MINI-REVIEW ARTICLE

Recent insights into the epidemiology and genetics of *Ascaris* in China using molecular tools

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SUMMARY

Ascaris is a large parasitic roundworm (nematode) of the small intestine of humans and pigs, which causes the socio-economically important disease, ascariasis. To better understand the relationship of *Ascaris* between the 2 host species, recent studies in China have focused on investigating the genetics and epidemiology of *Ascaris* from humans and pigs using a mutation scanning-based approach. Findings provided support for a low level of gene flow between the human and porcine *Ascaris* populations. Extending the studies of genotypic variability within *Ascaris* from humans and pigs, experimental infections of mice and pigs with selected genotypes of *Ascaris* were carried out. Initial results indicate that there is a significant difference in the ability of *Ascaris* eggs of genotype G1 (derived from human) and G3 (derived from pig) to infect and establish as adults in pigs, supporting the difference in the frequencies of these genotypes in natural *Ascaris* populations between pigs and humans in China. Taken together, current information supports that there is limited cross-infection of *Ascaris* between humans and pigs in endemic regions and that pigs are not a significant reservoir of human infection with the adult nematode in such areas.

Key words: *Ascaris*, China, mutation scanning, single-strand conformation polymorphism (SSCP) analysis, nuclear ribosomal DNA, genotypes, mitochondrial haplotypes, experimental infections, pigs.

INTRODUCTION

Ascaris is a large parasitic roundworm (nematode) of the small intestine of both humans and pigs. Ascariasis, the disease caused by this parasite genus, is of major human health importance in many parts of the world (WHO, 1987; Peng *et al*. 1998*a*; O'Lorcain and Holland, 2000; Crompton, 2001). *Ascaris* infects a quarter of the world's human population and clinically affects ~100–200 million people, particularly children (WHO, 1987; Crompton, 1989, 2001; Peng *et al*. 1996, 1998*a*, *b*; O'Lorcain and Holland, 2000; Stephenson, 2002). Also, ascariasis of pigs is of major economic significance due to production losses linked to reduced feed conversion efficiency and losses to the meat industry associated with the condemnation of 'milk-spot' livers (Stewart and Hale, 1988). Central to the implementation of control programmes against *Ascaris* is a clear understanding of fundamental aspects of its biology, genetics and epidemiology. While there is knowledge about the epidemiology and population biology of *Ascaris* for a number of countries (Crompton, 1989; O'Lorcain and Holland, 2000; Anderson, 2001; Crompton, 2001; Peng *et al*. 2002), there has been controversy as to whether *Ascaris*-infected pigs represent a significant reservoir for human infection in endemic and non-endemic regions (Anderson, 1995, 2001; Anderson *et al*. 1993, 1995; Peng and Zhou, 2001). Particularly for large endemic countries, such as China, questions regarding the genetic structure of *Ascaris* populations, host specificity and transmission patterns had scarcely been addressed (see Peng *et al*. 1996, 1998*a*, *b*). Clearly, investigating these areas is interesting scientifically and, importantly, contributes towards the improved prevention and control of *Ascaris*.

There has been debate about *Ascaris lumbricoides* Linnaeus, 1758 of humans representing a different

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species to *Ascaris suum* Goeze, 1782 of pigs, and various studies have attempted to address this problem (Barry and O'Rourke, 1967; Crompton, 1989; Anderson, 2001). Experimental cross-host species transmission studies have shown that *Ascaris* of human origin can infect the pig, and *vice versa* (Takata, 1951; Galvin, 1968). Other studies (Sprent, 1952; Ansel and Thibaut, 1973; Maung, 1973; Kurimoto, 1974; Kennedy *et al*. 1987; Nadler, 1987) have applied morphological, karyotypic, immunological and/or biochemical approaches to attempt to address this problem. More recently, molecular methods have been employed to investigate the genetic make-up of *Ascaris* populations (Anderson, 2001; Anderson *et al*. 1993, 1995; Peng *et al*. 1998*c*; Nadler *et al*. 1995; Nadler, 1996; Anderson and Jaenike, 1997; Nadler and Hudspeth, 1998). For example, PCR-coupled restriction fragment length polymorphism (PCR-RFLP) analyses of a nuclear ribosomal DNA (rDNA) region spanning the first (ITS-1) and second (ITS-2) internal transcribed spacers (= ITS) and the intervening 5.8S gene and of mitochondrial DNA regions had been used to study *Ascaris* populations (Anderson, 1995; Anderson *et al*. 1995; Anderson and Jaenike, 1997; Peng *et al*. 1998*c*), but there was no clear genetic delineation between human *Ascaris* and pig *Ascaris* (Anderson, 2001). While PCR-RFLP analysis of nuclear (including ribosomal) and of mitochondrial DNA markers has been used, nucleotide sequence data sets were lacking. However, Zhu *et al*. (1999) demonstrated a nucleotide sequence difference of 1·3% in a ~300 bp region of the ITS-1 (designated pITS-1) of nuclear ribosomal DNA between human and pig *Ascaris*. Due to the limited number of samples (from widely separated geographical locations) used, the interpretation from this study was guarded, but the sequence data had suggested population substructuring within the genus (based on sequence data).

Extending from previous studies, a series of recent investigations has been conducted to gain a better insight into the genetics and epidemiology of *Ascaris* in China. The main aims were (1) to study nucleotide variation in ribosomal and mitochondrial DNAs within and among *Ascaris* population from human and pig hosts from a range of different geographical regions in China employing a mutation scanning-coupled sequencing approach, (2) to establish whether there is a particular relationship between *Ascaris* genotype and host species, and (3) to establish an experimental infection of *Ascaris* of human origin in pigs, to be able to investigate the genetic and reproductive relationships between human *Ascaris* and pig *Ascaris*. The present article reviews recent findings and, based on them, makes conclusions regarding the biology and epidemiology of *Ascaris* and its prevention and control in China.

The genetic make-up of Ascaris *populations from humans and pigs in China*

Using the mutation scanning-based approach (i.e, single-strand conformation polymorphism (SSCP) and selective sequencing), Peng *et al*. (2003*a*) investigated nucleotide variation in a part of the first internal transcribed spacer (pITS-1) of nuclear ribosomal DNA within and among a large number (n=815) of *Ascaris* individuals from humans and pigs from 6 endemic regions in China, and recorded the frequency of the different genotypes of *Ascaris* in relation to host species and geographical origin. Five different genotypes (G1–G5) were detected for human *Ascaris*, of which 3 (genotypes G1–G3) were detected for pig *Ascaris*. The detection of more genetic variation than in some previous reports (Peng *et al*. 1998*c*; Zhu *et al*. 1999) was not unexpected, given the large numbers of *Ascaris* individuals examined and the use of the SSCP approach, which is highly sensitive to detecting nucleotide alterations (Gasser *et al*. 2002; Gasser, 2006). Of the five *Ascaris* genotypes detected, genotype G1 infected mainly humans (~63–74%), whereas genotype G3 infected predominantly pigs (~79–86%). Since the frequencies of the 3 other genotypes were substantially lower for the 2 host species, the results indicated that the rate of cross-infection of *Ascaris* between humans and pigs was low and that gene flow between the predominant genotypes (i.e., G1 and G3) was limited. These findings support other studies indicating that human and pig hosts tend to harbour a particular *Ascaris* genotype (Anderson *et al*. 1993; Peng *et al*. 1998*c*; Zhu *et al*. 1999), except in non-endemic regions (Anderson, 1995), and that gene flow between the *Ascaris* populations from humans and pigs is limited (Anderson, 1995; Peng *et al*. 2005). While it is not clear why pig *Ascaris* seems to be detected more frequently in humans in non-endemic than in endemic areas (Anderson, 1995, 2001; Nesjum *et al*. 2005), it may relate to a lack of exposure of humans to *Ascaris* eggs (of any host origin) and/or ease of sampling and detection (as infected individuals are more likely to consult a medical doctor).

The occurrence of particular genotypes of *Ascaris* in both the human and pig hosts has been interpreted to relate to one or more population genetic processes, such as introgression (= incorporation of genes of one species into the gene pool of another) or lineage sorting and retention of ancentral polymorphism (Anderson, 2001). However, the existence of genotypes of *Ascaris* (such as G2 and G5) which contain 2 distinct pITS-1 sequence types (i.e., with a G/C polymorphism at alignment position 133, consistent with a mixed profile in PCR-RFLP of ITS-1; Anderson *et al*. 1993; Peng *et al*. 1998*c*) had suggested that hybridization may occur between subpopulations of the human- and

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the pig-associated *Ascaris*. It had been proposed that interbreeding may occur between individuals representing different genotypes (e.g., G1 from humans and G3 from pigs) (Peng *et al*. 2003*a*), thus allowing variants of ITS-1 to be introduced and dispersed to a degree which exceeds the molecular 'homogenisation process'. Alternatively, it is possible that different host species-associated populations have diverged genetically, whereby the processes of concerted evolution (Elder and Turner, 1995) have led to polymorphism at particular nucleotide positions and to sequence length variation. The latter explanation is plausible, given that *Ascaris* has been reported previously to contain distinct types of ribosomal DNA (Back *et al*. 1984*a*, *b*). Theoretical studies have suggested that DNA turnover mechanisms, such as gene conversion, transposition, unequal crossing-over or/and slippage during DNA replication are involved in the process of homogenization (Elder and Turner, 1995), and these mechanisms may apply to *Ascaris*, but would need to be evaluated.

Since no unequivocal nucleotide differences were detected in the genetic marker (pITS-1) between human- and pig-derived *Ascaris* (in naturally infected hosts), cross infection (between hosts) could not be reliably detected (Peng *et al*. 2003*a*). Hence, under these circumstances, the proposal for the existence of separate species of *Ascaris* (i.e., *A*. *lumbricoides* and *A*. *suum*) cannot be supported or refuted (Peng *et al*. 2003*a*). However, the significant frequency differences in the genotypes detected between human- and pig-derived *Ascaris* suggested that cross-infection occur at a low level and that they are reproductively isolated. These findings are consistent with those from previous molecular studies conducted, for example, in Guatemala (Anderson *et al*. 1995) and in China (Peng *et al*. 1998*c*, 2005) using nuclear or mitochondrial gene markers. Taken together, these findings appear to support the 'two host-specialist parasite population with some cross-infection' model, as proposed by Anderson (2001).

Experimental infections of pigs and mice with eggs from Ascaris *of human or pig origin*

Extending recent molecular and epidemiological studies (Peng *et al*. 2003*a*, *b*, 2005), experimental infections of pigs and mice with eggs representing particular genotypes of *Ascaris* were carried out in China (Peng *et al*. 2006). *Ascaris* of human origin (genotype G1) did not establish readily in experimental pigs whereas *Ascaris* of porcine origin (genotype G3) did (Peng *et al*. 2006). These preliminary findings are in accordance with those from a recent molecular-epidemiological investigation in China, showing that the prevalence of genotype G1 in pigs is very low (2·4%, $n=329$) and inferring a limited level of cross-infection of *Ascaris* between human and pig (Peng *et al*. 2003*a*). Although a small number of different egg batches from single *Ascaris* females representing the 2 genotypes were used in the infection experiments (Peng *et al*. 2006), the findings support the proposal that *Ascaris* of human origin does not readily infect pigs. This statement is reinforced by other results that *Ascaris* eggs of genotypes other than G1 (being consistent with genotype G2 from human *Ascaris*; see Peng *et al*. 2003*a*) do not establish as adults in pigs (using appropriate control groups) (Peng *et al*. unpublished findings). It is also supported by various molecular-epidemiological investigations (Anderson, 2001; Peng *et al*. 2003*a*, 2005). Nonetheless, further study, using more egg batches from single female worms representing the different *Ascaris* genotypes/haplotypes from each of the two host species is required.

CONCLUDING REMARKS

The research findings of recent molecular-epidemiological and experimental investigations in China (Peng *et al*. 2003*a*, *b*, 2005, 2006) support the hypothesis that there is a specific affiliation of 'pig *Ascaris*' to pigs and 'human *Ascaris*' to humans and a limited gene flow between the 2 main types of *Ascaris*, in spite of a long history of contact between the two host species. This information reinforces other evidence (Peng *et al*. 1998*c*; Anderson, 2001; Peng and Zhou, 2001) that, in endemic regions of China (and Guatemala), there is very limited cross-infection between human and pig or between pig and human, and provides support that pigs are not a significant reservoir of infections for human ascariasis. Interestingly, these results contrast evidence for non-endemic regions, such as Denmark and North America, where the cross-infection of *Ascaris* from pigs to humans can occur (e.g., Anderson, 1995; Nejsum *et al*. 2005). Recently, Nejsum *et al*. (2005) studied *Ascaris* from Danish patients, with a history of contact with pigs or pig manure, employing amplified fragment length polymorphism (AFLP) and PCR-RFLP analyses of a range of *Ascaris* samples from different countries. Together with epidemiological information, the findings supported the proposal that *Ascaris* infection in Danish patients was acquired from domestic pigs. While the reasons for the differential ability of the parasite to cross-infect and establish in the 'heterologous' host species in non-endemic but not so readily in endemic areas are unclear, they are likely to relate to host- and parasite-factors. For instance, host genes linked to susceptibility (Wakelin and Bradely, 2002; Williams-Blangero *et al*. 2002) and/or the immune status of the host (Cooper, 2002; Jungersen, 2002) may be associated with the ability of some *Ascaris* genotypes to establish preferentially in humans and others in pigs. In non-endemic regions, humans may not have been exposed previously to pig *Ascaris*,

whereas in endemic regions (possibly due to the tradition of using pig excrement as a fertilizer) humans may have adequate immunological exposure to dead or viable porcine *Ascaris* eggs, thus blocking or inhibiting cross-infection. Also, susceptibility to cross-infection may be increased in hosts compromised by environmental or nutritional stresses, and/or concurrent infections with other infectious agents (Holland and Boes, 2002; Williams-Blangero and Blangero, 2002). Parasite factors could include differences in molecular processes governing the capacity of *Ascaris* to invade the 'appropriate' host. Another possibility is that the different genotypes of *Ascaris* induce varying immune responses *via* differentially expressed antigens (Abebe *et al*. 2002), and/or that the host defence and parasite invasion mechanism(s) or migratory routes differ in pigs compared with humans. The distinctiveness in recovery rates of worms from pigs and larvae from mice infected with eggs of genotypes G1 ('human') and G3 ('pig') (*cf*. Peng *et al*. 2006) suggests that investigations of the relationship between the genetic diversity of *Ascaris* and host susceptibility/resistance as well as immune responses will be worthwhile and informative. For example, the mouse model developed by Lewis *et al*. (2006) could be useful for the dissection of early host responses to experimental infection, given the limitation with reagents and cost associated with experiments in pigs. Another mouse model (Johnston *et al*. 2005) has been shown to be useful for exploring different adaptive immune responses against distinct isolates of *Trichuris muris*. At the molecular level, immunological responses and the parasite-host relationship of *Ascaris* could be explored by transcriptional profiling (temporal and spatial) utilizing a microarray platform, which could be extended to naturally infected humans of different ages groups and from non-endemic and endemic regions and, in the future (when the resources become available) to pigs experimentally infected with *Ascaris* of human or pig origin. With the major advances in genomic and proteomic expertise and resources, well-controlled comparative analysis of molecular differences between human *Ascaris* and pig *Ascaris* (employing a relatively large numbers of individual worms at the same and different stages of development, with biological replication) may also provide clues regarding the parasite-host interplay.

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REFERENCES

Abebe, W., Tsuji, N., Kasuga-Aoki, H., Miyoshi, T., Isobe, T, Arakawa, T., Matsumoto, Y. and Yoshihara, S. (2002). Species-specific proteins identified in *Ascaris lumbricoides* and *Ascaris suum* using two-dimensional electrophoresis. *Parasitology Research* **88**, 868–871.

Anderson, T. J. C., Romero-Abal, M. E. and Jaenike, J. (1993). Genetic structure and epidemiology of *Ascaris* populations: patterns of host affiliation in Guatemala. *Parasitology* **107**, 319–334.

Anderson, T. J. C., Romero-Abal, M. E. and Jaenike, J. (1995). Mitochondrial DNA and *Ascaris* microepidemiology: the composition of parasite populations from individual hosts, families and villages. *Parasitology* **110**, 221–229.

Anderson, T. J. C. (1995). *Ascaris* infections in humans from North America: molecular evidence for cross-infection. *Parasitology* **110**, 215–219.

Anderson, T. J. C. and Jaenike, J. (1997). Host specificity, evolutionary relationships and macrogeographic differentiation among *Ascaris* populations from humans and pigs. *Parasitology* **115**, 325–342.

Anderson, T. J. C. (2001). The dangers of using single locus markers in parasite epidemiology: *Ascaris* as a case study. *Trends in Parasitology* **17**, 183–188.

Ansel, M. and Thibaut, M. (1973). Value of the specific distinction between *Ascaris lumbricoides* Linnè 1758 and *Ascaris suum* Goeze 1782. *International Journal for Parasitology* **3**, 317–319.

Back, E., Müller, F. and Tobler, H. (1984*a*). Structural organization of the two main rDNA size classes of *Ascaris lumbricoides*. *Nucleic Acids Research* **12**, 1313–1332.

Back, E., Felder, H., Müller, F. and Tobler, H. (1984*b*). Chromosomal arrangement of the two main rDNA size classes of *Ascaris lumbricoides*. *Nucleic Acids Research* **12**, 1333–1347.

Barry, J. M. and O'Rourke, F. J. (1967). Ascariasis in pig and in man. *Science Proceedings of the Royal Dublin Society, Series A* **3**, 39–55.

Cooper, P. J. (2002). Immune responses in humans. In *World Class Parasites: Volume 2. The Geohelminths*: Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 89–107. Kluwer Academic Publishers, Boston.

Crompton, D. W. T. (1989). Biology of *Ascaris lumbricoides*. In *Ascariasis and Its Prevention and Control* (ed. Crompton, D. W. T., Nesheim, M. C. and Pawlowski, Z. S.), pp. 10–44. Taylor and Francis, London, New York and Philadelphia.

Crompton, D. W. T. (2001). *Ascaris* and ascariasis. *Advances in Parasitology* **48**, 285–375.

Elder, J. F. and Turner, B. J. (1995). Concerted evolution of repetitive DNA sequences in eukaryotes. *Quarterly Review of Biology* **70**, 297–320.

Galvin, T. J. (1968). Development of human and pig *Ascaris* in the pig and rabbit. *Journal of Parasitology* **54**, 1085–1091.

Gasser, R. B. (2006). Molecular tools – advances, opportunities and prospects. *Veterinary Parasitology* **36**, 69–89.

Gasser, R. B., Zhu, X. Q. and Chilton, N. B. (2002). The value of mutation scanning approaches for detecting genetic variation – implications for studying intestinal nematodes of humans. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 219–233. Kluwer Academic Publishers, Boston.

Holland, C. V. and Boes, J. (2002). Distribution and predisposition. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 1–24. Kluwer Academic Publishers, Boston.

Johnston, C. E., Bradley, J. E., Behnke, J. M., Matthews, K. R. and Else K. J. (2005). Isolates of *Trichuris muris* elicit different adaptive immune responses in their murine host. *Parasite Immunology* **27**, 69–78.

Jungersen, G. (2002). Immunity and immune responses in pigs. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 105–124. Kluwer Academic Publishers, Boston.

Kennedy, M. W., Qureshi, F., Haswell-Elkins, M. and Elkins, D. B. (1987). Homology and heterology between the secreted antigens of the parasitic larval stages of *Ascaris lumbricoides* and *Ascaris suum*. *Clinical and Experimental Immunology* **67**, 20–30.

Kurimoto, H. (1974). Morphological, biochemical and immunological studies on the differences between *Ascaris lumbricoides* Linnaeus, 1758 and *Ascaris suum* Goeze, 1782. *Japanese Journal of Parasitology* **23**, 251–267.

Lewis, R., Behnke, J. M., Stafford, P. and Holland, C. V. (2006). The development of a mouse model to explore resistance and susceptibility to early *Ascaris suum* infection. *Parasitology* **132**, 289–300.

Maung, M. (1973). *Ascaris lumbricoides* Linnè, 1758 and *Ascaris suum* Goeze, 1782: morphological differences between specimens obtained from man and pig. *Southeast Asian Journal of Tropical Medicine and Public Health* **4**, 41–45.

Nadler, S. A. (1987). Biochemical and immunological systematics of some ascaridoid nematodes: genetic divergence between congeners. *Journal of Parasitology* **73**, 811–816.

Nadler, S. A., Lindquist, R. L. and Near, T. J. (1995). Genetic structure of midwestern *Ascaris suum* populations: a comparison of isoenzyme and RAPD markers. *Journal of Parasitology* **81**, 385–394.

Nadler, S. A. (1996). Microevolutionary patterns and molecular markers: the genetics of geographic variation in *Ascaris suum*. *Journal of Nematology* **28**, 277–285.

Nadler, S. A. and Hudspeth, D. S. (1998). Ribosomal DNA and phylogeny of the Ascaridoidea (Nemata: Secernentea): implications for morphological evolution and classification. *Molecular Phylogenetics and Evolution* **10**, 221–236.

Nejsum, P., Parker, E. D. J. R., Frydenberg, J., Roepstorff, A., Boes, J., Haque, R., Astrup, I., Prag, J. and Skov Sorensen, U. B. (2005). Ascariasis is a zoonosis in Denmark. *Journal of Clinical Microbiology* **43**, 1142–1148.

O'Lorcain, P. and Holland, C. V. (2000). The public health importance of *Ascaris lumbricoides*. *Parasitology* **121** (Suppl.), S51–S71.

Peng W., Anderson, T. J. C., Zhou X. and Kennedy, M. W. (1998*c*). Genetic variation in sympatric *Ascaris* populations from humans and pigs in China. *Parasitology* **117**, 355–361.

Peng, W., Yuan, K., Hu, M., Zhou, X. and Gasser, R. B. (2005). Mutation scanning-coupled analysis of haplotypic variability in mitochondrial DNA regions reveals low gene flow between human and porcine *Ascaris* in endemic regions of China. *Electrophoresis* **26**, 4317–4326.

Peng, W., Yuan, K., Peng, G., Zhou, X., Hu, M. and Gasser, R. B. (2006). Experimental infections of pigs and mice with *Ascaris* eggs representing different genotypes indicates variability in host affiliation. *Parasitology* (in the Press).

Peng, W., Yuan, K., Zhou, X., Hu, M. and Gasser, R. B. (2003*a*). Molecular-epidemiological study of *Ascaris* genotypes in China based on single-strand conformation polymorphism analysis of ribosomal DNA. *Electrophoresis* **24**, 2308–2315.

Peng, W. and Zhou, X. (2001). Experimental epidemiological study on possible influence of pig-derived *Ascaris* on the transmission of human ascariasis. *Chinese Journal of Epidemiology* **22**, 116–118. (In Chinese, with English abstract).

Peng, W., Zhou, X. and Crompton, D. W. T. (1998*a*). Ascariasis in China. *Advances in Parasitology* **41**, 109–148.

Peng, W., Zhou, X. and Cui, X. (2002). Comparison of the structure of natural and re-established population of *Ascaris* in humans in a rural community of Jiangxi, China. *Parasitology* **124**, 641–647.

Peng, W., Zhou, X. and Gasser, R. B. (2003*b*). Faecal egg profiles of *Ascaris* from humans: biological and epidemiological implications. *Parasitology* **127**, 283–290.

Peng, W., Zhou, X., Cui, X., Crompton, D. W. T., Whitehead, R. R., Xiong, J., Wu, H., Peng, J., Yang, Y., Wu, W., Xu, K. and Yan, Y. (1996). *Ascaris*, people and pigs in rural community of Jiangxi Province, China. *Parasitology* **113**, 545–557.

Peng, W., Zhou, X., Cui, X., Crompton, D. W. T., Whitehead, R. R., Xiong, J., Wu, H., Yang, Y., Wu, W., Xu, K. and Yan, Y. (1998*b*). Transmission and natural regulation of infection with *Ascaris lumbricoides* in a rural community in China. *Journal of Parasitology* **84**, 252–258.

Sprent, J. F. A. (1952). Anatomical distinction between human and pig strains of *Ascaris*. *Nature, London* **170**, 627–628.

Stephenson, L. S. (2002). Pathophysiology of intestinal nematodes. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 39–61. Kluwer Academic Publishers, Boston.

Stewart, T. B. and Hale, O. M. (1988). Losses to internal parasites in swine production. *Journal of Animal Science* **66**, 1548–1554.

Takata, I. (1951). Experimental infection of man with *Ascaris* of man and the pig. *Kitasato Archives of Experimental Medicine* **23**, 49–59.

Wakelin, D. and Bradley, J. E. (2002). Parasite strain diversity and host immune responses. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 199–218. Kluwer Academic Publishers, Boston.

Williams-Blangero, S., VandeBerg, J. L., Subedi, J., Aivaliotis, M. J., Rai, D. R., Upadhayay, R. P., Jha, B. and Blangero, J. (2002). Genes on chromosomes 1 and 13 have significant effects on *Ascaris* infection. *Proceedings of the National Academy of Sciences, USA* **99**, 5533–5538.

Williams-Blangero, S. and Blangero, J. (2002). Host susceptibility to intestinal worm infections. In *World Class Parasites: Volume 2. The Geohelminths:* Ascaris, Trichuris *and Hookworm* (ed. Holland, C. V. and Kennedy, M. W.; Series ed. Black and Seed, J. R.), pp. 167–183. Kluwer Academic Publishers, Boston.

World Health Organization (1987). *Prevention and Control of Intestinal Parasitic Infections*. WHO Technical Report Series No. 749. WHO, Geneva.

Zhu, X. Q., Chilton, N. B., Jacobs, D. E., Boes, J. and Gasser, R. B. (1999). Characterisation of *Ascaris* from human and pig hosts by nuclear ribosomal DNA sequences. *International Journal for Parasitology* **29**, 469–478.

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