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Kent Journal of Psychiatry

Dedicated to Psychiatry trainees and trainers

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Agnieszka Klimowicz

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Kent Journal of Psychiatry is a psychiatric journal with emphasis on the publication of research by psychiatry trainees and their mentors. This traditional source of new medical knowledge has been under pressure due to the logarithmic growth of journal submissions; this journal attempts to redress this balance.

The editors will offer support to young authors in the presentation of their findings. This is followed by rigorous peer reviews. The procedure will enable the journal to fulfil a valuable educational and developmental role in addition to presenting high-quality research to a wider readership.

Kent Journal of Psychiatry is a free online publication which appears on biannual basis. It offers the best of new research from innovative young medical thinkers on the front line of mental health care.

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Editorial

Welcome to the second edition of KJPsych in 2025**.** This issue brings together a dynamic range of perspectives from psychiatry trainees and their trainers here and abroad, reflecting the profession's evolving challenges and ambitions. From cutting-edge research to candid reflections on training and practice, these contributions highlight both the vitality and the vulnerabilities of modern psychiatry.

A palpable unease lingers within Psychiatry; sparked not only by the recent Ten-Year Health Plan but also by deeper questions about its direction. While our leadership has often prioritised pragmatism and political sensitivities, many now argue that this approach has diluted the profession's intellectual rigor and innovative spirit. The result is a growing tension between maintaining stability and reclaiming the bold, visionary thinking that once positioned psychiatry at the forefront of medicine.

Today, we stand at a crossroads. Once pioneers in mental health, we risk fading into passivity; overshadowed by algorithmic medicine yet uncertain of our unique value. The rise of AI looms not as a distant threat but as a mirror, exposing our unresolved identity crisis. To thrive, psychiatry must reinvest in principled leadership, redefine its core mission, and reignite the curiosity that drives progress. The future is unwritten, but it begins here - with critical dialogue, dissent, and the courage to demand more.

Beyond the Episode: Why Treating Mental Illness Like a Soap Opera Won't Cut It

Agnieszka Klimowicz

Welcome, future Consultant Psychiatrists!

Get ready to embark on a journey deeper and richer than any medical drama you've binge-watched. Psychiatry isn't just about crisis management; it's about understanding the entire narrative arc of a person's mental health story. If you've ever thought, "I'll just treat this episode and they'll be fine", you're in good company. It's a common early-career instinct. After all, psychiatric presentations can feel like dramatic soap opera plotlines - manic highs, depressive crashes, psychotic detours, and anxiety spinning through every subplot. It's intense, gripping, and sometimes overwhelming.

But here's the twist: real-life mental illness rarely wraps up neatly between ad breaks. Most psychiatric conditions don't vanish after one heroic intervention. Instead, they evolve, sometimes subtly, sometimes dramatically across years. Think less season finale resolution and more long-running character development, complete with surprise reappearances, comorbid cameos, and tangled emotional backstories. And this doesn't just apply to severe mental illnesses like schizophrenia or bipolar disorder - chronic anxiety, obsessive-compulsive disorder, and even so-called "milder" conditions often follow unpredictable and looping trajectories.

That's why your role is about much more than acute care. As future consultants, you're not just episode-fixers; you're long-haul co-authors in someone's story. You help shape care plans that anticipate recurrences, build resilience, and support people through the highs, lows, and everything in between. So, gear up - not just with knowledge, but with curiosity, compassion, and a healthy respect for the complexity of the human mind. Psychiatry is less about tidy solutions and more about meaningful continuity. And trust me, the plot gets better the deeper you go.

The "Episodic Cure" Fantasy

Let's be honest: treating a single episode and assuming that the whole illness is handled? That's a charming fantasy; one that even seasoned psychiatrists sometimes secretly cling to. We all love closure. The DSM serves us clean-cut episodes like a Netflix series. Clinical trials obsess over symptom remission like it's the ultimate endgame. But here's the kicker - real life doesn't follow clinical protocols. It improvises, loops, and laughs at our tidy timelines and bullet points.

Conditions like schizophrenia, bipolar disorder, and depressive disorder don't just throw one dramatic episode and then fade into the credits. They're more like cunning houseguests - showing up uninvited, causing chaos, then lurking silently, moving your keys, draining your energy, and subtly reshaping how the person thinks, feels, and functions. As Insel [1] noted, the symptoms we can see often vanish long before the real disruption ends. The functional impacts on motivation, memory, and relationships can linger like background static.

Therefore, our job isn't just episode intervention; it's narrative insight. We're not just crisis responders; we're long-term collaborators, weaving recovery into a lifelong arc. Think of each episode as just one plot point in a longer saga. Our patients aren't just fighting flare-ups – they are often navigating a baseline that's already tough, even on a "good" day. And if we overlook the quiet aftermath, we miss the chance to build real and lasting support.

So, let's reframe our mindset. Psychiatry isn't about slaying dragons in single battles. It's

about staying for the whole story, from the noisy chaos to the eerie silence afterward. The work is messier than the textbooks, more layered than the diagnostic codes, and far more human than any randomised control trial can capture. But that's what makes it so important, and honestly, a kind of epic.

Case in Point: Meet Mr. Bipolar

Imagine Mr. X: he bursts onto the ward in a blaze of mania - decked out in neon clothes, overflowing with ideas, launching three businesses by breakfast and redesigning the hospital's workflow by lunch. Classic textbook mania, right? A few weeks of lithium, some stabilising therapy, and voilà - Mr. X is calm, cooperative, maybe even a little dull. Discharge papers are signed, as everyone's feeling victorious. But fast-forward five years: those wild highs have faded into something much greyer. Now, Mr. X is mostly dragging through depressive episodes. He is constantly exhausted, barely holding onto his job, and the only thing that moves fast is the fog clouding his brain. If we only focused on treating the firework display of mania, we'd have missed the slow burn; the chronic, smouldering dysfunction that follows long after the spotlight fades [2].

And here is where it gets even trickier. A few colleagues might start whispering, "ADHD?" After all, Mr. X has always been a bit scattered, impulsive, hyper-focused one minute and lost the next. But now, the disorganisation is worse, and his anxiety has hit new heights. Maybe he does have ADHD; or maybe those symptoms were actually early warning flares. See, ADHD and anxiety aren't just standalone labels - they often show up years before bipolar disorder fully unfolds. It's a bit like mistaking the trailer for the full movie. It is easy to retro-fit a neat diagnosis when we are looking for immediate explanations, but psychiatry rarely hands us clean timelines. The truth is, Mr. X's past may hold subtle clues that only make sense in hindsight - clues we often overlook unless we zoom out and take a long-term, developmental view. You didn't see that plot twist coming, did you?

Check this out:

BJPsych; 2018 Sep;213(3):555-560. doi: 10.1192/bjp.2018.111. Epub 21 Jun 2018.

Attention-deficit hyperactivity disorder and anxiety disorders as precursors of bipolar disorder onset in adulthood

Sandra M Meier , Barbara Pavlova , Søren Dalsgaard , Merete Nordentoft , Ole Mors , Preben B Mortensen , Rudolf Uher

It's a Marathon, Not a Sprint

Psychiatry isn't 999 - and yet, NHS mental health services often behave like it is. The model leans heavily on crisis containment: stabilise the risk, discharge the patient, move on. But our real job? It's not just about taming the chaos of an acute episode; it is about shaping the ongoing narrative of a person's mental health. We are here to support functional recovery, manage persistent symptoms, and help people reimagine who they can be beyond their diagnosis [3]. That takes time. That takes trust. That takes presence. And, let's be real, the NHS rarely allows for any of that. Anyone here interested in shaping the future of policy? Please stand up. We need you more than ever.

This chronic care model demands something bigger from us: the ability to embrace complexity, to tolerate ambiguity, and to resist the dopamine hit of a quick clinical "win". It is humbling, for sure, but it's also deeply rewarding. Think of it less like firefighting and more like gardening. You don't just hack away at visible weeds (AKA acute symptoms); you nurture the soil. You water regularly. You plant, prune, and hope the weather doesn't betray you. With patience, you begin to see real, meaningful growth. But how do you do that in a system where continuity is an endangered species? Where psychiatrists rotate like tourists on a schedule, and no one is left to witness the long-term fallout of well-intentioned but misguided decisions.

Let's talk about that fallout. One of the most glaring examples? The casual over-

prescription of antidepressants and ADHD stimulants in people who are clearly on the bipolar spectrum. Sure, in the short term, patients report feeling "better", as they are more energised and focused. Great, right? But what happens six months later? Cue the manic relapse, the hospital admission, and the emotional wreckage. And yet somehow, these dots are rarely connected. The system, so obsessed with short-term outcomes, fails to ask what the long-term costs might be. It is like applauding the fact that a fire was extinguished without noticing that the basement is still smouldering.

So yes, this work is messy. It is nonlinear. It demands long-term thinking in a short-term system. But here's the truth: you're not just doctors. You are story editors, systems thinkers, and cultural reformers. You are the future of a field that desperately needs to stop acting like a reactive emergency service and start becoming what it was always meant to be: a healing profession built on continuity, curiosity, and care. If you can hold on to that tension, and advocate fiercely for the structures that support it - you'll do more than treat the illness. You will change lives. And maybe, just maybe, change the system too.

So, What's the Take-Home for Trainees?

Don't be episode-obsessed

Yes, taming the wild peaks and dangerous dips is crucial, but it is just the opening act. Psychiatric care isn't about putting out fires and moving on. It's about recognising that those fires often leave smouldering embers - cognitive fog, social withdrawal, emotional numbness. These can quietly erode a patient's quality of life long after discharge. Symptom control is vital, but it is only chapter one.

Think long term - like, really long term

Imagine care not just over months, but over decades. Build treatment plans with that horizon in mind. We are talking relapse prevention, rehabilitation input, family support, and structured routines. And please, for the love of mood stability, don't reach for antidepressants every time someone with bipolar disorder or schizophrenia hits a rough patch. A "boost" in energy can quickly become a full-blown manic storm, and you will be left wondering why your seemingly helpful move backfired spectacularly. Think twice. Consult a colleague. Sleep on it. That pill isn't going anywhere.

Redefine recovery

It is not about getting rid of every symptom. It's about helping people rebuild a life worth living. For some, that means returning to work or school; for others, it might be cooking a meal, reconnecting with family, or going a week without being overwhelmed by dread. If your patient says they're "okay", dig deeper. Are they functioning? Are they engaged with life? Are they hopeful? Recovery is a mosaic; not a checkbox.

Stay curious and cautious

First presentations are especially tricky. A bit of anxiety? Could be. Or it could be the slow-build prodrome of a psychotic disorder. Don't be lulled into a false sense of security because the person isn't floridly unwell. Some of the most serious conditions take months, not days, to reveal their full shape. Early misdiagnosis (or overdiagnosis) can derail a person's care path for years. So, approach those first few months like you are watching the pilot of a very complex TV series: everyone seems normal... until they absolutely aren't.

Final Word: Stay Woke, Stay Weird

Remember: mental illnesses aren't soap operas you binge one weekend and forget by Monday. They are sweeping, nonlinear epics - think Game of Thrones meets Inception with a bit of Fleabag energy thrown in. There are spin-offs, prequels, side quests, and plot twists that can leave even the best clinicians scrambling for the script. You are not just a junior doctor; you're the showrunner. Your job is to keep the story moving toward meaning, connection, and growth.

And here's the uncomfortable truth. NHS mental health services are often built on the wrong

assumptions - crisis-focused, short-term, and paper-driven. And yes, you are being trained in that system, which means you are at risk of adopting a skewed, impoverished view of what mental illness actually is. If all you do is read RCTs and memorise diagnostic criteria, you are missing the real story. Spend time with patients. Learn from their arcs. Think outside the DSM or ICD-shaped box. Challenge what you are told; even by people like me.

Because psychiatry isn't just a job. It is a long game. And you? You are the one who decides how that game is played.

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Development and Implementation of a Comprehensive Induction Pack for Healthcare Professionals in an Integrated Recovery Hub

Amirhosein Banki and Hamid Rahmanian

Abstract

It is always challenging for newly joined healthcare professionals (HCPs) to understand their roles and the roles of other colleagues in an Integrated Recovery Hub (IRH). To address this issue, we prepared a detailed induction pack, with the aim of providing clear information about different roles in the IRH and other teams working collaboratively in the community-based mental health service.

We designed a survey and asked our doctors to share their experiences and expectations to enable us to identify their needs. Based on their feedback, we designed an induction pack with three main chapters: an introduction to the roles in the IRH, descriptions of other teams and how to refer a patient to them, and details about regular meetings.

After developing and implementing the induction pack, a follow-up survey was done, which confirmed that the doctors felt more familiar with different roles in the IRH, worked better with different teams, and felt more confident in managing patients' referrals.

The results show that a comprehensive and well-structured induction process can improve team integration and patient care.

Introduction

The Integrated Recovery Hub (IRH) is a specialised service within the community mental health framework that provides comprehensive, coordinated care for individuals with severe and complex mental health conditions. By integrating a multidisciplinary team of mental health professionals, including psychiatrists, psychologists, community psychiatric nurses, social workers, and occupational therapists. The IRH adopts a holistic, patient-centred approach to treatment and recovery. This model aligns with evidence showing that collaborative care improves outcomes for individuals with complex mental health needs [1].

New healthcare professionals (HCPs) joining the IRH often face challenges in understanding the distinct roles and responsibilities within the team and across collaborating services. Without clear role definitions, professionals may experience confusion, miscommunication, and inefficiencies, which can negatively impact patient care. Research indicates that role ambiguity in mental health teams contributes to fragmented care, delayed interventions and reduced job satisfaction among staff [2]. To address this, we developed a structured induction pack that clarifies the functions of different IRH team members, explains how various services work together, and outlines key communication and referral processes.

The induction pack offers multiple benefits for both staff and patients. First, it enhances HCPs' knowledge and confidence by providing clear expectations about their roles and how they fit within the broader team, reducing uncertainty and improving efficiency. Second, it improves job satisfaction and retention, as a well-structured onboarding process helps new staff feel supported and integrated, decreasing anxiety and increasing engagement [3]. Finally, it leads to higher-quality patient care by promoting better coordination, reducing miscommunication, and ensuring timely, person-centred interventions [4]. By fostering role clarity and collaboration, the induction pack strengthens team dynamics, supports professional

development, and ultimately enhances recovery outcomes for individuals accessing mental health services.

Method

We designed a survey to gather medical doctors' perspectives on the induction process they experienced at the beginning of their roles and to identify additional needs for the induction process. The survey comprised questions about their grades, tenure within the team, familiarity with the roles and services available in the IRH, prior induction experiences, and the benefits of the induction. Additionally, doctors were asked to share their ideas for evolving the induction process to meet their needs.

There were 7 doctors in the IRH, including one higher trainee, one core trainee, two non-trainees, and two foundation-level doctors. All of them participated and completed the survey. The questions and responses are provided in the following table.

Table 1: Initial survey questions and responses

Questions	Responses
Participant's job title	14.3% higher trainee doctors 14.3% core trainee doctors 42.9% non-trainee doctors 28.6% foundation year doctors
Time working within the team	57.1% more than 6 months 42.9% less than 6 months
Familiarity with the roles and services available in IRH	85.7% somewhat familiar 14.3% completely familiar
Completing an induction process before starting the current role	71.4% yes 28.6% no
Efficacy of those induction process in providing sufficient information about the roles and services available in the IRH	28.6% not beneficial. 28.6% very beneficial. 42.9% somewhat beneficial
Need for a comprehensive induction pack	85.7% yes 14.3% no

After completing the survey and identifying the needs, we designed a comprehensive induction pack comprising three main chapters. The first chapter introduces the different roles within IRH [5,6], providing practical explanations for each role and its responsibilities. We also included role holders' names and pictures with their permission to make knowing each other easier and prevent confusion in the office. The second chapter offers a concise description of other teams providing community-based mental health care alongside the IRH [5,6], highlighting the referral process. The third chapter includes additional information about routine meetings held in the IRH throughout the week. This induction pack is designed as an electronic document, easily accessible on phones or laptops.

We provided our medical doctors with the induction pack and asked them to complete the second survey to clarify its efficacy and comprehensiveness. The questions and responses are illustrated in the following diagrams.

Figure 1: What is your role in the IRH?

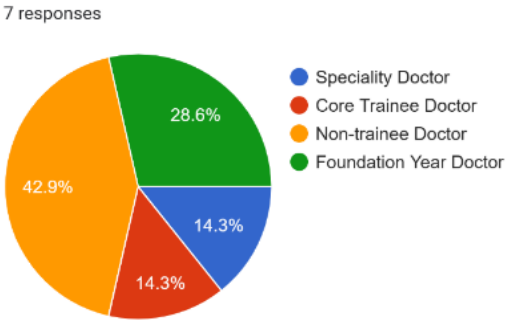


Figure 2: How useful did you find the induction pack overall?

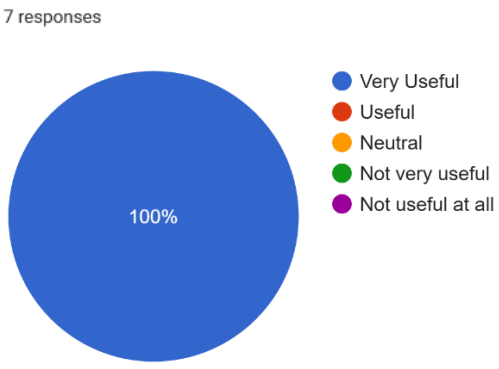


Figure 3: How clear and easy to understand was the information in the induction pack?

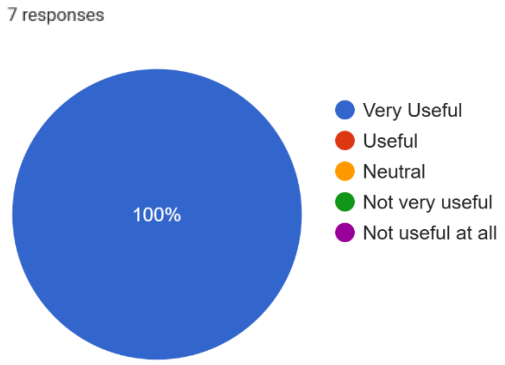


Figure 4: Did the induction pack provide sufficient information about each role within the community mental health team?

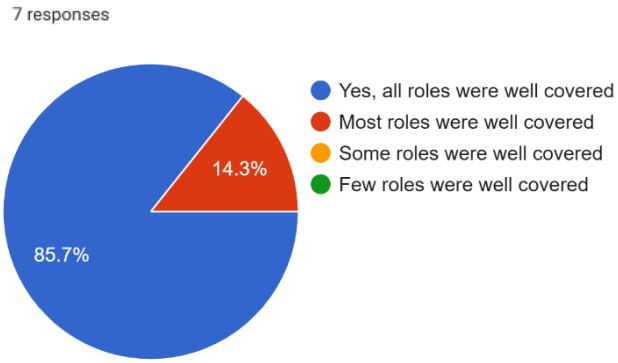


Figure 5: Did the induction pack provide sufficient information about each role within the community mental health team?

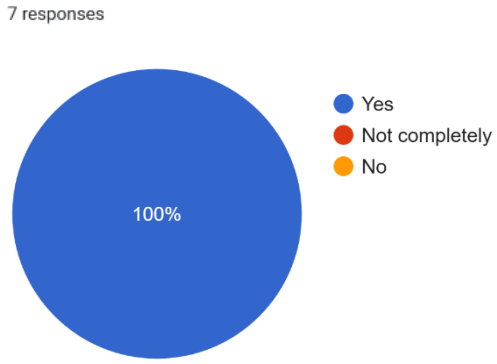
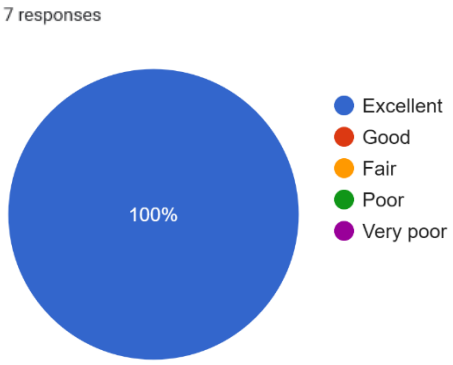


Figure 6: How would you rate the format and presentation of the induction pack?



Results

The survey captured a comprehensive cross-section of IRH doctors, representing all career stages: foundation year (28.6%), core trainees (14.3%), higher trainees (14.3%), and non-trainees (42.9%). This diversity ensured balanced perspectives from clinicians at different professional development phases. Experience levels varied significantly - 57.1% had worked in the IRH for over six months while 42.9% were relatively new (under six months), providing valuable insights from both established and incoming team members.

Induction attendance patterns revealed systemic gaps: 71.4% had received some form of onboarding, but 28.6% commenced duties without any formal orientation. This inconsistency in initiation processes created uneven starting points for new joiners. When assessing role familiarity, only 14.3% reported a complete understanding of IRH functions, with the majority (85.7%) having partial knowledge - a concerning gap for effective team functioning.

Satisfaction ratings with traditional inductions were mediocre at best: 28.6% found them "very beneficial," 42.9% "somewhat beneficial," and 28.6% "not beneficial." These lukewarm responses clearly signalled the need for reform. Notably, 85.7% of doctors actively requested a comprehensive induction resource, validating the development of the new pack.

The revamped induction pack achieved universal approval (100% satisfaction), demonstrating

its effectiveness in bridging knowledge gaps. Every respondent confirmed it successfully clarified roles, team structures, and referral pathways - critical elements for seamless service delivery. Doctors particularly praised its user-friendly design, noting how the thoughtful presentation enhanced information accessibility and retention.

Discussion

The initial induction process, attended by 71.4% of doctors, failed to adequately prepare them for the complexities of roles and services within the IRH. While 14.3% reported full familiarity with these aspects, the majority continued to experience confusion; a gap that directly impaired clinical performance, delayed treatment plans, and compromised care quality. These findings revealed critical deficiencies in onboarding, necessitating immediate intervention.

The redesigned induction pack, developed from feedback across all career stages (14.3% higher trainees, 14.3% core trainees, 42.9% non-trainees, and 28.6% foundation doctors), transformed this dynamic. It provided targeted guidance on IRH roles, inter-team collaboration, and referral protocols. Follow-up data showed unanimous satisfaction (100%), confirming its success in clarifying team structures and workflows. Notably, even experienced doctors (>6 months tenure) reported resolving persistent knowledge gaps, proving its value as both an onboarding tool and ongoing reference.

Doctors unanimously agreed that the pack improved role clarity, referral efficiency, and inter-team collaboration. Its accessible format was particularly praised, enabling quick information retrieval. However, the persistence of challenges among longer-serving staff highlighted a broader need: induction resources must evolve alongside service changes. Regular updates will be essential to maintain relevance and support continuous professional development.

This initiative underscores how structured onboarding directly enhances team cohesion and patient care. Its success stems from iterative design informed by frontline feedback—a model for addressing systemic gaps in workforce training. Future efforts should prioritise cyclical review processes to sustain these gains, ensuring the IRH's induction framework remains as dynamic as the teams it supports.

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Patterns of Sleep Disorder in Alzheimer's Dementia- A Scoping Review

Ayobami Yusuff and Ioana Toma

Abstract

Introduction

Alzheimer's disease (AD) is the most common type of dementia, characterised by progressive cognitive deterioration alongside other symptoms. Amongst the non-cognitive symptoms, 25 to 40 % of patients suffer from disturbed sleep patterns. Sleep is essential to the brain as it supports learning and memory, regulates synaptic plasticity, and enhances waste clearance.

Method

This review was set out to explore and understand the possible patterns of sleep disorder in AD, adding to the existing knowledge about this condition of growing health significance.

This is a systematic search of the literature in the EBSCO host databases. The keywords for the search criteria in the Boolean operators of multiple databases were words that describe 'Alzheimer AND sleep disorder', done up to 9 January 2024.

Results

A total of 1,511 records were obtained, 118 were read in full text and only 20 of these met the study criteria. The results demonstrated macroarchitectural sleep changes, which were 45% of the total results. Other results included reduced sleep drive, broad REM sleep changes and reversal of normal physiological sleep pattern; microarchitectural sleep changes are higher N1 and lower N3, reduced alpha activity during wakefulness and prominent EEG slowing in REM sleep.

Discussion

There are diverse sleep changes in AD which impact its presentation and course. Sleep disorders may also predate cognitive decline and may therefore be a potential biomarker for AD and may be used to discriminate the stages of its severity. We recommend that sleep history should be incorporated into the holistic assessment and management plans of patients with AD.

Introduction

AD accounts for most types of dementia cases and is characterised by insidious progressive cognitive deterioration alongside a constellation of other non-cognitive symptoms [1]. At the molecular level, it is a progressive neurodegenerative disorder characterised neuropathologically by the accumulation of amyloid-β as extracellular plaques and intracellular neurofibrillary tangles of hyperphosphorylated tau [2]. Cognitive symptoms include short-term memory dysfunction, executive dysfunction, reading and calculation impairment, comprehension and judgement difficulties, and attentional struggle. Within the category of non-cognitive symptoms, we find mood disturbances, psychotic symptoms, behavioural alterations such as agitation, aggression, wandering and pacing, and changed sleep patterns [3].

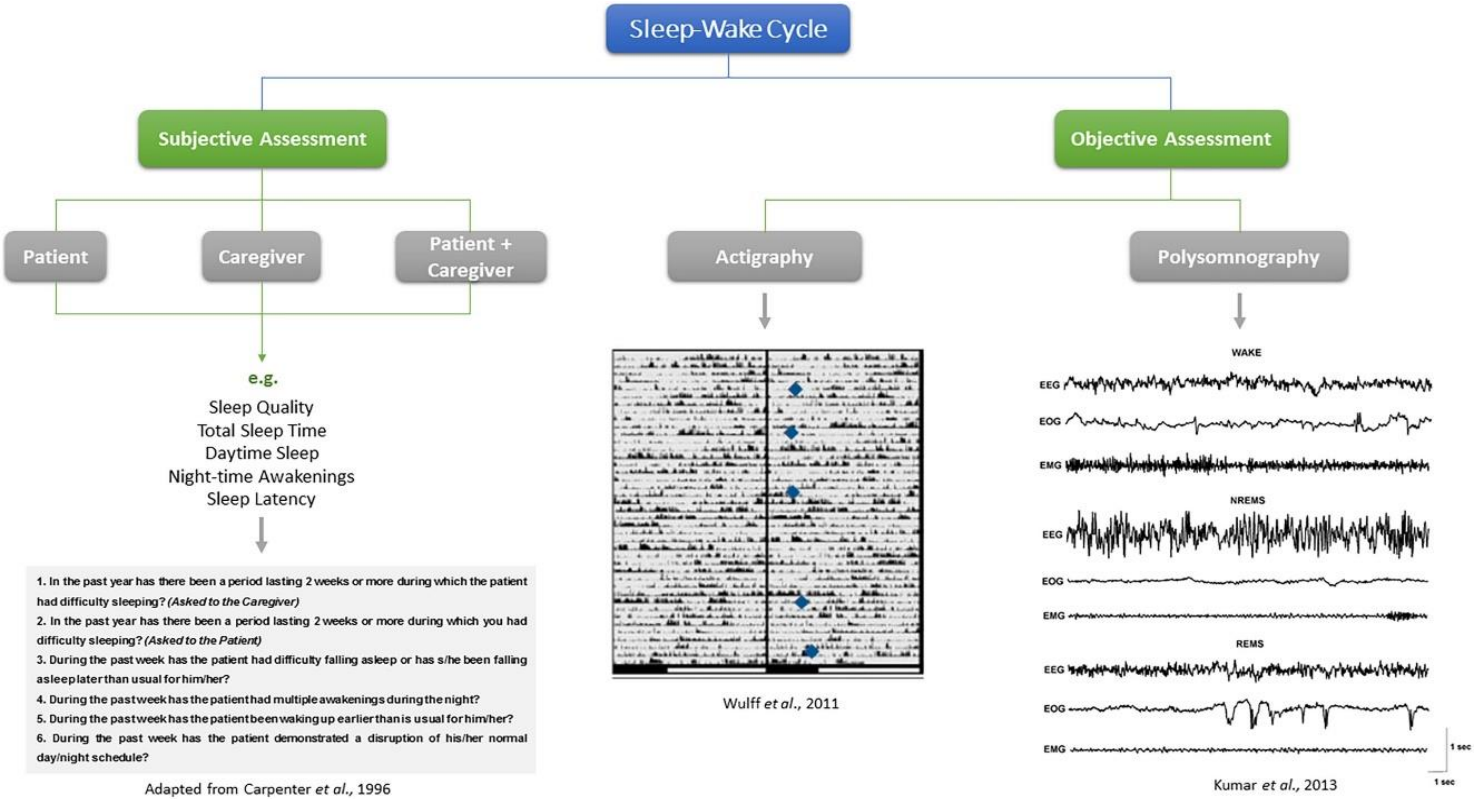
Disordered sleep patterns in AD

About 25% to 40% of AD patients suffer from disturbances of sleep, such as insomnia, excessive daytime sleepiness, rapid eye movement (REM) sleep behaviour disorder, sleep apnoea and circadian rhythm disturbance [3]. Another type of agitated behaviour in AD is sundowning, which is described as the occurrence or exacerbation of behavioural symptoms of AD in the afternoon and evening, closely related to disturbance of the circadian rhythm [4]. Sleep problems occur early in

the course of AD, and disordered sleep has been associated with an increased risk of developing AD [5].

When assessing sleep disorders in AD, several methods have been applied.

Figure 1: Subjective and objective assessments of sleep in patient populations. Image adapted from Winsky-Sommerer et al [6].



Epidemiology of Sleep Disorders in AD

Disturbed sleep is one of the most reported symptoms in people with dementia [7], but estimates of its prevalence vary significantly, ranging between 5 % and 86 % [8]. Differences in the measures used to identify the sleep problems, dementia subtypes, and dementia severity of people in the sample being studied might all contribute to this variability in estimates.

The prevalence of sleep disturbance may vary depending on the setting in which the persons with dementia live, as community-dwelling individuals and those living in long-term residential settings are subject to numerous environmental differences, such as privacy, noise levels and the presence of specialised carers versus family caregivers. As a result, it is crucial to differentiate these two population groups when analysing sleep disturbance patterns, as around 84% of people with dementia worldwide live in the community [9]. Moreover, sleep disturbances are a predictor of people with dementia living in the community moving into care homes [10].

The multifaceted nature of geriatric sleep impairment is believed to reflect a complex interaction between a variety of factors, including age-dependent decrease in the ability to sleep, neuropsychiatric comorbid disorders such as depression and dementia, pain and limitation of mobility with excessive bed-time, increased prevalence of sleep-disordered breathing and nocturnal myoclonic activity, and disturbed behaviour during sleep including nocturnal confusion (sundowning) [11].

Accumulating evidence shows that sleep disturbance contributes to cognitive decline and

might also increase the risk of AD [11]. Thus, it would be helpful to understand the patterns of sleep disorder (SD), as this can potentially guide clinicians in designing care plans and incorporate and address carer burden. Furthermore, emerging research has provided a direct link between several of these sleep disruptions and AD pathophysiology, suggesting that treating sleep disorders in this population may target basic mechanisms of the disease [12].

Aim

There is growing evidence that sleep disorder in AD is a core part of clinical presentation; however, little is known about the characteristics and manifestations of SD in AD. This review aims to identify and summarise current patterns and characteristics of sleep disorders in AD.

To the best of our knowledge, this is the first article synthesising multiple reviews that describe the patterns of presentation of sleep disorder in AD. To understand the implications of the potential relationship between the two conditions, we formulated the following research questions:

What are the relationships between sleep disorder and AD?

What are the patterns and characteristics of sleep disorder in AD?

What are the potential clinical indications for identifying them?

Method

Using the methodology of a systematic review but adopting a scoping review, we were able to qualitatively extract and describe the results. The Preferred Reporting Items for Systematic reviews and Meta Analyses (PRISMA) extension for scoping reviews was used as a guideline in writing this project. On the hierarchy of evidence, all studies with a level of III or above were considered. We used the Patient/Population, Intervention, Comparison, and Outcomes (PICO) framework to appraise each study.

A systematic search of literature in EBSCO host Research Databases, including: EMBASE, PubMed, MEDLINE, CINAHL, and GreenFILE was conducted until 9 January 2024; there was no limit to the starting search date. The keywords for the search criteria in the Boolean operators were words that describe 'Alzheimer AND sleep disorder'. The following keywords were used: neurodegeneration and sleep disturbance, neurodegeneration and sleep disorder, Alzheimer and sleep disturbance, dementia and sleep disorder, and dementia and sleep disturbance.

The study criteria used were: studies published in the English Language and performed on human subjects, not animal models, not AI-generated reports and clearly answer the aims of the literature review, i.e., focus on describing the patterns of sleep disorder in AD.

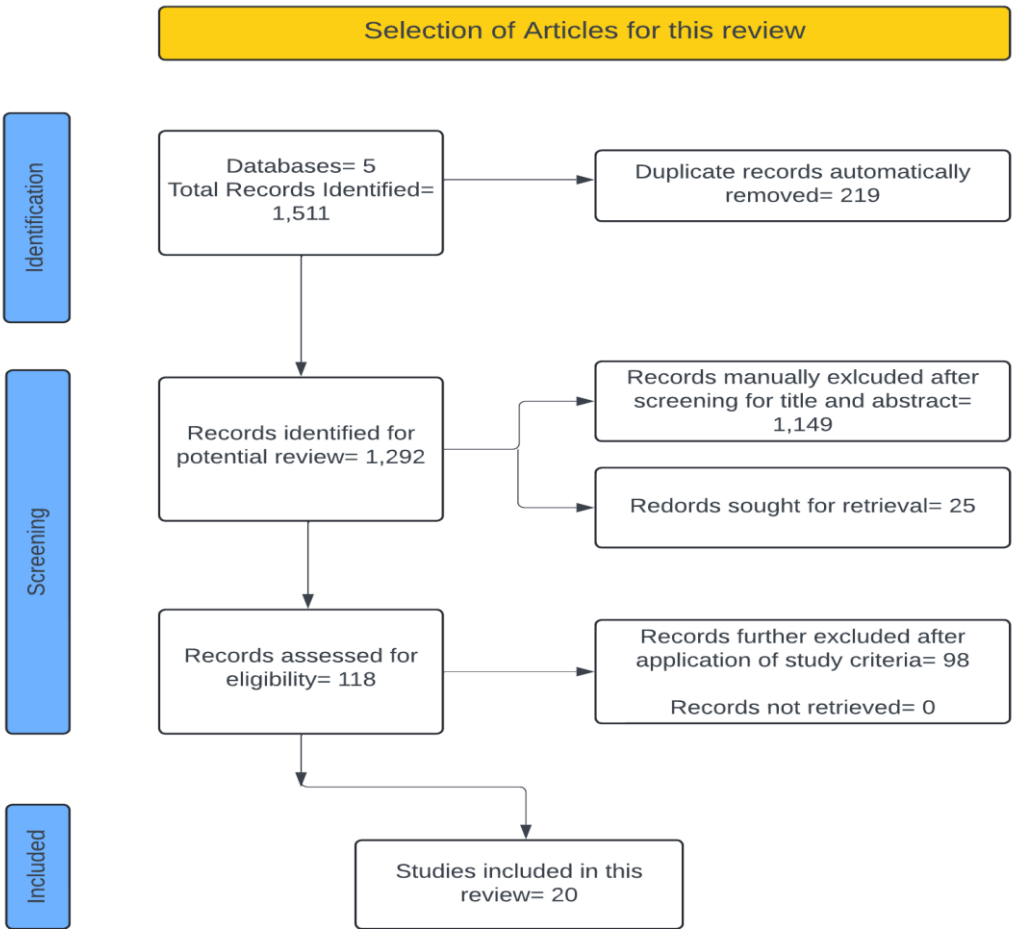
After an initial stage of reading the title and abstract of each study generated from the search, studies that met the research aim were extracted. The first grade of collected studies were then read in full text to identify those that specifically answer the research question.

We invited an independent reviewer to inspect the search criteria, the findings, all abstracts and then the full texts identified from the searches. The process was facilitated by the use of EBSCO host folder sharing.

As a literature review, it is generally considered low-risk research with no direct contact with human subjects and so no specific ethics application was sought; however, for each article referenced in this project, appropriate ethics have been considered as described by the authors.

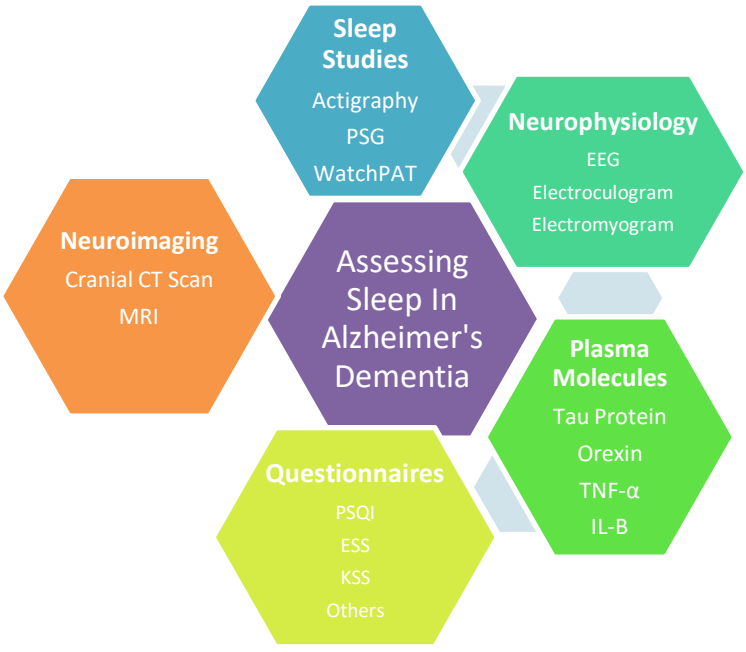
1,511 results were obtained in the initial search. 219 duplicate articles were automatically removed. 118 publications were read thoroughly and only 20 were included in this review.

Figure 2: Flowchart of the literature review



Results

Figure 3: Methodologies identified



We classified the disturbed sleep patterns broadly and vaguely into macroarchitectural changes and microarchitectural changes, though this may not be applicable in all the results of this review, as some articles correlated sleep disturbance with other parameters such as the proteinopathies and genetics of AD.

Macroarchitectural sleep changes

Table 1: P-Patients, C-Controls

Authors and year	Sample size	Method/ Parameters measured	Findings/Results	Values
Devulder et al 2023	P- 41 C-30	24h EEG PSG	Reduced REM Increased WASO Reduced SE Longer TIB	REM reduced by <6%; REM < 33 mins WASO > 72 mins; P=.001 SE reduced by < 12%, P=.087
Liguori et al 2016	P- 20 C- 26	EEG PSG MRI CSF Orexin	Negative correlation between CSF Orexin and REM	R = −0.65, p = 0.001
Vitiello et al 1990	P- 44 C- 45	PSG	WASO significantly greater in the AD group AD subjects spent significantly more TIB and had significantly less %3-4 TIB REM latency measures were significantly shorter in the AD group	P=.006 P=.03 P=.04; p=.01
Tadokoro et al 2020	P= 41 C= 22	PSQI WatchPAT	Bad self-check PSQI scores in AD vs NC Decreased REM sleep in AD Increased NREM sleep in AD	PSQI>5 (42.9%) vs (81.8%, * p < 0.05) P<0.05 P<0.05
Gagnon et al 2006	P= 15 C=15	EEG Infrared video	Shorter sleep time in AD Lower nr of REM periods in AD Higher percentage of stage 1 sleep in AD RSWA in Ads	

REM sleep and Sleep Efficiency (SE)

REM sleep changes and reduced SE have been consistently reported [13,14,15,16]. AD patients have been observed to have less REM sleep plus shorter REM latency than their healthy counterparts and this has even been observed at the stages of subjective cognitive impairment, prior to the clinical assessment or diagnosis of AD [15]. While REM sleep is generally associated with low muscle tone, a small number of patients with AD have abnormal motor activity in REM sleep, a significant number of them do have REM sleep without atonia (RSWA) [14].

Wake after sleep onset (WASO) has also been reported in AD and has been identified as a predictor of progression of mild cognitive impairment (MCI) to AD [13,16]. Sleep efficiency (SE) is described as a ratio of the time spent asleep to the total time in bed (TIB). A sleep efficiency of 85 % or higher is normal, while a sleep efficiency anywhere above 90 % is considered very good. While reporting SE, Alzheimer's sufferers spent shorter sleep time [14], more time awake, plus high number of nocturnal awakenings, and more TIB [17]. Overall, there is reduced SE in AD.

Some reports show slightly different results; for example, WatchPAT analysis showed REM sleep decreased significantly in AD and slightly in MCI compared with normal controls (NC) [18]. Non-REM light sleep increased significantly in AD and slightly in MCI compared with NC, but not deep sleep. These findings also demonstrated occasional differences between objective and subjective reports/assessments of sleep in AD, although objective assessments are still preferred options for clinicians.

Disturbance of physiological sleep timings

Table 2: P- Patients, C- Controls

Authors and year	Sample size	Method/ Parameters measured	Findings/Results	Values
Tractenberg, E., Singer, M., and Kaye, A. (2006)	263 AD 399 controls	SDSQ IADL Cornell Depression Scale	SP had higher SDSQ scores and more no of symptoms People with SP had worse depression scores Six symptoms were more frequent in AD + SP: number of wakeups per night; drowsy during day; daytime naps; equal day, night sleep time; trouble breathing; breathes irregularly	P<.001 P<.01 In both cohorts SL >30 mins
Ancoli-Israel et al (1994)	246 AD 94 controls	Sleep questionnaire (10) BDRS DRS POD	The more demented the patient, the more time they spent in bed, the more fragmented their sleep and more daytime naps they took. Correlation between prolonged sleep, disruptive behaviour and severity of dementing process	
McCurry et al. (1999)	205 AD	BDRS RMBPC	Most frequent sleep problems reported by caregivers was patient sleeping more than usual (40%) and patient awakening early (31%), which caused relatively little distress Other behaviours (e.g., waking the caregiver up at night) were much more distressing.	

Liguori et al 2014	P- 48 C- 29	PSG CSF Orexin CSF tau and Aβ1-42	Earlier bedtimes in severe AD Increased CSF orexin levels were correlated with decreased sleep efficiency, N3 and REM sleep.	P=.04, r = −0.38; P = .007, r = −0.32 P = .027, r = −0.31 P = .03 respectively.
Mulin et al 2011	103 AD	Actigraphy	Patients with AD and apathy have higher WASO, more TIB and mean duration of daytime inactivity periods	P<0.01 P<05 P<0.001
Park et al 2011	36 AD	UPDRS ESS, KSS PSQI MSLT PSG	Patients with AD+PF are more sleepy than those without PF	

Several reports have described a change in sleep timing, sleep scheduling and reversal of normal day-night sleep patterns. AD patients present with nocturnal awakening [16], earlier bedtimes with daytime sleepiness [19], night-time wakefulness and nocturnal wandering [17], higher duration of daytime inactive periods [20], and a worse daytime dysfunction [21].

In a retrospective cohort study, Tractenberg et al [22] analysed 21 sleep disturbance symptoms using The Sleep Disturbance Symptom Questionnaire (SDSQ), Instrumental Activities of Daily Living (IADL) questionnaire and The Cornell Depression Scale. For less than half of the symptoms in people with AD and a quarter of those in non-demented elderly (NDE), endorsement rates were significantly different depending on whether the reporter (or their sleep partner) did or did not report a sleep problem. In both the cohorts, people with sleep problems (SP+) had significantly higher SDSQ total scores and numbers of symptoms endorsed, and significantly worse depression scores. In both cohorts, SP+ reflected long sleep latencies (>30 min) and a tendency to lie awake worrying, as well as restless sleep, waking too early, waking during the night for at least 1 hour and the use of medication to get to sleep; having bladder and bowel problems at night also followed this trend. The authors suggested that daytime sleep is the most distinctive feature of sleep disturbance in AD patients.

It has been observed that the more demented the patient, the more time they spend in bed, the more fragmented their sleep is, and the more daytime naps they took. There was a correlation between lengthy sleep and disruptive behaviour [23]. Here, it was demonstrated that prolonged sleep is associated with a severe dementing process and behavioural disturbance.

Cluster analysis results revealed three distinct patterns of patient behaviour that were associated with patients awakening their caregivers at night:

(1) mildly demented patients who were fearful, fidgety, and occasionally sad;

(2) patients with few mood or behaviour problems but who spent considerable periods of time sitting alone during the day; and

(3) more severely demented patients with multiple cognitive, depressive, and disruptive behaviour problems [24].

The most frequent sleep problems reported (e.g., sleeping more than usual) caused relatively little caregiver distress, while other behaviours (e.g., waking the caregiver up at night) were much more distressing.

Molecular tests and other Alzheimer's proteinopathies

Table 3: P- Patients, C- Controls

Authors and year	Sample size	Method/ Parameters measured	Findings/Results	Values
Oh et al 2022	P- 53 C- 32	EEG and PSG Postmortem immunochemistry	Greater loss of wake-promoting neurons (LC, LHA & TMN) in AD	Shorter sleep time- LHA: r = −0.63; P = .009; TMN: r = −0.62; P = .008 Greater WASO- LHA: r = 0.85; P < .001; TMN: r = 0.78; P < .001 Greater number of LC neurons = shorter total sleep time (r = −0.68; P = .008) and greater REM latency (r = 0.71; P = .006).
Cooke et al 2006	P- 76 C-12	PSG; effect of AChEI	Lower stage 1 sleep in Donepezil vs Galantamine Higher stage 2 sleep in Donepezil vs controls	P=0.01 P= 0.04
Chen et al 2012	P- 43 C- 22	PSG ESS	SE and WASO higher in AD	
Durães et al 2023	P-78 C-70	PSQI CSF Biomarkers	Sleep duration score was lower in AD Daytime dysfunction worse in AD Negative weak correlations between Ab42 values and daytime dysfunction Weak correlation between t-tau values and daytime dysfunction	 R= - 0.21, P=0.011 R= - 0.16, P=0.046

Several Alzheimer's disease proteinopathies have been associated and correlated with sleep disturbance. In a recent discovery of the glymphatic system- the lymphatic system of the CNS - disturbance of the glymphatic system is mediated through aggregation, impaired clearance, and toxicity of AD proteinopathies (Aβ, APOE4, tau, and others). This leads to a cascade of hypoxia-ischemia and atrophy of the sleep regions in the brain [25]. Although the AD biomarkers were higher in AD patients than controls, there were no statistically significant differences in most studies [16,19].

Orexin system dysregulation may contribute to sleep impairment in MCI patients with a high likelihood of AD pathology, mainly affecting REM sleep and resulting in insomnia, prolonged sleep latency, and nocturnal awakenings [16]. Increased Orexin-A in CSF is associated with sleep fragmentation and sleep disruption. Higher levels of Orexin have been associated with decreased SE and decreased REM sleep [19].

These results imply that in AD, increased CSF orexin levels are linked to a parallel sleep deterioration, which further appears to be related to cognitive decline. Hence, in AD, orexinergic output and function seem to be overexpressed with disease progression and severity, possibly owing to an imbalance in the neurotransmitter networks

regulating the wake-sleep cycle toward the orexinergic system promoting wakefulness.

In another report, AD patients carrying the -31TT genotype in the IL-1β promoter plus the APOEε4 allele were susceptible to sleep disturbance [26]. This group showed less TIB, longer sleep latency and REM latency, more awakenings, and a lower slow wave sleep (SWS) percentage than those carrying CC/CT combined genotypes. This demonstrates that the synergistic effect of the IL-1β-31TT genotype and the APOEε4 allele led to an increased susceptibility of inducing IL-1β, IL-6 and TNF-α overexpression and sleep disturbance in AD patients.

Longer sleep duration has also been suggested as a sign of disease severity [27], i.e., sleep duration in AD (7.8 ± 1.5 hours) vs MCI (7.4 ± 1.4 hours). In terms of the qualitative measures, daytime dysfunction was worse in AD patients. A weak correlation was found between t-tau values and daytime dysfunction ($r = -0.16$, $P = 0.046$).

Chen et al [28] reported no significant correlation between serum levels of TNF-α and sleep parameters in AD patients. However, a significant difference in serum TNF-α was observed between AD patients with daytime sleepiness and those without daytime sleepiness and controls ($p<0.05$). SE and WASO were also significantly reduced in AD patients with and without daytime sleepiness when compared with controls on the PSG (polysomnography) reports. As such TNF-α may be a mediator of daytime sleepiness in AD patients due to an exaggerated secretion, thought to be triggered by activated microglia in AD patients.

We also identified that AChEIs have some effects on Sleep Architecture in mild to moderate AD. AChEI therapy had a significant effect on the percentage of stage 1 ($p = 0.01$) and stage 2 ($p = 0.03$) sleep, which was not attributable to the use of other medications [29]. Patients in the donepezil group had a significantly lower percentage of stage 1 sleep than patients in the galantamine group ($p=0.01$). and had significantly higher percentages of stage 2 sleep than patients in the no AChEI group ($p=0.04$). It is postulated that apart from different timing/administration schedules of donepezil, it is possible that the differential effects on sleep architecture of the AChEIs used in this study may reflect variations in the mechanisms of action of the drugs, despite being in the same AChEI group.

Mixed sleep microarchitecture and macroarchitecture

Table 4: P- Patients, C- Controls

Authors and year	Sample size	Method/ Parameters measured	Findings/Results	Values
Falgàs et al. (2023)	P- 19 naAD 15 aAD C- 14	Video EEG	Higher WASO in aAD vs control Higher N1 in a AD vs controls Lower sleep maintenance in aAD vs controls Lower N3 in aAD vs controls Higher N1 in naAD Less REM in naAD Longer REM latency in naAD Lower N3 in aAD vs naAD	P<.05 P>.01 P<.05 P>.05 P<.05 P<.01 P<.05 P<.01
Montplaisir, J. et al. (1998)	8 AD	EEG	Significant reduction in alpha activity in all give cortical regions during wakefulness Decrease in alpha waves associated with advanced disease	P<.04
Kim, Y. et al. (2020)	83 AD 331 controls	EEG NTF	Abnormal REM sleep in AD Patterns 4, 18, 28 and 30 positively related to AD Pattern 4 – delta and theta wave (0.5 -8 Hz) and Pattern 18 – theta wave (4-8 Hz) Represent abnormal sleep	P>.05

According to Devulder et al [13], in AD patients ages 55-85 years, IEDs (interictal epileptiform discharges) were present during sleep and wakefulness through TIB, whereas in control subjects, they occurred exclusively during sleep. In both groups, epileptiform spikes were most frequent during sleep stage N2. The authors concluded that decreased REM sleep and more severe sleep-disordered breathing are consistent with AD and this might be related to sleep epileptiform activity.

In a study assessing 8 AD patients in the sleep laboratory for 6 months [30], the authors reported that a significant reduction in absolute alpha activity was the only factor involved in EEG slowing, especially during wakefulness, for all five cortical regions – frontal, central, occipital, parietal and temporal (Wilcoxon $p<0.04$). The authors conclude that the decrease in alpha waves parallels the evolution of the disease, and persistent low alpha activity is a phenomenon observed in advanced stages of the disorder.

Falgàs et al [31] investigated the sleep architecture in different phenotypes of AD (amnestic/typical vs. non-amnestic/atypical) using overnight video-EEG in a cross-sectional study of 15 individuals with amnestic AD, 19 individuals with non-amnestic AD and 14 healthy controls. In their results, amnestic AD showed higher WASO and N1, and lower sleep maintenance and N3. In contrast to amnestic AD, atypical AD had less REM sleep and longer REM latency. Lastly, N3(%) was significantly lower in amnestic than in atypical AD. This study confirmed a sleep fragmentation pattern consisting of more time spent awake (WASO) and greater time in N1 sleep in AD overall compared to healthy controls.

Kim et al [32] factorised 5 patterns (using clinical variables and EEG signals; $p>0.05$, 4 patterns for AD likely and 1 pattern for normal ones- That is, Patterns 4, 18, 28, and 30 were positively related to AD, whereas Pattern 2 was negatively related). The abnormal patterns are Pattern 4 and 18,

representing abnormal REM sleep. Pattern 4 refers to Delta and Theta wave (0.5 Hz - 8 Hz) with slow oscillation and slow wave activity and also Gamma wave (30 Hz ~), Pattern 18 refers to Theta wave (4 - 8 Hz) with Gamma wave during the middle of sleep (4 - 7 hour); Pattern 28 represents drowsy sleep, focused on Theta wave 4-8 Hz and Alpha 8-10.5 Hz waves several times throughout the 8 hours of sleep; Pattern 30 represents insomnia, it has very strong signals on Beta and Gamma waves (15 Hz ~) during the first one hour and 5 - 8 hour of sleep. Pattern 2 represents healthy normal sleep, representing slow wave activity and spindle with Sigma wave (12 - 15 Hz). The authors conclude that there are distinctive and unique sleep patterns associated with AD, and sleep disturbances seem to be a risk factor for the development of AD and cognitive decline.

Sleep disorder in AD in relation with comorbidities

Mulin et al [20] reported apathy and sleep/wake disturbances in individuals with AD. AD patients with associated apathy have less consolidated nocturnal sleep than those without, with significantly higher WASO ($p<0.01$), more TIB ($p<05$) and mean duration of daytime inactivity periods ($p<0.001$). The confluence of apathy and sleep disturbance in individuals with AD is yet to be elucidated; however, it is believed that apathy may be regulated by the same circadian pacemaker as mood is, with involvement of the suprachiasmatic nucleus.

Park et al (2011) hypothesised that Parkinsonian features (PF) correlate with sleepiness in mild AD21. Based on the presence of PF, present (AD+PF, n=17) or absent (AD-PF, n=19), participants were categorised into 'sleepy' or 'non sleepy' according to MSLT scores (cut off score = 10/4). The AD+PF group has higher periodic limb movement associated with arousal than the AD-PF group. 75 % of the AD+PF were classified by the MSLF as 'sleepy' whilst only 31.6 % of the AD-PF group were classified as 'sleepy'. These results demonstrate that the presence of sleepiness can be seen as a possible risk factor for cognitive impairment and for the development of other neurodegenerative disorders, such as PD.

Discussion

This literature review was undertaken to further understand how sleep disorders manifest in AD and whether they have any specific pattern. We identified that several years before the onset of cognitive impairment, cerebrospinal fluid (CSF) biomarkers of AD, including amyloid β (Aβ) and tau, begin to accumulate in the brain. It has been hypothesised that sleep changes start in the preclinical stages of AD, even more as a possible risk factor for this neurodegenerative disorder by increasing the brain's Aβ burden [33]. Moreover, midlife sleep disturbances may contribute to pathological Aβ deposition, and the short-sleep duration during midlife influences the neurodegenerative process and accelerates the AD-related regional hypometabolism [34].

In this review, we have identified common disordered patterns of sleep in AD patients. These have been observed through objective and subjective reports and analysis and were broadly grouped into macroarchitectural and microarchitectural changes. Examples of macroarchitectural sleep changes include reduced sleep drive, REM sleep changes, reversal of normal physiological sleep pattern, reduced sleep efficiency and increased motor activity at night, while examples of microarchitectural sleep changes are higher N1 and lower N3, reduced alpha activity during wakefulness and prominent EEG slowing in REM sleep.

We have identified that sundowning, the occurrence or exacerbation of behavioural symptoms of AD in the afternoon and evening, is closely related to disturbance of the circadian rhythm, and is emphasised as being caused by circadian disturbance, particularly as a phase delay of body temperature [4,35].

We also identified that some medications, even AChEI used in treating AD, has effects on sleep, with differences observed between the types, such as Donepezil lowering the stage 1 sleep and increasing stage 2 sleep, compared to other

AChEIs. In a study by Grippe et [36], sleep parameters in AD patients were evaluated before and after trazodone use. The authors reported that sleep-wake cycle patterns were found to be significantly different across the two treatment arms, with a ratio much closer to a normal daily sleep pattern amongst medication users. Trazodone could potentially be used as a safe choice to treat sleep-related circadian disturbances in AD patients, as it shows a stabilising effect on circadian rhythms.

Although our review is focused on characterising the patterns of sleep disorders in AD, we identified that several factors contribute to these sleep disturbances and disruptions. They include genetic susceptibility between AD and sleep disorders sharing similar pathways, disruption of the circadian system (mediated through several other factors), increased orexinergic output and orexin system dysregulation, increased TNF-α, loss of wake promoting neurons in the CSF, co-occurrence of primary sleep disorders, other comorbid physical and psychiatric disorders particularly depression and Parkinson's disease [37,38].

Conclusion and Recommendation

This review summarises the multifaceted nature of sleep disorder in AD and the relationship between them. Sleep is essential to the brain as it supports learning and memory, regulates synaptic plasticity, and enhances waste clearance from the brain. Sleep disturbance and disruption impairs sleep-dependent memory and it predates cognitive decline and AD. We consider sleep to be a potential biomarker for AD and may even be used to discriminate the stages of its severity. Sleep history should routinely be factored into the assessment of patients with AD, with both objective and subjective measurements considered, as it would form a crucial part of the management of AD. Further studies describing how sleep may be used to discriminate severity and stages of MCI/AD are hereby advocated.

Declaration of competing interest

The authors declare that there are no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Rest of the references are available on request

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A Needs Assessment for the Development of an Inpatient Forensic Mental Health Service in Malta

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Background

Forensic mental health service specialises in the assessment, treatment and management of mentally disordered offenders. Despite having variations in legislation, forensic mental health teams worldwide share common aims of providing care for severely mentally ill patients who have a tendency towards delinquency, preventing relapse of offenders and reducing recidivism rates, as well as acting as the interface between psychiatry and the legal system, by liaising closely between both clinical and non-clinical professionals [1,2]. To achieve these goals, specialised knowledge and skills must be developed, especially in the assessment and management of violence and sexual deviance [3].

The incorporation of forensic mental health teams within the National Health Service (NHS) has been the standard practice since the early 1990s [4]. At present, there is no forensic service functioning within Mental Health Services (MHS) in Malta. This puts a significant burden on the general psychiatric services and fails to prevent further violent crime and safeguard the public.

A summary of the most common international forensic mental health service pathways is summarised in the figure below (Figure 1). Inpatient forensic mental health services are typically structured around varying levels of therapeutic security [5]. Furthermore, international practices place an emphasis on rehabilitation, through both the alleviation of psychiatric illness as well as the reduction of recidivism. A focus on recovery-oriented approaches has highlighted the importance of patient-centred and collaborative care, rather than more custodial or punitive models of forensic mental health services [3].

Figure 1: A summary of forensic mental health service pathways in UK Trusts.

Standards of Care

In July 2023, the Quality Network and the Royal College of Psychiatrists released updated guidelines outlining the standards of care for forensic mental health services, emphasising key priorities such as efficient admission and assessment pathways, high-quality physical and mental healthcare, multidisciplinary treatment plans, and robust recovery and rehabilitation support both in inpatient and community settings [6]. The guidelines also highlight the importance of patient involvement in care planning, family support, safe and appropriate ward environments, stringent security protocols, safeguarding, legal representation, a well-trained multidisciplinary workforce, and the reduction of restrictive practices—all of which should form the foundation for developing a new forensic service.

Objectives

This project prioritises a structured, phased approach to developing forensic mental health services, with the immediate focus on establishing an **inpatient forensic care** unit within Malta's Mental Health Services (MHS). We endeavoured to conduct a needs assessment to quantify the current need for inpatient forensic services in Malta. Subsequent phases will expand to include **community forensic care, prison in-reach services, and violent crime prevention** initiatives, ensuring a comprehensive and integrated system for managing mentally disordered offenders. The current proposal lays the foundation by addressing the most urgent need, specialised inpatient care, while setting the framework for future service enhancements.

Quantification of Risk

Risk can be quantified using the DUNDRUM-1 scale, which is an 11-item triage security scale. This can ensure consistency in accepting referrals to the proposed service. Each of the 11 items is rated on a 5-point scale from 0-5, as follows:

0 - No security needed or no mental disorder.
1 - Could be managed in an open hospital ward.
2 - Could be managed in a local PICU or low secure unit.
3 - Could be managed in a medium secure unit.
4 - Special or high secure unit required.

The Dundrum QUARTET [7,8] consists of four structured professional judgement instruments that are to be used for admission triage, determination of urgency, assessment of treatment completion and prognosis for recovery. The first of the four questionnaires (DUNDRUM-1) is used to triage admissions to secure units and determine the level of physical security that would be required for the safe management of the patients in question. Although related, this differs from other tools which assess risk of violence, such as the widely used Historical Clinical and Risk Management - 20 (HCR-20) [9], as it provides an indication of the level of security required for violent patients and thus, would give an indication as to whether a patient would benefit from being managed by a specialist forensic team. It is frequently used in the U.K. and Ireland as part of the pre-admission assessment and decision-making process for patients who are referred from the criminal justice system. For example, from prison in-reach teams or court liaison services, as well as from general adult psychiatric services. There is no current formal training to administer the DUNDRUM quartet; however, the authors of the toolkit provide detailed guidance on its use, including clinical examples and vignettes. The DUNDRUM-1 triage security items are mainly static in nature and so it is possible to utilise patient notes in the completion of this assessment.

DUNDRUM-1 is an 11-item triage security scale. Each of the 11 items is rated on a 5-point scale from 0-4 as follows:

>3: Indicates need for high security.
>2: Indicates need for medium security.
>1: Indicates need for low security (including PICU and long-term low security).
0-1: Indicates need for open hospital or community-supported placement.

Each of the 11 items can be scored from 0-4, indicating the level of security that is likely

needed, as shown above. The authors of the toolkit recommend that, to benchmark the need for therapeutic security for a group or cohort, items 2 and 4 be omitted from the score. Thus, the remaining nine items can be scored according to the 5-point scale and a mean score can be calculated.

The scale has shown good internal consistency and inter-rater reliability. Thus, the triage security scale can be used to ensure consistency in decision-making and to advise admission criteria for forensic mental health services [10]. Furthermore, the tool shows good predictive validity and is associated with more efficient use of resources, without compromising risk to the public [11]. The DUNDRUM-1 triage security scale has also been used in other European countries and was shown to have good reliability in these instances [12].

Method

A formal needs assessment was conducted for Malta's Mental Health Services (MHS) to evaluate service demands, using anonymised patient data without requiring ethical approval since no direct contact occurred. Data included a snapshot of all Mount Carmel Hospital (MCH) admissions on March 24, 2024, as well as admissions and discharges between 18th and 31st March 2024. Researchers analysed medical records to collect non-identifiable demographic and clinical data - including mental health diagnoses, risk of harm to others, and substance use history, which was securely stored in an encrypted Excel sheet.

Patients were initially filtered according to their clinical presentation to the hospital:

- Patients without an indication of increased risk to others were excluded.
- Patients who were currently detained in hospital under criminal jurisdiction (insanity defence) or court order were automatically included.
- The DUNDRUM-1 screening tool was used for patients with a relevant history of increased risk to others and who may be considered eligible for the service. After the 9 items (TS1,TS3,TS5-TS11) are classified, the average score will be used to identify the patient's security needs.

These groups inform the need for a forensic inpatient service, including the number of patients who need the service, and the size of wards specifically dedicated to forensic care. Furthermore, an arbitrary cut-off of 1 will be used to include all patients who require at least a low level of security and thus would possibly require specialist forensic care.

Proposed Inclusion & Exclusion Criteria

Proposed criteria for patients to be referred to and managed by the forensic mental health team are as follows:

- Patients who pose a high risk to others, as evidenced by both their mental state and behaviour and who cannot be managed safely in a general ward setting and by less restrictive means. This can be classified by a DUNDRUM-1 score of above 1.
- Patients who are sectioned under criminal sections of the Mental Health Act. This refers to all patients who were deemed to be mentally ill during the time of their index offence and were thus admitted to hospital instead of carrying out their prison sentence.
- Patients who are currently under court order for psychiatric treatment following a violent offence.
- Patients who have been recently admitted to the prison's forensic unit and have completed their prison sentence.

A DUNDRUM-1 score of above 1 indicates the need for at least a low level of security and thus, for patients to be admitted within a forensic service. Current inpatient services in Malta lack a secure unit and patients who generally score above 1 are treated in general adult wards. Patients are transferred to a PICU setting depending on acute risk to self and others and generally, admissions to a PICU are kept to a minimum number of days before transfer to a normal ward. Therefore, there is no available service for patients who require

longer admissions in a more secure setting or who pose a more chronic risk to others.

Needs Assessment Results

All inpatients on the 24th of March 2024 as well as all admissions and discharges from the hospital between the 18th and 31st March 2024 were included in the data sample. This adds up to a total data sample of 287 patients.

Out of the total data sample, 26 patients met the inclusion criteria. 24 of these patients were over the age of 18. There were two patients below the age of 18. The youngest was 13 years old and the eldest was 75 years old. The mean age was 40 years. Only 4 patients were female, resulting in an 84.62% male cohort.

16 out of 26 patients were admitted involuntarily (61.54%). 8 patients were admitted under criminal sections of the Mental Health Act and 7 patients were admitted under Court Order.

13 out of 26 (50%) have a confirmed diagnosis of psychotic disorder (including schizophrenia, schizoaffective, BPAD or substance-induced psychosis).

23 out of 26 patients had a history of violence. Although the other three patients did not have a history of violence documented in their notes, they were automatically included due to being detained under criminal sections.

13 out of 26 had been admitted from 200-365 days in 1 year. Only one patient was admitted for under 1 week - 15 y/o ASD. Average days admitted in 1 year: 191.84 days. An average of 4.16 psychiatric medications per patient was identified. The highest number was 9 psychiatric medications for one patient. 12 out of 26 were prescribed elevated supervision and had police involvement during the admission.

11 out of 26 had served a prison sentence, 5 of whom had been admitted to the prison forensic unit. 21 out of the 26 had at least 1 criminal case. 2 patients were on probation.

18 patients used substances (69.23%). 72.22% (n=13) of those used cocaine, 44.44% (n=8) used THC, 27.78% (n=5) used heroin. 18 patients had a history of violence associated with substance use, meaning 100% of patients who use substances and 69.23% of the total patients.

From the younger patients, the 15-year-old male required low security and the 13-year-old male required medium security. Out of the adult patients (n=24), 2 required medium security and the rest required low security (n=22) according to their DUNDRUM-1 scoring. This mimics the situation in forensic services overseas, where high security beds are rarely required and are often situated in separate hospitals.

Conclusion

The needs assessment confirms that Malta has a significant patient cohort requiring specialised forensic mental health services, as current care standards, provided by both CSA and MHS, fall short of international benchmarks such as those set by the Royal College of Psychiatrists and NICE guidelines. This gap highlights the urgent need to enhance care quality for mentally disordered offenders within both healthcare and criminal justice systems.

The study recommends establishing a **low secure forensic unit** with a limited number of medium secure beds, segregated by gender, to address this need. For younger patients, a small adjoining unit with 2-3 low secure beds (convertible to medium security if required) is proposed. Additionally, the findings underscore the necessity of a **multidisciplinary forensic team**, incorporating social work, occupational therapy, psychotherapy, and substance use specialists. A more cohesive collaboration between MHS and CSA is also essential to ensure consistent, high-quality care for mentally disordered offenders throughout their involvement with the criminal justice system.

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From Forensic Psychiatry to Healthcare Entrepreneurship: A Personal Journey

Neeraj Tripathi

My career has taken me from the world of forensic psychiatry to healthcare entrepreneurship, a journey that has been both challenging and deeply rewarding. It began in the high-stakes environment of secure psychiatric wards and outpatient clinics, where I specialised in the complex intersection of mental health and the legal system. Those early years provided an invaluable foundation in clinical care while also exposing me to systemic inefficiencies in healthcare delivery. Treating high-risk populations—assessing competency, managing severe mental illness in correctional settings, and navigating complex outpatient cases—taught me not just about patient care, but about the broader structural gaps in access, coordination, and resource allocation.

Over time, I realised that while individual clinical work was meaningful, creating larger-scale change required a different approach. The limitations of traditional healthcare models—fragmented systems, outdated technology, and barriers to mental health services—became impossible to ignore. This recognition led me to explore innovation beyond the clinic, where I could apply my frontline experience to develop solutions with wider impact. Transitioning from clinician to entrepreneur was a leap, but one that felt necessary. I immersed myself in healthcare business strategy, learning about digital health, value-based care, and startup ecosystems, always grounding new ideas in real clinical needs rather than abstract theories.

What excites me most about this dual perspective is the ability to create solutions that are both innovative and deeply practical. Whether building telepsychiatry platforms to expand access, designing risk-assessment tools, or advising organisations on forensic mental health programs, I've seen how clinical insight can shape better business strategies—and vice versa. The challenges of merging these worlds have only reinforced how much medicine needs interdisciplinary thinking. Today, my focus remains on bridging gaps: between patient care and systemic change, between clinical expertise and entrepreneurial execution. The future of healthcare depends on this kind of integration—where innovation isn't just cutting-edge, but truly responsive to the people it serves.

The COVID-19 Pandemic: A Catalyst for Innovation

The COVID-19 pandemic marked a pivotal turning point in my career, transforming my clinical insights into actionable entrepreneurial solutions. As the world grappled with unprecedented lockdowns and social isolation, the mental health crisis reached alarming proportions - exposing critical gaps in accessibility, scalability, and specialised care. Witnessing this urgent need first-hand, I channelled my expertise in forensic psychiatry into building innovative ventures designed to address these systemic challenges.

My first initiative, **HappiMynd**, emerged as a direct response to the surge in anxiety, depression, and emotional distress during the pandemic. Recognising that traditional in-person therapy could not meet the skyrocketing demand, I developed this digital platform to provide immediate, evidence-based mental health support. Combining therapeutic tools, self-guided interventions, and psychoeducation, HappiMynd empowered users to take charge of their emotional wellbeing—bridging the gap between clinical care and everyday resilience.

However, digital solutions alone could not address the needs of all vulnerable populations. For individuals with severe and complex mental health conditions, many of whom faced heightened

instability due to pandemic disruptions, I founded Symbiosis Care. This supported living service was designed to offer stability, dignity, and personalised care to those who needed it most. By integrating clinical oversight with compassionate community-based support, Symbiosis Care became a lifeline for individuals who might otherwise have fallen through the cracks of an overburdened system.

To further expand accessible, high-quality psychiatric care, I established Bucks Psychiatry, an independent clinic and supported living provider serving Buckinghamshire and surrounding regions. This venture brought together multidisciplinary expertise to deliver tailored treatment plans, ensuring continuity of care for patients across outpatient and residential settings.

Today, these organisations collectively serve clients from 13+ local authorities and employ over 200 dedicated professionals, reflecting the tangible impact of merging clinical knowledge with entrepreneurial innovation. The pandemic didn't just reveal the fragility of mental healthcare systems; it underscored the potential for transformative change. What began as crisis-driven solutions has now evolved into sustainable models of care, proving that necessity can indeed be the mother of meaningful innovation.

Overcoming Challenges Through Resilience and Strategy

Transitioning from clinician to healthcare entrepreneur while maintaining my clinical practice was an endeavour fraught with challenges, each demanding careful navigation and personal adaptation. The early stages of launching my ventures coincided with a period of significant professional and personal strain: financial pressures from reduced clinical hours, gruelling 70-hour work weeks that tested my time management to its limits, and the psychological adjustment required to reconcile my identity as both a physician and a business founder. Perhaps most daunting was ensuring rigorous regulatory compliance across all operations, particularly in delivering care services where patient safety and legal accountability were paramount.

To surmount these obstacles, I adopted a multipronged approach that blended strategic upskilling with purposeful relationship-building. Recognising the value of cross-sector collaboration, I actively expanded my professional network, forging connections with leaders in healthcare, technology, and business - partnerships that would later prove invaluable in refining service models and scaling operations. Concurrently, I enrolled in an Executive MBA program at Warwick Business School, a decision that equipped me with the frameworks to make informed financial, operational, and strategic decisions while grounding my entrepreneurial vision in academic rigour.

In the absence of prior technical expertise, I embraced a "learning by doing" philosophy; immersing myself in the intricacies of app development for HappiMynd and the operational complexities of care delivery for Symbiosis Care and Bucks Psychiatry. This hands-on experimentation, though demanding, allowed me to iterate quickly and align solutions with real-world user needs. Crucially, my clinical background served as both compass and differentiator: by applying frontline insights to venture development, I ensured that each initiative remained tethered to the realities of patient care rather than abstract business goals.

Perhaps the most transformative step was assembling multidisciplinary teams whose diverse skills complemented my own. From technologists to operations specialists, these collaborators didn't just fill gaps; they elevated the ventures through shared expertise and accountability. Looking back, the challenges that once seemed like roadblocks were in fact catalysts for growth, reinforcing that sustainable entrepreneurship in healthcare isn't about solitary brilliance, but the synergy of continuous learning, adaptive leadership, and teams united by mission. The lessons from this period continue to shape my work: that resilience isn't just endurance, but the wisdom to leverage every setback as a setup for smarter innovation.

Lessons from Scaling Impactful Healthcare Ventures:

The growth of my ventures provided powerful validation for the synergy between clinical expertise and innovative business models. Each initiative demonstrated how physician-led innovation could address critical gaps in healthcare delivery. HappiMynd democratised mental health support through technology, reaching users who might never walk into a clinic. Symbiosis Care redefined supported living by combining clinical oversight with compassionate, community-based care. Bucks Psychiatry not only expanded access to specialist psychiatric services but also created a new standard for integrated, patient-centred care in our region. These successes reinforced my conviction that clinicians possess unique insights to drive meaningful healthcare transformation.

For medical professionals contemplating entrepreneurial paths, I've distilled seven key lessons from my journey:

1. **Leverage Your Clinical Expertise as Competitive Advantage**

   Your medical training provides an invaluable perspective that most entrepreneurs lack. Use your first-hand understanding of patient needs, system inefficiencies, and care pathways to develop solutions with authentic clinical relevance.

2. **Diversify Across Complementary Healthcare Segments**

   By operating across digital health (HappiMynd), residential care (Symbiosis), and clinical services (Bucks Psychiatry), we created an ecosystem where each venture reinforced the others while mitigating sector-specific risks.

3. **Make Regulatory Compliance a Foundational Priority**

   In healthcare, cutting corners isn't an option. Invest early in legal and compliance expertise, particularly for care provision models where patient safety and governance requirements are non-negotiable.

4. **Assemble Teams That Complement Your Skills**

   Recognise what you don't know. Surround yourself with operational, technical, and financial experts who can transform clinical insights into sustainable business models.

5. **Anchor Innovation in Real Societal Needs**

   Let your medical specialisation guide you to unmet needs. The most impactful solutions emerge when clinical knowledge intersects with clear market gaps.

6. **Prepare for the Entrepreneurial Reality**

   The path demands sacrifice - financial uncertainty, relentless hours and constant problem-solving. Build personal and professional support systems before you need them.

7. **Adopt a 'Test and Learn' Mentality**

   Healthcare innovation requires agility. Be prepared to iterate based on real-world feedback while maintaining rigorous clinical standards.

The physician-entrepreneur journey is challenging but profoundly rewarding. By combining medical expertise with business acumen, clinicians can scale their impact far beyond individual patient encounters. Our profession's future needs more doctors willing to bridge these worlds - not just to build businesses, but to reshape healthcare systems for the better. What begins as clinical insight can, with perseverance and the right approach, grow into solutions that transform thousands of lives.

Advice for Trainees

My evolution from psychiatrist to healthcare entrepreneur has been one of the most challenging yet profoundly rewarding experiences of my professional life. This dual-path career has enabled me to amplify my impact on mental healthcare far beyond what would have been possible through clinical practice alone, while simultaneously fulfilling my aspirations for innovation and systemic change. Where I once measured success in individual patient outcomes, I now see it reflected in thousands of lives touched

through scalable solutions and transformed care models.

For the next generation of medical professionals considering this path, I can confidently state that healthcare entrepreneurship represents one of the most exciting frontiers in medicine today. As trainee doctors, you possess not just clinical expertise but also the unique potential to become architects of healthcare innovation. Your medical training has equipped you with something invaluable - an intimate understanding of patient needs and system gaps that most entrepreneurs can only guess at. This positions you extraordinarily well to develop solutions that truly move the needle on global health challenges.

The opportunities before you are vast. Digital health platforms can democratise access to care in underserved regions. Novel care models can address longstanding gaps in service provision. Data-driven solutions can personalise treatment at unprecedented scales. Your specialised knowledge - whether in psychiatry, surgery, paediatrics or any other field - could form the foundation of revolutionary approaches to some of healthcare's most persistent problems.

Yes, the path demands much. You'll need to become comfortable with uncertainty, master new skills outside medicine, and likely face setbacks along the way. The long hours and financial pressures can test even the most dedicated professionals. But I can attest that when you see your ideas translated into real-world solutions that improve patient outcomes at scale, these challenges pale in comparison to the rewards.

What makes this journey uniquely worthwhile is the opportunity to create lasting, systemic change. While clinical practice allows us to heal one patient at a time, healthcare entrepreneurship empowers us to transform entire systems of care. For those with the vision to see beyond traditional career paths and the perseverance to navigate the entrepreneurial landscape, the potential to reshape healthcare has never been greater.

As you consider your own path forward, remember that some of medicine's most important breakthroughs have come from clinician-innovators who dared to think differently. Your medical training has given you both the privilege and the responsibility to improve lives - entrepreneurship simply provides another powerful tool with which to fulfil that mission. The healthcare system needs more doctors willing to bridge these worlds, and the patients we serve will be better for it.

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Functional Neurological Disorders (FND) Emotions Skills Training and Guided Self-help for Improving Symptom Self-Management

K. Saleh, A. Dunlop, R. Faruqui

Background

Neuropsychiatric illnesses are associated with dysfunction in distributed prefrontal, fronto-limbic, and fronto-striatal neural systems that underlie perception, cognition, social interactions, emotion regulation, and motivation [1]. Functional neurological disorder (FND) is a core neuropsychiatric condition and is often characterised by disruptive and disabling neurological symptoms that do not conform to the known pathophysiology of neurological disease [2]. FND frequently presents with comorbid psychopathology; can become chronic, and lead to unemployment and disability for many patients. Psychosocial factors play an important role in the onset and perpetuation of symptoms. FND can present with many symptoms, either in isolation or combined. Common phenotypes include seizures (known as psychogenic nonepileptic seizures [PNES], dissociative seizures or functional seizures [FS]), gait difficulties, weakness, abnormal movements, sensory deficits, and cognitive complaints.

Non-epileptic attack disorder (NEAD) is a more common presentation of FND when people present with what looks like epileptic seizures, but they do not have epilepsy or associated electroencephalogram (EEG) changes or a physiological condition that may mimic seizures, such as syncope [3]. This condition is also known as psychogenic non-epileptic seizures (PNES), psychogenic seizures, hysterical seizures, dissociative seizures and so on. In ICD-10 (1992), they are classified as 'dissociative convulsions', in ICD-11 (2018) as 'dissociative neurological symptom disorder' and in DSM-5 (2013) as 'functional neurological symptom disorder – with attacks or seizures'. The change in terminology over the years reflects the changes in theoretical underpinning and clinical understanding.

NEAD are commonly seen in neurological and accident and emergency (A&E) settings, and up to 50% of emergency admissions to ITU for status epilepticus in some studies are eventually diagnosed as non-epileptic in origin (NEAD). Up to one in five patients in epilepsy clinics present with NEAD and 20–40% of patients in epilepsy-monitoring inpatient units have NEAD. The estimated prevalence of NEAD is 2–30/100,000 in the general population and population incidence has been estimated to be 1.4–4.9/100,000/year. NEAD most commonly presents in young adulthood and in women, but can occur in children, older adults and men.

The aetiology of NEAD, as with functional neurological disorders in general, is thought to be multifactorial [3]. Risk factors include female gender, Migraine, Chronic pain disorders, Hypermobility, childhood abuse and neglect, avoidant coping styles, previous functional disorders and somatisation, and recent stressful events, which may itself be 'organic' – such as a head injury or syncope. In terms of prognosis and outcomes, data suggest that 4 years after diagnosis, approximately a third of patients will be attack-free. However, being attack-free alone does not result in greatly improved levels of psychopathology or recovery of social function, as nearly half of patients who are attack-free are still in receipt of state benefits [3].

Multiple treatments have been trialled for NEAD, including cognitive–behavioural therapy (CBT) (both individual and group-based), paradoxical treatment, psychodynamic therapy, group therapy, family therapy, case management, eye movement desensitisation and reprocessing, acceptance, and commitment therapy [3]. Treatment of any comorbidities is an important part of treatment for NEAD. Although the data is limited by small numbers, comorbid prevalence rates of 63% with affective disorders, 50-60% with Migraine,46% with personality disorders, 32.5% with somatoform disorders, 27% with a chronic pain disorder and 19% with both anxiety disorders and post-traumatic stress disorder (PTSD) have been reported [3].

The management for all patients will naturally be different due to the different nature of their attacks and their individual triggers, but there are important overarching principles. These include making the diagnosis clearly and early (and ideally soon coming off anticonvulsants); biopsychosocial formulation including the role others play in reaction to attacks; psychoeducation about non-epileptic seizures; the management of comorbidities; the development of personal insight and understanding triggers to attacks (which can be achieved by tools such as attack diaries); offering trauma focused therapy if appropriate; and the personal testing and utilisation of different techniques such as grounding, relaxation and mindfulness [3].

FND universally incorporate psychoeducation and treatment rationale. Common elements include symptom tracking (to identify triggers and emotional dysregulation), relaxation/grounding techniques, and targeting trauma, maladaptive thoughts, and dysfunctional behaviours (e.g., avoidance). Most emphasise self-care (sleep, exercise). Some modalities employ unique tools like workbooks, DBT diaries, mindfulness, or behaviour chain analysis [2].

The symptoms of FND are a product of its pathophysiology. The pathophysiology of FND is reflective of dysfunction within and across different brain circuits that, in turn, affects specific constructs. Five constructs that are affected in FND are emotion processing (including salience), agency, attention and predictive processing/inference. Examples of underlying neural circuits include salience, multimodal integration, and attention networks [4]. The symptoms of each patient can be described as a combination of dysfunction in several of these networks and related processes.

Emotion dysregulation represents a core neuropsychological impairment in both primary neuropsychiatric disorders and their frequent comorbidities. Defined as the capacity to adapt emotional states to facilitate goal-directed behaviour, this regulatory function relies on diverse cognitive strategies [5]. While disrupted in functional neurological disorders, emotion regulation is also compromised by neurostructural alterations in classical neuropsychiatric conditions, including Parkinson's disease, Huntington's disease, and post-traumatic frontal lobe syndromes. These emotional disturbances significantly impair multiple functional domains - including social relationships, occupational performance, and daily living activities - often exacerbating and perpetuating neuropsychiatric symptoms [6,7,8]. Consequently, emotion regulation constitutes a critical therapeutic target in neuropsychiatry.

Box 1: Emotion skills for everyday life. A 6 – 8-week course for Neuropsychiatric outpatients

Aims

To improve the quality of life and reduce the distressing symptoms experienced by patients with Neuropsychiatric disorders in East Kent.

Training methods

The key focus of the course is on the acquisition of appropriate emotion regulation skills. Each of the 90-minute to 2-hour sessions includes:

1. A supportive "check in" and/or review of home practice
2. Teaching on the subject/skill introduced
3. The opportunity for practice of the skill in the session
4. Discussion of the content and practice delivered in each session
5. Written handouts or streaming resources (as appropriate) and worksheets/ diaries to support each session and a resource manual given at the end of the course

Course timetable

- Week 1: Understanding emotions and their basic regulation
- Week 2: Surviving crises
- Week 3: Optional – improving distress tolerance
- Week 4: Optional - Problem solving and opposite action
- Week 5: What is Mindfulness
- Week 6: Being right where you are
- Week 7: Introduction to mindful movement
- Week 8: Bringing kindness and compassion into focus

Emotion Regulation Group Therapy in Neuropsychiatric Disorders

Emotion regulation, the capacity to modulate emotional responses to facilitate adaptive functioning, constitutes a fundamental therapeutic target in neuropsychiatric care [9]. As a core component of emotional intelligence, these skills enable individuals to identify, process, and adapt emotional experiences, thereby improving decision-making, stress resilience, and symptom management across both functional and structural neuropsychiatric conditions.

a) Intervention Design

Our Managing Emotions Skills workshops integrate evidence-based techniques from cognitive behavioural therapy (CBT), dialectical behaviour therapy (DBT), and radically open DBT (RO-DBT), delivered through a structured yet engaging pedagogical model. The curriculum progresses from didactic skill-building to experiential practice, emphasising:

- Physiological regulation: Slow-paced breathing and progressive muscle relaxation to reduce baseline arousal and condition adaptive responses to emotional escalation.

- Cognitive-behavioural strategies: Symptom/emotion logging to identify triggers, coupled with fact-checking, problem-solving, and opposite-action techniques to modify maladaptive responses.

- Mindfulness and compassion: Exercises to enhance attentional control, reduce distress, and cultivate self-compassion, adapted from mindfulness-based interventions and compassion-

focused therapy (CFT) to address self-criticism and promote emotional safety.

b) Development and Adaptation

The protocol was developed through multidisciplinary consensus, with content derived from established treatment manuals and tailored for transdiagnostic neuropsychiatric applicability by AD, a psychiatric nurse specialist with advanced training in CBT, CFT, RO-DBT, and adapted mindfulness-based therapies. The intervention incorporates elements from our empirically supported group psychoeducation programs for NEAD and FND, as well as clinical insights from delivering individualised mindfulness training to neuropsychiatric patients. A targeted literature review informed the inclusion of brief, non-standard mindfulness techniques suitable for neurological populations.

a) Outcome Monitoring

Treatment efficacy will be evaluated using a battery of psychometric measures assessing emotion regulation pre- and post-intervention, alongside patient satisfaction metrics to ensure clinical relevance and acceptability.

Conclusion

There seems to be positive feedback so far, from service users and carers, to the proposed content and its impact on symptoms when delivered either individually or as part of other group treatments in our service. Clinicians have observed clinical improvements in mood, impulsivity, emotion management and in functional neurological symptoms (especially movement symptoms & NEAD) when these skills have been taught to patients with both structural and functional neurological disorders. We plan to write this up in detail when the project is completed.

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Why University Academia Fails to Deliver a Socially Optimal Output for Society: A Microeconomic Perspective

Musa Sami and Naveed Kausar

Universities have long been considered the bedrock of knowledge production and engines of innovation. Their contributions to scientific discovery, cultural enrichment, and policy development underscore their vital role in modern society. However, an emerging body of criticism suggests that university academia, especially in high-stakes fields like medicine, engineering, and public health, fails to deliver a level of scientific output that is socially optimal. Scientific advancements that could enhance well-being, reduce inequality, or accelerate innovation are often delayed, underfunded, or marginalised.

In this essay, we examine this dysfunction through a microeconomic lens. By conceptualising science as a good traded in a market regulated by prestige instead of price, we can better understand the structural inefficiencies that limit academia's societal impact. In particular, we explore the supply and demand dynamics of academic science, the distortions caused by prestige, and the institutional barriers that reinforce underproduction. Finally, we offer policy recommendations aimed at fostering a more inclusive, transparent, and efficient academic ecosystem.

In classical microeconomics, markets are driven by the forces of supply and demand. Consumers derive diminishing marginal utility from additional units of a good, leading to a downward-sloping demand curve. Producers, on the other hand, are more inclined to supply a good when they can command a higher price, resulting in an upward-sloping supply curve. The market-clearing equilibrium, the point where these curves intersect, reflects the quantity and price of goods that maximise welfare, assuming perfect competition. This model, although abstract, offers a valuable heuristic when applied to non-traditional goods such as academic research.

In academia, monetary gain is often secondary to the pursuit of prestige. Prestige encompasses recognition by peers, influence over policy and discourse, access to better funding and career advancement, and inclusion in elite networks. It is the primary "currency" traded in the academic marketplace. Scientific output, particularly in the form of peer-reviewed publications, conference presentations, patents, and grants, represents the good being exchanged. Just as consumers become saturated with additional units of a physical good, the public and indeed academic audiences themselves, experience diminishing marginal prestige with each successive contribution. The demand curve for scientific output thus reflects a diminishing societal and institutional appetite for new research, particularly in saturated or over-specialised areas.

On the supply side, prestige incentivises individuals to pursue academic careers, invest in long periods of training, and contribute novel ideas. As prestige becomes more attainable or better rewarded, more individuals enter the market. This produces an upward-sloping supply curve, as the number of researchers willing to produce scientific work increases with the perceived rewards. In a perfectly competitive system, where access to prestige is not unduly constrained and resources are distributed based on merit, the equilibrium would be reached when the marginal cost of producing an additional unit of knowledge equals the marginal prestige or utility derived from it.

However, modern academia deviates sharply from this idealised model. A number of market failures and structural inefficiencies skew

both the supply and demand of academic research, preventing society from reaching an optimal output. One of the most significant of these is the presence of high barriers to entry. The path to becoming an academic is notoriously difficult. Individuals must often endure over a decade of precarious employment, accumulating credentials, and competing for limited tenure-track positions. Even after attaining these positions, researchers face bureaucratic hurdles in acquiring funding, publishing work, and gaining recognition. This restricts the inflow of potentially innovative scientists and narrows the diversity of perspectives within the academy. Dasgupta and David [1] argue that science functions under a hybrid incentive structure, part competitive, part communal, which can paradoxically limit innovation when institutions become overly rigid.

Furthermore, success in academia is disproportionately determined by network effects. Connections to prominent scholars, elite institutions, and prestigious journals significantly affect one's chances of securing grants, publishing in high-impact venues, or receiving job offers. These advantages tend to reinforce themselves over time; a dynamic Robert Merton [2] described as the “Matthew Effect,” where the rich get richer. The result is a skewed distribution of recognition and resources. Those already embedded in influential networks can consolidate power, marginalising newcomers and non-traditional voices. The feedback loops generated by these networks distort the market for science, concentrating prestige in the hands of a few rather than distributing it according to merit or social relevance.

The concentration of power in academia also creates monopolistic and oligopolistic structures. In certain fields, a handful of elite labs or senior scholars wield disproportionate influence over funding decisions, journal gatekeeping, and the direction of research agendas. These actors may intentionally limit the production of high-quality but low-prestige work, such as replication studies, interdisciplinary approaches, or socially controversial topics, in order to safeguard their own status. This mirrors the behaviour of monopolists in economic theory, who restrict output to maximise their own utility rather than social welfare. The ongoing replication crisis in psychology and biomedical research, well-documented by Ioannidis [3], underscores this failure. Many published findings cannot be reproduced, yet the academic system disincentivises the validation of results in favour of novelty.

Another systemic distortion arises from the misalignment of incentives. Universities often evaluate researchers based on crude bibliometric indicators such as the number of publications, citations, or journal impact factors. This "publish or perish" mentality pushes academics to maximise output quantity rather than quality. The result is a proliferation of minimally incremental studies, questionable research practices, and salami-slicing of findings to inflate publication counts. The prestige earned by these publications does not necessarily correspond to their societal value, resulting in a classic negative externality, where private gains come at a cost to public understanding and trust [4].

Given these failures, some have advocated for increasing public demand for science by enhancing scientific literacy, engaging with media, and raising awareness of research contributions. In microeconomic terms, this approach aims to shift the demand curve outward, thereby raising the equilibrium quantity of research and the prestige associated with it. While this could help expand funding, create more academic positions, and improve societal appreciation for science, it has inherent limitations. For one, prestige is a relative and finite resource. Its value depends on exclusivity; the more researchers producing high-quality work, the more difficult it becomes for any individual to stand out. This inelasticity constrains the extent to which demand-side interventions alone can raise output. Additionally, if systemic bottlenecks, such as grant monopolies and journal hierarchies, remain in place, increased demand will primarily benefit those already entrenched in the system, exacerbating inequality rather than alleviating it.

To achieve a socially optimal academic system, comprehensive structural reform is needed. One important step is the decentralisation of funding mechanisms. By reducing reliance on elite gatekeepers and introducing participatory grant making, citizen review panels, or algorithmic allocation models, the distribution of research resources could become more democratic and less subject to institutional bias. Research by Lee et al [5] highlights how conventional peer review processes are vulnerable to implicit bias, conservatism, and nepotism, suggesting the need for alternative models.

Equally vital is the cultivation of a research culture that values replication, null results, and transparency. Journals and funding bodies should be encouraged to support studies that challenge prevailing paradigms or confirm earlier findings, even when such work lacks immediate novelty. This would not only improve the reliability of scientific knowledge but also shift incentives toward socially useful, if less glamorous, projects. Flattening academic hierarchies can further support this shift. Moving away from impact-factor-based assessments and toward holistic evaluations of research relevance, collaboration, and community engagement would reduce the prestige bottleneck and encourage broader participation.

The academic ecosystem must also embrace a pluralistic understanding of scholarly impact. Teaching, science communication, and policy advising are often undervalued relative to publications, despite their direct societal relevance. Broadening the definition of academic success to include such contributions would empower a wider array of scholars and enhance the utility of university research for the public. Finally, promoting open science and open access publishing would dismantle the information silos that prevent public access to taxpayer-funded research. By eliminating paywalls and encouraging data sharing, knowledge becomes a public good in the truest economic sense - non-rivalrous, non-excludable, and universally beneficial.

In conclusion, while the microeconomic model offers a simplified abstraction of academic production, it reveals important insights into the failures of university academia to deliver socially optimal outcomes. Prestige, like price, can serve as a powerful incentive. But when prestige is hoarded, distorted, or divorced from societal value, the system ceases to function efficiently. Addressing these structural failures requires both internal reforms within academic institutions and external pressure from policymakers, funders, and the public. A more open, equitable, and productive academia is not just desirable but necessary to meet the complex challenges of our time.

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