High-Throughput Genetics and Neuroimaging Integration for Identifying Neurobiological Alterations in Neurodegenerative Disorders

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Abstract

The integration of high-throughput genomics and neuroimaging technology has the promise of providing more information about neurobiological irregularities in neurodegenerative illnesses. Transcriptomics-derived connections provide insight into the molecular trajectory of neurodegeneration, prioritizing particular systems and networks while also considering other aspects, including neuropathology and cognition. Causal links between gene expression and brain morphology are unknown, however. If omics systems have a wide impact upstream, they can influence morphological changes identified by MRI. Gene expression is a signal indicating a process already underway in a diseased brain area if it is downstream of structural brain changes. More study on people in the early stages of disease may give insights into the temporal connection between anatomical and expression problems.

One such thought is molecular stereotactic propagation. Changes in gene expression may travel across the brain, according to this notion, through tractography trails. They follow the successive pattern of afflicted regions and the temporal distribution of sensitive locations. In addition, cell motility genes are often overexpressed in vulnerable locations. On the other hand, the data on gene expression and its relevance to structural change propagation is still conflicting. The role of immunological processes and motility-related genes in neurodegeneration appears to be validated by expression data.

There are no therapies available for neurodegenerative diseases. Symptom development and diagnosis is often delayed due to advanced MRI and clinical stages. Early diagnosis is crucial since therapy interventions should ideally aim at commencing pathogenic processes as soon as possible to avoid the onset of disease and restrict the course of disease. This requires dependable neurodegenerative biomarkers with diagnostic validity. Unfortunately, transcriptomics still has several important limitations. There is a paucity of high-quality expression data encompassing a large number of brain regions, expression data generally collected from healthy persons, comparisons with neuroimaging data from degenerative cohorts, and a lack of consistent approach for transcriptional imaging research. By overcoming this barrier, researchers can uncover prodromal stages of neurodegeneration and therapeutic molecular targets.

Introduction

In neuroimaging, omics technologies allow for the analysis of large amounts of data from complex networks, as well as the clustering and combining of disparate datasets. They allow researchers to look into the link between neuroanatomical and neuroimaging characteristics and gene expression. 1 The first investigations concentrated on a small number of gene candidates that were examined individually. However, a key drawback of this methodology is that it can only display one candidate gene and its expression at a time, with no ability to follow expression changes at the global expression network level (e.g., including data from upstream and downstream regulators). This limitation can be resolved with next-generation sequencing (NGS), which allows the whole collection of genes and transcripts to be studied simultaneously. Massively parallel, high-throughput sequencing methods for DNA, RNA, and methylation are referred to as NGS. This allows the entire expression network to be viewed at the same time. 2 and 3 Many studies have found some association between neuroimaging data and single nucleotide polymorphisms, a common sort of genetic variation among people that may predict an individual's reaction to specific medicines or sensitivity to environmental influences. Validation of their relevance, on the other hand, remains a challenge. 4–6 More data on spatial transcriptomics has been acquired since the focus of omics research has lately changed from DNA variations to expression analyses. Transcriptomics and neuroimaging data may now be combined to previously unreachable levels because of international initiatives, new algorithms, and a data-sharing culture. 7

The two-dimensional transcriptome of total brain tissue, on the other hand, was thought to be insufficient to explain the focally occurring molecular mechanisms driving neurodegenerative diseases.

8 – 10 The importance of geographical distribution and cellular subdivision in clarifying molecular mechanisms involved in neurodegeneration has been stressed in recent years, and as a result, a notion of expanding zones of susceptibility for neurodegenerative illnesses has been proposed. 11 and 12 We present an update on imaging transcriptomics, a methodology connected with expression and neuroimaging data, in this paper. Recent research on this issue, as well as the relevance of transcriptomics and connectomics in understanding neurodegenerative processes, will be discussed.

Transcriptomics Imaging

The phrase "imaging transcriptomics" describes a method for combining neuroimaging and transcriptomics datasets. Imaging transcriptomics connects genetic and expression data to structural, functional, connectivity, and biochemical changes in the brain. 1 – 13 Large-scale RNA sequencing data may be studied in specific neuronal areas or even the whole brain using this technology.

Furthermore, imaging transcriptomics can reveal how regional differences in gene expression relate to functional connectivity at different levels and at different stages of development, such as with canonical resting-state networks,14, 15 fiber tract connectivity between brain regions,16 temporal and topological properties of large-scale functional brain networks,17 and the specialization of large-scale functional brain networks. 20 – 23 Furthermore, imaging transcriptomics data may be integrated with epigenomes (genome alterations that do not impact DNA sequence; in this case, methylation), connectomes, or phenotypic information; they may be included directly in the native context of tissues with gene expression data. 24 Metabolomics, or the study of tiny molecules known as metabolites on a massive scale, may also be combined.

Neurodegeneration,24 psychiatric diseases,25 neuropharmacology,26 neurooncology,27 and neurotransmission have all used molecular imaging and imaging transcriptomics.28 The majority of research looking into transcriptomics in neurodegeneration focuses on Alzheimer's Disease (AD) and Parkinson's Disease (PD), as these are widespread and well-understood neurodegenerative disorders with readily available neurological databases for a wide audience.

Techniques and Resources

Genetic and imaging atlases use high-throughput technology to link expression data at one or more genetic loci to differences in one or more imaging-derived phenotypes (IDPs).

29 Variants linked with an IDP change gene expression or protein abundance, modify cellular function, and, eventually, impact the examined IDP, according to a prevalent premise in this type of research. The Allen Human Brain Atlas is one of the public datasets that provides high-quality expression and neuroimaging data for the whole brain (AHBA, ABA). 30, 31 AHBA covers 102-103 geographically distinct brain areas in great detail. It comprises almost 20,000 genes extracted from 3,702 geographically different tissue samples from six postmortem brains (five males and one female). 30 Although other atlases provide data on brain expression, they usually rely on limited, particular brain areas—so-called regions of interest (ROI) —that are altered in pathological states. The Alzheimer's Disease Neuroimaging Initiative (ADNI) 32 and the Parkinson's Progression Markers Initiative (PPMI), both consortia established to develop standardized imaging techniques and biomarker procedures in normal subjects and individuals with neurodegeneration, are examples of databases containing neuroimaging and phenotypic data for neurodegenerative diseases. However, the scarcity of high-quality expression data leads to a situation in which huge neuroimaging datasets from afflicted people are paired with AHBA expression datasets from a small number of healthy people, potentially leading to interpretation bias.

Neurodegenerative diseases have clinical and neuroimaging features.

Alzheimer's disease (AD) is the most prevalent neurodegenerative genetic illness, affecting over 44 million people globally.34 Memory loss, confusion, aphasia, apraxia, agnosia, and a change in executive functioning leading to difficulty completing daily regular activities are the most typical symptoms. Mild cognitive impairment (MCI) is a major predictor of Alzheimer's disease (AD) and is often seen as a prodromal stage. Despite the fact that the memory impairment is above what is expected given the individual's age and education, these individuals exhibit no indicators of dementia and their everyday activities are unaffected. 35 On a clinical/neuroimaging level, persons with Alzheimer's disease have a variety of distinct MRI characteristics, including atrophy in common locations: Slowly progressive localized cortical atrophy, which usually starts in the hippocampus and eventually spreads to the temporal lobe and posterior cortex. The bilateral hippocampus and amygdala in MCI patients reveal substantial shrinkage and microstructural alterations. 36

Parkinson's disease (PD) is a movement illness that affects the neurological system. Aside from the normal clinical symptoms of rigidity, tremor, and bradykinesia, afflicted people experience cognitive deterioration, confusion, and memory loss, as well as occasional visual hallucinations, in approximately 30% of cases. 37 T1 moderate hyperintensity of compact and reticular sections of the substantia nigra and red nuclei (due to iron buildup), lack of normal hyperintensity in substantia nigra owing to loss of neuromelanin, and the "absent swallow tail" sign on SWI scans are all common MRI characteristics in PD. Even in cognitively intact individuals, PD is linked with a general decline in cortical thickness and subcortical atrophy. 39

Features of Neuroimaging Correlate with Expression Data

In AD and PD, the MRI abnormalities that may be seen by clinical image interpretation frequently appear in the latter stages of the illness, when irreversible alterations have already occurred. This is why it is important to notice alterations at an early stage, such as MCI, or even earlier. Researchers have been able to partially recreate biological mechanisms that may play a role in the conversion of MCI into AD disease by combining large data from neuropsychological evaluation scores, expression data, single genetic variations, pathways, and neuroimaging-related aspects. Transcriptomics imaging combines expression data with neuroimaging biomarkers to identify and link early stages of neurodegeneration, such as transverse relaxation time (R2), as well as late disease characteristics, such as cortical atrophy.

Transverse Relaxation Time (R2)

Transverse relaxation time (R2) in specific brain areas has been linked to cognitive deterioration in later life. 40 R2 has been found to be decreased in PD41 and AD in several investigations. 42 The consequences of brain iron accumulation in AD and PD appear to dominate R2. 40

R2 values in subgroups with memory complaints were substantially greater in the right temporal cortex and considerably lower in the left internal capsule and left temporal and frontal white matter when compared to healthy controls. This implies that in people with memory problems, the quantity of iron in the temporal cortex rose, and myelin-related iron was lost from multiple white matter locations, which is consistent with incipient AD pathogenesis and biochemical findings. 40 R2 was decreased in people who had macroscopic infarcts or hippocampal sclerosis. Lower R2 was also linked to higher TDP-43 levels, as well as atherosclerosis and arteriolosclerosis. There were no substantial links between R2 and microinfarcts, Lewy body disease, or cerebral amyloid angiopathy, on the other hand. 43

Both DNA methylation and gene expression dysregulation were linked to R2 changes in late-life cognitive impairment, according to a study that combined neuroimaging characteristics with transcriptomics data.43 In two cohorts, the Religious Orders Study (ROS) and the Rush Memory and Aging Project, researchers looked at gene expression and DNA methylation in postmortem human brains, as well as their relationship with R2 changes (MAP). More than 500 patients had MRI data, and over 100 patients had gene expression data from the prefrontal cortex. Frozen tissue from the dorsolateral prefrontal cortex was used to obtain next-generation RNA sequencing and DNA methylation data. For signals related to R2 measurements, genome-wide association analysis was utilized to look at gene expression and, independently, DNA methylation. A variety of correlations were discovered, with PADI2, a citrullination-catalyzing posttranslational modification enzyme, being the top-ranked gene associated with the R2 signal. Citrullination of myelin basic protein, in particular, destabilizes the myelin structure. ZNF385A (Ensembl ENSG00000161642), which encodes one of the transcription factors, PSD2 (Ensembl ENSG00000146005), which is involved in phospholipid binding, and A2ML1, which encodes a protease inhibitor, were among the genes linked to delayed R2 (Ensembl ENSG00000166535).

In a separate study24, the same authors looked at molecular systems, white matter, methylation, and connectomics as well as transcriptomics and neuroimaging data.The study group included thoroughly phenotyped individuals from two longitudinal aging cohorts—ROS and MAP—from which transcriptomics data was generated. The correlations between molecular systems and R2 done ex vivo demonstrate where brain microstructure changes are connected with the average level of the molecular systems on the expression level. Hundreds of genes, including those involved in transcriptional regulation and cell morphology indicators, are influenced by these systems, which play a role in a variety of molecular activities. The genes connected to brain microstructure were preferentially associated with AD symptoms and performed tasks related to cell motility, transcriptional control, and nuclear activities. 24 Surprisingly, at the systemic level, there was no link between gene enrichment and cellular composition. There was also no link discovered between omics and neuropathology.

Atrophy of the Regions

Many neurodegenerative disorders are characterized by regional atrophy. A link between the network of gene expression and atrophic areas in PD was investigated by Freeze et al44. Two large datasets from the Parkinson's Progression Markers Initiative (PPMI) MRI component were examined for regional PD atrophy patterns. 44 T-scores were calculated using MR volumetric data to measure the atrophy. Seventeen genes linked to Parkinson's disease were studied on a whole-brain basis. Four genes involved in transsynaptic synuclein spread were investigated (LAG3, APLP1, NRXN1, and RAB5A). The expression of LAG3 (positive correlation) and RAB5A (negative correlation) corresponded with the PD brain MRI pattern. Additionally, NUSK1, a gene connected to brain shrinkage that has been connected to DNA damage but has yet to be completely defined gene function, has been found. In patients with hallucinations, high-expressing LAG3 areas in the occipital brain showed significantly greater atrophy, suggesting that this gene may be particularly crucial in mediating the visuospatial abnormalities that can occur in PD. Surprisingly, well-known PD genes including alpha-synuclein, tau, GBA, PARK, and SYNK did not appear to be linked to the atrophic pattern. One theory is that they operate as disease initiators rather than becoming dysregulated later in the illness, when macroscopic atrophy begins. 44 In all, various expression patterns were observed in various people for the analyzed collection of potential genes. This shows that, while these genes may all play a role in PD risk or etiology, gene expression is very variable among brain atrophy patterns.

Acosta et al45 analyzed MRI-derived regional atrophy quantified as a T-ratio from the ADNI database in conjunction with AHBA expression data in another investigation. Three genes—APP, MAPT, and PRNP—have a low but substantial correlation with regional expression patterns. However, they have a limited predictive value for regional atrophy in Alzheimer's disease. The greatest risk factor for Alzheimer's disease, APOE, did not contribute as a predictor of regional atrophy in AD. The Network Diffusion Model, a model of disease and protein transmission across brain networks under connectivity-driven processes, was found to be a stronger predictor of atrophy than regional gene expression, according to the scientists. Transsynaptic transmission genes were shown to be stronger predictors of cross-sectional atrophy than other genes linked to Alzheimer's disease. Transmission of protein products from the identified gene's regional expression did not adequately simulate regional vulnerability in Alzheimer's disease in this investigation. The study's main flaw was that it examined two separate MRI cohorts (ABA and ADNI), with the expression data from the ABA cohort having just a small number of healthy people. Another confusing aspect might be that atrophy was only detected in the late stages of Alzheimer's disease.

Epigenome is a term that refers to a

Epigenome data, particularly methylation data, is frequently explored in conjunction with neuroimaging and expression datasets. In research by Yu et al.43, no clear link between methylation and R2-related cognitive deterioration was discovered. Although there was no apparent association between methylation and R2 across the genome, it did demonstrate an inverse connection in several of the cytosine-guanine dinucleotides. This pattern implies that the link between DNA methylation and R2 isn't limited to a single locus. The authors came to the conclusion that a bigger cohort was needed to investigate this phenomenon further. Another research looked at methylation patterns again and compared them to changes in neuroimaging and the connectome. 24 The methylation-related white matter areas were larger than the co-expression-related white matter areas. Although expression-related areas were predominantly detected in the frontal lobes, methylation-related areas were discovered in both the temporal and frontal lobes, which are normally linked with Alzheimer's disease pathology. Methylation-associated white matter regions were anticipated to link the gray matter areas. R2 was linked to methylation patterns in a wide variety of frontal brain regions, as well as certain temporal and parietal locations. The correlation between co-methylation modules and phenotypic parameters was also found. 24 For various brain areas in Alzheimer's disease, a relationship between expression, methylation, and R2 has been shown.

Focal Changes' Spreading Potential

Correlating neuroimaging and expression data can be used to examine a notion of localized alterations spreading in a stereotactic prion-like pattern following network projection. 12, 46, and 47. In AD and frontotemporal dementia, as well as matching T1-weighted MRI volumetrics, the connectivity-based model of spread based on tractrography was seen. For AD, the spreading pattern followed the classic Braak pattern of progression, beginning 3 years prior to conversion in the anterior medial temporal regions, spreading to nearby temporal and parietal cortices, and eventually encompassing the classic temporo-parieto-frontal AD pattern after an overt AD diagnosis. 46 As a result, rather than being spread by proximity, the illness is spread through neural circuits. A similar noncell autonomous activity might be implicated in the pathogenesis of Parkinson's disease. In a research by Keo et al.48, susceptible areas and MAPT and SNCA microarray expression data in healthy participants from the ABA were linked with functional MRI and connectome in progressive supranuclear palsy, PD, and healthy patients (n = 128). The link strength in PD and executive cognition were connected with the regional expression of MAPT, the tau gene's haplotype variation. 28 The findings imply that MAPT expression spreads over a global neural network in a characteristic temporal and particular order, raising the potential that gene expression patterns and their products contribute to distinct brain networks' vulnerability to various neurodegenerative disorders.

According to scientists, areas with higher MAPT gene expression are more sensitive to neurodegeneration caused by the MAPT haplotype or tau pathology, and they are also better related to other brain areas in both PD and healthy individuals. The global network spreading pattern, on the other hand, did not correlate with the haplotype in the alpha-synuclein gene, suggesting that alpha-synuclein disease is not mediated by global network features. 48 In people who were not affected by any neurologic condition, the alpha-synuclein expression signature across certain brain areas followed an interesting pattern, with the lowest expression in preclinically implicated areas (brainstem) and the highest expression in the limbic system and cortex (which are affected first in PD individuals). The idea of specific network sensitivity to neurodegenerative diseases is supported by combining expression and imaging data. The authors build on their notion of highly linked hub areas' propensity to change in a variety of neurological and neuropsychiatric illnesses, including Alzheimer's disease.49, 50

To back up the neurological spreading idea, transcriptional imaging data indicated that genes involved in motility were overexpressed and sensitive to neurodegeneration in the afflicted regions. Ex vivo R2 was linked to the methylation of molecular systems involved in cell motility in research by Gaitieri et al24. Two genes implicated in synuclein transfer, LAG3 and RAB5A, were shown to have expression patterns that are strongly predictive of PD regional atrophy in research by Freeze et al44. Genes related to cell mobility, such as MAPT and PRNP (involved in transport and cell-cell communication), were identified as weak but stable predictors of regional atrophy in AD in another study by Acosta et al,45, where there was insufficient proof of the expression levels of subsequent network transmissions. MAPT has also been linked to atrophy in Parkinson's disease. 28

Neuroimmunological Processes Are Involved

Woo et al51 studied brain transcriptome datasets comprising 17 brain areas, linking them with connectome data, using data from the ADNI. AD-associated tracts linked to twenty-eight pairs: L-CAB, L-ILF, L-SLFT, and L-SLFP. Tissue-to-tissue transcriptional synchronization was used to analyze the brain connectome and expression data across 17 brain regions. Genes involved in synaptic transmission were enriched in areas that were altered in a distinctive way in AD, as predicted. TLR signaling pathways were shown to be overrepresented in both tract-bound and AD-associated tract-bound ROI pairings, and this was linked to increased TLR expression in the blood. TLRs are activated in proteinopathies and play an essential role in innate immunity in humans. TLR pathways may be important for region-to-region contact networks, suggesting that the immune system is engaged in Alzheimer's disease-related cross-brain communication. TLR signaling may play a role in region-to-region brain interactions via white matter tracts, according to the author, and routes in brain regions and the blood may be coordinated. Other transcriptome imaging investigations have found a link between genes implicated in neuroimmunological processes, confirming the idea that neuroimmunological processes and neuroinflammation play a central role in the pathophysiology of Alzheimer's disease.

The Studies' Limitations

The lack of high-resolution expression data is the most significant constraint in the analysis of transcriptomics and neuroimaging-omics data. AHBA is currently the primary source of such information. The AHBA databases, on the other hand, have certain flaws. For example, they only cover a small number of genes and are based on the expression of healthy people, whereas neuroimaging data is frequently obtained from sick people. 20 Because gene expression is altered in neurodegenerative illnesses, comparing genetic expression from healthy participants to neuroimaging findings from patients with neurodegenerative illnesses may lead to bias. Furthermore, AHBA only provides data for six people, which is deemed inadequate for large-scale expression investigations. Other public databases include expression data for specific brain areas, albeit their coverage can be inconsistent. Furthermore, when only a few genes are considered in the study, additional significant genes may be ignored. The absence of established techniques makes it difficult to achieve result repeatability. Another stumbling block is the scarcity of good neuroimaging measures of neurodegeneration at an early stage. The characteristic of numerous neurodegenerative illnesses is atrophy, which occurs mostly in the latter stages of the illness, although early stage indicators are the focus of scientific research. More research, with larger cohorts and consistent methodology, might lead to more certain results.

Conclusion

The combination of high-throughput genomics and neuroimaging technologies offers the potential to provide a more comprehensive knowledge of neurobiological anomalies in neurodegenerative disorders. Transcriptomics-derived associations provide light on the molecular course of neurodegeneration, prioritizing certain systems and networks while also taking into account other factors, including neuropathology and cognition. However, the causal relationships between gene expression and brain shape are yet unknown. If omics systems have a broad impact upstream, they may be able to influence morphological alterations detected by MRI. Gene expression is a signal for a process that is already underway in an afflicted brain region if it is downstream of structural brain alterations. More research on people in the early stages of illness might shed light on the temporal link between anatomical and expression abnormalities.

Molecular stereotactic propagation is one potential notion. Changes in gene expression may propagate through the brain through tractography trails, according to this theory. They follow the disease's sequential pattern of affected regions as well as the temporal distribution of susceptible spots. Furthermore, genes involved in cell motility are frequently overexpressed in susceptible areas. The evidence on gene expression and its relationship to the propagation of structural alterations, on the other hand, is still contradictory. It's worth noting that the influence of immunological processes and motility-related genes on neurodegeneration appears to be confirmed by expression data.

For neurodegenerative illnesses, there are no disease-modifying treatments available. The onset of symptoms and diagnosis is frequently delayed due to advanced MRI and clinical stages. Early diagnosis is critical since treatment interventions should ideally target the initiating pathogenic processes as soon as feasible in order to prevent illness starting as well as limit disease progression. This necessitates the development of reliable neurodegenerative biomarkers with diagnostic validity. Unfortunately, there are still a few key limitations to imaging transcriptomics. There is a scarcity of high-quality expression data spanning a large number of brain areas. Expression data is typically drawn from healthy people, comparisons with neuroimaging data from degenerative cohorts, and transcriptional imaging investigations lack a uniform technique. By overcoming this barrier, researchers may be able to detect prodromal phases of neurodegeneration and identify therapeutic molecular targets.

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