ERRATA

Listed below are abstracts that were presented at the 47th Annual Meeting, March 1–5, 2003, but not included in the Abstracts Issue.

Sunday, March 2

Workshop I

Global Analysis of Protein Activities Using Protein Chips
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The genomes of a wide variety of organisms have now been sequenced; a major challenge ahead is to understand the function, regulation and modification of the many encoded gene products. We have been carrying out proteomics approaches to the identification and analysis of signalling pathways in yeast. 121 of 122 protein kinases were cloned and purified from yeast as GST fusions and analyzed for their ability to phosphorylate 60 different yeast substrates. More than 93% of the kinases exhibited activities that are 5 fold or higher, relative to controls, including 18 of 24 previously uncharacterized kinases. Many protein kinases had novel activities; for example 27 yeast kinases were found to phosphorylate Tyr. In addition, we have now cloned 6000 open reading frames and overexpressed their corresponding proteins. The proteins were printed onto slides at high spatial density to form a yeast proteome microarray and screened for their ability to interact with a variety of different proteins, nucleic acids and phospholipids. As examples, we have probed yeast proteome chips with calmodulin and six different phospholipids. These studies revealed many new calmodulin and phospholipid-interacting proteins; a common potential binding motif was identified for many of the calmodulin-binding proteins. Thus, microarrays of an entire eukaryotic proteome can be prepared and screened for diverse biochemical activities. They can also be used to screen protein-drug interactions and to detect posttranslational modifications.

488.1-Pos Board # B29.1

Formation of the Folding Nucleus of src-SH3 Domain from Denatured Conformations Investigated Through Biased Molecular Dynamics Simulations
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The experimentally well-established folding characteristics of the SH3 domains, that comprise a description of their transition state[1-3] represent a sort of testing table for theoretical investigations on protein folding. We performed parallel all-atom molecular dynamics simulations of the SH3 protein domain with an implicit solvation model. Starting from denatured conformations, by rescuing and restarting only trajectories that got closer and closer to the transition state ensemble[4], we have been able to obtain conformations where the putative folding nucleus of the protein consisting in a three-stranded β-sheet [1] is completely formed. Several conformational pathways have been identified.


Monday, March 3

778.1-Pos Board # B363.1

Effects of Ca²⁺ and Temperature on the Force-generating Transition in Cardiac Muscle Studied by Photolysis of Caged-phosphate
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We have shown that below 20°C the step determining kPi in skinned guinea pig trabeculae may shift from the force generating transition to a different step, perhaps cross-bridge formation (Biophys J. 80:586a, 2001) implying kPi should be calcium sensitive at lower temperatures. To test this hypothesis, we have measured kPi at a fixed final [Pi] (initial + photoreleased @ 1.4mM) at different [Ca²⁺] and at either 24°C or 14°C. At 14°C and full activation (pCa 4.5) kPi was 5.87 ± 0.54 sec⁻¹ (mean ± sem, n= 9) decreasing ~40% to 3.42 ± 0.92 sec⁻¹ at pCa 5.34 (P/Po~0.4). At 24°C pCa 5.93 (P/Po–0.4) kPi was not significantly different than k Pi at full activation (26.30 ± 3.9 sec⁻¹ at pCa 5.93 vs. 28.4 ± 2.87 sec⁻¹ at pCa 4.5). This suggests that Ca²⁺ control of cross-bridge kinetics differs at the two temperatures: at higher temperatures the force-generating transition is Ca²⁺-insensitive, while both cross-bridge formation and the force-generating transition are Ca²⁺-sensitive at lower temperatures. Alternatively, Ca²⁺ control is exerted at the cross-bridge formation step at both temperatures, but at 14°C, kPi is governed by a force generating transition that is slow compared to a Ca²⁺-sensitive equilibrium at the cross-bridge formation step and therefore sensitive to it.