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植物毒素的分类及毒理学效应

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摘要：植物在人类生活中具有多重价值，包括营养、观赏及药用用途，但部分植物含有毒性成分，对人类和动物健康构成威胁。植物毒素主要源于其次生代谢产物，包括生物碱、糖苷类化合物、蛋白质类物质、氨基酸及其衍生物、多肽类毒素、脂类毒素、结合金属毒素、聚炔醇类毒素、醌类毒素、酚类毒素、倍半萜内酯类毒素等。这些化合物通过抑制酶活性、干扰代谢途径或破坏细胞结构引发中毒，临床表现为神经损伤、肝毒性、消化障碍、溶血性贫血等。本文系统综述了植物毒素的分类及对人和动物的影响，为植物毒素中毒的诊断和治疗提供理论依据。

关键词：植物；　中毒；　次生代谢产物；　植物毒素分类；　毒理机制

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Classification and Toxicological Effect of Plant Toxins

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Abstract Plants possess multifaceted values in human life, serving nutritional, ornamental, and medicinal purposes. However, some species contain toxic components that pose threats to humans and animals. Plant toxins primarily originate from secondary metabolites, including alkaloids, glycosides, proteinaceous compounds, amino acids and their derivatives, polypeptide toxins, lipidic toxins, metal-binding toxins, polyacetylenic alcohols, quinones, phenolic compounds, sesquiterpene lactones, etc. These compounds induce toxicity by inhibiting enzymatic activity, disrupting metabolic pathways, or damaging cellular structures, leading to clinical manifestations such as neurological damage, hepatotoxicity, gastrointestinal disturbances, hemolytic anemia, and others. This review systematically summarizes the classification of plant toxins and their effects on humans and animals, providing a theoretical basis for the diagnosis and treatment of plant toxin poisoning.

Keywords plants; poisoning; secondary metabolites; classification of plant toxins; toxicological mechanisms

多样性的植物世界对地球上的生命至关重要。自早期文明诞生以来，人类便利用植物服务于多种用途，尤其是其药用价值。尽管部分植物以毒性著称，但它们同时也含有对植物自身和人类均有益的化学成分，这体现了其双重特性[1]。“药用植物”含有的活性化学物质，可以起到协同治疗或减轻潜在不良反应的作用。它们常被用作提取活性化学物质的原料，广泛应用于药物生产领域[2-3]。然而，除了有益的植物外，还有有毒植物，即使摄入这些植物的茎、叶、种子、果实或根部的一小部分，也可能致命[4-5]。

1 有毒植物及毒素介绍

有毒植物通过其次生代谢产物——包括生物碱（菊科、旋花科、唇形科等）、氰苷（高粱属及禾本科植物）、硝酸盐（象草根、苋属等）、草酸盐（禾本科、苋科等）及苷类（蕨类）等化合物，对人和动物产生多系统毒性效应，如致畸与流产（疯草、羽扇豆等）、肝毒性（猪屎豆、马缨丹等）、光敏反应（马缨丹、空心莲子草等）以及呼吸循环系统损伤（亚硝酸盐及氰化物中毒）[6]。目前仍有若干重要有毒植物，其具体毒性成分尚未明确。

由于共同的生活环境，宠物（如犬、猫）与人类（尤其是儿童）常接触相同的有毒植物，中毒具有可比性，但也存在一些显著差异；而牲畜则因饲养环境的特殊性，易接触那些罕见于人类中毒案例的有毒植物[7-8]。不了解植物毒性的人在不知情的情况下，向牲畜提供有毒植物的碎屑，从而使牲畜中毒[9-10]。多数情况下摄入有毒植物后不会产生不良反应或症状较轻，通常无需治疗干预。然而某些植物具有剧毒，微量摄入即可迅速导致死亡[8]。

多种有毒植物对人类和动物具有相同影响，然而不同物种对植物中毒的敏感性存在显著差异[8]。例如，与牛或马相比，绵羊可以耐受较大的吡咯里西啶生物碱摄入量[11]。小动物在中毒敏感性方面也存在一些显著差异。例如，猫在食用百合后会出现急性肾衰竭，而狗则不受影响[12]。危险植物的分布存在地区差异，例如在某地区存在有毒植物，而在其他地区并不存在。另外，若一些有毒植物被引入，可能给当地人类和动物带来严重的中毒风险[13]。由于草药的广泛使用，草药植物中毒变得越来越普遍，部分草药中毒通常是对某种草药的错误识别或过度使用造成的[14-16]。

单一植物体内含有数千种化合物，其中大多数并不具有毒性。事实上，许多化合物具有极其重要的生物学意义，部分化合物更是地球上所有生命形式（包括动植物）生存所必需的[17-19]。初生代谢是指维持生命所必需的代谢途径，而初生代谢产物则是直接参与这些途径的代谢物。植物还通过次生途径合成一系列次生代谢产物，其中甚至包括葡萄糖等初生代谢产物（例如作为某些苷类化合物的组分）。植物次生代谢产物作为关键的防御物质，既能直接发挥抗虫功能，又能调控防御反应以保护植物免受侵害。并非所有次生代谢产物都是生命存续所必需，目前对次生代谢产物的认知仍不充分，毒性化合物正属于这类认知不足的次生代谢产物大类[20-23]。

2　植物毒素分类

目前尚缺乏对植物毒素进行全面而精确分类的体系。Cheeke 等[24]提出的基于毒素化学结构分类方法将植物毒素初步分为生物碱、糖苷类化合物、蛋白质类物质、氨基酸及其衍生物、多肽类毒素、脂类毒素、结合金属毒素、聚炔醇类毒素、醌类毒素、酚类毒素及倍半萜内酯类毒素等。

2.1　生物碱

生物碱是一类含氮的碱性化合物，其氮原子通常以杂环结构形式存在。这类物质多具苦味，且大多数具有毒性[25-26]。根据含氮杂环的化学特性，可将其进一步划分为以下亚类，见表 1。

表 1　植物常见有毒生物碱

分类	亚组	毒性作用	植物来源
吲哚生物碱（色氨酸的衍生物，具有苯环与五元吡咯环稠合的双环结构）[27]	阿马碱（ajmalicine）	致循环系统紊乱[28]	萝芙木属、长春花、帽柱木[28]
	士的宁、马钱子碱（strychnine）	对抗神经递质甘氨酸，阻断脊髓中的突触后抑制作用[29]	马钱子[29]
	3-甲基吲哚（3-methylindole）	引起动物急性肺水肿和肺气肿[30-31]	茉莉、黄钟花、橙花、甜菜根[32-33]
	相思子碱（abrine）	引起呕吐、胃肠出血、脱水、幻觉、肢体震颤、舞蹈手足徐动症、惊厥、强直-阵挛发作、器官衰竭[34-35]	相思子的种子、鸡骨草[36-37]
	N，N-二甲基色胺（N，N-Dimethyltryptamine）	致幻觉[38]	藨草属、龙骨葵属、金合欢属、山蚂蝗属、含羞草属、肉豆蔻属、九节属[39]
吡咯里西啶类生物碱（核心结构由两个五元环构成）[40]	千里光宁碱（senecionine）	肝毒性、遗传毒性和细胞毒性[41-42]	千里光属、蜂斗菜属、波叶短喉菊、梁子菜、兔儿伞属、猪屎豆属、狭萼驴蹄草、菊三七[43-48]
	野百合碱（monocrotaline）	肝、肺毒性及肺动脉高压、神经毒性[49-51]	猪屎豆属的种子、琉璃草属[52-54]
	天芥菜碱（heliotrine）	抑制所有细胞类型的核酸与蛋白质生物合成；肺淤血、胸腔积液、肝脏损伤、心脏损伤、肠道出血[55]	天芥菜属（大尾摇）[56-57]
	蓝蓟定（echimidine）	心脏发育抑制和功能损害、肝细胞损伤[58-59]	蓝蓟、聚合草属[54]
	暗黄猪屎豆碱（fulvine）	肺血管炎、肝肾毒性[60-63]	猪屎豆属[54]
哌啶类生物碱（以六元饱和杂环为特征）[64]	毒芹碱（coniine）	含有烟碱型乙酰胆碱受体拮抗剂，导致运动失调、惊厥及角弓反张、肌肉麻痹、呼吸抑制、致畸[65-66]	毒参属、瓶子草属、尖叶茴芹[66]
吡啶类生物碱（结构特征为含有一个吡啶环）[67]	尼古丁（nicotine）	成瘾性；导致衰老与动脉粥样硬化、血管功能障碍、心血管疾病、糖尿病；降低生育能力，增加母体、胎儿和婴儿的发病率和死亡率；抑制免疫力，诱发呼吸系统疾病，促进肿瘤的生长和转移；牙周炎、与年龄相关性黄斑变性[68-69]	烟草属、石松属、叙利亚马利筋[54,70]
	消旋毒藜碱（anabasine）	激动神经肌肉接头烟碱型受体，导致肌松、呼吸抑制及心动过缓，具有致畸作用[71-73]	无叶假木贼、光烟草、八角枫[70,73-74]
喹诺里西啶类生物碱（核心结构由两个六元环组成）[75]	白羽扇豆碱（lupanine）	阻断神经节传导、降低心肌收缩力以及引起子宫肌肉收缩[76]	金雀儿属、羽扇豆属、牡丹草属、风铃豆属、染料木属、彗豆属、沙冬青、霍州油菜[54]
	臭豆碱（anagyrine）	致畸、血管异常及红细胞再生障碍，可诱发心动过速[54,77-79]	彗豆、臭味崖金豆、霍州油菜、金雀儿属、染料木属、羽扇豆属、苦参属、银砂槐属、荆豆属[54]
	鹰爪豆碱（sparteine）	阻断神经节传导、产生抗胆碱能效应，抑制中枢神经系统及引发神经元坏死[80-82]	金雀儿属、羽扇豆属、染料木属、赝靛属、天竺葵属、厚果槐、银砂槐属[54]

续表 1 植物常见有毒生物碱

分类	亚组	毒性作用	植物来源
吲哚里西啶类生物碱(由吡咯环和吲哚环稠合而成的双环结构)[83]	苦马豆素(swainsonine)	α-甘露糖苷酶和甘露糖苷酶Ⅱ抑制剂,导致溶酶体贮积症;神经元凋亡,肝、脾、肠及肾小管毒性,出血及含铁血黄素沉积,动物流涎、体重下降、步态失调、站立困难、姿势异常、精神沉郁、行为异常、繁殖障碍及死亡[84-88]	黄芪属、棘豆属、黄花棘、沙耀花豆属、树牵牛[84,86,89]
甾体类生物碱(具有环戊烷多氢菲骨架)[90]	茄啶(solanidine)	食欲减退、皮疹、四肢疼痛或全身不适;呕吐与严重腹泻,并伴有弥漫性腹痛(轻重不等),偶含粘液但无血便,次日发热,严重者虚脱、休克或昏迷,致畸[91-93]	马铃薯、瓶子花、黑贝母[54]
	藜芦碱(cevadine/veratrine)	心血管毒性、骨骼肌毒性;强烈的局部刺激反应,惊厥、流涎[94-97]	药用羽柄花、绿花铁筷子、藜芦属[54]
	棋盘花辛碱(zygacine)	较弱的抑制呼吸功能和降低心率的作用,迅速代谢成钠通道毒剂(棋盘花碱),引起心血管功能异常[98-101]	沙盘花属[99]
二萜类生物碱(具有四环或五环二萜的杂环化合物)[102]	甲基牛扁碱(methyllycaconitine)	竞争性拮抗烟碱型乙酰胆碱受体,导致心动过速及心律不齐、肌无力、震颤、呼吸困难、虚脱、肌肉抽搐及强直性惊厥[103-106]	翠雀属[103]
	乌头碱(aconitine)	高剂量则通过激活 Na^+ 通道、抑制 L 型钙通道或调控基因表达等途径诱发心脏毒性、阻断神经干复合动作电位;呼吸衰竭、肝肾毒性、胚胎毒性[107-108]	乌头属[107-108]
托品烷生物碱(具有托品烷环系结构)[109]	阿托品(atropine)	瞳孔散大固定、皮肤干燥发红、心动过速、呼吸急促、体温升高,以及以躁动、意识混乱、精神症状、谵妄为特征的中枢神经系统兴奋症状,偶见癫痫发作;面部及躯干上部可能出现皮疹[110-111]	颠茄、曼陀罗、其他茄科植物[54]
	可卡因(cocaine)	急性中毒表现为心动过速、高血压、高热、多汗、震颤、癫痫发作、瞳孔散大、头痛、腹痛、出血性脑卒中、肝毒性、心脏毒性及多器官功能衰竭;慢性中毒表现为神经退行性变、脑组织早衰、抑郁症状及血管损伤[112-113]	古柯属[112]

2.2 糖苷类化合物

作为一类植物重要的次生代谢产物,糖苷类化合物是由苷元与糖基通过共价键连接而成,不同类型的糖苷具有不同生物活性。绝大多数植物糖苷以非活性形式储存,当植物组织因萎蔫、冻伤、咀嚼或践踏等因素受损时,经酶促水解作用后可释放活性苷元[114]。根据其化学结构不同可分为氰苷、致甲状腺肿糖苷、香豆素苷、甾体苷、硝基糖苷、吡啶衍生物糖苷、致钙化苷类化合物、二萜类糖苷、植物雌激素等,具体分类见表 2。

2.3 蛋白质类物质

植物体内存在的多种重要生物抑制剂都属于蛋白质类物质,其作用机制往往表现为抑制动物对植物蛋白质的利用[140-141]。Laskowski 与 Kato[142] 提出应依据序列中所含的特异性反应位点对蛋白酶抑制剂进行家族分类,该命名体系将蛋白酶抑制剂划分为四个主要家族:①半胱氨酸蛋白酶抑制剂;②金属蛋白酶抑制剂;③天冬氨酸蛋白酶抑制剂;④丝氨酸蛋白酶抑制剂。还可根据其结构、功能及生化特性进一步分类,如 Bowman-Birk 型丝氨酸蛋白酶抑制剂、谷物胰蛋白酶/α-淀粉酶抑制剂、半胱氨酸蛋白酶抑制剂、金属羧肽酶抑制剂、芥子胰蛋白酶抑制剂、马铃薯Ⅰ型蛋白酶抑制剂、马铃薯Ⅱ型蛋白酶抑制剂、丝氨酸蛋白酶抑制剂超家族、大豆胰蛋白酶抑制剂以及南瓜抑制剂等。随后,Rawlings 等[143] 提出了一种新的蛋白酶抑制剂分类系统,将其划分为不同的蛋白酶抑制剂、家族和族群三级分层结构,其核心目标是通过基因序列揭示蛋白酶抑制剂间的进化关系。另外还有两种分类方式尚在探索阶段,本文将结合 Laskowski 与 Kato 分类体系探讨部分蛋白质类植物毒素的效应。

表 2　植物有毒苷类化合物

分类(结构)	亚组	毒性作用	植物来源
氰苷(一个中心碳原子通过单键连接 CN 基团和两个取代基，并通过糖苷键与单糖或二糖相结合)[115]	苦杏仁苷(amygdalin)	使含三价铁离子(Fe^{3+})的酶类失活，可显著抑制氧代谢过程，尤其对心肌细胞和脑细胞影响最为显著；导致呼吸困难、头晕、腹泻、呕吐、腹痛等不良反应，严重时可导致死亡[116]	广泛分布于约 2 000 个物种中，涵盖 110 科(如蔷薇、禾本科、蝶形花科、大戟、玄参科等)，其中包括多种食用水果及其种仁，如桃、李、杏、苹果及食用杏仁[116]
	蜀黍苷(dhurrin)	发生水解释放氢氰酸引发生物死亡[117]	高粱属[117]
致甲状腺肿糖苷(含硫化合物)[118]	硫苷(glucosinolates)	致动物甲状腺肿大、血浆甲状腺激素水平降低、肝脏与肾脏等器官病理改变、生长发育迟缓、繁殖性能下降、贫血，甚至导致死亡[119]	芸薹属[118]
香豆素苷(由苯并吡喃酮结构组成的含氧杂环化合物)[120]	香豆素(coumarin)	通过抑制维生素 K 活性干扰凝血酶原合成，从而降低血液凝固性，引起出血毒性反应；肝坏死[120-122]	广泛分布于双子叶植物、单子叶植物等被子植物中；十字草属、九里香属[120,122-123]
甾体苷(苷元部分具有与甾体激素相似的基本骨架)[120]	洋地黄皂苷(digitonin)	急性中毒可引起神经、视觉及胃肠道效应，电解质紊乱及非特异性心脏症状，且症状显著滞后于血清峰浓度；慢性中毒胃肠道症状轻微，神经系统表现(嗜睡、意识模糊、谵妄、定向力障碍及乏力)更为显著；视觉改变更为常见，可表现为色觉异常、暗点形成甚至失明[124]	毛地黄属、铃兰属、夹竹桃属、黄花夹竹桃属、北美罗布麻、羊角拗属、海葱属[120]
	强心内酯(cardenolides)	Na^+/K^+-ATP 酶的特异性抑制剂[125]	马利筋属[125-126]
硝基糖苷(硝基与糖基通过糖苷键结合)[127-128]	硝基丙醇糖苷(nitropropanol glycosides)	经肝脏乙醇脱氢酶转化为硝基丙酸，硝基丙酸通过不可逆抑制琥珀酸脱氢酶活性，从而阻断 ATP 合成，导致高铁血红蛋白血症；呼吸困难、肌肉协调障碍、精神沉郁、体重减轻、心率加快、大小便失禁、运动失调、唾液分泌过多，甚至导致死亡，牛中毒会出现蹄跟叩击症[129-130]	黄芪属，其他能合成硝基丙酸的豆科植物还包括霉甘蔗、冠花豆属、木蓝属、百脉根属、马蹄豆属[129-130]
吡啶衍生物糖苷(糖基与嘧啶环 C-5 位羟基结合)[131]	蚕豆嘧啶葡糖苷(vicine)	引发溶血[132]	蚕豆[132]
致钙化苷类化合物(含有维生素 D 固醇核)[133]	1，25-二羟基维生素 D(1，25-dihydroxyvitamin D)	软组织钙化、高钙血症、高磷血症、甲状旁腺功能减退、降钙素分泌亢进、骨坏死及骨硬化[134]	茄属、夜香树属、穗三毛草属、赛亚麻属、钝叶草属[133-134]
二萜类糖苷(具有贝壳杉烷型结构)[135]	羧基苍术苷(carboxyatractyloside)	昏迷、呕吐、低血糖、癫痫发作、脑缺氧、上腹部疼痛、肌无力，毒性是苍术甘的 10 倍[136-137]	苍耳属[137]
	苍术苷(atractyloside)	昏迷、呕吐、低血糖、黄疸、尿毒症、肝、肾、心脏毒性、蛋白尿、糖尿、酮尿、出血[136-137]	苍耳属[137]
植物雌激素(分子结构可模拟雌二醇的构型)[138]	异黄酮(isoflavones)	干扰雌激素的生理效应，还会引发生殖系统发育异常[139]	大豆属、苜蓿属、豌豆属、菜豆属[139]

α-淀粉酶蛋白质抑制剂于 60 多年前在小麦胚乳中被发现[144]，对昆虫及哺乳动物的 α-淀粉酶具有抑制活性[145]，能产生毒性甚至致死效应，或可诱导其取食习性的适应性演化[146]。在小麦中，二倍体抑制剂可强烈抑制昆虫 α-淀粉酶活性，但对哺乳动物唾液或胰腺 α-淀粉酶的抑制活性较弱甚至完全缺失；相反，四、六聚体抑制剂通常对哺乳动物来源的酶具有更强抑制效果[147]。部分蛋白(如谷物胰蛋白酶/α-淀粉酶

抑制剂家族)是通过吸入或摄入谷物面粉引发过敏性疾病的主要过敏原[145]。动物实验显示,摄入高活性大豆胰蛋白酶抑制剂不仅降低了食物转化效率,还增加了胰腺重量和腺泡细胞分泌[148]。还有研究显示[149],生大豆抑制幼龄动物的生长,长期饲喂后增加胰腺对致癌物的敏感性及肿瘤发生率,改变人类胰液分泌。

植物凝集素是一类糖结合蛋白,具有调节细胞粘附、糖蛋白合成以及血液蛋白水平的作用[150]。早在 1888 年就观察到凝集素具有红细胞凝集特性[151]。这种存在于豆类及其他可食用植物产品中的膳食凝集素,经口服摄取后可进入循环系统,触发人嗜碱性粒细胞释放白细胞介素-4 和白细胞介素-13,在Ⅰ型过敏反应中发挥重要作用[152]。若食用处理不当的豆类可引发腹泻、呕吐、肌肉疼痛、横纹肌溶解及中毒性心肌炎等[153]。

研究表明[154-155],食物源所含的硫胺素酶会导致硫胺素耗竭,引发相关疾病的发病率和死亡率升高。该酶存在某些蕨类植物、细菌及鱼类(鲤科鱼类)中。

2.4 氨基酸及其衍生物

植物体内存在 300 余种非蛋白质氨基酸,并广泛存在于动物饲料和人类食品中。部分非蛋白质氨基酸存在于野生植物中,并在农作物采收过程中被无意混杂带入。一些非蛋白质氨基酸及其代谢物对人类具有毒性,潜在毒素可能通过动物中间体沿食物链传递[24,156]。

含羞草氨酸存在于热带饲用植物银合欢中,L-含羞草氨酸中毒所引发的症状包括营养不良、体重增长迟缓、白内障、脱毛、不育及流产,存在一定的致畸风险,还可导致动物甲状腺肿大,对雄性生殖系统具有直接毒性作用[157]。这种由 L-含羞草素引发的多重毒性效应,被认为是通过抑制酪氨酸代谢并取代该氨基酸在生物活性蛋白中的位置而实现的。

植物中色氨酸多见于土豆、大豆和坚果[158],色氨酸作为膳食必需氨基酸,已被证实与一种可能致命疾病——嗜酸性粒细胞增多-肌痛综合征相关。特征性临床表现包括剧烈且致残的肌痛以及显著的外周血嗜酸性粒细胞增多,可能伴有血管炎、神经病变及肺部受累等表现[159]。色氨酸及代谢产物还增加慢性肾脏病患者血栓形成风险[160],增加化学致癌物对肿瘤发生的诱导作用[161]。

蚕豆富含二羟基苯丙氨酸(左旋多巴)[162]。研究表明[163],左旋多巴是一种强效毒素,对多种神经元和非神经元细胞具有致死性,其通过触发细胞凋亡来杀死细胞。但没有证据表明它在体内会对动物和帕金森病患者的黑质纹状体多巴胺能神经元造成损害。

硒代氨基酸作为具有关键生物学功能的硒蛋白的组成成分,硒以特殊形式被掺入氨基酸中。部分蔬菜品种具有硒富集特性,如十字花科蔬菜、大蒜和洋葱[164],还有一些植物,如黄芪属、假含羞草属、木根菊属、向日葵属、紫菀属、亚麻荠属、苜蓿属[165]。超富集植物中积累的硒形态以有机态的甲基硒半胱氨酸为主,而非富集型及中等富集型植物中则仍以无机态的硒酸盐为主要存在形式[166]。由于硒与硫的化学性质相似,参与硫代谢的大多数酶无法区分这两种元素。因此,在酵母和高等植物中,无机硒(硒酸盐、亚硒酸盐)可利用硫同化途径形成硒代氨基酸(硒代蛋氨酸、硒代高半胱氨酸及硒代半胱氨酸)。硒代氨基酸可诱导活性氧依赖性脱氧核糖核酸链断裂和/或碱基氧化,进而通过诱导凋亡或坏死导致细胞死亡[167]。动物研究表明[168],高硒饮食可致体重下降、共济失调,甚至瘫痪或死亡。

山黧豆中毒的临床特征表现为对称性痉挛性下肢无力不伴感觉功能异常,亚急性或隐匿性发病。草豌豆中含有神经毒素 L-α,β-二氨基丙酸(亦称 β-草酰基-L-丙氨酸),若过量食用可导致山黧豆中毒[169]。部分山黧豆物种(如香豌豆)含有 β-氨基丙腈,该成分可在实验动物中导致骨骼("骨性山黧豆中毒")和血管("血管性山黧豆中毒")的病理改变,而不损伤神经系统。然而,其相关化合物二甲基氨基丙腈和 β,β'-亚氨基二丙腈分别对人类和动物具有慢性神经毒性[170]。

亚麻籽及亚麻籽粕中的亚麻素水解后会产生具有毒性的 1-氨基-D-脯氨酸,是维生素 B_6 拮抗物[171]。缺乏维生素 B_6 会出现贫血、皮炎、舌炎、脑电图异常、抑郁、困惑和易怒等症状,可导致人体免疫系统功能受损以及炎症反应发生[172],还可致大鼠肝中央静脉及肝血窦扩张、轻度脂肪变性及肝脏甘油三酯含量升高[173]。

穗花木兰氨酸是一种非蛋白质源性、具有肝毒性的精氨酸类似物,仅存在于木蓝属植物中,并可在采食该类植物的动物组织内累积,导致原本健康的动物出现肝功能异常[174]。犬类因食用受穗花木兰氨酸污染的肉类导致死亡,人类食用受污染肉类后可能会出现肝毒性[175]。

芸薹贫血因子的化学本质为 S-甲基半胱氨酸亚砜,主要存在于羽衣甘蓝、芜菁等芸薹属饲用植物中。在几种可食用芸苔属植物中,S-甲基半胱氨酸亚砜含量存在显著差异,西兰花和花椰菜花朵的含量为 2.4 mg/g(鲜重)、卷心菜和芥菜叶的含量为 0.3 mg/g,

老芜菁根中的含量为 0.043 mg/g。过量食用可导致动物溶血性贫血和血红蛋白尿[176-177]。

2.5 多肽类毒素

鹅膏(真菌)毒素是一类水溶性、热稳定的多肽类物质,主要存在于鹅膏属、盔孢伞属及某些环柄菇属真菌中。毒鹅膏的主要毒素为 α-鹅膏蕈碱,是一种环状八肽。该毒素可强效抑制 RNA 聚合酶活性,从而阻断肝细胞和肾细胞中信使核糖核酸及蛋白质合成[178],引发严重肝损伤,可迅速危及生命[179]。

2.6 脂类毒素

芥酸是一种单不饱和脂肪酸,主要合成于十字花科多种植物的种子中,其中甘蓝型油菜、芜菁及埃塞俄比亚芥被认为是其最丰富的来源[180]。由于部分研究提示,其可导致大鼠心脏脂肪浸润、肝脂肪变性,多年来芥酸一直被归类为应避免使用的有毒物质;然而,芥酸对人类的心脏不良反应尚未得到证实,且动物模型实验存在诸多局限性[181]。

棉籽中含有三种主要的环丙烯脂肪酸:二氢苹婆酸、锦葵酸和苹婆酸。研究表明[182],当动物饲料中含有棉籽产品时,这些酸类物质会干扰其体内的脂肪酸去饱和酶活性;家禽饲料中添加棉籽产品会导致蛋黄变色。研究显示[183],环丙烯脂肪酸在虹鳟鱼中可作为黄曲霉毒素及原发性肝脏致癌物的协同增强剂。

2.7 结合金属毒素

草酸是一种有机二元羧酸,具有腐蚀性,溶于水,并且对钙和镁具有显著的亲和力,其盐类在水中的溶解度相对较低,菠菜、大黄、君迭菜、苋菜富含草酸盐。草酸中毒可导致人类、家畜及家庭宠物发病甚至死亡。草酸盐中毒的死亡通常归因于低钙血症,而慢性中毒多归因于因肾脏损伤导致的尿毒症[184]。

植酸可通过螯合矿物质和微量元素(钙、铁、锌),限制其生物利用度并降低吸收率。全谷物、豆类、蔬菜、种子和坚果是健康膳食的核心组成部分,不过其中多数植酸含量相对较高[185-186]。

2.8 聚炔醇类毒素

毒芹素是毒芹属的主要毒性成分,属于 C17-聚乙炔类化合物,具有长链 π 键共轭体系[187]。口服后会感到恶心、头晕、胃痛和腹泻,随后昏迷、针尖样瞳孔(脑缺氧或缺血可能出现极度瞳孔散大)、结膜充血、紫绀、大量出汗和过度流涎、腮腺明显肿大、间歇性的肌肉痉挛、颈部过伸、有时出现角弓反张、化验可见严重的代谢性酸中毒及显著升高的肌酸激酶[188]。该毒素对中枢神经系统具有致命毒性,可导致呼吸麻痹、心律失常、横纹肌溶解症、肾功能衰竭和死亡,硫喷妥钠是此种情况下的首选巴比妥类药物[187-189]。

2.9 醌类毒素

醌类主要作为电子传递链的组成部分,参与细胞呼吸和光合作用,包括泛醌(辅酶 Q)、质体醌和甲基萘醌类(维生素 K)。醌的毒理学效应受到取代基的调节,这些取代基决定了其氧化性和亲电性[190]。

金丝桃素是贯叶连翘(俗称圣约翰草)中的萘骈二蒽酮类化合物,其作为主要光敏剂,可能引发光细胞毒性和光皮炎,进而导致皮肤损伤,也可能会引起胃肠道症状、白内障、头晕与疲倦[191-192]。

2.10 酚类毒素

酚类化合物是一大类植物次生代谢产物,其化学结构特征为至少含有一个酚羟基。仅具单个苯环结构的分子可称为“酚”,而含有两个及以上苯环的分子则称为“多酚”。根据化学骨架的不同,酚类化合物可分为四大类:酚酸、芪类、木脂素和类黄酮。酚酸的特征是一个酚环通过短烃链与羧酸共轭连接,芪类、类黄酮和木脂素则均含有两个酚环,分别通过二碳桥(芪类)、含氧杂环(类黄酮)或丙基侧链的 β-碳(木脂素)相连[193]。

棉酚是棉花植物茎、叶、种子和花蕾中产生的酚类化合物。棉籽粕作为棉花的副产品富含油脂和蛋白质,其中的游离棉酚可导致急性棉酚中毒,临床症状包括呼吸窘迫、体重增长受阻、厌食、虚弱、心力衰竭、嗜睡,可在数日后死亡,而最常见的毒性作用是对雄性和雌性动物生殖功能的损害。其与铁结合形成棉酚-铁复合物,抑制铁的吸收,影响红细胞生成。此外,棉酚会促进红细胞脆性增加,并通过提高胞质 Ca^{2+} 活性引发红细胞凋亡,导致贫血。棉酚的另一重要毒性效应是干扰免疫功能,降低动物对感染的抵抗力并削弱疫苗效力。加热处理可以降低游离棉酚浓度,但是结合态的棉酚在消化过程中可被重新释放为游离形式[194]。

单宁是一类水溶性多酚类植物提取物,其分子大小与结构复杂度各异。分子量为 500～3 000 Da 的单宁为有效的鞣制剂,而低分子量(<500 Da)与高分子量(>3 000 Da)酚类化合物则无此活性。单宁可分为可水解单宁(包括鞣花单宁和没食子单宁)和缩合单宁两大类[195]。牛和羊的急性中毒疾病与其摄食栎属植物及多种热带豆科植物中的可水解单宁酸有关[196]。缩合单宁不能被酶、碱或酸处理水解,长期储存亦不分解,常进一步缩合形成不溶于水的聚合物,即“红粉单宁”[195]。高粱与豆类中的单宁为缩合单宁,可与蛋白质相互作用形成单宁-蛋白质复合物,导致酶失活或蛋白质不溶性增加,进而抑制消化酶活性,降低蛋白质与氨基酸生物利用率,阻碍矿物质吸

收，干扰维生素代谢并抑制生长发育[197]。

2.11 倍半萜内酯类毒素

倍半萜内酯类化合物是菊科植物中典型的一类次生代谢产物[198]，具有广泛的生物活性，这些活性主要源于其烷基化能力，可导致接触性皮炎，并引起农场动物多种中毒综合征。近年来，其遗传毒性、胚胎毒性和致突变性受到关注[199-200]。

3 总结及展望

综上所述，植物虽为生命提供基础营养与药物资源，但其产生的次生代谢产物（如生物碱、苷类、蛋白质毒素及酚类化合物等）可通过抑制关键酶、干扰代谢通路或破坏细胞结构引发中毒，临床表现涵盖神经损伤、肝、肾毒性、溶血及器官衰竭等。毒性效应受物种敏感性、植物生长阶段、环境因素及管理方式共同影响。未来还需在植物毒素毒性机制深度解析、风险评估与精准防控、药用价值与毒性平衡、多学科协作与公众教育、环境与气候变化的关联研究等方面进一步探索。

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