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DJ-1 family Maillard deglycases prevent acrylamide formation

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ABSTRACT

The presence of acrylamide in food is a worldwide concern because it is carcinogenic, reprotoxic and neurotoxic. Acrylamide is generated in the Maillard reaction via condensation of reducing sugars and glyoxals arising from their decomposition, with asparagine, the amino acid forming the backbone of the acrylamide molecule. We reported recently the discovery of the Maillard deglycases (DJ-1/Park7 and its prokaryotic homologs) which degrade Maillard adducts formed between glyoxals and lysine or arginine amino groups, and prevent glycation damage in proteins. Here, we show that these deglycases prevent acrylamide formation, likely by degrading asparagine/glyoxal Maillard adducts. We also report the discovery of a deglycase from the hyperthermophilic archaea *Pyrococcus furiosus,* which prevents acrylamide formation at 100 °C. Thus, Maillard deglycases constitute a unique enzymatic method to prevent acrylamide formation in food without depleting the components (asparagine and sugars) responsible for its formation.

1. Introduction

In 2002, acrylamide was detected in food, especially in bakery products, chips, grilled cereals, instant coffees, evaporated milk, and baby food [1,2]. Since acrylamide is carcinogenic, reprotoxic and neurotoxic, authorities recommended to maintain acrylamide as low as reasonably achievable in food products [3,4]. Acrylamide is formed at elevated temperatures (60–200 °C) from the reaction of glucose, fructose, and glyoxals (R–CO–CHO) arising from their decomposition, with the amino acid asparagine via the Maillard reaction [5,6]. This reaction, discovered by Louis Camille Maillard in 1912 [7], involves the spontaneous formation of covalent adducts (Maillard adducts) between carbonyl and amino groups. In addition to its recently discovered role in acrylamide formation, the Maillard reaction is responsible for covalent damage by reducing sugars and glyoxals, of arginine and lysine side chains in proteins, and deoxyguanine in DNA [8]. Maillard adducts undergo a series of dehydrations, oxidations and rearrangements that lead to the formation of advanced glycation end-products (AGEs) [8], causing irreversible damage to proteins and nucleic acids. The overall process called glycation is responsible for aging, cancer, atherosclerosis, cataracts, neurovegetative disorders, and renal, autoimmune, and postdiabetic diseases [8]. Similar glycation reactions, activated by elevated temperatures, also occur during food processing, cooking, sterilization and storage, and dietary AGEs are detrimental because they accelerate oxidative stress and inflammation, especially in patients with diabetes or renal failure [3,4].

We discovered recently the Maillard deglycases, which belong to the PfpI/Hsp31/DJ-1 superfamily, include the parkinsonism-associated protein DJ-1/Park7 and its prokaryotic homologs, and prevent protein glycation by degrading Maillard adducts formed between glyoxals and cysteine, arginine, and lysine side-chains [9–11]. The ability of these deglycases to prevent protein glycation by degrading Maillard adducts suggested to us that they might also prevent acrylamide formation. Actual methods to reduce acrylamide levels in food are based on reducing levels of precursors

Abbreviations: MGO, methylglyoxal; GO, glyoxal; AGE, advanced glycation end product; AA, acrylamide; BSA, bovine serum albumin.

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(asparagine, sugars) or changing process parameters (temperature, pH, ionic strength), and affect the quality parameters of foods or even cause the formation of harmful products [12]. Here, we show that DJ-1 and its prokaryotic homologs prevent acrylamide formation in asparagine/glyoxal and asparagine/sugar mixtures. Moreover, since acrylamide formation occurs at elevated temperatures, we discovered and used a novel deglycase from *Pyrococcus furiosus* to prevent acrylamide formation at 100 °C.

2. Materials and methods

2.1. Purification of the deglycases

The deglycases DJ-1 and YhbO were purified as described previously [13,14]. We purified PfpI from a *P. furiosus* protein extract by heat treatment of the extract at 98 °C for 6 h in 50 mM sodium phosphate (pH 7.5), 1 mM dithiothreitol, and 1% SDS [15]. After 6 h, most proteins were hydrolyzed (autoproteolysis), and two major bands (with estimated molecular masses of 132 and 66 kDa) could be detected by Coomassie brilliant blue staining. These bands represent the trimeric and hexameric forms of PfpI (with a monomeric molecular weight of 20 kDa) [15]. This SDS-resistant PfpI preparation is highly resistant to thermodenaturation and displays an optimal activity at 105 °C (as judged by its peptidase activity [15]). PfpI was separated from low molecular weight compounds by chromatography on a Bio-Gel P2 column (Bio-Rad) equilibrated in N_2-gassed 50 mM sodium phosphate (pH 7.5), treated with Bio-Beads SM2 (Bio-Rad) for 2 h to remove SDS, and stored at −80 °C. We confirmed the peptidase activity of PfpI by measuring the degradation of the peptide AAF-amc [15], and we checked that the purified protein was not contaminated by an asparaginase.

2.2. Deglycation of thioredoxin

E. coli thioredoxin (30 μM) in 50 mM sodium phosphate (pH 7.0) was incubated with 7 mM MGO (from Sigma Aldrich) at 70 °C in the absence or presence of YhbO or PfpI (5 μM each), and activity was measured by the insulin assay in the presence of NADPH and thioredoxin reductase [16]. We confirmed that thioredoxin is stable at 70 °C (not shown). Glycation of thioredoxin (60 μM) by GO (2 mM) in 50 mM sodium phosphate (pH 7.0), in the absence or presence of 5 μM PfpI was assayed by immunoblotting with anti-AGEs antibodies (Cell Biolabs Inc, STA-817) [9]. The results are representative of three experiments.

2.3. Stimulation by BSA of the apparent glyoxalase activity of PfpI

PfpI (1.5 μM) in 50 mM HEPES (pH 7.0) was incubated at 85 °C with 3 mM MGO in the absence or presence of 20 μM BSA and MGO levels were measured using a dinitrophenylhydrazine assay [9].

2.4. Prevention of acrylamide formation in asparagine/glyoxals mixtures

We incubated asparagine (2–6 mM) and either glyoxal or methylglyoxal (2–6 mM) for 30 min in the presence of DJ-1 (5 μM) at 55 °C, YhbO (5 μM) at 70 °C or PfpI (5 μM) at 95 °C, in 200 mM sodium phosphate pH 7.5, and 1% NH_4HCO_3. Acrylamide formation was measured by analyzing the mixtures on a C18 reverse phase HPLC column equilibrated in 20 mM K_2HPO_4, and 100 mM NaCl or on a Hypercarb HILIC column [17] (hydrophilic interaction liquid chromatography, 100 mm × 4.6 mm i.d., 5 μm particle size). Chemicals were detected by their absorbance at 206 nm. The results are representative of three experiments.

2.5. Prevention of acrylamide formation in asparagine/glucose or asparagine/fructose mixtures

We incubated asparagine and glucose or fructose (25 mM each) in the presence of PfpI (5 μM), in 200 mM sodium phosphate pH 7.5, and 1% NH_4HCO_3 for 0–130 min at 95 °C. Acrylamide formation was measured by analyzing mixtures on a Hypercarb HILIC column (hydrophilic interaction liquid chromatography, 100 mm × 4.6 mm i.d., 5 μm particle size). Chemicals were detected by their absorbance at 206 nm. Acrylamide levels in asparagine/glyoxal and asparagine/sugar mixtures were determined by comparing chromatograms with those obtained with standard acrylamide solutions. The results are representative of three experiments.

3. Results

3.1. Mechanism of acrylamide formation and prevention of its formation by the DJ-1 family deglycases

Asparagine alone could in principle afford acrylamide by direct decarboxylation and deamination, but the reaction is inefficient with extremely low yields [18]. However, the Maillard adducts between asparagine and reducing sugars or glyoxals furnish acrylamide in the range of 1 mol% in model systems [18].

Proposed mechanism of acrylamide formation in asparagine/methylglyoxal mixtures [5,6]:

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}NH_2 + CHO{-}CO{-}CH_3$ (asparagine + methylglyoxal)

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}NH{-}CHOH{-}CO{-}CH_3$ (condensation into an aminocarbinol)

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}N{=}CH{-}CO{-}CH_3$ (Schiff base formation)

$NH_2{-}CO{-}CH_2{-}CH_2{-}N{=}CH{-}CO{-}CH_3$ (decarboxylation of the Schiff base)

$NH_2{-}CO{-}CH_2{-}CH_2{-}NH_2 + CHO{-}CO{-}CH_3$ (hydrolysis into 3-aminopropionamide and methylglyoxal)

$NH_2{-}CO{-}CH{=}CH_2 + CHO{-}CO{-}CH_3$ (deamination of 3-aminopropionamide into acrylamide)

Spontaneous condensation of the α-NH_2 of asparagine with methylglyoxal leads to formation of an aminocarbinol (Maillard adduct) which dehydrates into a Schiff base. The Schiff base activates decarboxylation of its asparagine moiety, and its subsequent hydrolysis leads to formation of methylglyoxal and 3-aminopropionamide, which deaminates into acrylamide. Similar reactions occur with glyoxal (CHO–CHO) or glucose (not shown).

Proposed mechanism for the prevention of acrylamide formation by the DJ-1 family deglycases:

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}NH_2 + CHO{-}CO{-}CH_3$ (asparagine + methylglyoxal)

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}NH{-}CHOH{-}CO{-}CH_3$ (condensation into aminocarbinol)

$NH_2{-}CO{-}CH_2{-}CH{-}(COOH){-}NH{-}CO{-}CHOH{-}CH_3$ (amide formation by catalyzed H migration)

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NH_2–CO–CH_2–CH–(COOH)–NH_2 + COOH–CHOH–CH_3 (catalyzed amidolysis to asparagine + lactate)

We propose a sequence of reactions similar to those involved in lysine/arginine deglycation by DJ-1 [9]. The spontaneous condensation of asparagine with methylglyoxal leads to formation of an aminocarbinol, as above. The deglycase then converts (by catalyzed H migration) the aminocarbinol into an amide, and subsequently cleaves the amide into asparagine and lactate in a reaction reminiscent of its peptidase activity [19]. Similar reactions would occur with glyoxal (CHO–CHO), leading to formation of asparagine and glycolate (COOH–CH_2OH).

3.2. The PfpI peptidase from P. furiosus functions as a deglycase

PfpI belongs to the PfpI/Hsp31/DJ-1 superfamily, which comprises proteins involved in environmental stress resistance. Several of these proteins were previously characterized as peptidases, notably PfpI [15], DJ-1 [19], and Hsp31 [20], but we recently characterized the deglycase activities of DJ-1 [9] and Hsp31 [10], and we show below that PfpI displays deglycase activity.

We first assessed the peptidase activity of PfpI, and found that it degraded AAF-amc at 85 °C with a specific activity of 18 nmol/min/mg protein (not shown). We then characterized the deglycase activity of PfpI by its ability to prevent glycation of thioredoxin. Thioredoxin was stable at 70 °C (not shown), but was inactivated by MGO at this temperature (Fig. 1A, empty circles). Thioredoxin inactivation by MGO was prevented by the *E. coli* deglycase YhbO (filled circles), and by PfpI (diamonds). Thioredoxin inactivation at 85 °C was also prevented by PfpI (not shown). As shown in Fig. 1B, in which thioredoxin was revealed with anti-AGEs antibodies, PfpI afforded full protection against glycation of thioredoxin by glyoxal. We reported previously that deglycases display an apparent glyoxalase activity (apparent degradation of methylglyoxal into lactate) in the presence of a substrate protein (9–11), because the latter is glycated by MGO and deglycated by the deglycase, resulting in lactate formation. Accordingly, MGO depletion by PfpI at 85 °C was stimulated 10-fold by bovine serum albumin (BSA) (BSA alone was unable to degrade MGO (no shown)) (Fig. 1C). In this experiment, PfpI degraded MGO with a kcat of 1.2 s^{-1}, similar to those of other Maillard deglycases (9–11).

3.3. Deglycases prevent acrylamide formation in asparagine/glyoxal mixtures

Methylglyoxal and glyoxal form quickly from fructose and glucose upon heating, especially in the presence of additives such as the baking agent NH_4HCO_3 [21]. They react 100–350-fold more efficiently with asparagine than fructose or glucose, and are responsible for most of the acrylamide formed in sugar/asparagine mixtures [21]. We incubated asparagine and glyoxal (2–6 mM as indicated in legend to Fig. 2) at 55 °C in the presence of DJ-1, at 70 °C in the presence of YhbO, and at 95 °C in the presence of PfpI, and measured acrylamide formation by analyzing the mixtures on a C18 reverse phase HPLC column or a Hypercarb HILIC column. Acrylamide formation in the glyoxal/asparagine mixture was reduced by 78%, 72%, and 98%, respectively, by YhbO, PfpI, and DJ-1 (Fig. 2A–C) (1.9 μM acrylamide was formed in the control glyoxal/asparagine mixture (Fig. 2A), in accordance with results previously reported [22]). Acrylamide formation in methylglyoxal/asparagine mixtures

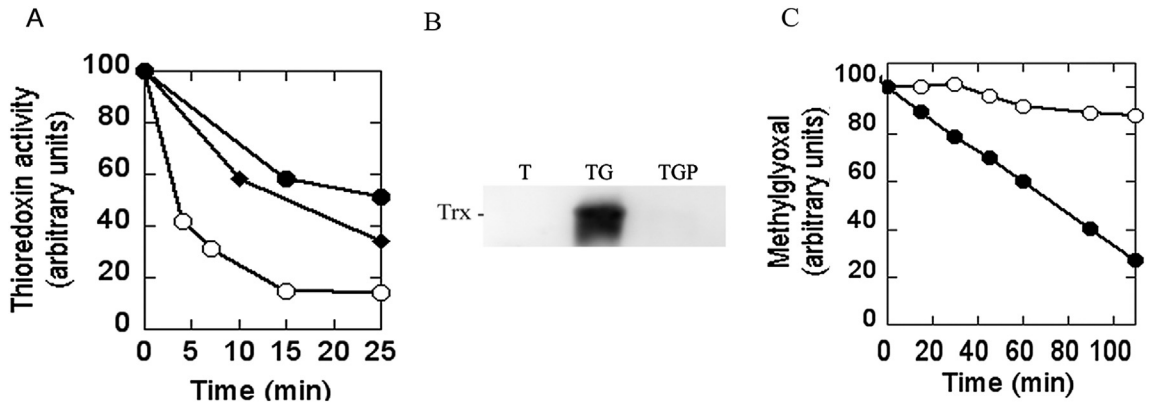

Fig. 1. Deglycase activity of PfpI. A) Thioredoxin was incubated with MGO in the absence (open circles) or presence of YhbO (filled circles) or PfpI (diamonds), and its activity was assayed at different times. B) Glycation of thioredoxin by GO was assayed by immunoblotting with anti-AGEs antibodies: Thioredoxin (T), thioredoxin glycated for 60 min at 70 °C by glyoxal, in the absence (TG) or presence (TGP) of PfpI. C) PfpI was incubated with MGO alone (open circles) or with MGO and BSA (filled circles), and MGO levels were measured.

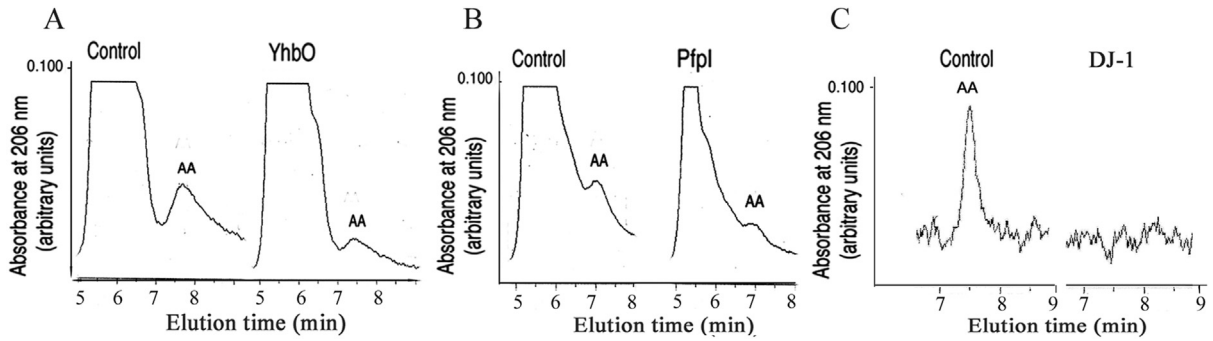

Fig. 2. Prevention of acrylamide formation in asparagine/glyoxal mixtures. A, B) Asparagine and glyoxal (2 mM each) were incubated for 30 min in the absence or presence of YhbO or PfpI as indicated, and mixtures were analyzed on a C18 RP-HPLC column. C) Asparagine and glyoxal (6 mM each) were incubated for 30 min at 55 °C in the absence or presence of DJ-1 as indicated, and mixtures were analyzed on a hydrophilic interaction chromatography Hypercarb column.

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was much lower than in glyoxal/asparagine mixtures, as reported previously [21], and was reduced by 72% and 74%, respectively, by YhbO and PfpI at 70 °C (not shown). DJ-1 and YhbO were inactive at 95 °C, and subsequent experiments were performed with PfpI.

3.4. Deglycases prevent acrylamide formation in asparagine/glucose and asparagine/fructose mixtures

Glucose and fructose by themselves react poorly with asparagine [21], and, as stated above, the glyoxals arising from their decomposition at high temperatures are responsible for most of the acrylamide formed in sugar/asparagine mixtures. For this reason, the DJ-1 family deglycases, whose substrates are glyoxal-containing Maillard adducts, prevent acrylamide formation in asparagine/sugar mixtures. We incubated asparagine and glucose (25 mM each) in the presence of PfpI (5 μM) for 0–130 min at 95 °C, and measured acrylamide formation by analyzing the mixtures on a Hypercarb HILIC column. Acrylamide formation in the glucose/asparagine mixtures was reduced by 55–60% by PfpI (Fig. 3A, B) (38 μM acrylamide was formed in 90 min in the control asparagine/

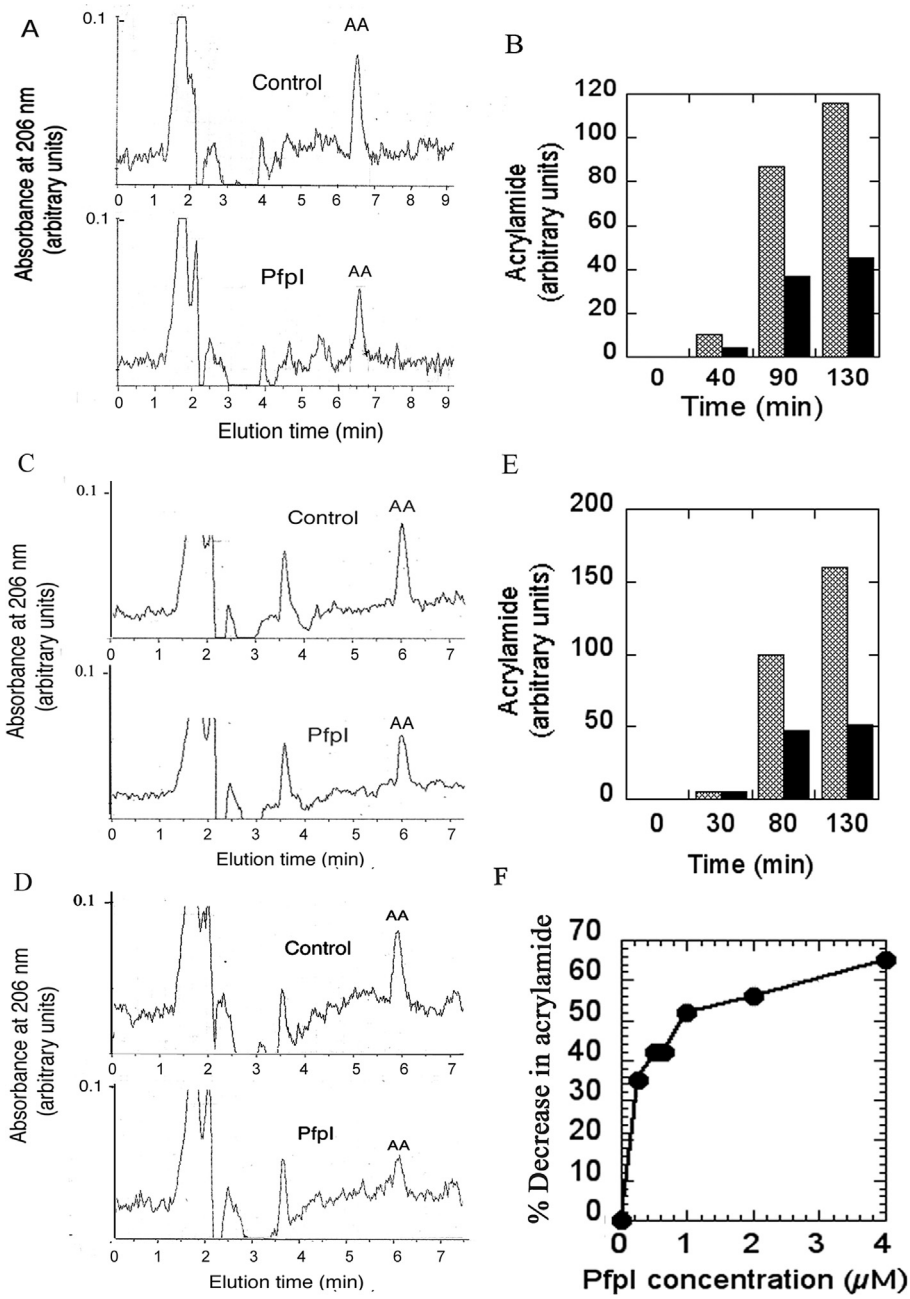

Fig. 3. Prevention of acrylamide formation in asparagine/sugar mixtures. A) Asparagine and glucose were incubated for 90 min at 95 °C in the absence or presence of PfpI as indicated, and the mixture was analyzed on a hydrophilic interaction chromatography Hypercarb column. B) Asparagine and glucose were incubated for different times at 95 °C in the absence (grey) or presence (black) of PfpI, and mixtures were analyzed a Hypercarb column for acrylamide determination. C, D) Asparagine and fructose were incubated for 80 min (C) or 130 min (D) at 95 °C in the absence or presence of PfpI as indicated, and mixtures (35 μl in A and 25 μl in B) were analyzed on a Hypercarb column. E) Asparagine and fructose were incubated for different times at 95 °C in the absence (grey) or presence of PfpI (black), and mixtures were analyzed on a Hypercarb column to assess acrylamide formation. F) Asparagine and fructose were incubated for 130 min at 95 °C in the presence of different concentrations of PfpI, and mixtures were analyzed on a Hypercarb column to assess acrylamide formation.

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glucose solution, in accordance with results previously reported [21]). We then incubated asparagine and fructose (25 mM each) in the presence of PfpI (5 μM) for 0–130 min at 95 °C, and measured acrylamide formation by analyzing mixtures on a Hypercarb column. Acrylamide formation in the fructose/asparagine mixtures was reduced by 55–70% by PfpI (Fig. 3C–E)), and PfpI concentrations of 0.2–2 μM were sufficient to prevent acrylamide formation (Fig. 3F) (62 μM acrylamide was formed in 80 min in the control asparagine/fructose solution (Fig. 3C), in accordance with results previously reported [21]). The higher efficiency of deglycases in asparagine/glyoxal mixture (70–100%) than in asparagine/sugar mixtures (50–70%) can be explained by the fact that, in asparagine/sugar mixtures, they are not likely to degrade Maillard adducts formed between asparagine and sugars, whose contribution to acrylamide formation represents approximately 25% of the whole [22].

A scheme describing acrylamide formation from asparagine, and the role of the Maillard deglycases in its prevention is presented (Fig. 4).

4. Discussion

The Maillard reaction is responsible for the formation of both advanced glycation endproducts (AGEs) [8] and acrylamide [5,6]. DJ-1 family deglycases prevent acrylamide formation, likely by degrading Maillard adducts formed between asparagine and glyoxals in a manner reminiscent of their degradation of Maillard adducts formed between lysine or arginine side chains and glyoxals [9–11]. Their ability to degrade Maillard adducts composed of different amino acids, linked to glyoxals via α- or ε-amino groups, suggests that their specificity is principally directed towards the glyoxal region of these adducts. Whereas DJ-1, Hsp31 and PfpI display peptidase activities [15,19,20], they also display deglycase activities which recruit most of their previously reported functions [9,10] (chaperone, peptidase, and glyoxalase). Since Maillard adducts are the substrates of the DJ-1 family deglycases, we propose renaming them DJ-1 family Maillard deglycases.

Prevention of acrylamide formation represents a novel property of these deglycases, which adds to protein repair [9–11]. This suggests that, despite the number of functions proposed for DJ-1 and its homologs [19], their primary function is likely the degradation of Maillard adducts and prevention of the formation of AGEs and acrylamide [9–11, this work]. Since acrylamide formation occurs at high temperatures (60–200 °C), it would be of interest to increase the thermal stability of PfpI from 100 °C to 120–140 °C, by protein engineering or immobilization on heterofunctional supports [23].

A variety of methods have been proposed to decrease the levels of acrylamide in food, including changing the process parameters, using microorganisms to decrease sugar content, incorporation of asparaginase to decrease asparagine content, addition of amino-acids to compete with asparagine, and introduction of multivalent cations or an acidic pH to inhibit the Maillard reaction, as well as genetic approaches to reduce asparagine and sugar levels in cereals and potato plants [12]. However, several of these methods are expensive, affect the quality parameters of foods, or even cause formation of other harmful products [12]. Moreover, several of these interventions result in only a moderate reduction in acrylamide formation, or require pre-incubation to decrease precursor levels, thereby delaying the production process. Thus, the DJ-1 family deglycases could constitute a viable means to reduce acrylamide formation in food: In contrast with methods which lead to depletion of sugars or asparagine, or which modify the pH or ionic strength, the deglycases are not likely to affect the quality parameters of foods. Moreover, they can be used directly during the thermal process, which avoids a time consuming pre-incubation step. Consequently, many foods whose heating processes occur at around 100 °C, such as baby foods, evaporated milk, dry soups and prune juices could be clients of the deglycase method, and it should be possible to raise its operating temperature by protein engineering or immobilization on heterofunctional supports [23]. Thus, the use of Maillard deglycases to decrease acrylamide levels represents a unique and original method, because it likely acts on Maillard adducts involved in its formation, and avoids depletion of asparagine and sugars, which constitute important nutritive compounds.

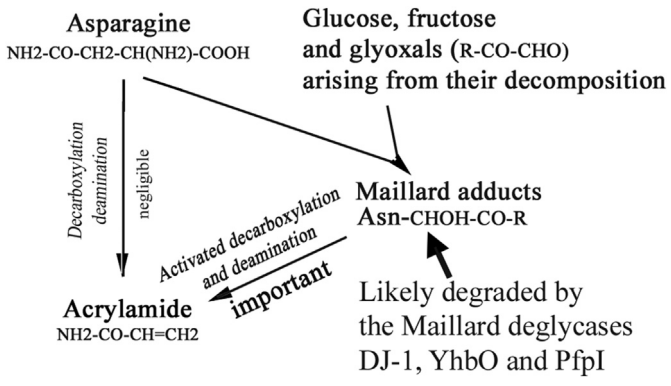

Fig. 4. Scheme describing acrylamide formation from asparagine, and the role of Maillard deglycases in its prevention. Asparagine alone could in principle afford acrylamide by direct decarboxylation and deamination, but the reaction is inefficient with extremely low yields. However, the Maillard adducts between asparagine and reducing sugars or glyoxals furnish acrylamide in the range of 0.1–1 mol% in model systems. The Maillard deglycases of the DJ-1 family prevent acrylamide formation, likely by degrading Maillard adducts responsible for its formation.

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References

[1] E. Tareke, P. Rydberg, P. Karlsson, S. Eriksson, M.J. Törnqvist, Analysis of acrylamide, a carcinogen formed in heated foodstuffs, Agric. Food Chem. 50 (2002) 4998–5006.
[2] P. Erkekoğlu, T. Baydar, Toxicity of acrylamide and evaluation of its exposure in baby foods, Nutr. Res. Rev. 23 (2010) 323–333.
[3] M.W. Poulsen, R.V. Hedegaard, J.M. Andersen, et al., Advanced glycation endproducts in food and their effects on health, Food Chem. Toxicol. 60 (2013) 10–37.
[4] F.J. Tessier, I. Birlouez-Aragon, Health effects of dietary Maillard reaction products: the results of ICARE and other studies, Amino acids 42 (2012) 1119–1131.
[5] R.H. Stadler, I. Blank, R. Varga, et al., Acrylamide from Maillard reaction products, Nature 419 (2002) 449–450.
[6] D.S. Mottram, B.L. Wedzicha, A.T. Dodson, Acrylamide is formed in the Maillard reaction, Nature 419 (2002) 448–449.
[7] L.C. Maillard, Action des acides aminés sur les sucres, C.R. Acad. Sci. Ser. 154 (1912) 66–68.
[8] P.J. Thornalley, Protein and nucleotide damage by glyoxal and methylglyoxal in physiological systems–role in ageing and disease, Drug metabol, Drug Interact. 23 (2008) 125–150.
[9] G. Richarme, M. Mihoub, J. Dairou, et al., Parkinsonism-associated protein DJ-1/Park7 is a major protein deglycase that repairs methylglyoxal- and glyoxal-glycated cysteine, arginine, and lysine residues, J. Biol. Chem. 290 (2015) 1885–1897.

[10] M. Mihoub, A. Abdallah, B. Gontero, J. Dairou, G. Richarme, The DJ-1 superfamily member Hsp31 repairs proteins from glycation by methylglyoxal and glyoxal, Biochem. Biophys. Res. Commun. 463 (2015) 1305–1309.
[11] J. Abdallah, M. Mihoub, V. Gautier, G. Richarme, The DJ-1 superfamily members YhbO and YajL from *Escherichia coli* repair proteins from glycation by methylglyoxal and glyoxal, Biochem., Biophys. Res. Commun. 470 (2016) 282–286.
[12] S. Mariotti, F. Pedreschi, J.A. Carrasco, K. Granby, Patented techniques for acrylamide mitigation in high-temperature processed foods, Recent Pat, Food Nutr. Agric. 3 (2011) 158–171.
[13] J. Abdallah, T. Caldas, F. Kthiri, R. Kern, G. Richarme, YhbO protects cells against multiple stresses, J. Bacteriol. 189 (2007) 9140–9144.
[14] F. Kthiri, H.T. Le, V. Gautier, et al., Protein aggregation in a mutant deficient in yajL, the bacterial homolog of the Parkinsonism-associated protein DJ-1, J. Biol. Chem. 285 (2010) 10328–10336.
[15] S.B. Halio, M.W. Bauer, S. Mukund, M. Adams, R.M. Kelly, Purification and characterization of two functional forms of intracellular protease pfpi from the hyperthermophilic archaeon *Pyrococcus furiosus*, Appl. Environ. Micr 63 (1997) 289–295.
[16] R. Kern, A. Malki, A. Holmgren, G. Richarme, Chaperone properties of Escherichia coli thioredoxin and thioredoxin reductase, Biochem. J. 371 (2003) 965–972.
[17] Y. Zhang, Y. Ren, J. Jiao, D. Li, Y. Zhang, Ultra high-performance liquid chromatography-tandem mass spectrometry for the simultaneous analysis of asparagine, sugars, and acrylamide in Maillard reactions, Anal. Chem. 83 (2011) 3297–3304.
[18] D.R. Lineback, J.R. Coughlin, R.H. Stadler, Acrylamide in foods: a review of the science and future considerations, Annu. Rev. Food Sci. Technol. 3 (2011), 10.1-10.21.
[19] M.A. Wilson, The role of cysteine oxidation in DJ-1 function and dysfunction, Antioxid. Redox Signal 15 (2011) 111–122.
[20] A. Malki, T. Caldas, J. Abdallah, et al., Peptidase activity of the Escherichia coli Hsp31 chaperone, J. Biol. Chem. 280 (2005) 14420–14426.
[21] T.M. Amrein, L. Andres, G.G. Manzardo, R.J. Amado, Investigations on the promoting effect of ammonium hydrogencarbonate on the formation of acrylamide in model systems, Agric. Food Chem. 54 (2006) 10253–10261.
[22] Y. Yuan, G. Zhao, F. Chen, et al., Correlation of methylglyoxal with acrylamide formation in fructose/asparagine Maillard reaction model system, Food Chem. 108 (2008) 885–890.
[23] C. Mateo, J.M. Bolivar, C.A. Godoy, et al., Improvement of enzyme properties with a two-step immobilizaton process on novel heterofunctional supports, Biomacromolecules 11 (2010) 3112–3117.

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