Supplementary information S1 (table) **| The role of pattern recognition receptors (PRRs) in regulating intestinal epithelial cells (IECs) and tissue homeostasis.**

PRRs and signalling adaptors in IECs		In vivo effect on IECs
TLRs	TLR1[1,2]	ND
	TLR2[3–7]	• Protects IECs from injury[3] • Enhances M cell transport[4] • Promotes gap junction communication[6] • Increased TFF3 production[7] • Promotes tight junction integrity[5]
	TLR3[8,9]	• Promotes IEC IL-15 production and IEL cytotoxicity[8,9]
	TLR4[3,10–14]	• Protects IECs from injury[3] • Promotes tumour development and growth[12,14] • Increases prostaglandin production[11] • Promotes necrotizing enterocolitis (in neonates)[15,16] • Increases serum amyloid A production[17] • Enhances M cell transport[4]
	TLR5[18–23]	• Promotes chemokine and cytokine production[18] • Protects against apoptosis[19,21] • Limits IL-1β mediated inflammation[22] • Prevents bacterial overgrowth[20]
	TLR9[16,24–28]	• Inhibits NF-κB activation and tolerizes subsequent TLR signalling[25] • Protects against necrotizing enterocolitis (in neonates)[16] • Promotes Paneth cell degranulation[24] • Promotes AMP production[24,26] • Protects IECs from injury[16,25,27,28]
	TLR10[29]	• ND
	TLR11*[30–32]	• Prevents epithelial entry and dissemination of pathogenic *Salmonella* species[31,32]
	TRIF[33]	• Promotes IEC apoptosis[33]
	MYD88[3,34–43]	• Protects IECs from injury[3,38] • Promotes AMP secretion[37,41,42] • Increases mucin production[42] • Promotes prostaglandin production[38] • Promotes tumour formation and growth[36,39] • Increases IEC IL-15 to promote IEL survival[34] • Promotes APRIL and BAFF production to promote B cell IgA production[35,44] • Increases pIgR expression[42] • Promotes IEC autophagy and defence against intracellular bacteria[43]
	TAK1[45–48]	• Prevents IEC apoptosis[45–48] • Promotes IEC proliferation[45,48] • Promotes tight junction formation[46] • Limits accumulation of ROS[46,47]
	TAB1–TAB2[47]	• Limits accumulation of ROS[47]
RLRs	RIG-I[49,50]	• ND
	MDA5[50]	• Promotes interferon production[50]
	MAVS[50,51]	• Protects IECs from injury[51]
NLRs	NOD1[52–54]	• Protects IECs from injury[53] • Inhibits tumour development[53] • Induces intestinal lymphoid tissue formation[52] • Recruits autophagy proteins[54]
	NOD2[54–59]	• Recruits autophagy proteins[54] • Promotes AMP production[57,58] • Promotes ROS production[59]
	NLRP3[60–66]	• Inhibits tumour development[61,64] • Promotes or limits colitis (depending on circumstances)[61–63,65] • Promotes AMP production[66] • Regulates commensal microbial composition[66]
	NLRP6[65,67–69]	• Protects IECs from injury[67,68] • Promotes colonic wound healing[68] • Inhibits tumour development [67,68] • Regulates commensal microbial composition[65] • Inhibits NF-κB and MAP kinase responses to bacterial infection[69]

NLRs (cont.)	NLRC4[70,71]	• Inhibits tumour development and proliferation[70] • Protects IECs from injury[70,71] • Protects against *Salmonella* infection[71]
	NLRP12[72–74]	• Limits inflammatory response to injury[72–74] • Limits tissue repair following injury[72] • Inhibits tumour formation and growth[72–74]
	Caspase 1[61,63,72,75]	• Protects IECs from injury[61,63,72] • Inhibits tumour development[61,72]
	Caspase 12[72]	• Inhibits epithelial repair[72] • Inhibits tumour development[72]
PRR signalling pathways in IECs		
NF-KB Signalling	**IKK1**[76]	• Inhibits IEC apoptosis[76,77] • Promotes AMP production[76,77] • Protects IECs from injury[77,78] • Promotes tumour development[79,80]
	IKK2[76,78–82]	
	NEMO[76]	
	RelA[77]	
MAP Kinase Signalling	**p38α**[82,83]	• Promotes goblet cell differentiation[83] • Inhibits proliferation[83]
IEC negative regulators of PRR signalling		
	SIGIRR[84,85]	• Inhibits TLR signalling[84,85] • Limits proliferation[85] • Protects IECs from injury[85] • Inhibits tumour development and progression[85]
	A20[86–92]	• Inhibits apoptosis[89,90,92] • Protects IECs from injury[89,90,92] • Inhibits NF-κB activation by PRRs and cytokine receptor[86,87,89,90]
	MKP1[93]	• ND
	miR-146a[94]	• Inhibits TLR signalling during the neonatal period[95]

Bold font designates evidence for IEC-specific role based on targeted deletion or transgenic expression in murine models

* Indicates expression only in mice, and not in humans

AMP, antimicrobial peptide; APRIL, a proliferation-inducing ligand; BAFF, B cell activating factor; IEC, intestinal epithelial cell; IEL, intraepithelial lymphocyte;
ND, not determine; pIgR, polymeric immunoglobulin receptor; ROS, reactive oxygen species; TFF3, trefoil factor 3; TLR, toll-like receptor;

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