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American Society of Parasitologists

Newsletter

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From the *Editor* of the Newsletter

The ASP newsletter accepts information and news of a parasitological nature from all disciplines. Consider publishing your parasite poems, posting a link to your favorite "parasite lecture" providing an actual parasite lecture, or otherwise send "something" in to the editor. Your contribution is valuable and anything sent in to me will be considered for publication.

Sincerely,

Scott L. Gardner

Curator, Harold W. Manter Laboratory of Parasitology
University of Nebraska-Lincoln

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MEETING DATA

PLAN FOR THE 2009 ASP MEETING [Knoxville, TN].

The 2009 ASP Annual meeting will occur at the Crowne Plaza Hotel in Knoxville, TN, from 13-17 August, 2009. You can check their web site, www.crowneplaza.com, for more information on the Knoxville edition of the hotel. Sharon Patton and Charles Faulkner will be the co-chairs of the Local Committee. Watch for more information both on the ASP web site and in the Journal.

Other Meetings.

EMOP 10 [August 24 - 29, 2008] THE 10TH EUROPEAN MULTICOLLOQUIUM OF PARASITOLOGY The Société Française de Parasitologie and the European Federation of Parasitology welcome you in Paris for the 10th European Multicolloquium of Parasitology (EMOP 10). During the same week the XXth International Congress of Zoology will be held in Paris. Combined registration fees will be proposed in order to attend both conferences.

THE XIITH INTERNATIONAL CONGRESS OF PARASITOLOGY (ICOPA) (2010). To be held in Melbourne, Australia, from **15-20th August 2010** at the new Exhibition and Convention Centre. All are invited to join the parasitology community at this exceptional facility that lies in the heart of Melbourne in close proximity to the scenic Yarra River and the associated parks, multicultural restaurants, cafes and bars.

WORLD ASSOCIATION FOR THE ADVANCEMENT OF VETERINARY PARASITOLOGY (WAAVP) 2009 CALGARY, CANADA, 8-13 AUG
The 54th Annual Meeting of AAVP will be held in conjunction with the 22nd International Conference of the World Association for the Advancement of Veterinary Parasitology (WAAVP) in Calgary, Canada, August 8-13, 2009.
www.WAAVP2009.com

57TH ANNUAL MEETING OF THE AMERICAN SOCIETY OF TROPICAL MEDICINE AND HYGIENE (ASTMH) 2008
The annual meeting of ASTMH will be held on December 7-11, 2008 at the Sheraton New Orleans, New Orleans, LA. Information about the annual meeting can be obtained by contacting ASTMH Headquarters at http://www.astmh.org.

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Wildlife Disease Association - 2008

The mission of the Wildlife Disease Association (WDA) is to acquire, disseminate, and apply knowledge regarding health and disease of wild animals in relation to their biology, conservation, and ecology including interactions with humans and domestic animals. The Journal of Wildlife Diseases is published quarterly and contributing authors and other WDA members work for universities, governmental agencies, zoological institutions, private businesses, and agricultural and livestock agencies throughout the world. To learn more about our organization, visit www.wildlifedisease.org. We invite you to join us at the annual WDA meeting in Edmonton, Alberta in August 2008. **The theme this year is "Wildlife Diseases: Northern and Western Frontiers."** For more information, please visit the WDA conference website: http://www.biology.ualberta.ca/parasites/WDA08/

Check the web site of David Gibson for more meeting data:

http://www.diplectanum.dsl.pipex.com/purls/index.htm

International Biogeography Society Fourth Biennial Meeting

January 8-12, 2009
Merida, Mexico
http://biogeography.org

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FIELD PHOTOGRAPHY *- NEW FEATURE OF THE ASP NEWSLETTER*

This photograph was made by slg during field work in Bolivia, 1993. The day before, the stream below was clear, with small deep clear pools of refreshingly cool water. We wondered why the cable was set up that we could see hanging across the valley about 30 feet above the little stream. We all went across to see the "Oilbird Caves" and we set about 200 traps for rodents on the other side of the little stream. That night it started raining and it continued all the next day. By nightfall it was clear why the cable was strung across the "little" stream. We all had fun learning how to hook into the rope sling to shoot across the stream with a box of Sherman traps hanging off of our shoulders.

To participate in the "Field Photography" section of the ASP Newsletter, send your digital photograph to slg@unl.edu along with an explanation of the content of the photograph and what field experience you were having at the time.

Happy Photographing in the Field.

Scott L. Gardner -editor

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PARASITIC DISEASE OF THE QUARTER

[Echinococcosis - *polycystic, cystic, alveolar hydatid disease*]

Human echinococcosis (hydatidosis, or hydatid disease) is caused by the larval stages of cestodes (tapeworms) of the genus *Echinococcus*. *Echinococcus granulosus* causes cystic echinococcosis, the form usually encountered in both North and South America; *E. multilocularis* a species with a primary distribution in the Holarctic, causes alveolar echinococcosis; *E. vogeli* with a southern Nearctic and Neotropical distribution causes polycystic echinococcosis; and *E. oligarthrus*, also occurring in the Neotropics, is an extremely rare cause of human echinococcosis.

Echinococcus vogeli, life cycle - the normal sylvatic definitive hosts include: Bush dog *(Speothos venaticus)* - intermediate hosts usually include the paca = *Cuniculus paca*.

Echinococcus oligarthrus, life cycle - the only species of *Echinococcus* known to use a felid as a primary definitive host uses Jaguar and Ocelot and other felids as primary hosts and usually rodents of the family Echimyidae as intermediates.

Echinococcus granulosus, life cycle - this species has a cosmopolitan distribution having been transported widely by humans with their domestic animals. With a primary definitive host of any of the family Canidae the intermediate host in the domestic cycle is can be a sheep, goat, or swine among others. The original sylvatic cycle was a boreal in origin occurring primarily in ungulates and wolves.

Echinococcus multilocularis, life cycle - a species that uses rodents of the family Arvicolidae (voles and kin) as intermediates and canids (rarely felids) as the definitive hosts in the sylvatic cycle.

This summary on echinococcosis was taken directly from the CDC web site: (http://www.dpd.cdc.gov/dpdx). Some data were modified by the editor.

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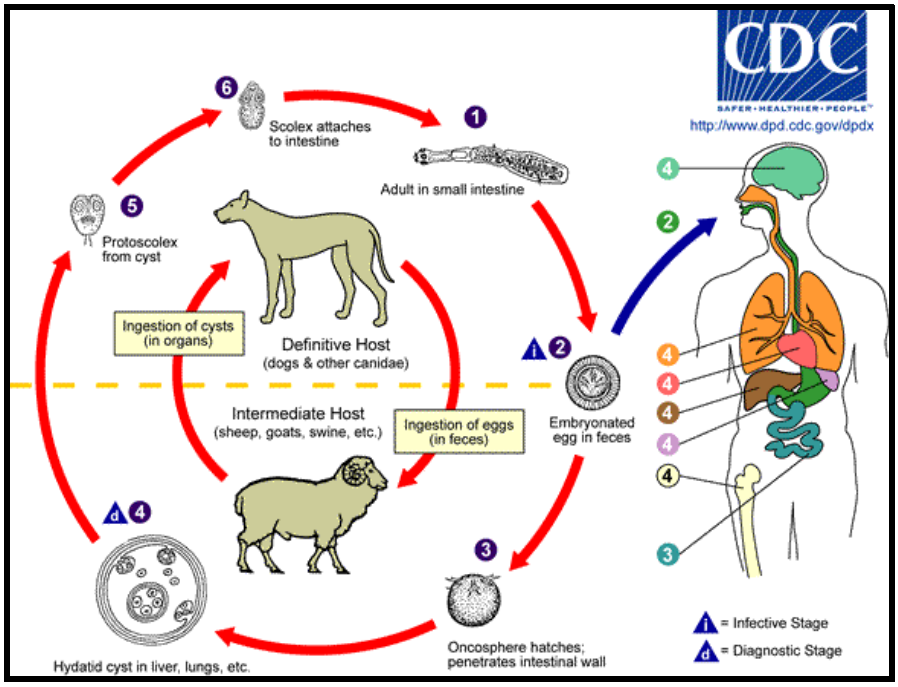

Fig. 1 shows the generalized life cycle of tapeworms of the genus *Echinococcus*. In the case of *E. vogeli* the definitive host is the bush dog and the intermediate host is the paca.

JOB OPENINGS

Associate Director Cedar Point Biological Station
University of Nebraska

Description of work:

Coordination and supervision of all activities at or relating to the Cedar Point Biological Station. Candidates must have a commitment to work with students and be prepared to handle all aspects of the field station operation. Hands-on duties are part of the position.

Significant daily effort in planning and supporting the summer programs in teaching and research (recruiting, logistics planning, and coordination), arranging housing/dining and facilities maintenance, arranging space for research projects, and

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managing a large natural area are expected. Supervision of full-time and temporary kitchen and maintenance personnel is included.

The Associate Director develops fund-raising programs to support facilities, teaching and projects, and implements new initiatives.

The Associate Director must live at the Station during the active summer season and be based at Lincoln the remainder of the year. The station is located in Ogallala, in Western Nebraska http://cedarpoint.unl.edu/ Map coordinates: 41.210745, -101.649988 on Google Maps.

A successful candidate should demonstrate a strong motivation for contributing professionally to the teaching and/or research and/or outreach mission of the station.

Outreach could include leading field trips for classes and the public, developing and maintaining a dynamic docent program, and/or establishing a K-12 education and other non-traditional outreach programs. In addition the Associate Director will be actively engaged in fund-raising supporting these programs.

Research could include any site based natural history, environmental or biological effort, especially focused on long-term data collection and developing funding to support these activities.

Teaching could include teaching summer courses and/or the development of new initiatives in teaching.

Qualifications:

MS required, Ph D preferred

Academic training in the life and/or environmental sciences and experience with field stations and/or field biology research and/or teaching field courses and/or public outreach.

Significant experience in one or more of the following: environmental education, environmental research, nonprofit management, program development, resource management, science or environmental administration, or a related field.

Ability to interact productively with students, station users, administrators and neighbors.

Self-sufficiency. Excellent oral and written communication/presentation skills. Strong problem-solving skills, organizational skills and ability to plan, and direct others in the formation and implementation of programs.

Computer skills (i.e. Word, Excel) required; database, GIS, and web design preferred.

Experience in administration or budget management preferred.

Special Requirements: Altered work schedule, must be available on weekends, evenings and holidays during the summer, as needed. Must live on site (housing provided) from May until August and in Lincoln from August to May.

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Application Process:
Application deadline: August 15, 2008
Starting Date: October 1, 2008
Salary range: $35,000 - $45,000

To apply you need to submit the following on the UNL website:

Application letter
Resume / CV
List of three references

http://employment.unl.edu
Position number: 00006929
Requisition Number: 080587
For more information contact:
Johannes (Jean) Knops
Director Cedar Point Biological Station &
Associate Professor
School of Biological Sciences
University of Nebraska
348 Manter Hall
Lincoln, NE 68588

Phone (402) 310-3904
Email: jknops2@unl.edu

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Ecology of Infectious Disease Postdoctoral Researcher

Postdoctoral Researcher sought to join a collaborative program examining the interactions between Sudden Oak Death (SOD) and tick-borne disease risk in California. Principal Investigators on the project are Dr. Richard S. Ostfeld (http://www.ecostudies.org/people_sci_ostfeld.html) at the Cary Institute of Ecosystem Studies, Dr. Cheryl Briggs (http://www.lifesci.ucsb.edu/eemb/faculty/briggs/) at UC Santa Barbara, and Dr. Robert Lane (http://espm.berkeley.edu/directory/fac/lane_r.html) at UC Berkeley. The postdoc will lead efforts to understand how SOD-induced changes in the community of vertebrate hosts for ticks affects tick infection with *Anaplasma phagocytophilum*, an emerging tick-borne pathogen. The postdoc will be centered at the Cary Institute of Ecosystem Studies in Millbrook, NY but could spend a portion of the year conducting field work in California. Ph.D. and excellent laboratory and/or quantitative skills are required. Desired start date in autumn 2008, for likely 2-year term. Please send CV, representative publications, and three letters of recommendation by email to: Dr. Richard S. Ostfeld, c/o Human Resources, Cary Institute of Ecosystem Studies, Job #08027, Millbrook, NY, Rostfeld@ecostudies.org (with a cc: to jobs@ecostudies.org). Closing date 20 August 2008.

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Cell and Molecular Biology of Leishmania

A Postdoctoral Fellowship is available in Cell Biology for an individual with a Ph.D. in biochemistry, microbiology or molecular biology. The successful candidate will join an active group studying the cell biology of Leishmania and related trypanosomatid parasites (a group of parasitic protozoa causing human disease). Ongoing project areas include: 1) characterization of secretory and surface membrane enzymes

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critical to parasite growth, development, and survival; 2) molecular dissection of genes encoding and regulating such proteins; 3) developmental expression and cellular targeting of such proteins; and 4) functional characterization of components of both the endo- and exocytic pathways in these organisms. Visit our Web site, http://www3.niaid.nih.gov/labs/aboutlabs/lpd/cellBiologySection/dwyer.htm, and check PubMed for our latest publications.

These positions offer a unique opportunity to have impact in a rapidly developing area highly relevant to human disease. No prior experience in parasite research is necessary.

NOTE: Fellowships available only to those with less than 3 years of postdoctoral experience. Salary is commensurate with experience.

Please send curriculum vitae, list of publications, and names of 3 references to:

Dr. Dennis M. Dwyer
Head, Cell Biology Section,
Laboratory of Parasitic Diseases,
Building 4, Room 126,
National Institute of Allergy
and Infectious Diseases,
National Institutes of Health
Bethesda, MD 20892-0425
Telephone: (301) 496-5969
Fax: (301) 402-0079
E-Mail: DDwyer@niaid.nih.gov

The NIH is dedicated to building a diverse community in its training and employment programs.

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Contributed Paper

Editors note: As I have been prodding readers of the newsletter to send in contributions, things have started to appear in my mail. Still, contributions are coming in at a somewhat slower pace than I and my associate editors would like. The following article was sent to me for consideration for publication in the "ASP Newsletter" and after it was reviewed by several colleagues, I have decided to publish it here. Although this is not the usual form that these kinds of data are put into print or onto the web, the data contained in this contribution does add to the knowledge base of world wide parasitology. Some is practical and some common sense, but it does make you think - and this is what we are trying to encourage - thinking and positive action. Comments are welcome.

To have your comments considered for publication, send them to the editor (Scott L. Gardner, slg@unl.edu).

Review Paper

Conflicting results in biomedical research: factors to be considered and implications of these studies.

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Science encompasses a system of acquiring and organizing knowledge based on the scientific method or via research that can be repeated, which seeks to explain the complexities of nature. As Wilson Jr. observes; advancement in science arises from the collective judgment of scientists, in so far as there is substantial agreement. "*The fact that there are very large areas of agreement, in spite of the individualistic, antiauthoritarian nature of science, is partial evidence for the validity of scientific methods*" (Wilson Jr. 1990). However, there are cases where agreement is difficult to come by, and what we have are several different "truths"; and yet in a few other cases, there has been *universal agreement for an untruth.* The latter has however

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mostly been the case with *"sweeping generalizations rather than basic scientific observations"* (Wilson Jr. 1990). The role of biomedical research in modern society cannot be overemphasized. Biomedical research is crucial as a source of new knowledge; a means to identify problems and solutions and as a means to implement, monitor and evaluate public health programs. Biomedical science research aims at advancing human welfare, knowledge and understanding of the numerous health problems afflicting local populations; with the ultimate goal of alleviation of human suffering caused by diseases and infections. A lot of biomedical/health research takes place around the world, focusing on a broad range of subjects. Interestingly, some of the research on the same areas has often produced conflicting results. Studies (ranging from clinical, behavioral, to laboratory) that have produced conflicting results are the focus of this review, which addresses some of these studies, highlights some factors to be taken into consideration, and provides comments on the implications of these conflicting results.

Malaria - Helminths co-infections

Research in parasite ecology has often focused on single parasites in individual hosts. However, we know that under natural conditions, one host may be infected by more than one parasite (as is the case with *Plasmodium* and most helminths occurring in the same endemic foci), which then makes the dynamics of interactions very complex. Because of this, the immunological networks activated as a result of these infections are multi-factorial and can be complicated to study (Halters and Yazdanbakhsh 2006). It is now widely acknowledged that the concomitance of various parasitic infections in human populations can induce modifications of immune response specific to each organism thereby induce changes in the clinical expression of each species. For example, malaria-helminth co-infections frequently occur in disease endemic areas. There is evidence that interactions occur between these species, although it is unclear whether this effect is beneficial, harmful, or neutral to the host. In general, control of malaria parasitemia is largely dependent on the type 1 immune response, while immunity to helminths is largely driven by the type 2 responses. The type 1-dependent control of malaria parasitemia might be impaired by the type 2 milieu of preexisting helminth infection. Alternatively, immunomodulatory effects of helminths might affect the likelihood of malarial immunopathology (Graham *et al.* 2005). In addition, immunological responses to malaria parasites may affect the balance between pro- and anti-inflammatory cytokines (tumour necrosis factor (TNF)-α, interleukin (IL)-6, and IL-10) on the one hand and T helper (Th) type 1 and 2 cytokines on the other. Infection with helminths has a profound effect on the immune system resulting in polarization towards Th2, characterized by high levels of cytokines such as interleukin-4 (IL-4), IL-5, IL-13 and high serum levels of immunoglobulin E (IgE) (Halters and Yazdanbakhsh 2006).

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Several studies on the effect of malaria-helminth co-infections, however, have produced conflicting results. The differences have been with respect to findings and conclusions about the strength and direction of any interaction between the parasites. Some studies found that infection with helminths increases the susceptibility to malaria infection. For instance, both *Schistosoma mansoni* and *Schistosoma haematobium* infections have been shown to increase the incidence of malarial fevers (Sokhna et al. 2004; Spiegel et al. 2003). An association of helminth infection with an increased risk for malaria incidence has also been confirmed in Thailand (Nacher et al. 2002). It is estimated that, if *Ascaris* has an odds ratio of 0.5 and a prevalence rate of 50% in malaria endemic countries (it is often higher in children), the number of cerebral malaria cases avoided as a consequence of *Ascaris* infection would be between 650,000 and 2,650,000 per year and the number of deaths avoided between 97,500 and 795,000 per year worldwide (Nacher et al. 2000).

In Zaire, a study on mothers and children described a positive association between infection with *Ascaris* and the occurrence of *Plasmodium falciparum* (Tshikuka et al. 1996). Helminth infections have been documented to slow down the development of antimalarial immunity (Druilhe et al. 2005). A greater risk (increased incidence) of *P. falciparum* malaria has been observed in people infected with intestinal nematodes than in their non-infected counterparts (Nacher et al. 2002; Spiegel et al. 2003). A significant increase in malaria was reported in children treated for helminth infections compared with a group of children who received a placebo (Keusch and Migasena 1982).

In contrast to studies described above, some recent studies have reported a protective effect of infection with helminths. In Senegal, *S. haematobium* was shown to have had a protective effect on infection by decreasing *P. falciparum* densities as compared to helminth-free children (Briand et al. 2005). However, this was only the case for light infections of *Schistosoma haematobium* (1–9 eggs/10 ml of urine), since no significant association with malaria densities was found for higher infection intensities determined by egg output. In this same study, no association between intestinal helminth infections (mainly *Ascaris lumbricoides*) and malaria densities was found, indicating that not all species of helminths might have the capacity to modify the course of malarial infections. Unfortunately, the study did not address clinical symptoms of malaria. In a recent study that compared *S. haematobium*-infected children with helminth-free children in Mali, infected children demonstrated a delayed time to first symptoms of clinical malaria, fewer malaria episodes and lower parasite densities, although only in the younger age group (4–8 years of age) (Lyke *et al.* 2005). Again, the age-associated protective effect on malaria seemed stronger in the children with light *S. haematobium* infections (< 50 eggs/10

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ml of urine) than in the children with higher egg loads, although this was only significant for the time to first malaria infection. They concluded that underlying schistosomiasis is associated with protection against clinical *falciparum* malaria in an age-dependent manner. Infection by *Ascaris lumbricoides* has been shown to protect subjects in Thailand from cerebral malaria (Nacher et al. 2000). In another study (Nacher et al. 2001), it was found that pre-existing *Ascaris* infection could increase tolerance of the host to different *Plasmodium* species, thus facilitating their coexistence.

Inhibition or exacerbation of allergic and autoimmune diseases by infection

Studies on the impact of infections on the inhibition or exacerbation of allergic and autoimmune diseases have also produced conflicting results. Data from some of these studies suggests that infections or the exposure to non-pathogenic bacteria or helminths protect individuals from developing some autoimmune and atopic disorders. Most of these findings support the 'hygiene hypothesis', which attributes the rise in autoimmune and atopic disorders to a lack of infections that normally keep the immune system balanced by inducing immunoregulation (Kamradt et al. 2005).

Autoimmune infections

It has been suggested that infections within the first year(s) of life decrease the risk of developing type 1 diabetes (T1D), inflammatory bowel disease (IBD) or multiple sclerosis (MS) (Bach 2002; Loftus 2004; Ponsonby et al. 2005). Exposure to a wide variety of infectious agents, including viruses (Bach 2002), *Mycobacterium* (Qin et al. 1993), *Salmonella* (Zaccone et al. 2004) and helminths (Zaccone et al. 2003), protects the diabetes-susceptible non-obese diabetic (NOD) mice from spontaneously developing T1D (Qin et al. 1993). There are reports suggesting a protective role for pathogens in murine models of IBD (Moreels et al. 2004; Reardon et al. 2001). It has also been established that the incidence and severity of experimental autoimmune encephalomyelitis (EAE), can be slightly reduced by a pre-established infection with parasites or *Mycobacterium* (La Flamme et al. 2003; Sewell et al. 2003). A protective role for pathogens in rodent models of arthritis has also been suggested (Moudgil et al. 2001).

However, other studies have shown that infections do not always protect from the development of autoimmunity. This is evidenced in some autoimmune diseases which have been shown to occur shortly after infection with other pathogens. Examples include; post-infectious encephalitis disseminata and rheumatic fever (Bach 2002; Kamradt and Mitchison 2001); lyme arthritis and spondylarthropathies develop after bacterial infections (Kamradt 2002); congenital rubella infection and T1D (Bach 2002) and exacerbation of MS is 2–3 times more likely to occur during, or shortly after, common respiratory, gastrointestinal or urological infections (Bach 2002; Kamradt and Mitchison 2001).

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Allergies and atopy

The development of asthma and allergy, is thought to be favored by the absence of infections (Herz et al. 2000; Maizels and Yazdanbakhsh 2003). Bacterial infection, (in particular with *Mycobacterium tuberculosis* and *Mycobacterium bovis*) (Kamradt et al. 2005) and *Chlamydia trachomatis*, *Listeria monocytogenes* or lactic acid bacteria (Han et al. 2004; Repa et al. 2003; Sayers et al. 2004; Trujillo and Erb 2003) is thought to be one factor associated with protection from allergic disorders. Since these infections induce strong Th1 responses and because IFN-γ has inhibitory effects on Th2 responses, it has been speculated that infections with *Mycobacterium* might protect humans from developing allergies (Kamradt et al. 2005). Indeed, it has been reported that a positive tuberculin test result was associated with a decreased risk of atopy and asthma in Japanese school children (Trujillo and Erb 2003). However, a subsequent evaluation in the same cohort failed to reproduce these findings (Trujillo and Erb 2003). Recently, the effects of BCG vaccination and its protection against allergic disorders have been documented (da Cunha et al. 2004; Marks et al. 2003; Trujillo and Erb 2003).

Other studies have produced results showing an opposing effect to allergy protection by bacterial infections. These studies found that some types of bacteria are associated with increased allergic responses. *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* can exacerbate asthma (Blasi 2004; Lieberman et al. 2003) or allergen-induced bronchial hyper-reactivity (Hardy et al. 2002) and *Staphylococcus aureus* can trigger the exacerbation of atopic dermatitis (Kamradt et al. 2005). It is argued that exotoxins from *S. aureus* induce vigorous T-cell activation and cytokine release, which increases the already established Th2 response in the skin (Herz et al. 2000). Allergic inflammation in the airways is enhanced by infections with *Bordetella pertussis* (Ennis et al. 2004).

Evidence exists suggesting that infections with viruses, e.g. hepatitis A (Marsland et al. 2004; Matricardi et al. 2000; Wohlleben et al. 2003) protect against atopy development, and these anti-allergic effects of influenza A virus infection are associated with Th1 responses (Wohlleben et al. 2003). However it has also been shown that Th1 responses do not necessarily protect from allergy but can sometimes even exacerbate allergic responses (Kamradt and Mitchison 2001). For instance, respiratory viruses, such as rhinovirus (RSV), influenza A and metapneumovirus, can exacerbate the symptoms of asthma in humans or can directly induce wheezing (Herz et al. 2000; Williams et al. 2004). In addition, RSV infections in the first year of life might be a risk factor for the development of childhood asthma and can directly induce airway eosinophilia and hyper-reactivity (Herz et al. 2000). Low grade RSV infection, however, has been shown to protect mice from

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allergen-induced inflammation in the airways (Kondo et al. 2004), suggesting that the severity and frequency of an RSV infection might also be important in determining what effect the infection will have on the development of asthma (Kamradt et al. 2005). In allergen-exposed mice, Influenza A virus induces increased airway responsiveness (Dahl et al. 2004; Herz et al. 2000; Marsland et al. 2004; Wohlleben et al. 2003). However, depending on the time point of infection, influenza A could also protect from allergen induced airway eosinophilia (Wohlleben et al. 2003).

Controversy continues as to whether the Th2 response to helminths is protective or acts to dampen potentially damaging inflammatory responses against abundant and well-established tissue parasites (Allen and Maizels 1997). Nevertheless, helminthic infections have been associated with a lower incidence of atopic diseases (Maizels and Yazdanbakhsh 2003). Infections with schistosomes or hookworms are associated with a reduced atopic phenotype (Herz et al. 2000; Maizels and Yazdanbakhsh 2003). Retrospective (Herz et al. 2000) and interventional studies (van den Biggelaar et al. 2004) have shown that anti-helminthic chemotherapy results in increased levels of skin-test reactivity against common allergens. Mice infected with *Strongyloides stercoralis* (Wang et al. 2001) or *Nippostrongylus brasiliensis* (Wohlleben et al. 2003) have been found to express suppressed pulmonary allergic responses.
Helminths are known to induce strong Th2-type responses in contrast to most other types of infections (Maizels and Yazdanbakhsh 2003). It is therefore plausible that helminths promote allergic disorders by generally enhancing Th2-type inflammation (Kamradt et al. 2005). Indeed, it has been established that infections with helminths can directly induce an asthma-like phenotype in mice and rats (Herz et al. 2000) or can lead to the breakdown of oral tolerance against allergen (Hurst et al. 2001). Frequent occurrence of allergic symptoms has been reported in children seropositive for *Toxocara* or *Ascaris* species than in seronegative children and anti-helminthic treatment ameliorates asthma (Herz et al. 2000; Maizels and Yazdanbakhsh 2003). Chronic helminth infections or the exposure to helminth-derived products have also been linked to urticaria (Demirci et al. 2003).

Scientific evidence exists that clearly shows that infections have multiple and seemingly opposing effects on both autoimmune and allergic diseases [for review see (Kamradt et al. 2005)]. Numerous types of infections have been shown to have an impact on atopy but not on autoimmunity and visa versa. Helminths and mycobacteria seem to be particularly good at protection and the timepoint (infections before the onset of atopy or autoimmunity have the greatest impact), age at infection, route, localization and dose of the infection all have a role (Kamradt et al. 2005).

Breast-feeding and intelligence in children

Currently, there is a debate within the

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scientific cycles as to whether breast-feeding promotes intelligence in children. The duration of breastfeeding has been shown to affect the intelligence quotient (IQ) (Angelsen et al. 2001). The study shows that babies who are breast-fed for at least six months grow to be more intelligent than their peers who are breast-fed for less time. This is supported by another study that involved a meta-analysis of 20 articles and showed that breast-fed babies' IQ is higher than that of formula-fed babies (Anderson et al. 1999). This study adjusted for factors that may influence intellect, including the mother's age and intelligence, birth order, race, birth weight, gestational age and socioeconomic status, and found that breastfeeding may raise a child's IQ by more than five. Results of an 18 year study of 1,000 New Zealand children concluded that children who were breast-fed achieved consistently higher IQ scores, higher grades, higher classroom performance ratings and better high school achievements than non-breastfed children (Horwood and Fergusson 1998). It has also been reported that children who were breast-fed performed better in tests of intellectual competence than those who were not (Lucas et al. 1992). Studies on neurological and cognitive outcomes in breastfed children found that early visual acuity and cognitive function of these children is greater than in non-breastfed children (Jorgensen et al. 1996; Lucas et al. 1992).

However, several other studies dispute the view that breast-feeding promotes intelligence. A recent study (Der et al. 2006) (which elicited the current debate), found a positive impact for breastfeeding on intelligence only when other potential contributors such as the mother's IQ and the parents' educational and economic status were not taken into consideration. This study included 5,475 children and mothers in the U.S. who participated in an ongoing youth development survey. The study found no significant difference in intelligence among the breastfed and nonbreast-fed siblings when they considered the variables above. It was also found that the mother's IQ was by far the most important variable, accounting for 70-75% of the difference [between children who were and were not breast-fed]. The position held by The US Department of Health and Human Services Office on Women's Health (USDHHS 2000) is that the observation that cognitive function of breast-fed children is greater than in non-breastfed children has not been conclusively proven. This position by US DHHS is supported by results from other studies (Jacobson et al. 1999; Richards et al. 1998). Although the majority of studies concluded that breastfeeding promotes intelligence, the evidence from higher quality studies is less persuasive (Jain et al. 2002). It is further argued that no convincing evidence exists regarding the comparative effects of breastfeeding and artificial feeding on intelligence (Jain et al. 2002).

Energetic costs of an immune response

Existing evidence for the direct mechanisms, particularly energetic costs, underlying indirect costs of immune

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activity in non-human vertebrates is currently equivocal. Unfortunately, a majority of studies on energetic costs of immune responses have involved the use of non-replicating or non-living antigens, and it remains to be investigated if other types of immune responses, to relevant living pathogens, are energetically more costly. Different authors (Demas et al. 1997; Martin et al. 2003; Smits et al. 1999) have argued that by using non-replicating antigens instead of an experimental infection with a parasite, it is easy to assess the energetic costs associated with an immune response alone, irrespective of the costs that would be imparted by parasite proliferation and subsequent tissue damage and repair. However, we know that this is far from what happens in natural populations in the field, and non-replicating antigen-induced immune response can only be a crude surrogate for a parasite-induced immune response. Therefore, there is a critical need for use of live replicating antigens, which will induce a more ecologically relevant antigenic challenge. Nevertheless, several studies have tried to quantify the energetic costs of an immune response, using resting metabolic rates (RMR) or basal metabolic rates (BMR) as an index of energy expenditure. Most of these studies have found significant increases in the RMR/BMR associated with an immune response. Table 1 summarizes some studies.

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Table 1. Summary of some studies on energetic costs (percentage increase in resting or basal metabolic rate as compared to controls) of an immune response to various antigenic challenges.

Species	Immune Challenge	Increase in RMR	References
Homo sapiens (Human)	AIDS	25%	Grunfeld et al., 1992
	AIDS	9%	Hommes et al., 1990
	HIV	17%	Grinspoon et al., 1998
	HIV	12%	Melchior et al. 1991
	Sepsis	30%	Kreymann et al. 1993
	Sepsis	30%	Carlson et al., 1997
	Sepsis and injury	57%	Clark et al., 1996
	Sepsis	49%	Plank et al., 1998
	Typhoid vaccination	16%	Cooper et al., 1992
	Sickle cell disease	15%	Borel et al., 1998
Rattus norvegicus (lab rat)	IL-1 infusion	18%	Tocco-Bradley et al. 1987
	Inflammation	28%	Cooper et al., 1994
Mus musculus (lab mouse)	KLH challenge	30%	Demas et al., 1997
	Heligmosomoides bakeri	9% & 14%	Kristan and Hammond 2000, Kristan and Hammond 2001
	Heligmosomoides bakeri	0%	Kristan and Hammond 2006
Ovis aeries (sheep)	Endotoxin	28%	Fewell et al., 1991
	Endotoxin	10-49%	Baracos et al., 1987
Great tits *Parus major*	SRBCs	9% (BMR)	Ots et al., 2001
Blue tits *Cyanistes caeruleus*	Diptheria-tetanus vaccine	0% (BMR)	Svensson et ao., 1998
Chicken (domestic)	SRBCs	0%	Henken and Brandsma, 1982

NOTE: 0 % indicates no significant increase.
AIDS - Acquired immune deficiency syndrome; HIV - Human immunodeficiency virus; KLH - Keyhole limpet hemocyanin; SRBCs - Sheep red blood cells.

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It is interesting to note that some of the studies in Table 1 above (Henken and Brandsma 1982; Kristan and Hammond 2006; Svensson et al. 1998) found no significant differences in the change in metabolic rates between the experimental and control groups. Whereas these three studies are not conflicting sensu stricto, the fact that they observed no significant change in RMR/BMR deserves mention here.

Antibody responses in mice infected with *Heligmosomoides bakeri* (Nematoda)

Numerous laboratory studies (rodent-helminth systems) continue to employ Heligmosomoides bakeri, a trichostrongyloid nematode that has been proposed (Monroy and Enriquez 1992) as a model of human hookworm disease and nematode infections of veterinary importance e.g Ostertagia ostertagi. A recent study (Behnke et al. 2003), found a clear difference in anti-adult IgG1 levels between two strains of mice (SWR and CBA) infected with H. bakeri. This was in tandem with earlier reports that adult worms induce a polyclonal B cell stimulation, with CBA mice, having the greater number of adult worms, showing the larger response (Chapman et al. 1979). They acknowledged that this finding contrasted with results of earlier studies utilizing single pulse infections where the responder mice strains such as SWR, SJL and NIH were shown to have faster and more intense adult worm specific IgG1 responses (Ben-Smith et al. 1999; Wahid and Behnke 1993). In addition, evidence exists that IgG1 antibodies to adult worms, in repeatedly infected mice, are host protective (Pritchard et al. 1983). It was not clear why CBA were unable to control worms under the trickle infection protocol used in their study.

Are BALB/c mice a weak or strong responder to *Heligmosomoides bakeri* (Nemata) infection?

There is close agreement between different researchers as to which mouse strains are weak responders to H. bakeri infection, with the exception of BALB/c. Some authors (Behnke and Robinson 1985) found the performance of BALB/c to range from weak (<20% protection) to strong (almost 100% protection), and therefore designated this strain as an intermediate responder. Some studies using BALB/c have found them to be inferior responders relative to NIH mice (Behnke and Wakelin 1977) and LAF1/J mice (Jacobson et al. 1982). However, other studies (Hurley and Vadas 1983; Mitchell and Prowse 1979; Prowse et al. 1979) have found BALB/c mice to be among the strong responder strains. In addition, BALB/c have been reported to be much more resistant than C57BL/6 to challenge infections (Cypess and Zidian 1975; Enriquez et al. 1988a; Scott 1991).

Factors to be taken into consideration to address conflicting results and implications of such studies.

Several factors need to be addressed in dealing with conflicting studies. The epidemiological settings and

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methodology used in some of the studies must be evaluated if any standard comparisons are to be made.

In malaria-helminth co-infections, there is need to study each type of helminth infection separately, since each might have different effects on the course of malaria (Hartgers and Yazdanbakhsh 2006). The intensity of a helminth infection must be reported, since the intensity might be an important determinant for the outcome of immune responses to the malaria parasite (Briand et al. 2005; Lyke et al. 2005; Sokhna et al. 2004). The analysis of the immunological profiles at the level of the cellular immune system will shed more light on the mechanisms that are involved, and murine co-infections will help to dissect the molecular immunological pathways involved in controlled models (Hartgers and Yazdanbakhsh 2006). Understanding, at the molecular level, how these parasites modify the responses of cells such as dendritic cells or macrophages, central to regulation of the immune system, will enhance our understanding of how infection and disease are controlled during co-infection (Hartgers and Yazdanbakhsh 2006).

There is need to control for confounding factors or effect modifiers in scientific research. For instance, the traditional approach among researchers towards controlling for confounding and modification in parasitological surveys is either through matched case control designs or by stratifying on common confounders such as age and sex (Booth 2006). Location of residence is among significant confounders in malaria-helminth co-infections. Simply observing an association between two species of parasite (or their associated morbidities) in the absence of controlling for location cannot be interpreted as evidence of biological interaction (Booth 2006). Controlling for confounding factors can help reduce heterogeneity in several parameters that frequently remains unexplained, and will ultimately lead to correct inferences.

Another area of concern is the impact pre-existing co-infections may have on vaccine testing and efficacy. Chronic helminth infections induce strong type 2 and regulatory immune responses, and are known to influence immune activity to other antigens such as allergens and vaccines (Hartgers and Yazdanbakhsh 2006). Many malaria vaccines have been shown to target parasite antigens that have the capacity to induce specific antibodies capable of inhibiting parasite growth. In the ideal sense, vaccines should also stimulate an efficient CD4+ Th1 response (Hartgers and Yazdanbakhsh 2006). Infections with helminths have been shown to affect immune responses to tetanus vaccines and to (Cooper et al. 1998; Elias et al. 2005; Sabin et al. 1996). It has been suggested that helminth infections may also affect the induction of an efficient Th1 type of immune response to a potential malaria vaccine, and the presence of helminth infections in a population should be considered as a

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confounding factor for the assessment of the efficacy of malaria vaccines (Hartgers and Yazdanbakhsh 2006). These authors further argue that a better answer to the question of whether helminth infections affect course of malarial infection and disease will come from longitudinal studies with a placebo-controlled anti-helminth treatment design. All these factors need to be taken into consideration when designing and evaluating vaccines.

It is clear that malaria-helminth co-infections pose a significant challenge with real world consequences. Arguing from the point that helminth co-infection ameliorates cerebral malaria (Nacher et al. 2000), mass anti-helminthics will be disastrous and of no benefit where malaria is endemic. On the other hand, the additional burden of helminth infection may increase the severity of malaria infection (Spiegel et al. 2003). This stresses the importance for clear, accurate and consistent results on malaria-helminth co-infections. Scientists must carefully evaluate the impact that conflicting results on malaria-helminth co-infections might have on the implementation and efficacy of control interventions such malaria vaccines. The interaction between malaria parasites and helminths is complex on its own, and this is worsened when there is no consensus among studies in this subject.

It is without doubt that the immune system benefits from regular encounters (priming) with some pathogens, which clearly has implications for interventions in autoimmunity and atopy. Available evidence indicates that under certain circumstances some infections can inhibit, induce or exacerbate allergic or autoimmune diseases (see (Kamradt et al. 2005)). The current, most popular, hypothesis on how infections inhibit the development of both types of disease, suggests that microbial molecules trigger certain programs, such as IL-10 production by dendritic cells (DCs), which thereupon instruct the development of regulatory T (Tr) cells (Adams et al. 2004; McGuirk et al. 2002; Wakkach et al. 2003). Some authors (Kamradt et al. 2005) have argued that although increasing evidence for this hypothesis is currently being published, it is far from proven and other mechanisms might contribute to infection-mediated protection. Data from these results needs careful interpretation because it can have far reaching ramifications regarding the development of novel therapeutic intervention strategies aimed at reducing autoimmune responses in humans by using pathogens or commensals. Several studies ranging from BCG vaccination and T1D (Allen et al. 1999; Elliott et al. 1998), BCG and MS (Ristori et al. 1999), probiotic bacteria and pouchitis in ulcerative colitis (Gionchetti et al. 2003), *Trichuris suis* and ulcerative colitis (Summers et al. 2005b), *Trichuris suis* eggs and Crohn's disease (Summers et al. 2005a), *Trichuris suis* and IBD (Summers et al. 2003) provide us with an insight of how conflicting results in this area can play out in the real world.

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The debate on whether breastfeeding promotes intelligence in children is an interesting one. There is a clear need to carry out evaluations on the impact of controlling for maternal intelligence and other confounding factors (e.g. socioeconomic status and maternal education) on this association. The importance of controlling for maternal IQ has been demonstrated (Der et al. 2006) and it has been shown that the association of breast feeding with cognitive performance in children drops from 4.7 to 0.5 points after adjusting for mother's cognitive competence and other socio-environmental measures. Other studies (Jacobson et al. 1999; Rao et al. 2002) support this finding that maternal intellectual enrichment and genetic endowment, as measured by maternal IQ, are crucial potential confounders of the effect of breast feeding on cognitive competence in children. It remains to be determined what really accounts for breast milk's brain boosting power.

There is a general consensus that all physiological processes, including immune responses, consume energy (Coop and Kyriazakis 2001; Demas et al. 1997; Lochmiller and Deerenberg 2000; Svensson et al. 1998); the only fundamental question being how much. The possible reasons given for the no significant change in RMR in some of the studies above (Table 1) include: differences in parasite cultures that produce different levels of infectivity and thereby elicit different degrees of immune response in mice that could differentially affect RMR (Kristan and Hammond 2006); very low sample sizes (Svensson et al. 1998) and the effect of age of chicken (Henken and Brandsma 1982). Science could benefit through the empirical evaluation of the relationship between immune responses to various parasites and RMR/BMR, and between immune eliciting capacity and different parasite cultures in order to make concrete conclusions as to why there are no significant changes in some studies on energetic costs of immune responses.

The SWR and CBA mice share different MHC-linked background genes, with SWR expressing the H-2q haplotype and CBA the H-2K haplotype (Behnke and Robinson 1985). Indeed, it has been established that the immune response phenotype in H. bakeri infection in mice is influenced by both background and major histocompatibility complex (MHC) genes (Behnke and Robinson 1985). This implies therefore, that thoughtful considerations should be made in comparisons between infection with H. bakeri using different mouse strains.

Whether BALB/c should be designated as a weak or strong responder to *H. bakeri* is still controversial. It is apparent that differences in the immunizing protocols that are employed in some of the studies are a likely cause of variation in the results. Indeed, differences in immunizing schedules have been suggested previously to be responsible for these incompatible results (Behnke and Robinson 1985; Scott 1991). This therefore calls for control of the

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immunizing schedules for any meaningful comparisons and concrete conclusions to be made.

CONCLUSION

How can the scientific community benefit from the knowledge from conflicting results in research? Parasitic diseases, caused by a diverse spectrum of eukaryotic organisms represent a major global health problem in terms of social and economic burden. Elimination of these diseases is probably not feasible in our lifetime. As scientists, we must strive towards bridging the gap between some of these conflicting results. One novel way would be to use the "old rule of thumb": - encourage checks and repetitions of scientific work for consistency and reproducibility. In addition, researchers must explore concrete explanations or discussions for lack of consistency and reproducibility within their study settings. Conflicting results may not be a negative phenomenon after all, science is all about stimulation, creativity and critical thinking; and conflicting studies provide the foundation for reflections and new approaches/designs in biomedical research.

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