UNIVERSIDAD AUTÓNOMA AGRARIA ANTONIO NARRO

SUBDIRECCIÓN DE POSTGRADO

EFECTOS DEL ESTRÉS POR CALOR EN ÚTERO EN VACAS HOLSTEIN SOBRE SU SUBSECUENTE DESEMPEÑO PRODUCTIVO Y REPRODUCTIVO

Tesis

Que presenta MARÍA INÉS CHÁVEZ VÁZQUEZ
como requisito parcial para obtener el Grado de
DOCTOR EN CIENCIAS EN PRODUCCIÓN AGROPECUARIA

Torreón, Coahuila Mayo 2021

EFECTOS DEL ESTRÉS POR CALOR EN ÚTERO EN VACAS HOLSTEIN SOBRE SU SUBSECUENTE DESEMPEÑO PRODUCTIVO Y REPRODUCTIVO

Tesis

Elaborada por MARÍA INÉS CHÁVEZ VÁZQUEZ como requisito parcial para obtener el grado de Doctor en Ciencias en Producción Agropecuaria con la supervisión y aprobación del Comité de Asesoría

Dr. Miguel Ángel Mellado Bosque
Asesor principal

Dr. José Eduardo García Martínez
Asesor

Dr. Francisco Gerardo Véliz Deras
Asesor

Dra. Leticia Romana Gaytán Alemán
Asesor

Dra. Ma. De los Angeles de Santiago M.
Asesor

Dra. Leticia R. Gaytán Alemán
Jefe del Departamento de Postgrado

Dr. Marcelino Cabrera De La Fuente
Subdirector de Postgrado

Torreón, Coahuila

Mayo 2021

Agradecimientos

Dr. Miguel Ángel Mellado Bosque

Gracias por su paciencia, apoyo, comprensión y por los consejos brindados durante estos años de formación, un honor ser su discípulo y poder contar siempre con su acompañamiento. Mi admiración y respeto para Usted, siempre.

Dr. Francisco Gerardo Véliz Deras

Agradecida enormemente con Usted por darme la oportunidad de ser parte del programa, por siempre impulsarme a ser mejor y por todas sus enseñanzas y exigencias.

Dr. Eduardo García Martínez

Dra. María de los Ángeles de Santiago Miramontes

Dra. Leticia R. Gaytán Alemán

Gracias por todo el apoyo y acompañamiento en esta etapa de mi vida, gracias por sus enseñanzas y consejos.

CONACyT

Por haberme proporcionado la beca que me hizo poder cubrir mis necesidades, tanto económicas como de salud para mi familia y para mí.

Mi familia

Es para mí un orgullo contar con Ustedes en toda esta travesía, pues sin el apoyo no hubiera sido posible alcanzar mi sueño, gracias por la paciencia y por siempre estar aquí conmigo.

Dios

Agradezco a Dios por no soltarme nunca de su mano cuando he querido desfallecer, por darme la salud y la fortaleza necesarias para terminar con bien todo lo que me he propuesto. Me siento bendecida por tan noble gesto hacia mi persona.

Dedicatoria

A Dios por todas las bendiciones que ha otorgado a mi vida durante todos estos años.

A mis padres Felipa Vázquez Lara (+) y Miguel Chávez Luna (+) que sin ellos no sé dónde estuviera yo en estos momentos.

A mi esposo Edgar Saavedra Porras porque ha estado siempre a mi lado brindándome apoyo incondicional, siendo paciente, comprensivo y motivándome a ser mejor cada día.

A mis hijas Diana Cristina y Jessica Jazmín por ser mi motor de arranque y darme fuerzas para seguirme preparando.

A mis amigos Julieta Ordoñez, Lupita Machado, Homero González, Alan Alvarado, Aurelio Morales, que han estado en las buenas y en las malas conmigo, ayudándome durante todo este proceso, pues se lograron crear lazos fuertes de amor fraterno entre nosotros.

A la doctora Dalia Carrillo por ayudarme siempre que la he necesitado y resolver mis dudas cuando no sé algo.

ÍNDICE DE CONTENIDO

RESUMEN

EFECTOS DEL ESTRÉS POR CALOR EN ÚTERO EN VACAS HOLSTEIN SOBRE SU SUBSECUENTE DESEMPEÑO PRODUCTIVO Y REPRODUCTIVO

MARÍA INÉS CHÁVEZ VÁZQUEZ
Doctor en Ciencias en Producción Agropecuaria
UNIVERSIDAD AUTÓNOMA AGRARIA ANTONIO NARRO
Dr. Miguel Ángel Mellado Bosque
Director de tesis

El objetivo fue determinar cómo afecta el estrés térmico en el útero en el subsecuente desempeño reproductivo y producción de leche en vaquillas Holstein El estudio incluyó 4976 vaquillas Holstein. Las vaquillas nacidas de vacas que no experimentaron estrés por calor tres meses antes del parto, pero con un índice de temperatura humedad; THI> 83 al momento del parto, fueron mayores ($p < 0.05$) al primer parto (743 ± 67 vs.729 ± 55 días) que las novillas gestadas bajo condiciones maternas de estrés por calor. Un aumento de dos veces ($p < 0.01$) en la tasa de preñez ocurrió en vaquillas gestadas en condiciones maternas sin estrés por calor durante dos o tres meses antes del parto y ausencia de estrés por calor en el parto, en comparación con novillas gestadas en condiciones maternas sin estrés por calor. La mediana de días para quedar gestantes fue mayor (140 días) para las novillas cuyas madres estuvieron expuestas a THI> 83 al momento del parto que para las novillas cuyas madres estuvieron expuestas a THI<76 o 76-83 (117 y 114 d) al momento del parto. Estos datos mostraron que el estrés por calor en el útero durante los últimos tres meses de gestación afecta negativamente al rendimiento reproductivo de novillas Holstein. En otro estudio (n= 5278 vaquillas) no se presentaron diferencia entre las novillas con y sin estrés térmico en el útero. Las vaquillas sin estrés térmico produjeron 170 kg más leche ($P < 0.05$) a 305 d en comparación con las expuestas a hipertermia en el útero. Las vaquillas sin estrés térmico durante la gestación produjeron 0.7 kg más leche/día. La duración de la producción de leche fue mayor ($P < 0.05$) en vaquillas nacidas de madres sin estrés térmico (73.5 ± 2.4%) comparadas con las expuestas a hipertermia (73.5 ± 2.4%). Se concluyó que la producción de leche fue alterada y la persistencia de la lactancia entre las novillas con estrés en el útero en comparación con las novillas sin estrés. Sin embargo, estos efectos fueron menos pronunciados en las siguientes lactaciones; así, los efectos del estrés térmico en el útero sobre la producción de leche no fueron más allá del impacto inmediato en la primera lactancia.

Palabras clave: Tasa de concepción; Servicios por concepción; Edad a primer parto; Producción acumulada; Estrés por calor; Duración de la lactancia.

ABSTRACT

EFFECTS OF HEAT STRESS IN UTERO OF FIRST CALVING HOLSTEIN COWS ON MILK YIELD AND REPRODUCTIVE PERFORMANCE.

MARÍA INÉS CHÁVEZ VÁZQUEZ
Doctor of Science in Agricultural Production

AUTONOMOUS AGRARIAN UNIVERSITY ANTONIO NARRO

Dr. Miguel Ángel Mellado Bosque
Thesis's director

The objective was to determine how heat stress affects reproductive performance, milk production and its duration in Holstein heifers gestated under a hot environment. The study included 4,976 heifers. Cows at first calving without heat stress (ITH > 83) calved later (p <0.05) (743 ± 67 vs. 729 ± 55 d) than those with heat stress. Heifers gestated in conditions without heat stress, the pregnancy rate increased twice more compared to those exposed, the pregnancy rate (all services) was higher for heifers with heat stress (p <0.05) (66.7 vs 51, 1%). The median for the heifers to become pregnant, the mothers were exposed to stress at the time of calving ITH> 83 was greater than 140 d, with ITH <76 or 76 to 83 it was 114 and 117 d. The second study included 5,278 cows, with an average lactation of 451 d (SD = 81), it did not have differentiation from heifers with and without heat stress. Heifers without heat stress produced 170 kg more milk (P <0.05) at 305 d compared to those exposed. Heifers without heat stress during gestation produced 0.7 kg more milk / day. The duration of milk production was greater (P <0.05) in heifers born to dams without heat stress (73.5 ± 2.4%) compared to those exposed (73.5 ± 2.4%). It was concluded that milk production and lactation length in first calving heifers that experienced heat stress in utero is affected by hyperthermia in utero.

Keywords: Conception rate; Services per pregnancy; Age at first calving; Cumulative milk yield / Extended lactations / In utero heat stress / Lactation length / Lactation persistency

I. INTRODUCCIÓN

El clima es el factor ecológico más importante que determina el crecimiento, desarrollo y productividad de los animales domésticos (Herbut *et al*., 2919). Los cambios climáticos impactan la viabilidad económica de los sistemas de producción ganadera en todo el mundo, a través de una variedad de rutas (Baumgard *et al.,* 2012). Estos incluyen cambios en la disponibilidad y calidad de los alimentos, cambios en las poblaciones de plagas y patógenos, alteración de la inmunidad e impactos directos e indirectos en el rendimiento animal, como el crecimiento, la reproducción y la lactancia. La falta de acondicionamiento previo (aclimatación) al cambio repentino del clima a menudo resulta en pérdidas catastróficas en la industria ganadera nacional (Collier *et al.,* 2019). El aumento global en la temperatura también influirá en los patógenos y las enfermedades (Campos *et al.,* 2017).

Se espera que la población humana aumente de 7.200 a 9.600 millones para 2050 (FAO, 2013). Esto representa un aumento de la población del 33%, pero a medida que aumenta el nivel de vida mundial, la demanda de productos agrícolas aumentará aproximadamente 70% en el mismo período. Los productos pecuarios son un producto agrícola importante para la seguridad alimentaria mundial porque proporcionan el 17% de consumo de kilocalorías y 33% del consumo mundial de proteínas (Rosegrant *et al.,* 2009). El sector ganadero contribuye a el sustento de mil millones de la población más pobre del mundo y emplea a cerca de 1.1 mil millones de personas. Existe una demanda creciente de productos pecuarios y se considera la "revolución ganadera" debido a su rápido crecimiento en los países en desarrollo (Thornton, 2010; Wright *et al.,* 2012). Se espera que la producción mundial de leche aumente de 664 millones de toneladas (cifras de 2006) a 1077 millones de toneladas (para 2050), y la producción de carne se duplicará de 258 a 455 millones toneladas.

A nivel mundial el cambio climático afecta económicamente los sistemas de producción sin importar las fronteras geográficas ya que no solo afecta a las regiones tropicales o subtropicales sino también las regiones templadas (Collier *et al.,* 2017; Rahman *et al.,* 2018). El medio ambiente confortable para el ganado lechero fluctúa entre 5 y 25 º C, la cual es considerada como zona termo neutral (ZTN) ya que el animal tiene un gasto mínimo fisiológico y una máxima productividad para mantener su temperatura corporal (Kadzere *et al.,* 2002; Aggarwal y Upadhyay, 2013).

El estrés por calor afecta negativamente la rentabilidad de las granjas lecheras en los Estados Unidos (Sejian *et al.,* 2012). De acuerdo a St-Pierre *et al.* (2003), el clima cálido reduce la producción de leche, y las pérdidas económicas totales estimadas durante el verano para la industria láctea de EE. UU. superan los $ 897 millones de dólares anuales, dicha disminución de leche es un fenómeno común que registra una reducción entre 30 y 40% (Nienaber y Hahn, 2007; Pragna *et al.,* 2016). En Florida y Texas, las pérdidas económicas por vaca lactante se han estimado en $ 337 y $ 383 USD / vaca por año, respectivamente.

Los animales que son expuestos a temperaturas elevadas reducen el apetito, disminuyen la tasa de crecimiento, la producción de leche y carne, reducen su rendimiento reproductivo y son más susceptibles a enfermarse (Dahl, 2018; Rahman *et al.,* 2018). El aumentó en la temperatura corporal central reducirá la producción de leche, los porcentajes de proteína de la leche, las grasas, los sólidos y la lactosa (Pragna *et al.,* 2016). Por unidad de aumento del Índice de temperatura y humedad (THI) superior a 72, se registró en vacas lecheras una reducción de 0.2 kg en el rendimiento de la leche (Pragna *et al.,* 2016).

El estrés lo podemos definir como un evento o condición externa propia del medio ambiente que provoca un efecto sobre un sistema biológico. Existen diversos factores que provocan estrés tales como la temperatura ambiental, el manejo, el ruido, los contaminantes ambientales, las enfermedades, etc. (Collier *et al.,* 2017).

II. HIPÓTESIS Y OBJETIVO GENERAL

Hipótesis

Las vacas Holstein que sufren estrés por calor en su vida uterina en el último tercio de la gestación, podrían presentar una disminución en la producción de leche, un aumento en la ocurrencia de enfermedades y una menor eficiencia reproductiva, en comparación con vacas que no experimentaron estrés térmico en el útero.

Objetivo general

El objetivo general de esta investigación fue determinar los efectos que causa el estrés por calor en el útero y como éste afecta en el desempeño reproductivo, y en la producción de leche en vaquillas Holstein gestadas bajo condiciones de estrés térmico en un ambiente caluroso.

III. REVISIÓN DE LITERATURA

Es probable que la producción ganadera se vea afectada negativamente por el cambio climático, la competencia por la tierra, el agua y la seguridad alimentaria en el momento en que más se necesita (Thornton, 2010). El cambio climático global es causado principalmente por emisiones de gases de efecto invernadero (GEI) que resultan en el calentamiento de la atmósfera. El sector ganadero contribuye con el 14.5% de las emisiones globales de GEI (Gerber *et al.,* 2013) y, por lo tanto, puede aumentar el calentamiento de la tierra, la contaminación del aire y del agua y disminución de la biodiversidad (Reynolds *et al.,* 2010; Thornton y Gerber, 2010). Al mismo tiempo, el cambio climático afectará la producción ganadera a través de competencia por los recursos naturales, cantidad y calidad de los alimentos, enfermedades del ganado, estrés por calor y pérdida de biodiversidad (Grossi *et al*., 2019).

3.1 Estrés térmico

El impacto térmico del medio ambiente sobre los animales es consecuencia de la combinación e interacción del animal con las variables climáticas (temperatura del aire, humedad, velocidad del aire, temperatura de las superficies de contacto, radiación solar, disponibilidad de agua y conductividad térmica) (Church, 1988). Un aumento del estrés por calor (THI >72) trae como consecuencia un aumento en la tasa respiratoria y en la temperatura corporal de las vacas y aquí es cuando la vaca lechera empieza a bajar su productividad (Baumgard *et al.,* 2006; Ahmed *et al.,* 2017), además de sufrir alteraciones en los marcadores de estrés oxidativo, así como cambios a nivel sanguíneo de glucosa, sodio, potasio y cloro (Jeelani *et al.,* 2019).

El estrés térmico se activa cuando las condiciones ambientales superan el nivel crítico superior o inferior en la temperatura de los animales de la granja que requieren un aumento en el metabolismo para hacer frente al estrés, generando una respuesta a un factor que involucra cambios de comportamiento, metabólicos y fisiológicos en diferentes niveles (Bernabucci *et al.,* 2010; Collier *et al.,* 2019). Los animales sometidos a estrés térmico presentan temperaturas rectales elevadas, cambios en la frecuencia cardiaca y respiratoria, además de modificaciones en el volumen de hematocrito y número de glóbulos rojos (Cardoso *et al.*, 2015; Fabris *et al.,* 2017).

Todos los procesos de la vida al igual que los eventos fisiológicos gastan energía, pues ésta es necesaria para que un animal viva y se mantenga, por lo cual el medio ambiente repercute en los animales mediante el intercambio de ella, ya que si el animal recibe más energía de la que libera aumentará su temperatura corporal y posiblemente muera. Por el contrario, si pierde más energía de la que recibe se enfriará y no sobrevivirá, por lo que es necesario un ambiente compatible con sus requerimientos fisiológicos de energía, debido a que el ganado regula su temperatura corporal a partir de 38 ºC (Carroll *et al.,* 2012; Collier y Gebremedhin, 2015).

El ganado lechero tiene una tasa metabólica alta lo que incrementa los mecanismos de termorregulación para poder mantener su temperatura corporal y está influenciado por la edad, la especie, el consumo de alimento, la composición de la dieta, la producción de leche, el estado fisiológico, el alojamiento, la condición del corral, aislamiento del tejido (piel y grasa), pelo y el comportamiento animal (Aggarwal y Upadhyay, 2013).

Los mamíferos y las aves poseen una estrategia térmica para mantener una temperatura corporal por encima de la temperatura ambiente que les permita disipar el calor a través de cuatro mecanismos: la conducción, la convección, la evaporación y la radiación, como rutas sensibles de perdida de calor dado que si éstas se pierden la ruta final/única restante de pérdida de calor sería la evaporación (sudoración y jadeo) que requieren de un gradiente de presión de vapor por lo cual la humedad relativa es un factor importante que controla la tasa de pérdida de calor por evaporación (Habeeb *et al.,* 2018; Collier *et al.*, 2019).

La activación de la respuesta aguda al estrés es iniciada por receptores térmicos ubicados en la piel, por los cuales las vías aferentes trasmiten información hacia el sistema nervioso central incluyendo el tálamo y el hipotálamo con la finalidad de poder responder a los cambios del medio ambiente (Collier y Gebremedhin, 2015).

Por lo tanto, en lo que se refiere al ganado bovino lechero en época de verano, es importante el proporcionar sombras, ventiladores o rociadores con la finalidad de disminuir el estrés por calor, además de hacer modificaciones en la dieta para que éstas puedan desafiar las condiciones climáticas (Mader, 2003; Bernabucci *et al.,* 2014).

3.2. El período seco y el estrés por calor

El período seco es un estado no lactante que se inicia de seis a ocho semanas antes del parto y se caracteriza por fases de involución de la glándula mamaria a través de muerte celular programada (apoptosis) y redesarrollo a través de la división y crecimiento celular (proliferación celular) (Zhao et al., 2019). El propósito de este período es promover el recambio de células epiteliales mamarias (MEC), donde las células epiteliales senescentes se reemplazan con células completamente funcionales en preparación para la siguiente lactancia. En el tejido mamario, el estrés por calor altera la síntesis de proteínas MEC, altera la organización de los filamentos de queratina y actina y regula a la baja los genes involucrados en el crecimiento celular y la ramificación ductal mientras promueve la expresión de genes involucrados en respuestas apoptóticas, fagocíticas y de supervivencia celular (Senn *et al.,* 2019).

Dreiling *et al.* (1991), reportó que en ovejas gestantes disminuyó 20-30 % el flujo sanguíneo uterino por cada 1 ^{0}C que se elevaba la temperatura ambiental, lo que provoca cambios en el metabolismo fetal, lo cual conduce a un retraso en el crecimiento y desarrollo, además de una reducción del 60 y 100 % de las hormonas oxitocina y antidiurética, respectivamente.

Durante los últimos dos meses de gestación, el feto crece al ritmo más rápido y se acumula aproximadamente 60% de su peso al nacer. Los terneros nacidos de madres que sufrieron estrés por calor cuando se secaron son más livianos desde el nacimiento hasta el año de edad y tienen una función inmunológica comprometida. Además, estas terneras (es decir, novillas) producen menos leche durante su primera lactancia en relación con vaquillas nacidas de madres enfriadas durante el periodo seco (Skibiel *et al.,* 2018).

Las vacas que se encuentran en el período seco, producen menos calor metabólico en comparación con las vacas lecheras lactantes y, por lo tanto, teóricamente son menos susceptibles al estrés por calor (Collier *et al.,* 2017). El estrés por calor durante el período seco reduce la producción de leche en la siguiente lactancia (Tao *et al.,* 2011; Tao *et al.,* 2012; Fabris *et al.,* 2017).

La exposición de las vacas al enfriamiento durante el período seco aumenta la producción de leche en relación con los animales expuestos al estrés por calor (do Amaral *et al.,* 2009). Aunque las vacas son vulnerables al estrés por calor durante la lactancia, el estrés por calor ambiental durante el período seco también afecta adversamente la fisiología de la vaca y la producción de leche en la lactancia subsiguiente (Skibiel *et al.,* 2018). Similar a las vacas lactantes, el estrés por calor durante el período seco disminuye el consumo de materia seca (Adin *et al.,* 2009; do Amaral *et al.,* 2009; Yue *et al.,* 2020). Como resultado de la energía reducida por el bajo consumo de alimento, las vacas secas estresadas por calor tienen menor ganancia de peso corporal y gestación tardía en comparación con las que no sufren estrés por calor (Fabris *et al*., 2017).

El estrés por calor materno durante el período seco afecta a la descendencia. En comparación con los de vacas con enfriamiento, los terneros nacidos de vacas secas con estrés por calor, tienen una función inmunológica pasiva y mediada por células deteriorada (Tao *et al.,* 2012; Monteiro *et al.,* 2014) y metabolismo alterado durante el período previo al destete (Tao *et al.,* 2014; Monteiro *et al.,* 2016). Además, Monteiro *et al*. (2013), informaron que el estrés por calor materno durante el período seco redujo la producción de leche de las vaquillas durante la primera lactancia.

El enfriamiento durante el período seco puede aumentar la producción de leche como lo hace durante la lactancia. Los resultados indican un posible beneficio de enfriar las vacas secas incluso bajo estrés por calor leve (Wolfenson *et al.,* 1988). El enfriamiento también mejora el crecimiento mamario durante el período seco y mejora el rendimiento de la lactancia después del parto (Tao *et al.,* 2013).

El desarrollo de la glándula mamaria durante el período seco se ve afectado por el estrés por calor principalmente en lo que se refiere a la involución de ésta y su desarrollo, esto tiene importancia ya que al iniciar la lactancia la capacidad de la glándula mamaria para sintetizar y almacenar leche dependerá del número de células mamarias y su actividad secretora, debido a que estás vacas tienen menos alvéolos comparadas con vacas sujetas a sistemas de enfriamiento, lo que podría conducir a que la glándula mamaria sea menos productiva, y por ende, tenga menor producción de leche (Tao *et al.,* 2011; Laporta *et al.,* 2020).

El enfriamiento antes del parto mejora el estado inmunológico de las vacas en transición y la evidencia sugiere que la señalización alterada de prolactina en las células inmunes media los efectos del estrés por calor en la función inmunológica (Bagath et al., 2019). El estrés por calor de la gestación tardía compromete el desarrollo de la placenta, lo que resulta en hipoxia fetal, desnutrición y, finalmente, retraso del crecimiento fetal (Tao y Dahl, 2013) además, se ven alterados los metabolitos sanguíneos, así como la captación de insulina (Monteiro *et al*., 2016).

Por otro lado, López *et al*. (2017), reportó que los terneros que son expuestos a estrés por calor reducen el consumo de alimento concentrado en la etapa de lactancia lo que conlleva a bajo peso al destete y esto tiene repercusiones en el crecimiento posterior. Además, dicho estudio proporciona información sobre la importancia del enfriamiento de las vacas secas, con la finalidad de prevenir el nacimiento de terneros livianos por causa del estrés por calor (López *et al*., 2017).

3.3. Síntesis y secreción de hormonas

Los aminoácidos son la base para la síntesis de hormonas de bajo peso molecular, por ejemplo, tirosina o fenilalanina, siendo precursor de la síntesis de hormonas como epinefrina, norepinefrina, dopamina y tiroides (Guoyao, 2009). Por otro lado, en el metabolismo de la glucosa y ácidos grasos, la insulina tiene un papel importante ya que su secreción y respuesta en los fetos de corderos y ovejas se ven alterados durante la gestación (Tao *et al.,* 2014).

Las hormonas tiroideas triyodotironina (T3) y tiroxina (T4) juegan un papel importante en la regulación de la termogénesis ya que son un indicador para evaluar la termo-tolerancia en los animales de la granja, al encontrarse reducidas en animales que experimentan estrés por calor, efecto que puede atribuirse a la disminución a nivel eje hipotálamo-pituitario-tiroideo al disminuir la producción de la hormona liberadora de tirotropina (TRH), que limita el metabolismo basal (Sejian *et al.,* 2018).

La disminución del sustrato y hormonas y el aumento de la temperatura corporal inhibe la actividad enzimática, que disminuyen el metabolismo y consecuentemente perjudican la producción de leche, el crecimiento corporal y la reproducción. Además, la escasez de energía, de los sustratos y de la hormona triyodotironina (T3) pueden ser responsables de la depresión en la producción y composición de la leche (Habeeb *et al.,* 2018).

La hormona prolactina (PRL) es primordial para la mamogénesis y lactogénesis en el ganado (Tucker, 2000), la perturbación de la función de la PRL durante el período seco puede afectar la producción de leche futura. De hecho, la señalización de PRL alterada inducida por un fotoperiodo variable afecta el desarrollo de la glándula mamaria durante el período seco

(Wall *et al.,* 2005) y la posterior producción de leche (Auchtung *et al.,* 2005) y tiene efectos negativos en lo que concierne a la reproducción (Noordhuizenn y Bonnefoy, 2015).

El estrés por calor durante el final de la gestación reduce la secreción placentaria de sulfato de estrona, que mejora el crecimiento mamario. El estrés por calor puede atenuar la involución mamaria a través de la secreción reducida de sulfato de estrona y el aumento de la concentración de PRL en la sangre en el período seco temprano (Tao *et al.,* 2018).
Las vacas que experimentan estrés por calor tienen una reducción del tamaño de la placenta, y, por ende, el peso de la cría se ve disminuido por la reducción de las hormonas placentarias como el sulfato de estrona, lactógeno placentario, y glucoproteína asociada a la preñez, además de una menor ingesta de alimento como otra posible causa de éste (Dahl *et al.,* 2016). De igual manera el retraso en el crecimiento de las ovejas se atribuye a una disminución en la actividad de las hormonas tiroideas, así como, baja secreción de progesterona y del lactógeno placentario por causa del estrés térmico (Bell *et al.,* 1989).

3.4. Fisiología del rumen e ingesta de alimento

El aumento de la temperatura ambiental tiene un efecto negativo directo sobre el centro del apetito del hipotálamo ocasionando la disminución en la ingesta de alimento. El consumo de alimento en vacas Holstein comienza a disminuir a una temperatura ambiental de 25-26 °C en vacas lactantes y se reduce más rápidamente por encima de los 30 °C en condiciones climáticas templadas y a 40 °C puede haber una disminución de hasta un 40%, en las cabras lecheras puede llegar hasta un 22-35% y 8-10% en novillas búfalo. El estrés por calor a largo plazo disminuyó la producción de leche en un 17.2% y la ingesta de materia seca en un 12.6% en vacas lecheras, lo que indica que el estrés por calor a largo plazo provocó una disminución más severa en la producción de leche y la ingesta de alimento que el estrés por calor de corto plazo (Hou et al., 2021).

Reducir la ingesta de alimento es una forma de disminuir la producción de calor en ambientes cálidos, así como, el incremento de calor de la alimentación es una fuente importante de producción de calor en los rumiantes. Como resultado, los animales experimentan una etapa de balance energético negativo (NEB), y en consecuencia pérdida de peso y baja condición corporal (Das *et al.,* 2016; Noordhuizen y Bonnefoy, 2015).

La causa principal de la disminución de la producción de leche se debe, en primer lugar, a que las vacas disminuyen el consumo de materia seca, lo cual trae como resultado una disminución de la productividad láctea (Baumgard *et al.,* 2011; Dahl *et al.,* 2016), ya que se disminuye la rumia, la producción de saliva, la masticación y la fermentación del alimento, todo esto a causa del jadeo elevado, producto del estrés por calor (Pawar *et al.,* 2018; Liu *et al.,* 2019). Sin embargo, esta disminución representa solo el 35% de la baja en la producción de leche, el resto pudiera atribuirse a los NEFA basales del metabolismo postabsortivo (Rhoads *et al.,* 2009). West (2003), reportó una reducción en la ingesta de materia seca de 0.85 kg por cada 1 ^{0}C en la temperatura ambiental por encima de la ZTN de una vaca, que representa el 36% de la disminución de la producción de leche.

Por otro lado, esta disminución de consumo reduce la concentración de ácidos grasos de cadena corta (AGCC) en el rumen encontrándose elevado el butirato y disminuido el acetato como respuesta a las altas temperaturas (Tajima *et al.,* 2007), lo cual podría traer como consecuencia disminución en la proteína de la leche y la grasa a causa del estrés por calor (Gao *et al.,* 2017). Sin embargo, Xie *et al. (*2016), reportó que existe resistencia a la insulina por el bajo consumo de alimento debido al impacto que tiene el estrés térmico, pero la señalización en el hígado y en el músculo esquelético permanecieron sin cambios en su estudio.

Otro factor es la hipercetonemia que afecta negativamente la producción de leche en mayor proporción en vacas multíparas que primíparas, lo cual es razonable porque las vacas de primera lactancia no tienen el estado NEB de la lactancia previa como factor de riesgo potencial, y en promedio tienen una mejor condición corporal y producen menos leche que vacas multíparas (Benedet *et al.,* 2019).

Los ácidos grasos monoinsaturados aumentaron con la hipercetonemia. La disminución de la síntesis de Novo de ácidos grasos en la leche podría sugerir una glándula mamaria menos activa metabólicamente, mientras que el incremento de ácidos grasos de cadena larga y ácidos grasos insaturados podría estar relacionada con una mayor fermentación ruminal acidógena debido a una menor ingesta de materia seca y una mayor velocidad de paso (Benedet *et al.,* 2019).

Debido al metabolismo sistémico alterado, se prevé que el estrés por calor influya en la disponibilidad de nutrientes y la absorción de la glándula mamaria. La absorción mamaria de nutrientes está determinada por la diferencia de concentración arteriovenosa de nutrientes y el flujo sanguíneo mamario (Pacheco-Rios *et al.,* 2001; Chaiyabutr, 2012), y la explicación de los efectos del estrés por calor en ambos factores es fundamental para comprender la absorción de nutrientes mamarios. El flujo de sangre a la glándula mamaria bovina juega un papel clave en el suministro de nutrientes adecuados para apoyar la síntesis de leche (Tao *et al.,* 2018).

3.5. El crecimiento fetal y la placenta

El crecimiento y desarrollo fetal pueden verse afectados por los cambios en el medio ambiente, así como la susceptibilidad a enfermedades en la vida futura de las crías, por lo que es importante la detección de nutrientes placentarios ya que esta información permite conocer las vías de señalización en relación a la secreción de hormonas, la transferencia de nutrientes placentarios y oxigenantes (Dimasuay *et al.,* 2016). Limesand *et al.* (2004), en un estudio realizado en ovejas concluyó que hubo una reducción en el GLUT 8 el cual ha disminuido la capacidad de transporte de la glucosa placentaria e hipoglucemia fetal.

Por otro lado, Macko *et al.* (2013), reportó que elevaciones en la norepinefrina plasmática fetal suprime las concentraciones de insulina lo que provoca una respuesta compensatoria con la secreción de células b (islotes pancreáticos) presentes antes de la inducción del desarrollo intrauterino.

En un estudio realizado por Regnault *et al.* (2002), se encontró que en ovejas existe una disminución del flujo sanguíneo de la arteria aorta y umbilical lo que trae como consecuencia una disminución del peso de la placenta, así como su desarrollo y por ende deficiencias en cuanto a la función de ésta trayendo como consecuencia fallas en el crecimiento del feto por causa del estrés térmico.

El estrés por calor que experimentan las crías durante la gestación provoca efectos en ésta en lo que concierne a la respuesta a la insulina, provocando una reducción en la ingesta y el crecimiento del ternero y alterando los metabolitos sanguíneos y elevando la captación de glucosa no dependiente de insulina (O´Brien *et al.,* 2010; Monteiro *et al.,* 2016).

Las crías nacidas de vacas que son expuestas a estrés por calor durante la gestación (período seco), tienen menor peso al nacer 12.4 % (4.6 kg) y al destete 10.4 % (7.1 kg), así como baja inmunidad pasiva comparados con los que son nacidos de vacas que son provistas de enfriamiento, por lo cual se ve comprometida la supervivencia y la producción de leche de estas terneras hasta la primera lactancia (Noordhuizen y Bonnefoy, 2015; Habeeb *et al.,* 2018; Monteiro *et al.,* 2016; Tao *et al.,* 2012; Dahl *et al.,* 2019; Ouellet *et al.,* 2020).

3.6. La reproducción y el estrés por calor

El estrés térmico afecta de manera negativa la tasa de concepción en el ganado lechero, ya que ésta se reduce del 31 % al 12 % cuando el ITH está en 73 o más unidades calor tanto antes como después de que se inseminan (Schüller *et al.,* 2014; De Rensis *et al.,* 2015; Mellado *et al.,* 2016). La duración prolongada del folículo preovulatorio dominante se debe a que emerge de manera temprana por efectos del estrés por calor, lo que trae como consecuencia disminución en la tasa de fertilidad, debido a que se reduce la capacidad esteroidogénica del folículo, por una menor actividad de la aromatasa en las células de la granulosa y las bajas concentraciones de estradiol en el folículo dominante (De Rensis y Scaramuzzi, 2003; Wolfenson y Roth, 2018).

En corderos el estrés térmico afecta el estro, la producción de embriones y el peso al nacer de los cabritos, tamaño y función de la placenta y la tasa de crecimiento fetal, sin embargo, factores como la nutrición o el entorno físico juegan un papel importante (Sejian *et al.,* 2012). El estrés por calor provoca efectos negativos como bajos niveles de estradiol y trae como consecuencia la duración del celo, provocando anestro o bien originando una ovulación silenciosa, así como un menor número de montas, por otro lado, la baja liberación de LH durante el pico preovulatorio, trae como resultado alteraciones en la ovulación, maduración de ovocitos o en ambos, que se formen quistes ováricos, además de falla del funcionamiento lúteo tales como baja en la producción y secreción de progesterona, mala calidad de los ovocitos, pérdidas embrionarias y fetales tempranas, bajo flujo sanguíneo uterino (De Rensis *et al.,* 2015; Roth, 2015; Roth, 2017; Lees *et al.,* 2019).

La tasa de preñez se ve altamente afectada por el estrés por calor en vacas cuando la temperatura alcanza los 30 ^{0}C y en las vaquillas la tasa de concepción se ve reducida cuando éstas alcanzan los 35 ^{0}C, por otro lado, las vaquillas requirieron 1.5 servicios por concepción compradas con 2.3 en las vacas lactantes (Sejian *et al.,* 2012; Habeeb *et al.,* 2018).

Las cerdas para disipar el calor por las temperaturas elevadas redireccionan el flujo de sangre de la glándula mamaria hacia la piel, lo que trae como consecuencia baja producción de leche y por ende un crecimiento disminuido en los lechones, además de celos reducidos en los 15 días posteriores al destete, como consecuencia de una baja ingesta de alimento por causa del estrés térmico (Baumgard *et al.,* 2015).

3.7. La producción de leche y el estrés térmico

En vacas con mayor mérito genético para la producción de leche, la lipólisis puede ser mayor debido a un aumento de la respuesta a la estimulación β-adrenérgica, aumentó de la actividad de la lipasa sensible a hormonas (LIPE), y una lipogénesis disminuida en comparación con los animales de mérito genético promedio. Debido a una lipólisis más rápida, generalmente las vacas lecheras de alta producción tienen una condición corporal moderada (> 2.5 y ≤ 3.0) al comienzo del período de transición, que en consecuencia hará que tengan una menor movilización de las reservas corporales y la concentración de NEFA y BHBA (Ácido betahidroxibutírico) (Tao *et al.,* 2012; Baumgard *et al.,* 2015; Barletta *et al.,* 2017; Tao *et al.,* 2018).

Las vacas con una producción mayor son más propensas al estrés por calor ya que generan más calor metabólico (Kadzere *et al.,* 2002; Bernabucci *et al.,* 2010). Las vacas multíparas son más sensibles al estrés por calor o ITH elevado, lo que trae como consecuencia una producción menor hasta de 1 kg de leche por día y una disminución en cuanto a la exhibición del celo (Collier *et al.,* 2006; Bernabucci *et al.,* 2014; Cowley *et al.,* 2015; Liu *et al.,* 2019). Las vaquillas hijas de vacas que son expuestas a estrés por calor en la gestación presentaron alvéolos mamarios más pequeños y menos células epiteliales secretoras comparadas con las que recibían enfriamiento, lo que puede contribuir a un menor rendimiento en la producción láctea hasta por tres lactancias (Wheelock *et al.,* 2010; Dahl *et al.,* 2019; Tao *et al.,* 2019; Laporta *et al.,* 2020).

3.8. Salud de las vacas

Otro indicador encontrado en las vacas que experimentan estrés térmico es la alta incidencia de mastitis clínica y subclínica, ya que se tomó como referencia el conteo de células somáticas para ver la salud de la ubre (Lambertz *et al.,* 2014; Gantner *et al.,* 2017; Roth y Wolfenson, 2016; Hamel *et al.,* 2021), además de mayor incidencia en la retención de membranas fetales, enfermedades respiratorias (Thompson *et al.,* 2014) y laminitis (Cook *et al.,* 2007; Sander *et al.,* 2009). Las vacas que son expuestas a un ITH >78 presentaron un aumento en patógenos como Staphylococcus *aureus* y Escherichia *coli*, además de una disminución en la grasa de la leche y la proteína (Vitali *et al.,* 2016; Dahl, 2018).

3.9. Inmunidad en los terneros

El sistema inmunológico tiene como función proteger a los individuos de los efectos ambientales y factores de estrés que estos pudieran experimentar, está constituido por los glóbulos blancos, los glóbulos rojos, la hemoglobina, plaquetas, concentración de glucosa y proteínas en sangre que pueden alterarse por el estrés térmico (Das *et al.,* 2016).

El estrés térmico afecta la inmunidad en los terneros y su crecimiento posterior, ya que los animales que nacen de madres que experimentan estrés por calor presentan menor inmunidad pasiva como consecuencia de un aumento en la producción de cortisol a nivel eje HPA (Hipotálamo – Pituitaria - Adrenales) axis, inhibiendo la producción de citocinas, lo que trae como consecuencia un aumento en la ocurrencia de enfermedades en los animales, debido a la baja absorción de IgG (Tao *et al.,* 2012; Monteiro *et al.,* 2014;; Thompson *et al.,* 2014; Laporta *et al.,* 2017; Bagath *et al.,* 2019).

IV. LITERATURA CITADA

Adin, G., Gelman, A., Solomon, R., Flamenbaum, I., Nikbachat, M., Yosef, E., Zenou, A., Shamay, A., Feuermann, Y., Mabjeesh, S., Miron, J. (2009). Effects of cooling dry cows under heat load conditions on mammary gland enzymatic activity, intake of food water, and performance during the dry period and after parturition. Livest. Sci. 124, 189–195.

Aggarwal, A. Upashyay, R. (2013). Heat Stress and Animal Productivity. Springer India, p.2.

Ahmed, B., Younas, U., Asar, T., Dikmen, S., Hansen, P., Dahl, G. (2017). Cows exposed to heat stress during fetal life exhibit improved thermal tolerance. J. Anim. Sci. 95, 3497–3503.

Bagath, M., Krishnan, G., Devaraj, C., Rashamol, V. P., Pragna, P., Lees, A. M., Sejian, V. (2019). The impact of heat stress on the immune system in dairy cattle: A review. Res. Vet. Sci. 126, 94-102.

Barletta, R., Fiho, M., Carvalho, P., Del Valle, T., Netto, A., Rennó, F., Mingoti, R., Gandra, J., Mourao, G., Fticke, P., Santori, R., Madureira, E., Wiltbank, M. (2017). Association of changes among body condition score during the transition period with NEFA and BHBA concentrations, milk production, fertility, and health of Holstein cows. Theriogenology 1-34.

Baumgard, L., Keating, A., Ross, J., Rhoads, R. (2015). Effects of heat stress on the immune system, metabolism and nutrient partitioning: implications on reproductive success. Rev. Bras. Reprod. Anim. 39, 173-183.

Baumgard, L., Rhoads, R., Rhoads, M., Gabler, N., Ross, J., Keating, A., Boddicker, R., Lenka, S., Sejian, V. (2012). Impact of Climate Change on Livestock Production. Chapter 15, Springer-Verlag Berlin Heidelberg.

Baumgard, L., Wheelock, J., Sanders, S., Moore, C., Green, H., Waldron, M., Rhoads, R. (2011). Postabsorptive carbohydrate adaptations to heat stress and monensin supplementation in lactating Holstein cows. J. Dairy Sci. 94, 5620–5633.

Baumgard, L., Wheelock, J., Shwartz, G., O´Brien, M., VanBaale, M., Collier, R., Rhoads, M., Rhoads, R. (2006). Effects of Heat Stress on Nutritional Requirements of Lactating Dairy Cattle. Proceedings of the 5th Annual Arizona Dairy Production Conference, pp. 1–10.

Bell, A., McBride, B., Slepetis, R., Early, R., Currie, W. (1989). Chronic Heat Stress and Prenatal Development in Sheep: I. Conceptus Growth and Maternal Plasma Hormones and Metabolites. J. Anim. Sci. 67, 3289 – 3299.

Benedet, A., Manuelian, C., Zidi, A., Penasa, M., De Marchi, M. (2019). Invited review: β-hydroxybutyrate concentration in blood and milk and its associations with cow performance, The Animal Consortium, 1-14.

Bernabucci, U., Biffani, S., Buggiotti, L., Vitali, A., Lacetera, N., Nardone, A. (2014). The effects of heat stress in Italian Holstein dairy cattle. Journal Dairy Sci. 97, 471–486.

Bernabucci, U., Lacetera, N., Baumgard, L. H., Rhoads, R. P., Ronchi, B., Nardone, A. (2010). Metabolic and hormonal acclimation to heat stress in domesticated ruminants. Animal 4, 1167-1183.

Boddicker, R., Lenka, S., Sejian, V. (2012). Impact of Climate Change on Livestock Production. In Environmental Stress and Amelioration in Livestock Production. Springer, 413–468.

Campos, P., Le Floc´h, N., Noblet, J., Renaudeau, D. (2017). Invited Review Physiological responses of growing pigs to high ambient temperature and/or inflammatory challenges. Braz. J. Anim. Sci. 46, 537- 544.

Cardoso, C., Peripolli, V., A mador, S., Brandao, E., Esteves, G., Sousa, C., Franca, M., Goncalves, F., Barbosa, F., Montalvao, T., Martins, C., FonsecaNeto, A., Manus, C. (2015). Physiological and thermographicresponse to heat stress in zebu cattle. Livest. Sci. 182, 83–92.

Carroll, J., Burdick, N., Chase, C., Coleman, S., Spiers, D. (2012). Influence of environmental temperature on the physiological, endocrine, and immune responses in livestock exposed to a provocative immune challenge. Domestic Animal Endocrinology, 43,146–153.

Church, D. (1988). The Ruminant Animal: Digestive Physiology and Nutition. Editorial Acribia, Zaragoza, España, p. 527.

Collier, R., Baumgard, L., Zimbelman, R., Xiao, Y. (2019). Heat Stress: Physiology of acclimation and adaptation, Anim. Front. 9,11-19.

Collier, R., Dahl, G., VanBaale, M. (2006). Major Advances Associated with Environmental Effects on Dairy Cattle. J. Dairy Sci. 89, 1244–1253.

Collier, R., Gebremedhin, K. (2015). Thermal Biology of Domestic Animals. The Annual Review of Animal Biosciences, 10-20.

Collier, R., Renquist, B., Xiao, Y. (2017). A 100 Year Review: Stress physiology including heat stress. J. Dairy Sci. 100,10367- 10380.

Cook, N., Mentink, R., Bennett, T., Burgi, K. (2007). The Effect of Heat Stress and Lameness on Time Budgets of Lactating Dairy Cows. J. Dairy Sci. 90, 1674–1682.

Cowley, F., Barber, D., Houlihan, A., Poppi, D. (2015). Immediate and residual effects of heat stress and restricted intake on milk protein and casein composition and energy metabolism. J. Dairy Sci. 98, 1–13.

Dahl, G. (2018). Impact and Mitigation of Heat Stress for Mastitis Control. Vet Clin Food Anim 34, 473–478.

Dahl, G., Skibiel, A., Laporta, J. (2019). In utero heat stress programs reduced performance and health in calves. Vet. Clin. Food Anim. 35, 343–353.

Dahl, G., Tao, S., Monteiro, P. (2016). Effects of late-gestation heat stress on immunity and performance of calves. J. Dairy Sci. 99, 1–6.

Das, R., Sailo, L., Verma, N., Bharti, P., Saikia, J., Imtwati, Kumar, R. (2016). Impact of heat stress on health and performance of dairy animals: A review, Veterinary World, EISSN: 2231-0916, 260-268.

De Rensis, F., Garcia-Isprieto, I., López-Gatius, F. (2015). Review: Seasonal heat stress: Clinical implications and hormone treatments for the fertility of dairy cows. Theriogenology 84, 659–666.

De Rensis, F., Scaramuzzi, R. (2003). Heat stress and seasonal effects on reproduction in the dairy cow—a review. Theriogenology 60, 1139–1151.

Dimasuay, K., Boeuf, P., Powell, T., Jansson, T. (2016). Placental Responses to Changes in the Maternal Environment Determine Fetal Growth. Front. Physiol., 1-9.

Do Amaral, B., Connor, E., Tao, S., Hayen, M., Bubolz, J., Dahl, G. (2009). Heat-stress abatement during the dry period: Does cooling improve transition into lactation? J. Dairy Sci. 92, 5988–5999.

Dreiling, Ch., Carman, F., Brown, D. (1991). Maternal Endocrine and Fetal Metabolic Responses to Heat Stress. J. Dairy Sci. 74, 312-327.

Fabris, T., Laporta, J., Corra, F., Torres, Y., Kirk, D., McLean, D., Chapman, J., Dahl, G. (2017). Effect of nutritional immunomodulation and heat stress during the dry period on subsequent performance of cows. J. Dairy Sci. 100, 1–10.

FAO 2013. Greenhouse gas emissions from ruminant supply chains, a global life cycle assessment. FAO, Rome.

Gantner, V., Bobici, T., Gantner, R., Gregic, M., Kuterovac, K., Novakovic, J., Potocnik, K. (2017). Differences in response to heat stress due to production level and breed of dairy cows. Int J Biometeorol. 61 1675–1685.

Gao, S., Guo, J., Quan, S., Nan, X., Sanz, M. Baumgard, L., Bu, D. (2017). The effects of heat stress on protein metabolism in lactating Holstein cows. J. Dairy Sci. 100, 5040–5049.

Gerber, P., Hristov, A., Makkar, H., Oh, J., Lee, C., Meinen, R., Montes, F., Ott, T., Frirkins, J., Rotz, A., Dell, C., Adesogan, A., Yang, W., Tricarico, J., Kebreab, E., Waghorn, G., Dijkstra, J., Oosting, S. (2013). Technical options for the mitigation of direct methane and nitrous oxide emissions from livestock: a review. Animal 7, 220–234.

Grossi, G., Goglio, P., Vitali, A. Williams, A.G. (2019). Livestock and climate change: Impact of livestock on climate and mitigation strategies. Anim. Front. 9(1) doi: 10.1093/af/vfy034.

Habeeb, A., Gad, A., Atta, M. (2018). Temperature- hUmidity Indices as Indicators to Heat Stress of Climatic Conditions with Relation to Production and Reproduction of Farm Animals; Review Article. Int. J. Biotechnol. Recent Adv. 1, 35-50.

Habeeb, A., Gad, A., El – Tarabany, A., Atta, M. (2018). Negative Effects of Heat Stress on Growth and Milk Production of Farm Animals. J. Anim. Husb. Dairy Sci. 2, 1-12.

Hamel, J., Zhang, Y., Wente, N., Krömker, V. (2021). Heat stress and cow factors affect bacteria shedding pattern from naturally infected mammary gland quarters in dairy cattle. J. Dairy Sci. 104, 786–794.

Herbut, P., Angrecka, S., Godyń, D., Hoffmann, G. (2019) The physiological and productivity effects of heat stress in cattle-A review. Ann. Anim. Sci. 19, 579–594.

Hou, Y., Zhang, L., Dong, R.Y., Liang, M.Y., Lu, Y., Sun, X.Q., Zhao, X. (2021). Comparing responses of dairy cows to short-term and long-term heat stress in climate-controlled chambers. J. Dairy Sci. 104, 2346-2356.

Jeelani, R., Konwara, D., Khana, A., Kumarb, D., Chakrabortyb, D., Brahmaa, B (2019). Reassessment of temperature-humidity index for measuring heat stress in crossbred dairy cattle of a sub-tropical region. J. Therm. Biol. 82, 99–106.

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.

Lambertz, C., Sanker, C., Gauly, M. (2014). Climatic effects on milk production traits and somatic cell score in lactating Holstein-Friesian cows in different housing systems. J. Dairy Sci. 97, 319–329.

Laporta, J., Fabris, T., Skibiel, A., Powell, J., Hayen, M., Horvath, K., Miller- Cushon, E., Dahl, G. (2017). In utero exposure to heat stress during late gestation has prolonged effects on the activity patterns and growth of dairy calves. J. Dairy Sci. 100, 1–9.

Laporta, J., Ferreira, F., Ouellet, V., Dado-Seen, B., Almeida, A., De Vries, A., Dahl, G. (2020). Late-gestation heat stress impairs daughter and granddaughter lifetime performance. J. Dairy Sci. 103, 7555–7568.

Lees, A., Sejian, V., Wallage, A., Steel, C., Mader, T., Lees, J., Gaughan, J. (2019). Review: The Impact of Heat Load on Cattle. Animals 9, 1-20.

Limesand, S., Regnault, T., Hay W. (2004). Characterization of Glucose Transporter 8 (GLUT8) in the Ovine Placenta of Normal and Growth Restricted Fetuses. Placenta 25, 70–77.

Liu, J., Li, L., Chen, X., Lu, Y., Wang, D. (2019). Effects of heat stress on body temperature, milk production, and reproduction in dairy cows: a novel idea for monitoring and evaluation of heat stress — A review. Asian-Australas J Anim Sci. 39, 1332-1339.

López, E., Mellado, M., Martínez, A., Véliz, F., García, J., de Santiago, A., Carrillo, E. (2017). Stress-related hormonal alterations, growth and pelleted starter intake in pre-weaning Holstein calves in response to thermal stress. Int J Biometeorol, Springer, 1-8.

Macko, A., Yates, D., Chen, X., Green, A., Kelly, A., Brown, L., Limesand, S. (2013). Elevated plasma norepinephrine inhibits insulin secretion, but adrenergic blockade reveals enhanced b-cell responsiveness in an ovine model of placental insufficiency at 0.7 of gestation. J. Dev. Origins Health Dis. 4, 402–410.

Mader, T. (2003). Environmental stress in confined beef cattle. J. Anim. Sci. 81, Suppl. 2, 110–119.

Mellado, M., López, R., de Santiago, A., Veliz, F., Macías-Cruz, U., Avendaño-Reyes, L., García, E. (2016). Climatic conditions, twining and frequency of milking as factors affecting the risk of fetal losses in high-yielding Holstein cows in a hot environment. Trop. Anim. Health Prod. 48, 1-6.

Monteiro, A., Guo, J., Weng, X., Ahmen, B., Hayen, M., Dahl, G., Bernard, J., Tao, S. (2016). Effect of maternal heat stress during the dry period on growth and metabolism of calves. J. Dairy Sci. 99, 3896–3907.

Monteiro, A., Tao, S., Thompson, I., Dahl, G. (2014). Effect of heat stress during late gestation on immune function and growth performance of calves: Isolation of altered colostral and calf factors. J. Dairy Sci. 97, 1–14.

Nienaber, J.A., Hahn, G.L. (2007). Livestock production system management responses to thermal challenges. Int. J. Biometeorol. 52, 149–157.

Noordhuizen, J., Bonnefoy, J. (2015). Heat stress in dairy cattle: major effects and practical management measures for prevention and control. symbiosis, SOJ. Vet. Sci. 45, 1-7.

O´Brien, M., Rhoads, R., Sanders, S., Duff, G., Baumgard, L. (2010). Metabolic adaptations to heat stress in growing cattle. Dom. Anim. Endocrinol. 38, 86–94.

Ouellet, V., Laporta, J., Dahl, G. (2020). Late gestation heat stress in dairy cows: Effects on dam and daughter. Theriogenology, 150, 471-479.

Pawar, M., Srivastava, A., Chauhan, H., Damor, S. (2018). Nutritional strategies to alleviate heat stress in dairy animals – a review. Int. J. Livest. Res. 8, 8-18.

Pragna, P., Archana, P., Aleena, J., Sejian,V., Krishnan, G., Bagath, M., Manimaran, A., Beena, V., Kurien, E., Varma, G., Bhatta, R. (2016). Review Article Heat Stress and Dairy Cow: Impact on Both Milk Yield and Composition, Int. J. Dairy Sci. 55, 1-12.

Polsky, L., von Keyserlingk, M. (2017). Invited review: Effects of heat stress on dairy cattle welfare. J. Dairy Sci. 100, 8645–8657.

Rahman, M., Schellander, K., Luceño, N., Soom, A. (2018). Heat stress responses in spermatozoa: Mecanisms and consequences for cattle fertility, Theriogenology, 1-47.

Regnault, T., Orbus, R., de Vrijer, B., Davidsen, M., Galan, H., Wilkening, R., Anthony, R. (2002). placental expression of vegf, plgf and their receptors in a model of placental insufficiency—intrauterine growth restriction (PI-IUGR), Placenta 23, 123–144.

Reynolds, C., Crompton, L., Mills, J. (2010). Livestock and climate change impacts in the developing world. J Agric. 39, 245–248.

Rhoads, M., Rhoads, R., VanBaale, M., Collier, R., Sanders, S., Weber, W., Crooker, B., Baumgard, L. (2009). Effects of heat stress and plane of nutrition on lactating Holstein cows: I. Production, metabolism, and aspects of circulating somatotropin. J. Dairy Sci. 92, 1986–1997.

Rosegrant, M., Fernandez, M., Sinha, A. (2009). Looking into the future for agricultura and AKST, In; Mclntyre, B. D., Herren, H. R., Wakhungu, J., Watson, R. Ed. International Assessment of Agricultural Knowledge, Science and Tecnology for Development, Washington, DC. pp. 307-376.

Roth, Z. (2015). Physiology and endocrinology symposium: Cellular and molecular mechanisms of heat stress related to bovine ovarian function. J. Anim. Sci. 93, 2034–2044.

Roth, Z. (2017). Effect of Heat Stress on Reproduction in Dairy Cows—Insights into the Cellular and Molecular Responses of the Oocyte. Annu. Rev. Anim. Biosci. 5, 2–20.

Roth, Z., Wolfenson, D. (2016). Comparing the effects of heat stress and mastitis on ovarian function in lactating cows: basic and applied aspects. Domestic Animal Endocrinology 56, S218–S227.

Sanders, A., Shearer, J., De Vries, A. (2009). Seasonal incidence of lameness and risk factors associated with thin soles, white line disease, ulcers, and sole punctures in dairy cattle. J. Dairy Sci. 92, 3165–3174.

Schüller, L., Burfeind, O., Heuwieser, W. (2014). Impact of heat stress on conception rate of dairy cows in the moderate climate considering different temperature – humidity index thresholds, periods relative to breeding, and heat load índices. Theriogenology 81,1050–1057.

Sejian, V., Bhatta, R., Gaughan, J., Dunshea, F., Lacetera, N. (2018). Review: Adaptation of animals to heat stress. Animal 12, 431–444.

Sejian, V., Naqvi, S., Rattan, J. (2012). Environmental Stress and Amelioration in Livestock Production. Springer.

Senn, B., Skibel, A., Fabris, T., Dahl, G., Laporta, J. (2019). Dry period heat stress induces microstructural changes in the lactanting mammary gland. Plos One 1-15.

Skibiel, A., Senn, B., Fabris, T., Dahl, G., Laporta, J. (2018). In utero exposure to thermal stress has longterm effects on mammary gland microstructure and function in dairy cattle. Plos one 13, 1-13.

St-Pietre, N., Cobanov, B., Schnitkey, G. (2003). Economic losses from heat stress by US livestock industries. J. Dairy Sci. 86, E52-E77.

Tajima, K., Nonaka, I., Higuchi, K., Takusari, N., Kurihara, M., Takenaka, A., Mitsumori, M., Kajikawa, H., Aminov, R. I. (2007). Influence of high temperature and humidity on rumen bacterial diversity in Holstein heifers. Anaerobe 13, 57-64.

Tao, S., Bubolz, J., do Amaral, B., Thompson, I., Hayen, M., Johnson, S., Dahl, G. (2011). Effect of heat stress during the dry period on mammary gland development. J. Dairy Sci. 94, 5976–5986.

Tao, S., Dahl, G. (2013). Invited review: Heat Stress effects during late gestation on dry cows and their calves. J. Dairy Sci. 96, 1–15.

Tao, S., Dahl, G., Laporta, J., Bernard, J., Orellana, R., Marins, T. (2019). Physiology Symposium: Effects of heat stress during late gestation on the dam and its calf. J. Anim. Sci. 97, 2245-2257.

Tao, S., Monteiro, A., Hayen, M., Dahl, G. (2014). Short communication: Maternal heat stress during the dry period alters postnatal whole-body insulin response of calves. J. Dairy Sci. 97, 897–901.

Tao, S., Monteiro, A., Thompson, I., Hayen, M., Dahl, G. (2012). Effect of late-gestation maternal heat stress on growth and immune function of dairy calves. J. Dairy Sci. 95, 7128–7136.

Tao, S., Orellana, R., Weng, X., Marins, T., Dahl, G., Bernard, J. (2018). Symposium review: The influences of heat stress on bovine mammary gland function. J. Dairy Sci. 101, 1–13.

Tao, S., Thompson, I., Monteiro, A., Hayen, M., Young, L., Dahl, G. (2012). Effect of cooling heat-stressed dairy cows during the dry period on insulin response. J. Dairy Sci. 95, 5035–5046.

Thompson, I., Tao, S., Monteiro, A., Jeong, K., Dhal, G. (2014). Effect of cooling during the dry period on immune response after Streptococcus uberis intramammary infection challenge of dairy cows. J. Dairy Sci. 97, 7426–7436.

Thornton, K. (2010). Livestock production: recent trends, future prospects. Philos. Trans. Roy. Soc. 365, 2853–2867.

Thornton, P., Gerber, J. (2010). Climate change and the growth of the livestock sector in developing countries. Mitig. Adapt. Strat. Global Change 15, 169–184.

Vitali, A., Bernabucci, U., Nardone, A., Lacetera, N. (2016). Effect of season, month and temperature humidity index on the occurrence of clinical mastitis in dairy heifers. Advances in Animal Biosciences, 7, 250–252.

Wall, E., Auchtung-Montgomery, T., Dahl, G., McFadden, T. (2005). Short Communication: Short-Day Photoperiod During the Dry Period Decreases Expression of Suppressors of Cytokine Signaling in Mammary Gland of Dairy Cows. J. Dairy Sci. 88, 3145–3148.

West, J. W. (2003). Effects of heat-stress on production in dairy cattle. J. Dairy Sci. 86, 2131–2144.

Wheelock, J., Rhoads, R., VanBaale, M., Sanders, S., Baumgard, L. (2010). Effects of heat stress on energetic metabolism in lactating Holstein cows. J. Dairy Sci. 93, 644–655.

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.

Wolfenson, D., Roth, Z. (2018). Impact of Heat Stress on Cow Reproduction and Fertility. Feature Article, Am. Soc. Anim. Sci. 20, 1–7.

Wright, I., Tarawali, S., Blummel, M., Gerard, B., Teufel, N., Herrero, M. (2012). Integrating crops and livestock in subtropical agricultural systems. J. Sci. Food Agric. 92, 1010–1015.

Xie, G., Cole, L., Zhao, L., Skrzypek, M., Sanders, S., Rhoads, M., Baumgard, L., Rhoads, R. (2016). Skeletal muscle and hepatic insulin signaling is maintained in heat-stressed lactating Holstein cows. J. Dairy Sci. 99, 1–11.

Zhao, X., Ponchon, B., Lanctôt, S., Lacasse, P. (2019). Invited review: Accelerating mammary gland involution after drying-off in dairy cattle. J. Dairy Sci. 102, 6701-6717.

V. ARTÍCULOS

El desarrollo de esta tesis está sustentado en los siguientes artículos:

5.1 In utero heat stress effects on cows' cumulative milk yield

Animal Science Papers and Reports vol. 38 (2020) no. 4, 345-353
Institute of Genetics and Animal Biotechnology
of the Polish Academy of Sciences, Jastrzębiec, Poland

In utero heat stress effects on cows´ cumulative milk yield

María I. Chavez[2], José E. García[1], Francisco G. Véliz[2], Leticia Gaytán[2], Ángeles de Santiago[2], Miguel Mellado[1]*

[1] Department of Animal Nutrition, Autonomous Agrarian University Antonio Narro Saltillo, Coahuila, 25315 Mexico

[2] Department of Veterinary Science, Autonomous Agrarian University Antonio Narro, Department of Veterinary Science, Torreon, Coahuila, 27054 Mexico

(Accepted August 26, 2020)

The objective of this study was to determine whether in utero heat stress (IUHS) affects milk yield and lactation traits in Holstein cows in a hot environment. A total of 5278 cows with three consecutive lactations were used. Temperature-humidity index (THI) classes were defined as <76, 76-83 and >83 units. Average lactation length for first-lactation heifers was 415 days (SD=81) with no difference between IUHS heifers and non-IUHS heifers. First-calving heifers born to cows not exposed to heat stress (HS) during delivery produced 170 more kg of milk ($P<0.05$) in 305-d lactations than heifers whose dams suffered HS (adjusted for the effects of THI one, two and three months previous to

calving). Total milk yield for the first lactation was 12477±2828 kg with no effect of THI at any stage of gestation. Heifers not exposed to IUHS at the day of delivery produced 0.7 more kg of milk daily compared to IUHS heifers at the day of delivery. Milk yield persistence was greater ($P<0.05$) in first-calving heifers born to dams suffering heat stress at calving (73.5±2.4%) compared to heifers born to HS cows at calving (72.9±2.3%). IUHS at any stage of pregnancy did not influence subsequent cumulative total milk yield (37574±5845; across THI). It was concluded that there was evidence for altered milk yield and lactation persistence among IUHS first-calving heifers compared to non-IUHS heifers. However, these effects were less pronounced in subsequent lactations; thus, the IUHS effects on milk production did not go beyond the immediate impact in the first lactation.

**KEY WORDS: cumulative milk yield / extended lactations / in utero heat stress /
lactation length / lactation persistency**

---

*Corresponding authors: mmelladomiguel07@gmail.com

Maternal exposure to continuous high ambient temperatures during the last months of pregnancy has a direct impact on fetal development. During this period of rapid fetal growth, heat stress (HS) negatively affects both placental development and function [Limesand *et al*. 2018], which is reflected in lighter calves at calving [Tao and Dahl 2013, López *et al*. 2017]. HS leads to reduced placental mass and function [de Vrijer *et al*. 2004, Macko *et al*. 2013], which results in the development and progressive decline in fetal glucose and oxygen concentrations over the final third of gestation when fetal growth rate is at a maximum [Limesand *et al*. 2018, Rozance *et al*. 2018].

Mechanisms explaining placental and fetal growth constraints include the redistribution of blood to the body surface and reduced perfusion of the placental vascular bed [Dreiling *et al*. 1991, McCrabb *et al*. 1993]. Reduced placental function and mass increases vascular resistance in the placenta due to a reduction of angiogenesis by an abnormal expression of vascular endothelial growth factor and its receptors and placental growth factor [Regnault *et al*. 2002, Hagen *et al*. 2005]. This reduces glucose transport across the placenta [Thureen *et al*. 1992] by lower abundance of facilitated glucose transporters [Limesand *et al*. 2004, Wallace *et al*. 2005], slows growth of fetuses and alters their endocrine and metabolic profiles [Limesand *et al*. 2018].

Additionally, relative to calves born to cooled cows, calves from non-cooled dry cows present slower insulin clearance after insulin challenges [Monteiro *et al*. 2016a], while both passive and cell-mediated immune function of calves is also impaired by prenatal heat stress [Tao *et al*. 2019]. Thus, placental nutrient provision plays an important role in modulating maternal-fetal resource allocation, thereby affecting not only fetal growth, but altering the life-long health of the offspring [Fouden *et al.* 2006, Dimasuay *et al*. 2016].

Although evidence exists for epigenetic mediation of intergenerational effects in offspring from heat-stressed dams [Sinclair *et al*. 2016], it seems that prenatal heat stress undergoes adaptations in endocrinology, metabolism and organ functions, which alter the developmental programming and may have long-lasting effects throughout life. Pronounced interest is currently observed in the potential role of epigenetics in underlying the long-term effects of prenatal stress on the development of the fetus and these epigenetic changes in the fetus after prenatal stress have been documented in animal models [Jensen Peña *et al*. 2012]. This point is particularly important in dairy cattle, as in utero heat stress (IUHS) seems to alter the mammary gland microstructure and cellular processes during the cow's first lactation [Skibiel *et al*. 2018].

Although there is evidence that prenatal heat stress alters growth, the immune systems and metabolism of calves [Tao *et al*. 2012, Monteiro *et al*. 2016a], as well as milk production up to and through the first lactation of offspring [Monteiro *et al.* 2016b], and reproductive performance [Dahl *et al*. 2016], there is limited research, as yet, linking prenatal heat stress to milk yield in subsequent lactations of offspring. Additionally, studies that investigated the effects of IUHS on milk yield used only one lactation; therefore, it would be convenient to assess if hyperthermia during the fetal life affects subsequent milk yields during various lactations. Thus, this study aimed to establish whether the temperature-humidity index (THI) experienced by dams at calving, one, two or three months previous to parturition could have carryover effects on the heifer's future milk production when considering multiple lactations.

Material and methods

The research protocol was reviewed and approved by the Institutional Animal Care and Use Committee at the Autonomous Agrarian University Antonio Narro.

Farm and cows management

Data were collected from one large commercial dairy farm (2800 cows) located in northeastern Mexico (25°N; 1120 m above sea level). Average annual temperature is 23.7°C and THI ranges from 70.1 to 89.8 units. Cows were housed in open dry-lot pens (161.5 m/cow) with shades (14.6 m/cow) in the center of each pen oriented north-south. Shade dimensions were 121.9 m long by 9.1 m wide by 4.0 m high. All animals had *ad libitum* access to feed and water and they were fed a total mixed ration balanced to meet or exceed nutrient requirements for prepartum Holstein cows weighing 650 kg (NRC 2001). Cows were fed two times daily a mixed ration with approximately 50% concentrate and approximately 5% leavings were removed immediately before each morning feeding.

Cows were inseminated after 50 days postpartum. Heifers and cows were observed for estrus three times a day, for approximately 30 min. Estrus detection was secured with the aid of pedometers and AI was conducted after visual observation of estrous behavior following the a.m. - p.m. guideline. Commercial sexed (for heifers) and non-sexed (pluriparous cows) frozen-thawed semen from multiple sires from the USA was used across all months of the year. Pregnancy was detected by palpation of the uterus per rectum about 45 days post-insemination. Services per pregnancy were 4.5, consequently lactation length was far beyond the traditional 10-11 months (>400 days).

Data collection

A retrospective study was conducted using 5278 cows with three consecutive lactations (from 2015 to 2019). Information was obtained from the farm software and consisted of birth date, calving dates, parity, date of dry-off, 305-d milk yield, days in milk and total milk yield. Cows became eligible for the study when they completed three consecutive lactations, regardless of reproductive difficulties which lead to extended lactations in many of the cows. Additional criteria for inclusion of cows in the study were that they had not initiated their lactation with abortion or hormonal treatment. Cows were dried off 60 d before expected parturition or when milk yield reached 20 kg/day. Milk yield was measured electronically at each milking using the Dairy Comp 305 software (Valley Agricultural Software, Tulare, CA) for each cow.

For the duration of the study, meteorological data were obtained from a climatic station located 3 km away from the dairy operation. Maximum temperatures and relative humidity were used to calculate the temperature-humidity index (THI; highest daily temperature in degrees Celsius; RH refers to maximum relative humidity) for each day using the following equation [Mader 2003]:

$$\text{THI} = [0.8 \times \text{temperature}] + [[\% \text{ RH}/100] \times [\text{temperature} - 14.4]] + 46.4$$

THI classes were defined as <76, 76-83 and >83 units. These cut-off values were based on THI values associated with changes in rectal temperature. For example, cows subjected to THI <76 show normothermia, whereas cows exposed to THI = 76 – 83 present rectal temperature ≈40°C and animals exposed to THI >83 present rectal temperature ≥40.5°C [Dikmen and Hansen 2009, Jeelani *et al*. 2019].

Statistical analyses

Milk production traits were analysed according to a completely randomized design with cow as the experimental unit. The statistical model included fixed effects of THI categories, with cow as a random effect. THI at calving, during the ninth, eighth and seventh month of pregnancy were included in the model, so that adjusted weather effects for particular months of gestation were calculated controlling for THI at different months of pregnancy. The computations were performed using the MIXED procedure of the SAS package programmes (SAS Institute Inc., Cary, N.C.)

Milk trait means were compared using the probability of a statistical difference (PDIFF option of SAS). The occurrence of extended lactations was analysed using the PROC GENMOD of SAS. Statistical differences were considered significant at $P<0.05$.

Results and discussion

First-lactation milk yield data of Holstein heifers experiencing heat stress at delivery and previous to delivery (in utero) are summarized in Table 1. Average lactation length for first lactation heifers was 415 days (SD=81) with no difference between groups for THI groups. These lactation lengths are far above the traditional 305 days in milk for cows with a 12-13 month calving interval and were caused by the prolonged warm weather in the study site, which provokes a high number of services per pregnancy and consequently a prolonged days open period. Thus, these results suggested that maternal HS during pregnancy does not negatively affect offspring lactation length.

First-calving heifers born to cows not exposed to HS during delivery produced 170 kg more ($P<0.05$) milk in 305-d lactation than heifers, whose dams suffered HS at calving (adjusted for the effects of THI one, two and three months previous to calving). Likewise, compared with heifers born to cows exposed to HS during the eight-month of pregnancy, 305-d milk yield was higher ($P<0.05$) in heifers from dams not suffering HS or under HS two months before parturition (adjusted for THI at calving, one and three months before delivery). These results suggest a direct effect of IUHS on lactational physiology in first-lactation heifers and a carryover effect of

Table 1. The effect of temperature-humidity index (THI) in first-lactation Holstein heifers born to cows exposed to heat stress at calving or one to three months prior to parturition Values are means (standard deviations in parenthesis)

Variables	THI at calving	THI one month before calving	THI two months before calving	THI three months before calving
Lactation length 1st lact (days)				
THI <76	412 (83)	410 (77)	413 (77)	416 (84)
THI 76-83	416 (82)	415 (85)	414 (84)	413 (80)
THI >83	418 (79)	419 (79)	417 (81)	417 (82)
305-d milk yield (kg)				
THI <76	9850^{a} (983)	9815 (1046)	9803^{a} (1004)	9760 (1066)
THI 76-83	9680^{b} (1090)	9768 (1031)	9707^{b} (1102)	9790 (1028)
THI >83	9803^{a} (1191)	9823 (1165)	9850^{a} (1158)	9835 (1148)
Total milk yield 1st lact (kg)				
THI <76	12433 (2805)	12397 (2705)	12469 (2873)	12550 (2944)
THI 76-83	12437 (2983)	12495 (2959)	12409 (2792)	12429 (2824)
THI >83	12568 (2749)	12517 (2745)	12533 (2788)	12483 (2743)
Milk/day in 305-d lact (kg)				
THI <76	32.3^{a} (3.2)	32.1 (3.5)	32.1^{a} (3.3)	31.9 (3.5)
THI 76-83	31.6^{b} (3.6)	31.9 (3.4)	31.8^{b} (3.6)	32.0 (3.4)
THI >83	32.1^{a} (3.9)	32.2 (3.9)	32.2^{a} (3.8)	32.2 (3.8)
Persistence 305-d milk yield (%)				
THI <76	73.5^{a} (2.4)	73.4^{a} (2.7)	73.4^{a} (2.4)	73.1 (2.3)
THI 76-83	72.9^{b} (2.3)	73.3^{a} (2.6)	73.0^{b} (2.3)	73.3 (2.4)
THI >83	73.0^{b} (2.3)	73.1^{b} (2.3)	73.2^{a} (2.4)	73.3 (2.4)

lact=lactation.

^{ab}Within variables, means in the same column differ significantly at P<0.05.

IUHS on the calf's future milk performance. The present results are in line with results of Monteiro *et al*. [2016b] and Laporta *et al*. [2018], who observed that heifers born to late gestation HS cows had lower milk yields during their first lactations compared to heifers not suffering from IUHS.

This reduced milk yield of IUHS heifers during late pregnancy occurs because as cows undergo thermal stress in late gestation, so do the fetuses that must maintain core temperatures by adjusting blood flow and nutrient utilisation to limit heat production. This hyperthermia decreases the alveolar area of mammary glands of lactating heifers [Skibiel *et al*. 2018]. Given the positive association between the luminal area of mammary alveoli and the quantity of mammary secretory epithelial cells they possess, the lower the alveoli area, the lower the milk synthesis and storage, which appears to reduce milk production observed in IUHT cows. Another possible explanation for the reduced milk yield in IUHS heifers is related to epigenetic effects, because maternal HS during the dry period alters DNA methylation of the mammary gland during their first lactation [Skibiel *et al*. 2018, Reynolds *et al*. 2019]; therefore, these epigenetic marks due to IUHS may contribute to the reduced performance of the offspring in their adult life. Further, IUHS heifers have a lower percent of proliferating mammary cells [Skibiel *et al*. 2018] and present altered endocrine systems through early life that are consistent with metabolic adaptations to accumulate energy in peripheral tissues and reduce lean growth. For instance, calves born to HS-dams present greater blood insulin in the first week of life relative to those born to cooled dams [Tao and Dahl 2013b]. Additionally, adiposity in puberty is related to subsequent milk yield [Silva *et al*. 2002]; therefore, the potentially greater accretion of fat in calves from HS dams could predispose those calves to alterations in mammary growth and reduced milk yield.

Due to the long lactations of first-calving heifers, total milk yield for the first lactation was 12477 kg (SD=2828) with no effect of IUHS at any stage of gestation. Heifers not exposed to IUHS at the day of delivery produced 0.7 more kg/day of milk compared to heifers exposed to IUHS at the day of calving. Surprisingly, daily milk yields of heifers suffering from severe IUHS at calving were not affected. These results were also found in animals exposed to IUHS two months previous to calving. Milk yield persistence was greater ($P<0.05$) in first-calving heifers born to dams not suffering from heat stress at calving, and one and two months before calving compared to IUHS heifers at calving and one or two months before calving.

For greater milk production and efficiency it is desirable to have cows with high lactation persistency. In the present study, IUHS heifers presented a marginal, but significant reduction in lactation persistence. These results further suggest that IUHS alters mammary growth throughout postnatal life and possibly restrict the maintenance of the number and activity of milk-secreting cells and increased cell death via apoptosis with advancing lactation, which ultimately reduced lactation persistence in IUHS cows.

None of the milk variables were affected by IUHS during the seventh month of pregnancy. In developmental programming, the timing and duration of stressors will influence particular responses [Reynolds *et al*. 2019]. Hence, the windows to IUHS reflected in subsequent milk yield seem to be active during the eighth and ninth month of pregnancy. Growth of bovine fetuses follows an exponential trend with the maximal rate of body weight gain by 7.6 months [Prior and Laster 1979]. Thus, biological changes in the mother during the most active growth of the fetus were involved in fetal programming induced by prenatal stress, which altered subsequent milk yield of offspring.

Maternal HS exposure during calving as well as at one, two or three months before calving did not influence subsequent cumulative days in milk, total milk yield (sum of three lactations), and percentage of extended lactations (>500 days) during three consecutive lactations (Tab. 2). This is contrary to observations of Laporta *et al*. [2018], who reported that granddaughters born to late gestation heat-stressed dams produced less energy-corrected milk in their second lactation compared with granddaughters born to cows without heat stress during the dry period. This discrepancy appears to be due to the extended lactations practiced in the present study, prolonging the cumulative days in milk and milk yield, which completely compensated for the milk reduction by IUHS, by the additional milk yield per lactation. The reduction in milk yield in IUHS cows likely resulted from irregular mammary morphology as a result of alterations

Table 2. The effect of temperature-humidity index (THI) experienced by first-calf Holstein heifers at delivery and previous to delivery (in utero) on the subsequent cumulative total milk yield (kg) during three consecutive lactations. Values are means (standard deviations in parenthesis)

Variables	THI at calving	THI one month before calving	THI two months before calving	THI three months before calving
Days in milk, sum 3 lact (days)				
THI <76	1249 (191)	1243 (1870	1250 (193)	1254 (197)
THI 76-83	1248 (199)	1254 (201)	1250 (197)	1249 (194)
THI >83	1259 (196)	1257 (189)	1257 (192)	1255 (192)
Total milk yield, sum 3 lact (kg)				
THI <76	37497 (5751)	37357 (5681)	37524 (5804)	37632 (5937)
THI 76-83	37449 (5987)	37655 (6022)	37512 (5919)	37497 (5844)
THI >83	37772 (5879)	37639 (5734)	37712 (5760)	37626 (5781)
Lactations >500 d (%)[1]				
THI <76	12 (272/2337)	11 (134/1248)	12 (137/1196)	13 (167/1305)
THI 76-83	13 (204/1557)	13 (326/2544)	12 (309/2523)	11 (256/2248)
THI >83	11 (157/1384)	12 (173/1486)	12 (187/1559)	12 (210/1725)

lact = lactation.

[1] Values in parenthesis are number of cows with extended lactations/total.

in the development of the fetal mammary gland [Skibiel *et al*. 2018]. However, the extended lactations of cows in the present study possibly stimulated more renewal of mammary epithelial cells throughout lactation, which would result in higher secretory activity. This is so because the absence of pregnancy during lactation suppresses secretion of estrogens, thus increasing milk yield and preventing the increased mammary epithelial cells loss [Herve *et al*. 2016].

Conclusion

These data provide compelling support for the view that prenatal heat stress, particularly during the eighth and ninth months of pregnancy, can have long-lasting effects on milk yield at maturity of in utero heat-stressed heifers. However, the extent of this milk loss due to late gestation heat stress is less prononunced and disappears over three consecutive lactations. Thus, under the present conditions of exposure to heat stress for most of the year, the reduced milk yield caused by in utero heat stress may be completely compensated by improved health and the adoption of extended lactations, which would increase cow longevity and cumulative milk yield.

REFERENCES

1. DAHL G.E., TAO S., MONTEIRO A.P.A., 2016 – Effects of late-gestation heat stress on immunity and performance of calves. ***Journal of Dairy Science*** 99, 3193-3198.
2. DE VRIJER B., REGNAULT T.R., WILKENING R.B., MESCHIA G., BATTAGLIA F.C., 2004 – Placental uptake and transport of ACP, a neutral nonmetabolizable amino acid, in an ovine model of fetal growth restriction. ***American Journal of Physiology: Endocrinology*** and ***Metabolism*** 287, E1114-1124.

3. 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? ***Journal of Dairy Science*** 92, 109-116.
4. DIMASUAY K.G., BOEUF P., POWELL T.L., JANSSON T., 2016 – Placental responses to changes in the maternal environment determine fetal growth. ***Frontiers in Physiology*** 7, 12.
5. DREILING C.E., CARMAN F.S., BROWN D.E., 1991 – Maternal endocrine and fetal metabolic responses to heat stress. ***Journal of Dairy Science*** 74, 312-327.
6. FOWDEN A.L., GIUSSANI D.A., FORHEAD A.J., 2006 – Intrauterine programming of physiological systems: causes and consequences. ***Physiology*** [Bethesda] 21, 29-37.
7. HAGEN A.S., ORBUS R.J., WILKENING R.B., REGNAULT T.R., ANTHONY R.V., 2005 – Placental expression of angiopoietin-1, angiopoietin-2 and tie-2 during placental development in an ovine model of placental insufficiency-fetal growth restriction. ***Pediatric Research*** 58, 1228-1232.
8. HERVE L., QUESNEL H., LOLLIVIER V., BOUTINAUD M., 2016 – Regulation of cell number in the mammary gland by controlling the exfoliation process in milk in ruminants. ***Journal of Dairy Science*** 99, 854-863
9. JEELANI R., KONWAR D., KHAN A., KUMAR D., CHAKRABORTY D., BRAHMA B., 2019 – Reassessment of temperature-humidity index for measuring heat stress in crossbred dairy cattle of a sub-tropical region. ***Journal of Thermal Biology*** 82, 99-106
10. JENSEN PEÑA C., MONK C., CHAMPAGNE F.A., 2012 – Epigenetic effects of prenatal stress on 11beta-hydroxysteroid dehydrogenase-2 in the placenta and fetal brain. ***PLOS ONE*** 7, e39791.
11. LAPORTA J., FERREIRA F.C., DADO-SENN B., DE VRIES A., DAHL G.E., 2018 – Dry period heat stress reduces dam, daughter, and grand-daughter productivity ***Journal of Dairy Science*** 101[Suppl.2], 151. [Abstr.]
12. LIMESAND S.W., CAMACHO L.E., KELLY A.C., ANTOLIC A.T., 2018 – Impact of thermal stress on placental function and fetal physiology. ***Animal Reproduction*** 15, 886-898.
13. LIMESAND S.W., REGNAULT T.R., HAY W.W. JR., 2004. Characterization of glucose transporter 8 [GLUT8] in the ovine placenta of normal and growth restricted fetuses. ***Placenta*** 25, 70-77.
14. LÓPEZ E., MELLADO M., MARTÍNEZ A.M., VÉLIZ F.G., GARCÍA J.E. DE SANTIAGO A., CARRILLO E., 2018 – Stress-related hormonal alterations, growth and pelleted starter intake in pre-weaning Holstein calves in response to thermal stress. ***International Journal of Biometeorology*** 62, 493-500.
15. MACKO A.R., YATES D.T., CHEN X., GREEN A.S., KELLY A.C., BROWN L.D., LIMESAND S.W., 2013 – Elevated plasma norepinephrine inhibits insulin secretion, but adrenergic blockade reveals enhanced b-cell responsiveness in an ovine model of placental insufficiency at 0.7 of gestation. ***Journal of Developmental Origins of Health and Disease*** 4, 402-410.
16. MADER T.L., 2003 – Environmental stress in confined beef cattle. ***Journal of Animal Science*** 81E-Suppl., E110-E119.
17. MCCRABB G.J., MCDONALD B.J., HENNOSTE L.M., 1993 – Lamb birthweight in sheep differently acclimatized to a hot environment. ***Australian Journal of Agricultural Research*** 44, 933-943.
18. MONTEIRO A.P.A., TAO S., THOMPSON I.M.T., DAHL G.E., 2016b – In utero heat stress decreases calf survival and performance through the first lactation. ***Journal of Dairy Science*** 99, 8443-8450.
19. MONTEIRO A.P.A., GUO J.R., WENG X.S., AHMED B.M., HAYEN M.J., DAHL G.E., BERNARD J.K., TAO S., 2016a – Effect of maternal heat stress during the dry period on growth and metabolism of calves. ***Journal of Dairy Science*** 99, 3896-3907.
20. NATIONAL RESEARCH COUNCIL, 2001 – Nutrient requirements of dairy cattle. 7th rev. ed. Washington: National Academy Press.

21. PRIOR R.L., LASTER D.B., 1979 – Development of the bovine fetus. ***Journal of Animal Science*** 48, 1546-1553.
22. REGNAULT T.R., ORBUS R.J., DE VRIJER B., DAVIDSEN M.L., GALAN H.L., WILKENING R.B., ANTHONY R.V. 2002 – Placental expression of VEGF, PlGF and their receptors in a model of placental insufficiency-intrauterine growth restriction [PI-IUGR]. ***Placenta*** 23, 132-144.
23. REYNOLDS L.P., BOROWICZ P.P., CATON J.S., CROUSE M.S., DAHLEN C.R., WARD A.K., 2019 – Developmental programming of fetal growth and development. ***Veterinary Clinics of North America*: *Food Animal Practice*** 35, 229-247.
24. ROZANCE P.J., ZASTOUPIL L., WESOLOWSKI S.R., GOLDSTROHM D.A., STRAHAN B., CREE-GREEN M., SHEFFIELD-MOORE M., MESCHIA G., HAY W.W. JR, WILKENING R.B., BROWN L.D., 2018 – Skeletal muscle protein accretion rates and hind limb growth are reduced in late gestation intrauterine growth-restricted fetal sheep. ***Journal of Physiology*** 596, 67-82.
25. SILVA L.F., VANDEHAAR M.J., WHITLOCK B.K., RADCLIFF R.P., TUCKER H.A., 2002 – Short communication: relationship between body growth and mammary development in dairy heifers. ***Journal of Dairy Science*** 85, 2600-2602.
26. SINCLAIR K.D., RUTHERFORD K.M.D., WALLACE J.M., BRAMELD J.M., STÖGER R., ALBERIO R., SWEETMAN D., GARDNER D.S., PERRY V.E., ADAM C.L., ASHWORTH C.J., ROBINSON J.E., DWYER C.M., 2016 – Epigenetics and developmental programming of welfare and production traits in farm animals. ***Reproduction, Fertility and Development*** 28, 1443-1478.
27. SKIBIEL A.L., DADO-SENN B., FABRIS T.F., DAHL G.E., LAPORTA J., 2018 – In utero exposure to thermal stress has longterm effects on mammary gland microstructure and function in dairy cattle. ***PLoS ONE*** 13:e0206046
28. TAO S., DAHL G.E., 2013 – Invited review: heat stress effects during late gestation on dry cows and their calves. ***Journal of Dairy Science*** 96, 4079-4093. 12.
29. 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. ***Journal of Dairy Science*** 95, 7128-7136.
30. TAO, S., DAHL, G.E., LAPORTA, J., BERNARD, J.K., ORELLANA RIVAS, R.M., MARINS, T.N., 2019 – Physiology Symposium: Effects of heat stress during late gestation on the dam and its calf12. ***Journal of Animal Science*** 97, 2245-2257.
31. THUREEN P.J., TREMBLER K.A., MESCHIA G., MAKOWSKI E.L., WILKENING R.B., 1992 – Placental glucose transport in heat-induced fetal growth retardation. ***American Journal of Physiology*** 263, R578-R585.
32. WALLACE J.M., REGNAULT T.R., LIMESAND S.W., HAY W.W. JR, ANTHONY R.V., 2005 – Investigating the causes of low birth weight in contrasting ovine paradigms. ***Journal of Physiology*** 565, 19-26.

Copyright of Animal Science Papers & Reports is the property of Institute of Genetics & Animal Breeding of the Polish Academy of Sciences and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

5.2 Effects of in utero heat stress on subsequent reproduction performance of first-calf Holstein heifers

Spanish Journal of Agricultural Research
18 (2), e0404, 9 pages (2020)
eISSN: 2171-9292
https://doi.org/10.5424/sjar/2020182-15721
Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria (INIA)

RESEARCH ARTICLE

OPEN ACCESS

Effects of *in utero* heat stress on subsequent reproduction performance of first-calf Holstein heifers

María I. Chavez (Chavez, MI)[1], José E. García (García, JE)[2], Francisco G. Véliz (Véliz, FG)[1], Leticia R. Gaytán (Gaytán, LR)[1], Ángeles de Santiago (de Santiago, A)[1] and Miguel Mellado (Mellado, M)[2]

[1] *Autonomous Agrarian University Antonio Narro, Dept. Veterinary Science, Torreon, Coahuila, 27054 Mexico.*
[2] *Autonomous Agrarian University Antonio Narro, Dept. Animal Nutrition, Saltillo, Coahuila, 25315 Mexico.*

Abstract

Aim of study: To determine the reproductive performance of heifers gestated under maternal conditions of heat stress in late gestation.

Area of study: Northern Mexico (25° 32' N, 103° 23' W).

Material and methods: The study included reproductive records of 4976 first-calf Holstein heifers in a hot environment.

Main results: Heifers born to cows experiencing no heat stress three months before parturition but with a THI >83 at calving were older (p<0.05) at first calving (743 ± 67 *vs.* 729 ± 55 days) than heifers gestated under maternal conditions of heat stress. A two-fold increase (p<0.01) in pregnancy rate occurred in heifers gestated under maternal conditions of no heat stress during two or three months before pregnancy and no heat stress at parturition, compared with heifers gestated under maternal conditions of no heat stress. Overall, across *in utero* heat stress one, two or three months before calving, pregnancy rate to all services was higher (p<0.05) for first-calf heifers gestated under maternal conditions of no heat stress during delivery, compared with heifers gestated under maternal conditions of heat stress (66.7 *vs.* 51.1%). Median days for getting pregnant was higher (140 d) for heifers whose dams were exposed to THI >83 at calving than heifers whose mothers were exposed to <76 or 76-83 (117 and 114 d) at calving.

Research highlights: These data suggest that in utero heat stress during the last three months of gestation negatively affects the reproductive performance of first-calf Holstein heifers.

Additional keywords: conception rate; twining rate; services per pregnancy; age at first calving; fetal losses.

Abbreviations used: AI (artificial insemination); AFC (age at first calving); BCS (body condition score); DM (dry matter); FTAI (fixed-time artificial insemination); IgG (immunoglobulin G); THI (temperature-humidity index).

Authors' contributions: Data acquisition: MIC, LG. Study design and drafted the manuscript: MM. Analyzed the results: JEG, MM.

Citation: Chavez, MI; García, JE; Véliz, FG; Gaytán, LR; de Santiago, A; Mellado, M (2020). Effects of *in utero* heat stress on subsequent reproduction performance of first-calf Holstein heifers. Spanish Journal of Agricultural Research, Volume 18, Issue 2, e0404. https://doi.org/10.5424/sjar/2020182-15721

Received: 13 Sep 2019 **Accepted:** 18 Jun 2020

Copyright © 2020 INIA. This is an open access article distributed under the terms of the Creative Commons Attribution 4.0 International (CC-by 4.0) License.

Funding: The authors received no specific funding for this work

Competing interests: All authors declare that there are no actual or potential conflicts of interest among the authors and other people or organizations that could inappropriately bias their work.

Correspondence should be addressed to Miguel Mellado: mmellbosq@yahoo.com

Introduction

High body temperature during late pregnancy causes distresses in the intrauterine environment, which can induce functional alterations in the fetus that could persist at maturity (Skibiel *et al.*, 2018). A positive consequence of maternal heat stress during late gestation on calf function during postnatal life is the enhancement thermo-tolerance at maturity by increasing the ability to dissipate body heat which reduces their core body temperature, which is reflected in lower sweating rate and rectal temperature when exposed to high ambient temperatures (Ahmed *et al.*, 2017).

However, other potential physiological processes which alter fetal programming in cows born from heat-stressed cows have a detrimental effect on productive and reproductive response in dairy cows. Lower body weight at 12 months of age has been observed *in utero* heat-stressed cows, although no difference in this trait at maturity was detected, but the productive life of these cows in the herd was reduced (Monteiro *et al.*, 2016). Also, maternal heat stress in late gestation depress birth weight of calf at parturition and hampers mammary growth and the absorption of IgG, which lower serum IgG concentration in young calves (Tao *et al.,* 2012, Tao & Dahl, 2013; Monteiro *et al.,* 2014; Laporta *et al.,* 2017).

While the negative effects of *in utero* heat stress on birth weight, weaning weight, passive immune transfer and milk yield in dairy cattle have been well documented, information on the exposure of developing fetuses to heat stress *in utero* in late gestation on subsequent reproductive performance in dairy cattle is incomplete. Likewise, the stages of pregnancy in which heat stress has the most adverse effects on fetuses have not been well established. Therefore, this study was carried out to determine if bovine fetal exposure to heat stress during the last three months of pregnancy negatively alters the subsequent reproductive performance of these animals. It was hypothesized that first-calf heifers born to cows exposed to heat stress during late gestation would decrease reproductive performance by increasing age at first calving, decreasing pregnancy rate, and requiring greater services per pregnancy.

Material and methods

Animal management and facilities

The experimental procedures and animal care conditions were approved by the Ethics Committee of the Research Department of the Autonomous Agrarian University Antonio Narro. This study included 4976 first-lactation Holstein heifers from a large commercial dairy operation in a hot arid environment (26° N, elevation 1140 m, mean annual rainfall 230 mm, mean annual temperature 23.7 ºC). The dairy operation consisted of approximately 3000 lactating Holstein cows housed in open-dirt pens equipped with a fixed metal framework shade in the center of pens that provided 4 m^2 of shade per cow. Shades also covered feed alleys. The study took place from 2015 to 2018.

All diets were offered as total mixed rations (49% forage and 51% concentrate; DM basis) and were served at 07:00 and 14:00 h. Feed was offered calculating 35 g refusals for each kg offered. Diets were formulated to meet the net energy requirements (1.62 Mcal/kg net energy of lactation and 18% crude protein) for lactating Holstein cows weighing 650 kg and producing 42 kg of milk containing 35 g/kg of fat and 30 g/kg of protein when consuming 24 kg/d of DM (NRC, 2001). Cows in this study were born to dams not exposed to heat stress (temperature-humidity index, THI <76), exposed to moderate heat stress (THI 76-83), or exposed to high heat stress (THI >83) during one, two or three months before delivery and at parturition.

The THI value of 76 was chosen because it was deemed as the upper critical temperature, as hyperthermia (based on increased rectal temperature) would be expected at THI above this value (Dikmen & Hansen, 2009). Kaufman *et al.* (2018) found that THI of 76.4 practically did not alter rectal temperature of Holstein cows. It is worth mentioning that the upper critical THI used in the current study is higher than values of 72 for dairy cows in Georgia and 74 for cows in Arizona (Bohmanova *et al.,* 2007), although these values derived from studies relating THI with losses in milk production.

Reproductive management

All cows in this herd were vaccinated against diseases impairing fertility, such as brucellosis (*Brucella abortus* strain RB51 vaccine), infectious bovine rhinotracheitis, bovine respiratory syncytial virus, bovine viral diarrhea, para-influenza, *Campylobacter fetus* and leptospirosis (5-serovars). Herd veterinarians examined fresh cows to identify and treat cows with postpartum infection and inflammation in the reproductive tract such as retained placenta, metritis, and clinical endometritis.

Cows became eligible for artificial insemination (AI) after 50 days postpartum. Technicians kept trying to achieve pregnancy until 360 days in milk or when milk yield dropped below 25 kg/d; this practice leads to more than 12 services per cow in some animals, and consequently to extended lactations (>500 days). First-calf heifers not pregnant around 200 post-partum and with more than 3 services were submitted for fixed-time artificial insemination (FTAI) using the Ovsynch protocol.

Nonpregnant first-calve heifers were observed for estrus three times a day, for approximately 30 min. Estrus detection was reinforced with the aid of pedometers, and AI was conducted based on visual observation of estrous behavior, according to the a.m. - p.m. guideline. Commercial non-sexed frozen-thawed semen from multiple sires was used across all months of the year. Pregnancy was detected by palpation of the uterus per rectum about 45 days post-AI.

Data collection

Data from reproductive examinations and AI were collected by the herd veterinarians during daily herd health revision for cows included in this study. For each animal, the following variables were recorded: age at first calving (AFC), occurrence of dystocia (prolonged assisted extraction), retained fetal membranes (retained fetal membranes for more than 24 h), occurrence of twin pregnancies, metritis, premature calves (<265 days, as determined by AI records) and fetal losses (expulsion of a fetus or presence of extraembryonic membranes and vaginal discharges; return to service after being confirmed pregnant). Also, pregnancy rate at first service, pregnancy rate to all services, and time from calving to pregnancy were recorded. Cows experiencing calving-related disorders were appropriately treated by the herd veterinarians, so that they were healthy by the time the first breeding postpartum occurred. Body condition scores (BCS) were determined at calving using the scoring system suggested by Edmonson *et al.* (1989).

Additional variables were THI, the day dams delivered the first-calf heifers included in the study, THI one, two or three months before calves were born. Climatic data were obtained from a meteorological station situated 3 km away from the dairy operation for the duration of the study. Information recorded was daily maximum temperatures (mercury thermometer under full shade) and relative humidity. The following equation was used for calculation of THI (highest daily temperature in Celsius degrees; RH refers to maximum relative humidity; Mader *et al.* 2006):

$$\text{THI} = (0.8 \times \text{temperature}) + (\%\text{RH}/100) \times (\text{temperature} - 14.4)] + 46.4$$

Statistical analyses

To analyze the combined effect of maximum ambient temperature and humidity contributing to pregnancy (binary outcome), fetal losses, twin births, premature births and dystocic parturition, a logistic regression model of SAS (SAS Inst. Inc., Cary, NC, USA) was used. The model included the following potentially explanatory variables of interest: THI when first-calf heifers were born, THI one, two or three months before the first-calf heifers were born and one-way interactions. Interactions were only retained if significant at the 0.15 level. The SAS model included fixed effects of THI groups with first-calf heifers within the THI groups as a random effect. Covariates used in the model included BCS at calving and year of breeding.

AFC was analyzed using the MIXED procedure of SAS. The model included the main effects of THI at various stages of fetal life and one-way interactions; year and BCS at calving were included as covariates. The number of services per pregnancy was evaluated by the bivariate Wilcoxon rank-sum test (non-parametric; proc npar1way of SAS). The effect of THI at various periods in late gestation on the interval from calving to pregnancy was analyzed using survival plots generated by Kaplan-Meier survival analysis performed with Statgraphics Centurion version XVII software (Statgraphics Technol., Inc., The Plains, Virginia). The final models were assessed for collinearity using the variance inflation factor (VIF option) in SAS; collinearity did not exist. Statistical significance was set at $p<0.05$.

Results

The percentage of fetal losses (16%), twining rate (0.7%), dystocic parturition (10%), and premature calves (5%) were not affected ($p>0.10$) by the THI prevailing during the last three months of the uterine life of first-calf heifers included in this study. THI when calves were born affected ($p<0.05$) AFC of these first-calf heifers. THI one, two, or three months previous to delivery did not affect this trait. There was a THI at parturition by THI one month before calving interaction ($p<0.05$). When heat stress was moderate or high for two months previous to parturition, the age at first calving was shorter ($p<0.05$) in cows born to dams not exposed to heat stress the day of parturition (Table 1). Likewise, heifers born to cows not exposed (THI <76) or exposed to severe heat stress (THI>83) three months before parturition but with no heat stress at parturition of their dams presented shorter AFC than heifers whose mothers experienced mild or severe heat stress at parturition.

Pregnancy rates at first service are summarized in Table 2. Pregnancy rate at first service was severely depressed ($p<0.05$) in first-calf heifers born to dams exposed to moderate (THI 76-83) heat stress one month before calving and severe heat stress (THI >83) the day of parturition. Regardless of the magnitude of heat stress exposure of dams in late gestation, first-calf heifers born to dams exposed to heat stress the day of calving presented a marked reduction in first-service pregnancy rates compared with heifers born to dams not exposed to severe heat stress. There was a THI at calving of dams by THI one month before calving interaction ($p<0.05$).

Pregnancy rates to all services are summarized in Table 3. THI at calving and during the first, second, and third month before parturition of dams affected ($p<0.01$) this reproductive variable. THI at calving of dams × THI one month before calving was an interaction term significantly associated with pregnancy rates to all services. Overall, across THI one, two or three months before calving of dams, pregnancy rate was depressed ($p<0.05$) when parturition of their dams coincided with severe heat stress.

The number of services per pregnancy confirmed at d 45 after the last AI was smaller in first-calf heifers from dams experiencing moderate and severe heat stress one, two and three months before calving and THI 76-83 at

Table 1. The effect of the temperature-humidity index (THI) experienced by first-calf Holstein heifers at delivery and previous to delivery *(in utero)* on the subsequent age at first calving in a hot environment (mean annual temperature 23.7 °C).

Variable	**THI 1-month before delivery**		
	<76	76-83	>83
THI at delivery <76[1]	732 ± 55	737 ± 61	740 ± 68
THI at delivery 76-83	737 ± 56	740 ± 60	740 ± 62
THI at delivery >83	737 ± 64	746 ± 66	741 ± 62
	THI 2-months before delivery		
	<76	76-83	>83
THI at delivery <76	732 ± 58	736 ± 57^{a}	733 ± 57^{a}
THI at delivery 76-83	736 ± 57	738 ± 63^{ab}	745 ± 63^{b}
THI at delivery >83	739 ± 64	745 ± 65^{b}	741 ± 61^{b}
	THI 3-months before delivery		
	<76	76-83	>83
THI at delivery <76	729 ± 55^{a}	735 ± 59	735 ± 58^{a}
THI at delivery 76-83	735 ± 56^{ab}	737 ± 58	747 ± 65^{b}
THI at delivery >83	743 ± 67^{b}	742 ± 62	742 ± 62^{ab}

[1]THI at parturition × THI one month before calving interaction ($p<0.05$). ^{ab}Within groups, values in columns with different superscript letters differ ($p<0.05$).

Table 2. Pregnancy rate at first service of first-calf Holstein heifers gestated under maternal conditions of no heat stress (temperature-humidity index; THI<76), moderate heat stress (THI 76-83), or severe heat stress (THI>83) at delivery and one, two or three months before calving in a hot environment.

Variable	**THI 1-month before delivery**		
	<76	76-83	>83
THI at delivery <76 [1]	149/1130 (13.2)^{a}	37/343 (10.8)^{a}	9/105 (8.6)^{a}
THI at delivery 76-83	46/477 (9.6)^{b}	118/909 (13.0)^{a}	73/651 (11.2)^{b}
THI at delivery >83	10/85 (11.8)^{a}	28/416 (6.7)^{b}	71/860 (8.3)^{a}
	THI 2-months before delivery		
	<76	76-83	>83
THI at delivery <76	99/840 (11.8)^{a}	61/ 437 (14.0)^{a}	35/301 (11.6)^{a}
THI at delivery 76-83	104/874 (11.9)^{a}	57/449 (12.7)^{a}	76/714 (10.6)^{a}
THI at delivery >83	13/233 (5.6)^{b}	48/520 (9.23)^{b}	48/608 (7.9)^{b}
	THI 3-months before delivery		
	<76	76-83	>83
THI at delivery <76	51/386 (13.2)^{a}	77/693 (11.1)a	67/499 (13.4)^{a}
THI at delivery 76-83	87/780 (11.2)^{a}	75/567 (13.2)a	75/690 (10.9)^{a}
THI at delivery >83	18/300 (6.0$^{)b}$	57/597 (9.6)b	34/464 (7.3)^{b}

[1]THI at calving of dams × THI one month before calving interaction ($p<0.05$). ^{ab}Within groups, values in columns with different superscript letters differ ($p<0.05$).

Table 3. Pregnancy rate to all services of first-calf Holstein heifers gestated under maternal conditions of no heat stress (temperature-humidity index; THI<76), moderate heat stress (THI 76-83), or severe heat stress (THI >83) at delivery and one, two or three months before calving in a hot environment.

Variable	THI 1-month before delivery		
	<76	76-83	>83
THI at delivery <76[1]	787/1130 (69.7)^{a}	223/343 (65.0)^{a}	42/105 (40.0)^{a}
THI at delivery 76-83	292/477 (61.2)^{b}	565/909 (62.2)^{a}	378/651 (58.1)^{b}
THI at delivery >83	30/85 (35.3)^{c}	212/416 (51.0)^{b}	453/860 (52.7)^{c}
	THI 2-months before delivery		
	<76	76-83	>83
THI at delivery <76	585/840 (69.6)^{a}	292/437 (66.8)^{a}	175/301 (58.1)
THI at delivery 76-83	293/ 581 (66.5)^{a}	260/449 (57.9)^{b}	394/714 (55.2)
THI at delivery >83	103/233 (44.2)^{b}	276/520 (53.1)^{b}	316/608 (52.0)
	THI 3-months before delivery		
	<76	76-83	>83
THI at delivery <76	290/386 (75.1)^{a}	480/693 (63.3)^{a}	282/499 (56.5)^{a}
THI at delivery 76-83	537/780 (68.9)^{b}	335/567 (59.1)^{b}	363/690 (52.6)^{a}
THI at delivery >83	158/300 (52.7)^{c}	317/597 (53.1)^{c}	220/464 (47.4)^{b}

[1] THI at calving of dams × THI one month before calving interaction ($p<0.05$). ^{ab}Values in rows with different superscript letters differ ($p<0.05$).

Table 4. Services per conception in first-calf Holstein heifers gestated under maternal conditions of no heat stress (temperature-humidity index; THI<76) moderate heat stress (THI 76-83) or severe heat stress (THI>83) at delivery and one, two or three months before calving in a hot environment.

Variable	THI 1-month before delivery		
	<76	76-83	>83
THI at delivery <76[1]	4.6 ± 3.3^{a}	5.4 ± 3.7^{a}	4.7± 3.8^{a}
THI at delivery 76-83	4.5 ± 3.1^{a}	4.2 ± 3.1^{b}	4.2 ± 3.3^{b}
THI at delivery >83	3.3 ± 3.0^{b}	4.6 ± 3.1^{b}	5.0 ± 3.6^{a}
	THI 2-months before delivery		
	<76	76-83	>83
THI at delivery <76	4.8 ± 3.4	4.8 ± 3.4	5.0 ± 3.7^{a}
THI at delivery 76-83	4.3 ± 3.1	4.3 ± 3.5	4.2 ± 3.1^{b}
THI at delivery >83	4.7 ± 3.2	4.8 ± 3.5	4.9 ± 3.4^{a}
	THI 3-months before delivery		
	<76	76-83	>83
THI at delivery <76	4.7 ± 3.5^{ab}	5.0 ± 3.4^{a}	4.5 ± 3.5^{ab}
THI at delivery 76-83	4.3 ± 3.1^{a}	4.1 ± 3.2^{b}	4.2 ± 3.3^{a}
THI at delivery >83	5.0 ± 3.3^{b}	4.8 ± 3.5^{a}	4.6 ± 3.4^{b}

^{ab}Values in rows with different superscript letters differ ($p<0.05$).

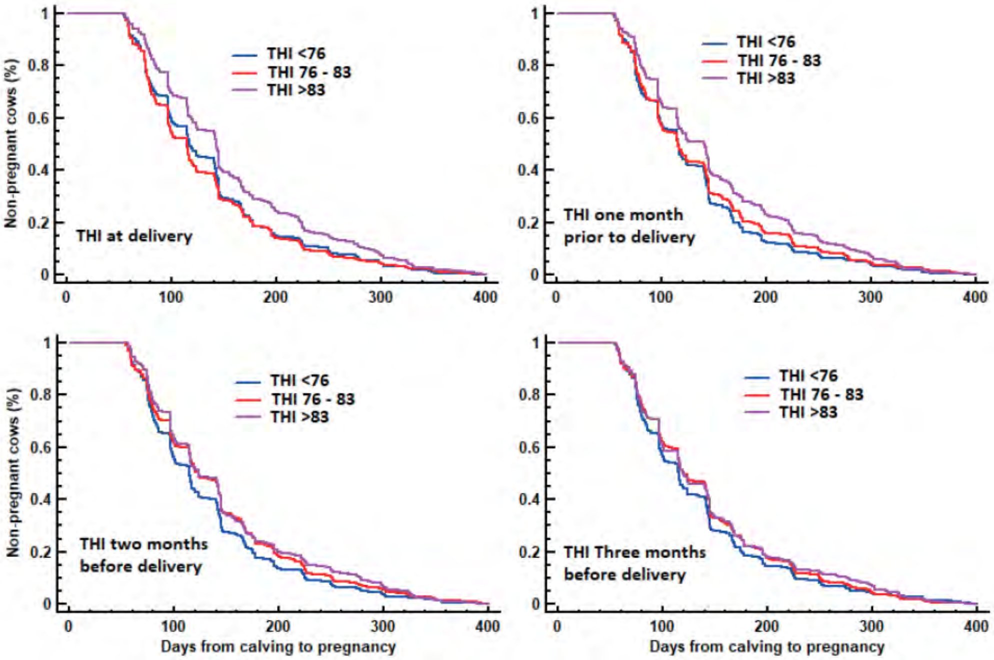

Figure 1. Kaplan-Meier survival curves for the proportion of first-calf heifers gestated under maternal conditions of heat stress in late gestation. Days open were lower for heifers whose dams were exposed to THI <76 and those born at 76-83 and >83 three months before calving (p<0.001; Wilcoxon test). Median days for getting pregnant were 117, 114, and 140 for heifers whose dams were exposed to THI <76, 76-83, and >83 at calving, respectively. Median days for getting pregnant were 115, 115, and 120 for heifers whose dams were exposed to THI <76, 76-83, and >83 one month before calving, respectively. Median days for getting pregnant was 114, 119 and 117 for heifers whose dams were exposed to THI <76, 76-83 and >83 two months before calving, respectively. Median days for getting pregnant were 115, 117, and 117 for heifers whose dams were exposed to THI of <76, 76-83, and >83 three months before calving, respectively.

calving (Table 4). For first-calf heifers born to dams exposed to different THI categories at birth, one, two and three months before calving, the median days open for heifers undergoing severe heat stress *in utero* was larger (p<0.05) than that of heifers exposed to moderate or no heat stress *in utero* (Fig. 1).

Discussion

The prevalence of fetal losses in the present study corresponds well with literature reports in hot environments (Pontes *et al.*, 2015; Mellado *et al.*, 2016), but it is much higher than the 4.8-9.8%% reported for Holstein heifers in temperate climates (Ettema & Santos, 2004; Bach, 2011). This reproductive disorder was not affected by the exposure of dams to heat stress at parturition or various periods before calving. The same was true for twining rate, dystocic parturition, and premature calves. Thus, there was no evidence of the negative effects of maternal heat stress on these periparturient disorders on offspring. Dystocia, premature deliveries, stillbirths, and abortions, are linked, in part, to endocrine changes in late pregnancy which impair pregnancies (Kindahl *et al.*, 2002; Sandman *et al.*, 2003); thus, these results do not support the view that *in utero* heat stress altered hormones related to calves delivery.

An important finding of this study was that *in utero* heat stress was associated with an increase in the AFC of heifers. This response seems to be linked to the fact that *in utero* heat-stressed calves are lighter at birth compared to non-heat stressed animals (Tao *et al.*, 2012; Monteiro *et al.*, 2014). Additionally, these lighter calves born from heat-stressed cows in late pregnancy do not have any compensatory growth before puberty, as they remained smaller and lighter up to one year of age (Monteiro *et al.*, 2016). Thus, heifers suffering heat stress *in utero* apparently do not achieve rapid growth rates due to reduced fetal secretion of insulin-like growth factor-I, prolactin, and insulin (Guo *et al.*, 2016), which did not enable them to achieve breeding size earlier, lessening AFC which increases costs of raising replacement heifers (Davis Rincker *et al.*, 2011).

Overall, first-service pregnancy rate was very low in first-calf Holstein heifers (10.9%) in this hot environment, but is in line with values observed in dairy operations in this hot environment (Flores *et al.*, 2019). In temperate and tropical zones the percentage success at first service in dairy cattle has ranged between 26.7% and 49.6% in previous studies (Tillard *et al.*, 2008; Fodor *et al.*, 2018), which is much higher than the figure found in the present study, even when careful attention was given to reproductive management. This poor response is due to chronic heat stress suffered by cows in this zone which causes impaired fertilization (Sartori *et al.*, 2002; Moghaddam & Karimi, 2009), oocyte quality (Hansen, 2009; Souza-Cácares *et al.*, 2019), sperm viability and hampers embryonic development (Sakatani *et al.*, 2015; Wolfenson & Roth, 2019).

First-calf heifers born to cows with moderate (THI 76-83) and severe (THI >83) heat stress, one, two or three months before calving and severe heat stress at calving consistently presented a marked reduction in pregnancy rate at first service. The rise of internal body temperature in high-milk yielding dairy cows subjected to heat stress (Wolfenson & Roth, 2019) causes a hyperthermic uterine environment which seems to influence the phenotypic variation of offspring mediated by epigenetic modifications regulating gene expression (Skibiel *et al.*, 2018). Thus, heat stress in late pregnant Holstein cows seems to produce indirect developmental and post-natal effects in bovine offspring, adversely impacting reproduction function as evidenced by a noticeable reduction in pregnancy rate at first service. Reports suggest that epigenetic mechanisms are the link between early-life environment and fitness later in life (Gabory *et al.*, 2011; Jammes *et al.*, 2011); however, the underlying mechanisms involved in the association between the uterine environment of developing fetuses and postnatal reproductive function remain unclear.

In general, regardless of THI in the last trimester of gestation, pregnancy rate to all services was reduced in first-calf heifers gestated under maternal conditions of heat stress compared to heifers gestated with no heat stress in late gestation. The associations between late gestational exposure to heat stress and subsequent heifer reproductive function are complex and have yet to be elucidated but could be attributed, in part, to the fact that late-gestation heat stress compromises placental growth due to reductions in secretion of progesterone and placental lactogen (Bell *et al.*, 1989), which leads to fetal malnutrition, hypoxia, and fetal growth retardation (Tao & Dahl, 2013). Additionally, heat stress during late gestation depresses cell proliferation in tissues, thereby reducing cell number and restricting tissue growth (Harding & Johnson, 1995; Fowden *et al.*, 1998). *In utero* heat stress also reduces immune function and alters systemic metabolism (Flouris *et al.*, 2009; Tao *et al.*, 2012). Thus, *in utero* acute heat-stressed calves probably presented alterations in their reproductive organs which was reflected in lower reproductive capacity in adulthood. Insults during late gestation alter cell proliferation and program their morphology in postnatal life, which seems to contribute to the reduced reproductive performance of *in utero* heat-stressed heifers (Skibiel *et al.*, 2018).

Services per pregnancy in the present study were extremely high (4.6 ± 3.4; Table 4). This value was far higher than the 2.7 services per conception in Holstein herds in southeastern United States (Norman *et al.*, 2009), but is close to the mean number of services per pregnancy > 6 (Flores *et al.*, 2019) typical of dairy herds in this zone. To put this value into perspective, services per pregnancy for the whole herd (only pregnant cows) was 6.7 ± 3.4. High ambient temperature for the most of the year was responsible for the impaired reproduction in this dairy herd (Mellado *et al.*, 2013). The present study provides no clear evidence that services per pregnancies were reduced in first-calf heifers gestated under maternal conditions of heat stress (THI >83) compared to heifers born to dams not exposed to heat stress in late pregnancy. Therefore, measurement of ambient and humidity during late pregnancy in Holstein dams do not help to predict services per pregnancy in their first-calf offspring under the management and ambient conditions of the present study.

However, the calving-to-conception interval was longer in first-calf heifers gestated under maternal conditions of heat stress compared to heifers gestated with no heat stress the last trimester of pregnancy. It is worth mentioning that, the effect of *in utero* heat stress on calving-to-conception interval was not adjusted for milk yield and this variable could have an important impact on this variable. Given that animals in this dairy operation received adequate diets throughout lactation, deficient dietary protein or energy early in lactations were not responsible for this response. Intervals from calving to first ovulation neither seemed to be involved in this response as most first-calf heifers presented behavioral estrus repeatedly before getting pregnant. Thus, the greater struggle to become pregnant in first-calf heifers born to cows exposed to heat stress in late pregnancy apparently derive from induced structural and functional changes of the reproductive tract or disruption of the endocrine systems controlling the reproductive organs in the fetus that apparently persisted through adulthood (Skibiel *et al.*, 2018), and possibly hindered the production of viable ovum, its fertilization and implantation in the uterus.

These data support the hypothesis that exposure of high-milk producing dairy cows to heat stress during the third trimester of pregnancy and at calving is negatively associated with reproductive function of their first-calf heifers, but the causes of this fertility depression are unclear. Thus, intervention strategies for minimizing the duration and degree of heat stress at the end of gestation could result in offspring with enhanced reproductive ability.

References

Ahmed BMS, Younas U, Asar TO, Dikmen S, Hansen PJ, Dahl GE, 2017. Cows exposed to heat stress during fetal life exhibit improved thermal tolerance. J Anim Sci 95: 3497-3503. https://doi.org/10.2527/jas.2016.1298

Bach A, 2011. Associations between several aspects of heifer development and dairy cow survivability to second lactation. J Dairy Sci 94: 1052-1057. https://doi.org/10.3168/jds.2010-3633

Bell AW, McBride BW, Slepetis R, Early RJ, Currie WB, 1989. Chronic heat stress and prenatal development in sheep: I. Conceptus growth and maternal plasma hormones and metabolites. J Anim Sci 67: 3289-3299. https://doi.org/10.2527/jas1989.67123289x

Bohmanova J, Misztal I, Cole JB, 2007. Temperature-humidity indices as indicators of milk production losses due to heat stress. J Dairy Sci 90: 1947-1956.

Davis Rincker LE, VandeHaar MJ, Wolf CA, Liesman JS, Chapin LT, Weber Nielsen MS, 2011. Effect of intensified feeding of heifer calves on growth, pubertal age, calving age, milk yield, and economics. J Dairy Sci 94: 3554-3567. https://doi.org/10.3168/jds.2010-3923

Dikmen S, Hansen PJ, 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. https://doi.org/10.3168/jds.2008-1370

Edmonson AJ, Lean IJ, Weaver LD, Farver T, Webster G, 1989. A body condition scoring chart for Holstein dairy cows. J Dairy Sci 72: 68-78. https://doi.org/10.3168/jds.S0022-0302(89)79081-0

Ettema JF, Santos JEP, 2004. Impact of age at calving on lactation, reproduction, health, and income in first parity Holsteins on commercial farms. J Dairy Sci 87: 2730-2742. https://doi.org/10.3168/jds.S0022-0302(04)73400-1

Flores J, García JE, Mellado J, Gaytán L, De Santiago A, Mellado M, 2019. Effect of growth hormone on milk yield and reproductive performance of subfertile Holstein cows during extended lactations. Span J Agric Res 17 (1): e0403. https://doi.org/10.5424/sjar/2019171-13842

Flouris AD, Spiropoulos Y, Sakellariou GJ, Koutedakis Y, 2009. Effect of seasonal programming on fetal development and longevity: links with environmental temperature. Am J Human Biol 21: 214-216. https://doi.org/10.1002/ajhb.20818

Fodor I, Baumgartner W, Abonyi-Tóth Z, Lang Z, Ózsvári L, 2018. Associations between management practices and major reproductive parameters of Holstein-Friesian replacement heifers. Anim Reprod Sci 188: 114-122. https://doi.org/10.1016/j.anireprosci.2017.11.015

Fowden AL, Li J, Forhead AJ, 1998. Glucocorticoids and the preparation for life after birth: are there long-term consequences of the life insurance? Proc Nutr Soc 57: 113-122. https://doi.org/10.1079/PNS19980017

Gabory A, Attig L, Junien C, 2011. Developmental programming and epigenetics. Am J Clin Nutr 94: 1943S-1952S. https://doi.org/10.3945/ajcn.110.000927

Guo JR, Monteiro APA, Weng XS, Ahmed BM, Laporta J, Hayen MJ, Dahl GE, Bernard JK, Tao S, 2016. Short communication: Effect of maternal heat stress in late gestation on blood hormones and metabolites of newborn calves. J Dairy Sci 99: 6804-6807. https://doi.org/10.3168/jds.2016-11088

Hansen PJ, 2009. Effects of heat stress on mammalian reproduction. Philos Trans R Soc London B. 364: 3341-3350. https://doi.org/10.1098/rstb.2009.0131

Harding JE, Johnson B, 1995. Nutrition and fetal growth. Reprod Fert Dev 7: 538-547. https://doi.org/10.1071/RD9950539

Jammes H, Junien C, Chavatte-Palmer P, 2011. Epigenetic control of development and expression of quantitative traits. Reprod Fertil Dev 23: 64-74. https://doi.org/10.1071/RD10259

Kaufman JD, Saxton AM, Ríus AG, 2018. Short communication: Relationships among temperature-humidity index with rectal, udder surface, and vaginal temperatures in lactating dairy cows experiencing heat stress. J Dairy Sci 101: 6424-6429. https://doi.org/10.3168/jds.2017-13799

Kindahl H, Kornmatitsuk B, Königsson K, Gustafsson H, 2002. Endocrine changes in late bovine pregnancy with special emphasis on fetal well-being. Dom Anim

Endocrinol 23: 321-328. https://doi.org/10.1016/S0739-7240(02)00167-4

Laporta J, Fabris TF, Skibiel AL, Powell JL, Hayen MJ, Horvath K, Miller-Cushon EK, Dahl GE, 2017. *In utero* exposure to heat stress during late gestation has prolonged effects on the activity patterns and growth of dairy calves. J Dairy Sci 100: 1-9. https://doi.org/10.3168/jds.2016-11993

Mader TL, Davis MS, Brown-Brandl T, 2006. Environmental factors influencing heat stress in feedlot cattle. J Anim Sci 84: 712-719. https://doi.org/10.2527/2006.843712x

Mellado M, Sepulveda E, Meza-Herrera C, Veliz F, Arevalo J, Mellado J, De Santiago A, 2013. Effects of heat stress on reproductive efficiency of high yielding Holstein cows in a hot-arid environment. Rev Colomb Cienc Pec 26: 193-200.

Mellado M, López R, de Santiago A, Veliz FG, Macías-Cruz U, Avendaño-Reyes L, García JE, 2016. Climatic conditions, twining and frequency of milking as factors affecting the risk of fetal losses in high-yielding Holstein cows in a hot environment. Trop Anim Health Prod 48: 1247-1252. https://doi.org/10.1007/s11250-016-1084-8

Moghaddam A, Karimi IP, 2009. Effects of short-term cooling on pregnancy rate of dairy heifers under summer heat stress. Vet Res Commun 33: 567-575. https://doi.org/10.1007/s11259-009-9205-8

Monteiro APA, Tao S, Thompson IM, Dahl GE, 2014. Effect of heat stress during late gestation on immune function and growth performance of calves: Isolation of altered colostral and calf factors. J Dairy Sci 97: 6426-6439. https://doi.org/10.3168/jds.2013-7891

Monteiro APA, Guo JR, Weng X, Ahmed BM, Hayen MJ, Dahl GE, Bernard JK, 2016. Effect of maternal heat stress during the dry period on growth and metabolism of calves. J Dairy Sci 99: 3896-3907. https://doi.org/10.3168/jds.2015-10699

Norman HD, Wright JR, Hubbard SM, Miller RH, Hutchison JL, 2009. Reproductive status of Holstein and Jersey cows in the United States. J Dairy Sci 92: 3517-3528. https://doi.org/10.3168/jds.2008-1768

NRC, 2001. Nutrient requirements of dairy cattle, 7th rev. ed. National Research Council, National Academic Science, Washington, DC, USA.

Pontes GCS, Monteiro PLJ, Prata AB, Guardieiro MM, Pinto DAM, Fernandes GO, Wiltbank MC, Santos JE Sartori R, 2015. Effect of injectable vitamin E on incidence of retained fetal membranes and reproductive performance of dairy cows. J Dairy Sci 98: 2437-2449. https://doi.org/10.3168/jds.2014-8886

Sakatani M, Yamanaka K, Balboula AZ, Takenouchi N, Takahashi M, 2015. Heat stress during in vitro fertilization decreases fertilization success by disrupting anti-polyspermy systems of the oocytes. Mol Reprod Dev 82: 36-47. https://doi.org/10.1002/mrd.22441

Sandman CA, Glynn L, Wadhwa PD, Chicz-DeMet A, Porto M, Garite T, 2003. Maternal hypothalamic-pituitary-adrenal dysregulation during the third trimester influences human fetal responses. Dev Neurosci 25: 41-49. https://doi.org/10.1159/000071467

Sartori R, Rosa GJ, Wiltbank MC, 2002. Ovarian structures and circulating steroid in heifers and lactating cows in summer and lactating and dry cows in winter. J Dairy Sci 85: 2813-2822. https://doi.org/10.3168/jds.S0022-0302(02)74368-3

Skibiel AL, Peñagaricano F, Amorín R, Ahmed BM, Dahl GE, Laporta J, 2018. *In utero* heat stress alters the offspring epigenome. Sci Rep 8 (1): 14609. https://doi.org/10.1038/s41598-018-32975-1

Souza-Cácares MB, Fialho ALL, Silva WAL, Cardoso CJT, Pöhland R, Martins MIM, Melo-Sterza FA, 2019. Oocyte quality and heat shock proteins in oocytes from bovine breeds adapted to the tropics under different conditions of environmental thermal stress. Theriogenology 130: 103-110. https://doi.org/10.1016/j.theriogenology.2019.02.039

Tao S, Dahl GE, 2013. Invited review: Heat stress effects during late gestation on dry cows and their calves. J Dairy Sci 96: 4079-4093. https://doi.org/10.3168/jds.2012-6278

Tao S, Monteiro APA, Thompson IM, Hayen MJ, Dahl GE, 2012. Effect of late-gestation maternal heat stress on growth and immune function of dairy calves. J Dairy Sci 95: 7128-7136. https://doi.org/10.3168/jds.2012-5697

Tillard E, Humblot P, Faye B, Lecomte P, Dohoo I, Bocquier F, 2008. Postcalving factors affecting conception risk in Holstein dairy cows in tropical and subtropical conditions. Theriogenology 69: 443-457. https://doi.org/10.1016/j.theriogenology.2007.10.014

Wolfenson D, Roth Z, 2019. Impact of heat stress on cow reproduction and fertility. Anim Front 9: 32-38. https://doi.org/10.1093/af/vfy

VI. CONCLUSIÓN GENERAL

En conclusión, estos datos indican que el estrés por calor en el útero durante los últimos tres meses de gestación afecta de manera negativa el desempeño reproductivo de las vaquillas Holstein de primer parto; además, se encontró evidencia sobre la reducción de la producción de leche y la duración de la lactancia entre las vaquillas de primer parto que experimentaron estrés por calor en útero.

Sin embargo, estos efectos fueron menos pronunciados en las siguientes lactancias, por lo que los efectos sobre la producción de leche no fueron más allá del impacto inmediato en la primera lactancia.