



# A DIAGNOSTIC AND IMMUNOLOGICAL STUDY OF *SARCOPTES SCABIEI*, CAUSES SCABIES IN BABYLON PROVINCE, IRAQ

Nebras Mohammed Sahi<sup>1</sup>, Hawraa Sabah Al-musawi<sup>2</sup>, Maani Seher Abid AL-Kafaji<sup>3</sup>  
Nada Naji Shaalan<sup>4</sup>, Hawraa Jwad Khadem<sup>5</sup> and Mohammed K. Alhattab<sup>6</sup>

<sup>1,2,5</sup>Department of Biology, Faculty of Science for Women, Babylon University, Iraq.

<sup>3</sup>Department of Microbiology, College of Medicine, Babylon University, Iraq.

<sup>4</sup>Collage of Education for Pure science, Kerbala University, Iraq.

<sup>6</sup>Department of Microbiology, College of Hammurabi Medicine, Babylon University, Iraq.

## Abstract

This research was carried out to explore *Sarcoptes scabiei* parasite illness. This research included 56 samples from the Marjan Specialist Hospital, Dermatology Unit (21 (38%) female samples and 35 (62%) males). These samples were immunologically diagnosed by assessing concentrations of interleukin 6, 4. The outcomes were found to be high in IL6 and IL4.  $969971, 35.932 \pm 61.051 \pm 7.899$  for the 37-47 year age group, whereas the reduced percentage of the two interleukins studied was  $23.418 \pm 5.884 \pm 40.194 \pm 1.349$  for the 15-25 age group compared to the control group where the level was  $2.373 \pm 0.727, \pm 2.246, 969971$ , respectively. For both IL6, IL4, the findings of the present research were statistically evaluated at a substantial rate ( $P < 0.05$ ).

**Key word:** Scabies, IL6, IL4, parasite *Sarcoptes scabiei*.

## Introduction

Scab is a global illness that impacts millions of people. This sort of disease has infected a human body's skin and exterior layers. The symptoms and signs of this disease are rash, rot, itching and blisters. Moreover, there are many of the clinical characteristics of scab ranging from mild to strong destruction. The immune system and infectious reply connected with various clinical Themes stayed typical of poverty. Many microorganisms such as primates have been infected with other types of scab, like lots terrestrial animals and domestic animals. It is bring about the itchiness and parasite is *Sarcoptes scabiei*, which penetrates the lower Epithelial tissue of skin. Scab mites are mains material source that modulates Some aspects of both kinds of immunity, inflammatory host innate and acquired immune response enabling it to avoid detection by the host until a flourishing population can be established (Arlian *et al.*, 1996; Arlian *et al.*, 2003, 2004).

In particular, The regions of the Pacific and Central America have a big proportion from infection of scab illness and the currency in kids is substantially greater

than in teenagers and adults, (Arlian *et al.*, 2003, 2004).

Secondary interactions such as Rheumatic Heart Disease (RHD) and Acute Post-Streptococcal Glomerulonephritis (APSGN) are also associated with this natural distribution (Hoy, *et al.*, 2011). If left untreated, This secondary impact may lead to serious life conditions (Engelman, *et al.*, 2013).

On the other hand, *Sarcoptes scabiei* was primarily due to the genus *Acarus* with nomenclature *Acarus scabiei* DeGeer, 1778 s. This naming has included, so has the division of *S. scabiei* is now placed in the super family Sarcoptoidae and family Sarcoptidae along with lots other ectoparasitic mites of mammalian (Romani, *et al.*, 2015; Zhang, 2011).

Furthermore, scab mite, has clinical and economic significance in human ectoparasitic and other mammals worldwide. All active mite life steps (larvae, protonymph, tritonymph and adults) are mandatory lasting parasites needing to extracellular fluid from the host (plasma) Infiltrate the haven as a natural benefit (Arlian *et al.*, 1988).

\*Author for correspondence : E-mail : wsci.nebras.m@uobabylon.edu.iq

## Mites Pathogenicity

The mites motivate epidermal keratinocytes and dendritic cells with particles in their eggs, feces, excreta, saliva and other secretory goods (glue proteins and hormones), chelicerae, pedipalps and ankles to protect physical activity and crack down organs after extinction. (Arlian *et al.*, 1984).

Sit scab enhance anti-inflammatory cytokine production such as interleukin-1 receptor antagonist (IL-1ra) as keratinocytes and fibroblasts from immune cells in human skin (Morgan and Arlian, 2010). IL-1ra suppresses action of IL-1 ligand Linking cytokine by past the IL-1 receptor existing in many macrophages, neutrophils T-cells, B-cells and natural killer cells, (Morgan and Arlian, 2010; Arend *et al.*, 1998).

Scabies mites can also rein co-stimulatory relationships between cells and T-cells that present antigen. Scab mite essence produces human T-regulating cells for IL-10 secretion. IL-10 performs as powerful anti-inflammatory cytokine by hinder generation and expression of proinflammatory cytokines on antigen-presenting MHC-cells particles. As a result, the interference between MHC-II-antigen complex and T-cell receptor would be significantly reduced to stimulate and proliferate B-cells into plasma cell-generating antibodies (Arlian *et al.*, 2006).

In 2010 (Arlian *et al.*, 2006; Hay *et al.*, 2010) nearly 100 million individuals globally suffer from scab illness and happen in different areas ranging from 0.2% to 71.4%.

In reaction to vital scab, animal skin analogs and monocultures of ordinary animal epidermal keratinocytes and dermal fibroblasts increase the production of the Vascular Endothelial Growth Factor (VEGF). (Morgan and Arlian, 2010; Arlian *et al.*, 2006). VEGF would enhance blood vessels and fluid (plasma) in the pest cave close to jaw parts and mouth mite opening. We find that this liquid is the main source of water and mite nutrition in dried corneal coating (Arlian *et al.*, 1988).

## Material and Method

Blood specimens were taken from March to May from the Marjan Specialist Hospital in Babylon Governorate with age (15-58) for 56 patients with scab. The serum was segregated for 5 minutes by 5000 rpm centrifuges. The sera was subsequently frozen below -20°C until further use. Enzyme Linked ImmunoSorbent Analysis (ELISA) was used to evaluate concentrations of IL4 and IL6 (Elabscience).

## Result and Discussion

New studies targeted at advancing a serological test for skin disease such as scab disease have long been affected by the lack of antigens produced by this kind of parasite. Research has earlier used all body aqueous extract of identical of scab mite to detection that these mites are the exporters of lots antigenic and allergic proteins (Arlian *et al.*, 1988a; Arlian *et al.*, 1994; Arlian *et al.*, 1996a; Arlian *et al.*, 2004a; Morgan *et al.*, 1994; Arlian and Morgan, 2000; Schumann *et al.*, 2001).

Further (Arlian *et al.*, 2006; Arlian *et al.*, 2006; Elder *et al.*, 2006; Elder *et al.*, 2009; Bergstrom *et al.*, 2009; Morgan and Arlian, 2009; Morgan and Arlian, 2010; Mika *et al.*, 2012; Morgan *et al.*, 2013; Reynolds *et al.*, 2014; Swe *et al.*, 2014). Showed that these hidden molecules of the mites behave as modulators of an infested host's immune system. There was no determination of these molecules responsible for the observed antigenic and immunomodulators act of crusty pest extracts. Scab determined IL6 and IL4 by ELISA assay in this present research.

From table 1 shows the level of infection by patient age compared to IL6 pg/ml. Firstly, high IL6 concentration in age 37-47 y was found to be 969971, 35.932 compared to control is  $4.449 \pm 0.761$  for this class of L.S.D scab under ( $P < 0.05$ ) = 24.547. Next, in the control group for the same division,  $35.817 \pm 14.032$  were recorded at 26-36y with  $3.761 \pm 0.816$ . While the low IL6 concentration appeared at 15-25 years of age is  $23.418 \pm 5.884$  control grade of  $2.373 \pm 0.727$ . Finally, in infection and control groups, 48-58 y showed  $31.531 \pm 1.268$ ,  $2.578 \pm 0.462$  respectively.

These findings due to IL-6's impact in infection control since IL-6 is recognized to be a variable in the manufacture of IgA, our study has determined IL-6 was produced during mite infection scab (Hirano *et al.*, 1986).

From table 2 the patient age-specific concentration

**Table 1:** The level of IL-6 in patients with scab disease.

Age group	Groups	Concentration of IL-6 pg/ml Mean $\pm$ S.D
15-25 y	Control	2.373 $\pm$ 0.727
	infection	23.418 $\pm$ 5.884 *
26-36 y	Control	3.761 $\pm$ 0.816
	infection	35.817 $\pm$ 14.032*
37-47 y	Control	4.449 $\pm$ 0.761
	infection	68.573 $\pm$ 35.932*
48-58 y	Control	2.578 $\pm$ 0.462
	infection	31.531 $\pm$ 1.268 *

\*L.S.D under ( $P < 0.05$ ) = 24.547

**Table 2:** The level of IL-4 in patients with scab.

Age group	Groups	Concentration of IL-4 pg/ml Mean ± S.D
15-25 y	Control	2.246±0.564
	infection	40.194 ± 1.349 *
26-36 y	Control	2.493±0.632
	infection	49.295± 8.256*
37-47 y	Control	3.671±0.589
	infection	61.051± 7.899*
48-58 y	Control	4.239±0.398
	infection	59.786 ± 24.086 *

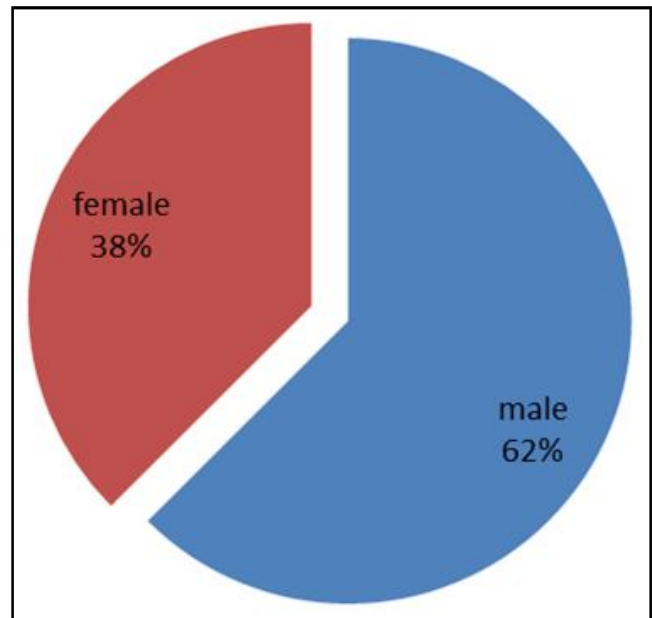
L.S.D under (P<0.05)= 39.345

of IL 4 pg/ml appears. ELISA assay appeared to have notice elevated concentration of IL4 at age 37-47 y being 969971, 7.899 compared to control 3.671± 0.589 for this class of patients with scab. While the IL4 has reported a small concentration of 40.194 ± 1.349, at L.S.D below (P<0.05).

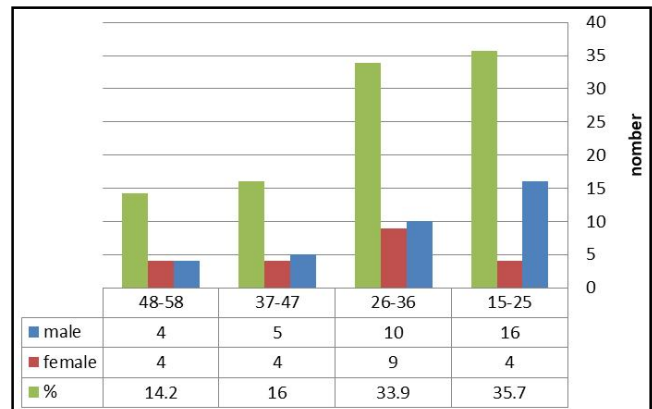
This study agreed with study (Simona *et al.*, 2003) while the findings are still poorly known due to the work of interleukin-4 in controlling immunity to infection with *Leishmania donovani*. Here we show that rise parasite download notice in receptor IL-4 and IL-4 receptor integrates with postponed granuloma maturity and antileishmanial action and that the increased parasite download observed in receptor mice associated with increased NOS<sub>2</sub> mRNA and decreased serum gamma interferon levels, IL-4 and IL-13 show to play little role in adjusting collagen sedimentation in *L. donovani*-induced granulomas. Nevertheless, interleukin-4 (IL-4) is often affiliated with evolution of protective type 2 immune responses in models of dermal scab (Launois *et al.*, 1997; Mohrs *et al.*, 1999; Satoskar *et al.*, 1995).

IL-4 performs a main part in influencing the essence of immune response. Simple peripheral CD4 + T cells start synthesizing and secretion cytokines when activated. This cytokine function act on growth and differentiation regulates, leading to the reproduction and Discrimination of simple T cells into effector cells Different subsets of effector T helper (Th) cells can be differentiated based on the cytokine pattern they secrete (Paul *et al.*, 1997).

Type 1 Th cells secrete IL-2, Interferon gamma and Tumor Necrosis Factor (TNF), while Type 2 secretes IL-4, IL-5, IL-6 and IL-13, IL-4 is a 15 KD polypeptide that has multiformity on many cell kinds. Its cell has different diameter consist of α subunit with a holding affinity to IL-4 and a common π subunit which is also minerals of other cytokine proteins. Binding IL-4 to its T-cell receptor results in development and differentiation into Th2-cell (Paul *et al.*, 1997).



**Fig. 1:** Distribution of infection according to the sex.



**Fig. 2:** Distribution of infection according to the age group.

Fig. 1 has shown the distribution of the disease by sex, where the percentage of infection in males is 62% compared to the rate of infection in the female is 38%.

The highest distribution rate is 33.9% among the 26-36 age group. The percentage of this group was close to both sexes, while the 48-58 age group had the lowest distribution rate of 14.4% and 50% of males and females as in Fig. 2.

### Conclusion

We conclude from this research that scabies infection contributes to the induction of cellular immune response by grow cytokine secretion, especially IL6, IL4 and its connection with age and sex.

### Recommendation

- 1- Investigate of the scab connection with immune cell mediated factors.
- 2- Knowledge of the influences of scabies on defence

cells such as big macrophage cells, lymphocytes, skin cells and other physiological cells such as mast cells.

#### Interest conflict: None to declare

Ethical Clearance: All laboratory procedures have actually supported in this research paper under the Department of Biology, Women's Science College, Babylon University, Hillah City, Iraq and all examinations have been performed in full compliance with allowed systems.

### References

- Arlian, L.G., D.L. Vyszanski-Moher, C.M. Rapp and B.E. Hull (1996). Production of IL-1 alpha and IL-1 beta by human skin equivalents parasitized by *Sarcoptes scabiei*. *J. Parasitol.*, Oct; **82(5)**: 719–23. PMID:8885878.
- Arlian, L.G., M.S. Morgan and J.S. Neal (2003). Modulation of cytokine expression in human keratinocytes and fibroblasts by extracts of scabies mites. *Am. J. Trop. Med. Hyg.*, Dec; **69(6)**: 652–6. PMID:14740884.
- Arlian, L.G., M.S. Morgan and J.S. Neal (2004). Extracts of scabies mites (Sarcoptidae: *Sarcoptes scabiei*) modulate cytokine expression by human peripheral blood mononuclear cells and dendritic cells. *J. Med. Entomol.*, Jan; **41(1)**: 69–73. PMID:14989348.
- Arlian, L.G., R.A. Runyan and D.L. Vyszanski-Moher (1988). Water balance and nutrient procurement of *Sarcoptes scabiei* var. *canis* (Acari: Sarcoptidae). *J. Med. Entomol.*, **25**: 64–68.
- Arlian, L.G., R.A. Runyan, S. Achar and S.A. Estes (1984). Survival and infectivity of *Sarcoptes scabiei* var. *canis* and var. *hominis*. *J. Am. Acad. Dermatol.*, **11**: 210–215. Pub Med.
- Arend, W.P., M. Malyak, C.J. Guthridge and C. Gabay (1998). Interleukin-1 receptor antagonist: Role in biology. *Annu. Rev. Immunol.*, **16**: 27–55. [PubMed].
- Arlian, L.G., M.S. Morgan and C.C. Paul (2006). Evidence that scabies mites (Acari: Sarcoptidae) influence production of interleukin-10 and the function of T-regulatory cells (Tr1) in humans. *J. Med. Entomol.*, **43**: 283–287. [PubMed].
- Arlian, L.G., R.A. Runyan and D.L. Vyszanski-Moher (1988). Water balance and nutrient procurement of *Sarcoptes scabiei* var. *canis* (Acari: Sarcoptidae). *J. Med. Entomol.*, **25**: 64–68. [PubMed].
- Arlian, L.G., R.A. Runyan and D.L. Vyszanski-Moher (1988a). Water balance and nutrient procurement of *Sarcoptes scabiei* var. *canis* (Acari: Sarcoptidae). *J. Med. Entomol.*, **25**: 64–68. [PubMed].
- Arlian, L.G., D.L. Vyszanski-Moher, S.G. Ahmed and S.A. Estes (1991). Cross-antigenicity between the scabies mite, *Sarcoptes scabiei* and the house dust mite, *Dermatophagoides pteronyssinus*. *J. Invest. Dermatol.*, **96**: 349–354. [PubMed].
- Arlian, L.G., M.S. Morgan, D.L. Vyszanski-Moher and B.L. Stemmer (1994). *Sarcoptes scabiei*: the circulating antibody response and induced immunity to scabies. *Exp. Parasitol.*, **78**: 37–50. [PubMed].
- Arlian L.G., M.S. Morgan and J.J. Arends (1996a). Immunologic cross-reactivity among various strains of *Sarcoptes scabiei*. *J. Parasitol.*, **82**: 66–72. [PubMed].
- Arlian, L.G., M.S. Morgan, S.A. Estes, S.F. Walton, D.J. Kemp and B.J. Currie (2004a). Circulating IgE in patients with ordinary and crusted scabies. *J. Med. Entomol.*, **41**: 74–77. [PubMed].
- Arlian, L.G. and M.S. Morgan (2000). Serum antibody to *Sarcoptes scabiei* and house dust mite prior to and during infestation with *S. scabiei*. *Vet. Parasitol.*, **90**: 315–326. [PubMed].
- Arlian, L.G., M.S. Morgan and C.C. Paul (2006). Evidence that scabies mites (Acari: Sarcoptidae) influence production of interleukin-10 and the function of T-regulatory cells (Tr1) in humans. *J. Med. Entomol.*, **43**: 283–287. [PubMed].
- Arlian, L.G., N. Fall and M.S. Morgan (2007). *In vivo* evidence that *Sarcoptes scabiei* (Acari: Sarcoptidae) is the source of molecules that modulate splenic gene expression. *J. Med. Entomol.*, **44**: 1054–1063. [PubMed].
- Bergstrom, F.C., S. Reynolds, M. Johnstone, R.N. Pike, A.M. Buckle, D.J. Kemp, K. Fischer and A.M. Blom (2009). Scabies mite inactivated serine protease paralogs inhibit the human complement system. *J. Immunol.*, **182**: 7809–7817. [PubMed].
- Engelman, D., K. Kiang, O. Chosidow, J. McCarthy, C. Fuller, P. Lammie, *et al.*, (2013). Toward the global control of human scabies: introducing the international alliance for the control of scabies. *PLoS Negl Trop Dis.*, **7(8)**: e2167. doi: 10.1371/journal.pntd.0002167. [PMC free article] [PubMed] Cross Ref.
- Elder, B.L., L.G. Arlian and M.S. Morgan (2006). *Sarcoptes scabiei* (Acari: Sarcoptidae) mite extract modulates expression of cytokines and adhesion molecules by human dermal microvascular endothelial cells. *J. Med. Entomol.*, **43**: 910–915. [PMC free article] [PubMed].
- Elder, B.L., L.G. Arlian and M.S. Morgan (2009). Modulation of human dermal microvascular endothelial cells by *Sarcoptes scabiei* in combination with proinflammatory cytokines, histamine and lipid-derived biologic mediators. *Cytokine*, **47**: 103–111. [PMC free article] [PubMed].
- Hay, R.J., N.E. Johns, H.C. Williams, I.W. Bolliger, R.P. Dellavalle, D.J. Margolis, *et al.*, (2014). The global burden of skin disease in 2010: an analysis of the prevalence and impact of skin conditions. *J. Invest. Dermatol.*, **134(6)**: 1527–1534. doi: 10.1038/jid.2013.446. [PubMed] [CrossRef].
- Hoy, W.E., A.V. White, A. Dowling, S.K. Sharma, H. Bloomfield, B.T. Tipiloura, *et al.*, (2012). Post-streptococcal glomerulonephritis is a strong risk factor for chronic kidney disease in later life. *Kidney Int.*, **81**: 1026–1032. doi:

- 10.1038/ki.2011.478. [PubMed] [CrossRef].
- Hirano, T., K. Yasukawa, H. Harada, T. Taga, Y. Watanabe, T. Matsuda, S. Kashiwamura, K. Nakajima, K. Koyama, A. Iwamatsu, *et al.*, (1986). Complementary DNA for a novel human interleukin (BSF-2) that induces B lymphocytes to produce immunoglobulin. *Nature*, **324**: 73–76.
- Launois, P., I. Maillard, S. Pingel, K.G. Swihart, I. Xenarios, H. Acha-Orbea, H. Diggelmann, R.M. Locksley, H.R. MacDonald and J.A. Louis (1997). IL-4 rapidly produced by V beta 4 V alpha 8 CD4 T cells instructs Th2 development and susceptibility to *Leishmania major* in BALB/c mice. *Immunity*, **6**: 541–549.
- Mika, A., S.L. Reynolds, F.C. Mohlin, C. Willis, P.M. Swe, D.A. Pickering, V. Halilovic, L.C. Wijeyewickrema, R.N. Pike, A.M. Blom, *et al.*, (2012). Novel scabies mite serpins inhibit the three pathways of the human complement system. *PLoS One*, **7**: e40489. [PMC free article][PubMed].
- Morgan, M.S. and L.G. Arlian (2009). Response of human skin equivalents to *Sarcoptes scabiei* mites and extract. *Am. J. Trop. Med. Hyg.*, **81**: S218.
- Morgan, M.S. and L.G. Arlian (2010). Response of human skin equivalents to *Sarcoptes scabiei*. *J. Med. Entomol.*, **47**: 877–883. [PMC free article] [PubMed].
- Morgan, M.S., L.G. Arlian and M.P. Markey (2013). *Sarcoptes scabiei* mites modulate gene expression in human skin equivalents. *PLoS One*, **8**: e71143. [PMC free article] [PubMed].
- Morgan, M.S. and L.G. Arlian (2010). Response of human skin equivalents to *Sarcoptes scabiei*. *J. Med. Entomol.*, **47**: 877–883. [PMC free article] [PubMed].
- Morgan, M.S. and L.G. Arlian (1994). Serum antibody profiles of *Sarcoptes scabiei* infested or immunized rabbits. *Folia Parasitol. (Praha)*, **41**: 223–227. [PubMed].
- Mohrs, M., B. Ledermann, G. Kohler, A. Dorfmueller, A. Gessner and F. Brombacher (1999). Differences between IL-4- and IL-4 receptor alpha-deficient mice in chronic leishmaniasis reveal a protective role for IL-13 receptor signaling. *J. Immunol.*, **162**: 7302–7308.
- Paul, W.E. (1997) Interleukin 4: signaling mechanisms and control of T cell differentiation. *Ciba Found Symp.*, **204**: 208–16. [PubMed] [Google Scholar].
- Reynolds, S.L., R.N. Pike, A. Mika, A.M. Blom, A. Hofmann, L.C. Wijeyewickrema, D. Kemp and K. Fischer (2014). Scabies mite inactive serine proteases are potent inhibitors of the human complement lectin pathway. *PLoS Negl. Trop. Dis.*, **8**: e2872. [PMC free article] [PubMed].
- Romani, L., A.C. Steer, M.J. Whitfield and J.M. Kaldor (2015). Prevalence of scabies and impetigo worldwide: a systematic review. *Lancet Infect Dis.*, **15**(8): 960–967. doi: 10.1016/S1473-3099(15)00132-2. [PubMed] [CrossRef].
- Romani, L., A.C. Steer, M.J. Whitfield and J.M. Kaldor (2015). Prevalence of scabies and impetigo worldwide: a systematic review. *Lancet Infect Dis.*, **15**(8): 960–967. doi: 10.1016/S1473-3099(15)00132-2. [PubMed] [CrossRef].
- Satoskar, A., H. Bluethmann and J. Alexander (1995). Disruption of the murine interleukin-4 gene inhibits disease progression during *Leishmania Mexicana* infection but does not increase control of *Leishmania donovani* infection. *Infect. Immun.*, **63**: 4894–4899.
- Schumann, R.J., M.S. Morgan, R. Glass and L.G. Arlian (2001). Characterization of house dust mite and scabies mite allergens by use of canine serum antibodies. *Am. J. Vet. Res.*, **62**: 1344–1348. [PubMed].
- Simona Staiger, James Alexander, K. Christine Carter, Frank Brombacher and Paul M. Kaye (2003). Both Interleukin-4 (IL-4) and IL-4 Receptor Signaling Contribute to the Development of Hepatic Granulomas with Optimal Antileishmanial Activity. *Infection and Immunity*, **71**(8): p.4804–4807.
- Swe, P.M. and K. Fischer (2014). A scabies mite serpin interferes with complement-mediated neutrophil functions and promotes staphylococcal growth. *PLoS Negl. Trop. Dis.*, **8**: e2928. [PMC free article][PubMed].
- Zhang, Z.Q. (2011). Animal biodiversity: an outline of higher-level classification and survey of taxonomic richness. *Zoo taxa.*, **3148**: 237.