Lead Article

Burden of anemia among indigenous populations

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An international perspective of the magnitude of anemia in indigenous peoples is currently lacking. The present systematic review was performed to characterize the global prevalence, severity, and etiology of anemia in indigenous peoples by conducting a systematic search of original research published in English from 1996 to February 2010 using PubMed, Medline, and Embase. A total of 50 studies, representing the following 13 countries, met the inclusion criteria: Australia, Brazil, Canada, Guatemala, India, Kenya, Malaysia, Mexico, New Zealand, Sri Lanka, Tanzania, the United States, and Venezuela. Results indicate major deficiencies in the coverage and quality of anemia monitoring data for indigenous populations worldwide. The burden of anemia is overwhelmingly higher among indigenous groups compared to the general population and represents a moderate (20–39.9%) to severe (≥40%) public health problem. For the most part, the etiology of anemia is preventable and includes inadequate diet, poor living conditions, and high infection rates (i.e., malaria and intestinal parasites). A concerted global effort is needed to reduce the worldwide burden of anemia in these marginalized populations.

INTRODUCTION

Recent estimates report that one-quarter of the world's population is suffering from anemia (95% CI: 22.9–26.7%).[1] Anemia is a condition characterized by decreased oxygen-carrying capacity caused by below-normal levels of hemoglobin (Hb) in the body.[2] This often unrecognized and consequently undertreated condition impairs the delivery of oxygen to tissues and can manifest clinically as fatigue or lethargy, depression, and impaired cognitive function.[3] Maternal anemia during pregnancy is an independent risk factor for low-birth-weight infants and preterm delivery.[4]

Anemia has multiple precipitating factors.[5] From a clinical perspective, anemia commonly occurs in patients with chronic conditions of inflammation, infection, or malignancy, such as cancer, rheumatoid arthritis, renal disease, and heart failure.[6] In populations not suffering from chronic disease, anemia is frequently caused by micronutrient deficiencies, infectious diseases, and genetic predispositions.[5] Iron-deficiency anemia (IDA) is the most common nutritional cause of anemia worldwide.[5] Folate, vitamin B_{12}, and vitamin A deficiencies also cause anemia.[7] Folate deficiency slows down DNA synthesis and impairs cell proliferation, which, in turn, leads to the intramedullary death of many of these abnormal cells and shortened lifespan of circulating red blood cells.[7] Vitamin B_{12} is necessary for the synthesis of red blood cells and is, therefore, another etiological factor in anemia development.[7] Vitamin A is thought to positively influence Hb levels by stimulating human erythroid precursors.[7] Vitamin A enhances iron availability to the bone marrow by mobilizing it from storage forms such as ferritin and enhancing absorption of iron from the gut.[7]

Anemia can occur when excessive amounts of blood are lost due to gastrointestinal infections associated with

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diarrhea.[8] Thus, infections related to hygiene, sanitation, safe water, and water management are significant contributors to anemia.[8] Infections with heavy loads of the helminth *Trichuris trichiura* and/or *Trichuris* dysentery syndrome (TDS) are characterized by anemia and growth stunting.[2] Hookworm infections produce a high degree of long-term morbidity by causing IDA. The primary form of morbidity caused by human hookworm infection is chronic intestinal blood loss resulting from adult parasites.[2] Malaria-related anemia is one of the leading causes of death, with pregnant women and children being the most frequently affected groups.[2] Other causes of anemia are inherited disorders, such as sickle cell disease and thalassemia (defects in genes producing Hb).[2] Elevated rates of anemia are observed more frequently in developing countries, due to increased risk of infectious diseases, poverty, and malnutrition, and in pregnant women and young children because of higher iron requirements during periods of accelerated growth.[9] In addition, infant and toddler diets are often poor in bioavailable iron, particularly in the post-weaning period.[7]

An important and often unreported group at risk for anemia is indigenous peoples. Indigenous peoples represent a heterogeneous group within and across countries; however, these populations experience striking and persistent disparities in social determinants of health, including access to healthcare services, employment, housing, and food security.[10] There is an acknowledged lack of information on many indigenous groups; however, in those populations for whom data do exist, indigenous peoples have worse health and social indicators than non-indigenous groups in the same society.[11]

One of the challenges in understanding anemia among indigenous peoples is the current ambiguity in determining how indigenous is defined. Instead of adopting a single definition, the World Health Organization (WHO) has developed a list of items that describe the world's indigenous peoples, such as those recognized and accepted by their community as indigenous, or those that demonstrate historical continuity with pre-colonial societies; other definitions include peoples that have distinct social, economic, or political systems and groups that maintain distinct languages, cultures, and beliefs.[10] According to the WHO, there are an estimated 370 million indigenous peoples living in 70 countries worldwide.[10]

While certain countries, such as Canada and Australia, have established national bodies to represent their indigenous populations and report regularly on their aboriginal populations,[12–14] there is no consolidated source to date that systematically reports on the prevalence of anemia in indigenous peoples nationally or internationally. Such a compilation of data on the prevalence of anemia among indigenous peoples would be useful for informing national and international prevention strategies and could be used by several already existing national and international agencies dedicated to improving morbidity and mortality among indigenous peoples. As a starting point for understanding anemia among indigenous populations, the present systematic review was undertaken to synthesize and critically appraise what is currently known within the peer-reviewed scientific literature about the prevalence of anemia in indigenous populations worldwide.

METHODS

Literature search

Studies reporting on the prevalence of anemia in free-living healthy populations and cohorts were identified using structured searches in PubMed, Medline, and Embase from January 1996 through February 2010 and supplemented by cross-checking the reference lists of relevant publications. Since there appear to be no previously published summaries of evidence on anemia prevalence among indigenous populations across various countries, as a first step to understanding the issue, searches for this review were limited to the peer-reviewed literature in the English language. The World Health Organization's online database, which compiles studies on the prevalence of anemia by country (for the general population only, not specifically on indigenous groups) was consulted to ensure the search findings were complete.[9]

To ensure greater completeness, the search strategy was completed in two stages (Figure 1). Stage one of the search process involved searches in Medline, Embase, and PubMed using the search terms: exp Anemia OR exp Anaemia AND indigenous.mp. or exp. Indians, North American/or exp Oceanic Ancestry Group/or aboriginal.mp or exp Health Services, Indigenous/ or tribe.mp. or tribal.mp. for Medline, exp. exp Anemia OR exp Anaemia AND exp. Indigenous people AND exp. Aborigine. or tribe.mp. or tribal.mp. for Embase and anemia (Title/Abstract) OR anaemia (Title/Abstract) AND indigenous (Title/Abstract) OR aboriginal (Title/Abstract) OR "tribe" OR "tribes" OR "tribal" for PubMed. After the selection procedure was fully completed for records identified in stage one of the search process, the studies selected for inclusion were categorized by country.

Finding a reliable and comprehensive list of names for indigenous populations worldwide proved unsuccessful. Therefore, stage two of the search process involved PubMed searches for countries that were identified from stage one of the search process. These additional PubMed

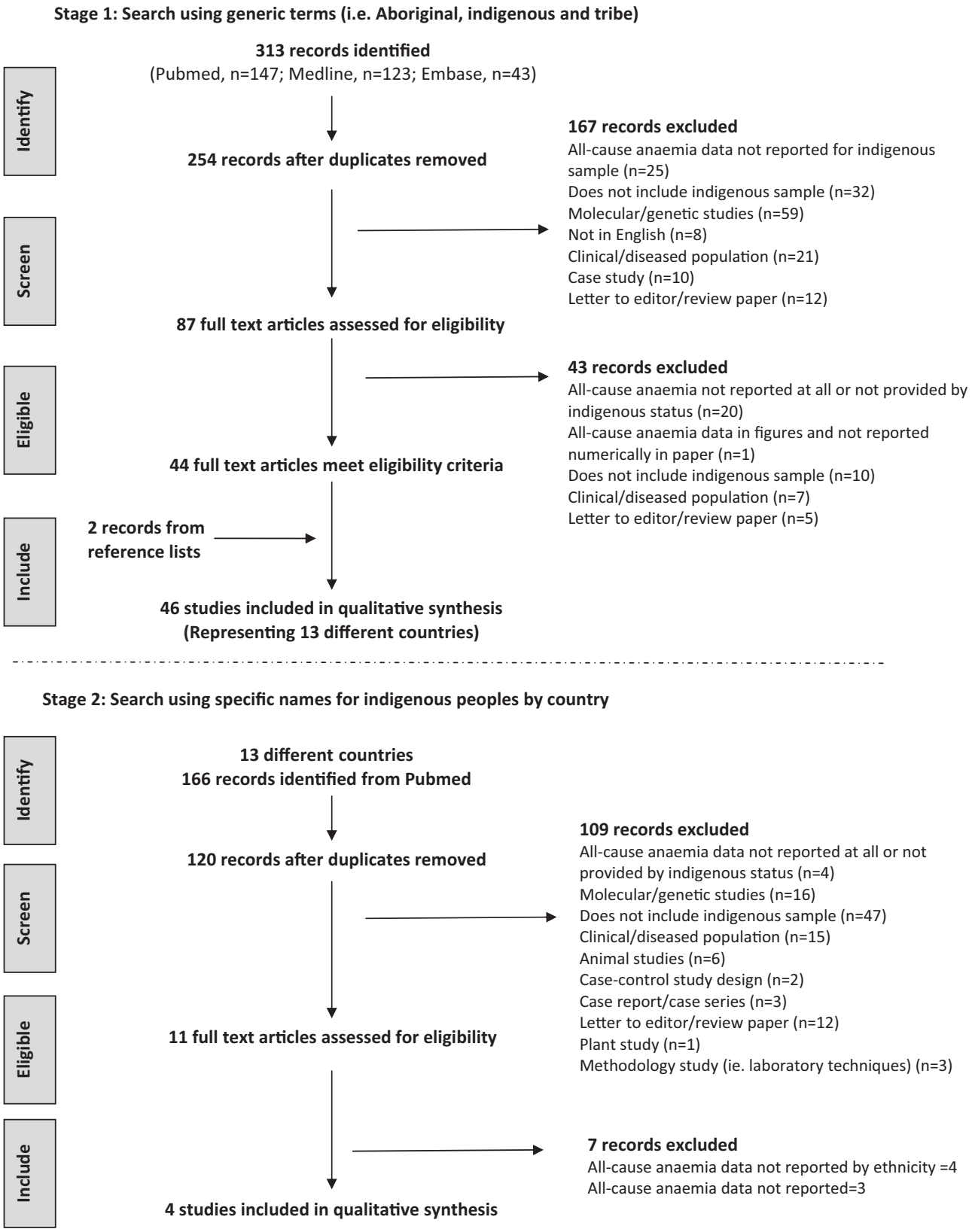

Figure 1 **Flowchart of literature selection procedure.**

searches included the country name or the name(s) of indigenous people within countries (based on web searches) (Table 1).

Inclusion and exclusion criteria

Studies were included in the current systematic review if the following criteria were met: 1) the study population included aboriginal or indigenous people, broadly defined as people of the same race or nationality that existed from the earliest known period and who share a culture that is distinct from colonizers or new settlers; 2) the study reported a prevalence estimate of anemia in a free-living indigenous population as a primary or secondary outcome; 3) the study was conducted on human beings (studies that examined indigenous plants, animals, genetic disorders, or specific genes were excluded). Studies examining individuals according to specific disease groups (e.g., diabetes, rheumatoid arthritis, renal disease) were excluded. There can be many causes of anemia, such as vitamin B_{12} deficiency, folic acid deficiency, anemia of unknown cause, and iron-deficiency anemia. The prevalence of anemia of all causes in a population is virtually always greater than the prevalence of anemia due to one cause (e.g., iron-deficiency anemia). Thus, so as not to underestimate the prevalence of anemia, all of the articles reviewed for inclusion had to separately present data for all-cause anemia.

Selection strategy and data extraction

Screening of articles was conducted in two stages. The first stage involved screening the titles and abstracts of all the articles identified during the electronic searches. The second stage involved screening the full text of articles that were included after the first stage of screening. Two investigators (AK, AA) independently screened abstracts for eligible studies and assessed the full text of potentially eligible studies using a standardized inclusion form. Discrepancies from stage one of screening were examined again in stage two of screening. Any discrepancies at stage two of screening were resolved through discussion. Data were extracted directly into tables by both reviewers or by one reviewer and checked for completeness and accuracy by a second reviewer.

For each study, the following descriptive information was extracted: country, study design, study period, sampling frame, sampling/selection method (e.g., random sampling, inclusive cohort), response rate, age of participants and description of the indigenous population studied (Table 2). The following data on study findings were included: sample size, criteria used to define anemia, prevalence of anemia, comparisons of anemia prevalence between indigenous and non-indigenous populations (where available), and causes of anemia (Table 3).

Quality assessment and data synthesis

A quality assessment of the studies to examine the validity of the sampling approach and how well the population of interest was represented was conducted based on the following four criteria: study design, sampling frame, sampling method, and response rate reporting (Table 2). Inspection of the included studies revealed that a meta-analysis was not possible given the heterogeneity of age groups, the diversity of indigenous peoples within countries, and variability in the definitions of anemia used. To perform the data synthesis, studies were grouped by country and the study population's life stage (i.e., children, pregnant women, adults) to report on the prevalence of anemia, the severity of anemia, and the etiology of anemia, where data was available. In order to interpret findings and classify the prevalence of anemia in specific indigenous populations according to public health significance, the following classifications from the WHO were used: no public health significance (anemia prevalence $\leq$4.9%), mild public health problem (anemia prevalence 5.0–19.9%), moderate public health problem (anemia prevalence 20.0–39.9%), and severe public health problem (anemia prevalence $\geq$40%).[15]

RESULTS

General description of findings

A total of 50 studies met the inclusion criteria (Figure 1). Data on the prevalence of anemia among indigenous and non-indigenous populations was available from 13 different countries: Australia, Brazil, Canada, Guatemala, India, Kenya, Malaysia, Mexico, New Zealand, Sri Lanka, Tanzania, the United States, and Venezuela. Overall, there was only one nationally representative study, which was conducted in Mexico and received the highest quality rating. Four studies examined populations that were representative of an entire state within a country, such as parts of Australia. Otherwise, the studies sampled from particular areas or communities known to be densely inhabited by or including people from an indigenous population. General limitations included the lack of reporting on anemia definitions in retrospective studies, the absence of confidence intervals around prevalence estimates of anemia, and the lack of statistical significance testing between anemia rates in indigenous and non-indigenous samples.

As might be expected, Hb, the most cost-efficient and commonly used measure to screen for anemia,[7] was used

Table 1 **Search terms and selection procedure for search strategy using country-specific terms for indigenous people.**

Country	Terms for indigenous groups	Search terms in PubMed	Search findings	Reasons excluded based on titles and abstracts	Screening of full-text articles
Australia	Indigenous; Aboriginal; Torres Strait Islander.[78]	(anaemia (Title/Abstract) OR anaemia (Title/Abstract) AND (Aboriginal(Title/Abstract) OR Indigenous(Title/Abstract) OR Aborigine(Title/Abstract)OR Torres Strait Islander(Title/Abstract)) AND Australia(Title/Abstract)	Records: $n = 12$ Duplicates: $n = 10$ Screened: $n = 2$	No anemia data: $n = 2$	Screened: $n = 0$
Brazil	Indigenous; historically called "Indians"[79]	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND (Indian*(Title/Abstract)OR Indigenous(Title/Abstract)OR Aborigine(Title/Abstract)) AND Brazil(Title/Abstract)	Records: $n = 2$ Duplicates: $n = 1$ Screened: $n = 1$	Genetic: $n = 1$	Screened: $n = 0$
Canada	Aboriginal; Native American; American Indian[80]	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND (Aboriginal(Title/Abstract)OR Indigenous(Title/Abstract)OR Aborigine(Title/Abstract)or Native(Title/Abstract)OR Indian*(Title/Abstract) AND Canada(Title/Abstract)	Records: $n = 9$ Duplicates: $n = 4$ Screened: $n = 5$	Non-indigenous sample: $n = 1$ Clinical sample: $n = 2$ Animal study (fish): $n = 1$	Screened: $n = 1$ Excluded: $n = 1$ (data not presented by ethnicity)
Guatemala	Indigenous; Indians; Maya, Xinca and Garifuna[81]	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND (Indigenous(Text Word)OR Maya(Text Word)OR Xinca(Text Word)OR Garifuna(Text Word)OR Indian*(Text Word)) AND Guatemala(Text Word)	Records: $n = 5$ Duplicates: $n = 2$ Screened: $n = 3$	Non- indigenous sample: $n = 1$ Case-control study: $n = 1$ Discussion paper: $n = 1$	Screened: $n = 0$
India	Government uses "Scheduled Tribes". Representatives of indigenous peoples in northeastern region use "indigenous peoples"[82]	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND (tribe*(Title/Abstract)OR tribal(Title/Abstract))OR Indigenous(Title/Abstract)) AND India(Title/Abstract)	Records: $n = 29$ Duplicates: $n = 13$ Screened: $n = 16$	Genetic: $n = 6$ Discussion paper: $n = 5$ Clinical population: $n = 1$ Animal study (rat): $n = 1$	Screened: $n = 3$ Included: $n = 2$ Excluded: $n = 1$ (data not presented by ethnicity)
Kenya	Indigenous people comprise 42 ethnic groups or tribes[83]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND (tribe*(Title/Abstract)OR tribal(Title/Abstract)OR Indigenous(Title/Abstract)) AND Kenya(Text Word)	Records: $n = 1$ Duplicates: $n = 1$ Screened: $n = 0$	Not applicable: $n = 0$	Screened: $n = 0$
Malaysia	Indigenous; Bumis; Negrito; Senoi; Orang Asli; Kadazan; Dusun; Rungus; Murut; Sungai; Lundayeh; Iban; Penan; Kenyah; Kayan; Kelabit; Ukit; Sekapan; Lahanan; Punan Bah[82]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND (Negrito(Title/Abstract)OR Senoi (Title/Abstract)OR Aboriginal(Title/Abstract)OR Orang Asli(Title/Abstract)OR Indigenous (Title/Abstract)OR Kadazan(Title/Abstract)OR Dusun(Title/Abstract)OR Rungus(Title/Abstract)OR Murut(Title/Abstract)OR Sungai(Title/Abstract)OR Lundayeh(Title/Abstract)OR Iban(Title/Abstract)OR Penan(Title/Abstract)OR Kenyah(Title/Abstract)OR Kayan(Title/Abstract)OR Kelabit(Title/Abstract)OR Ukit(Title/Abstract)OR Sekapan(Title/Abstract)OR Lahanan(Title/Abstract)OR Punan Bah(Title/Abstract)OR Bumis(Title/Abstract)) AND Malaysia (Title/Abstract)	Records: $n = 7$ Duplicates: $n = 1$ Screened: $n = 6$	Genetic: $n = 2$ Discussion paper: $n = 1$	Screened: $n = 3$ Included: $n = 1$ Excluded: $n = 2$ (no anemia data)

Table 1 **Continued**

Country	Terms for indigenous groups	Search terms in PubMed	Search findings	Reasons excluded based on titles and abstracts	Screening of full-text articles
Mexico	Indigenous peoples is the preferred term[84]	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND (Indigenous(Title/Abstract)) AND Mexico(Title/Abstrat)	Records: $n = 7$ Duplicates: $n = 7$ Screened: $n = 0$	Not applicable: $n = 0$	Screened: $n = 0$
New Zealand	Māori[85]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND Maori (Title/Abstract)AND New Zealand(Text Word)	Records: $n = 9$ Duplicates: $n = 2$ Screened: $n = 7$	Genetic: $n = 1$ Clinical sample: $n = 3$ Letter to editor: $n = 1$	Screened: $n = 2$ Excluded: $n = 2$ (data not presented by ethnicity: $n = 1$; no anemia data: $n = 1$)
Sri Lanka	Veddas[86]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND (Vedda*(Title/Abstract)OR Wanniya-laeto (Title/Abstract)) AND Sri Lanka(Text Word)	Records: $n = 1$ Duplicates: $n = 1$ Screened: $n = 0$	Not applicable: $n = 0$	Screened: $n = 0$
Tanzania	There are more than 120 ethnic groups/tribes.[87] After a broad search, abstracts were searched for any mention of ethnicity	(anaemia (Title/Abstract)OR anaemia (Title/Abstract)) AND prevalence (Title/Abstract)AND Tanzania(Title/Abstract)	Records: $n = 49$ Duplicates: $n = 1$ Screened: $n = 48$	Discussion paper: $n = 1$ Clinical sample: $n = 5$ Animal study: $n = 2$ No anaemia data: $n = 1$ Case study: $n = 1$ Laboratory methods/tools: $n = 1$ Not indigenous sample: $n = 37$	Screened: $n = 0$
United States	Native American; American Indian[88]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND (native American (Title/Abstract)OR American Indian [Title/Abstract))	Records: $n = 10$ Duplicates: $n = 0$ Screened: $n = 10$	Genetic: $n = 3$ Clinical sample: $n = 3$ Plant study: $n = 1$ Case study: $n = 1$ Case-control study: $n = 1$	Screened: $n = 1$ Data not presented by ethnicity: $n = 1$
Venezuela	Indigenous[89]	(anaemia (Title/Abstract)OR anaemia [Title/Abstract)) AND Venezuela [Title/Abstract))	Records: $n = 25$ Duplicates: $n = 2$ Screened: $n = 23$	Clinical sample: $n = 1$ Lab methods: $n = 2$ No anaemia data: $n = 1$ Discussion paper: $n = 3$ Not indigenous: $n = 8$ Genetic: $n = 3$ Animal study: $n = 2$ In vivo study: $n = 1$ Case series: $n = 1$	Screened: $n = 1$ Included: $n = 1$

Table 2 **General description of studies reporting on the prevalence of anemia among indigenous populations by country.**

Country	Sample	Study	Design	Study period	Sampling frame	Sampling method	Response rate	Age	Indigenous population	Quality score
Australia ($n = 7$)	Children	Heath and Panaretto (2005)[21]	CS	NR	3 schools with high indigenous enrollment in state of Queensland	Inclusive	NR	4–12 y	Aboriginal and Torres Strait Islander	Poor
	Children	Mackerras et al. (2003)[23] and Mackerras and Singh (2007)[24]	CS nested in PC	1987–90 1998–01	Single obstetric hospital in Northern Territory to select all singleton infants born to Aboriginal mothers born 1987–90. Recruited 686 of 1238 births and followed 572 infants living in study area	Inclusive	83.4%	8–14 y	Aboriginal	Good
	Children	Paterson et al. (1998)[25]	CS	1993	All schools in 11 of 18 communities in Northern Territory. Screened 774 of 858 (90%) children	Inclusive	58%	3–18 y	Aboriginal	Moderate
	Children and adults	Hopkins et al. (1997)[22]	CS	1992	Single community in Western Australia	NR	NR	≥5 y	Aboriginal	Poor
Australia	Pregnant women	Lewis et al. (2009)[27]	R-CS	2004–6	Selected nulliparous mothers with singleton and full-term births from perinatal database in single obstetric hospital in Western Australia	Inclusive	NA	12–18 y	Indigenous	Poor
	Pregnant women	Westenberg et al. (2002)[28]	R-CS	1995–9	Perinatal database on all births in state of South Australia	Inclusive	NA	15–19 y	Aboriginal	Moderate
	Pregnant women	Wills and Coory (2008)[29]	R-CS	2005–6	Perinatal database of all singleton births in state of Queensland	Inclusive	NA	All ages	Indigenous	Moderate
Brazil ($n = 2$)	Infants	Morais et al. (2005)[31]	CS	2002	Two villages in district of Acquidauana in Mato Grosso do Sul state	Inclusive	>90%	6–120 mo	Terena Indians	Moderate
	Infants	Orellana et al. (2006)[32]	CS	2005	All children in 9 of 11 villages in Sete de Setembro Indian reservation	Inclusive	100%	6–119 mo	Surui Indians	Good
Canada ($n = 4$)	Infants	Christofides et al. (2005)[34,35]	CS	2001–3	Two Aboriginal communities in James Bay area in Ontario, and one Inuit community in Nunavut. List of all infants from health records	Inclusive	77.7%	4–18 mo	Inuits and Cree First Nations	Moderate
	Infants	Willows et al. (2000)[39]	CS	1995–98	6 of 9 Cree villages in James Bay area, northern Quebec. Cree infants screened at 9-month well-baby clinic visit	Inclusive	83.6%	9 mo	First Nations	Good
	Infants	Willows and Gray-Donald (2002; 2003; 2004)[36–38]	PC	1998–2000	9 Cree communities in James Bay area, northern Quebec. Cree infants screened at 9-month well-baby clinic visit.	Inclusive	87.3%	9 mo	First Nations	Good
	Infants and pregnant women	Hodgins et al. (1998)[33]	R-CS	1989–92	Multiple sources of surveillance data. Prenatal records (pregnant women), study on environmental contaminants (infants), and health survey in Nunavik, Northern Quebec	Unclear	NA	Infants: 9–14 mo Women: all ages	Inuits	Poor
Guatemala ($n = 1$)	Women	Neufeld et al. (2004)[18]	CS	1994	House visits in 17 villages in rural highland region	Inclusive	NR	15–45 y	Mayans	Moderate

Table 2 **Continued**

Country	Sample	Study	Design	Study period	Sampling frame	Sampling method	Response rate	Age	Indigenous population	Quality score
India ($n = 12$)	Children	Arlappa et al. (2010)[42]	CS	2002–3	State of West Bengal	Random sampling	NR	1–5 y	Schedule Tribe	Moderate
	Children	Chakma et al. (2000)[46]	CS	NR	3 tribal areas of Madhya Pradesh, Jalbalpur	Random sampling	52.7%	6–14 y	Baiga, Abujhmaria and Bha"d tribes	Moderate
	Children	Rao et al. (2005)[52]	CS	2000–1	House-to-house survey in 27 of 197 villages in Jabalpur district in Madhya Pradesh	Unclear	NR	NR	Gond tribe	Poor
	Children and adults	De et al. (2006)[47]	CS	NR	3 states Assam, Arunachal Pradesh, and Tripura in Northeastern India	Random sampling	NR	All ages	Unspecified tribes	Moderate
	Children and adults	Rao et al. (1998)[51]	CS	NR	House-to-house survey of all remaining 36 members of the Great Andamanese tribe	Inclusive	100%	All ages	Andamanese tribe	Very good
	Adolescents	Ghosh et al. (2002)[49]	CS	NR	Single boarding school in Maharashtra State	Unclear	NR	12–18 y	Bhil and Pawar tribes	Poor
	Adolescent girls	Deshmukh et al. (2008)[48]	CS – part of RCT	2000–1	Nashik district in Maharashtra state. Cluster sampling in tribal, rural, and urban-slum areas	Unclear	NR	14–18 y	Unspecified tribes	Poor
	Adolescent girls	Kotecha et al. (2009)[43]	CS	2000–1	All 426 schools in 3 areas in Vadodara district in Gujarat. Girls in grades 8–12	Non-random	NR	12–19 y	Tribes	Poor
	Non-pregnant women	Ghosh and Bharati (2003)[41]	CS	2000	Periurban area in Kolkata City	Unclear	NR	15–42 y	Munda tribe	Poor
	Non-pregnant women	Menon et al. (2010)[44]	CS	2007	Community in Nagpur district. Stratified cluster sampling of households	Random selection	92%	18–30 y	Unspecified tribes	Good
	Pregnant women	Singh et al. (1998)[45]	PC	1995–6	Pregnant women living in 25 tribal villages in area located in Madhya Pradesh	Random selection	NR	18–40 y	Gond tribe	Moderate
	Elderly adults	Kerketta et al. (2009)[50]	CS	NR	4 tribal dominated states in Orissa	Unclear	NR	≥60 y	Langia Saora, Paudi Bhuiyan, Kutia Kongh, and Dongria Kondh tribes	Poor
Kenya ($n = 1$)	Pregnant women	Van Eijk et al. (2001)[53]	CS	1996–8	Single prenatal clinic in Kisumu. Enrolled healthy pregnant women visiting prenatal clinic with uncomplicated singleton pregnancy at ≥32 weeks gestation	Inclusive	85%	All ages	Luo tribe	Moderate
Malaysia ($n = 3$)	Children	Aini et al. (2007)[55]	CS	2003–4	Eight villages in state of Selangor	Unclear	NR	2–15 y	Orang Asli	Poor
	Children	Al-Mekhlafi (2008)[54]	CS- part of RCT	2006–7	Single school in state of Pahang. Baseline data of randomized controlled trial for vitamin A supplementation trial	Inclusive	82.5%	7–12 y	Orang Asli	Moderate
	Children and adults	Sagin et al. (2002)[56]	CS	NR	7 villages in 5 remote communities in upper Rejang River basin, state of Sarawak	Unclear	NR	5–85 y	5 tribes	Poor

Mexico ($n = 5$)	Women	Monárrez-Espino et al. (2001)[64]	CS	1998	Guachochi municipality in Northern Mexico. Multistage proportional sampling of localities	Random sampling	NR	12–49 y	Tarahumara	Moderate
	Women and children	Shamah-Levy et al. (2003); Villalpando et al. (2003)[57–61,90]	CS	1999	National probabilistic sample of women and children from 18,000 households	Random sampling	82.3%	12–49 y; 0–11 y	Multiple indigenous groups	Excellent
	Children	Monarrez-Espino et al. (2004)[62]	CS	2001	5 of 38 boarding schools in 3 different municipalities. Sample selected based on expert panel	Non-random sampling	NR	6–14 y	Tarahumara	Poor
Mexico	Children	Villalpando et al. (2009)[61]	CS	1999–2006	Data from Mexican National Health Nutrition Survey, 2006 and Mexican National Nutrition Survey, 1999	Random sampling	82.3% for 1999, NR in English for 2006	0–11 y	Multiple indigenous groups	Excellent
	Children	Valencia et al. (1999)[63]	CS	NR	26 rural communities in Northwest. Children with reliable birth dates	Unclear	NR	6–10 y	Yaqui	Poor
New Zealand ($n = 1$)	Adolescent girls	Schaaf et al. (2000)[65]	CS	1997–8	High schools in Auckland city. Randomly selected 10/32 schools with high proportion of Pacific Island students. 8 high schools participated	Inclusive	61%	NR Grades 5–7	Maori and Pacific Islanders	Moderate
Sri Lanka ($n = 1$)	Children	Chandrasena et al. (2004)[67]	CS	1999	Two primary schools in Kelaniya. Included children in y 1–3 at Dambana (Veddha) and Wewatta (Sinhalese) primary schools	Inclusive	NR	6–15 y	Veddha	Poor
Tanzania ($n = 1$)	Children and Adults	Hinderaker et al. (2001)[17]	CS	1995–6	Two rural divisions of Mbulu and Hanang districts. 12 antenatal clinics in a single Lutheran hospital	Inclusive	57%	14–49 y	Mostly, Datoga and Iraqw tribes	Moderate
USA ($n = 1$)	Children and pregnant/ postpartum women	Gessner (2009)[68,69]	R-C	1999–2006	Birth certificate database linked to database with hemoglobin levels from US Supplemental Nutrition Program for Women, Infants and Children	Inclusive	NA	Infants: 10–59 mo Women: all ages	Alaskan Natives	Poor
Venezuela ($n = 2$)	Children and adults	García-Casal et al. (2008)[70]	CS	NR	Attendants of single medical facility in Amazon region. Sampling process executed over 3 consecutive days	Inclusive	NR	All ages	Piaroa	Poor
	Adults	Diez-Ewald et al. (1997)[71]	CS	NR	NR	NR	NR	9 mo–69 y	Bari Indians	Poor
Venezuela and Brazil ($n = 1$)	Children and adults	Grenfell et al. (2008)[30]	CS	2005	Ocamo and Alto Ocamo areas in Amazon region of south Venezuela. Data used from recent census of 11 communities	Random selection	NR	All ages	Yanomami	Moderate

Abbreviations: CS, cross-sectional; R-CS, retrospective cross-sectional; PC, prospective cohort; RCT, randomized controlled trial; NR, not reported.

Table 3 **Prevalence, severity, and etiology of anemia among indigenous populations by country.**

Country; age group	Study	Age	Sex	No. of anemic subjects in each group	Definition of anemia	Prevalence of anemia by group (%)	Etiology of anemia			
							Nutritional	Infectious	Genetic	Other
Australia; children	Heath and Panaretto (2005)[21]	4–12 y	Both	I: 55 NI: 71	Hb < 115 g/L	I: 16.4; NI: 4.2 ($P = 0.02$)*	ID (SF < 15 µg/L and MCV < 74 fL) was 3.6% among indigenous and 0% non-indigenous ($P = 0.11$). IDA (ID and Hb < 115 g/L) was 3.6% among indigenous children and 0% among non-indigenous children ($P = 0.11$)	Not reported	Not reported	Not reported
Australia; children	Mackerras (2003)[23]	8–14 y	Both	I: 442	Hb < 115 g/L (5–11 y); Hb < 120 g/L (12–13 y)	I: 14*	Not reported	Lack of association between eosinophilia and anemia. The authors state that hookworm in Northern Territory is now rare	Not reported	Not reported
Australia; children	Mackerras and Singh (2007)[24]	9–13 y	Both	I: 517	Hb < 115 g/L (5–11 y) Hb < 120 g/L (12–13 y)	I: 13.2 (95% CI: 10.4–16.4)*	Not reported	Not reported	Not reported	Not reported
Australia; children	Paterson et al. (1998)[25]	3–18 y	Both	I: 657	Hb < 105 g/L	I: 24*	Causes not examined. Authors state most likely cause would be dietary	Not reported	Not reported	Not reported
Australia; children and adults	Hopkins et al. (1997)[22]	All ages	Both	I: 188 NI: 19	Hb < 120 g/L (5–14 y) Hb < 130 g/L (men, ≥14 y); Hb < 120 g/L (women, ≥14 y)	(5–14 y) I: 75.0; NI: 0*	ID (SF ≤ 20µg/L or serum iron ≤ 8 µg or TfR ≥ 4.05 g/L) among non-Aboriginals was 10% for women and 0% for men. Among Aboriginals, IDA was 75% for children, 0% for men, and 31% for women. None of the non-Aboriginal adults had IDA. In hookworm-negative Aboriginals, ID was found in 100% of children, 10% of men, and 50% of women, suggesting inadequate dietary iron is a causal factor	None of the non-Aboriginals had hookworm infection. Among Aboriginals, 77% had hookworm infections. Hookworm infections were associated with anemia ($P < 0.01$) and ID ($P < 0.01$) in Aboriginals >14 y old. Other parasites included: *Hymenolepideana* (23%), *Giardia duodenalis* (21%). and *Entamoeba coli* (30%)	Not reported	Not reported
Australia; pregnant women	Lewis et al. (2009)[27]	12–18 y	Females	I: 449 NI: 4625	Hb < 100 g/L	I: 23.0; NI: 8.0 ($P < 0.001$)*	Not reported	Not reported	Not reported	Not reported
Australia; pregnant women	Westenberg (2002)[28]	5–19 y	Females	I: 449 NI: 4625	Not reported	I: 25.2; NI: 9.9 (RR = 2.54; 95% CI: 2.11–3.04)*	Not reported	Not reported	Not reported	Not reported
Australia; pregnant women	Wills and Coory (2008)[29]	All ages	Females	I: 4228 NI: 75575	Not reported	I: 3.3 NI: 1.1*	Not reported	Not reported	Not reported	Not reported
Brazil; infants	Morais et al. (2005)[31]	6–120 mo	Both	I: 167	Hb < 110 g/L (6–72 mo); Hb < 115 g/L (73–120 mo)	I: 62.3*	Not reported	Not reported	Not reported	Not reported
Brazil; infants	Orellana et al. (2006)[32]	6–119 mo	Both	I: 268	Hb < 110 g/L (6–59 mo) Hb < 115 g/L (60–119 mo)	I: 80.6*	Not reported	Not reported	Not reported	Not reported
Canada; infants	Christofides et al. (2005)[34,35]	4–18 mo	Both	I: 115	Hb < 110 g/L	I: 36.0*	Depleted iron stores (SF < 12 µg/L) was present in 53.3% of infants. ID (TfR > 8.5 mg/L) was present in 27.6%. Multivariate modelling found cow's/evaporated milk was only significant predictor of anemia	High (29%) infection rates were found. Prevalence *of H. pylori* infection was 39%	Not reported	Not reported

Canada; infants	Willows et al. (2000)[39]	9 mo	Both	I: 354	Hb < 110 g/L Hb < 105 g/L Hb < 100 g/L	Hb < 110 g/L : I: 7.9 (95% CI: 5.3–11.2) Hb < 105 g/L I: 31.1 (95% CI 26.2–36.1) Hb < 100 g/L I: 17.2 (95% CI 13.4–21.5)	Prevalence of ID (Hb <110 g/L and low MCV) was 5.6–10.8% among Cree infants	Not reported	Not reported	Adjusted analysis found OR for Hb < 105 g/L was 7.9 (95% CI: 3.4–18.2) for infants fed breast milk versus formula
Canada; infants	Willows and Gray-Donald (2002; 2003; 2004)[36–38]	9 mo	Both	I: 274	Hb < 110 g/L	I: 25.0*	ID (SF < 10 μg/L) was 22.7% and IDA (SF < 10 μg/L and anemia) was 7.9%	Not reported	Not reported	Not reported
Canada; infants	Hodgins et al. (1998)[33]	9–14 mo	Both	I: 172	Hb < 110 g/L Hb < 105 g/L	Hb < 110 g/L I: 58.0 Hb < 105 g/L I: 49.0 when infants were 4–9 mo	64% had ID (low ferritin or MCV values – specific cut-offs not reported)	Not reported	Not reported	Not reported
Canada; pregnant women	Hodgins et al. (1998)[33]	All ages	Pregnant women	I: 187	Hb < 120 g/L Hb < 115 g/L	Hb < 120 g/L I: 39.0 Hb < 115 g/L I: 22.0	Not reported	27% of the Nunavik sample had positive or equivocal *Helicobacter* serology results	Not reported	Not reported
Guatemala; women	Neufeld et al. (2004)[18]	15–45 y	Females	I: 253	Hb < 130 g/L (low altitude) Hb < 134 (high altitude)	I: 25.00*	78 (31%) of women had ID (SF ≤ 12 μg/L). There was no significant association between smoky fire versus smokeless stove and anemia ($P = 0.67$) or ID ($P = 0.83$)	Not reported	Not reported	Not reported
India; children	Arlappa et al. (2010)[42]	1–5 y	Both	I: 49 NI: 388	Definition: 90 > Hb < 110 g/L	I: 100.0; NI: 82.8 (Schedule Caste), 76.8 (Others defined as "Forward Caste"). ($P < 0.01$)*	Among all rural children of West Bengal, diets were found to be deficient in micronutrients, especially iron	Not reported	Not reported	Multivariate analyses found the OR for anemia for children ≥2 y old was 3.0 (95 CI: 1.5–6.0) versus 7.7 (95% CI: 2.6–22.4) among 1 y olds. Children in the Scheduled Caste and Schedule Tribe had higher risk of anemia compared to other children (OR: 2.3, 95% CI: 1.3–3.9)
India; children	Chakma et al. (2000)[46]	6–14 y	Both	I: 776	90 > Hb < 110 g/L 70 > Hb < 90 g/L Hb < 70 g/L	90 > Hb < 110 g/L I: 21.4 70 > Hb < 90 g/L I: 23.6 Hb < 70 g/L I: 30.3	Not reported	Prevalence of ascariasis was 18.5% and 16.3% for hookworm. Multiple infestations of intestinal parasites were detected in 23% of samples	Not reported	Not reported
India; children	Rao et al. (2005)[52]	Not reported	Both	I: 1022	Hb < 110 g/L 90 > Hb < 110 g/L 70 > Hb < 90 g/L Definition: Hb < 70 g/L	Hb < 110 g/L I: 86.7 90 > Hb < 110 g/L I: 15.6 70 > Hb < 90 g/L I: 63.8 Hb < 70 g/L I: 7.3	Not reported	Not reported	Not reported	More than 60% of children were underweight (≥2 standard deviations below reference median as recommended by WHO) and more than 25% had severe undernutrition (≥3 standard deviations below reference median as recommended by WHO)

Table 3 **Continued**

Country; age group	Study	Age	Sex	No. of anemic subjects in each group	Definition of anemia	Prevalence of anemia by group (%)	Etiology of anemia			
							Nutritional	Infectious	Genetic	Other
India; children and adults	De et al. (2006)[47]	All ages	Both	I: 1263	Hb < 110 g/L	Prevalence by state Arunachal Pradesh state: I: 53.8 Assam state: I: 59.8; Triprura state: I: 57.5*	Not reported	Not reported	Not reported	Not reported
India; children and adults	Rao et al. (1998)[51]	All ages	Both	I: 35	Hb < 110 g/L	I: 94.3*	Results from dietary survey showed diet was poor in iron	Of 30 people examined, 96.7% had one or more types of intestinal parasites. The Great Andamanese tribe has been declining since its first estimation in 1858. Decline is mostly attributable to infectious diseases (i.e., measles, malaria and syphilis)	Not reported	35.7% were deficient based on recommended dietary allowance) and, as per Gomez classification, 57.1% of children ≤6 y had moderate-to-severe malnutrition
India; adolescents	Ghosh et al. (2002)[49]	12–18 y	Both	I: 481	Hb < 110 g/L	I: 16.2 (boys); 38.3 (girls)*	Not reported	Not reported	Beta-thalassemia was uncommon (1.6% of males and 2.4% of females). Sickle cell trait and disease was present in 21.3% of males and 14.4% of females	Not reported
India; adolescent females	Deshmukh et al. (2008)[48]	14–18 y	Both	I: 248	100 > Hb < 119 g/L 70 > Hb < 99 g/L Hb < 70 g/L	100 > Hb < 119 g/L I: 40.8 70 > Hb < 99 g/L I: 24.4 Hb < 70 g/L I: 3.6	After weekly iron and folic acid supplementation program, anemia was significantly reduced from 69.9% to 48.6% ($P < 0.001$)	Not reported	Not reported	Not reported
India; adolescent females	Kotecha et al. (2009)[43]	12–19 y	Females	I: 895 NI: 1965	Hb < 120 g/L	I: 73.7; NI: 74.5 (rural); NI: 75.8 (urban)*	ID (SF < 12 ng/mL) was evident in 49.7% of females	Not reported	Not reported	Not reported
India; non-pregnant women	Ghosh and Bharati (2003)[41]	15–42 y	Females	I: 105 NI: 138 (NI were members of lowest caste)	100 > Hb < 119 g/L 70 > Hb < 99 g/L Hb < 70 g/L	100 > Hb < 119 g/L I: 37.1; NI: 48.6 ($P < 0.001$) 70 > Hb < 99 g/L I: 55.2; NI: 9.7 Hb < 70 g/L I: 7.6; NI: 2.9	100% of the Munda (tribal) women had low Hb (<120 g/L). Low Hb was associated with age (<30 y), low BMI, low socioeconomic status, poverty, low literacy rates, and high live births	Not reported	Not reported	Not reported
India; non-pregnant women	Menon et al. (2010)[44]	18–30 y	Females	I: 56 NI: 53	Hb < 120 g/L	I: 72; NI: 53 ($P = 0.07$)*	Prevalence rates of zinc (<10.7 μmol/L), vitamin B_{12} (<148 pmol/L), vitamin A (retinol <0.7 μmol/L), and folate (<6.8 nmol/L) deficiencies were 52%, 34%, 4%, and 2%, respectively	Not reported	3 women tested positive for sickle cell disease.	Not reported Other: low BMI, concurrent micronutrient deficiencies, and a low intake of animal products together may explain the high prevalence of anemia

India; pregnant women	Singh et al. (1998)[45]	18–40 y	Female	I: 456	70.1 > Hb < 100 g/L Hb < 70 g/L	70.1 > Hb < 100 g/L Prevalence with malaria I: 76 (parity = 0); 80 (parity ≥ 1) Prevalence without malaria I: 54 (parity = 0); 69.6 (parity ≥1) Hb < 70 g/L Prevalence with malaria I: 4 (parity = 0); 7.5 (parity ≥ 1) Prevalence without malaria I: 2.7 (parity = 0); 2.5 (parity ≥ 1)	More than 60% of pregnant women without malaria were anemic, suggesting there are other causes, such as dietary inadequacy	21.1% of women were infected with malaria, of which *Plasmodium falciparum* accounted for 64% and *P. vivax* for remaining 36%	Not reported	Not reported
India; elderly	Kerketta et al. (2009)[50]	≥60 y	Both	I: 312	Mild: Hb < 130 g/L (men); Hb < 120 g/L (women). Definition: not provided for moderate or severe anaemia	Mild anemia I: 24.4 (men); 24.3 (women) Moderate anaemia I: 43.0 (men); 41.8 (women) Severe anaemia I: 29.6 (men); 27.7 (women)	Not reported	Not reported	Not reported	Not reported
Kenya; pregnant women	Van Eijk et al. (2001)[53]	All ages	Females	I: 3645 NI: 963	Hb < 110 g/L Hb < 70 g/L	Hb < 110 g/L Gravidae 1–2 I: 81.6; NI: 70.6 (RR:1.16; 95% CI: 1.10–1.22) Gravidae ≥3 I: 76.5; NI: 69.5 (RR: 1.10; 95% CI: 1.02–1.19) Hb < 70 g/L Gravidae 1–2 I: 7.5; NI: 5.1 (RR:1.47; 95% CI: 1.02–2.12) Gravidae ≥3 I: 7.1; NI: 4.5 (RR:1.57; 95% CI: 0.92–2.68)	Not reported	Not reported	Not reported	Not reported
Malaysia; children	Aini et al. (2007)[55]	2–15 y	Both	I: 368	Hb < 120 g/L Hb < 80 g/L	Hb < 120 g/L I: 41.5 Hb < 80 g/L I: 11.3	ID (SF < 10 µg/L) accounted for 88% of anemia; 61% of children had ID (serum iron <10.6 µmol/L and high TIBC (>75 µmol/L) and/or SF < 10 µg/L) and 36.5% had IDA (ID and anemia)	Of the 281 children tested, all were infected with at least one soil-transmitted helminth, *A. lumbrocoides* (61.9%), *T. trichiurara* (98.2%), and hookworm (37%). Adjusted analyses found severe trichuriasis was the main predictor of IDA (OR: 3.1; 95% CI: 1.7–5.8)	Not reported	Adjusted analyses found underweight (z-score below 2 standard deviations of National Centre of Health Statistics reference value) as a significant predictor of IDA (OR: 2.2; 95% CI: 1.3–3.6)
Malaysia; children	Al-Mekhlafi (2008)[54]	7–12 y	Both	I: 241	Hb < 120 g/L	I: 48.5 (95% CI: 42.3, 54.8)*	Prevalence of ID (SF < 10 µg/L) was 16.8% for children ≤10 y and 23.0% for those >10 y. Prevalence of IDA (anemic and low SF and/or serum iron <10.6 µg/L, TS < 16% and high total iron-binding capacity >75 µmol/L) was 34% (95% CI: 28.3–40.2) and accounted for 70.1% of anemia cases	Prevalence of ascariasis was 22.3%, trichuriasis was 29.8%, hookworm infection was 13.4%, mixed soil-transmitted helminths was 62.7%, and giardiasis was 17.8%	Not reported	Not reported

Table 3 **Continued**

Country; age group	Study	Age	Sex	No. of anemic subjects in each group	Definition of anemia	Prevalence of anemia by group (%)	Etiology of anemia			
							Nutritional	Infectious	Genetic	Other
Malaysia; children and adults	Sagin et al. (2002)[56]	5–85 y	Both	I: 365	Hb < 110 g/L (children) Hb < 120 g/L (women) Hb < 130 g/L (men)	I: 24.4%*	Not reported	Of 83 anemic individuals, 75 (90.4%) had no intestinal parasitic infection. Of the 9.6% with intestinal parasitic infection, the most common was *Trichuris trichiura* (6%). Hookworm (2.4%) contributed to a small proportion of parasitic causes	Not reported	Not reported
Mexico; women	Monárrez-Espino et al. (2001)[64]	12–49 y	Female	I: 446 non-pregnant and 35 pregnant women	100 > Hb < 119 g/L 80 > Hb < 99 g/L Hb < 80 g/L	100 > Hb < 119 g/L I: 17.1 (pregnant women) and 12.1 (non-pregnant women) 80 > Hb < 99 g/L I: 8.6 (pregnant) and 2.9 (non-pregnant) Hb < 80 g/L I: 0 (pregnant) and 1.1 (non-pregnant)	In subsample, ID (SF < 12 ng/mL) was found in 59.4% of non-pregnant and 68.8% of pregnant women. IDA (ID and anemia) was present in 69.6% of non-pregnant and 100% of pregnant women. Of women with no temperature or reported history of infection in past 2 weeks (urinary, respiratory, gastrointestinal), ID was found in 61.2% of non-pregnant and 71.4% of pregnant women	25 women reported a history of infection in past 2 weeks (urinary, respiratory, and gastrointestinal)	Not reported	Not reported
Mexico; women	Shamah-Levy et al. (2003)[59]	12–49 y	Females	I: 1453 NI: 14, 686	Hb < 120 g/L	I: 24.8 (95% CI: 22.2, 27.4); NI: 20.4 (95% CI: 19.5, 21.4)*	Adjusted OR for anemia in I versus NI non-pregnant women was 1.29 (95% CI: 1.11–1.51)	Not reported	Not reported	Significant factors in adjusted analyses for anemia were: age ($P = 0.0001$), southern region ($P < 0.0001$), parity ($P < 0.0001$), no schooling ($P < 0.05$), maternal illiteracy ($P = 0.006$), low SES ($P < 0.0001$), and indigenous status ($P = 0.001$)
Mexico; children	Monarrez-Espino et al. (2004)[62]	6–14 y	Both	I: 331	115 < Hb < 130 g/L depending on age	I: 13.0*	In subsample, ID (SF < 12 ng/mL and TfS > 14%) was 11.1% and IDA (anemia and ID) was 10.1%. Proportion of anemia unrelated to ID was 4.4% in boys and 7.4% in girls	Not reported	Not reported	Not reported
Mexico; children	Valencia et al. (1999)[63]	6–10 y	Both	I: 296	Hb < 110 g/L	I: 0.7*	8.5% had ID (SF < 12 ng/mL and TfS > 16%); 0.0% had iron deficient erythropoiesis (SF < 12 ng/mL and TfS < 16%); 0.4% had IDA (SF < 12 ng/mL, TfS < 16, MCV < 80 fL, and Hb < 110 g/L)	Not reported	Not reported	Not reported

Mexico; children	Villalpando et al. (2003)[60]	0–11 y	Both	0–4 y I: 614; NI: 4,912 5–11 y I: 1,068; NI: 9,150	95 < Hb < 120 g/L (depending on age)	6–71 mo I: 35.8 (95% CI: 32.0–39.5); NI: 26.1 (95% CI: 24.9–27.4) 5–11 y I: 24.0 (95% CI: 21.3–26.7); NI: 19.0 (95% CI: 18.1–19.9)*	Association between Hb and beneficiary food assistance program was significant in children 2 y or older	Not reported	Not reported	No association was found between Hb concentration and indigenous status ($P = 0.81$), but height-for-age, SES, maternal education, and altitude above sea level were significant in children 2 y or older
Mexico; children	Villalpando et al. (2009)[61]	0–11 y	Both	<5 y 1999 data I: 587; NI: 4614 <5 y 2006 data I: 286; NI: 6329 5–11 y 1999 data I: 1,067; NI: 9151 5–11 y 2006 data I: 842; NI:13,821	Hb < 110 g/L (ages 12–71 mo); Hb < 120 g/L (ages 6–11 y).	<5 y 1999 data I: 39.6; NI: 30.1 <5 y 2006 data I: 26.7; NI: 25.8 5–11 y 1999 data I: 31.3; NI: 24.8 5–11 y 2006 data I: 20.6; NI: 17.8*	Among children 6–11 y, adjusted analyses for anemia found statistically significant benefits of national social program including nutrition interventions ($P = 0.004$)	Not reported	Not reported	Among children <2 y significant factors in adjusted analyses for anemia were: age ($P = 0.005$), low SES ($P = 0.002$), and living in northern region ($P = 0.01$). Among children 6–11 y, adjusted analyses for anemia were age ($P = 0.0001$), beneficiary of national social program including nutrition interventions ($P = 0.004$), and central region ($P = 0.03$)
New Zealand; adolescent females	Schaaf et al. (2000)[65]	Not reported	Females	I: 556 NI: 145	Hb < 120 g/L	I: 11 (Maori) NI: 4 (European) (Adjusted RR: 2.92, 95% CI: 1.15–7.43)*	Adjusted RRs for ID (2 or more of: SF < 12 µg/L, TfS < 14%, red blood cell distribution width >14.5%) were higher for Maori (OR: 3.12, 95% CI: 1.74–5.59) compared to European students	Not reported	Not reported	Not reported
Sri Lanka; children	Chandrasena et al. (2004)[67]	6–15 y	Both	I: 77 NI: 189	Hb < 115 g/L Hb < 70 g/L	Hb < 115 g/L I:67; NI: 36 ($P < 0.05$) Hb < 70 g/L I: 0 (males); NI: 0 (males); I: 3.57 (females); NI: 0 (females	Not reported	One or more intestinal parasites were detected in 43.7% of I and 32.09% of NI children. Hookworm was the primary infection	Not reported	Not reported
Tanzania; children and adults	Hinderaker et al. (2001)[17]	14–49 y	Female	I: 3836	Hb < 110 g/L Hb < 90 g/L Hb < 70 g/L	Hb < 110 g/L I: 22.7 Hb < 90 g/L I: 4.6 Hb < 70 g/L I: 0.5 (Hb levels adjusted for altitude)	Not reported	Among anemic women, 31.5% had malaria parasitemia compared to 17.5% among non-anemic women ($P < 0.0001$)	Not reported	Not reported
USA; children	Gessner (2009)[68]	6–59 mo	Both	I: 51 NI: 3083	Hb < 110 g/L Hb < 90 g/L	Hb < 110 g/L I: 30.0 versus NI: 18.0 (6–59 mo); I: 35.0 versus NI: 21.0 (10–23 mo); I: 22.0 versus NI: 12.0 (24–59 mo) Hb < 90 g/L I: 1.5 versus NI: 0.62 (6–59 mo); data on other age groups not reported	Not reported	Not reported	Not reported	Adjusted analyses found Alaska Native status was not significantly related to Hb levels for children 10–23 mo ($P = 0.07$) or those 24–59 mo ($P = 0.07$)

Table 3 **Continued**

Country; age group	Study	Age	Sex	No. of anemic subjects in each group	Definition of anemia	Prevalence of anemia by group (%)	Etiology of anemia			
							Nutritional	Infectious	Genetic	Other
USA; pregnant and postpartum women	Gessner (2009)[69]	All ages	Female	I: 8541 NI: 21,613	Hb < 110 g/L (first trimester) Hb < 105 g/L (second trimester) Hb < 90 g/L	Hb < 105 g/L: I: 25 versus NI: 15 Hb < 90 g/L: I: 3.5; NI: 1.3	Not reported	Not reported	Not reported	Adjusted analyses found Alaska Native status was associated with anemia (OR: 1.5; 95% CI: 1.4–1.6)
Venezuela; children and adults	García-Casal et al. (2008)[70]	All ages	Both	I: 182	110 < Hb < 130 g/L (depending on age and sex)	I: 89.6*	ID (SF < 10 to <12 ng/mL depending on age) prevalence was 37.1%. Of those anemic, 35.6% had ID, 57.4% had folic acid deficiency (<3 ng/mL) and 38.2% had both	Not reported	Not reported	Not reported
Venezuela; adults	Diez-Ewald et al. (1997)[71]	69 mo–69 y	Both	I: 406	107 < Hb < 130 g/L (depending on age and sex)	I: 53.6 (Campo Rosario community); 30.6 (Saimadoyi community)*	Prevalence of low serum iron (30 µg/L for children <6 y old and 50 µg/L for other ages), ID (SF < 10 µg/L for children <6 y old and 12 µg/L for other ages), folate deficiency (folic acid <3 µg/L), and vitamin B_{12} deficiency (<150 mµg/L) were 27.5%, 20.3%, 91%, and 64.4% for the Campo Rosario community and 27.7%, 5%, 5.1%, and 0% for the Saimadoyi community	Not reported	Not reported	Not reported
Venezuela and Brazil; children and adults	Grenfell et al. (2008)[30]	Sex: both Age: all ages		I: 183	95 < Hb < 130 g/L (depending on age and sex)	I: 70.5*	Not reported	Prevalence of malaria was 12.6%	Not reported	In adjusted analyses significant factors associated with Hb concentration were: age ($P < 0.001$), sex ($P = 0.01$), splenomegaly ($P = 0.5$), malaria infection ($P < 0.001$), and access to healthcare ($P = 0.01$)

*No data on severity of anemia.
Abbreviations: CI, confidence interval; Hb, hemoglobin; I, indigenous; ID, iron deficiency; IDA, iron-deficiency anemia; MCV, mean cell volume; NI, non-indigenous; OR, odd ratio; NR, not reported; RR, relative risk; SES, socioeconomic status; TfR, serum transferrin receptor; TfS, percent transferrin saturation.

to diagnose anemia across studies (Table 3). Among the studies that reported on the definition of anemia that was used, the most commonly used definitions were those suggested by the WHO nearly 40 years ago, which account for age, sex, and pregnancy status.[16] Studies have found that Hb values are affected by such variables as sex, age, the race of the individual, altitude, and smoking.[16] Adjustments in Hb values were reported for sex, age, pregnancy, and altitude (Table 3). Studies conducted in areas located above sea level recognized the influence of altitude on Hb levels and adjusted the values accordingly. The study by Hinderaker et al.[17] in Tanzania adjusted Hb values using a formula that accounts for altitudes above sea level. In Guatemala, the study by Neufeld et al.[18] defined anemia as Hb ≤130 g/L at lower altitudes and as Hb ≤134 g/L at higher altitudes.

Variations in the definition of anemia by ethnicity were not considered. While there has been evidence to suggest ethnic differences in the cutoffs for Hb and other biochemical measures, the search for consensus on the strength of this evidence and the appropriate cutoffs to use is ongoing. For example, large-scale studies have conclusively established that North American blacks have a lower population-mean of serum Hb concentrations than North American whites.[19] However, these studies have also reported higher erythrocyte sedimentation rates and higher mean serum ferritin levels in the black population, which, in association with low Hb levels, are indicative of anemia due to chronic disease.[19] These findings indicate that the difference in Hb concentrations between blacks and whites in the United States is the result of factors other than iron intake and iron status. Higher serum ferritin levels, despite lower Hb levels, may result from a higher prevalence of acute infections and chronic inflammatory diseases, suggesting that lower Hb levels in blacks and whites may be a consequence of environmental factors related to disease rather than racial causes.[19] Results from a literature review found that hematological means in blacks differ from those in whites, regardless of socioeconomic class.[19] More specific investigations of both the genetic and environmental determinants of iron utilization in blacks are needed. Until studies show that the lower Hb levels in blacks represent a normal physiological variation, race-specific Hb standards are not recommended.

Definitions of iron deficiency (ID) were less consistent across studies. ID is a reduction in body iron to the extent that cellular stores of iron are fully exhausted, and it can occur with or without anemia.[20] There are several laboratory methods that can be used to measure iron in the body, each with its own strengths and limitations, including mean corpuscular volume (MCV), erythrocyte zinc protoporphyrin (ZnPP), transferrin saturation, serum ferritin (SF), and serum transferrin receptor (TfR).[20] The majority of studies in this review used SF to define ID; however, several studies used a combination of iron measures (Table 3). Other micronutrients that can cause anemia when levels are inadequate, such as folate, vitamin B_{12}, and vitamin A, were infrequently reported in studies, but the definitions used are reported where available (Table 3).

Results by country

Australia. Seven studies reported on anemia among Australia's indigenous Aborigines. The validity of the sampling approach and representativeness of the study population were rated as poor in three studies, as moderate in three studies, and as good for a single study (Table 2). Four studies examined modest sample sizes (<500) of children from select communities. Only one study defined the indigenous group specifically as Torres Strait Islander participants[21]; the remaining studies referred to the indigenous study populations simply as "Aboriginal" (Table 3).

Two studies, which were rated as poor in terms of validity and representativeness of sampling method, reported the prevalence of anemia among Aboriginal children to be 16.4% [21] and 75.0%.[22] The study by Heath and Panaretto,[21] which found a prevalence of anemia of 16.4% among Aboriginal children found a significantly lower prevalence of anemia among non-Aboriginal children (4.2%, $P = 0.02$).[21] There were no statistically significant differences between Aboriginal and non-Aboriginal children for eosinophilia (a possible indicator of parasitic infections), ID, and IDA.[21] The prevalence of ID (SF < 15 μg/L and MCV < 74 fL) was 3.6% among Aboriginal and 0% among non-Aboriginal ($P = 0.11$) children. The prevalence of IDA (ID and Hb < 115 g/L) was 3.6% among Aboriginal children and 0% among non-Aboriginal children ($P = 0.11$). The causes of anemia, as indicated in the study report, remain unclear; however, based on a 24-hour food diary, the Aboriginal children reportedly consumed less dairy ($P = 0.007$), meat ($P = 0.013$), and vegetables ($P = 0.014$) compared to the non-Aboriginal children.[21] An association between ID and dietary intakes was not reported, but one could speculate that lower meat and vegetable intakes would negatively impact iron levels over time, since meat contains heme iron, and vitamin C from vegetables aids in iron absorption; however, lower intakes of dairy may positively impact iron levels, since calcium is an iron inhibitor.[7]

The high prevalence of anemia (75%) in the study by Hopkins et al.[22] is likely due to the high hookworm rates that were present at the time the study was conducted in 1992. Infections with hookworm were present in 77% of Aboriginals and were found to be significantly associated

with anemia ($P < 0.001$) and ID ($P < 0.01$), suggesting that hookworm infection was a major contributor to IDA.[22] While dietary intakes were not measured in the study, the authors allude to the possibility that inadequate dietary iron intake is also a major cause of anemia, particularly in children and women; subjects in these groups had higher rates of ID than men, and hookworm-negative Aboriginal women over the age of 14 years showed high levels of ID (50%) and IDA (31%).[22]

Only one study reported on anemia among non-Aboriginal children.[21] This study was rated as poor because of the small sample size, lack of reporting of the response rate, and selective sampling frame (i.e., children aged 4–12 years from three schools).[21] The prevalence of anemia was significantly higher among Aboriginal children (16.4%) compared to non-Aboriginal children (4.2%, $P = 0.02$). Two studies, rated as good and moderate in quality, reported the prevalence of anemia among Aboriginal children to be 14%[23,24] and 24%[25]; however, neither of these studies examined the causes of anemia. In a national study on lead exposure, Mackerras et al.[26] found that the prevalence of anemia among Australian children between the ages of 1 and 4 years, weighted to 1996 census data, was 2% (95% CI: 1.3–3.1).[26] Overall, when comparing the prevalence of anemia among Australian children reported in these studies, it is clear that Aboriginal children fare far worse than their non-Aboriginal counterparts.

Three studies reported on anemia among pregnant women using retrospective data from statewide perinatal databases. In Western Australia, Aboriginal adolescent mothers (age range: 12–18 years) from a single obstetric hospital had a significantly higher prevalence of anemia, defined as Hb < 100 g/L (23%) compared to non-Aboriginal adolescent mothers (8%, $P < 0.001$).[27] In South Australia, the prevalence of anemia (not defined) among pregnant women between 15 and 19 years of age was 25.2% among Aboriginals and 9.9% among non-Aboriginals.[28] Adolescent Aboriginal mothers were more than twice as likely to be anemic compared to their non-Aboriginal counterparts (relative risk [RR]: 2.54; 95% CI: 2.11–3.04).[28] In the state of Queensland, the prevalence of anemia (Hb cut-off not reported) among pregnant women of all ages was 3.3% among Aboriginals and 1.1% among non-Aboriginals.[29] Differences in anemia prevalence may be related to the definition of anemia. Since pregnancy is characterized by increased blood volume, a normal physiological change that causes a reduction in the blood concentration of Hb, a lower Hb cutoff is required for defining anemia in pregnant women.[7] Westenberg et al.[28] and Wills and Coory[29] did not report the Hb level used to define anemia (Table 3).[28,29] The lower prevalence of anemia among pregnant women in Queensland compared to those in South Australia and Western Australia may be a result of particular conditions in the geographic area or of the age of the women examined (all ages versus adolescent females). Indigenous women were found to have higher teenage pregnancy rates, higher teenage birth rates, and higher smoking rates compared to non-indigenous women.[27–29] In all three settings, Aboriginal women had higher rates of anemia compared to non-Aboriginal women. Overall, these results suggest anemia is more prevalent among Aboriginals compared to non-Aboriginal Australians and, based on the WHO criteria, is a moderate public health problem.[15]

Brazil. Among the studies reviewed, two were conducted in Brazil and one was among indigenous people living in the Amazon in Brazil and Venezuela.[30] The study by Morais et al.[31] sampled Terena Indian infants aged 6–120 months from two villages and was given a moderate quality rating. By age group, the prevalence of anemia was 86.1% among infants aged 6–24 months, 50.8% among infants aged 24–60 months, and 40.7% for children aged 6–120 months.[31] The study by Orellana et al.[32] was rated as good and sampled all infants between the ages of 6 and 119 months in 9 of the 11 villages inhabited by Surui Indians. The prevalence of anemia was 62.3% among Terena Indian infants and 80.6% among Surui Indian infants (Table 3).[31,32]

There was no data on the etiology of anemia; however, based on previous data from Brazil, Morais et al.[31] suggested anemia is mostly due to ID and blood loss from intestinal parasites. In the study by Orellana et al.,[32] one of every four children was undernourished and, according to previous data, the infant mortality rate for Surui Indians (70/1,000 in 2004) was more than twice as high as the average for the general Brazilian population. Both authors referred to poor living conditions, including inadequate water and sewage treatment systems, as likely causes of anemia. In terms of infectious causes of anemia, malaria was apparently interrupted two decades ago and a recent parasitological survey found a low prevalence of hookworm infection (<5%).[32]

The study conducted in the Brazilian and Venezuelan regions of the Amazon was a cross-sectional survey of Yanomami people of all ages living in 11 communities that were randomly selected from census data.[30] The study found that 70.5% of the Yanomami people living in the Ocamo area of the Amazon in Brazil and Venezuela were anemic.[30] Unfortunately, the prevalence of anemia was not reported separately by age group, thus limiting our understanding of anemia in high-risk age groups such as infants and women in this region. However, as in the two other studies performed in Brazil, the high prevalence of anemia suggests the condition is a severe public health problem among various indigenous peoples in the country.[15]

Canada. Among the four studies (seven articles) conducted in Canada that met the inclusion criteria, none included a non-indigenous comparison group (Table 2). One study of Nunavik pregnant women found that 39% were mildly anemic (Hb < 120 g/L) and 22% were moderately anemic (Hb < 115 g/L).[33] Among the Nunavik women sampled, 27% had positive or equivocal *Helicobacter* serology results. Further information on possible nutritional causes of anemia were not available due to the retrospective nature of the study design.[33]

Four studies examined infants of Cree, Inuit, or First Nations descent between the ages of 3 and 60 months.[33–38] Between 1995 and 1998, Willows et al.[39] examined 354 Cree infants at their 9-month well-baby clinic visit and found that 7.9% (95% CI: 5.3–11.2) had anemia (Hb < 110 g/L), 31.1% (95% CI: 26.2–36.1) had moderate anemia (Hb < 105 g/L), and 17.2% (95% CI: 13.4–21.5) had severe anemia (Hb < 100 g/L). Between 1998 and 2000, Willows and Gray-Donald reported on 274 Cree infants at their 9-month well-baby clinic visit and found that 25.6% were anemic (Hb < 110 g/L).[36–38] Between 2001 and 2003, Christofides et al. found 36% of Inuit and First Nation infants (4–18 months old) to be anemic.[34,35] A study by Hodgins et al.[33] in 1989–1992 found that 58% of Inuit infants were anemic and 39% (Hb < 120 g/L) of Inuit women were anemic.

In terms of the etiology of anemia, Christofides et al. and Willows et al. discuss infant feeding practices and the need to address socioeconomic conditions that prevent healthy feeding practices from being adopted, such as the continued use of cow's milk/evaporated milk, which is low in iron and nutritionally replete instead of formula, because cow's milk is less expensive and more readily available.[34–38] In their study, Christofides et al.[34] found anemia to be significantly associated with *Helicobacter pylori* infection (OR: 3.10, 95% CI: 1.01–9.51), consumption of cow's/evaporated milk (OR: 2.84, 95% CI: 1.24–6.50), and prolonged breastfeeding (OR: 2.47, 95% CI: 1.04–5.85); formula intake was significantly associated with risk of ID (sTfR > 8.5 mg/L) (OR: 0.35, 95% CI: 0.15–0.84). The high prevalence of *H. pylori* suggests a need to improve water quality and sanitation in the indigenous communities studied.[34,35] In the studies by Willows and Gray-Donald,[36–38] the authors found that concentrations of microcytic erythrocytes were higher among infants who were breastfed (OR: 10.7, 95% CI: 3.9–29.0), fed cow's milk (OR: 9.2, 95% CI: 3.2–26.8), and fed mixed milks (OR: 7.3, 95% CI: 2.2–23.9) in comparison with formula-fed infants. The authors also reported that thalassemia minor is not present in Cree infants. Based on the available evidence, the level of anemia among indigenous groups in Canada appears to represent a moderate public health problem.[15]

Guatemala. A single study of Mayan people in Guatemala was found for the present review.[18] The study collected data on 253 women from 17 villages with the aim of examining the association between smoky indoor cooking fires and elevated Hb concentrations, but it did not indicate how representative these 17 villages were of the total Mayan community nor did it report the survey response rate. The study was conducted in 1994 and found that 25% of Mayan women (15–45 years old) were anemic and 31% had depleted iron stores. Since no other studies that reported on anemia in indigenous populations in Guatemala were found, the prevalence of anemia in the Mayan community and in other indigenous populations in Guatemala is essentially unknown. Nevertheless, this study indicates anemia among Mayan women is a health issue that warrants clinical attention.

India. Twelve of the studies reviewed were conducted in India. These studies varied widely in terms of the age and tribal groups investigated. Of the four comparison group studies, only the study by Ghosh and Bharati[41] included a significance test, and a statistically significant difference in the rates of anemia was found between tribal and non-tribal groups. The study examined married women between the ages of 15 and 42 years in a periurban area in Kolkata city and found that anemia, of varying degrees, was more prevalent among women belonging to the Munda tribe compared to women belonging to the Poundrakshatriya Hindu caste ($P < 0.001$).[41] The rates of mild (Hb 100–119 g/L), moderate (Hb 70–99 g/L), and severe (Hb < 70 g/L) anemia were 37.1%, 55.2%, and 7.6%, respectively, among Munda tribe women compared to 48.6%, 9.7%, and 2.9% of 9.7%, respectively, among women belonging to the Poundrakshatriya Hindu caste (Table 3).[41]

A population-based study conducted in the state of West Bengal by the National Nutrition Monitoring Bureau found a significantly higher prevalence of anemia (Hb < 110 g/L) among Scheduled Tribe children between the ages of 1 and 5 years compared to other children in the community.[42] This study in West Bengal among rural children highlighted the high prevalence of anemia among all children due to diets with inadequate micronutrient content, most notably iron, and poor hygienic conditions – the majority of children practiced open defecation, as sanitary facilities were absent in 81% of households.[42] While the authors did not have data on hookworm infection, they reported the importance of periodic deworming among children as a preventive measure for anemia and IDA.[42]

The study by Kotecha et al.[43] examined 895 tribal and 1,965 non-tribal girls aged 12–19 years from schools in the Vadodara district of Gujarat. While the authors did not report a significance test, the prevalence of anemia

was relatively similar between tribal (73.7%), rural (74.5%), and urban (75.8%) areas.[43] After 17 months of educational intervention about iron and folic acid supplementation, the overall rates of anemia decreased by 21.5 % (from 74.7% to 53.2%, $P < 0.05$).[43]

In Nagpur district in Maharashtra, Menon et al.[44] examined non-pregnant women between the ages of 18 and 30 years and found that anemia rates in tribal versus non-tribal women were 72% versus 53%, respectively ($P = 0.07$). In a district in central India, Singh et al.[45] studied 456 tribal women between the ages of 18 and 40 years and found that anemia was commonly present in most (80%) of the women. Approximately 21% of the women in this study were found to be infected with malaria.[45] While the authors did not report on any nutritional or genetic causes of anemia, they reported the high probability that dietary inadequacy played an important role. More than 60% of pregnant women without malaria were also anemic. Among women with malaria, anemia was more frequent in multigravidae versus primigravidae women (69.6% versus 54%, respectively); thus, highly prevalent anemia of nutritional origin appears to be further aggravated by pregnancy.[45]

Six non-comparison studies were conducted in different states and among diverse tribal peoples of varying ages, making comparisons among studies very difficult, if not impossible.[46–52] The prevalence of anemia (Hb < 110 g/L) ranged from 16.2% among male Bhil and Pawar adolescents (12–18 years old) in Jalbalpur state to 86.7% among Gond children of all ages in Madhya Pradesh (Table 3).

Studies reported a high proportion of parasitic infections related to unhygienic living conditions as well as poor nutrition and a high prevalence of low-birth-weight infants.[41,46,47] While not measured in any of the studies described, authors also discussed previous studies in Indian populations in which thalassemia and hemoglobinopathies were found.[47] Levels of anemia across the heterogeneous group of studies suggest that anemia is a severe public health problem for women and children in both tribal and non-tribal groups in India.[15]

Kenya. A single study from Kenya was included, which was conducted in Kismu. This study sampled 3,645 healthy pregnant women of all ages from a single hospital to assess risk factors for anemia in late pregnancy.[53] Results were reported by gravidity. Among women in primi- and secundi-gravidae, those from the Luo tribe had a higher prevalence of anemia (Hb < 110 g/L) relative to non-tribal women (81.6% versus 70.6%, respectively; RR: 1.16; 95% CI: 1.10–1.22) and a higher prevalence of severe anemia (Hb < 70 g/L) (7.5% versus 5.1%, respectively; RR: 1.47; 95% CI: 1.02–2.12) (Table 2).[53] Among women in gravidae three or more, those from the Luo tribe had a higher prevalence of anemia compared to non-tribal women (76.5% versus 69.5%, respectively; RR: 1.10; 95% CI: 1.02–1.19) and a higher, but not significantly different, prevalence of severe anemia (7.1% versus 4.5%, respectively; RR: 1.57; 95% CI: 0.92–2.68).

The study did not measure iron status or other micronutrient causes of anemia; however, a high seroprevalence of malaria and HIV was found among all women (tribal and non-tribal). The level of anemia reported in this single study suggests that among both tribal and non-tribal pregnant women in Kenya anemia is a severe public health issue.[15]

Malaysia. Three studies on anemia among indigenous populations in Malaysia were identified. Two studies were conducted among the indigenous Orang Asli population.[54,55] One study examined 368 Orang Asli children aged 2–15 years living in eight villages in the state of Selangor and found that 41.5% were anemic (Hb < 120 g/L).[55] Similar findings were reported in a single school-based study in the state of Pahang that evaluated 241 Orang Asli children; in this study, 48.5% (95% CI: 42.3–54.8) of Orang Asli children aged 7–12 years were anemic (Hb < 120 g/L) (Table 3).[54] Iron deficiency accounted for 61% of anemia cases in the Selangor study and 70.1% of cases in the Pahang study.[54,55] The Selangor study examined stool samples from a subsample of children ($n = 281$) and found the prevalence rates for hookworm, trichuriasis, ascariasis, and giardiasis were 19%, 26%, 3%, and 24.9%, respectively.[55] In the Pahang study, the prevalence rates for hookworm and ascariasis were 13.4% and 22.3%, respectively.[54]

In a study of indigenous people by Sagin et al.[56] seven villages were selected in five remote communities in Sarawak. Among indigenous people between the ages of 5 and 85 years, the researchers found that 29.2% of males were anemic and 17.2% of females were anemic.[56] Anemia was almost twice as common in males (29.2%) than females (17.2%), except for adolescent and reproductive females (31.6%). The authors speculated that the higher incidence of anemia in men aged 40 years and older may be a result of nutritional deficiency or alcoholism.[7]

According to Sagin et al.,[56] the area of the Amazon Basin they examined has undergone large-scale development projects and the diet of the indigenous peoples has changed from traditional nomadic diets to imported and processed foods. The two studies described here indicate anemia is a moderate public health problem in the Sarawak population and a severe public health problem among Orang Asli children.[15]

Mexico. Mexico produced the highest quality of evidence regarding anemia in indigenous populations. Five articles

reported data for women and children from the National Nutrition Survey (NNS) in 1999.[57–60] The prevalence of anemia among indigenous and non-indigenous groups in various age groups were as follows: 35.8% versus 26.1% for children younger than 5 years, 24% versus 19% for children aged 5–11 years, and 24.8% versus 20.4% for adult mothers.[57–60] Data comparing rates of anemia among children from the Mexican National Health and Nutrition Survey (2006) and the Mexican National Nutrition Survey (1999) indicate the prevalence of anemia has decreased by 1.8 percentage points per year among indigenous toddlers.[61] Compared to the national data, lower estimates of anemia were reported in smaller studies among Tarahumara (13%)[62] and Yaqui (0.7%)[63] indigenous children in Mexico. The lower prevalence of anemia among Tarahumara children compared to national data is postulated to be a result of selection bias (boarding school) and a feeding intervention that had been implemented in the school.[64] A study among Tarahumara women in Northern Mexico found that 12.1% of non-pregnant ($n = 446$) and 17.1% of pregnant ($n = 35$) women (age range: 12–49 years) were anemic.[64] The low rate of anemia among the Yaqui people is likely due to adequate amounts of iron in the Yaqui diet.[63] The Yaqui Valley is located in one of the most important agricultural regions in Mexico.[63] According to the study's authors, the amount of protein in the diet of Yaqui Indians is adequate, with sufficient amounts of vegetable protein and smaller quantities of animal proteins derived from eggs, dairy products, and occasional fresh or processed meats.[63]

Among studies that reported on the etiology of anemia, causes included ID and poor socioeconomic conditions.[60,64] The median intake of dietary iron for those younger than 5 years of age corresponded to 50% of the recommended daily allowance and reached 80.8% in subjects aged 9–10 years. Iron sources were also found to contain high amounts of phytic acid (>500 mg/day) and tannins.[60] Another cause of anemia discussed by Shamah-Levy et al.[59] is poverty. The authors state it is well known that indigenous populations are the poorest in Mexico and have the least access to social support services. Based on nationally representative data from the nutrition survey, anemia in indigenous women and children in Mexico is a moderate public health problem.[15]

New Zealand. Results from a study in Auckland, New Zealand conducted among adolescents in grades 5–7 (ages not specified) in eight schools found a higher prevalence of anemia (Hb < 120 g/L) among female Maori versus non-indigenous students (11% versus 4%).[65] The risk ratio of anemia was 2.92 (95% CI: 1.15–7.43) in Maori versus non-indigenous female students (Table 3).[65] The prevalence of anemia in this single study suggests anemia may be a mild public health problem among Maori females in this student group; however, the small and selective sample from this single study in a single urban city prevents any conclusions from being made about the level of anemia among indigenous populations in New Zealand in general. Furthermore, the study did not provide information on the etiology of anemia and the authors requested that further research be conducted to examine possible risk factors.

New Zealand conducts population monitoring of dietary intakes and biochemical indices. Published data in the peer-reviewed literature on 15–49-year-old women ($n = 1751$) from the National Nutrition Survey in 1997 (NNS97) provide estimates of ID and IDA among Maori and non-indigenous New Zealanders.[66] Nationally representative population estimates among women for ID (defined as SF < 12 μg/L) were reported as 4.8% (95% CI: 1.9–7.6) among Maori and 4% (95% CI: 2.4–5.6) among non-indigenous New Zealanders.[66] IDA defined as Hb < 120 g/L and SF < 12 μg/L was 7.3% (95% CI: 3.2–11.4) among the Maori and 2.5% (95% CI: 1.2–3.9) among non-indigenous New Zealanders.[66] While the findings for women from the NNS97 found marginal and generally non-significant differences in the prevalence of IDA or ID across age and ethnic groups, rates were higher among the Maori compared to non-indigenous groups.[66] In their commentary, the authors expressed surprise at the lack of significance in these findings, given the higher prevalence rates of IDA and ID reported among Maori students compared with their Caucasian counterparts in the Schaaf et al. study.[65] The authors speculated that marked ethnic group differences may only occur in childhood and adolescence.[66] The number of adolescents in ethnic groups was limited in the NNS97 data and therefore could not be analyzed separately.[66] Another possibility is differential response rate bias for the response to blood collection in the NNS97 (e.g., 73% among non-indigenous New Zealanders compared to 61% among Maori).[66] Unfortunately, as the study did not report data on the prevalence of anemia it did not meet eligibility criteria for this review.[66]

Sri Lanka. A single study in Kelaniya, Sri Lanka compared Veddah (tribal) children to Sinhalese (non-tribal) children between the ages of 6 and 15 years and found anemia (Hb < 115 g/L) was present in 67% of Veddah children compared to 36% of Sinhalese children ($P < 0.05$) (Table 3).[67] Both wasting and anemia were higher among indigenous children. Causes of anemia were reported to be inadequate diet, undernutrition, and high rates of intestinal infections as a result of contaminated drinking water. While there was only one study on anemia among indigenous people in Sri Lanka, its results indicate anemia is a severe public health problem for Veddha children in this country.

Tanzania. One study in northern Tanzania collected data on women aged 14–49 years from 12 antenatal clinics in a single Lutheran hospital.[17] The women represented the following tribal groups: Iraqw, Datoga, and others. The overall prevalence of anemia was 22.7%. Among the anemic women, 31.5% had malaria compared to 17.5% of non-anemic women ($P < 0.0001$). This single study indicates anemia is common in this particular high-altitude, rural region of northern Tanzania, albeit less common than the authors expected, based on previous studies in most other parts of the country.[17] The authors cite previous studies of pregnant women in the general population in Tanzania among whom anemia prevalence ranged from 41% to 95%.[17] A lower prevalence of diseases such as intestinal worms and sickle cell disease were cited as possible reasons for the lower prevalence of anemia among women in the rural area studied compared to previous estimates from other areas in Tanzania. The authors indicate further studies on the micronutrient status of the women are needed in order to identify more specific etiological factors.[17]

United States of America. One study from the United States that examined anemia among an indigenous population met the inclusion criteria. Two articles by Gessner[68,69] reported results from a retrospective, 1999–2006 cohort based on data from the US Supplemental Nutrition Program for Women and Infants (WIC). The prevalence of anemia was higher among Native Alaskan infants compared to non-Native Alaskan infants at 10–23 months of age (35% versus 21%, respectively) and at 24–59 months of age (22% versus 12%, respectively).[68] The relative risk of anemia among Alaskan Natives versus non-Native Alaskans was 1.7 (95% CI: 1.6–1.7) for infants 10–23 months old, and 1.9 (95% CI: 1.8–2.0) for infants 24–59 months old.[68] Among pregnant or postpartum women, Alaskan Natives had higher rates of anemia (25%) and severe anemia (3.5%) compared to non-Native women (15% and 1.3%, respectively).[69] Analyses adjusted for Hb levels found the following significant factors: age, Alaskan Native status, rural residence, number of previous pregnancies, maternal prenatal tobacco use, and female gender.[69] The magnitude of anemia among Alaskan Native women and children, based on the studies by Gessner, suggests anemia is a moderate public health problem in this segment of the US population.[68,69]

Venezuela. There have been few hematological studies on indigenous people in Venezuela, but two studies were identified that reported on anemia since 1996. One study, conducted in the Amazon, reported on anemia among 182 Piaroa people of all ages using data from a single medical facility.[70] The study found that 89.6% of Piaroa people were anemic, and 35.6% of them were iron deficient. A second study collected data from two groups of Bari Indians; 179 subjects were from the Campo Rosario community and 287 were from the Salmadoyi community.[71] The two communities differ greatly in their location and access to food. As described by the study authors, the Bari Indians from the Campo Rosario community live in an arid area with impoverished agriculture and maintain a diet of pasta or rice cooked with onions, sweet peppers, and yuca (a starchy vegetable).[71] Vegetables, fruit, and meat are scarce, aside from the occasional smoked monkey. In contrast, the Salmadoyi community is in a fertile valley with access to vegetables, fruit, and domestic animals.[71] In the Campo Rosario community, anemia was 53.6%, ID was 20.3%, folate deficiency was 91%, and vitamin B_{12} deficiency was 64.4% (Table 3). Micronutrient deficiencies in the Campo Rosario community are likely the result of inadequate diet and parasitic infections from food and water contamination.[71] The more nutritious Salmadoyi diet is reflected in lower levels of observed micronutrient deficiencies, i.e., anemia was 30.6%, ID was 5%, folate deficiency was 5.1%, and vitamin B_{12} deficiency was 0% (Table 3). Based on evidence from these two studies, anemia is a severe public health problem among secluded indigenous populations in Venezuela that have limited access to a diverse diet and to governmental health programs (Table 3).

DISCUSSION

The results of this systematic review of the peer-reviewed literature indicate a number of important findings regarding the documentation and prevalence of anemia among indigenous populations worldwide. First, in more than a decade of scientific literature, there has been very little published evidence that characterizes the prevalence, severity, and etiology of anemia among indigenous populations. There are an estimated 370 million indigenous peoples living in 70 countries worldwide,[10] yet this review found only 50 studies pertaining to 13 countries. The majority of studies in this review reported on anemia among the Inuit and First Nation populations in Canada, the Aborigines of Australia, indigenous peoples in Mexico, and tribal groups in India. Other indigenous populations in the world were entirely absent, such as the Indians of the Americas, the Saami of northern Europe, and the indigenous people that comprise more than 60% of Bolivia's population.[72] China and India have more than 150 million indigenous and tribal people, and Myanmar has about 10 million indigenous people,[72] but none of them are currently represented in the English-language, peer-reviewed scientific literature on anemia. The lack of published studies on anemia in indigenous populations in China may reflect a limitation of the search strategy or a genuine gap in the literature. However, the latter was

suggested in a World Health Organization report on indigenous peoples worldwide, which mentioned that while China has the largest population of minority peoples, very few data on indigenous health are available for the country.[11]

Second, based on the available evidence reviewed here, the magnitude of anemia as a public health problem among indigenous populations internationally ranges from a moderate (20–39.9%) to a severe (≥40%) level. For the most part, anemia is a moderate problem among indigenous populations living in developed countries, such as Australia and Canada, and it reaches a severe level in poorer parts of the world, where rates of anemia are likely to be high among the general population as well. In either case, the burden of anemia is overwhelmingly higher among indigenous groups compared to the general population. This speaks to the etiology of anemia, which includes inadequate diet and poor living conditions, as well as an association with malaria and intestinal parasites.

Third, the results of this review indicate the quality of published data on anemia in indigenous peoples is generally poor. Mexico was the only country with peer-reviewed studies reporting on anemia in a nationally representative sample of indigenous women and children. The quality of evidence from the remaining 12 countries was generally sparse and poor in quality. Information on anemia among indigenous peoples from Brazil, Guatemala, Malaysia, New Zealand, Sri Lanka, Tanzania, Venezuela, and the United States were based on one or two small studies examining select samples. In Brazil, for example, national census data confirms higher mortality rates among indigenous compared to non-indigenous people,[73] yet there is no national information about the health or nutrition of indigenous people because such surveys have only reported on non-indigenous populations.[32] Australia, Canada, and India were the only other countries, aside from Mexico, with three or more published studies in more than a decade of research on anemia in indigenous peoples; however, none of these countries provided nationally representative estimates. Australia provided state-based studies; however, these studies were retrospective and relied on prenatal data from medical records. While it is easy to be critical of the lack of high-quality data on this topic, it must be recognized that collecting data on indigenous populations is difficult and costly. Indigenous communities are often located in rural and remote areas and usually represent a small proportion of a country's total population. Furthermore, nationwide surveys may not collect blood samples to analyze markers of anemia or ID due to high costs and limited resources.

A limitation of this study is the inclusion of peer-reviewed literature from 1996 onwards and in the English language only. There were no studies identified in the search strategy that were published in languages other than English, and the threshold for the publication date was designed to include only current estimates of anemia for present-day policy relevance. In addition, the definition of "indigenous" is ambiguous. It is therefore possible that studies on indigenous populations were not identified in the search strategy. In an effort to increase the comprehensiveness of the literature search, variations of the search terms "aboriginal", "tribe", and "indigenous" were included, multiple electronic databases were searched, and references from relevant articles were cross-checked. In addition, the World Health Organization's online database of studies reporting on the prevalence of anemia was used as a resource to cross-check references, and it demonstrated completeness in our search strategy among the countries that had studies identified in our search.[9] Country reports were beyond the scope of this review because there is no systematic procedure or source for ensuring all of the 70 estimated countries with an indigenous population were searched. However, to ensure greater completeness of the search strategy and to account for the fact that studies may refer to indigenous people by a specific name rather than using the generic terms aboriginal, indigenous, or tribe, stage two of the search strategy included a detailed search for indigenous studies on anemia for countries that were already identified in stage one. In order to comprehensively understand the extent and scope of anemia for all indigenous populations worldwide, a global database of anemia similar to the existing WHO database on anemia[9] needs to be created for indigenous peoples by name, country, and language."

The fact that a high prevalence of anemia among indigenous populations is widely distributed around the world, in countries such as Australia, Mexico, Sri Lanka, Malaysia, Guatemala, and Canada, suggests that common disparities in health among these diverse groups of people are attributing to a higher risk of anemia. Conversely, there are other possible risk factors that may not be applicable to all indigenous groups worldwide, such as changes in traditional food systems. In Alaska, for example, there appears to be a paradox between higher rates of anemia among Alaskan Natives despite a traditional diet that is high in meat content.[68,69,74,75] While access to fresh produce and meat may have increased over time, scientists have reported higher rates of anemia in these communities as far back as 1955.[76] Studies suggest that *H. pylori* and other infections, as well as idiopathic causes, are unlikely at the population level.[77] In Alaskan Natives, however, a possible genetic base for higher rates of anemia has not been ruled out.[69]

The three major causes of anemia among indigenous populations identified in this review, i.e., ID, malaria, and

helminth infections, can be addressed using a combination of interventions, such as iron supplements targeted to at-risk groups, fortification of staple foods with iron and other micronutrients, prevention and treatment of malaria, use of insecticide-treated materials and bed nets, and deworming (anthelminthics) in at-risk groups. Additional strategies for preventing and controlling anemia include fully immunizing children; treating communicable diseases; managing obstetric complications, particularly excessive bleeding; promoting birth spacing through use of modern family planning methods; exclusive breastfeeding for the first 6 months of life followed by appropriate complementary feeding with iron-rich foods; and improving water quality as well as sanitation facilities and practices.

In parts of Australia, interventions to eradicate hookworm infections have been implemented and found to be effective in reducing infection rates.[23] In Mexico, results from three National Nutrition and Health surveys, conducted in 1996, 1999, and 2006, indicate a decrease in the prevalence of anemia from 1996 to 2006 among indigenous and non-indigenous children.[61] The authors attributed the improvements in child health in Mexico to national social programs, including nutritional interventions such as *Oportunidades*, a federal poverty alleviation program that combines cash transfer with financial incentives for positive health and social behaviors (e.g., regular school attendance and health clinic visits).[57] Mexico has also endorsed and supported a large-scale multiple-micronutrient supplementation program aimed at preventing anemia and micronutrient deficiencies in infants in predominantly indigenous communities.[57] Evidence from the national surveys in Mexico provide promising results; however, anemia among indigenous children and women in Mexico is still a moderate public health problem (about 30% at the national level), which warrants more public health interventions and research on risk factors.

In India, the National Nutritional Anaemia Prophylaxis Programme (NNAP) has been in operation for more than 30 years, yet anemia remains a major public health problem.[42] The effectiveness of daily iron-supplementation programs has been questioned because of poor efficiency of health services and lack of compliance by targeted groups.[48] Positive findings from the intervention trial of Deshmukh et al.,[48] which provided weekly iron supplementation to control anemia, is evidence that such interventions can be effective for correcting anemia. A critical issue in ensuring the efficacy of interventions aimed at correcting anemia is managing the operational side. For example, the study by Arlappa et al.[42] in West Bengal found that none of the rural preschool children (indigenous and non-indigenous) had received the government-funded iron plus folic acid tablets, indicating that strategies and funds to improve administrative efficiency and ensure transparency are still needed.

CONCLUSION

To the best of our knowledge, this is the first systematic review to report on the prevalence of anemia among indigenous populations globally. The magnitude of anemia among indigenous groups worldwide carries public health importance, particularly in planning programs to improve maternal and infant health. Further research examining common challenges in monitoring, preventing, and treating anemia and other nutritional deficiencies in these diverse peoples may be useful for developing a concerted global effort to reduce the burden of anemia in these marginalized populations worldwide.

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