Review Article

Giardia lamblia in children and the child care setting: A review of the literature

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Social and workforce changes have resulted in young infants experiencing group care outside the family at a younger age, resulting in earlier exposure to infectious illnesses, at a time when they are immunologically inexperienced and therefore more susceptible. Not only is susceptibility increased, but exposure is likely to be greater in non-toilet-trained children who have yet to learn basic hygiene. In the child care context, diarrhoeal illness raises additional public health concerns, because of the potential for spread to other children and through families to the wider community. This review concentrates on describing *Giardia lamblia*, its microbiological identification and clinical features, evidence regarding the natural history of infection with this protozoan, and the prevalence of *Giardia* in surveys undertaken in Australian children. Prospective studies of diarrhoeal illness carried out in children, with particular emphasis on child care settings, are examined to give perspective to the possible role for *Giardia lamblia* as a pathogen in the child care setting.

GIARDIA LAMBLIA (G. INTESTINALIS)

Giardia are flagellated protozoa, which exist in two forms. The trophozoite is the motile flagellate form that derives from the cyst after it has been acted upon by the acid environment of the stomach. The trophozoite attaches firmly to the mucosa of the small intestine, and since it divides by binary fission every 5 h or so, it may rapidly establish itself in great numbers.[1,2] Trophozoites are fragile, found only in diarrhoeic stools, and do not survive outside the body of the host. The cyst is the normal form found in the stool and, although rapidly destroyed by drying, may survive in wet conditions and remain infective for months.[1–3]

Followings its identification, *Giardia* was thought to be a commensal, and an association with clinical disease was not made until many years later. Infectivity appears dependent upon a number of parasite and host factors. There is no rigid host specificity,[3] and circumstantial and epidemiological evidence suggests that humans can acquire infection from other animals.[4–6] *Giardia* may be spread by faeces from a carrier,

sewage contaminating a water supply or by hand-to-mouth transfer of cysts. Standard concentrations of chlorine in public water supplies fail to destroy *Giardia* cysts, and conventional water testing commonly does not detect small degrees of contamination.[7,8] *Giardia lamblia* occurs worldwide, and is associated with overcrowding, poor sanitary conditions and poor water quality control, although in the developed world most cases are probably acquired by person-to-person transmission.[9]

In experiments in which human volunteers were infected, Rentdorff showed that infection could occur following the ingestion of as few as 10 cysts, and that 100 *Giardia* organisms caused *Giardia* infection in 50% of recipients.[10,11] These studies also demonstrated that infection may be asymptomatic, or cause short-lived diarrhoeal illness. Most subjects (86%) eradicated the parasites spontaneously, only two out of 21 infested volunteers continuing to excrete cysts in their stools after 3 months. Sagi *et al.* found that infestation disappeared spontaneously in six out of seven positive children re-examined after a year,[12] but some asymptomatic carriers may excrete the cysts for many years.[13]

Longitudinal data on *Giardia* excretion indicate the natural history in children commonly exposed to infection. In 45 Guatemalan children followed from birth through 3 years, all children acquired at least one *Giardia* infection, and more than 40% of infections lasted 2–6 weeks or more.[14] Prevalence to incidence ratios were low in the first 9 months of life indicating that *Giardia* events were short-lived, but increased substantially during the second and third years of life, consistent with more persistent infections. Simultaneous infection with other enteropathogens was a feature of that environment, which may in part account for the observation that weight–velocity growth was lower in the second year in *Giardia*-positive children. Another longitudinal investigation of the health effects of *Giardia* was undertaken in Egypt, where 42 2–4 year olds had stools examined weekly for 6 months. All but one child acquired *Giardia* during the study.[15] Forty-two per cent of specimens examined were *Giardia*-positive, and the mean duration of excretion in *Giardia*-positive children was 7½ weeks, with a range of 1–17 weeks. Clinical symptoms of illness occurred frequently within 1 month before or after *Giardia* excretion, but no statistical association could be demonstrated.

In a waterborne outbreak of *Giardia lamblia*, the majority of infections were asymptomatic and ran a self-limited course without treatment.[16] Data from other waterborne outbreaks of giardiasis support evidence from animal experiments that some degree of immunity to *Giardia* can be acquired.[17,18] In a mouse

model of giardiasis, recovery from infection with *Giardia muris* is accompanied by the acquisition of prolonged resistance to reinfection with the same parasites.[19] The effect of giving anti-*Giardia* chemotherapy on development of immunity is unknown.

CASE DEFINITION OF GIARDIASIS

A clinical description of giardiasis is 'an illness caused by the protozoan *Giardia lamblia* and characterized by diarrhoea, abdominal cramps, bloating, weight loss, or malabsorption'.[20] The clinical case definition necessitates laboratory confirmation of *Giardia* in the stools: a person is confirmed symptomatic when one or more of the symptoms described above are present, and asymptomatic when *Giardia* is present without any of the above symptoms.[20]

Spectrum of illness with *Giardia lamblia*

Most persons who acquire *Giardia* infections remain asymptomatic[2,3,8,13-16,21] However, *Giardia lamblia* can cause a range of illnesses ranging from acute, self-limiting diarrhoea with or without abdominal discomfort and vague upper gastrointestinal symptoms to chronic, intermittent diarrhoea with distension and flatulence, or severe malabsorption. Infected patients may remain symptomatic for years.[22] Children with chronic giardiasis may present with a coeliac-like syndrome, characterized by anorexia, growth retardation, weight loss, abdominal pain, chronic diarrhoea, wasted musculature, abdominal distension, anaemia and steatorrhoea.[9,23] Pathological findings on small bowel biopsy, however, do not correlate well with the disease state.[24] Malabsorption occurring in children with *Giardia*, which responds rapidly to anti-parasitic medication, is one extreme of the spectrum of infection.

Pathological changes might depend on the presence of large numbers of the protozoa or on host factors, such as lowered immunity (systemic or in the gut) or disturbances of gastrointestinal function and/or flora from other causes. The factors responsible for converting an asymptomatic into symptomatic *Giardia* infection are unknown, but may be inherent in the host and not in the organism.[3] Toxigenicity and invasiveness are two qualities, possessed to a limited degree by *Giardia*, which are usually counterbalanced by the immune mechanisms of the normal host. A change in the host may tip the host–parasite balance in favour of the parasite, resulting in a heavy infection that may include the expression of toxicity or invasiveness.[3] Intestinal damage by another enteropathogen, may cause *Giardia* to appear as the primary pathogen where asymptomatic *Giardia* infection has pre-existed.

Giardiasis is more frequent in children than in adults.[9,25] Males have been reported to be infected more commonly than females,[26-29] and there is an increased incidence in homosexual men.[30,31] Since infants and young children have the highest age-specific incidence of diarrhoea from other infections (such as rotavirus, *Salmonella, Campylobacter*), they may be more likely to have *Giardia lamblia* identified even if it is a bystander.

Laboratory identification

The laboratory criterion for diagnosis is demonstration of *Giardia lamblia* cysts in stool; demonstration of *Giardia lamblia* trophozoites in stool, duodenal fluid, or small bowel biopsy; or

demonstration of *Giardia lamblia* antigen in stool by a specific test such as enzyme-linked immunosorbent assays (ELISA) which have a sensitivity of 92–95% and a specificity of 95–98%.[32-34] However, their use at present represents an additional cost given the need to prepare concentrated specimens and perform microscopic examination for other protozoa.[35] These considerations are particularly important in day care centres (DCC) where there may be multiple enteropathogens and a high background prevalence of *Giardia*.

Intermittent shedding of *Giardia lamblia* in faeces occurs. Rentdorff found stool specimens negative for *Giardia* for periods lasting 20–30 days in some infected volunteers,[10] a phenomenon confirmed by many others. Danciger and Lopez described three patterns of parasite excretion: (i) high excreters, with the parasite present and abundant in nearly all stools; (ii) low excreters, with the parasite undetectable in the stool for long periods and scanty when present; and (iii) mixed excreters, where periods of high excretion lasting 1–3 weeks alternated with generally shorter periods of low excretion.[13]

The ability to identify cysts in the stool is increased by use of a concentration technique to supplement direct faecal smears. Traditionally, three separate stool examinations have been regarded as necessary to exclude giardiasis. Wolfe reported that of persons with *Giardia* infection identified by stool examination, 76% were positive on the first specimen, 90% were identified with two specimens and 97.6% were found with three specimens.[36] Others have reported a higher pick-up rate on a single stool: 86%,[37] and 82%.[38] Others have reported that cysts were not found in the faeces of 10–50% of infected symptomatic individuals identified by more sensitive techniques such as intestinal biopsy.[39,40]

The approach to microbiological investigation of diarrhoeal illness will depend upon the clinical context, and investigation for protozoal infections is warranted when there is a convincing clinical presentation that includes diarrhoea persisting for greater than 1 week. The diagnosis of the majority of clinically significant infections is made on the first faecal examination, so it has been suggested that the ordering of a second or third faecal specimen for examination for parasites should only be undertaken when the result of the first examination is negative, the patient's symptoms persist and other causes of diarrhoea have not been found.[41,42] One stool yields the correct diagnosis in 93.9–95.6% of cases. A change in practice to initially ordering a single stool examination would result in a cost saving of perhaps 50% without compromising the standard of care,[41] although for a few children this change may require an extra medical consultation. With a very high index of clinical suspicion (persistent diarrhoea, malabsorption, malodorous diarrhoea, bloating, crampy abdominal pain, failure-to-thrive or weight loss, unexplained gastrointestinal symptoms, history of contact or travel to a highly endemic area), it is reasonable to pursue the diagnosis with extra stool examinations and, in the absence of cysts or trophozoites in the faeces, by undertaking duodenal aspirates, performing the 'string' test, or even duodenal biopsy for severe and persistent symptoms. In practice, therapeutic trial is often undertaken without pursuing microbiological diagnosis.

When investigating clusters of diarrhoeal illness within the day care setting, a policy of collecting single stool specimens must be balanced against the delay and difficulty in receiving and processing stool specimens, and the urgency for and value of complete outbreak investigation in order to achieve control. Searching for asymptomatic excretors of infectious agents such as *Giardia* is not warranted.

PREVALENCE OF *GIARDIA LAMBLIA* IN AUSTRALIAN CHILDREN

Relatively few studies have been published on prevalence rates of *Giardia* in the Australian population, and those identified are summarized in Table 1.[43-52] Prevalence rates range between 2 and 46%, but a number of common findings emerge from these population studies: (i) *Giardia* carriage is generally asymptomatic and is not associated with any increase in gastrointestinal symptoms; and (ii) it is most common in the 1–5 year age group and is more prevalent in children who attend preschool group care.

There are difficulties in interpreting these reports of 'outbreaks' and their management. For example, Cameron and Elliot reported two outbreaks of *Giardia* occurring in child care centres in Adelaide.[46] Few details of symptoms or of the microbiological investigation were given, yet the mass administration of tinidazole was used as an adjunct to control in one centre. In the second centre (60 children, 13 staff), microscopy of faecal specimens revealed an infection rate of 33%. Details of how many stool specimens were provided were not given, half the cases were asymptomatic at the time, and details of symptoms in the non-infected group were not reported. In their investigation of a presumed epidemic of giardiasis in Coffs Harbour, Walker *et al.* noted the propensity for a diagnosis of *Giardia* based upon symptoms, without laboratory confirmation.[49] A bacterial or viral pathogen was identified in 18% of those provisionally diagnosed as having giardiasis. Their investigation established a prevalence of *Giardia* (6%) that was comparable to that of 250 asymptomatic preschoolers in Sydney (6.8%).[50]

GIARDIASIS AND DIARRHOEAL ILLNESS IN CHILDCARE CENTRES

Illness caused by *Giardia* occurring in DCC must be viewed within the broader context of gastrointestinal infections. Appreciable discrepancies in the incidence of diarrhoeal illness have been found between DCC of similar composition within the same study and demographic area, although there is a

Table 1 Prevalence and incidence studies of *Giardia lamblia* in Australian children

Authors	Study design	Place	No. children	Age	Infected (%)	Comments
Willis[43]	Prevalence survey	Townsville, Qld	96	—	33	High prevalence of protozoal infections including *Giardia*
Court and Stanton[44]	Prevalence survey	Three institutions in Victoria	148	—	21	No significant difference in incidence of symptoms (abnormal bowel habits, diarrhoea or abdominal signs including pain and distension) between infected and non-infected children
Boreham *et al.*[45]	Random sample, Prevalence survey	Logan, SE Qld		<11 years	5.7 2.0	No correlation between the sex or age of the children, symptoms of diarrhoea or socio-economic conditions
Cameron and Elliot[46]	Microbiological investigation of outbreak	Child care centres Adelaide, SA, 1 2	55 25 ?	<6 years Staff	21.8 8.0 33	No details of symptoms, but mass administration of tinidazole used as an adjunct to control Half the cases were asymptomatic. No details given of symptoms in non-infected group
Boreham and Shepherd[47]	Microbiological investigation following a case	Child care centre Brisbane, Qld	71	<6 years Toddlers	19.7 46.2	
Boreham and Phillips[48]	Prevalence study, Random survey of 680 dwellings	Mt Isa, Qld		Community 1–5 years	4.5 12.0	Most infected persons were asymptomatic Children more likely than adults to have symptoms consistent with giardiasis No difference between infected and non-infected group for presence of gastrointestinal symptoms (abdominal cramps, unusual fatigue, bloating, nausea, foul-smelling stools, weight loss, loss of appetite, fever) 1–5 year olds who attended school or preschool childcare centre were significantly more likely to have *Giardia*
Walker *et al.*[49]	'Epidemic' investigation Community prevalence survey	Coffs, Harbour, NSW			6	Noted the propensity for *Giardia* to be diagnosed on the basis of symptoms, without laboratory confirmation A bacterial or viral pathogen was identified in 18% of those provisionally diagnosed as giardiasis No correlation between *Giardia* cysts or trophozoites and symptoms
Walker *et al.*[50]	Prevalence survey	Sydney, NSW	250	Pre-schoolers	6.8	
Grimmond *et al.*[51]	Prevalence survey	Adelaide, SA	178	≤6 years	10.7	All carriers were 1–4 years No significant association between *Giardia* carriage and gastrointestinal symptoms or stool consistency Urban Aboriginals had no greater prevalence than white children unless they had recently travelled to a rural Aboriginal settlement or had contact with rural visitors
Gill and Jones[52]	Prevalence survey Rural Aborigines	Rural WA		<6 years	26.3	

considerably higher incidence in children under 3 years of age.[53] Factors repeatedly shown to be associated with a higher risk of acquiring infectious disease in DCC have been: (i) the ratio of personnel to children; (ii) the more hours each day the centre is open, as well as the more days each week; (iii) whether the centre accepts 'drop-ins' for occasional care; (iv) admission of pre-toilet-trained children; (v) large enrolment; (vii) attendance of children from low income families; and (vii) staff with responsibilities for food preparation as well as care of children.[53-56]

In 1977, Black et al. reported evidence of person-to-person transmission of giardiasis in three DCC in Atlanta.[37] The overall prevalence of infection with *Giardia* in two DCC not experiencing an outbreak of diarrhoea was 29 and 38%, respectively, in infants aged six months to 3.5 years compared to 2% in the general population. Further evidence of person-to-person transmission in two Toronto DCC followed an investigation of an 'outbreak', with prevalences of *Giardia* of 39 and 17% in children in the two nurseries.[57] Household contacts had infection rates of 7 and 23%, and the infected children and household contacts on whom information could be obtained were symptomatic in 26 and 30% of cases, respectively.

Following these cross-sectional studies, Pickering et al. undertook a landmark prospective study of children attending 20 DCC, aimed at determining the occurrence, causes and transmission of gastroenteritis among children, staff and family members during a 19 month period.[58] Microbiological processing included use of selective media, and infection with rotavirus was ascertained by the use of electron microscopy. The presence of *Giardia lamblia* cysts or trophozoites in a child with diarrhoea was taken to incriminate *Giardia* as the causative organism. The authors identified an enteropathogen in only 3 of the 68 single cases of diarrhoea that occurred, although an enteropathogen was identified in all 15 outbreaks. *Shigella* was the only pathogen identified in five outbreaks, rotavirus in two, and *Giardia lamblia* in one. In the remaining seven outbreaks, multiple enteropathogens were identified (four with rotavirus and *Giardia*; three with other multiple pathogens). Rotavirus and *Giardia* occurred only in children less than 3 years of age; shigellosis occurred at all ages. In only one of the single cases of diarrhoea was there secondary spread (to one of 175 family members = 0.6%), whereas in 'outbreaks' the occurrence of diarrhoeal illness in families whose DCC child had shigellosis was 26%, for *Giardia* enteritis it was 17%, and for rotavirus infection it was 15%. The average duration of outbreaks was 2.4 weeks; this duration was considered in part due to inadequate exclusion of ill children.[58] Parents of children with diarrhoea would often deny that their children were ill, resulting in the continued attendance of sick children at DCC; there was an absence of centre policies regarding the exclusion of ill children and their eventual re-admission, paralleling the absence of State regulations. The authors concluded that DCC might play an important part in the epidemiology and transmission of gastroenteritis in the United States.[58] For the majority of the diarrhoeal episodes, aetiology remained undetermined and was attributed to viruses other than rotavirus. Viruses subsequently shown to be associated with childhood diarrhoea but which were not tested for in that study were human caliciviruses, enteric adenoviruses (types 40 and 41), astroviruses, coronaviruses, and unclassified, small, round viruses.[59]

Another prospective study within DCC identified enteric pathogens in only 20% of cases of diarrhoea.[60] *Giardia lamblia*, rotavirus and *Campylobacter jejuni* were the most common pathogens, with *Giardia* found in 19% of asymptomatic child

contacts of symptomatic infected children. More illness occurred in children with shorter enrolment, in non-toilet-trained children, and in centres with poor hygiene and child-handling practices. The incidence of diarrhoea in infants and children attending DCC was significantly higher than in children using family day care homes or in home care.[61]

An evaluation of *Giardia lamblia* occurrence in DCC combined two prevalence studies of 600 children enrolled in 30 centres, with an 18 month longitudinal study of 82 children in one centre.[62] The prevalence studies found *Giardia* cysts in 72 (21%) and 67 (26%) children, and trophozoites in 15 (4%) and 8 (3%) of those who provided stool specimens. Enteric symptoms reported by a parent or teacher did not differ significantly when groups of cyst excreters, cyst and trophozoite excreters, and non-excreters were compared. None of the three children assessed as underweight on World Health Organization international standards was *Giardia* positive. The high prevalence of infection was attributed to the susceptibility of young children to gut colonization, and the close-clustering that occurs in child care centres with its opportunities for person-to-person transmission.[62]

The high prevalence and incidence of giardiasis in children in day care and the lack of association of parasite excretion with symptoms has been borne out by many other studies in young children, particularly those in group care. A number of studies are summarized in Table 2 and support the following conclusions: (i) *Giardia lamblia* infection is prevalent in children cared for within DCC, with a peak incidence in the 1–3 year age group; (ii) the appearance of *Giardia* cysts or trophozoites in the stools is not always associated with gastrointestinal symptoms, nor is their continued passage; (iii) some children continue to excrete *Giardia* for prolonged periods; and (iv) there is no evidence of impaired nutritional status as judged by height, weight or haemoglobin level.[62-66] The variability in the effects of giardiasis is unexplained, but heterogeneity of strains could contribute to this.[67]

Concern has been raised about the spread of *Giardia* from infected children to parents and household contacts; this has been considered a possible indication for early recognition and treatment.[68] Polis et al. reported an association between the presence of *Giardia lamblia* in stools and clinical symptoms,[68] but the study was flawed by a poor response rate to the questionnaire so may have been unrepresentative. Even more importantly, stool examination did not include examination for other enteropathogens, so infection with *Giardia* may have just been a sign of poor hygiene practice and overall risk for infection with agents that cause gastrointestinal symptoms.

TREATMENT OF GIARDIAL INFECTIONS

Children in DCC are exposed to a higher risk of infection, and there is no likelihood that protection against *Giardia lamblia* will be afforded in the near future by the development of a safe and effective vaccine.[69] Nor is there oral medication that can be taken to prevent infection from ingested *Giardia* cysts.[3,70]

As with any acute infectious diarrhoea, for those infants who acquire *Giardia* the most essential treatment is adequate hydration. Antimicrobial therapy for giardiasis is of established benefit,[71] and the standard treatment in Australia is metronidazole (Flagyl, Mayand Baker; Metrozine, Searle, Sydney, NSW, Australia; Metrogyl, Alphapharm, Sydney, NSW, Australia), or

Table 2 Prevalence and incidence of *Giardia lamblia* in children attending day care centres

Authors	Study design	No. children	Age	% Infected Cysts (Trophozoites)		Comments
Black et al.[37]	Prevalence studies					
	1 'Outbreak' DCC	48	1–3.5 years	54		Overlapping epidemic of shigellosis (43% of children had *S. sonnei*)
	2 DCC	8	<3.5 years	38		Prevalence of *Giardia* significantly higher in children in day care
	3 DCC	28	<3.5 years	29		*Giardia* infection associated with flatulence and diarrhoea, especially if lasts >10 days
	Not in day care	42	<3.5 years	2		No recent history of diarrhoea in positives
Harter et al.[27]	Prevalence survey 2 State Counties	518	1–3 years	7		No difference in histories of diarrhoea in *Giardia* positive and negative cases
						Infection unrelated to domestic water source, DCC attendance, parental occupation
						Risk factors were drinking untreated surface water and ≥2 sibs aged 3–10 years
Sealy and Schuman[38]	Prevalence studies	1731				
1971	Countrywide		Grades 1–3			
	Whites			4		
	Blacks			6		
1980	Survey					
	Whites			11		Disproportionate use of DCC by whites
	Blacks			2		Children <2 years had a quarter the prevalence of 2–3 year olds
1981	White		DCC attendees	26		Acute or recurrent symptoms were reported for <5% of children
						None of these children had *Giardia* positive stools
1981			Entering 1st graders	10		Relative risk 6.7 for *Giardia* if they had been attending day care
Sagi et al.[12]	Prevalence survey	77	1–3 years	30		
	Single DCC	23	<12 months	4		*Giardia* positive children tended to have higher weight and height percentiles
	Three stools	56	1–2 years	26		
		29	2–3 years	34		No increase in GI symptoms in positive children
Pickering et al.[56]	Prevalence studies	600	<6 years			
	1	(30 DCC)		21	(4)	No correlation between frequency of recent diarrhoeal episodes and finding *Giardia*
	2			26	(3)	Cysts were more frequent in 13- to 30-month old children than infants <12 months (*P*<0.001)
						Children attending DCC >3 months more likely to be excreting *Giardia*
	Longitudinal study Weekly stool specimens	82 (1 DCC)	<6 years	33		12 children had *Giardia* cysts in a weekly stool specimen for a mean of 6.2±1.2 months, trophozoites in a weekly stool specimen for a mean of 3.3±1.2 months
						Enteric symptoms and nutritional status not significantly different between infected and non-infected children
Woo et al.[26]	Prevalence Surveys					
	DCC 1	97	2–5 years	8		No clinical signs of infection
	DCC 2	147	3–5 years	6		No clinical signs of infection
Grimmond et al.[51]	Prevalence on single stool	178	DCC attendees ≤6 years	11		No significant association between *Giardia* carriage and GI symptoms
	Aboriginal			8		No significant association between stool consistency and carriage of cysts
	Non-Aboriginal			34		All cyst excreters were 1–4 years old
Ish-Horowitz et al.[63]	Prospective study Single DCC;	83	DCC attendees	37		Infection was asymptomatic; usually associated with prolonged carriage
	12 months Monthly stools; weekly questionnaire		3 months–3 years			No significant differences for height, weight or mean haemoglobin but *Giardia* positive children tended to have fewer GI and respiratory symptoms

Table 2 continued

Authors	Study design	No. children	Age	% Infected Cysts (Trophozoites)	Comments
Novotny et al.[64]	Prevalence survey		Toddlers		Infection was not associated with symptoms
	DCC	236		16	Risk factors for those attending DCC were:-
	Home care	79		9	increasing duration of attendance
					time per week attending DCC
					low family income
					large family size
Rauch et al.[65]	15 month longitudinal study 1 DCC Weekly stools	82	0–24 months	33	*Giardia* infections observed in 14 of the 15 months of the study
					Rotavirus infection occurred in 45% over the same time period
					Range of 0–23% of children infected with *Giardia* in any one month
					6/48 (12%) of episodes of *Giardia* infection associated with symptoms
					Symptoms lasted 1 day to 7 weeks.
					No correlation between frequency of recent diarrhoeal episodes and detection of *Giardia*
Varga and Delage[66]	Prevalence survey 1 DCC	75	6–65 months	23	No difference in height, weight, skinfold thickness or GI symptoms between *Giardia* positive and
	Three stool specimens	10	6–18 months	10	negative children
	Positives	14	19–30 months	50	9/17 (53%) of those initially positive were still
	repeated at	14	31–42 months	21	excreting cysts 6 months later
	6 months	19	43–54 months	17	
		19	55–65 months	16	

GI, gastrointestinal; DCC, day care centre.

tinidazole (Fasigyn, Pfizer, Sydney, NSW, Australia).[72] Metronidazole is also effective in treatment of amoebiasis, trichomonas, *Clostridium difficile* and anaerobes. The imidazole derivatives have a mildly unpleasant metallic after-taste and can cause other side effects, especially as the duration of treatment increases. Efficacy of treatment for giardiasis varies from 50% for single-dose regimens, up to 97% for 7–10 days of therapy. The potential for side effects, the lack of complete efficacy in eliminating infection and the inadvisability of treating pregnant women (especially if asymptomatic) with metronidazole,[73] must be borne in mind in any attempt to eliminate *Giardia* from the DCC setting.

Management of *Giardia* in day care attendees

There is little argument about the need for treatment of symptomatic children, found on stool examination to have *Giardia* present, although even in this circumstance *Giardia* may be only a bystander or secondary pathogen following mucosal damage inflicted by another less readily identified enteropathogen. However, the treatment of asymptomatic children with *Giardia* who attend DCC is controversial. Some have argued for case-finding and treatment of all asymptomatic carriers within a closed community for effective public health control purposes.[9,39,47,53,69] However, as other aetiologic agents causing diarrhoea have been documented, and the evidence from prospective studies shows a lack of adverse outcomes of asymptomatic carriage of *Giardia*,[12,14–16,63–66] case-finding and treatment of asymptomatic carriers in DCC appears unwarranted. Control of diarrhoeal illness may be difficult to achieve even when measures that include treatment and isol-

ation of both symptomatic and asymptomatic carriers are implemented.[74,75]

The conclusions of many investigators who have addressed the issue of treatment of asymptomatic carriers based upon the data from their own and other studies are as follows: (i) there is no evidence to support the routine treatment or exclusion from DCC of children who are asymptomatic carriers of *Giardia lamblia*, or of routine testing of asymptomatic contacts of symptomatic infected children;[64,65] and (ii) complete eradication of the parasite in this setting is impractical because of the failure to detect all carriers on stool examination, treatment failures from currently available anti-parasitic drugs, poor tolerance of these agents, problems with compliance, cost of the medication, concerns about the safety of the medications and the constant potential for reinfection. In view of the lack of adverse consequences associated with *Giardia* carriage, treatment of asymptomatic infections is not justified. However, parents of stool-positive children should seek medical advice in the event of prolonged diarrhoea, and contacts of children with confirmed giardiasis need only seek medical advice if diarrhoea, symptoms of malabsorption or failure to thrive develop.[58]

A prospective controlled trial of three different strategies for the control of *Giardia lamblia* randomized 31 centres to three interventions;[76] from rigorous exclusion and treatment for symptomatic and asymptomatic infections to no exclusion applied, with only symptomatic children treated and followed up in the centre. Following the intervention, prevalences declined in all groups, but less dramatically in the no exclusion group at 1 month, although by 6 months *Giardia* prevalences between intervention groups were not significantly different. Children newly admitted to the centres during the intervention period had

a *Giardia* prevalence of 10.6%, and accounted for approximately 40% of *Giardia* infection present at 6 months. The authors concluded that the stricter intervention policy resulted in greater cost in terms of child day care and parents' work days lost, but not in significantly better control of Giardia infections in the day care environment, and that stricter policies might result in children being moved to different centres.

RECOMMENDATIONS

Giardia infection is not a clinical diagnosis and, although Giardia has a high prevalence in the childcare setting, it is not usually associated with illness. What then is a reasonable approach to diagnosis and management of diarrhoeal illness occurring in a child who attends a DCC? Both the severity and duration of symptoms will influence a decision to investigate. Trial of metronidazole should not be used to incriminate *Giardia* in the absence of a microbiological diagnosis, since an apparent response may be the result of the self-limiting nature of most diarrhoeal illness or, occasionally, the effectiveness of anti-parasitic drugs in treating other pathogens. Repeated and inappropriate anti-protozoal treatment should be avoided. Unless there is a high index of clinical suspicion, there is little value in pursuing a diagnosis of *Giardia* by ordering multiple stool specimens. Where *Giardia* is identified, it should be treated in a child who is experiencing gastrointestinal illness, but not in asymptomatic excretors, and children need not be excluded once they are well and have stools of normal consistency. There is no place for active case-finding in DCC in an attempt to eliminate giardial carriage from asymptomatic contacts.

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