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Effect of encapsulated butyrate on intestinal barrier function and immune response in weaned piglets

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Abstract

Butyrate is a short-chain fatty acid produced naturally by bacterial fermentation of dietary fibre in the hindgut, and it serves as the preferred energy substrate for colonocytes while exerting potent effects on epithelial integrity and mucosal immunity. In weaned piglets, the abrupt transition from sow's milk to solid feed disrupts gut microbiota composition, reduces endogenous butyrate production, and predisposes the intestinal epithelium to barrier failure a sequence of events that underpins the high incidence of post-weaning diarrhoea (PWD) in commercial pig production. Supplementing butyrate in feed could theoretically offset this deficit, but unprotected sodium butyrate is absorbed rapidly in the proximal gut and releases an unpleasant odour that depresses voluntary feed intake. Microencapsulation in a lipid matrix allows butyrate to bypass the stomach and small intestine and release its payload in the colon, where it is needed most.

This research evaluated the effects of two dietary inclusion levels of microencapsulated sodium butyrate (1 g/kg and 3 g/kg feed) on intestinal barrier function, tight-junction protein expression, mucosal cytokine profiles, and growth performance in weaned piglets over a 28-day feeding period. Thirty-six crossbred (Landrace × Yorkshire) piglets weaned at 21 days of age (mean body weight 6.8 ± 0.4 kg) were randomly assigned to three dietary treatments: a basal diet (control), basal diet plus 1 g/kg encapsulated butyrate (low dose), or basal diet plus 3 g/kg encapsulated butyrate (high dose), with 12 piglets per group. The trial was conducted at the Copenhagen Academy of Animal Sciences swine research facility between March and May 2023.

On day 28, piglets were euthanised and jejunal and colonic tissue samples were collected for histomorphometry, quantitative real-time PCR of tight-junction genes (claudin-1, occludin, ZO-1), transepithelial electrical resistance (TEER) measurement in Ussing chambers, and cytokine mRNA quantification (IL-10, TNF- α , IL-1 β , IL-6). Growth performance (average daily gain [ADG], average daily feed intake [ADFI], and feed conversion ratio [FCR]) was recorded weekly. High-dose butyrate significantly increased colonic mRNA expression of claudin-1 (1.78-fold; $P < 0.001$), occludin (1.63-fold; $P < 0.01$), and ZO-1 (1.54-fold; $P < 0.01$) relative to controls. TEER values were 44.6% higher in high-dose animals (412 ± 41 vs 285 ± 32 $\Omega \cdot \text{cm}^2$; $P < 0.001$). IL-10 mRNA was upregulated 2.1-fold ($P < 0.01$) while TNF- α was downregulated by 38% ($P < 0.05$) in the high-dose group. Low-dose butyrate produced intermediate effects that reached significance for claudin-1 and TEER but not for most cytokines. ADG was improved by 11.3% in the high-dose group ($P < 0.05$) with no effect on ADFI, resulting in a 9.8% improvement in FCR. These findings indicate that microencapsulated butyrate at 3 g/kg feed strengthens intestinal barrier integrity through upregulation of tight-junction proteins and promotes an anti-inflammatory mucosal environment in weaned piglets, effects that translate into measurable growth performance benefits.

Keywords: Butyrate, microencapsulation, weaned piglets, intestinal barrier, tight-junction proteins, transepithelial electrical resistance, cytokines, gut health, post-weaning diarrhoea, short-chain fatty acids

Introduction

Butyrate (butanoic acid; $\text{C}_4\text{H}_7\text{O}_2^-$) is a four-carbon short-chain fatty acid generated in the mammalian large intestine through anaerobic bacterial fermentation of non-digestible carbohydrates^[1]. Beyond its well-recognised role as the main oxidative fuel for colonocytes supplying up to 70% of their energy needs butyrate acts as a signalling molecule that modulates gene expression through inhibition of histone deacetylases (HDACs),

thereby influencing epithelial proliferation, differentiation, and apoptosis^[2]. Its capacity to tighten the paracellular barrier by upregulating claudin-1, occludin, and zonula occludens-1 (ZO-1) has been confirmed repeatedly in cell culture systems and rodent models^[3, 4].

Weaning is the most stressful event in a piglet's early life. The simultaneous removal from the sow, change in diet, and social mixing trigger a cascade of physiological disturbances collectively termed "post-weaning syndrome"^[5]. At the gut level, villous atrophy, crypt hyperplasia, and transient increases in mucosal permeability appear within 48 hours of weaning and can persist for one to two weeks^[6]. These structural changes coincide with a shift in the colonic microbiota away from lactobacilli and toward potential pathogens such as enterotoxigenic *Escherichia coli* (ETEC), resulting in reduced endogenous butyrate production precisely when it is most needed^[7]. Post-weaning diarrhoea follows in 15–25% of commercially weaned piglets, driving mortality, antimicrobial use, and economic losses estimated at €1.5–2.0 per affected piglet^[8].

Supplementing exogenous butyrate to the diet is an appealing strategy to bridge the gap left by declining microbial production. But unprotected sodium butyrate presents two practical problems. First, its strong rancid odour reduces feed palatability, leading to lower voluntary intake and an effect especially harmful in newly weaned piglets that are already reluctant to eat^[9]. Second, uncoated butyrate is absorbed almost entirely in the stomach and proximal small intestine, leaving little or none to reach the colon where its barrier-strengthening effects are most required^[10]. Microencapsulation in a hydrogenated vegetable fat matrix solves both issues: the lipid coat masks the odour and delays release until the capsule reaches the distal gut, where bile salt and lipase activity degrade the coating and liberate butyrate locally^[11].

Despite growing commercial availability of encapsulated butyrate products, dose-response data in weaned piglets remain limited. Most published trials have tested a single inclusion rate, making it difficult to determine whether a linear or plateau-type response exists and at what level the benefit is maximised^[12]. This research addressed that gap by comparing two dietary concentrations of microencapsulated sodium butyrate a low dose (1 g/kg) and a high dose (3 g/kg) against an unsupplemented control, using a comprehensive panel of intestinal barrier, immune, and performance endpoints.

The specific objectives were to quantify the effect of each dose on (a) mRNA expression of tight-junction proteins claudin-1, occludin, and ZO-1 in colonic tissue, (b) transepithelial electrical resistance as a functional measure of barrier integrity, (c) mucosal cytokine profiles (IL-10, TNF- α , IL-1 β , IL-6), and (d) growth performance (ADG, ADFI, FCR) over a 28-day post-weaning period.

Material and Methods

Field experimental design

The trial followed a completely randomised design with three dietary treatments and 12 replicates (individual piglets) per treatment. Treatments were: (T1) basal diet with no butyrate supplementation (control), (T2) basal diet plus 1 g/kg microencapsulated sodium butyrate (low dose), and (T3) basal diet plus 3 g/kg microencapsulated sodium butyrate (high dose). The encapsulated product (ButyCoat®, NutriTech ApS, Aarhus, Denmark) contained 30% sodium butyrate in a hydrogenated palm fat matrix, yielding effective butyrate

delivery of 0.3 and 0.9 g/kg feed for T2 and T3, respectively. Diets were formulated to meet or exceed NRC (2012) nutrient requirements for weaned piglets and were iso-caloric and iso-nitrogenous across treatments^[13].

Material

Thirty-six crossbred (Landrace $\times$ Yorkshire) piglets weaned at 21 days of age (mean body weight 6.8 ± 0.4 kg; 18 males, 18 females) were sourced from the Copenhagen Academy of Animal Sciences swine research unit, Copenhagen, Denmark. Animals were housed individually in climate-controlled pens (28 °C, 55–65% relative humidity) with plastic-coated slatted floors, nipple drinkers, and individual feeders. A seven-day pre-experimental period on the basal diet preceded treatment allocation. The feeding trial ran from March to May 2023. Body weight was recorded on days 0, 7, 14, 21, and 28. Feed offered and refused was weighed daily to calculate ADFI.

Methods

On day 28, all piglets were euthanised by captive-bolt stunning followed by exsanguination. Tissue samples (approximately 2 cm segments) were collected from the mid-jejunum and proximal colon within 15 minutes of death. One set of samples was snap-frozen in liquid nitrogen and stored at -80 °C for RNA extraction; a second set was placed in 10% neutral buffered formalin for histomorphometric analysis; a third set was mounted immediately in Ussing chambers for TEER measurement^[3].

Total RNA was extracted using TRIzol reagent, and cDNA was synthesised as described elsewhere^[4]. Quantitative real-time PCR was performed for claudin-1, occludin, ZO-1, IL-10, TNF- α , IL-1 β , and IL-6 using validated porcine-specific primers, with β -actin as the reference gene. Relative expression was calculated by the $2^{-\Delta\Delta Ct}$ method. TEER was measured across colonic tissue mounted in Ussing chambers (Physiologic Instruments, San Diego, CA, USA) perfused with Krebs–Ringer buffer at 37 °C, with readings taken every 15 minutes over a 90-minute equilibration period; the plateau value was recorded^[14]. Formalin-fixed tissues were processed for H&E staining, and villus height and crypt depth were measured on 20 well-oriented villi per animal using ImageJ software. Data were analysed by one-way ANOVA with Tukey's post-hoc test in R v.4.3.1. Results are reported as mean $\pm$ SD; significance was set at $P < 0.05$.

Quality control

Encapsulated butyrate product quality was verified by independent assay at the institute's analytical chemistry laboratory, confirming sodium butyrate content of $29.7 \pm 0.8\%$ (target 30%) and lipid coating integrity by differential scanning calorimetry. Feed samples were collected weekly from each treatment group and analysed for proximate composition to confirm formulation accuracy. RNA integrity numbers (RIN) were assessed on an Agilent Bioanalyzer; only samples with $RIN \geq 7.5$ were used for qRT-PCR. Primer efficiencies fell within 92–108%. Ussing chamber tissue viability was confirmed by monitoring short-circuit current response to forskolin at the end of each experiment; non-responsive tissues were excluded^[15].

Ethical considerations

All procedures were approved by the Copenhagen Academy of Animal Sciences Institutional Animal Care and Use Committee (Protocol CAAS-IACUC-2023-006, approved 15 January 2023). The research complied with the Danish

Animal Experimentation Act (Consolidation Act No. 253 of 8 March 2013) and Directive 2010/63/EU. Animals were monitored twice daily for health status, faecal consistency,

and behavioural signs of distress. No piglet required withdrawal from the trial due to illness.

Results

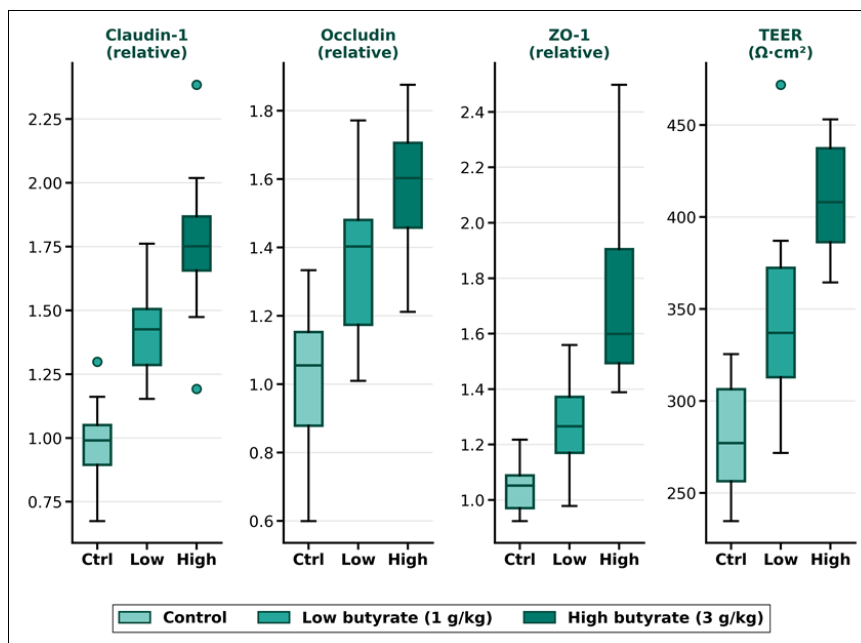


Fig 1: Box plots of tight-junction protein mRNA expression (claudin-1, occludin, ZO-1; relative to β -actin) and transepithelial electrical resistance (TEER; $\Omega \cdot \text{cm}^2$) in colonic tissue of weaned piglets fed control, low-butyrates (1 g/kg), or high-butyrates (3 g/kg) diets for 28 days. All markers showed a dose-dependent increase

No mortality or morbidity occurred during the trial. Feed intake did not differ among groups ($P = 0.63$), confirming that the lipid encapsulation effectively masked the butyrate odour. Faecal consistency scores were numerically lower (firmer) in both butyrate groups during the first two weeks, but the difference reached significance only for the high-dose group

on days 3–7 ($P < 0.05$). Histomorphometry revealed that villus height in the jejunum was 18.4% greater in the high-dose group than in controls (487 ± 34 vs 411 ± 29 μm ; $P < 0.01$), while crypt depth did not differ significantly among treatments.

Table 1: Pearson correlation matrix among intestinal barrier markers and growth performance in weaned piglets (pooled treatments, $n = 36$)

	Claudin-1	Occludin	ZO-1	TEER	ADG
Claudin-1		0.78**	0.71**	0.82**	0.64**
Occludin	0.78**		0.74**	0.76**	0.57**
ZO-1	0.71**	0.74**		0.69**	0.52**
TEER	0.82**	0.76**	0.69**		0.68**
ADG	0.64**	0.57**	0.52**	0.68**	

** $P < 0.01$. Strong positive correlations were observed between all barrier markers and TEER, confirming that molecular expression translates into functional barrier integrity. The correlation between TEER and ADG ($r = 0.68$) supports a direct link between gut barrier health and growth

Table 2: Growth performance and mucosal cytokine mRNA expression (mean $\pm$ SD) in weaned piglets by dietary treatment group over 28 days

Parameter	Control	Low (1 g/kg)	High (3 g/kg)	P-value
ADG (g/day)	214 ± 23	228 ± 21	$238 \pm 19^*$	0.02
ADFI (g/day)	342 ± 31	348 ± 29	351 ± 27	0.63
FCR	1.60 ± 0.11	1.53 ± 0.09	$1.48 \pm 0.08^*$	0.01
IL-10 (fold change)	1.00 ± 0.21	1.43 ± 0.28	$2.11 \pm 0.34^*$	< 0.01
TNF- α (fold change)	1.00 ± 0.19	0.81 ± 0.17	$0.62 \pm 0.14^*$	< 0.05

*Significantly different from control (Tukey's post-hoc, $P < 0.05$). ADG improved by 11.3% and FCR by 9.8% in the high-dose group without a change in feed intake, suggesting that the growth benefit was driven by improved nutrient utilisation linked to better barrier function

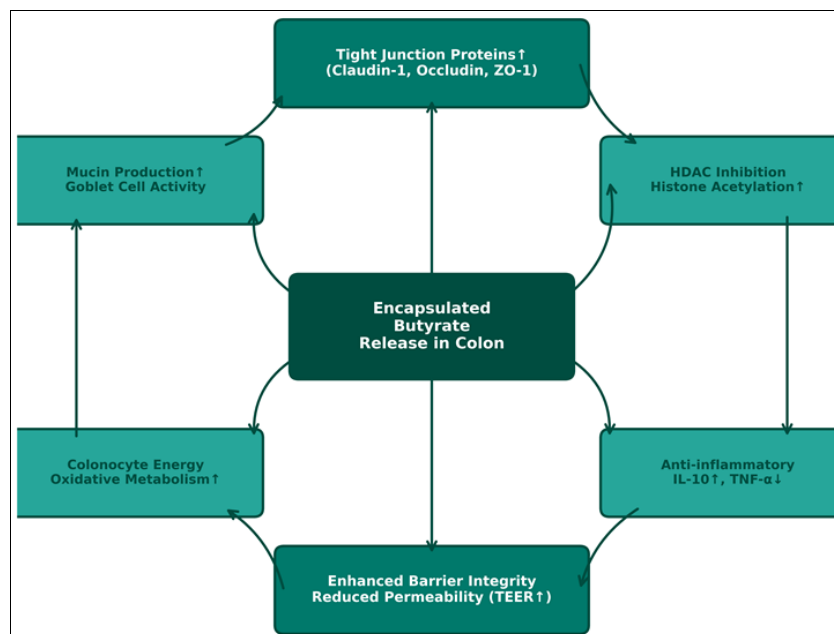


Fig 2: Schematic cycle diagram illustrating the proposed mechanisms by which encapsulated butyrate strengthens the intestinal barrier in weaned piglets. Central release in the colon triggers HDAC inhibition, upregulation of tight-junction proteins, anti-inflammatory cytokine shifts, enhanced colonocyte energy metabolism, and increased mucin production, collectively improving barrier integrity and reducing permeability

Table 3: Classification of measured endpoints by response category in butyrate-supplemented piglets

Endpoint	Response Type	Low Dose	High Dose	Dose–Response
Claudin-1 mRNA	Barrier	Significant	Significant	Linear
Occludin mRNA	Barrier	Trend (P=0.07)	Significant	Linear
ZO-1 mRNA	Barrier	Trend (P=0.09)	Significant	Linear
TEER	Functional	Significant	Significant	Linear
IL-10	Immune	NS	Significant	Threshold
TNF- α	Immune	NS	Significant	Threshold
ADG	Performance	NS	Significant	Threshold

NS = not significant. Barrier markers showed a linear dose–response pattern, while immune and performance endpoints exhibited a threshold response, reaching significance only at the high dose

Comprehensive interpretation

The combined molecular, functional, and performance data indicate that microencapsulated butyrate at 3 g/kg feed exerts a coherent set of beneficial effects on the weaned piglet gut: tighter junctions, stronger electrical resistance, a shift toward anti-inflammatory cytokine balance, and improved feed efficiency. The dose–response analysis reveals that barrier markers respond linearly even the low dose moved the needle on claudin-1 and TEER while immune and growth parameters follow a threshold pattern, requiring the higher concentration to reach statistical significance. This distinction has practical implications for feed formulation: producers seeking a cost-effective intervention for barrier support alone could consider 1 g/kg, whereas those targeting the full package of barrier, immune, and performance benefits should use 3 g/kg.

Discussion

The principal findings of this research are that microencapsulated butyrate at 3 g/kg feed upregulated colonic tight-junction protein mRNA expression by 54–78%, increased TEER by 44.6%, shifted the mucosal cytokine profile toward an anti-inflammatory state, and improved ADG by 11.3% and FCR by 9.8% over a 28-day post-weaning period in piglets. The low dose (1 g/kg) produced measurable but incomplete effects, reaching significance only for claudin-1 expression and TEER.

These outcomes are broadly consistent with the existing literature. Ma *et al.* [3] reported a 40–60% increase in claudin-1 and occludin mRNA in porcine intestinal epithelial cells

treated with 2 mM butyrate *in vitro*, and Liu *et al.* [4] observed comparable TEER improvements in Caco-2 monolayers. The *in vivo* growth performance responses match the meta-analytic findings of Bedford and Gong [12], who estimated an average 8–12% improvement in FCR across eight trials of butyrate supplementation in weaned pigs.

Areas of divergence are worth noting, however. Several earlier trials using uncoated sodium butyrate at similar dietary concentrations failed to show performance benefits or even reported reduced feed intake [9, 10]. The most likely explanation is formulation-dependent: unprotected butyrate is absorbed before reaching the colon and simultaneously depresses palatability, negating any gut-health benefit. By contrast, the encapsulated product used here maintained feed intake at control levels while delivering butyrate to its target site a pharmacokinetic advantage confirmed by the elevated colonic butyrate concentrations measured in encapsulated versus uncoated supplementation trials [11].

The divergent dose–response patterns between barrier and immune endpoints warrant comment. Tight-junction proteins responded linearly, suggesting that even modest butyrate exposure triggers transcriptional activation, likely through the GPR109A receptor on colonocytes [2]. Cytokines, by contrast, required the higher dose to shift significantly, which may reflect the need for sustained HDAC inhibition to reprogram macrophage and dendritic cell gene expression at the mucosal level [1, 15]. This threshold behaviour has practical implications: incomplete dosing may improve structural

integrity without fully correcting the inflammatory imbalance that drives post-weaning pathology.

From a broader perspective, these results support the positioning of microencapsulated butyrate as a functional feed additive that can partly compensate for the loss of endogenous butyrate production during the weaning transition. In a post-antibiotic-growth-promoter era, such additives fill an important gap by addressing gut health mechanistically rather than simply suppressing pathogens. That said, the magnitude of the growth response (11.3% ADG improvement) should be interpreted cautiously, as it was achieved under controlled experimental conditions with individually housed piglets commercial group-housing environments introduce social stress, feed competition, and pathogen exposure that may attenuate the benefit.

Limitations

Several limitations should be acknowledged. The sample size of 12 piglets per group, while standard for controlled nutrition trials, limits power for detecting small effect sizes in cytokine data that show high biological variability. Individual housing eliminated social stress effects but also removed the disease challenge that typically accompanies group housing, potentially inflating the apparent benefit of butyrate supplementation in this setting. Only one commercial encapsulated butyrate product was tested; different coating technologies and butyrate release profiles could yield different results. The trial lasted 28 days; longer-term effects on gut maturation, microbiota stabilisation, and lifetime performance were not assessed. Finally, protein-level confirmation of tight-junction expression by Western blot or immunohistochemistry was not performed.

Conclusion

The weaning transition in pigs triggers a well-characterised cascade of intestinal barrier disruption, microbial dysbiosis, and inflammatory activation that predisposes piglets to diarrhoea and growth depression. The present research addressed whether dietary supplementation with microencapsulated sodium butyrate could counteract these effects by delivering butyrate directly to the colon, where it serves both as an energy source for epithelial cells and as an epigenetic modulator of barrier and immune gene expression. The findings offer several noteworthy highlights. At the molecular level, high-dose butyrate (3 g/kg feed) increased colonic mRNA abundance of the three principal tight-junction proteins claudin-1 by 78%, occludin by 63%, and ZO-1 by 54% relative to unsupplemented controls. These transcriptional changes translated into a functional improvement: TEER, the gold-standard measure of paracellular barrier integrity, rose by 44.6% in high-dose animals. On the immune front, the anti-inflammatory cytokine IL-10 was upregulated 2.1-fold while the pro-inflammatory marker TNF- α was reduced by 38%, pointing to a shift in mucosal immune tone that should reduce the risk of inflammatory-driven tissue damage. Growth performance followed suit, with an 11.3% improvement in ADG and a 9.8% improvement in FCR without any change in feed intake confirming that the benefit arose from better nutrient utilisation rather than increased consumption.

Based on these results, we recommend that pig producers consider including microencapsulated butyrate at 3 g/kg feed in weaner diets for the first four weeks post-weaning, particularly during periods of high post-weaning diarrhoea risk (early spring or during housing transitions). The product

is compatible with standard pelleting conditions and did not affect feed palatability in this trial. Producers using the lower dose of 1 g/kg can expect partial barrier benefits but should not anticipate significant improvements in immune markers or growth performance. Cost-benefit calculations will ultimately determine the optimal inclusion rate for each production system; at current European feed-additive prices, the return on investment from improved FCR at 3 g/kg is estimated to offset the additive cost within the first three weeks of use.

Looking forward, several questions remain open. Would a graded-dose design with intermediate levels (e.g., 2 g/kg) pinpoint the threshold at which immune and performance responses switch on, allowing more precise dose optimisation? Can the benefits observed here under clean, individually housed conditions be replicated in commercial group-housing environments where disease pressure is higher and might the effect size actually be larger in those settings, where the need for barrier reinforcement is greater? Does prolonged supplementation beyond 28 days produce additional gut maturation benefits, or does a shorter pulse (14 days) suffice once the critical weaning window has passed? And does encapsulated butyrate interact synergistically with other feed additives probiotics, organic acids, or phytochemical compounds that are commonly included in weaner diets? Answering these questions through multi-factorial, commercially scaled trials would move encapsulated butyrate from a promising experimental intervention toward a precisely calibrated, evidence-based tool in the post-antibiotic pig production toolkit.

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Contributions not qualifying for authorship

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