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EPIDEMIOLOGICAL STUDY OF BRUCELLOSIS IN DOMESTIC ANIMALS

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Abstracts: Brucellosis is a disease that causes severe economic losses for livestock farms worldwide. *Brucellamelitensis, B. abortus*and *B. suis*, which are transmitted between animals both vertically and horizontally, cause abortion and infertility in their primary natural hosts – goats and sheep *(B. melitensis*), cows (*B. abortus*) and sows (*B. suis*). *Brucella*spp. infects not only their preferred hosts but also other domestic and wild animal species, which in turn can act as reservoirs of the disease for other animal species and humans. Brucellosis is therefore consideredto be a major zoonotic disease that are transmitted by direct contact with animals and/or their secretions, or by consuming milk and dairy products.
Keywords: Brucellaabortus – Brucellamelitensis – Brucellasuis– Brucellosis – Epidemiology

Introduction

The main characteristic features of the *Brucella*genus is its ability to survive within phagocytic and non-phagocytic cells. While a wide variety of factors explain the capacity of the *Brucella* genus to multiply and spread to new cells, so far no single factor has been shown to be responsible for its virulence (Celli *et al*., 2004).

Brucellae usually enter in the body via the oral route and lodge in the mucosa, where the bacteria are ingested by professional phagocytes beneath the sub-mucosa. Once internalised, *Brucella*is localised in a vacuole that matures from an early to a late endosome and, unless destroyed, goes on to multiply in the endoplasmic reticulum of macrophages. These bacteria multiply abundantly in the placental cotyledons, chorion and fetal fluids, where they cause lesions in the organ wall, inducing endometriosis ulcerosa in the intercotyledonary spaces and destruction of the villi, leading to death and expulsion of the fetus (Xavier*et al*., 2009). Three species of *Brucella*affect humans: *B. melitensis, B. abortus*and *B. suis*(other species can cause infection in humans, but only rarely). Out of these three species, infections by *B. melitensis*are the most common in humans (Pappas *et al*., 2005).

Epidemiology of *Brucellaabortus*

Bovine brucellosis is usually caused by *B. abortus*. *Brucellaabortus*has seven recognized biovars, the most reported of which are biovars 1, 2, 3, 4 and 9. The primary source of

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infection for cattle is believed to be elk (*Cervuselaphus*) (Van Campen*et al*., 2010). Ruminants are generally susceptible to *B. abortus*, which is of particular relevance in areas where eradication programmes are in operation. Buffaloes, camels, deer, goats and sheep are highly susceptible to infection (Cvetnic*et al*., 2005). The manifestations of brucellosis in these animals are similar to those of bovine brucellosis and can become epidemiologically important in sustaining the infection in cattle where they share pasture and water holes (Centers for Disease Control and Prevention CDC., 2011). The infection is prevalent in horses cohabiting with cattle, it presents with characteristic swelling of the supraspinous bursa, known as fistulous withers. The infection is usually transmitted to pigs by feeding them whey as a by-product from cheese-making (Muñoz et al., 2010; Musa M.T. &Jahans K.L. 1990). The main route of entry for *Brucella*is oral, by the ingestion of food or water contaminated with secretions or aborted fetal remains from infected cows, or by licking the vaginal secretions, genitals, aborted fetuses or newborn calves of infected cows. Infected cows shed *Brucella*in their milk and this is key in its transmission to calves.

The disease is usually asymptomatic in non-pregnant females, but pregnant adult females infected with *B. abortus*develop placentitis, which normally causes abortion between the fifth and ninth month of pregnancy. Adult males can develop orchitis, and brucellosis can cause sterility in both sexes. Neutered males used for fattening are not important in the distribution of the disease (Samartino L. 2003;CarvalhoNeta, 2010; World Organisation for Animal Health (OIE) ,2010).

Epidemiology of *Brucellasuis*

Domestic pigs are mainly infected by *B. suis*.. There are five biovars of *B. suis*, with 1, 2 and 3 being responsible for porcine brucellosis worldwide. *Brucellasuis*biovars 1 and 3 are distributed worldwide in most areas where there are pigs. They affect both sexes, causing infertility, abortion, orchitis and bone and joint lesions.

*Brucellasuis*is moderately influenced by environmental factors, the bacteria often survive dessication and can survive freezing temperatures for over two years (Public Health Agency of Canada (PHAC), 2009).

The *B. suis*entry sites are similar to those identified for other types of *Brucella*infection, being essentially the oral, nasopharyngeal, conjunctival and vaginal mucosa. These are not usually visible in young animals, and their occurrence will depend mainly on the age, sex and physiological state of animals at the time they are infected.

In a primary infection with *B. suis* in pig farms, the bacteria can spread within a few months from one infected pig tomore than 50% of animals on the farm. The infection can often reach 70% to 80% of infected animals at the start of the outbreak (Philipon et al., 1970; Beer J. 1980). However, recently infected herds may manifest major signs of infection, such as a high percentage of abortions, increased neonatal mortality and infertility, causing adverseeconomic consequences. Porcine brucellosis is believed to affect both sexes equally and age is no determinant of susceptibility, although thisis not proven. It has also been reported that some pig breeds, such as Duroc and Jersey Red crosses, may be lesssusceptible to experimental challenge with *B. suis*, which suggests the existence of genetic resistance to infection (Cameron*et al*., 1942).

Brucellosis infection caused by *B. suis*biovar 2 differs from that caused by biovars 1 and 3 in terms of geographicaldistribution, host and virulence, and is considered less pathogenic for humans than the highly infectious biovars1 and 3; humans must be immunocompromised to become infected with biovar 2 (Garin-Bastuji et al., 2006;Lagier, 2005; Meirelles-Bartoli et al., 2012).

Epidemiology of *Brucellamelitensis*

*Brucellamelitensis*is the most virulent species of the *Brucella*genus and has three biovars, with biovars 1 and 3 beingthe ones isolated most frequently in small ruminants (Lucero et al., 2008; Blasco J.M. & Molina-Flores B. (2011). Brucellosis causes a significant losses from abortion, as well as being a serious zoonosis (Banai M. 2007; Benkirane A. 2006; Seleem *et al.,* 2010).

Goats are the classic and natural host of *B. melitensisn* and, together with sheep, are its preferred hosts. The main clinical manifestations of brucellosis in ruminants are abortion and stillbirths, which usually occur in the lastthird of the pregnancy following infection and usually only once in the animal's lifetime (Blasco J.M. & Molina-Flores B. 2011; Elzer *et al*., 2002). *B. melitensis* can be transmitted congenitally *in utero* but only a small proportion of lambs and kids are infected in this way and most latent infections of *B. melitensis* are probably acquired by ingesting colostrum or milk (Grillo M.J., Barberán M. & Blasco J.M. 1997). It is therefore recommended that infected females and their offspring be culled as part of an eradication programme in infected herds (Banai M. (2007). The exact mechanism enabling latent *Brucella* infection to develop is unknown (Blasco J.M. & Molina-Flores B. 2011).

Some female hoggets testing seropositive to brucellosis havebeen found to shed *B. melitensis* in milk postpartum, whereas others do not shed brucellae despite being infected. A previously unreported fact is that *B. melitensis* was successfully isolated from the vaginal discharge of a goat that had aborted but tested seronegative for brucellosis, making the animal a potential risk for spread undetectable by serological diagnosis (Herrera *et al*., 2011).

While orchitis and epididymitis are uncommon in rams and billy goats, they do occur (Chand P., Sadana J.R. & Malhotra A.K. 2002). *Brucellamelitensis* biovar 3 has been isolated from a testicular hygroma of a ram (Musa M.T. & Jahans K.L. 1990). *Brucellamelitensis* can infect not only cattle but also calves, through the ingestion of infected milk. The isolation of *B. melitensis* in dogs has been demonstrated and this has been observed to favour incidence of the disease, as dogs can drag placentas or aborted fetuses to uninfected areas.

In extensive goat and sheep farms, it is common practice for herds to share pasture and water holes before returningto their pens. Such mixing of animals is a factor of risk for spreading the disease from infected to free herds and makes it harder to control. In this case, all goats sharing such sites must be considered as a single, large herd, and all goat farmers must carry out control activities, e.g. vaccination and the separation of positive and negative animals.

Conclusion

Any strategy for the control or eradication of brucellosis in domestic animals should be begin by establishing the different epidemiologicalcontexts within a country or even a region or district, and must have the support and collaboration of farmers. Aboveall, the effectiveness of any such strategy will rely heavily on the quality of the Veterinary Services and administrative organizations involved, because the requisite diagnostic and prophylactic tools are already fully validated and standardized.

References

[1] Abernethy D.A., Moscard-Costello J., Dickson E., Harwood R., Burns K., McKillop E., McDowell S. & Pfeiffer D.U. (2011).–Epidemiology and management of a bovine brucellosis cluster in Northern Ireland. *Prev. vet. Med*., **98** (4), 223–229.

[2] Alton G.G. (1990). – *Brucellasuis*. *In* Animal brucellosis (K. Nielsen & J.R. Duncan, eds). CRC Press, Boca Raton, Florida, 411–422. Beer J. (1980).

[3] Banai M. (2007). – Control of *Brucellamelitensis*. Memorias del IV ForoNacional de Brucelosis, Facultad de Medicina Veterinaria y Zootecnia de la Universidad NacionalAutónoma de México (FMVZ-UNAM), 26–27 November, Mexico, DF.

[4] Benkirane A. (2006). – Ovine and caprine brucellosis: world distribution and control/eradication strategies in West Asia/North Africa region. *Small Rum. Res.*, **62** (1–2), 19–25.

[5] Blasco J.M. & Molina-Flores B. (2011). – Control and eradication of *Brucellamelitensis* infection in sheep and goats.*Vet. Clin. N. Am. (Food Anim. Pract.)*, **27** (1), 95–104.

[6] Cameron H.S., Hughes E.H. & Gregory P.W. (1942). – Genetic resistance to brucellosis in swine. *J. Anim. Sci.*, **1**, 106–110.

[7] Carvalho Neta A.V., Mol J.P., Xavier M.N., Paixão T.A., Lage A.P. & Santos R.L. (2010). – Pathogenesis of bovinebrucellosis. *Vet. J.*, **184** (2), 146–155.

[8] Celli J. & Gorvel J.P. (2004). – Organelle robbery: *Brucella*interactions with the endoplasmic reticulum. *Curr. Opin. Microbiol.*,**7**(1), 93–97.

[9] Centers for Disease Control and Prevention (CDC) (2011). – Summary of notifiable diseases: United States, 2009. *MMWR*,**58** (53), 1–100.

[10] Chand P., Sadana J.R. & Malhotra A.K. (2002). – Epididymoorchitis caused by *Brucellamelitensis* in breeding rams in India.*Vet. Rec.*, **150** (3), 84–85.

[11] Cvetnic Z., Spicic S., Curic S., Jukic B., Lojkic M., Albert D., Thiébaud M. & Garin-Bastuji B. (2005). – Isolation of *Brucellasuis* biovar 3 from horses in Croatia. *Vet. Rec.*, **156** (18), 584–589.

[12] Elzer P.H., Hagius S.D., Davis D.S., DelVecchio V.G. & Enright F.M. (2002). – Characterization of the caprine modelfor ruminant brucellosis. *Vet. Microbiol.*,**90** (1–4), 425–431.

[13] Fosgate G.T., Diptee M.D., Ramnanan A. & Adesiyun A.A. (2011). – Brucellosis in domestic water buffalo (*Bubalusbubalis*) of Trinidad and Tobago with comparative epidemiology to cattle. *Trop. anim. Hlth Prod.*, **43** (8), 1479–1486.

[14] Garin-Bastuji B., Vaillant V., Albert D., Tourrand B., Danjean M.P., Lagier A., Rispal P., Benquet B., Maurin M., De Valk H. & Mailles A. (2006). – Is brucellosis due to the biovar 2 of *Brucellasuis* an emerging zoonosis in France? Two case reports in wild boar and hare hunters. *In* Proceedings of the International Society of Chemotherapy Disease Management Meeting, 1st International Meeting on Treatment of Human Brucellosis, 7–10 November, Ioannina, Greece.

[15] Gomo C., de Garine-Wichatitsky M., Caron A. & Pfukenyi D.M. (2012). – Survey of brucellosis at the wildlife– livestock interface on the Zimbabwean side of the Great Limpopo Transfrontier Conservation Area. *Trop. anim. Hlth Prod.*, **44** (1), 77–85.

[16] Grillo M.J., Barberán M. & Blasco J.M. (1997). – Transmission of *Brucellamelitensis* from sheep to lambs. *Vet. Rec*., **140** (23),602–605.

[17] Herrera E., Rivera A., Palomares E.G., Hernández-Castro R. & Díaz-Aparicio E. (2011). – Isolation of *Brucellamelitensis* from a RB51-vaccinated seronegative goat. *Trop. anim. Hlth Prod*., **46** (6), 1069–1070.

[18] Hinić V., Brodard I., Petridou E., Filioussis G., Contos V., Frey J. & Abril C. (2012). – Brucellosis in a dog caused by*Brucellamelitensis*. Rev 1. *Vet. Microbiol*.,**141** (3–4), 391–392.

[19] Infektionskrankheiten der Haustiere (Infectious diseases of domestic animals), 2nd Ed. Jena, VEB Fischer-Verlag, 759 pp.

[20] Kahler S.C. (2000). – *Brucellamelitensis*infection discovered in cattle for fi rst time, goats also infected. *J. Am. vet. med.Assoc*., **216** (5), 648.

[21] Lagier A., Brown S., Soualah A., Julier I., Tourrand B., Albert D., Reyes J. & Garin-Bastuji B. (2005). – Brucelloseaiguë à *Brucellasuis*biovar 2 chez un chasseur de sanglier. *Méd. Mal. infect.*, **35**, 185.

[22] Lucero N.E., Ayala S.M., Escobar G.I. & Jacob N.R. (2008). – *Brucella* isolated in humans and animals in Latin America from1968 to 2006. *Epidemiol. Infect*., 136 (4), 496–503.

[23] Meirelles-Bartoli R.B., Mathias L.A. & Samartino L.E. (2012). – Brucellosis due to *Brucellasuis*in a swine herd associatedwith a human clinical case in the State of São Paulo, Brazil. *Trop. anim. Hlth Prod*., **44** (7), 1575–1579.

[24] Mikolon A.B., Gardner I.A., Hernandez de Anda J. & Hietala S.K. (1998). – Risk factors for brucellosis seropositivity of goat herds in the Mexicali Valley of Baja California, Mexico. *Prev. vet. Med*., **37** (1–4), 185–195.

[25] Muñoz P.M., Boadella M., Arnal M., de Miguel M.J., Revilla M., Martínez D., Vicente J., Acevedo P., Oleaga A., Ruíz-Fons F., Marín C.M., Prieto J.M., de la Fuente J., Barral M., Barberán M., de Luco D.F., Blasco J.M. & Gortázar C. (2010). – Spatial distribution and risk factors of brucellosis in Iberian wild ungulates. *BMC infect. Dis*., **5**, 10–46.

[26] Musa M.T. & Jahans K.L. (1990). – The isolation of *Brucellamelitensis* biovar 3 from a testicular hygroma of a ram in anomadic flock of sheep and goats in Western Sudan. *J. comp. Pathol*.,**103** (4), 467–470.

[27] Ocholi R.A., Bertu W.J., Kwaga J.K., Ajogi I., Bale J.O. &Okpara J. (2004). – Carpal bursitis associated with *Brucellaabortus*in a horse in Nigeria. *Vet. Rec*., **155** (18), 566–567.

[28] Pappas G., Akritidis N., Bosilkovski M. &Tsianos E. (2005). – Brucellosis. *N. Engl. J. Med*., **352**, 2325–2336.

[29] Philipon A., Renoux G. & Plommet M. (1970). – Experimental bovine brucellosis. 3. The vaginal excretion of *Brucellaabortus*before and after parturition. *Ann. Rech. vét*.,**1** (2), 215–224.

[30] Public Health Agency of Canada (PHAC) (2009). – *Brucella*spp. (*B. abortus*, *B. canis*, *B. melitensis*, *B. suis*). Material SafetyData Sheets (MSDS). Available at: www.phac-aspc.gc.ca/msdsftss/ msds23e-eng.php (accessed on 12 July 2012).

[31] Samartino L. (2003). – Conceptosgeneralessobre brucellosis bovina. Jornada de actualizaciónsobrebrucelosis bovina,Rocha, InstitutoNacional de TecnologíaAgropecuaria (INTA), Castelar, Argentina. Available at: www.mgap.gub.uy/DGSG/Capacitación/JornadasBrucelosis/ (accessed on 12 July 2012).

[32] Seleem M.N., Boyle S.M. &Sriranganathan N. (2010). – Brucellosis: a re-emerging zoonosis. *Vet. Microbiol*.,**140** (3–4), 392–398.

[33] Van Campen H. &Rhyan J. (2010). – The role of wildlife in diseases of cattle. *Vet. Clin. N. Am. (Food Anim. Pract.)*, **26**, 147–161.

[34] Verger J.M., Garin-Bastuji B., Grayon M. &Mahé A.M. (1989). – Bovine brucellosis caused by *Brucellamelitensis*in France.*Ann. Rech. vét*.,**20** (1), 93–102.

[35] World Organisation for Animal Health (2012). – World Animal Health Information Database(WAHID).Availableat:www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail (accessed on 12 December 2012).

[36] World Organisation for Animal Health (OIE) (2010). – Bovine brucellosis, Chapter 2.4.3. [Version adopted by the WorldAssembly of Delegates of the OIE in May 2009]. *In* Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. OIE,Paris. Available at:www.oie.int/fi leadmin/Home/eng/Health_ standards/tahm/2.04.03_bovine_brucell.pdf (accessed on13 November 2012).

[37] Xavier M.N., Paixão T.A., Poester F.P., Lage A.P. & Santos R.L. (2009). – Pathological, immunohistochemical and bacteriological study of tissues and milk of cows and fetuses experimentally infected with *Brucellaabortus*. *J. comp. Pathol*.,**140**(2–3), 149–157.