

活性羰基化合物及其对食品加工过程中化学危害物形成的影响

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摘 要: 活性羰基化合物(reactive carbonyl species, RCSs)是一类活泼的醛酮类物质。RCSs在体内通过脂质氧化和糖酵解等反应形成,也可在食品加工过程中通过脂质氧化和美拉德反应形成。RCSs在体内通常维持一个低浓度水平,当过量存在时会引起机体系列病变发生。人体摄入RCSs主要源于加工食品,与此同时,食品加工过程中的RCSs还会诱导其他加工危害物的形成,因此控制食品中RCSs的形成对于营养健康至关重要。本文对RCSs的形成、生物学活性,及其对食品加工中杂环胺、晚期糖基化终末产物、丙烯酰胺以及多环芳烃等加工危害物形成的影响进行综述,以期对食品加工中RCSs及其相关食品加工安全控制提供参考。

关键词: 活性羰基化合物; 食品安全; 杂环胺; 晚期糖基化终末产物; 丙烯酰胺; 多环芳烃

An Update on Reactive Carbonyl Species and Their Effects on the Formation of Chemical Hazards during Food Processing

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Abstract: Reactive carbonyl species (RCSs) are a class of active aldehydes and ketones. RCSs are formed in the body through a series of reactions such as lipid oxidation and glycolysis as well as through lipid oxidation and the Maillard reaction during food processing. RCSs are usually kept at a low concentration level in the body, while high level of these substances can cause diseases in the body. The intake of RCSs by the human body mainly comes from processed foods. Furthermore, RCSs can induce the formation of other hazards during food processing. Therefore, controlling the formation of RCSs in foods is crucial for nutritional health. This article reviews the formation and biological activity of RCSs as well as their impacts on the formation of chemical hazards such as heterocyclic amines, advanced glycation end products (AGEs), acrylamide, and polycyclic aromatic hydrocarbons in food processing. The aim of this study is to provide a reference for the safety control of RCSs during food processing.

Keywords: reactive carbonyl species; food safety; heterocyclic amines; advanced glycation end products; acrylamide; polycyclic aromatic hydrocarbons

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活性羰基化合物 (reactive carbonyl species, RCSs) 是含有一个或多个羰基官能团的亲电试剂, 通常是醛酮类物质。RCSs不仅来源于内源性代谢、脂质过氧化和微生物组活性, 还来源于外源性食品和饮料摄入^[1]。根据相对分子质量和结构不同, RCSs分为3类, 即相对分子质量小于120的单羰基物质、介于120~200之间的单醛类物质和二羰基类物质, 具体为: 1) α,β -不饱和醛类 (4-羟基壬烯醛、壬烯醛、丙烯醛 (acrolein, ACR) 等); 2) 醛酮类 (甲基乙二醛 (methylglyoxal, MGO)、4-氧代壬烯醛等); 3) 二醛类 (乙二醛 (glyoxal, GO)、丙二醛 (malondialdehyde, MDA) 等)。大多数RCSs的代谢产物都是醛类物质, 能形成大量水合物^[2], 可通过体内和食物中的脂质氧化等化学反应产生。目前经鉴定, 已鉴别出了20多种不同结构的RCSs, 常见的结构如图1所示。同时研究表明, RCSs超过特定的生理浓度 (具体不同类型的RCSs的正常浓度标准不统一) 会诱导人体众多疾病形成, 例如糖尿病、不孕不育、阿尔茨海默病和癌症等一系列严重的慢性疾病^[3]。

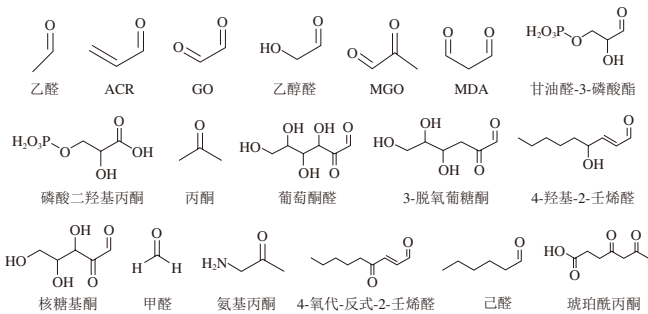


图1 常见RCSs化学结构式

Fig. 1 Chemical structures of common RCSs

1 RCSs的来源

RCSs的来源主要可分为内源性和外源性。

1.1 内源性

大到人类, 小到细菌, 生物体内RCSs都保持在一定浓度范围内。RCSs主要源于糖类^[4]、氨基酸^[5]和脂质^[6]的氧化。研究表明, RCSs主要通过非酶促反应和酶促反应形成, 非酶促反应主要包括糖化、氨基酸氧化和脂质过氧化等反应途径, 酶促反应包括多元醇途径和糖酵解途径, 具体如表1所示。

表1 酶促/非酶促反应形成的RCSs^[7]

Table 1 RCSs from enzymatic/non-enzymatic reactions^[7]

酶促反应		非酶促反应		
糖酵解途径	多元醇途径	脂质氧化	氨基酸氧化	糖化
乙醛、二氧丙酮、磷酸盐、MGO、甘油醛-3-磷酸酯	3-脱氧葡萄糖酮	4-羟基-反-2-壬烯醛、MDA、GO、MGO、ACR、4-氧代-反-2-壬烯醛、己醛、巴豆醛	GO、MGO、2-羟基丙酮、乙醇醛、糖酮、3-脱氧葡萄糖酮、ACR	GO、MGO、葡萄糖酮、ACR

1.1.1 非酶促反应

在生物体内, 内源性4-羟基壬烯醛 (4-hydroxynonenal, 4-HNE) 和MDA被视为脂质氧化的代表产物, 可通过检测其含量判断生物体内脂质氧化程度^[8], 因此是相对重要的两种RCSs。

4-HNE主要源于亚油酸 (linoleic acid, LA) 和花生四烯酸 (arachidonic acid, ARA) 等 ω -6多不饱和脂肪酸的氧化反应, 首先是脂肪酸氧化生成脂质过氧化氢 (lipid hydroperoxides, LOOH), 然后LOOH氧化分解形成4-HNE^[9]。以LA为例, 脂肪酸自氧化生成LOOH时, LA中C11上的活性双烯丙基亚甲基先失去一个氢原子形成离域的戊二烯基自由基, 自由基末端位置C9和C13与氧发生反应生成9-氢过氧-10,12-十八碳二烯酸 (9-hydroperoxy-10,12-octadecadienoic acid, 9-HpODE) 和13-氢过氧-9,11-十八碳二烯酸 (13-hydroperoxy-9,11-octadecadienoic acid, 13-HpODE) 等氢过氧混合物, 两种混合物进一步反应生成4-HNE^[10]。具体的步骤为: 9-HpODE生成的烷氧基自由基环化形成烯丙基自由基, 烯丙基自由基进一步自动氧化成环状过氧化氢, 环状氢过氧化物再还原成烷氧基自由基, 并经过 β -断裂生成以碳为中心的自由基, 最后, 碳为中心自由基氧化水解形成4-HNE; 13-HpODE生成的烷氧基自由基环化成环过氧化氢, 在路易斯酸和伯酸的催化下生成环氧-氢过氧化物, 再转化形成4,5-环氧醛, 4,5-环氧醛不稳定, 在温和条件下容易水解生成4-HNE^[8]。

1.1.2 酶促反应

酶促反应也可生成MDA: ARA通过环加氧酶氧化生成前列腺素 (prostaglandin, PG) G2, 前列腺素氢过氧化物酶使PGG2获得两个电子, 从而还原为PGH2, 然后在血栓素合成酶作用下形成MDA, 同时生成血栓素A2^[11]。此外, 精胺可在多胺氧化酶作用下反应形成3-羟基丙醛, 3-羟基丙醛再在氨氧化酶作用下氧化形成MDA^[12]。这是内源性MDA形成的主要两种途径。

糖类的氧化酵解可形成GO和MGO等: 葡萄糖通过C2或C3的羟基去质子化诱导逆向羟醛缩合反应直接裂解形成GO, 乙醇醛中间体氧化也可形成GO^[13]; MGO的形成是糖发生酵解过程时, 随着反应过程中甘油醛-3-磷酸和磷酸二羟基丙酮的降解、氨基丙酮的氧化以及脂质过氧化产物的分解形成^[14]。脂质氧化和糖酵解反应是RCSs形成的主要原因。

1.2 外源性

外源性RCSs主要源于加工食品的摄入和环境污染物的暴露。ACR、巴豆醛、GO和甲醛等RCSs普遍存在的工业污染物,很容易从环境和大气中通过呼吸进入人体^[15-16]。有机药物、香烟烟雾、酒精^[17]和加工食品等也是外源性RCSs来源的重要途径^[18-20]。研究表明,ACR是香烟烟雾中的主要成分,含量达到54~155 $\mu\text{g}/\text{支}$ ^[21]。外源性RCSs主要源于各类加工食品, α -二羟基类化合物可由富含碳水化合物的食品原料经高温热加工形成,各类食品在油炸过程中可产生 α,β -不饱和羰基化合物,美拉德反应终产物中也存在大量RCSs^[22]。MGO是美拉德反应的标志性中间产物,具有较强的反应活性^[23]。在美拉德反应期间,葡萄糖和果糖与氨基酸反应生成席夫碱,进一步转化形成Amadori重排产物,Amadori重排产物经过烯醇化、脱氢和脱水后形成1-脱氧葡萄糖酮和3-脱氧葡萄糖酮,两种化合物裂解后均可形成MGO^[24]。此外,一些含糖量较高的食物会发生自氧化生成MGO^[25]。肉制品加工过程中RCSs主要源于脂肪的氧化裂解以及糖类的高温氧化裂解,具体的形成通路如图2所示。

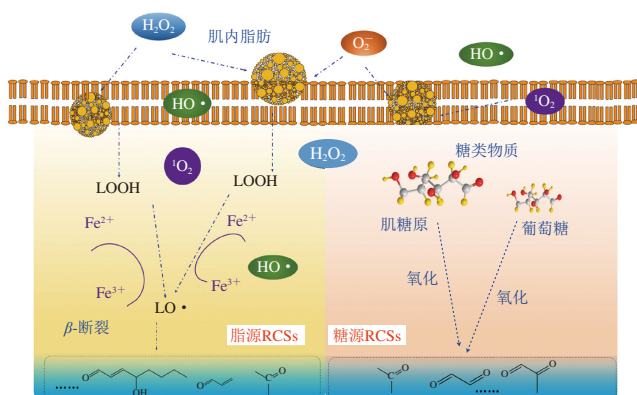
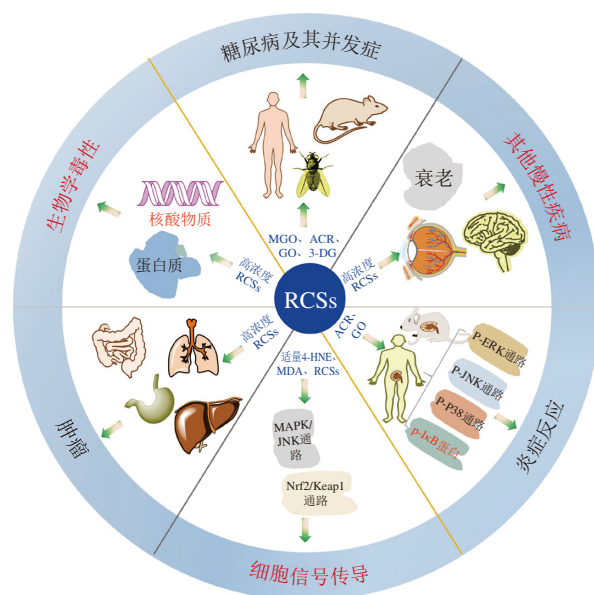


图2 肉制品高温加工过程RCSs主要内源形成途径

Fig. 2 Major intrinsic formation pathways of RCSs in meat products under high temperature treatment

2 RCSs的生物学活性

在细胞中RCSs的浓度并没有统一的标准,这是由于细胞具有不同种类、结构和生长时期,所处代谢强度、氧浓度以及温度等原因也会导致其出现差异,且细胞中RCSs的含量是一个动态平衡状态,会同时产生、降解和排泄。不同浓度的RCSs具有不同的生理活性,具体如图3所示。



3-DG. 3-脱氧葡萄糖醛酮 (3-deoxyglucosone); MAPK. 丝裂原活化蛋白激酶 (mitogen-activated protein kinase); JNK. Jun 氨基末端激酶 (Jun N-terminal kinase); Nrf2. 核因子-红血细胞2相关因子2 (nuclear factor erythroid 2-related factor 2); Keap1. Kelch样ECH相关蛋白1 (Kelch-like ECH-associated protein 1); P-ERK. 磷酸化细胞外信号调节激酶 (phospho extracellular signal regulated kinase); P-JNK. 磷酸化c-Jun N末端激酶 (phospho c-Jun N-terminal kinase); P-P38. 磷酸化p38激酶 (phospho p38 kinase); p-IκB. 磷酸化核因子κB抑制蛋白 (p-inhibitor of nuclear factor-κB)。

图3 RCSs的生物学活性及毒性

Fig. 3 Biological activities and toxicity of RCSs

2.1 细胞信号转导

人体血液中MGO的生理浓度为0.12~0.65 $\mu\text{mol}/\text{L}$ 时,可以调节细胞信号转导和基因表达^[26],MGO在体内可以被乙二醛酶、醛糖还原酶和醛脱氢酶作用失去毒性,进而防止蛋白质和DNA糖基化^[27],从而避免机体一些疾病发生及衰老进程的加快。RCSs可以通过抑制噻唑类化合物的活化、激活线粒体凋亡途径以及通过抑制脂氧合酶、细胞色素P450和环加氧酶的表达来抑制前列腺癌细胞的增殖^[28-30]。适量4-HNE通过直接结合或间接激活人体氧化还原敏感的MAPK的级联反应激活JNK的信号通路^[31-32]。4-HNE和MDA可以通过Nrf2抗氧化应激通路与体内的亲电性物质反应元件和抗氧化反应元件结合调节Nrf2/Keap1信号通路参与应激适应^[33-34],从而促进抗氧化基因的转录,最终保护细胞免受过度的应激反应造成细胞损伤。

2.2 介导慢性疾病

2.2.1 羰基应激

高浓度RCSs通过共价修饰使蛋白质构象发生变化,会引起催化位点畸变,造成蛋白质功能受损,进而破坏正常生理功能^[35-36],引起“羰基应激”效应。羰基应激可

导致碳水化合物和脂质的氧化加剧,特别是活性氧稳态水平的增加^[37],以及细胞代谢紊乱^[38-39]。高浓度RCSs引起的氧化应激还可以通过损伤睾丸来阻止精子的生成、诱导间质细胞凋亡和减少类固醇合成等方式,进而损害男性生育能力^[40]。氧化应激还可以通过损伤DNA、抑制信号传输和免疫应答等多种方式诱导人体癌症的发生^[41]。

2.2.2 糖尿病及其并发症

果蝇模型实验中,随着果蝇体内的MGO浓度逐渐升高至对照组的1.5倍,果蝇出现了肥胖、高血糖和胰岛素抗性增加等症状,同时伴随着2型糖尿病的出现,表明MGO升高可能会导致2型糖尿病出现^[42]。Hanssen等^[43]随访了1 010名1996年1月至2006年3月患1型和2型糖尿病患者,对他们的体内MGO、GO和3-DG浓度进行检测,结果发现,相较于健康人而言,患者体内的MGO、GO和3-DG高出近2~4倍。连续28 d给雄性SD大鼠注射60 mg/(kg·d) MGO或饮水中添加1 mg/100 mL MGO,会导致大鼠体内的葡萄糖水平升高至正常水平的1.72倍,从而使胰岛素受体底物失活和脂肪细胞中葡萄糖转运蛋白4表达分别降低至对照组的1/2和1/4,诱导了胰岛素抵抗的发生并伴随糖尿病的形成^[23,44]。因此,血浆中MGO浓度水平可以反映出人体或者其他生物一定的健康水平。

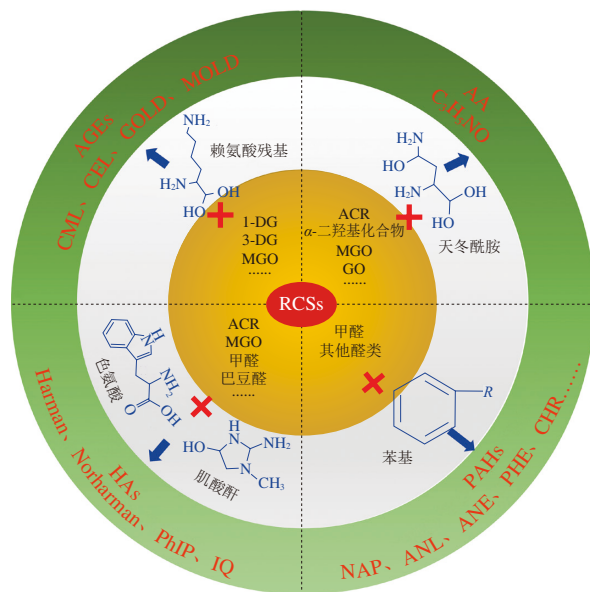
2.2.3 诱发炎症反应

当人的主动脉内皮细胞暴露于100 μmol/L GO后,细胞内的MAPK途径被激活,磷酸化的MAPK途径表达水平提高,包括P-ERK、P-JNK和P-P38通路,从而加重细胞的炎症损伤^[45]。Liu Dan^[46]和Zhu Jinjin^[47]等在研究GO对人胚胎肾细胞中核因子-κB (nuclear factor-κB, NF-κB) 通路影响时发现,当GO浓度为0.5、1 mmol/L和2 mmol/L时,p-IκB蛋白的表达逐渐上调,并且p-IκB/IκB的比率显著增加。ACR可通过以下两种方式来激活细胞中NF-κB通路,进而促进炎症的形成^[48]:1)与生物大分子结合来产生过量的活性氧,随后进一步激活NF-κB通路^[34];2)ACR通过影响人体巨噬细胞来增加炎症细胞因子肿瘤坏死因子-α (tumor necrosis factor-α, TNF-α) 的生成和表达,这种因子可以激活NF-κB^[49]。

3 RCSs介导的食品加工危害物的形成

杂环胺(heterocyclic amines, HAs)、晚期糖基化终末产物(advanced glycation end products, AGEs)、丙烯酰胺(acrylamide, AA)以及多环芳烃(polycyclic aromatic hydrocarbons, PAHs)等是高脂肪、高碳水和高蛋白质食物原料在热加工过程中形成的典型食品加工危害物。Ding Xiaoqian等^[50]在烤羊肉中检测到9H-吡啶[4,3-b]吡啶(9H-pyrido[4,3-b]indole, Norharman)、2-

氨基-1-甲基-6-苯基咪唑[4,5-b]吡啶(2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine, PhIP)和1-甲基-9H-吡啶[4,3-b]吡啶(1-methyl-9H-pyrido[4,3-b]indole, Harman)等多种HAs。Xue Chaoyi等^[51]在烤牛肉饼中检测到N^ε-羧甲基赖氨酸(N^ε-(carboxymethyl)-L-lysine, CML)和N^ε-羧乙基赖氨酸(N^ε-(carboxyethyl)-L-lysine, CEL)两种AGEs。Shi Haonan等^[52]在油炸鱼饼时发现,AA含量达到26 μg/kg。高脂肪、高碳水和高蛋白质等食物原料在高温热处理过程中往往生成种类各异、含量各异的RCSs。Papastergiadis等^[53]研究表明,使用腌制以及热加工处理肉制品时,HNE含量平均值达110 μg/kg,最高可达2 047 μg/kg;而MDA的含量平均值为106 μg/kg,最高达882 μg/kg。近年来,越来越多的研究表明食品加工过程中形成的RCSs与HAs、AGEs、AA和PAHs等加工危害物的形成具有密切的关联性,一些RCSs是诱导上述加工危害物形成的主要原因,具体如图4所示。



NAP.萘(naphthalene); ANL.苊烯(acenaphthylene); ANE.范(acenaphthene); PHE.菲(phenanthrene); CHR.蒽(chrysene); IQ. 2-氨基-3-甲基咪唑[4,5-f]喹啉(2-amino-3-methylimidazo[4,5-f]quinoline); GOLD.乙二醛-赖氨酸二聚体(glyoxal-lysine dimer); MOLD.丙酮醛-赖氨酸二聚体(methylglyoxal-lysine dimer)。

图4 RCSs介导的食品加工过程中形成的危害物

Fig. 4 Formation of hazardous compounds induced by RCSs during food processing

3.1 RCSs对HAs形成的影响

3.1.1 HAs

HAs是一类在高温热处理加工中由蛋白质、氨基酸等经美拉德反应或热解反应形成的有毒有害物质^[54]。根据生成温度和化学结构差异,可以分为极性HAs(主要可分为喹啉类、喹啉类、吡啶类和咪唑吡啶

类)和非极性HAs(主要可分为 α -吡啶类、 β -吡啶类、 γ -吡啶类和 ξ -吡啶类)^[55-56]。常见的极性HAs主要有IQ、2-氨基-3,4-二甲基咪唑[4,5-*f*]喹啉(2-amino-3,4-dimethylimidazo[4,5-*f*]quinoline, MeIQ)和PhIP等,非极性HAs有Norharman、Harman等。HAs对人体的危害较多,主要有致突变性、致癌性、心肌毒性、神经毒性,因此受到食品安全控制高度重视^[57]。

3.1.2 RCSs对食品加工过程中HAs形成的影响

RCSs中的醛类物质在HAs形成过程中,发挥着至关重要的作用,是构成各类HAs形成的基础物质。例如,巴豆醛是形成MeIQ的底物,ACR是形成IQ和3,8-二甲基咪唑并[4,5-*f*]喹啉-2-胺(2-amino-3,8-dimethylimidazo[4,5-*f*]quinoxaline, MeIQx)的基础物质,MGO和甲醛是形成MeIQx的关键反应物^[58]。在PhIP形成过程中,苯丙氨酸发生Strecker降解反应生成苯乙醛、肌酸发生环化反应生成肌酸酐,同时,苯乙醛自身会继续反应生成苯甲醛和甲醛,当苯乙醛和肌酸酐反应到最后一步时,甲醛会将自身的碳原子掺入PhIP分子中,进而形成完整的PhIP化学分子式^[59]。Hidalgo等^[58]指出,控制RCSs可以显著抑制HAs形成。

Jing Meilin等^[60]研究表明,在PhIP化学模型中,当ACR的浓度为0.16 mmol/5 mL时,PhIP生成量较不添加时增加了622%。Hidalgo等^[61]研究发现,在反应模型体系中,ACR和MGO是可以参与并形成MeIQx的RCSs。Zamora等^[62]的研究也表明,若是化学反应体系中无巴豆醛存在,那在模型体系中就没有MeIQ形成。Hidalgo等^[63]推测,食品中产生具有氨基咪唑并氮杂芳烃结构的HAs种类和数量,是根据现有活性羰基的相对含量比例决定的。

当然,也可以添加一些物质来削弱RCSs对形成HAs的作用。例如Zhang Lang等^[64]发现,添加2%花青素可以通过减少熏鸡腿中苯丙氨酸(及其产生的苯乙醛)、肌酸酐和色氨酸等前体物质含量来抑制PhIP、Norharman和Harman的形成,其抑制率能达到64.33%、17.09%和39.36%;0.07 mmol/L姜黄素可以通过控制甲醛和乙醛等RCSs物质来抑制1,2,3,4-四氢- β -吡啶-3-羧酸,从而进一步抑制炖猪肉中Harman和Norharman的形成^[65]。与此同时,通过调控羰基-胺反应过程中的pH值、温度和加热持续时间等参数,也可有效减缓羰基和肌酐之间的相互作用^[66]。

3.2 RCSs对食品加工过程中AGEs形成的影响

3.2.1 AGEs

AGEs是由葡萄糖和果糖等糖类物质上的羰基与脂质、蛋白质或核酸上的游离氨基酸反应而生成的一系列对人体有害的物质^[67-68]。AGEs也可分为内源性和外源性,内源性AGEs是在人体器官、组织或体液中形成,外源性则是从加工食品中摄入。脂肪和蛋白质含量

高的动物源性食物中AGEs含量远高于植物源性食物,且热处理后食品中AGEs水平较未经处理的食品高出10~100倍^[69]。AGEs也被证明与女性不孕症密切相关,AGEs在卵巢上的积累会加速卵巢氧化应激并改变该部位类固醇激素水平,从而导致功能障碍,例如会出现多囊卵巢综合征、子宫内膜异位症和卵巢衰老等^[70-71]。CML和CEL是两种典型的AGEs。通过日粮中添加(76.0 \pm 15.3) mg/100 g CML和(436.9 \pm 88.1) mg/100 g CEL饲喂雌性Wistar大鼠,发现大鼠体内的血清葡萄糖、胰岛素、睾酮水平升高,雌激素、孕激素水平降低^[72]。通过小鼠模型实验研究发现,AGEs的添加能够引起小鼠睾丸和附睾的组织病理学损伤,导致附睾精子数量减少和精子异常率增加。高浓度AGEs在体内还会引起人体氧化应激、动脉粥样硬化以及一些神经性疾病等其他疾病^[73]。

3.2.2 RCSs对食品加工过程中AGEs形成的影响

AGEs主要是通过美拉德反应形成,过程主要分为两个部分。

首先,含有羰基的还原糖与蛋白质和氨基酸的游离氨基发生可逆反应,形成不稳定的席夫碱,席夫碱加合物经过环化和分子重排,转化为相对稳定的Amadori重排产物,也被称为早期糖基化产物。小部分Amadori产物可以通过Hodge途径,经历不可逆氧化、环化或水解过程,直接转化为AGEs^[74];剩余的Amadori重排产物可以通过脱水、氧化裂解或环化转化为GO、MGO、3-DG和 α -二羰基化合物等AGEs前体物质,这些RCSs化合物与未结合或已结合的氨基酸反应形成AGEs^[67]。同时,还原糖的自发氧化(Wolff途径)、席夫碱的氧化裂解(Namiki途径)和脂质过氧化(Acetol途径)可以形成相应的 α -二羰基化合物等中间体,这些中间体进一步反应最终可形成AGEs^[75]。

迄今为止,前后累计共鉴定出20多种不同类型的AGEs^[76]。RCSs可参与加热过程中的美拉德反应诱导AGEs形成,例如,在美拉德反应过程中,MGO会与赖氨酸残基反应生成CML,3-DG可以与赖氨酸残基反应生成CEL。除此之外,加工食品中还存在GOLD、MOLD和吡咯啉等形态的AGEs,它们的形成均与RCSs有一定的关系^[77]。胡本伦等^[78]在研究油炸鱼饼时发现,MDA含量与鱼饼中AGEs含量呈现一定正相关关系。Liu Guimei等^[79]通过构建赖氨酸-葡萄糖模拟体系发现,添加100 μ mol/L槲皮素可显著减少GO和MGO含量,同时进一步减少了AGEs生成,实验结果间接证明了GO和MGO是参与AGEs形成的重要物质。

3.3 RCSs对食品加工过程中AA形成的影响

3.3.1 AA

AA是富含碳水化合物的食品发生美拉德反应而产生的一种有毒副产物,具有潜在的致癌性、生殖毒

性和神经毒性,可经过呼吸道、消化道、皮肤等途径直接进入人体^[80]。Johnson等^[81]发现,当大鼠暴露于2.0 mg/(kg·d) AA时,雌性大鼠患乳腺、甲状腺、中枢神经系统、口腔组织和子宫肿瘤的发病率显著提升,雄性大鼠患甲状腺肿瘤的几率也显著增加。AA对人体具有神经毒性的机制与细胞骨架蛋白的异常变化、氧化应激、蛋白质结合、神经末梢轴突损伤和离子反应等有关^[82]。

3.3.2 RCSs对食品加工过程中AA形成的影响

美拉德反应中天冬酰胺途径是AA形成的主要途径。在美拉德反应中间阶段,天冬酰胺中的游离氨基水分被脱去,然后与具有羰基的葡萄糖、果糖和GO等RCSs缩合形成极不稳定的席夫碱,席夫碱发生Amadori重排产生重排产物,重排产物进一步脱水和脱氨形成重要的二羟基化合物等RCSs,然后RCSs通过Strecker降解途径或N-糖苷途径形成AA^[83]。与此同时,AA还可通过其他反应途径生成。葡萄糖能通过逆向醇醛反应直接转化为GO等RCSs化合物,RCSs进一步利用天冬酰胺脱氨形成AA;脱羧的席夫碱和Amadori产物可以通过形成3-氨基丙酰胺的中间产物直接或间接生成AA^[84]。

Yaylayan等^[85]研究发现,在化学模型体系添加羰基化合物,天冬酰胺会迅速降解形成AA。天冬酰胺的热解化学模型实验结果表明,添加25 mmol/L MGO时,模型系统中AA生成量能达到19 µg/g,分别是添加相同浓度葡萄糖和果糖时的3.5倍和2.4倍^[86]。许多研究结果均指出,RCSs中的ACR、GO和MGO可以诱导食品加工过程中AA的生成^[87-93]。

目前,抑制食品加工过程中AA的形成主要通过3种方式完成:1)减少原料中天冬酰胺和还原糖等AA的前体物质浓度;2)改变食品加工温度、时间和水分来减缓AA形成^[94];3)添加黄酮类、多酚类、皂苷类和萜类、醚类、生物碱等其他天然产物来抑制AA前体物形成以及羰氨反应的进程^[95]。

3.4 RCSs对食品加工过程中PAHs形成的影响

3.4.1 PAHs

PAHs是热加工食物中最常见的致癌物之一,能够引发人类乳腺癌、胰腺癌、肺癌和结肠癌等^[96],是一组由两个或两个以上苯环稠合在一起的芳香族化合物及其衍生物,常见的PAHs化合物呈无色、白色或浅黄色固体,具有疏水性、亲脂性和低生物降解性^[97]。目前鉴定出约有18种不同类型的PAHs,分别为萘、苊烯、苊、苝、菲、蒽、荧蒽、芘、苯并[a]蒽、蒾、苯并[b]蒽、苯并[k]蒽、苯并[a]芘、茚并[1,2,3-c,d]芘、二苯并[a,h]蒽、

苯并[g,h,i]花、苯并[j]荧蒽和苯并[e]芘。除了致癌性外,PAHs还具有致畸性、基因毒性和免疫毒性^[98]。

目前,食品经热加工后形成PAHs主要有3种方式:

1)在熏烤过程中,由于木柴、木炭等热源出现不完全燃烧现象,导致产生含PAHs的燃烧烟雾,随着烟雾上升,食品与燃烧产物直接接触,PAHs迁移并沉积在食品表面^[99];2)肉类制品中由于脂肪含量较高,烤制时肉中的脂肪滴落在热源上发生化学反应,其产物也会发生热反应并聚合生成PAHs,随后附着在肉制品表面,随着贮藏时间的延长,PAHs逐渐向内部渗透,从而产生更严重的污染^[100],研究表明,若是阻止掉落的油脂滴到热源上,则烧烤制品中的PAHs含量相对较低^[101];3)由于烟熏、烧烤的加工过程中温度较高,肉制品中的PAHs前体物质,如脂肪、蛋白质和碳水化合物等受热分解,经一系列反应后也会形成PAHs,进而使肉制品中PAHs含量增加^[102];同时,肉制品中的蛋白质在高温下热解能产生游离氨基酸,如天冬氨酸、脯氨酸等,它们可与葡萄糖等还原糖反应形成Amadori化合物,并进一步热解产生PAHs^[103]。

3.4.2 RCSs对食品加工过程中PAHs形成的影响

RCSs对PAHs的生成具有一定的影响。Bittner等^[104]认为,在低温环境加工条件下,苯环脱氢生成苯基,甲醛发生羟醛缩合等一系列反应生成乙烯,然后乙烯与苯基发生取代反应生成2-苯基乙烯基,苯乙烯基接着与甲醛反应生成苯丁烯基,最后通过环化生成萘。Nie Wen等^[103]通过在烤猪肉香肠中添加醛糖和酮糖判定其对PAHs生成的影响,结果表明,添加酮糖的实验组与空白组相比,总PAHs含量并无显著性差异,但添加醛糖后的处理组中总PAHs的生成率较空白组增加了2.20倍。推测是由于醛糖中含有游离醛基,在高温烤制的条件下生成了小分子醛类物质,这些醛类物质进一步发生复杂的裂解、聚合或缩合反应,最终产生更多的PAHs。

美拉德反应、脂质氧化反应均与PAHs的形成息息相关^[105]。一方面,正如前文所述,脂质氧化会生成较多的RCSs,这些物质与PAHs的形成可能有较大关系;另一方面,脂质氧化是食品系统中典型的自由基反应,氧化过程中产生的大量自由基也可能与PAHs生成有关,高反应性自由基会攻击脂质酰基链,导致脂酰基链被裂解成大量的小分子产物,这些自由基和小分子产物会发生反应,形成不稳定的不饱和烃自由基,这些自由基会重新组合,从而形成PAHs^[106]。对于美拉德反应而言,在食品中可以诱导PAHs的含量增加^[107],但具体发生了哪些化学反应诱导PAHs的生成还不可得知,推测与美拉德反应生成的醛类产物有一定关系。

4 结 语

人体摄入RCSs主要源于膳食,特别是加工食品。体内过量的RCSs对机体具有潜在的生物毒性,会引起糖尿病、炎症反应、肿瘤以及阿尔茨海默病等疾病的发生。越来越多的研究表明,RCSs会介导食品加工过程中HAs、AGEs、AA和PAHs等化合物的形成,这些化合物被证实为致畸、致突变等危害物,对人体的健康具有重要影响。因此,控制食品加工过程中RCSs的形成对于食品安全的主动防控具有重要的意义。首先,目前的研究表明,在食品热加工过程中,RCSs的形成受到加热温度、时间等反应介质条件的调控,同时也受到多酚等外源添加物的影响,因此,阐明食品加工过程中RCSs的主要形成来源、形成分子机理以及关键影响因素对于RCSs的控制至关重要。其次,食品加工过程中HAs、AGEs、AA以及PAHs等加工危害物的形成受到RCSs的影响或介导,因此,挖掘热加工过程中介导这些加工危害物形成的RCSs标志物对于加工危害物的精准靶向控制具有重要的意义。最后,RCSs种类繁多,当前并没有多种RCSs的同步检测分析方法,因此,开发食品加工过程中多种类的RCSs同步检测分析方法十分必要。

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