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大豆胞囊线虫抗性研究现状及其对大豆育种的启示

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摘要

大豆胞囊线虫（SCN，*Heterodera glycines*）是危害全球大豆生产最严重的线虫。作物轮作和培育抗线虫品种是防治线虫的主要方法。随着越来越多能够克服常用抗源的毒性 SCN 群体出现，有必要寻找更多新的抗源或拓宽抗性遗传背景。本文综述了大豆抗 SCN 基因的研究进展，以及这些基因如何相互作用，从而使大豆能够抗 SCN。本文还提供了最新分子作图和分子标记，可用于不同抗性品系和品种的大规模选择和鉴定，以加速常规育种计划。深入了解 SCN 寄生蛋白和大豆对病原体的抗性是通过基因修饰、基因叠加、回交或基因工程引入新的抗源而使抗源多样化的关键。

1. 引言

大豆[*Glycine max*（L.）Merr.]是人类种植的最古老的作物之一[1]。大豆可能起源于中国北部和中部地区[2]。有证据表明，早在公元前3500年大豆就已被驯化[3]，公元前200年前后被引入韩国，公元300年前后传入日本和俄罗斯[1]。大豆的世界年产值约为1045亿美元。

1899年，在中国由大豆胞囊线虫（SCN，*Heterodera glycines*）引起的危害被称为“火烧苗”[3]。由于大豆起源于中国，在大豆大规模传播之前，线虫在中国引起“火烧苗”病，所以中国被认为是线虫的起源地。SCN持续影响中国大豆生产，产量损失估计超过1.2亿美元[4–7]，并且在许多大豆生产国都有报道。SCN最初于1915年在日本被发现[8]，稍后在美国也观察到SCN[9]，年产量损失超过12亿美元[9–11]。目前SCN遍及整个美国南部地区。

在美国中北部地区，用于作物轮作的SCN抗源包括PI 88788、PI 54840（Peking）和PI 437654[12,13]。遗憾的是，随着时间的推移，由于这些抗源的连续应用，已进化出毒性更强的SCN群体。根据4个大豆基因型的反应将SCN群体的毒力型描述为小种[14]。随后，为了确定SCN群体的毒力而发展为HG型系统（HG代表线虫属种学名*Heterodera glaycines*的第一个字母）。由于小种不适合描述多样性、杂合SCN群体，所以该系统采用7个具有不同形式抗性的大豆标志性品系[15]。越来越多的证据表明，SCN群体能够克服抗性[16–18]。因此，随着更多的SCN群体能够克服PI 88788的抗性[19–23]，迫切

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需要在大豆种质资源中扩大抗源，拓宽优良大豆种质资源的抗性遗传背景。

2. 专性寄生的分子适应性

与其他胞囊线虫一样，大豆胞囊线虫是一种专性的根寄生物，与寄主具有复杂而亲密的相互作用。土壤中二龄幼虫（J2）利用其口针侵染大豆植株的根。然后，J2通过根皮层细胞在细胞间移动至根髓部，通过食道腺细胞的分泌物诱导一个初始细胞形成合胞体，开始取食，经过3次连续蜕皮后，J2变成成虫。合胞体是一种作为取食点的代谢库，提供发展到成虫所需的营养物质[24]。线虫完全依赖于合胞体而存活，这意味着合胞体细胞的破坏或死亡将导致线虫死亡。

分子研究发现线虫的食道腺细胞分泌的蛋白质对这种亲密关系至关重要[25,26]。一项传统遗传学研究报道，SCN毒力基因（*ror*）可使其能够在抗虫大豆品种上繁殖[27]。同时，在侵染大豆根之前和之后的SCN中发现了一些纤维素酶和果胶裂解酶[28–32]。这些纤维素酶存在于可侵染根组织的幼虫和需要3次蜕皮并脱离根的雄虫中，可能起到软化根组织的作用[30]；其他SCN基因（如鸟苷酸基因），可能在化学感觉识别中起作用[33]。

SCN的分支酸变位酶（CM）基因具有多态性[34]。这种酶存在于植物的莽草酸途径中，在动物中不存在。它可以改变一个或多个莽草酸途径下游产物，可能在合胞体维护中发挥作用。在这种线虫中存在一种与CLAVATA3配体同源的分泌性CLAVATA3/胚胎周围区域相关（CLE）肽，其信号肽诱导植物（原生质体）的细胞分裂[35]。最近在一项离体研究中发现，SCN的一个CLE肽（HgCLE）与大豆CLE受体相互作用，并且受体的沉默提高了对SCN的抗性[36]。

最近在SCN中发现的其他基因包括生物素合酶和推测的*N*-乙基马来酰亚胺敏感因子激活蛋白受体（SNARE）域基因[37]。SNARE-结合蛋白 HgSLP-1可与大豆*N*-乙基马来酰亚胺敏感因子连接蛋白α（α-SNAP）相互作用，在非亲合互作中触发防御反应。HGSLP-1似乎不存在，表明其作为无毒的SCN蛋白的作用。因此，大豆中无毒的SCN的HgSLP-1与*Rhg1* α-SNAP之间的互作引起抗性反应[37]。虽然生物素参与了植物中的几种细胞过程，但在植物中线虫生物素的作用问题尚待确定。然而，推测无毒和强毒SCN之间生物素的氨基酸差异有助于确定其功能[37]。此外，从SCN克隆并鉴定了3个新的ran结合蛋白基因，其中包含N端和B30.2的信号肽、C端的spla激酶以及利阿诺定受体（SPRY）结构域。RNAi介导的*Hg-rbp-2*沉默导致线虫的抑制和寄生能力[38]。

虽然SCN在大豆上寄生的分子细节和大豆抗性机制尚不清楚，但是这种知识对于寻找新的抗药性策略至关重要。

3. 亲合互作

当线虫能够成功地侵染寄主并在其中繁殖时，这种相互作用被称为亲合互作。在这种情况下，侵染性J2能够侵入大豆植株根，穿透根表皮和皮层细胞，并继续向髓部移动，在其中诱导合胞体细胞、发育和维持生长。穿透和迁移通过表皮和皮层细胞都是通过机械性[39]和酶反应[29]。来自两性体、腺细胞和内唇感觉器的分泌物在口针与合胞体之间形成一个短管[40–42]。在取食时，口针在完整的取食塞中进出[26]。因此，大豆通过基因表达修饰和细胞变化作出反应，特别是在受影响的细胞内[43,44]。

首先，合胞体细胞的细胞质变得致密，核糖体和粗面内质网（RER）数目增加[42]。细胞壁逐渐通过受影响的细胞壁的开口溶解。初始细胞的胞间连丝扩张，导致相邻细胞的原生质体融合，最终细胞壁溶解，产生代谢活性增强的多核取食细胞（合胞体）。细胞质变得致密，细胞器和细胞壁向内生长[45,46]。细胞壁增厚，主要细胞液泡被许多次生液泡所取代[42]。距离初始合胞体细胞较远的细胞壁溶解更大，而距离初始合胞体细胞较近的细胞壁加厚更广泛[47]。薄壁细胞可能发生增生，而距合胞体较远的细胞发生肥大。合胞体内的细胞核是否复制尚不清楚。线虫取食，在变为成虫之前经历4次蜕皮[图1（a）和（b）]。雌虫产卵，由雄虫受精，从而开始新一代的亲合关系。

4. 非亲合互作

在非亲合互作中，J2侵入根部并诱导合胞体的形成。然而，互作关系建立后不久，合胞体坏死并退化，导致线虫死亡[图1（c）和（d）]。坏死形成时间和最终合胞体死亡的时间取决于宿主植物[48]。细胞核退化和坏死是过敏反应的结果[图1（e）][45,48,49]。不

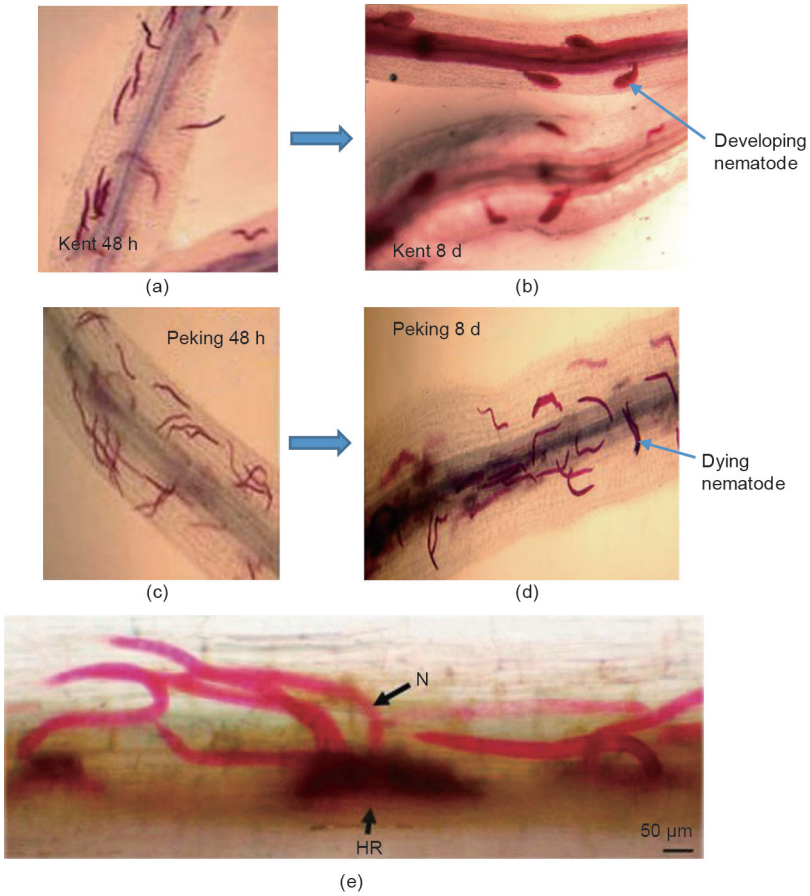

图1. 大豆与SCN（*Heterodera glycines*）的亲合与非亲合互作。（a）接种48 h后（DAI）感染宿主（Kent）根中的J2;（b）接种8 d后感病宿主（Kent）根中的J2;（c）接种48 h后抗病宿主（Peking）根中J2;（d）8 DAI;（e）抗病大豆品种Forrest的根在侵染SCN J2的部位上表现出过敏反应（HR）样细胞死亡，被酸性品红（N）染成粉红色[图（a）~（d）由美国农业部B. Mathews提供；图（e）由密苏里大学Xiaohong Liu提供]。

同抗源的抗性反应速度不同：在Peking中合胞体退化很快，而在PI 88788和PI 209332中，合体细胞死亡缓慢[48,50–52]。大豆品种Peking中的大多数合胞体在接种后5 d（DAI）内停止发育并坏死[53]，可能于2 DAI从RER的扩张开始[52]。到5 DAI，伴随着发育中的合胞体坏死而形成细胞壁沉积[50,54]。另外，到开始后4~5 d，细胞发生了以下变化：合胞体细胞壁不规则增厚，细胞壁向内生长，细胞质内含有大量微管的质膜内陷[41,42]。在PI 437654中观察到不规则的细胞壁增厚、细胞壁附着、细胞坏死和细胞核退变现象，其抗性反应类似于Peking [55]。

P1 88788或这类抗源培育的大豆品种产生的初始合胞体细胞具有广泛的内胞浆网槽和RER [42]，而没有像Peking中的抗性反应如细胞壁增厚、沉积或坏死层。有趣的是，发育的中合胞体周围的非合胞体细胞的细胞壁发生坏死，8~10 DAI整个合胞体坏死[50,54]。随后细胞核退化，胞质内的染色质样物质形成。

一般而言，合胞体在非亲合互作中的消亡可能归因于细胞核退化、细胞壁沉积物形成、非功能内质网和程序性细胞死亡[53]。尽管具有保守的转录背景，但在PI 88788和Peking的退化合胞体中的基因表达使基因型特异性的基因表达[56]，这说明大豆品种的抗SCN机制可能与不同品种有关。

5. 大豆中发现的抗 SCN 基因

经典的遗传学研究表明，大豆对SCN的抗性是由隐性基因*rhg1*、*rhg2*和*rhg3* [57]与显性基因*Rhg4*和*Rhg5* [58,59]控制的。随后的研究表明，这种相互作用更复杂，既涉及主效基因又包括微效基因[60]。近20年来，遗传标记技术的发展极大地促进了控制大豆抗SCN的主效数量性状基因座（QTL）的发现、定位和分析[16,61–64]。许多QTL实验的结果表明，*Rhg1*和*Rhg4*的遗传区域贡献了大部分SCN抗性（图2）[65]，因此为了选择大豆品系中的这些基因座而开发了这些区域的分子标记[63,66,67]。

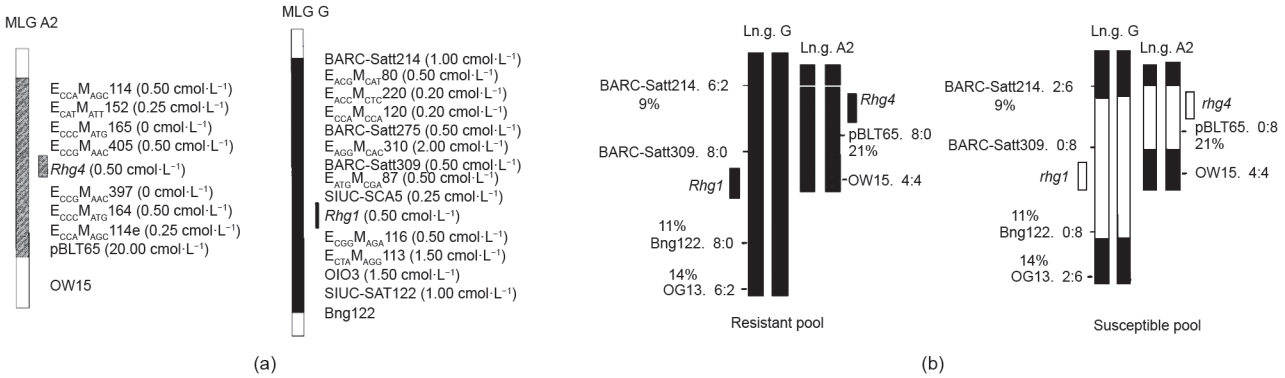

图2.（a）携带*Rhg1*和*Rhg4*基因座染色体片段的高密度遗传图谱；（b）它们在大豆抗病和感病池中的存在或缺失。MLG：分子标记连锁群（引自参考文献[65]）。

大豆18号染色体上的*Rhg1*基因表现为不完全显性。该基因在SCN抗性中起重要作用[16]。不同抗源之间存在不同的*Rhg1*等位基因[68]。美国约90%的SCN抗源使用了*rhg1-b*等位基因。*Rhg4*被定位到8号染色体上，呈显性遗传，并且在某些抗源中可能需要完全抗性[68]。

为了进一步精细定位*Rhg1*和*Rhg4*基因座，并分析SCN大豆互作中涉及的候选基因特征，综合采用了过去10年的很多技术发展的功能基因组学工具、大豆基因组测序、图位克隆、诱变、定向诱导基因组局部突变技术（TILLING）和基因沉默等方法。特别是发现*rhg1-b*基因的一部分编码3种蛋白质，即氨基酸转运蛋白（AAT）、α-SNAP和创伤诱导域蛋白（WI12），这有助于大豆抗SCN的突破（图3）[75]。在感病品种中发现*rhg1-b*中长度为31 kb的单拷贝，但在抗性品种中发现了多个拷贝（图3）。该片段的拷贝数增加导致抗性品种中基因的表达增加。此外，这些基因在感病品种中的过度表达对SCN产生某种程度的抑制[75]。值得注意的是，这一结果表明，大豆的SCN抗性不仅受抗性基因座*Rhg1*存在与否的影响，而且还受*rhg1-b*基因座重复的长度为31 kb多基因片段拷贝数变异的影响。其他研究还表明，*Rhg1*和*Rhg4*在抗SCN中的作用与含QTL基因座的富亮氨酸重复受体样激酶（LRR-RLK）无关[74,76–78]。有趣的是，在长度为31 kb的多基因片段重复的基因中没有一个与具有核苷酸结合位点的经典植物抗性基因[富亮氨酸重复序列（NBS-LRR）域]相似，表明这是植物的一种新

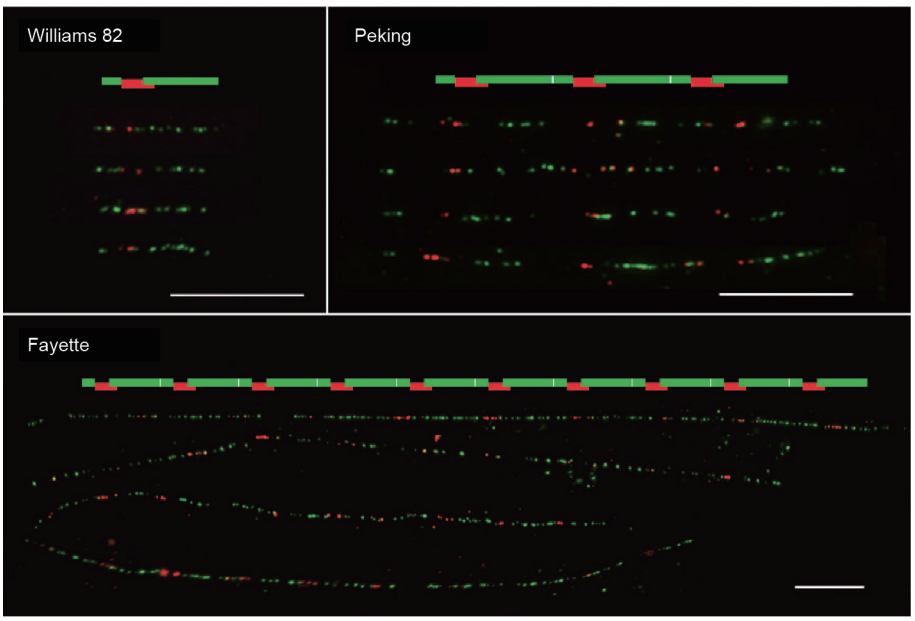

图3. 纤丝荧光原位杂交（FISH）检测被广泛利用的大豆品系中*Rhg1*拷贝数的变异：探针示意图和每个基因型4个纤丝-FISH（4个DNA纤丝）整合图，显示抗SCN材料Fayette和Peking中的长度为31 kb的*Rhg1*片段的10个或3个直接重复拷贝和感病材料Williams 82中每个*Rhg1*单倍型1个拷贝。白色标尺代表10 mm，对应于3.21 kb·mm^{-1}的转化率大约32 kb（引自参考文献[75]）。

的抗性形式[76,78]。

目前的研究已经证实，拷贝数变异、基因序列差异和重复片段甲基化介导大豆对SCN的抗性[79–81]。根据这些发现，大豆抗性被分为*Rhg1*的31 kb部分的高拷贝数和低拷贝数材料[23,79]。感病品种α-SNAP等位基因与抗性品种α-SNAP等位基因的C端结构域不同[79,80]。此外，在具有不同重复数的抗病品系中观察到3种不同形式的α-SNAP蛋白，表明*Rhg1*的α-SNAP蛋白拷贝数和序列在大豆抗SCN中起重要作用[80]。同时从PI 88788类型抗性（*GmSNAP18*）的α-SNAP多个拷贝同时对*rhg1-b*抗性起作用[75]，Peking类型*rhg1-a*的*GmSNAP18*与*Rhg4*一起使该基因座具有SCN抗性[23]。最近的遗传分析表明，*GmSNAP11*作为一种新的微效抗性基因，有助于SCN的加性抗性[82]。

此外，图位克隆实验表明，Peking的*Rhg4*抗性基因座编码预测的胞浆丝氨酸羟甲基转移酶（SHMT）在序列上与感病形式不同[74]。在SHMT的维生素B_6结合位点将精氨酸（R）转化为脯氨酸（P）以及将酪氨酸（Y）转化为天冬酰胺（N）的错义突变可能是其抗SCN功能的关键。这些氨基酸多态性可能是不同形态的SHMT之间酶活性差异的原因[74,83,84]。最近的研究结果表明，含有低拷贝*Rhg1*的SCN抗性种质需要*Rhg4*[80]，然而，目前尚不清楚这两个基因座抗性的分子机制[23]。Peking的*Rhg1*需要*Rhg4*同时表达才能发挥功能，证明Peking的SCN抗性可能有上位性作用参与[68,74]。*rhg1-a* Peking型*GmSNAP18*与*Rhg4*结合足以抗SCN，这表明来源于Peking的*GmSNAP18*与来源于PI 88788中*GmSNAP18*的SCN抗性在功能上是不同的[85]。SHMT的突变揭示了结构稳定性、配体结合、酶活性和蛋白质相互作用的关键残基，因此，这些令人信服的遗传证据表明，不论是否携带抗性的*Rhg1*等位基因，SHMT对于Peking型抗病品种中有效SCN抗性是必需的。

6. 大豆抗 SCN 机制的认识

尽管几项遗传、细胞学和分子作图研究有助于鉴定和分析大豆中SCN抗性基因，关于SCN抗性的分子机制还有很多需要研究的地方。目前，大豆中已发现了两种类型的抗SCN抗性，即PI 88788型抗性和Perking型抗性。这两种类型的抗性是由携带*Rhg1*和*Rhg4*基因的两个主要QTL基因座控制的。PI 88788的SCN抗性仅需要*rhg1-b*等位基因发挥功能；然而，Peking的抗性需要*rhg1-a*和*Rhg4*等位基因同时发挥功能[16,23,85,65]。另外，在Peking型抗性中，还有一种快速而有效的局部超敏反应影响SCN J2；而在PI 88788型抗性中，抗性响应更长，并影响SCN三龄期和四龄期幼虫。大豆的两种SCN抗性比较列于表1 [1,8,14–17,30,49,51,53,55,56,68,69,72,76,80,81,83,86]。

仍有待解决的难题是，*rhg1*和*rhg4*等位基因在SCN-大豆非亲合互作中上调或下调几个基因的作用[56,87–93]。茉莉酸和苯丙烷、腺苷甲硫氨酸、乙烯等几种化学产物的生物合成途径参与了抗性反应[94]。尽管在SCN-大豆非亲合互作中存在分子事件网络，但在Peking和PI 88788中存在保守的差异基因表达，这表明在大豆中存在与SCN抗性相关的特定基因[56]。水杨酸（SA）途径相关基因在抗性反应过程中具有上调作用[56,89,95,96]。在非亲合互作中这些分子事件最终导致合胞体退化。这些防御相关基因在合胞体中被上调作为直接抗性反应[23,89]。

已经报道了大豆抗SCN反应中*S*-腺苷-*L*-蛋氨酸

表1 大豆两种类型SCN抗性的比较

Resistance type	Perking-type	PI 88788-type	References
Resistance allele(s)	*rhg1-a*, *Rhg4*	*rhg1-b*	[8,14,51,76,83]
Resistance requirement	*rhg1-a*, *Rhg4*	*rhg1-b*	[14,68,81]
rhg1 copies	Low (1–3)	High (7–10)	[16,17]
rhg1 genes	AAT, α-SNAP, WI12	AAT, α-SNAP, WI12	[15–17,83]
α-SNAP + *Rhg 4*	Resistance	Susceptibility	[68,80]
α-SNAP polymorphism	High	Low	[69,80,86]
Nuclear degeneration	Rapid	Slow	[1,53,55,56,72]
Cytoplasmic degeneration	4–5 DAI	8–10 DAI	[49,53,55,56]
Site of necrosis initiation	Inside syncytium	Outside syncytium	[53,56,83]
Cell wall appositions	Observed	Not observed	[30,53,72,83]
Dilation of RER	Observed	Not observed	[53,55,83]

（SAM）依赖型水杨酸羧甲基转移酶1（GmSAMT1）的活性[96]。*GmSAMT1*在不同感病大豆品系中表达量过大时，对SCN的抗性有增强作用[97,98]。然而，SA和*Rhg1*或*Rhg4*之间的相互作用在抗性响应中尚未可知。在另一种非亲合互作中，当Peking和PI 88988被SCN侵害时，聚半乳糖醛酸酶的水平降低[99]；而在抗病材料PI 437654的第3天和第6天，受侵害的大豆根中乙烯相关蛋白（GmEREBP1）的水平升高，但在6 d后降低[100]。同样，在Centennial品种（Peking抗源）的非亲合反应中，植保素谷胱甘肽I在SCN J2侵入8 h后提高，侵入6 d 后提高到最高浓度23 μg·g^{-1}[101]；相反，侵害后6 d，在亲合互作中，谷胱甘肽I浓度为7 μg·g^{-1}。此外，其他蛋白质如4-香豆酰Co A连接酶和苯丙氨酸解氨酶在非亲合宿主中比在亲合宿主中增加了，并且在Hartwig品种中表现出比在具有Peking抗源的品种Forest中更高的活性[102]。

另一个编码受体样膜蛋白（7.9 kDa）的大豆基因*GmDS1*，诱导病原与有害生物相关分子模式（PAMP）触发的对多种害虫（如SCN）和真菌病原菌的免疫反应[103]。显然，SCN-大豆互作是复杂的，并且在*Rhg1*和*Rhg4*基因座上由多个基因控制，其表达依赖于拷贝数和核苷酸变异、甲基化，以及这3个因素之间的上位关系，同时有其他微效基因的参与。了解这些基因的功能及其相互作用或甲基化模式如何影响SCN-大豆互作，对于培育抗SCN品种是至关重要的。

7. SCN 抗性的分子标记辅助选择

传统上，植物育种工作者根据温室和田间条件对SCN感染的响应选择抗SCN品系[16]。这个过程不仅是困难的，而且是费力、费时和耗资巨大的，并且因SCN群体的遗传变异而变得更加复杂[16]。然而，标记辅助选择（MAS）是根据与SCN抗性基因的等位基因连锁的遗传标记而实现的[104]。利用简单重复序列（SSR）标记的每个数据点基因分型的成本估计为0.25~1美元，需要1~2 d；相比之下，利用温室生物测定每个数据点的成本为1.50~5美元，需要30 d时间[16]。

为了促进大豆育种，已经确定的分子标记包括限制性片段长度多态性（RFLP）、扩增片段长度多态性（AFLP）、随机扩增多态性DNA（RAPD）、序列特征扩增区域（SCAR）和SSR标记以及单核苷酸多态性（SNP）[16,66,67,77,86,105–109]。除了利用来自双亲杂交的群体进行传统QTL定位外，多样性和自然发生群体的全基因组关联分析（GWAS）也是用于定位QTL和阐明胞囊线虫抗性遗传基础的有效策略[107–111]。植物GWAS越来越受欢迎，主要是由于基因组测序技术的进步，产生了足够数量的可识别的遗传标记，提高了QTL定位的准确性[109]。已开发了用于选择*rhg1*抗性等位基因或*Rhg4*抗性等位基因并区分PI 88788和Peking型抗性的功能性SNP标记，用于高通量MAS[111,112]。目前，分子检测可用于预测大豆抗性品系中的*Rhg1*拷贝数[80,113,114]，有助于提高抗性选择和育种准确率。

8. 抗 SCN 育种的启示

大豆中控制SCN抗性的主效基因座*Rhg1*和*Rhg4*似乎具有不同的进化起源。野生大豆品种中不存在编码SHMT酶的*Rhg4*基因座，而感病等位基因*rhg4*存在于野生大豆品种中[83]。*Rhg4*基因座含有两个关键的错义突变，而在感病等位基因*rhg4*中不存在，表明在驯化过程中可能通过选择产生的*Rhg4*等位基因参与SCN抗性[74,83,84]。这表明可以在*Rhg4*基因座内产生人工、定向的错义突变，从而在新的育种材料中产生具有增加酶活性的SHMT异构体。

*Rhg1*基因的进化似乎与*Rhg4*有很大不同。*Rhg1*基因座在野生大豆（*Glycine soja*）和栽培性大豆（*Glycine max*）中都存在，说明*Rhg1*的起源可能早于驯化和分化[80]。来自野生性大豆的品系PI 468916和来自栽培大豆的品系PI 438489B或PI 89772对可以抗几个SCN群体，携带新的抗性基因[115,116]。同样，其他抗SCN品系不携带*Rhg1*和*Rhg4*抗性基因座[111]。这些结果表明，新的抗性基因一旦被成功发现、分析和导入到大豆中，将为抗SCN提供替代基因。

野生大豆在*Rhg1*中含有3个拷贝的长度为31.2 kb的串联重复单元，而一些栽培大豆可有多达10个串联重复单元[75]。一般认为重复最初来自复制和重组[80]。这种基因重复事件可能说明在抗SCN栽培大豆种质中存在更多重复。

抗性的进一步提高可以通过聚合各种抗源的基因来实现，这可能导致抗性等位基因拷贝数的增加。*Rhg4*和*Rhg1*等位基因的叠加与*Rhg1*等位基因拷贝数的增加拓宽了对SCN的抗性。通过回交将不同来源的SCN抗性等位基因组合，表明不同抗源的基因叠加可能拓宽SCN抗性背景[117]。此外，植物防御相关基因的过表达可

以增强抗性。有趣的是，在野生大豆中发现了与*Rhg1*和*Rhg4*不同的其他SCN抗性QTL[118]。野生大豆的抗性基因可以与*Rhg1*和*Rhg4*等位基因叠加，可以提高对SCN的抗性[119]。最近的遗传操作研究表明，大豆植株中某些基因的沉默或过表达可能导致SCN抗性的增强[36,97]。在另一项研究中，从SCN中敲除ran-结合蛋白基因（*Hg-rbp-2*）可减少SCN根的侵害和发育[38]。许多实验表明，利用非*Rhg1*基因和*Rhg4*基因的遗传操作来实现大豆抗SCN是可行的，在提高育种效率和扩大转基因抗SCN大豆品系的创制方面还有许多工作要做。

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Compliance with ethics guidelines

Guiping Yan and Richard Baidoo declare that they have no conflict of interest or financial conflicts to disclose.

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