Biomarkers for Assessing Diet-Related Neurocognitive Deficits in Children—A Systematic Review

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Abstract: Neurocognitive deficits in children could be irreversible and detrimental to the overall wellbeing of children. Typically, children with this illness live lives below their mental and intellectual potential. The aim of this paper was to review primary evidence on the association between inflammatory biomarkers on neurocognition in children. Electronic databases such as Scopus, Cochrane Library, and PubMed were systematically searched to include all published data from 2000 to October 2023. The keywords included serum biomarker, cognition, executive function, intellectual ability, brain-derived neurotrophic factor, neurocognitive deficits, tau proteins, and children. A total of 8512 journal publications were obtained, but after the removal of duplicates, commentaries, and review papers, 9 papers were accepted for review. C-reactive protein, brain-derived neurotrophic factor (BDNF) tumor necrosis factor alpha (TNF-α), fibrinogen, plasma leptin, soluble tumor necrosis factor receptor 1 (sTNFR1), and copper were associated with neurocognition in the subjects. This review revealed that there is no research published in sub-Saharan Africa and most of the sample sizes in the studies were small.

Keywords: cognition; C-reactive protein; brain-derived neurotrophic factor

1. Introduction

Neurocognitive deficit is a reduction or impairment of cognitive function in the neural pathways or cortical networks in the brain and occurs especially when the brain experiences physical changes such as aging-related physiological changes of after neurological illness, mental illness, drug use, or brain injury [1]. They are usually characterized by a significant decline in at least one of the domains of cognition, which include executive function, complex attention, language, learning, memory, perceptual-motor function, or social recognition [2]. Infant and child cognitive development is dependent on several factors including genetics, mental health, social context, adequate nutrients, and their metabolism [3] Further, children who do not receive sufficient nutrition are at risk of exhibiting impaired cognition skills [4]. It is interesting to know that a child’s brain is highly plastic to accommodate a wide range of environmental interactions [5]. Accumulating evidence indicates that the food habits a child adopts during early years can have a big impact on their neurocognitive development [6–8]. Severe malnutrition contributes to inflammation characterized by increased levels of pro-inflammatory cytokines, especially interleukin 6 (IL-6) and TNF-α [9].

In recent years, there has been a growing concern over the prevalence of neurocognitive deficits in children such as mood disorders, learning difficulties, and Attention Deficit Hyperactivity Disorder (ADHD) [10]. Nutrition has been connected to neurocognition in several studies [11,12]. Blood-based biomarkers can successfully predict cognitive decline and the pathological progression of neurocognitive disorders such as Alzheimer’s disease [13]. For instance, vitamin D levels, iron status, omega-3 fatty acids, homocysteine
levels, C-reactive protein (CRP), brain-derived neurotrophic factor (BDNF), glucose, insulin, and antioxidant levels [14–21] may provide valuable insights into how diet may be influencing cognitive development [22]. They have the potential to give accurate, cost-effective results, and they are usually accessible to be widely used clinically, facilitating timely diagnoses [20]. These biomarkers serve as objective indicators, allowing for the identification of potential deficiencies or imbalances that can be associated with neurocognition [22]. The use of biomarkers in assessing neurocognition in children has significant implications for public health policies and also for the creation of intervention strategies [23]. Exploring biomarkers that are linked to cognitive development would equip scientists with validated tools to detect neurocognitive deficits in children, tailoring interventions and preventive strategies to suit the needs of a population before the physiological onset of cognitive impairment.

There have been a number of studies to examine the relationship between inflammatory biomarkers and cognition in humans, but these studies have focused on two extremities of the lifespan: preterm infants and elderly populations [23–26]. Further, despite numerous potential causes of childhood adversities and inflammation in low-resource settings [27], there are few studies that have examined the relationship between inflammation and neurocognition in children [28]. Although several commentaries exist, to the best of our knowledge, there is no comprehensive review on the association between inflammatory biomarkers and neurocognition in children. We hypothesize that some inflammatory biomarkers are associated with neurocognition in children. Therefore, the aim of this review is to assess primary evidence on the relationship between inflammatory biomarkers and neurocognition in children.

2. Method
2.1. Literature Search

This study was carried out in accordance with the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) [29]. A systematic search for all published articles on the relationship between inflammatory biomarkers and neurocognition in children was conducted by two independent reviewers on the databases Scopus, Cochrane Library, and PubMed from 2000 to October 2023. Based on the literature, the exposure variables were serum biomarkers, amyloid beta and tau proteins, CRP, BDNF, and homocysteine, while the outcome variables were cognition, executive function, neurocognition, and intellectual ability.

These search terms were applied to PubMed, Cochrane Library, and Scopus on said day between 11:20 a.m. and 11:47 a.m. GMT.

2.2. Selection of Studies
2.2.1. Eligibility Criteria

This review employed the following inclusion criteria for the systematic selection of articles: (i) the articles had to constitute original research; (ii) they needed to be composed in English; (iii) access to the full text of the article was required; (iv) they needed to present findings on biochemical markers, and the outcomes were mandated to be directly relevant to the relationship between biochemical markers and at least one outcome variable; (v) the exposure variables were inflammatory markers such as serum biomarkers, amyloid beta and tau proteins, CRP, BDNF, and homocysteine; (vi) the outcomes measured included cognition parameters such as executive function neurocognition and intellectual ability; (vii) the study designs considered were observational studies, specifically cross-sectional studies; and (viii) the study population encompassed infants and children up to 15 years of age.

2.2.2. Exclusion Criteria

The exclusion criteria were (i) articles that were published as conference papers, review papers, editorial letters, or commentaries; (ii) studies where participants were given
interventions; (iii) studies that recruited diseased individuals; (iv) animal studies; and (v) studies wherein participants were older than 15 years.

2.3. Data Extraction

Data extraction was completed for all included studies by two reviewers (MA and FFAPE). The characteristics extracted included author, year of publication, country, study design, sample size, age, the specific biomarker(s) studied, the cognitive parameter(s) used, and the study outcome.

3. Results

3.1. Literature Search Results

A comprehensive dataset comprising a total of 8512 records was retrieved from the databases PubMed \((n = 992)\), Cochrane Library \((n = 154)\), and Scopus \((n = 7366)\). The meticulous review process to eliminate duplicate entries resulted in 6340 studies. Subsequently, the screening of titles and abstracts led to the identification of nine full-text articles that rigorously adhered to the predefined systematic review criteria. This procedural framework is visually presented in Figure 1 for enhanced clarity.

![Figure 1. Summary of the screening process.](image)

3.2. Study Designs

Of the nine \((9)\) studies retrieved after our search, eight \((8)\) were cross-sectional designs. Only one study \([30]\) was a longitudinal cohort study where biomarkers related to cognition were assessed prospectively at defined intervals.
3.3. Study Population, Setting, and Country

The study population had similar characteristics: the subjects were all healthy males and females, mostly in their formative and early adolescence years, aged between 6 weeks and 15 years. Most studies (3) reviewed in this paper were conducted in Spain [31–33]. There were two studies from Bangladesh [30,34], while there was a study each from the USA [35], Korea [36], Brazil [37], and China [38]. There was no study conducted in Africa.

3.4. Biomarkers Considered in This Study

The studies found considered the following inflammatory biomarkers: CRP, IL-6, IL-1β, TNF-α, BDNF, serum cortisol, redox status of leptin, Adiponectin, Resistin, soluble tumor necrosis factor receptor 1 (sTNFRI), sTNFR2, chemokines, total plasma homocysteine concentration (tHcy), neuron-specific enolase (NSE), myelin-basic protein (MBP), calcium-binding protein (CBP), Granulocyte Monocyte Chemoattractant Protein-1 (MCP-1), Chemokine CC Motif Ligand 22 (ccl-22), GCSF, and Granulocyte Colony-Stimulating Factor (GCSF).

3.5. Main Findings

The findings of the review are presented in Table 1. An important variable for assessing cognition in the studies was intelligent quotient, as reported by most studies [30,32,34–36]. There were other crucial cognition assessment variables that were utilized, including academic performance, executive function [31], working memory, verbal memory, fine motor speed, decision making, and inhibition [33]. The Wechsler Preschool and Primary Scale of Intelligence for IQ was used in three studies [30,32,36]. Other assessment tools used were Muller’s scale of Early learning [34] and the Stanford–Binet Intelligence Scale (IV). The Delis–Kaplan Executive Function System (D–KEFS) was used to test for executive function [31], and the Bayley-III test was used to evaluate infant and toddler development [37].

Table 1. Summary of study characteristics and outcomes listed in alphabetical order by first author’s surname.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Country</th>
<th>Study Design</th>
<th>Sample Size</th>
<th>n(M), n(F)</th>
<th>Age</th>
<th>Biomarker(s)</th>
<th>Cognitive Parameter(s)</th>
<th>Outcome(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adelantado-Renau et al., 2019 [31]</td>
<td>Spain</td>
<td>Cross-sectional</td>
<td>107</td>
<td>44, 63</td>
<td>10.0 ± 1.1 years</td>
<td>WBC, IL-6,IL-1β, TNF-α, and CRP</td>
<td>Academic performance, executive function, cognitive inhibition, and working memory</td>
<td>CRP was associated with behavioral and emotional functioning; TNF-α was associated with mathematics scores</td>
</tr>
<tr>
<td>Arija et al., 2006 [32]</td>
<td>Spain</td>
<td>Cross-sectional</td>
<td>83</td>
<td>43, 40</td>
<td>6.0 ± 0.0 years</td>
<td>Complete hematological profile and tHcy</td>
<td>Intelligence quotient (IQ)</td>
<td>No biochemical parameter was a significant predictor of IQ</td>
</tr>
<tr>
<td>Bach et al., 2022 [30]</td>
<td>Bangladesh</td>
<td>Longitudinal</td>
<td>122</td>
<td>66, 56</td>
<td>6 to 53 weeks</td>
<td>CRP</td>
<td>Intelligence quotient (IQ)</td>
<td>CRP was associated with IQ</td>
</tr>
<tr>
<td>Beers et al., 2007 [35]</td>
<td>USA</td>
<td>Cross-sectional</td>
<td>30</td>
<td>13, 17</td>
<td>3 months to 12 years</td>
<td>NSE, CBP, and MBP</td>
<td>Intelligence quotient (IQ)</td>
<td>NSE and MBP were associated with IQ</td>
</tr>
<tr>
<td>Caldu et al., 2023 [33]</td>
<td>Spain</td>
<td>Cross-sectional</td>
<td>105</td>
<td>49, 56</td>
<td>12 to 21 years</td>
<td>IL-6, TNF-α, CRP, and fibrinogen</td>
<td>Working memory (WM), cognitive flexibility (CF), inhibitory control (IC), decision making (DM), verbal memory (VM), and fine motor speed (FMS)</td>
<td>CRP was associated with DM and VM, TNF-α was associated with IC, and fibrinogen was associated with VM</td>
</tr>
<tr>
<td>Camargos et al., 2017 [37]</td>
<td>Brazil</td>
<td>Cross-sectional</td>
<td>50</td>
<td>50</td>
<td>6–24 months</td>
<td>Leptin, Adiponectin, Resistin, sTNFRI, sTNFR2, chemokines, BDNF, serum cortisol, and redox status</td>
<td>Cognitive development</td>
<td>Plasma leptin and sTNFRI were associated with cognitive development</td>
</tr>
</tbody>
</table>
Table 1. Cont.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Country</th>
<th>Study Design</th>
<th>Sample Size</th>
<th>n(M), n(F)</th>
<th>Age</th>
<th>Biomarker(s)</th>
<th>Cognitive Parameter(s)</th>
<th>Outcome(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xie et al., 2019 [34]</td>
<td>Bangladesh</td>
<td>Cross-sectional</td>
<td>130</td>
<td>56, 74</td>
<td>6 to 104 weeks</td>
<td>CRP</td>
<td>Intelligence quotient (IQ)</td>
<td>No significant association between CRP and IQ</td>
</tr>
<tr>
<td>Yeom et al., 2016 [36]</td>
<td>Korea</td>
<td>Cross-sectional</td>
<td>28</td>
<td>13, 15</td>
<td>5–7 years</td>
<td>BDNF</td>
<td>Intelligence quotient (IQ)</td>
<td>BDNF showed a significant correlation with IQ</td>
</tr>
<tr>
<td>Zhou et al., 2015 [38]</td>
<td>China</td>
<td>Cross-sectional</td>
<td>826</td>
<td>439, 387</td>
<td>10–14 years</td>
<td>Copper</td>
<td>Working memory</td>
<td>Significant association between high copper levels and poor working memory</td>
</tr>
</tbody>
</table>

n (M) = number of males, n (F) = number of females, WBC = white blood cell, BDNF = brain-derived neurotrophic factor, IL-6 = interleukin-6, IL-1β = interleukin-1β, TNF-α = tumor necrosis factor α, and CRP = C-reactive protein (CRP), tHcy = total plasma homocysteine concentration, NSE = neuron-specific enolase, MBP = myelin-basic protein, CBP = calcium-binding protein, soluble tumor necrosis factor receptors 1 and 2 = sTNFR1 and sTNFR2, MCP-1 = Granulocyte Monocyte Chemoattractant Protein-1, CCL22 = Chemokine CC Motif Ligand 22, GCSF = Granulocyte Colony-Stimulating Factor.

CRP, the most reported biomarker, was associated with almost all cognitive assessment tools in specific studies. Specifically, CRP was associated with behavioral and emotional functioning [31], intelligent quotient [30], decision making (DM), and verbal memory (VM) [33]; however, it was not associated with IQ in another study [34]. TNF-α was also associated with mathematics scores and decision making [33], while serum neuron-specific enolase (NSE) and myelin-basic protein (MBP) were associated with IQ [35]. Fibrinogen was found to have an association with verbal memory [33]. Plasma leptin and sTNFR1 were both associated with cognitive development [37]. BDNF showed a correlation with intelligence quotient [36], while higher copper levels were associated with poor working memory [38].

4. Discussion

This systematic review aimed to evaluate primary evidence on the association between inflammatory biomarkers and neurocognition in children. All participants (n = 1481) were below the age of 15 years. This cut-off was chosen since there is evidence that cognitive abilities and brain structures undergo significant changes prior to this age [39]. Also, the brain is highly plastic during childhood, allowing for greater potential for rehabilitation and intervention [40]. Lastly, cognitive deficits in childhood can have long-term consequences, affecting academic, social, and emotional development. Focusing on this age range will help contribute to the development of tools and strategies that can have a lasting positive impact on a child’s future [41].

The most studied cognition parameter was IQ (55.6%), followed by working memory (33.3%). IQ is usually measured by the Wechsler Preschool and Primary Scale of Intelligence for IQ, Mullen’s scale of Early learning [34], and the Bayley-III test. Assessing IQ is crucial because IQ scores provide an estimate of the person’s general intelligence levels and can help diagnose intellectual disabilities [42]. The most studied biomarker among the studies was CRP, which was examined in multiple studies, accounting for 44.4% of the total studies included in this review. On the other hand, copper was the least studied biomarker, featured in only one study, constituting 11.1% of the total studies. BDNF, IL-6, and TNF-α were the second most studied biomarkers (22.2%). CRP showed a significant inverse association with cognitive parameters, specifically IQ in Bangladesh [30], decision making (DM) and verbal memory (VM) in Spain [33], and emotional and behavioral function also in Spain [31]. BDNF showed a significant inverse association with IQ in Korea [36]. TNF-α also showed a significant inverse association with mathematics scores [31] and inhibitory control [33]. Other biomarkers, such as soluble TNF receptor 1 (sTNFR1) and leptin, showed significant inverse association with cognitive development in a study in Brazil [37].

CRP may induce cerebral atherosclerosis and trigger microvascular and macrovascular lesions that can improve the uptake of low-density lipoproteins by macrophages while...
promoting foam cell formation, by impounding endothelial function, or by inducing the abnormal migration and proliferation of human vascular smooth cells. Further, BDNF induces inhibitory and excitatory neurotransmission in the brain, causing loss of synaptic refinement and impairment of learning memory, and was observed to be significantly inversely associated with IQ [36]. Although some other studies have evaluated the role of BDNF in synaptic plasticity, synaptic connectivity formation, and neuronal survival [36], research has found out that high levels of BDNF show significant impairment in learning and memory. In addition, TNF-α was also noted to be inversely associated with mathematics scores [31] and inhibitory control [33]; the exact reason for this is not known, but TNF-α has crucial roles in the inhibition of neurogenesis [43].

Other biomarkers, such as soluble TNFR1, are associated with increases in cognitive and motor development [37], and this could be attributed to their functions in increasing the number of surviving motoneurons in the postnatal nervous system [44]. Leptin, on the other hand, was found to be inversely associated with cognition. Although leptin has a central role in hippocampus and plays a crucial role in learning and memory processing, obesity is associated with elevated leptin levels and with limited cognitive functions; hence, leptin levels could contribute to memory impairment due to leptin resistance in the brain [45,46]. Copper is involved in several enzyme systems and involved in neurotransmitter generation; however, excess copper levels could induce cytotoxicity and cognitive decline [38], and this could be the reason why participants in one of the studies reviewed [38] experienced poor working memory with excess copper.

The cause of inflammation in these children is diverse. This is because, of the studies reviewed, children in eight out of nine studies were reported in healthy children; however, there were some reported factors worth noting. Specifically, some of the children were without any apparent risk of malnutrition and with normal or high intellectual capacity [32]; some were living in limited resource settings [30], while others were reportedly obese [37]. The study in the United States [35] reported that some of the children were inflicted with traumatic brain injury, and this could contribute to inflammation [35]. Children from low-resource settings are at an elevated risk of exposure to biological, environmental, and psychological adversities, including polluted environments, food insecurity, and infectious diseases [47]. Food insecurity contributes to malnutrition in populations [48]. It is interesting to note that systemic inflammation increases in children with severe acute malnutrition [49], releasing inflammatory biomarkers such as CRP, BDNF, and TNF-α that could affect brain development. In some populations, inflammation and malnutrition coexist as part of a two-way causal malnutrition–infection cycle, whereby undernutrition increases the risk and severity of inflammation and infection, and inflammation impairs nutritional status by decreasing food intake and impairing micronutrient absorption [50]. On the other hand, overweight and obesity in childhood are associated with a cascade of neuroendocrine inflammatory changes that activates a chronic low-grade inflammation state with high levels of leptin and sTNFR1 [51]. Numerous biomarkers are related to neurocognition in children, and this makes the pursuit of identifying an inflammatory biomarker associated with neurocognition an exciting venture.

The results from this review indicate that there is no published work on the relationship between inflammatory biomarkers and cognition in sub-Saharan Africa and Australia compared to three (3) papers published in Europe, four (4) in Asia, one (1) in America, and one (1) in South America. Research states that there is limited data on the burden of mental disorders among children in the general population in Africa [52,53]. The situation could be alarming in Africa because they usually have low incomes, where poverty, diseases, and conflict dominate, and these conditions increase the risk of these disorders. Additionally, cognition deficits appear to be widespread in many of these populations [54]. The absence of routine cognitive tests in early childhood, integrated into child welfare clinic protocols, makes it challenging to identify cognitive deficits at an early stage. Therefore, the identification of a robust biomarker for early-life cognition assessment could serve as a crucial preventative measure. Such a biomarker would play a pivotal role
in the early detection of neurocognitive deficits, laying a solid foundation for suggesting and implementing targeted interventions within vulnerable groups. Another point worth noting is that many developing countries may prioritize research in other healthcare areas, such as infectious diseases, maternal and child health, and communicable diseases, due to their immediate public health impact. Nonetheless, a recent study reported poor cognition scores among school-aged Ghanaian children [55]. Also, the stigmatization that comes with neurocognitive deficits and the cultural beliefs about the causes of such conditions may be plausible reasons for the data paucity in this part of the world. This observation may be explained by these factors and an apparent publication bias.

4.1. Research Gaps

This review further observes a lack of focus on diverse age groups across the human lifecycle in the existing literature. Prevailing knowledge indicates that distinct age groups are prone to exhibit varying characteristics with regard to nutrition and health. This complexity arises due to the unique milestones and developmental stages associated with each age group. Therefore, recognizing and applying age distinctions within the various age groups is imperative for understanding the interplay between nutrition and cognition at different life stages. This was not found in the review as different age groups were clustered together; for instance, if a study considers age groups from 3 months to 12 years [35], it may be difficult to clearly understand the problem as this age will have infants, toddlers, preschoolers, and school-aged children lumped together in a single study. Consequently, the distinct age groups were not representative enough; hence, it would be key for research studies to explore biomarkers and cognition in distinct age groups with similar or different characteristics. This aspect was not identified in this review, as various age groups tended to be grouped together.

From this review, inflammation may be associated with cognition [30,31,34]. From mid-fetal development through middle and late childhood, a number of elements of brain development, including alterations in synapse quantity and myelin integrity, are influenced by environmental factors [30]. The early development of the brain may be impacted by the type and timing of inflammatory insults. If inflammation happens during a critical neurodevelopmental process, such as cell migration or dendritic sprouting, it may have a deleterious effect on the development of the fetus’s brain [30]. The effects of inflammation observed at the molecular level may in turn contribute to changes in brain structure. Consequently, blood-based inflammatory mediators may enter the central nervous system via many routes and hence impact brain function. [31]. However, there is a need for further studies to measure sustained systemic inflammation and evaluate specific neurological changes that can mediate the relationship between inflammation and developmental outcomes [30]. Consequently, it will be important to understand the pathways by which inflammation is linked to brain health while assessing the long-term and short-term role of systemic inflammation [31]. Specifically, studies on BDNF metabolism, those on clarifying its role, and those on the relevance of a difference in peripheral BDNF as a potent biomarker for neurocognitive development [36] would be groundbreaking. Currently, only nine papers in this field have been published in 23 years. They reported the variable biomarkers assessed and the cognitive function tools that were utilized. In general, these reports indicate that this area requires more exploration with carefully targeted age groups.

4.2. Limitations and Future Directions

While all the studies scrutinized presented encouraging outcomes regarding biomarkers and cognition, a common limitation across them was the use of small sample sizes. Hence, there is a pressing need to carry out these investigations on a larger scale, employing longitudinal and interventional approaches and conducting multi-center studies to corroborate these findings. This will not only validate the reported results but also ascertain the broader applicability of biomarkers in assessing cognition. A limitation to our
work could be the timeframe with which the manuscripts were retrieved. We may have missed some papers that were published later than October. We only accessed electronically published papers, so we could have missed information from other databases that are not electronic yet.

The future for biomarker exploration and cognition is vast because it promises to serve as a preventive approach to reducing the prevalence of neurocognitive disorders in children of distinct age groups. This could be achieved effectively by investigating biomarkers in distinct age groups with similar or different characteristics and its role in cognition. Curative medicine is paving the way for preventive medicine; hence, this approach can be part of the preventive tools that will set into motion the ‘future of biomedicine’.

5. Conclusions

The current systematic review reveals that a number of biomarkers such as CRP, TNF-α, BDNF, sTNFR1, leptin, and copper influenced cognitive development to various degrees, although CRP and TNF-α appear to be the most practical and effective biomarkers for assessing neurocognitive deficits. While some showed an increase in cognition, others gave contrasting results. The variation in biomarker associations establishes the complexity of cognitive development and the need for further investigation into the role of these markers in children’s cognitive function. These biomarkers could be explored further to identify a potent biomarker for cognition assessment in various populations. This could help with routine checks of cognition deficits and improve preventive healthcare approaches in solving the growing incidence of neurocognitive deficits in populations.

Author Contributions: M.A. and R.K. conceived the idea. F.A.A.P.E., M.A. and R.K. searched for the papers and wrote the manuscript. F.A.A.P.E., M.A. and R.K. critically edited the manuscript. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflicts of interest.

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