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Parasite Body Size and Interspecific Variation in Levels of Aggregation among Nematodes

Robert Poulin and Serge Morand*, Department of Zoology, University of Otago, P.O. Box 56, Dunedin, New Zealand; and *Centre de Biologie et d'Ecologie tropicale et méditerranéenne, Laboratoire de Biologie Animale (UMR 5555 CNRS), Université de Perpignan, 66860 Perpignan, France

ABSTRACT: The aggregation of parasites among individual hosts is one of the best documented features of parasite populations; we still do not know, however, why certain parasite species are more highly aggregated than other, related species. Here we search for a general explanation of interspecific variation in aggregation levels, based on the relationship between parasite body size and fecundity, transmission success, and intensity-dependent population regulation. We test the prediction that larger-bodied parasite species are more weakly aggregated than smaller-bodied related species, in a comparative analysis across parasitic nematode species. Across species, the variance-to-mean abundance ratio correlated negatively and significantly with nematode body sizes, as predicted. All other tests, however, including the more robust analyses controlling for phylogenetic influences, failed to support this result. This is mainly because the variance in infection levels is almost completely explained by mean parasite abundance. For this reason, it may prove difficult to identify a general biological explanation for interspecific variability in aggregation levels among parasites.

Aggregation is a ubiquitous characteristic of natural populations of helminth and arthropod parasites (Crofton, 1971; Anderson and Gordon, 1982; Dobson and Merenlender, 1991; Shaw and Dobson, 1995). The processes likely to generate aggregated distributions of parasites include heterogeneity among hosts in susceptibility to parasites and temporal or spatial clumping of parasite infective stages (Anderson and Gordon, 1982; Poulin, 1998). Levels of aggregation vary among parasite species, however, and this variation remains poorly understood. A universal explanation of why certain parasite species are more intensely aggregated than other related species would be very useful to epidemiologists because the level of aggregation is a key parameter in the population dynamics of host–parasite interactions (Anderson and May, 1978, 1979; May and Anderson, 1978, 1979).

The only comparative study to examine interspecific variation in aggregation levels among parasites has been that of Shaw and Dobson (1995). On a log–log scale, the variance in parasite abundance is expected to relate linearly with the mean abundance among parasite populations of the same or different species, with a slope greater than 1 indicating aggregation (Taylor, 1961). Across published data from a large number of parasite species, Shaw and Dobson (1995) found a strong linear relationship (slope = 1.55) between the variance and mean abundance, with 87% of the spread in variance explained by mean abundance. In other words, for a given mean abundance, there are constraints on the variance in numbers of parasites per host and hence on levels of aggregation. Shaw and Dobson (1995) attempted, with limited success, to account for the remaining 13% of the variation in log variance not explained by log mean abundance by identifying taxonomic or ecological groups of parasites with consistently higher or lower levels of aggregations. Here, we test for a general, biological explanation of interspecific variation in aggregation unaccounted for by mean abundance, by seeing whether helminth parasite body size is a predictor of aggregation levels.

To understand how parasite body size may relate with aggregation, it is useful to consider the graphical shape of an aggregated distribution. Aggregation is best characterized by the negative binomial distribution (Crofton, 1971; Shaw et al., 1998), whereas the Poisson distribution describes a random dispersion. The main differences between these 2 distributions are that the negative binomial distribution has a higher intercept with the y-axis and a longer tail along the x-axis than the Poisson distribution. In biological terms, this means the negative binomial distribution is characterized by a lower prevalence, i.e., more uninfected hosts (see Poulin, 1993), and more heavily parasitized hosts than the Poisson distribution.

How could parasite body size influence these 2 features of the distribution in ways that either increase or decrease aggregation? First, there is comparative evidence that helminth body size correlates positively with prevalence (Poulin, 1999). This can be explained by the fact that larger helminth species are

more fecund than smaller ones (Skorping et al., 1991; Morand, 1996a; Poulin, 1998; Trouvé et al., 1998); they release more propagules in the environment and, all else being equal, infect a higher proportion of the host population. Epidemiological models have emphasized the key role of propagule production in transmission success (Anderson and May, 1978; May and Anderson, 1978). Clearly, in parasite species with complex life cycles, these propagules infect intermediate hosts, whereas in species with direct life cycles they infect definitive hosts. There is, however, no difference in prevalence in the vertebrate host among nematodes with different life cycles (Morand, 1996b), and the complexity of the life cycle may not influence the postulated effect of adult parasite body size on prevalence.

Second, there is comparative evidence that helminth body size correlates negatively with intensity of infection, or the mean number of parasites per infected host only (Arneberg et al., 1998; Poulin, 1999). This is most likely due to intensity-dependent parasite mortality, a process commonly observed in helminths (Keymer, 1982; Shostak and Scott, 1993) and capable of decreasing aggregation (Anderson and Gordon, 1982). What matters here may be parasite body size relative to host size rather than absolute parasite body size. For a given host size, intensity-dependence may be more severe in large-bodied parasite species than in small-bodied ones, preventing the accumulation of high numbers of large-bodied parasites in individual hosts. Parasite body size (absolute or relative) can, therefore, influence the shape of the frequency distribution of parasites among hosts, i.e., the level of aggregation. We might thus expect a decrease in aggregation levels with increasing body size among related parasite species.

Here, we test this prediction in a comparative analysis across species of gastrointestinal nematode parasites of mammalian hosts. Specifically, we examine empirical relationships between absolute and relative nematode body size, mean abundance, and its variance, to see whether they reflect the causal chain of arguments presented above.

We used data from published studies in which both mean abundance (mean number of conspecific nematodes per host, including uninfected hosts) and its variance were reported and in which at least 30 hosts had been examined (sources of data given in Shaw and Dobson [1995] and Morand and Guégan [1999]). When more than 1 estimate of mean abundance and its variance were available for a nematode species, they were averaged to obtain a single specific value. Body sizes of nematode species were taken as adult body volume, computed as $(\pi l w^2)/4$, where l is mean adult body length and w mean adult maximal body width, both in mm; host mass was recorded in kg. Data were obtained from a variety of sources (see Morand et al., 1996; Poulin and Morand, 1997). The entire data set is presented in Table I.

Mean abundance, its variance, nematode body size, and host mass were transformed (natural logarithms) to normalize their distributions prior to analyses. Two approaches were used to test the body size-versus-aggregation relationship. First, as in previously published analyses of patterns of aggregation (Dobson and Merenlender, 1991; Shaw and Dobson, 1995), we treated each nematode species as statistically independent. Second, we repeated the same analyses using phylogenetically independent contrasts (Felsenstein, 1985; Harvey and Pagel, 1991). This method controls for the potential pseudoreplication arising from inherited similarities between species in body size or even aggregation levels. We obtained contrasts from a nematode phylogeny derived from the cladistic analysis of Blaxter et al. (1998) and from a nematode taxonomy (Anderson, 1992, and references therein). Contrasts were computed using the computer package CAIC version 2.0 (Purvis and Rambaut, 1994); they were not standardized because branch lengths in the phylogeny were not known (see Purvis et al., 1994, for justification). All correlations and regressions using contrasts were forced through the origin (see Garland et al., 1992).

Log variance and log mean abundance were strongly correlated (Fig. 1), both across species (n = 79, $r^2 = 0.964$, $P < 0.0001$) and across phylogenetic contrasts (n = 37, $r^2 = 0.929$, $P < 0.0001$). This indicates that mean abundance explains 93–96% of the spread in the variance in nematode infections among host individuals and thus more than the 87% value reported by Shaw and Dobson (1995) from their survey of a wide range of metazoan parasites. There is very little variation left to explain, but this small amount of variation is not trivial or merely the product of measurement error in the estimates of variance we obtained from published sources. In a simulation, if random deviations in variance are added to the expected variances computed for the 79 species using the regression equation of Figure 1A, and if these estimates of variance now including error are used in a regression against mean abundance, only 0.1% of the variation is left unexplained and not the 4–7% observed in this study (A. Shostak, pers. comm.). Thus the residual variation, although limited, seems to be due to more than just measurement error.

Could nematode body size account for some of it? We computed residuals from the regressions shown in Figure 1 and used them as estimates of the residual, unexplained variation in aggregation levels. These residuals correlated negatively, but not significantly, with absolute nematode body size across species (n = 59, $r = -0.151$, $P = 0.253$) and across phylogenetically independent contrasts (n = 37, $r = -0.182$, $P = 0.280$). However, nematode body size tends to covary with host mass (e.g., across contrasts, n = 37, $r = 0.339$, $P = 0.04$; see also Morand et al. [1996]). Using residuals of regressions of nematode body size versus host mass as measures of relative nematode body size, we also found that nematode body size correlates negatively but not significantly with residual variance (across species, n = 59, $r = -0.158$, $P = 0.233$; across contrasts, n = 37, $r = -0.102$, $P = 0.549$). These results suggest that aggregation levels, measured as the deviation between the observed variance in numbers of parasites per host and the predicted variance, are so constrained by mean abundance that nematode body size, and possibly any other variable, cannot explain the residual noise.

There is an interesting relationship that came out of our analyses and that is worth reporting, however. The variance-to-mean ratio is one of the most widely used indices of parasite aggregation in the parasitology literature; aggregation increases as the value of the ratio increases beyond 1. When using this index as a measure of aggregation (Fig. 2), we found that it correlated negatively and significantly with absolute nematode body size

TABLE I. Nematode species used in the comparative analysis; data from sources listed in Shaw and Dobson (1995) or Morand and Guégan (1999).

Species	Mean abundance	Variance	Body size (mm^3)	Host mass (kg)
Ancylostoma caninum	29.0	356.7	—	57.3
Ancylostoma tubaeforme	5.6	50.3	—	110.0
Apteragia odocoilei	1.3	9.4	—	145.0
Ascaris suum	0.2	0.03	300.0	125.0
Ascarops strongylina	1.7	14.5	23.7	125.0
Aspicularis tetraptera	5.8	37.3	3.8	0.1
Baylisascaris transfuga	3.1	24.8	240.0	140.0
Capillaria aerophila	27.0	388.6	47.6	140.0
Capillaria plica	0.2	0.8	50.0	140.0
Capillaria putorii	1.5	12.3	11.0	140.0
Carolinensis kinsellai	94.9	1,286.6	—	0.2
Cooperia oncophora	2,385.3	55,303.5	11.0	711.3
Craterostomum acuticaudatum	2,416.3	55,992.1	—	350.0
Crenosoma sp.	0.5	3.0	—	140.0
Crossocephalus viviparus	535,763.0	22,641,957.6	—	350.0
Cyathospirura sp.	0.1	0.1	15.0	140.0
Cyathostomum alveatum	717.3	14,720.0	13.0	350.0
Cyathostomum catinatum	65.0	1,391.2	9.0	350.0
Cyathostomum labratum	23.0	422.6	9.5	350.0
Cyathostomum montgomeryi	2,081.1	51,284.6	6.5	350.0
Cyathostomum tetracanthum	4,828.6	142,850.2	12.0	350.0
Cylicocyclus adersi	244.7	4,554.6	16.0	350.0
Cylicocyclus auriculatus	2,841.7	82,586.6	26.0	350.0
Cylicocyclus gyalocephaloides	416.8	10,163.8	15.0	350.0
Cylicocyclus nassatus	60.0	1,219.0	12.5	350.0
Cylicocyclus triramosus	2,076.3	52,576.4	14.0	350.0
Cylicodontophorus reineckei	3.8	29.9	13.5	350.0
Cylicodontophorus schuermanni	1,212.0	28,526.4	20.0	350.0
Cylicospirura subaequalis	27.3	346.5	21.3	110.0
Cylicostephanus bidentatus	883.0	24,962.2	8.0	350.0
Cylicostephanus calicatus	3,163.2	84,536.1	8.0	350.0
Cylicostephanus longiconus	2,588.0	71,715.3	—	350.0
Cylicostephanus minutus	309.8	6,210.6	6.0	350.0
Dictyocaulus viviparus	3.0	—	57.5	145.0
Dirofilaria immitis	1.4	16.9	190.0	57.3
Dracunculus sp.	0.1	0.2	—	140.0
Draschia megastoma	77.3	1,202.8	13.0	350.0
Globocephalus urosubulatus	0.8	5.2	9.4	125.0
Gnathostoma sp	0.2	0.05	—	140.0
Gongylonema pulchrum	4.7	34.3	745.0	110.0
Habronema muscae	26.0	486.0	22.0	350.0
Haemonchus contortus	64.6	1,035.3	34.0	145.0
Haemonchus mitchelli	18.0	215.4	21.0	350.0
Lagochilascaris sp.	1.2	11.2	22.7	140.0
Litosomoides carinii	0.2	—	65.0	0.1
Metastrongylus apri	18.9	189.7	48.5	125.0
Metastrongylus pudendoctectus	3.5	28.4	40.0	125.0
Metathelazia californica	12.7	132.2	20.0	110.0
Molineus barbatus	4.3	49.8	6.6	140.0
Monodontus floridanus	5.2	42.7	—	0.2
Nematodirus helvetianus	1,840.3	—	25.0	900.0
Nematodirus odocoilei	71.0	—	8.5	145.0
Oesophagostomum quadrispinulatum	11.7	166.7	10.4	125.0
Oesophagostomum venulosum	1.8	10.0	20.0	145.0
Oslerus osleri	24.9	362.6	—	16.0
Ostertagia mossi	321.6	—	9.0	145.0
Ostertagia ostertagi	0.1	0.06	9.2	145.0
Oxyuris equi	199.4	3,281.5	90.0	350.0
Parabronema pecariae	0.3	2.8	20.0	30.0
Parabronema skrjabini	528.0	10,379.0	36.6	350.0
Parascaris equorum	55.0	—	370.0	350.0

TABLE I. Continued.

Species	Mean abundance	Variance	Body size (mm^3)	Host mass (kg)
Pharyngostomoides procyonis	0.01	0.05	—	140.0
Physaloptera praeputialis	3.2	26.9	48.0	110.0
Physaloptera rara	1.8	16.0	24.0	70.5
Physocephalus sexalatus	28.5	384.7	18.7	77.5
Placoconus lotoris	0.9	3.5	—	140.0
Poteriostomum ratzii	108.5	1,937.0	17.0	350.0
Poterostomum imparidentatum	210.0	—	18.0	350.0
Probstmayria vivipara	11,138,654.5	573,037,793.5	—	350.0
Protospirura numidica	4.0	32.3	—	16.0
Protostrongylus macrotis	3.0	—	47.0	145.0
Pterygodermatites peromysci	0.6	—	29.3	0.1
Sertaria yehi	10.6	—	89.0	145.0
Setaria equina	3.6	28.5	190.0	350.0
Skrjabinema parva	55.0	—	4.0	145.0
Skrjabinema sp.	327.0	6,182.0	—	350.0
Spirocerca lupi	0.5	0.9	15.4	16.0
Stephanus dentatus	10.2	89.3	45.0	125.0
Strongyloides sigmodontis	46.3	805.3	4.7	0.2
Strongyloides sp.	171.5	3,502.4	—	140.0
Strongyloides westeri	1.6	9.5	—	350.0
Syphacia peromysci	5.7	—	2.3	0.1
Syphacia sigmodontis	2.5	—	3.0	0.2
Texicospirura turki	97.0	2,119.0	—	30.0
Toxascaris leonina	51.6	763.7	100.0	47.3
Toxocara cati	0.3	1.3	70.0	110.0
Trichostrongylus affinis	0.1	—	9.3	0.1
Trichostrongylus axei	20.6	—	5.5	247.5
Trichostrongylus colubriformis	3.0	—	6.0	145.0
Trichostrongylus thomasi	51.6	793.1	5.6	350.0
Trichuris vulpis	7.5	124.3	75.0	16.0
Triodontophorus brevicaudata	330.0	—	19.0	350.0
Triodontophorus minor	1,252.7	33,236.7	16.0	350.0
Triodontophorus nipponicus	41.0	803.2	19.5	350.0
Triodontophorus serratus	32.7	529.8	20.0	350.0
Vogeloides felis	1.3	7.3	7.1	110.0

across parasite species (n = 59, $r = -0.332$, $P = 0.0102$). The scatter of points forms a triangular pattern (Fig. 2A), suggesting that small-bodied nematodes show a wide range of aggregation levels, whereas large-bodied nematodes occur mostly at low levels of aggregation. The same relationship (n = 59, $r = -0.375$, $P = 0.0034$) and triangular scatter were found when using relative nematode body size instead of absolute size. This pattern disappeared, however, once we used contrasts and controlled for potential phylogenetic influences (absolute nematode size, n = 37, $r = 0.076$, $P = 0.650$; relative nematode size, n = 37, $r = 0.049$, $P = 0.773$).

These last results suggest that there may be a weak relationship between parasite body size and aggregation levels. The signal is very weak though. The linear relation between the variance and mean abundance is stronger in nematodes than in other groups of parasites (Shaw and Dobson, 1995; S. Morand, unpubl. obs.). It may be that aggregation is too constrained in nematodes, and that the influence of body size may be apparent in other groups of parasites where variance in infection levels shows more spread. The search for what, if anything, determines aggregation levels in parasite population may need to turn toward these other parasite taxa. Along these lines, it is interesting to note that certain types of parasites with large body sizes relative to host size, such as parasitoids and parasitic castrators, do not have aggregated distributions (Kuris, 1996).

An effect of parasite body size or of some other predictor of aggregation would have implications for the population biology of host–parasite interactions. For instance, population dynamics models have highlighted the importance of parasite aggregation levels in determining whether a parasite population can regulate its host population (Anderson and May, 1978; May and Anderson, 1978). Host regulation by parasites becomes impossible when parasites are very highly aggregated. Also, from a population genetics perspective, aggregation levels may influence the effective population size if they lead to density-dependent reproductive output and inequalities in egg production among parasite individuals (Dobson, 1986; Szalai and Dick, 1989). There is some evidence that within-population genetic variation in nematodes is lower in large-bodied species, e.g., *Ascaris* (Nadler et al., 1995) than in small-bodied ones, e.g., *Ostertagia* (Blouin et al., 1992) in hosts of comparable sizes. The above implications illustrate the sort of far-reaching consequences that

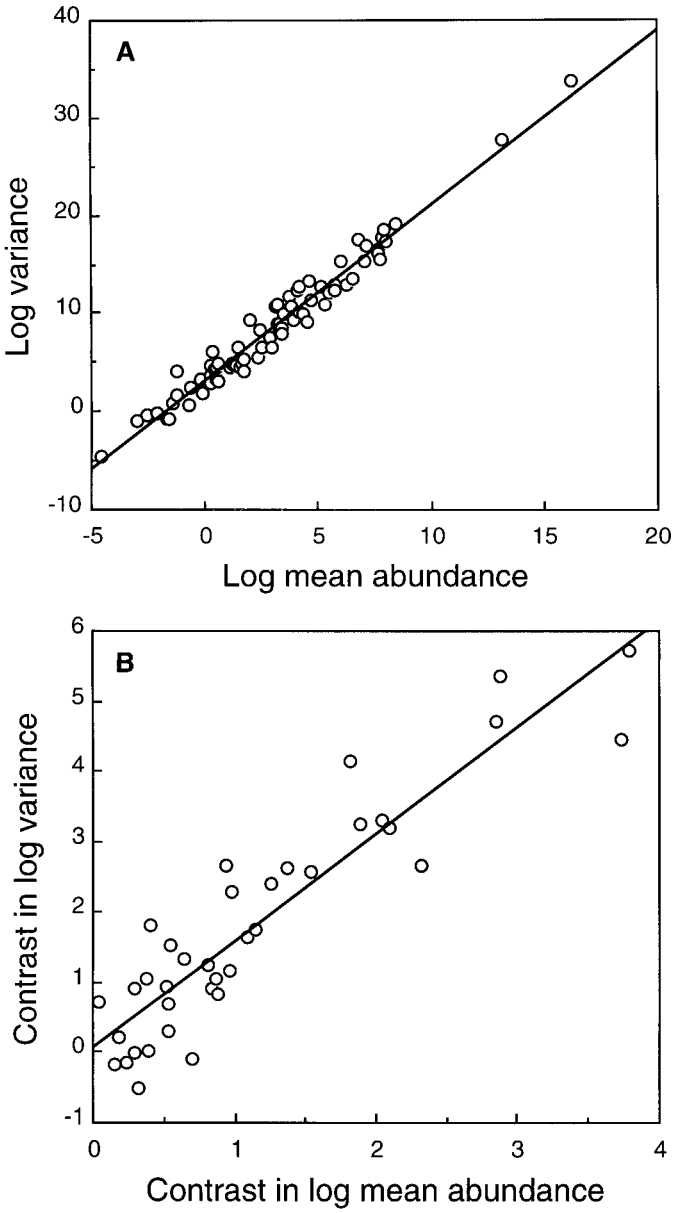

FIGURE 1. Relationship between log variance and log mean abundance: **A**, across 79 nematode species, y = 1.793x + 3.088, and **B**, across 37 phylogenetically independent contrasts derived from those species, y = 1.524x.

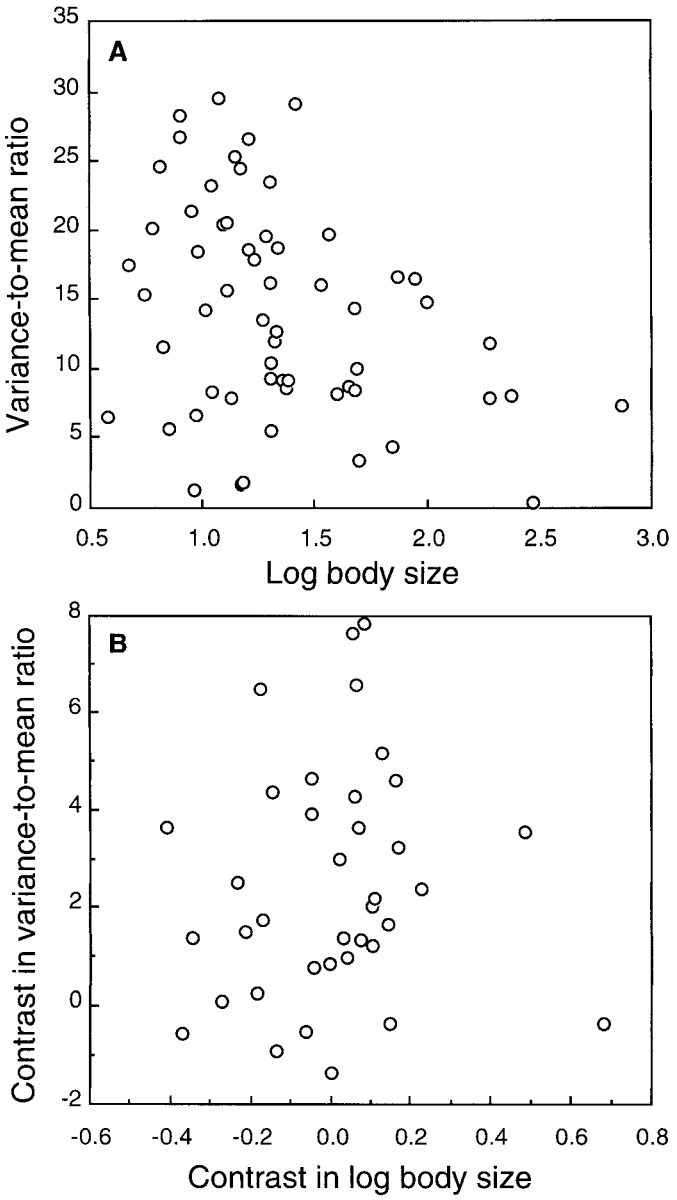

FIGURE 2. Relationship between the variance-to-mean ratio and log body size (volume, in mm^3) of parasitic nematodes: **A**, across 59 species, and **B**, across 37 phylogenetically independent contrasts derived from those species.

are linked with aggregation levels, and the importance of searching for the biological causes behind their variability.

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Reassignment of *Lamanema* from Nematodirinae to Molineinae (Nematoda: Trichostrongyloidea)

Lora G. Rickard and Eric P. Hoberg*, College of Veterinary Medicine, P.O. Box 9825, Mississippi State University, Mississippi State, Mississippi 39762; and *Biosystematics and National Parasite Collection Unit, USDA, Agricultural Research Service, BARC East No. 1180, 10300 Baltimore Avenue, Beltsville, Maryland 20705 (order of authorship is arbitrary)

ABSTRACT: The monospecific *Lamanema* historically has been assigned to the Nematodirinae within the Molineidae. Inconsistencies in morphological characters, within a phylogenetic context for Nematodirinae, led to a re-evaluation of the putative relationships and taxonomic placement of *Lamanema.* Among 7 putative synapomorphies for Nematodirinae, *Lamanema* possesses only 1, large eggs. Large eggs, sporadically present in phylogenetically disparate taxa of trichostrongyles, are equivocal with respect to placement of *Lamanema*; it is considered that possession of this single homoplasious character alone is insufficient justification to retain the genus in Nematodirinae. Affinities with the Trichostrongylidae (Cooperiinae or Haemonchinae) have also been proposed; however, *Lamanema* possess neither of 2 synapomorphies that diagnose monophyly of the family. *Lamanema* is retained in the Molineidae and transferred to the Molineinae as it possesses all characters of the family as currently defined. The origin of *Lamanema* represents a secondary colonization of ruminants by molineids and provides no context for elucidating the history of the Nematodirinae and *Nematodirus.*

Lamanema chavezi Becklund, 1963, a distinctive trichostrongyloid nematode parasitizing alpacas (*Lama pacos* (L.)) and vicuña (*Vicugna vicugna* (Molina)) in Peru (Becklund, 1963), is the only member of the genus *Lamanema.* Subsequent to the original description, a unique enterohepatic migration by parasitic third- and fourth-stage larvae was recognized and considered to be the cause of significant pathology associated with infections of the parasite (Chavez et al., 1967; Guerrero et al., 1981). This nematode has been considered a characteristic helminth of South American camelids (Guerrero et al., 1981) and has yet to be reported outside of the Neotropical region (see Rickard and Bishop, 1991). *Lamanena chavezi* has also been identified in the chinchillid rodent, *Lagidium viscacia* (Molina), in Argentina, the only known noncamelid host (Sutton and Durette-Desset, 1985).