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REVIEW ARTICLE / ПРЕГЛЕДНИ РАД

Primitive reflexes in developing and adult brain – from intellectual disability to dementia

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SUMMARY
Primitive reflexes are involuntary motor responses elicited by sensory stimuli, playing essential roles in feeding, survival, and protection during the neonatal period. Although primitive reflexes are grounded in the central nervous system integrity and are crucial for motor development and sensory processing, they are progressively inhibited and integrated as the frontal cortex matures. The persistence of primitive reflexes beyond 12 months of age may signify underlying neurodevelopmental delays, including intellectual disability, cerebral palsy, autism spectrum disorder, and other related conditions. At the other end of the lifespan spectrum, the reemergence of primitive reflexes or frontal release signs is frequently observed in older patients with various types of dementia, reflecting diffuse cerebral dysfunction and frontal lobe lesions that impair cortical inhibition of brainstem activity. This paper aims to summarize the key clinical implications of persistent primitive reflexes in cognitively affected individuals (such as intellectual disability and dementia) at both ends of the lifespan and to compare their neurobiological bases and prognostic significance to enhance understanding of these phenomena.
Keywords: primitive reflexes; intellectual disability; dementia; motor development; cognitive development

INTRODUCTION

Primitive reflexes (PRs) are automatic motor responses triggered by sensory stimuli, essential for newborns' feeding, survival, and protection [1, 2, 3]. Emerging around 25–26 weeks of gestation, these reflexes become fully functional at birth and dominate infant motor patterns during the first months of life [4]. Although PRs are grounded in central nervous system (CNS) integrity and crucial for motor and sensory development, their persistence beyond 12 months of age may indicate neurodevelopmental delays such as intellectual disability (ID), cerebral palsy (CP), autism spectrum disorder (ASD), and related conditions [3–7].

At the other end of the lifespan spectrum, the intriguing reemergence of primitive reflexes, also known as frontal release signs, is frequently observed in patients with various dementias. This finding is believed to reflect diffuse cerebral dysfunction and frontal lobe lesions that diminish cortical inhibition of brainstem activity [8]. However, PRs can also be observed in healthy elderly individuals, suggesting they may represent normal physiological aging and raising questions about their clinical significance. Nevertheless, a recent meta-analysis demonstrated that PRs in the elderly are strongly associated with an increased risk of dementia, underscoring the importance of their careful assessment during routine physical examinations of patients with cognitive decline [8].

This paper aims to summarize the key clinical implications of persistent PRs in cognitively challenged individuals with ID and dementia at opposite ends of the lifespan and to compare their neurobiological bases and prognostic significance to enhance understanding of these phenomena.

PRIMITIVE REFLEXES IN TYPICAL DEVELOPMENT

Primitive reflexes are involuntary motor responses that enable infants to interact with their environment and serve as foundational mechanisms for developing voluntary movement, supporting maturation of the motor and sensory cortex as well as the temporal–parietal–occipital association area [2, 9, 10]. During typical development (TD), brainstem-mediated PRs are gradually suppressed, integrated, and replaced by cortically controlled voluntary movements, reflecting normal CNS maturation [11–14]. As motor development advances, goal-directed actions and communicative gestures emerge through the inhibition of specific PRs, particularly those involving the hands and mouth [11, 15]. This process depends on the frontal neocortex, which exerts top-down control to inhibit PR pathways [10–13].

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The presence of PRs can be readily assessed in clinical practice as part of standard neurological examinations in both pediatric and adult populations; moreover, specific assessment scales have been developed for children [11, 12, 14]. Most of the over 20 PRs, with major ones listed in Table 1, typically disappear by 4–6 months postnatally, allowing the development of voluntary movements [1, 2, 3].

Retained PRs are relatively common in TD children, with prevalence rates among preschoolers ranging from 43.2% to 80%, varying by assessment methods [9, 15–19]. According to some data, over 90% of healthy children aged 4–6 years exhibit at least one PR to some degree [15]. These findings highlight significant variability in CNS maturation, extending over broader timeframes than previously recognized. The underlying determinants, likely involving genetic and environmental factors, remain largely unknown [1, 2, 3].

PRIMITIVE REFLEXES IN NEURODEVELOPMENTAL DISORDERS

The phenomenon of retained PRs is attributed to brain functional dysconnectivity caused by maturational delays in specific cortical networks. This can lead to compensatory growth in other networks, resulting in asynchronous development and inconsistent functional abilities [10]. When PRs fail to be suppressed, they continue to drive bottom-up interference with the cortex, hindering brain maturation and the establishment of top-down regulatory control. This may cause widespread dysregulation of the nervous, immune, and endocrine systems [20]. Persistence of PRs beyond the first year of life typically indicates psychomotor developmental delays or neurological impairments. This has been well documented in conditions such as CP, ASD, ID, attention-deficit/hyperactivity disorder (ADHD), developmental coordination disorder, Tourette's syndrome, and learning disorders [1, 3, 6, 11, 20–26]. Retained PRs negatively impact motor skill development, cognitive processing, and emotional regulation [5, 27]. When these reflexes persist beyond their typical integration period, they may interfere with a child's ability to coordinate movements, focus attention, and regulate emotional responses effectively [5, 16, 27].

Frontal lobe maturation delays, the primary cause of persistent PRs, are also linked to delayed postural reflex development, hindering sensory-motor milestones such as crawling and walking. Additionally, these delays often coincide with impaired executive functions, a common feature of neurodevelopmental disorders [10]. Early detection of persistent PRs may aid in diagnosing neurodevelopmental disorders, but its reliability is debated [9]. The persistence of PRs in individuals with ID and other neurodevelopmental disorders can facilitate early identification of at-risk children, clarify underlying pathological mechanisms, and improve understanding of cognitive impairment pathways. Additionally, it supports targeted selection for early intervention programs [3, 28].

Primitive reflexes in intellectual disability

Limited epidemiological data exist on PRs in individuals with ID because this group is often excluded from studies or not distinctly separated from other neurodevelopmental disorders [15, 20–26, 29]. A recent review noted frequent retention of the Moro reflex in individuals with ID, including those with Down syndrome, athetoid CP, and occasionally spastic CP. However, as the primary sources are not in English, detailed data remain limited [5, 30, 31].

Besides delayed cortical maturation and network dysfunction, especially in the frontal lobes, persistent PRs have also been associated with abnormal asymmetric development of the brain hemispheres [23, 32]. The causes of retained PRs often remain unclear, but may include insufficient external stimuli essential for TD and exposure to harmful environmental factors [9, 12]. While structural and functional neuroimaging studies have investigated the mechanisms of PRs in elderly populations with neurological disease, no comparable research has been conducted in individuals with ID.

Clinical significance of persistent primitive reflexes in intellectual disability

Retained PRs often signify maturational delay, manifested by structural and functional changes, alongside motor and cognitive delays [27]. High-risk newborns frequently exhibit abnormal, asymmetrical, or absent reflex response [10]. Children with ID typically show reduced motor skills relative to age-matched peers and neurodevelopmental norms, correlating with ID severity and manual dexterity [33, 34]. In isolated ID cases, PR persistence is found in 24.6% of cases and is more common in comorbidities like ASD or CP. The prevalence of retained PRs increases with ID severity, being higher in severe and profound than in mild or moderate cases [29].

An early study reported a higher prevalence of the palmomental (47% *vs*. 7%) and snout reflexes (14% *vs*. 0%) in individuals with Down syndrome compared to healthy controls [35]. However, with a mean age of 37 years in the Down syndrome group, these findings rather represent reemergence of PRs than their retention [8].

In ASD, Minderaa et al. [29] observed increased prevalence of snout and visual rooting reflexes (VRR) compared to TD controls. Teitelbaum et al. (2002) [36] noted persistence of some PRs in ASD cases, particularly the asymmetrical tonic neck reflex. De Buildt et al. [37] found the VRR more common in ASD individuals with ID (43.9%) than in those with ID alone (24.6%), and its persistence correlated with lower global cognition and adaptive functioning. Chinello et al. [11] reported greater PRs persistence in 12–17-month-old infants whose parents exhibited higher subclinical autistic traits.

Healy et al. [13] found a higher prevalence of the snout and VRR in children with ASD compared to peers with developmental delays or TD, matched for age (4–6 years). They proposed that persistence of these reflexes may serve as developmental biomarkers for ASD, potentially

Table 1. Major primitive reflexes in infancy, their time-line and purpose [1, 2, 3]

PR	Time-line	Purpose
Moro	From 34th GW to 3–4 months post birth	Instant arousal of survival systems
Palmar grasp	From birth until 5–6 months post birth	Aid clinging (protection from fall)
Walking/stepping	From birth until 6 weeks	Preparation for voluntary locomotion
Doll's eye (oculocephalic reflex)	Disappears within a week or two after birth	Gaze fixation on stationary objects
Rooting (tactile or visually evoked)	From 28 GW to 3–4 months of age	Food finding; breastfeeding
Sucking	Appears at 28 GW and disappears within 24 months	Feeding
Snout	Appears at 26 GW and disappears within 3–6 months	Feeding
Glabellar	Disappears within 3–6 months	Blinking in response to tactile stimulation – eyes protection
Asymmetrical tonic neck reflex	From 18 GW to 3–6 months after birth	Assists through birth canal and in developing cross pattern movements
Galant	From birth until 3–9 months	Assists baby with birth process, crawling and creeping

PR – primitive reflex; GW – gestational week

Table 2. Primitive reflexes across the lifespan – prevalence, clinical significance, associated conditions [1, 2, 3, 8, 40]

Lifespan segment	Prevalence	Clinical significance	Associated conditions
Prenatal	Present in all healthy fetuses since the third trimester	Normal finding	/
Infancy and toddlerhood (birth to 2 years)	Present in all healthy infants until 6 months	Normal finding within 6 months after birth	May indicate maturation delay if present beyond 6/24 months
Early childhood (2–6 years)	Present in 40–90% healthy children	Isolated PRs can be detected in healthy subjects	May indicate maturation delay or neurodevelopmental disorders, cerebral palsy
Middle childhood and adolescence (6–18 years)	Up to 65% of healthy individuals	Isolated PRs can be detected in healthy subjects	May indicate brain injury affecting frontal lobe or diffuse cortical lesions
Adulthood (18–65 years)	6–47% of neurological intact individuals	Isolated PRs can be detected in healthy subjects	May be sign of brain injury or development of progressive neurological condition
Late adulthood (older than 65 years)	40–100% of neurologically and cognitively intact individuals	Isolated PRs can be detected in healthy subjects but correlates with cognitive status; may be warning sign of cognitive deterioration	May be sign of brain injury or development of progressive neurological condition

PRs – primitive reflexes

facilitating earlier diagnosis, especially in cases with comorbid ID [13].

In a cohort of 81 infants with ID without motor disturbances, Futagi et al. [38] observed tendencies toward retained Galant reflex, asymmetric tonic neck reflex, and plantar grasp. An earlier prospective study of 53 infants with developmental delays but no physical disabilities reported delayed postural adjustment reflexes relative to chronological age [39].

PRIMITIVE REFLEXES ACROSS THE LIFESPAN

In Table 2, the reported prevalence, clinical significance, and associated conditions of PRs across the lifespan are summarized [1, 2, 3, 40]. While PRs are typically present during early childhood as part of normal neurodevelopment, reflecting immature cortical control, particularly within the frontal lobes, their gradual suppression within the first year after birth signifies cortical maturation and enables the development of voluntary movements. Persistence of these reflexes beyond infancy or their reemergence in advanced aging often indicates delayed or disrupted frontal cortical development or neurodegeneration predominantly affecting cortical networks and their connections, sometimes heralding the onset of cognitive decline [41].

PRIMITIVE REFLEXES IN NORMAL AGING

Persistent PRs, including the palmomental reflex, snout reflex, and the sucking reflex can be detected across all ages in individuals with TD, including older adults without neurological or cognitive impairments, with their reappearance increasing with age [8, 42]. Early studies reported that about one in six healthy adults exhibits at least one PR, with palmomental reflex present in 6–27% of young adults (20–50 years) and 28–60% of those over 60 years [8, 10, 42]. Results of the Maastricht aging study reported at least one PR in 47% of men and 51% of those aged 25–45 years, with no associated cognitive dysfunction [41]. Snout reflex appears in 13% of individuals aged 40–57 years and 22–33% of those above 60; the sucking reflex is observed in 6% of normal individuals aged 73–93 years [8, 10, 40, 42]. Retained PRs have been detected in 33% of neurologically and cognitively healthy aging subjects (age range 45–91 years), and their presence correlates with decreased performance on cognitive tests compared to those without PRs, although suntil within the normal range [40]. This indicates that retained PRs may be a warning sign of incipient cognitive decline.

Among isolated, subtle, neurological abnormalities (ISNAs) in neurologically and cognitively healthy individuals, PRs are the most common. They have been linked to

magnetic resonance imaging (MRI) markers of small vessel disease, the most frequent form of vascular brain changes associated with cognitive decline and dementia, including deep and subcortical white matter hyperintensities, lacunar infarcts, and subcortical atrophy [43, 44, 45]. In addition, ISNAs, including PRs, also correlate with vascular risk factors such as hypertension, hypertriglyceridemia, and apolipoprotein E (APOE) ε4 carrier status [44, 46]. These findings suggest vascular brain damage as a potential cause and emphasize the clinical relevance of PRs as early indicators of underlying pathology [44, 46]. Vascular and degenerative changes accumulate over decades with no overt cognitive or functional symptoms but likely impair brain networks [44]. These changes are likely causing PRs reemergence as an epiphenomenon of covert brain damage that may precede cognitive decline [44].

PRIMITIVE REFLEXES IN DEMENTIA

The reemergence of PRs is frequently observed in conditions affecting the frontal lobes or their associative areas, including Alzheimer's disease (AD), vascular dementia, frontotemporal dementia, Parkinson's disease, and other neurodegenerative disorders [8, 45, 47]. Early studies reported PR prevalence rates of 16% in healthy controls, 32% in mild cognitive impairment, and 58% in dementia, indicating a progressive increase linked to cognitive decline [6]. Additionally, the presence of any PR increased the likelihood of other neurological signs, such as bradykinesia [46].

A recent meta-analysis of observational and cohort studies demonstrated that individuals with dementia have a four- to 16-fold higher risk of exhibiting PRs, particularly the grasp reflex, compared to healthy controls [8]. The snout reflex was the most commonly detected, present in about one half of cases, while the grasp reflex posed the highest dementia risk, suggesting links between specific PRs and frontal cortex volume or connectivity [8]. Further insights into PR reemergence come from 18F-fluorodeoxyglucose positron emission tomography studies, revealing hypometabolism in the superior frontal gyrus and putamen and implicating corticostriatal motor circuit dysfunction (supplementary motor area–putamen–thalamus) in dementia patients [48].

Clinical significance of primitive reflexes in dementia

A strong link between PRs and cognitive dysfunction is well documented. The presence of PRs correlates with poorer performance in global cognition, executive function, attention, and language in older adults, suggesting they may serve as early indicators of cognitive impairment [8]. Patients with degenerative dementia and PRs exhibit more severe cognitive and functional decline than those without PRs [48]. Notably, cerebral metabolism was lower in patients with PRs in the bilateral superior frontal gyri, bilateral putamina, and thalami, with no brain regions showing increased metabolism compared to those without, indicating distinct mechanisms of PR reemergence *vs.* persistence [48]. Analysis of individual reflexes showed similar hypometabolic brain regions, yet these did not overlap with primary affected areas in AD or frontotemporal dementia, highlighting a unique pathophysiology associated with PRs [49]. Additionally, PR incidence increases with advancing age.

The presence of ISNAs, including PRs, is observed across all mild cognitive impairment subtypes, particularly in cases involving multiple cognitive domains and carriers of the *APOE* ε4 allele. These abnormalities correlate with MRI evidence of silent small vessel disease and subcortical atrophy [49]. Cognitive decline and PR presence likely share neurodegenerative mechanisms, with prefrontal neural networks being especially susceptible to aging-related deterioration manifested as structural degradation, connectivity loss, and functional inefficiency [50].

CONCLUSION

In infants, PRs serve as evolutionary adaptations that enhance survival during early life by providing immediate protection, aiding feeding, and stimulating neural pathways crucial for subsequent motor and sensory development. As infants mature, most PRs gradually integrate into more complex behaviors that support motor control, cognitive processing, and emotional regulation. The persistence of PRs in ID remains understudied, with unclear underlying mechanisms and clinical impacts. While retained PRs may indicate neurodevelopmental disorders such as ID, many children exhibiting these reflexes develop without impairments. In adults, particularly the elderly, persistent PRs are considered pathological, signaling frontal lobe dysfunction or disrupted connectivity between frontal and other brain regions, and serve as a warning sign for potential cognitive decline.

Across the lifespan, there is strong evidence linking motor and cognitive functions both in children with developmental and intellectual delays and in adults experiencing progressive cognitive decline and dementia. CNS maturation involves a transition from brainstem-mediated reflexes to cortically controlled responses; however, vulnerability of the frontal cortex to aging, vascular, and degenerative processes leads to loss of top-down regulation.

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Примитивни рефлекси и мозак у развоју и одраслом добу – од интелектуалне ометености до деменције

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САЖЕТАК

Примитивни рефлекси су невољни моторички одговори који се активирају сензорним стимулусима и имају кључну улогу у храњењу, преживљавању и заштити у неонаталном периоду. Иако су засновани на интегритету централног нервног система и неопходни у моторичком развоју и сензорном процесирању, током сазревања фронталног кортекса инхибирају се и интегришу. Њихово трајање након 12 месеци од рођења може указивати на неуроразвојно кашњење, укључујући интелектуалну ометеност, церебралну парализу, поремећај из спектра аутизма и друге поремећаје. На другом крају животног спектра поновна појава примитивних рефлекса или присуство дезинхибиционих феномена често се јавља код старијих пацијената са свим врстама деменције, што указује на дифузну церебралну дисфункцију и лезије фронталног режња које слабе кортикалну инхибицију активности мождано стабла. Циљ овог рада је да сумира главне клиничке импликације перзистентних примитивних рефлекса код особа са когнитивним изазовима (као што су интелектуална ометеност и деменција) на различитим крајевима животног спектра и да упореди њихову неуробиолошку основу и прогностичку вредност ради бољег разумевања овог феномена.

Кључне речи: примитивни рефлекси; интелектуална ометеност; деменција; моторички развој; когнитивни развој