

**FACTORES QUE INFLUYEN EN LA MORTALIDAD NEONATAL DE
BECERROS HOLSTEIN EN UN CLIMA CÁLIDO**

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TESIS

Presentada como requisito parcial para optar al grado de:

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FACTORES QUE INFLUYEN EN LA MORTALIDAD NEONATAL DE
BECERROS HOLSTEIN EN UN CLIMA CALIDO

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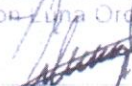


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I. INTRODUCCIÓN

La salud de los terneros de reemplazo son componentes importantes de la rentabilidad total de la operación lechera intensiva. La productividad del hato puede verse afectada negativamente por las altas tasas de mortalidad de los becerros , ya que aumenta los costos veterinarios, limita la selección genética y aumenta la necesidad de adquisición de animales de reemplazo. Entre los animales de la granja, las tasas de mortalidad más altas ocurren en periodo pre - destete de terneros lecheros criados artificialmente (Curtis et al, 1988; Wells et al, 1996).

La crianza de becerras es, quizá, la operacion más trascendente en la ganaderia lechera. La becerria que actualmente se encuentra en alguna etapa del proceso de crianza, en un periodo de 1 a 2 años se convertirá en una vaca en fase de produccion. En la mayoría de los hatos lecheros, se desechan de 20 a 30% de los animales en produccion cada año, lo cual significa que se debe contar con suficientes remplazos para mantener constante el número de cabezas del hato adulto.

Se ha indicado que diversos efectos de manejo tales como: tamaño del hato, métodos de alimentacion, tipo y tamaño de caseta y personal que alimenta a las becerras tienen influencia sobre el porcentaje de mortalidad de crías en el establo. De igual forma, se reconoció que diversos efectos ambientales como: epoca de nacimiento, temperatura, viento y precipitación pluvial, tienen relevancia sobre el índice de sobrevivencia de las crías del establo, al igual que diversos

efectos de tipo genético, como: el número de parto de la vaca incidencia de partos múltiples en el establo, sexo y raza de la cría.

Por ser la etapa de crianza la de mayor vulnerabilidad de los animales, se debe poner atención especial en el proceso de cría, ya que el mayor índice de mortalidad se presenta en el periodo, principalmente en el primer mes de vida. Lo que se haga y no con acierto, se traducirá en satisfacción o frustración para el ganadero y tendrá impacto directo sobre la economía de la empresa, sin importar sus dimensiones (Ben,1999).

II. REVISIÓN DE LITERATURA

2.1 Factores que desencadenan la mortalidad en becerros de lechería

Los resultados del primer estudio nacional de EE.UU. en salud de la vaca lechera indicaron que los factores asociados con la mortalidad en los primeros 21 días de vida incluyen como primer factor el método de alimentación con calostro, la sincronización y el volumen, el tiempo del destete; facilidad de parto y el nacimiento de mellizos. Las estimaciones atribuibles a la fracción de la población demostraron la importancia de estos factores en la prevención de la mortalidad de terneros. Este análisis indicó que hasta el 31% de la mortalidad de terneros durante los primeros 21 días de vida se podría prevenir por los cambios en la alimentación con calostro como primer método, tiempo y volumen (Wells et al., 1996).

Berglund et al. (2003), indican que la tasa de muerte en becerros a aumentado probablemente debido a una causa multifactorial. Como por ejemplo, terneros nacidos de novillas, presencia de patógenos, partos distócicos, defectos genéticos y el tamaño del hato.

Las condiciones climáticas, además de partos distócicos, la edad de la madre, el tamaño del becerro, y el sexo de la cría afectan la sobrevivencia de terneros desde el nacimiento hasta la primera semana de edad. Estos resultados demuestran que los terneros nacidos de vacas con partos distócicos tenían cinco veces más probabilidades de morir que los terneros nacidos de parto normal. De

todos los becerros que murieron, 43.6 % habían nacido con dificultad. De estos terneros, la supervivencia fue más baja para aquellos que eran pequeños en relación con su grupo genético, sexo, y edad de la madre. Los terneros grandes tenían marcado aumento de la mortalidad sólo cuando nacieron de vacas de más de 2 años de edad. La temperatura y precipitación en el día del parto influyeron en el pronóstico de mortalidad y la magnitud del efecto dependía de la edad de la madre, el sexo, el tamaño, y la incidencia de distocia. Los terneros nacidos de vacas de 2 años de edad, fueron más susceptibles a las condiciones meteorológicas adversas que los terneros nacidos de las vacas más viejas. El efecto negativo de la precipitación en la supervivencia incrementado con la disminución de temperatura. De los terneros nacidos con dificultad, la supervivencia fue más baja para los que eran pequeños en relación a su grupo genético, sexo, y edad de la madre (Berguer et al., 1992; Azzam et al., 1993).

2.2. Factores que desencadenan la distocia

Diversos factores afectan la distocia en el ganado. Los factores se agrupan en cuatro categorías principales: factores directos, factores fenotípicos relacionados con el ternero y la vaca, no genética y los factores genéticos. El primer grupo incluye presentaciones anormales y de torsión uterina. La segunda

incluye: el peso de los terneros al nacer, los partos múltiples, la mortalidad perinatal, el área pélvica de la vaca, el peso y la condición corporal de la vaca al parto y duración de la gestación. Los factores no genéticos son: la edad de la vaca, año y época de parto, lugar de parto, las prácticas de alimentación, los trastornos de salud, el sexo de la cría y la nutrición. Otros factores no genéticos son el nivel de hormonas en el periodo periparto, la producción *in vitro* de embriones y la clonación de embriones. Por último, los genotipos de la vaca, el toro, la endogamia, la hipertrofia muscular, la selección y los rasgos cuantitativos constituyen el cuarto grupo de factores genéticos (Zaborski et al., 2008). Un parto distócico afecta la mortalidad de terneros en las 24 h siguientes al nacimiento. El peso al nacer y la forma en que se presente el ternero explican la mayor parte de las variaciones observadas en la distocia (Nix et al., 1997).

2.3. Estrés calórico

El estrés por calor durante el período de sequía no sólo afecta negativamente al rendimiento de una vaca, también afecta a su descendencia. Estudios previos indican que los terneros nacidos de vacas estresadas por el calor durante la gestación tardía tienen menor peso al nacer (Tao et al., 2013). El estrés por calor también aumenta la temperatura rectal de los terneros recién nacidos (Kume et al., 1998).

El estrés por calor generalmente causa una disminución en el consumo de alimento. Por lo tanto, las altas temperaturas reducen el crecimiento y el aumento de la masa muscular en el ganado, dependiendo del genotipo, la edad, y

adaptabilidad al calor. Por otra parte un ganado estresado puede aumentar la ingesta de agua, estar de pie en lugar de acostarse, y aumentar la tasa de respiración y la temperatura corporal, así como, en los casos más graves producirse la muerte, lo que resultaría pérdidas económicas para la industria ganadera. Debido a que el principal beneficio del ganado lechero es la producción de leche, se protege a las vacas lecheras contra el estrés calórico. Sin embargo, éste es generalmente olvidado y no se toma en cuenta en terneros, casi todos los estudios se centran en las vacas lecheras o ganado de engorda. Las enfermedades que se asocian con la mortalidad neonatal y postnatal son una importante causa de pérdidas económicas en la producción lechera. En un estudio de Brouce et al. (2009) los terneros nacidos durante el período con temperaturas más altas tenían menores ganancias de peso vivo, bebían una mayor cantidad de agua hasta el destete, y consumían menos alimento desde la primera semana de vida hasta el destete.

El estrés, el transporte, un cambio repentino en la alimentación, el contacto con animales de otros establos, y el suministro interrumpido de la madre de Inmunoglobulinas después del destete crea terneros jóvenes susceptibles a las infecciones virales y bacterianas. La inmunización pasiva con calostro o un concentrado de inmunoglobulinas, la inmunización activa por la vacunación con antígenos virales son alternativas para reducir la mortalidad de los becerros (Vandenbossche et al., 1994; Sun et al., 2010).

En otro estudio se desarrolló un método para dividir el riesgo de mortalidad en terneros lecheros en las primera 16 semanas de vida. La proteína de suero

concentrado se utilizó para determinar la tasa de mortalidad de referencia de la población y la tasa de mortalidad debida a la transferencia pasiva inadecuada de inmunoglobulinas del calostro. Se estudiaron un total de 3,479 terneros, el 8.2% de los cuales falleció antes de las 16 semanas de edad. La tasa de mortalidad de referencia de la población fue de 5.0% y la tasa de mortalidad debido a la insuficiente transferencia pasiva de inmunoglobulinas fue del 3.2%. Treinta y nueve por ciento de la mortalidad observada se atribuyó a la inadecuada transferencia pasiva. Esta división del riesgo entre las fuentes de la transferencia-relacionadas y no relacionadas pasivas puede ser muy útil en la realización de investigaciones sobre los problemas de mortalidad de terneros en hatos lecheros (Danovan., 1988; Tyler et al., 1999).

2.4. Estrés por frío

Las causas del frío en los terneros recién nacidos, y sometidos a hipotermia, sumergiéndolos en agua; la temperatura varió de 15 a 17°C. Este estrés retardó el inicio de la absorción de inmunoglobulinas IgM, IgG e IgG2. Trabajos con perros han demostrado que la hipotermia es responsable de la disminución del flujo de la salida venosa desde el intestino delgado, disminución de la motilidad intestinal y tiene una reducción neta en el transporte de sustancias de la luz intestinal a la sangre. Por lo tanto, la hipotermia puede disminuir la absorción de calostro y por consiguiente disminuir la absorción de inmunoglobulinas de terneros (Olson et al., 1980).

2.5. Enfermedades infecciosas

Un estudio retrospectivo se llevó a cabo en 845 terneras nacidas durante 1991 en 30 granjas lecheras Holstein en el sureste de Minnesota. Los objetivos del estudio fueron describir la epidemiología de la morbilidad y la mortalidad en terneros lecheros desde el nacimiento hasta las 16 semanas de edad (con énfasis en las enfermedades respiratorias), se examinaron individualmente y las prácticas de manejo del hato como factores de riesgo de morbilidad y mortalidad de terneras, y para validar el diagnóstico de la mortalidad. Las tasas de incidencia de la morbilidad, enteritis y neumonía fueron 0.20, 0.15 y 0.10 casos por 100 en situación de riesgo durante el período del estudio. El riesgo de enteritis fue mayor en las 3 primeras semanas de vida. Siendo el riesgo de neumonía más alta a las 10 semanas de edad. Las tasas de letalidad promedio de 11.8 %, 17.9 % y 9.4 % para todos los diagnósticos de enteritis y neumonía, respectivamente. Las tasas promedio diaria de ganancia desde el nacimiento hasta las 16 semanas de edad fueron diferentes entre las granjas que tenían alojamiento de terneros insuficiente (0.8 kg día⁻¹) frente a los que tienen una vivienda adecuada ternero (1.0 kg/ día). Aproximadamente a la mitad de terneros se les tomaron muestras de sangre mensualmente desde el nacimiento hasta las 16 semanas de edad. De los terneros en la muestra, sólo 19 mostraron un aumento en los títulos séricos de virus respiratorios. Dieciséis terneros al VDVB, dos becerros a IBRV, y un becerro al virus de PI3. A 98 terneros de menos de 10 días de edad se les realizó la prueba de adecuación de la transferencia pasiva, 35 (35.7 %) tenían niveles de inmunoglobulinas en suero de menos de 800 mg dl⁻¹. No hubo diferencias

significativas en la mortalidad o la morbilidad entre los terneros que tenían la transferencia pasiva adecuada y los que no lo hicieron. La incidencia de mortalidad fue de 0.08 muertes por cada 100 días; 64 terneros murieron durante los 16 meses del estudio. El riesgo de muerte fue más alta a las 2 semanas de edad. La enteritis fue la causa más común de muerte (28 muertes, 44 % de todas las muertes), seguido de la neumonía (19 defunciones, 30 % de todas las muertes). Al comparar el diagnóstico productor de la mortalidad con los resultados de la necropsia arrojó sensibilidad de 58.3 % y 56 % y especificidad de 93 % y 100% para el diagnóstico de enteritis y neumonía, respectivamente. Se realizó una prueba estadística kappa de comparación para el diagnóstico del resultado de la necropsia y ésta fue de 0.47. Los patógenos más frecuentes aislados de becerros que murieron de enteritis fueron rotavirus (cinco terneros) y *Escherichia coli* (cuatro terneros). Los patógenos aislados de pulmones neumónica incluyen *Pasteurella multocida* (tres terneros), *Haemophilus somnus* (tres terneros) y *Pasteurella haemolytica* (una cría) (Sivula et al., 1996).

La diarrea en terneros de menos de 30 días de edad es una causa importante de la mortalidad neonatal en el oeste de Canadá. Durante un período de tres años, el promedio de incidencia de la diarrea, la mortalidad y las tasas de letalidad asociadas con diarrea fueron 16.7 % 1.51 % y 9.02 % - respectivamente- en terneros de menos de 30 día de edad. En otra encuesta la incidencia de la diarrea neonatal fue de 21.98 % con una tasa de mortalidad del 3.06 % y una tasa de letalidad del 13.96 %. En ambas encuestas, entre el 70 y el 80 % de todos los

hatos experimentaron algo de diarrea en el período neonatal, pero el 65% de estos hatos tenían mortalidades por diarrea (Mitchell et al., 1981).

En un estudio con becerras de lechería los factores de riesgo potenciales para la ocurrencia de diarrea fueron el Rotavirus y *Cryptosporidium*. Los terneros seropositivos se asociaron a un hato con mayor riesgo de diarrea. Otros factores que se encontraron que aumentan el riesgo de diarrea fueron el uso de suelo de hormigón de rejilla en corrales de grupos frente a otros tipos de, terneros nacidos en la manada frente a los terneros que son comprados, y terneros nacidos durante el invierno en comparación con otras estaciones del año (Gulliksen et al., 2009).

La enfermedad respiratoria bovina es una enfermedad multifactorial del ganado que involucran a muchos agentes patógenos y se puede utilizar para explorar el impacto de diferentes factores etiológicos o de otro tipo de esta compleja enfermedad. *P. multocida* fue la bacteria más común que se encontró en el líquido de los pulmones de los terneros; *Mannheimia haemolytica* (antes de *Pasteurella haemolytica*) no se detectó en absoluto. Según otros estudios, *P. multocida* y *M. haemolytica* son los predominantes (Nikunen et al., 2007).

Las enfermedades respiratorias aumentan el riesgo de muerte en todos los grupos de edad con los cocientes de riesgo (HR) de 6.4 , 6.5, 7.4 , y 5.6 durante la primera semana de vida, de 8 a 30 días de edad, 31 a 180 días de edad, y de 181 a 365 días de edad, respectivamente. La diarrea aumentó el riesgo de muerte entre los terneros de menos de 180 días de edad, pero la influencia sólo fue significativa durante la primera semana de vida y entre 8 y 31 d de edad (HR = 2.4

y 2.9. respectivamente). Los terneros nacidos durante el invierno eran más propensos a morir durante la primera semana de vida que los terneros nacidos durante el verano (OR = 1.2), y tenían más probabilidades de morir durante el primer mes de vida que los terneros nacidos durante el otoño (OR = 1.2). Las tasas de mortalidad de los becerros en todos los grupos de edad se incrementaron con el aumento de tamaño del hato. Los terneros alojados en un corral de grupo de 2 semanas de edad tenían más probabilidades de morir durante el primer mes de vida que los terneros alojados individualmente (HR = 1.5). La bronconeumonía y enteritis fueron los diagnósticos postmortem más frecuentes, con tasas proporcionales de 27.7 y 15.4 %, respectivamente (Gulliksen et al., 2009).

Un brote grave de enfermedades entéricas y respiratorias asociadas con la infección por coronavirus bovino (BCoV) se presentó en un hato lechero en verano. El brote se produjo en un hato lechero del sur de Italia en la primera década de septiembre de 2006, cuando todavía se registraron temperaturas de verano, afectando a los terneros, novillas y vacas adultas (Decaro et al., 2008).

El efecto de los factores ambientales y las rutinas de manejo sobre el riesgo de diarrea, enfermedades respiratorias y otras enfermedades infecciosas se investigó en 3,081 terneras 0-90 días de edad en 122 hatos lecheros suecos. El riesgo de las enfermedades respiratorias se asoció significativamente con una concentración de amoníaco por debajo de 6 ppm (OR : 0.42, $P < 0.05$), mientras que la probabilidad de moderada a grave se asoció significativamente con una infección de VDVB en el hato (OR : 2.39; $P < 0.05$) y el clima frío (OR : 3,7 , $P < 0.02$). La ausencia de calostro de buena calidad se asoció significativamente con

el riesgo de las enfermedades infecciosas diferentes a la diarrea y las enfermedades respiratorias (OR: 0.42) (Lundorg et al., 2005). Especialmente coccidias que se acumulaban, la mala ventilación y la transmisión directa de agentes patógenos de los animales de más edad para los recién nacidos (Hill et al., 2011).

III. LITERATURA CITADA

- Azzam, S.M., Kinder, J.E., Nielsen, M.K., Werth, L.A., Gregory, K., Cundiff, V. 1993. Environmental effects on neonatal mortality of beef calves. *J. Anim. Sci.* 71, 282-290.
- Ben, A.A. 1999. Manual de cría de becerras. Editorial Acribia. pp. 3-9.
- Berger, P.J., Cubas, A.C., Koehler, K.J., Healey, M.H. 1992. Factors affecting dystocia and early calf mortality in Angus cows and heifers. *J. Anim. Sci.* 70, 1775-1786.
- Berglund, B., Steinbock, L., Elvander, M. 2003. Causes of stillbirth and time of death in Swedish Holstein calves examined post mortem. *Acta Vet. Scand.* 44,111-120.
- Brouce, J., Kisac, P., Uhrincat, M. 2009. Effect of hot temperatures on the hematological parameters, health and performance of calves. *Int. J. Biometeorol.* 53, 201–208.
- Decaro, N., Mari, V., Desario, C., Campolo, M., Elia, G., Martella, V., Greco, G., Cirone, F., Colaianni, M.L., Cordioli, P., Buonavoglia, C. 2008. Severe outbreak of bovine coronavirus infection in dairy cattle during the warmer season. *Vet. Microbiol.* 126, 1-3.
- Gulliksen, S.M., Jor, E., Lie, K.I., Hamnes, I.S., Loken, T., Akerstedt, J. Osteras, O. 2009. Enteropathogens and risk factors for diarrhea in Norwegian dairy calves. *J. Dairy Sci.* 92, 5057-5066.
- Gulliksen, S.M., Jor, E., Lie, K.I., Loken, T., Akerstedt, J., Osteras, O., 2009b. Respiratory infections in Norwegian dairy calves. *J. Dairy Sci.* 92, 5139-5146.
- Hill, T.M., Bateman II, H.G., Aldrich, J.M., Schlotterbeck, R.L. 2011. Comparisons of housing, bedding, and cooling options for dairy calves. *J. Dairy Sci.* 94, 2138-2146.
- Lundborg, G. K., Svensson, E.C., Oltenacu, P.A. 2005. Herd-level risk factors for infectious diseases in Swedish dairy calves aged 0-90 days. *Prev. Vet. Med.* 68, 123-143.
- Mitchell, P.J., Hooper, P.T., Collyer, D.N., Letter, S. 1981. Heat stress and diarrhoea in neonatal calves. *J. Vet. Aust.* 57, 392-398.
- Nix, J.M., Spitzer, J.C., Grimes, L.W., Burns, G.L., Pliier, B.B. 1997. A retrospective analysis of factors contributing to calf mortality and dystocia in beef cattle. *Theriogenology* 49, 1515-1443.

- Olson, D.P., Papasian, C.J., Ritter, R.C. 1980. The effects of cold stress on neonatal calves. *Can. J. Comp. Med.* 44, 19-23.
- Nikunen, S., Hartel, H., Orroc, T., Neuvonend, E., Tanskanene, R., Kivela S.L. Sankarif, S., Ahog, P., Pyorala, S., Saloniemi, H., Soveri, T. 2007. Association of bovine respiratory disease with clinical status and acute phase proteins in calves. *Comp. Immunol. Microbiol. Infect. Dis.* 30, 143-151.
- Kume, S., Toharmat, T., Kobayashi, N. 1998. Effect of restricted feed intake of dams and heat stress on mineral status of newborn calves. *J. Dairy Sci.* 81, 1581-1590 .
- Sivula, N.J., Ames, T.R., Marsh, W.E., Werdin, R.E. 1996. Descriptive epidemiology of morbidity and mortality in Minnesota dairy heifer calves. *Prev. Vet. Med.* 27, 155-171.
- Sun, P., Wang, J.Q., Zhang, H.T. 2010. Effect of stereochemistry of avermectin-like 6,6- spiroketals on biological activities and endogenous biotransformations in *Streptomyces avermectinius*. *J. Dairy Sci.* 93, 3168-3173.
- Tao, S., Monteiro, A.P.A., Hayen, M.J., Dahl, G.E. 2014. Effect of late-gestation maternal heat stress on growth and immune function of dairy calves. *J. Dairy Sci.* 97, 897-901.
- Tyler, J.W., Hancock, D.D., Thorne, J.G., Gay, C.C. Gay, J.M. 1999. Partitioning the mortality risk associated with inadequate passive transfer of colostral immunoglobulins in dairy calves. *J. Vet. Int. Med.* 13, 335–337.
- Vandenbossche, C.M.R., Vermeerscr, H., Embrecht, E., Theys, H., Baeck, N., Remon, J.P. 1994. Effect of adjuvant administration on weight gain, infectious diseases, and mortality of veal calves, *Vet. Quart. J. Anim. Sci.* 94, 2138-2146.
- Wells, S.J., Garber, L.P., Hill, G.W. 1996. Health status of preweaned dairy heifers in the United States. *Prev. Vet. Med.* 29, 185-199.
- Zaborski, D., Grzesiak, W., Szatkowska, I., Dybus, A., Muszynska, M., Jedrzejca, M. 2008. Factors affecting distociaa in cattle. *Reprod. Domest. Anim.* 44, 540-551

Factors associated with neonatal dairy calf mortality in a hot-arid environment

ABSTRACT

A field study involving 7734 Holstein calves from eight large intensive dairy herds in northern Mexico (26° N; 24.2°C mean annual temperature; same location) was conducted to determine factors associated with early postnatal (one to 21-d of age) dairy calf mortality. The effects of season of birth, size of dairy operation, birth type (single or twin), sex of calves, type of feeding (milk or milk replacer), herd and temperature-humidity index (THI) shortly before, during or shortly after calving were analyzed by the GENMOD procedure of SAS. The total mortality rate was 14% (range among dairy operations 7.0-29.1%). Mortality of calves was associated with the THI during birth, with a decreased ($P<0.05$) mortality rate from 16 to 12% with THI above 81 units. Mortality rates were 4 percentage point higher ($P<0.05$) in winter than all other seasons. Single-born calves had fewer deaths (14%) than twin-born calves (19%). The mortality rate was higher ($P<0.05$) in male than in female calves (17 vs. 12%). Calves that were born in dairy operations >1800 cows were more likely to die than calves born in smaller dairies. Both respiration rate (74.4 ± 15.6 vs. 67.8 ± 16.3) and heart rate (119.2 ± 19.6 vs. 113.9 ± 17.0) were higher ($P<0.05$) in calves born in winter than in summer. Rectal temperature was not altered by high ambient temperature and panting was not observed in calves during days with high ambient temperature. It was concluded that in this particular zone characterized by an intense heat load for most of the year, calf mortality was not linked to high ambient temperature around calving, rather, winter weather

negatively affected survival of calves. Thus, efforts to protect calves from cold weather would likely alleviate calf losses.

Keywords: Panting score, Twin calving, Temperature-humidity index, Calf mortality, Rectal temperature

1. Introduction

The health of replacement calves is an important component of total dairy operation profitability (Razzaque et al., 2009). The productivity of the herd can be negatively affected by high rates of calf mortality, because it increases veterinary costs, limits genetic selection and increases the need for acquisition of replacement dairy animals. Among farm animals, the highest mortality rates generally occur in pre-weaning dairy replacement calves raised under typical dry-lot management conditions (Curtis et al., 1988; Wells et al., 1996). Death rate in dairy calves in the first 16 weeks of life in temperate climates varies considerably among herds, but usually ranges from 8 to 12% (Tyler et al., 1999; Magalhães et al., 2008; Torsein et al., 2011). This mortality rate can be higher in regions with chronic high ambient temperatures, because the new-born calf is not fully capable for regulating its core body temperature (Spain and Spiers, 1996). Potential shortcomings on performance of new-born calves subjected to intense heat load might reflect impairment of immune function, because heat stress of the dam during the dry period not only compromises the fetal growth but also the immune function of offspring from birth through weaning (Tao et al., 2012). Additional shortcomings are increased incidence of diseases (Trotz-Williams et al., 2007), decreased feed intake (Brouce et al., 2009), decreased birth weight (Collier et al.,

1982; Wolfenson et al., 1988), reduced weight gain (Brouce et al., 2009; Hill et al., 2011), which increased heifers' age at first calving (Heinrichs et al., 2005), and reduced energy status (Hahn, 1999). Heat stress may be responsible for impairment of the protective value of bovine colostrums (Nardone et al., 1997; Morin et al., 2001). There is abundant research on heat stress effects on lactating dairy cows (Mellado et al., 2013), but less information is available for the effect of heat stress on dairy calves, particularly in hot-arid environments. Knowing the risks and understanding the impact of extreme weather on dairy calves mortality can help producers implement management practices to reduce these losses.

Thus, it was hypothesized that hyperthermia either in uterus (two weeks prior to calving) and the day of calving increase the likelihood of dyeing of Holstein calves. Objectives were to determine how temperature-humidity index (THI) shortly before and after calving, sex of calves, season of calving, single or twin calving, feeding regime, herd and dairy operation size affect mortality rate of Holstein calves during the first three weeks of life. An additional objective was to assess the effect of cold or warm weather on physiological variables of calves indicative of heat stress.

2. Material and Methods

2.1 Animals and management

Seven thousand seven hundred thirty-four Holstein calves from 8 large commercial dairy farms close to each other (within a radius of 20 km) and located in northern Mexico (26°N) were included in the study. The criteria for recruiting herds into the present study were closeness between herds, similarity in calf rearing facilities, presence of large numbers of milking cows (most over 1000 animals), and

presence of individual maternity pens. These dairy herds were truly representative of “La Laguna” dairy zone (the biggest dairy region in Mexico). The average cow inventory for the 8 herds was 1805 heads (SD = 445), and ranged from 410 to 2132 cows. Parturitions were spread fairly evenly throughout the year. Calves were born between January 1, 2009, and December 31, 2010. Calves were followed from birth to 21 days of age. Calves were housed in individual outdoor 1.2 × 2.4 m portable pens with tube sides and plywood roofs with a covered area of approximately 1.8 m². These pens had adequate ventilation at a low and high level, they were clean and dry with no bedding and good drainage. Each pen had a double pail holder for 6.15 liter pails. Pens were about 0.5 meters apart from one another.

When parturition approached cows were moved to isolated quiet and clean individual maternity pens. After parturition calves remained with their dams during the first two to six hours of life. Upon separation from their mothers all calves received 2 feedings of 1.5 to 2 L of colostrum (depending of calf size; total of 3 to 4 litres per day) each in the first 3 d of life. Thereafter, calves were fed either nonmedicated milk replacer (approximately 20% crude protein and 20% fat) or milk twice daily. Calves were offered 2 L in buckets at each feeding during the first 45 d of age and then once a day until 50 to 60 d of age, when calves were weaned from milk. Quality of assistance provided to calves was not registered.

In general, calves were fed a mixture of grains (20 to 22% CP), alfalfa hay (17 to 19% CP), and water from a bowl drinker ad libitum. This diet met or exceeded nutrient requirements for pre-weaned Holstein calves to achieve maximum growth

rate (NRC, 2001). Grain was fed once a day in the morning, immediately after milk feeding, for ad libitum intake during the first 60 d of age.

Weather data were obtained from a meteorological station located 2 to 7 km away from the dairy operations throughout the study. Information consisted of daily maximum temperatures and relative humidity 15, 10 and 5 d prior to calving as well as the day of calving. This information was used to calculate the THI for each day, using the following equation (Hahn, 1999; highest ambient daily temperature in Celsius degrees):

THI = 0.8 * ambient temperature + [(relative humidity/100) * (ambient temperature - 14.3)] + 46.4. Additionally, the occurrence of precipitation the day of calving was recorded.

Information at birth for calves included herd identification, calving date, multiple births, calf gender, and calf mortality from birth to 21 d of age. Editing of data removed individuals having calved with less than 275 (n= 122) or greater than 285 (n=1654) days of gestation, as well as calves coming from difficult births (n= 621, regardless of parity). All data were verified with the producer's veterinarians and cross-checked with an external veterinarian who monthly gathered the information from the eight dairy operations included in this study.

Respiratory and heart rates as well as rectal temperature and panting score were measured at noon in one-to five-days-old calves kept on roofed individual pens on three adjacent large dairy operations, as indicators of the degree of heat stress experienced by new-born calves. These measurements were made by an experienced veterinarian. Half of the measurements were made in January and the

other half in June. Mortality rate was defined as the proportion of dead calves registered during the first three weeks of life, out of all calves born alive.

2.2 Statistical analyses

A preliminary analysis of the data was carried out to detect and remove from the model variables not statistically related to calf mortality. For this purpose a multiple logistic regression using SAS software (SAS Institute, Inc., Cary, NC, USA) was carried out to assess the statistical significance of calf gender, type of birth (single or twin), operation size, feeding regime (use of milk or milk replacer), calving season, herd, THI (15, 10 and 5 d prior to calving), THI at calving and THI 5 d after calving, precipitation the day of calving, as well as calf gender x season and type of birth x season interactions on calf mortality. The backward stepwise procedure was used with the log-likelihood ratio and the criteria $P=0.05$ to enter and $P=0.10$ to remove. This preliminary stepwise selection did not identify THI previous or after calving, precipitation the day of calving and single interactions as prognostic factors for calf mortality, and consequently were removed from the model. Data were also tested for multicollinearity (Proc Reg procedure of SAS with the vif, tol, and Collin options) and evidence of it was found for THI before, during and after parturition, therefore only THI the day of calving was included in the final model.

For the analysis of the reduced model the GENMOD procedure of SAS with the logit link function was used. The model included:

model Logit (Mortality) = Season + Birth type + Gender + Operation size + Feeding type + Calving season + Herd + THI the day of calving; year was included as covariate. Model fit to the data was tested using Hosmer–Lemeshow Goodness-

of-fit test (LACKFIT option of SAS). This test ($\chi^2= 19.7$; $P=0.011$) suggested that the model fit the data soundly well. The LSMEAN (PDIF) statement was used for means separation among seasons of birth and THI categories at birth. Confidence limits for calf mortality were calculated with the proc freq statement of SAS with the riskdiff option.

Months were grouped into seasons, winter months being January–March; spring, April–June; summer, July–September; and fall, October–December. Calves that died or survived were coded as a dichotomous variable (0 = dead; 1 = alive); size of dairy operations was grouped in two classes: < or >1,800 cows (four dairy operations per category).

The relationship between THI the day of calving and mortality rate was analyzed by fitting a curve using a nonlinear regression. The model was fitted using the CurveExpert Professional program (version 1.6.5; Levenberg-Marquardt method), which selected the best-fitting model. For this regression analysis THI was divided into 6 classes, with the first class being <78; subsequent classes were set at each three units thereafter until the last class, which was >87. The LSMEAN (PDIF) statement of SAS was used to compare mortality rate among categories of THI the day of calving. The MIXED procedure of SAS was used to compare continuous variables indicative of heat stress in calves born either in January or June.

3. Results and Discussion

Overall mortality rate (14%) was higher than values reported for well-managed Holstein cattle in temperate (<8%, Svensson et al., 2006; Lombard et al., 2007; Magalhaes et al., 2008) and subtropical environments (<12%, Donovan et al.,

1998). Given the similarities in climate, facilities and management in dairy operations in the zone where this study took place, it is presumed that the high mortality rate found in the dairy farms studied can be extrapolated to the entire arid and semi-arid region of northern Mexico.

Differences between herds in mortality rates were found (range 7.0-29.1%; $P < 0.01$). Herd environment, genetic differences between herds and interactions between these variables seems to provide an appreciable source of variation.

Mortality rates were not different between THI categories < 78 units (Fig. 1). On the other hand, mortality rates for calves born in days with a THI > 78 were lower ($P < 0.05$) than those born in days with cooler temperatures. Mortality of calves born in days with extreme heat load (> 87 units) were similar to values observed in calves born in days with milder climatic conditions.

A negative relationship existed between mortality rate of calves and THI at calving (Fig. 1). High THI had a minimal influence on mortality rate but low THI markedly increased mortality rate. This is in line with data of Stull et al. (2008), who also observed greater mortality rates in calves subjected to cold versus warm ambient temperatures. The regression suggests that the net effect of increased THI at calving would be favorable even at extreme heat stress (THI > 85). In fact, calves born on days with severe hyperthermia were less likely to die compared with calves born during cold weather (THI < 78). Thus, newborn dairy calves appear to be more stressed by cold than by heat exposure.

These results are at odds with the notion that heat stress has significant consequences for dairy calves. It has been reported that heat stress may be responsible for impairment of the protective value of bovine colostrums, via a lower

concentrations of IgG and IgA (Nardone et al., 1997; Tao et al., 2012), and alteration of plasma immunoglobulins of calves (Kelley et al., 1988; Lacetera, 1998). In the present study calves showed good heat tolerance. The thermo neutral zone for calves less than 21 d of age is 15 to 25°C (NRC, 2001), and heat stress can occur at temperatures greater than 32°C (60% relative humidity; THI= 83; Neuwirth et al., 1979; Gebremedhin et al., 1981). During this observational study, calves were exposed to temperatures exceeding their thermo neutral zone throughout most of the year, but this did not appear to impair physiology or behavior of the calf and was not reflected in higher mortality rates. The lack of effect of heat load on calf mortality seems to be due to the fact that calves under heat stress activate responses to adapt to high heat loads, reaching homeothermy without difficulty (Hahn, 1997); thus, fitness seems not to be hampered and health is not necessarily affected (Silanikove, 2000). Moreover, all calves were reared in roofed pens with open sides, and shade diminishes the severity of heat stress experienced by calves housed in these structures (Spain and Spiers, 1996).

High THI during the final days of pregnancy did not alter calf mortality. These results conflict with the generalized idea that heat stress affects calf viability by limiting fetal growth at the end of gestation and immune function (Tao et al., 2012). Heat stress alters endocrine dynamics of the mother (Collier et al., 1982), reduces placental weight (Thureen et al., 1992; Ross et al., 1996), uterine blood flow (Fowden et al., 2010), vascular endothelial growth factor (Regnault et al., 2002) and gravid uterine and umbilical glucose uptakes (Bell et al., 1999). The end result of these alterations is a reduction in birth weight of calves (Collier et al.,

1982; Tao et al., 2012). Cows in late gestation during hot weather have reduced feed intake, which can also result in lower calf birth weights.

In the present study the lack of effect of high ambient temperature on fetuses shortly before birth on subsequent calf mortality possibly was due to the chronic heat stress in this zone; cows present certain degree of acclimation, as heat-stress conditions do not provoke a drastic reduction in milk yield (Mellado et al., 2011). At or below the upper critical temperature (28.4°C; Dikmen and Hansen, 2009), Holstein cattle are capable of maintaining a stable body temperature (Kadzere et al., 2002), and they activate a series of mechanisms to counter the stressful effects induced by high heat loads (Collier et al., 2008).

Calf mortality rate differed among seasons of birth, peaking in winter and decreasing in all other seasons. However, offspring delivered in summer and fall was not more or less likely to die than were calves delivered in spring (Fig. 2). This demonstrates a clear environmental impact on the mortality rate of calves. These results are in line with other studies in temperate regions where the negative effects of winter calving (Godden et al., 2005; Silva del Río et al., 2007; Gulliksen et al., 2009a) or exposure to cold temperatures (Nonnecke et al., 2009) on calf mortality has been documented.

The higher mortality rates observed in winter does not appear to be related to a lower energy intake, as cold environments do not alter ingestion behavior of calves (Borderas et al., 2009). Higher mortality rates in winter could be due to the impaired absorption of immunoglobulins from colostrum (Norheim and Simensen, 1985; Beam et al., 2009), or colostrum with lower quality during winter than

summer (Shearer et al., 1992). Also, there is evidence that calves born in winter have a greater risk for developing diarrhea (Gulliksen et al., 2009c).

Another cause of high mortality rate in winter in the present study probably was insufficient protection of calves from drafts and cold weather (bedding was not used in pens of calves in the dairies studied, and this management practice seems to be fundamental for ameliorating cold stress in newborn calves (Lago et al., 2006). Another reason for the cold-related deaths could be the increase in respiratory infections, which spread more readily in cold weather. Apart from that, breathing of cold air stimulates coughing and running of the nose and this helps to spread respiratory viruses and bacteria. Cold stress also tends to suppress immune responses to infections (Godden et al., 2005; Beam et al., 2009).

The mortality rate was higher ($P<0.05$) in male than in female calves (Table 1), which is similar to what has been reported by Dhakal et al. (2013) and Nix et al. (1998). One reason for the higher mortality of male calves is that male calves, on average, weigh more than heifer calves at birth (McDermott et al., 1992; Johanson and Berger, 2003). This increased calf weight may cause a mismatch of fetal-maternal size, which increases the occurrence of dystocia (Azzam et al., 1993), and calves under these circumstances are more prone to die (Lombard et al., 2007). However, male calves have higher mortality rates than female calves even when dystocia and relative size of the calf is accounted for (Azzam et al., 1993). Also, Riley et al. (2004) found that Brahman male calves had greater odds of poor vigor (score based on capacity to nurse) than females. It is worth mentioning that male calves are not as valuable to the dairy operation as females and therefore

may not receive the attention the heifers do, possibly accounting for the higher mortality in the males.

Calf mortality rate from twin births was five percentage points higher than single birth calves (Table 1). These findings are in line with various reports in Holstein-Friesian calves (Silva del Rio et al., 2007; Brickell et al., 2009; Gulliksen et al., 2009a). Twin births have been associated with decreased gestation length and increased dystocia (primarily due to malpresentation) and decreased perinatal viability (Gregory et al., 1996; Echtenkamp and Gregory, 1999).

Mortality rate was highly dependent on the number of cows present on each dairy farm. Calf mortality rate increased ($P<0.05$) with increasing herd size, as has previously been reported in numerous other studies (Silva del Rio et al., 2007; Gulliksen et al., 2009a). As herd size increases and there is more technological developments used in dairy operations, the time spent with calves decreases. These factors could contribute to increased mortality rates. Gulliksen et al. (2009b) found that the time of the first colostrum feeding was highly correlated with herd size, therefore, a satisfactory colostrum-feeding regimen appears to be more difficult to implement in large dairy herds. Caution must be applied when interpreting the effect of herd size on perinatal calf mortality because this effect can be the effect of herd expansion rather than the effect of herd size itself.

Variables indicative of heat stress for calves born in winter or summer are presented in Table 2. Both heart and respiratory rate were higher ($P<0.01$) in calves born in winter compared to calves born in summer. Panting was not observed in calves experiencing intense heat load. Likewise, rectal temperature did not differ between calves born in January or June.

Responses observed in the present study in calves under cold temperature have been observed by other authors (Marques et al., 1981), and this response is an indication of cold acclimatization, which involves higher peak metabolic capability and a temporary elevation in resting metabolism. The higher respiration rate observed in calves born in winter is an attempt by these animals to maintain regular body temperature by pumping blood faster to keep normothermia. As pulse increases, the need for oxygen also increases, because lungs transfer oxygen to the blood and this explicate the higher respiratory rate. Both panting and increased rectal temperature were not apparent in calves during high heat loads, which imply that calves at an early age and in this particular environment seem to suffer little from heat stress. Calves during their first days of life generate much less metabolic heat than adult animals, have greater surface area relative to internal body mass and are expected to be much more tolerant to heat stress (West, 2003). Rectal temperature is strongly associated with many physiological attributes related with heat stress (McManus et al., 2009), but in this study this trait probably was not suitable to evaluate heat stress because body temperature changes take some time to react to ambient temperature changes (Brown-Brandl et al., 2005).

4. Conclusions

Under the weather conditions of the current study, in which chronic heat loads prevail for most of the year, these data do not support the concept that sustained high ambient temperatures increases neonatal mortality of Holstein calves. On the contrary, cold weather was detrimental for survival of calves. Also, there was no evidence that heat stress shortly before parturition has significant consequences

for calf survival. Finally, this study showed a marked seasonal effects on calf mortality, which indicate that efforts should be conveyed to minimize environmental stress of calves born in cold months.

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References

- Azzam, S.M., Kinder, J.E., Nielsen, M.K., Werth, L.A., Gregory, K.E., Cundiff, L.V., Koch, R.M. 1993. Environmental effects on neonatal mortality of beef calves. *J. Anim. Sci.* 71, 282–290.
- Beam, A.L., Lombard, J.E., Koprak, C.A., Garber, L.P., Winter, A.L., Hicks, J.A., Schlater, J.L. 2009. Prevalence of failure of passive transfer of immunity in newborn heifer calves and associated management practices on US dairy operations. *J. Dairy Sci.* 92, 3973–3980.
- Bell, A.W., Hay Jr, W.W., Ehrhardt, R.A. 1999. Placental transport of nutrients and its implications for fetal growth. *J. Reprod. Fertil. Suppl.* 54, 401-410.

- Brickell, J.S., McGowan, M.M., Pfeiffer, D.U., Wathes, D.C. 2009. Mortality in Holstein-Friesian calves and replacement heifers, in relation to body weight and IGF-I concentration, on 19 farms in England. *Animal* 3, 1175-1182.
- Brown-Brandl, T.M., Eigenberg, R.A., Nienaber, J.A., Hahn, G.L. 2005. Dynamic response indicators of heat stress in shaded and non-shaded feedlot cattle. *Anal. Indic. Biopr. Biosyst. Engin.* 90, 451–462.
- Borderas, F.T., de Passillé, A.M.B., Rushen, J. 2009. Temperature preferences and feed level of the newborn dairy calf. *Appl. Anim. Behav. Sci.* 120, 56-61.
- Brouce, J., Kisac, P., Uhrincat, M. 2009. Effect of hot temperatures on the hematological parameters, health and performance of calves. *Int. J. Biometeorol.* 53, 201–208.
- Collier, R.J., Beede, D.K., Thatcher, W.W., Israel, L.A., Wilcox, L.S. 1982. Influences of environment and its modification on dairy animal health and production. *J. Dairy Sci.* 65, 2213-2227.
- Collier, R.J., Collier, J.L., Rhoads, R.P., Baumgard, L.H. 2008. Genes involved in the bovine heat stress response. *J. Dairy Sci.* 91, 445–454.
- Curtis, C.R., Erb, H.N., White, M.E. 1988. Descriptive epidemiology of calfhood morbidity and mortality in New York Holstein herds. *Prev. Vet. Med.* 5, 293-307.
- Dhakal, K., Maltecca, C., Cassady, J.P., Baloché, G., Williams, C.M., Washburn, S.P. 2013. Calf birth weight, gestation length, calving ease, and neonatal calf mortality in Holstein, Jersey, and crossbred cows in a pasture system. *J. Dairy Sci.* 96, 690-698.

- Dikmen, S., Hansen, P.J. 2009. Is the temperature-humidity index the best indicator of heat stress in lactating dairy cows in a subtropical environment? *J. Dairy Sci.* 92, 109–116.
- Donovan, G.A., Dohoo, I.R., Montgomery, D.M., Bennett, F.L. 1998. Associations between passive immunity and morbidity and mortality in dairy heifers in Florida, USA. *Prev. Vet. Med.* 34, 31-46.
- Echternkamp, S.E., Gregory, K.E. 1999. Effects of twinning on gestation length, retained placenta, and dystocia. *J. Anim. Sci.* 77:39–47.
- Fowden, A.L., Ward, J.W., Wooding, F.B.P., Forhead, A.J. 2010. Developmental programming of the ovine placenta. *Soc. Reprod. Fert. Supl.* 7, 41-57.
- Gebremedhin, K.G., Cramer, C.O., Porter, W.P. 1981. Predictions and measurements of heat production and food and water requirements of Holstein calves in different environments. *Trans. Am. Soc. Agric. Engin.* 24, 715-720.
- Godden, S.M., Fetrow, J.P., Feirtag, J.M., Green, L.R., Wells, S.J. 2005. Economic analysis of feeding pasteurized nonsaleable milk versus conventional milk replacer to dairy calves. *J. Am. Vet. Med. Assoc.* 226, 1547–1554.
- Gregory, K.E., Echternkamp, S.E., Cundiff, L.V. 1996. Effect of twinning on dystocia, calf survival, calf growth, carcass traits, and cow productivity. *J. Anim. Sci.* 74, 1223–1233.
- Gulliksen, S.M., Lie, K.I., Løken, T., Østerås, O. 2009a. Calf mortality in Norwegian dairy herds. *J. Dairy Sci.* 92, 2782–2795.
- Gulliksen, S.M., Jor, E., Lie K.I., Loken, T., Akerstedt, J., Osteras, O. 2009b. Respiratory infections in Norwegian dairy calves. *J. Dairy Sci.* 92, 5139-5146.

- Gulliksen, S.M., Jor, E., Lie, K.I., Hamnes, I.S., Loken, T., Akerstedt, J., Osteras, O. 2009c. Enteropathogens and risk factors for diarrhea in Norwegian dairy calves. *J. Dairy Sci.* 92, 5057-5066.
- Hahn, G.L. 1999. Dynamic responses of cattle to heat heat loads. *J. Anim. Sci.* 77, 10–20.
- Heinrichs, A.J., Heinrichs, B.S., Hare, O., Rodgers, W., Place, N.T. 2005. A prospective study of factors affecting age, body size and body condition score at first calving of Holstein dairy heifers. *J. Dairy Sci.* 88, 2828-2835.
- Hill, T.M., Bateman, I.I., H.G., Aldrich, J.M., Schlotterbeck, R.L. 2011. Comparisons of housing, bedding, and cooling options for dairy calves. *J. Dairy Sci.* 94, 2138-2146.
- Johanson, J.M., Berger, P.J. 2003. Birth weight as a predictor of calving ease and perinatal mortality in Holstein cattle. *J. Dairy Sci.* 86, 3745–3755.
- Kadzere, C.T., Murphy, M.R., Silanikove, N., Maltz, E. 2002. Heat stress in lactating dairy cows. A review. *Livest. Prod. Sci.* 77, 59–91.
- Kelley, K.W., Osborne, C.A., Evermann, J.F., Parish, S.M., Gaskins, C.T. 1988. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. *J. Dairy Sci.* 65, 1514-1528.
- Lacetera, N., Bernabucci, U., Ronchi, B., Scalia, D., Nardone A. 2002. Moderate summer heat stress does not modify immunological parameters of Holstein dairy cows. *Int. J. Biomet.* 46, 33-37.
- Lago, A., McGuirk, S.M., Bennett, T.B., Cook, N.B., Nordlund, K.V. 2006. Calf respiratory disease and pen microenvironments in naturally ventilated calf barns in winter. *J. Dairy Sci.* 89, 4014–4025.

- Lombard, J.E., Garry, F.B., Tomlinson, S.M., Garber, L.P. 2007. Impacts of dystocia on health and survival of dairy calves. *J. Dairy Sci.* 90, 1751–1760.
- Magalhaes, V.J.A., Susca, F., Lima, F.S., Branco, A.F., Yoon, I., Santos, J.E.P. 2008. Effect of feeding yeast culture on performance, health, and immunocompetence of dairy calves. *J. Dairy Sci.* 91, 1497-1509.
- Marques, P.R., Illner, P., Williams, D.D., Gren, W.L., Kendall, J.W., Davis, S.L., Johnson, D.G., Gale, C.C. 1981. Hypothalamic control of endocrine thermogenesis. *Am. J. Physiol. Endocrinol. Metab.* 241, E420-E427.
- McDermott, J.J., Allen, O.B., Martin, S.W., Alves, D.M. 1992. Patterns of stillbirth and dystocia in Ontario cow-calf herds. *Can. J. Vet. Res.* 56, 47–55.
- McManus, E., Prescott, C., Paludo, G.R., Bianchini, E., Louvandini, H., Mariani, A.S. 2009. Heat tolerance in naturalized Brazilian cattle breeds. *Livest. Sci.* 120, 256–264.
- Mellado, M., Antonio-Chirino, E., Meza-Herrera, C., Veliz, F.G., Arevalo, J.R., Mellado, J., de Santiago, A. 2011. Effect of lactation number, year, and season of initiation of lactation on milk yield of cows hormonally induced into lactation and treated with recombinant bovine somatotropin. *J. Dairy Sci.* 94, 4524–4530.
- Mellado, M., Sepulveda, E., Meza-Herrera, C.A., Veliz, F.G., Arevalo, J.R., Mellado, J., de Santiago, A. 2013. Effect of heat stress on reproduction efficiency in high-milk yielding Holstein cows in a hot arid environment. *Rev. Colomb. Cienc. Pec.* 26, 193-200.

- Morin, D.E., Constable, P.D., Maunsell, F.P., McCoy, G.C. 2001. Factors Associated with Colostral Specific Gravity in Dairy Cows. *J. Dairy Sci.* 84, 937–943.
- Nardone, A., Lacetera, N., Bernabucci, U., Ronchi, B. 1997. Composition of colostrum from dairy heifers exposed to high air temperatures during late pregnancy and the early postpartum period. *J. Dairy Sci.* 80, 838-844.
- NRC (National Research Council). 2001. *Nutrient Requirements of Dairy Cattle* 7th Rev. Ed. Nat. Acad. Press, Washington, DC.
- Neuwirth, J.G., Norton, J.K., Rawlings, C.A., Thompson, F.N. 1979. Physiologic responses of dairy calves to environmental heat stress. *Int. J. Biomet.* 23, 243-246.
- Nix, J.M., Spitzer, J.C., Grimes, L.W., Burns, G.L., Plyler, B.B. 1998. A retrospective analysis of factors contributing to calf mortality and dystocia in beef cattle. *Theriogenology* 49, 1515-1523.
- Nonnecke, B.J., Foote, M.R., Miller, B.L., Fowler, M., Johnson, T.E., Horst, R.L. 2009. Effects of chronic environmental cold on growth, health, and select metabolic and immunologic responses of preruminant calves. *J. Dairy Sci.* 92, 6134–6143.
- Norheim, K., Simensen, E. 1985. An epidemiological study of factors affecting serum IgG levels in dairy calves. *Nord. Vet.* 37, 121–135.
- Razzaque, M.A., Bedair, M., Abbas, S., Al-Mutawa, T. 2009. Economic impact of calf mortality on dairy farms in Kuwait. *Pakistan Vet. J.* 29, 97-101.

- Regnault, T.R., Galan, H.L., Parker, T.A., Anthony, R.V. 2002. Placental development in normal and compromised pregnancies – a review. *Placenta*, 23Suppl A, S119–S129.
- Riley, D.G., Chase, Jr C.C., Olson, T.A., Coleman, S.W., Hammond, A.C. 2004. Genetic and nongenetic influences on vigor at birth and preweaning mortality of purebred and high percentage Brahman calves. *J. Anim. Sci.* 82, 1581–1588.
- Ross, J.C., Fennessey, P.V., Wilkening, R.P., Battaglia, F.C., Meschia, G. 1996. Placental transport and fetal utilization of leucine in a model of fetal growth retardation. *Am. J. Physiol.* 270, E491–E503.
- Shearer, J., Mohammed, H.O., Brenneman, J.S., Tran, T.Q. 1992. Factors associated with concentrations of immunoglobulins in colostrum at the first milking post-calving. *Prev. Vet. Med.* 14,143-154.
- Silanikove, N. 2000. Effects of heat stress on the welfare of extensively managed domestic ruminants. *Livest. Prod. Sci.* 67, 1–18.
- Silva del Rio, N., Stewart, S., Rapnicki, P., Chang, Y.M., Fricke, P.M. 2007. An observational analysis of twin births, calf sex ratio, and calf mortality in Holstein dairy cattle. *J. Dairy Sci.* 90, 1255–1264.
- Spain, J.N., Spiers, D.E. 1996. Effects of supplemental shade on thermoregulatory response of calves to heat challenge in a hutch environment. *J. Dairy Sci.* 79, 639-646.
- Stull, C.L., McV Messam, L.L., Collar, C.A., Peterson, N.G., Castillo, A.R., Reed, B.A., Andersen, K.L., VerBoort, W.R. 2008. Precipitation and temperature

- effects on mortality and lactation parameters of dairy cattle in California. J. Dairy Sci. 91, 4579–4591.
- Svensson, C., Linder, A., Olsson, S.O. 2006. Mortality in Swedish dairy calves and replacement heifers. J. Dairy Sci. 89, 4769–4777.
- Tao, S., Monteiro, A.P.A., Thompson, I.M., Hayen, M.J., Dahl, G.E. 2012. Effect of late-gestation maternal heat stress on growth and immune function of dairy calves. J. Dairy Sci. 95, 7128-7136.
- Thureen, P.J., Trembler, K.A., Meschia, G., Makowski, E.L., Wilkening, R.B. 1992. Placental glucose transport in heat-induced fetal growth retardation. Am. J. Physiol. 263, R578–R585.
- Torsein, M., Lindberg, A., Sandgren, C.H., Waller, K.P., Törnquist, M., Svensson, C. 2011. Risk factors for calf mortality in large Swedish dairy herds. Prev. Vet. Med. 99, 136-147.
- Trotz-Williams, L.A., Wayne Martin, S., Leslie, K.E., Duffield, T., Nydam, D.V., Peregrine, A.S. 2007. Calf-level risk factors for neonatal diarrhea and shedding of *Cryptosporidium parvum* in Ontario dairy calves. Prev. Vet. Med. 82, 12–28.
- Tyler, J.W., Hancock, D.D., Thorne, J.G., Gay, C.C., Gay, J.M. 1999. Partitioning the mortality risk associated with inadequate passive transfer of colostral immunoglobulins in dairy calves. J. Vet. Int. Med. 13, 335–337.
- Wells, S.J., Garber, L.P., Hill, G.W. 1996. Health status of preweaned dairy heifers in the United States. Prev. Vet. Med. 29, 185-199.
- West, J.W. 2003. Effects of heat-stress on production in dairy cattle. J. Dairy Sci. 86, 2131–2144.

Wolfenson, D., Flamenbaum, I., Berman, A. 1988. Dry period heat stress relief effects on prepartum progesterone, calf birth weight, and milk production. J. Dairy Sci. 71, 809-818.

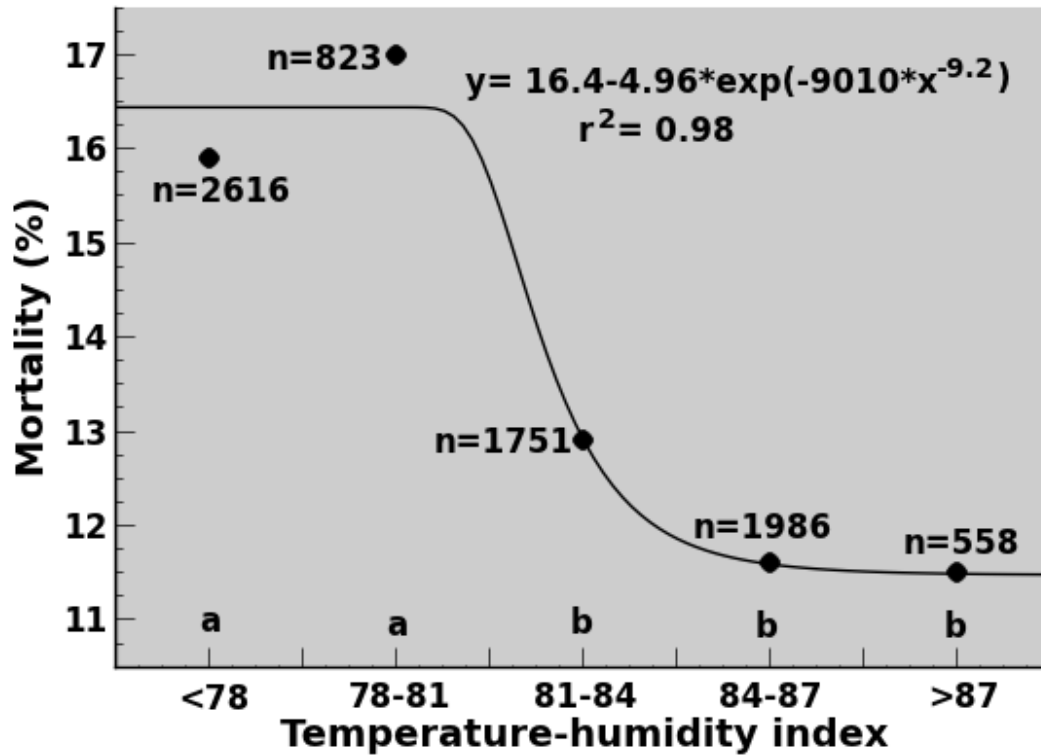


Fig. 1. Association between temperature-humidity index (THI) the day of birth and calf mortality rate from birth to 21 d of age in intensive large dairy operations in a hot environment (26°N). THI with dissimilar letters differ ($P < 0.05$).

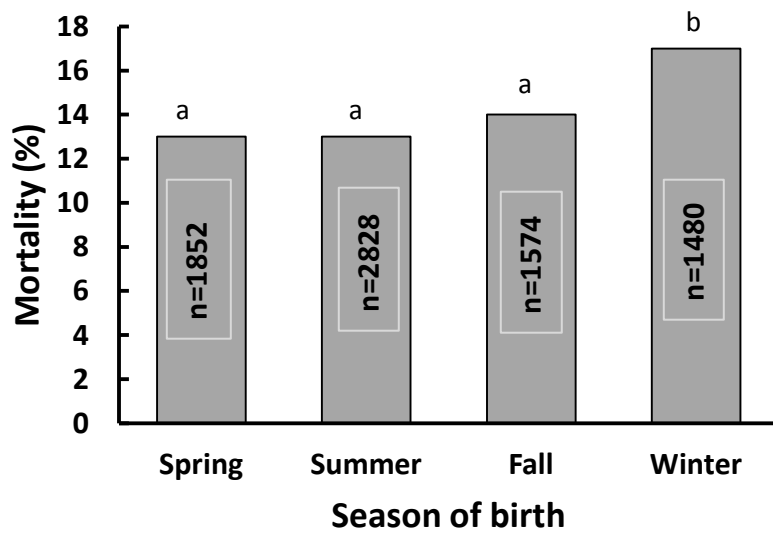


Fig 2. The effect of season of birth on mortality rate during the first 21 d of life for Holstein calves raised in large scale intensive dairy herds in a hot environment (26°N).

Table 1

Factors associated with calf mortality in large scale intensive dairy herds in a hot environment (26°N).

Variable	% mortality	95% confidence limits	P-value
Gender			0.00001
Males	17 (618/3740)	16 - 18	
Females	12 (472/3994)	11 - 14	
Birth type			0.00001
Single	14 (998/7241)	13 - 15	
Twin	19 (92/493)	18 - 21	
Operation size			0.0001
>1800 cows	16 (490/3154)	15 - 18	
<1800 cows	13 (601/4580)	12 - 14	
Type of feeding			0.0001
Milk	16 (413/2657)	15 - 18	

Milk replacer	13 (677/5067)	12 - 14
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Table 2

Environmental conditions and variables indicative of thermal stress in newborn (one to five-day-old) Holstein calves in a hot-arid environment (26°N). Values are means ± SD.

Variable	January	June
Number of calves	100	102
Mean maximum ambient temperature (°C)	28.1 ± 3.3	38.0 ± 3.4
Mean relative humidity (%)	37.5 ± 7.3	23.7 ± 8.8
Mean temperature-humidity Index ¹	73.9 ± 3.2	82.3 ± 3.3
Respiratory rate	74.4 ± 15.6 ^a	67.8 ± 16.3 ^b
Heart rate (beats per minute)	119.2 ± 19.6 ^a	113.9 ± 17.0 ^b
Rectal temperature (°C)	38.8 ± 0.5	38.8 ± 0.5
Panting score ²	0.0 ± 0.0	0.0 ± 0.0

¹Temperature-humidity index (THI) = $0.8 \times \text{ambient temperature} + [(\% \text{ relative humidity} \div 100) \times (\text{ambient temperature} - 14.4)] + 46.4$.

²Panting score: 0=normal respiration, 4=Severe open-mouthed panting with protruding tongue and excessive salivation.

^{a,b}Within rows means with different superscript letters differ ($P < 0.01$).