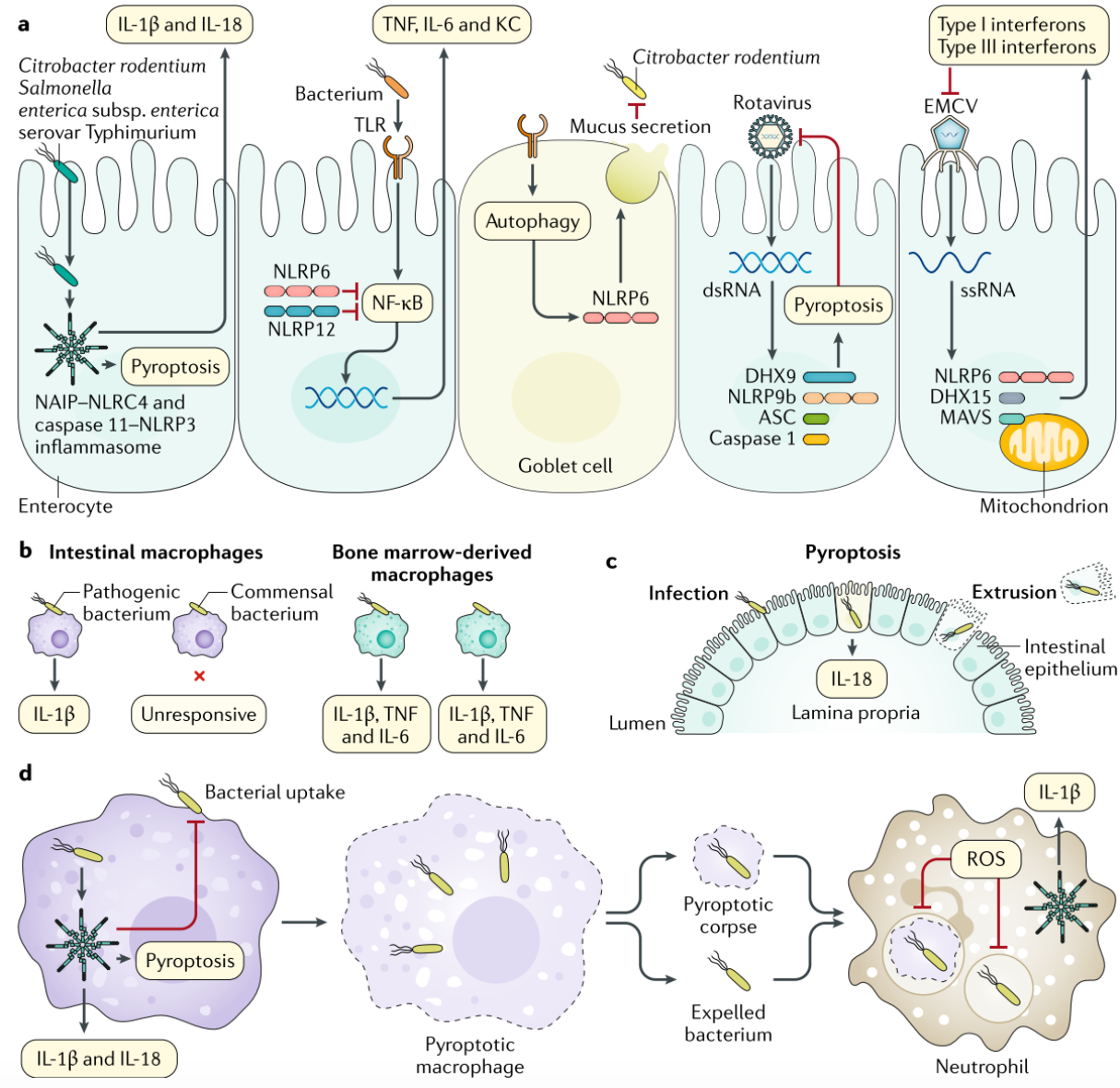
胃肠道中的炎症小体：感染，癌症和肠道微生物群稳态

Inflammasomes in the gastrointestinal tract: infection, cancer and gut microbiota homeostasis【3】



炎症小体和其相关的分子在肠道细胞和免疫细胞中促进杀死和清除胃肠道病原菌

a| 炎症小体通过诱导分泌 IL-1β，IL-18 和细胞焦亡介导保护宿主免受革兰氏阴性菌侵袭[1-8]。NLRP6 和 NLRP12 可以负向调节炎症[9-12]。NLRP6 在杯状细胞中对 Toll 样受体诱导的自噬作出响应从而介导粘液的分泌[13]。RNA 结合的 DEAH 盒蛋白9（DHX9）与 NLRP9b 相互作用，诱导炎症小体复合物的组装[14]。NLRP6–DHX15 复合物结合病毒 RNA 并且诱导 1 型和 3 型干扰素的产生[15]。

b| 肠道巨噬细胞可 以区分病原菌和共生菌[16]。

c | 激活 caspase 1，caspase 8 或者 caspase 11 导致细胞死亡，进而促使从上皮清除和排出被感染的肠上皮细胞[17-19]。

d | 炎症小体可以通过抑制细菌摄入减少细菌负荷，这会限制巨噬细胞的运动和刚度（stiffness）并且促进活性氧的产生(ROS)[20]。焦亡的巨噬细胞释放完整的细菌或者陷入细胞内陷阱的细菌[21]，这些释放的实体会进一步被中性粒细胞吞噬[3,22] 。

TIPs：

pyroptosis: 细胞焦亡,是一种新的程序性细胞死亡方式，其特征为依赖于半胱天冬酶-1(caspase-1)，并伴有大量促炎症因子的释放。细胞焦亡的形态学特征、发生及调控机制等均不同于凋亡、坏死等其他细胞死亡方式[01]；
Caspase：半胱天冬酶；
ASC：apoptosis-associated speck-like protein containing a caspase activation and recruitment domain (CARD)： 含有半胱天冬酶激活和募集结构域的凋亡相关斑点样蛋白
dsRNA , double-stranded RNA：双链 RNA ;
EMCV, encephalomyocarditis virus：脑心肌炎病毒;
KC，keratinocyte chemoattractant：角质形成细胞化学引诱物（也称为CXCL1）；
MAVS, mitochondrial antiviral-signalling protein：线粒体抗病毒信号蛋白;
NAIP, neuronal apoptosis inhibitory protein：神经细胞凋亡抑制蛋白;
NF-κB, nuclear factor-κB：核因子-κB;
NLRC4, nucleotide-binding domain, leucine-rich repeat-containing protein (NLR) family CARD domain-containing protein 4： 核苷酸结合结构域，富含亮氨酸重复序列的蛋白（NLR）家族 CARD 结构域蛋白4（NLRC4）
ssRNA, single-stranded RNA： 单链RNA;

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