

PROTECTIVE EFFECTS OF CAMEL MILK ON ACUTE AND CHRONIC INFECTION OF *Toxoplasma gondii* IN MICE

Xinlei Yan^{1,3*}, Wenying Han^{1,3}, Zhili Yang², Hejing Wang¹ and Ruifeng Li^{2*}

¹Food Science and Engineering College of Inner Mongolia Agricultural University, Hohhot 010018, China

²Department of Pediatrics, Inner Mongolia Maternal and Child Health Hospital, Hohhot 010020, China

³These authors contributed equally to this work

ABSTRACT

In this study, the effects of camel milk in mice infected with *T. gondii* was evaluated. We established acute and chronic infection mouse models, as well as a dexamethasone-based immunocompromised model. All mice were treated with camel milk, milk, or phosphate-buffered saline (PBS), followed by analyses of survival rate, cyst count, serum cytokine levels and brain inflammation in mice. There were significant differences in linear trend ($P < 0.05$) in the survival curve of treating by camel milk, milk and PBS. And serum levels of IL-2 ($P < 0.05$) of camel milk-treated mice were lower compared with milk group, while serum levels of IL-4 and IFN- γ ($P < 0.05$ for both, $P < 0.01$ for both) were higher of camel milk-treated mice than milk and PBS group. Additionally, camel milk reduced the extent of brain inflammation in mice with chronic *T. gondii* infection and immunocompromised mice. Importantly, camel milk alleviated the clinical symptoms of toxoplasmosis in mice. In conclusion, our findings suggest that camel milk exhibits promise for preventing or treating *T. gondii* infections.

Key words: camel milk, infection, mouse, *Toxoplasma gondii*

Toxoplasma gondii is an obligate intracellular protozoan parasite that can infect almost all warmed-blooded animals, including humans (Liu *et al*, 2015; Yang *et al*, 2013). Approximately one-third of the human population has been exposed to *T. gondii* (Zhang *et al*, 2016). In humans, *T. gondii* infections predominantly occur via consumption of water or raw meat contaminated with *T. gondii* oocysts (Dubey, 2008; Montoya and Liesenfeld, 2004). During acute infection, tachyzoites proliferate and subsequently transform into cysts to establish a chronic infection in the brain preferentially (Suzuki, 2020). Infections with *T. gondii* can be life-threatening, especially in immunodeficient patients, as they cause cerebral and ocular damage and even death (Zhang *et al*, 2016). Furthermore, reactivation of latent infection in immunocompromised individuals can result in fatal toxoplasmic encephalitis, myocarditis, and pneumonitis (Eza and Lucas, 2006 and Saadatnia and Golkar, 2012). Infections with *T. gondii* also lead to the induction of potent cellular immune responses. Upon activation, macrophages and T lymphocytes produce various cytokines, including interleukin (IL) and interferon- γ (IFN- γ), which inhibit parasite

replication in haematopoietic and nonhaematopoietic cells, prevent the activation process of latent infection, and promote extracellular parasite lysis (Benevides *et al*, 2019; Khan *et al*, 1994; Kugler *et al*, 2016 and Xing *et al*, 2017).

Camel milk is an important nutritional source for pastoralists in many African and Asian countries. Camel milk has exceptionally high nutritional and medical value (Abrhaley and Leta, 2018). Camel milk is low in cholesterol and high in minerals (e.g., sodium, potassium, iron, copper, zinc cobalt, magnesium, manganese, and molybdenum) (Abrhaley and Leta, 2018 and Saini *et al*, 2007). Additionally, studies have shown that camel milk may alleviate liver conditions, malnutrition, vitamin deficiency, and allergic reactions, as well as prevent diabetes and improve immune system function (Khan *et al*, 2021; Wernery, 2006). Moreover, rats consuming camel milk have been shown to have higher IFN- γ levels compared to those consuming yak milk or cow milk (Wen *et al*, 2017).

Based on the complexity of the *T. gondii* life cycle, the diversity of its pathogenesis, and biological

SEND REPRINT REQUEST TO XINLEI YAN and RUIFENG LI [email: yanxinlei1987620@foxmail.com](mailto:yanxinlei1987620@foxmail.com) • 2317078007@qq.com

characteristics, there are currently no effective preventive or therapeutic agents for *T. gondii* infection (Konstantinovic *et al*, 2019). The sulfonamides are commonly used in the clinical (e.g., sulfadiazine) may alleviate symptoms in patients infected with *T. gondii*, they do not provide a cure and may cause significant toxicity (Ben-Harari *et al*, 2017). Therefore, more effective and safer therapeutic agents are urgently required. In this study, we examined the ability of camel milk to treat acute and chronic *T. gondii* infection in mice.

Materials and Methods

Animals, parasites, and drugs

Six-week-old female Kunming mice were purchased from Inner Mongolia University. Mice (five per cage) were given *ad libitum* access to food and water and housed with a 12-h light/dark cycle. Animal protocols were reviewed and approved by the Inner Mongolia Agricultural University Laboratory Animal Welfare and Animal Experimental Ethical Inspection Committee (approval no.: 2020-037). Prugniald strains of *T. gondii* were obtained from the National Animal Protozoa Laboratory of China Agricultural University. Camel milk was obtained from Alxa Bactrian camels in Alxa Left Banner, Inner Mongolia, China. Dexamethasone was purchased from Henan Runhong Pharmaceutical Co., Ltd. Enzyme-linked immunosorbent assay (ELISA) kits for IL-2, IL-4, and IFN- γ were purchased from Shanghai Jingmei Bioengineering Co., Ltd.

Acute infection

Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or phosphate-buffered saline (PBS), respectively (n=12 mice per group). Five days later, the mice were intraperitoneally injected with 100 μ L of PBS containing 20 cysts and the control with 0.1 mL PBS only. On 14 day post-infection (PI), the trial was over and the survival curve was drawn. Another group was treated as described above. On day 7 PI, the mice were sacrificed. Additionally, blood samples were collected and serum levels of IL-2, IL-4, and IFN- γ were measured via ELISA following the manufacturer's instructions.

Chronic infection

Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or PBS (n=12 mice per group). Five days later, the mice were intraperitoneally inoculated with 100 μ L PBS including 3 cysts and the control injected with 100 μ L of PBS alone. On 56 day PI, the mice were sacrificed, and cysts were counted. Brain tissue sections were prepared and stained

with hematoxylin and eosin (H&E) to assess tissue inflammation (Afifi and Al-Rabia, 2015; Chen *et al*, 2020).

Immunosuppression

Mice were treated by oral gavage with 100 μ L of camel milk, milk, or PBS (n=12 mice per group). Five days later, mice were intraperitoneally injected with 100 μ L PBS including 3 cysts or 100 μ L of PBS as a control. On 56 day PI, the mice were injected intraperitoneally with 3 mg/kg of dexamethasone every 2 days. The modified SHIRPA protocol was used to assess infection severity every 2 or 3 days, and on the last day of the experiment (Rogers *et al*, 1997). On 63 day PI, the mice were sacrificed by cervical dislocation, and brain tissue sections were stained with H&E.

Toxoplasma gondii cyst counting

The brains of sacrificed mice were homogenised in 2 mL of PBS. A 20- μ L sample of each homogenate was absorbed to blood counting chamber and counted three times microscopically (only count intact cysts were counted). Then the mean number of cysts count per brain was calculated.

Clinical scores

Clinical examination of mice was performed using the modified SHIRPA protocol included a series of individual tests, which provided quantitative data about an individual performance (Rogers *et al*, 1997). The tests were performed in the following order, and one point was given for conformity: piloerection, abdominal writhing, weight loss, diarrhoea, lacrimation, palpebral closure, moving speed, reflexive escape from touch, spontaneous tremors, reduced grip strength, hunched posture, and changes in respiration rate (hyperventilation) (Estado *et al*, 2018). The control group was regarded as no changes in the clinical score.

Statistical analysis

GraphPad Prism software was used in plotting and data analysis. Data are expressed as the means \pm standard deviations (SD). Statistical significance was determined using two-tailed t-tests. P-values \leq 0.05 were considered to indicate statistical significance (*, P \leq 0.05; **, P \leq 0.01; ***, P < 0.001).

Results

Effects of camel milk on the survival rate and cytokine levels of mice with acute *T. gondii* infection

All non-infected control mice survived. By contrast, infected mice treated with camel milk,

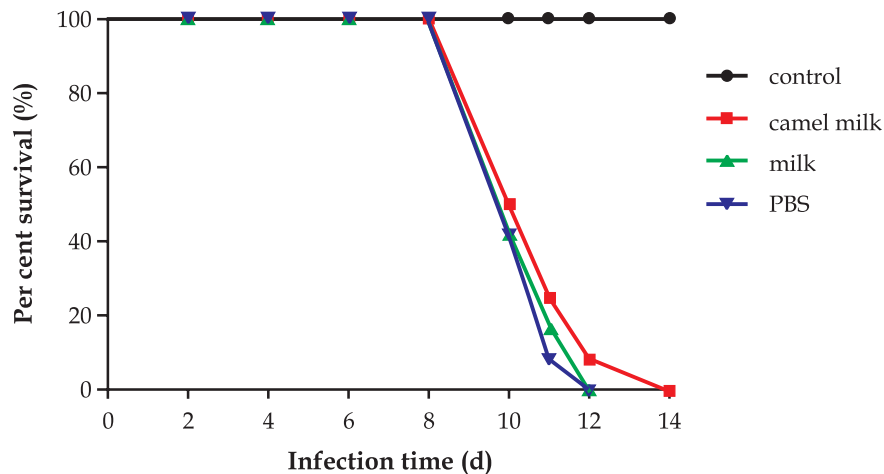


Fig 1. Survival of mice with acute *T. gondii* infection. Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or PBS, respectively. Five days later, the mice were intraperitoneally injected with 100 μ L of PBS containing 20 cysts and the control with 0.1 mL PBS only. No deaths were observed in the control group. Camel milk treatment prolonged the survival rate of mice with acute *T. gondii* infection.

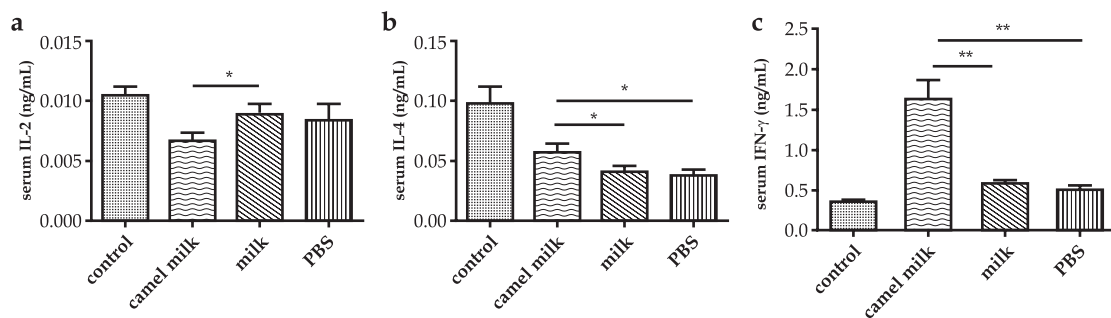


Fig 2. Serum cytokines levels in mice with acute *T. gondii* infection. Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or PBS, respectively. Five days later, the mice were intraperitoneally injected with 100 μ L of PBS containing 20 cysts and the control with 0.1 mL PBS only. On 7 day PI, the mice were sacrificed and the levels of serum cytokines were measured. Serum levels of IL-2 (a), IL-4 (b), and IFN- γ (c). Data are shown as the means \pm standard deviations (SDs; n=12 mice per group). *, $P < 0.05$; **, $P < 0.01$.

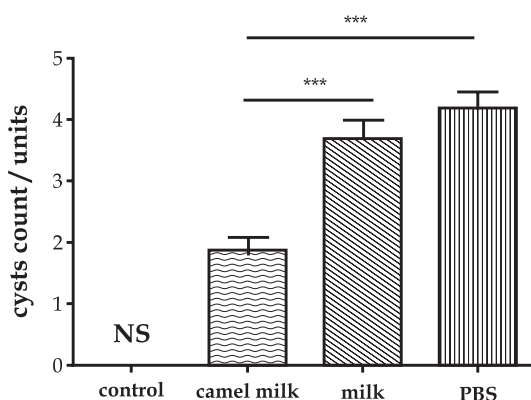


Fig 3. Effects of camel milk on the number of cysts in the brains of mice with chronic *T. gondii* infection. Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or PBS. Five days later, the mice were intraperitoneally inoculated with 100 μ L PBS including 3 cysts and the control injected with 100 μ L of PBS alone. On 56 day PI, the mice were sacrificed, and cysts were counted. Data are shown as the means \pm SDs (n=12 mice per group). ***, $P < 0.001$.

milk, or PBS started dying at 8 day PI. The 10-day survival rate of mice treated with camel milk was 50.0%. On 11 day PI, 16.7% of the mice treated with milk were alive (Fig 1). There was no significant difference in the survival rate of mice treated with camel milk, milk, or PBS. However, linear regression analysis revealed significant differences ($P < 0.05$) in the survival curves of mice treated with camel milk, milk, and PBS.

The serum levels of IL-2 and IL-4 were lower in mice with acute *T. gondii* infection than in non-infected mice, whereas the levels of IFN- γ were higher. There were significant differences in the levels of IL-2 between mice treated with camel milk and those treated with milk ($P < 0.05$; Fig 2a). Additionally, serum IL-4 levels differed significantly between the camel milk group and the milk group, as well as between the camel milk group and the PBS group ($P < 0.05$ for both; Fig 2b). Compared with

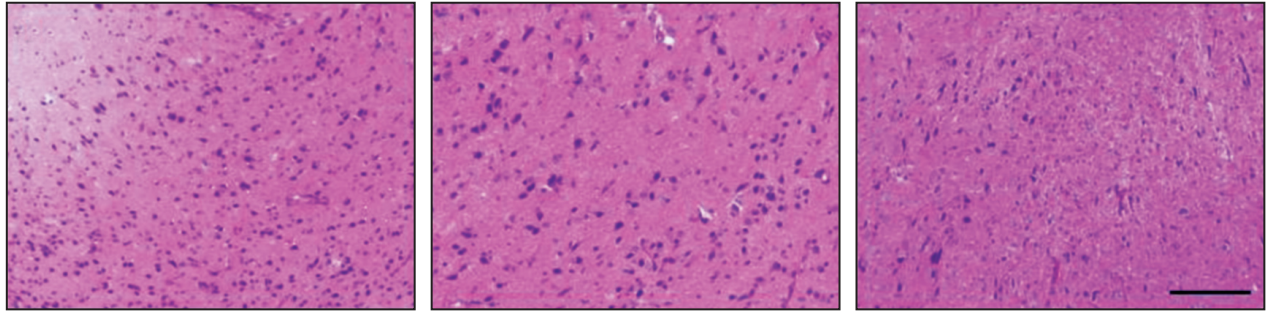


Fig 4. Effects of camel milk on brain inflammation in mice with chronic *T. gondii* infection. Mice were treated daily by oral gavage with 100 μ L of camel milk, milk or PBS. Five days later, the mice were intraperitoneally inoculated with 100 μ L PBS including 3 cysts and the control injected with 100 μ L of PBS alone. On 56 day PI, the mice were sacrificed, brain tissue sections were prepared and stained with hematoxylin and eosin (H&E). (a–c) Brain tissue sections with different extents of inflammatory cell infiltration. Brain tissue section from *T. gondii*-infected mice treated with PBS (a), milk (b), or camel milk (c). Scale bar: 50 μ m.

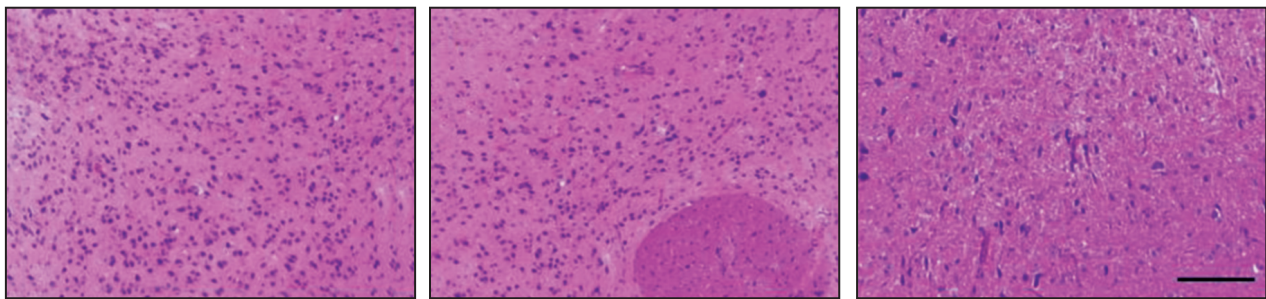


Fig 5. Effects of camel milk on brain inflammation in immunocompromised mice. Mice were treated by oral gavage with 100 μ L of camel milk, milk, or PBS. Five days later, mice were intraperitoneally injected with 100 μ L PBS including 3 cysts or 100 μ L of PBS as a control. On 56 day PI, the mice were injected intraperitoneally with 3 mg/kg of dexamethasone every 2 days. On 63 day PI, the mice were sacrificed, brain tissue sections were prepared and stained with hematoxylin and eosin (H&E). (a–c) Brain tissue sections with different extents of inflammatory cell infiltration. Brain tissue section from *T. gondii*-infected mice treated with dexamethasone and PBS (a), dexamethasone and milk (b), or dexamethasone and camel milk (c). Scale bar: 50 μ m.

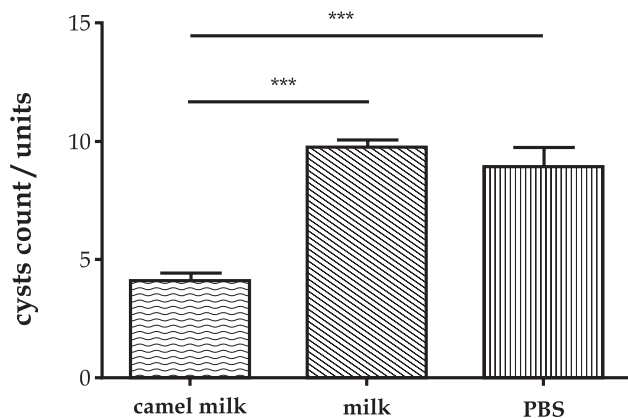


Fig 6. Effects of camel milk on clinical performance in *T. gondii*-infected immunocompromised mice. Mice were treated by oral gavage with 100 μ L of camel milk, milk, or PBS (n=12 mice per group). Five days later, mice were intraperitoneally injected with 100 μ L PBS including 3 cysts or 100 μ L of PBS as a control. On 56 day PI, the mice were injected intraperitoneally with 3 mg/kg of dexamethasone every 2 or 3 days, and on the last day of the experiment. The control group was regarded as no changes in the clinical score.

IFN- γ levels in the camel milk group, those in the milk and PBS groups were significantly lower ($P < 0.01$; Fig 2c).

Camel milk reduces the cyst counts and brain inflammation in mice with chronic *T. gondii* infection

As expected, no cysts were observed in the brains of non-infected mice. The cyst counts in the brains of chronically infected mice treated with camel milk, milk, and PBS were 1.86/unit, 3.72/unit, and 4.19/unit, respectively (Fig 3). The cyst count differed significantly between the camel milk and milk groups ($P < 0.001$), as well as between the camel milk and PBS groups ($P < 0.001$). These findings suggest that camel milk can reduce the number of cysts in the brain of mice with chronic *T. gondii* infection. Furthermore, we found that chronic *T. gondii* infection resulted in extensive infiltration of inflammatory cells into the brain (Fig 4a). Notably, camel milk reduced the extent of immune cell infiltration (Fig 4c).

Camel milk reduces the extent of brain inflammatory responses in immunocompromised mice

In immunosuppressed mice, *T. gondii* infection induced extensive infiltration of inflammatory cells into the brain (Fig 5a). Importantly, camel milk profoundly reduced inflammatory responses in the brain in response to *T. gondii* infection (Fig 5c).

Effects of camel milk on clinical performance in *T. gondii*-infected immunocompromised mice

At up to 8 days after *T. gondii* infection, we did not observe any obvious clinical signs, which was consistent with the clinical course of toxoplasmosis. Clinical signs were observed starting at 3 day PI and peaked at 12 day PI. Dexamethasone treatment improved the clinical performance of the infected mice. Severe clinical signs included palpebral closure, a hunched posture, and changes in the respiration rate (hyperventilation). Interestingly, clinical signs differed significantly between mice treated with camel milk and those treated with milk ($P < 0.001$) or PBS ($P < 0.001$) based on the Modified-SHIRPA protocol. By contrast, there were no significant differences in clinical signs between the milk and PBS groups ($P > 0.05$; Fig 6). These results were consistent for up to 63 day PI as just showed because the mice was infected chronically all the time (chronic infection).

Discussion

We found that camel milk prolonged the survival of mice with acute *T. gondii* infection, although the differences in the survival rate among groups were not significant. This discrepancy may be explained by the high virulence and rapid proliferation of *T. gondii*. Additionally, mice with acute *T. gondii* infection exhibited significantly lower serum IL-2 and IL-4 levels and higher IFN- γ levels compared to control mice. Type 1 helper (Th1) cells induce cellular immune responses by releasing IL-2 and IFN- γ , which are key inflammatory mediators in mice infected with *T. gondii* (Yang *et al*, 2008). IL-2 further enhances IFN- γ production, establishing a positive feedback loop that induces potent immune responses (Fang *et al*, 2000). Consistent with our findings, previous *in vivo* studies have shown that IL-2 significantly prolongs the survival of *T. gondii*-infected mice (Cheng *et al*, 2010; Fang *et al*, 2000). However, we found that the serum levels of IL-2 were lower in mice infected with *T. gondii* than in non-infected mice, possibly due to the dual-directional regulation of IL-2 and IFN- γ production. IFN- γ has been demonstrated to prevent tachyzoite

proliferation and growth by activating cerebral cells and promoting innate and T-cell-mediated immune responses (Suzuki, 2020; Suzuki *et al*, 2011); this effect of IFN- γ might have contributed to the prolonged survival of mice with acute infection. Type 2 helper (Th2) cells activate humoral immune responses by releasing IL-4 and other cytokines that induce B-cell activation. Cytokines secreted by Th1 and Th2 cells often have contradicting effects (Cheng *et al*, 2010; Vander *et al*, 2000). Although our results suggest that camel milk has protective effects in mice with acute *T. gondii* infection, the role of the Th1/Th2 balance in the protective effects of camel milk merits further investigation.

Compared with acute infection, camel milk also played an important role in inhibiting chronic infection of *T. gondii* in mice. Previous studies have shown that a suitable animal model of chronic *T. gondii* infection can be established with intraperitoneal *T. gondii* inoculation in mice (Liu *et al*, 2005). Notably, camel milk profoundly reduced the number of cysts in the brains of *T. gondii*-infected mice. Moreover, camel milk markedly inhibited inflammatory responses in the brain, pinpointing the protective effects of camel milk against chronic *T. gondii* infection.

Dexamethasone treatment in immunocompromised mice further impairs the ability of the immune system to control *T. gondii* infection. In this study, we found that camel milk strongly inhibited inflammatory responses in the brain of *T. gondii*-infected immunocompromised mice, providing further evidence of the protective effects of camel milk. It is worth noting that compared with immunocompetent mice with chronic *T. gondii* infection, *T. gondii*-infected immunocompromised mice exhibited stronger inflammatory responses, suggesting that the severity of *T. gondii* infection may be regulated by host immunity. A previous study showed that the numbers of cysts were higher in the hippocampus and cerebral cortex of *T. gondii*-infected mice than in the brain stem and ependymal region (Bao *et al*, 2006), in accordance with the ability of *T. gondii* to impair learning and memory in mice (Witting, 1979). Interestingly, we found that mice with severe toxoplasmosis displayed palpebral closure, a hunched posture, and changes in the respiration rate (hyperventilation). Camel milk significantly alleviated these clinical signs of toxoplasmosis. In conclusion, our findings indicate that camel milk exhibits promise for preventing or treating *T. gondii* infections. The mechanisms underlying the protective effects of camel milk warrant further investigation.

Acknowledgements

This work was financially supported by Research project of high level talents in Inner Mongolia Agricultural University (No. RZ1900002817) and the Program of Inner Mongolia Natural Science Foundation of China (No.2018BS03015).

Conflict of interest

All individual authors declare that they have no conflict of interest (financial, personal or other).

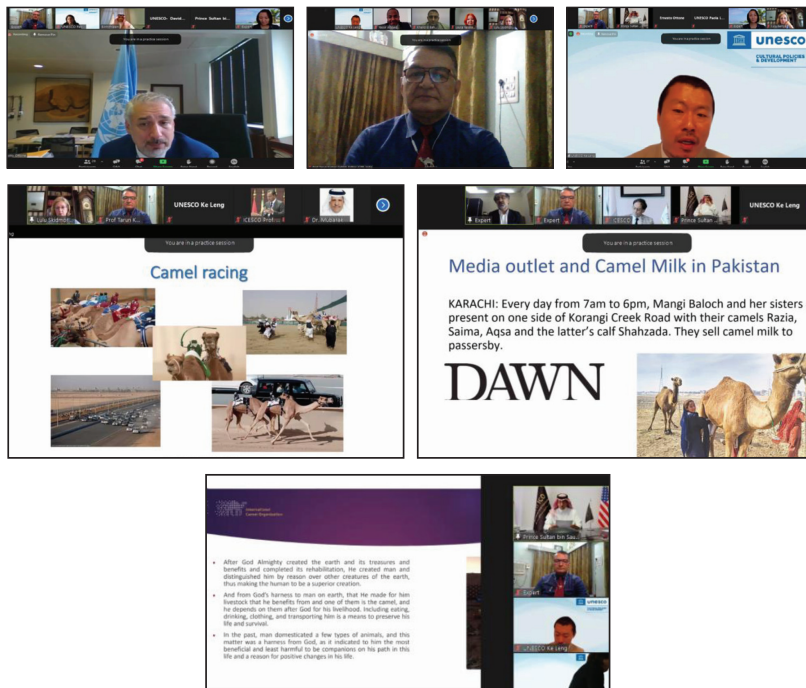
References

- Abrahale A and Leta S (2018). Medicinal value of camel milk and meat. *Journal of Applied Animal Research* 46(1):552-558.
- Afifi MA and Al-Rabia MW (2015). The immunomodulatory effects of rolipram abolish drug-resistant latent phase of *Toxoplasma gondii* infection in a murine model. *Journal of Microscopy and Ultrastructure* 3(2):86-91.
- Bao AY, Wang HL, Wang GH, Dong HF, Guo Y and Jiang MS (2006). Studies on the distribution of cysts in the brain of rats chronically infected with *Toxoplasma gondii*. *Chinese Journal of Zoonoses* (09):837-839. (in Chinese)
- Benevides L, Saltarelli VM, Pioto F, Sacramento LA, Dias MS, Rodríguez GR, Viola JPB, Carregaro V, Silva JS (2019). NFAT1 Regulates Ly6Chi Monocyte Recruitment to the CNS and Plays an Essential Role in Resistance to *Toxoplasma gondii* Infection. *Frontiers of Immunology* 10:2105. doi: 10.3389/fimmu.2019.02105. PMID: 31555297; PMCID: PMC6742953.
- Ben-Harari RR, Goodwin E and Casoy J (2017). Adverse Event Profile of Pyrimethamine-Based Therapy in Toxoplasmosis: A Systematic Review. *Drugs in R&D* 17(4):523-544.
- Cheng Y, Li JH and Peng JX (2010). Advances in studies on the relationship between cytokines and parasitic infection. *Journal of Pathogen Biology* 5(05):381-384. (in Chinese)
- Chen JX, Wang YP, Zhang X, Li GX, Zheng K and Duan CZ (2020). lncRNA Mtss1 promotes inflammatory responses and secondary brain injury after intracerebral hemorrhage by targeting miR-709 in mice. *Brain Research Bulletin* 162:20-29.
- Dubey JP (2008). The history of *Toxoplasma gondii*-the first 100 years. *Journal of Eukaryotic Microbiology* 55(6):467-475.
- Estate V, Stipursky J, Gomes F, Mergener TC, Frazao-Teixeira E, Allodi S, Tibirica E, Barbosa HS and Adesse D (2018). The neurotropic parasite *Toxoplasma gondii* induces sustained neuroinflammation with microvascular dysfunction in infected mice. *American Journal of Pathology* 188(11):2674-2687.
- Eza DE and Lucas SB (2006). Fulminant toxoplasmosis causing fatal pneumonitis and myocarditis. *HIV Medicine* 7(6):415-420.
- Fang YQ, Xu SF and Tan Y (2000). The effects of TNF- α , IL-6, IL-4 and IL-2 on the anti-toxoplasma infection of IFN- γ . *Journal of Immunology* (03):196-199. (in Chinese)
- Khan IA, Ely KH and Kasper LH (1994). Antigen-specific CD8+ T cell clone protects against acute *Toxoplasma gondii* infection in mice. *Journal of Immunology* 152(4):1856-1860.
- Khan MZ, Xiao JX, Ma YL, Ma JY, Liu S, Khan A, Khan JM and Cao ZJ (2021). Research development on anti-microbial and antioxidant properties of camel milk and its role as an anti-cancer and anti-hepatitis Agent. *Antioxidants* 10(5):788.
- Konstantinovic N, Guegan H, Stajner T, Belaz S and Robert-Gangneux F (2019). Treatment of toxoplasmosis: Current options and future perspectives. *Food and Waterborne Parasitology* 15:e00036.
- Kugler DG, Flomerfelt FA, Costa DL, Laky K, Kamenyeva O, Mittelstadt PR, Gress RE, Rosshart SP, Rehmann B, Ashwell JD, Sher A and Jankovic D (2016). Systemic toxoplasma infection triggers a long-term defect in the generation and function of naive T lymphocytes. *The Journal of Experimental Medicine* 213(13):3041-3056.
- Liu JY, Yang XZ, Wu ZQ and Yang SS (2005). A study on the formation of cysts in the brain in mice infected by *Toxoplasma gondii* by different ways. *Chinese Journal of Zoonoses* 21(7):616-620. (in Chinese)
- Liu Q, Wang ZD, Huang SY and Zhu XQ (2015). Diagnosis of toxoplasmosis and typing of *Toxoplasma gondii*. *Parasites & Vectors* 8: 292-326.
- Montoya JG and Liesenfeld O (2004). Toxoplasmosis. *Lancet* 363:1965-1976.
- Rogers DC, Fisher EM, Brown SD, Peters J, Hunter AJ and Martin JE (1997). Behavioral and functional analysis of mouse phenotype: SHIRPA, a proposed protocol for comprehensive phenotype assessment. *Mammalian Genome* 8(10):711-713.
- Saadatnia G and Golkar M (2012). A review on human toxoplasmosis. *Scandinavian Journal of Infectious Diseases* 44(11):805-814.
- Saini N, Bhati AK, Singh N and Tuteja FC (2007). Trace mineral and vitamin C content of camel milk: a comparative study. *Veterinary Practitioner* 8(1):20-21.
- Suzuki Y (2020). The immune system utilizes two distinct effector mechanisms of T cells depending on two different life cycle stages of a single pathogen, *Toxoplasma gondii*, to control its cerebral infection. *Parasitology International* 76:102030.
- Suzuki Y, Sa QL, Gehman M and Ochiai E (2011). Interferon-gamma- and perforin-mediated immune responses for resistance against *Toxoplasma gondii* in the brain. *Expert Reviews in Molecular Medicine* 13:e31.
- Vander Veen RC, Dietlin TA, Pen L, Gray JD and Hofman FM (2000). Antigen presentation to Th1 but not Th2 cells by macrophages results in nitric oxide production and inhibition of T cell proliferation: interferon-gamma is essential but insufficient. *Cellular Immunology* 206(2):125-135.
- Wen YP, He QW, Ding J, Wang HY, Hou QC, Zheng Y, Li CK, Ma YZ, Zhang HP and Kwok LY (2017). Cow, yak, and camel milk diets differentially modulated the systemic immunity and fecal microbiota of rats. *Science Bulletin* 62(6):405-414.

- Wernery Ulrich (2006). Camel milk, the white gold of the desert. *Journal of Camel Practice and Research* 13(1):15-26.
- Witting PA (1979). Learning capacity and memory of normal and toxoplasma-infected laboratory rats and mice. *Zeitschrift Fur Parasitenkunde-parasitology Research* 61(1):29-51.
- Xing MN, Wang DW, Guo XG, Li CH, Li JQ, Wu AH, Li HK, Li DC and Yang N (2017). Immunity of CD8+ T lymphocytes in mice induced by *Toxoplasma gondii* infection. *Heilongjiang Animal Husbandry and Veterinary Medicine* (13):57-61. (in Chinese)
- Yang N, Li HK, He JB, Mu MY and Yang SH (2013). Seroprevalence of *Toxoplasma gondii* Infection in Domestic Sheep in Liaoning Province, Northeastern China. *The Journal of Parasitology* 99(1):174-175.
- Yang S, Qian JX, Zhu JY, Feng P and Zhu XM (2008). Effects of recombinant human IL-2 and IFN- γ on T cell subsets in pregnant rats infected with *Toxoplasma gondii*. *Chinese Journal of Schistosomiasis Control* (05):360-363. (in Chinese)
- Zhang N, Wang S, Wang D, Li CY, Zhang ZC, Yao ZJ, Li TT, Xie Q, Liu SG and Zhang HZ (2016). Seroprevalence of *Toxoplasma gondii* infection and risk factors in domestic sheep in Henan province, central China. *Parasite (Paris, France)* 23:53-57.

First ICO International Experts Congress (ICOIEC)

First online meeting of ICO International Experts Congress (ICOIEC) in collaboration with UNESCO on 'Cultural practices related to camel traditions to advance the SDGs' was held on 29 June 2021. Opening remarks were given by the H.E. Mr. Fahad F. bin Hithleen, Founder & President of International Camel Organization and Mr. Ernesto Ottone R. Assistant Director-General for Culture, UNESCO. There were four thematic sessions, i.e. Safeguarding the living cultural heritage enshrined in the camel traditions; Sustaining the local practices of camel herding for socio-economic development; Leveraging camel-based practices for biodiversity preservation and climate change adaptation and mitigation, and Bolstering evidence on the scientific and educational dimensions of the camelid traditions. The rapporteurs were Mr. Ahmed Skounti, Morocco; Dr. Ilse Köhler-Rollefson, LPPS, India and Germany; Ms. Hindou Oumarou Ibrahim, Chad and Dr. Laura Yereshekova, Kazakhstan. Various speakers were H.H Prince Sultan bin Saud bin Mohamed, Vice President of ICO to the State of the Headquarters, Saudi Arabia; Dr. Ed Emiri, London, Ms. Ayjarkin Kojobekova, Expert on living heritage; Dr. Hanan Abdel-Mawla, Sudan; Prof. Abdel-Razek Kakar, UAE; Prof. Hani Hayajneh, Jordan; Prof. Lulu Eskidmore, UAE, Ms. Delaram Kiramat, Uzbekistan; Prof. Abdul Malik Ibrahim Khalaf Allah, UAE and Prof. T K Gahlot, India. Closing remarks were given by the Secretary-General of ICO. The one-day event organised by ICO in collaboration with UNESCO was aimed to highlight the connections between camel traditions and the achievement of the UN 2030 Agenda for Sustainable Development, and notably their contribution to SDG 1 on reducing poverty, SDG 2 on fighting hunger, SDG 4 on education for sustainable development, SDG 8 on decent work and employment, SDG 11 on sustainable communities, SDG 12 on responsible consumption and production, and SDG 15 on environmental sustainability.



Glimpses of online presentations of First ICO International Experts Congress

First ICO International Experts Congress (ICOIEC)

ICO - UNESCO Organizing committee
 Contact information: Mr. Yousif A. Al-Hmed (1966) 543 751002 secretary@ico.org.sa
 Mr. Ka Leng - k.leng@unesco.org

