South African Journal of Psychiatry
ISSN: (Online) 2078-6786, (Print) 1608-9685

Key biopsychosocial lessons from a South African birth cohort: A review and reflection

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Dates:
Received: 05 Feb. 2025
Accepted: 26 Sept. 2025
Published: 06 Nov. 2025

How to cite this article:
Koopowitz S-M, Hoffman N, Zar HJ, Stein DJ. Key biopsychosocial lessons from a South African birth cohort: A review and reflection. S Afr J Psychiat. 2025;31(0), a2478. https://doi.org/10.4102/sajpsychiatry.v31i0.2478

Background: Birth cohorts provide evidence regarding factors promoting child neurodevelopment, including optimal maternal mental health and a secure early environment. Few birth cohorts exist in low- and middle-income countries (LMICs), and it is unclear how findings from high-income birth cohorts generalise elsewhere.

Aim: This study aims to undertake a narrative review of psychosocial publications from the Drakenstein Child Health Study (DCHS) and to reflect on overarching themes.

Setting: The DCHS is based in the Western Cape, South Africa.

Methods: The DCHS is a birth cohort that has followed ~1000 mother–infant dyads since 2012, collecting maternal mental health and child neurodevelopment data, including brain imaging and genomics. This review (2013–2025) synthesises psychosocial publications and emerging themes within prior literature.

Results: Seventy-five psychosocial articles were reviewed. Four themes emerged: (1) prenatal and postnatal maternal mental health risk factors are associated with negative infant birth and child neurodevelopment outcomes; (2) prenatal and postnatal maternal physical health risk factors are associated with negative infant birth and child neurodevelopment outcomes; (3) we are beginning to understand the neural circuitry, genomic alterations, immunological changes and other mechanisms that underlie impaired neurodevelopment; and (4) an integrative biopsychosocial perspective is needed to fully understand and promote optimal child neurodevelopment.

Conclusion: Findings indicate that many high-income countries' observations generalise to a peri-urban South African setting while also highlighting context-specific pathways and intervention targets. These themes inform research priorities, interventions and policies to optimise child neurodevelopment in LMICs.

Contribution: This review highlights context-specific risk and resilience factors and emerging mechanistic pathways, underscoring the value of LMIC birth cohort research.

Keywords: psychosocial; birth cohort; LMICs; review; child development; DCHS.

Introduction

Birth cohorts prospectively collect data and employ multidisciplinary collaboration to examine factors associated with optimal child health and development.[1] In high-income countries (HICs), a broad range of such cohorts have been established (e.g. Avon Longitudinal Study of Parents and Children – ALSPAC,[2] Amsterdam Born Children and their Development – ABCD,[3] Norwegian Mother, Father and Child Cohort – MoBa,[4] UK Millennium Cohort Study[5] and Growing Up in Singapore Towards Healthy Outcomes – GUSTO[6]). Systematic review of this work indicates that birth cohorts have yielded knowledge of factors, such as maternal mental disorders, maternal substance use and early maternal–infant attachment, which influence child neurodevelopment.[7] Determining risk and resilience factors that impact neurodevelopment in childhood may provide important targets for intervention.[8]

There are relatively few birth cohorts in low- and middle-income countries (LMICs).[9,10,11] Studies in these settings tend to examine specific sub-populations rather than representative samples and have tended to be smaller and of shorter duration.[11] Potential challenges in this context include a lack of research infrastructure, difficulty with participant enrolment and retention, and limited funding.[11] Nevertheless, work on LMIC birth cohorts (e.g. Birth-to-Twenty Plus[12]; Pelotas Birth Cohort[13]) has

Note: Additional supporting information may be found in the online version of this article as Online Appendix 1.

been undertaken and has emphasised that poor childhood nutrition, maternal exposure to traumatic events and adverse childhood experiences can adversely impact neurodevelopment.[8,14,15]

Given the relative lack of birth cohorts from LMICs, the extent to which lessons from high-income cohorts generalise to these settings is unclear. This article reviews psychosocial publications from an ongoing South African birth cohort, the Drakenstein Child Health Study (DCHS), against the context of previous work on factors influencing child neurodevelopment. The DCHS collects data on maternal mental health and child neurodevelopment, including measures of maternal mental disorders, substance use and exposure to traumatic stressors, as well as investigations of brain imaging and genomics.[16,17,18]

This is a narrative review of psychosocial publications published between 2013 and early 2025, reflecting on the overarching themes that emerge against the context of previous work.

Methods

The research question for this study was: what overarching themes emerge from psychosocial publications in the DCHS? To address this question, we conducted a narrative review of publications from this study. The DCHS team maintains a comprehensive, continuously updated database of all publications arising from the cohort. For this review, we drew on this database to identify all psychosocial publications. The initial search was conducted in March 2024 and was updated in February 2025.

Inclusion criteria for this review included original peer-reviewed research articles conducted on DCHS psychosocial data, including articles focused on neurodevelopmental outcomes. Exclusion criteria included reviews, methods papers, commentary pieces and grey literature, such as preprints, dissertations, conference articles and articles in which DCHS data were pooled with other cohort data. For this review, the first author screened all publications in the database, and uncertainties about inclusion were discussed with senior co-authors until consensus was reached. From each included article, sample size, exposures, outcomes and main findings were extracted using a standardised template. We then synthesised the findings thematically, grouping them into four overarching domains. The overarching themes were developed through discussion by all authors.

Mothers in the DCHS provided informed consent at enrolment and were re-consented annually. Assent was obtained annually from children from 7 years of age depending on their neurocognitive ability.

Ethical considerations

The DCHS was approved by the Faculty of Health Sciences, Human Research Ethics Committee, University of Cape Town (401/2009) and by the Western Cape Provincial Health Research Committee. Mothers provided informed consent at enrolment and were re-consented annually. Consent was performed in the mother's preferred language: English, Afrikaans or isiXhosa.

Results

Eighty-one articles were identified in the database. Six articles were excluded (*n* = 3 reviews; *n* = 2 methods; *n* = 1 conceptual framework) from this review. Online Appendix Table 1-A1 reflects each of the 75 included articles, including sample size, outcomes and overarching themes. Reflection on the extracted data suggested four overarching themes:

- Prenatal and postnatal maternal mental health risk factors are associated with negative infant birth and child neurodevelopment outcomes.[19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48]
- Prenatal and postnatal maternal physical health risk factors are associated with negative infant birth and child neurodevelopment outcomes.[49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73]
- We are beginning to understand the neural circuitry, genomic alterations, immunological changes and other mechanisms that underlie impaired neurodevelopment.[21,36,39,42,44,51,52,55,59,60,61,67,71,72,73,74,75,76,77,78,79,80,81,82]
- Understanding neurodevelopment requires an integrative biopsychosocial approach.[23,40,83,84,85,86,87,88,89,90,91,92,93]

Discussion

In this narrative review of 75 psychosocial DCHS publications, we identified four overarching themes: (1) prenatal and postnatal maternal mental health risks are associated with negative infant birth and child neurodevelopment outcomes; (2) prenatal and postnatal physical health exposures are associated with negative infant birth and child neurodevelopment outcomes; (3) we are beginning to understand the neural circuitry, genomic alterations, immunological changes and other mechanisms that underlie impaired neurodevelopment; and (4) understanding neurodevelopment requires an integrative biopsychosocial approach. These findings indicate that many HIC observations not only generalise to this peri-urban South African setting but also highlight context-specific pathways and intervention targets. Each of the four themes is explored next in more detail in the context of prior work.

Prenatal and postnatal maternal mental health risk factors are associated with negative infant birth and child neurodevelopment outcomes

In HIC birth cohorts, maternal mental health risk factors, such as depression and anxiety symptoms, have consistently been linked with poorer infant birth and child neurodevelopment outcomes.[7] For example, the Cohort for Childhood Origin of Asthma and Allergic Diseases study in Korea reported that maternal prenatal depression was associated with lower gestational age,[94] and the Danish National Birth Cohort reported that emotional stress during pregnancy was associated with shorter gestation.[95] In South African birth cohorts, maternal antenatal depression and anxiety have similarly been linked with lower gestational age[36] and adverse child neurodevelopmental outcomes.[96]

In DCHS, maternal mental health problems are frequently observed.[22,33,34,38] For example, we identified five depression trajectories in mothers: the most severe trajectory was associated with stressful life events, sexual intimate partner violence (IPV) and tobacco use.[34] Suicidal ideation and behaviour were reported by 19.9% of mothers antenatally, rising to 22.6% postnatally.[38] Mothers often report maltreatment during childhood (34%), IPV during pregnancy (33%),[31,32] and a lifetime history of post-traumatic stress disorder (PTSD).[37,41,45,91] Up to 75% of DCHS children have been exposed to some form of violence, either within the home or community by the age of 4.5 years.[47]

In DCHS, maternal mental disorders, psychological distress and exposure to traumatic stressors were negatively associated with infant outcomes.[25,26,35,43,46] Firstly, infants of mothers with antenatal depression and IPV exposure exhibited lower weight-for-age-*z*-scores, smaller head circumference and lower length-for-age *z*-scores.[19,22,25] Secondly, maternal antenatal trauma exposure was associated with smaller infant head-circumference-for-age *z*-scores.[26] Thirdly, maternal PTSD was negatively associated with infant gestational epigenetic age.[28]

In DCHS, a number of maternal mental health risk factors were negatively associated with early childhood outcomes. Maternal antenatal and postnatal IPV and PTSD were associated with poorer language and motor scores,[20] and with poorer fine motor and adaptive behaviour[27] at 2 years of age. Furthermore, postnatal maternal psychological distress and IPV exposure were associated with offspring having decreased functional residual capacity, severe lower respiratory tract infection and wheezing episodes in early childhood.[24,29,30] Conversely, an increase in maternal education and socioeconomic status was ssociated with improved infant developmental outcomes on measures of cognition, fine and gross motor, and receptive and expressive language.[23]

Associations between maternal mental health risk factors and childhood infection found in the DCHS may not be confined to LMIC contexts. The MoBa study reported a significant association between maternal prenatal stressful life events and infectious diseases in children up to the age of 11 months.[97] The EDEN Study reported an association between prenatal maternal depression and child allergic rhinoconjunctivitis.[98] More work is needed on the mechanisms underlying these associations,[99] and we will return to this point next.

Prenatal and postnatal maternal physical health risk factors are associated with negative infant birth and child neurodevelopment outcomes

In a systematic review of HIC cohorts, maternal physical health variables, such as exposure to air pollution, were associated with negative infant birth and child neurodevelopment outcomes.[100,101] Evidence from LMIC systematic reviews suggests similar findings, for example, air pollution has been associated with childhood asthma and impaired neurodevelopment in this context.[102,103] In addition, a systematic review of LMIC studies examined the adverse effects of HIV exposure on child neurodevelopment.[11] Given that mental disorders have physical underpinnings and sequelae, any distinction between mental and physical health risk factors is somewhat arbitrary; we include substance use in this section because as in the case of other physical health variables, the mechanisms that underlie its impact on infant outcomes are comparatively well understood.[104,105,106,107]

In DCHS, there was an association between prenatal indoor air pollution (PM10) exposure and impairment of cognitive, language, motor function and general adaptive behaviour domains at 2 years of age.[49,52,62] Furthermore, prenatal exposure to environmental pollutants, which included PM10 and prenatal smoking exposure, was associated with increased internalising behaviours in children aged 24–60 months.[50]

In DCHS, maternal HIV infection was consistently associated with altered neurodevelopment.[58,60,66,80] At 24 months of age, HIV-exposed uninfected (HEU) children scored lower than HIV-unexposed uninfected (HUU) children for receptive and expressive language.[58] The proportion of HEU children with developmental delay was higher than that of HUU children for receptive language.[58] At 3.5 years of age, HEU was associated with impaired expressive language,[53] indicating persistence of this association.

In DCHS, associations of prenatal alcohol and tobacco exposure with brain changes and neurodevelopment were studied.[63,65,68,69,71,72] Alcohol and tobacco use during pregnancy was associated with several negative infant birth and child neurodevelopment outcomes.[51,54,70] Firstly, maternal perinatal alcohol use or prenatal alcohol exposure (PAE) was associated with negative infant birth outcomes (low birth weight, shorter infant length, smaller head length, smaller head circumference and early gestational age).[55,76] Secondly, PAE was associated with decreased gross and fine motor functioning at 2 years of age[54] as well as subcortical surface area and subcortical volume differences.[56] Finally, antenatal maternal smoking was associated with a decrease in infant birthweight-for-age *Z*-score and adaptive behaviour outcomes.[49,57]

Several DCHS findings are consistent with work elsewhere. Thus, DCHS findings of associations of maternal PAE with adverse infant birth and child neurodevelopment outcomes are in line with existing research from LMICs.[102,108,109,110] While the literature on HEU has inconsistencies,[111] a systematic review found HEU children exhibited poorer expressive language and gross motor function than HUU children, but HEU children performed similar to HUU children on measures of cognitive development, receptive language and fine motor skills.[111] Taken together, this literature highlights the importance of targeting maternal PAE to ensure optimal child neurodevelopment, as well as the potential importance of targeting mechanisms that underlie HEU-associated neurocognitive deficits.

We are beginning to understand the neural circuitry, genomic alterations, immunological changes and other mechanisms underlying impaired neurodevelopment

In HIC cohorts, there is growing understanding of how associations of maternal mental and physical health with impaired neurodevelopment are underpinned by neural circuitry and genomic alterations.[112] There is, however, less evidence regarding underlying neural circuitry, genomic alterations, immunological changes and other mechanisms from LMIC cohorts.[11]

In DCHS, antenatal maternal depression was associated with reduced neonate amygdala and caudate nucleus volumes.[44] Furthermore, maternal antenatal IPV exposure was associated with a larger caudate nucleus among males and smaller amygdalae among females at 2 years.[77] These findings highlight the possible impacts of maternal psychosocial stressors on the development of subcortical regions and indicate that these associations may be persistent.

In DCHS, maternal antenatal alcohol use was associated with alterations in neurodevelopment and the mediating role of neuroimaging was investigated.[51,55,73,76] Firstly, the association between maternal antenatal alcohol use and neonatal neurobehavioural scores was mediated by lower fractional anisotropy and higher mean diffusivity in the inferior cerebellar peduncle at birth.[51] Secondly, the association between maternal antenatal alcohol use and adverse neurobehavioural outcomes at 6 months of age was mediated by decreased regional volumes in the temporal and prefrontal lobes of neonates.[73] These findings are important in indicating that early life brain alterations mediate the association between maternal alcohol use and adverse child neurodevelopment.

In DCHS, maternal HIV status and CD4 count have been associated with brain alterations in children.[79] Firstly, HEU infants exhibited reduced total grey matter volume relative to HUU infants.[59] Secondly, decreased infant grey matter volumes, particularly in the bilateral caudate, were associated with low maternal CD4 cell count (< 350 cells/mm^3); the dose–response association between maternal CD4 count and infant grey matter volume points to maternal immune activation altering brain development.[59] Thirdly, at ages 2–3 years, there were increased ratios of myo-inositol to total creatine in parietal brain regions in HEU children; these suggest ongoing neuroinflammatory processes.[75] Finally, the association between HIV exposure and poor language outcomes was mediated by cortical thickness in the medial orbital frontal cortex.[60] These findings indicate that early life brain structural and molecular changes mediate the association between maternal HIV and adverse child neurodevelopment.[59]

In DCHS, maternal anaemia during pregnancy was associated with structural brain differences in children aged 2–3 years,[61,67] with smaller bilateral caudate volumes, left putamen and corpus callosum relative to children of mothers without anaemia during pregnancy.[67] This study was repeated 4 years later in children aged 6–7 years and demonstrated that decreased volume in these regions persisted, and that maternal anaemia status was also associated with smaller volumes of the corpus callosum as well as the bilateral caudate nucleus at this time point.[61] These findings suggest that perinatal anaemia has persistent associations with brain development in regions associated with motor control, executive function and interhemispheric communication.

In DCHS, there is some evidence that epigenetic mechanisms, alterations in gene expression, immunological changes and other mechanisms are mediators of the relationship between prenatal exposures and neurodevelopmental outcomes.[36,39,42,81,82] Firstly, at 6 months of age, the association between maternal antenatal alcohol use and infant motor domain scores was mediated by 16 CpG sites.[74] Secondly, the association between prenatal PM10 (indoor air pollution) exposure and cognitive neurodevelopment at 2 years was mediated by DNA methylation of GOPC, RP11-74K11.1, DYRK1A and RMNT.[52] Thirdly, gene expression in neonatal cord blood differed in those born to mothers with and without psychopathology; co-expression network analysis identified two depression-related modules implicated in axon guidance and mRNA stability and two PTSD-related modules implicated in TNF signalling and cellular response to stress.[19] Fourthly, the association between maternal HIV status and poorer motor function at 2 years of age was mediated by reduced serum inflammatory markers (GM-CSF, IFN-γ, IL-10, IL-12p70, IL-1β, IL-2, IL-4, IL-6 and NGAL) at 6 weeks of age.[78] Finally, maternal antenatal psychological distress and maternal lifetime IPV were associated with altered bacterial profiles in infant and maternal faecal bacteria.[36] These findings highlight the role of genomic and immunological alterations in mediating the relationship between prenatal exposures and neurodevelopmental outcomes.

The DCHS findings complement HIC studies addressing how neural circuitry and genomic mechanisms underlie child neurodevelopment. The Avon Longitudinal Study of Parents and Children, for example, has made particular contributions to understanding epigenetic mediators of the association of maternal alcohol and tobacco use with adverse neurodevelopment[113] and genetic mediators of the association of maternal prenatal depression symptoms with childhood internalising and externalising behaviour.[114] Further research is needed, in both HICs and LMICs, to fully understand mechanisms underlying the association between maternal psychosocial risk factors and impaired child neurodevelopment. The next section focuses on how such research will require an integrative approach.

Understanding neurodevelopment requires an integrative biopsychosocial approach

We have observed that there is a growing understanding of the mechanisms that underlie associations between maternal psychosocial risk factors and impaired child neurodevelopment. This section focuses on the point that such mechanisms play out at different levels – the biological, the psychological and the societal – and that an integrative biopsychosocial approach is therefore required. Previous research in HIC birth cohorts has also outlined the complex interactions between biological, psychological and social risk and resilience factors; thus, for example, an interdisciplinary and integrative approach has been recommended to address perinatal mental health and its associations with infant neurodevelopmental outcomes.[115]

Emerging evidence from the DCHS highlights, for example, how maternal substance use influences a range of biopsychosocial pathways that may influence child neurodevelopmental outcomes. Thus, the relationship between increased IPV exposure and decreased infant growth from birth through infancy was mediated by prenatal maternal alcohol and tobacco use, while the relationship between increased IPV and decreased growth was mediated by postnatal maternal tobacco use.[19] Furthermore, prenatal substance exposure was associated with increased externalising behaviour, which was partially mediated by infant temperament (negative emotionality).[84] Maternal substance use is an important target for public health interventions in the DCHS context.

Mother–infant interactions and parenting may also be important mediators of infant growth and childhood behaviour. Notably, in DCHS, higher gestational age at delivery and higher infant birth weight were associated with increased frequency of shared pleasure moments in mother–infant interactions.[85] However, DCHS mothers with a history of childhood trauma had lower-rated (poorer quality) interactions with their infants, which, in turn, were associated with reduced child growth at 1 year of age.[86] Furthermore, the combination of lower socioeconomic status and maternal prenatal depression was associated with increased child externalising behaviour, and this association was mediated by coercive parenting.[84] Parenting interventions may have promise in the DCHS context.

In DCHS, a range of biopsychosocial factors are associated with mother–infant interactions[92,93] and breastfeeding.[89] Thus, for example, maternal childhood trauma and smoking were associated with poorer mother–infant interactions, while initiated breastfeeding was associated with better mother–infant interactions.[93] Employment and HIV diagnosis during pregnancy predicted a lower likelihood of breastfeeding initiation among HIV-infected mothers,[89] while in HIV-uninfected mothers, employment was associated with earlier breastfeeding discontinuation.[89] Additional support is clearly required for women who may feel they must discontinue breastfeeding because of work obligations.

As one example of the complex relationships between mental and physical health, DCHS findings have highlighted associations between maternal mental health and childhood respiratory function. Maternal smoking is associated with altered lung development.[116,117,118,119] Both maternal smoking and IPV were associated with recurrent wheeze.[120] Furthermore, prenatal IPV exposure was associated with a reduced respiratory resistance at 6–10 weeks, postnatal IPV was associated with a reduced ratio of time to peak tidal expiratory flow over total expiratory time (tPTEF/tE) at 12 months, and prenatal depression was associated with a lower respiratory rate at 6–10 weeks and at 12 months. Longitudinal analysis found an association of prenatal IPV with reduced tPTEF/tE; postnatal IPV with decreased functional residual capacity; prenatal PTSD with increased FRC; prenatal depression with increased FRC; and postnatal depression with increased FRC.[24]

Emerging evidence from the DCHS indicates the importance of socioeconomic status in influencing child neurodevelopment.[40,88,90] In the DCHS, mothers with a history of childhood trauma had lower-rated (poorer quality) interactions with their infants, which were in turn associated with reduced child growth at 1 year of age.[86] However, when this model was adjusted for socioeconomic status and maternal age, there was no longer a significant association between maternal childhood trauma and mother–infant interactions, and socioeconomic status predicted reduced child growth.[86] There is growing interest in combining psychosocial interventions with interventions that address the social determinants of mental health in LMICs.[121]

In HIC birth cohorts, multiple interacting biopsychosocial determinants are also apparent. A systematic review of IPV risk factors found, for example, that perinatal IPV exposure was associated with unemployment, drug use, being part of a minority group, exposure to early childhood abuse, and stress.[122] Intimate partner violence was in turn influenced by childhood maltreatment and the family environment (such as low socioeconomic status) and by child externalising behaviour.[14] The bidirectionality of various risk factors further complexifies models. For example, increased stress levels may lead to IPV, while IPV may lead to increased stress levels.[123] This bidirectionality of risk factors must be considered when developing interventions and targeted strategies.

Similarly, in DCHS, the complexity of the mechanisms underlying neurodevelopment has implications for interventions. Thus, for example, a book-sharing randomised controlled trial intervention was implemented within the DCHS to determine the efficacy of book-sharing on neurocognitive and socio-emotional development of 3.5-year-old children.[83] This intervention reported no differences between intervention and control groups on neurocognitive and socio-emotional measures from pre- to post-intervention.[83] A range of other factors impacting participants, including community violence and food security, may have influenced outcomes.[83]

A biopsychosocial approach has informed ongoing community engagement and stakeholder involvement in the DCHS. The study has built partnerships with several key stakeholders that facilitate community engagement, social responsiveness and research sustainability. Firstly, accessible communication has been key to disseminating information to participants. The DCHS has provided infographics and posters placed at study sites, and participants have been directed to a range of social and health resources. Secondly, the DCHS has helped address several community needs; these include establishing a library at a local school, a beautification project in which murals were painted on a local school, and the building of a multipurpose outdoor sports field. During the (coronavirus disease 2019) COVID-19 pandemic, a food insecurity project was launched in collaboration with local non-profit organisations to distribute food parcels. Public engagement workshops with DCHS participants have resulted in the designing and distribution of health awareness posters. Finally, DCHS participants and fieldworkers originate from a variety of backgrounds; participants' and fieldworkers' knowledge and lived experiences were continually incorporated into the design of the study.[124]

Several limitations of both the DCHS publications and this review warrant acknowledgment. Firstly, although the DCHS is set in a peri-urban low socio-economic status (SES) in South Africa with the intention of allowing generalisability to other regions, such generalisation has constraints. For example, other LMIC or high-income settings may be characterised by different exposures. Secondly, this review did not include grey literature, dissertations, or conference articles – we wished to emphasise peer-reviewed research. Thirdly, we did not conduct a journal database search, as the DCHS maintains an up-to-date database of all publications. A wider search would be unlikely to yield additional publications. Finally, the DCHS psychosocial publications are rich, and other authors may have found different key themes to emerge from them.

In conclusion, while birth cohorts in HICs have provided key lessons about factors influencing neurodevelopment, this review shows that LMIC birth cohorts may also yield valuable insights into child neurodevelopment. The Drakenstein Child Health Study has replicated some HIC cohort findings and has also contributed a number of novel findings. Firstly, regarding prenatal and postnatal maternal health risk factors, the DCHS highlights that psychological distress and traumatic stressors are negatively associated with physical and neurocognitive outcomes in offspring. Secondly, regarding prenatal and postnatal maternal physical health risk factors, maternal HIV and alcohol use, as well as air pollution exposure, are associated with impaired child neurodevelopmental outcomes. Thirdly, regarding understanding the mechanisms underlying impaired neurodevelopment, the DCHS highlights that prenatal indoor air pollution exposure and cognitive neurodevelopment at 2 years of age are mediated by DNA methylation of specific genes. Finally, regarding an integrative biopsychosocial approach to understanding neurodevelopment, the DCHS highlights the importance of intersecting biological, psychological and societal determinants that influence neurodevelopment. Insights from both HIC and LMIC birth cohorts, including the DCHS, not only deepen the understanding of determinants of child neurodevelopment but also provide actionable lessons that could drive policy changes and interventions.

Acknowledgements

The authors would like to thank the mothers and their children for participating in the study and the study staff, the clinical and administrative staff of the Western Cape Government Health Department at Paarl Hospital and at the clinics for support of the study.

This article is forms part of a larger study. A number of related articles are available with the latest correlating articles focusing on the joint effects of prenatal indoor air pollution and maternal psychosocial factors on child behavior trajectories and factors associated with mother-infant interaction has been published in https://doi.org/10.1093/aje/kwae046 and https://doi.org/10.1007/s10826-024-02997-7, respectively The present article undertakes a narrative review of psychosocial publications from the Drakenstein Child Health Study (DCHS) and to reflect on overarching themes.

Competing interests

The author, D.J.S, serves as an editorial board member of this journal. The peer review process for this submission was handled independently, and the author had no involvement in the editorial decision-making process for this manuscript. D.J.S. has received consultancy honoraria from Discovery Vitality, Kanna, L'Oreal, Lundbeck, Orion, Servier, Seaport Therapeutics, Takeda and Wellcome. The authors have no additional competing interests to declare.

Authors' contributions

S-.M.K. synthesised the data and took the lead on writing the manuscript with input from all authors; H.J.Z. and N.H. supervised the project; D.J.S. conceived the original idea, developed the theoretical framework and supervised the project. All authors contributed to the final manuscript.

Funding information

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Data availability

The data that support the findings of this study are available from the corresponding author, S-.M.K., upon reasonable request. The Drakenstein Child Health Study is committed to the principle of data sharing; thus, de-identified data will be made available to requesting researchers as appropriate. Requests for collaborations to undertake data analysis are welcome. More information can be found on our website [http://www.paediatrics.uct.ac.za/scah/dclhs].

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