Accounts of Chemical Research	38	Quang Luu-Nguyen
ACS Central Science	**MOOK**	Colin McKinlay
ACS Chemical Biology	**MOOK**	Clayton Hardman
ACS Nano	38	Nancy Benner
Advanced Drug Delivery Reviews	N/A	N/A
Angewandte Chemie International Edition	39	Katie Near
Bioconjugate Chemistry	N/A	N/A
Biomacromolecules	41	Nancy Benner
Bioorganic and Medicinal Chemistry	N/A	N/A
Bioorganic and Medicinal Chemistry Letters	N/A	N/A
Chemical Communications	41	Katie Near
Chemical & Engineering News	**MOOK**	Colin McKinlay
Chemical Reviews	42	Jefferson Tyler
Chemical Science	**MOOK**	Jack Sloane
Chemistry, A European Journal	**MOOK**	Clayton Hardman
European Journal of Organic Chemistry	**MOOK**	Jack Sloane
Journal of the American Chemical Society	43	Melanie Huttner (odd)
		Akira Shimizu (even)
JAMA	43	Stephen Ho
Journal of Medicinal Chemistry	N/A	N/A
Journal of Organic Chemistry	N/A	N/A
Molecular Pharmaceutics	N/A	Xiaoyu Zang (Janice)
Natural Product Reports	44	Nancy Benner
Nature	44	Stephen Ho
Nature Chemistry	45	Stephen Ho
Nature Chemical Biology	45	Xiaoyu Zang (Janice)
New England Journal of Medicine	46	Stephen Ho
The New York Times	N/A	N/A
The Onion	N/A	N/A
Organic Letters	46	Quang Luu-Nguyen
Organometallics	N/A	Ryan Quiroz
PNAS	**MOOK**	Colin McKinlay
Science	47	Xiaoyu Zang (Janice)
Science Translational Medicine	48	Jefferson Tyler
Synlett	N/A	N/A
Synthesis	N/A	N/A
Tetrahedron	N/A	N/A
Tetrahedron Letters	N/A	N/A

Next Due Date: Monday, April 17, 2017

Identify articles to abstract in the journals you have been assigned. Try to pick things that the group (or specific subgroups) would like to read or should be aware of. This does not need to be limited to chemistry! If you encounter interesting pieces of media elsewhere (The Economist being a recent example) don't hesitate to let the group know. If you are splitting a journal with another group member, talk with him/her to be sure you are not reviewing redundantly. If you are not able to cover your journal for some reason, get someone to cover it for you—as if it were your group job.

Create an Abstract

Abstract submissions are usually prepared using ChemDraw. The editors of the *Lit Review* strongly encourage the copying of graphical material from PDF files and wish to point out the following. Graphics stored in PDF files are typically of postscript or >300 dpi quality. When an image is copied into a ChemDraw document, a screen snapshot is taken, and the image is captured at the present screen resolution. If the PDF file is being viewed zoomed-in, this typically results in the transfer of a high quality image. If the PDF is being viewed zoomed-out, a low quality image typically results. Text can be copied from a PDF file and pasted as text using the text select or column select tool. Once pasted, this text behaves as if it were input from the keyboard.

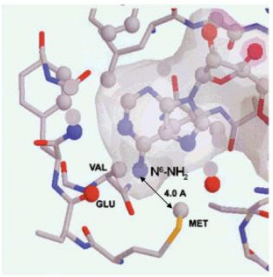
Include a brief textual summary of the article; an example of a completed abstract is shown below. The list of topics and subgroups on the right is useful to highlight which subgroups should pay attention to your abstract and roughly what kind of chemistry the article contains.

Please email the files to knear@stanford.edu. Late abstracts will be included in the Lit Review for the following month**. PCs please send .cdx and macs please send .pdf files.**

Citation: Abeyweera, T.P.; Rotenberg, S.A. *Biochemistry* **2007**, *46*, 2364-2370

Design and Characterization of a Traceable Protein Kinase C-alpha

Protein kinase CR (PKCR) is a critical component of pathways that govern cancer-related phenotypes such as invasion and proliferation. Proteins that serve as immediate substrates for PKCR offer potential targets for anticancer drug design. To identify specific substrates, a mutant of PKCR (M417A) was constructed at the ATP binding site such that it could bind a sterically large ATP analogue derivatized through the N6 amino group of adenosine (1ç-32Pl-N6-phenyl-ATP). Because this analogue could be utilized by the mutant kinase but not by wild-type PKCR (or presumably other protein kinase) to phosphorylate peptide or protein substrates, 32P-labeled products were the direct result of the mutant PKCR.



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Citation: Dictionary.com (search term = "mook")

For those of you who always wanted to know what it meant....
mook **Pronunciation Key** (mk) *n. Slang*
An insignificant or contemptible person.

methods
synthesis

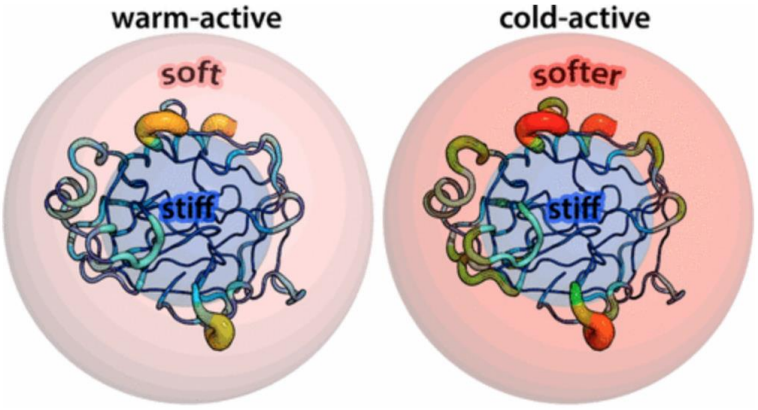
DON'T BE A MOOK!

Lit Review MOOKS include those who:
- fail to submit their abstracts in a timely fashion (or at all), or
- claim there was nothing to abstract in *JACS*, *JOC*, *Org. Lett.*, etc.

Penalties for being a Lit Review MOOK:
- You will get last choice when it's time to pick new journals.

Citation: Brandsdal *et al. Acc. Chem. Research.* **2017**, *50*(2), 199-207

Entropy and Enzyme Catalysis



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synthesis
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review
other

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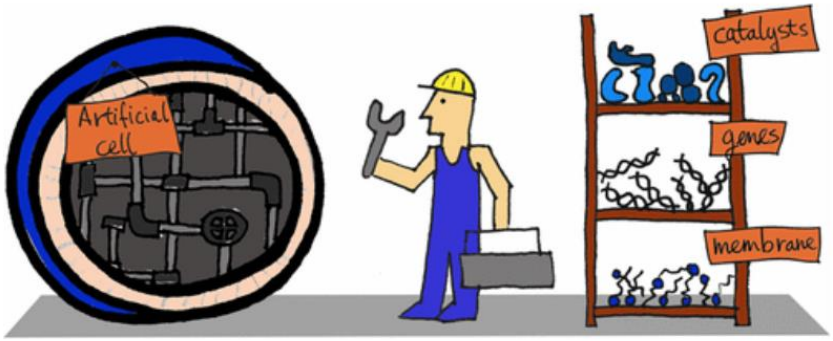
Hybrid
Drug Deliv.
Prostratin

Citation: Bastiaan C. Buddingh, Jan C. M. van Hest. *Acc. Chem. Research.* **2017**. *ASAP*

Artificial Cells: Synthetic Compartments with Life-Like Functionality and Adaptivity



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synthesis
mechanism
review
other

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Hybrid
Drug Deliv.
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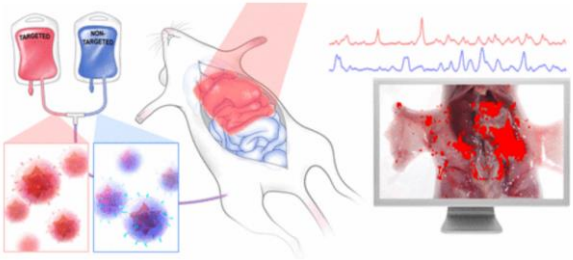
Citation: Oseledchyk, A. et al. *ACS Nano,* **2017**, *11 (2)* 1488-1497

Folate-Targeted Surface-Enhanced Resonance Raman Scattering Nanoprobe Ratiometry for Detection of Microscopic Ovarian Cancer



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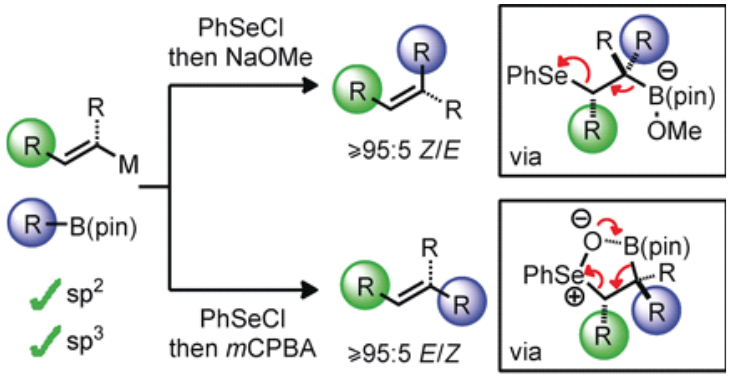
Citation: Armstrong, R. J. *et al*. *Angew. Chem. Int. Ed.* **2017**, *53* (3), 786.

Stereodivergent Olefination of Enantioenriched Boronic Esters



· efficient · stereodivergent · stereospecific

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synthesis
mechanism
review
other

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Drug Deliv.
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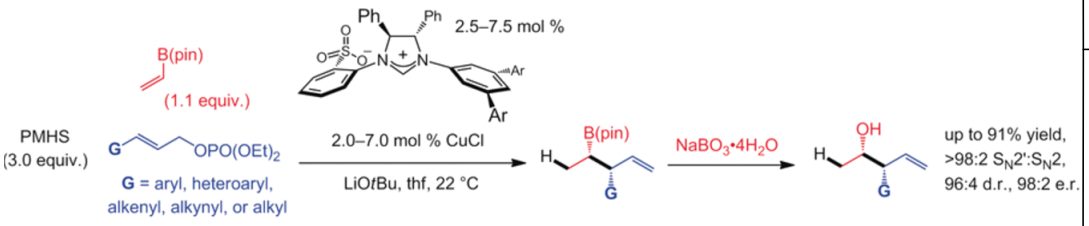
Citation: Lee, J.; Torker, S.; Hoveyda, A. H. *Angew. Chem. Int. Ed.* **2017**, *53* (3), 821.

Versatile Homoallylic Boronates by Chemo-, SN2'-, Diastereo- and Enantioselective Catalytic Sequence of Cu-H Addition to Vinyl-B(pin)/Allylic Substitution



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methods
synthesis
mechanism
review
other

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Drug Deliv.
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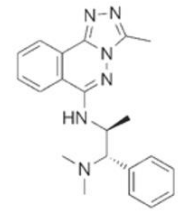
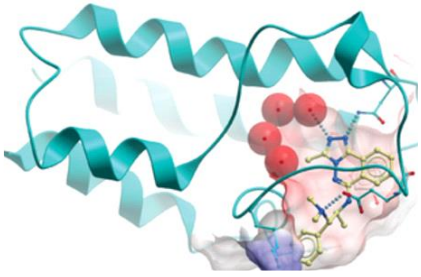
Citation: Moustakim, M.; *et al. Angew. Chem. Int. Ed.* **2017**, *53* (3), 827.



***L*-Moses**

PCAF ITC K_D 126 nM
HTRF K_i 47 nM
GCN5 ITC K_D 600 nM
>4500-fold BRD4 selectivity



Discovery of a PCAF Bromodomain Chemical Probe

Let my PCAF go: The first potent, selective, and cell-active inhibitor of PCAF bromodomains (Brd) is reported. **L-Moses** was shown to disrupt the PCAF-Brd/histone H3.3 interaction in cells using a nanoBRET assay, and a co-crystal structure of **L-Moses** with the homologous Brd PfGCN5 helps explain the high selectivity for PCAF and GCN5 bromodomains.

bioorganic
methods
synthesis
mechanism
review
other

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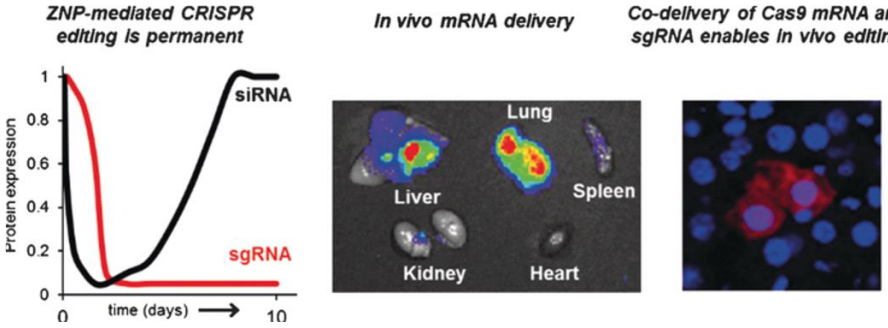
Bryo
DDOs
Hybrid
Drug Deliv.
Prostratin

Citation: Miller, J. B.; *et al. Angew. Chem. Int. Ed.* **2017**, *53* (4), 1059.

Non-Viral CRISPR/Cas Gene Editing In Vitro and In Vivo Enabled by Synthetic Nanoparticle Co-Delivery of Cas9 mRNA and sgRNA



Special delivery: The synthesis and development of zwitterionic amino lipids (ZALs) is reported. ZALs are uniquely able to deliver long RNAs (Cas9 mRNA and targeted sgRNA) from a single ZAL nanoparticle (ZNP) to enable CRISPR/Cas gene editing.

bioorganic
methods
synthesis
mechanism
review
other

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Bryo
DDOs
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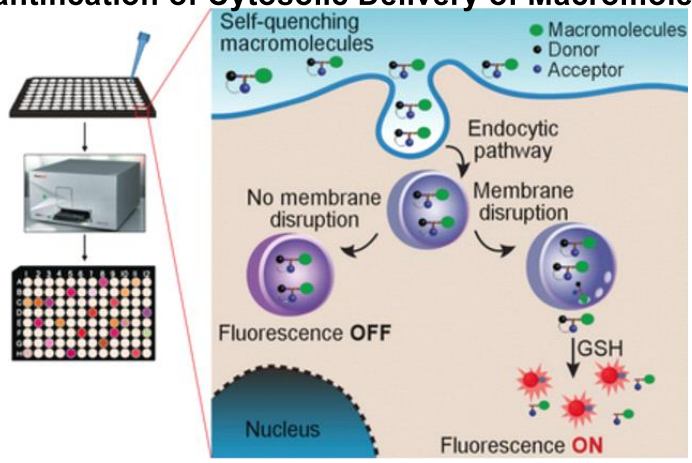
Drug Deliv.
Prostratin

Citation: Wang, Z..; *et al. Angew. Chem. Int. Ed.* **2017**, *53* (5), 1319.

A Redox-Activatable Fluorescent Sensor for the High-Throughput Quantification of Cytosolic Delivery of Macromolecules



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methods
synthesis
mechanism
review
other

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Drug Deliv.
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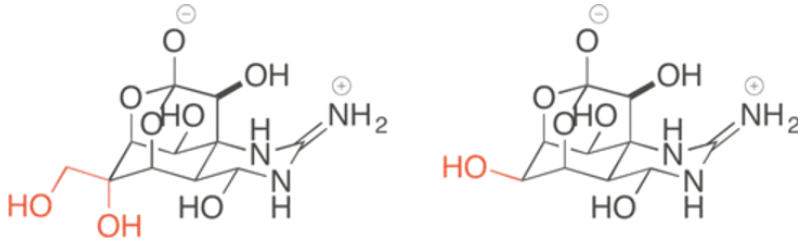
Citation: Maehara, T.; *et al. Angew. Chem. Int. Ed.* **2017**, *53* (6), 1549.

Total Synthesis of (-)-Tetrodotoxin and 11-norTTX-6(R)-ol



Pick your poison: Total synthesis of the voltage-gated sodium channel inhibitor (-)-tetrodotoxin (TTX) was achieved from p-benzoquinone in 31 steps. The synthesis, which is also applicable to TTX congeners like 11-norTTX-6(R)-ol, features efficient stereoselective oxygenation at C-6, C-7, and C-8 by taking advantage of the tricyclic skeleton, diastereoselective [3,3]-sigmatropic rearrangement of an allylic cyanate, and intramolecular 1,3-dipolar cycloaddition of a nitrile oxide.

bioorganic
methods
synthesis
mechanism
review
other

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DDOs
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Drug Deliv.
Prostratin

Citation: Yang, C.; *et al. Angew. Chem. Int. Ed.* **2017**, *53* (9), 2356.

Dual Fluorescent- and Isotopic-Labelled Self-Assembling Vancomycin for in vivo Imaging of Bacterial Infections

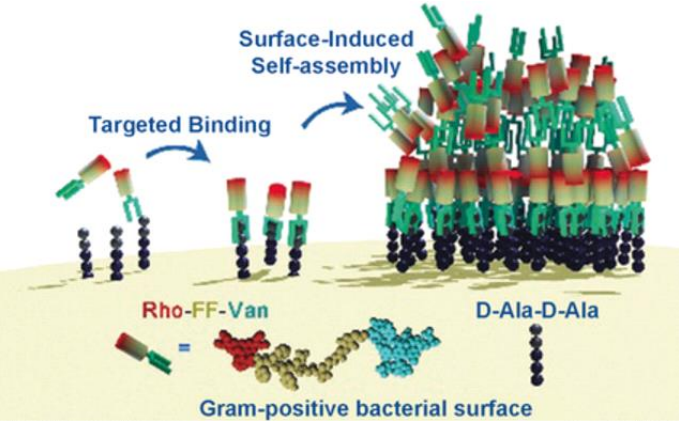

Image building: A dual fluorescent/nuclear probe based on the self-assembly of vancomycin on Gram-positive bacteria images bacterial infection. The probe aggregates on the surface of methicillin-resistant Staphylococcus aureus (MRSA) and can image MRSA-infected myositis and lungs in mice.

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDOs
Hybrid
Drug Deliv.
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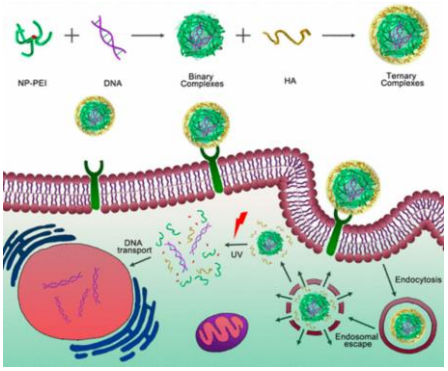
Citation: Jiang, B. et al.. *Biomacromolecules* **2017**, 183), 877-885

Harmonizing the Intracellular Kinetics toward Effective Gene Delivery Using Cancer Cell-Targeted and Light-Degradable Polyplexes



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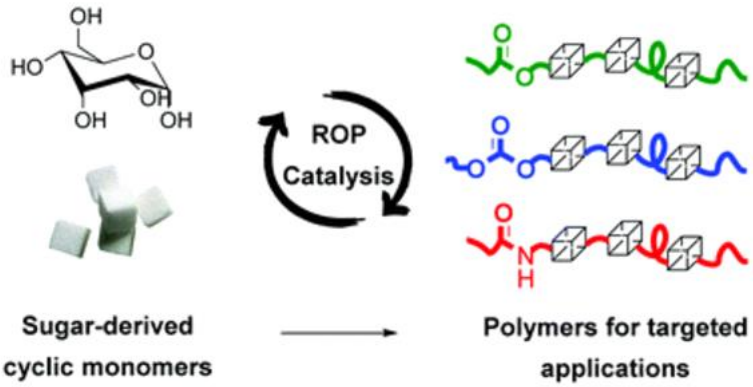
Citation: Gregory, G. *et al. Chem Commun.* **2017**, *53,* 2198.

Polymers from sugars: cyclic monomer synthesis, ring-opening polymerisation, material properties and applications



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other

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Drug Deliv.
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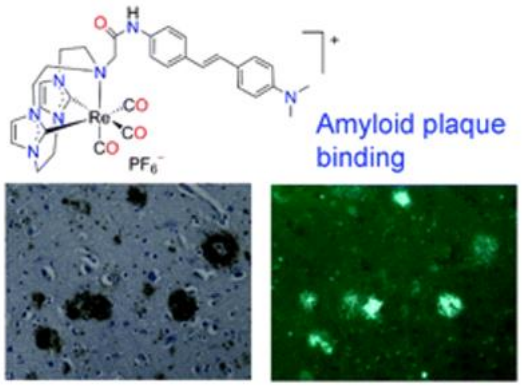
Citation: Chan, C. Y.; *et al. Chem. Commun.* **2017**, *53,* 2311.

Rhenium(I) complexes of N-heterocyclic carbene ligands that bind to amyloid plaques of Alzheimer's disease



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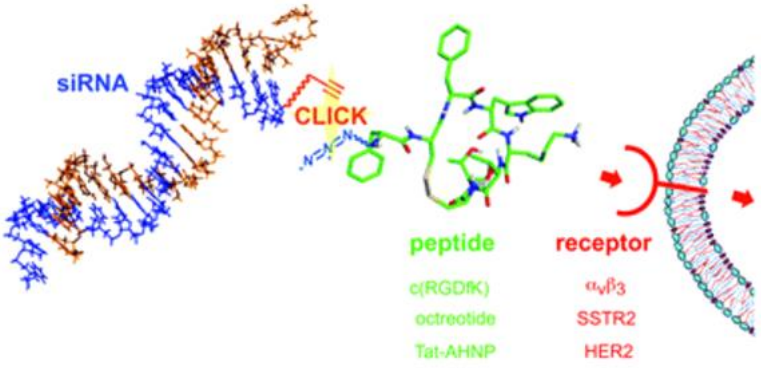
Citation: Gandioso, A.; *et al. Chem. Commun.* **2017**, *53,* 2870.

Efficient siRNA–peptide conjugation for specific targeted delivery into tumor cells



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methods
synthesis
mechanism
review
other

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Drug Deliv.
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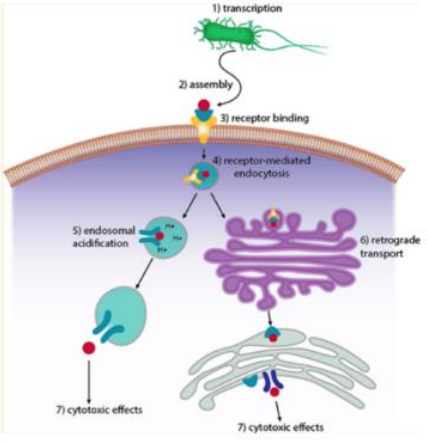
Citation: Garland, M.; Loscher, S.; Bogyo, M. *Chem. Rev.*, **2017**, *117*, 4422

Chemical Strategies To Target Bacterial Virulence

Antibiotic resistance is a significant emerging health threat. Exacerbating this problem is the overprescription of antibiotics as well as a lack of development of new antibacterial agents. A paradigm shift toward the development of nonantibiotic agents that target the virulence factors of bacterial pathogens is one way to begin to address the issue of resistance. Of particular interest are compounds targeting bacterial AB toxins that have the potential to protect against toxin-induced pathology without harming healthy commensal microbial flora. Development of successful antitoxin agents would likely decrease the use of antibiotics, thereby reducing selective pressure that leads to antibiotic resistance mutations. In addition, antitoxin agents are not only promising for therapeutic applications, but also can be used as tools for the continued study of bacterial pathogenesis. In this review, we discuss the growing number of examples of chemical entities designed to target exotoxin virulence factors from important human bacterial pathogens.



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methods
synthesis
mechanism
review
other

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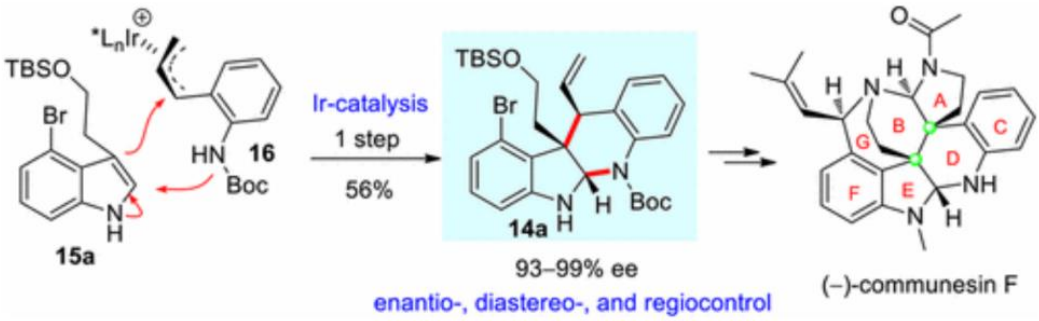
Citation: *JACS*, **2017,** *139*, 3364.

Ir-Catalyzed Asymmetric Total Synthesis of (-)-Communesin F



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synthesis
mechanism
review
other

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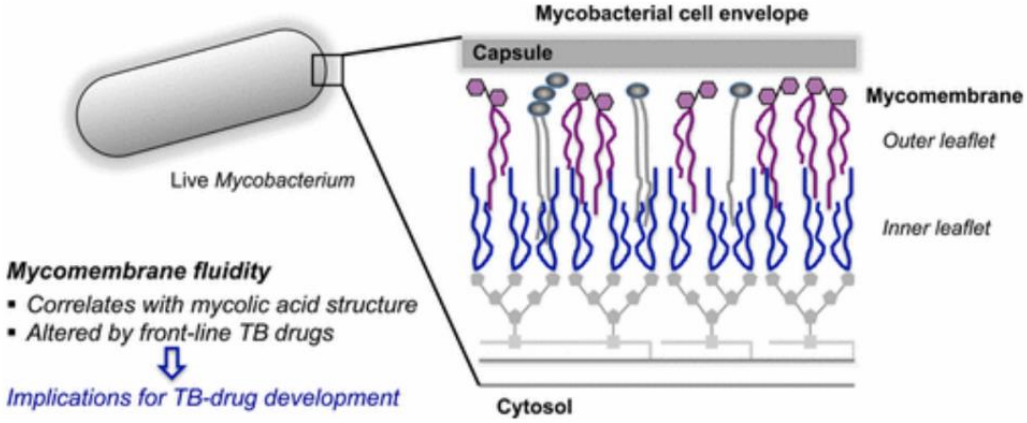
Citation: *JACS*, **2017,** *139*, 3488.

Visualization of mycobacterial membrane dynamics in live cells

Bertozzi paper



bioorganic
methods
synthesis
mechanism
review
other

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Hybrid
Drug Deliv.
Prostratin

Citation: *JAMA*. 2017;317(9):896-898. doi:10.1001/jama.2017.0276

Alzheimer Outlook Far From Bleak

bioorganic
methods
synthesis
mechanism
review
other

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Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: *JAMA*. 2017;317(9):899. doi:10.1001/jama.2017.1388

Technique Monitors T Cells as They Target Cancer

bioorganic
methods
synthesis
mechanism
review
other

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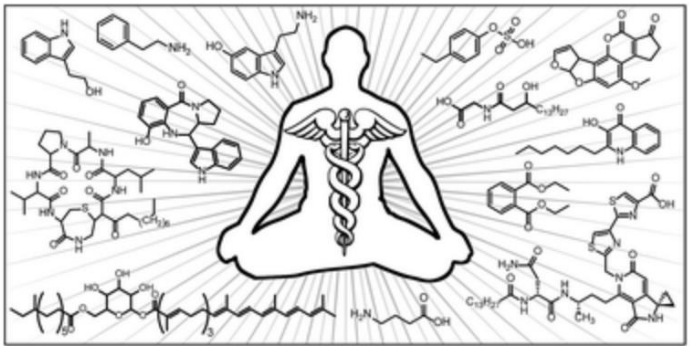
Citation: Garg, N. et al. *Nat. Prod. Rep.*, **2017**, 34, 194-219

Natural products as mediators of disease



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methods
synthesis
mechanism
review
other

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DDO
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Drug Deliv.
Prostratin

Citation: *Nature* **542,** 401 (23 February 2017) doi:10.1038/nature.2017.21502

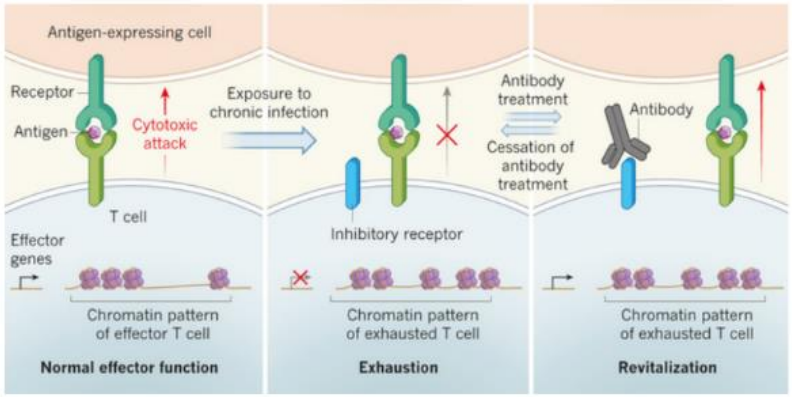
Broad Institute wins bitter battle over CRISPR patents

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: *Nature* **543**, 190–191 (09 March 2017) doi:10.1038/nature21508

Immunology: The chronicles of T-cell exhaustion



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methods
synthesis
mechanism
review
other

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Bryo
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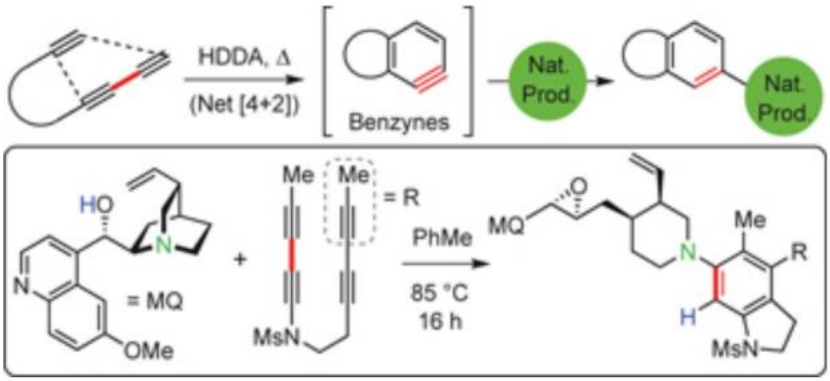
Citation: *Nature Chemistry* (2017) doi:10.1038/nchem.2732

Reactions of hexadehydro-Diels–Alder benzynes with structurally complex multifunctional natural products



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methods
synthesis
mechanism
review
other

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Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: Dubach, J. M.; *et al. Nature Chem. Bio.* **2017**, *13*, 168.

Quantitating drug-target engagement in single cells in vitro and in vivo

Quantitation of drug target engagement in single cells has proven to be difficult, often leaving unanswered questions in the drug development process. We found that intracellular target engagement of unlabeled new therapeutics can be quantitated using polarized microscopy combined with competitive binding of matched fluorescent companion imaging probes. We quantitated the dynamics of target engagement of covalent BTK inhibitors, as well as reversible PARP inhibitors, in populations of single cells using a single companion imaging probe for each target. We then determined average in vivo tumor concentrations and found marked population heterogeneity following systemic delivery, revealing single cells with low target occupancy at high average target engagement in vivo.

bioorganic
methods
synthesis
mechanism
review
other

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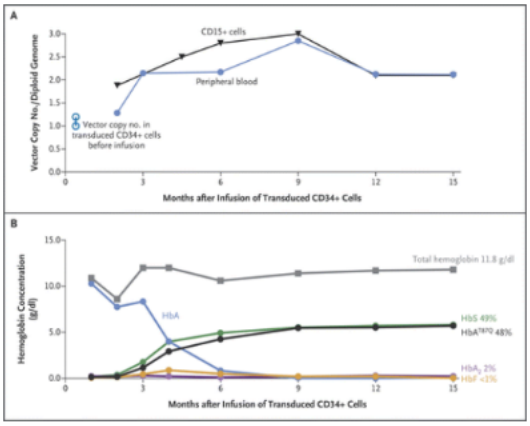
Drug Deliv.
Prostratin

Citation: N Engl J Med 2017; 376:848-855

Gene Therapy in a Patient with Sickle Cell Disease



bioorganic
methods
synthesis
mechanism
review
other

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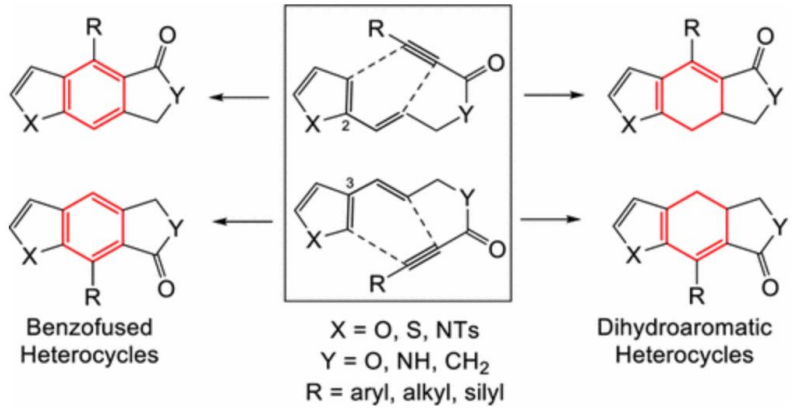
Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: Brummond *et al. Org. Lett.* **2017**. *ASAP*

Intramolecular Didehydro-Diels–Alder Reaction for the Synthesis of Benzo- and Dihydrobenzo-Fused Heterocycles



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methods
synthesis
mechanism
review
other

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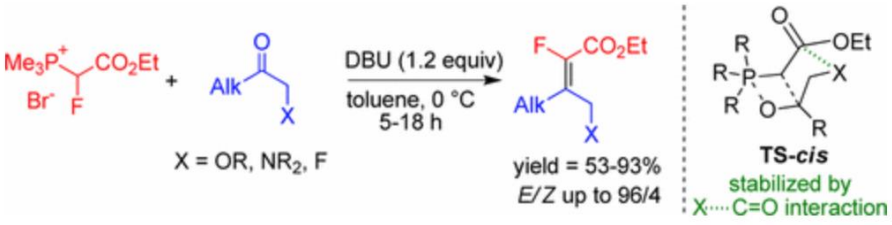
Citation: Robiete *et al. Org. Lett.* **2017**. *ASAP*

Origin of the *E*/*Z* Selectivity in the Synthesis of Tetrasubstituted Olefins by Wittig Reaction of



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methods
synthesis
mechanism
review
other

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Drug Deliv.
Prostratin

Citation: Cohen, J. *Science* **2017**, *355* (6326), 677.

Easier cure for resistant TB

A new treatment strategy has had astonishing success against extensively drug-resistant tuberculosis (XDR TB), which kills more than 70% of patients. XDR and other drug-resistant forms of TB are burgeoning among people with HIV, and current treatments are so prolonged and toxic that many patients fail to adhere to them. But a small study now shows that a simpler, safer regimen can cure the disease. Called Nix-TB, the trial has had 34 people in South Africa with XDR on three antibiotics that have never been combined before to treat TB. After 6 months, the TB bacillus could not be cultured from anyone's sputum, a sign that they had cleared the infection. More impressive, 20 people stopped taking the drugs at that point and just one relapsed.

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDOs
Hybrid
Drug Deliv.
Prostratin

Citation: Lewis, K; Shan, Y. *Science* **2017**, *355* (6327), 769.

Why tolerance invites resistance

Bacteria use two strategies to avoid being killed by antibiotics: resistance and tolerance. Resistance mechanisms such as destruction of a drug or modification of its target allow bacteria to grow in the presence of antibiotics. Tolerance is a property of dormant, nongrowing bacterial cells in which antibiotic targets are inactive, allowing bacteria to survive. The two phenomena are mechanistically distinct and assumed to be unrelated. On page 826 of this issue, Levin-Reisman et al. (1) show that tolerance nevertheless leads to resistance.

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDOs
Hybrid
Drug Deliv.
Prostratin

Citation: Levin-Reisman, I.; *et al. Science* **2017**, *355* (6327), 826.

Antibiotic tolerance facilitates the evolution of resistance

Controlled experimental evolution during antibiotic treatment can help to explain the processes leading to antibiotic resistance in bacteria. Recently, intermittent antibiotic exposures have been shown to lead rapidly to the evolution of tolerance-that is, the ability to survive under treatment without developing resistance. However, whether tolerance delays or promotes the eventual emergence of resistance is unclear. Here we used in vitro evolution experiments to explore this question. We found that in all cases, tolerance preceded resistance. A mathematical population-genetics model showed how tolerance boosts the chances for resistance mutations to spread in the population. Thus, tolerance mutations pave the way for the rapid subsequent evolution of resistance. Preventing the evolution of tolerance may offer a new strategy for delaying the emergence of resistance.

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDOs
Hybrid
Drug Deliv.
Prostratin

Citation: Nakatsuji *et al.*, *Sci. Trans. Med.* **2017**, *9*, eaah4680

Antimicrobials from human skin commensal bacteria protect against Staphylococcus aureus and are deficient in atopic dermatitis

The microbiome can promote or disrupt human health by influencing both adaptive and innate immune functions. We tested whether bacteria that normally reside on human skin participate in host defense by killing Staphylococcus aureus, a pathogen commonly found in patients with atopic dermatitis (AD) and an important factor that exacerbates this disease. High-throughput screening for antimicrobial activity against S. aureus was performed on isolates of coagulase-negative Staphylococcus (CoNS) collected from the skin of healthy and AD subjects. CoNS strains with antimicrobial activity were common on the normal population but rare on AD subjects. A low frequency of strains with antimicrobial activity correlated with colonization by S. aureus. The antimicrobial activity was identified as previously unknown antimicrobial peptides (AMPs) produced by CoNS species including Staphylococcus epidermidis and Staphylococcus hominis. These AMPs were strain-specific, highly potent, selectively killed S. aureus, and synergized with the human AMP LL-37. Application of these CoNS strains to mice confirmed their defense function in vivo relative to application of nonactive strains. Strikingly, reintroduction of antimicrobial CoNS strains to human subjects with AD decreased colonization by S. aureus. These findings show how commensal skin bacteria protect against pathogens and demonstrate how dysbiosis of the skin microbiome can lead to disease.

bioorganic
methods
synthesis
mechanism
review
other

OM
Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: Bonsignori *et al.*, *Sci. Trans. Med.* **2017**, *9*, eaai7514

Staged induction of HIV-1 glycan–dependent broadly neutralizing antibodies

A preventive HIV-1 vaccine should induce HIV-1–specific broadly neutralizing antibodies (bnAbs). However, bnAbs generally require high levels of somatic hypermutation (SHM) to acquire breadth, and current vaccine strategies have not been successful in inducing bnAbs. Because bnAbs directed against a glycosylated site adjacent to the third variable loop (V3) of the HIV-1 envelope protein require limited SHM, the V3-glycan epitope is an attractive vaccine target. By studying the cooperation among multiple V3-glycan B cell lineages and their coevolution with autologous virus throughout 5 years of infection, we identify key events in the ontogeny of a V3-glycan bnAb. Two autologous neutralizing antibody lineages selected for virus escape mutations and consequently allowed initiation and affinity maturation of a V3-glycan bnAb lineage. The nucleotide substitution required to initiate the bnAb lineage occurred at a low-probability site for activation-induced cytidine deaminase activity. Cooperation of B cell lineages and an improbable mutation critical for bnAb activity defined the necessary events leading to breadth in this V3-glycan bnAb lineage. These findings may, in part, explain why initiation of V3-glycan bnAbs is rare, and suggest an immunization strategy for inducing similar V3-glycan bnAbs.

bioorganic
methods
synthesis
mechanism
review
HIV

OM
Bryo
DDO
Hybrid
Drug Deliv.
Prostratin

Citation: Alam *et al.*, *Sci. Trans. Med.* **2017**, *9*, eaai7521

Mimicry of an HIV broadly neutralizing antibody epitope with a synthetic glycopeptide

A goal for an HIV-1 vaccine is to overcome virus variability by inducing broadly neutralizing antibodies (bnAbs). One key target of bnAbs is the glycan-polypeptide at the base of the envelope (Env) third variable loop (V3). We have designed and synthesized a homogeneous minimal immunogen with high-mannose glycans reflective of anative Env V3-glycan bnAb epitope (Man_9-V3). V3-glycan bnAbs bound to Man_9-V3 glycopeptide and native-like gp140 trimers with similar affinities. Fluorophore-labeled Man_9-V3 glycopeptides bound to bnAb memory B cells and were able to be used to isolate a V3-glycan bnAb from an HIV-1–infected individual. In rhesus macaques, immunization with Man_9-V3 induced V3-glycan-targeted antibodies. Thus, the Man_9-V3 glycopeptide closely mimics an HIV-1 V3-glycan bnAb epitope and can be used to isolate V3-glycan bnAbs.

bioorganic
methods
synthesis
mechanism
review
HIV

OM
Bryo
DDO
Hybrid
Drug Deliv.
Prostratin