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IMMUNOLOCALIZATION AND EXPRESSION OF AQUAPORIN 7 IN THE MAMMARY GLAND OF MURRAH BUFFALO

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ABSTRACT:
Aquaporins (AQPs) are integral membrane channel proteins that mediate the transcellular movement of water and selected small solutes. Among these, aquaporin 7 (AQP7) is classified as an aquaglyceroporin, enabling the transport of water, glycerol, and urea. The mammary gland, as a secretory organ, must transfer substantial quantities of fluid and solutes to sustain milk synthesis, with water comprising approximately 87% of milk volume. Despite this high demand for transcellular water flux, the specific contribution of AQP7 to mammary gland function in ruminants has not been fully elucidated. In this study, we investigated the expression, regulation, and immunolocalization of AQP7 in the mammary gland of Murrah buffalo (Bubalus bubalis) across distinct physiological stages and under defined hormonal stimuli. Histological examination revealed clear structural differences among heifer, lactating, and dry mammary tissues, reflecting stage dependent remodeling consistent with secretory activity. RT qPCR analysis demonstrated a robust upregulation of AQP7 transcript levels during lactation, indicating a strong association between AQP7 expression and the active milk producing state. Immunohistochemical localization further showed AQP7 protein in both secretory epithelial cells and myoepithelial cells, suggesting roles in milk secretion and alveolar function.

KEYWORDS: AQP7, buffalo mammary epithelial cells, Murrah buffalo, Lactation

INTRODUCTION

The buffalo mammary gland is a dynamic organ that undergoes repeated cycles of remodeling throughout the animal's life (during puberty, pregnancy, lactation, and involution) to carry out its main biological function: the synthesis and secretion of milk [Macias & Hinck 2012, Jiang et al. 2025, Oliveira et al. 2026]. Sustained milk production is essential for the survival of the buffalo calf, and the gland's development and function are tightly regulated by systemic hormones and locally produced growth factors [Hennighausen & Robinson 2001, Macias & Hinck 2012, Jiang et al. 2025]. Milk is produced by mammary secretory epithelial cells, which release its major components (water, lipids, proteins, and lactose) into the alveolar lumen [Shennan & Peaker 2000]. Water is the main component of milk, about 87% of its volume, so moving water across the epithelium is a key step in milk production. Even so, the molecular mechanisms that control water transport across mammary epithelial cells have only recently begun to be understood [Calamita & Delporte 2023].

Aquaporins are highly conserved membrane proteins that mediate bidirectional transport of water and small solutes across cellular membranes (Sabir et al., 2026). Since the initial identification of AQP1 in human erythrocytes, 13 mammalian aquaporins have been characterized, each exhibiting distinct tissue distribution and substrate selectivity (Yadav et al., 2025). Based on permeation properties, they are broadly categorized into orthodox aquaporins (AQP0–2, AQP4–6, AQP8), which are primarily selective for water, and aquaglyceroporins (AQP3, AQP7, AQP9–10), which conduct water as well as glycerol and urea (Portincasa et al., 2026). Structurally, aquaporins comprise six transmembrane helices and highly conserved Asn–Pro–Ala (NPA) motifs that assemble into a narrow aqueous pore (Wang et al., 2025). In aquaglyceroporins, the pore diameter is enlarged relative to orthodox AQPs, enabling passage of larger solutes such as glycerol; in particular, AQP7 has emerged as a pivotal channel in metabolic and secretory processes, linking transmembrane glycerol flux to cellular energy balance and lipid metabolism (Kitchen et al., 2015). In humans, the gene AQP7, located on chromosome 9, forms homotetrameric transmembrane channels that allow water, glycerol, and urea passage (Costa et al., 2025; Azad et al., 2021). AQP7 is highly abundant in adipose tissue and acts as a conduit for glycerol released after lipolysis, thus maintaining systemic energy homeostasis (de Mare, 2020). Absence of AQP7 in murine models leads to adipocyte hypertrophy, insulin resistance, glycerol accumulation, and higher obesity susceptibility (Costa et al., 2025; Pimpão et al., 2022). Besides adipose tissue, AQP7 is expressed in the kidney, where it participates in glycerol reabsorption, and in pancreatic beta-cells, where it mediates insulin secretion (Lamsal, 2022). Expression of AQP7 in mammary glands is developmentally regulated and linked with milk production. AQP7 is expressed in adipocytes and mammary epithelial cells, predominantly with plasma membrane localization, during the lactation period, suggesting its participation in transepithelial transport (Ferré-Dolcet and Rivera del Alamo, 2023).

The mammary gland undergoes morphological and physiological changes during the reproductive cycle and milk

secretion (Biswas et al, 2022). It has branched ducts ending in secretory alveoli surrounded by stromal matrix comprising adipocytes, fibroblasts, and immune cells (Jaswal et al., 2022). Alveolar epithelium includes luminal secretory cells for milk synthesis and myoepithelial cells responsible for milk ejection. The lactation process requires tremendous synthetic and transport capacity as dairy cows produce 60 liters of milk per day (Webster, 2020). Milk composition includes about 87% of water, 3-5% of lipids, 3-4% of proteins, 4-5% of lactose, and 0.7% of minerals (Kumar et al, 2025). The large need for water content in milk makes aquaporins essential for efficient water transport. Glycerol is also a structural molecule essential for fat synthesis and is transported via AQP7 aquaglyceroporin (Liu et al., 2024).

Several aquaporin isoforms are expressed in mammary tissues of different species, including AQP1, AQP3, AQP4, AQP5, and AQP7 in human, bovine, and mouse mammary glands (Fujimoto et al., 2024). In the lactating bovine mammary gland, both AQP3 and AQP7 are expressed in secretory epithelial cells, ductal epithelial cells, and smooth muscle bundles, providing evidence of their involvement in milk synthesis and milk ejection (Jiang et al., 2025). AQP1 is highly expressed in capillary endothelia and myoepithelial cells, while AQP5 is expressed in gland cisterns (Calamita and Delporte, 2023). Glycerol transport for milk fat synthesis and waste disposal is facilitated by aquaglyceroporins AQP3, AQP7, and AQP10 (Soares, 2020). Stage-dependent expression patterns during pregnancy, lactation, and involution periods indicate specific regulatory mechanisms (Oliveira et al., 2026). AQP3 and AQP5 expression in mouse mammary glands differ before and after parturition, suggesting their involvement in functional maturation of the gland (Oliveira et al., 2026).

Water buffalos (Bubalus bubalis) have economic importance worldwide as more than 200 million of these animals are mostly located in Asia, the Mediterranean, and South America (Liang and Xu, 2025). Buffalo milk is produced worldwide and is known for high fat content and high-value dairy products (Mota-Rojas et al., 2026). Murrah buffalo, being native to the Indian sub-continent, is known for high milk yield (1,800-2,000 kg per lactation) (Bhat and Qureshi, 2025). Owing to advancements in buffalo genomics, the aquaporin gene family in water buffalo was characterized, allowing to detect conserved and species-specific features. AQP0, AQP2, AQP4, AQP5, AQP7, AQP9, and AQP12 have been shown to have tissue-restricted expression, while AQP1, AQP3, AQP8, and AQP11 are ubiquitously expressed (Rajput et al., 2024). Transcript variants and positive selection at NPA motifs and ar/R selectivity filters suggest functional adaptation (Liu et al., 2024).

This study was undertaken with the following objectives:

1. To verify the physiological states of the mammary tissue samples by histological examination.
2. To measure the amount of AQP7 mRNA in different developmental stages.
3. To determine AQP7 protein immunolocalization.
4. To investigate AQP7 regulation in buffalo mammary epithelial cells (BuMEC) in response to lactose, cortisol, estrogen, prolactin, and growth hormone.

MATERIALS AND METHODS

Ethical statement

Ethical approval was not required from the Institutional Animal Ethics Committee (IAEC) of ICAR-National Dairy Research Institute, Karnal, Haryana as buffalo tissue samples were collected from municipal slaughterhouse (Gazipur, New Delhi) for the study.

Histological Analysis

Mammary gland tissue samples were obtained from Murrah buffaloes of three different physiological states, that is, heifers (pre-pubertal, nulliparous), lactating (from early to mid-lactation) and dry (non-lactating). The tissue samples were fixed in 10% neutral buffered formalin and then used for histology and immunohistochemistry or stored snap-frozen in liquid nitrogen for RNA extraction.

The formalin-fixed tissue samples were dehydrated in a series of ethanol, cleared with xylene and embedded in paraffin. Paraffin blocks were sectioned into 5-7µm thickness and stained with haematoxylin and eosin. De-paraffinized sections were stained with Harris haematoxylin for 8-10 minutes, differentiated in 1% acid alcohol, blued in Scott's tap water, counterstained with 1% eosin, dehydrated, cleared, and mounted. The morphological assessment was done through microscopic examination (light microscope, Olympus BX51).

RNA Extraction and Evaluation

Isolation of RNA was carried out using Trizol reagent from tissue samples and cultured BuMEC. Tissue samples or 1×10^6 cells were homogenized in 1ml of Trizol, incubated for 5 minutes at room temperature, addition of 200µl of chloroform and then centrifugation at 12,000 × g for 15 minutes at 4°C, followed by isolation of RNA from the aqueous phase with isopropanol. RNA pellets were washed with 75% ethanol, air dried and then dissolved in RNase free water. RNA quality (OD 260/280 ratio of 1.9-2.0) and RNA integrity (18S and 28S bands on 1.5% agarose gel) were evaluated.

Complementary DNA Synthesis and Quantitative RT-PCR

Approximately 1 µg of RNA was used for reverse transcription into cDNA using RevertAid First Strand cDNA

Synthesis Kit. SYBR green master mix was performed on CFX96 Real-Time PCR System (RT-qPCR). The primers for the AQP7 and β-actin genes were designed based on buffalo specific sequence information. Reaction mixture was prepared by adding 10µl SYBR green master mix, 1 µl of each primer (10µM concentration), 2µl cDNA and 6µl water. RT-qPCR cycling program included 95°C for 3 minutes, 40 cycles of 95°C for 10 seconds, 60°C for 30 seconds and 72°C for 30 seconds. Gene expression was quantified by $2^{-\Delta\Delta Ct}$ method with β-actin as an endogenous control.

Immunohistochemistry

Immunohistochemistry of AQP7 was carried out using the technique based on the avidin-biotin-peroxidase complex method. Antigen retrieval was done by treating sections in 0.01M citrate buffer (pH 6.0). Afterwards, the paraffin-embedded sections were incubated for 16 hours at 4°C with a 1:200 dilution of rabbit polyclonal anti-AQP7 antibodies. Following washing of the sections, incubation was performed with biotinylated goat anti-rabbit antibodies and ABC kit. Presence of peroxidase was detected with DAB chromogen, while haematoxylin was used for counterstaining. The sections were examined under a light microscope and photographed.

Cell Culture and Treatments

Buffalo mammary epithelial cells were cultured in DMEM/F12 with 10% FBS, antibiotics, insulin, hydrocortisone and EGF at 37°C in 5% CO_2 atmosphere. Cells were grown in 6-well plates until reaching approximately 70% confluence, after which the culture medium was replaced with fresh medium without serum for 12 hr. After 12 hr, BuMECs were treated with lactose (2, 4, 8 µg/ml), cortisol (6.25, 12.5, 25 ng/ml), estrogen (20, 40, 60 pg/ml), prolactin (62.5, 125, 250 ng/ml), and growth hormone (4, 8, 12 ng/ml) for 24 hours. Control cells were grown without serum. Three biological replicates were used for each experiment.

Total RNA was isolated from BuMECs following treatment with the hormones using TRIzol reagent, according to the manufacturer's specifications.

2.7 Statistical Analysis

Data are presented as mean ± SEM of at least three independent experiments. One way ANOVA and Tukey's post-hoc test were applied for statistical analysis. Significance was set at *p* < 0.05; level of significance is as follows: *****p* ≤ 0.0001, ****p* ≤ 0.001, ***p* ≤ 0.01, **p* ≤ 0.05, ns for non-significant changes.

RESULTS

Histological Characterization

Histological examination of mammary tissue revealed distinct morphological characteristics at each physiological stage. In heifers, the mammary gland contained developing alveoli embedded in a stroma rich in adipocytes and connective tissue, with only a few poorly developed ductal structures and alveolar buds. In contrast, lactating mammary glands exhibited well-developed tubuloalveolar structures lined by cuboidal to columnar epithelial cells with abundant eosinophilic cytoplasm, and lumina filled with eosinophilic secretory material. Dry mammary glands showed marked involution, characterized by loss of secretory parenchyma, a reduction in the number of tubuloalveolar structures, replacement of alveolar tissue by adipose tissue, collapse of inactive alveoli and the presence of apoptotic cells.

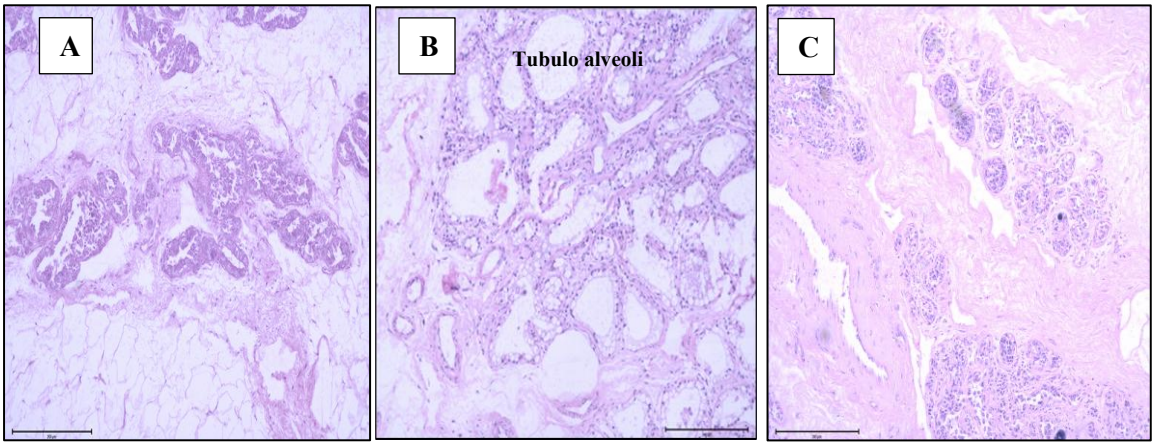


Fig 1. Histology of mammary gland of murrah buffaloes. **A)** Section of the Heifer mammary gland showing developing alveoli surrounding adipocytes and stroma, **B)** Section of lactating mammary gland showing active tubuloalveoli and ducts filled with milk droplets, **C)** Section of Dry mammary gland showing reduction in milk production, loss of tubuloalveolar structures, apoptosis and the replacement of epithelial tissue with adipocytes.

RNA Quality and AQP7 Expression

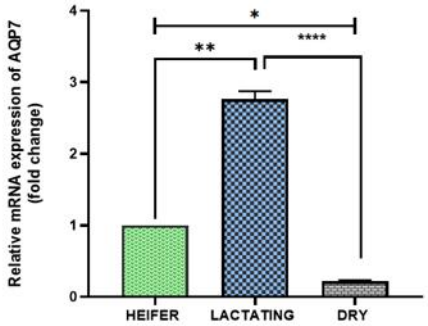


Fig 2: Relative mRNA expressions ($2^{-\Delta\Delta ct}$) of AQP7 in mammary gland of murrah buffalo during different developmental stages. Results are shown as mean ± SEM from three independent experiments. Statistical significance as per Tukey's test is indicated by: ****p ≤ 0.0001, *** p ≤ 0.001, ** p ≤ 0.01, * p ≤ 0.05, and ns for non-significant changes.

RNA samples exhibited good integrity, with OD 260/280 ratios between 1.9 and 2.0 and clear 28S and 18S ribosomal bands on agarose gel electrophoresis. RT-qPCR analysis revealed significantly higher AQP7 mRNA expression in lactating mammary glands compared with heifer and dry stages, with lactating tissue showing approximately 4.5- and 6.8-fold increases, respectively ($p < 0.0001$). In addition, AQP7 expression in mammary glands of dry animals was significantly lower than in heifers, indicating a marked reduction in secretory activity at the dry stage.

Immunolocalization of AQP7

Immunohistochemical analysis showed stage-dependent localization of AQP7 in the mammary gland. In heifer tissue sections, AQP7 immunoreactivity was observed in the ductal and alveolar epithelial linings, with moderate cytoplasmic staining. In contrast, lactating mammary glands displayed intense and widespread immunostaining, particularly in secretory epithelial cells and myoepithelial cells. In dry mammary gland tissue, immunoreactivity in these same cellular components was markedly reduced, indicating a decline in AQP7 expression concurrent with decreased secretory activity.

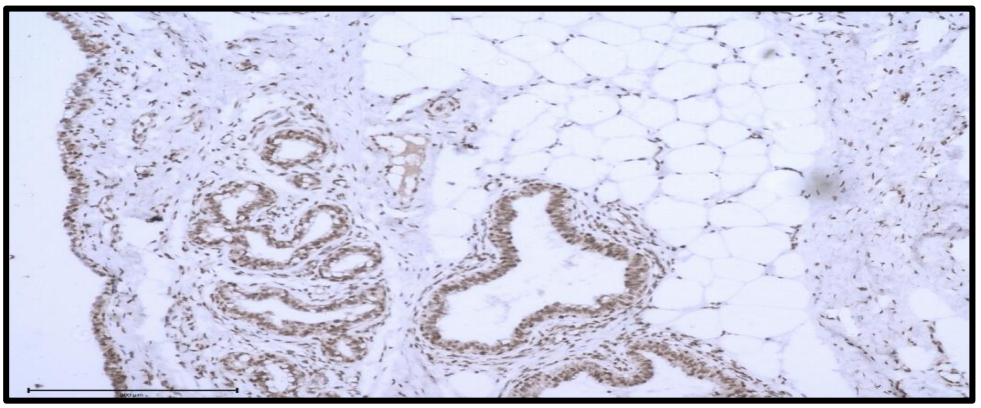


Fig 3: Immunohistochemical localization of AQP7 in ductal and alveolar MECs of heifer mammary gland of Murrah buffalo. Ductal and alveolar epithelial lining are immunostained with anti-AQP7 antibody (Brown colour).

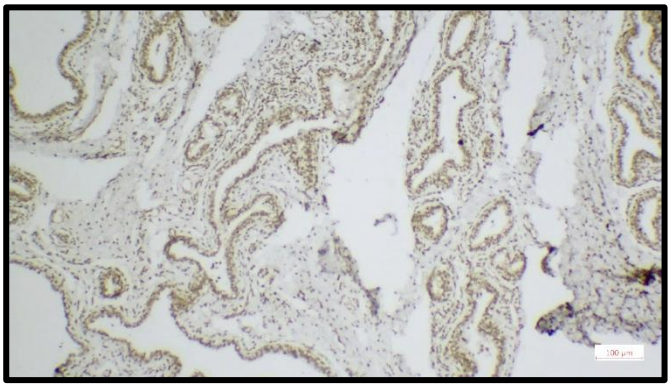


Fig 3.32: Immunohistochemical localization of AQP7 in the mammary gland of a lactating murrah buffalo. Staining shows strong cytoplasmic expression of AQP7 (brown colour) in myoepithelial cells and secretory

epithelial cells, indicating its involvement in glycerol transport for milk lipid synthesis during lactation.

Fig 4: Immunohistochemical localization of AQP7 in the mammary gland of a lactating murrah buffalo. Staining shows strong cytoplasmic expression of AQP7 (brown colour) in myoepithelial cells and secretory epithelial cells, indicating its involvement in glycerol transport for milk lipid synthesis during lactation.

Effect of Lactose on AQP7 Expression

Lactose treatment produced a biphasic effect on AQP7 expression. At a concentration of 2 µg/ml, AQP7 expression was significantly reduced to 0.5-fold compared to the control ($p < 0.001$). In contrast, higher concentrations led to an increase in expression, reaching 1.1-fold at 4 µg/ml ($p < 0.0001$) and 1.7-fold at 8 µg/ml ($p < 0.0001$).

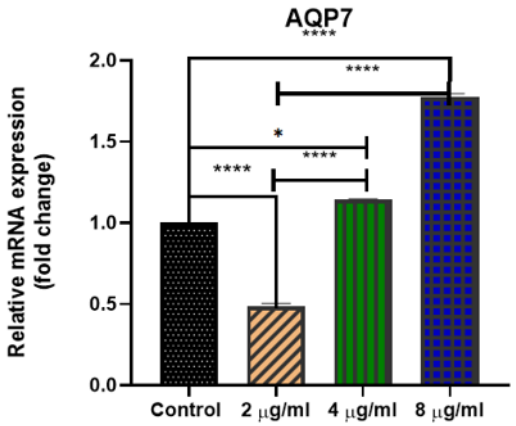


Fig 5: Relative expression levels of AQP genes in BuMEC treated with various concentrations of lactose, assessed using RT-PCR. RNA isolated from treated cell pellets was reversed transcribed into cDNA, and the expression of A) AQP7 was quantified. Expression data were normalized to β-actin as an internal control. Results are shown as mean ± SEM from three independent experiments. Statistical significance is indicated by: ****$p \leq 0.0001$, *** $p \leq 0.001$, ** $p \leq 0.01$, * $p \leq 0.05$, and ns for non-significant changes.

Effect of cortisol on AQP7 expression

Cortisol treatment induced dose-dependent changes in AQP7 expression. At 6.25 ng/ml, AQP7 expression increased significantly to 2.4-fold compared to the control ($p \leq 0.001$). However, at higher concentrations, expression decreased to 1.2-fold at 12.5 ng/ml and was markedly inhibited to 0.2-fold at 25 ng/ml ($p \leq 0.0001$).

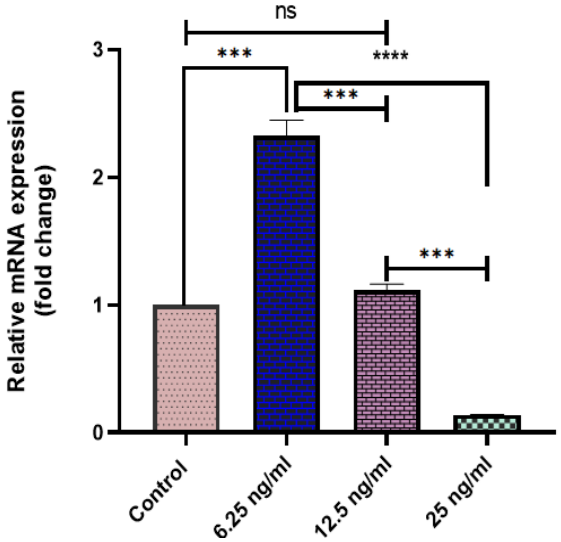


Fig 6: Relative expression levels of AQP genes in BuMEC treated with various concentrations of cortisol, assessed using RT-PCR. RNA isolated from treated cell pellet was reversed transcribed into cDNA, and the expression of A) AQP7 was quantified. Expression data were normalized to β-actin as an internal control. Results are shown as mean ± SEM from three independent experiments. Statistical significance is indicated by: ****$p \leq 0.0001$, *** $p \leq 0.001$, ** $p \leq 0.01$, * $p \leq 0.05$, and ns for non-significant changes.

Effect of estrogen on AQP7 expression

Estrogen treatment resulted in a dose-dependent increase in AQP7 expression. At 20 pg/ml, expression increased to 2.5-fold compared to the control ($p \leq 0.0001$). This increase was more pronounced at higher concentrations, reaching 4.5-fold at 40 pg/ml ($p \leq 0.0001$) and 7.5-fold at 60 pg/ml ($p \leq 0.0001$).

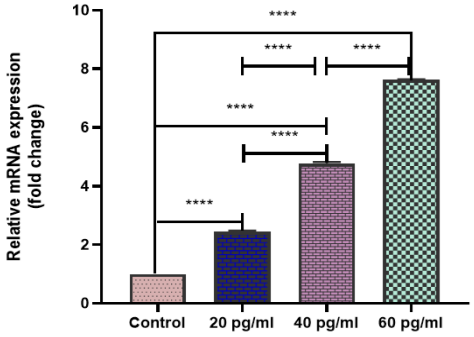


Fig 7: Relative expression levels of AQP genes in BuMEC treated with various concentrations of estrogen, assessed using RT-PCR. RNA isolated from treated cell pellets was reversed transcribed into cDNA, and the expression of A) AQP7 was quantified. Expression data were normalized to β-actin as an internal control. Results are shown as mean ± SEM from three independent experiments. Statistical significance is indicated by: ****p ≤ 0.0001, *** p ≤ 0.001, ** p ≤ 0.01, * p ≤ 0.05, and ns for non-significant changes.

Effect of prolactin on AQP7 expression

Prolactin treatment resulted in a dose-dependent increase in AQP7 expression. At 62.5 ng/ml, AQP7 expression showed a non-significant increase to 0.8-fold compared to the control. At higher concentrations, expression increased significantly to 2.3-fold at 125 ng/ml ($p < 0.01$) and further to 3.4-fold at 250 ng/ml ($p < 0.0001$).

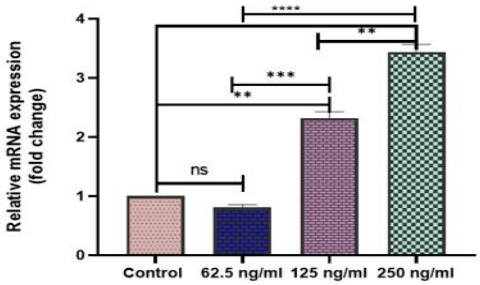


Fig 8: Relative expression levels of AQP genes in BuMEC treated with various concentrations of prolactin, assessed using RT-CR. RNA isolated from treated cell pellet was reversed transcribed into cDNA, and the expression of A) AQP7 was quantified. Expression data were normalized to β-actin as an internal control. Results are shown as mean ± SEM from three independent experiments. Statistical significance is indicated by: ****p ≤ 0.0001, *** p ≤ 0.001, ** p ≤ 0.01, * p ≤ 0.05, and ns for non-significant changes.

Effect of growth hormone on AQP7 expression

Growth hormone proved to be the most potent stimulator. At 4 ng/ml, AQP7 expression was 4-fold (*p* < 0.05). At 8 ng/ml, expression was 10-fold (*p* < 0.0001), and at 12 ng/ml, was 13-fold (*p* < 0.0001) compared to controls.

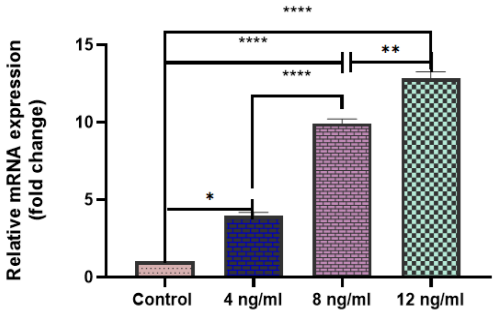


Fig 9: Relative expression levels of AQP genes in BuMEC treated with various concentrations of growth hormone, assessed using RT-PCR. RNA isolated from treated cell pellets was reversed transcribed into cDNA, and the expression of A) AQP7 was quantified. Expression data were normalized to β-actin as an internal control. Results are shown as mean ± SEM from three independent experiments. Statistical significance is indicated by: ****p ≤ 0.0001, *** p ≤ 0.001, ** p ≤ 0.01, * p ≤ 0.05, and ns for non-significant changes.

DISCUSSION

The present study provides comprehensive evidence for the expression, localization and hormonal regulation of aquaporin 7 (AQP7) in the mammary gland of Murrah buffalo, thereby identifying AQP7 as a lactation-associated factor of functional importance. AQP7 mRNA and protein were markedly upregulated during the lactation period

compared with heifer and dry periods, in agreement with reports from other species and supporting a conserved role of AQP7 in milk secretion. Immunohistochemical analysis demonstrated strong AQP7 staining in secretory epithelial cells and myoepithelial cells of lactating tissue, which is consistent with observations in bovine mammary gland and suggests that AQP7 contributes both to secretory function and to the microenvironment of contractile elements.

The marked increase of AQP7 expression in lactation is biologically relevant, as milk contains a high proportion of water and coordinated water transport is essential for milk volume formation. In addition, AQP7 belongs to the aquaglyceroporins and facilitates glycerol transport, providing substrate for triglyceride synthesis in the mammary epithelium. Milk triglyceride synthesis depends on a continuous supply of glycerol-3-phosphate derived from circulating or locally generated glycerol; thus, the decline in AQP7 expression during the dry period and involution supports a close association between active lactation and AQP7-mediated water and glycerol flux.

In vitro experiments using buffalo mammary epithelial cells (BuMEC) revealed that AQP7 expression is under complex endocrine control. Estrogen induced a robust increase in AQP7 mRNA, consistent with the established role of estrogen in mammary growth and lactogenesis. In-silico prediction of estrogen response elements within the AQP7 promoter further supports direct transcriptional regulation. Prolactin also upregulated AQP7, in line with its central role in mammary epithelial differentiation and milk protein gene expression via the JAK2–STAT5 pathway. These data indicate that AQP7 is integrated into the prolactin-responsive gene network that coordinates biosynthetic capacity with membrane transport during lactation.

Growth hormone (GH) exerted the greatest stimulatory effect on AQP7, with a 13-fold increase at the tested concentration. Given the galactopoietic activity of GH in dairy species and its known signaling through JAK2–STAT5 and MAPK pathways in mammary epithelium, the strong GH responsiveness of AQP7 suggests that AQP7 may act as a downstream mediator of GH-driven nutrient partitioning and mammary metabolism. The observed hierarchy of hormonal effects, with GH > estrogen > prolactin, is consistent with the coordinated endocrine regulation of the lactation process described in earlier work.

Cortisol exhibited a biphasic effect on AQP7 expression, with low concentrations upregulating and high concentrations downregulating the gene. This dual response is compatible with the physiological role of glucocorticoids in supporting mammary differentiation and lactation at moderate levels, but impairing lactation under chronic stress when cortisol is elevated. Similarly, lactose exposure resulted in a biphasic AQP7 expression pattern, with downregulation at low concentration followed by upregulation at higher concentrations. Because lactose synthesis in the Golgi apparatus is a major driver of osmotic water flux into secretory vesicles, this pattern may reflect a feedback mechanism in which AQP7 is adjusted according to secretory activity and osmotic demands.

Immunohistochemical localization data support the gene expression findings. Cytoplasmic and membrane-associated AQP7 staining indicates roles in intracellular glycerol handling and transepithelial transport. Localization in both basolateral and apical membranes of secretory epithelial cells is consistent with vectorial water and glycerol transport from blood to milk along a transcellular pathway. AQP7 positivity in myoepithelial cells suggests that these cells may also participate in local water and glycerol homeostasis or utilize glycerol for metabolic needs during oxytocin-mediated milk ejection.

Characterization of AQP7 in Murrah buffalo contributes to understanding of aquaporin biology in ruminant lactation. The conservation of AQP7 across mammalian species underscores its importance in water and glycerol homeostasis. Species differences in mammary structure, teat canal length, lactation curve and milk fat content between water buffalo and dairy cattle may be partly explained by distinct patterns of AQP7 expression and regulation. In particular, the high fat content of buffalo milk may be linked to strong AQP7 expression during lactation and its pronounced stimulation by GH, which together enhance glycerol availability for milk fat synthesis.

Implications for Milk Production and Animal Health

The present findings have practical relevance for dairy buffalo production. Identification of AQP7 as an important lactation factor suggests that modulation of its expression or activity could be a strategy to improve milk yield and composition. Optimizing GH status and maintaining adequate estrogen and prolactin levels through nutritional or hormonal management may enhance AQP7-mediated transport and support higher milk fat and volume. Conversely, chronic stress leading to sustained elevation of cortisol may reduce AQP7 expression and negatively affect lactation performance. A better understanding of AQP7 regulatory pathways could aid in designing management practices that minimize stress and promote a favorable endocrine milieu for lactation. Furthermore, given the role of AQP7 in glycerol transport and milk fat synthesis, polymorphisms in the AQP7 gene may contribute to variation in milk production traits, making AQP7 a potential candidate marker for genetic selection in breeding programs.

CONCLUSION

In conclusion, AQP7 is expressed in the mammary gland of Murrah buffalo and is significantly upregulated during the lactation stage relative to heifer and dry stages. Secretory epithelial and myoepithelial cells of lactating tissue show strong AQP7 immunoreactivity, supporting its involvement in milk production. AQP7 expression is regulated by estrogen, prolactin, growth hormone and cortisol, indicating complex endocrine control of mammary gene expression during lactation. Growth hormone exerts the most potent stimulatory effect, suggesting that AQP7 participates in mediating galactopoietic actions of GH. Overall, these results identify AQP7 as an essential molecular component of the lactation machinery in water buffalo and provide a useful framework for future studies aimed at improving milk production and understanding mammary physiology in this economically important species.

Conflict Of Interest

The authors declare no conflicts of interest associated with this manuscript.

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