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Transcriptomic Association Between Poliovirus Receptor (PVR/CD155) and Claudin Signaling Pathways in Colorectal Cancer

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Abstract

Background/Aim: Enterotoxigenic *Bacteroides fragilis* promotes colorectal carcinogenesis through toxin-mediated cleavage of E-cadherin, a process facilitated by membrane-associated Claudin-4 (CLDN4). Separately, the poliovirus receptor (PVR/CD155) modulates tumor epithelial and immune dynamics. This study explored potential transcriptomic interactions and co-expression frameworks between PVR and claudin signaling pathways in colorectal cancer.

Materials and Methods: Transcriptomic and proteomic data from the The Cancer Genome Atlas-colon adenocarcinoma cohort (TCGA-COAD) were evaluated. An exploratory E-cadherin Cleavage Index was modeled to capture transcript-protein discordance. To control for tissue composition heterogeneity without mathematical circularity, a de-circularized, non-parametric partial rank residual model adjusted for independent CLDN4 expression was deployed within the stable microsatellite-stable (MSS) sub-cohort (N=473).

Results: Multivariable survival models showed no independent associations between overall survival and continuous PVR (p=0.79) or CLDN3 (p=0.56) expression. Robust linear modeling revealed no significant baseline interaction between PVR and CLDN4 regarding the exploratory Cleavage Index (p=0.82). However, de-circularized partial correlation analysis revealed a highly stable, positive co-expression between PVR and CLDN3 (rho=0.2459, $p=3.23\times10^{-7}$). Both epithelial markers retained modest inverse correlations with the infiltrating lymphocytic axis (TIGIT and CD96).

Conclusion: Baseline PVR expression is coordinated with CLDN3 tissue programs independent of general epithelial cellularity but does not interact with the CLDN4 axis or impact overall survival in an unexposed cohort. Because TCGA lacks virome or active microbial exposure tracking, these findings serve as baseline benchmarks for future context-dependent mechanistic studies.

Keywords: Colorectal cancer, poliovirus receptor, CD155, Claudin-4, Claudin-3, Bacteroides fragilis toxin, E-cadherin.

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Introduction

Colorectal cancer (CRC) remains a major cause of cancer-related mortality worldwide. In addition to dietary and lifestyle risk factors, increasing evidence suggests that interactions between the intestinal epithelium and the microbiome contribute to colorectal carcinogenesis (1).

Enterotoxigenic *Bacteroides fragilis* (ETBF) has emerged as a microbial contributor to CRC development. ETBF secretes *Bacteroides fragilis* toxin (BFT), a zinc-dependent metalloprotease capable of disrupting epithelial integrity through E-cadherin cleavage. Recent structural studies identified Claudin-4 (CLDN4) as a membrane-associated binding partner facilitating localization of BFT and subsequent epithelial junction disruption (2-4). These processes may contribute to β-catenin activation, inflammatory signaling, and tumor-promoting immune responses (5, 6).

Separately, historical ecological analyses have reported inverse correlations between regional poliomyelitis incidence or oral poliovirus vaccination exposure and subsequent colorectal cancer mortality (7). Although intriguing, ecological associations cannot establish causality and remain highly vulnerable to confounding related to demographic variation, screening practices, migration, and secular changes in lifestyle-associated risk factors.

The poliovirus receptor (PVR/CD155) is an adhesion-related immunoglobulin superfamily protein frequently expressed in epithelial malignancies, including colorectal cancer. PVR has recognized roles in immune regulation, cellular adhesion, and membrane-associated signaling pathways (8). Whether PVR-associated transcriptional programs relate to claudin-associated epithelial biology in colorectal tumors remains unclear.

The present study was designed as an exploratory computational analysis to evaluate whether baseline transcriptomic associations exist between PVR expression and claudin-associated epithelial signaling patterns in The Cancer Genome Atlas (TCGA) colorectal tumors.

Materials and Methods

Data acquisition. Transcriptomic and proteomic data from The Cancer Genome Atlas (TCGA) colon adenocarcinoma (TCGA-COAD) were obtained through the National Cancer Institute Genomic Data Commons using the TCGAbiolinks R package. RNA sequencing data were derived from upper-quartile normalized Fragments Per Kilobase of transcript per Million mapped reads upper quartile (FPKM-UQ) counts. Proteomic measurements were obtained from reverse phase protein array (RPPA) datasets. Patient-level identifiers were harmonized by truncating TCGA barcodes to the first 12 characters. Replicate profiles were averaged when present.

Gene and protein mapping. The following Ensembl identifiers were used: CDH1 (E-cadherin): ENSG00000039068; CLDN4 (Claudin-4): ENSG00000189146; CLDN3 (Claudin-3): ENSG00000169855; PVR (CD155): ENSG00000073008. RPPA peptide targets corresponding to E-cadherin were identified using case-insensitive regular expression matching.

Construction of the exploratory cleavage index. To model discordance between CDH1 transcript abundance and E-cadherin protein expression, we defined an exploratory Cleavage Index: Cleavage Index=log2(CDH1 mRNA + 1) / [E-cadherin protein – minimum (E-cadherin protein) + 0.1]. The denominator adjustment was introduced to stabilize values across negative RPPA measurements. This index was designed as a hypothesis-generating surrogate integrating transcriptomic and proteomic measurements and has not been validated against direct biochemical measurements of extracellular E-cadherin cleavage or membrane-localized protein loss. Both CLDN3 and CLDN4 were examined as relevant claudin family members, with CLDN4 used for the interaction model based on its established role in BFT binding and CLDN3 examined for co-expression given its epithelial expression patterns.

Ethics. The present study analyzed publicly available, de-identified transcriptomic and proteomic data from TCGA and the National Cancer Institute Genomic Data Commons (GDC).

All data were previously collected under protocols approved by participating institutions within TCGA. The current secondary analysis of de-identified public data did not require additional institutional review board approval or informed consent.

Statistical analysis. The primary model evaluated whether PVR expression modified the relationship between CLDN4 expression and the exploratory Cleavage Index: $Y=\beta0 + \beta1(CLDN4) + \beta2(PVR) + \beta3(CLDN4 \times PVR) + \varepsilon$.

Three analytical stages were performed: (i) Ordinary least squares regression in the unstratified cohort; (ii) Robust linear modeling using M-estimation to reduce the influence of outliers; (iii) Analysis restricted to microsatellite-stable (MSS) and microsatellite instability (MSI)-low tumors to reduce biological heterogeneity. Co-expression analysis between PVR and CLDN3 transcript abundance was evaluated using Pearson correlation and ordinary least squares regression. Overall survival was assessed using Kaplan-Meier analysis and multivariable Cox proportional hazards regression adjusting for age, sex, and tumor stage. All analyses were performed using R 4.5.2 (The R Foundation for Statistical Computing, Vienna, Austria).

Results

Unstratified cohort analysis. Initial ordinary least squares regression in the full TCGA-COAD cohort demonstrated residual skewing caused by an extreme outlier with a markedly elevated Cleavage Index. The interaction coefficient between CLDN4 and PVR expression was not statistically significant (Figure 1). Application of robust linear modeling reduced variance inflation and stabilized coefficient estimates. Under robust estimation, the interaction term remained small and statistically non-significant.

MSS/MSI-low subset analysis. Restriction to MSS/MSI-low tumors reduced residual heterogeneity and yielded the coefficients shown in Table I. The interaction term remained statistically non-significant, with confidence intervals compatible with small positive or negative effects. These findings indicate that baseline transcript abundances of PVR and CLDN4 did not demonstrate detectable interaction in TCGA colorectal tumors.

Survival analysis. Kaplan-Meier analysis demonstrated no significant difference in overall survival between tumors stratified by median PVR expression (log-rank p=0.9; Figure 2). To further evaluate whether continuous transcript abundance was associated with prognosis, multivariable Cox proportional hazards modeling was performed adjusting for age, sex, and tumor stage (Figure 3). Continuous PVR expression was not significantly associated with overall survival [hazard ratio (HR)=1.054, 95% confidence interval (CI)=0.72-1.55, p=0.7887], nor was CLDN3 expression (HR=1.075, 95%CI=0.84-1.37, p=0.56). Age at diagnosis remained significantly associated with mortality risk (HR=1.026, 95%CI=1.01-1.04, p=0.0035). These findings indicate that baseline variation in PVR and CLDN3 transcript abundance was not independently associated with survival outcomes within TCGA-COAD.

PVR and CLDN3 co-expression. Across TCGA-COAD tumors, PVR and CLDN3 transcript abundance demonstrated a modest positive correlation (Pearson r=0.3100, 95%CI=0.224-0.391, $p=6.57\times10^{-13}$; Figure 4). Although statistically significant, the magnitude of the correlation was moderate and may reflect shared epithelial transcriptional programs, tumor composition, or other biological factors not directly assessed in this study.

To isolate the intrinsic relationship between the tight-junction component *CLDN3* and the immune-evasive ligand *PVR* independent of macroscopic tissue composition or varying epithelial cell density across bulk tumor biopsies, we performed a non-parametric partial correlation analysis using rank-order residuals (9) across the stable MSS cohort (N=473). Rather than using internal combination metrics, the models were conditioned on *CLDN4 (average)* expression derived from an independent tracking matrix to serve as an uncoupled proxy for general epithelial tissue cellularity. Under this de-circularized framework, a statistically significant positive co-expression pattern

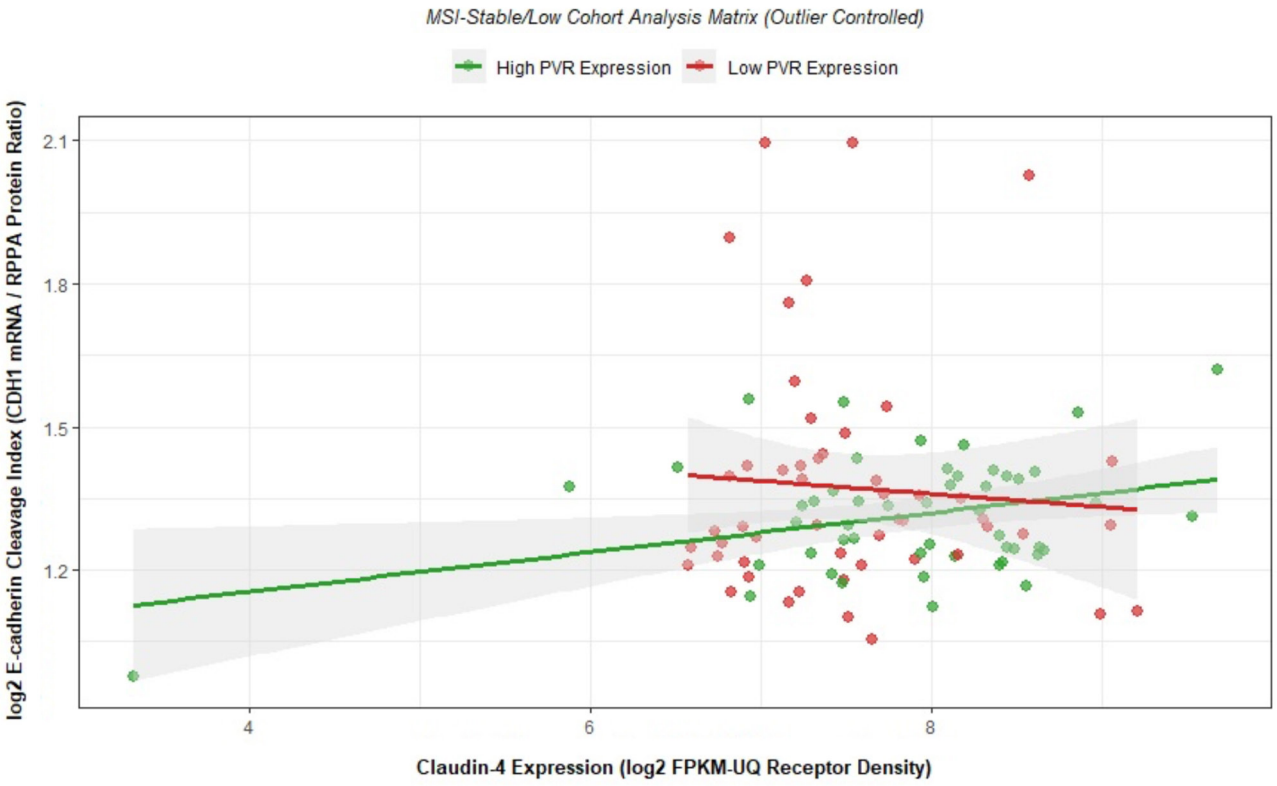

Figure 1. *Relationship between CLDN4 expression and the exploratory E-cadherin cleavage index in microsatellite-stable/microsatellite instability-low (MSS/MSI-low) TCGA-colon adenocarcinoma (COAD) tumors stratified by median polio virus receptor (PVR) expression. Scatter plot demonstrating a log-transformed relationship between CLDN4 transcript abundance and the exploratory E-cadherin Cleavage Index in microsatellite-stable and MSI-low TCGA-COAD tumors. Tumors were stratified by median PVR expression into high-PVR (green) and low-PVR (red) groups. Regression lines with shaded 95% confidence intervals are shown for each subgroup. No statistically significant interaction between CLDN4 and PVR expression was identified in relation to the exploratory Cleavage Index.*

Table I. *Interaction model coefficients for CLDN4 and polio virus receptor (PVR) expression in the microsatellite-stable/microsatellite instability-low patient sub-cohort.*

Variable	Estimate	Standard error	*t* Value
Intercept	1.7993	1.9121	0.9410
log2_CLDN4	−0.0195	0.2527	−0.0773
log2_PVR	−0.1614	0.5100	−0.3164
log2_CLDN4 × log2_PVR	0.0155	0.0669	0.2321

was preserved between *PVR* and *CLDN3* (rho=0.2459, $p=3.23\times10^{-7}$). Both epithelial components maintained modest, independent inverse relationships with the infiltrating lymphocytic receptor axis (*PVR vs. TIGIT*: rho=-0.1350; *CLDN3 vs. CD96*: rho=-0.0919). These exploratory data indicate that the baseline transcriptomic coordination between *PVR* and *CLDN3* is an independent architectural feature of these tumor profiles, distinct from artifactual index overlap or variations in advancing tissue dedifferentiation (Figure 5).

Exploratory stratification by age at diagnosis, excluding intermediate ages (51-64 years; n=171), suggested that PVR-CLDN3 partial correlations may differ numerically between early-onset (≤50 years; n=48; ρ=0.39, $p=0.007$) and late-onset (≥65 years; n=254; ρ=0.31, $p<0.001$) cohorts; however, formal interaction testing did not reach significance ($p=0.921$). Given the limited sample size in the early-onset stratum, the absence of interaction effect, and

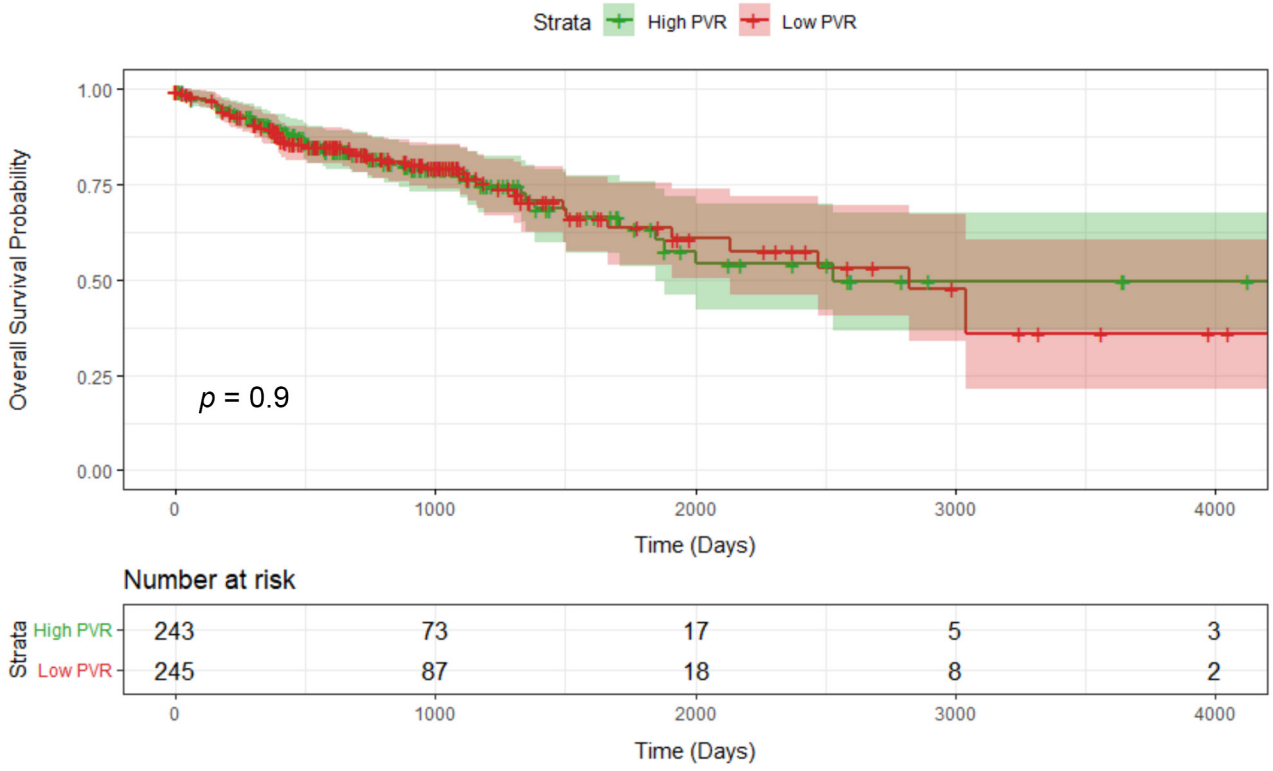

Figure 2. *Kaplan-Meier estimates of overall survival stratified by median polio virus receptor (PVR) expression in TCGA-colon adenocarcinoma (COAD). Kaplan-Meier survival curves compare overall survival between tumors with high (green line; n=243) and low (red line; n=245). PVR transcript abundance is based on median cohort expression values. Shaded regions represent 95% confidence intervals. The accompanying number-at-risk table summarizes patient follow-up across the observation period. No statistically significant difference in overall survival was observed between the two groups (log-rank p=0.9).*

the lack of exposure data in TCGA, this observation requires validation in independent cohorts with documented clinical histories.

To evaluate whether embryonic tissue origin influenced the observed associations, a secondary analysis stratified the MSS cohort into proximal midgut-derived subsites (cecum, ascending colon, and hepatic flexure; n=154) and distal hindgut-derived subsites (splenic flexure, descending colon, sigmoid colon, rectosigmoid junction, and rectum; n=108, anatomical subsite data were available for 302 of 473 MSS tumors). Transverse colon cases (n=40) were excluded due to ambiguous embryonic classification. De-circularized partial correlation models, adjusting for CLDN4 expression, demonstrated similar PVR-CLDN3 associations in both distal (ρ=0.29, p=0.002) and proximal (ρ=0.29, p<0.001) subsites; formal interaction testing was not significant (p=0.847). These findings suggest that the observed co-expression relationship is consistent across microsatellite-stable colorectal tumors regardless of anatomical location.

Discussion

In this exploratory analysis of TCGA colorectal cancer data, we observed a modest positive correlation between PVR and CLDN3 transcript abundance but found no significant interaction between PVR and CLDN4 expression in relation to an exploratory E-cadherin Cleavage Index. In addition, neither PVR nor CLDN3 expression was independently associated with overall survival.

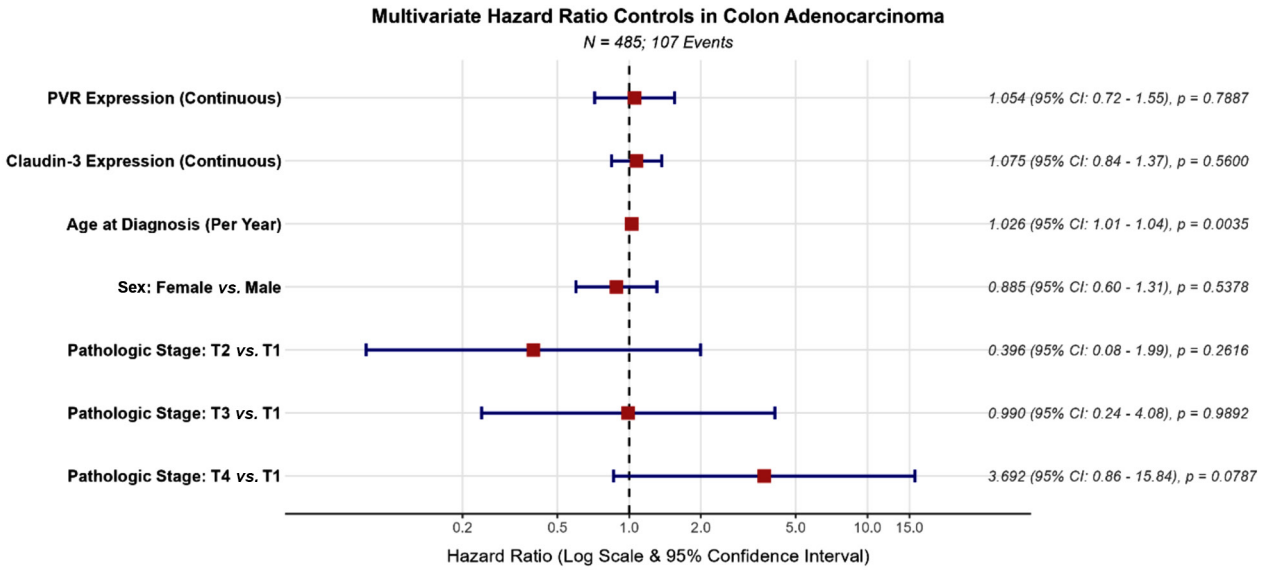

Figure 3. *Multivariable Cox proportional hazards analysis in TCGA colon adenocarcinoma. Forest plot displays adjusted hazard ratios and 95% confidence intervals derived from multivariable Cox proportional hazards regression models evaluating continuous transcriptomic variables and clinical covariates (n=485 patients; 107 survival events). Continuous polio virus receptor (PVR) expression (HR=1.054, 95%CI=0.72-1.55, p=0.7887) and CLDN3 expression (HR=1.075, 95%CI=0.84-1.37, p=0.5600) were not significantly associated with overall survival. Age at diagnosis remained significantly associated with mortality risk (HR=1.026, 95%CI=1.01-1.04, p=0.0035). Hazard ratios are displayed on a logarithmic scale.*

Recent experimental and structural studies have characterized the roles of CLDN3 and CLDN4 in BFT-mediated E-cadherin cleavage, identifying CLDN4 as the primary high-affinity receptor and CLDN3 as a partial compensatory binding partner (4). Structural analyses demonstrated that the Extracellular Segment (ECS1) sequence of CLDN4 differs from CLDN3 by only three amino acids (T33S, V41I, and T45N), with the T45N substitution abolishing stable BFT binding (5). The observed co-expression between PVR and CLDN3 in TCGA tumors may reflect shared epithelial differentiation programs, common upstream transcriptional regulation, or tumor composition. However, whether this co-expression has functional relevance in the context of microbial exposure cannot be determined from transcriptomic data alone, and PVR has not been implicated in BFT-mediated signaling.

Importantly, TCGA datasets do not contain information regarding active enterotoxigenic *Bacteroides fragilis* colonization, bacterial toxin exposure, enteroviral infection status, or prior poliovirus immunity. Consequently, the present findings cannot directly evaluate whether microbial or viral exposures modify epithelial signaling pathways involving PVR or claudins.

The absence of a detectable baseline interaction between PVR and CLDN4 expression should therefore be interpreted cautiously. The findings neither establish nor exclude biologically relevant context-dependent interactions under conditions not represented within TCGA tissue repositories.

The exploratory E-cadherin Cleavage Index used in this study also requires careful interpretation. This construct was designed as a hypothesis-generating surrogate integrating transcriptomic and proteomic measurements and has not been validated against direct biochemical measurements of E-cadherin cleavage or membrane localization.

Prior ecological observations suggesting inverse relationships between poliovirus exposure and colorectal cancer incidence or mortality remain difficult to interpret because of potential confounding related to vaccination practices, demographics, healthcare access, and secular

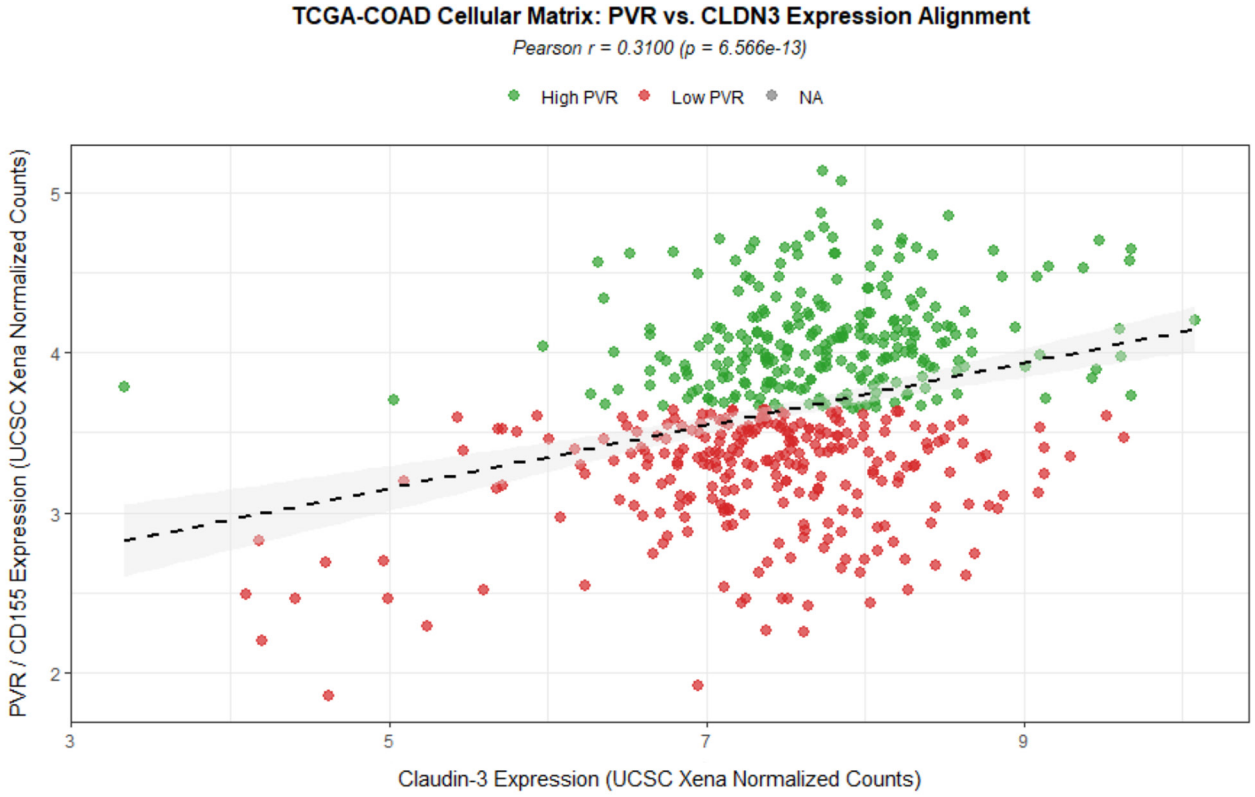

Figure 4. *Co-expression relationship between polio virus receptor (PVR) and CLDN3 in TCGA- colon adenocarcinoma (COAD) tumors. Scatter plot demonstrates the relationship between normalized PVR (CD155) and CLDN3 transcript abundance across TCGA colon adenocarcinoma samples. The dashed line represents the ordinary least squares regression fit, and the shaded ribbon indicates the 95% confidence interval. Tumors are stratified by median PVR expression into high-PVR (green) and low-PVR (red) groups. PVR and CLDN3 expression demonstrated a modest positive correlation across tumors (Pearson r=0.3100, p=6.57×10^{-13}).*

changes in colorectal cancer risk factors. The present study was not designed to test those epidemiological associations directly. Nevertheless, the synchronized tissue framework, CLDN3, CLDN4, PVR, takes on added mechanistic significance when integrated alongside recent evidence characterizing multi-kingdom viral-bacterial partnerships within the colorectal niche. Specifically, Damgaard *et al.* (2026) demonstrated that colorectal cancer-associated *Bacteroides fragilis* driver strains are infected with unique, highly specific Caudoviricetes prophages (*Bacteroides phage FU* and *ODE*; OR=2.05, p=2.52×10^{-7}) that facilitate distinct pathobiological or lysogenic conversions (10). We hypothesize that such upstream prophage-mediated alterations expand the local enterotoxigenic secretome, subjecting neighboring epithelial cells to chronic, localized toxin-mediated barrier stress. Our findings demonstrate that under these structural pressures, the mucosal epithelium maintains a highly synchronized *PVR-CLDN3* transcriptomic coordination axis (rho=0.2459). While this baseline coordination likely represents an intrinsic epithelial response to microenvironmental disruption, it concurrently provides an intact *PVR* checkpoint shield that suppresses localized immune surveillance via the *TIGIT/CD96* pathway. Crucially, this structural-immune checkpoint coupling may represent a critical point of vulnerability. In birth cohorts with historical exposure to live-attenuated oral poliovirus vaccines or matching sub-

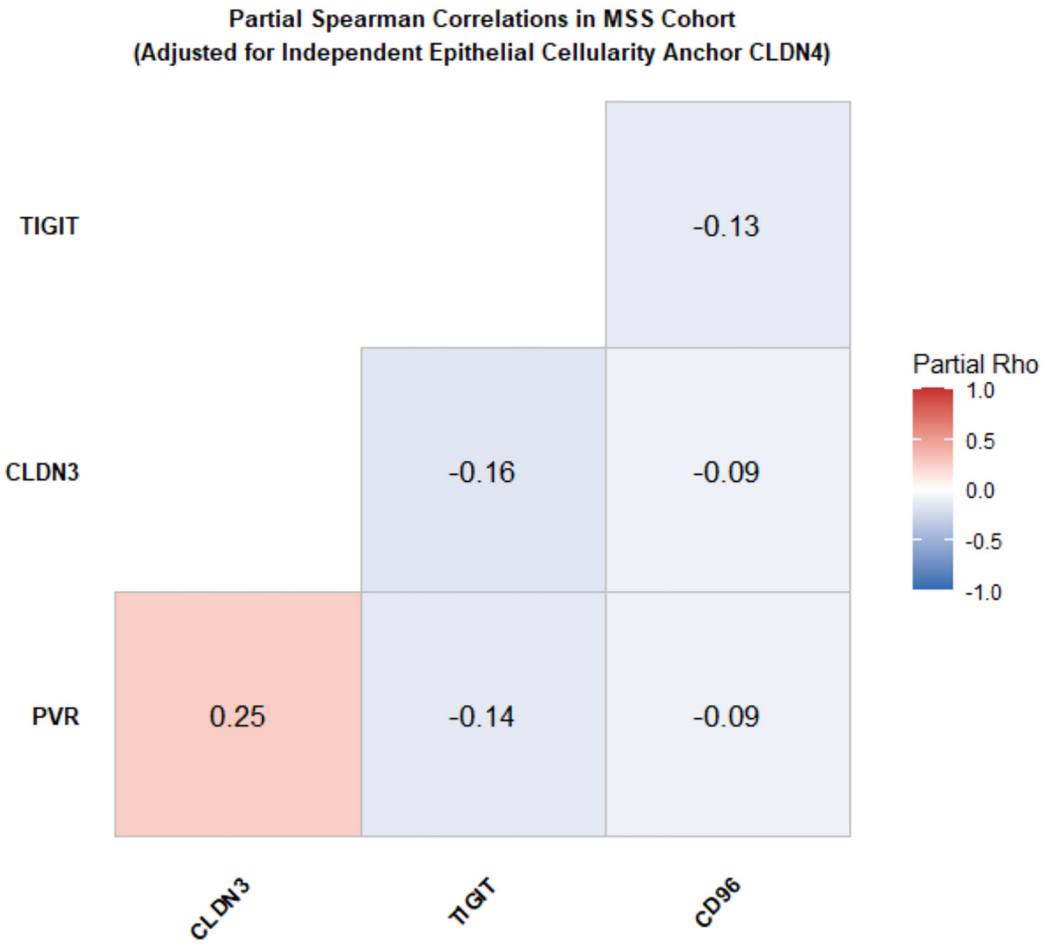

Figure 5. *Partial Spearman correlation matrix of epithelial and lymphocytic axis transcripts. Lower triangular heatmap displaying adjusted non-parametric Spearman rank-order residual correlation coefficients (rho) across the primary microsatellite-stable (MSS) patient sub-cohort (N=473). Values are mathematically adjusted using a rank-order residual framework to control for variation in bulk tissue epithelial content, using CLDN4 (average) expression as an independent, non-circular conditioning covariate. A stable, statistically significant positive co-expression pattern is preserved between the tight-junction component CLDN3 and the ligand PVR (rho=0.2459, p=3.23×10^{-7}), demonstrating that their transcriptomic coordination persists independent of general tissue cellularity. Concurrently, both structural markers exhibit modest, independent inverse relationships with the infiltrating lymphocytic receptor axis (TIGIT and CD96), consistent with expected co-expression patterns in bulk tumor transcriptomic profiles. Color intensity scales with the direction and magnitude of the partial rank coefficient are to the right; text inserts denote explicit rounded rho values.*

clinical enteroviral strains, these *PVR*-dense mucosal sites serve as natural targets for viral-mediated oncolytic lysis and host immune-priming. Conversely, in sanitized, modern cohorts lacking these enteric viral clearing pressures, this prophage-driven bacterial disruption and matching *PVR* checkpoint synchronization can proceed entirely unchecked, offering a plausible translational framework for the rising incidence of early-onset distal malignancies.

Future studies integrating microbiome profiling, spatial transcriptomics, direct toxin exposure models, and experimental validation systems will be necessary to determine whether microbial factors influence PVR-associated epithelial signaling in colorectal carcinogenesis.

Study limitations. First, the study lacked direct microbiome or virome data. Neither ETBF colonization status nor enteroviral

exposure history was available within TCGA. Second, the exploratory Cleavage Index has not been validated against direct biochemical measurements of extracellular E-cadherin cleavage or membrane protein localization. Third, bulk RNA sequencing integrates epithelial, stromal, and immune cell populations, introducing biological heterogeneity (11).

Fourth, the MSS/MSI-low subset sample size was modest (n=473), resulting in broad confidence intervals compatible with small positive or negative effects. Fifth, the epidemiological observations motivating the poliovirus/colorectal cancer inverse relationship hypothesis are ecological in nature and vulnerable to substantial confoundings, including demographic shifts, migration, screening practices, and secular changes in colorectal cancer risk factors. Finally, the study design cannot distinguish between biologically dormant pathway interactions and the absence of a true mechanistic relationship.

Conclusion

This exploratory TCGA analysis identified modest co-expression between PVR and CLDN3 transcripts in colorectal tumors but did not identify significant baseline interactions between PVR and CLDN4 expression or associations with overall survival. Because TCGA lacks microbial exposure and virome data, these findings should not be interpreted as evidence for or against context-dependent biological interactions. Experimental and microbiome-integrated studies will be required to determine whether microbial or viral exposures influence PVR-associated epithelial signaling in colorectal carcinogenesis.

Conflicts of Interest

The Authors declare no competing interests in relation to this study.

Authors' Contributions

Steven Lehrer: Conceptualization, methodology, data analysis, statistical analysis, interpretation of results, visualization, and manuscript writing. Peter H. Rheinstein: Interpretation of results, critical revision of the manuscript, and supervision. Both Authors reviewed and approved the final manuscript.

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None.

Artificial Intelligence (AI) Disclosure

During the preparation of this manuscript, a large language model (ChatGPT, OpenAI) was used solely for language editing and stylistic improvements in select paragraphs. No sections involving the generation, analysis, or interpretation of research data were produced by generative AI. All scientific content was created and verified by the authors. Furthermore, no figures or visual data were generated or modified using generative AI or machine learning-based image enhancement tools.

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