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Amyloid peptides with antimobicrobial and/or microbial agglutination activity

Dongru Chen[1#], Xiangqi Liu[2#], Yucong Chen[3], Huancai Lin[3*]

1 Department of Orthodontics, Hospital of Stomatology, Guanghua School of Stomatology, Sun Yat-sen University. Guangdong Provincial Key Laboratory of Stomatology. Guangzhou, Guangdong, China

2 Department of Oral and Maxillofacial Surgery, Hospital of Stomatology, Guanghua School of Stomatology, Sun Yat-sen University. Guangdong Provincial Key Laboratory of Stomatology. Guangzhou, Guangdong, China

3 Department of Preventive Dentistry, Hospital of Stomatology, Guanghua School of Stomatology, Sun Yat-sen University. Guangdong Provincial Key Laboratory of Stomatology. Guangzhou, Guangdong, China

Adress: Lingyuan Xi 56, Guangzhou, Guangdong, China.

[*]Corresponding author: Huancai Lin; Email: linhc@mail.sysu.edu.cn. Telephone and fax numbers: 020-83744816.

Table S1: Effects and mechanisms of amyloid peptides against bacteria and fungus

First author	Year	Amyloid peptides or sequence	Derives	functions	Specific or broad effects	Mechanism	Cytotoxicity	Monomers or fibers effect
Antimicrobes (antibacteria and antifungus)								
Wei (Wei et al. 2021)	2021	Hen Egg-White Lysozyme	Lysozyme	Antibacteria	Broad (G^+ bacteria, G^- bacteria)	Pores formation	-	Lysozyme fibrils showed significantly enhanced antibacterial activity compared to monomers and oligomers.
Salinas (Salinas et al. 2021)	2021	Uperin 3.5	Skin of Uperoleia mjobergii	Antibacteria	G^+ bacteria (Only G^+ bacteria tested)	Membranes disruption	-	Both monomers and fibers, but the effect of fibrils is weaker than that of monomers
Tu (Tu et al. 2021)	2021	UP(EFVAKLFKFFK), UP-RR(EFVAKLFRFFR), UP-RWR(EFVAKLFRFWR)	PSMα3 (phenol-soluble modulins α3)	Antibacteria	Broad (G^+ and G^- bacteria)	Membranes disruption	Excellent biocompatibility	Monomers were added, but the authors think the antibacterial activity derived from formed nanostructures.
Engelberg (Engelberg & Landau 2020)	2020	$LL\text{-}37_{17\text{-}29}$	LL-37 (37-residue peptide whose N-terminal sequence is LL)	Antibacteria	G^+ bacteria (Only G^+ bacteria tested)	Membranes disruption	-	Only monomers tested

Schnaider (Schnaider et al. 2020)	2020	dhvar2 (KRLFKELLFSLRKY), L7F (KRLFKEFLFSLRKY)	Histatin 5	Antibacteria and antifungus	Broad (G^+ bacteria, G^- bacteria and fungus)	Membrane permeabilization	-	Only monomers tested
Khodaparast (Khodaparast et al. 2018)	2018	Aggregation prone regions (APRs) derived from *Escherichia coli* proteome	*Escherichia coli* proteome	Antibacteria	Broad (G^- bacteria tested)	APRs lead to massive and lethal inclusion body formation containing a large number of proteins, inducing the collapse of protein homeostasis.	Non-cytotoxic	Only monomers tested
Salinas (Salinas et al. 2018)	2018	LFKFFK	PSMα3	Antibacteria	G^+ bacteria (*Micrococcus luteus* and *Staphylococcus hominis*), but nontoxic to S*taphylococcus aureus*	-	-	Only monomers tested
Schnaider (Schnaider et al. 2017)	2017	Diphenylalanine (FF)	-	Antibacteria	Broad (G^+ and G^- bacteria)	Membrane permeabilization and depolarization	Non-cytotoxic	Both monomers and nanotubes?
Bouaziz (Bouaziz et al. 2017)	2017	Lysozyme monomers, fibers, Zn2Al-NO3-LYS, Zn2Al-NO3-AMY	Lysozyme	Antibacteria	G^+ bacteria (Only G^+ bacteria tested)	The enzymatic activity depends on the active site integrity and accessibility, and the enzymatic site is not totally denatured in	-	Both monomers and fibers, but the effect of fibrils is weaker than that of monomers.

						amyloid fibrils, making the fibrils retain a weaker antibacterial effect.		
Bednarska (Bednarska et al. 2016)	2016	C29, C30 , Hit1 and Hit50	Proteome of *Staphylococcus epidermidis*	Antibacteria	Higher antibacterial activity against G^+ bacteria than against G^- bacteria.	Peptides are internalized by bacteria, inducing intracellular aggregation, disturbing cell division.	Non-cytotoxic	Only monomers tested
Park (Park et al. 2016)	2016	α-synuclein (α-Syn)	α-Syn	Antibacteria and antifungus	Broad (G^+ bacteria, G^- bacteria and fungus)	The α-Syn interacts with bacterial membrane and fugal cytoplasmic compounds in microbial cells to inhibit cell growth.	-	Only monomers tested
Wang (Wang et al. 2012)	2012	Human islet amyloid polypeptide (hIAPP)	hIAPP	Antibacteria	Higher antibacterial activity against G^+ bacteria than against G^- bacteria.	-	-	All the three states are effective, but monomer's antimicrobial effect was greater than those of annular protofibrils and fibrils.
Soscia (Soscia et al. 2010)	2010	Abeta (Aβ)	Aβ	Antibacteria and antifungus	Broad (G^+ bacteria, G^- bacteria and fungus)	-	-	Only monomers tested
Pasupuleti (Pasupuleti et al. 2009)	2009	PrP_{23-231} and PrP_{23-144}	Prion protein (PrP)	Antibacteria and antifungi	Broad (G^+ bacteria, G^- bacteria and fungus)	-	-	Only monomers tested

Walsh (Walsh et al. 2014)	2014	$PrP_{106-126}$	PrP	Antibacterial	Broad (G+ bacteria, G- bacteria)	Interacts with the bacterial membrane, results in physical removal of peptide-lipid micelles, causing membrane leakage and cell death	Cytotoxic	Only oligomers exhibit antimicrobial activity
Hirakura (Hirakura et al. 2002); Hari-Dass R (Hari-Dass et al. 2005); Zheng (Zheng et al. 2020)	2002, 2005, 2020	serum amyloid A (SAA)	SAA	Antibacteria	Broad (G^+ bacteria, G^- bacteria)	Ion-channel formation and membrane disruption	-	Only monomers tested
Microbes agglutination								
Chen (Chen et al. 2021)	2021		C123 protein, and other species	Pathogen agglutination	Broad (G^+ bacteria, G^- bacteria and fungus)	Forming amyloid fibrils agglutinating microbes through binding microbial surface carbohydrates	Slight or no cytotoxicity.	Both monomers and fibers, but the effect of fibrils is weaker than that of monomers
Kumar (Kumar et al. 2016)	2016	Aβ	Aβ	Pathogen agglutination	Broad (bacteria and fungus)	Forming amyloid fibrils agglutinating microbes through binding microbial surface carbohydrates	-	All the monomers, oligomers and fibrils are effective. Compared with monomers, oligomers exhibited potentiated, and protofibrils attenuated.
Voth (Voth et al. 2020)	2020	Amyloids including Aβ and tau	Supernatant of type Ⅲ secretion	Pathogen agglutination	Bacteria	Amyloid oligomers bind to the bacteria membrane and cause agglutination	Cytotoxic	Oligomers

			system (T3SS) incompetent *Pseudomonas aeruginosa* infected pulmonary microvascular endothelial cells (PMVECs)					
Chu (Chu et al. 2012)	2012	Human α-defensin 6 (HD6)	HD6	Pathogen agglutination	Bacteria (Only bacteria tested)	Forming amyloid fibrils agglutinating microbes through binding proteinaceous surface appendages	-	Only monomers tested
Microbes agglutination and antimicrobes								
Kurpe (Kurpe et al. 2020)	2020	R23I(RKKRRQRRRGGSar#(A)GVTDFGVFVEI) R23T(RKKRRQRRRGGSar#(A)GVVEGTVVEVT) V10I(VTDFGVFVEI)	Ribosomal S1 protein	Antibacteria	Not clear? Only G- bacteria *Thermus Thermophilus* tested.	Coaggregating S1 protein, suppressing the intensity of protein biosynthesis and thus decreasing total numbers of proteins.	Decrease cell viability when using 20 μg/mL and more, suppress mitotic activity and migration activity.	Only monomers tested.
Hu (Hu et al. 2020)	2020	phase-transited BSA(PTB)	BSA	Antibiofilm (Inhibit microbes adhesion to fibrils)	Broad	-	-	Fibrils

Hu (Hu et al. 2018)	2018	Epigallocatechin gallate-binding lysozyme amyloid fibrils hydrogels; lysozyme amyloid fibrils	Lysozyme	Bacteria agglutination firstly and antibacteria	Broad (G^+ and G^- bacteria)	Inducing the agglomeration of the bacterial cells firstly and then killing bacteria might through membrane disintegration.	Non-cytotoxic	Fibrils
Spitzer (Spitzer et al. 2016)	2016	$A\beta_{x-42}$, $A\beta_{1-40}$	Aβ	$A\beta_{x-42}$: antimicrobes and pathogen agglutination; $A\beta_{1-40}$: antifungus	$A\beta_{x-42}$: broad; $A\beta_{1-40}$: moderate against fungus, non effect to bacteria.	Pathogen agglutination: forming amyloid fibrils agglutinating microbes; Antimicrobes: not stated.	-	Only monomers tested
Torrent (Torrent et al. 2012)	2012	Eosinophil cationic protein (ECP)	ECP	Antimicrobes and pathogen agglutination	Specific to G^- bacteria	Specifically binding to lipopolysaccharide (LPS) and forming amyloid fibrils, and thus disrupting lipopolysaccharide bilayer, exposing the internal cytoplasmatic membrane to the protein action, promoting the membrane disruption.	-	Only monomers tested

Note: Previous well-known antimicrobial peptides or host defense peptides with the amyloid forming ability are not listed in this table. -: Not declared.

Table S2: Effects of amyloid peptides against viruses and their possible mechanisms

Author	**Year**	**Amyloid peptides**	**Derives**	**Monomers or fibers effect**	**Functions**	**Virus species**	**Specific or not**	**Mechanism**	**Cytotoxicity**	**Others**
Palika (Palika et al. 2021)	2021	β-lactoglobulin (BLG)	BLG	β-lactoglobulin amyloid fibril-Fe (BLG AF-Fe) has strong effect, BLG monomer-Fe has moderate effect, while BLG AF or Fe alone have no effect.	Filtration membrane trap eliminate viruses from water by retaining and inactivating virus.	Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), *H1N1*, *enterovirus 71*	Broad	The viruses are elongated perpendicular to the surface, and such elongation was driven by the shrinkage of the membrane on drying.	-	Enveloped, non-enveloped, airborne and waterborne viruses
Michiels (Michiels et al. 2020)	2020	Peptide 12B(WDLIQLIVSDGSDLIQLIVSD)	Basic polymerase 2 (PB2) APR ($_{381}$LIQLIVS$_{387}$)	Monomers and oligomers.	1. Antivirus 2. Not specifically interfering with viral entry into cell.	Influenza A	Specific to influenza A strains, not to influenza B strains.	Peptides 12B induces aggregation and inactivation of PB2, which occurs in the cell.	Dose-dependent red blood cells lysis. No accumulation.	Enveloped virus
Ezzat (Ezzat et al. 2019)	2019	NNFGAIL from IAPP, GNNQQNY from yeat prion protein, Aβ		Monomers tested	Virus agglutination	Respiratory syncytial virus (RSV), herpes simplex virus type (HSV)	-	Viruses bind amyloidogenic peptides in their corona and catalyze amyloid formation via surface-assisted heterogeneous nucleation.	-	RSV catalyzes the amyloid aggregation of NNFGAIL, but not to GNNQQN

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Zabrodskaya (Zabrodskaya et al. 2018) Egorov (Egorov et al. 2013) Matusevich (Matusevich et al. 2015)	2018, 2013, 2015	PB1(6–13), PB1(6-25)	PB1	Monomers tested	Antivirus by inhibiting influenza A virus replication in cell culture	Influenza A	Effective to Influenza A of different subtypes	PB1(6–13) monomer interact with PB1 subunit N-terminal region, causing a change of PB1 from an alpha helix to a beta structure, causing PB1 not binding to acidic polymerase (PA) protein.	Non-cytotoxic	Another mechanism is assumed to be PB1 derived peptides competes with the full-size PB1 protein for binding sites on the PA protein.
Eimer (Eimer et al. 2018)	2018	Aβ	Aβ	Aβ oligomers	Viral agglutination	HSV-1, 6A and 6B	Not specific	Aβ oligomers bind herpesvirus surface glycoproteins.	Non-cytotoxic	Enveloped virus
Bourgade (Bourgade et al. 2015)	2015	$A\beta_{42}$ and $A\beta_{40}$	Aβ	Monomers tested	1. Inhibit HSV-1 replication 2. Inhibit HSV-1 infection cells	HSV-1	-	Aβ interference with HSV-1 replication could involve its insertion into the HSV-1 envelope	Non-cytotoxic	D not prevent non-enveloped human adenovirus replication.
White (White et al. 2014)	2014	$A\beta_{42}$ and $A\beta_{40}$	Aβ	Monomers tested	3. Reduce viral uptake by epithelial cells 4. Antivirus 5. Virus agglutination 6. Increase uptake of virus by	H3N2 and H1N1 influenza A virus	Not specific	-	Non-cytotoxic	Enveloped virus

					neutrophils 7. Reduce viral protein synthesis in monocytes					
Münch (Munch et al. 2007)	2007	$PAP_{248-286}$	Prostatic acidic phosphatase (PAP)	$PAP_{248-286}$ fibrils	Viral agglutination	Human immunodeficiency virus (HIV)	Independently of HIV-1 subtype or group.	PPI-2480 and α-synuclein, but not Aβ, also enhance HIV-1 infection. Fibrils agglutinate HIV and enhance viral infection into cells, independent of the viral Env glycoprotein and hence not restricted to retroviruses.	Non-cytotoxic	Enveloped virus. Monomers lost viral agglutination effect.

Note: Previous well-known antimicrobial peptides or host defense peptides with the amyloid forming ability are not listed in this table. -: Not declared.

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