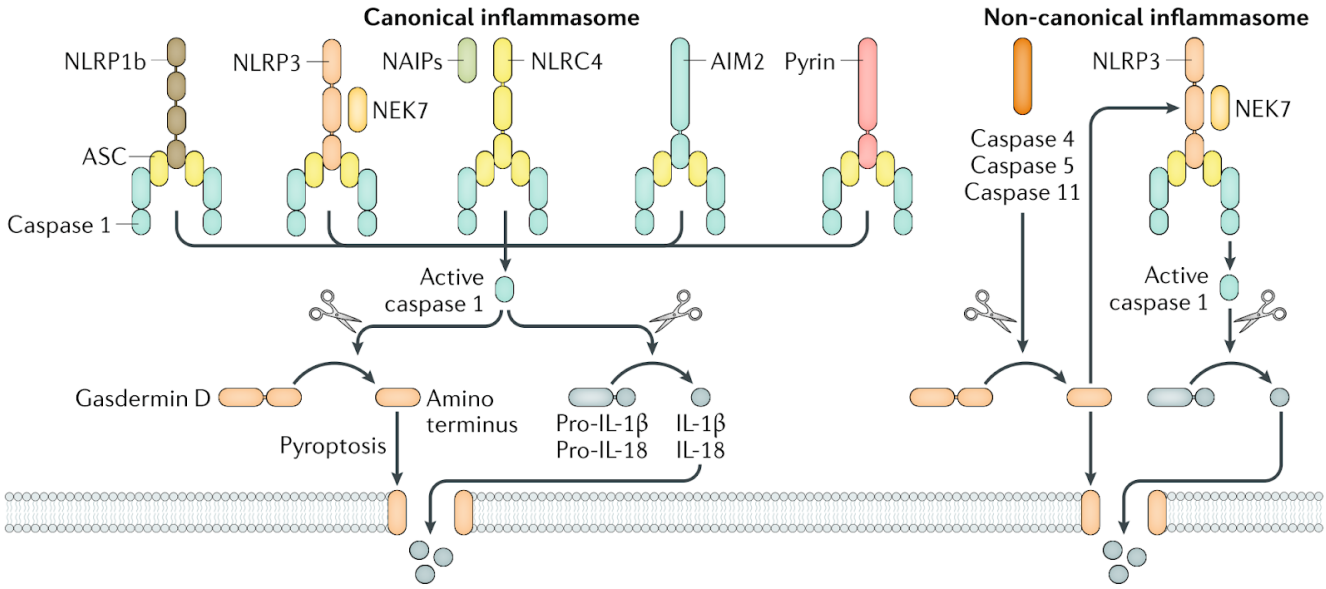
# 胃肠道中的炎症小体：感染，癌症和肠道微生物群稳态

Inflammasomes in the gastrointestinal tract: infection, cancer and gut microbiota homeostasis【1】




## 炎症小体复合物

炎症小体传感受器 NLRP1b，NLRP3，NLRC4，AIM2 和 pyrin 都能够形成含有衔接蛋白ASC和半胱氨酸蛋白酶 caspase 1 的经典炎症小体复合物。NLRP1b 和 NLRC4 也在没有 ASC 的情况下募集 caspase 1，因为它们的结构中存在 CARD结构域[1-3] 。NLRP3 和 NLRC4 的激活分别需要 NEK7 激酶和 NLR 家族成员神经元凋亡抑制蛋白（NAIP）。Caspase 1裂解前体细胞因子 pro-IL-1β 和 pro-IL-18 以及成孔蛋白 gasdermin D。Gasdermin D 的活性片段寡聚化导致细胞膜上形成孔洞，引发细胞焦亡(pyroptosis)[4-11]。这些孔洞允许从细胞中被动释放具有生物活性的 IL-1β 和 IL-18。非经典炎症小体的定义是需要人类 caspase 4 和 caspase 5 或小鼠 caspase 11激活的 NLRP3 炎症小体复合物[12]。这些半胱天冬酶（Caspase）的活化导致 gasdermin D 的裂解和细胞焦亡[4-6]。gasdermin D 的成孔片段（pore-forming fragment）激活 NLRP3 炎症小体以及 IL-1β 与 IL-18 的 caspase 1 依赖性成熟[4,5]。

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[13]；

Caspase：半胱天冬酶；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