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Biomarkers in Neurodegenerative Diseases: A Bibliometric Analysis Using VOSviewer

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Abstract

The accurate and timely diagnosis of neurodegenerative diseases is a major challenge, driving considerable efforts to identify biomarkers that can detect presence of disease, progression and therapeutic response. A bibliometric analysis of the literature on biomarker research in relation to Parkinson's disease, Alzheimer's disease, and more neurodegenerative disorders is conducted in this study to trace the intellectual structure, working groups, and thematic development of the research. A dataset of 1,431 publications from 2010–2025 was analyzed using bibliometric and visual analysis techniques in VOS viewer. The analysis of co-authorship demonstrated that Swedish establishments, especially the Sahlgrenska Academy and Skan University Hospital dominate the research market, with Henrik Zetterberg (1, 203 citations) and Kaj Blennow (1, 134 citations) being the most influential authors. The United States was had the maximum number of publications (452 documents, 22,101 citations), then the United Kingdom and Sweden. The most cited paper was Dammer (2022) on multi-platform proteomic analysis of Alzheimer CSF and plasma biomarkers (2,293 citations). Thematic analysis showed a chronological shift from CSF-based protein markers and biomarker ratios (2010–2015) through neuroinflammatory markers and microRNAs (2015–2020) toward blood-based alternatives, digital biomarkers, and multi-modal diagnostic frameworks (2020–2025). The findings highlight the need for protocol standardisation across studies, increased collaboration with underrepresented regions, and continued development of blood-based and minimally invasive diagnostic tools. The paper has practical implications to researchers who want to collaborate and future trends in neurodegenerative biomarker studies.

Keywords: *Alzheimer's Disease; Parkinson's Disease; Neurodegeneration; Bibliometric Analysis; VOSviewer, Biomarkers*

Introduction

Traditionally, biomarker studies on neurodegenerative diseases combine clinical diagnostics, which examine protein biomarkers, imaging biomarkers and genetic susceptibility factors, with mechanistic studies that consider what is known about pathological processes underlying neuronal loss. The identification of this dual point of view is critical to designing diagnostic methods that are clinically available and biologically significant. Neurodegenerative disorders are multifactorial conditions that involve the nervous system, which are characterized by protein misfolding, mitochondrial dysfunction,

oxidative stress, and environmental circumstances and present in about 30 million people globally (Sheikh et al., 2013). These conditions include Parkinson's disease (PD), Alzheimer's disease (AD), Lewy body dementia (LBD), and related disorders, each disease presenting with distinct clinical and pathological features, yet sharing common mechanisms of progressive neuronal loss. With world populations aging, the prevalence of these disorders continues to increase, causing significant strain to healthcare facilities, care givers and patients. Diagnosis remains essential during the management of a disease; however, the diagnosis is frequently done with clinical signs and symptoms that have been developed after considerable amounts of neuronal damage having already occurred. This fact has spurred a desperate and growing hunt to find dependable biomarkers, objectively measurable biological markers that reflect normal physiological processes, pathogenic processes or treatment response and are used in diagnosis, disease staging, prognosis and treatment response monitoring (Jack et al.,2018).

Initial research in this area focused mainly on cerebrospinal fluid (CSF) biomarkers, whereby CSF is the biological fluid that encloses the brain and the spinal cord and that captures biochemical activities in the central nervous system (Blennow et al., 2010). Amyloid -2 (A2) peptide, the extracellular deposition of which is a key pathogenic process in Alzheimer (Hardy and Selkow, 2002), and tau protein, the abnormal hyperphosphorylation of which results in the formation of neurofibrillary tangles contributing to cognitive impairment (Wang and Mandelkow, 2016). In the case of Parkinson and synucleinopathies, alpha -synuclein became a primary target, an X-synaptic protein whose pathogenic accumulation in Lewy bodies is a characteristic of these diseases (Spillantini et al., 1997). These approaches established a foundation for understanding of the molecular changes associated with neurodegeneration but involved invasive lumbar puncture techniques hence being unsuitable for large scale industrial use.

Later studies used blood-based substitutes that were more accessible. Neurofilament light chain (NfL), which is released into biofluids in response to axonal injury, turned out to be a reliable marker of different disorders (Gaetani et al., 2019), and phosphorylated tau 181 (p-tau181) was a disease-specific biomarker that could differentiate between Alzheimer and other forms of dementia (Janelidze et al., 2020). More recent frameworks have utilised both genetic and neuroimaging dimensions, examining how risk factors such as the apolipoprotein E ε4 allele (APOE ε4) interact with fluid biomarkers to predict cognitive decline (Liu et al., 2013). This combines molecular findings with imaging markers including white matter hyperintensities and medial temporal atrophy (Wardlaw et al., 2015; Frisoni et al., 2010). Newer technologies such as seed amplification assays and proximity extension assays have widened the range of possible diagnostics (Mastrangelo et al., 2024; Pedersen et al., 2023), whereas new studies have also looked at extracellular vesicles, cell-free mitochondrial DNA, and synaptic proteins as potential sources of biomarkers (Thompson et al., 2016; Lowes et al., 2020; Brinkmalm et al., 2014).

Over the past few decades, governments, healthcare institutions, and research organisations have augmented aid to biomarker studies in order to guide clinical choice on matters of early detection by way of therapeutic monitoring. However, there is a disparity in research distribution, with a comparatively limited number of very active collaboration institutions in Europe and North America leading to massive validation research and technology-building, and clinical centres in other areas providing case series of particular patient groups and disease subtypes. Researchers increasingly correlate fluid based biomarkers with neuroimaging and genetic data with multi-platform strategies to understand the complexity of the neurodegenerative process. This accumulating literature offers a more plentiful but nonetheless piecemeal evidence base on neurodegenerative biomarkers, constraining the ability to make generalisable conclusions and to establish useful diagnostic regimens across populations.

Importance of the Study

This work has great importance to researchers and clinicians who are keen on the changing landscape of biomarker research in neurodegenerative disorders. It is a systematic mapping of 1,431

articles, which assesses the trends and results of this field over time and in different institutions and countries. The discovery of biomarkers has been an area of research inquiry across various fields; however, the transfer of laboratory findings into the clinic is still difficult as studies tend to differ in terms of design, sample manipulation, and the platform of analysis. There is yet no single, scientifically agreed biomarker panel, which can diagnose and stage all neurodegenerative diseases, a fact that represents the biological heterogeneity of such diseases as well as this methodological uncertainty which surrounds the validation of biomarkers. Following the development of the initial cerebrospinal fluid-based markers, through blood based substitutes to the current multi-modal frameworks, the current study helps the researchers to identify where the current methods meet and where the gaps exist, especially in such areas as protocol standardisation across sites, cross-disease biomarkers validation, and how to integrate the emerging markers into clinical practice. Significant differences in pre-analytical variability of sample collection and processing between large research consortia have been demonstrated to affect biomarker values significantly, which is why harmonisation efforts are essential (Stewart et al., 2019). To readers, the study provides a better insight into the evolution of biomarker studies that used to be single-marker studies of specific diseases to multifactorial, multi-disease models that could reflect the complexity of neurodegeneration and its multifactorial pathogenesis.

Methodology

Information on the topic of biomarkers in neurodegenerative diseases was searched for upon the Scopus database. The articles were obtained using the query as follows: TITLE-ABS-KEY (BIOMARKERS AND NEURODEGENERATIVE) from 2010 to 2025 with filters with subject areas of biochemistry, neurology, and medicine. Additional filters were applied for the document type: article, the source type: journal, and the inclusion of English language. After this, a total of 1,431 documents were obtained following this query, with zero duplicates using Mendeley. The database was then exported to VOSviewer for further visualization and bibliometric analysis.

Table 1
Keywords used for searching literature and the no. of results obtained from Scopus.

Keywords	Scopus
BIOMARKERS AND NEURODEGENERATIVE	1,431
Duplicates	0
Total (net of duplicates)	1,431

Analysis

Couthorship and author

Among 4,987 authors, having at least three documents per author and ten citations per author, 137 were found to meet the threshold. Cluster1: The parameters of alpha-synuclein seed amplification assay (SAA) in Lewy body disease were evaluated, with the results demonstrating that Nrep and Lag are correlated with disease stage and progression to dementia, making them suitable to use in measuring disease progression and treatment response over time (Mastrangelo et al., 2024). Cluster 2: The initiation, nature, and persistence of treatment, and comorbidities both significantly impact the levels of circulating cell-free mitochondrial DNA (ccf-mtDNA) in Parkinson's disease and may affect the ccf-mtDNA homeostasis and use as a biomarker (Lowes et al., 2020). Other recent investigations have attributed cell-free mitochondrial DNA to neurodegeneration in both Alzheimer and Parkinson disease, and it is therefore possible that the same would be found with other diseases that exhibit a neurodegenerative aspect. One of the constituents of PMS is reduced ccf -mtDNA, implying that it, in fact, could be a

signature of larger-scale neurodegeneration (Lowes et al., 2019). Exception: Blood extracellular vesicles with synaptic-functional and brain proteins are under study as possible biomarkers of Alzheimer disease, which will fill the immediate requirement of objective and available Alzheimer disease AD and other dementia biomarkers (Tian et al., 2022). There were pre-analytical variability of CSF collection and processing in ADNI and PPMI studies, which considerably affected the values of biomarkers, especially A. There is a need to standardise and harmonise protocols to classify biomarkers correctly in neurodegenerative diseases (Stewart et al., 2019). Cluster 4: This paper described a seven biomarker panel of Parkinson disease (PD) and a four biomarker panel of Alzheimer disease (AD) discovered by proximity extension assay technology. 9 inflammatory biomarkers in patients with newly diagnosed PD were identified with the disease and demonstrated an well adjusted AUC of 0.82 to distinguish PD and controls. Four biomarkers used in the AD panel differentiated between Alzheimer's disease and controls with an AUC of 0.87. The results indicate that diagnostic panels of PD and AD could have biomarkers related to inflammation (Pedersen et al., 2023).

Table 2
Coauthorship and Author

Author	Documents	Citations	Total link strength
weintraub, daniel a.	5	1397	17
aarsland, dag	15	1340	38
zetterberg, henrik h.	20	1203	65
blennow, kaj	20	1134	64
ballard, clive g.	7	1056	19
mollenhauer, brit	7	979	9
hansson, oskar	18	884	66
trojanowski, john quinn	5	686	13
el-agnaf, omar mukhtar ali	4	663	16
janelidze, shorena	8	615	35

Figure 1
Couthorship and Author overlay visualisation

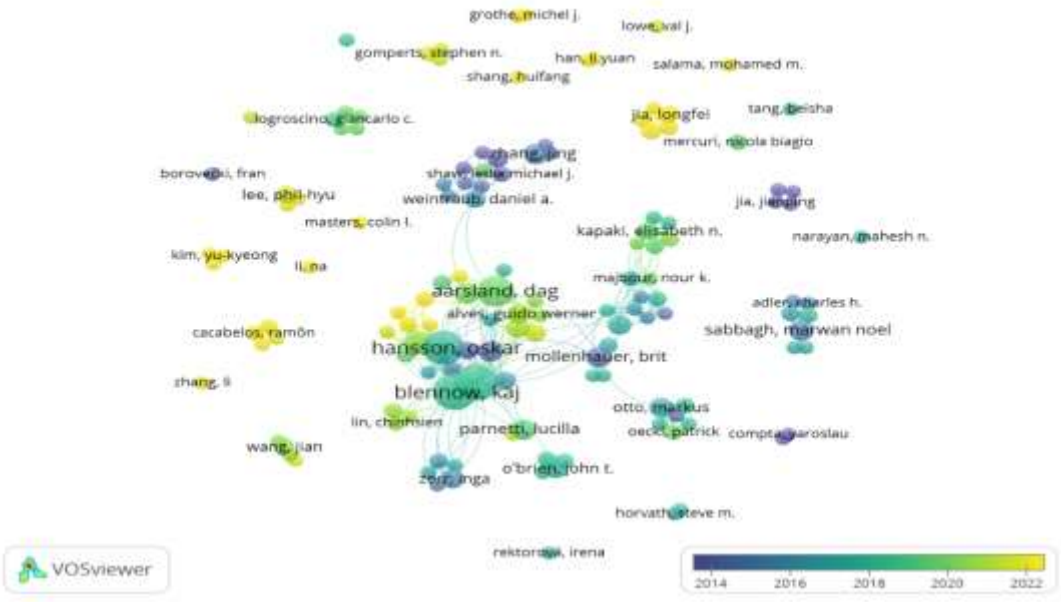
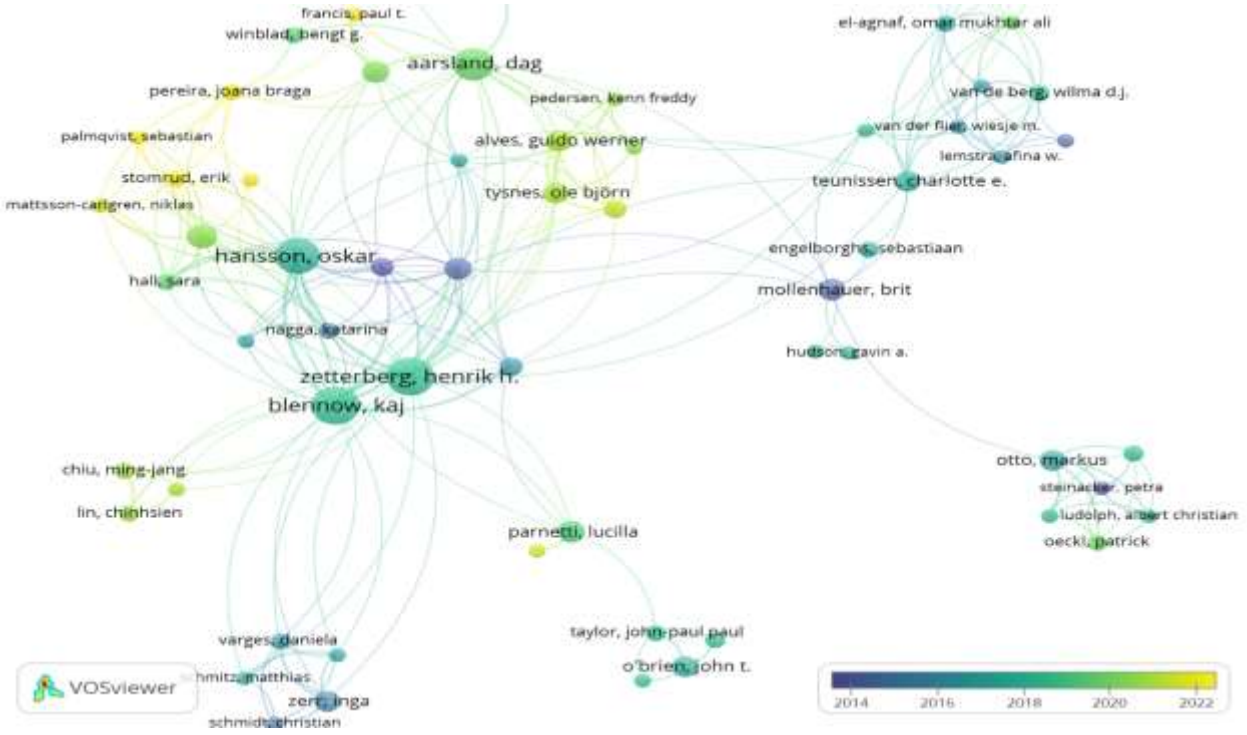


Figure 2
Couthorship and Author overlay visualisation



Coauthorship and organisation

Out of 4654 organisations, with minimum no. of documents of an organisation as 4, and minimum no. of citations of an organisation as 15, 184 met the threshold. Cluster 1 found that APOE E4 allele, thought disorder, and SPARE-AD score were found as important predictors of cognitive deterioration in patients afflicted by Parkinson disease. The greatest impact was on the APOE 4 genotype, which elevated the risk of cognitive impairment in all the evaluated areas (Tropea et al., 2018). In Cluster 2, the researchers determined specific presynaptic identities in Lewy body dementia (LBD) that were higher than controls, but less presynaptic modules in Alzheimer disease (AD). This brings out distinctive pathophysiological modifications in LBD compared to AD (Shantaraman et al., 2024). Cluster 3 found the analysis shows that melanoma brain metastases contain the transcriptomic resemblances of neurodegenerative diseases, with 53 dysregulated genes associated with the extracellular matrix and development, and 195 genes linked to cell differentiation and chromatin remodeling, and thus, identifying potential biomarkers, including ITGA10 and DNAJC6 (Soler-Saez et al., 2024). In DLB, cerebrovascular and Alzheimer disease biomarkers were also evaluated using high white matter hyperintensity loads, medial temporal atrophy, and connections with the medial temporal atrophy, all affected by 6 existence and mediation by 5 amyloid pathology compared to other types of dementia, and cognitively unimpaired subjects (Rennie et al., 2024). Cluster 4 found that Ab-related pathology was found to be a characteristic feature of AD and the majority of patients who were diagnosed with Alzheimers disease had biomarker proof of this process although the same had been found in other dementias, although less often. Concentrations of AD-like pathology cerebrospinal fluid biomarkers were linked to cognitive function but were mostly seen in patients with an AD-related diagnosis (early- and late-onset AD) (Skillback et al., 2025).

Table 3
Coauthroship and Organisation

Organization	Documents	Citations	Total link strength
Dept of Psychiatry and Neurochemistry, Sahlgrenska Akademin, Gothenburg, Västra Götaland, Sweden	45	3449	233
Memory Clinic, Skånes Universitetssjukhus, Lund, Skåne, Sweden	27	3354	131
Clinical Neurochemistry Laboratory, Sahlgrenska Universitetssjukhuset, Gothenburg, Västra Götaland, Sweden	37	2556	221
Dept. of Clinical Sciences, Lunds Universitet, Lund, Skåne, Sweden	14	2500	48
Dept. of Neurology, Skånes Universitetssjukhus, Lund, Skåne, Sweden	17	2423	92
Dept. of Pathology, University of Washington School of Medicine, Seattle, WA, United States	12	2332	59
Dept. of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, WA, United States	14	2228	81
Dept. of Neurology, University of Washington School of Medicine, Seattle, WA, United States	14	2228	81
Dept. of Neurodegenerative Disease, UCL Queen Square Institute of Neurology, London, United Kingdom	25	2145	161
OHSU School of Medicine, Portland, OR, United States	10	2155	62

Figure 3
Coauthorship and organisation overlay visualisation

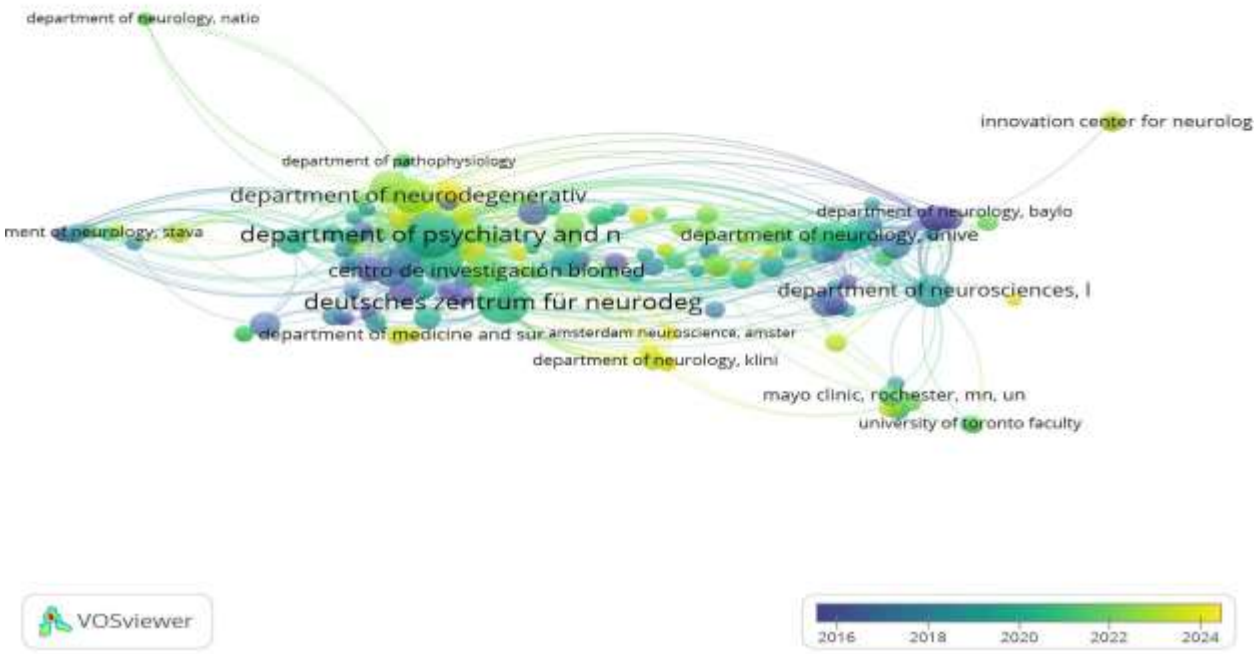


Figure 4
Coauthorship and organisation overlay visualisation

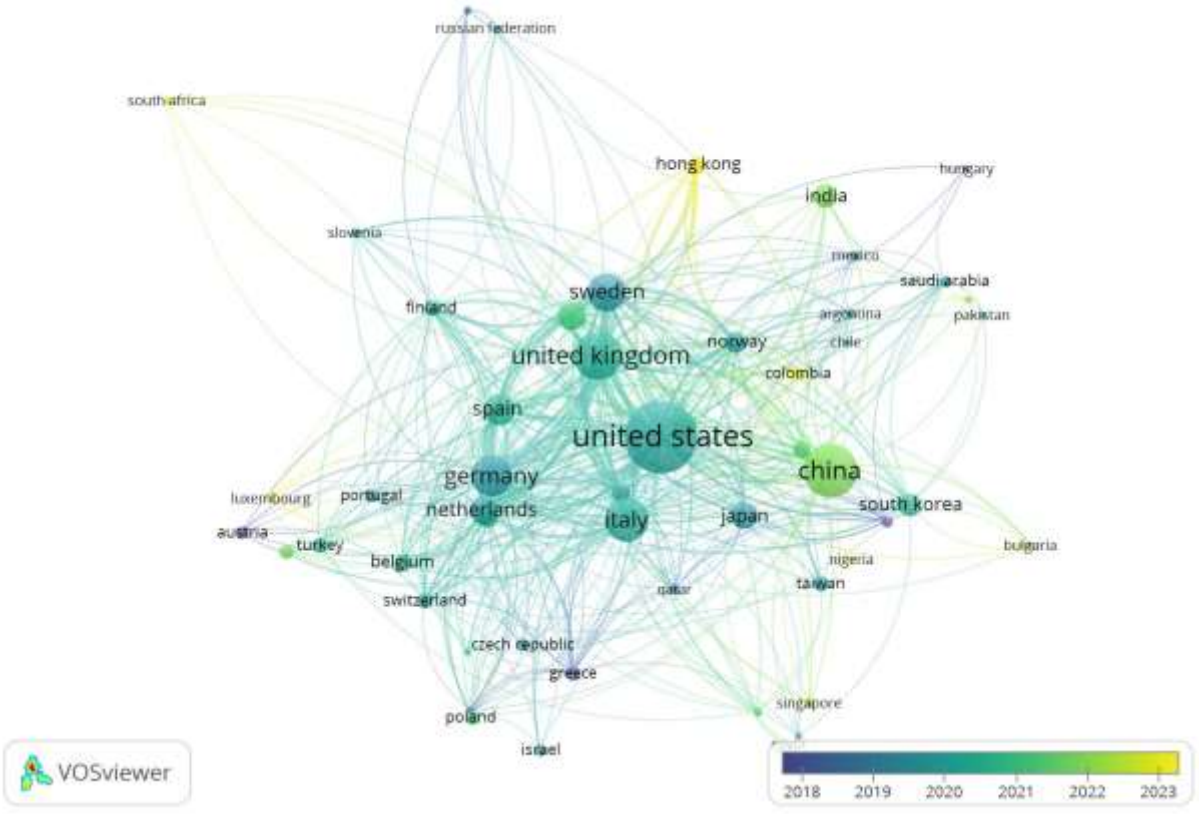
Coauthorship and country

Out of 77 countries, with the minimum no. of documents of a country as 3, and minimum no. of citations of a country as 11, 55 met the threshold. Cluster 1 concentrates on natural products with antioxidant properties, especially on the phytoconstituent, polyphenolic antioxidants comprised of herbs, fruits, and vegetables. These substances are theorized to inhibit neurodegeneration and improve cognitive performance, and thus serve the control of neurodegenerative diseases like Parkinson's disease and Alzheimer's disease by means of their anti-inflammatory and neuroprotective effect (Rahman et al., 2021). Cluster 2 established a framework that found disease-specific signals at the behavioral, neuroanatomical, and neurofunctional levels and, thus, highlights the sensitivity and specificity that can be reached by one naturalistic task. The results represent the future translational agenda to combine ecological aspects with multimodal cognitive neuroscience to study neurodegeneration further (Birba et al., 202. Cluster 3 presents a study of 29022 cerebrospinal-fluid samples done in a decade and confirms that 1433 protein testing retains a high specificity of 92% in the detection of Creutzfeldt-Jakob disease in the context of increasing test referral and variation in specificity among the various rapidly progressing dementia diagnoses (Stoeck et al., 2012). Cluster 4 reports a central biobank of samples of over 400 subjects with neurodegenerative conditions accompanied by a virtual biobank of over 8 600 subjects across 21 research centres, which generalise biomarker validation of Parkinson disease and Alzheimer disease (Reijs et al., 2015). Cluster 5 describes the diagnostics of probable DLB, having sensitivity of 73 and a specificity of 93. Despite the acceptable performance of the criteria, the occurrence of false positive and negative values implies the need to improve the method and possibly include biomarkers (Skogseth et al., 2017). Cluster 6 states that lesser levels of SNAP25 and synaptic proteins Rab3A demonstrating a strong correlation with the cognitive impairment in Alzheimer’s disease and in DLB, which thus suggests that they can be used in the future as biomarkers of progression of disease and the targets of subsequent therapeutic intervention (Bereczki et al., 2016).

Table 4
Coauthorship and country

Country	Documents	Citations	Total link strength
United States	452	22,101	473
United Kingdom	206	12,459	484
Sweden	129	9,498	325
Italy	165	7,504	237
Germany	151	6,674	267
China	255	6,355	134
Netherlands	72	5,153	207
Canada	73	4,774	157
Spain	88	3,535	163
Norway	43	2,839	126

Figure 5
Coauthorship and Country overlay visualisation



Co-occurrence and keywords

On the basis of co occurrence with unit of analysis as all keywords with a minimum no. of 216 occurrences, there were 37 keywords which met the thresholds of 12,365 keywords. After screening for duplicates and removal of irrelevant terms, 10 keywords had been selected. The most frequent keyword was Alzheimer (1111 occurrences), then Parkinsons disease (1076 occurrences), and Controlled study (847 occurrences). The maximum period of these high-frequency keywords was between the middle of 2020 and the beginning of 2021 (2020.5, 2020.6). Since the presented dataset encompasses the years 2010-2025, it shows that a significant fraction of research focused on the Alzheimer and PD disease was concentrated in this time. The keywords that trended during the period of August 2019 (2019.8) were Cerebrospinal fluid, α-synuclein, and Sensitivity and specificity. The words TAO protein and Amyloid 2 protein became trending keywords between 2020.2 and May 2020 (2020.2-2020.5). Between May 2020 and August 2020 (2020.5, 2020.8), the topic of study shifted to terms like Biomarkers, Cohort analysis, Pathology, Genetics, Cognitive defect, Degenerative disease among others.

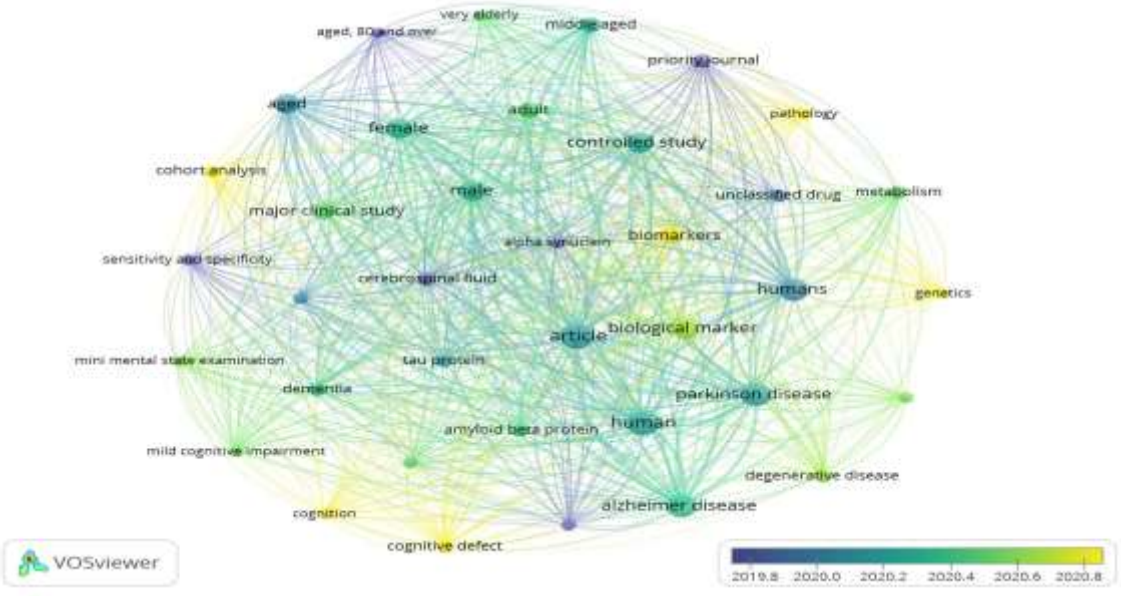
Table 5

Co Occurrence and all keyword

Keyword	Occurrences	Total link strength
alzheimer disease	1111	14949
parkinson disease	1076	14299
controlled study	847	12245
biological marker	828	11714
biomarkers	635	9433
major clinical study	584	9486
metabolism	385	5216
tau protein	383	6347

Figure 6

Co occurrence and all keyword overlay visualisation



Citation and Documents

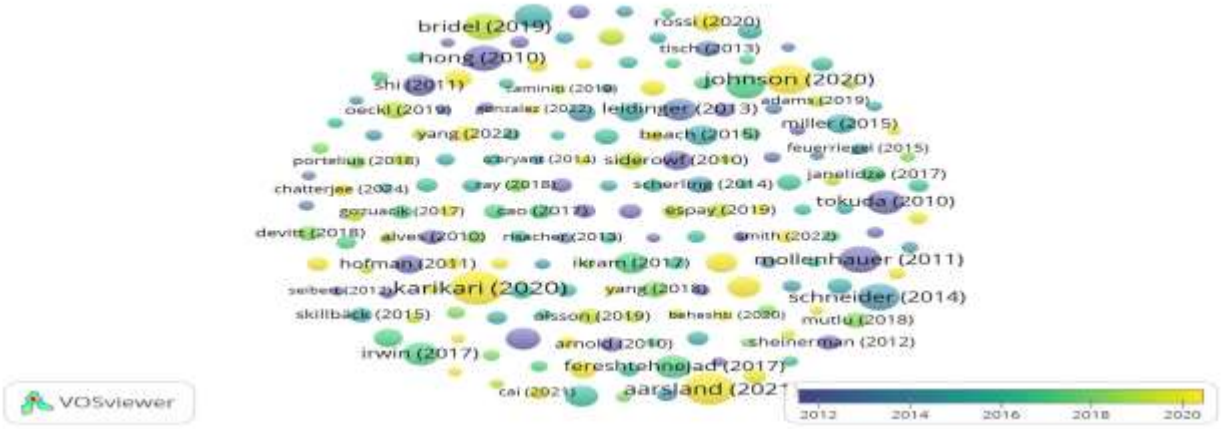
On the basis of citation with the unit of analysis being documents with a minimum of no. 70 citations, 183 documents had met the thresholds out of a total of 1431 documents.

Table 6
Citation and Documents

Document	Citations	Links
Dammer (2022)	2293	0
Tadayon (2020)	957	0
Ashton (2021)	878	0
Karikari (2020)	707	0
Saiki (2019)	602	0
Scherling (2014)	590	0
Berti (2011)	576	0
Reesink (2010)	573	0
Janelidze (2017)	532	0
Fan (2015)	494	0

Dammer (2022) has the highest number of citations with 2293 citations, followed by Tadayon (2020) with 957 citations, Ashton (2021) with 878 citations, Karikari (2020) with 707 citations, and Saiki (2019) with 602 citations. In a study titled Multi-platform proteomic analysis of the Alzheimer disease cerebrospinal fluid and plasma reveals network biomarkers of proteostasis and the matrisome, Dammer (2022) indicates some divergent protein changes in AD cerebrospinal fluid (CSF), and SMOC1 was determined to be increased in both plasma and CSF. According to Tadayon (2020) in the article titled *Choroid plexus volume is associated with levels of CSF proteins: relevance to Alzheimer's and Parkinson's disease, the change in choroid plexus functioning could be related to the pathophysiology of Alzheimer's disease (AD), specifically in non-demented patients. In a study published by Ashton (2021) under the name A multicentre validation study of the diagnostic value of plasma neurofilament light, it is established that plasma neurofilament light (NfL) is a good biomarker that can detect neurodegeneration in thirteen different disorders. Karikari (2020) named the article Blood phosphorylated tau 181 as a biomarker of AD: a diagnostic performance and prediction modelling study using four prospective cohorts evidence that blood p -tau181 predicts Alzheimer pathology and cognitive impairment, and that plasma p -tau181 may discriminate between AD and other neurodegenerative disorders. The article by Saiki (2019), entitled A metabolic profile of polyamines in Parkinson disease: A promising biomarker, indicates that spermidine and spermine levels are potential biomarkers of Parkinson disease that can be used to increase lifespan and that polyamine metabolites are potential age-related and severity-dependent biomarkers of Parkinson disease.

Figure 7
Citation and Documents overlay visualisation



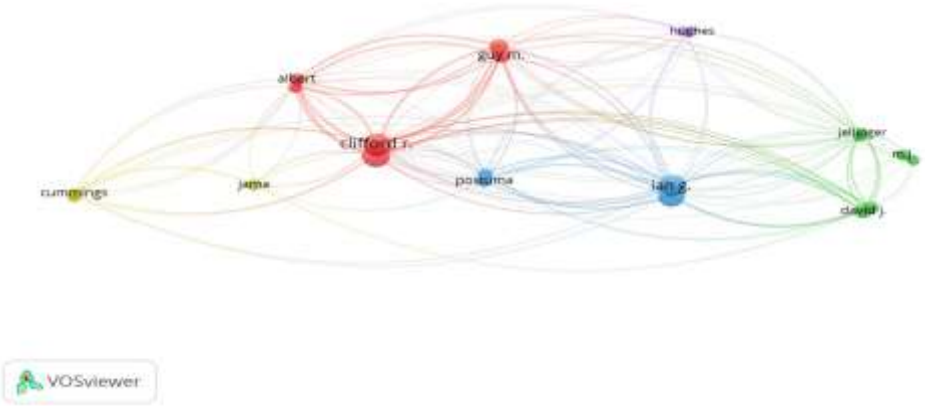
Co citation and cited Authors

On the basis of co citation and authors, with the minimum number of citations had been set to 27, the leading 25 authors of a total of 4870 in were taken. Cluster 1 found that The PredictND tool improved clinicians' diagnostic confidence and led to changes in clinical diagnoses in dementia cases, but did not particularly ameliorate diagnostic accuracy. The study involved 779 patients and highlighted the potential of CDSS in dementia diagnostics (Bruun et al., 2019). Cluster 2 found the study identifies over a number of seventy putative caspase substrates in mammalian brain synapses, including novel targets like ATP6V1B2 and NSF, highlighting the role of caspases in synaptic plasticity and loss, with potential implications for neurodegenerative disease biomarkers (Victor et al., 2018). Cluster 3 found that there was a new robust and reliable rating scale that captured the visual appearance of 123I-Ioflupane brain images. It showcased high accuracy in autopsy validated cases and offered advantages over the previously existing visual rating scales (Lloyd et al., 2018).

Table 7
Co citation and Cited Authors

Author	Citations	Total Link Strength
clifford r.	121	306
jack	121	306
ian g.	103	264
mckeith	103	264
guy m.	53	161
mckhann	53	161
david j.	47	140
irwin	41	138
jama	38	34
postuma	36	92

Figure 8
Co Citation and Cited Documents network visualisation



Bibliographic Coupling and Cited Documents

On the basis of bibliographic coupling and cited documents, with the minimum number of citations set to 159, out of 1431, 65 met the threshold. Cluster 1 validates plasma neurofilament light (NfL) as a biomarker for neurodegeneration across 13 disorders, demonstrating its utility in differentiating atypical parkinsonian disorders, dementia in Down syndrome, and psychiatric dementia, with significant age-related diagnostic cut-offs (Ashton et al., 2021). Cluster 2 showed the existence of a group of patients with Parkinson disease dementia (PDD), who displayed cerebrospinal fluid (CSF) and positron emission tomography (PET) patterns that resembled those of Alzheimer disease (AD), with significant changes in amyloid-9 and to lower levels tau levels (Buongiorno et al., 2011). Cluster 3 showed that a combination of these 21 biomarkers could separate the patients with PD against the healthy control as well as the patients with AD and multiple system atrophy. Specifically, CSF Flt3 ligand measurement allowed making a clear distinction between PD and multiple system atrophy, which is both a clinical entity, phenotypically overlapping with PD and yielding an impressive sensitivity of 99 000 and specificity of 95 000 (Shi et al., 2011).

Table 8
Bibliographic Coupling and Cited Documents

Document	Citations	Total Link Strength
yang (2022)	2293	2
betts (2019)	957	0
ashton (2021)	878	4
karikari (2020)	707	6
nakajima (2019)	602	0
scherling (2014)	590	0
tokuda (2010)	576	4
janelidze (2017)	532	6
casas (2014)	494	0
montine (2010)	464	5

Figure 9
Bibliographic Coupling and Cited Documents overlay visualisation

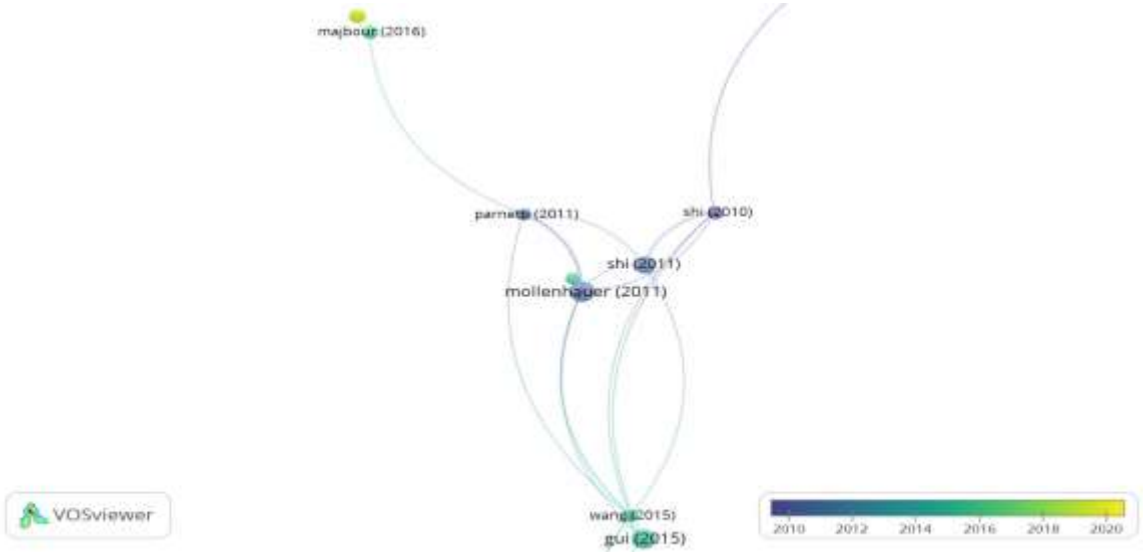
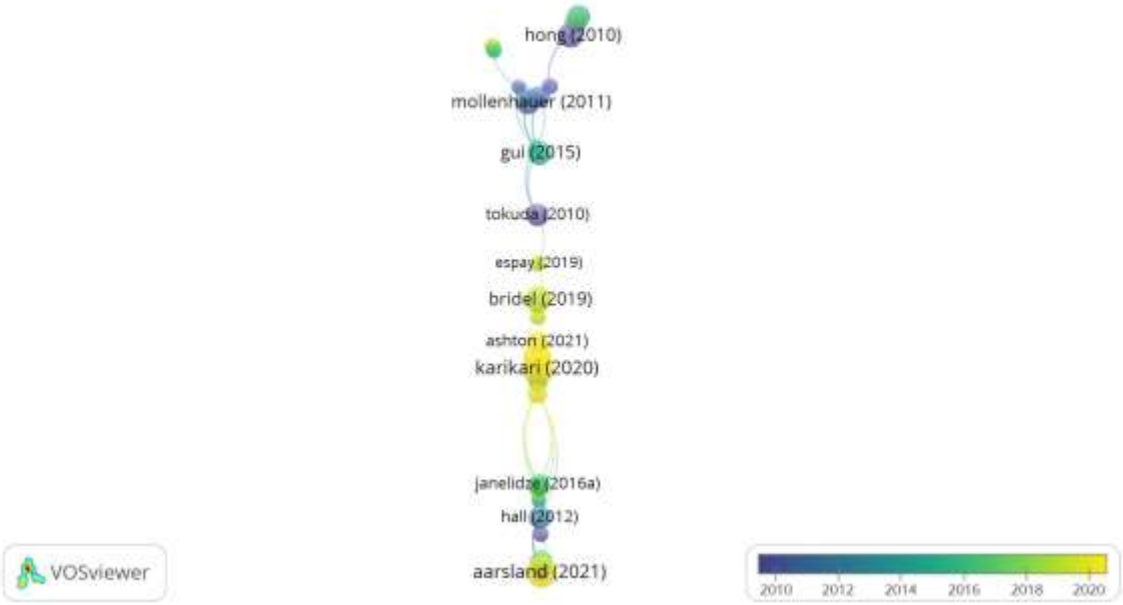


Figure 10
Bibliographic Coupling and Cited Documents overlay visualisation



Research Dynamics

In the 2010-2025 set, there are three overall chronological stages of biomarker research in neurodegenerative disease, in which there has been a macro-trend shift toward more accessible, multimodal biomarker models, as opposed to the traditional cerebrospinal fluid (CSF) protein biomarkers.

Between 2010 and 2015, most studies have been based on the classical biochemical paradigm, and amyloid -2 and tau became the main markers of the pathology of the Alzheimer disease. Alpha -synuclein

by itself lacked diagnostic specificity in the synucleinopathies. It was shown that adding alpha-synuclein to ratio based measures including total tau/alpha-synuclein and phosphorylated tau/alpha-synuclein was more effective in distinguishing between Parkinson disease and other neurodegenerative processes (Parnetti et al., 2011). Initial studies indicated that CSF biomarkers would be able to reflect coexisting pathologies. In particular, the alpha synuclein/ p -tau181 ratio has become a possible marker of comorbid Lewy body disease in Alzheimer disease patients, thus narrowing down prognostic estimates of cognitive impairment (Toledo et al., 2013). Tauopathies were distinguished by different CSF tau profiles; for example, progressive supranuclear palsy had lower levels of both N-terminal and C-terminal tau in case of pure neuron damage (Wagshal et al., 2015). Another notable development of this time was the invention of non invasive methods of sampling with a target of overcoming the shortcomings of lumbar puncture. Salivary A2 levels was found as significantly elevated in the cases of mild Alzheimer's disease as compared to controls, suggesting that it could be utilised in the future as non-invasive biomarker in early Alzheimer disease diagnosis and in differentiating between it and other illnesses like Parkinson's disease (Bermejo-Pareja et al., 2010). This was a significant attempt at evidence of scalable biomarker development, although sensitivity and standardization was crude, this was a pivotal demonstration of proof-of-concept that would drive other research efforts.

In the period between 2015 and 2020, the growing interest in clinical diagnostic uncertainty was accompanied by the acknowledgment of the need to support the current diagnostic criteria with biomarkers. Critera of Dementia with Lewy bodies used internationally as a consensus with respect to the condition demonstrated moderate sensitivity and specificity, with scores of 73 and 93, respectively; however, a standard residual false- suggests that the criterion ought to be improved to incorporate biomarkers, which would further enhance diagnostic accuracy (Skogseth et al., 2017). Studies had broadened scope in the research beyond classical misfolded protein paradigms to the neuroinflammation and glial biology. Neuron-specific enolase (NSE) became a reliable indicator of neuronal damage; activated glial cells produced YKL -40, which became a reliable biomarker of neuroinflammation, an exceptionally large expression of this protein in neurodegenerative conditions suggested that glial participation might be more notable than previously identified (Dichev et al., 2020). This change indicated an increase in the significance of the immune-gliial axis as a central mechanistic pathway in neurodegeneration, which has not been limited to protein aggregation. Simultaneously, the appearance of non-protein molecular biomarkers was also a characteristic feature of the time. Cell-free microRNAs in cerebrospinal fluid were described as stable extracellular structures that may be useful in diagnosing and monitoring Parkinsonian syndromes, as an acceptance of the fact that regulatory biology and gene-expression dynamics can be informative equal to structural proteins (Starhof et al., 2018). Moreover, prodromal clinical phenomena were brought closer to the biomarker, and research revealed that self-reports of hyposmia in patients could be used to help diagnose the presence of a Parkinsonian disease even in the absence of objective measures, and the inability to detect hyposmia was linked with cognitive impairment in non-Parkinsonian elderly participants (Shill et al., 2016). The observation presaged the later development of digital phenotyping approaches.

Biomarkers based on blood were consolidated strongly between 2020-2025 along with a growing consideration of digital and functional biomarkers. It was discovered that there existed is a significant, positive correlation between plasma p -tau and plasma A1 -42 concentrations in individuals with Alzheimer disease and cognitively unimpaired controls but not in individuals with fronto temporal dementia or Parkinson's disease, plasma A1 -42 explained about 47.7 percent of plasma p -tau variance in individuals with Alzheimer after confounding factors were controlled (Chiu et al., 2021). These results highlighted disease-specific synergistic effects and shed light on the continued problem of universality biomarkers, since underlying processes vary fundamentally between disorders. Extracellular vesicles have been identified as signals of brain-relevant signals that are measurable in peripheral blood. Myeloid-derived extracellular vesicles isolated in cognitively impaired multiple sclerosis patients exhibited unique microRNA signatures compared to cognitively preserved patients, with increased miR 150 5 p and

decreased levels of let 7b 5p; these results were suggested as possible biomarkers of cognitive impairments across neurological conditions (Scaroni et al., 2022). Yet another important change has been the introduction of digital biomarkers, which are non-invasive, scalable modalities enabling early detection and longitudinal tracking. Recent hypotheses suggest that technologies of virtual-reality and artificial-intelligence are able to create ecologically viable simulations of everyday activities and thus, acquire the kinematics and gait of patients when performing instrumental activities. Clinical and neuropsychological data can be combined with these digital biomarkers by the machine-learning algorithms and determine patterns of cognitive and motor impairment, even in the early stages of the disease (Cavedoni et al., 2020). Biomarkers in the form of speech have been explored in a continuum of neurodegenerative diseases. Patients with mild impaired cognitive as a result of the Alzheimer disease have reduced application of functional words and higher content density; patients with the Parkinson and mild impaired cognitive diseases show abbreviated sentences with longer pauses; and patients with the Lewy body impaired mild cognitive impairment show increased lexical repetition (Kovac et al., 2025). Empirical data show that machine-learning models have the capacity to boost the accuracy of classification more than baseline clinical scores, which favors the possibility of introducing speech biomarkers into scalable systems of cognitive surveillance.

Taken together, these trends represent a fundemental shift in the goal of research on biomarkers. The focus has moved out of the aim of searching inert biomarkers to dynamic biomarkers that are able to trace subtle decline before the emergence of categorical clinical thresholds. Single markers have been replaced by signatures and panels, and multi-markers are based on ratios and combined analytes. The emphasis of research has shifted towards biological axes cutting across numerous diseases, recognizing that phenotype-specific patterns are more realistic and a more useful clinically than general dementia biomarkers. Most crucially, there is evidence that hybrid diagnostic ecosystems, which combine fluid biomarkers, digital measures, neuroimages, and genetic data, are the next stable direction in the field.

Implications for Researchers

The current review shows that initial biomarker studies used general proteomic techniques to query amyloid- 2, tau, and 2-synuclein in cerebral spinal fluid, but recent investigations are focused on mechanisms, such as blood-based approaches, neuroinflammatory biomarkers, digital biomarkers, and extracellular vesicles. This shifting terrain creates openings to other meta-analyses, which might attempt to relink these discrete strands and be able to assess the interaction in various biomarker modalities between different neurodegenerative diseases. The integration of multi-center and longitudinal data would improve future research by determining how fluid biomarkers, neuroimaging values, and genetic predispositions are matched in population heterogeneous patient groups and evaluating whether minimal invasive markers are effective. There is still scanty evidence (gathered mostly in small regional studies) that is not yet consolidated to determine unequivocally the generalizability and permanence of the effects observed in different clinical settings.

Implications of Social Support

The reviewed evidence suggests that people with neurodegenerative disorders often face late diagnoses, limited access to specialized tests, and a lot of caregiver burden due to the progressive nature of these disorders. This gives policy makers and health-care infrastructure a motivation to expand the access to validated biomarker testing so that testing does not require an invasive lumbar puncture, thus reducing the obstacles to early diagnosis in the primary-care and underserved locations. Patient advocate groups and caregiver networks could supplement such efforts by providing educational materials on the importance of biomarker testing, as well as, assisting families along the diagnostic process.

Practical Implications

The results have obvious implications to the stakeholders involved in the management of neurodegenerative disorders. Validated blood-based biomarkers, including plasma p -tau181 and neurofilament light chain, can be used by clinicians to improve early detection, differentiate between diagnoses, and monitor disease progression, though expertise in interpretation guideline and awareness of pre-analytical variability is needed. Health-care systems will gain the advantage of investment in harmonized procedures and multi-modal diagnostic systems incorporating fluid biomarkers together with neuroimaging and cognitive testing, and, at the same time, investing in training and quality control in order to have reliable implementation. Patients will have earlier and more accurate diagnoses enabling them to easily get the intervention and plan the care to do but they will face challenges concerning the availability of tests, their cost, and the psychosocial implications of being diagnosed in advance. Potential gains in pharmaceutical businesses and in research teams carrying out clinical trials could be made by using biomarker-based stratification of patients and tracking of the therapeutic effect, although in the context of continuing regulatory complications related to novel biomarkers. By careful understanding of these trade-offs, the development of interventions that maximize benefits among all parties can be developed furthering the goal of precision medicine in the care of neurodegenerative diseases.

Conclusion

This review shows how the field of research on biomarkers in neurodegenerative diseases has changed concerning geographical distribution and conceptual focus. Co-author analysis shows that few institutions, mainly in Sweden (the Sahlgrenska Academy and Skane University Hospital) are highly collaborative and produce most of the leading research on the topic of cerebral spinal fluid markers, blood-based substitutes and multimodal diagnostic models. The co-citation analysis of the proximal complementary nature shows that whereas the initial research on biomarkers concentrated on classical CSF protein markers such as amyloid 2 and tau, recent studies concentrate on smaller processes such as blood-based biomarkers, digital phenotyping, and neuroinflammatory markers. Despite the fact that theoretical and methodological innovation is still concentrated in a limited number of hubs in Sweden, the United States, and the United Kingdom, underrepresented areas are making more contributions. Future studies ought to hencebuild multi-center, longitudinal data to determine the interaction of fluid biomarkers with neuroimaging and genetic data, and also determine how minimally invasive markers can be established over time in a large variety of patient populations. Other areas of priority regarding future research are comparative studies between emerging markers, including extracellular vesicles and cell-free mitochondrial DNA, and disease progression, and additional research on the standardization of pre-analytic protocols to minimise inter-site variation. In practice, clinicians can make use of validated blood-based biomarkers, such as plasma p-tau181 and neurofilament light chain, to improve the process of early diagnosis and differentiation, and health-care systems should invest in the standardized protocols and multimodal diagnostic systems to bridge the gap between research findings and clinical practice. On the whole, it is important to enhance stronger partnership between major research centres and underrepresented clinical populations to generate more biomarker research that has an appropriate representation of diverse patient groups, facilitates more precise diagnostic results and enhances more equitable patient outcomes with neurodegenerative diseases across the globe.

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