World View

AI policies are currently being devised globally in reaction to this enormous change in policy, legality and particularly to protect human endeavour and knowledge and prevent it being 'owned ' by those who did not author it and who are quickly building their AI storage units and disguising it as new work. It is not new work. It is human knowledge, repackaged. As such its human authorship needs to be acknowledged legally and where applicable, financially. Most countries are doing this.

The policies of most countries currently protect Intellectual Property, Copyright and human rights. Most countries of the world are designing new AI policies along the lines of only human knowledge and endeavour can be copyright. In the Middle East for example, IP and copyright laws typically require a natural human author, meaning fully autonomous AI-generated content falls into a legal grey zone. However, AI-assisted works can be protected if they reflect sufficient human contribution, and authorities are actively enforcing unauthorized AI use.

Middle East
Navigating AI and intellectual property in the Middle East requires attention to distinct regional developments:

Human Authorship Requirement: Copyright laws in key jurisdictions like the UAE and Saudi Arabia only recognize natural or legal persons as authors. Outputs generated purely by AI without meaningful human intervention are often considered to be in the public domain.
AI-Assisted Protections: AI works lack copyright protection, works that feature significant human input, such as iterative prompts, detailed editing, and modifications can be copyrighted.
Copyright Infringement: Regulators are actively policing unauthorized AI usage.
Patent and Trademark Rules: Patent systems in countries like the UAE require a human inventor. AI can be used to ideate and create new trademarks, but the resulting marks must still satisfy traditional IP and brand registration requirements.
Data Privacy and Scraping: Businesses must navigate regional data privacy laws. In both the UAE and Saudi Arabia, strict Personal Data Protection Laws (PDPL) apply to data scraping, making the sourcing of AI training datasets a major compliance focus.

Australia
In Australia Copyright and AI interact in three ways:
1. No AI training exception
The Australian Government has rejected a 'text and data mining exception for AI'
2. No Copyright for AI Output
Copyright only protects original works with human intellectual effort
3. Active policy updates
4. No copyright for AI generated content.
- The law requires that an original work must have a human author. AI generated material 'does not belong to anyone'
- If uploading copyrighted material to AI the copyright remains with the original author or creator.
- If a human uses AI to assist in developing material the resulting work may be eligible for copyright protection.

United States
US copyright law includes a broad fair use doctrine, meaning a greater variety of reproductions can be classified as non-infringing. Despite a broader definition of fair use, both jurisdictions assess fairness in relation to similar factors, including the purpose and character of the use, the nature of the work, the amount used and the market impact of the use.

The US Copyright Office Report considered the key question of whether training or use of generative AI models involves reproduction of copyright works so as to potentially give rise to a cause of action for copyright infringement. It identifies several ways reproduction of copyright works may occur, including:
• through the downloading, copying, and modification of copyright works for training datasets; and
• through the process of an AI system memorising copyrighted works and making temporary copies during model optimisation; and
• when an AI system produces "material that replicates or closely resembles copyrighted works".

European Union
The EU has adopted a comprehensive and binding framework for regulating the intersection of AI and copyright. This dual regime includes:

The AI Act, which imposes obligations on developers of general-purpose and high-risk AI systems to document training data, label AI-generated content, and implement safeguards against the use of copyrighted material in certain circumstances. These obligations are enforceable by EU national regulators with penalties of up to 7% of global turnover for serious non-compliance.

Developers training AI on EU-based content must verify whether a valid opt-out applies and secure licences where required. Failure to do so may result in copyright infringement.

China
In China, the legal framework surrounding AI-generated content is also developing. A ruling by the Beijing Internet Court in November 2023 recognized copyright protection for an AI-generated image, provided that the work demonstrates originality and reflects a human's intellectual effort. This decision signifies a progressive stance toward acknowledging AI-assisted creations under Chinese copyright law. Furthermore, China's regulatory approach mandates that AI-generated content must be clearly labeled, and AI companies are held liable for any misinformation or unlawful content produced by their models.

AI and Academia

Most Universities have their own AI policies as do Academic journals. That said, they usually follow already accepted conventions.

National schools and universities already house the world's knowledge which is made available to students. Along with knowledge acquisition, comes reasoning and invention and research. It has got us to where we are now. This knowledge and reasoning built AI systems in the first place.

Knowledge belongs to all humanity and all humanity authored it. If AI is to be used for profit then proceeds should go to everyone on the planet. The 'AI providers' are just providing the vehicle to distribute the knowledge, they don't own the knowledge. They only own the interface.

It is up to students and staff to decide if AI is doing the job it claims to do.

Conclusion

We have seen inventions and systems born and developed since humans first walked the earth approximately 300,000 years ago. Making tools is what we are good at. Inventions and knowledge have improved our collective lives and were born of our collective minds and endeavour.

We need to ensure that the age of 'artificial intelligence' is not monopolised by those who have just 'added the last knob to our collective knowledge machine' and that it serves the true purpose of humankind where the dream and endeavour for most of us has always been a world of sharing and mutual growth.

We must also be vigilant and curb the commercial aspirations of 'AI suppliers' to ensure they do not charge us for what we already own.

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