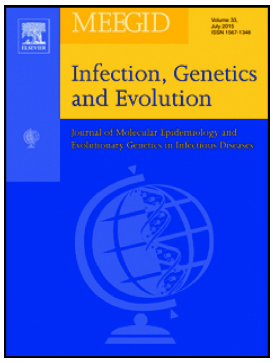
Ng-Hublin, J.S.Y., Combs, B., Reid, S.A. and Ryan, U. (2017) Differences in the occurrence and epidemiology of cryptosporidiosis in Aboriginal and non-Aboriginal people in Western Australia (2002 − 2012). Infection, Genetics and Evolution, 53. pp. 100-106.



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PII: S1567-1348(17)30178-8
DOI: doi: 10.1016/j.meegid.2017.05.018
Reference: MEEGID 3154

To appear in: *Infection, Genetics and Evolution*

Received date: 15 February 2017
Revised date: 16 May 2017
Accepted date: 20 May 2017

Please cite this article as: Josephine S.Y. Ng-Hublin, Barry Combs, Simon Reid, Una Ryan , Differences in the occurrence and epidemiology of cryptosporidiosis in Aboriginal and non-Aboriginal people in Western Australia (2002−2012), *Infection, Genetics and Evolution* (2017), doi: 10.1016/j.meegid.2017.05.018

Differences in the occurrence and epidemiology of cryptosporidiosis in Aboriginal and non-Aboriginal people in Western Australia (2002-2012)

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Abstract

Cryptosporidiosis is a diarrhoeal illness caused by the protozoan parasite *Cryptosporidium*. In Australia, very little is known about the epidemiology of cryptosporidiosis in Aboriginal peoples. The present study analysed long-term cryptosporidiosis patterns across Western Australia (WA) (2001–2012), combined with genotyping and subtyping data at the 18S and glycoprotein 60 (*gp60*) loci respectively. Comparison of cryptosporidiosis notifications between Aboriginal and non-Aboriginal people in WA, revealed that notification rates among Aboriginal people were up to 50 times higher compared to non-Aboriginal people, highlighting the burden of the disease in this population. More than 90% of notifications were in Aboriginal children aged 00-04 years, who had a notification rate 20.5 times higher than non-Aboriginal children in the same age group. *Cryptosporidium hominis* was the predominant species infecting both Aboriginal and non-Aboriginal people. However, Aboriginal people were mainly infected with the *C. hominis* IdA15G1 subtype, whereas non-Aboriginal people were predominantly infected with the IbA10G2 subtype. To control cryptosporidiosis in Aboriginal populations in Australia, effective health interventions/promotions need to be a priority for public health research and action.

Keywords: Cryptosporidium; Aboriginal; Non-Aboriginal; Western Australia; *gp60*

1.0 Introduction

Cryptosporidium is a protozoan parasite of medical and veterinary importance that causes gastroenteritis in a variety of vertebrate hosts (Xiao, 2010). In addition to diarrhea, symptoms associated with cryptosporidiosis include vomiting, nausea, lack of appetite and cramps (Chalmers and Davies, 2010). Malnutrition, which impairs cellular immunity, is an important risk factor for cryptosporidiosis (Gendrel et al., 2003) and *Cryptosporidium* infection in children is also associated with persistent growth retardation and cognitive deficits (Guerrant et al., 1999; Mondal et al., 2009). Currently 33 species of *Cryptosporidium* are recognized as valid and of these, *C. hominis* and *C. parvum* account for more than 90% of human cases of cryptosporidiosis worldwide, infecting 8-10 million people per year (Xiao, 2010; Jezkova et al., 2016; Ryan et al., 2016; Seeber and Steinfelder, 2016).

Human cryptosporidiosis is a notifiable disease in several, mainly European countries (ECDC, 2014). In Australia, the National Notifiable Diseases Surveillance System (NNDSS) was established in 1990, under the auspices of the Communicable Diseases Network Australia and in 2001, cryptosporidiosis was listed as a national notifiable disease (Blumer et al., 2003). At the time of study, only confirmed cases (based on microscopy or PCR) are reported to the NNDSS. However, molecular identification of *Cryptosporidium* species is not routinely carried out, and therefore the *Cryptosporidium* species responsible for the infections reported to the NNDSS are generally unknown.

To date, in humans in Australia, *C. hominis, C. parvum, C. meleagridis, C. fayeri, C. andersoni, C. bovis, C. cuniculus*, a novel *Cryptosporidium* species most closely related to *C. wrairi* and the *Cryptosporidium* mink genotype have been reported (Morgan at al., 1997; Morgan et al., 1998; Gasser et al., 2004; Chalmers et al., 2005; Jex et al., 2007; Ng et al., 2008; Jex et al., 2008; Alagappan et al., 2008; Pangasa et al., 2009; Waldron et al., 2009; Ng

et al., 2010a; 2010b; Waldron et al., 2010; Power et al., 2011; Waldron et al., 2011a; Ng et al., 2012; Jex et al., 2012; Ng-Hublin et al., 2013; Sari et al., 2013 unpublished - KF279538; Yang et al., 2013; Koehler et al., 2013; Koehler et al., 2014a; 2014b; Ebner et al., 2015; Ng-Hublin et al., 2015).

Very little is known about cryptosporidiosis in Aboriginal people, although previous studies have reported high rates of cryptosporidiosis in Aboriginal children (Assadamongkol et al., 1992; Lal et al., 2015a; 2015b). The present study therefore aimed to provide insights into the prevalence, disease patterns of cryptosporidiosis and species and subtypes of *Cryptosporidium* in Aboriginal and non-Aboriginal people in Western Australia (WA), from the first full year of reporting in 2002 through to 2012.

2. Material and Methods

2.1 Case definition

The Australian case definition for cryptosporidiosis notification, which is also adopted by WA, requires laboratory definitive evidence through the detection of *Cryptosporidium* oocysts (NNDSS, 2016).

2.2 Data source and extraction

Cryptosporidiosis notification data used in the present study were obtained from the Western Australian Notifiable Infectious Disease Database (WANIDD) by the data custodian, the Communicable Disease Control Directorate (CDCD) at the WA Department of Health. The WA surveillance system relies on mandatory reporting of notifiable diseases by

laboratories and medical practitioners. WANIDD, is an intranet-based real-time application with notifications from laboratories uploaded electronically and notifications from medical practitioners added manually.

WANIDD data from January 2002 to December 2012 were extracted and included in the present study. The data was saved in Microsoft Excel de-identified through removal of case name, address and date of birth, under Murdoch Human Ethics approval number 2012/208 and the WA Department of Health Ethics approval number 2009/48. The ODOO is defined as the 'true' date of onset provided by the notifying doctor or obtained during case follow-up, the date of specimen collection for laboratory notified cases, and when neither of these dates is available, the date of notification by the doctor or laboratory, or the date of receipt of notification, whichever is earliest.

Variables exported from WANIDD included public health unit (PHU), sex, country of birth, Aboriginal status, age, travel history, location where infection was acquired and hospitalization. Additional information such as clinical symptoms, duration of diarrhea and the infecting *Cryptosporidium* species based on molecular typing data (when available), were included in variables exported.

2.3 Molecular typing

Molecular typing was conducted on a total of 324 *Cryptosporidium*-positive human faecal samples provided by WA clinical pathology laboratories. These samples were matched to the basic demographic data of cases from WANIDD. Molecular genotyping was carried out on isolates with ODOO from 2005 to 2008 and have been detailed in two previous studies Ng et al. (2010a) (n = 198) and Ng et al. (2010b) (n = 42). Molecular genotyping for the

remaining 84 samples were carried out from June 2010 to December 2012 as part of the present study.

Total DNA was extracted using a QIAmp DNA Stool Kit (Qiagen, Germany). Initial genotyping of the samples was carried out by PCR-RFLP and sequencing of an 830 bp fragment of the *Cryptosporidium* 18S rRNA gene as described by Xiao et al. (2001). For samples that failed to amplify or produced ambiguous banding patterns, a two-step nested PCR and sequencing of 540 bp of the 18S rRNA gene was carried out (Ryan et al., 2003). Samples that were positive for *C. hominis* and *C. parvum* were subtyped at the glycoprotein 60 (*gp60*) gene using a two-step nested PCR that amplifies an 830 bp fragment (Strong et al., 2000; Sulaiman et al., 2005). Secondary PCR products were purified and sequenced as described by Ng et al. (2006).

2.4 Data analysis

Statistical analyses were conducted using Microsoft Excel 2010 and OpenEpi version 3.01 (http://openepi.com/v37/Menu/OE_Menu.htm) and STATA 13. Notification rates were calculated using annual census population data available through the Rates Calculator software (Department of Health, WA) and age standardised to the Australian population. Notification Rate Ratios were calculated by dividing the notification rate in Aboriginal people by the notification rate in non-Aboriginal people. WA population estimates for public health unit (PHU) areas, year, sex, age and Aboriginality status was used to calculate notification rates and described as number of notified cases per 100,000 populations/year. Public Health Units in Western Australia are divided into different health administrative regions –Metropolitan (North and South), Kimberley, Pilbara and Goldfields region

(classified as remote) and the Midwest, Wheatbelt, South West and Great Southern (classified as rural).

3. Results

A total of 2,988 cryptosporidiosis cases, with an average notification rate of 12.7 cases/100,000 population, were reported in WA during the 11-year period from January 2002 to December 2012. Individuals that were not residents of WA accounted for 17 of the cases notified during this period, of which three cases were Aboriginal people, nine were non-Aboriginal and the Aboriginality status for five of the remaining cases was not recorded.

3.1 Cryptosporidium notification rates in Aboriginal and non-Aboriginal people

Of the 2,988 cryptosporidiosis notifications, 24.6% (734/2,988) were Aboriginal people, 53.1% (1,585/2,988) were non-Aboriginal people and Aboriginal status was not recorded for 22.4% of cases. Notification rates in Aboriginal and non-Aboriginal people over the 11-year period ranged from 42.9-135.8 and 2.4-20.4 per 100,000 population per year, respectively (Table 1). The highest difference in notification rate between Aboriginal people and non-Aboriginal people occurred in 2004, with a notification rate ratio of 53.1 and the lowest in 2007 with a notification rate ratio of 5.6 (Table 1).

A bimodal distribution of notifications was observed for non-Aboriginal age groups, with peaks in the 0-4 years age group (33.3% of cases, notification rate = 36.5 notifications per 100,000/year)) and the 30-34 year age group (9% of cases, notification rate = 8.7 notifications per 100,000/year) was observed. In contrast, there was a unimodal distribution of notifications for age groups in Aboriginal people, with 93.2% of cases reported in the 0-4 years age group (749.6 cases per100,000/year) (Fig. 1, Table 2). The notification rate ratio in

the 0-4 years age group in Aboriginal and non-Aboriginal people was 20.5 (Table 2). Within the 0-4 years age group, Aboriginal children ≤ 1 years of age had the highest average rate at 1,549 cases/100,000 population, followed by 2 year olds with a rate of 564 cases/100,000 population. Similarly, non-Aboriginal children in the 0-4 years age group with the highest number of notification rates were children ≤ 1 years of age and at 2 years of age with average rates of 41 cases/100,000 population.

Aboriginal people had higher notification rate ratios compared to non-Aboriginal people in the 45-49 (notification rate ratio = 4.8), 50-54 (notification rate ratio = 3.7), 55-59 (notification rate ratio = 5.8) and 60-64 (notification rate ratio = 4.9) age groups (Table 2). Male Aboriginal people had a higher notifications rate (95.3 per 100,000 population, notification rate ratio = 14.7) compared to male non-Aboriginal people (7.5 per 100,000 population). Similarly, notification rates among female Aboriginal people were higher (90.5 per 100,000 population, notification rate ratio = 12.1) compared to female non-Aboriginal people (7.5 per 100,000 population).

Rates of cryptosporidiosis notifications were higher among Aboriginal people compared to non-Aboriginal people across all PHUs, ranging from 11.9-246.6 per 100,000 population (Table 3). The remote regions (Goldfields, Kimberley and Pilbara), had the highest average number of cases notified among the Aboriginal people, particularly in the Kimberley, with an average of 38.5 cases notified each year between 2002-2012, at a rate of 246.6 per 100,000 per year (Table 3). Among non-Aboriginal people, the highest number of notifications was reported from the Metropolitan region (91.5 per year; notification rate = 5.6 per 100,000 per year), while the highest notification rate was in the Kimberley (notification rate = 51.2 per 100,000 per year (Table 3).

Hospitalisation data were available for 55% (1,268/2,320) of cases with recorded Aboriginal status, comprising 251 Aboriginal people and 1,017 non-Aboriginal people. A

total of 244 cases of cryptosporidiosis were hospitalised between 2002 and 2012. A significantly (P<0.01) higher proportion of Aboriginal people were hospitalised (53% 133/251) compared to non-Aboriginal people (10.8% 110/1,017). Children aged 0-4 years comprised the majority of both Aboriginal and non-Aboriginal cases hospitalised, at 89.5% (119/133) and 34.2% (38/111) respectively.

3.2 Molecular Epidemiology

Data for molecular analyses carried out were available for the specimens tested from April 2005 to January 2008 and from June 2010 to December 2012 (n = 324 cases), with Aboriginal status recorded in only 290 of these cases. In total, *C. hominis* was identified in 259/324 (79.9%) cases, *C. parvum* in 54 (16.6%) and *C. meleagridis* in 10 (3.1%) cases, with one case (0.3%) identified with a mixed infection of *C. meleagridis*, *Cryptosporidium* Mink genotype and an unknown *Cryptosporidium* genotype (Table 4). The latter two genotypes have been previously analysed in more depth (Ng-Hublin et al., 2013).

Of the cases identified with *C. hominis* (n = 259), 58.7% (152/259) were non-Aboriginal people, 32% (83/259) were Aboriginal people and the status of 9.3% were unknown (Table 4). However, Aboriginal people were 1.3 times more at risk (RR 1.3; 95% CI, 1.21-1.44, $p < 0.05$) than non-Aboriginal people of being infected with *C. hominis*. Only a small proportion of Aboriginal people (2.4%; 2/85) were diagnosed with *C. parvum,* with 97.6% (83/85) of cases diagnosed with *C. hominis* (Table 4). Non-Aboriginal people were 8.7 times more likely to be infected with *C. parvum* compared to Aboriginal people (RR 8.7; 95% CI, 2.16-35.16, $p < 0.05$). All cases infected with *C. meleagridis* were non-Aboriginal people. Thus, zoonotic *Cryptosporidium* (*C. parvum, C. meleagridis* and the mink genotype) were identified in 25.8% (53/205) of non-Aboriginal people.

The most common *C. hominis* subtype identified in Aboriginal people was the IdA15G1 subtype (59%; 49/83), followed by IgA17 (30.1%; 25/83) and IbA10G2 (8.4%; 7/83). In non-Aboriginal people, *C. hominis* IbA10G2 was the most common (61.8%; 94/152), followed by IgA17 (16.4%; 25/152) and IdA15G1 (15.1%; 23/152) (Table 4). Aboriginal people were 3.9 times more likely to be infected with the *C. hominis* IdA15G1 subtype (RR 3.90; 95% CI, 2.57-5.92, $p < 0.05$) and 1.8 times more likely to be infected with IgA17 (RR 1.8; 95%CI, 1.13-2.98, $p < 0.05$) compared to non-Aboriginal people. Compared to Aboriginal people, non-Aboriginal people were 7.2 times more likely to be infected with the IbA10G2 subtype (RR 7.24; 95%CI, 3.52-14.87, $p < 0.05$). The two most common *C. parvum* subtypes identified in non-Aboriginal people were the IIaA17G2R1 subtype (21%; 9/42) and IIaA18G3R1 (66.6%; 28/42).

The majority of typed cases from Aboriginal people came from remote areas, particularly the Kimberley. Of the 113 typed cases from remote areas, 70.8% (80/113) were from the Kimberley, and Aboriginal people consisted of 67.5% (54/80) of these. Very few typed Aboriginal cases came from metropolitan Perth (2% - 2/101) compared to Non-Aboriginal cases (98% - 99/101). Similarly, 87.1% (954/62) of non-Aboriginal cases came from rural areas compared to Aboriginal people (12.9% - 8/62).

Distribution of *gp60* subtypes indicated that in the remote regions of WA, the majority (60.8% - 45/74) of infections in Aboriginal people were attributed to the IdA15G1 subtype, followed by the IgA17 subtype (29.7% - 22/74), whereas in non-Aboriginal people, infection was attributed to the IbA10G2 subtype (46.2% - 18/39) and the IdA15G1 subtype (30.8% - 12/39) (Table 5). In the rural regions of WA, 37.5% (3/8) of Aboriginal people were infected with the IdA15G1 subtype, followed by the IbA10G2 and IgA17 subtypes in 25% (2/8) of cases each. Non-Aboriginal people were mainly infected with the IbA10G2 subtype, (22.2% - 12/54), followed by the IgA17 and IdA15G1 subtypes in 16.7% (9/54) and 14.8%

(8/54) of cases respectively (Table 5). In the Metropolitan area, the IbA10G2 subtype was identified in 63.3% (62/99) of non-Aboriginal people, followed by the *C. parvum* IIaA18G3R1 subtype (13.3% - 13/99) and IgA17 (11.1% - 11/99) (Table 5). Only two Aboriginal people were identified from the Metropolitan area and the IbA10G2 and IgA17 subtypes were identified in a case each (Table 5).

Molecular genotyping data were available for 168 hospitalised cases, for which Aboriginal status was known. Aboriginal people comprised 27/168 of cases, of which 63.0% (17/27) were hospitalised, whilst non-Aboriginal people comprised 141/168 of cases, of which 19.9% (28/141) were hospitalised. All 17 hospitalised Aboriginal people were infected with *C. hominis* and 16 of these were children (aged 0-4 years). The IdA15G1 subtype was identified in 10/17 cases, the IbA10G2 and IgA17 subtypes identified in three cases each and the IeA11G3T3 subtype identified in one case. *Cryptosporidium hominis* was detected in 15/28 hospitalised non-Aboriginal people, followed by *C. parvum* in 11/28 cases and *C. meleagridis* in two cases, one of which was a mixed infection with Mink genotype. The *C. hominis* IbA10G2 subtype was the most common (11/15), followed by the IdA15G1 subtype in two cases and the IfA12G1 subtype in one case. For cases infected with *C. parvum*, the IIaA18G3R1 subtype was the most common subtype, identified in eight cases, followed by the IIaA17G2R1 subtype in two cases and the IIaA15G2R1 subtype in one case.

4. Discussion

Rates of reported cryptosporidiosis in Australia are higher than many comparable developed countries (Lal et al., 2015a), with an average rate of 12.5 cases/100,000 population/year across Australia between 2001-2016 (NNDSS, 2016). However, the actual number of cryptosporidiosis cases in Australia and worldwide is likely to be much higher as

routine surveillance systems detect only a small fraction of the pathogen infections that occur in the community because: (1) it is estimated that less than 10% of individuals with gastroenteritis visit their local doctor, and of these, less than 10% have a faecal specimen collected (2) not all individuals presenting with gastroenteritis will have faecal samples tested for microorganisms and (3) identification of *Cryptosporidium* and other pathogens via microscopy lacks specificity and sensitivity) (cf. Ryan et al., 2017). For example, a national survey of gastroenteritis in Australia in 2002, suggested a ratio of about 500 community cases to one notified (Hall et al., 2006). A more recent study reported that in 2010 alone, cryptosporidiosis was estimated to be responsible for 195,495 cases of acute gastroenteritis (AGE) and 333 disability-adjusted life years (DALYs) in Australia (Gibney et al., 2014).

Under-reporting of cryptosporidiosis is even more pronounced in Aboriginal communities due to the difficulty they have accessing health services. For example, in 2008, the National Aboriginal and Torres Strait Islander Social Survey (NATSISS) found that about 26% of Indigenous people aged 15 and over living in non-remote areas, had difficulty accessing health services (AIHW, 2011); in contrast, only 2.6% of the general population had difficulty (ABS, 2012). The main reasons cited were long waiting times, services not being available when needed, difficulties with transport and health-care costs. Less commonly reported reasons included lack of engagement, fear of discrimination and poor treatment arising from previous experiences, and the lack of culturally appropriate services (AIHW, 2011).

The disparity of health status between Aboriginal people and non-Aboriginal people in Australia has been well documented, with diarrhoea caused by gastrointestinal parasites recognised as issues of major importance in Aboriginal populations (Gracey and Cullinane, 2003; Bramley et al., 2004; Holt et al., 2010; Hotez, 2014; Shield et al., 2015). As the majority of Aboriginal people reside in remote areas, the significantly higher

cryptosporidiosis notification rates in Aboriginal people compared to non-Aboriginal people (up to 50 times higher), especially in children 0-4 years of age, is likely due to factors associated with remote living such as poor socioeconomic status and household conditions, as well as limited access to nutritious food, potable drinking water, adequate sewage and solid waste disposals; all of which facilitate the faecal-oral transmission of *Cryptosporidium*, thus increasing the risk of infection (Bailie et al., 2004, Bailie et al., 2010, McDonald and Bailie, 2010, Becker et al., 2015).

Among both Aboriginal and non-Aboriginal people, the largest number of notifications occurred in children aged 0-4 years, in particular those aged 1 year or less, consistent with studies worldwide (Ryan et al., 2016; Kotloff et al., 2016). The bimodal age related pattern observed in non-Aboriginal cases is consistent with the general trend observed in previous studies in WA, other states in Australia, as well as in the US, Canada and UK (Laupland and Church, 2005; Ng et al., 2010b; Yoder and Beach, 2010; Chalmers et al., 2011; Kent et al., 2011; Waldron et al., 2011a). The high cryptosporidiosis rates, particularly in females in the 20-39 years age group, has been attributed to the occurrence of transmission between children and their caregivers (family members, childcare staff, household contact), possibly through child minding and nappy changing activities (Robertson et al., 2002; Hunter et al., 2004).

Aboriginal people comprise 30-40% of the population in rural and remote areas, in comparison to just 1.5% of the population in metropolitan areas, which may partly explain the increased rates of cryptosporidiosis cases among Aboriginal people in the remote regions of WA, particularly in the Kimberley. Notifications were highest during the Kimberley wet season (data not shown), which occurs from November to April (late spring to autumn), when there is flooding and swimming in natural water holes or catchment is a common practice, resulting in increased potential for transmission.

The predominantly anthroponotically transmitted *C. hominis* was the most common species identified among Aboriginal and non-Aboriginal people, suggesting person to person transmission. A higher proportion of zoonotic *Cryptosporidium* species were detected in non-Aboriginal cases (25.8% v 2.5%), with *C. parvum* and *C. meleagridis* identified mainly from the metropolitan area, but also from rural regions (e.g. South West and Great Southern), where there is greater density of livestock. Four different *C. parvum* subtypes were identified from cases in this region; the IIaA17G2R1 and IIaA18G3R1, which have previously been identified from cattle in the South West region (Ng et al., 2011) and the IIaA19G4R1 and IIaA21G2R1 subtype. There have been no previous reports of the *C. parvum* IIaA19G4R1 and IIaA21G2R1 in livestock in WA, although these subtypes have been identified from cattle in New South Wales (NSW) (Ng et al., 2008). These and the other *C. parvum* IIa subtypes identified in the present study, particularly the IIaA18G3R1 subtype, are common *C. parvum* subtypes identified in humans and cattle in Australia (Jex et al., 2007; Jex et al., 2008; Ng et al., 2008; O'Brien et al., 2008; Waldron et al., 2009; Ng et al., 2011; Ng et al., 2012). In the present study, the *C. parvum* IIdA15G1 subtype was identified in one case, which has been reported in buffalo from the Northern Territory (Zahedi et al., 2016a). As not all *C. parvum* and *C. meleagridis* infections are attributed to zoonotic transmission, it is difficult to deduce whether the source of these infections were zoonotic or anthroponotic, without carrying out extensive molecular genotyping of both humans and animals in the regions. However, the identification of these *C. parvum* genotypes in both humans and livestock (Xiao, 2010), indicates that animals may play a role in the dissemination of *Cryptosporidium* oocysts. Therefore, further investigation should be carried to determine the extent of zoonoses and the risk to public health as well as to catchment water quality in the area.

Aboriginal people were mainly infected with the *C. hominis* IdA15G1 subtype (59%) whereas non-Aboriginal people, were predominantly infected with the IbA10G2 subtype (61.8%). The IdA15G1 subtype was previously identified as the most common subtype in WA (Ng et al., 2010) and has also been identified in NSW and Victoria (VIC) (Jex et al., 2007; Ng et al., 2008). It has been responsible for waterborne outbreaks in the U.S. (Feng et al., 2014). The IbA10G2 subtype is recognised as a major cause of sporadic cryptosporidiosis in WA, VIC, South Australia (SA), and NSW (Jex et al., 2007; Jex et al., 2008; Waldron et al., 2009; Ng et al., 2010; Waldron et al., 2011a), is highly virulent and has been associated with numerous waterborne cryptosporidiosis outbreaks in Australia (Ng et al., 2010; Mayne et al., 2011; Waldron et al., 2011b; Ng-Hublin et al., 2013) and worldwide (cf. Li et al. 2013).

The identification of distinct *C. hominis gp60* subtypes circulating among Aboriginal and non-Aboriginal populations highlights the unique endemicity of cryptosporidiosis in Aboriginal people. Due to low numbers of cases from Aboriginal people genotyped from other regions in WA, it is difficult to ascertain from this study, whether this endemicity of the *C. hominis* IdA15G1 subtype is unique to the Kimberley area, as direct comparisons could not be made in the metropolitan or other rural or remote regions. This finding however, provides an avenue for more focussed public health attention, better tracking of sources of infection and implementation of intervention measures to prevent transmission of the disease and warrants further investigations for a better understanding of the *gp60* subtypes circulating among the populations and communities of Aboriginal and non-Aboriginal people in the Kimberley and other areas in WA.

Overall, the findings from the present study shows that the risk of cryptosporidiosis in Aboriginal people, especially those < 5 years of age is disproportionately greater than in non-Aboriginal people, with the risk of the disease increasing with remoteness in WA. The predominance of *C. hominis* infections in both Aboriginal and non-Aboriginal people

indicates that the main transmission route of the disease is person to person; however, infection with distinct subtypes within the population suggests that the source of infection and transmission may differ. Factors associated with remote living such as poverty, access to nutritious food and sanitation resources that may impede the transmission of *Cryptosporidium*, are likely the underlying factor for the disparities observed in the present study. More focussed attention on interventions for disease control that includes handwashing with soap and water, sanitation and hygiene promotion, are most likely to reduce diarrhoeal illness, especially in children (McDonald et al., 2008) and are currently being carried out by environmental health practitioners working in Aboriginal communities in remote and rural areas http://www.public.health.wa.gov.au/2/1395/2/aboriginal_health.pm. The importance of zoonotic transmission of cryptosporidiosis in WA however should not be discounted, particularly in light of the identification of *C. hominis* in kangaroos in Australia (Zhaedi et al., 2016b) and in cattle in New Zealand (Abeywardena et al., 2012). Further studies and molecular genotyping involving larger human, livestock and wildlife populations, particularly in livestock dense areas, as well as animals in Aboriginal communities, should be carried out to examine the extent of zoonoses in these communities.

Limitations of the present study include the use of passive surveillance data, which is subjected to under-reporting especially of asymptomatic cases (Lal et al., 2015b). Molecular data was also not available for every case analysed and specimens genotyped were sent from various pathology laboratories from different regions in WA, depending on whether adequate amount for analysis was available, which did not allow for standardisation of sample sizes for cross regional comparisons. However, the use of molecular data to complement existing surveillance data was one of the highlights of the present study, as it provided supporting evidence, and a better explanation and understanding of the surveillance data. Molecular surveillance should be routinely conducted for a better understanding of cryptosporidiosis

epidemiology in WA and across Australia, to allow for better detection and more targeted public health interventions.

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Fig. 1. Comparison of number of cryptosporidiosis cases notified between Aboriginal people and non-Aboriginal people by age groups.

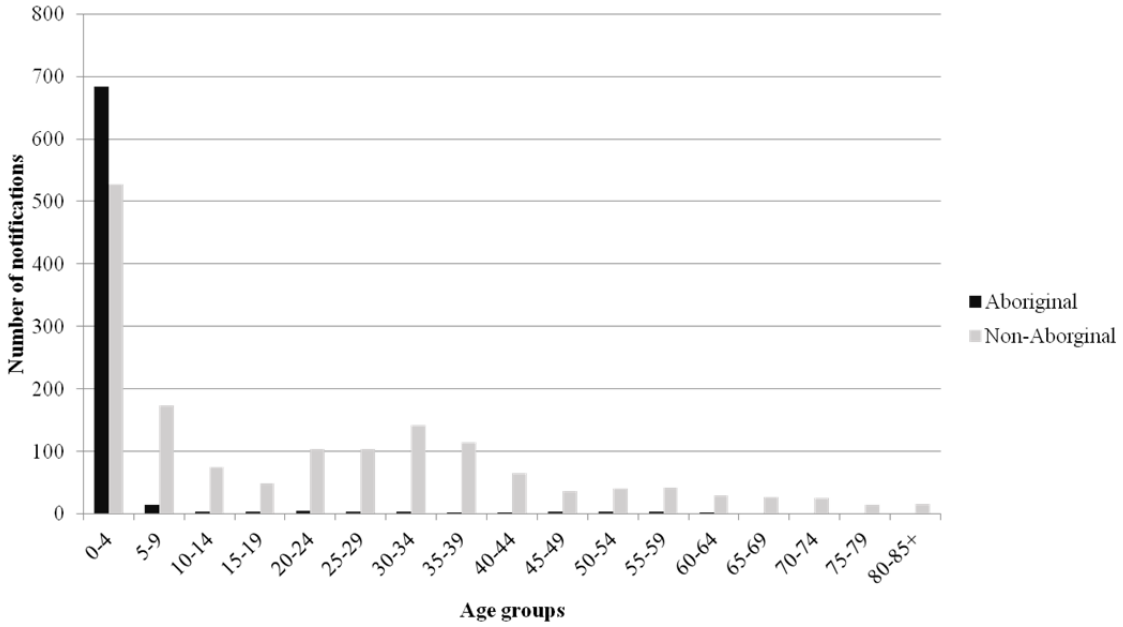

Fig. 1

Table 1.
The annual number, notification rate, NR (notifications per 100,000/year) and notification rate ratio (NRR) of cryptosporidiosis notification in Aboriginal and non-Aboriginal people in Western Australia (2002-2012).

Year	Aboriginal people		Non-Aboriginal people		NRR
	Number	NR	Numbers	NR	
2002	62	92.8	45	2.4	38.7
2003	92	135.8	130	6.9	19.7
2004	51	74.3	27	1.4	53.1
2005	93	133.6	67	3.4	39.3
2006	61	86.1	148	7.4	11.6
2007	82	114.2	414	20.4	5.6
2008	60	82.3	83	4	20.6
2009	66	89.4	113	5.2	17.2
2010	37	49.3	89	4	12.3
2011	97	127.8	343	15.1	8.5
2012	33	42.9	126	5.4	7.9
Total	**734**		**1,585**		

Table 2.
The age distribution, notification rate, NR (notifications per 100,00/year) and notification rate ratio (NRR) of cryptosporidiosis notification in Aboriginal and non-Aboriginal people in Western Australia (2002-2012).

Age Group	Aboriginal (Aborg)	Non-Aborginal (Non-Aborg)	NRR	
			Aborg:Non-Aborg	Non-Aborg: Aborg
0-4	749.6	36.5	20.5	0.0
5-9	15.5	12.2	1.3	0.8
10-14	3.2	5	0.6	1.6
15-19	3.6	3.1	1.2	0.9
20-24	7	6.3	1.1	0.9
25-29	6.4	6.3	1.0	1.0
30-34	5.2	8.7	0.6	1.7
35-39	3.7	6.8	0.5	1.8
40-44	4.3	3.8	1.1	0.9
45-49	10.1	2.1	4.8	0.2
50-54	9.6	2.6	3.7	0.3
55-59	17.9	3.1	5.8	0.2
60-64	13.3	2.7	4.9	0.2
65-69	0	3.2	0.0	-
70-74	0	3.7	0.0	-
75-79	0	2.6	0.0	-
80-85+	0	2.3	0.0	-

Table 3.
Average number of notifications and notification rates (NR) of cryptosporidiosis cases in each PHU from 2002-2012 by Aboriginal status.

PHUs	Aboriginal people		Non-Aboriginal people	
	Average no. of notifications (range)	NR (per 100,000)	Average no. of notifications (range)	NR (per 100,000)
Wheatbelt	1.6 (0-6)	44.9	6.9 (0-15)	9.7
Goldfields	5.6 (2-17)	96.8	6.2 (0-18)	12.5
Great Southern	0.36 (0-1)	17.1	5.2 (2-10)	9.3
Kimberley	38.5 (12-59)	246.6	9.5 (4-22)	51.2
Metropolitan	3.2 (0-9)	11.9	91.5 (9-319)	5.6
Midwest	4.9 (0-14)	67.9	6.2 (0-25)	11.1
Pilbara	11.3 (4-16)	151.8	5.5 (2-10)	13.9
South West	1.0 (0-3)	31.7	12.4 (6-30)	8.4

Table 4.

Cryptosporidium species and subtypes identified in Aboriginals and non-Aboriginals in WA.

	Aboriginal	Non-Aboriginal	Unknown	Total
C. hominis	**83**	**152**	**24**	**259**
IbA10G2	7	94	14	112
IdA15G1	49	23	4	75
IdA16	-	3	3	6
IdA17	-	1	-	1
IeA11G3T3	1	3	-	4
IfA12G1	1	3	-	4
IgA17	25	25	3	53
C. parvum	**2**	**42**	**10**	**54**
IIaA15G2R1	-	1	-	1
IIaA17G2R1	-	9	-	10
IIaA18G3R1	1	28	9	37
IIaA19G4R1	1	1	1	3
IIaA21G2R1	-	1	-	1
IIdA15G1	-	1	-	1
ND*	-	1	-	1
C. meleagridis	**-**	**11^**	**-**	**10**
Mink genotype	**-**	**1^**	**-**	**1**
Total	**85**	**205**	**34**	**324**

*ND – subtype not determined.

^Mixed infection – *C. meleagridis* and *Cryptosporidium* Mink genotype was identified from one case

Table 5.
Distribution of *C. hominis* and *C. parvum gp60* genotypes in remote, rural and metropolitan regions of WA by Aboriginality.

	REMOTE		RURAL		METROPOLITAN	
	Aboriginal	**Non-Aboriginal**	**Aboriginal**	**Non-Aboriginal**	**Aboriginal**	**Non-Aboriginal**
C. hominis						
IbA10G2	4	18	2	12	1	62
IdA15G1	45	12	3	8	-	3
IdA16	-	-	-	1	-	2
IdA17	-	-	-	-	-	1
IeA11G3T3	1	1	-	1	-	1
IfA12G1	1	2	-	-	-	1
IgA17	22	5	2	9	1	11
C. parvum						
IIaA15G2R1	-	-	-	1	-	-
IIaA17G2R1	-	1	-	5	-	3
IIaA18G3R1	-	-	1	15	-	13
IIaA19G4R1	1	-	-	1	-	-
IIaA21G2R1	-	-	-	1	-	-
IIdA15G1	-	-	-	-	-	1
Unknown	-	-	-	-	-	1
Total	**74**	**39**	**8**	**54**	**2**	**99**

Highlights

- 1st study to compare cryptosporidiosis in Aboriginal v non-Aboriginal communities
- Notification rates among Aboriginal people were up to 50 times higher
- Aboriginal people were mainly infected with the *C. hominis* IdA15G1 subtype
- Non-Aboriginal people were predominantly infected with the IbA10G2 subtype
- Unique endemicity of cryptosporidiosis in Aboriginal people