







Non- and minimalinvasive procedures in paediatric dentistry

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HEALTH AND PREVENTION



□ WHO:

HEALTH = Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity..

- Physical status;
- Subjective factors;
- Progressive/ goals:

- ✓ ***Primary:*** to stop development of illness or make it reversible;
- ✓ ***Secondary:*** early diagnosis and early treatment of the diseases;
- ✓ ***Tertiary: substitution*** rehabilitation and .

- ✓ ***Primary primary prevention!***

From: WHO, Nyárasdy I, Bánóczy J: Preventív Fogászat, Medicina Könyvkiadó Zrt., Budapest 2009, p: 19.

PEDODONTIC PREVENTION - STAGES

➤ *Primary prevention:*

- ❑ Caries;
- ❑ Periodontal diseases;
- ❑ Dental anomalies;
- ❑ Malignant processes – oral cavity

➤ *Primary-primary prevention:*

- ❑ during pregnancy, prenatal period.



**Prevention of
development**

➤ *Secondary prevention:*

- ❑ Early diagnosis: screening;
- ❑ (Early) treatment – orthodontics, precancerous lesions;

➤ *Tertiary prevention:*

- ❑ Treatment, rehabilitation;

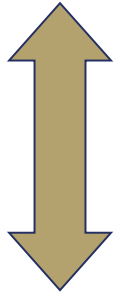
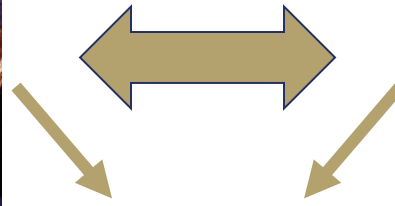
➤ *Primordial prevention: „risk prevention”*

- ❑ To prevent risk factors;

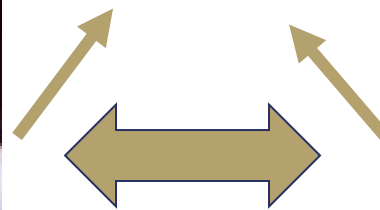
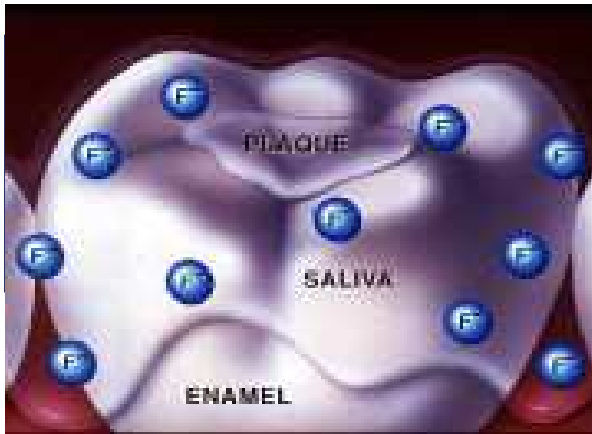


From: Nyárasdy I, Bánóczy J: Preventív Fogászat, Medicina Könyvkiadó Zrt., Budapest 2009, p:20-21.

Komplex prevention in Paediatric Dentistry



CARIES
Dental Anomalies
Periodontal diseases



Prevention/Profilaxix

- *Modern dentistry's goal should be to preserve healthy, natural tooth structure.*
- *The loss of even a part part of a human tooth should be considered „a serious injury” .*

M. Markley, 1951

From: Murdoch-Kinch CA, Mclean ME. Minimally Invasive Dentistry. Journal of American Dental Association, 2003; 134 (1): 87-95.

Minimal (Minimum) Invasive Dentistry (MI)

= is defined as a philosophy of professional care, concerned with the occurrence, early detection and earliest possible cure of disease on a micro level, followed by minimally invasive treatment in order to repair irreversible damages caused by such disease.

New paradigm: an increase in interest in recreating the natural appearance of dentition.

Preservation of the teeth with minimally invasive care is essential in today's dentistry.

The current philosophy of Minimal Invasive Dentistry is to combine aesthetics, prevention, healing, adhesion, and restoration to remove a carious lesion in the least invasive manner.

Paediatric dentistry: Non-invasive



Minimum Invasive

From: Garg S, Goel M, Verma S, et al. Minimal Invasive Dentistry- a comprehensive review. British Journal of Medicine and Medical Research, 2016; 17 (5): 1-9.

Non-invasive therapy modell guidelines Meyer-Lückel and Paris, 2011

- Non-invasive preventive methods used by the patients at home on daily basis;
- professional, risk orientate non-invasive therapy;
- Fissuresealing: conventional and modern techniques;
- Approximal cariesinfiltration;
- Pulp protective cariesescavation;
- Old fillings repaired with adhesive techniques;
- Minimal invasive adhesive filling techniques.

Minimal invasive caries therapy model guidelines Tyrel, 2001

- Early caries diagnosis;
- X-ray for caries depth and progression assessment;
- Individual caries risk evaluation;
- Reduction of pathogenic microorganisms to prevent demineralisation and later cavitation;
- Treatment of active lesions;
- Remineralisation, observation and monitoring of inactive lesions;
- Treatment of existing cavitated caries using minimal invasive techniques for cavity preparation;
- Old fillings repaired with adhesive techniques;
- Regular check-up of the patients.

Proper, caries-free nutrition

POSTRESORPTIVE:

- *after eruption;*
- *albumins, vitamins (A, B, C, D), minerals (Ca, P, Mg), trace elements (F, Mo, V);*
- *quantities and proportions;*



DISZPOZITION PROFILAXIS PRERESORPTIVE:

- *local effects: quantity, quality, frequency.*

EXPOZITION PROFILAXIS

SUGAR



REFINED CARBOHYDRATES

- Sugar is the most fabricated chemical in the world: more than 10 M tonnes/year.
- Hungary: 1419; first inscriptions about sugar during King Sigismund.
- 1476:, the wedding of King Mathias and Queen Beatrix: „ *the rabble was of golden sugar, ornamented with angels and little animals, and a garden of trees and flowers with singing little birds made of sugar...* ”



DISZPOZTION PROFILAXIS

- sugar substituents – polyols/sugar alcohols
 - xylitol, sorbitol, mannitol, maltitol, lactitol, erythritol - cardioprotective effects
 - ↓- decreasing dental plaque quantities and also the plaque-index
 - ↓ - S. mutans: plaque, saliva
 - ↓ - extra-, intracellular polysaccharide matrix secretion
 - ↓ - S. mutans less adherent to the acquired enamel pellicula (AEP)
 - xilitol E967
 - ↓ - decreases S. mutans vertical transmission
- > 10% : „CAVE”



• Sweeteners

DISZPOZTION PROFILAXIS

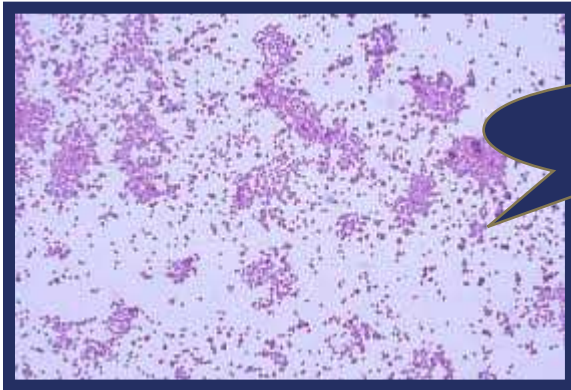
- „sweetener”, Süßstoffe - 1878 SZACHARIN – C. Fahlberg
- aspartame, acesulfame, saccharin, cyclamate,
- agriculture: nutritional supplement; flavor enhancers;
- their sweetening power is many times that of sucrose - a much smaller dosage is required, they can be low-calorie or even calorie-free;
- microorganisms in the mouth do not break down → do not form a substrate;
- synergistic effect: greater sweetening power: aspartame + acesulfame-K: 350x; combinations: with each other and with sugar substitutes;
- long-term effect: UNknown;
- natural origin: stevia (stevia, licorice, jasmine pomace), thaumatin, neurohesperidin.
- Not recommended for under 12s
- **CAVE: FENOLKETONURIA - PKU** → foods or drinks, that contain aspartame must be labelled "**contains a source of phenylalanine**"



CARIESPROTEKTIVE MICROORGANISMS

□ Veillonella ?

- lactic acid break down

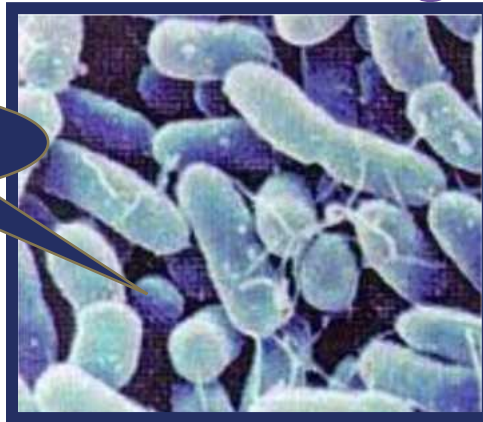


Veillonella

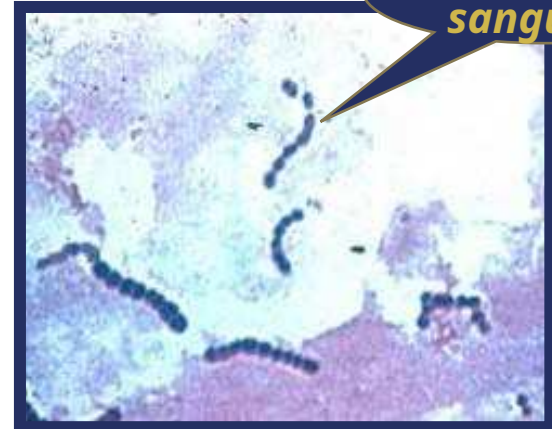
- arginin deaminase secretion ⇨
urea and ammónia ⇨ biofilm: ↑pH

□ *S. salivarius*, *S. sanguinis*

S. salivarius



*S.
sanguinis*



From: Garcia-Godoy & Hicks, 2008

CARIOGEN MICROORGANISMS

- Acid secretion („ACIDOGENIC“)
- Acid resistant („ACIDURIC“)



S. mutans

■ **S. mutans**, *S. sobrinus*

■ *Lactobacillus*, *Actinomyces*

■ *S. mitis*, *S. oralis*, *S. gordonii*,
S. anginosus

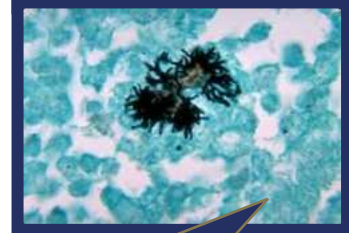
Garcia-Godoy & Hicks, 2008



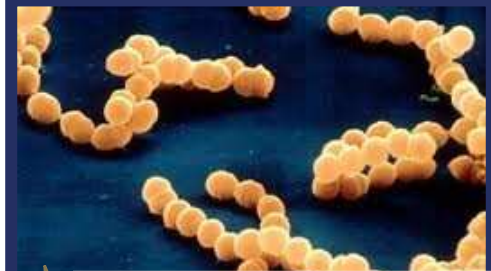
Lactobacillus



A. israelii



A. naeslundii



sobrinus

S. anginosus

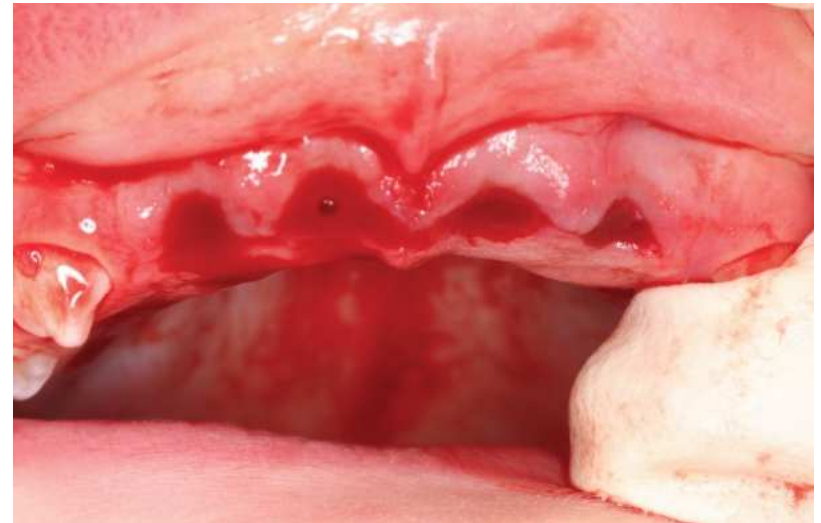
Early S. mutans colonization is the primordial risk factor for ECC

S. mitis



S. gordonii







Interkingdom assemblages in human saliva display group-level surface mobility and disease-promoting emergent functions

Zhi Ren^{a,b,c,d,1}, Hannah Jeckel^{e,f,1}, Aurea Simon-Soro^{a,b,c,2}, Zhenting Xiang^{a,b,c}, Yuan Liu^{a,b,c,g}, Indira M. Cavalcanti^{a,b,c,3}, Jin Xiao^h, Nyi-Nyi Tinⁱ, Anderson Hara^j, Knut Drescher^{k,4}, and Hyun Koo^{a,b,c,d,4}

Edited by Edward DeLong, University of Hawaii at Manoa, Honolulu, HI; received June 10, 2022; accepted August 31, 2022

Fungi and bacteria often engage in complex interactions, such as the formation of multicellular biofilms within the human body. Knowledge about how interkingdom biofilms initiate and coalesce into higher-level communities and which functions the different species carry out during biofilm formation remain limited. We found native-state assemblages of *Candida albicans* (fungi) and *Streptococcus mutans* (bacteria) with highly structured arrangement in saliva from diseased patients with childhood tooth decay. Further analyses revealed that bacterial clusters are attached within a network of fungal yeasts, hyphae, and exopolysaccharides, which bind to surfaces as a preassembled cell group. The interkingdom assemblages exhibit emergent functions, including enhanced surface colonization and growth rate, stronger tolerance to antimicrobials, and improved shear resistance, compared to either species alone. Notably, we discovered that the interkingdom assemblages display a unique form of migratory spatial mobility that enables fast spreading of biofilms across surfaces and causes enhanced, more extensive tooth decay. Using mutants, selective inactivation of species, and selective matrix removal, we demonstrate that the enhanced stress resistance and surface mobility arise from the exopolymeric matrix and require the presence of both species in the assemblage. The mobility is directed by fungal filamentation as hyphae extend and contact the surface, lifting the assemblage with a “forward-leaping motion.” Bacterial cell clusters can “hitchhike” on this mobile unit while continuously growing, to spread across the surface three-dimensionally and merge with other assemblages, promoting community expansion. Together, our results reveal an interkingdom assemblage in human saliva that behaves like a supraorganism, with disease-causing emergent functionalities that cannot be achieved without coassembly.

<https://www.pnas.org/doi/pdf/10.1073/pnas.2209699119>

Fig. 1. Interkingdom microbial assemblages in saliva attach to surfaces as structured cell groups. (A) Using fluorescent staining and confocal microscopy, native fungal–bacterial assemblages are found in saliva of patients with early childhood caries, but not in healthy individuals. (B) *S. mutans* and *C. albicans* were found in high levels in the diseased plaque. Data are presented as median with interquartile range. * $P < 0.05$ by Mann–Whitney U test. (C) Interkingdom assemblages and surface colonization is recapitulated using an in vitro model based on human saliva and hydroxyapatite surfaces, to mimic the tooth enamel. (D) Spatially structured *S. mutans* and *C. albicans* assemblage on the tooth-mimetic surface. (E) Surface-colonized assemblage using different fluorescent markers, as indicated underneath the images. EPS: extracellular α -glucan matrix produced by *S. mutans*. Asterisk: inner core harboring a mix of *C. albicans* and *S. mutans*. Arrowheads: peripheral areas containing mostly *Candida* hypha covered with bacterial-derived EPS. (F) Spatial distribution of *S. mutans* and *C. albicans* within the assemblage. Lines correspond to mean and shaded region to SD of $n = 4$ independent replicates. (G) Schematic diagram describing the spatial arrangement of the two species inside the assemblage, based on the computational image analysis results from F and H. (H) Spatial organization of the fungal and bacterial species relative to the surface. Lines correspond to mean and shaded area to SD of $n = 4$ independent replicates. (I) Confocal images of initial surface colonizers, for WT–WT assemblages and different WT–mutant assemblages (using *C. albicans* $\Delta\Delta\text{efg1}$ or *S. mutans* ΔgtfBC mutants). (Scale bars, 10 μm .)

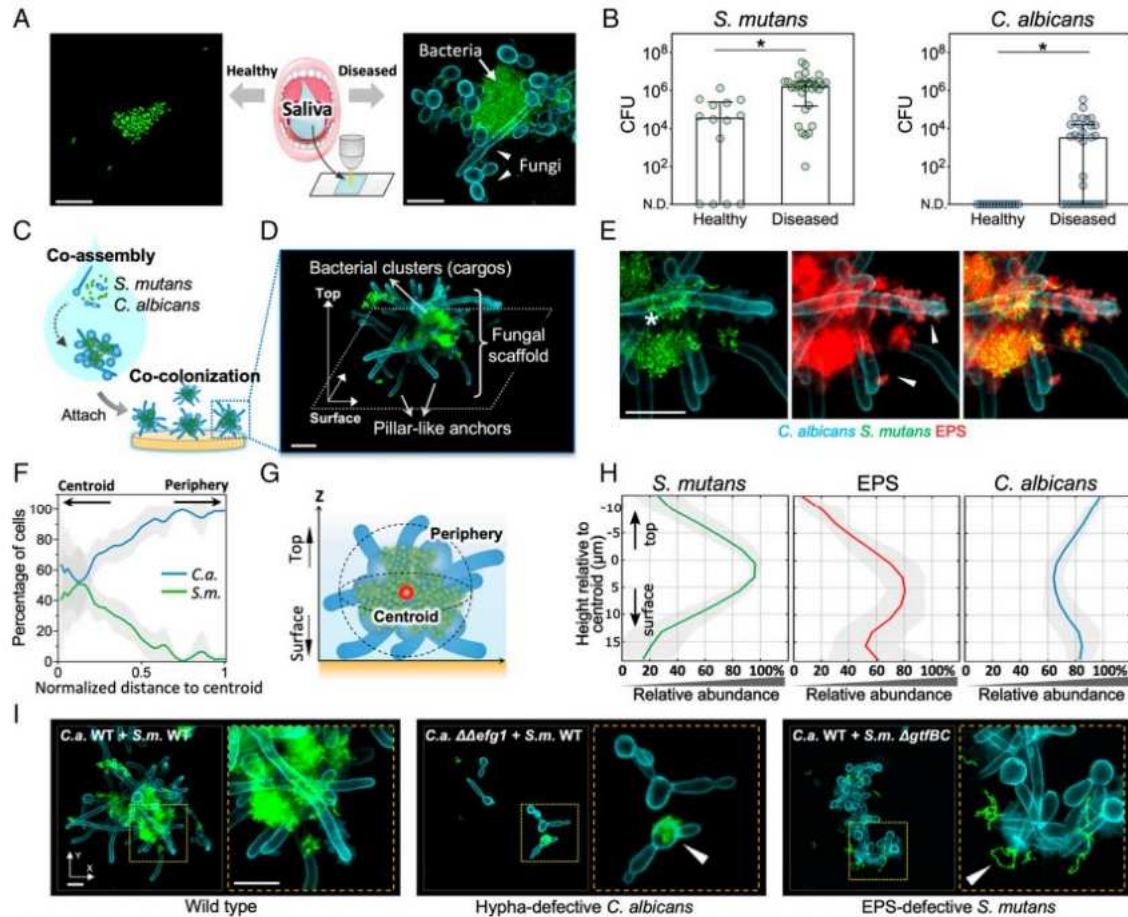
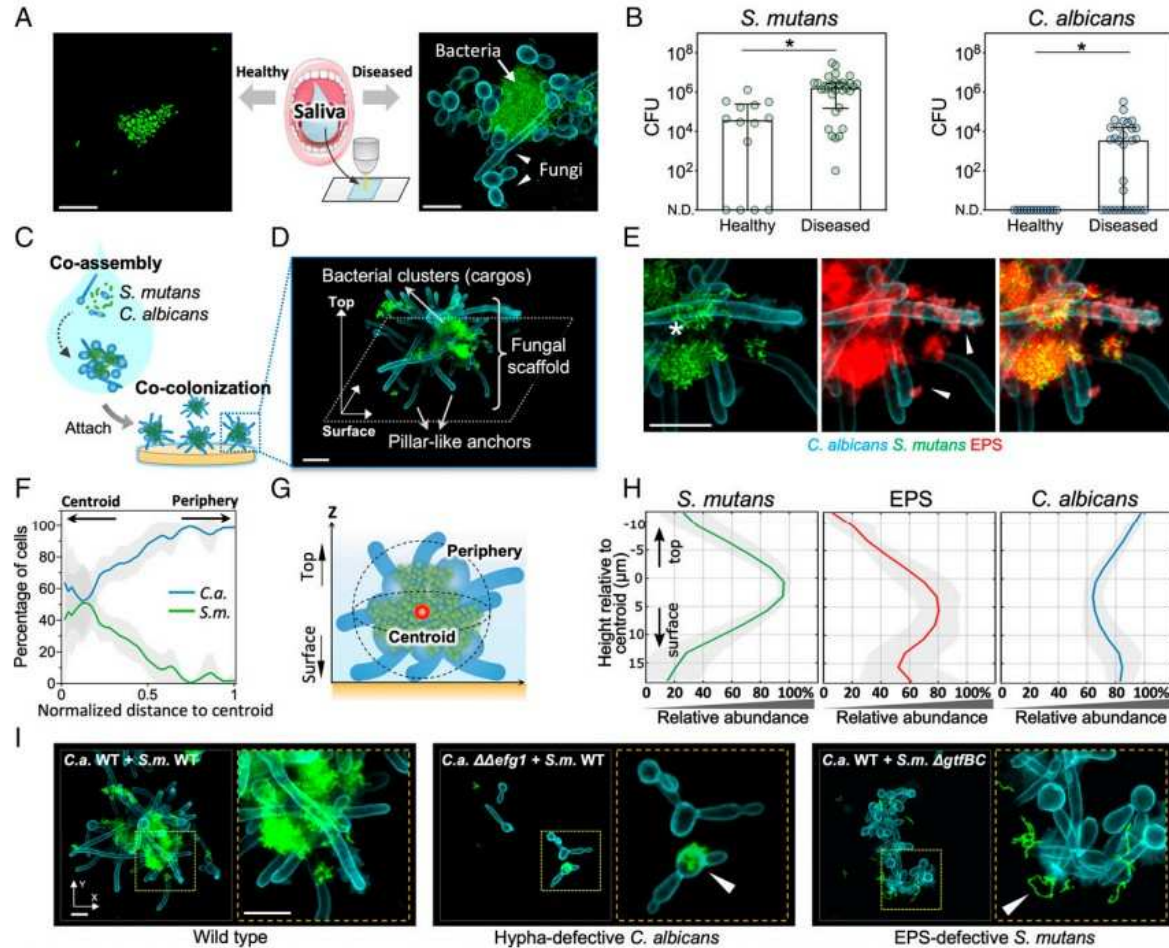


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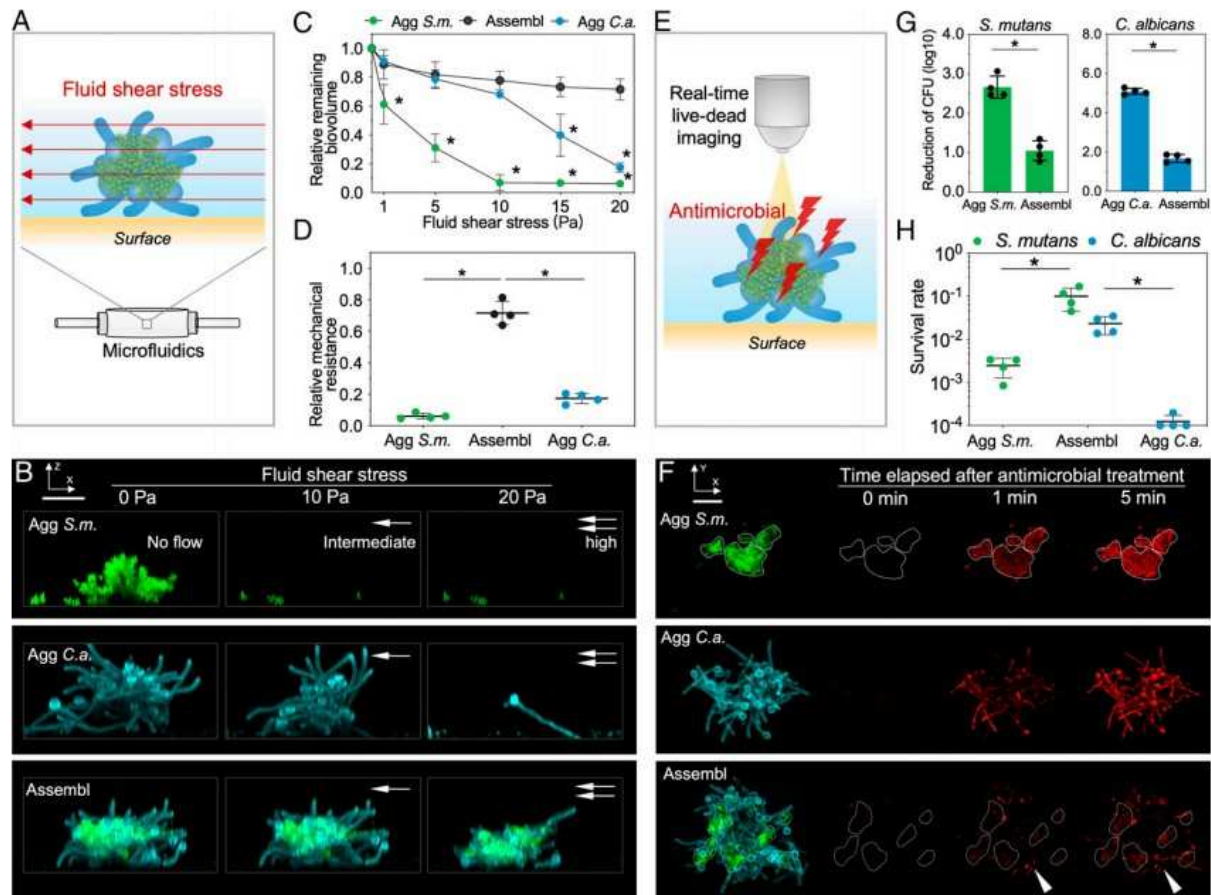


Fig. 2. Enhanced mechanical resistance and antimicrobial tolerance of surface-attached interkingdom assemblage. (A) Schematic diagram of the microfluidic device used to test the mechanical resistance of surface-attached assemblages. (B) Time-lapse confocal imaging of the cellular response to the surface detachment force. *Left image* (0 Pa) illustrates surface-attached biostructures prior to shear stress exposure. *Center and Right images* show the same biostructures after being exposed to intermediate (10 Pa) or high shear stress (20 Pa). Green, *S. mutans*; cyan, *C. albicans*. (Scale bar, 20 μm .) (C) Relative remaining biovolume of the surface-attached biostructures after applying different shear stress. For interkingdom assemblages, the total biovolume of *S. mutans* and *C. albicans* was calculated. (D) Relative mechanical resistance of surface-attached assemblages after exposed to high shear stress (20 Pa), which is defined as the ratio of remaining biovolume on the surface to the original biovolume. (E) Schematic diagram of the experimental setup for the real-time antimicrobial killing assay for surface-attached assemblages, using 100 $\mu\text{g/mL}$ chlorhexidine. (F) Time-lapse confocal image of antimicrobial killing. Dead cells (bacteria and fungi) are visualized using Toto-3 iodide (in red). *Left image* illustrates biostructures prior to antimicrobial exposure. Green, *S. mutans*; cyan, *C. albicans*. Images on the *Right* show the real-time killing profile (red channel only) within the same surface-attached biostructure. White solid lines indicate the bacterial clusters within aggregated *S. mutans* or interkingdom assemblage. Green, *S. mutans*; cyan, *C. albicans*; red, dead cells (*S. mutans* and *C. albicans*). (Scale bar, 20 μm .) (G) Reduction of *S. mutans* and *C. albicans* CFU in different biostructures after 5-min antimicrobial treatment. (H) Survival rate for each species in the biostructures after 5-min treatment. Abbreviation: Agg *C.a.*, aggregated *C. albicans*; Agg *S.m.*, aggregated *S. mutans*; Assembl, interkingdom assemblages. * $P < 0.05$ by one-way analysis of variance with Dunnett's multiple-comparison test (C and D) or by Student's *t* test (G and H).

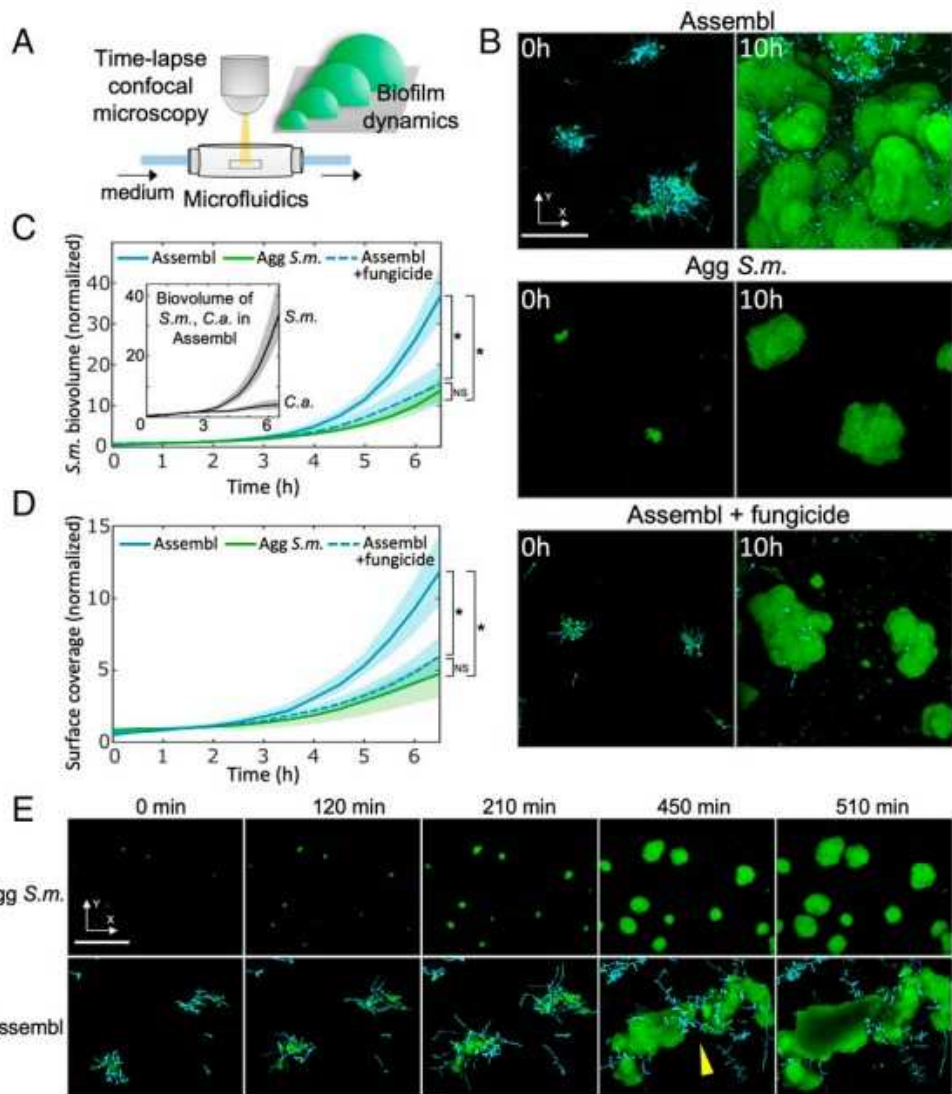


Fig. 3. Biofilm growth dynamics of interkingdom assemblages on tooth-mimetic surface. (A) Schematic diagram of the flow-cell microfluidic culture system coupled with time-lapse confocal microscopy for visualizing biofilm growth. (B) Confocal images of the initial colonizers on the surface at 0 h, and the biofilm structure after 10 h, for interkingdom assemblage (Assemblage), for aggregated *S. mutans* (Agg *S.m.*), and for fungicide-treated assemblage (250 $\mu\text{g}/\text{mL}$ nystatin for 30 min). Green, *S. mutans*; cyan, *C. albicans*. (Scale bar, 100 μm .) (C) Time-resolved biofilm biovolume of *S. mutans* during the biofilm development. (Inset) Time-resolved biovolume of each *S. mutans* and *C. albicans* within the assemblage. Lines correspond to mean, shaded region to SD of $n = 4$ independent replicates. (D) Quantification of the dynamics of biofilm surface spreading. Lines correspond to mean, shaded region to SD of $n = 4$ independent replicates. * $P < 0.05$ by one-way analysis of variance with Tukey's multiple-comparison test ($t = 6.5$ h). (E) Confocal image time series showing merging behavior (yellow arrowhead) of multiple individually developing interkingdom assemblages on the tooth-mimetic surface. Green, *S. mutans*; cyan, *C. albicans*. (Scale bar, 100 μm .)

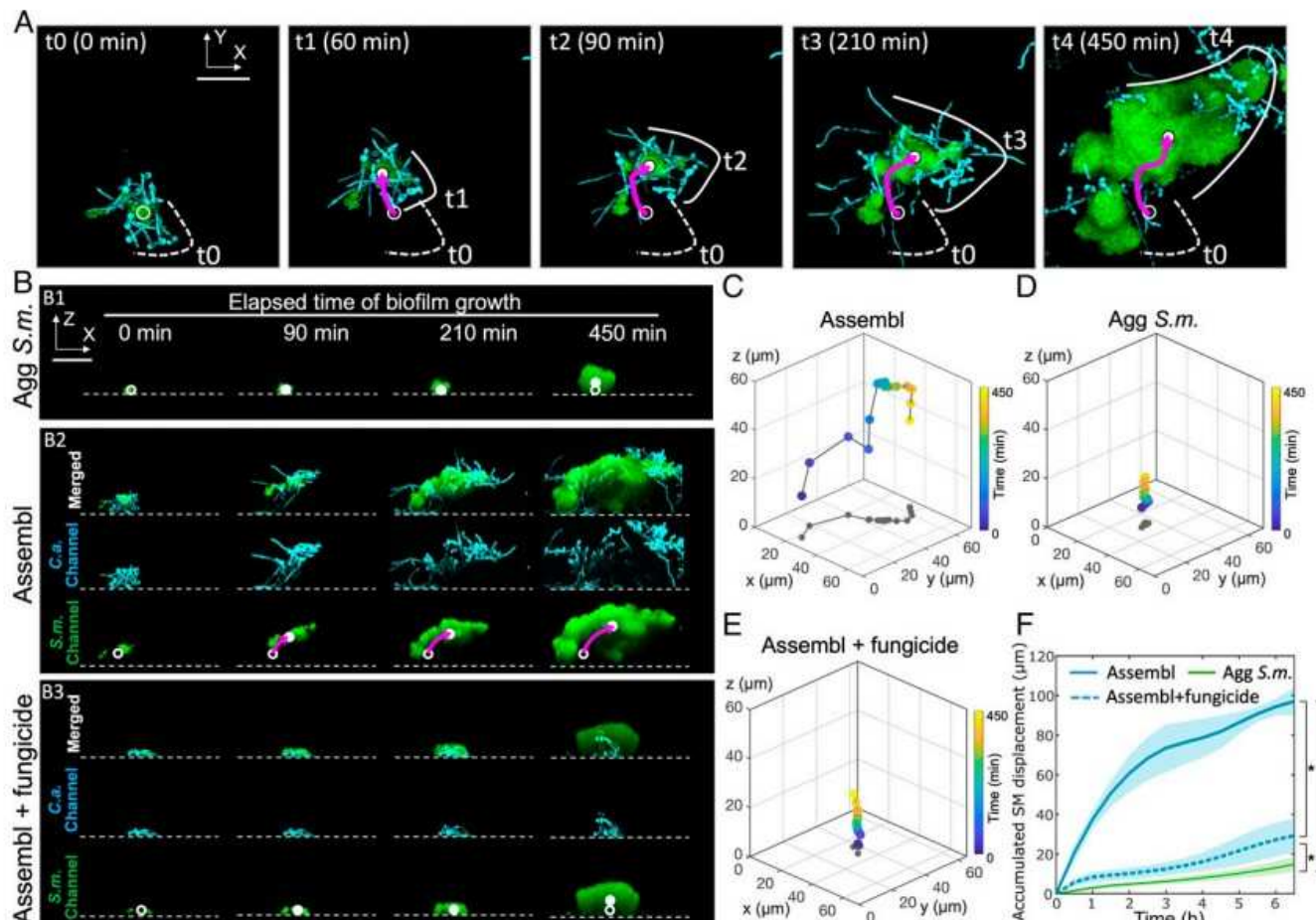


Fig. 4. Dynamics of interkingdom group-level surface mobility and spreading. (A) Surface mobility of the colonized interkingdom assemblage during biofilm initiation. Solid line, leading edge of the interkingdom assemblage at different timepoints; dotted line, leading edge at initial position; solid dot, bacterial biovolume centroid at different timepoints; hollow dot, bacterial biovolume centroid at initial position; purple arrow, path of the bacterial biovolume centroid over time. Green, *S. mutans*; cyan, *C. albicans*. (Scale bar, 50 μm.) (B) Side-views (orthogonal projections) of selected time-frames obtained by time-lapse confocal microscopy for B1) aggregated *S. mutans* (Agg *S.m.*); B2) interkingdom assemblages of *S. mutans* and *C. albicans* (Assembl); B3) fungicide-treated assemblages (Assembl+fungicide). See Movies S1–S3 for B1–B3. Green, *S. mutans*; cyan, *C. albicans*. Solid dot, bacterial biovolume centroid at different timepoints; hollow dot, bacterial biovolume centroid at the initial position. (Scale bar, 50 μm.) (C–E) Four dimensional time-resolved trajectories of the bacterial biovolume centroids within surface-attached assemblage, aggregated *S. mutans*, and fungicide-treated assemblage. Color code: gray, projections of track onto xy-plane; other colors, time elapsed during the biofilm growth (0 to 450 min). (F) Accumulated *S. mutans* displacement (total path length) of the bacterial centroid relative to the initial position. Lines correspond to mean, shaded region to SD of $n = 4$ independent replicates. $*P < 0.05$ by one-way analysis of variance with Tukey's multiple-comparison test ($t = 6.5$ h).

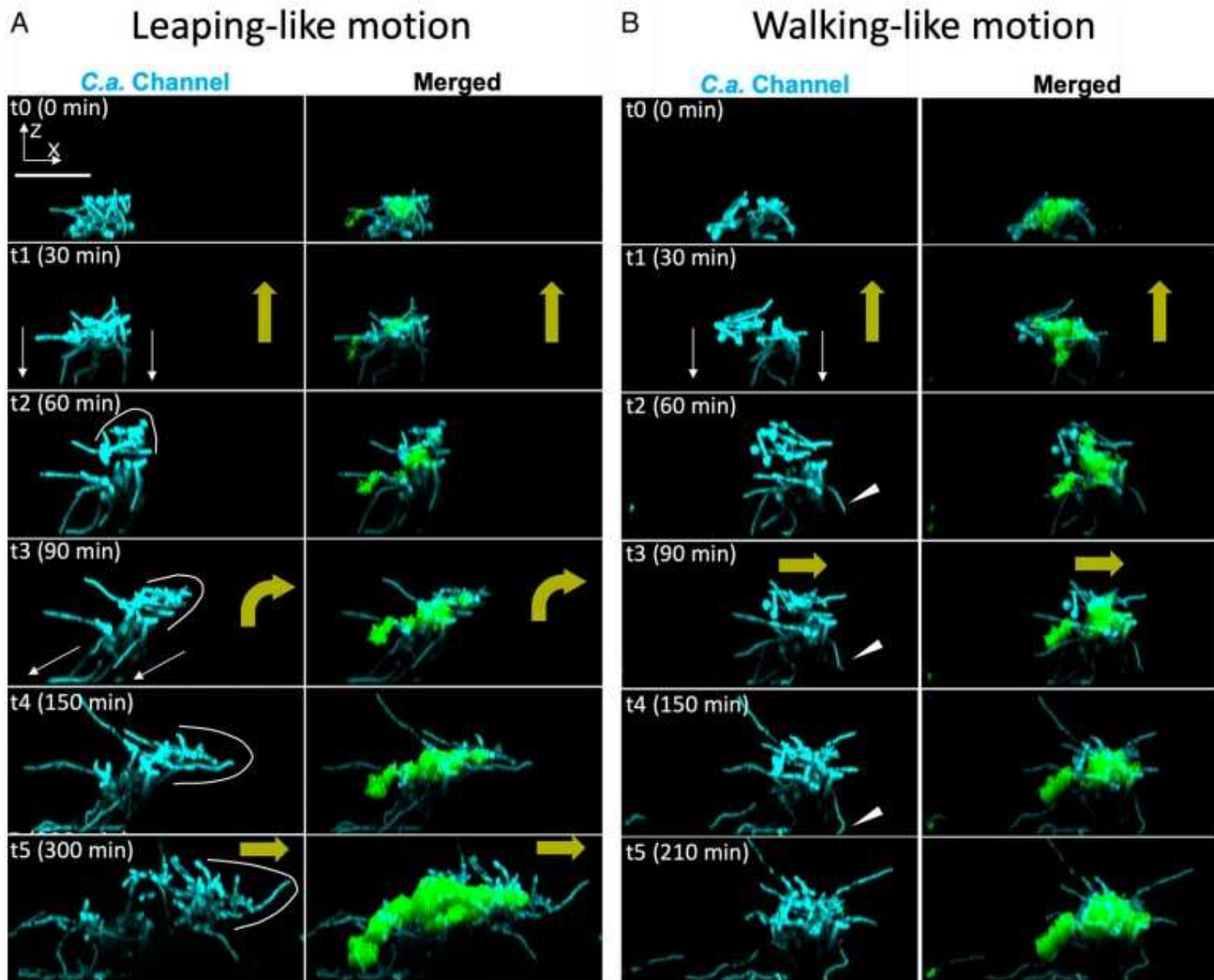


Fig. 5. Assemblage mobility, bacterial hitchhiking, and spatial growth dynamics. Confocal image time series (xz-projections) for growing interkingdom assemblages. (A) Interkingdom assemblage displays a leaping-like motion. White lines indicate the leading edge of the growing assemblage. White thin arrows indicate the direction of fungal hyphal elongation toward the surface. (B) Walking-like motion. White arrowheads, newly formed fungal hyphae establish new anchoring points on the surface. Green, *S. mutans*; cyan, *C. albicans*. Yellow thick arrows, moving directions of the assemblage. Abbreviation: *C.a.*, *C. albicans*. (Scale bar, 50 μ m.)

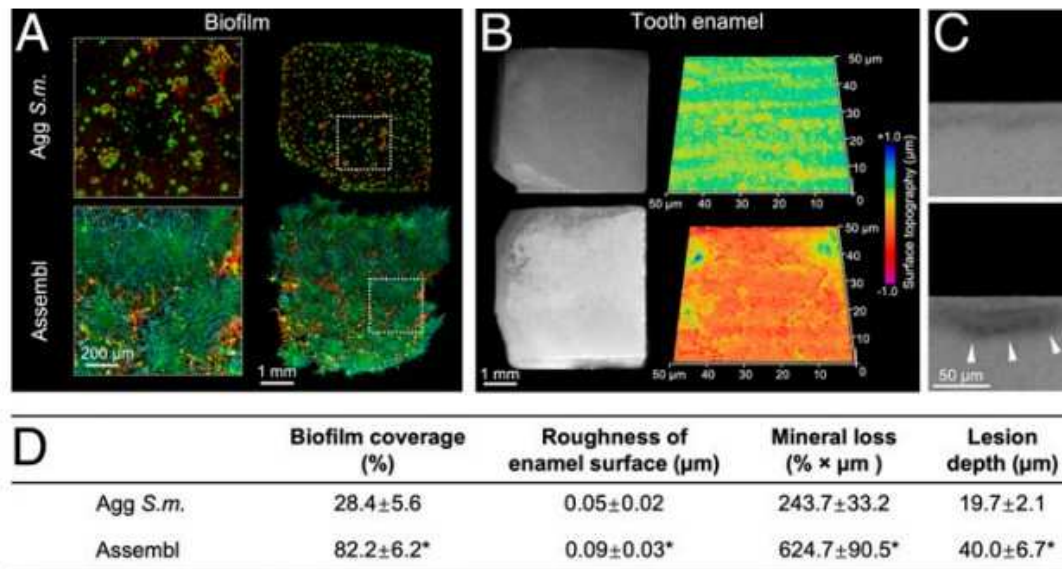


Fig. 6. Interkingdom assemblage-mediated biofilm and tooth decay on human enamel. (A) On human enamel surfaces, biofilms derived from interkingdom assemblages (Assembl) or from aggregated *S. mutans* (Agg *S.m.*) were imaged using confocal microscopy. *Insets* with dotted lines show high-resolution view of magnified areas. Green, *S. mutans*; cyan, *C. albicans*; red, EPS matrix. (B) Multiscale analyses of the tooth-enamel underneath the biofilms. (Left) Macroscopic demineralized lesions (brighter, chalky areas) developed on the enamel surface when biofilms derived from interkingdom assemblages are present; (Right) corresponding surface topography analysis showing microcavities formed on the enamel surface. The surface topography is color-coded to visualize the microcavities. (C) Transverse microradiography of the human enamel surface underneath the biofilm derived from aggregated *S. mutans* (Upper) or biofilms derived from interkingdom assemblages (Lower). White arrowheads indicate area of enamel demineralization caused by biofilms derived from assemblages. (D) Quantitative analysis of biofilm surface coverage on human tooth-enamel, tooth surface topography, and tooth mineral analysis (mean ± SD of $n = 4$ independent replicates. * $P < 0.05$ by Student's t test).

Interkingdom Supraorganism

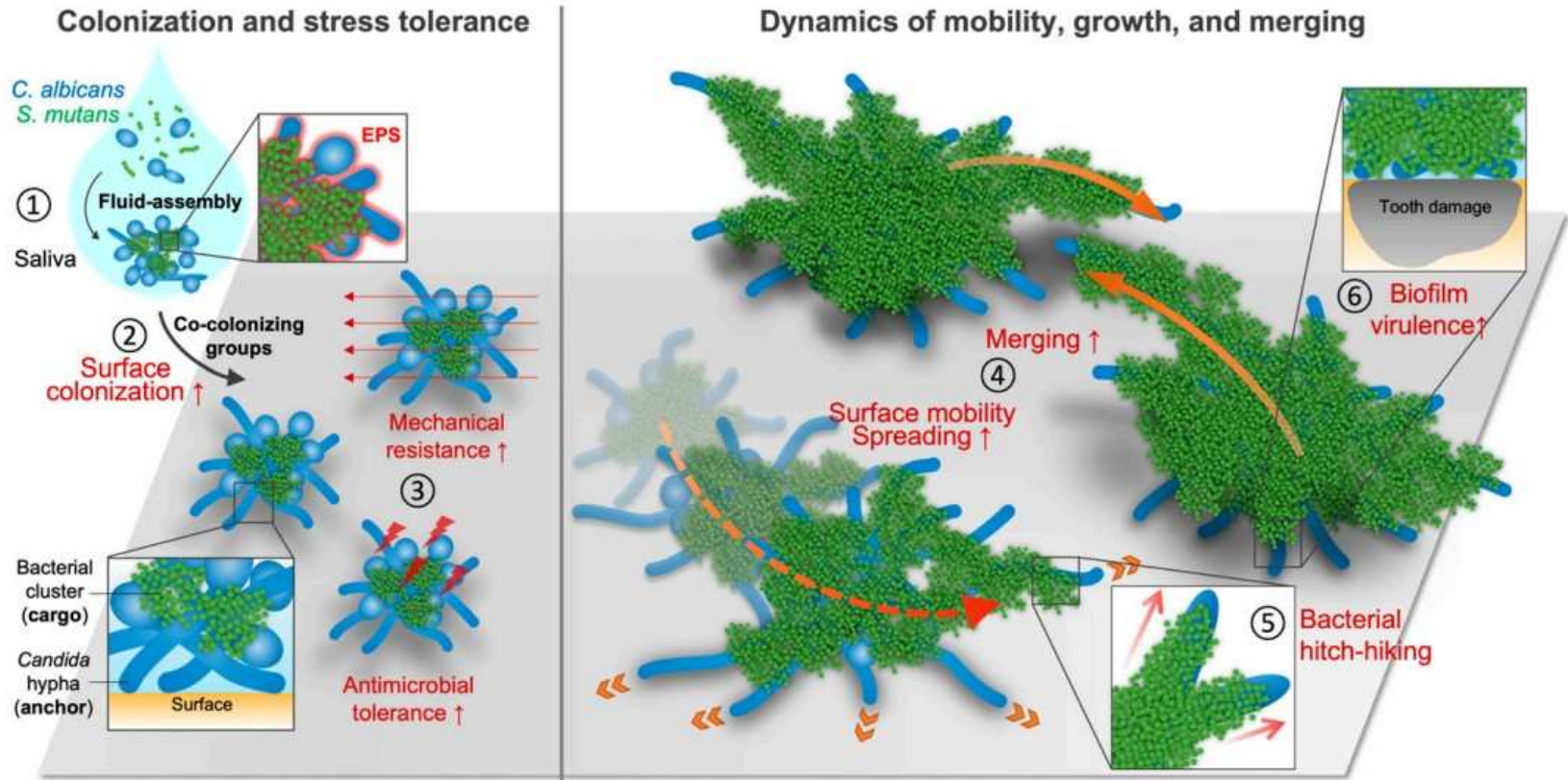
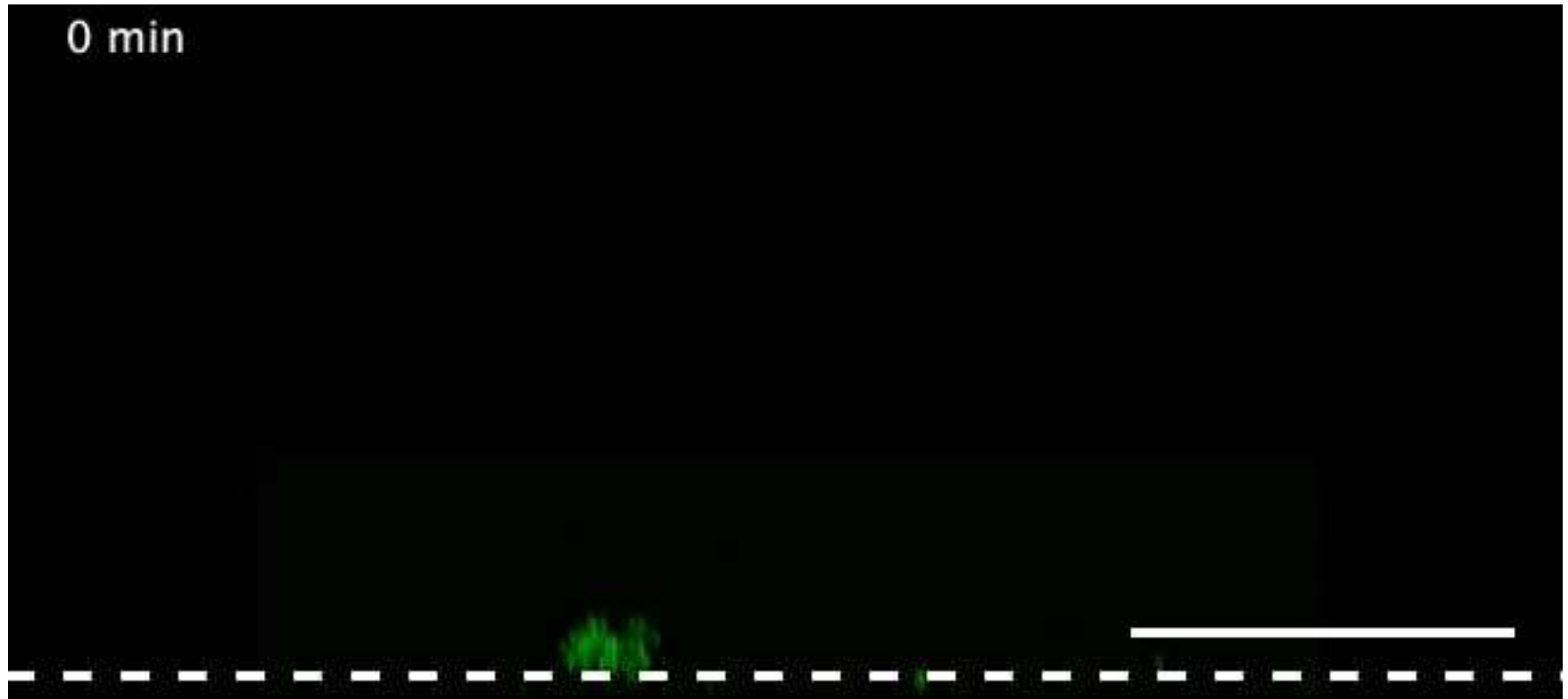


Fig. 7. Interkingdom assemblages in human saliva behave like supraorganisms with new functionalities and disease-promoting activity. 1) *C. albicans* and *S. mutans* coassemble into structured cell groups in human saliva, which are remarkably similar to the native interkingdom aggregates found in intact saliva from diseased patients. 2) Bacteria and fungi collectively colonize the surface as a structured cell group with enhanced binding affinity. 3) The assemblage displays enhanced tolerance to shear stress and antimicrobials. 4) The assemblages behave as single units that grow faster than single-species aggregates, spreading three-dimensionally and merging with each other, resulting in high surface coverage. 5) The interkingdom assemblages display a novel mode of migratory group-level mobility with forward motions and a hitchhiking growth mechanism during biofilm initiation that allows nonmotile bacteria to relocate after surface colonization, which promotes biofilm spatial surface spreading. 6) The interkingdom assemblages cause extensive and severe damage of the tooth-enamel surface.

Aggregated *S. mutans* alone remained in their initial position on the surface during the growth. Scale bar, 50 µm.



<https://www.pnas.org/doi/pdf/10.1073/pnas.2209699119>

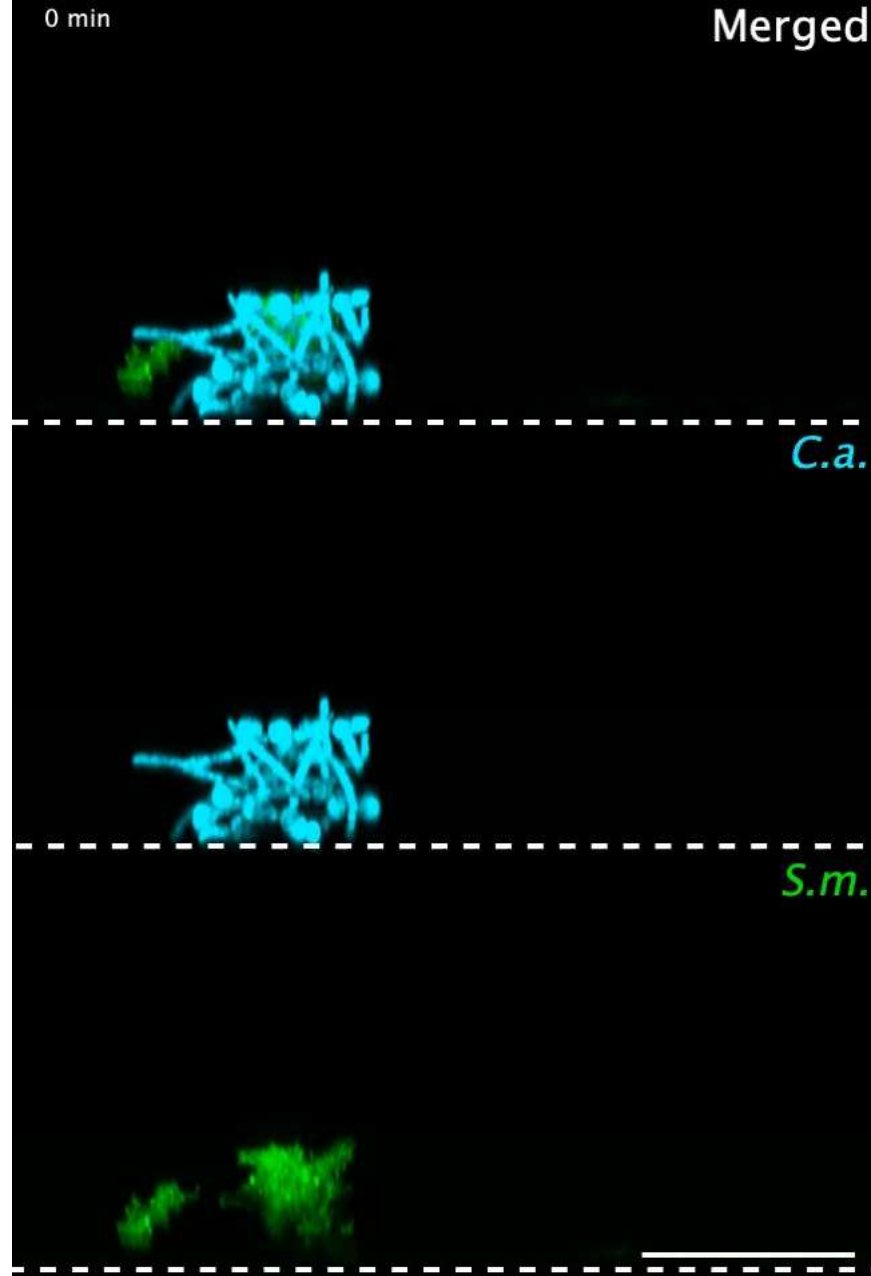


Interkingdom assemblages in human saliva display group-level surface mobility and disease-promoting emergent functions

Zhi Ren^{1,2,3,4,5}, Hannah Jocke^{6,7,8}, Aurea Simon-Soto^{9,10,11}, Zhenling Wang^{12,13}, Yuan Li^{14,15}, Indira M. Chawikan^{16,17,18}, Jin Xie¹⁹, Nyl-Nyi Tin²⁰, Anderson Hara²¹, Knut Drescher^{22,23}, and Hyun Koo^{24,25}

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In the interkingdom assemblage, bacterial clusters were lifted away from the surface and transported laterally while continuously growing along with fungi, thus “hitch-hiking” on the elongating hyphae. A forward-leaping motion was observed during the growth of the interkingdom assemblage. The fungal network rapidly protruded forward to move the biostructure laterally. Green, *S. mutans*; cyan, *C. albicans*. Scale bar, 50 μm .



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Interkingdom assemblages in human saliva display group-level surface mobility and disease-promoting emergent functions

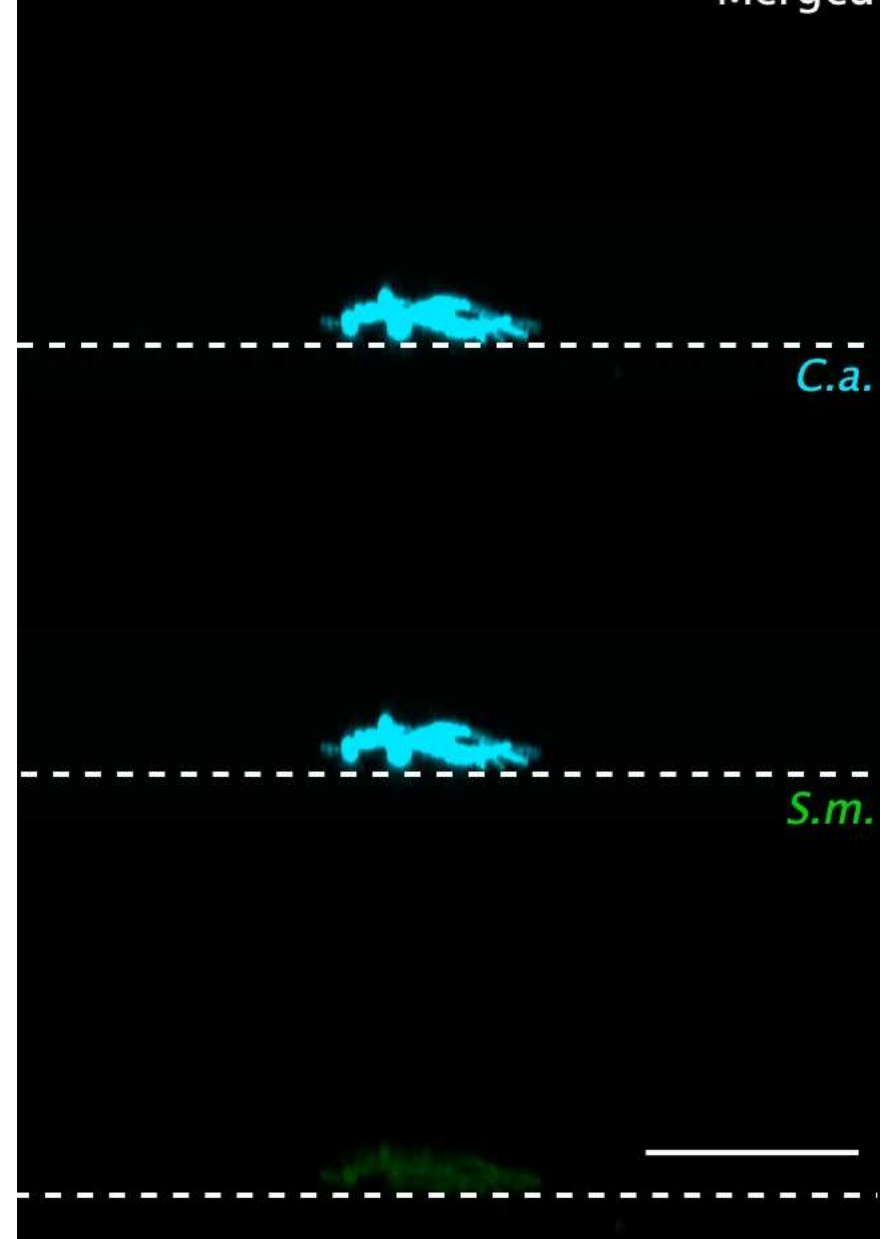
Zhi Ren^{1,2,3,4}, Hannah Jekka^{4,5}, Aurore Simon-Sora^{4,6,7}, Zhering Wang^{4,8,9}, Yuan Liu^{4,10}, Indira M. Cavicani^{4,11,12}, Jin Xiao⁴, Nyl-Nyl Tin⁴, Anderson Hara⁴, Knut Drescher⁴, and Hyun Kook^{4,13,14}

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Specific deactivation of fungal growth in the interkingdom assemblage using nystatin impaired fungal filamentation and resulted in complete loss of “hitch-hiking” mobility. Green, *S. mutans*; cyan, *C. albicans*. Scale bar, 50 μm .

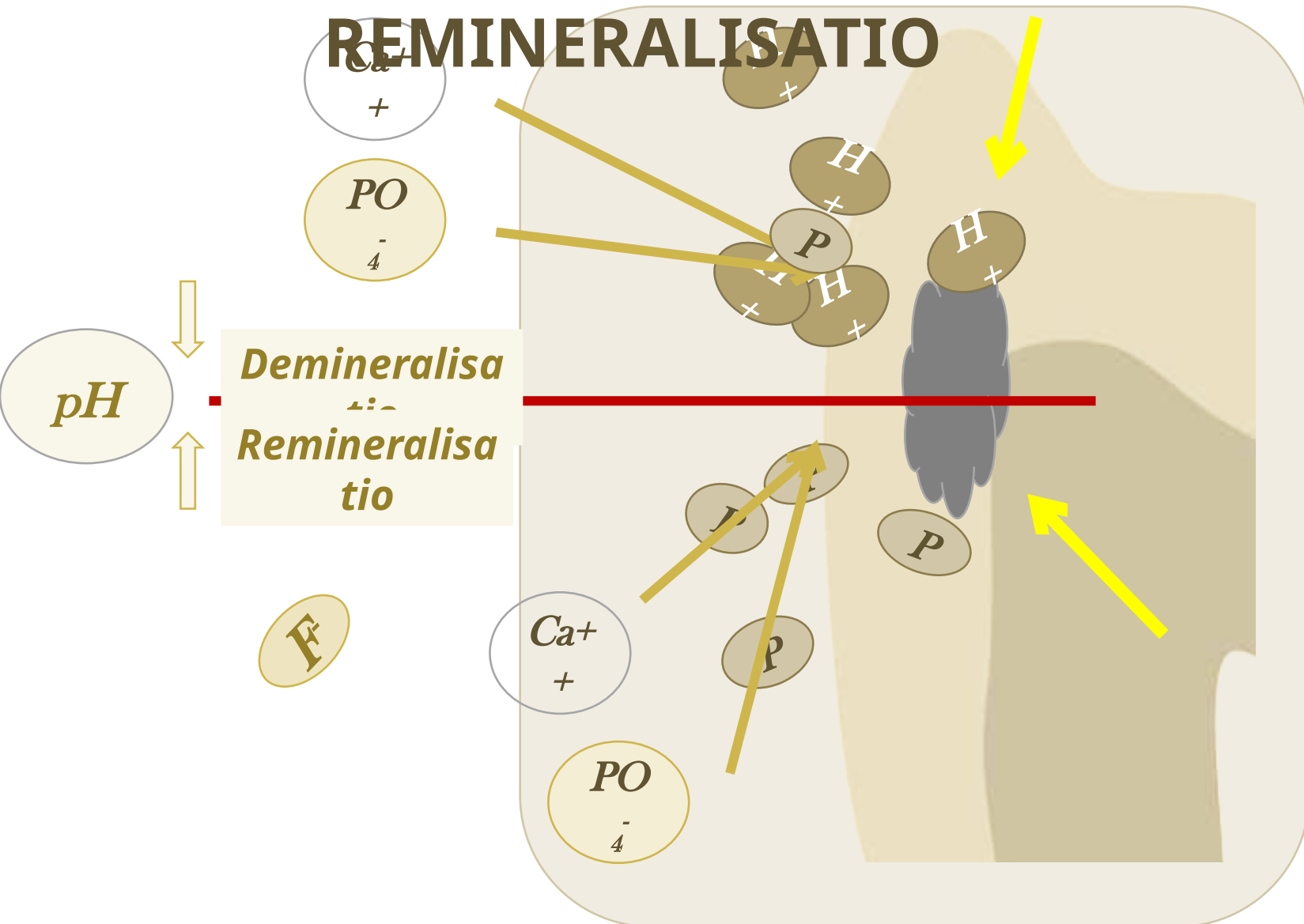
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Merged



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DEMINERALISATIO - REMINERALISATIO



BIOFILM MODULATION

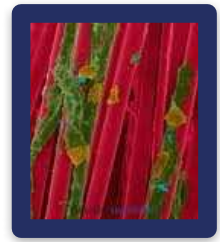


How can we change biofilm and saliva contents for preventive and therapeutic purposes?

- **Amorph calcium-phosphate (ACP) combinations:**
 - **casein-phosphopeptid (CPP);**
 - **stannous fluoride (SnF);**
- **Hydroxyapatite; nano-hydroxiapatite (n-HAp)**
- **Fluoride combinations;**
- **Xylit;**
- **Calcium-sodium-phosphosilicate (NovaMin[®]);**
- **Tricalcium-phosphát (TCP);**



BIOFILM MODULATION



How can we change biofilm and saliva contents for preventive and therapeutic purposes?

- **Amorph calcium phosphate (ACP)**

- ✓ chewing gums, gels
- ✓ casein phosphopeptide (CCP) – lactic albumin which stabilise Ca and P ions in ACP solutions:
 - 25 Ca⁺² □ 15 P □ 5 F⁻
- ✓ CPP+ACP with the help of the biofilm, it adheres to the enamel surface as nanoparticles, then in case of acid production, Ca, P and F ions are released from here
- ✓ GIC – filling materials
- ✓ **CAVE!** Milk protein sensibility (allergy!)



BIOFILM MODULATION

Remineralisation CPP-ACP: Recaldent® GC Tooth Mousse (Plus) INDICATIONS



- During orthodontic treatment
- After bleaching
- After professional dental cleaning
- Prevention and treatment of hypersensitivity
- Treatment of localized enamel hypoplasia
- Xerostomia
- Chemotherapy
- *Frequent consumption of carbonated, acidic soft drinks*

- Children: medium and high caries risk
- For both professional and home use, it should be applied in a thick layer after brushing
- Keep in mouth for at least 3 minutes, then spit out, but do not rinse
- Do not eat or drink for at least 30 minutes after application



✓ **CAVE!** Milk protein allergy! (IgE casein)

BIOFILM MODULATION

Remineralization: Remin Pro® (VOCO) INDICATIONS



- ❑ Hydroxiapatite
- ❑ Fluoride: 1450 ppm
- ❑ Xylit
- During orthodontic treatment
- After bleaching
- After professional dental cleaning
- Prevention and treatment of hypersensitivity
- Treatment of localized enamel hypoplasia

- From the age of 6, for professional use only
- From the age of 12, home use also
- Keep in mouth for at least 3 minutes, then spit out, but do not rinse
- Do not eat or drink for at least 30 minutes after application



- Parabene,
Fluoride

✓ **CAVE! Allergy!**

BIOFILM MODULÁCIÓ



Remineralization: Enamelon® (Premier) INDICATIONS

- ACP - stannous fluoride, Fluorid: 970 ppm
 - Ultramulsion® -
 - Medicinal plant: Spilanthes – stimulates salivary secretion
 - Menthol flavour;
 - RDA 8
 - Gluten-, dye- and SLS-free
- In combination with toothpaste
 - Enamelon® toothpaste:
 - 0,45% stannous fluoride (1150 ppm)
 - RDA 39

SLS – natrium-lauryl-sulphate, foaming detergent



➤ For both professional and home use, no age restrictions!

Spilanthes acmella oleracea „The toothache plant“

- Medicinal plant;
- Stimulates salivary secretion;
- Flavour: ginger, Echinacea;
- Leaves, flower petals;



REGENERATE Enamel Science™

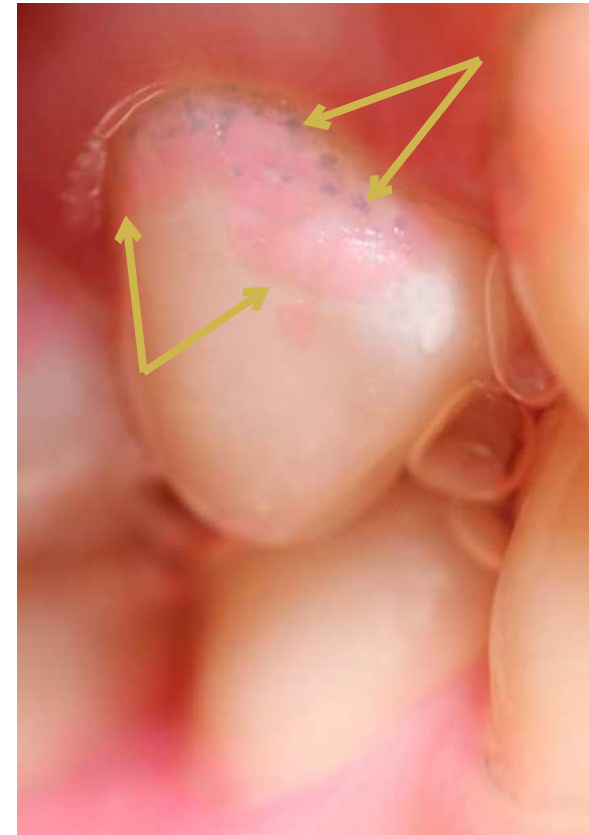
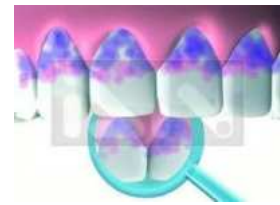
- Advanced Toothpaste
- Boost Serum
- 82%-os repaire
- Hydroxiapatite-formation \approx enamel

- calcium-silicate
- natrium-phosphate

- Toothpaste: 2x daily
- Serum:
 - 3 days/month
 - application: splint
 - 2 layers: serum, activator gel
 - 3 min, mild rinsing
 - during the evening tooth cleaning.



ORAL HYGIENE



- Caries risk assessment
- Plaque pH assessment – colour coded
- Plaque accumulation assessment
- Old/new plaque assessment





GC Tri Plaque ID Gel



- 48h older
- blue, purple



- mature, acidic plaque,
- ↑ caries risk
- light blue

- new plaque,
- pink, red

- For professional use only!
- Do not swallow!
- rinse!
- colour coded – plaque pH
- cleaning
- **CAVE!** Allergy: preservatives!



From: GC und DH lida







10 éves ♀





Tooth brushing techniques



- Technique; KAI – age!
- Toothpaste type and quantity;
- Under parent supervision;
- Motivation;
- Regular dental check-up.

Children's toothpaste – new EU guidelines

FLUORIDE

age-dependent application

Age	Fluoride concentration PPM	Application frequency	Quantity
From the eruption of the first primary tooth until the age of 2 years	500 ppm	daily 2x	pea
	<i>Alternatives</i>		
	1000 ppm	daily 2x	rice
Between the age of 2 and 6 years	1000 ppm	daily 2x	pea

From: <https://www.dzw.de/neue-fluorid-empfehlungen-fuer-kinderzahnpasten>; 2018.06.

CARIESINFILTRÁATION - ICON® (DMG)

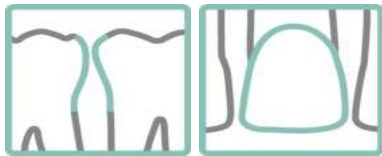
INDIKATIONS



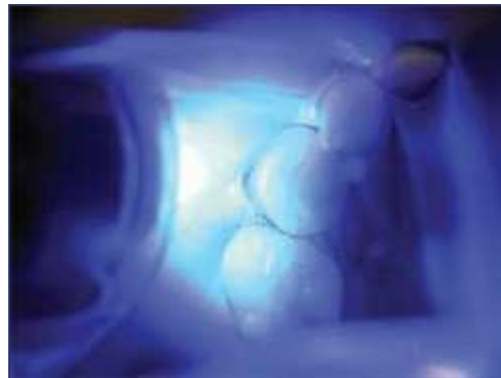
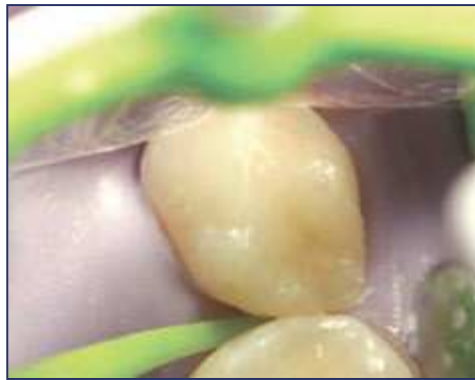
- Caries in the outer 1/3rd of the dentin – D1;
- *Primary molars: clinically not evident, not cavitated approximal caries incipiens (X-ray!)*
- Non- or minimal invasive – no drilling, no cavity preparation!;
- Capillary effect – *difusion of the low viscosity resin*;
- Aesthetics! - *white spot laesio (WSL)*);

CAVE! - 15% HCl – hydrochloric acid as etching agent!

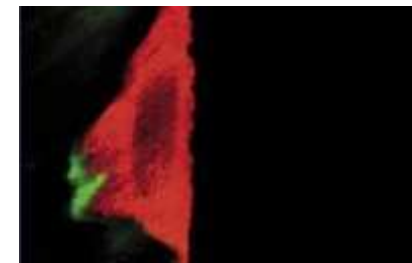
- Not very effective in severe tooth discolorations after tetracycline, fluorosis;
- No x-ray opacity! → documentation!



CARIES INFILTRATION - ICON® (DMG)



PRIMARY TEETH



*FROM: Paris S, Chadtzidakis AJ, Meyer-Lueckel H. Influence of application time on caries infiltration in primary teeth. *Int J Paediatr Dent* 2009; 19 Suppl. 1 S. 9.*

*FROM: Berg JH, Dunn J. Infiltration of Fluid Resin without Cutting into Primary and Permanent Teeth in Children. *Inside Dentistry* 2009; 5(8).*

CONDITIONING: 15% HCL!

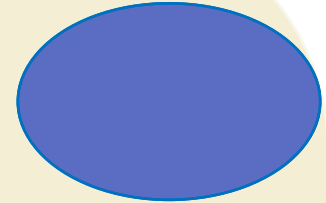
15% HCL

2x2 min

ICON[®]

ICON[®] Dry

30 sec

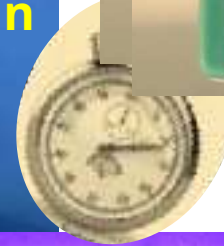
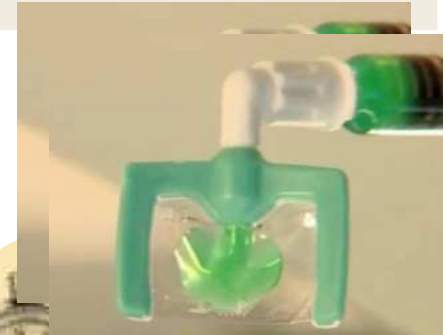


From: Case presentation – SE-FOK Department of Paediatric Dentistry and Orthodontics , I. Simon DMD, DMS

ICON[®] application

ICON[®]

✓ 3 min
✓ 1 min



40 sec



From: Case presentation – SE-FOK Department of Paediatric Dentistry and Orthodontics , I. Simon DMD, DMS

12 years old, ♂

before



after



ICON®

From: Case presentation – SE-FOK Department of Paediatric Dentistry and Orthodontics , I. Simon DMD, DMS

MIH – „Molar-incisor hypomineralisation”



- hypomineralisatio, hypoplasia
- **localisation: first permanent molars and permanent incisors;**
- aetiology?
- retrospective studies;
- complex therapy;
- monitoring



From: Weerheijm et al. Molar-Incisor Hypomineralisation. *Caries Research* 2001; 35:390-391

Pictures: SE FOK Gyermekfogászati és Fogszabályozási Klinika fotótár. Case: Rózsa N. DMD, DMS, MSc, PhD

TERMINOLOGY

- Nonendemic enamel (Jackson, 1961)
- Idiopathic enamel-hypomineralisation (Koch et al., 1987)
- „cheese molars” (van Ameringen and Kreulen, 1995)
- Non fluoride induced hypomineralisation of the first permanent molars, (Leppäniemi et al., 2001)
- ***Molar-incisor-hypomerisation (Weerheim et al., 2001)***
- ***VI. Congress of the EAPD, 2003.***

Forrás: Kellerhof NM, Lussi A. Molar-Incisor-Hypomineralization. Schweizer Monatsschr Zahnmed. Vol 114:3/2004

MOLARIS INCISIVUS HYPOMINERALISATIO MIH - SYNDROME

DIAGNOSIS

AETHIOLOGY

DIFFERENTIAL DIAGNOSIS

CLASSIFICATION

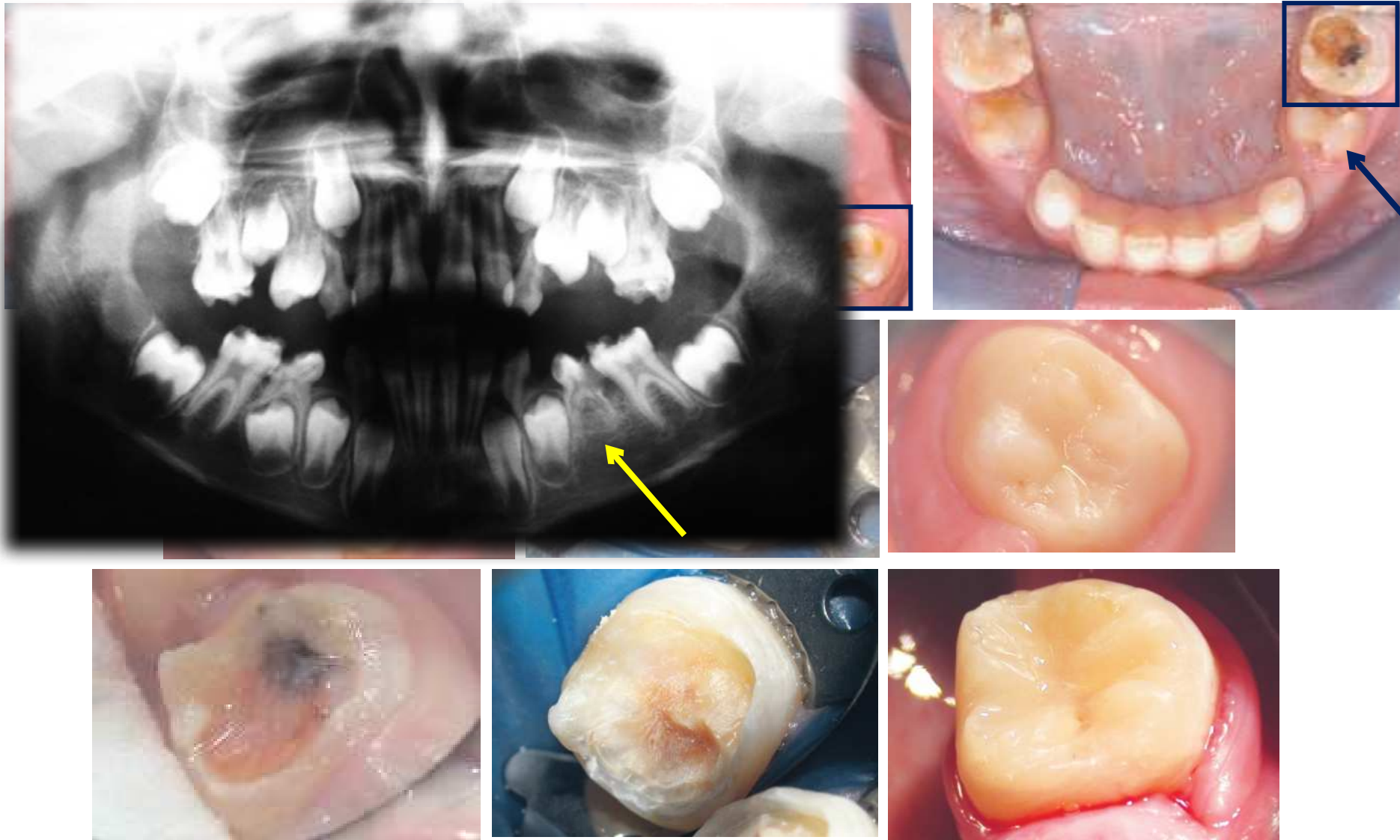
THERAPY



Forrás: Weerheijm et al.

SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga, Mlinkó É., Rózsa N.

MIH – Molar-incisor hypomineralisation



Forrás: Giraki M: **Konservierende Restauration von Zähnen mit Schmelzhypoplasien.** *Oralprophylaxe und Kinderzahnheilkunde* 2006; 28(4):164-168.

A HALL-TECHNIQUE



A HALL-TECHNIKA



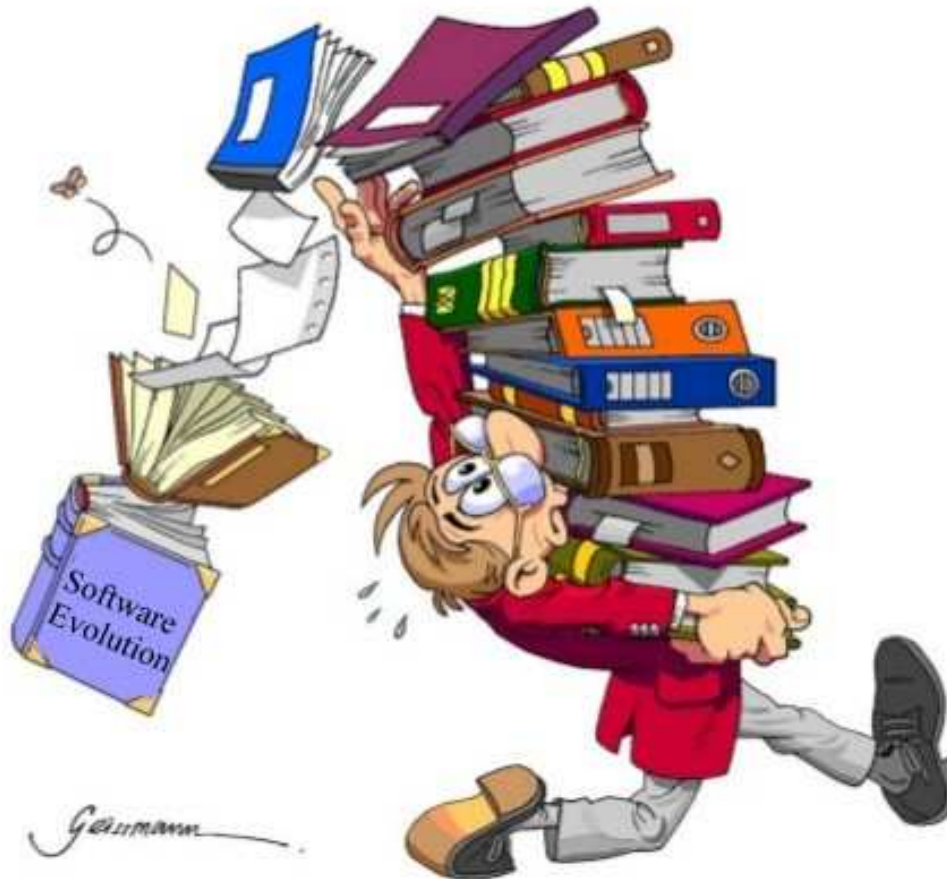
84: Caries media/ approx.



*FROM: Santamaria R,
Splieth Ch. Ist die
Kariesentfernung an den
Milchmolaren notwendig?
ZMK; 2013.*

https://www.zmk-aktuell.de/fachgebiete/kinderzahnheilkunde/story/ist-die-kariesentfernung-an-den-milchmolaren-notwendig_973.html

Take Home Messages



***Thank You for Your kind
Attention!***





Coffee Break

SUGAR



REFINED CARBOHYDRATES

- Sugar is the most fabricated chemical in the world: more than 10 M tonnes/year.
- Hungary: 1419; first inscriptions about sugar during King Sigismund.
- 1476:, the wedding of King Mathias and Queen Beatrix: „ *the rabble was of golden sugar, ornamented with angels and little animals, and a garden of trees and flowers with singing little birds made of sugar...* ”

