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Research Progress of PSP/Reg Protein Family in Chronic Pancreatitis

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Abstract

Pancreatic stone protein (PSP), pancreatitis-associated protein (PAP) and Regenerating protein I (Reg I) have been independently found in pancreatic exocrine and endocrine diseases. Subsequent studies showed that PSP and Reg I are the same protein and belong to the same protein family as PAP. PSP/Reg and PAP have the same selective specific tryptic cleavage site and are digested by trypsin to form insoluble fiber. Since there is a hypothesis that PSP has a function of inhibiting pancreatic formation, PSP is also called lithostathine. However, the function of inhibiting stone formation by PSP has been questioned. At present, most studies have shown that PSP has a promoting effect on the formation of stones, rather than inhibition.

Keywords

Pancreatic Stone Protein, Regenerating Protein, Pancreatitis-Associated Protein, Chronic Pancreatitis

PSP/Reg蛋白家族在慢性胰腺炎中研究进展

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摘　要

胰石蛋白(pancreatic stone protein, PSP)，胰腺炎相关蛋白(pancreatitis-associated protein, PAP)和

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再生蛋白I (Regenerating protein I, Reg I)先后被独立地发现于胰腺的外分泌和内分泌疾病。随后的研究表明，PSP和RegI是同一种蛋白并且其与PAP属于同一种蛋白家族。PSP/Reg和PAP具有相同的选择性特异性的胰酶切割位点，并被胰酶降解形成不溶性纤维。由于有假说提出PSP具有抑制胰石形成的功能，所以PSP又称为胰石蛋白(lithostathine)。但是胰石蛋白的抑制结石形成的功能受到了质疑。目前大部分研究结果表明PSP对于结石形成具有促进作用，而非抑制。

关键词

胰石蛋白，再生蛋白，胰腺炎相关蛋白，慢性胰腺炎

1. 前言

PSP 和 Reg I 分别在胰腺炎和糖尿病领域的研究中被独立发现，但后来的研究发现，两者是同一种蛋白质。1979 年，Sarles H.等在尝试分离胰腺结石内的蛋白成分时发现了一种分子量为 14kDa 的蛋白质，并将其命名为胰石蛋白(pancreatic stone protein, PSP) [1]。

1980 年，法国的 Gross J.团队于中性的牛胰液中发现了一种蛋白质纤维沉淀，并将其命名为胰丝蛋白(pancreatic thread protein, PTP) [2]。在人类中发现的与前者相似特征的蛋白质随后被列入同一蛋白质家族[3]。Terazono K.团队报道称，在老鼠的胰腺中该蛋白的 mRNA 的克隆表达仅出现于胰腺再生小岛，成熟胰岛反而不存在，故将其命名为 Reg [4]。

胰腺炎相关蛋白(PAP)及其亚型也属于前面提及的蛋白质家族。PAP 首次发现于急性胰腺炎老鼠[5]，现今被重新命名为 RegIII。PSP/Reg 和 PAP 具有相同的选择性特异性的胰酶切割位点，并被胰酶降解形成不溶性纤维，这种纤维来自于人类，牛类，鼠类的 PSP/Reg 和 PAP 蛋白[6]。

目前，这种蛋白的功能作用尚未完全阐明。本文我们将对慢性胰腺炎(CP, chronic pancreatitis)中 PSP/Reg 家族的功能和分子结构进行回顾。

2. 结构分析

PSP 被认为是 CP 中抑制结石形成的关键蛋白质。Sarles 团队[1] [7]于胰腺结石中首次发现一种分子量为 14kDa 的糖基化酸性磷蛋白，他们认为这种蛋白具有抑制胰液中碳酸钙结晶的能力，并将其命名为 PSP。

PSP $S_{2\text{-}5}$ 由四个基团组成(分泌形式，14~19kDa)，由胰腺腺泡细胞分泌，进入胰管最终汇入十二指肠。PSP 来源于 PSP $S_{2\text{-}5}$，后者被胰蛋白酶剪切 Arg11-Ile12 肽键，余下的带有 N 端的 11 个氨基酸组成的单链多肽即为 PSP。PSP 和 PSP S1-5 统一来源于 PSP $S_{2\text{-}5}$ 的降解，不同之处在于肽链的长度。通过对 PSP 的 mRNA 进行克隆和序列分析证实，该蛋白仅有一条转录体并且其编码蛋白的氨基端序列与已知的 PSP $S_{2\text{-}5}$ 多肽的相一致[7]-[15]。PSP $S_{2\text{-}5}$ 的多态性可能是由于单一多肽翻译成熟所造成的。在胰腺组织或胰液中发现 PSP S_1 提示胰酶已经被激活[16]。

胰腺炎相关蛋白(PAP)，胰腺腺泡细胞在炎症急性期的分泌产物，与 PSP/Reg 和 PTP 有极强的序列同源性。在寻找 PAP 与其他胰腺蛋白的同源性研究中，仅有 Reg 蛋白被观测到与 PAP 由很高水平的相

似性，两者的氨基酸序列相似性达到了 63%。这一结果提示 PSP/Reg 和 PAP 基因也许起源于同一原始基因[17]。

3. PSP/Reg 家族蛋白功能和病理生理分析

3.1. 胰腺结石的促进剂还是抑制剂？

慢性胰腺炎最早的胰腺损伤被认为是蛋白栓子沉积于胰腺导管内[18]。如果胰液蛋白质浓缩沉积是蛋白栓子形成的基本原因，那么候选蛋白的浓度必定会增加。有文献报道在胰液中形成不溶性的聚集物或纤维的能力似乎比起始蛋白浓度升高更为重要[19]，如果往这方面考虑，PSP 可以作为形成蛋白栓和结石的候选蛋白。

胰液中过度饱和的碳酸钙和 PSP 作为胰腺结石的主要成分，导致科学家假设抑制碳酸钙晶体形成的抑制剂必须存在于胰液内。这种抑制剂必须干扰晶体成核和生长[1] [7]-[14]，这种假说似乎可以解释为什么 CP 患者会有结石形成，而正常人没有。因此，科学家将 PSP 更名为 lithostathine [20]，其意义在于更加侧重其抑制碳酸钙结晶和生长的能力，而非之前提到的抑制不溶性纤维形成和蛋白沉淀的能力。

照此推断，CP 患者胰液中的 PSP 水平应该会降低，但是目前关于 PSP 水平的报道存在很大的争议。利用多种方法测定的钙化性 CP 患者胰液中 PSP $S_{2\text{-}5}$ 含量，最终得出结果是 PSP 含量显著降低[21] [22] [23]。与此相反，多项研究表明[24] [25]，慢性胰腺炎与对照组的 PSP 含量无明显差异。两组报道的结果存在争议，可能的原因是用于 PSP 含量测定的抗 PSP 抗体的特异性或许存在差异。

如果过早的胰蛋白酶激活，将导致胰液中的可溶性的 PSP $S_{2\text{-}5}$ 转变为不溶性的 PSP S_1。对于 CP 患者，采取基于 PSP $S_{2\text{-}5}$ 方法测定的 PSP 含量将会降低。并且有大量文献报道，CP 患者有反复的胰腺炎发作史。这一点支持 CP 患者的胰酶提前激活十分频繁[26] [27]。

Bimmler 等证实，纯化的鼠胰石蛋白在体外实验中可以抑制碳酸钙结晶。但是，牛胰蛋白酶和人血清白蛋白和磷酸根等离子似乎有同样的作用。因此，他们对胰石蛋白抑制胰腺结石形成的能力提出了质疑[28] [29]。同样的，De Reggi 等将胰石蛋白抑制结石形成的能力归因于非特异性反应。多项研究报道，PSP/lithostathine 的 C 端有沉淀的趋势[3] [7] [12] [16] [30] [31]。故前面提到的两位学者认为，在胰石蛋白的功能阐明之前是否应该继续使用 lithostathine 这一名字。

对 CP 患者胰腺导管钙化和沉淀进行蛋白质分析是阐明 PSP 在胰腺结石形成机制的重要手段。PSP 是胰腺结石的重要组成成分。但是胰腺结石还含有多种蛋白成分，提示 PSP 不是唯一的与胰腺结石形成相关的蛋白质。

3.2. 支架栓塞

CP 患者接受内镜治疗对胰管进行引流，特别是胰管支架置入，该治疗方法可以很好地控制疼痛。但是很容易发生胰管支架闭塞，经研究发现支架闭塞的蛋白质成分主要为：胰石蛋白，白蛋白，胰蛋白酶[32] [33] [34]。胰管支架闭塞的详细机制尚未阐明，但是具有较大侧孔直径的支架更少发生闭塞[35]。胰石蛋白似乎促进支架的闭塞。

3.3. 细菌聚集

Iowanna 等研究发现在含有大肠杆菌的培养皿中加入 PAP/RegIII，可观察到细菌聚集现象。同样，从胰液中纯化得到的 PSP/lithostathine/Reg 也能产生相似的细菌聚集现象[36]。蛋白多糖绑定和细菌聚集需要被胰酶裂解蛋白[37]。蛋白栓子的细微结构显示：细菌和酸性糖蛋白包含在细微的网状结构之中。提示 PSP/lithostathine/Reg 和 PAP/RegIII 参与了纤维的形成[38]。Marotta 等报道胰液具有抗菌作用[39]。然而，

胰液的细菌聚集和抗菌作用的机制尚未阐明[40]。

3.4. 抑制胰腺星形细胞(pancreatic stellate cells, PSCs)活性

PSP/Reg 家族蛋白在正常胰腺和胰液中呈低水平，在 CP 时腺泡细胞大量合成分泌 PSP/Reg [41]，因此有学者推测 PSP/Reg 分泌可能是一种机体自我保护机制[42]。CP 的最重要的病理特征之一是纤维化，而 PSCs 在 CP 相关纤维化被公认为胰腺纤维化过程中的关键环节，李玲[43]等报道发现分离纯化 CP 患者的 PSCs 在不同浓度的 PSP/Reg 家族蛋白干预下表现出不同的增殖特性，提示高浓度的 PSP/Reg 家族蛋白能抑制 PSCs 的活性，可能与参与抑制胰腺纤维化的过程。

4. 未来展望

PSP，PAP 和 Reg 是在慢性胰腺炎、急性胰腺炎、糖尿病领域研究中分别被发现的蛋白质。后续的研究发现，三种蛋白质和其亚型属于同一家族蛋白质，尽管其功能尚未完全阐明。更多的研究方向应该指向 Reg，PAP 及其亚型的再生功能，有丝分裂，致癌作用，抗炎和抗凋亡作用。我们期待在不远的将来，有更多的有效的标记物对慢性胰腺炎预后与治疗效果的进行准确的评估与预判。

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附录

缩略词：

胰石蛋白(pancreatic stone protein, PSP)；胰腺炎相关蛋白(pancreatitis-associated protein, PAP)；再生蛋白 I (Regenerating protein I, Reg I)；胰丝蛋白(pancreatic thread protein, PTP)；胰腺星形细胞(pancreatic stellate cells, PSCs)；慢性胰腺炎(chronic pancreatitis, CP).