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MITOTIC AND MEIOTIC CELL CYCLE CONTROL AND EXECUTION

CONTROLE ET EXECUTION DU CYCLE CELLULAIRE EN MITOSE ET MEIOSE

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Rapport sur la Conférence

Conference Report

The complementation of the generous financial and logistical support from CNRS with the superb setting of the Station Biologique de Roscoff and its environs made the Jacques Monod conference "*Contrôle et exécution du cycle cellulaire en mitose et méiose/Mitotic and meiotic cell cycle control and execution*" a resounding success. The outstanding reputation of the previous 14 CJM Cell Cycle meetings in Roscoff meant that the topics covered by the invited speakers could be complemented by selection of further speakers, from a very strong field of applicants. The quality and breadth of applications supported a seamless continuum between the themes within the different sessions. Although the official feedback data is yet to be compiled, several attendees remarked upon how the quality of the selected talks was so high that it was impossible to differentiate the invited from selected speakers. The intimate nature of the meeting and engaging personalities of the speakers drove lively debate to maintain the long-standing reputation of these meetings as the key forum for the inception and development of novel concepts in cell cycle control and execution. The constellation of speakers not only struck a gender balance with 21 female and 23 male presenters but sampled the cross section of career progression from PhD students, through post-docs/junior faculty, to world leaders in their respected areas of cell cycle research. To maximise inclusion and exposure, session chairs were selected from those for whom it was not logistically possible allocate a slot to present a platform presentation. Further integration and opportunities for dialogue were ensured by both the packed and dynamic poster sessions and the popular excursion to Isle de Batz. As a consequence, several students and post-doc attendees commented on how surprised they were at the ease with which they could spend extended periods of quality time with pre-eminent figures in the field.

The exceptional organisational skills of Mme Babic deserve special mention. Her prodigious talents and unimposing attention to detail ensured that we were able to focus entirely upon the scientific delivery of the meeting. We cannot speak too highly of the quality of her support in all matters including programme reorganisation to accommodate catering arrangements and inconvenient flight schedules from Brest. One pre-eminent speaker made a point of telling us that the help she received juggling her travel plans was the best she had ever experienced after her increasingly chaotic travel conflicts were swiftly resolved once Mme Babic stepped in to guide her personal assistants.

In keeping with the tradition of CJM Cell Cycle meetings, there was a modest shift in emphasis from the impact of distinct environmental settings upon cell division at the previous meeting, "*Le cycle cellulaire dans tout ses états/Cell cycle inside out*", towards the conserved regulatory networks that control progression through the cell division cycle. The different elements of this theme of control came together in the penultimate session on the subversion and exploitation of these controls in the initiation, progression and treatment of Cancer. The diversity of model systems and approaches used to study cell cycle progression meant that many exciting, novel and state of the art technologies were covered, ranging from structural biology, through highly innovative approaches for high resolution imaging of living cells, to discussions about some pitfalls encountered when using CRISPR/Cas9 technology for genome editing.

Flanked by Plenary Sessions from Profs. Kim Nasmyth and Geneviève Almouzni, the main sessions covered:

Session II: Meiotic and embryonic cell cycles and divisions
Session III: Controlling the cycle I
Session IV: Kinetochores in division and control
Session V: Mechanics of cell cycle control
Session VI: Protein phosphatases in cell cycle control
Session VII: Controlling the cycle II
Session VIII: Context
Session IX: Cancer

Cell Cycle Biology

Cell cycle progression is driven by the sequential gain and loss of the activities of the CDK-Cyclin family of protein kinases. The regulatory, cyclin, subunits of the principle CDK-Cyclin complexes are subjected to sustained destruction for distinct, discrete, cell cycle phases. This periodicity means that the activity of each kinase exhibits a specific periodicity to drive the timely execution of cell cycle events. To put the talks in this meeting into context, it is important to understand how the mitotic B type Cyclin is targeted for destruction: ubiqutinylation by a multi-subunit ubiquitin ligase complex called the Anaphase Promoting Complex (APC/C). APC/C also targets securin for destruction. Securin restrains the activity of a protease called separase. Separase cleaves a component of the cohesin complex that holds sister chromatids together from S phase until anaphase. Consequently, APC/C activity promotes sister chromatid separation at the same time as Cdk1-Cyclin B activity is lost, to push the cell from metaphase through to choromosome segregation in anaphase. Both proteins are recruited to the APC/C through their association with an activator called Cdc20. A pathway called the Spindle Assembly Checkpoint (SAC) ensures that APC/C activity is only released when all of the chromosomes are attached to spindle microtubules. A key SAC component, Mad2, undergoes a conformational switch at unattached kinetochores that enables it to form a Mitotic Checkpoint Complex (MCC) and sequester Cdc20 in order to halt Cdc20's ability to take substrates to the APC/C for ubiquitinylation. Consequently, once the last chromosome attaches to the spindle, APC/C activity is released and sister chromatids move to opposite poles in anaphase. A number of other Mad and Bub proteins join Mad2 in the SAC pathway.

Meeting report
We were absolutely delighted to be able to follow the welcome reception and dinner with a plenary talk from Prof. Kim Nasmyth (*Oxford, UK*). Prof. Nasmyth has been a leading light in the cell cycle field since his PhD studies in Prof. Murdoch Mitchison's lab in the 1970s when he worked with Sir Paul Nurse to isolate the fission yeast cell cycle mutants that led Prof. Nurse to his Nobel Prize winning discovery of the ubiquity of cell cycle control by Cdk-cyclin kinases. After defining sex determination in budding yeast, the architecture of cell cycle transcriptional control networks and the means by which a relay of Cdk-Cyclin kinases controlled progression through the budding yeast cell cycle, Prof. Nasmyth has spent the past 20 years identifying, characterising and defining the role of the cohesin complexes. His remarkable contributions have dominated the field to earn recognition with his receipt of the 2018 Breakthrough Prize. As anticipated, Prof. Nasmyth delivered an electrifying talk describing his latest studies on the mechanisms by which the cohesin complex could embrace sister chromatids. This is particularly fascinating issue as the complexes must be loaded to bind the two sisters as each chromosome is replicated in S phase and yet cohesin association must remain flexible enough to support multiple levels of local and higher order chromatin remodelling, to continuously accommodate the transcriptional demands, imposed by dynamic fluctuations in environmental cues. Prof. Nasmyth's exquisite combination of structural, biophysical, biochemical and genetic technologies are revealing multiple modes of chromosome capture by this versatile complex.

The first general session, on Tuesday morning, picked up from where Prof. Nasmyth had left off by covering the particular challenges presented by the demands of the two sequential meiotic divisions. In the first meiotic division the two homologous chromosomes are separated away from one another, while the cohesin binding the two sister chromatids of each homologue together is maintained. This cohesin, that faithfully held sisters together throughout meiosis I, is then removed in the second meiotic division to produce the single copies of each homologous chromosome in each of the four products of the meiotic divisions. A key aspect to the flip between these modes of division is the maintenance of the centromeric cohesion during meiosis I as a consequence of the recruitment of Protein Phosphatase 2A (PP2A) to centromeres by Shugoshin 2 (Sgo2). Three talks in session I described how a multitude of protein kinases regulate meiotic centromeric cohesion. Katja Wassmann (*Paris*) described how a combination of Mps1 phosphorylation of histone H1 and a Histone chaperone promote Sgo2 recruitment to ensure protection of centromeric cohesion in meiosis I, while Adele Marston (*Edinburgh, UK*) described the means by which recruitment of Polo kinase to centromeres by Spo13 during

meiosis I counteracts the drive by the kinases Hrr25 and DDK to promote centromere segregation. Soni Lacefield (*Bloomington, USA*) described further levels of control by protein kinases by revealing how the SAC components Bub1 and Bub3 recruit aurora kinase to regulate chromosome integrity and segregation. Wolfgang Zacchariae's (*Martinsried, Germany*) presentation addressed the long-standing question of the impact of tension across the centromeres in regulating local cohesion during meiosis II. Contrary to some models, her reported that tension was required to control progression through division by the Anaphase Promoting Complex (APC/C), rather than the protection of cohesion at centromeres. The complexity of the control of chromosomal architecture during meiosis was highlighted later in the meeting, by Sylvie Tournier (*Toulouse*) who described how condensin recruitment to telomeres during meiosis I but not meiosis II relied upon aurora B activity.

The remaining talk in Session I, by Thomas Mayer (*Konstanz, Germany*), focused upon meiotic control of APC/C activity by a protein phosphatase called calcineurin (CaN). This calcium regulated phosphatase plays a major, yet poorly understood, role in regulating cell cycle activation and control in many oocyte systems. Mayer removed much of this mystery by recounting how, in *Xenopus* eggs, CaN contributes to timely APC/C activation by both negatively regulating the activity of an APC/C inhibitor called XErp1 (Emi1 in human cells) and positively regulating the activity of one of the two APC/C activating proteins, Cdc20. This theme of APC/C control extended into session II as David Morgan (*San Francisco, USA*) described an epic biochemical journey that culminated in the finding that APC/C activity is exquisitely sensitive to activator ejection by poly-anions, while Prasad Jallepalli (*New York, USA*) described how the assembly of the APC/C inhibitory mitotic checkpoint complex by Mad1 and Mad2 relies upon their recruitment to kinetochore by Rod-Zw10-Zwilch (RZZ).

Unexpected roles for Mad2 regulation of APC/C activity in cell cycle control were revealed in a fascinating presentation by Arshad Desai (*San Diego, USA*). His team used *C. elegans* to make the unanticipated observation that Mad2 regulates APC/C activity in G2 phase, long before cells get into mitosis and use Mad2 in SAC control of APC/C. They found that G2 phase Mad2 sets the rate at which cells can accumulate the APC/C target Cyclin B. Further reinforcement of APC/Cs activity towards Cyclin B in G2 emanates from Cdk1-Cyclin B phosphorylation of the APC/C activator, Cdc20 to reduce its affinity for APC/C. These two controls converge on APC/C to determine the rate at which Cyclin B levels rise in G2 towards the critical threshold that promotes mitotic commitment in their system.

This question of how Cdk-Cyclin activities are regulated to control the timing with which cells commit to division was explored in several more presentations. After Lionel Pintard (*Paris*) revealed the importance of aurora kinase control of Polo kinase 1 (Plk1) in determining when Plk1 supported Cdk1-Cyclin B activity to drive mitotic commitment, Iain Hagan (*Manchester, UK*) described how the fission yeast spindle pole played a key role in regulating the activation of polo kinase activity. As in other systems, fission yeast polo is activated by Cdk1-Cyclin B in feedback controls that repress the activity of the Cdk1-Cyclin B inhibitory kinase Wee1 and boost the activity of the counteracting phosphatase Cdc25. Hagan showed how this feedback control is reinforced, at several levels, by Cdk1-Cyclin B and polo kinase control of the spindle pole component's pro-mitotic activity. Meanwhile, systematic time-lapse assays of Plk1 activation in human cell lines by Arne Lindqvist's lab (*Stockholm, Sweden*) revealed how the DNA replication checkpoint regulates Wee1 activity to set the timing of mitotic commitment. Their unanticipated finding that it is actually Cyclin A-CDK complexes that trigger Plk1 activation was echoed in a fascinating dissection of the G2/M transition described by Helfrid Hochegger (*Brighton, UK*) later in the meeting. His lab has been at the forefront of the development and application of chemical genetic approaches to dissect Cdk-Cyclin control of cell cycle progression. They made the surprising finding that Cyclin A alone was able to drive cells into division when Cyclin B had been depleted.

Impressive work from Susanne Lens's (*Utrecht, Netherlands*) team revealed how Polo and Aurora B kinase activities are each employed during mitotic exit to regulate cytokinesis. Plk1 releases a centralspindlin complex from the inter-digitating, spindle mid-zone, to enable Aurora B to direct it's oligomerisation to form a ring at the cell cortex that will ultimately mature into the cytokinetic ring that pinches the mother cell in two to generate the two daughters.

Session IV focused upon the role played by the formation of the kinetochore, on the centromeric DNA sequences, to capture the spindle microtubules. Unfortunately, compelling personal reasons meant that Andrea Musacchio (*Dortmund, Germany*) had to withdraw from the meeting, however, comprehensive insight into the structural basis for kinetochore assembly came from the other speakers in this session. Suzanna Storchova (*Martinsried, Germany*) described how the shugoshin protein, Sgo1, recruits the cohesin related condensin complex to provide structural integrity to kinetochores, while Sara Carvalhal (*Oeiras, Portugal*) recounted how important complementary structural integrity provided by cohesin is to the fidelity of chromosome attachment and segregation. Talks from Viji Draviam (*London, UK*) and Jennifer de Luca (*Fort Collins, USA*) provided molecular insights into the assembly of the kinetochore corona, a topic that had been beautifully introduced earlier in the meeting by Prasad Jallepalli's dissection of Mps1 function in kinetochore formation. Draviam and DeLuca's work focused upon the means by which Ndc80 associated complexes govern the recruitment of other key kinetochore components, including the SKAP-Astrin complex that anchors microtubule ends to the kinetochore.

Session V saw a set of talks defining some exquisite work on the structural molecular biology of the APC/C and the spindle assembly checkpoint network, alongside striking inroads into the similarities and complexities of genome segregation in plants. Jon Pines (*London, UK*) got the session off to a sensational start with an account of a totally new twist to SAC control; the recruitment of Cdk1-Cyclin B to unattached kinetochores through its association with Mad2 partner, Mad1. This association supports the recruitment of Cdk1-Cyclin B to kinetochores 10-15 minutes before nuclear envelope breakdown marks the irreversible commitment to mitosis. Abolition of this recruitment significantly compromised SAC signalling. Further insight into the finer points of SAC control by Mad1 were covered by Hongtao Yu's (*Dallas, USA*) presentation about a metazoan kinetochore specific receptor for Mad1: the ROD-ZW10-ZWILCH complex. Intriguingly, this complex plays a greater role in SAC signalling in response to tension at the kinetochore, than it does when kinetochores cannot bind microtubules at all. These accounts of Mad1 function were complemented by a stunning account from Claudio Alfieri (*Cambridge, UK*) of his cryo-electron microscopy studies of how the structure of the APC/C changes as the MCC binds to inhibit its ability to ubiquitinylate substrates before re-configuration supports APC/C activation upon departure of the MCC when the SAC is turned off. Hyun Sook Lee's (*Seoul, South Korea*) presentation complemented this study of SAC attenuation at the APC/C with a striking impact of BubR1 acetylation at the kinetochore upon SAC silencing.

A further remarkable twist to Mad2 control of the anaphase switch came from Olaf Stemmann's group (*Bayreuth, Germany*) as he described how Mad2 turns the meiotic PP2A recruiter Sgo2 (introduced in session II above) into a separase inhibitor to reinforce APC/C dependent imposition of metaphase arrest by unattached kinetochores. This, pseudo-substrate inhibition is relieved by the action of the Mad2 regulators TRIP13 and $p31^{comet}$. The theme of securin control and cohesion was extended into plants with remarkable live cell imaging of meiosis in *Arabidopsis* by Raphael Mercier (*Versailles*) and Arp Schnittger (*Hamburg, Germany*). The Mercier lab used their finding that PATRONUS is the long sought after plant securin to identify securin homologues in all systems in which a homologue was yet to be identified, while Schnittger's imaging identified a novel regulator of cohesin loading.

Dynamic control by protein phosphorylation plays a key role in driving cell cycle progression. Session VI was dedicated to the protein phosphatases that ensure that all phosphorylation events are transient and thereby drive cell cycle progression. Jean-Paul Javerzat (*Bordeaux*) kicked off

the session with an account of the antagonism between protein phosphatase 4 (PP4) and CDK5 protein kinase in loading cohesin at fission yeast centromeres. Jakob Nilsson (*Copenhagen, Denmark*) provided deep insight into how PP4 maybe exerting such control by defining the motif that recruits the trimeric PP4 holoenzymes to substrates. The potential breadth of impact of PP4 on cell cycle control was highlighted by the presence of this motif in a broad range of proteins, including the Cdk1/Cdk2 regulator Cdc25.

PP2A phosphatases play major roles in regulating progression through and exit from mitosis. As in PP4 holoenzymes, the catalytic subunit of PP2A is anchored to a structural scaffolding subunit alongside a regulatory subunit. Of the four types of regulatory subunit, B55 and B56 are found in the principle mitotic phosphatases. PP2A-B55 is inhibited by ENSA and ARPP19 once they have been phosphorylated by Greatwall kinase. Anna Castro's group (Montpellier) has been generating mouse models to determine the relative contribution of ENSA and ARPP19 to PP2A regulation throughout development while Myreille Larouche's (*Montreal, Canada*) PhD studies are addressing how the regulation of Greatwall shuttling between the cytoplasm and nucleus controls its access to cytoplasmic ENSA. Julia Kamenz (*Stanford, USA*) has been combining mathematical modelling of PP2A-B55 antagonism with Cdk1-Cyclin B kinase activity with meticulous assays of PP2A-B55 activity in *Xenopus* egg extracts as they progress through the cell cycle. Her results revealed a fascinating stimulation of PP2A-B55 activity by Cdk1-Cyclin B as cells approach mitosis. She proposed that this unanticipated relationship between Cdk1-Cyclin B and its antagonist represents an incoherent feedforward system that sharpens the transitions between interphase and mitosis. Adrian Saurin's team (*Dundee, UK*) has been addressing the functional diversity between the four different B56 isoforms. After finding that each version of the regulatory subunit directs the respective phosphatase to a distinct part of the mitotic spindle they generated chimeric fusions of different isoforms to precisely define the domains conferring specificity.

The ability of checkpoint control pathways to restrain cell cycle progression, in order to ensure that the cycle does not progress until all events required for faithful genome transmission have been completed, has been studied in great detail. However, although the fact that none of the arrests is permanent has been noted, little is known about how this built in obsolescence is engineered. This "leakiness" is important because checkpoint persistence for an extended period is indicative of a major perturbation that will cause serious damage. It is important that the cell move on to another state in which it will either execute apoptosis (metazoans), or it will have another throw of the dice in the next cell cycle if it is a unicellular organism, such as a yeast. For the spindle assembly checkpoint response to unattached chromosomes, the departure from checkpoint arrest, without resolving the problem, or segregating the chromosomes, is called mitotic slippage. Understanding slippage is of critical importance, because many cancer cells use slippage to avoid the toxic impact of mitotic arrest by taxol. It has been shown that blocking this slippage with a non-destructible cyclin B can flip cancer cells from resisting taxol, to becoming exquisitely sensitive to its lethal impact. The talk from Simonetta Piatti (*Montpellier*) about slippage was therefore one of the major highlights of the meeting as she described how protein phosphatase 1 (PP1) control of the phosphorylation status of a single residue in Mad3 regulates slippage. Mutating this single residue to block phosphorylation traps budding yeast cells in a state of permanent SAC arrest.

This fascinating talk by Piatti was followed by another stand out presentation on the means by which human cells decide to enter the cell division cycle. Tobias Meyer's (*Stanford, USA*) live cell imaging of cell cycle reporters has enabled him to add exquisite detail to define the hurdles that must be passed in order for cells to commit to the cell division cycle. It has long been established that Cdk4/6-Cyclin D control of a transcriptional switch involving the Rb and E2F proteins regulates commitment to cell division from both a quiescent and actively cycling state. For over 25 years, this control has been assumed to be the only means by which the transcriptional cascade that drives division can be initiated. Remarkably, Meyer presented compelling data to indicate that, while this dogma is true in some cell lines, other cell lines use

Cdk2-Cyclin E/A complexes to trigger commitment. This revelation will have immediate impact in the clinic, as it provides a means to identify cancers in which the Cdk4/6 inhibitors that are already being used to great effect to treat breast cancer, can be more widely applied to treat other tumour types.

After Giselene Pereira's (*Heidelberg, Germany*) fascinating account of how the position within the cell of the anaphase B spindle, with its correctly segregated genomes, determines whether budding yeast cells can exit the cycle or not, Monica Gotta (*Geneva, Switzerland*) described the equally important positioning of the mitotic spindle by Polo kinase in *C. elegans* embryos. A novel spin on the question of spatial control of division was provided by Marie-Emilie Teret's (*Paris*) account of the impact of cortical stiffness upon cell division in mouse oocytes. Their finding that a simple perturbation of cortical stiffness to generate soft oocytes leads to chromosome alignment errors provides major insights into a novel route by which aneuploidy maybe generated in the 45 year-long meiotic arrest of human oocytes.

One aspect of cell cycle control that is of critical clinical significance is the mechanism by which microtubule poison taxol selectively eliminates cancer cells. As prolonged SAC dependent mitotic arrest triggers apoptosis, it has long been assumed that their altered genome renders cancer cells much more sensitive to strain on SAC control than neighbouring normal tissues. However, the mechanistic basis for this switch to death has remained a frustrating mystery. There was therefore great interest in an engaging presentation from Hironori Funabiki (*New York, USA*) in the penultimate session. He described how the c-GAS-STING pathway that detects cytoplasmic DNA, as an anti-viral system, is initially silenced as cells enter mitosis, to prevent a c-GAS-STING inflammatory death response being prompted each time the nuclear envelope breaks down to expose mitotic chromosomes to the cytoplasm. Intriguingly however, this repression of c-GAS-STING signalling declines with prolonged arrest to eventually flip c-GAS-STING signalling into positively triggering apoptosis. Remarkably, a second mechanism for the triggering of mitotic death may well have emerged at this meeting as, a tantalising observation from Olaf Stemmann described how simultaneous removal of Sgo2 and securin immediately triggers apoptosis to suggest that separase may also act as a long sought inducer of cell death from mitotic arrest.

Hyun Sook Lee's accounts of high rates of tumour formation in her Bub1 acetylation deficient mouse resonated with the long known karyotypic abnormalities in tumours. However, despite such clear-cut correlations between chromosomal instability and cancer, just how the initial chromosome segregation errors arise and are selected for, in order to set a cell on the long journey to transformation, have not been clear. There was therefore great interest in Sarah McClelland's (*London, UK*) account of the loss of specific chromosomes following exposure to distinct stresses. Stephen Taylor (*Manchester, UK*) then described how absolutely chaotic the divisions of cancer cells can be. His heroic establishment of a bank of primary patient ovarian cancer isolates revealed such abnormal spindle structures and chromosome complements that it is hard to understand just how these tumours can survive. However, as these lethal tumours clearly do survive repeated rounds of completely chaotic divisions, Taylor suggested that we may have to radically re-think some of the strategies for developing novel therapies based on cell cycle manipulation of tumours because mitotic perturbation may actually help, rather than hinder tumours.

Some hope for new avenues for therapy were provided by the remaining two talks in this session. Renata Basto (*Paris*) described how ovarian cancer patients whose tumours exhibited an abnormally high numbers of centrosomes responded better to therapy than those where centrosome numbers were closer to normal. Franz Meitinger's (*San Diego, USA*) discovery of TRIM37 as a factor that promoted the oligomerisation of the centrosome duplication regulating Polo kinase 4 (Plk4), led him to the realisation that TRIM37 is in a chromosomal region that is frequently amplified in a range of cancers. Strikingly, cell lines derived from such cancers harbouring the amplification are exquisitely sensitive to the Plk4 inhibitor centrinone.

The meeting was then drawn to a close in the fine style with which it began as Prof. Geneviève Almouzni (*Paris*) gave a brilliant plenary lecture. She described her exquisite studies of the means by which histone chaperones support the specialised identity of the centromeres. Her fascinating talk bridged the scales from specific structural studies of histone remodelling through to the impact upon development of perturbations in the control of centromeric identity.

The ebullient atmosphere at the banquet and heartfelt thanks from many attendees suggested that this 15th meeting in the series on cell cycle controls maintained the tradition of being, by far, the most engaging and stimulating meeting in cell cycle field. Despite many years of insightful research, entirely new insights and concepts continue to emerge. Particularly important insights into the mitotic cycle revealed at this meeting include the revelation of an entirely new route to enter the cycle, two fascinating leads into the means by which apoptosis maybe triggered by extended cell cycle arrest, an insight into the mysterious phenomenon of mitotic slippage, a major rethink of how to view cell cycle controls of primary tumours and extended complexity to Mad2's role in cell cycle control. The complexity of cohesin control throughout the meiotic cycle seems to know no boundaries, with more and more kinases are being linked to a variety of phosphatases in redundant controls that ensure that this most important of divisions is executed with the highest fidelity. There can be no doubt that the journey towards an understanding of cell cycle control and execution is a long way from finished, yet it remains as enthralling as it was at the first CJM Cell Cycle conference at Roscoff in 1988.

A final point to note is our gratitude to the *European Molecular Biology Organisation* and *Company of Biologists* for their support of the plenary talks, a generous grant from *Fondation ARC pour la recherche sur le cancer* to support Session X and funds from the journal *PLOS Biology* towards the opening and closing drinks receptions.

Le soutien financier généreux, et le soutien logistique du CNRS combinés au cadre superbe de la Station Biologique de Roscoff ont fait de la conférence Jacques Monod "Contrôle de l'exécution du cycle cellulaire en mitose et méiose" du 8 au 12 avril 2019, un succès retentissant. Le président scientifique (Iain Hagan) et la vice-présidente (Katja Wassmann) ont pu compléter les présentations des conférenciers invités avec des conférenciers supplémentaires sélectionnés parmi un très grand nombre de résumés. Ceci a créé un continuum passionnant entre les thématiques des différentes sessions. L'équilibre des genres parmi les présentateurs, avec 21 femmes et 23 hommes, a représenté un échantillonnage des différentes étapes de la carrière au niveau internationnal, avec des doctorants, des post-doctorants, des professeurs au début de leur carrière, et des directeurs d'équipe, dans le domaine respecté du cycle cellulaire. Les présidents de séance sélectionnés parmi les autres résumés soumis ont permis une exposition maximale pour promouvoir les interactions. Deux sessions de posters très dynamiques et l'excursion à l'île de Batz ont été l'occasion de discussions prolongées. La science de pointe passionnante présentée à cette conférence a été guidée par certaines technologies très novatrices qui ont fait l'objet de discussions passionnées. La qualité de la science, l'enthousiasme suscité par les progrès dans le domaine, le cadre magnifique et le soutien organisationnel de Mme Babic en ont fait un congrès formidable.

Iain Hagan & Katja Wassmann 11th May 2019