NEUROLOŠKA PODLOGA MUCANJA

NEUROLOGICAL BASIS OF STUTTERING

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Sažetak: *Mucanje je neurorazvojni govorni poremećaj složene etiologije. O uzrocima mucanja se sve više zna, premda neurološka podloga mucanja ostaje još uvijek nedovoljno istražena i dokazana. Sa sigurnošću se može reći kako postoje strukturalne i funkcionalne razlike mozga osoba koje mucaju i osoba koje ne mucaju, a te razlike prisutne su u kortikalnom i subkortikalnom moždanom području. Ovim preglednim radom daje se osvrt na postojeća aktualna saznanja o neurološkoj podlozi mucanja, gledajući teorijske okvire s obzirom na strukturalne temelje mucanja i znanstvenu utemeljenost. Također, dat će se uvid u buduća istraživanja te sukladno tome, u moguće nove teorijske okvire.*

Ključne riječi: *mucanje, neurološka podloga, kortikalna područja mozga, subkortikalna područja mozga*

Abstract: *Stuttering is a neurodevelopmental speech disorder of complex aetiology. Although we are continually learning about the causes of stuttering, there is insufficient research and evidence regarding the neurological basis of stuttering. It can be said with certainty that there are structural and functional differences between the brains of people who stutter and those who do not stutter, and these differences are present in the cortical and subcortical areas of the brain. This review paper provides an overview of the existing knowledge on the neurological basis of stuttering by examining theoretical frameworks with regard to the structural foundations of stuttering and its scientific basis. In addition, this paper provides insight into future research and, accordingly, possible new theoretical frameworks.*

Keywords: *stuttering, neurological basis, cortical brain areas, subcortical brain areas*

UVOD

Mucanje je neurorazvojni govorni poremećaj složene etiologije. Glavni simptomi mucanja vežu se za gubitak kontrole nad govorom koji rezultira pojavom mucajućih netečnosti. Simptomi mucanja mogu se podijeliti na ponovljene pokrete (npr. ponavljanje sloga) i na fiksacijske položaje (Onslow, 2025). Ponovljeni pokreti društveno su najpoznatiji te su ujedno i prva asocijacija na mucanje. Fiksacijski položaji odnose se na izostanak tipičnih govornih pokreta uslijed čega (najčešće) usnice, čeljust i jezik ostaju nepomični. Trajanje fiksiranog položaja može biti kratko, a ako traje duže od pola minute postaje komunikacijski najviše nelagodno. Ako je fiksacijski položaj popraćen čujnom zračnom strujom, to nazivamo produljivanjem (Onslow, 2025). Ako se radi o nečujnom fiksiranju položaja, tada to nazivamo blokadom (Onslow, 2025). Simptomi mucanja uključuju i vanjska ponašanja koja se mogu podijeliti na verbalna i neverbalna ponašanja (Onslow, 2025). Mucanje se najčešće javlja između

INTRODUCTION

Stuttering is a neurodevelopmental speech disorder of complex aetiology. The main symptoms of stuttering are associated with loss of speech control that subsequently results in stuttering. The symptoms of stuttering can be categorised into repeated movements (e.g., repeating syllables) and fixation positions (Onslow, 2025). Repeated movements are the most socially recognised and are often the first signs associated with stuttering. Fixation positions refer to the absence of typical speech movements, during which (most often) the lips, jaw, and tongue remain motionless. The duration of a fixed position may be brief, but if it lasts longer than half a minute, it becomes communicatively uncomfortable. If the fixation is accompanied by audible airflow, it is referred to as prolongation (Onslow, 2025). If the fixation is silent, it is called a blockage (Onslow, 2025). Stuttering symptoms also include external behaviours, which can be categorised as verbal and non-ver-

3. i 6. godine (Sommer i sur., 2021). O uzrocima mucanja se sve više zna: o genetičkim, psihološkim, jezičnim te okolinskim uzrocima, no poprilično nejasan ostaje neurološki mehanizam koji je u podlozi razvoja mucanja.

Jedan od razloga zašto je to tako jest i činjenica kako je govor uvjetovan širokom neurološkom mrežom, koja uključuje različita područja mozga, kortikalna i subkortikalna. Drugim riječima, nepoznat je neurološki mehanizam u podlozi procesa govora, pa je utoliko otežano razumijevanje i objašnjavanje neurološke podloge razvoja mucanja.

Pregledom dijela literature uviđa se kako se razlike mozga osoba koje mucaju (OKM) i osoba koje ne mucaju (OKNM) mogu pronaći u kortikalnom i subkortikalnom području, a same razlike mogu biti strukturalne i funkcionalne. Od strukturalnih se sve veći naglasak stavlja na razliku u volumenu korpusa kalozuma, a razlika postoji i u povećanom volumenu bijele tvari u desnoj hemisferi (Choo i sur., 2011). Od funkcionalnih razlika dobro su potkrijepljena saznanja o pretjeranoj i neuobičajenoj aktivaciji desne hemisfere (Fox i sur., 1996; Preibisch i sur., 2003; Choo i sur., 2012), deaktivaciji slušnog područja (Budde i sur., 2014), aktivaciji motoričkog područja i malog mozga (Fox i sur., 1966, Ingham, 2001). Poznato je da su subkortikalna područja poput bazalnih ganglija, talamusa i malog mozga, također uključena u proces nastajanja mucanja (Fox i sur., 1996; Kell i sur., 2018). Uloga subkortikalnog područja u nastanku mucanja jest još uvijek nedovoljno jasna, naročito zbog složenosti strukture i funkcija pojedinih subkortikalnih dijelova mozga, kao što su primjerice bazalni gangliji.

Cilj ovog polusistemskog preglednog rada jest osvrt na postojeća saznanja iz područja neurološkog aspekta razvoja mucanja, prateći vremenski slijed i napredak u istraživanjima. Pomoću sadržajne analize pokušat će se dati narativni uvid u polje neurologije i mucanja kroz kronološki tijek. Gledajući strukturalne temelje mucanja, ističu se tri teorije (Alm, 2020) koje pokušavaju objasniti neurološke temelje mucanja. O njima će

bal behaviours (Onslow, 2025). Stuttering is most commonly recognised between the ages of 3 and 6 years (Sommer et al., 2021). Our understanding of the causes of stuttering continues to grow - there are genetic, psychological, linguistic, and environmental causes. What remains rather unclear is the neurological mechanism that forms the basis of the development of stuttering.

One underlying reasons is the fact that speech is conditioned by an extensive neurological network, which includes different areas of the brain such as the cortical and subcortical areas. In other words, given that the neurological mechanism behind the speech process is unknown, it is difficult to understand and explain the neurological basis of the development of stuttering.

Through a review of the literature, it becomes evident that there are distinct differences between the brains of people who stutter (PWS) and those who do not stutter (non-PWS) in both cortical and subcortical areas, and these differences can be both structural and functional in nature. When it comes to structural differences, there is an increasing emphasis on the variation in the volume of the corpus callosum, as well as an increase in the volume of white matter in the right hemisphere in PWS (Choo et al., 2011). Functional differences between PWS and non-PWS are well-documented by findings of excessive and unusual right hemisphere activation (Fox et al., 1996.; Preibisch et al., 2003.; Choo et al., 2012), deactivation of the auditory area (Budde et al., 2014), as well as the activation of the motor area and cerebellum (Fox et al., 1966; Ingham, 2001). It is known that subcortical areas such as the basal ganglia, thalamus, and cerebellum are also involved in the process of stuttering (Fox et al., 1996.; Kell et al., 2018). The role of the subcortical area in the onset of stuttering remains unclear, especially due to the complexity of the structure and functions of certain subcortical parts of the brain, such as the basal ganglia.

The aim of this semi-systematic review paper is to examine the existing knowledge regarding the neurological aspects of the development of stuttering by following the timeline of research in this field and seeing how it has progressed over time. By using content analysis, we aim to pro-

se pisati dalje u tekstu, kao i o smjeru prema kojem idu buduća istraživanja.

TEORIJE O NEUROLOŠKOJ OSNOVI RAZVOJA MUCANJA

Neurološka osnova mucanja objašnjava se brojnim predloženim teorijama, no gledajući mucanje kao neurorazvojni poremećaj, ističu se tri teorije: *teorija cerebralne dominantnosti, teorija o poremećaju mijelinizacije te teorija o poremećaju funkcije bazalnih ganglija*.

Teorija cerebralne dominantnosti

Kako bi govorni organi funkcionirali sinkronizirano, govorni impulsi koji dolaze iz lijeve i desne moždane hemisfere moraju stići istovremeno do govornih organa. Jedna hemisfera treba biti dominantna te kontrolirati, upravljati i usklađivati pokrete. Teorija koja polazi od ove teze jest *Teorija cerebralne dominacije*, a prvi put se spominje u zapisu Stiera, 1911. godine (prema Alm, 2020). Temelje ove teorije poslije su postavili Sam Orton i Lee Travis, 1920-ih godina (Orton, 1927; Orton i Travis, 1929; Travis, 1931, 1978; prema Ingham, 2001). Ova teorija prikazuje mucanje kao rezultat neuspješne lateralizacije dominacije govornih centara između lijeve i desne hemisfere. Prema toj teoriji OKM nemaju potrebnu dominantnost među hemisferama, koja je neophodna za izvođenje sinkroniziranih govornih pokreta. Sve navedeno dovodi do konfuzije hemisferične kontrole. Godine 1986., neposredno prije svoje smrti, Travis je izjavio: „*Mucavac se značajno razlikuje od urednog govornika samo u svojoj neuroanatomskoj organizaciji za govo*r" (str.119, Travis, 1986; prema Onslow, 2025). Prvi formalni prijedlog ove teorije o interhemisferskoj interferenciji koji uključuje suplementarno motoričko područje, nastao je 1987. godine (Onslow, 2025), a kao takav ima dva dijela. Prvi dio ukazuje na suplementarno motoričko područje mozga koje je neučinkovito, dok drugi dio implicira da je sustav hemisferske aktivacije pretjerano reaktivan. Ova dva faktora smatraju se nužnima i dovoljnima za razvoj mucanja, ali nijedan od njih pojedinačno nije presudan (Onslow, 2025). Otad svjedočimo brojnim istra-

vide a narrative insight into the field of neurology and stuttering through a chronological course. By looking at the structural foundations of stuttering, three theories (Alm, 2020) that attempt to explain the neurological foundations of stuttering stand out. We will discuss these theories in detail and outline the direction for future research.

THEORIES ON THE NEUROLOGICAL BASIS OF THE DEVELOPMENT OF STUTTERING

The neurological basis of stuttering has been explained through numerous proposed theories. When we consider stuttering as a neurodevelopmental disorder, three theories stand out: the theory of cerebral dominance, the theory of myelination disorder, and the theory of basal ganglia function disorder.

Cerebral dominance theory

In order for the organs of speech to function synchronously, speech impulses coming from the left and right cerebral hemispheres must reach these organs at the same time, and one hemisphere should be dominant and be able to control, manage, and coordinate speech movements. The theory that stems from this thesis is the *Theory of Cerebral Domination*, which was first mentioned by Stier in 1911 (according to Alm, 2020). The foundations of this theory were further outlined by Sam Orton and Lee Travis in the 1920s (Orton, 1927; Orton and Travis, 1929; Travis, 1931, 1978; according to Ingham, 2001). This theory presents stuttering as a result of the unsuccessful lateralisation of the dominance of speech centres between the left and right hemispheres. According to this theory, PWS do not exhibit the necessary dominance between the hemispheres, which is essential for performing synchronised speech movements. This culminates in hemispheric control confusion. In 1986, shortly before his death, Travis stated: "*A stutterer differs significantly from the normal speaker only in their neuroanatomical organisation for speech*" (p. 119, Travis, 1986; according to Onslow, 2025). The first formal proposal of the theory on interhemispheric interference, which includes the supplementary motor area, emerged in 1987 (Onslow, 2025). This theory consists of two parts: the first part points to

živanjima neurooslikavanja mozga, a dokazuju kako je desna hemisfera neuobičajeno aktivnija kod OKM-a, u usporedbi s lijevom hemisferom (Fox i sur., 1996; Preibisch i sur., 2003; Choo i sur., 2012). Kod OKM-a učestalo se uviđa pretjerana i neuobičajena aktivacija desne hemisfere, i to područja koja su analogna govornim područjima lijeve hemisfere. Dugotrajna je diskusija o ulozi takve aktivacije, a odgovor je gotovo uvijek kompenzacijska uloga (Neef i Chang, 2024). Kao potvrda takvom zaključku ističe se kako se takva pretjerana aktivacija desne hemisfere ne pronalazi kod djece koja mucaju (DKM) (Zablotsky i sur., 2019, prema Neef i Chang, 2024). Prednost je ove teorije ekonomičnost jer specificira dva nužna uvjeta za razvoj mucanja (Packam i Attanasio, 2017; prema Onslow 2025), no nedostatak je to što je najviše puta opovrgnuta gledajući istraživanja do 2017. godine. Naročito se to odnosi na dokaze o lateralizaciji jezika tijekom različitih govornih zadataka (Packam i Attanasio, 2017; prema Onslow 2025). Današnji model interhemisferske interferencije proširuje sada već napuštenu Orton-Travisovu teoriju, ali se od nje udaljava navodeći da osobe koje mucaju imaju tipičnu lateralizaciju govornih funkcija. Najnovija inačica modela navodi: „*Anomalija u interhemisferskim odnosima i deficit u mehanizmima govorno-motoričke kontrole svakako su nužan, ali ne i dovoljan uvjet za mucanje*" (Onslow, 2025).

Teorija poremećaja mijelinizacije

Teorija o cerebralnoj dominaciji usmjerava istraživače prema shvaćanju kako etiologija mucanja ima čvrsto neurološko uporište. Istraživački nalazi jasno potvrđuju povezanost mucanja s neurorazvojnim promjenama, što je dovelo do dubljeg istraživanja moždanih struktura, posebice bijele tvari. Brojni autori istraživali su poremećaj mijelinizacije kao uzroka nastanka mucanja (Salmelin i sur., 2000; Sommer i sur., 2002; Neumann i sur., 2003; Neumann i sur., 2005; Chang i sur., 2008; Chang i sur., 2011; Choo i sur., 2012; Chang i sur., 2015; prema Galić-Jušić, 2021). Sommer i sur. (2002) radili su istraživanje koje jasno pokazuje postojanje znakova kortikalne nepovezanosti lijevog senzomotoričkog korteksa koji obuhvaća

the supplementary motor area of the brain as being ineffective, while the second part implies that the hemispheric activation system is excessively reactive. These two factors are considered necessary and sufficient for the development of stuttering, but neither of them are key determinants on their own (Onslow, 2025). Since then, numerous brain neuroimaging studies have reported that the right hemisphere in PWS is noticeably more active compared to the left hemisphere (Fox et al., 1996; Preibisch et al., 2003; Choo et al., 2012). Excessive and unusual activation of the right hemisphere is often observed in PWS, namely of the areas that are analogous to the speech areas of the left hemisphere. There has been an ongoing discussion about the role of such activation, and the most consistent answer is that it plays a compensatory role (Neef and Chang, 2024). To confirm this conclusion, it is emphasised that such excessive activation of the right hemisphere is not found in children who stutter (CWS) (Zablotsky et al., 2019, according to Neef and Chang, 2024). The advantage of this theory is its simplicity, as it specifies two necessary conditions for the development of stuttering (Packam & Attanasio, 2017; according to Onslow, 2025), but its drawback is that it has been frequently refuted in research studies published before 2017. This particularly relates to evidence of language lateralisation during different speech tasks (Packam & Attanasio, 2017; according to Onslow, 2025). The current model of interhemispheric interference expands upon the now-abandoned Orton-Travis theory, but deviates from it by stating that individuals who stutter exhibit typical lateralisation of speech functions. The latest version of the model states: "Anomalies in interhemispheric relationships and deficits in speech-motor control mechanisms are certainly necessary, but not sufficient conditions for stuttering." (Onslow, 2025).

Myelination disorder theory

The theory of cerebral dominance directs researchers towards understanding that the aetiology of stuttering has a strong neurological basis. Research findings clearly confirm the link between stuttering and neurodevelopmental changes, which has led to further exploration of brain structures, particularly white matter. Several authors have in-

funkcije larinksa i jezika. Sommer i sur. (2002) to interpretiraju kao dokaz atrofije mijeliniziranih vlakana koji povezuju područja bitna za govor. Demijelinizacija vlakana i/ili propadanje mijeliniziranih vlakana posljedično dovodi do smanjene brzine prijenosa informacija od mozga do ciljanih dijelova tijela, kao i do simptoma koji su određeni funkcijama oštećenog neurona (poremećaj govora, gubitak pamćenja i sl.). Sommerova teza (i njegovih suradnika) može se povezati sa starijom teorijom o etiologiji mucanja, koju je postavio Isaac Karlin (1947), a govori o zakašnjeloj mijelinizaciji (sazrijevanju) govornih područja mozga, što uzrokuje i veći postotak mucanja kod muške djece. Promjene u bijeloj tvari pronađene su i u dijelovima koji su odgovorni za izmjenu informacija između područja mozga odgovornih za govornu produkciju i motoričku kontrolu (npr. *arcuate fascikulus, superiorni longitudinalni fascikulus, frontalni aslantni trakt, kortikobulbarni trakt, cerebellarni pendunkules*) (Neef i Chang, 2024).

Neurooslikavanje mozga prikazuje zanimljivost o neurološkoj osnovi prirodnog oporavka od mucanja tijekom djetinjstva. Djeca koja se oporave od mucanja pokazala su povećan volumen bijele tvari u *motoričkim projekcijskim vlaknima, lijevom fascikulus arkuatusu, korpus kalozumu i cerebelarnom pedunkulu oko dentatnih jezgara*, što sugerira normalizaciju ili uspješnu kompenzaciju neuroloških deficita povezanih s mucanjem (Chow i sur., 2023). Također, slična povećanja volumena *korpusa kalozuma* i *cerebelarnog pedunkula* opažena su kod DKM-a, što ukazuje na nepotpunu kompenzaciju. Ovi rezultati pružaju značajne nove uvide u moguće neurološke osnove nastanka perzistencije i oporavka od mucanja tijekom djetinjstva. Jasno je kako mucanje zbog svog multidimenzionalnog okvira ne može nastati samo kao posljedica poremećene mijelinizacije te da na njegov razvoj utječu i jezični faktori, okolinski, psihološki. Kako navodi i Galić-Jusić (2021), svi navedeni faktori objedinjeni su u Packam i Attanasio trofaktorskom modelu mucanja. Model pretpostavlja problem središnjeg živčanog sustava koji kod neke djece uzrokuje poremećenu neurološku obradu govornog jezika (Onslow,

vestigated myelination disorder as a cause of stuttering (Salmelin et al., 2000; Sommer et al., 2002; Neumann et al., 2003; Neumann et al., 2005; Chang et al., 2008; Chang et al., 2011; Choo et al., 2012; Chang et al., 2015; according to Galić-Jušić, 2021). Sommer et al. (2002) conducted a study that clearly shows the existence of signs of cortical disconnection in the left sensorimotor cortex, which includes the functions of the larynx and the tongue. Sommer et al. (2002) interpreted this as evidence of atrophy of myelinated fibres that connect speech-relevant areas. Demyelination of fibres and/or degradation of myelinated fibres consequently leads to a reduced rate of transmission of information from the brain to the target parts of the body, as well as to symptoms that are determined by the functions of the damaged neuron (speech disorder, memory loss, and so on). Sommer's (and his associates') thesis can be linked to an older theory on the aetiology of stuttering, presented by Isaac Karlin (1947), which talks about the delayed myelination (maturation) of the speech areas of the brain, which causes a higher percentage of stuttering in male children. Changes in white matter were also found in the parts of the brain responsible for the exchange of information between the areas responsible for speech production and motor control (e.g., *arcuate fasciculus, superior longitudinal fasciculus, frontal aslant tract, corticobulbar tract, cerebellar peduncle*; Neef and Chang, 2024).

Neuroimaging of the brain shows interesting information about the neurological basis of natural recovery from stuttering during childhood. Children who recover from stuttering showed an increased volume of white matter in the *motor projection fibres, left arcuate fasciculus, corpus callosum, and cerebellar peduncle around the dentate nuclei*, suggesting normalisation or successful compensation of neurological deficits associated with stuttering (Chow et al., 2023). Similar increases in corpus callosum and cerebellar peduncle volume were also observed in CWS, indicating incomplete compensation. These results provide significant new insights into the possible neurological causes of persistence and recovery from stuttering during childhood. It is clear that stuttering, due to its multidimensional nature, cannot arise solely as a result of impaired myelination, and that its de-

2025), odnosno atipičnu povezanost bijele tvari kao vjerojatan problem u neurološkoj obradi.

Teorija poremećaja funkcije bazalnih ganglija

Osim istraživanja kortikalnih struktura, njihove povezanosti i ukupnog utjecaja na pojavu mucajućih netečnosti, proučava se i uloga subkortikalnih područja mozga. Još 1995. godine istraživači su se bavili aktivnostima bazalnih ganglija kod odraslih osoba koje mucaju, a pronašli su sniženu aktivnost u repatoj jezgri (*nucleus caudate*, dio bazalnih ganglija) (Wu i sur., prema Galić-Jusić, 2021). U fokusu istraživanja jest utjecaj bazalnih ganglija (Alm, 2004) i kortiko-striato-talamo-kortikalnih veza na mucanje (Smits-Bandstra i De Nill, 2007). Nefunkcionalnost bazalnih ganglija povezuje se s mucanjem u vidu pravovremenog pokretanja govorno motoričkih programa (Alm, 2004). Upravo to i jest mogući razlog zbog kojeg osobe koje mucaju, uz uvođenje pravovremenog iniciranja i reguliranja pokreta (kao što su ritmički govor, govor uglas s drugom osobom, pjevanje), govore uz smanjeni postotak mucajućih netečnosti. Civier i sur. (2013) povezuju u svom istraživanju abnormalnost bijele tvari te poremećenu funkciju bazalnih ganglija. Postavili su dvije hipoteze: (1) abnormalnosti bijele tvari remete krug putem kortikostriatalnih projekcija koje prenose kopije izvedenih motoričkih naredbi i (2) dopaminergičke abnormalnosti remete krug putem striatuma. Rezultati simulacije koju su proveli u svom istraživanju podržavaju obje hipoteze: "*.. u oba scenarija, neuralne abnormalnosti odgađaju očitavanje motoričkog programa sljedećeg sloga, što dovodi do netečnosti u govoru.*" (Civier i sur., 2013).

S poremećajem funkcije bazalnih ganglija može se povezati i utjecaj emocionalne komponente te stresa, koji utječu na količinu mucajućih netečnosti u govoru. Poznato je kako je uredno funkcioniranje bazalnih ganglija usko povezano s aktivacijom dopamina. Dobro regulirana razina dopamina ključna je za funkcioniranje neuronskih krugova bazalnih ganglija u regulaciji motoričkih i endokrinih funkcija, emocija i radnog pamćenja (Ayano, 2016; prema Galić-Jusić 2021). Upravo terapija dopaminergičkim lijekovima utječe na

velopment is also influenced by linguistic, environmental, and psychological factors. As mentioned in Galić-Jusić (2021), all these factors are integrated into the Packam and Attanasio three-factor model of stuttering. The model proposes that, in some children, an issue in the central nervous system causes impaired neurological processing of speech language (Onslow, 2025) or that atypical white matter connectivity is likely to cause a problem in neurological processing.

Basal ganglia dysfunction theory

In addition to researching cortical structures, their connectivity, and the overall impact on the emergence of stuttering disfluencies, the role of the subcortical brain regions have also been studied. As early as 1995, researchers examined the activity of the basal ganglia in adults who stutter and found reduced activity in the caudate nucleus, which is a part of the basal ganglia (Wu et al., according to Galić-Jusić, 2021).The focus of the research is the influence of the basal ganglia (Alm, 2004) and cortico-striato-thalamo-cortical connections on stuttering (Smits-Bandstra and De Nill, 2007). The dysfunction of the basal ganglia is associated with stuttering in the form of timely initiation of speech and motor programmes (Alm, 2004). This is one possible reason why people who stutter – with the introduction of timely initiation and regulation of movement (such as rhythmic speech, speaking in unison with another person, singing) – speak with a reduced percentage of stuttering dysfluencies. Civier et al. (2013) linked abnormalities in white matter with impaired functioning of the basal ganglia. They proposed two hypotheses: (1) abnormalities in white matter can disrupt the circuit through corticostriatal projections that carry copies of motor commands, and (2) dopaminergic abnormalities disrupt the circuit through the striatum. The results of the simulation that they conducted in their study supported both hypotheses: „*... in both scenarios, neural abnormalities delay the reading of the motor programme for the next syllable, leading to disfluencies in speech*." (Civier et al., 2013). Disruption of the basal ganglia function can be linked to the influence of emotional components and stress, which affect the quantity of stuttering dysfluencies

poboljšanje/pogoršanje simptoma mucanja, što ukazuje na povezanost funkcije bazalnih ganglija i mucanja (Alm, 2004). Mucanje se može povezati i s ostalim motoričkim poremećajima nastalih zbog poremećene funkcije bazalnih ganglija, npr. hipo i/ili hiperkinetičkom disfunkcijom (Alm, 2020). Zanimljivo je što se kod djece koja će razviti perzistentno mucanje (rezultat na testu SSI-4 jednak je ili veći od 10), ranije u neurorazvoju uočava značajno smanjen volumen putamena (koji je dio bazalnih ganglija) (Chow i sur., 2023). Ta diskrepancija smanjuje se s odrastanjem, a zanimljivost je ta da se u odrasloj dobi upravo u putamenu (Chang i sur., 2008) i nukleusu kaudatusu pronalazi povećana neurološka aktivacija (Giraud i sur., prema Neef i Chang, 2024). Jedna od mogućih hipoteza prema Neef i Changu (2024) jest da rano pojavljivanje strukturnih razlika u bazalnim ganglijima može u početku pridonijeti uočenoj razlici među skupinama, ali se ta razlika s vremenom smanjuje zbog razvojnih kaskada koje utječu na međusobno povezane moždane strukture. Primjerice, strukturna razlika u bazalnim ganglijima u ranom razvoju i njihova povezanost sa suplementarnim motoričkim područjem mogla bi potencijalno utjecati na različite razvojne putanje u regijama kao što su talamus i mali mozak (Neef i Chang, 2024). Daljnja istraživanja potrebna su kako bi se razjasnili precizni mehanizmi koji stoje iza ovih razvojnih dinamika kod OKM-a.

BUDUĆA ISTRAŽIVANJA NEUROLOŠKE OSNOVE MUCANJA

Sva navedena istraživanja i teorije od kojih polaze dovode do stvaranja novog smjera razmišljanja uz primjese već pronađenog i dokazanog. Jedno od najnovijih načina razmišljanja o uzorku moguće podgrupe mucanja odnosi se na pregledni rad dr. Per A. Alma (2020).

Prema istraživanju temeljenom na medicinskim zapisima djece koja mucaju (Berry, 1938; prema Alm 2020) glavni je uzrok mucanju sredinom 1900. godine bila infekcija β-hemolitičkim streptokokom skupine A (BHSA), sve do pojave penicilina 1943. Pojavu iznenadnog i naglog mucanja nakon infekcije grla moguće je povezati s

in speech. It is widely known that the normal functioning of the basal ganglia is closely related to the activation of dopamine. A well-regulated level of dopamine is crucial for the functioning of the neural circuits of the basal ganglia with respect to the regulation of motor and endocrine functions, emotions, and working memory (Ayano, 2016; according to Galić-Jusić, 2021). Dopaminergic drug therapy can affect the improvement/exacerbation of stuttering symptoms, indicating an association between basal ganglia function and stuttering (Alm, 2004). Stuttering may also be associated with other motor disorders caused by impaired basal ganglia function, for example, hypo and/or hyperkinetic dysfunction (Alm, 2020). It is interesting that children who are likely to develop persistent stuttering (based on the results of the SSI-4 test being equal to or greater than 10) tend to show a significantly reduced volume of the putamen (part of the basal ganglia) earlier in their neurodevelopment (Chow et al., 2023). This discrepancy decreases as they grow up, and interestingly, in adulthood, increased neurological activation is found in the putamen (Chang et al., 2008) and the caudate nucleus (Giraud et al., according to Neef and Chang, 2024). One possible hypothesis according to Neef and Chang (2024) is that the early appearance of structural differences in the basal ganglia may initially contribute to the observed difference (between the children with persistent stuttering and those who will later recover from stuttering), but this difference decreases over time due to developmental cascades affecting interconnected brain structures. For example, the structural difference in the basal ganglia in early development and their association with the supplementary motor area could potentially affect different developmental trajectories in regions such as the thalamus and cerebellum (Neef and Chang, 2024). Further research is needed to clarify the precise mechanisms behind these developmental dynamics in PWS.

FUTURE RESEARCH ON THE NEUROLOGICAL BASIS OF STUTTERING

The above-mentioned research and theories have led to the development of a new line of thought, combining previously discovered and

BHSA infekcijom iz triju razloga: 1) medicinski zapisi iz 1930-ih pokazuju snažnu povezanost između početka mucanja kod djece i BHSA infekcije, posebice tonzilitisa, šarlaha i reumatske groznice (koja je autoimuna posljedica BHSA infekcije, učestali medicinski problem sve do početka 1960.) (Berry, 1938, prema Alm, 2020) , 2) zabilježena je visoka korelacija između pada u pojavi mucanja i reumatske groznice s pojavom penicilina (oko 0.95) (Alm, 2020), 3) potvrđeni biološki mehanizmi povezuju BHSA tonzilitis s imunološkim utjecajem na mozak. U prilog hipotezi povezanosti mucanja i BHSA infekcije idu i rezultati manjeg broja prikaza slučajeva (primjerice Maguire i sur., 2010, Lewin i sur., 2011, Ray i sur., 2013, prema Alm, 2020), kao i rezultati istraživanja na većoj skupini djece koja mucaju (Fidan i sur., 2024). Maguire (2020) zaključuje kako prikazi slučajeva upućuju na to da antitijela koja su usmjerena protiv streptokokne infekcije reagiraju unakrsno te napadaju bazalne ganglije u razvoju. Fidan i suradnici (2024) pronašli su povišenu razinu antistreptokoknih antitijela kod djece koja mucaju, što ukazuje na moguću aktivaciju postinfekcijskog imunološkog mehanizma koji reagira zbog BHSA infekcije te tako utječe na patogenezu mucanja.

BHSA infekcije mogu dovesti do dječjeg autoimunog neuropsihijatrijskog poremećaja (PANDAS), koji uključuje različite neurološke manifestacije (poput opsesivno-kompulzivnog poremećaja ili tikova) kod djece nakon preboljele streptokokne infekcije. Brojni prikazi slučajeva sugeriraju kako je mucanje jedan od simptoma PANDAS-a, povezujući mucanje s Tourettovim sindromom s kojim dijeli određene kliničke karakteristike (početak poremećaja u djetinjstvu, prevalencija na strani muškog spola, fluktuirajućeg rasta i pada simptoma, postojanje tikova, pogoršanje simptoma pri tjeskobi i pri dopamin agonistima) (Fidan i sur., 2024). Ako BHSA infekcije i danas nastavljaju utjecati na pojavu mucanja (i potencijalne podgrupe mucanja), rano dijagnosticiranje takvih slučajeva bilo bi od velike važnosti.

proven concepts. One of the latest approaches to understanding a possible subgroup of stuttering is highlighted in the work of Dr Per A. Alm (2020).

According to the study based on medical records (Berry 1938; according to Alm, 2020), the main cause of stuttering in mid-1900s was infection with the group A beta-haemolytic streptococcus (BHSA), until the appearance of penicillin in 1943. It is possible to connect the occurrence of unexpected and sudden stuttering after a throat infection with a BHSA infection for three reasons: 1) Medical records from the 1930s show a strong association between the onset of stuttering in children and BHSA infection, especially tonsillitis, scarlet fever, and rheumatic fever (which is an autoimmune consequence of BHSA infection, a common medical problem until the beginning of 1960; Berry, 1938, according to Alm, 2020), 2) There was a high correlation between the decline in the occurrence of stuttering and rheumatic fever and the appearance of penicillin (about 0.95; Alm, 2020), 3) Research confirmed that biological mechanisms can link BHSA tonsillitis to an immune effect on the brain. The hypothesis of the connection between stuttering and BHSA infection is supported by the results of a smaller number of case reports (for example, Maguire et al., 2010, Lewin et al., 2011, Ray et al., 2013, according to Alm, 2020), as well as the results of research on a larger group of children who stutter (Fidan et al., 2024). Based on case reports, Maguire (2020) concluded that antibodies directed against streptococcal infection cross-react and attack the developing basal ganglia. Fidan et al. (2024) found elevated levels of anti-streptococcal antibodies in children who stutter, indicating a possible activation of a post-infective immune mechanism that responds to BHSA infection, and thus, affects the pathogenesis of stuttering.

After a streptococcal infection, BHSA infections in children can lead to Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcal Infections (PANDAS), which includes various neurological manifestations (such as obsessive-compulsive disorder or tics). Numerous case reports suggest that stuttering is one of the symptoms of PANDAS, linking stuttering to

Zanimljivo je i istraživanje australskog istraživačkog tima (*Australian Research Center*) koje sugerira kako je uzrok mucanju organski i prisutan već pri rođenju. Istraživanje je provedeno na 18 djece starosne dobi od osam do 18 tjedana. Šestero djece ima genetičku podlogu kao rizik od razvoja mucanja. Rezultati pokazuju smanjeni integritet bijele tvari u korpusu kalozumu kod novorođenčadi koja su u riziku od razvoja mucanja u odnosu na kontrolnu skupinu (Packman i sur., 2022). To je prvo istraživanje kroz skeniranje dječjeg mozga prije razvoja mucanja, a rezultati svakako trebaju daljnja istraživanja. Važnost ovog istraživanja je i razbijanje vječnog pitanja jesu li razlike u neurološkim funkcionalnim/strukturalnim razlikama uzrok ili rezultat mucanja.

Istraživanje etiologije mucanja ne bi bilo potpuno bez istraživanja genetičkih mutacija koje svojim višestrukim djelovanjem pridonose promjenama u funkciji i strukturi mozga. Poznata su četiri gena povezana s pojavom mucanja (GNPTAB; GNPTG; NAGPA; AP4E1) (Cvjetković Grginović, 2022). Navedeni geni imaju ulogu u intracelularnom trgovanju kroz enzime koje kodiraju. Intracelularno trgovanje uključuje premještanje proteina na prava mjesta, a prema jednom istraživanju deficiti u ovom procesu mogu dovesti do perzistentnog mucanja („NIH Researchers“, 2015, prema Cvjetković Grginović 2022). Kako je naglašeno u uvodu, nepoznavanje neurološkog mehanizma procesa govora, otežava razumijevanje i objašnjavanje neurološke podloge razvoja mucanja. To se posebno reflektira u razumijevanju povezanosti varijanti gena, koji upravljaju metaboličkim procesima i recikliranjem starih dijelova stanica, s razvojem nekih perzistentnih mucanja. Brojna istraživanja donose ograničen broj jasnih uzročno-posljedičnih veza, ostavljajući prostor za daljnja istraživanja. Primjeri za to su istraživanja: Barnes i sur. (2016; varijante u genu GNPTAB i promjena vokalizacije u miševa), Han i sur. (2019; varijante gena GNPTAB i deficiti u vokalizaciji miša povezane s manjkom astrocita u korpusu kalozumu), Stamurai i sur., (2020; nasljeđivanje samo

Tourette's syndrome, with which it shares certain clinical characteristics (onset in childhood, prevalence in boys, fluctuating growth and decline of symptoms, existence of tics, worsening of symptoms in situations of anxiety, as well as in patients undergoing therapy with dopamine agonists; Fidan et al., 2024).

If BHSA infections continue to affect the onset of stuttering (and potential stuttering subgroups), early diagnosis of such cases would be of great importance.

An interesting study by the Australian Research Centre suggested that the cause of stuttering is organic and present from birth. The study was conducted on 18 children, aged 8 to 18 weeks, including six children who had a genetic predisposition that was a risk factor for developing stuttering. The results show a reduced integrity of white matter in the corpus callosum in newborns at risk of developing stuttering compared to the control group (Packman et al., 2022). This is the first study to scan the brains of infants before the onset of stuttering, and the results certainly warrant further research. The significance of this study also lies in addressing the long-standing question of whether differences in neurological functional/structural differences are a cause or a result of stuttering.

Research into the aetiology of stuttering would not be complete without studying genetic mutations that, through their multiple effects, contribute to changes in brain function and structure. The four known genes associated with the onset of stuttering are GNPTAB, GNPTG, NAGPA, and AP4E1) (Cvjetković Grginović, 2022). These genes play a role in intracellular trafficking through the enzymes they encode. Intracellular trafficking involves the movement of proteins to their correct locations, and according to one study, deficits in this process can lead to persistent stuttering (NIH Researchers, 2015, according to Cvjetković Grginović, 2022). As emphasised in the introduction, a lack of understanding of the neurological mechanism of the speech process complicates the understanding and explanation of the neurological basis of development of stuttering.

jedne „oštećene“ kopije GNPTAB, GNPTG, NAGPA dovodi do mucanja) (Cvjetković Grginović, 2022).

ZAKLJUČAK

Neuobičajena aktivacija desne hemisfere dobro je potkrijepljena činjenica koja vjerojatno ima kompenzacijsku ulogu, moguće zbog anomalije u interhemisferskim odnosima i deficitu u mehanizmima govorno-motoričke kontrole. Strukturalna razlika mozga OKM-a i OKNM-a većim se dijelom odnosi na razlike u volumenu bijele tvari, moguće zbog atrofije mijeliniziranih vlakana koji povezuju područja bitna za govor. Poremećena mijelinizacija dovodi do promijene u brzini prijenosa impulsa bitnih za produkciju govora (prekid prijenosa impulsa), što se može povezati sa simptomatologijom mucanja u vidu prekida govornog tijeka.

Velik utjecaj na razvoj mucanja imaju i subkortikalne strukture, u vidu pravovremenog iniciranja izvođenja pokreta što je uvelike funkcija bazalnih ganglija. Istraživanja striatuma (nucleus caudatus i putamen, dijelovi bazalnih ganglija) jasno pokazuju da organizacija striatuma čini osnovu za istraživanje načina na koji striatum doprinosi obradi motoričkih, limbičkih i heteromodalnih informacija kroz više kortikostrijatalne krugove (Choi i sur., 2012). Bit će potreban niz istraživanja s ciljem razjašnjavanja uloge bazalnih ganglija i njegove uloge u nastanku mucanja.

Povezanost BHSA infekcije i pojave mucanja, uz relaciju s bazalnim ganglijima, jest područje koje se tek počinje istraživati. Ako se slučajevi nastanka mucanja nakon takve infekcije pojavljuju još i danas, od izuzetne je važnosti detektirati ih te spriječiti. Istraživanjem ovog područja u mucanju dolazi do razvijanja prevencije moguće podgrupe mucanja nastale BHSA infekcijom.

Genetička istraživanja pridonose razumijevanju neurološke osnove mucanja. Kompleksnost genskih mutacija i njihov utjecaj na funkciju i strukturu mozga ključni su za rasvjetljavanje neurološke podloge mucanja.

Za kraj, potrebno je imati na umu tri bitne stavke kada govorimo o neurološkoj podlozi

This is particularly reflected in understanding the connection between gene variants that regulate metabolic processes and the recycling of old cell parts, and the development of persistent stuttering in some cases. Numerous studies provide a limited number of clear cause-and-effect connections, leaving room for further research. Examples of such studies include Barnes et al. (2016; variants in the GNPTAB gene and changes in vocalisation in mice), Han et al. (2019; variants of the GNPTAB gene and deficits in mouse vocalisation linked to a lack of astrocytes in the corpus callosum), and Stamurai et al. (2020; inheritance of only one “damaged” copy of GNPTAB, GNPTG, NAGPA leads to stuttering) (Cvjetković Grginović, 2022).

CONCLUSION

The unusual activation of the right hemisphere is a well-established fact that potentially has a compensatory role in maintaining speech fluency, possibly due to anomalies in interhemispheric relationships and deficits in speech-motor control mechanisms. The structural difference between the brains of the PWS and non-PWS mostly refers to differences in the volume of white matter, possibly due to atrophy of myelinated fibres connecting speech-relevant areas. Disturbed myelination leads to a change in the speed of transmission of impulses essential for speech production (interruption of pulse transmission), which can be associated with the symptomatology of stuttering in the form of interruption of speech flow.

Subcortical structures also have a major influence on the development of stuttering, in the form of timely initiation of movement, which is largely a function of the basal ganglia. Research on the striatum (caudate nucleus and putamen, parts of the basal ganglia) clearly shows that the organisation of striatum forms a basis of reference for exploring how it contributes to the processing of motor, limbic, and heteromodal information through higher corticostriatal circuits (Choi et al., 2012). A series of studies must be conducted to clarify the role of the basal ganglia and its association with the onset of stuttering.

mucanja, a isto naglašava nekolicina autora (primjerice Mark Onslow, Nicole E. Neef, Soo-Eun Chang). Prva je ta da ne postoji jedna specifična regija mozga odgovorna za govor i jezik. Govor i jezik procesi su na razini neuralne mreže koja uključuje mnogo različitih područja mozga. Druga, istraživanje neurooslikavanja mozga uglavnom se provodilo s obzirom na vidljive, otvorene trenutke mucanja, što automatski upućuje na nepotpuno neurobiološko znanje o mucanju. I naposljetku, mozgovi djece i odraslih koji mucaju izgledaju uredno u odnosu na govornike koji ne mucaju. Mucanje ne pokazuje očite znakove poput vidljive „frakture na rendgenskoj snimci“, kako je to naveo profesor Mark Onslow u svojim zapisima o mucanju (2025). Potrebni su sofisticirani instrumenti i grupne statističke analize kako bi se uočile razlike u odnosu na vršnjake. Ovaj treći čimbenik od posebne je važnosti pri prenošenju informacija klijentima i roditeljima prilikom opisivanja neuroloških saznanja.

Research on BHSA infection and the onset of stuttering, in relation to the basal ganglia, is an area that is only beginning to be explored. If cases of stuttering after such an infection continue to be reported, it is of utmost importance to detect and prevent them. Examining this aspect related to stuttering can help prevent a possible subgroup of stuttering caused by BHSA infection. Genetic research contributes to understanding the neurological basis of stuttering. The complexity of genetic mutations and their impact on brain function and structure are key to elucidating the neurological foundation of stuttering.

Finally, it is necessary to keep three important points in mind when talking about the neurological basis of stuttering, as emphasised by several authors (for example, Mark Onslow, Nicole E. Neef, Soo-Eun Chang). The first is that no single region of the brain is responsible for speech and language. Speech and language are neural network-level processes that involve many different areas of the brain. Second, the study of brain neuroimaging was mainly conducted with regard to visible moments of stuttering, which automatically indicates incomplete neurobiological knowledge of stuttering. And finally, the brains of children and adults who stutter look normal when compared to speakers who do not stutter. Stuttering does not show obvious signs, such as a visible “X-ray fracture”, as Professor Mark Onslow noted in his records about stuttering (2024). Sophisticated instruments and group statistical analyses are needed to detect any differences from peers. This third factor is of particular importance when communicating information to clients and parents regarding neurological findings.

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