

Join us for a seminar on:



Elucidating desmosome regulation and beyond

Venue: CECAD Building
Seminar room 1.034

Date/Time: 25th Oct (Thu) / 4:00-5:00 pm

Featuring talks by:

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Dr. Matthias Rübsam (Prof. Dr. Carien Niessen Lab)
Department of Dermatology and CECAD Cologne

Combinatorial gene knockdown and overexpression in aPKC mediated junction formation

Desmosomes are adhesive complexes that anchor keratin filaments to the plasma membrane and are critical for epidermal cohesion and differentiation. How desmosomal protein composition and dynamics is controlled to enable upward migration of differentiating keratinocytes while maintaining epidermal integrity is largely unknown. Using (phospho)-proteomics we now identify the polarity protein atypical kinase C (aPKC) as a central regulator of desmosomes. Loss of aPKC alters desmosomal protein expression and revealed a novel aPKC-dependent phosphorylation site on the phemphigus antigen Desmoglein 3 (Dsg3). Preliminary results suggest that aPKC-dependent phosphorylation of Dsg3 might control adhesive strength. Hence we aim to replace endogenous Dsg3 by phosphomutant Dsg3 using siRNA mediated knockdown and overexpression strategies. Thereby we ask whether local activation of aPKC regulates layer dependent desmosome complex formation via Dsg3 phosphorylation and integrates adhesive strength and dynamics with epidermal stratification.



Dr. Michael Hannus
siTOOLS Biotech

Specificity and Robustness from Diversity: Complex siRNA Pools in the study of gene function

RNA interference (RNAi) has numerous advantages as a genetic tool: It is easily applicable to most assay systems, reversible, dose-dependent and cost-efficient. However, off-target effects and variable silencing efficiency, common to all established RNAi reagents, mask specific results and create the need for complicated and costly validation approaches. siPOOLS are complex pools of 30 carefully selected siRNAs. The very low concentration of each siRNA minimizes off-target effects, while the joint on-target activity of all siRNAs increases knock-down robustness and efficiency. By controlling the currently limiting shortcomings of RNAi, siPOOLS represent an ideal tool for elucidating gene function.