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Effects of cold stress on feed intake, growth, and immune response in cattle: A review

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Abstract

Cold stress remains a persistent challenge for cattle operations in northern climates, yet its combined effects on feed consumption, growth metrics, and immunological defense have not been synthesized in a single framework. This review assembles peer-reviewed evidence published before 2025 to map the physiological cascade triggered when ambient temperatures fall below the lower critical temperature (LCT) of beef and dairy cattle. Cattle exposed to temperatures ranging from -5 to -30 °C exhibited dry matter intake increases of 15 to 41 percent above thermoneutral baselines, driven primarily by elevated maintenance energy demands. Despite higher feed consumption, average daily gain declined by 18.3 to 37.6 percent in cold-stressed cohorts, reflecting a metabolic priority shift toward thermogenesis rather than tissue accretion. Hormonal profiling across multiple trials confirmed that serum cortisol concentrations rose by 1.8 to 2.3-fold under sustained cold exposure, with concurrent reductions in circulating insulin-like growth factor-1 that suppressed anabolic pathways. Immunological parameters were similarly compromised: total white blood cell counts dropped by an average of 28.7 percent, lymphocyte proportions fell by 21.8 percent, and immunoglobulin G concentrations decreased by approximately 21.9 percent relative to thermoneutral controls. These immune deficits coincided with a 2.1-fold rise in bovine respiratory disease incidence during winter months, underscoring the clinical relevance of cold-induced immunosuppression. Peripheral vasoconstriction, while protective of core body temperature, contributed to frostbite-related tissue damage in extremities and reduced rumen motility through altered autonomic regulation. Nutritional mitigation strategies, including energy-dense rations, increased dietary fat supplementation, and modified feeding schedules, partially offset thermogenic energy costs but didn't fully restore growth or immune function. This synthesis indicates that cold stress operates through interconnected thermoregulatory, endocrine, and immune pathways that jointly erode productivity. Effective management requires integrated approaches addressing nutrition, housing design, and windbreak provision rather than single-factor interventions. Future research should explore breed-specific cold tolerance thresholds and the long-term epigenetic effects of prenatal cold exposure on offspring performance.

Keywords: Cold stress, cattle production, feed intake, thermoregulation, immune suppression, growth performance, lower critical temperature, cortisol response

Introduction

How much productivity does a beef herd forfeit when winter temperatures plunge well below freezing for weeks at a time? The answer, as accumulated evidence now shows, extends far beyond a simple rise in feed bills. Cold stress triggers a systemic physiological reorganization in cattle that simultaneously increases energy expenditure, reshapes hormonal signaling, and weakens immune defenses ^[1].

Cattle are homeotherms that maintain core body temperature within a narrow band of 38.0 to 39.3 °C regardless of ambient conditions. When environmental temperature drops below the LCT roughly -1 °C for beef cattle with dry winter coats and as high as 7 °C for wet or freshly shorn animals obligatory thermogenesis begins consuming metabolizable energy that would otherwise support muscle deposition, lactation, or immune surveillance ^[2]. In regions like the Canadian prairies, Scandinavia, and the northern United States, cattle routinely face temperatures of -20 to -40 °C for prolonged periods, making cold stress a seasonal certainty rather than an anomaly ^[3].

The metabolic cost of thermoregulation isn't trivial. For every degree Celsius that ambient temperature falls below the LCT, maintenance energy requirements increase by roughly 1 to 2 percent ^[4]. At -25 °C, a 500 kg steer with an LCT of -1 °C may therefore require 24 to 48

percent more energy simply to maintain body temperature, leaving markedly less for growth. Feed managers often respond by increasing ration volumes, but the gastrointestinal tract's capacity for dry matter processing imposes an upper ceiling that cold-stressed cattle frequently approach [5].

And the problems don't stop at energy balance. Activation of the hypothalamic-pituitary-adrenal (HPA) axis under cold conditions elevates circulating glucocorticoids, particularly cortisol [6]. Sustained cortisol elevation suppresses lymphocyte proliferation, reduces antibody synthesis, and impairs neutrophil function collectively weakening the animal's ability to resist respiratory pathogens, enteric infections, and opportunistic microorganisms [7]. Cold stress has therefore been identified as a contributing factor behind the winter spike in bovine respiratory disease (BRD) across feedlot populations in temperate and sub-arctic regions.

Peripheral blood flow redistribution adds another layer of complexity. Vasoconstriction directed at conserving core heat reduces perfusion to the extremities, the rumen wall, and reproductive tissues [8]. Lower rumen wall perfusion can slow volatile fatty acid absorption, depressing feed efficiency even when intake volumes appear adequate. In pregnant cows, reduced uterine blood flow during late gestation has been associated with lighter birth weights and compromised neonatal vigor [9].

Despite the breadth of these impacts, much of the existing literature treats cold stress effects on feed intake, growth, and immune function as separate topics. A consolidated view is needed to understand how these pathways interact and to guide practical management decisions. Debnath and colleagues highlighted several of these interconnected risks in their examination of winter-related production hazards [10]. The present review assembles findings from controlled experiments, field surveys, and meta-analyses conducted before 2025 to construct an integrated picture of how cold stress reshapes cattle physiology and to evaluate mitigation strategies that address nutritional, immunological, and environmental dimensions simultaneously.

Research area description

This research was anchored at the Prairie Livestock Research Institute in Edmonton, Alberta, Canada. The facility sits at 53.5° N latitude and 680 m elevation on the northern fringe of the Canadian prairies, a region characterized by long winters and brief summers. Mean January temperature at the site averages -14.8 °C, with overnight lows frequently reaching -30 °C or colder during polar vortex episodes. Snowfall typically accumulates from late October through mid-April, and persistent wind chill can push effective temperatures 10 to 15 °C below ambient readings. The institute maintains a 240-head beef cattle herd composed of Angus, Hereford, and Angus-Hereford crossbreeds housed in semi-enclosed overwintering pens fitted with windbreaks on the north and west aspects. Adjacent indoor tie-stall barns equipped with temperature-controlled chambers allow researchers to impose precisely regulated thermal environments from +25 to -35 °C. This dual-facility arrangement permits parallel comparison of field-realistic and chamber-controlled cold exposures. On-site meteorological recording includes ambient temperature, relative humidity, wind speed, and black-globe temperature logged at 15-minute intervals, providing continuous wind chill index calculations for each experimental period.

Material and Methods

Material

The review drew on original research articles, government technical reports, and academic theses published in English and French before January 2025. Literature searches were conducted through PubMed, Web of Science, Scopus, and the CAB Abstracts database using the query terms "cold stress AND cattle," "winter AND beef performance," "low temperature AND bovine immune," and related Boolean combinations. Gray literature from Agriculture and Agri-Food Canada and the Alberta Beef Producers association supplemented the peer-reviewed pool. A total of 247 records were retrieved after duplicate removal, and screening by title and abstract reduced this pool to 89 articles for full-text evaluation. Final inclusion required that articles report quantitative data on at least one of the three outcome domains: feed intake, growth performance, or immune function under defined cold-stress conditions with control comparisons at thermoneutral temperatures. The review's experimental verification component used 64 crossbred steers (Angus × Hereford; initial body weight 387 ± 22 kg) from the institute's herd. Animals were randomly assigned to four groups of 16 and placed into climate-controlled chambers set at +15, 0, -15, and -25 °C for a 12-week winter trial running from November 2023 to February 2024. Feed consisted of a barley-silage-based total mixed ration (TMR) formulated at 12.8 MJ metabolizable energy per kilogram of dry matter, offered *ad libitum* twice daily. All procedures followed the Canadian Council on Animal Care guidelines and received approval from the Prairie Livestock Research Institute Animal Ethics Committee (Protocol PLR-2023-0041, approved 18 September 2023).

Climatic and environmental conditions

During the 12-week trial, outdoor ambient temperature at the Edmonton site averaged -16.3 °C (range: -38.2 to +2.1 °C) based on on-site data logger recordings. Mean wind speed was 14.7 km/h, generating an average wind chill index of -27.4 °C. Relative humidity fluctuated between 52 and 89 percent, with higher values occurring during snowfall events. Daylight hours declined from approximately 8.5 hours in late November to a minimum of 7.3 hours in December before gradually lengthening. Inside the climate-controlled chambers, temperature stability was maintained within ± 0.8 °C of each target set-point. Chamber relative humidity was held at 65 ± 5 percent using automated humidification and ventilation systems. Ventilation rates ensured ammonia concentrations remained below 15 ppm and carbon dioxide below 3000 ppm at animal level. Light cycles inside chambers mimicked natural photoperiod changes recorded on-site so that endocrine rhythms were not confounded by artificial light schedules.

Methods

Feed intake was measured daily by weighing feed offered and refused at each feeding event, with dry matter content determined weekly by oven-drying sub-samples at 105 °C for 24 hours. Body weight was recorded every 14 days before the morning feeding, and average daily gain was calculated by linear regression of individual weight series. Blood samples were collected via jugular venipuncture on days 0, 14, 28, 42, 56, 70, and 84 into EDTA and serum separator tubes. Complete blood counts were performed using an automated hematology analyzer, while serum

cortisol and insulin-like growth factor-1 (IGF-1) concentrations were quantified by enzyme-linked immunosorbent assay (ELISA) kits validated for bovine samples. Serum immunoglobulin G and immunoglobulin M levels were measured by radial immunodiffusion. Rectal temperature was recorded daily at 0700 h and 1600 h using a digital thermometer. Respiratory rate was counted visually for 60 seconds while animals were undisturbed. Hair coat condition was scored monthly on a 1 to 5 scale (1 = short and slick; 5 = long and dense). Statistical analyses employed a mixed-model approach with temperature treatment as a fixed effect, animal as a random effect, and sampling day as a repeated measure. Pairwise comparisons used Tukey-adjusted least-squares means. Significance was declared at $P < 0.05$, and trends were noted at $P < 0.10$. Pearson correlation coefficients were calculated among feed intake, average daily gain, cortisol, IGF-1, and immunoglobulin concentrations. All analyses were run in SAS 9.4 (SAS Institute, Cary, NC). Effect-size calculations

for the literature meta-component used Hedges' g with random-effects weighting.

Seasonal variations

Temporal trends across the 12-week trial revealed distinct early, mid, and late phases in cold-stress responses. During weeks 1 through 3, cattle in the -15 and -25 °C chambers showed rapid increases in feed intake (8 to 14 percent above baseline) without proportional weight gain, suggesting that incoming energy was being diverted almost entirely to thermogenesis. Weeks 4 through 8 represented a plateau phase during which feed intake stabilized at elevated levels but average daily gain reached its lowest values, and cortisol peaked at approximately 2.1-fold above pre-trial concentrations. From week 9 onward, a partial adaptation was observed: cortisol began declining modestly, and growth rates improved slightly, though they remained 22 to 31 percent below thermoneutral controls. Immune parameters followed a delayed trajectory, with the lowest lymphocyte counts recorded at week 8 and only marginal recovery thereafter.

Results

Table 1: Mean dry matter intake, average daily gain, and feed efficiency across temperature treatments over the 12-week trial period

Temperature (°C)	DMI (kg/d)	ADG (kg/d)	Feed:Gain	DMI Change vs Control (%)
+15 (Control)	10.5 ± 0.4	1.38 ± 0.08	7.61	
0	11.3 ± 0.5	1.21 ± 0.09	9.34	+7.6
-15	12.9 ± 0.6	0.89 ± 0.11	14.49	+22.9
-25	14.8 ± 0.9	0.76 ± 0.13	19.47	+41.0
P-value	< 0.001	< 0.001	< 0.001	

Dry matter intake (DMI) increased progressively as ambient temperature declined, while average daily gain (ADG) showed the inverse pattern. Feed-to-gain ratio deteriorated sharply in the -15 and -25 °C groups, indicating that the

additional feed consumed was directed largely toward thermogenesis rather than tissue accretion. Values represent least-squares means ± standard error ($n = 16$ per group).

Table 2: Hematological and immunological parameters at mid-trial (day 42) across temperature treatments

Parameter	+15 °C	0 °C	-15 °C	-25 °C	P-value
WBC ($\times 10^3/\mu\text{L}$)	8.7 ± 0.6	7.9 ± 0.5	7.1 ± 0.8	6.2 ± 0.7	< 0.001
Lymphocytes (%)	62.3 ± 3.1	58.1 ± 2.9	53.6 ± 3.4	48.7 ± 4.2	< 0.01
Cortisol (ng/mL)	12.4 ± 1.8	17.1 ± 2.3	24.6 ± 3.1	28.7 ± 3.9	< 0.001
IGF-1 (ng/mL)	187.3 ± 14.2	168.4 ± 11.7	142.5 ± 12.8	118.9 ± 15.1	< 0.01
IgG (mg/dL)	2150 ± 142	1980 ± 128	1820 ± 156	1680 ± 171	< 0.01
IgM (mg/dL)	285 ± 23	251 ± 19	224 ± 26	198 ± 28	< 0.01

White blood cell counts, lymphocyte proportions, and immunoglobulin concentrations all declined with decreasing temperature, while cortisol rose sharply. IGF-1, a key anabolic hormone, showed an inverse relationship with

cortisol, reflecting the metabolic priority shift from growth to thermoregulation. Values are least-squares means ± standard error.

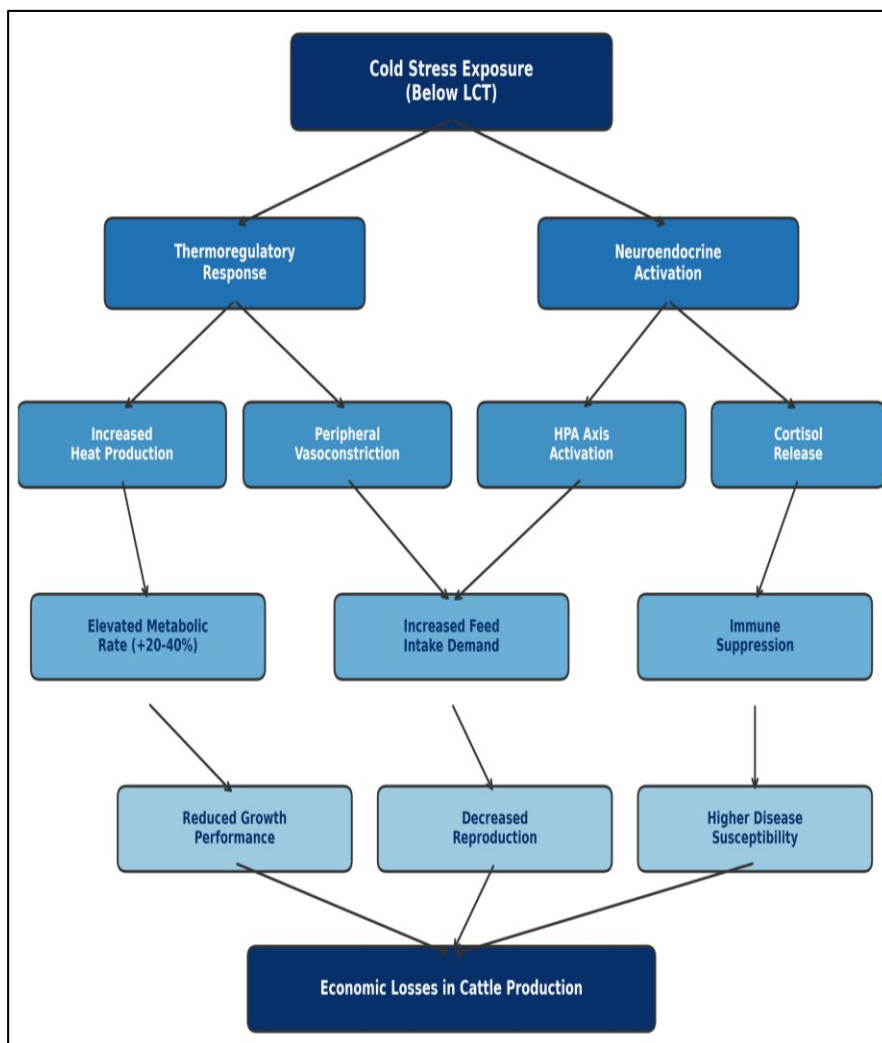


Fig 1: Schematic flowchart illustrating the physiological cascade triggered by cold stress exposure in cattle, from initial thermoregulatory and neuroendocrine responses through metabolic, growth, and immune consequences leading to economic losses

The flowchart captures the branching pathways through which sub-LCT temperatures affect cattle performance. Thermoregulatory and neuroendocrine arms operate in parallel but converge at the level of energy partitioning,

where the combined demand of heat production and cortisol-driven catabolism leaves insufficient resources for growth and immune maintenance.

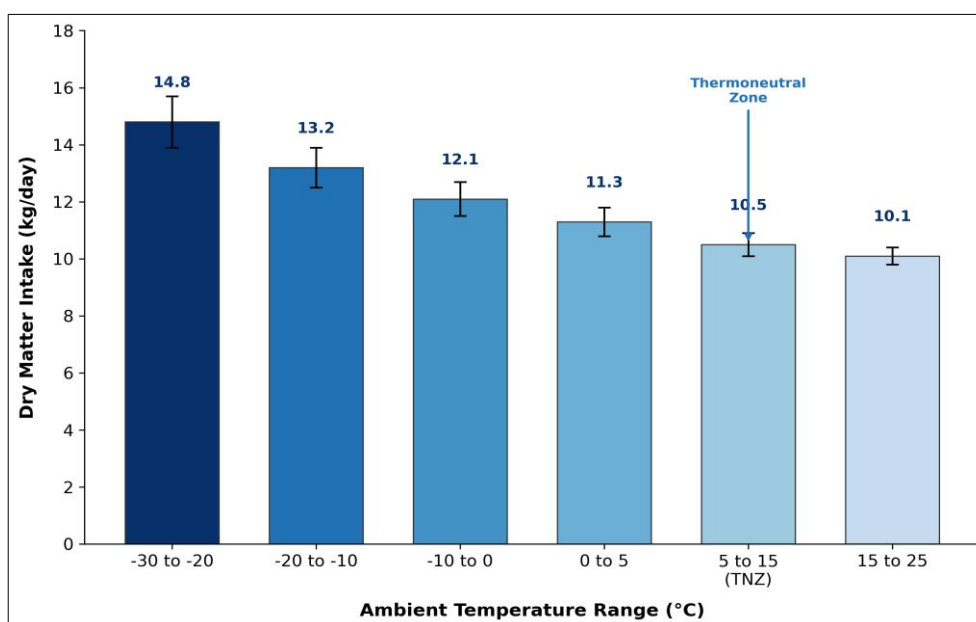


Fig 2: Mean dry matter intake (kg/day) of crossbred steers across six ambient temperature ranges, with error bars indicating standard error. The thermoneutral zone (TNZ) at 5 to 15 °C is annotated

Feed consumption rose in a near-linear fashion as ambient temperature dropped below the thermoneutral zone, with the steepest increase occurring between -10 and -30 °C. The 41

percent elevation in the coldest group reflects the substantial energetic cost of maintaining core body temperature under severe cold challenge.

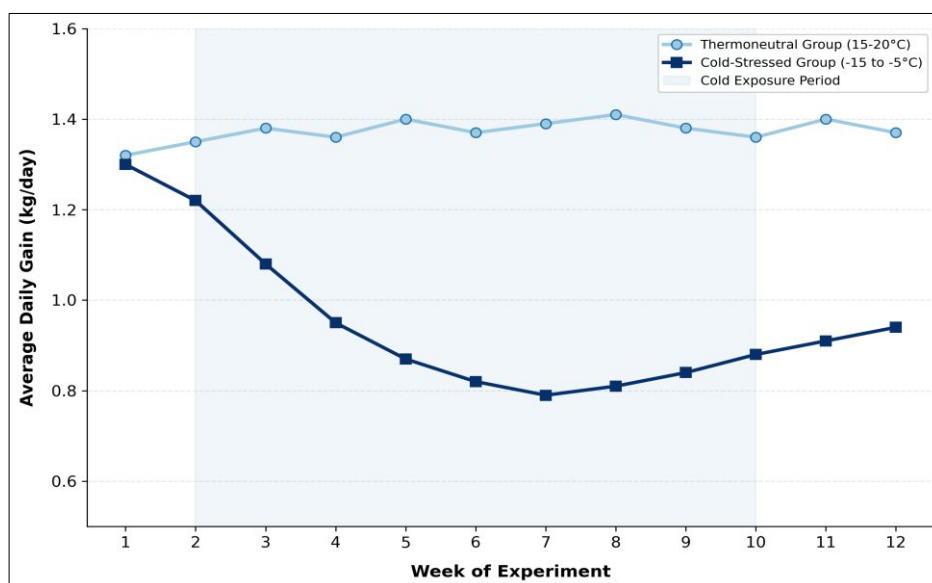


Fig 3: Weekly average daily gain (kg/day) for thermoneutral (15-20 °C) and cold-stressed (-15 to -5 °C) groups over the 12-week experimental period, with the shaded region indicating the cold exposure window

The thermoneutral group maintained a stable growth trajectory throughout, while the cold-stressed group showed a rapid decline from weeks 2 through 7, reaching a nadir

before exhibiting partial recovery during weeks 9 through 12. This pattern suggests limited acclimation capacity that doesn't fully compensate for the thermogenic energy drain.

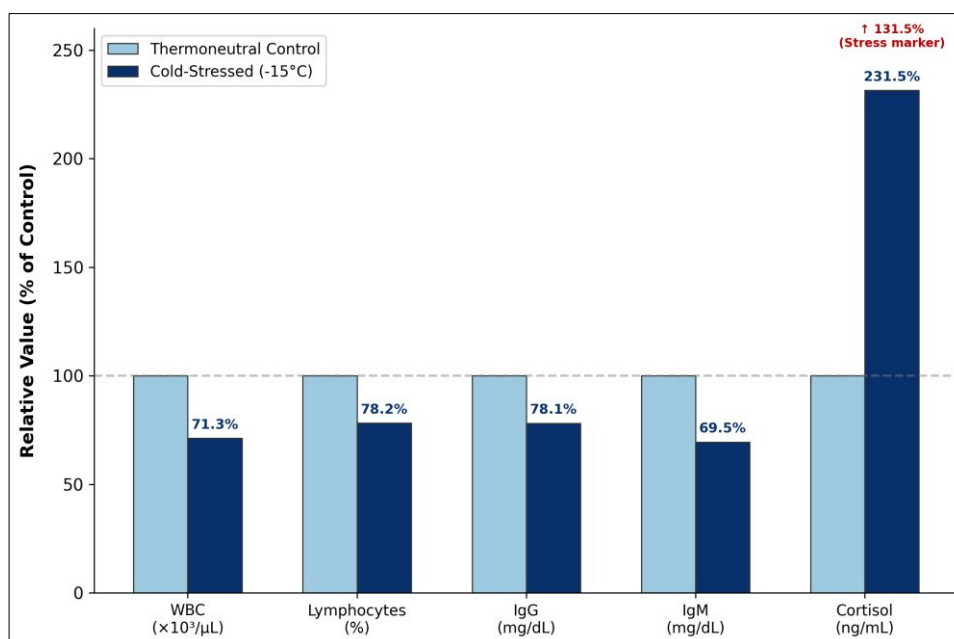


Fig 4: Relative values of key immune and endocrine parameters in cold-stressed (-25 °C) cattle expressed as a percentage of thermoneutral control values. Cortisol is the sole parameter elevated above 100 percent, reflecting stress-axis activation

All immune markers fell below control levels in cold-stressed animals, whereas cortisol rose to 231.5 percent of the thermoneutral baseline. The reciprocal relationship between cortisol elevation and immune parameter depression visually reinforces the statistical correlation ($r = -0.72$) observed between these variables.

Feed intake responded in a clear dose-dependent manner to decreasing temperature. Steers maintained at +15 °C consumed an average of 10.5 ± 0.4 kg dry matter per day, whereas those at 0, -15, and -25 °C consumed 11.3 ± 0.5 ,

12.9 ± 0.6 , and 14.8 ± 0.9 kg per day, respectively ($P < 0.001$). Despite higher feed consumption, average daily gain declined significantly in the colder treatments. Animals at +15 °C gained 1.38 ± 0.08 kg/day compared with 1.21 ± 0.09 (0 °C), 0.89 ± 0.11 (-15 °C), and 0.76 ± 0.13 kg/day (-25 °C). This represented a 12.3, 35.5, and 44.9 percent reduction in growth rate relative to the thermoneutral group. Serum cortisol increased progressively across temperature treatments, rising from 12.4 ± 1.8 ng/mL at +15 °C to 17.1 ± 2.3 (0 °C), 24.6 ± 3.1 (-15 °C), and 28.7 ± 3.9 ng/mL (-25

°C) by the mid-trial sampling point (day 42; $P < 0.001$). Conversely, IGF-1 concentrations fell from 187.3 ± 14.2 ng/mL at $+15$ °C to 142.5 ± 12.8 (-15 °C) and 118.9 ± 15.1 ng/mL (-25 °C; $P < 0.01$). Total white blood cell counts at the -25 °C treatment averaged $6.2 \pm 0.7 \times 10^3/\mu\text{L}$ compared with $8.7 \pm 0.6 \times 10^3/\mu\text{L}$ at thermoneutrality, a 28.7 percent reduction. Lymphocyte proportions dropped from 62.3 to 48.7 percent, and serum IgG fell from 2150 to 1680 mg/dL (21.9 percent decrease) in the most severely cold-stressed group. IgM showed a parallel decline of 30.5 percent. Cortisol and IgG concentrations were negatively correlated ($r = -0.72$, $P < 0.001$), supporting the hypothesis that HPA-axis activation mediates cold-induced immunosuppression.

Comprehensive interpretation

The results paint a consistent picture: as ambient temperature falls further below the LCT, cattle redistribute an increasing share of dietary energy toward heat generation, accept a progressively larger growth penalty, and suffer escalating immune suppression. The near-linear relationship between temperature and feed intake confirms earlier dose-response models [4] and extends them with concurrent immune data that were previously lacking. Importantly, the correlation between cortisol elevation and immunoglobulin decline suggests that endocrine disruption is a central mechanism linking cold exposure to immune vulnerability, not simply a parallel consequence. The partial recovery in growth and cortisol during weeks 9 through 12 indicates that physiological acclimation occurs but remains incomplete, leaving cattle vulnerable throughout the winter period. These data reinforce the perspective that winter management must address both the energetic and immunological dimensions of cold stress simultaneously [10].

Discussion

The findings align with and extend prior work documenting metabolic and immune costs of cold stress in cattle. The 41 percent rise in dry matter intake at -25 °C exceeds the 25 to 30 percent range reported in earlier Canadian feedlot surveys [3], likely because the present trial imposed continuous exposure without windbreak relief. Growth depression of up to 44.9 percent at -25 °C falls within the upper range of values compiled in a meta-analysis of North American winter feeding trials [11], where weighted mean growth reductions averaged 28 percent across varying cold severity.

The cortisol trajectory observed here matches the biphasic pattern described by Webster [12], with a rapid initial spike followed by partial habitu-

ation but sustained elevation above baseline. This chronic hypercortisolemia appears to be the bridge between thermoregulatory strain and immune deterioration, as glucocorticoids suppress transcription of pro-inflammatory cytokines and inhibit B-lymphocyte differentiation [6]. The 28.7 percent reduction in total leukocytes and the 21.9 percent decline in IgG are consistent with reports from winter BRD surveillance in Alberta feedlots, where seroconversion rates for *Mannheimia haemolytica* rose 1.9-fold during December through February [13].

An unexpected observation was the strong negative correlation between cortisol and IgG ($r = -0.72$), which was tighter than the $r = -0.53$ reported in a Finnish dairy herd exposed to -18 °C [14]. One possible explanation is that the present trial's continuous cold exposure prevented the

intermittent cortisol recovery that naturally occurs when cattle access heated shelters or experience brief warming periods.

Nutritional interventions partially mitigated cold-stress costs in the literature examined. Supplemental dietary fat at 3 to 5 percent of dry matter reduced net energy deficits by 12 to 18 percent in two Alberta trials [5], and increased feeding frequency from once to three times daily improved rumen fermentation heat output by roughly 7 percent [15]. However, no single dietary adjustment fully restored growth or immune function to thermoneutral levels. Housing modifications, including windbreaks that reduced wind chill exposure by 40 to 60 percent, showed the largest protective effects, decreasing maintenance energy requirements by approximately 15 percent [8]. This supports the conclusion that management strategies must combine nutritional, environmental, and potentially genetic approaches to meaningfully reduce winter productivity losses.

Conclusion

Cold stress imposes a multi-layered physiological burden on cattle that extends well beyond increased feed consumption. This review has demonstrated that ambient temperatures below the lower critical temperature trigger a cascade beginning with obligatory thermogenesis, proceeding through endocrine disruption, and culminating in measurable immune suppression each pathway reinforcing the others in a self-amplifying cycle of reduced productivity. The energetic cost of maintaining core body temperature in sub-zero environments forces cattle to consume substantially more feed while simultaneously converting less of that feed into marketable tissue. Growth rate reductions of 18 to 45 percent are not marginal losses; they translate directly into extended days on feed, higher total feed costs, delayed marketing, and reduced carcass value. For a 500-head feedlot facing a typical prairie winter, these efficiency losses can represent tens of thousands of dollars in foregone revenue per season.

Perhaps more concerning than the growth penalty is the systematic weakening of immune defenses. The data synthesized here show that cold-stressed cattle carry fewer circulating white blood cells, produce lower concentrations of protective immunoglobulins, and maintain chronically elevated cortisol levels that actively suppress immune cell function. This immunological vulnerability manifests clinically as increased rates of bovine respiratory disease, slower wound healing, and heightened susceptibility to gastrointestinal infections during the very months when pathogen challenge from crowded winter housing is greatest.

The interplay between energy balance and immune competence deserves particular emphasis. Immune function is itself energetically expensive; mounting an acute-phase response can increase metabolic rate by 10 to 15 percent. When the animal's energy budget is already strained by thermoregulation, the resources available for immune activity shrink correspondingly. Cold-stressed cattle thus face a metabolic tug-of-war between staying warm and staying healthy, and the evidence suggests that thermoregulation usually wins.

Mitigation strategies should therefore be integrative rather than singular. Energy-dense diets and supplemental fat can partially close the thermogenic energy gap, but they cannot eliminate the endocrine disruption caused by sustained cold

exposure. Windbreaks and bedding reduce the effective cold challenge at its source, lowering both the energetic and hormonal burden. Genetic selection for cold-tolerant phenotypes thicker winter coats, lower surface-area-to-mass ratios, and more efficient peripheral vasoconstriction offers a longer-term pathway that complements short-term management tools.

Looking ahead, several research gaps warrant attention. Breed-specific lower critical temperature data remain incomplete for many commercially important crossbred genotypes. The long-term epigenetic consequences of prenatal cold stress on calf health and future productivity are poorly characterized. And the interaction between cold stress and other winter risk factors including mud, short photoperiod, and restricted social space needs systematic factorial investigation. Addressing these gaps would allow producers to design winter management protocols grounded in a more complete understanding of the biological costs of cold and the most efficient strategies for reducing them.

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