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From Bench to Byte: Small Molecule Development for Alzheimer's Disease in the AI Era

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Abstract:

Introduction:

Alzheimer's disease (AD) is becoming a significant global health problem that requires urgent and effective treatments. In the past, drug development for AD has faced high failure rates, with most clinical trials not providing effective treatments. This report reviews the literature on small molecules developed or in development for AD. It highlights the current state, key drug targets, and the role of artificial intelligence (AI) in speeding up discovery and improving drug design.

Objective:

The pipeline for AD therapies shows a shift from focusing only on amyloid-beta to a broader, multi-target approach, where small molecules make up a large part of therapies aimed at the disease.

Methods:

Repurposed small molecules are becoming more popular because they offer a safer and cheaper way to develop new treatments. The shortcomings of current symptomatic treatments highlight the need for new agents that can modify the disease. New strategies focus on changing basic cellular processes, like lysosomal function and microglial activation. These processes can affect multiple disease-related issues at once.

Discussion:

AI technologies are changing drug discovery at every stage, from identifying targets and optimizing leads to predictive modeling and drug repurposing. This addresses the long timelines, high costs, and low success rates that have troubled the field. Based on these insights, new small molecule designs should focus on AI-driven rational poly pharmacology, precise adjustment of microglial states, restoration of lysosomal balance, and targeting of cellular aging.

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Conclusion:

This present review directs the more effective, diverse, and potentially personalized treatments for AD over AI.

1. Introduction to Alzheimer's Disease and the Urgent Need for Therapeutics

1.1. Pathophysiology and Global Burden of AD

Alzheimer's disease is a relentless, progressive brain disorder that seriously affects cognitive skills, memory, independence, and behavior. It mainly impacts people in their mid-60s and older [1]. The main features of Alzheimer's are the buildup of amyloid-beta (Aβ) peptides outside cells, which form plaques, and the clumping of hyperphosphorylated tau proteins inside cells, leading to neurofibrillary tangles (NFTs) [2]. These protein clusters do not just sit idly; they interfere with normal brain function and eventually cause widespread cell death. A key part of Alzheimer's progression is its long preclinical stage. During this time, amyloid plaques and NFTs can be in the brain for years or even decades before any noticeable symptoms appear. This long asymptomatic period poses a significant diagnostic challenge and provides an important opportunity for treatment, possibly before permanent damage happens [3].

The global prevalence and economic impact of Alzheimer's disease (AD) are increasing rapidly. Right now, AD affects about 5.1 million people in the United States and 13 million worldwide. Projections suggest these numbers could rise to 13 million in the US and over 100 million globally by 2050 [4]. The financial toll is serious; the cost of AD care in the US is estimated at $267 billion each year. This amount could exceed $1 trillion annually by 2050 if we don't develop effective new treatments. As the most common type of dementia, AD is already the second leading cause of death in Australia and the top cause of disease burden in people over 65 [5]. The magnitude of this looming crisis highlights the urgent need for new therapies that can delay the onset, slow the progression, or ideally, reverse the effects of this devastating disease [6].

The path to developing drugs for Alzheimer's disease has been very difficult, with a long history of serious setbacks. Until recently, no new drugs had been approved since 2003 [7]. The overall failure rate for new treatments has been over 99%. A close look at drug development from 2002 to 2012 highlights this high drop-off rate. Traditionally, diagnosing Alzheimer's has had low accuracy, and there have been few reliable biomarkers for a definitive diagnosis. This lack of precision has made it hard to select patients for clinical trials, often leading to diverse groups being enrolled. This can obscure real treatment effects [8].

Small molecules remain central to Alzheimer's disease (AD) drug development, comprising 43% of the 2025 pipeline, with many being repurposed drugs [8]. Their advantages—oral bioavailability, BBB penetration, and ease of manufacturing—make them well-suited for chronic conditions like AD. A shift away from amyloid-centric strategies toward multi-target approaches reflects a deeper understanding of AD's complexity [9]. Repurposed small molecules, representing 43% of disease-targeted therapies, offer a cost-effective path forward by leveraging known safety profiles, reducing development risk, and addressing the economic urgency posed by the rising burden of AD care [10].

2. Promising Small Molecule Therapies for Alzheimer's Disease (AD) in 2025

Despite the formidable challenges, small molecules remain a cornerstone of Alzheimer's disease drug development. The 2025 pipeline demonstrates their prominent role, with small molecule Disease-Targeted Therapies (DTTs) accounting for a

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significant 43% of the total pipeline [11]. These advantages include oral bioavailability, the ability to effectively cross the blood-brain barrier (BBB) to reach central nervous system targets, and more straightforward chemical synthesis and manufacturing processes.

Several repurposed small molecules are set to complete Phase 3 trials in 2025 (Table 1):

• Semaglutide (GLP-1 agonist): Originally developed for diabetes and obesity, it is now in Phase 3 for Alzheimer's disease.

• Nilotinib (Tyrosine kinase inhibitor): Repurposed from oncology.

• Dextromethorphan + Quinidine, Xanomeline + Trospium, Dextromethorphan + Bupropion: Combination therapies in Phase 3.

• Nabilone: Synthetic cannabinoid in Phase 3.

- Additional Phase 3 candidates include:

• Piromelatine, Masupirdine, AR1001, Valiltramiprosate, Rotigotine + Rivastigmine.

• NU-9: This targets lysosomal protein clearance through cathepsin B and shows a lasting reduction in amyloid and inflammation in preclinical models.

• Simufilam: Currently in Phase 3, it targets tau and neuroinflammation.

• CT1812 (Phase 1): This blocks the toxic Aβ-receptor interaction.

• ALZ-801 (Phase 2): This inhibits Aβ aggregation.

• Posiphen (Phase 2): This blocks APP translation.

• Varoglutamstat (Phase 2): This prevents pGlu-Aβ formation.

• ALX-001 (Phase 2): This disrupts harmful Aβ oligomer-prion-glutamate receptor binding.

Unlike FDA-approved monoclonal antibodies, such as Aducanumab and Lecanemab, the newer small molecules focus on more than just Aβ. They also target tau, inflammation, and cellular cleanup processes [Table 1]. This marks a shift towards a broader, multi-targeted approach that reflects the complex nature of Alzheimer's disease [12].

Table 1: Key Small Molecule Candidates in AD Clinical Trials

Drug Name	**Primary Target(s)**	**Mechanism of Action (MoA)**	**Clinical Trial Phase**	**Repurposed**	**Source IDs**
NU-9	Lysosomal function, Cathepsin B	Rescues cellular pathway for toxic protein clearance, reduces Aβ build up and inflammation	Preclinical/Research	No	13
Simufilam	Tau pathology, Neuroinflam mation	Diminishes tau build up, decreases neuroinflammation	Phase III	No	14
Semaglutide	GLP-1 receptor	GLP-1 agonism (meta	Phase III	Yes	15

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		bolic modulation)			
Nilotinib	Tyrosine Kinase	Tyrosine kinase inhibition	Phase III	Yes	16
Dextromethorphan + Quinidine	NMDA receptor, CYP2D6	Combination for cognitive enhancement/NPS	Phase III	Yes	17
Xanomeline + Trospium	Muscarinic acetylcholine receptors	Combination for cognitive enhancement/NPS	Phase III	Yes	18
CT1812	Oligomeric Aβ-receptor interaction	Prevents Aβ-induced synaptic toxicity	Phase I	No	19
ALZ-801	Aβ aggregation	Disrupts/hinders Aβ aggregate formation	Phase II	No	20
Posiphen	Amyloid Precursor Protein (APP)	Hinders APP translation, reduces Aβ generation	Phase II	No	21
Varoglutamstat	pGlu-Aβ formation	Prevents formation of toxic Aβ variant	Phase II	No	22
ALX-001	Aβ oligomers, cellular prion protein, glutamate receptor	Blocks pathogenic binding, reduces synaptic toxicity	Phase II	No	23
Nabilone	Cannabinoid receptors	Symptomatic /NPS (synthetic cannabinoid)	Phase III	Yes	24
Piromelatine	Melatonin/Serotonin receptors	Sleep regulation, cognitive enhancement	Phase III	No	25

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Masupirdine	Histamine H3 receptor	Cognitive enhancement	Phase III	No	26
AR1001	Unknown (DMT)	Disease-modifying therapy	Phase III	No	27
Valiltramiprosate	Aβ clearance	Facilitates Aβ elimination	Phase III	No	28
Rotigotine + Rivastigmine	Dopamine/Serotonin receptors, AChE	Combination for motor/cognitive symptoms	Phase III	Yes	29
Dextromethorphan + Bupropion	NMDA receptor, Dopamine/Norepinephrie reuptake	Combination for NPS/cognitie enhancement	Phase III	Yes	30

3. Core Pathological Targets

There has been a greater understanding of Alzheimer disease (AD) pathophysiology, as drug development is beginning to move beyond an amyloid-centric approach. Treatments will be more effective when they can simultaneously address multiple, connected mechanisms contributing to the disease (Table 2). The amyloid cascade hypothesis suggests that abnormal Aβ production and increased clearance magnify the disease process in AD [31]. Aβ oligomers are believed to be primarily responsible for the neurotoxicity observed in AD and their role as key proximal driver of neuronal damage is supported by evidence from both animal and human studies. There are small molecules designed to inhibit Aβ production on the market, including BACE inhibitors (Verubecestat, Lanabecestat) with the exception of a few, which failed in development without significant evidence to help discriminate which drug class was involved [32]. A few even worsened cognition levels and presumably had off-target effects. While some gamma-secretase inhibitors such as Semagacestat and Avagacestat were halted in development despite their potential value in treating AD, the adverse side effects, such as severe skin cancer, outweighed the potential benefits derived. There are a number of modulators (e.g., NIC5-15, Phase II) still in development. Posiphen (Phase II) is a different mechanism because it lowers levels of Aβ by reducing APP translation. Multiple compounds aim to prevent Aβ aggregation or enhance Aβ clearance. For example, ALZT-OP1 (Phase III), Bexarotene, ALZ-801, PBT2, Varoglutamstat (all Phase II). The only reasonable way for Aβ clearance is through the action of microglia to clear Aβ. The use of NU-9, a newer agent which enhances lysosomal Aβ clearance, could be viable way for enhancing Aβ clearance in the brain through actions of lysosomal and cathepsin B-mediated Aβ degradation. Despite the prominence and influence of the amyloid hypothesis on treatments for AD, practically all small-molecule, Aβ-targeting drugs, are failed drugs - and continuing with the notable exception of monoclonal antibodies which are capable of clearing Aβ, whether it has functional benefit for cognition level remains [33].

4. Emerging and Complementary Targets

i) Neuroinflammation: Chronic neuroinflammation resulting from activated microglia is a critical

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characteristic of AD. These cells are the brain's resident immune cells and not only function in amyloid clearance but are also involved in modulation of external signals and synaptic reorganization and regulation of inflammation. Small-molecule targeting strategies for neuroinflammation focus on modulating microglial function on activation states, the most notable of which are M1 (pro-inflammatory) and M2 (anti-inflammatory) [34,35]. It is hypothesized that switching microglial state from M1 to M2 may be beneficial and lead to decreased neurodegeneration. Anti-inflammatory compounds such as minocycline allow for switching microglial activation state to M2 [36].

ii) Cytokine Modulators: The pro-inflammatory cytokines IL-1β and TNF-α should be avoided and pharmacological agents that work as TNF-α inhibitors (etanercept) may impact microglial mediated neuroinflammation [37].

iii) NLRP3 Inflammasome Inhibitors: The NLRP3 inflammasome is primed by NF-κB and activates production of pro-inflammatory cytokines. Inhibitors like MCC950 may have a positive effect by decreasing neuroinflammation and Aβ accumulation [38].

iv) CSF1R Inhibition: CSF1R inhibition may inhibit non-plaque associated microglia better than plaque associated microglia leading to robust decreases in tau pathologies in mouse models of AD [39].

v) Metabolic Modulators: Metformin acts through the activation of AMPK and inhibition of mTOR, and metformin in effect can modulate an anti-inflammatory microglial phenotype and induce phagocytosis. Metformin administration to mouse models of AD reduced both Aβ deposits and tau pathologies. Several other AI-scanned novel molecules presented with promise for the treatment of neuroinflammatory processes [40].

Table 2: Alzheimer's Disease Drug Targets and Corresponding Candidates

Target Pathway/Mechanism	**Specific Target (if applicable)**	**Small Molecule Candidates**	**Clinical Trial Phase/Status**	**Key Mechanism/ Note**	**Source IDs**
Acetylcholine Deficiency	Acetylcholinesterase (AChE)	Donepezil, Rivastigmine, Galantamine	Approved	AChE inhibition, increases ACh levels	41
Glutamate Excitotoxicity	NMDA Receptor	Memantine	Approved	NMDA receptor antagonism	42
Amyloid-beta production	BACE1	(Verubecestat, Lanabecestat - failed)	Failed Phase	BACE 1	43
Amyloid-beta production	Gamma-secretase	NIC5-15, (Semagacestat)	Phase II, Failed	Gamma-secretase Modulation Inhibition	44
Amyloid-beta Production	APP Translation	Posiphen	Phase II	Hinders APP translation	45

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Amyloid-beta Aggregation/ Clearance	Aβ Aggregation	ALZ-801, PBT2, Varoglutamstat	Phase II	Disrupts/ Hinders Aβ aggregation	46
Amyloid-beta Clearance (Lysosomal)	Lysosomes,	NU-9	Preclinical/ R	Rescues protein clearance pathway	47
Tau Pathology	Tau build up, Neuroinflammation	Simufilam	Phase III	Diminishes tau buildup, decreases Neuroinflammation	48
Neuroinflammation	Microglial Activation (M1-M2 shift)	Minocycline	Research	Shifts microglia to anti-inflammatory state	49
Neuroinflammation	NLRP3	MCC950	Research	Reduces neuroinflammation ,Aβ accumulation	50
Neuroinflammation	Metabolic Modulation	Metformin	Research	Promotes anti-inflammatory microglial phenotype, enhances phagocytosis	51
Neuroinflammation	Janus Kinase (JAK)	JAK inhibitors (identified by AI)	Research	Anti-inflammatory potential	52
Synaptic Dysfunction	Aβ oligomer-receptor interaction	CT1812	Phase I	Prevents Aβ-induced synaptic toxicity	53
Synaptic Dysfunction	Glutamate Receptor	ALX-001	Phase II	Blocks pathogenic binding of Aβ oligomers	54
Synaptic Dysfunction	Glucose Utilization	Nasal insulin	Phase III	Restructuring of synapses, glucose utilization	55
Oxidative Stress/ Mitochondrial Dysfunction	Mitochondrial efficiency	Coenzyme Q10 (CoQ10)	Research	Improves mitochondrial efficiency, reduces ROS	56
Oxidative Stress/ Mitochondrial	NAD+ synthesis	Nicotinamide Riboside (NR)	Research	Supports mitochondrial function,	57

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Dysfunction				modulates microglia	
Oxidative Stress/ Mitochondrial Dysfunction	Autophagy, Neuroinflammation	Spermidine	Research	Enhances autophagy, reduces neuroinflammation	58
Oxidative Stress/ Mitochondrial Dysfunction	Antioxidant defense	N-Acetylcyst eine (NAC), Taurine, Thiamine	Research	Bolsters antioxidant defenses, protects neurons	59
Oxidative Stress/ Mitochondrial Dysfunction	Glucose Metabolism	2-Deoxy-D-glucose (2-DG), Resveratrol	Research	Limits pro-inflammatory responses, reduces oxidative stress	60
Oxidative Stress/ Mitochondrial Dysfunction	Lipid Metabolism	Omega-3 Fatty Acids, Fenofibrate	Research	Anti-inflammatory, enhances Aβ phagocytosis	61
Lysosomal Function	TFEB Activation	Trehalose, Spermidine	Research	Enhances lysosomal function, autophagy	62
Cellular Senescence	Senescent cells/ pathways	(Senolytics/Senomorphics - general class)	Research	Targets senescent cells, anti-aging effect	63
Neuropsychiatric Symptoms (NPS)	Various (combination)	Dextromethorphan + Bupropion, Nabilone	Phase III	Ameliorates NPS	64
Cognitive Enhancement	Various (combination)	Dextromethorphan +Quinidine, Xanomeline+ Trospium, Piromelatine, Masupirdine, Rotigotine+ Rivastigmine	Phase III	Improves cognition	65

5. Rationale for Polypharmacology

Alzheimer's disease is increasingly seen as a complex disorder with many interacting harmful processes. The past failures of single-target approaches, especially those focused only on amyloid, have shown that a single-strategy approach is unlikely to produce meaningful clinical benefits. This understanding comes from the consistent problems with single-target methods, particularly the long-standing amyloid-beta monotherapy, along with the recognition of Alzheimer's as a complex disease. This suggests that using multiple drugs is not just an option but a

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necessary step in developing Alzheimer's treatments. The clear statement that a single-strategy approach is unlikely to be enough highlights this change in thinking. This view directly results from the challenges of earlier failures and emphasizes that effective Alzheimer's treatment must tackle several related disease pathways at once. Thus, future therapies will likely need to be multimodal, addressing multiple pathways together for lasting and significant outcomes. Artificial intelligence is playing an important role in this transition. AI-driven techniques are opening up new ways to create multi-target medications that can improve treatment effectiveness and possibly minimize side effects by approaching the disease in a comprehensive manner.

6. Current strategies for developing multi-targeted agents for AD

This simple approach to poly pharmacology combines two or more small molecules, often with different mechanisms, to achieve synergistic effects or lessen side effects. Examples currently in Phase 3 clinical trials include: Dextromethorphan plus Quinidine. Xanomeline plus Trospium. Rotigotine plus Rivastigmine. Dextromethorphan plus Bupropion. A more advanced approach involves designing or finding single small molecules that naturally modulate multiple targets or essential cellular processes, leading to broad therapeutic effects [66]. NU-9, While not an explicit combination is a single small molecule with a wide impact. It acts on a "common mechanism" that affects different proteins in various neurodegenerative diseases like ALS and AD. By rescuing the cellular pathway responsible for clearing toxic protein clumps, which involves lysosomes and cathepsin B, NU-9 effectively targets multiple forms of protein aggregation, including amyloid beta in AD and other mutated proteins in ALS. Another example, Metformin activates AMPK and inhibits mTOR, encouraging an anti-inflammatory microglial phenotype and improving phagocytic function. This action reduces both Aβ deposits and tau pathology. This illustrates how one small molecule can affect multiple hallmarks of AD [67]. While, Spermidine a polyamine boosts autophagy and reduces neuroinflammation. It addresses oxidative stress while improving microglial function. While explicit combination therapies are valid for multi-targeting, the mechanisms of NU-9 and Metformin suggest a deeper and potentially more effective form of poly pharmacology. This form targets essential cellular processes such as lysosomal function, protein clearance, or metabolic regulation, impacting multiple downstream pathologies like Aβ and tau aggregation, neuroinflammation, and oxidative stress [68,69]. This approach is not only more elegant but also potentially more effective than merely combining two drugs. It tackles root causes that contribute to several disease hallmarks. This suggests a future where small molecules are designed to modulate key cellular homeostatic mechanisms, leading to widespread therapeutic benefits, rather than just targeting specific pathological proteins in isolation [70].

7. Artificial Intelligence in Alzheimer's Drug Development:

A Revolutionary Force Artificial Intelligence (AI), encompassing machine learning (ML) and deep learning (DL), has emerged as a revolutionary force in drug discovery, particularly for Alzheimer's Disease (AD). With the integration and interrogation of enormous genomic, chemical, and clinical data, AI expedites the otherwise sluggish and expensive drug development process, which takes 10–15 years and costs up to $5.6 billion per drug [71]. AI resolves crucial AD drug development issues in target identification, lead compound optimization, predictive modelling, drug repurposing. It transforms drug discovery from hypothesis- to data- and patient-centric strategies, particularly critical for AD's multifactorial, complex pathology [72].

1. Target Identification & Validation

AI analyzes high-throughput omics data (genomic, proteomic, metabolomic) to discover new genes/proteins associated with AD. NETTAG:

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Created by Cleveland Clinic, it is a deep learning platform combining genetic information and protein-protein interaction networks, finding 156 AD-related genes. Multi-omics + GWAS integration: Discovered 103 AD risk genes and repositioned drugs such as pioglitazone [73].

2. Lead Discovery & Optimization:

AI facilitates improvement at every step, Virtual Screening aids in fast screening of chemical libraries with docking and energy models. Whereas, QSAR Modeling works with deep learning models forecast compound activity from chemical structures directly. The Generative AI the alternate sorce generates new molecules by employing GANs and neural networks. In pharmacophore modelling, dyphAI integrate ligand- and complex-based models to discover effective inhibitors (e.g., AChE) [74].

3. Predictive Modeling

AI forecasts pharmacokinetics and pharmacodynamics, ADME properties (Absorption, Distribution, Metabolism, Excretion), Toxicity, blood-brain barrier permeability, Side-effect profiling based on adverse event and drug–protein interaction databases [75].

4. Drug Repurposing

AI speeds up new indication discovery for already approved drugs, DREAM Study, a key monitor, identified 35 FDA-approved drugs for 20 AD-related pathways, narrowing to 15 leads. Gemfibrozil: Identified through NETTAG as lowering AD risk. DRIAD Platform ranked JAK inhibitors with high AD-relevant activity [76].

Some Key AI Tools in AD Research

AlphaFold: Uses 3D protein structure prediction for accurate target identification [77].

Exscientia's Centaur Chemist: AI platform that resulted in several clinical candidates, including DSP-0038 for AD psychosis [78].

Insilico Medicine's PandaOmics: Identified top AD-related proteins such as MARCKS, CAMKK2, and p62 [79].

NETTAG: Harmonizes network topology and genetics towards repurposing and target identification [80].

DyphAI: Unites AI with pharmacophore modeling towards virtual screening of enzyme inhibitors (Tabl3 3).

Table 3: AI platform and tools used in various stages of Drug discovery in Alzheimer's Disease

Development Stage	**AI Tool / Platform**	**Primary Function**	**Notable Example(s)**
Target Identification & Validation	Panda Omics (Insilico) disease-relevant targets	Integrates multi-omics /text to prioritize	Identified phase-separation targets MARCKS, CAMKK2, p62 [81,82]
	NETTAG (Cleveland Clinic)	Network-topology DL on genetic and PPI data	Prioritized 156 AD risk genes; flagged repurposing hits like gemfibrozil [83]
	AlphaFold / AlphaFold 3 (DeepMind / Isomorphic Labs)	Predicts protein/complement structures for drug targeting	Accurate 3D structures, complex modeling aids target selection
Lead Discovery & Optimization	Chemistry42 (Insilico)	De novo molecule design via generative AI	AI-designed hits progressed into

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			preclinical pipelines [85]
	dyphAI	Ensemble pharmacophore + ML screening	Virtual screening against AChE; identified 18 novel inhibitors [86]
Predictive ADME/ Tox Modeling	(Various ML/DL QSAR & ADMET tools)	Predicts drug-like properties, BBB permeability, toxicity, side effects	Frameworks highlighted in review [87]
Drug Repurposing	NETTAG, Dream, DRIAD	Leverages omics, EHR, network data	NETTAG → gemfibrozil; Dream → 35 FDA drugs across 20 pathways; DRIAD → JAK inhibitors [88]
Structural Biology Support	AlphaFold / AlphaFold 3	High-accuracy structure prediction for proteins, complexes	Open platform enabling drug design pipelines [89]

8. Conclusions and Future Directions

The landscape of Alzheimer's disease drug development is undergoing a radical transformation. Instead of focusing primarily on amyloid-beta, the highly simplistic single-target approaches, to re-emerge in this new chapter of AD drug development, it is important to acknowledge the wholly confirmed realization that the hallmark characteristics of AD are a multi-factorial disorder, and thus require therapeutic approaches that are multi-modal. This evolution is taking place and becoming evident in the increasing multiplicity and dimensions of our small molecule pipeline that is now specifically targeting a broad spectrum of pathogenic mechanisms, types of toxicity, both chronic and acute, including tau pathology, neuroinflammation, synaptic dysfunction, oxidative stress, mitochondrial dysfunction, lysosomal dysfunction, and cellular senescence. Small molecules have become a highly versatile resource as they have significant advantages including oral bioavailability and the ability to penetrate the blood-brain barrier. Therefore, as we have described, the hard focus on drug repurposing for small molecules is a pragmatic and effective approach to systematically reduce risk, costs and development timelines which ideally aligns development with market demands, all the while addressing key pain-points in drug development; research attrition rates of around 70% based on developer experience and acceptance develop timelines that can go to over 5000 days! Artificial intelligence has acted as a catalyst, and completely changed the landscape of drug discovery. For example, omics data by itself is a substantial evolution in drug discovery, where AI and/ or machine learning have further accelerated our ability to utilize omics data, identify new targets, optimize lead compounds, more accurately predict ADME/toxicity profiles or enable drug repurposing in a highly efficient manner. AI represents a major evolution in its ability to sift through extensive, high dimensional datasets.

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Conflict of Interest:

The authors declare there is no conflict of interest for the present article submitted.

Authors Contribution

All the authors contribute equally in the writing the manuscript

1. S.Amuthalakshmi - The conception and design of the study, and acquisition of data,
2. C.N.Nalini - analysis and interpretation of data.
3. Ramalakshmi & Dhatchana Moorty - Drafting the article or revising it critically for important intellectual content.
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