News Letter

IBASM

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Message from the Outgoing President- Jim Mitchell

We had an outstanding meeting at the IU School of Dentistry this year. Over 75 microbiologists, molecular biologists and immunologists attended this event. I would especially like to thank Richard Gregory for arranging use of the exceptional lecture hall and poster frames. Both he and Jeanne Barnett put together an informative and memorable rendezvous.

The Friday evening ASM Waksman Foundation speaker Dr. Tim Ford discussed biofilms in aqueducts as ideal environments for pathogen survival and proliferation. These biofilms harbor hundreds of species of bacteria including many pathogens. Dr. Ford presented data from Russia and India demonstrating with an increase in socioeconomic status comes a decrease of waterborne diseases.

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The IBASM thanks the Indiana University School of Medicine-Fort Wayne for financially supporting the publication of this newsletter.

Message from the President
Jeanne Barnett

The 2004 meeting in Indianapolis was a great success. We had 44 students and 28 full members register for the meeting. The poster session was enthusiastic and informative. The students did a great job presenting their work and learned a great deal from the interactions with everyone. A big "THANK YOU" to Dr. Rich Gregory for his involvement and arranging the use of the Dental School facilities.

Now is the time to plan to attend the 2005 meeting at Abe Martin Lodge in Brown County State Park. The meeting will be Friday, April 15 to Sunday, April 17. Rooms have been reserved and we're working on the weather! Currently, it looks

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WHAT'S INSIDE...

J. Mitchell's message (continued from page 1)

In the USA biofilms contribute to a substantial amount of corrosion that occurs in drainage pipes. USA analyses demonstrated that *Mycobacterium avium* is 3X as prevalent as *M. intercellulare* and that *M. intercellulare* was found in larger amounts in cold water compared to larger amounts of *M. avium* being found in hot water. *M. avium* is of utmost concern because it is a prime killer of AIDS patients and when growing within aqueducts is extremely hard to control with chemical agents because of the protective waxy outer-layer of its biofilm.

Dr. Dominique Galli (IU School of Dentistry) continued with the biology of biofilms by discussing conjugative resistance gene acquisition in biofilms of the oral cavity. Dr. Galli described how dental plaque (biofilm) functions as a reservoir for antibiotic resistance genes that are acquired from bacteria/microbes passing through the oral cavity. A unique DNA inversion system isolated from the conjugative plasmid (pVT745) of the capnophylic periodontitis-causing bacterium *Actinobacillus actinomycetemcomitans* might serve as a model system to monitor horizontal gene transfer amongst (biofilm) microbes.

Dr. Charles Kulpa, the recipient of the 2004 IBASM Academic Scientific Achievement award, rounded out our seminar series by discussing microbial biodegradation and biotransformation of toxic wastes. Until the 1980's toxic waste was either burned or buried. Dr. Kulpa discussed a number of projects investigating the potential of utilizing microbes to remediate different toxic waste materials. The original approach of utilizing selected pure cultures worked well in the laboratory by bioaugmenting single-compound wastes, but often with lower efficacy when applied to actual waste sites. This is attributed to a combination of mixed waste found at most sites, nutritional profile of the site, competition with other microbes and adverse environmental conditions. He discussed new approaches in adjuncting (e.g. 0.1% molasses) pure and microbial consortia to improve detoxification efficacy at these types of sites.

There were a total of 25 posters presented at the meeting. The quality of the student presentations was awesome and it was very informative for me to see the range of different research areas. Jonathan Willett (IU-Bloomington) won 1st place and Katina Moore (BSU) won 2nd place in the undergraduate category. In the MS category Mike Adam (BSU) received the McClung award for 1st place and Evelyn Toh (IU-B) the 2nd place award. Elisha Rahe (IU-B) received the McClung award for 1st place and Rick Alvey (IU-B) 2nd place in the Ph.D division. Congratulations !!! The socializing which occurred during the judging segment was almost deafening at times, but a great opportunity for students to visit with each other and also interact with professionals who can provide valuable ideas and advice for future education and employment. All of us who viewed the poster session look forward to a similar number of participants next year, and I hope to possibly see students compete in the high school division. First place winners received a complimentary ASM membership and all winners will receive a certificate and monetary gift when a short paper is published in the IBASM newsletter.

We discussed several issues at the Indiana branch business meeting. We chose May 12th to meet and plan details of the 2005 IBASM meeting which will be held at Abe Martin Lodge in Brown Co. state park. Although we will have met prior to publication of this newsletter, I wanted to announce that any full member is welcome to attend this meeting. This was my last meeting as President. Jeanne Barnett will take over the reins beginning in Spring 2005. I wanted to especially thank all of the executive officers for their support in making my tenure as President a wonderful experience.

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There are several upcoming meetings not listed on the ASM website which you may be interested in attending. The Mycological Society of America (MSA) will hold its annual meeting July 18-21 in Ashville, NC. The International Workshop for the Morphological and Molecular Identification of the Stramenopiles: Phytopthora and Pythium will be held July 23-27, Raleigh, NC. The Society for Industrial Microbiology Annual Meeting will be held July 25-29, Anaheim, CA. The American Phytopathological Society Annual Meeting is July 31 - August 4 in Anaheim, CA. The Southern Great Lakes section of the Society for Industrial Microbiology (SGL-SIM) will be meeting Saturday, October 9th at Argonne National Laboratory (ANL). I especially encourage everyone to attend this annual event held each October in the Great Lakes region. At least 2 vans of students and faculty from Ball State University travel to this rendezvous each year and I would like to see more members from Indiana attend this very informative meeting. Although there are no student presentations, it will provide an outstanding avenue for students and Hoosier microbiologists to meet with industrial microbiologists from both the private and government sectors. This years' program has not yet been released, but will include speakers from ANL, Cognis, Integrative Biology Management, Eli Lilly, Abbott Laboratories, and the Illinois Institute of Technology. Presentations are usually divided between industrial and environmental microbiology topics. If you are interested in more information you can email me or SGL-SIM president Chuck Kulpa (kulpa.1@nd.edu) or visit the website http://www.simhq.org/html/localsecs/sgreatlakes.html.

Looking forward to seeing you all at one of these meetings.....mark April 15-17, 2005 on your calenders now!

Special Thanks to All Judges!

On behalf of all of the students in the poster competition I would like to express sincere appreciation to all of the members who volunteered their time to judge at the meeting. Students were evaluated in 4 different categories: scientific thought, creativity, thoroughness and presentation (abstract, oral and poster). This was no easy task! Next time you see any of these persons please thank them for sweating through a very difficult challenge:

Graduate Team #1 = McKillip (BSU) and McDowell (BSU)
Graduate Team #2 = Fuqua (IUB) and Levinthal (Purdue)
Graduate Team #3 = Deloney-Marino (USI) and Vann (BSU)
Undergraduate Team #1 = Cecil (Rose-Hulman) and Bruns (BSU)
Undergraduate Team #2 = Selvaratnam (Wabash), Bauer (IUB) and Kehoe (IUB)

J. Barnett's message (continued from page1)

like we will have 2 ASM speakers. Dr. Ralph Tanner, Professor of Microbiology at University of Oklahoma, is an applied microbial physiologist with particular experience with acetogens, methanogens, SRB, clostridia and other anaerobes. His topic will be "A Funny Thing Happened on the Way from the Sewage Plant". This is a study of bluegills living in treated sewage water and the implications of the large number of antibiotic-resistant bacteria found in these situations. Dr. James Tiedje, from Michigan State University, is the in-coming president of ASM and has agreed to also present his research at the 2005 meeting. Dr. Tiedje is a microbial ecologist and physiologist. His research is centered on the interactions of organisms within a habitat. The interactions are what determine the success of the organism in that habitat. We are privileged to have 2 fine speakers for the meeting. The meeting promises to be both informative and enjoyable.

Look for more information on registration and abstract submission for the 2005 meeting in the fall newsletter. Dr. Dominique Galli, the new president-elect, will be handling those details.

The 2006 meeting has been scheduled for McCormick Creek State Park on April 21-23, 2006. Please mark those dates to join us.

Dr. Dominique Galli, the President-Elect of IBASM presenting her talk on biofilms in the oral cavity.

Second Place Graduate (Ph.D.) Winner from 2004

Regulation of *pcyA* in *Fremyella diplosiphon*:
Bilin Biosynthesis Sheds New Light on Chromatic Adaptation

Rick Alvey, Nicole Frankenberg-Dinkel, and David Kehoe
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Through a process known as complementary chromatic adaptation (CCA), the cyanobacterium *Fremyella diplosiphon* changes color in response to ambient light color. If grown in red light (RL), cells turn green. In green light (GL) they turn red. This allows it to optimize absorption of light for photosynthesis. The analysis of mutants defective in this process led to the identification of several components in the CCA signal transduction pathway. A photoreceptor controlling CCA is RcaE, a sensor kinase protein with sequence similarity to plant phytochromes (Kehoe, *et al*, 1996). Two response regulators controlling CCA have also been identified: RcaF acts after RcaE and contains a single receiver module, while RcaC acts after RcaF and contains a histidine phosphotransfer domain, a helix-winged-helix DNA binding domain and two receiver domains (Chiang, *et al*, 1992). The focus of our lab is to better understand these factors and the signal transduction pathways that control CCA.

The difference in color between RL and GL grown cells is due to differences in composition of the phycobilisomes, the light harvesting structures that collect light energy and funnel it to photosynthetic reaction centers. These structures are large multi-protein complexes that compose up to 50% of the total cellular protein (Bogorad, 1975). They consist of both pigmented proteins and largely non-pigmented linker peptides that bind the structure together. Differences in the protein composition of the phycobilisomes determine the color of the cells. In GL the outer portions contain red-colored phycoerythrin (PE) while in RL they contain blue-green pigmented phycocyanin (PC). Linker peptides specific for each of these two types of proteins are differentially synthesized as well. CCA is mediated almost entirely at the level of transcription (Oelmüller, et al, 1988a, and 1988b). Transcripts from *cpeBA*, encoding PE, and *cpeCDE*, encoding the PE linkers, accumulate to high levels in GL and low levels in RL while mRNA from the *cpcB2A2H2I2D2* (abbreviated *cpc2*) operon, encoding inducible PC and its associated linkers together, is abundant in RL and undetectable in GL.

Two additional forms of PC are produced. "Constitutive" PC is present in the inner portion of both RL and GL grown phycobilisomes. Transcripts from this operon, *cpcB1A1* (*cpc1)*, do not change in response to light color. A third PC is the product of the *cpcB3A3* operon (*cpc3)*, and contains almost no sulfur containing amino acids. mRNA from this operon is only detected in extracts from cells that were limited for sulfur. This appears to allow the cells to continue to produce and utilize phycobilisomes when starved of sulfur (Mazel, *et al*, 1989).

My efforts towards understanding the control of CCA have focused on understanding the regulation the genes encoding the bilin biosynthetic enzymes that catalyze the reduction of biliverdin IXa to either phycocyanobilin or phycoerythrobilin. Phycoerythrobilin is the chromophore attached to PE and phycocyanobilin is the chromophore attached to PC.

PebA and PebB are the enzymes that synthesize phycoerythrobilin. They were first identified in our organism through the complementation of a mutant unable to produce PE (Alvey, *et al*, 2003). We found that the *pebAB* operon was up regulated in GL and down regulated in RL. The promoters of *pebAB* and *cpeBA* (encoding PE) shared a common sequence that was previously shown for *cpeBA* to be the site of a GL specific binding factor (Sobczyk, et al, 1993). We also demonstrated that *pebAB* expression requires the activator CpeR which was previously shown to be required for the expression of *cpeBA* and is thought to be cotranscribed as part of the *cpeCDE* operon (Cobley, *et al*, 2002).

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We wanted to determine if the expression of *pcyA* was coordinated with the expression of the PC genes. We expected to see an induction of *pcyA* in RL, but not complete attenuation in GL as seen for *cpc2* because PC is required in both RL and GL in the inner portion of the rods and core components of the phycobilisomes. Additionally, we predicted that, unlike *cpc2*, *pcyA* would still be expressed under sulfur-limited conditions.

Northern analysis was conducted on RNA from RL and GL cultures from both replete and low sulfur media to determine how *pcyA* expression was coordinated with the PC operons,. Although *cpc2* transcripts only accumulated under RL sulfur replete conditions, *cpeBA* transcripts accumulated to high levels in GL grown cells in both sulfur replete and limiting conditions. *cpc3* RNA was undetectable in sulfur replete conditions but present at approximately equal levels in both red and GL in low sulfur. *cpc1* transcripts were only present under normal sulfur conditions and were equally abundant in red and GL.

We cloned the *pcyA* gene in *F. diplosiphon* using degenerate oligonucleotides. In collaboration with Dr. Nicole Frankenberg-Dinkel the product of this gene was purified and shown to be a phycocyanobilin oxidoreductase *in vitro*.

Having shown that the gene we identified functions as expected we conducted Northen analysis on it to determine the accumulation patterns of its mRNA. *pcyA* transcripts were present in all conditions we examined. There was an approximately five fold higher accumulation of *pcyA* in RL than in GL under sulfur replete conditions but there was no significant increase in expression of *pcyA* in response to sulfur deprivation. By analyzing *pcyA* transcript accumulation in *rcaE, rcaF,* or *rcaC* mutants we determined that the proteins encoded by these genes were required for the RL specific increase in *pcyA* RNA abundance under sulfur replete conditions, suggesting that this gene and *cpc2* were controlled by a common light responsive pathway.

This finding prompted us to search for possible coordination mechanisms. One possibility was that *pcyA* expression could be regulated by PC levels. This was examined by Northern analysis of *pcyA* in a *cpc2* null mutant. If *pcyA* expression depended on PC production then a five fold increase in *pcyA* transcript abundance in RL should not be seen in this mutant. *pcyA* RNA accumulation was normal in this mutant suggesting that PC levels do not significantly control *pcyA* expression.

Another alternative is that *cpc2* and *pcyA* could share promoter elements that coordinate their expression. When we compared the two sequences, no such promoter sequences could be identified. Surprisingly however there is an element consisting of four tandem repeats of ACGAATT 300bp upstream of the putative *pcyA* coding region that is also present in three locations in and around the *cpeCDE* operon. Two copies are between *cpeD* and *cpeE,* while the third is about 1Kbp upstream of *cpeC*. If these sequences are involved in light responsive regulation of *cpeCDE* and *pcyA* they must function to simultaneously repress and activate since their transcripts are inversely regulated by light.

We have developed a testable model that explains how these elements may function, based on the regulation of the genes encoding the outer membrane proteins OmpF and OmpC in response to changes in osmolarity in *Escherichia coli* (Pratt, et al, 1996). These responses are controlled by the EnvZ/OmpR two component system. *ompC* and *ompF* expression is controlled by the binding of OmpR at binding sites present in the promoters of these genes. *ompF* transcription is repressed by the binding of phosphorylated OmpR to multiple sites in and upstream of the *ompF* promoter, which induces a looping of the DNA. *ompC*, however, is directly activated by phosphorylated OmpR. A similar mechanism could be involved in repressing transcription of *cpeCDE* and activating *pcyA* in RL. Binding of a factor to the ACGAATT repeats in RL could simultaneously up regulate *pcyA* transcription and induce a looping of the DNA at *cpeCDE* that could prevent transcription at that operon. Furthermore this looping might also inhibit the transcription of the gene downstream of *cpeCDE, cpeR.* Since CpeR is an activator required for *cpeBA* and *pebAB* transcription, and the GL response would be completely attenuated. In GL the

Alvey's paper (continued from page 6)

the binding of a factor at these sites would be reduced, resulting in the transcription of *pcyA* at its basal level and a lack of looping of the DNA at *cpeCDE* which would allow transcription to occur. The subsequent production of CpeR would result in up regulation of *cpeBA* and *pebAB*.

We are currently in the process of replacing the ACGAATT repeats upstream of *pcyA* and will compare the transcript accumulation patterns of this gene in mutant and wild-type cells grown in red and GL. We also will replace the three copies of this element in and around *cpeCDE* to determine their role in its expression. If the phenotypes are consistent with our model, we will begin to investigate binding at these sites using gel shift assays and footprinting.

From these experiments, the significance of these ACGAATT repeats to the regulation of chromatic adaptation should be apparent. If our model is correct, the identity of the transcription factor will remain to be resolved. RcaC is a good candidate for the transcription factor in this model, since it has an OmpR-class DNA binding domain and an *rcaC* null mutant is unable to shut down *cpeCDE* transcription. In the future, we will also need to develop a model that accounts for the coregulation of *pcyA* and *cpc2*.

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Second Place Graduate (Masters) Winner from 2004

Characterization of Adhesive Holdfast Synthesis Genes in *Caulobacter crescentus*

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Caulobacter crescentus is a ubiquitous aquatic bacterium that leads a dimorphic lifestyle, generating a sessile stalked cell, and a motile swarmer cell. Stable attachment of *Caulobacter* stalked cells to surfaces requires an adhesive polar organelle, the holdfast. The holdfast is composed in part oligomers of the sugar N-acetylglucosamine (NAG) which are localized to the tip of the stalk. A previous genetic screen identified adhesion defective mutants which had transposon insertions in a gene cluster involved in polysaccharide export, *hfsDABC*. Adjacent to these genes are another four genes which we named *hfsEFGH* which have sequence similarity to polysaccharide biosynthesis genes.

HfsE shares homology to UDP-sugar lipid carrier transferases and is thought to catalyze the transfer of NAG from UDP-NAG to undecaprenol phosphate carrier. HfsG is similar to glycosyl transferases that transfer the sugar from UDP-glucose and UDP-NAG to the growing repeat unit. HfsH is similar to polysaccharide deacetylases. Finally, HfsF is similar to *E.coli* Wzx, which catalyzes the translocation of undecaprenol-phosphate-linked polysaccharide across the inner membrane.

Clean in-frame deletion mutants of *hfsE, hfsF, hfsG*, and *hfsH* were constructed and analyzed for adherence to polystyrene using a crystal violet staining assay and for the presence of holdfast NAG using lectin staining. Mutants with deletions in *hfsE, hfsF, hfsG*, and *hfsH* exhibited 25%, 38%, 8%, and 5% binding to polystyrene, respectively, as compared to wild-type CB15 cells (100%). Lectin binding assays using wheat-germ agglutinin (FITC-WGA) reveal that approximately 18% of Ä*hfsE* predivisional cells bound lectin, as compared to 30% for Ä*hfsF* cells, 4% for Ä*hfsG*, and 2% for Ä*hfsH* cells, and 80% for wild-type CB15 cells.

Complementation mutants of the deletion mutants for each of *hfsE, hfsF, hfsG*, and *hfsH* were constructed and analyzed for adherence to polystyrene using a crystal violet staining assay and for the presence of holdfast NAG using lectin staining. Mutants complemented with the following genes *hfsE, hfsF, hfsG*, and *hfsH* exhibited 75%, 72%, 68%, and 60% binding to polystyrene, respectively, as compared to wild-type CB15 cells (100%). Lectin binding assays using wheat-germ agglutinin (FITC-WGA) reveal that approximately 71% of Ä*hfsE* predivisional cells bound lectin, as compared to 70% for Ä*hfsF* cells, 62%

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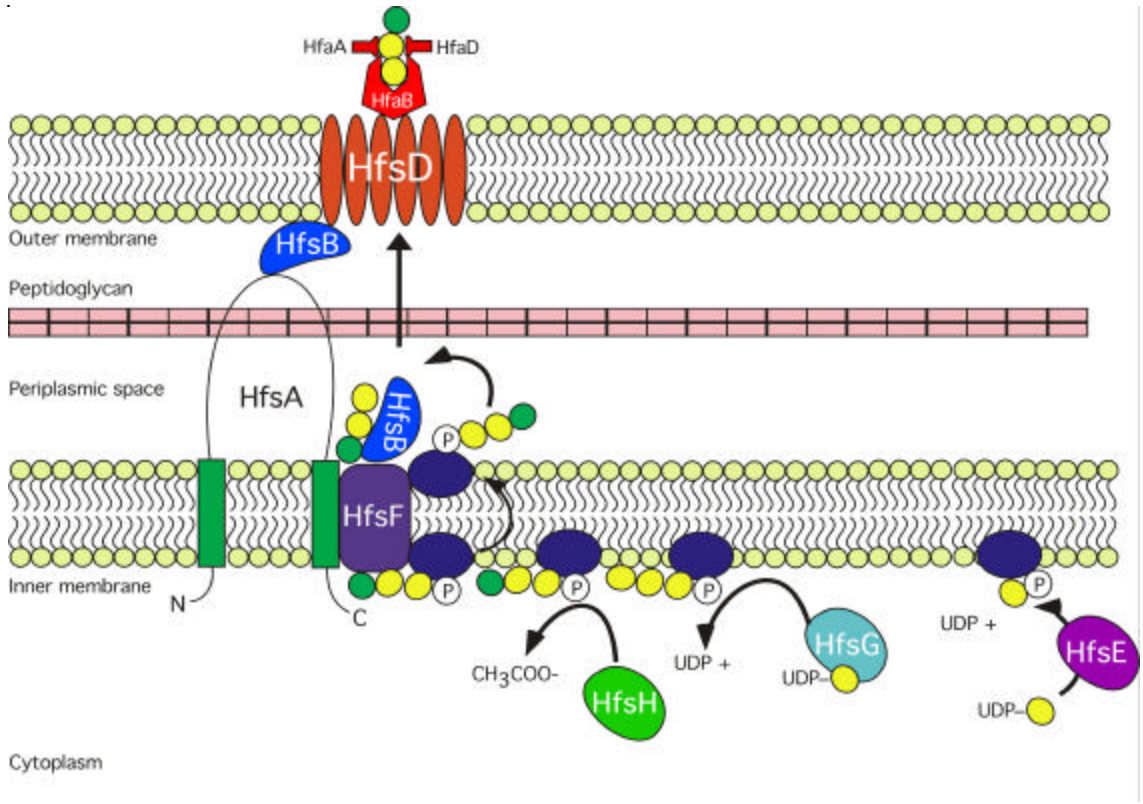
for Ä*hfsG*, and 55% for Ä*hfsH* cells, and 80% for wild-type CB15 cells.

Based on these results and on the sequence similarity of HfsEFGH to polysaccharide synthesis proteins, we propose a mechanism for the roles of HfsEFGH in the synthesis of the holdfast NAG-containing polysaccharide.



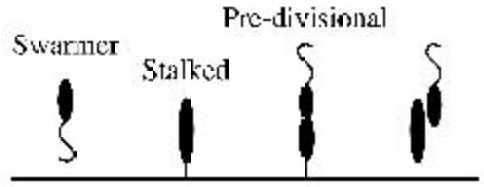
Evelyn Toh, receiving her certificate from Jim Mitchell

Second Place Graduate Winner from 2003

Cyclic Degradation of FtsA and FtsQ Allows the Coordination of DNA Replication and Cell Division

Miriam E. Martin and Yves V. Brun, Indiana University, Bloomington, IN 47405

In order to survive, cells must complete DNA replication and chromosome partitioning before cell septation. The consequences of completing cell division before the chromosomes have been properly stowed into each daughter cell can be severe. These consequences include asymmetric division, where all the DNA is inherited by one cell and the other is devoid of genetic information, and guillotination of the chromosomes, in which case neither cell inherits a full complement of DNA. *Caulobacter crescentus*, an oligotropic Gram-negative, avoids these undesirable fates through a checkpoint that stalls cell division when DNA replication is interrupted.



The Lifecycle of *Caulobacter crescentus*.

Caulobacter has an unusual life cycle in which a motile, non-replicating swarmer cell metamorphoses into a benthic, stalked cell that undergoes many rounds of DNA replication and cell division, giving rise to new swarmer cells (see figure). In *Caulobacter*, if DNA replication is initiated and then inhibited, cells can initiate constriction at the mid-point, but linger indefinitely as pre-divisional cells. Presumable the cell takes advantage of this delay in cell division to restore DNA replication. The SOS response is triggered by inhibition of DNA replication, but this replication-division checkpoint is not dependent upon the SOS response (Wortinger et al, 2000). Instead, the checkpoint is enacted through the response regulator CtrA. CtrA regulates genes involved in many aspects of the Caulobacter life cycle, including development, cell division and DNA replication (Laub et al., 2002). CtrA activates the transcription of the *ftsQA* operon in the pre-divisional cell, but shortly after a block in DNA replication, CtrA concentration drops precipitously and the *ftsQA* promoter (*Pqa*) is no longer expressed (Wortinger et al., 2000). This provides a tidy solution for cells when chromosome duplication is bogged down and septation would be ill-fated: simply turn off transcription of proteins that are required for the late stages of cell division. Cells depleted of *ftsA* and *ftsQA* are similarly able to initiate constriction but unable to complete cell division, suggesting that these proteins are essential for progression of cell division. These results are in agreement with previous observations of temperature-sensitive alleles of *ftsA*(*divE*) and *ftsQ* (*divB*) (Osley and Newton, 1980).

Turning off transcription of *ftsQA* in response to inhibited DNA replication will successfully impede cell division only if new synthesis is required for each round of cell division. To quantify FtsA and FtsQ concentration during the cell cycle, wildtype swarmer cells were isolated, resuspended in fresh media and allowed to grow. Protein samples were collected at regular intervals for SDS-PAGE and Western analysis, and the protein level was compared to dilutions of purified proteins to determine the molarity of each protein. As seen for the transcription from *Pqa* (Sackett et al., 1998), protein concentration peaked in pre-divisional cells, only to dramatically diminish directly after cell division. The rapid disappearance of FtsA and FtsQ following cell division suggested these proteins might undergo rapid proteolysis, as seen for FtsZ (Kelly et al., 1998). To distinguish between an intrinsically short half-life, or cell type-specific degradation of either FtsA and FtsQ transcript or protein, they were placed under the control of a xylose-inducible promoter (Meisenzahl et al., 1997), the cells were synchronized and then allowed to grow in fresh media with xylose. Samples were again

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collected at intervals and processed for Western analysis. Despite constitutive transcription, FtsA was entirely absent in swarmer cells and became apparent only just before cell division, as in wildtype *C. crescentus*. FtsQ concentration was moderate in swarmer cells, negligible in stalked cells and peaked in the pre-divisional cell. These results demonstrate that some form of post-transcriptional processing is responsible for the disappearance of FtsA and FtsQ from the cell. In addition, this regulated processing allows the accumulation of FtsA and FtsQ in different cell cycle patterns, both of which are distinct from the timing of FtsZ's accumulation and degradation earlier in the cell cycle (Kelly et al., 1998). It seems important to *Caulobacter*'s lifestyle to carefully regulate many components of cell division at the level of both the transcript and protein, rather than maintain tight control over FtsZ, and the initiation division, alone.

To ascertain if FtsA is subjected to cell cycle-dependent proteolysis, rather than transcript processing or tranlation interference, we measured the half-life of FtsA in swarmers, stalked cells, pre-divisional cells and the post-divisional mixed population. As FtsA is typically absent from wildtype swarmer cells, we introduced a plasmid carrying *ftsA* under the control of the xylose-inducible promoter. Xylose was added to stimulate FtsA synthesis in each cell type, protein was labeled with 35S-Methionine/Cysteine, and then a chase of cold Methionine/Cysteine was added to inhibit further labeling. Samples were collected at regular intervals and FtsA from each sample was immunoprecipitated and subjected to SDS-PAGE to determine the fraction of labeled protein remaining.

From the results of this experiment, we can make two main observations. Firstly, FtsA is a relatively short-lived protein, with a half-life of at most a third of the cell cycle. This is not true of the majority of proteins in *Caulobacter* as the total labeled protein in these cells remained relatively constant over time. In addition, most *E. coli* proteins are stable for several generations (Maurizi, 1992). Secondly, FtsA is turned over most rapidly in swarmer cells, where it has a half-life of 12 minutes, compared to the 50-55 minute half-life seen in stalked and pre-divisional cells. Models to explain the shorter half-life of FtsA in swarmers include swarmer-specific targeting of the protein to a protease, or the formation of stabilizing interactions of FtsA with FtsZ and other divisome components in stalked and pre-divisional cells. It will be of interest to learn if all the components of the division machinery are short-lived, or if the turnover of key components, such as FtsA, FtsQ and FtsZ, is enough to reset the division cycle.

Caulobacter can thus effectively coordinate late stages of cell division with DNA replication through *ftsQA* expression, because completion of cell division is contingent upon addition synthesis of FtsA and FtsQ. These proteins may not be the only targets of this checkpoint; recent DNA array results indicate that other proteins involved in cell division may also be dependent upon CtrA for expression (Laub et al, 2002). It is interesting to note that Caulobacter has set up mechanisms to coordinate different stages of DNA replication and cell division. FtsZ will not localize at the mid-cell to initiate cell division unless DNA replication has been initiated (Quardokus et al., 2001?), thus both the initiation and progression of DNA replication and cell division are tightly coupled. This line of thinking leads to the unanswered question: what *in situ* forces select for the close coordination of DNA replication and cell division (and development) during Caulobacter's lifecycle?

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Photos From the April 2004 IBASM Meeting

Chuck Kulpa receiving the IBASM Academic Scientific Achievement award from Jim Mitchell.

Judges talking to a student presenter.

Poster presentations.

Rick Alvey receives his award from Jim Mitchell.

Dr. Tim Ford, the ASM Waksman Foundation speaker, presenting his talk on biofilms in aqueducts.

IBASM Archives

We are in the process of updating branch archives including past meeting sites and branch presidents. As you can observe in the following paragraphs there are major gaps in time that a president and meeting site have not been identified. If anyone could supply useful information to fill in these empty spaces please email Jim Mitchell (jkmitchell@bsu.edu).

Past-Presidents of IBASM

H.M. Powell, whom the IBASM Powell service award was named for, was extremely active in the branch for many years serving as Vice President, President and Secretary/Treasurer (probably all 3-4 offices at the same time!). Other persons who may have possibly been past presidents (I really need your help on this) include: Chuck Hershberger and Steve Queener (Eli Lilly), Alvin Levine and Jack Bauer (I.U. School of Medicine), a woman (Ernie?) from Bayer in Elkhart and Mike Tansey (IU Bloomington)?

W.A. Jamieson, Eli Lilly & Co: 1936-1938 (First Branch President)
Bill Baldwin, I.U. School of Medicine, Gary, In: 1980
Chuck Kulpa, Notre Dame: 1988-1989
Paul Skatrud, Eli Lilly: 1991
Arun Sirvastavia, I.U. School of Medicine: 1992
Glenn Merkel, I.U. School of Mecicine: 1993
Anne Miller, Ely Lilly: 1994
Richard Gregory, I.U. School of Dentistry: 1995
Carolyn Vann, Ball State University: 1996-1998
Nancy Behforouz, Ball State University: 1998-2000
Kara Eberly, St. Mary's College: 2000-2002
Jim Mitchell, Ball State University 2002-2004
Jeanne Barnett, University of Southern Indiana: 2004-

Meeting Sites of IBASM

The branch met for a number of years at Ely Lilly. This last decade our venue has switched to state parks as often as possible. I have meeting site data going back to 1996 so would appreciate earlier information if anyone has this.
2005 Abe Martin Lodge, Brown County State Park
2004 IU Dental School, Indianapolis, IN
2003 Spring Mill Inn, Spring Mill State Park
2002 Clifty Inn, Clifty Falls State Park
2001 Abe Martin Lodge, Brown County State Park
2000 Courtyard by Marriott, Indianapolis, IN
1999 Canyon Inn, McCormick's Creek State Park
1998 Turkey Run Inn, Turkey Run State Park
1997 Potawatomi Inn, Pokagon State Park
1996 Hueston Woods State Park, College Corner, OH
For additional historical background on the Indiana Branch please read Rich Gregory's "History of the Indiana Branch" and Eugene Weinberg's lecture from the 2001 Branch Meeting at IBASM website: http://users.ipfw.edu/merkel/IndianaASM.html

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New Patch May Enhance Effects of Flu Virus Vaccine in Aging Population

Researchers from Maryland have developed a patch designed to stimulate the immune system and enhance the effectiveness of the injected flu vaccine. Their findings appear in the July 2004 issue of the *Journal of Virology*.

The elderly population is one of the groups hardest hit during outbreaks of infectious diseases, such as influenza, due to a decrease in immune system function with age. High mortality rates in humans over the age of sixty-five continue to challenge vaccinologists to explore new means of improving immune responses to the flu virus vaccine.

"During influenza epidemics, the rate of hospitalization in the elderly is high, with up to a 90% mortality rate," say the researchers.

LT-IS, an immunostimulating patch containing the enterotoxin *Escherichia coli*, is to be applied directly over the flu vaccine injection site. In the study young and old mice were administered the flu virus vaccine, after which some were treated with LT-IS for a period of eighteen hours. Both age groups showed an enhanced antibody response to the vaccine after wearing the patch, while the older mice that received the patch in addition to the vaccine showed responses equal to or greater than that of young mice receiving only the vaccine.

These results suggest that addition of an LT-IS patch may compensate for the deficient immune function seen in the aged in response to influenza virus vaccination," say the researchers. "Therefore, use of an LT-IS patch could be a new, safe, and simple immunization strategy that may significantly improve the outcome of influenza virus vaccination in the elderly."

(**M. Guebre-Xabier, S.A. Hammond, L.R. Ellingsworth, G.M. Glenn.** 2004. Immunostimulant patch enhances immune responses to influenza virus vaccine in aged mice. *Journal of Virology*, 78. 14: 7610-7618.)

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Shower Curtains May Harbor Harmful Microbes

Filmy residue, or "soap scum", on household shower curtains may be a breeding ground for potentially harmful bacteria say researchers from San Diego State University, California, University of Colorado at Boulder, and Washington University in St. Louis, Missouri. Their findings appear in the July 2004 issue of the journal *Applied and Environmental Microbiology*.

"One household environment that potentially accumulates microbial biofilms is that provided by vinyl shower curtains. Over time, vinyl shower curtains accumulate films, commonly referred to as "soap scum," which microscopy reveals are constituted of lush microbial biofilms," say the researchers.

In the study DNA samples were taken from four vinyl shower curtains in different homes. A broad range of RNA gene sequences were revealed with *Sphingomonas* spp. and *Methylobacterium* spp. appearing as the most prominent, both of which are considered to be opportunistic pathogens commonly associated with water reservoirs.

"These results show that shower curtains are a potential source of opportunistic pathogens associated with biofilms," say the researchers. "Frequent cleaning or disposal of shower curtains is indicated, particularly in households with immune-compromised individuals."

(**S.T. Kelley, U. Theisen, L.T. Angenent, A. St. Amand, N.R. Pace. 2004**. Molecular analysis of shower curtain biofilm microbes. *Applied and Environmental Microbiology*, 70. 7: 4187-4192.)

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2004 Membership Application/Renewal

*If you have not paid your dues for **2004**, you can do it now. Dues are $15.00 for non-students and $5.00 for students (per year). Please return the completed form with check, payable to IBASM, to*

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