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AGENDA AND ABSTRACTS OF SATELLITE SYMPOSIA

Tuesday, 18 June 2013 from 13:00 to 15:00 h (room B II)

PHARMACYCLICS, INC./JANSSEN PHARMACEUTICALS
ADVANCEMENTS IN THE TREATMENT OF B-CELL MALIGNANCIES
Chair: B.D. Cheson, Washington DC (USA)

INTRODUCTION
B.D. Cheson, Washington DC (USA)

B-CELL RECEPTOR PATHWAY INHIBITORS—RATIONALE AND POTENTIAL
J. Gribben, London (UK)

POTENTIAL FOR ELIMINATING CHEMOTHERAPY IN INDOLENT NHL
B.D. Cheson, Washington DC (USA)

THE CHANGING LANDSCAPE IN CHRONIC LYMPHOCYTIC LEUKEMIA
S. O'Brien, Houston, TX (USA)

NOVEL APPROACHES IN MANTLE CELL LYMPHOMA
A. Goy, Hackensack, NJ (USA)

Tuesday, 18 June 2013 from 16:00 to 18:00 h (room B I)

Supported by an unrestricted educational grant from
GILEAD SCIENCES, INC.

PI3 KINASE δ INHIBITORS AND THEIR ROLE IN LYMPHOID MALIGNANCIES
Chair: F. Cavalli, Bellinzona (Switzerland)

A THERAPEUTIC QUANDARY: 17P DELETION IN PATIENTS WITH LOW TUMOR BURDEN AND POOR CYTOGENETICS
S. O'Brien, Houston, TX (USA)

OPTIMAL PHARMACOTHERAPEUTIC MANAGEMENT OF CHRONIC LYMPHOCYTIC LEUKEMIA: BEST UPFRONT TREATMENT, CONSIDERATIONS IN THE ELDERLY
P.L. Zinzani, Bologna (Italy)

TARGETING SIGNAL TRANSDUCTION: TURNING KINASES INTO EFFECTIVE DRUGGABLE TARGETS
M. Hallek, Cologne (Germany)

MINIMAL RESIDUAL DISEASE: IS RELAPSE INEVITABLE WITH CHRONIC LYMPHOCYTIC LEUKEMIA?
S.E. Coutre, Stanford, CA (USA)

CLINICAL ACTIVITY OF PI3 KINASE δ INHIBITORS IN LYMPHOID MALIGNANCIES
I.W. Flinn, Nashville, TN (USA)

DOI: 10.1002/hon

Tuesday, 18 June 2013 from 16:00 to 18:00 h (room B II)

PFIZER ONCOLOGY

TREATING AGGRESSIVE LYMPHOMAS: BETWEEN GUIDELINES, CLINICAL TRIALS AND REAL LIFE
Chair: M. Ghielmini, Bellinzona (Switzerland)

GUIDELINES FOR AGGRESSIVE LYMPHOMAS: A COMPARISON OF THE AVAILABLE ONES
A. Lopez-Guillermo, Barcelona (Spain)

DLBCL: A YOUNGER PATIENT WITH A HIGH IPI SCORE
P.W.M. Johnson, Southamptom (UK)

MCL: A PATIENT IN RELAPSE AFTER AUTOLOGOUS TRANSPLANT
M. Dreyling, Munich (Germany)

PTCL-NOS: 1st LINE TREATMENT STRATEGY IN AN ELDERLY PATIENT WITH ADVANCED DISEASE
F. D' Amore, Aarhus (Denmark)

FINAL DISCUSSION & CLOSING REMARKS
M. Ghielmini, Bellinzona (Switzerland)

DOI: 10.1002/hon

Tuesday, 18 June 2013 from 19:00 to 21:00 h (room A)

F. HOFFMANN-LA ROCHE LIMITED

INNOVATIONS IN ANTIBODY THERAPY FOR LYMPHOID MALIGNANCIES

Chair: F. Cavalli, Bellinzona (Switzerland)

CHAIR'S WELCOME: 50 YEARS IN ONCOLOGY

F. Cavalli, Bellinzona (Switzerland)

NEW APPROACHES TO ANTIBODY DELIVERY

G. Salles, Lyon (France) and N. Crosbie, Plymouth (UK)

PHARMACOKINETIC AND CLINICAL PARAMETERS IN SUBCUTANEOUS THERAPY

A. Davies, Southampton (UK)

IMPLICATIONS OF SUBCUTANEOUS THERAPY FOR PHARMACOECONOMICS, HEALTHCARE PROVIDERS AND PATIENTS

J.V. Pinto Neto, Brasilia (Brasil)

PANEL DISCUSSION AND Q&A

All faculty

NEW OPTIONS IN ANTIBODY THERAPY

G. Cartron, Montpellier (France)

ADDRESSING UNMET MEDICAL NEEDS IN CHRONIC LYMPHOCYTIC LEUKAEMIA

M. Hallek, Cologne (Germany)

PANEL DISCUSSION AND Q&A

All faculty

CHAIR'S CLOSE

F. Cavalli, Bellinzona (Switzerland)

DOI: 10.1002/hon

Tuesday, 18 June 2013 from 19:00 to 21:00 h (room B I)

Symposium organized and funded by
CTI Life Sciences

CURRENT PERSPECTIVE ON THE TREATMENT OF RELAPSED/REFRACTORY AGGRESSIVE B-CELL NHL
Chair: M. Ghielmini, Bellinzona (Switzerland)

INTRODUCTION
M. Ghielmini, Bellinzona (Switzerland)

TREATMENT OF RELAPSED/REFRACTORY AGGRESSIVE B-CELL NHL: UPDATED ESMO GUIDELINES AND PRACTICAL CONSIDERATIONS
M. Ghielmini, Bellinzona (Switzerland)

ANTHRACYCLINE-MEDIATED CARDIOTOXICITY: RECENT RESEARCH
P.L. Zinzani, Bologna (Italy)

RECENT DEVELOPMENTS IN THE TREATMENT OF RELAPSED/REFRACTORY AGGRESSIVE B-CELL NHL
R. Pettengell, London (UK)

PERSPECTIVES ON TREATMENT OPTIONS ILLUSTRATED THROUGH CASE STUDIES
A. Gaiger, Wien (Austria)

Q&A
M. Ghielmini, Bellinzona (Switzerland)

CONCLUSIONS AND CLOSE
M. Ghielmini, Bellinzona (Switzerland)

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Tuesday, 18 June 2013 from 19:00 to 21:00 h (room B II)

prIME Oncology (*supported by an unrestricted educational grant from* Takeda & MILLENNIUM: THE TAKEDA ONCOLOGY COMPANY)

NEW OPPORTUNITIES AND NEW CHALLENGES IN IMPROVING OUTCOMES FOR PATIENTS WITH CD30+ HEMATOLOGIC MALIGNANCIES

Chair: T. Illidge, Manchester (UK)

WELCOME AND INTRODUCTION

T. Illidge, Manchester (UK)

RELAPSED/REFRACTORY CD30+ HEMATOLOGIC MALIGNANCIES: THE SCOPE OF THE PROBLEM AND EVOLVING SOLUTIONS

T. Illidge, Manchester (UK)

CASE #1: TRANSPLANT NAÏVE RELAPSED/REFRACTORY HODGKIN LYMPHOMA (R/R HL)

A. Engert, Cologne (Germany)

Q & A

CASE #2: RELAPSED/REFRACTORY ANAPLASTIC LARGE CELL LYMPHOMA (R/R ALCL)

C. Gisselbrecht, Paris (France)

Q & A

CASE #3: OPPORTUNITIES TO IMPROVE OUTCOMES: A LOOK TO THE FUTURE

A. Younes, New York, NY (USA)

Q & A

CLOSING COMMENTS

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Wednesday, 19 June 2013 from 19:00 to 21:00 h (room A)

MUNDIPHARMA INTERNATIONAL LTD.

MAKING SENSE OF THE RAPIDLY CHANGING WORLD OF LYMPHOMA

Chairs: M. Ghielmini, Bellinzona (Switzerland) and M. Czuczman, Buffalo, NY (USA)

INTRODUCTION: WHAT DO WE KNOW NOW, WHAT DO WE NEED TO KNOW NEXT?

M. Ghielmini, Bellinzona (Switzerland)

THE LYMPHOMA CELL—WHAT USEFUL SECRETS IS IT NOW REVEALING?

R. Dalla-Favera, New York, NY (USA)

CASE STUDIES—CAN WE APPLY 'PERSONALISED MEDICINE' IN THE INDOLENT LYMPHOMA CLINIC TODAY?

M. Rummel, Giessen (Germany)

A PEAK THROUGH THE LOOKING GLASS—WHAT DOES THE FUTURE PROMISE?

B.D. Cheson, Washington D.C. (USA)

CLOSING REMARKS FROM THE CHAIR—MY HOPES AND FEARS FOR PATIENTS WITH LYMPHOMA

M. Czuczman, Buffalo, NY (USA)

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Wednesday, 19 June 2013 from 19:00 to 21:00 h (room B)

CELGENE CORPORATION

FROM BENCH TO BEDSIDE—TRANSLATING PRECLINICAL RESEARCH INTO CLINICAL REALITY

Chair: B.D. Cheson, Washington DC (USA) and J. Gribben, London (UK)

WELCOME AND INTRODUCTION
Chair

WHAT IS THE ROLE OF CEREBLON IN THE MECHANISM OF ACTION OF IMIDS®?
R. Chopra, Summit, NJ (USA)

HOW DOES GENOMICS CONTRIBUTE TO OUR UNDERSTANDING OF B-CELL LYMPHOMA PATHOGENESIS AND THE IDENTIFICATION OF THERAPEUTIC TARGETS?
L. Staudt, Bethesda, MD (USA)

THE LYMPHOMA-MICROENVIRONMENT INTERACTION AS TARGET FOR THERAPY
U. Jäger, Wien (Austria)

TO WHAT EXTENT CAN PRECLINICAL DATA CONTRIBUTE TO THE DESIGN OF NOVEL TREATMENT CONCEPTS IN CLL?
J. Gribben, London (UK)

WHY SHOULD WE EXPLOIT THE SYNERGY OF LENALIDOMIDE AND ANTI-CD20 ANTIBODY THERAPY AS A BACKBONE FOR FUTURE TREATMENT COMBINATIONS IN NHL?
Chair

CONCLUSION
B.D. Cheson, Washington DC (USA)

SS1

WHAT IS THE ROLE OF CEREBLON IN THE MECHANISM OF ACTION OF IMIDS®?

Rajesh Chopra (USA)

Although the pathogenesis of lymphoid malignancies is a multistep process, the range of histologies typically reflect features of the tissue in which the primary genetic lesion occurred [1]. This observation may explain the dependence of lymphoid tumours on their microenvironment for survival and proliferation and suggests that the microenvironment is a valid therapeutic target [1,2]. Knowledge of molecular pathology can inform us on the mode of action of therapeutic agents and may direct the development of novel compounds [1]. Lenalidomide and other immunomodulatory drugs (IMiDs®, Celgene Corporation, Summit, NJ, USA) are indicated for use in MM and have demonstrated significant activity in a number of other lymphoid malignancies by acting on both the tumour cells and components of the microenvironment [3,4]. One protein target of the IMiDs® is cereblon, a substrate receptor within an E3 ubiquitin ligase complex. Binding of lenalidomide and other IMiD® drugs to cereblon is hypothesized to alter the function of the cereblon-containing E3 ubiquitin ligase [5,6]. Depending on the cellular context and type of IMiD® drug, there is either inhibition or activation of E3 ligase activity, resulting in either substrate accumulation or proteasomal degradation and subsequent loss of protein homeostasis [5-7]. It has been hypothesized that cereblon mediates the disease modifying immunomodulatory and microenvironmental effects observed with IMiDs® [5,6,8], and recent studies have examined cereblon as a potential biomarker for clinical outcomes. However, current evidence is limited by the lack of standardized assays for this protein [9]. Development of a validated cereblon assay and incorporation of this assay into prospective clinical trials is needed to determine the clinical utility of measuring cereblon and predicting treatment outcomes in lymphoma and myeloma patients treated with IMiDs®. In addition, further research on cereblon-IMiD® interactions may help in the design of novel compounds.

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SS2

HOW DOES GENOMICS CONTRIBUTE TO OUR UNDERSTANDING OF B-CELL LYMPHOMA PATHOGENESIS AND THE IDENTIFICATION OF THERAPEUTIC TARGETS?

Louis Staudt (USA)

The destabilization of mechanisms required for the differentiation and activation of B-cells and for the diverse repertoire of antibodies they produce are largely responsible for the formation of B-cell lymphomas [1]. The subtype of the resulting tumour depends on where and when these subversive events occur to block B-cell differentiation and have classically been defined morphologically. However, the marked heterogeneity of prognosis and response to therapy that is observed even within discrete morphologic categories suggests that pathogenetically distinct disease entities are not identified by traditional diagnostics [2]. Gene expression profiling techniques can identify molecularly distinct subtypes of cancer and the identification of potential targets for the rational design of novel therapeutic approaches [1–4]. Gene expression profiling has been used to refine classification and prognostication in a number of adult and paediatric forms of NHL including mantle-cell and diffuse large-B-cell (DLBCL) lymphoma [5–8]. DLBCL has thus been sub-classified into three groups (germinal centre B-cell-like (GCB) DLBCL, activated B-cell-like (ABC) DLBCL and primary mediastinal B-cell lymphoma with genetic signatures suggesting that they arise during different stages of B-cell development [2,9]. The ABC DLBCL accounts for approximately 30% of DLBCL cases and is the least likely to respond to conventional therapy [2,13]. It is characterized by chronic active B-cell receptor signalling (not seen in other DLBCL subtypes) that leads to constitutive activation of NF-κB [14,15]. Blockade of B-cell receptor signalling with agents like ibrutinib has been shown to be particularly effective in patients with ABC DLBCL [16]. The activity of lenalidomide was also shown to be higher in patients with non-GCB DLBCL than GCB-like DLBCL [17], and preclinical synergy between ibrutinib and lenalidomide suggests a potential therapeutic strategy for these subtypes [18]. Activating mutations in the adapter protein MYD88 and its associated kinases IRAK1 and IRAK4 have also been shown to be essential to ABC DLBCL survival [13], and inhibitors of IRAK4 are in development [13,19]. Ongoing trials will help determine how to best integrate the information gained from gene expression profiling into clinical trial designs and clinical practice.

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SS3
THE LYMPHOMA-MICROENVIRONMENT INTERACTION AS TARGET FOR THERAPY

Ulrich Jäger (Austria)

Although the primary treatment target in oncology is typically the tumour itself, there are potential opportunities to target other cell types in many malignancies due to the crosstalk between tumour cells and their environment. In chronic lymphocytic leukaemia (CLL), the tumour microenvironment is critical for CLL-cell survival and the PI3K/Akt pathway seems to play an important role in this interaction [1]. The microenvironment is also involved in several non-Hodgkin lymphomas [2,3] where, for example, the platelet-derived growth factor receptor supports growth of anaplastic large cell lymphomas [2]. Interestingly, in some cases, like for Hodgkin lymphoma, the percentage of tumour cells is about 0.1%–10% of the total cellular infiltrate [4], indicating that there is considerable interaction with bystander cells.

Therefore, it is important not only to consider tumour cells in isolation of surrounding cells (e.g. T-cells, macrophages and mesenchymal stem cells/stromal cells) but to study their interactions to identify potential targets for novel therapies. *In vitro* models, in which tumour cells are cultured on a layer of stromal cells, are used to investigate these interactions and the effect of drugs on the tumour cell and their microenvironment. Several compounds, such as pan-PI3K inhibitors, platelet-derived growth factor receptor inhibitors (e.g. imatinib) and lenalidomide have shown promising effects on the microenvironment, interfering with survival of CLL and lymphoma cells, which may prove to be clinically relevant [1,3,5,6]. Further research in this field is needed to elucidate the true potential of agents targeting the microenvironment in the treatment of lymphoma and leukaemia, either as monotherapy or in combination.

References
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SS4
TO WHAT EXTENT CAN PRECLINICAL DATA CONTRIBUTE TO THE DESIGN OF NOVEL TREATMENT CONCEPTS IN CHRONIC LYMPHOCYTIC LEUKEMIA?

John Gribben (UK)

Chronic lymphocytic leukemia (CLL) exhibits profound immune deficiency associated with defects in T-cell function, resulting in failure of antitumour immunity and susceptibility to recurrent infections [1,2]. Chemotherapy-based regimens, adapted according to patient fitness and other characteristics, are the current standards of care in both the front-line and relapsed/refractory settings. Despite recent improvements in outcomes, such regimens are not curative and typically exacerbate the inherent immunosuppression [3].

Preclinical research has identified T-cell defects in CLL that may potentially be targeted by novel agents. For example, in comparison with normal controls, changes in the expression of cytoskeletal genes in T-cells from CLL patients manifest as defective immunologic synapse formation with antigen-presenting cells, which was improved *in vitro* by treatment with the immunomodulatory drug lenalidomide [4]. Further work demonstrated that dysregulated Rho-GTPase signalling is a major target pathway for understanding and repairing tumour cell-induced T-cell defects [5]. It was recently demonstrated that the T-cell compartment of CLL patients is skewed in favour of antigen-experienced cells with an increased expression of some, but not all, markers of T-cell exhaustion [2]. These cells showed decreased proliferation and capacity for killing target cells *ex vivo*.

Restoration of T-cell-mediated immune defects thus represents an avenue for future clinical research paradigms [1]. Examples may include enhancing adaptive immunity via CD40 ligand gene therapy, adoptive transfer of T-cells bearing chimeric antigen receptors, targeting inhibitory receptors like PD-1 and immunomodulatory agents such as lenalidomide that act on the microenvironment, T-cells and CLL cells [1,6,7]. In conclusion, therapeutic strategies promoting immune restoration may improve outcomes for patients with CLL in the future.

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SS5

WHY SHOULD WE EXPLOIT THE SYNERGY OF LENALIDOMIDE AND ANTI-CD20 ANTIBODY THERAPY AS A BACKBONE FOR FUTURE TREATMENT COMBINATIONS IN NHL?

Bruce D. Cheson (USA)

Rituximab-based chemo-immunotherapy regimens are established standards of care for lymphoma, but many patients relapse and some are unable to tolerate intensive chemotherapy. Thus, novel treatment strategies are needed. Lenalidomide is active when given as monotherapy in patients with relapsed/refractory lymphomas, producing response rates of 23%–49%, varying with histologic subtype[1–4]. Preclinical evidence suggests that lenalidomide enhances the antitumor effects of rituximab, including induction of apoptosis and antibody-dependent cell mediated cytotoxicity [5–7]. In patients with relapsed/refractory indolent lymphoma, the combination of lenalidomide and rituximab (the R^2 regimen) has generated response rates of 52%–73% and median event-free or progression-free survival of 1–2 years [4,8–10]. In a recently reported randomized trial comparing lenalidomide with or without rituximab in patients with recurrent follicular lymphoma, R^2 therapy was associated with a higher response rate (75% vs. 49%, including complete response rates of 32% and 13%, respectively), which translated into a significantly longer median event-free survival (2.0 vs. 1.2 years; $p = 0.0063$) [4]. More modest and less durable response rates (33%–35%) have been reported when R^2 was used in patients with relapsed/refractory diffuse large B-cell lymphoma [11,12]. In patients with untreated indolent lymphoma, the response rate following treatment with R^2 has been >90% with a complete response rate that exceeded 60% [13,14]. The primary adverse event associated with R^2 therapy is myelosuppression, including neutropenia and thrombocytopenia. Current research is focusing on developing novel treatment regimens based on R^2. Encouraging results have been achieved with the combination of R^2 and chemotherapy agents like bendamustine [15] or regimens, such as CHOP [16,17]. Other studies are evaluating the addition of novel targeted therapies to R^2 with the aim of rationally developing better-tolerated, 'chemotherapy-free' regimens [18]. Several novel agents with demonstrated activity and acceptable safety profiles are good candidates for combination with R^2; these agents include idelalisib, IPI-145 and ibrutinib, among others. Additional research to assess R^2-based therapy in non-Hodgkin lymphoma is warranted.

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Thursday, 20 June 2013 from 19:00 to 21:00 h (room A)

PRIME ONCOLOGY

OPTIMIZING OUTCOMES FOR INDOLENT NON-HODGKIN LYMPHOMA AND MANTLE CELL LYMPHOMA

Chair: J.O. Armitage, Omaha, NE (USA)

WELCOME AND INTRODUCTION

J.O. Armitage, Omaha, NE (USA)

CONTEMPORARY THERAPY FOR INDOLENT NON-HODGKIN LYMPHOMA (NHL) AND MANTLE CELL LYMPHOMA (MCL): WHERE ARE WE NOW?

J.O. Armitage, Omaha, NE (USA)

FRONT-LINE THERAPY OF INDOLENT NHL AND MCL: CONSIDERATION OF NEW THERAPEUTIC STRATEGIES

S. Stilgenbauer, Ulm (Germany)

CONSOLIDATION THERAPY FOR INDOLENT NHL: READY FOR PRIME TIME?

T. Illidge, Manchester (UK)

Q & A

RELAPSED INDOLENT NHL AND MCL: HOW CAN WE IMPROVE PATIENT OUTCOMES

G. Salles, Lyon (France)

Q & A

FACULTY PANEL DISCUSSION—'HAS THE TIME COME TO REVISE OUR APPROACH TO INDOLENT NHL?'

All Faculty

SYMPOSIUM 'PEARLS' AND CLOSING COMMENTS

J.O. Armitage, Omaha, NE (USA)

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Thursday, 20 June 2013 from 19:00 to 21:00 h (room B)

MUNDIPHARMA INTERNATIONAL LTD.

CAN WE ERADICATE THE CNS DISEASE IN LYMPHOMA?

Chairs: C. Gisselbrecht, Paris (France) and E. Thiel, Berlin (Germany)

SHOULD INTRATHECAL THERAPY BE PART OF TREATMENT FOR PATIENTS WITH AGGRESIVE NHL?

E. Thiel, Berlin (Germany)

DETECTING DISEASE, IDENTIFYING PATIENTS AT RISK AND DETERMINING BENEFIT OF TREATMENT

L. Martin, Salamanca (Spain)

TREATMENT AND OUTCOME OF OCCULT LEPTOMENINGEAL AGGRESSIVE B-CELL LYMPHOMA

W. Wilson, Bethesda, MD (USA)

CONCEPTS OF EARLY TREATMENT TO ERADICATE SECONDARY CNS LYMPHOMA

S.H. Bernstein, Rochester, MN (USA)

SECONDARY CNS LYMPHOMA—LONG TERM REMISSIONS WITH RISK ADAPTED THERAPY

A. Korfel, Berlin (Germany)

A PHASE 2 STUDY WITH RITUXIMAB AND DEPOCYTE IN COMBINATION WITH C5R PROTOCOL IN PCNSL: A LYSA TRIAL

H. Ghesquieres, Lyon (France)

CNS DISEASE IN LYMPHOMA—CONCEPTS FOR CLINICAL PRACTICE

C. Gisselbrecht, Paris (France)

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Friday, June 21, 2013 from 18:45 to 19:45 (room B)

IOSI - ONCOLOGY INSTITUTE OF SOUTHERN SWITZERLAND
(*Supported by an unrestricted educational grant from MUNDIPHARMA INTERNATIONAL LTD.*)
PROS AND CONS OF FRONT LINE R-CHOP PLUS MAINTENANCE IN FL
Chair: M. Ghielmini, Bellinzona (Switzerland)

WELCOME AND INTRODUCTION
M. Ghielmini, Bellinzona (Switzerland)

DEBATE 1: DOES BR REPLACE CHOP-R IN FRONT LINE FL?
M. Rummel, Giessen (Germany) vs. G. Salles, Lyon (France)

DEBATE 2: IS R-CHOP THE STANDARD FOR GRADE 3 FL?
B.S. Kahl, Madison, WI (USA) vs. A. Davies, Southampton (UK)

DEBATE 3: DO WE NEED MAINTENANCE AFTER FRONT LINE TREATMENT?
G Salles, Lyon (France) vs. B.D. Cheson, Washington D.C. (USA)

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