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How biological elements interact with language: The biolinguistic inquiry

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TABLE OF CONTENTS

1. ABSTRACT

Biolinguistics realizes a scientific approach to study language both as a biological object (the language faculty) and an internal, intensional and individual language system (I-language), spurring a cross-disciplinary exploration of the biological nature of human language. The poverty of stimulus (POS) in language acquisition, together with the roles played by neurobiological factors in linguistic aphasia, specific language impairment and mirror deficits, confirms the biological nature of the language faculty and I-language. Based on the property, the classic molecular genetic study reveals how human genetic endowments canalize the development of human language, and they interact with specific linguistic experience during the maturation of human language. Further, the rapid development of biological research promotes an increasing emphasis on a more nuanced molecular network system, along with the existing interest in one-gene-one-behavioral phenotype. Thus, a synthetic perspective on the study of the biological part of language will function as a new departure for the incoming biolinguistic inquiry.

2. INTRODUCTION

Since the publication of Lenneberg's (1) classic *Biological Foundation of Language*, the resemblances and discrepancies between human language and other biological communication systems, such as ape signal systems, bee communication, have been explored in depth. The approach to study the human language as a biological object or an I-language in a biological context, viz., biolinguistic research1[1], has concentrated on the verification of a basic hypothesis. To wit, the primary object in the study of the human language is "the Language Faculty", a mental organ that facilitates the acquisition of linguistic knowledge (e.g., I-language) and use of language (2, 3). In this sense, the human language is fundamentally different from other biological systems in terms of several essential properties, although mature language systems (I-languages) and linguistic performance are superficially similar to certain animal communication systems. In other words, the distinct biological elements (e.g., neurobiological basis and genetic endowments) contribute to the formation of human language core—"language faculty in the narrow sense" (FLN)[2]— at 50, 000 to 80,000 years ago (4, 3). Unique to human as the FLN is, it, along with the "language faculty in the broad sense" (FLB)[3] (4), also attributes to the stable maturational route map, and interactions

between the maturational processes and the specific experience that is indispensable to reach the exact species-specific final stage.

Up to now, with the rapid development of biological research, the progress made in genetics, biological techniques for behavioral research and the investigation of connections between brain properties and linguistic capacities have greatly improved the knowledge of the classic issues. Along this line, it is helpful to examine the achievements and lessons in the study of the biological part of language, clarifying the classic issues against current scientific advancements. This will pave the way for the further research of language both as a biological object and an I-language in the generative tradition of biolinguistic paradigm, especially, in the current Minimalist framework.

The paper is organized into five parts. The third part exemplifies the neurobiological foundation of human language—the language faculty in both the narrow and the broad sense and I-language—in different stages of the generative linguistic inquiry. The fourth and the fifth parts respectively discuss the classic molecular genetic interpretation of and the new approach in genetic studies of the same targets in the generative tradition such as the Minimalist framework. Conclusions are provided in the last part.

3. NEUROBIOLOGICAL FOUNDATION OF LANGUAGE

As is well-known, the reason why birds are able to fly is the same as the fact that human beings are capable of speaking. Specifically, the similarity ascribes to the specific neurobiological basis which attributes to the genetic endowment (5). Chomsky (2, 6) credits human linguistic capacity with the human mental organ, particularly with the FLN, a recently-evolved computational mechanism of recursion due to the species-specific biological endowment. Language development thus starts from the initial state—a Universal Grammar (UG), a kind of biological properties initially set in the language faculty and consisting of invariant principle (Merge) and an inventory of universal linguistic features (7, 8). Language-specific grammars or I-languages grow up from UG based on primary linguistic data available to language learners.

One piece of evidences to support this assumption is that children can master their native tongues given a rather slender database or primary linguistic data (9). That is to say, even though the POS seems obstructive to the language acquisition, children are able to acquire their mother tongues with much less effort, no matter for the acquisition of spoken or sign languages. For instance, Hellen Keller becomes deaf and blind at the age of 19 months, but she earns a Bachelor of Arts degree and becomes a successful writer later (10).

Some researchers report a case of Simon's acquisition of sign language (11). Simon is a deaf child and his deaf parents both learned American Sign Language (ASL) after the age of 15. The only ASL inputs to Simon come from his late-learner parents. A study compares 7-year-old Simon's performance with eight children who have native signing parents, and compares with his own parents. The results show that Simon's production of ASL substantially surpasses that of his parents, and equal to that of children exposed to a native signing model. But Simon's parents, like some other late learners of ASL, perform below adult native signing level, with many errors in their use of ASL morphology. These cases exhibit that the earlier the acquisition occurs, the better the linguistic competence is attained, even if the linguistic input is defective. In other words, it is the neurobiological maturation of the language faculty in the narrow sense that decides the acquisition of linguistic competence and I-language. Meanwhile, as a researcher attests, the intrinsic linguistic properties identified as neurobiological endowments in the generative tradition, like recursion, also hold in the sign language under the Minimalist framework (12).

Proceed along this line, the examination of the biological part of human language has closely related to the neurobiological studies. And only in this way can a comprehensive exploration of "language" as both a biological object and neurobiologically determined mental structures be reached. As exhibited through fMRI by Shultz *et al.* (13), neural responses to speech and biological non-speech

sounds in 1- to 4-month-old infants are different. They find out a left-lateralized response in temporal cortex for speech, compared with biological non-speech sounds by 1 month of age. That is to say, at that time, human cortical circuitry defining both FLN and FLB is specialized for processing speech. And during the next 3 months the brain region becomes increasingly selective for speech as neural substrates become less responsive to biological non-speech sounds. This type of neural specialization for language is also revealed by other researchers.

Brederoo *et al.* (14) investigate global and local lateralization. As the research reveals, the two types are triggered by non-linguistic stimuli presented in the left visual field and linguistic stimuli in the right visual field respectively. The brain undergoes a left hemisphere (LH) specialization for local detail, and a right hemisphere (RH) specialization for global form. They verify that the global and local lateralization could be independently modulated. And the local lateralization accounts for a robust phenomenon, yet the global lateralization can be modulated by stimulus type. As is clear, the difference can date back to their own different neurobiological underpinnings, namely, the neurobiological endowments have shaped the human-unique FLN, which locates on LH and fixes the local lateralization.

It is also found that the latency or insufficiency of the neurobiological endowment is closely related to linguistic aphasia (15, 16), specific language impairment (SLI) (17, 18, 19) and “mirror deficits” (20). Hoshi *et al.* (21) revisit Lenneberg’s (1) biolinguistic framework and view on child aphasiology. Specifically, Lenneberg’s original version of the critical period hypothesis (CPH, language acquisition is constrained by neurobiological maturation of the language faculty) and child aphasiology demonstrates a child aphasia of epileptic origin (Landau-Kleffner syndrome, LKS), and indicates a possibility of recovery of the disease. On the other, the language disorder in LKS functions as living evidence for Lenneberg’s view on CPH and child aphasiology.

Besides, mirror deficits can be regarded as the solid evidence for the neurobiological nature of the language faculty and the modularity of language (20). In detail, mirror deficits indicate that the general cognition is below normal, but language is intact, thus, the mirror case of language-specific deficits. Williams Syndrome (WS) is a case in point to prove how cognitive disorders mirror linguistic capacities.

The WS is a neuro-developmental disorder. WS subject suffers from cognitive deficits, e.g., visuospatial processing ability, counting, planning and implicit learning are severely impaired, and their social communication is inappropriate, even though they seem extraordinarily friendly (22, 23). Researchers attribute WS to a genetic anomaly: A hemizygous microdeletion on the long arm of chromosome 7 (7q11.23), affecting 25-28 genes (24). Basically, the cognitive disorders are relevant to an elastin gene (*ELN*). In detail, both the gene for elastin and an enzyme called LIM kinase are deleted. The genes map to the same small area on the chromosome. In normal cells, elastin is a key component of connective tissue, conferring its elastic properties. Mutation or deletion of elastin lead to the vascular disease observed in WS; Further, LIM kinase is strongly expressed in the brain, and deletion of LIM kinase results in the impaired visuospatial constructive cognition in WS (25). Recently, more cases are reported about the WS subjects concerning 7q11. 23 microdeletion including the fragment deletion mutation of elastin gene and 7q11. 21q11. 23 deletion (26, 27).

But the language developmental sequence of WS subjects is significantly normal, even if their language development is delayed throughout childhood. For instance, by adolescence, their linguistic production and comprehension are strikingly delicate and complicated, and their spoken language goes with rich morphological and syntactic structures, including passives, embedded relative clauses and a range of conditionals (28, 29). In this case, even if the deletion of a set of genes brings about the low general intelligence, the remarkably sophisticated linguistic abilities are not affected (30). As a result, this provides a unique opportunity to identify genetic factors and the neurobiological basis that are directly involved in language ability (concerning both FLN and FLB) (31, 32).

Subsequently, researchers have probed into some robust linguistic phenomena revealing the relationship between neurobiological basis and

language disorders/ impairments (e.g., SLI) via the methods in molecular genetic studies. SLI refers to the failure to produce grammatical and inflectional morphemes, including SLI1 and SLI2. The former relates to phonological working memory (phonological SLI); The latter is related to grammar, such as syntactic SLI, semantic SLI, lexical SLI and pragmatic SLI (33, 34, 35). They are characterized by intact non-linguistic cognitive and perceptual abilities, but clear syntactic deficits (36, 37), e.g., a specific impairment in syntactically-driven lexical acquisition. Some researchers, based on the analysis of grammatical SLI, argue for the existence of a genetically- and neurologically-determined specialized mechanism required for normal development of grammar (38, 39, 40, 41, 43). Other researchers rely on the analysis of 'optional infinitive' (OI), putting forward that the errors in the production of grammar are closely connected to the neurobiological basis (44, 45, 46). It is because the simplification or omission of morphemes is very specific and not created by children, e.g., to replace finite forms with infinitive ones (**Her have* a big mouth.). Accordingly, it is a universal maturational-developmental neurobiological process that normal children undergo. Following this logic, Falcaro *et al.* (47) explain the link between the tense development to a region SLI2 of chromosome 19, and SLI1 to chromosome 16 (also see The SLI Consortium (48, 49). Cautiously, as some researchers point out that there is a chromosomal region somehow related to the optional infinitive genotype, perhaps to the growing away of the computational constraint (34). Others identify SLI with FOXP2 (forkhead box P2) ((50); for different views, see next section).

4. CLASSIC MOLECULAR GENETIC EXAMINATION OF LANGUAGE

As revealed in the various research, the genetic correlates of language disorders and neurodegenerative disorders can be uncovered via examination of correlations between genetic loci and language disorders. For instance, a disruption of one copy of FOXP2 causes severe speech and language disorder observed in the British KE family suffered from WS (51). In this case, to expound the phenotypic complexity of language and speech impairments is closely related to the developments in molecular genetic analysis. Although it is a principal challenge to unravel the genetic 'truth' underlying the phenotypic complexity, the past years have witnessed rapid technical progress in the genetic study of human language disorders (52, 53). This has set a solid foundation for the promising molecular investigation that aims to explain the language and speech impairments. This type of analyses reveals the situation of molecular genetic study of the language faculty and I-languages in the current Minimalist Program of biolinguistic paradigm.

As is well known, there are about 25, 000 genes in the human genome, which are shared by all humans, but only with different forms of combinations. Among the human genome, only about 5% of the human genome encodes for proteins. 95% of the genome has regulatory functions and dominates the activation of structural genes at certain proper time and places. Classically, as indicated in the early genetic findings, e.g., in Mendel's genre, a single mutation of gene causes a corresponding disease (52). Along with this trend, researchers try to justify a genetic-linguistic correspondence during the analysis of language and/or language disorders.

The SLI Consortium (48) lists out some specific chromosomal regions or genetic loci for specific language impairment. Specifically, 16q24 affects non-word repetition, and 19q13 is connected with expressive language. Further, molecular genetic analyses of developmental dyslexia have indicated loci on various chromosomes, like 2, 3, 6, 15 and 18 (although no risk gene has been identified. cf. (54)). Meanwhile, 13q21 is related to dominant reading impairment and 2p22 to recessive reading impairment, and one of the most consistent findings of molecular studies of autism is linkage to a locus on 7q31, also referred to as *AUTS1* (55).

A first census has been reached by Stromswold (56). 11 chromosomes and 25 genes are examined to be relevant to language disorders. In detail, 8 or 9 loci are found to be linked with written language disorders, such as 1p34-36, 2p15-16, 3p12-q13, 6p21.3, 6q12-13, 11p15.5, 15q21, and 18p11.2 (the additional one is 7q32 (57)). And 13q21-22 regions are also linked to specific language

impairment in non-ASD (autism spectrum disorder) groups (58), but CNTNAP2, a gene associated with specific language impairment /normal language development, is later shown to be the most likely candidate responsible for the linkage between SLI and ASD (59). Two novel chromosomal loci, 15q23-26 and 16p12 are identified for both SLI and ASD, more specifically, the two linkage signals showed specificity for oral language impairments for 15q and for written language impairment for 16p (60).

The orthographically-based (or surface) dyslexia and the phonologically-based dyslexia are associated with a candidate gene at the 15q21 locus and the 6p21 locus respectively (61). And the spoken language impairments have also been found to be linked with 6 loci or genes, including the FOXP2 gene on 7q3 (51), a region near the CFTR gene at 7q31, a region near D7S3052 at 7q31, 13q217 (62), a locus at 16q24, and a locus at 19q13 (48). What's more, loci at 2p22 (62), at 1p36, 2p15, 6p21, and 15q21 are unraveled to relate to the spoken language impairment (63). Furthermore, some cases report that there are mutations associated with spoken language impairments that refer to loci at 15q13, 1p22 and/or 2q31 (64).

Along with this tendency, researchers further investigate whether the genetic factors affect all aspect of language and how language-specific these factors are through the analyses of dyslexia and SLI. Specifically, many loci are linked to dyslexia, such as 1p34-p36, 2p12-p16, 3p12-q13, 6p21.3-p23, 6q13-q16.2, 7q32, 11p15.5, 15q21-q23 and 18p11.2 (65). Meanwhile, loci are argued not to be specific to types of dyslexia, i.e., 15q does not mean orthographic impairment and 6p does not mean phonological impairment; Likewise, most loci are also linked to other neuropsychological disorders, e.g., 2p15-p16 is associated with schizophrenia, 7q32 with autism and 18p11.2 with bipolar disorder and schizophrenia (65). Such being the case, some researcher doubts whether these bi-linkages are merely coincidental given that the loci encompass thousands of genes (56). This question is intensified with the investigation of SLI in molecular genetic studies.

On the other, several studies display that FOXP2 gene is not a common cause of SLI (66), and other spoken language impairment loci exist, including D7S3052, CFT2, 13q21, 16q24 and 19q13 (65). For instance, 13q21 and 19q13 have been linked to autism respectively. These studies suggest that there is no simple relationship between dyslexia or SLI and subcomponents of language system or the language faculty (65), which pushes researchers to re-investigate the molecular genetics of language.

Even though the research foretells an innovation in the molecular genetic study of language, the genetic factors undoubtedly play a non-negligible role in language. For example, twin studies show that genetic factors affect language development. Some researcher, based on Perinatal Environment & Genetic Interactions (PEGI) study, demonstrates that 68% phonological disorder attributes to genetic factors, and 40% lexical disorder and 69% syntactic disorder are connected with genetics (67). In other words, the corresponding percentages are related to environmental factors, and the genetic factors function maximum for syntax but the minimum for the lexicon. This conclusion is contrary to the Deviant Linguistic Environment Hypothesis (DLEH), which argues that it is difficult to separate the role of genetics and environment (20). For instance, its predications of SLI are not borne out—the most type of impairment as their relatives.

The above research shows that some genetic factors are not specific to language, and some are specific to language or parts of language, e.g., genetic factors influence articulation and syntax but not vocabularies, or influence syntax but not articulation. Even so, the role played by genetic factors in language is not trivial. And it can be safe to say that genetic factors affect all aspects of language in proper time and places. This indicates the decisive role of genetics and environmental factors over language development. To wit, perinatal shared environment affects language and motor development, because perinatal factors are mainly biological ones that affect neuro-development. While postnatal shared environment affects cognition because postnatal factors are mainly psychological (65). The idea is also supported by the successful acquisition of mother tongue by a child who keeps silent at an early stage, without negative evidence (correct, repeat, etc.) and language production at the

early stage (i.e., the child experiences 16-month language delay at early stage of language acquisition) (68). This evidences that language production is not necessary for the child who is mute at the early stage to completely master syntax, morphology, phonology and lexicon. More specifically, even if the child who is mute at very young age had a severe disorder of language production, the child still had apparently normal language comprehension and metalinguistic knowledge, because all milestones are acquired on schedule (20). As a result, compared with the psychosocial environment, the biological factors are more important for children's language development. Also, this favors the modularist theories of language, i.e., language is distinct or dissociable from general cognitive ability (68).

The genetic basis for modularist views of the language is reinforced by the exploration of the endophenotypes in syntactic developmental disorders (69). Basically, endophenotypes refer to the cognitive, neuroanatomical, neurophysiological, endocrine, or biochemical quantifiable components of the space between genes and diseases (70). Researchers show that the 'syntactic fingerprints', characterized by the child's ability to combine syntactic items (words or morphemes) in real samples of speech at different stages of development, confidently reflect how the typically developed faculties of language unfold within the child's mind (69). This is due to different language faculties being implemented in the child's mind. Most importantly, this results from different brain architectures emerging from different molecular backgrounds, e.g., gene mutations and changes in protein homeostasis.

One of the most attractive molecular genetic studies of language comes from the continuous interest in the investigation of the relation between FOXP2 and linguistic and cognitive disorders occurred in the members of British KE family. The KE family, a multiplex family with AD (Attention-Deficit) disorder, suffer from speech dyspraxia (difficult to make the complex, oral motor movements necessary for speech) (71, 72), grammatical deficits (73), also low nonverbal IQ and nonverbal learning disorders (74). PET and fMRI analyses of the affected members of the KE family have revealed severe abnormal bilateral development of the caudate nucleus; and the "core" of the KE family deficit has been identified as an impairment in tracking and producing sequential movements, rhythm, procedural learning or working memory (75, 76). For instance, KE family members exhibit the words/non-word repetition task deficits (77).

FOXP2, as one of the largest and most complex regulatory genes currently known, locates in the locus of chromosome 7 in the q31 region (34). The brain regions under its control, e.g., caudate nucleus and cerebellum are devoted to motor control and sequential learning (perhaps working memory). In this case, FOXP2 is connected to grammatical impairments via effects on selective brain regions, because the loss of one functional copy of FOXP2 in humans affects language and speech (78). The language disorders result from the orofacial dyspraxia caused by the mutation of FOXP2, i.e., in one copy of the gene only, in exon 14, a G goes to an A—arginine goes to histidine (51, 20). Some research shows that impaired phonological analysis resulting from the poor subvocal rehearsal of incoming speech could interfere with the ability to draw analogies between words with articulation patterns in common and, particularly in a developmental context, to learn implicitly the rules of syntax (79). Thus, a clear-cut, one-gene-one-behavioral phenotype is assumed at the moment (34).

Researchers testify that FOXP2 closely connects to and fully down-regulates CNTNAP2 (contactin associated protein-like 2, or a gene that encodes a neurexin and is expressed in human cortex development) (80). Based on the involvement of the genetic FOXP2-CNTNAP2 pathway into the language capacity, other researchers probe into whether a common variant of CNTNAP2 (rs7794745) is relevant for syntactic and semantic processing in the general population (81). Specifically, through examination of 49 healthy adults' visual sentence processing paradigm and their ERPs, they find that AA homozygotes (homozygous for the A allele) and T-carriers (T for Thymine) exhibit a standard N400 effect in terms of semantic anomalies, but the

reaction to subject-verb agreement violations (morphosyntactic mapping violations) vary from AA group to T-carriers. That is, T-carriers present an anterior negativity preceding the P600 effect, while AA homozygous people only display a P600 effect. These results show that the neuronal architecture of the human language faculty is shaped differently by genetically determined effects.

Even though the molecular genetic investigation of the language faculty is revealing, some research suggests that there is something more than the one-gene-one-behavioral phenotype hypothesis. For example, the spoken language impairment loci connected with dyslexia are also linked with other neurodevelopmental disorders. The WNT2 of 7q31 has involved in autism (82). The D7S2459 loci at 7q31 are near the *IMMP2L* gene that has been implicated in Tourette syndrome (83). What's more, even if the FOXP2 mutation disengages from affectedness in the KE family, the affected family members also suffer from speech dyspraxia, grammatical deficits, depressed nonverbal IQ, and developmental learning disorders that do not appear to be verbal in nature (56). Thus, it seems unclear of the phenotypical specificity resulting from the genetic mutation.

In addition, the evolution and development (Evo-Devo) study of the language faculty also intensifies the concern. If it is reasonable that the Evo-Devo is a departure from standard neo-Darwinism rather than an extension of it (20), the adaptationist neo-Darwinism doctrine of natural selection might not facilitate the molecular genetics to address the Evo-Devo of the language faculty. In the new interpretation of Evo-Devo, discrete variation at a finite number of key points results in major morphological variations, which is based on the conservation of genes and gene complexes. As Christiane Nüsslein-Volhard (84), a Nobel laureate in Physiology or Medicine 1995, points out in her Nobel lecture that many drosophila genes have been shown to have homologs in vertebrates, and this homology is not restricted to amino acid sequence and to their biochemical function, but extends to the biological role played in development. Thus, genes are considerably conservative. And genes are probably more often followers than leaders in evolutionary change, and phenotypic novelty is largely reorganizational rather than a product of innovative genes (85). This idea agrees with another view—the natural selection is just one, and maybe not even the most fundamental source of biological order (86). Thus, evolutionary-genomic studies show that natural selection is only one of the forces that shape genome evolution and is not quantitatively dominant, whereas non-adaptive processes are much more prominent than previously suspected (87, 88). In this case, it seems right to say that there is a universal genome (89). Even if the mutation is random, its retention in a genome often is not, and the phenotypic variation is also not random. As a result, the evolutionary of the language faculty might not attribute to the new gene but new regulations of preexisting genes, which are often modular (because binding to a particular enhancer may be independent of transcription-factor-binding at another enhancer) (20). In Sean Carroll's (90) metaphor, to teach old genes new tricks. In all, the majority of language disorders or impairments and evolutionary facts concerning human language reveal that all the relevant issues are genetically related to many genes and connected to a whole spectrum of molecular genetic mutations. It is this complex interaction and other gene-environmental factor (s) that usher in the new approach to examining the molecular genetics of language.

5. NEW APPROACHES TO GENETICS OF LANGUAGE

As indicated in the previous section, the molecular genetic study of the language faculty in both the narrow and the broad sense and I-language has witnessed an increasing emphasis on the genetics of complex traits besides the existing focus on classical Mendelian genetics. In other words, the foci gradually extend from one-gene-one-behavioral phenotype scenario to a more nuanced molecular network system (91, 92). For instance, the classical approach to language relies on the recognizable pattern of inheritance, such as dominant and recessive modes. However, the new tendency in molecular genetics of language development focuses on multifactorial mode involving multiple genes, environmental factors and their interactions (93, 94). In this case, instead of single gene mutation, genetic

heterogeneity attributes to more than one gene, that is to say, more than one gene contributes to (linguistic) phenotypes.

More specifically, the gene-gene combination influences complex traits (gene-gene interaction); Meanwhile, gene susceptibility is only observed in the presence of specific environmental triggers (gene-environment interactions) (95). Or in special ways, some phenotype is caused by environmental factors only (phenocopies), and modification of histones or DNA sequences is only triggered by environmental factors (epigenetics) (20).

Along this vein, Genome-Wide Association Studies (GWAS) plays an important role in the investigation of the molecular genetics of human language (96). Basically, a GWAS means to quickly scan markers across the complete sets of DNA or genomes of lots of people, finding out genetic variations associated with a particular disease. In this case, if certain genetic variations are discovered to be significantly more frequent in people with the disease against those people without the disease, the variations are considered to be associated with the disease. Cautiously, the associated variants might not bear a direct relation to the disease, but accompanying the real causal variants (20). And the common variants or alleles, identified in all human populations, are known to exist in coding and regulatory sequences of genes, some of which result in susceptibility to complex polygenic diseases. This situation indicates that GWAS in many common diseases has identified multiple genes, i.e., most with a small effect which do not fully explain the trait heritability (97). As a result, multiple SNPs (single nucleotide polymorphisms) are necessarily taken into considerations, or many rare variants, instead of common variants, lead to complex diseases.

Against the trend in molecular genetics, more and more studies have probed into the relation or suggestive causality between genes and linguistic impairment or disorders during the maturation of the language faculty and I-languages. For instance, to examine the shared genetic etiologies, some researchers initiate a genome-wide association study on individuals affected by reading disability (RD) and language impairment (LI) in the Avon Longitudinal Study of Parents and Children (ALSPAC) (98). As the results indicate, for both RD and LI, the strongest associations were found with markers in the gene of ZNF385D and the gene of COL4A2. Also, some risk genes contribute to both RD and LI, such as DCDC2, KIAA0319, FOXP2, CNTNAP2 and CMIP (for the same conclusion see (99)). In addition, the strongest associations with LI are mainly detected with markers in NDST4, while markers on Chromosome 10, 8 and the OPA3 gene have the strongest associations with RD.

Other researchers report a genome-wide association meta-analysis of two large cohorts in terms of reading and language disorders (100). The population-based samples include Australian twins and siblings aged 12-25 (N=1777), and UK ALSPAC (N=5472) as in (98). Specifically, the researchers adopt reading measure of non-word with 3-5 syllables and spelling measures, and implement a GWAS of non-word repetition (NWR) marker of SLI, revealing phonological decoding and orthographic skill of NWR (100). The suggestive association is established between NWR and SNPs in ABCC13 and DAZAP1 (there are 25 potential SNPs in the two cohorts). ABCC13 is the most significant, though, there is no association with reading measures and then it could be an SLI locus. Further, four genes, like CDC2L1, CDC2l2, LOC728661 and RPS15, are significant in both the reading and spelling measures. Interestingly, results across cohorts show variability in individuals. The inconsistency indicates the role played by epigenetics, because age is regarded as an influential factor in (100). Thus, epigenetic mechanisms might be involved in shaping the linguistic phenotype given that language is a complex trait. In other words, the linguistic phenotype can be attributable to the interactions of unchangeable genes with their environment. Thus, the identification of the real language gene (s) will facilitate the investigation of the biology of language and the interactions between genes and environment in language development.

In response to the change in the molecular genetic study of language, the neurolinguistic study of the brain has also seen a paradigm shift, i.e., from inside the neuron to large-scale interactions. In detail, the macroscopic functional stability and high

efficiency of the brain depend on the coherent oscillations of assemblies of millions of neurons, not solely by the properties of a single neuron (101). In this case, although the classic neuroscientific approach emphasizing on the study of single neurons is necessary, it is inadequate for researchers to probe into how to fill the gap between neurons and the brain. To wit, it is ineffective to investigate how to fill the gap between the fluctuations in the biochemical cellular and molecular microscopic activity and the stable and efficient global functional performances of the brain (101). As a result, it is necessary to demonstrate the possible solution to this problem, namely, the answer might come from certain aspects of the dissipative quantum model of the brain, if taking into consideration the fractal-like self-similarity properties of the brain functional activity (101).

6. CONCLUSIONS

The exploration of the biological part of language is closely related to the knowledge of human genetic architecture, neurobiological basis and advancements from other relevant disciplines. As alluded to previously, a more comprehensive understanding of the biological part of human language, such as the molecular genetic basis for the language acquisition, evolution and use, should be implemented interdisciplinarily, involving normal and pathological linguistic phenotype, the molecular genetic theories and techniques, and neurobiological analyses. This trend echoes the newly-established interest on GWAS studies of speech, reading or writing phenotypes from both normal variation and language impairments, supplementary to the existing one-gene-one-behavioral phenotype.

Cautiously, some factors delimit significant large-scale GWAS studies of language as Deriziotis (102) indicates, such as insufficient sample sizes for detecting SNPs with anticipated small effects, the absence of people with developmental language disorders in routine clinical or neuropsychiatric screening in the biomedical fields, and the complex properties of language as a multifaceted phenotype. However, these challenges will not impede the new genetic approach to the study of language faculty and/or I-language, but facilitate the explanation of some (ab)normal linguistic phenotypes. For instance, GWAS in geographical isolates with striking SLI disorders reveals rare variants' or novel genetic contributions to speech or language phenotypes (20). As a result, the question is how to use it and solve the insufficiency when conducting a molecular genetic study of language.

As normally assumed, human beings are endowed with the language faculty (especially FLN), and have the unique capacity to acquire complex I-language in a short time given the POS. To solve this puzzle in molecular genetics, or understanding the biology of human language need integrating much more efforts from different fields, such as the key aspects discussed here. Such being the case, there will be more to explore about the language-related genes in neighboring animal genetic system, i.e., to proceed the study of biological part of language via "concomitant evolutionary analysis" (34), given that neurogenetic pathways involved in human language rely on mechanisms with deeper evolutionary histories (103). Without doubt, a synthetic perspective on the biology of language will disclose more in the near future about how the biological factors interact deeply with the human language faculty in both the narrow and the broad sense and I-language under the Minimalist framework.

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Footnotes: [1]As for *Biolinguistics*, Chomsky (2018-9, pers. comm.) recently points out, "As I understand the term, and have always used it, most of the work we do is biolinguistics—studying language in a biological context, therefore with concerns about acquisition, use, evolution, neural representation, to the extent that they provide evidence about the nature of language. But *Biological Foundation of Language* may interpret the term more narrowly, to refer to work that seeks close integration with biological processes, like the neural basis of language." [2]FLN is a computational system (narrow syntax) that generates internal representations via Merge and maps them into the sensory-motor system and the conceptual-intentional system (4). [3]FLB includes an internal computational system (FLN) and at least two performance systems—sensory-motor system and conceptual-intentional system (4).

Abbreviations: I-language: internal, intensional and individual language; POS: poverty of stimulus; FLN: language faculty in the narrow sense; FLB: language faculty in the broad sense; UG: Universal Grammar; ASL: American Sign Language; fMRI: functional magnetic resonance imaging; LH: left hemisphere; RH: right hemisphere; SLI: specific language impairment; CPH: critical period hypothesis; LKS: Landau-Kleffner syndrome; WS: Williams Syndrome; LIM kinase: homeodomain proteins Lin11, Isl-1 and Mec-3 associated with kinases; ELN: elastin; OI: optional infinitive; FOXP2: forkhead box P2; ASD: autism spectrum disorder; PEGI: Perinatal Environment & Genetic Interactions; DLEH: Deviant Linguistic Environment Hypothesis; AD: Attention-deficit; PET: Positron emission tomography; ERP: Event-related potential; AA homozygotes: homozygous for the A allele; N400: negative-going deflection that peaks around 400 milliseconds post-stimulus onset (semantic ERP); P600: positive-going deflection that peaks around 600 milliseconds post-stimulus onset (syntactic ERP); *WNT2* gene: wingless-type MMTV integration site family, member 2; *IMMP2L* gene: inner mitochondrial membrane peptidase 2 like; Evo-Devo: evolution and development; DNA: DeoxyriboNucleic Acid; GWAS: Genome-Wide Association Studies; SNPs: single nucleotide polymorphisms; RD: reading disability; LI: language impairment; ALSPAC: Longitudinal Study of Parents and Children; NWR: non-word repetition

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