

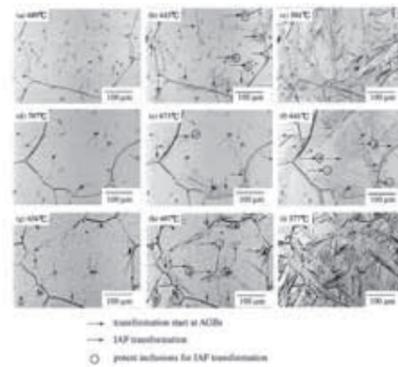
In Situ Observation of the Formation of Intragranular Acicular Ferrite at Non-metallic Inclusions in C-Mn Steel

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(Joining and Welding Research Institute)

Acta Materialia, 58, 1369-1378 (2010)

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This paper presents a direct observation of the morphological development of acicular ferrite in C-Mn steel by using laser scanning confocal microscopy. The effects of austenite grain size, chemical content and characteristic of inclusions on nucleation of acicular ferrite were systematically studied. The microstructure of specimen containing titanium and boron with an austenite grain size larger than 250 μm contains mostly acicular ferrite. The potent inclusions for nucleation are TiO, TiN and MnS. Boron segregation at the austenite grain boundary of ferrite typically suppressed the nucleation at the grain boundary, resulting in improving nucleation sites of acicular ferrite at the inclusions.

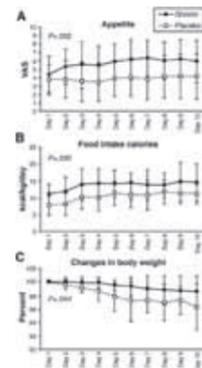


Biology

Effects of Ghrelin Administration After Total Gastrectomy: A Prospective, Randomized, Placebo-Controlled Phase II Study

Adachi, S.; Takiguchi, S.; Okada, K.; Yamamoto, K.; Yamasaki, M.; Miyata, H.; Nakajima, K.; Fujiwara, Y.; Hosoda, H.; Kangawa, K.; Mori, M.; Doki, Y.
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Gastroenterology, 138, 1312-1320 (2010)



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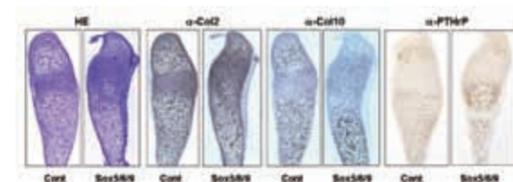
This paper presented that short-term administration of synthetic ghrelin was safe and successfully lessened postoperative BW loss and improved appetite and food intake after total gastrectomy (TG). In this randomized phase II study, 21 patients undergoing TG were assigned to a ghrelin (11) or placebo group (10). They received intravenous infusion of synthetic human ghrelin (3 g/kg) or saline twice daily for 10 days after starting oral food intake following surgery. 20 patients completed the study. Food intake and appetite were significantly higher with ghrelin compared with placebo (average, 13.8 vs 10.4 kcal/kg/day ($P=0.030$) and 5.7 vs 3.9 cm ($P=0.032$)). BW loss was significantly lower in the ghrelin than in the placebo group (1.4% vs. 3.7%; $P=0.044$).

Sox9 family members negatively regulate maturation and calcification of chondrocytes through up-regulation of parathyroid hormone-related protein.

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Molecular Biology of the Cell, 20, 4541-4551 (2009)

▲Reprinted from *Molecular Biology of the Cell*, 20, 2009, 4541-4551, Sox9 family Members Negatively Regulate Maturation and Calcification of Chondrocytes through Up-regulation of Parathyroid Hormone-related Protein, Nishimura, R. et al., with permission from The American Society for Cell Biology.

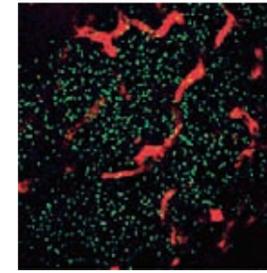


The figure is reprinted from some photographs of the Figure 5 of the original article.

Chondrogenesis is a very unique biological event consisting of several different steps. Sox9 family, Sox5, Sox6 and Sox9, plays essential roles for cartilage development by regulating expression of chondrogenic genes. In this study, we demonstrate

that Sox9 family members promote the early stage of chondrogenesis (Col2 expression) but inhibit the late stage of that (Col10 expression). We also found that Sox9 family members stimulated expression of PTHrP, a negative regulator for maturation of chondrocytes, and that treatment with anti-PTHrP neutralizing antibody rescued the late stage chondrogenesis from inhibition by Sox9 family. These data indicate that Sox9 family conducts chondrogenesis by forming negative-feedback loop with PTHrP.

Biology



Lymphoid chemokines, such as CCL19/CCL21 (CCR7 ligands) and CXCL12, play a vital role in lymphocyte trafficking

▲Reprinted from *The Journal of Immunology*, 182, Bai, Z. et al., CXCL12 Promotes CCR7-Dependent Naive T Cell Trafficking to Lymph Nodes and Peyer's Patches, 1287-1295, 2009. Copyright 2009, The American Association of Immunologists, Inc.

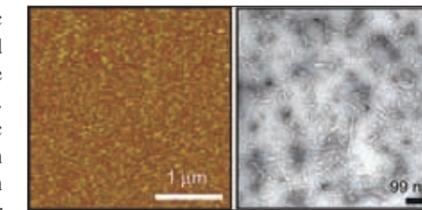
into lymph nodes (LNs) and Peyer's patches (PPs). In this paper, we demonstrate that these chemokines are localized in close proximity around high endothelial venules (HEVs), and that they collaborate to induce T cell migration across HEVs. *In vivo*, CXCL12 acted through its receptor CXCR4, and enhanced CCR7-dependent T cell chemotaxis at suboptimal CCR7-ligand concentrations. In *in vivo* adoptive transfer experiments, CXCL12 promoted T cell trafficking to LNs and PPs in a CCR7 ligand-dependent manner. We propose that CXCL12 provides an efficient mechanism for T cell entry into LNs and PPs by enhancing CCR7-ligand-dependent T cell migration across the HEVs.

CXCL12 Promotes CCR7-dependent Naïve T-cell Trafficking to Lymph Nodes and Peyer's Patches

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The Journal of Immunology, 182, 1287-1295 (2009)

Because of the insolubility and polymeric properties of amyloid fibrils, techniques used conventionally to analyze protein structure and dynamics have often been hampered. Ultrasonication can induce the monomeric solution of amyloidogenic proteins to form amyloid fibrils. However, ultrasonication can break down preformed fibrils into shorter fibrils. Here, combining these 2 opposing effects on β2-microglobulin, a protein responsible for dialysis-related amyloidosis, we present that ultrasonication pulses are useful for preparing monodispersed amyloid fibrils of minimal size with an average molecular weight of ≈1,660,000



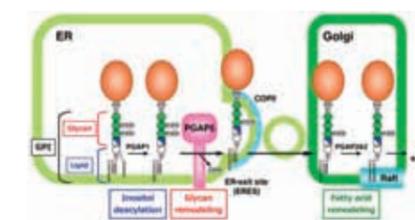
(140-mer). The production of minimal and monodispersed fibrils is achieved by the free energy minimum under competition between fibril production and breakdown. The small homogeneous fibrils will be of use for characterizing the structure and dynamics of amyloid fibrils.

Ultrasonication-Dependent Production And Breakdown Lead to Minimum-Sized Amyloid Fibrils

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Proceedings of the National Academy of Sciences of the United States of America, 106, 11119-11124 (2009)

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Glycosylphosphatidylinositol (GPI) anchoring of proteins is a conserved post-translational modification in eukaryotes, which is essential for embryonic development, immune

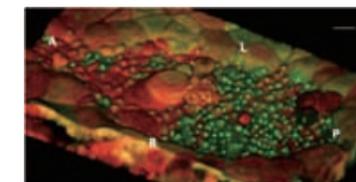
responses and neurogenesis in mammals. GPI-anchored proteins (GPI-APs) are formed in the endoplasmic reticulum (ER) and trafficked to the cell surface via the Golgi apparatus. We identified a gene, *PGAP5* (post-GPI-attachment to proteins 5), being responsible for the mutant cells defective in transport of GPI-APs. *PGAP5* catalyzes the remodeling of glycan moiety of GPI-APs. *PGAP5* activity is prerequisite for the efficient exit of GPI-APs from the ER. Our data demonstrate that GPI glycan acts as an ER-exit signal and further suggest that glycan remodeling mediated by *PGAP5* regulates GPI-AP transport in the early secretory pathway.

GPI Glycan Remodeling by PGAP5 Regulates Transport of GPI-anchored Proteins from the ER to the Golgi

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Cell, 139, 352-365 (2009)



The posterior tilt of node cilia is necessary for the generation of leftward fluid flow 'nodal flow' and future Left-Right asymmetric morphogenesis in mouse development, but its molecular mechanism remained unknown. Here, we show that the basal bodies of node cilia gradually shift from the center to the posterior in node cells by timelapse imaging

system and quantitative analysis. We have also discovered that the posterior shift of the basal body was regulated by posteriorly polarized localization of Dishevelled protein. These findings suggest that Anterior-Posterior polarity is translated into the Left-Right polarity by planar cell polarity pathway in the node.

Planar polarization of node cells determines the rotational axis of node cilia

Hashimoto, M.; Shinohara, K.; Wang, J.; Ikeuchi, S.; Yoshida, S.; Meno, C.; Nonaka, S.; Takada, S.; Hatta, K.; Wynshaw-Boris, A.; Hamada, H.
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Nature Cell Biology, 2, 170-176 (2010)

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