the biological crystallization pathway given by a liquid amorphous precursor (PILP), a phase intermediate to the solid ACC precursor phase proposed by Addadi.

Liquid PILP droplets coalesce to patches. Nucleation starts after about 24 hours of PILP formation with tablet crystals with hexagonal shapes and segment assemblages forming within the PILP patches. EBSD analysis shows that c*-axis orientation of the crystals is only slightly out of the plane of growth (by about 5-10 degrees), *a**-axes orientation is almost perpendicular to the plane of growth. For the calcite phase in the given orientation the hexagonal shape is surprising and is produced despite symmetrically not equivalent facets. Thus, crystal shape is also determined by the given space allowance during growth: here a two-dimensional thin film. Assuming that [0001] is the fastest direction of growth, then the largest crystals that form have the [0001]-direction within the plane of the PILP. AFM studies show that these crystals consist of an internal mesostructure consisting of nanoparticles and the hexagonal shape is due to the 2D aggregation of nanoparticles and not to atomic-scale crystal growth of calcite. In addition, we observed calcite crystallite formation from natural ACC in the shell of the modern brachiopod *Megerlia truncata*. We found an unhydrated amorphous domain in direct contact to a solid inclusion that has been incorporated into the shell. The ACC phase has been used as a precursor prior to shell calcite crystallization during shell repair [5]. Under TEM conditions this ACC domain crystallizes to vaterite and calcite [5]. Crystallization starts at the border of a fiber at the organic membrane lining of the fiber [5]. By keeping the electron beam on the amorphous shell region crystals develop in situ on both sides of the fiber, grow towards the center and meet along a growth front in the center of the fiber. The newly formed crystallites highly resemble in morphology, habit and texture the crystallites that form the primary shell layer of *M. truncata* [5].

[1] L. Gover, *Chem. Rev.* **2008** *108*, 4551. [2] S. Raz, S. Weiber, L. Addadi, *Adv. Mater.* **2000**, *12,* 38. [3] Y. Politi, T. Arad, E. Klein, S. Weiner, L. Addadi, *Science* **2004,** *306,* 1161. [4] J. Mahamid, A. Sharir, L. Addadi, S. Weiner, *PNAS* **2008,** *105,* 12748. [5] 6. E. Griesshaber, K. Kelm, S. Sehrbrock, W.W. Schmahl, W. Mader, J. Mutterlose, U. Brand, *Eur. J. Mineral.* **2009,** *21*, 715.

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Crystallization in gels and microgravity: a comparative study

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For crystals grown from solution it is generally accepted that a diffusive mass transport is beneficial for crystal quality as it promotes a slow and regular supply of growth units from the solution to the crystal interface. Microgravity and gels are two methods that have been used to reduce convection in protein crystallization experiments. While microgravity provides a chemically cleaner environment than gels, the associated noise in the microgravity of orbital facilities prevents a convection-free environment in space experiments. In addition, recent studies have shown [1] that gels can even be helpful in reducing impurity effects during protein crystal growth.

In this work we present the results of a comparison of the quality of several model and non-model protein crystals grown in microgravity on the un-manned spacecraft Foton, and in gels on ground. The experiments were performed with the counterdiffusion technique using the Granada Crystallization Facility-2 [2]. To ensure the validity of the comparison critical parameters like protein common batches, thermal history, duration of the experiments and diffraction data collection were carefully controlled. Our results show: 1) that crystals obtained in these experiments are of the highest quality as compared with those obtained in classical techniques, and 2) no statistically clear difference in crystal quality between growth in gels on Earth, and in the excellent microgravity environment on board of Foton, was observed for the studied proteins.

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[1] A.E.S Van Driessche, F. Otalora, J.A. Gavira, G. Sazaki, C*rystal Growth & Design* **2008**, *8,* 3623-3629. [2] L.A. González-Ramírez, J. Carrera, J.A. Gavira, E. Melero, J.M. García-Ruiz, *Crystal Growth & Design* **2008**, *8,* 4324-4329.

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MS

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Structure basis of ligand−receptor interaction in the IL-1 family of cytokines

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Interleukin 1 (IL-1) is a family of cytokines consisting of IL-1 α, IL-1 β, IL-1 receptor antagonist (IL-1Ra), IL-18, IL-33 and IL-1F5−IL-1F10, which play significant roles in inflammation and immune regulation [1]. The IL-1 family members interact with ligand-binding receptor (α chain) and co-receptor (β chain) on the surface of target cells, resulting in the formation of a cytokine/receptor ternary complex necessary and sufficient for triggering intracellular signaling [2]. IL-1 β is a prototype member in the IL-1 family. It first binds the ligand-binding α chain−IL-1 receptor type I (IL-1RI) and then recruits co-receptor β chain−IL-1 receptor accessory protein (IL-1RAcP) to form a signaling IL-1 β/IL-1RI/IL-1RAcP ternary complex. The co-receptor IL-1RAcP is also utilized by other agonists in the IL-1 family including IL-1 α, IL-33, IL-1F6, IL-1F8, and IL-1F9 as a necessary receptor chain in their signaling complexes. The IL-1 β signaling is negatively regulated by native antagonist IL-1Ra that competitively binds IL-1RI and further inhibits the association of IL-1RAcP. The decoy receptor IL-1RII also inhibits IL-1 β activities by binding IL-1 β and IL-1RAcP to form a non-signaling IL-1 β/IL-1RII/IL-1RAcP ternary complex.

We recently determined the structure of non-signaling IL-1 β/IL-1RII/IL-1RAcP complex at a resolution of 3.3 Å. The extracellular regions of IL-1RII and IL-1RAcP have a similar domain organization as that of IL-1RI, consisting of three Ig-like domains (D1−D3). However, structural analysis showed that the linker between D2 and D3 domains in IL-1RAcP is not as flexible as that in IL-1RI and IL-1RII, which may prevent IL-1RAcP from binding ligand in the absence of ligand-binding α chain. IL-1RII inhibits the binding between IL-1 β and IL-1RI by directing blocking sites I and II on IL-1 β for interaction with IL-1RI. The IL-1 β −IL-1RII interaction generates a composite surface contributed by both IL-1 β and IL-1RII to associate with IL-1RAcP. Biochemical analysis demonstrated that preformed IL-1 β/IL-1RI and IL-1 β/IL-1RII complexes bind IL-1RAcP in a similar manner, supporting that the signaling IL-1 β/IL-1RI/IL-1RAcP complex has a similar architecture. It also showed the importance of two loops of IL-1Ra in determining its antagonism. These results together provide a structural basis for assembly and activation of IL-1 β with its receptors and offer a general cytokine/receptor architecture that governs the IL-1 family of cytokines.