Dynamic Functional Connectivity as a Candidate Neuroimaging Biomarker Feature for Mild Cognitive Impairment and Alzheimer's Disease Progression

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Network neuroscience suggests that aspects of neurodegenerative disease progression can be explained by changes in how brain regions communicate, rather than solely by local neuronal damage. Traditional molecular models focused on amyloid and tau pathology have limited ability to explain variability in symptom timing, rate of cognitive decline, and cognitive reserve. The goal of this narrative review is to evaluate whether dynamic functional connectivity (DFC), as evidenced by resting-state fMRI, may serve as a candidate biomarker feature for mild cognitive impairment (MCI) and Alzheimer's disease (AD). A peer-reviewed literature search was performed using PubMed, Google Scholar, Web of Science, and ScienceDirect with keywords including time-varying connectivity, dynamic functional connectivity, resting-state fMRI, MCI, AD, default mode network, graph-theoretical methods of connectivity assessment, and machine-learning algorithms used for classification of neuroimaging data. Studies were selected based on assessment of dynamic brain connectivity, disease stage, degree of cognitive decline, and/or comparison with static connectivity, structural MRI, or molecular biomarkers. Although this review focuses mainly on studies involving MCI and AD, broader claims about neurodegenerative disease are treated cautiously. Across the studies examined within this review, network flexibility, time-dependent variability, and global efficiency of brain networks were frequently reported as reduced in individuals diagnosed with MCI or AD. The existing research base is insufficient to support dynamic functional connectivity as a validated biomarker that can be reliably used clinically. Before DFC-derived measures can be employed clinically, longitudinal validation, methodological standardization, independent replication in external samples, and comparisons with well-established biomarkers will be needed.

Keywords: Dynamic functional connectivity, Resting-state fMRI, Alzheimer's disease, Mild cognitive impairment, Default mode network, Connectomics, Graph theory, Machine learning neuroimaging

Introduction

Neurodegenerative conditions are being viewed increasingly as a network disorder of large scale communication among neurons; thus not solely as local damage to one area of the brain[1,2]. The classical pathological framework has focused upon various molecular hallmarks of disease, including amyloid-β deposition and neurofibrillary tangle formation with tau[3,4]. Molecular pathology alone does not account for the variation in how quickly patients decline cognitively, how variable symptoms can be, and how different each patient's rate of disease progression is[5]. Recent neuroimaging studies support the viewpoint that neurodegenerative conditions disrupt progressively distributed cortical networks involved in integrating information from multiple sensory inputs[6].

It is proposed in current studies of modern connectomes that many neurodegenerative disorders selectively disrupt hub areas where communication across variously dispersed networks is coordinated[7]. The structural complexity and metabolically demanding role of hub regions may make them especially vulnerable within network-level disease models. These vulnerabilities may interact with cellular and molecular disruptions associated with neurodegenerative disease, including oxidative stress, mitochondrial dysfunction, and abnormal protein accumulation[8]. Rather than indicating that hub regions are always the first or primary sites of brain damage, this interpretation suggests that their high connectivity and metabolic burden may make them more vulnerable within the broader process of disease progression. Several large-scale neuroimaging collaborations have shown that those brain regions that have a high degree of "centrality" or importance in terms of their role in the connectome tend to experience the earliest signs of disease-related pathology[9]. This supports the hypothesis that some neurodegenerative disease processes may progress along the organizational structure of the brain's

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connectome [10].

Hub vulnerability and white matter damage are related because highly connected hub regions require long-distance structural connections to coordinate communication with other brain regions. Structural network studies suggest that anatomical connectivity patterns provide important context for understanding later functional disorganization in neurodegenerative disease [11,12]. Because these tracts support communication between high-interaction cortical regions, anatomical disconnection may contribute to later functional disorganization by reducing the efficiency of signal transmission across cortical networks [12].

Functional Magnetic Resonance Imaging (fMRI), when done while resting (rs-fMRI), can measure fluctuations in the BOLD signal that occur at rest [13]. The BOLD signals are used to identify intrinsic connectivity networks; this is an example of functional connectivity where synchronized activity exists in functionally different areas of the brain and represents one way researchers study how large-scale organizations exist within the brain [14]. Dynamic functional connectivity captures changes in network organization over time, as summarized in Figure 1.

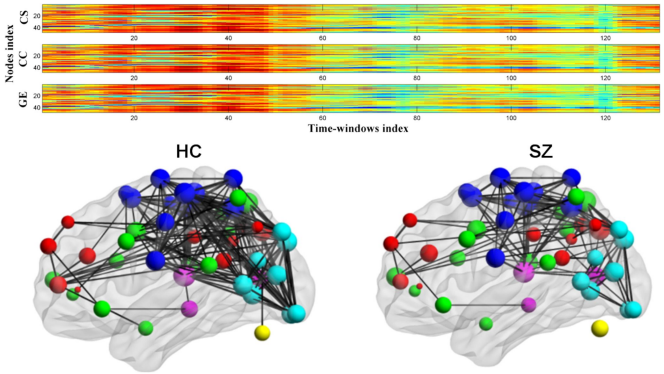

Figure. 1 Conceptual illustration of dynamic functional connectivity [15,16]. The upper part of the figure shows variations in the strength of a node's connections over time; the lower part of the figure compares network architecture for healthy controls (HC) vs. schizophrenia (SZ). Evidence from neurodegenerative disease studies suggests that mild cognitive impairment and Alzheimer's disease may involve reduced temporal variability and altered time spent in connectivity states, while psychiatric-disorder evidence is used here only for methodological and analogical comparison [17].

Resting state imaging, in particular, has the advantage of examining neurodegenerative diseases without requiring the subject to complete a task (performance). Instead, resting-state fMRI examines the "spontaneous" oscillations of neural activity as they occur naturally during rest [18]. In addition, Independent Component Analysis (ICA) is commonly employed to identify and isolate resting-state networks such as the Default Mode Network, the Salience Network and the Executive Control Network [19,20].

Among all of the intrinsic brain systems, one of the most vulnerable to early pathological processes is the default mode system [21]. Alterations within the default mode network have been reported in normal aging, individuals at risk for Alzheimer's disease, mild cognitive impairment, and Alzheimer's disease [22–25]. These findings suggest that neurodegeneration may be best understood as a progressive connectome-level disorder characterized by breakdown of communication efficiency across distributed neural circuits [26]. The large-scale systems most relevant to this review are shown in Figure 2.

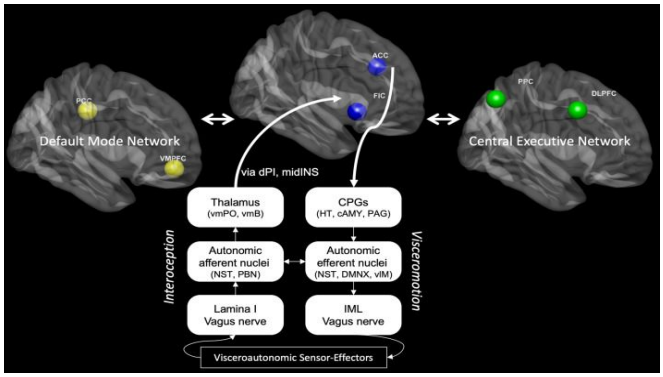

Figure. 2 Brain networks are affected early in neurodegenerative disease [22]. The three major brain systems shown here include the default mode network, central executive network, and salience network. Disruption of communication among large-scale networks has been associated with memory and cognitive changes in mild cognitive impairment and Alzheimer's disease.

Although DFC methods remain methodologically debated, apparent time-varying connectivity may be influenced by factors such as sliding-window length, motion artifacts, physiological noise, parcellation choice, global signal regression, and assumptions about whether connectivity is truly changing over time [27,28]. Therefore, caution is needed when interpreting DFC findings as evidence for biomarker use.

This review focuses on dynamic functional connectivity (DFC), a resting-state functional magnetic resonance imaging (rs-fMRI) approach that examines how functional relationships among brain regions vary over time rather than relying on a single static connectivity estimate across the entire scan. The purpose is not to suggest that DFC has already been established as a clinical biomarker, but to examine whether DFC-derived measures may serve as candidate research features for detecting or monitoring network-level changes in individuals with mild cognitive impairment and early Alzheimer's disease.

Methods

A narrative literature review with a structured search strategy was conducted to evaluate whether dynamic functional connectivity has been studied as a candidate research feature associated with mild cognitive impairment and Alzheimer's disease progression. Searches were conducted in 2025 for peer-reviewed studies published between 2004 and 2020. A literature search was conducted across PubMed, Google Scholar, Web of Science, and ScienceDirect. The search strategy used the following Boolean logic: ("dynamic functional connectivity" OR "time-varying functional connectivity") AND ("mild cognitive impairment" OR "Alzheimer's disease" OR "neurodegeneration"); ("resting-state fMRI" OR "functional connectivity") AND ("default mode network" OR "salience network" OR "executive control network") AND ("Alzheimer's disease" OR "mild cognitive impairment"); and ("graph theory" OR "machine learning") AND ("neuroimaging" OR "functional connectivity") AND ("Alzheimer's disease" OR "mild cognitive impairment"). Reference lists of relevant review articles and included studies were also screened to identify additional eligible sources.

Because the central focus of this review is dynamic functional connectivity in mild cognitive impairment and Alzheimer's disease, structural imaging studies were included only when they provided background information about network-level vulnerability, white matter disruption, or comparisons with functional connectivity findings. Structural MRI, diffusion tensor imaging (DTI), and tractography studies were not treated as primary evidence for validating DFC as a clinical biomarker unless they were directly connected to functional connectivity, disease staging, or multimodal neuroimaging comparisons.

Studies were excluded if they were not peer reviewed, were not published in English, did not involve human neuroimaging or directly relevant dynamic functional connectivity methodology, focused only on non-neurodegenerative conditions without methodological relevance to DFC, or did not address functional connectivity, structural connectivity, disease stage, cognitive decline, or neuroimaging-based classification. Structural-only studies were excluded unless they provided relevant background on network vulnerability, white matter disruption, or multimodal imaging comparisons with functional connectivity findings.

Time-varying changes in functional connectivity were selected for review because they directly addressed the central focus of this review: whether measures derived from DFC are associated with network-level changes in mild cognitive impairment and Alzheimer's disease, and whether these measures provide useful research-level information about disease stage, cognitive decline, or progression. Graph-theoretical studies were included when they provided network-level context for functional connectivity changes, but they were not given formal weighting over DFC-focused studies.

Studies were not treated as having equal evidentiary strength. Greater interpretive emphasis was placed on studies that specifically evaluated mild cognitive impairment or Alzheimer's disease, clearly identified disease stage, used human neuroimaging data, explained their connectivity methods, and included quantitative comparisons, longitudinal information, diagnostic performance metrics, or prognostic performance metrics. Methodological reviews and structural imaging studies were generally used for background, comparison, or interpretation rather than as primary evidence for establishing DFC as a validated clinical biomarker.

For each included study, extracted information included participant demographics, sample size, disease group, disease stage, neuroimaging modality, functional connectivity method, brain regions or networks examined, statistical approach, and main connectivity findings. When available, information was also recorded on diagnostic biomarker performance, prognostic prediction of cognitive decline, longitudinal analysis, and comparisons with structural imaging or molecular biomarkers.

Because the reviewed studies differed in disease stage, imaging modality, DFC method, network metric, and reported outcome measures, a quantitative meta-analysis was not conducted. Instead, the findings were integrated using a qualitative narrative thematic synthesis. Studies were categorized by disease focus, neuroimaging method, connectivity metric, and whether they provided direct evidence from mild cognitive impairment or Alzheimer's disease, methodological support, or broader comparative context. Each study was also grouped by methodology, including sliding-window correlation, independent component analysis, seed-based connectivity analysis, graph-theoretical modeling, and machine-learning classification, to compare how different methods identified connectivity abnormalities.

Results

The results are organized to distinguish evidence directly involving mild cognitive impairment and Alzheimer's disease from broader methodological or analogous DFC evidence drawn from non-neurodegenerative conditions. The studies identified in the literature review covered mild cognitive impairment (MCI), Alzheimer's disease (AD), functional connectivity disruption, graph-theoretical network changes, and neuroimaging-based classification. Findings varied across studies based on disease stage, imaging modality, connectivity metric, and whether the study design was cross-sectional, longitudinal, or classification-based. Therefore, this Results section summarizes common patterns reported in the literature, including disease-related alterations in connectivity,

commonly affected networks, graph-theoretical findings, and limitations in the reporting of diagnostic or prognostic performance metrics.

AD and MCI Evidence

Several studies report altered patterns of connectivity across mild cognitive impairment (MCI) and Alzheimer's disease populations[29]. Connectivity within the default mode network (DMN) was identified as a common area of altered connectivity among MCI and AD patients[21,22]. Reduced connectivity and synchronization between the posterior cingulate cortex and hippocampal regions have been linked to memory-related dysfunction and cognitive decline in mild cognitive impairment and Alzheimer's disease[30–35]. Numerous research studies have documented reduced coherence between the medial temporal lobe (MTL) structures and posterior cortical regions indicating disrupted long-range communication[34]. Impaired coordination of the hippocampus and the posterior cingulate cortex has also been associated with poor episodic memory performance and lower efficacy for integrating new information into an individual's knowledge base[35,36]. Functional connectivity analyses have reported altered dynamic functional connectivity in mild cognitive impairment and Alzheimer's disease, although the direction, magnitude, and diagnostic significance of these findings vary across methods and cohorts[17]. These graph-theoretical measures are illustrated in Figure 3. Together, these measures provide a simplified way to describe network-level disruption, but they should be interpreted as supportive network-context evidence rather than direct proof that DFC is a validated clinical biomarker.

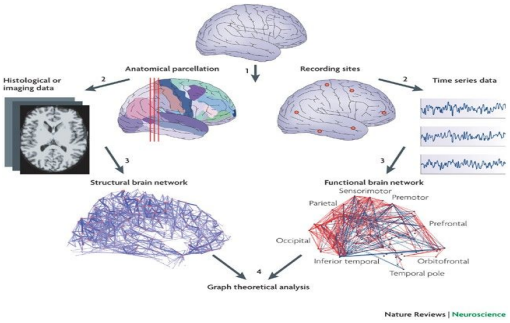

Figure. 3 Graph-theoretical representation of network disruption[37,38]. Global efficiency, modularity, and small-world organization are graph-theoretical metrics that provide a quantitative description of the structural characteristics of large-scale brain networks. These characteristics have been reported as disrupted in mild cognitive impairment and Alzheimer's disease.

Dynamic Connectivity and Network-State Findings

In this context, metastability refers to the brain's ability to shift flexibly among multiple connectivity patterns rather than remaining fixed in a single state. Dwell time refers to the amount of time a network remains in one connectivity configuration before transitioning to another. Small-world organization describes a network structure that balances local specialization with efficient long-distance communication, while modularity refers to the extent to which a network is divided into distinct but interacting modules[2,39]. Time-resolved analyses have also been used to track whole-brain connectivity patterns, identify recurring resting-state network configurations, and classify dynamic connectivity states[40–42].

Functional connectivity networks can transition quickly through different connectivity states in healthy individuals, whereas MCI subjects demonstrate longer periods of time spent in a single state[15]. Temporal variability in connectivity is generally reduced in those that are less cognitively flexible[16]. Healthy individuals usually display a pattern of metastable connectivity which is indicative of constant transitions among different network configurations[43]. Reduced metastability has been interpreted in the literature as reflecting less flexible coordination of neural resources across changing cognitive demands[16,43].

Several graph-theoretical studies suggest that individuals experiencing early cognitive decline may show lower global efficiency and altered small-world organization compared with cognitively healthy adults[37,38,44]. Hub regions such as the hippocampus and posterior cingulate cortex have been discussed in the literature as important regions for efficient information transfer across interconnected brain networks[9].

Across the studies examined in this review, the most frequently reported direction of change involved reduced or altered connectivity within memory-related and large-scale association networks, including the default mode network and hippocampal-cortical circuitry, as well as graph-theoretical measures such as global efficiency and small-world organization. However, the reviewed literature did not consistently report comparable effect sizes, diagnostic thresholds, or standardized performance metrics across MCI and Alzheimer's disease studies. Therefore, findings were summarized primarily by direction of network change, disease stage, imaging method, and whether the evidence came from MCI/Alzheimer's disease cohorts or broader methodological studies.

Discussion

Numerous large-scale studies of brain connectivity demonstrate that the way in which neurodegenerative injury develops can reflect the topological characteristics of existing brain

networks as opposed to being distributed at random throughout disparate areas of the brain[1,2,5]. Brain "hubs" are typically characterized by a very high level of interconnectivity; such hubs frequently serve cross-cortical communication functions and also have higher levels of energy expenditure than other parts of the brain[7,8]. The network approach offers an explanation for how early stages of neurodegenerative diseases can initially manifest through impairments in several different cognitive domains, including memory, attention, and executive functioning, while still recognizing that molecular and structural pathophysiology remains central to disease progression[6,26].

Emerging disease-progression models suggest that anatomical pathways and structural connections may influence how pathological proteins spread across neural systems[4]. In theory, regions with higher resting energy consumption may be affected earlier because they may be more vulnerable to oxidative stress, mitochondrial dysfunction, and related cellular stressors[8]. This supports the use of network-level approaches to examine patterns of structural and functional disruption alongside molecular and anatomical markers of disease[10,45]. These network-level patterns provide the background for considering whether time-varying functional connectivity measures can capture disease-related changes that may not be fully described by static connectivity alone. Dynamic functional connectivity (DFC), which refers to changes in connectivity between brain regions over time, has also been studied in relation to neurodegenerative disease assessment. Unlike static connectivity approaches, DFC allows researchers to examine how network relationships fluctuate across time rather than relying on one averaged connectivity estimate[46]. In mild cognitive impairment and Alzheimer's disease, decreased temporal variability and increased time in less adaptive connectivity patterns have been investigated as possible indicators of reduced network adaptability[17]. The default mode network and hippocampal-cortical connections are especially relevant to early cognitive decline because they support memory encoding, retrieval, and integration[21,30]. However, it remains premature to treat DFC as a reliable diagnostic tool because methodological consistency and clinical utility have not yet been established across the reviewed studies.

Dynamic Functional Connectivity (DFC) is better viewed as a candidate biomarker feature than as an established clinical biomarker. To be a valid clinical biomarker, there must be strong, consistent evidence to support its use as either a diagnostic or prognostic marker, which can include metrics such as sensitivity, specificity, ROC-AUC, positive and negative predictive values, independent external validation and the incremental value provided over other existing methods such as structural MRI, amyloid/tau biomarkers, APOE genotype, cognitive testing and static functional connectivity[47]. Across the studies examined in this review, some DFC-derived measures were associated with mild cognitive impairment and Alzheimer's disease. However, due to the heterogeneity of the methodologies used, variability in reported performance metrics, and lack of longitudinal validation across multiple studies, the reviewed evidence does not support DFC as ready for standard-of-care clinical diagnosis or prediction[28,46]. Before DFC-derived findings can be interpreted as clinically meaningful, it is necessary to consider the methodological limitations that affect how dynamic connectivity is measured and compared across studies.

Methodological Limitations of DFC

The dynamic functional connectivity (DFC) findings should be interpreted in light of several methodological constraints. Sliding-window analysis can be sensitive to window size, the number of estimated states, and assumptions about whether observed BOLD correlations reflect true neural transitions rather than statistical or physiological variability[27]. In addition to sliding-window parameters, motion artifacts, physiological noise, parcellation choice, temporal filtering, bandpass filtering, and global signal regression can all influence estimated connectivity patterns[19,46]. Leonardi et al. further caution that sliding-window correlation methods may yield spurious fluctuations if methodological constraints are not carefully controlled[28]. Because studies use different preprocessing pipelines and DFC metrics, direct comparison across cohorts remains difficult. These issues do not negate the potential utility of DFC, but they limit the strength of claims that DFC is ready for clinical biomarker use. More statistically structured approaches, such as hidden Markov models and carefully validated graph-theoretical metrics, may improve reliability; however, these approaches still require replication in larger longitudinal cohorts of individuals with neurodegenerative disease[7,15]. These methodological limitations are directly relevant to the question of whether DFC should be viewed as a validated biomarker or as a candidate research feature.

Status as Candidate Biomarker and Clinical Utility

Future work incorporating multimodal neuroimaging techniques could help elucidate how cortical thinning, structural disconnection, and functional brain abnormalities interact as disease progresses[48–50]. Using a combination of resting state functional connectivity, diffusion MRI, cortical thinning measures, and molecular imaging biomarkers may increase the ability to predict an individual's likelihood of experiencing rapid cognitive decline[47,51]. Multi-modal assessments could also identify complementary aspects of both anatomical disconnections and abnormal neuronal signal processing (dynamics)[52].

The standardization of preprocessing methodologies, in-

Table 1 Summary of DFC Biomarker Evidence by Disease Stage and Validation Status. This table summarizes selected studies by disease stage, DFC-related feature, study design, biomarker comparisons, reported diagnostic or prognostic metrics, evidence of incremental value, basis for early-stage claims, and current biomarker status. It distinguishes DFC-related candidate research features from validated clinical biomarkers.

Study	Disorder / stage	DFC-related metric or method	Study design	Comparison with static / structural / molecular biomarkers	Diagnostic or prognostic performance reported?	Incremental value of DFC shown?	Basis for early-stage claim	Biomarker status
Fiorenzato et al. 2020	Neurodegenerative disease cohort	Dynamic connectivity alterations	Cross-sectional / group comparison	Not consistently compared with molecular biomarkers or structural MRI as competing clinical biomarkers	Group-level DFC alterations reported; clinical validation metrics not sufficient to establish DFC as a clinical biomarker	Not established	Based mainly on disease stage at scan time; longitudinal temporal ordering not established	Candidate research feature only
Preti et al. 2017	General DFC methodology	Time-varying functional connectivity methods	Review / methodological synthesis	Not a direct clinical biomarker comparison study	Diagnostic or prognostic performance metrics not established	Not established	Methodological review; not direct evidence of early neurodegenerative progression	Methodological foundation only
Leonardi et al. 2015	General DFC methodology	Sliding-window correlation and spurious dynamic fluctuations	Methodological study	Not a clinical biomarker comparison study	Warns that apparent dynamic fluctuations can be artifactual if methodological constraints are insufficient	Not established	Methodological caution; not direct evidence of early neurodegenerative progression	Cautionary methodological evidence
Brier et al. 2012	Alzheimer's disease	Default mode network functional connectivity	Cross-sectional disease comparison	Static functional connectivity focus	Disease-related connectivity differences reported; DFC-specific diagnostic performance not established	Not assessed for DFC	Based on disease group/stage rather than DFC-specific longitudinal prediction	Static FC evidence, not validated DFC biomarker evidence
Sheline et al. 2010	Alzheimer's disease / amyloid burden	DMN connectivity and amyloid burden	Cross-sectional association study	Includes association with amyloid burden	Association between DMN connectivity and amyloid burden reported; DFC-specific diagnostic performance not established	Not assessed for DFC	Supports association with amyloid burden but not DFC-specific temporal ordering	Molecular/static FC association, not validated DFC biomarker evidence
Tijms et al. 2013	Alzheimer's disease	Network topology / graph-theoretical measures	Disease comparison study	Network topology rather than direct DFC clinical comparison	Altered network topology reported; DFC-specific clinical performance metrics not established	Not established for DFC	Supports network disruption in Alzheimer's disease but not DFC-specific early prediction	Network-level disease evidence, not validated DFC biomarker evidence
Arbabshirani et al. 2017	Brain disorders broadly	Single-subject prediction using neuroimaging features	Review / predictive-modeling synthesis	Discusses machine-learning prediction using neuroimaging features	Performance metrics vary across studies; DFC-specific clinical validity and independent incremental value are not consistently isolated	Not established for DFC	Predictive-modeling review; DFC-specific early-stage claim not established	Predictive-modeling support, not DFC clinical validation
Bron et al. 2020	Alzheimer's disease / MCI classification	Deep-learning classification	Review / classification-focused synthesis	Often includes structural or multimodal imaging features	Classification performance discussed, but DFC-specific clinical validity and incremental value are not established	Not established for DFC	Classification-focused review; DFC-specific early-stage claim not established	Research-level classification evidence

cluding motion-correction algorithms, temporal-filter parameters, network definitions, and connectivity metrics, is critical to reduce variability and enhance study-to-study replicability[19]. The differences in motion correction algorithms, temporal filter parameterizations, and definitions of networks used in studies have been major contributors to variability in results reported across the literature. A consensus on preprocessing techniques may increase the direct comparability of data sets and support future evaluation of connectivity measures in translational research[13].

Longitudinal data sets will be especially helpful in discovering if changes in connectivity can indicate with a degree of reliability that a person's condition is likely to deteriorate in the near future[53]. Identifying similar patterns of instability across independent cohorts could help determine whether connectivity measures have reliable predictive value before they are considered for clinical diagnostic use[54].

Further advancements in machine learning and computational neuroscience could allow for more personalized risk assessment based on an individual's connectome[47,54,55]. These cited works support neuroimaging classification and prediction at the research level, but they do not establish either DFC or machine-learning-based connectomic models as standard-of-care biomarkers. Furthermore, there is insufficient clinical trial evidence demonstrating that early detection using DFC-based methods results in interventions that slow neurodegeneration. Therefore, additional evaluations should determine whether DFC-derived measures provide meaningful predictive value beyond current biomarkers before they are used as part of clinical decision-making.

Conclusion

Dynamic functional connectivity represents a useful research approach for studying time-varying alterations in brain networks associated with mild cognitive impairment and Alzheimer's disease. Across the reviewed literature, DFC-related measures have been associated with altered temporal variability, reduced network adaptability, and disruptions in large-scale brain systems, including the default mode network and hippocampal-cortical circuitry. However, DFC is best understood as a candidate biomarker feature rather than a validated clinical biomarker. Future studies will need to provide longitudinal validation of DFC-derived measures, standardized preprocessing methods, independent replication across

datasets, and direct comparisons with established structural, molecular, genetic, and cognitive biomarkers before DFC-derived features are considered for clinical use.

References

1 Olaf Sporns, The Human Connectome: A Complex Network. Annals of the New York Academy of Sciences, 2011., https://doi.org/10.1111/j.1749-6632.2010.05888.x.

2 Ed Bullmore and Olaf Sporns, Complex Brain Networks: Graph Theoretical Analysis of Structural and Functional Systems. Nature Reviews Neuroscience, 2009., https://doi.org/10.1038/nrn2575.

3 Randy L. Buckner et al., Molecular, Structural, and Functional Characterization of Alzheimer's Disease. Journal of Neuroscience, 2005.

4 William Jagust, Imaging the Evolution and Pathophysiology of Alzheimer Disease. Nature Reviews Neuroscience, 2018.

5 Danielle S. Bassett and Olaf Sporns, Network Neuroscience. Nature Neuroscience, 2017., https://doi.org/10.1038/nn.4502.

6 Randy L. Buckner and Jessica R. Andrews-Hanna and Daniel L. Schacter, The Brain's Default Network. Annals of the New York Academy of Sciences, 2008.

7 Mikail Rubinov and Olaf Sporns, Complex Network Measures of Brain Connectivity: Uses and Interpretations. NeuroImage, 2010., https://doi.org/10.1016/j.neuroimage.2009.10.003.

8 Sophie Achard and Ed Bullmore, Efficiency and Cost of Economical Brain Functional Networks. PLoS Computational Biology, 2007., https://doi.org/10.1371/journal.pcbi.0030017.

9 Olaf Sporns and Richard F. Betzel, Modular Brain Networks. Annual Review of Psychology, 2016.

10 Danielle S. Bassett and Ed T. Bullmore, Small-World Brain Networks. The Neuroscientist, 2006.

11 Y. He et al., Small-World Anatomical Networks. Cerebral Cortex, 2007.

12 X. Chen et al., Disrupted Network Efficiency in Alzheimer's Disease. Brain Connectivity, 2013.

13 Karl J. Friston, Functional and Effective Connectivity: A Review. Brain Connectivity, 2011., https://doi.org/10.1089/brain.2011.0008.

14 Marcus E. Raichle, The Brain's Default Mode Network. Annual Review of Neuroscience, 2015.

15 Diego Vidaurre et al., Discovering Dynamic Brain Networks. Proceedings of the National Academy of Sciences (PNAS), 2017.

16 Vince D. Calhoun et al., The Chronnectome. Neuron, 2014.

17 E. Fiorenzato et al., Dynamic functional connectivity changes associated with dementia in Parkinson's disease. Brain. 2019;142(9):2860–2872., https://doi.org/10.1093/brain/awz192.

18 Jessica S. Damoiseaux, Resting-State fMRI as a Biomarker for Alzheimer's Disease. Alzheimer's Research & Therapy, 2012.

19 Stephen M. Smith et al., Network Modelling Methods for fMRI. NeuroImage, 2011.

20 Jessica R. Andrews-Hanna et al., Functional-Anatomic Fractionation of the Brain's Default Network. Neuron, 2010.

21 Michael D. Greicius et al., Default-Mode Network Activity Distinguishes Alzheimer's Disease. Proceedings of the National Academy of Sciences (PNAS), 2004.

22 Yvette I. Sheline et al., Default Mode Network Connectivity and Amyloid Burden. Proceedings of the National Academy of Sciences (PNAS), 2010.

23 Christian Sorg et al., Selective Changes of Resting-State Networks in Individuals at Risk for Alzheimer's Disease. Proceedings of the National Academy of Sciences (PNAS), 2007.

24 Jessica S. Damoiseaux et al., Reduced Resting-State Brain Activity in the Default Network in Normal Aging. Proceedings of the National Academy of Sciences (PNAS), 2008.

25 Matthew R. Brier et al., Loss of Intranetwork Connectivity in Alzheimer's Disease. Journal of Neuroscience, 2012.

26 Michela Pievani et al., Functional Network Disruption in the Degenerative Dementias. Lancet Neurology, 2011.

27 R. Matthew Hutchison et al., Dynamic Functional Connectivity. NeuroImage, 2013.

28 N. Leonardi et al., Spurious Fluctuations in Dynamic Connectivity. NeuroImage, 2015.

29 Anouk Hafkemeijer et al., Imaging the Default Mode Network in Aging and Dementia. Biochimica et Biophysica Acta, 2012.

30 Li Wang et al., Altered Hippocampal Connectivity in Mild Cognitive Impairment. Human Brain Mapping, 2006.

31 H. Li et al., Abnormal Hippocampal Connectivity Patterns. Brain Connectivity, 2012.

32 M. H. Lee et al., Disrupted Hippocampal Functional Connectivity. Neurobiology of Aging, 2014.

33 F. Bai et al., Abnormal Hippocampal Connectivity in Mild Cognitive Impairment. Journal of Alzheimer's Disease, 2015.

34 D. T. Jones et al., Functional Connectivity Alterations in Hippocampal Circuits. NeuroImage Clinical, 2018.

35 W. S. Sohn et al., Progressive Hippocampal Network Changes. Brain Imaging and Behavior, 2020.

36 Randy L. Buckner, Memory and the Hippocampus. Neuron, 2004.

37 B. M. Tijms et al., Alzheimer's Disease Network Topology. Cerebral Cortex, 2013.

38 C. J. Stam et al., Graph Theoretical Analysis of Dementia. NeuroImage, 2012.

39 J. Wang et al., Graph Theoretical Analysis of Functional Brain Networks. PLoS ONE, 2010.

40 Elena A. Allen et al., Tracking Whole-Brain Connectivity Dynamics. Cerebral Cortex, 2014.

41 Andrew Zalesky et al., Time-Resolved Resting-State Networks. Proceedings of the National Academy of Sciences (PNAS), 2014.

42 B. Rashid et al., Dynamic Connectivity States Classification. Frontiers in Neuroscience, 2016.

43 James M. Shine et al., Dynamic Network Communication. Nature Neuroscience, 2016.

44 Z. Dai et al., Altered Small-World Network Topology. Human Brain Mapping, 2015.

45 B. M. Tijms et al., Pathological Connectomics. NeuroImage Clinical, 2018.

46 Maria Giulia Preti et al., Dynamic Functional Connectivity: State-of-the-Art. NeuroImage, 2017.

47 M. R. Arbabshirani et al., Single Subject Prediction of Brain Disorders. NeuroImage, 2017.

48 Bradford C. Dickerson et al., MRI Derived Cortical Thinning in Alzheimer's Disease. Cerebral Cortex, 2009.

49 D. Zhang et al., Multimodal Classification of Alzheimer's Disease. NeuroImage, 2011.

50 H. Li et al., Multimodal Neuroimaging Classification. Frontiers in Neuroscience, 2018.

51 S. Vieira et al., Using Machine Learning in Neuroimaging. NeuroImage, 2017.

52 H. I. Suk et al., Deep Learning for Neuroimaging Biomarkers. NeuroImage, 2014.

53 C. Y. Wee et al., Identification of MCI Individuals Using Connectivity Patterns. NeuroImage, 2012.

54 E. E. Bron et al., Deep Learning Classification Accuracy in Alzheimer's Disease. Frontiers in Aging Neuroscience, 2020.

55 A. Payan et al., Predicting Alzheimer's Disease with Deep Learning. NeuroImage Clinical, 2015.