

**67th ASH Annual Meeting 2025 (Orlando, USA) –
Overview of CML related sessions**

Timings in Eastern Standard Time (EST)

Time slots	Sessions
Satellite Symposia	
<p>Dec 5 (Friday)</p> <p>11.00 a.m. – 2.00 p.m.</p> <p>Hyatt – Regency Ballroom R</p>	<p>Satellite Symposium: A Master Class in CP-CML: New Agents, Treatment Goals, and Clinical Challenges</p> <p>This program is supported by an educational grant from Novartis.</p> <p>Chair: Jerald R. Radich (Fred Hutchinson Cancer Center, Seattle, USA)</p> <p>Speakers:</p> <ul style="list-style-type: none"> • Neil Shah (San Francisco, USA) • Douglas B. Smith (Baltimore, USA) <p>Program:</p> <p>This symposium will review the latest advancements in chronic phase chronic myeloid leukemia (CP-CML) management. This activity will feature experts discussing cases illustrating clinical applications of the latest data and strategies to optimise the diagnosis, treatment, and management of CP-CML.</p> <p>Participants will be able to engage directly with leading experts through multiple discussion session and present questions and real-world cases for personalised guidance.</p> <p>Navigate the evolving evidence for individualised frontline CP-CML treatment, optimise response monitoring strategies, and gain insights into current recommendations for managing treatment intolerance and failure.</p>

Time slots	Sessions
Education Spotlight Sessions	
Dec 7 (Sunday) 4.30 – 5.45 p.m. OCCC – W414AB	<p>Education Spotlight Session: The changing face of chronic myelogenous leukemia</p> <p>Chair: Gabriela Soriano Hobbs (Massachusetts General Hospital)</p> <p>Speakers:</p> <ul style="list-style-type: none"> • Jorge Cortes (Augusta, USA): Frontline Treatment Selection for CML in 2025 • Francois-Xavier Mahon (Bordeaux, France): Can I Stop Taking My TKI Yet?
Oral Abstract Sessions	
Dec 6 (Saturday) 9.30 – 11.00 a.m. OCCC – Valencia Room W415D	<p>Oral Abstract Session: Chronic Myeloid Leukemia: Clinical and Epidemiological: Decoding the molecular drivers of response and resistance</p> <p>Program:</p> <ul style="list-style-type: none"> • 9.30 a.m.: Distinct patterns of mutant ASXL1 over time and their implications for treatment failure (TF) and BCR::ABL1 mutation development in newly diagnosed patients with chronic myeloid leukemia in chronic phase (CML-CP) treated with asciminib (ASC) vs investigator-selected tyrosine kinase inhibitors (IS-TKI) in the ASC4FIRST study (Branford et al.) (ID 73) • 9.45 a.m.: A single-cell atlas of diagnostic bone marrow to uncover the origins of CML relapse following therapy cessation (Krishnan V et al. (ID 74) • 10.00 a.m.: Somatic mutations at diagnosis in patients with chronic Phase CML receiving frontline

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	<p>imatinib are associated with a higher rate of treatment failure: First analysis from the international CML foundation (iCMLf) genomics alliance on the harmony platform (Branford S et al.) (ID 75)</p> <ul style="list-style-type: none"> • 10.15 a.m.: A machine learning approach identifies a transcriptomic signature predicting treatment-free remission in chronic myeloid leukemia (Alcazer V et al.) (ID 76) • 10.30 a.m.: Molecular landscape and clonal evolution in minor versus major BCR::ABL1 chronic myeloid leukemia under tyrosine kinase inhibition : A Study from the french group fi-LMC (Podvin B et al.) (ID 77) • 10.45 a.m.: DNA-based MRD monitoring enhances risk stratification during TKI dose reduction in CML: Evidence from the clinical trial (Machova Polakova K et al.) (ID 78)
<p>Dec 6 (Saturday) 4.00 – 5.30 p.m. OCCC – W414AB</p>	<p>Oral Abstract Session: Myeloproliferative Syndromes and Chronic Myeloid Leukemia: Basis and Translational: Drivers, vulnerabilities and resistance in CML and MPN</p> <p>Program:</p> <ul style="list-style-type: none"> • 4.00 p.m.: Bone marrow endothelial progenitor cells drive TKI resistance in CML via SEMA4D-plexin B1-mediated metabolic reprogramming (Yu S et al.) (ID 355) • 4.15 p.m.: Single cell analysis of chronic myeloid leukemia bone marrow from time of TKI discontinuation reveals stem cell alterations associated with recurrence versus treatment free remission (Freeland T et al.) (ID 356)
<p>Dec 8 (Monday) 2.45 – 4.15 p.m. OCCC - Chapin Theater (W320)</p>	<p>Oral Abstract Session: Chronic Myeloid Leukemia: Clinical and Epidemiological: Therapeutic agents to enhance patient outcomes</p> <p>Program:</p> <ul style="list-style-type: none"> • 2.45 p.m.: CARDINAL: A Phase 1 study of TERN-701, a novel investigational allosteric BCR::ABL1 inhibitor for patients with previously treated CML (Jabbour E et al. (ID 901) • 3.00 p.m.: Efficacy of tgrx-678, a potent BCR::ABL1 allosteric inhibitor, in CML-CP and CML-AP

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	<p>patients harboring T315I mutation: Results from a phase 1 study (Jiang Q et al.) (ID 902)</p> <ul style="list-style-type: none"> • 3.15 p.m.: Improved long-term tolerability and efficacy of asciminib-based combination therapies in newly diagnosed CML patients - the fascination trial. (Ernst T et al.) (ID 903) • 3.30 p.m.: Management and outcomes of patients diagnosed with chronic myeloid leukemia in blast Phase: A multicenter analysis by the H Jean Khoury cure CML consortium (Jain A et al.) (ID 904) • 3.45 p.m.: International, prospective study comparing nilotinib versus imatinib with early switch to nilotinib to obtain sustained treatment-free remission (TFR) in patients with chronic myeloid leukemia: The TFR rate at the end of follow-up (Pane F et al.) (ID 905) • 4.00 p.m.: Asciminib (ASC) in chronic myeloid leukemia in chronic Phase (CML-CP): Efficacy and safety results of the Phase 2 ASC2ESCALATE trial in the cohort of patients (pts) with 1 prior tyrosine kinase inhibitor (TKI) (Cortes J et al.) (ID 906)
Poster Presentations (for in-person participants)	
<p>Dec 6 (Saturday)</p> <p>5.30 – 7.30 p.m.</p> <p>OCCC - West Halls B3-B4</p>	<p>Poster Abstract Session: Myeloproliferative Syndromes and Chronic Myeloid Leukemia: Basic and Translational: Poster I</p> <p>Poster:</p> <ul style="list-style-type: none"> • Differential impacts of Lama4 deficient microenvironment on fitness of BCR::ABL1-expressing cells during chronic myeloid leukemia initiation and progression (Mansson A et al) (ID 1974) • Immunophenotypic and cytokine profiling reveal robust immunomodulation after dasatinib treatment and identify a subgroup with rapid and deep molecular response in CML patients: A multi-center prospective study (Cho SF et al) (ID 1975) • Immune Dysregulation and State-Transition Transcriptomic Signatures Underlying Pediatric Chronic Myeloid Leukemia Pathogenesis (Ybarra T et al) (ID 1982) • A collective non-mutational transition governs the development of chronic myeloid leukemia (Van Etten R et al) (ID 1984)

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	<ul style="list-style-type: none"> Immune deconvolution from whole transcriptomic data suggests expansion of exhausted T-cells and natural killer cells following tyrosine kinase inhibitor therapy in chronic myeloid leukemia patients (Morgenstern Y et al) (ID 1991)
<p>Dec 6 (Saturday)</p> <p>5.30 – 7.30 p.m.</p> <p>OCCC - West Halls B3-B4</p>	<p>Poster Abstract Session: Chronic Myeloid Leukemia: Clinical and Epidemiological: Poster I</p> <p>Poster:</p> <ul style="list-style-type: none"> Identification of predictive protein signatures of treatment-free remission in patients with chronic myeloid leukaemia (Nosratzadeh I et al) (ID 1992) ASC4OPT 96-week results: Asciminib once or twice daily continues to be highly efficacious and demonstrates favorable safety in patients with chronic myeloid leukemia and suboptimal response, resistance or intolerance to two or more tyrosine kinase inhibitors (Breccia M et al) (ID 1993) Risk of secondary chronic myeloid leukemia in cancer survivors: A nationwide population-based study in Taiwan (Liu CJ et al) (ID 1994) Nanopore sequencing to detect BCR::ABL1 and associated genomic rearrangements in CML (Cross N et al) (ID 1995) Real-world cardiovascular risk of ponatinib in chronic Phase CML: Insights from a nationwide Korean claims-based cohort study (Lee JO et al) (ID 1996) Asciminib (ASC) demonstrates continued improvement in patient-reported outcomes (PROs) vs investigator-selected tyrosine kinase inhibitors (IS-TKIs) in newly diagnosed chronic myeloid leukemia (CML): ASC4FIRST week 96 analysis (Cortes J et al) (ID 1997) Chronic myelogenous leukemia: A real-world experience from a Los Angeles county safety net hospital (Zheng D et al) (ID 1998) Olverembatinib-mediated deep remission improves allogeneic stem cell transplantation outcome in

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	<p>patients with blast crisis chronic myeloid leukemia: First real-world practice report (Zhang X et al) (ID 1999)</p> <ul style="list-style-type: none"> • Somatic mutations in cancer-related genes in chronic myeloid leukemia patients receiving asciminib (Kuzmina E et al) (ID 2000) • What is the optimal threshold for aberrant lymphoblasts at diagnosis to predict lymphoid transformation in chronic myeloid leukemia? (Jiang Z et al) (ID 2001) • Predictors of successful tyrosine kinase inhibitor discontinuation in chronic myeloid leukemia: Long-term, real-world evidence on the role of faster molecular decline and duration of response (Lee S et al) (ID 2002) • Prognostic utility of the ELTS score in pediatric chronic myeloid leukemia: Is it a valid clinical tool? (Ranga S et al) (ID 2003) • Comparative impact of tyrosine kinase inhibitor dose-reduction on ability to achieve treatment-free remission among TKI-intolerant patients with chronic myeloid leukemia (Rebechi M et al) (ID 2004) • Long-term imatinib therapy leads to significant reduction in GFR (Mishra K et al) (ID 2005) • Is initial treatment with dasatinib for newly diagnosed chronic Phase chronic myeloid leukemia, followed by switching to imatinib for those that achieve MR3.0 at 12 months, an effective strategy? Results of the Phase 2 kinase inhibition with sprycel startup study (Browett P et al) (ID 2006) • Sequential monitoring using NGS allows detection of ABL1 KD mutations in patients with chronic Phase CML: Results from the prospective calls trial (Baymul I et al) (ID 2007) • Frontline tyrosine kinase inhibitor selection influences eventual treatment-free remission eligibility and sustained treatment-free remission: A registry analysis of patients treated at CML referral sites in Australia (Shanmunagathan N et al) (ID 2008) • Impact-CML: Indian multicentric data supporting safe pregnancy and molecular control in women with chronic myeloid leukemia (Malhotra P et al) (ID 2009)

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	<ul style="list-style-type: none"> Phase II study of clia (cladribine, idarubicin, cytarabine) plus tyrosine kinase inhibitor (TKI) in patients with chronic myeloid leukemia in myeloid blast Phase (Abdelmalek M et al) (ID 2010) Standard tyrosine kinase inhibitor dose versus dose de-escalation prior to treatment-free remission attempt in chronic myeloid leukemia: A single-center 25-year retrospective study (Pusung M et al) (ID 2011) Longitudinal analysis of first and subsequent-line tyrosine kinase inhibitor use in chronic myeloid leukemia: A 16-year single-center experience (Ali MM et al) (ID 2012)
<p>Dec 7 (Sunday) 6.00 – 8.00 p.m.</p> <p>OCCC – West Halls B3-B4</p>	<p>Poster Abstract Session: Myeloproliferative Syndromes and Chronic Myeloid Leukemia: Basic and Translational: Poster II</p> <p>Program:</p> <ul style="list-style-type: none"> FBXO3-mediated DUSP9 ubiquitination reprograms MAPK signaling to eradicate tyrosine kinase inhibitor-resistant CML stem cells (Li X et al) (ID 3754) Different kinetic behavior of molecular reduction by BCR::ABL1 transcript types in frontline treatment of chronic myeloid leukemia patients: A GIMEMA labnet CML network database survey. (Stagno F et al) (ID 3755) Allele-dependent activity of promoters of genes SLC22A4 and SLC22A5 encoding imatinib transporters in chronic myeloid leukemia (Burda P et al) (ID 3756) Therapeutic targeting of focal adhesion kinase (FAK) modulates oncogenic and immune pathways in myeloid progenitor cells expressing oncogenic Janus kinase 2-V617F. (Chary Nimmagadda S et al) (ID 3757) Evaluating reverse transcriptase efficiency in CML: Consequences for deep molecular response classification and treatment-free remission decisions (Machova Polakova K et al) (ID 3762) Emergence of new somatic mutations in CML patients optimally responding to tyrosine kinase inhibitor therapy: Proposal of long-term genomic monitoring (Morgenstern Y et al.) (ID 3767)

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<p>Dec 7 (Sunday) 6.00 – 8.00 p.m. OCCC - West Halls B3-B4</p>	<p>Poster Abstract Session: Chronic Myeloid Leukemia: Clinical and Epidemiological: Poster II</p> <p>Poster:</p> <ul style="list-style-type: none"> • Integrated machine learning, cosmic signatures, and AI-driven genomic profiling of highly heterogeneous blast crisis CML at single patient level resolution: Implication as a versatile risk stratification and precision oncology approach for refractory and relapsed cancers in AI/ML era (Iqbal Z et al) (ID 3769) • Whole genome sequencing reveals that the Philadelphia chromosome (Ph) can evolve and gain complex chromoplectic sequence rearrangements associated with tyrosine kinase inhibitor (TKI) resistance (Branford S et al) (ID 3770) • Preliminary safety and efficacy of ELVN-001, a selective active site inhibitor of BCR::ABL1, in patients with CML driven by atypical fusion transcripts (Hochhaus A et al) (ID 3771) • Survival and risk of second primary malignancies in patients with chronic myeloid leukemia in the era of tyrosine kinase inhibitors: A retrospective US population-level analysis (Pramanik D et al) (ID 3772) • Additional genetic abnormalities (AGA) and microhomology end-joining DNA repair (MMEJ) signatures influence the outcome of newly diagnosed chronic phase (CP) CML: Ancillary study of the Trial of imatinib after ponatinib induction (TIPI) (Nicolini F et al) (ID 3773) • Frontline nilotinib vs dasatinib in newly diagnosed chronic phase chronic myeloid leukemia: A propensity score analysis based on AIFA monitoring registries (Breccia M et al) (ID 3774) • Asciminib (ASC) in chronic myeloid leukemia in chronic Phase (CML-CP): Interim analysis (IA) efficacy and safety results of the Phase 2 ASC2ESCALATE trial in the cohort of newly diagnosed (1L) patients (pts) (Levy M et al) (ID 3775) • Flumatinib versus nilotinib as first-line therapy for chronic myeloid leukemia in chronic Phase: A propensity score-matched cohort study (Xu N et al) (ID 3776) • Exploring co-mutations in CML and ph(+) b-ALL with ABL1 variants: Correlations with survival and

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	<p>treatment response (Dykes K et al) (ID 3777)</p> <ul style="list-style-type: none"> • Ponatinib as a consolidation strategy for a second attempt at TKI discontinuation in chronic myeloid leukemia: Interim results from the restop Trial (Zaratiegui Vergara A et al) (ID 3778) • The efficacy and safety of switching to olverembatinib or continuing original TKI therapy in CML-CP patients treated with at least two prior TKIs: A prospective, multicenter, controll trial (Wen B et al) (ID 3779) • A randomized study of ropeginterferon in combination with bosutinib in newly diagnosed chronic myeloid leukemia (CML) patients in chronic Phase. the bosupep trial from the Nordic CML study group (Hjorth-Hansen H et al) (ID 3780) • Identification of factors associated with a high success rate of treatment-free remission in chronic myeloid leukemia: A multicenter study in Taiwan (Chen NC et al) (ID 3781) • Updated efficacy and safety of olverembatinib (HQP1351) as second-line therapy in patients (pts) with chronic Phase-chronic myeloid leukemia (CP-CML) (Weiming L et al) (ID 3782) • Early reduction of BCR-ABL at 3 months predicts deeper molecular remission in pediatric CML (Wang X et al) (ID 3783) • Changes in disease responses after discontinuation of tyrosine kinase inhibitors (TKI) for pregnancy in established chronic myeloid leukaemia (CML): Evidence for the basis of advice for women of child-bearing age (Chan J et al) (ID 3784) • Integrating cytokine profiles with clinical and molecular biomarkers to predict molecular relapse after TKI discontinuation in CML patients (Pavlovsky C et al) (ID 3785) • Treatment-free remission outcomes in pediatric chronic myeloid leukemia: Second-generation TKIs vs. Imatinib—a real-world multicenter study from China (Liang LX et al) (ID 3786) • Hematopoietic architecture drives biological and metabolic heterogeneity and predicts treatment-free remission outcomes in chronic myeloid leukemia (Vergez F et al) (ID 3787) • Olverembatinib (HQP1351) demonstrates efficacy vs. best available therapy (BAT) in patients (pts)

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	<p>with tyrosine kinase inhibitor (TKI)-resistant chronic-Phase chronic myeloid leukemia (CML-CP) in a registrational randomized Phase 2 trial: Up to 4-year follow-up including patients without T315I mutations (Jiang Q et al) (ID 3788)</p> <ul style="list-style-type: none"> Two decades of pregnancy outcomes in women with CML: Insights from a real-world single-center cohort (Gundeti S et al) (ID 3789)
<p>Dec 8 (Monday) 6.00 – 8.00 p.m. OCCC - West Halls B3-B4</p>	<p>Poster Abstract Session: Chronic Myeloid Leukemia: Clinical and Epidemiological: Poster III</p> <p>Poster:</p> <ul style="list-style-type: none"> Five-year interim analysis of multi-institutional collaborative study for estimating the persistence of treatment free remission in chronic myeloid leukemia after stopping tyrosine kinase inhibitor in Japan: J-SKI (Takahashi N et al) (ID 5545) Reattempt of tyrosine kinase inhibitor discontinuation after maintenance therapy with ponatinib in patients with chronic myeloid leukemia in the chronic phase: Result of JALSG CML RE-STOP219 study (Iriyama N et al) (ID 5546) XS003, an amorphous re-formulation of nilotinib, demonstrates improved dose linearity and minimal food effect, reducing the risk of QT-prolongation at fed conditions (Andersson P et al) (ID 5547) Beyond survival in chronic myeloid leukemia: A systematic review of secondary malignancies in the era of tyrosine kinase inhibitors (Rola G et al) (ID 5548) Improved long-term tolerability with asciminib (ASC) vs investigator-selected (IS) tyrosine kinase inhibitors (TKIs) in patients (pts) with newly diagnosed chronic myeloid leukemia in chronic phase (CML-CP): Week 96 exploratory analysis of the phase 3 ASC4FIRST trial (Larson R et al) (ID 5549) Predictive factors for successful treatment-free remission in patients with chronic myeloid leukemia: A pooled analysis (Haddad F et al) (ID 5550) ASXL1 and NOTCH1 mutations independently predict TKI treatment failure in chronic myeloid leukemia (Huang J et al) (ID 5551)

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	<ul style="list-style-type: none"> • High rates of deep molecular response (DMR) in patients (pts) with chronic myeloid leukemia in chronic Phase (CML-CP) who have not achieved dmr after ≥ 1 year of prior imatinib (IMA) in the asciminib (ASC) monotherapy arm of the Phase 2 ASC4MORE study (Cortes J et al) (ID 5552) • BCR::ABL1/GUSB halving times predict molecular response to asciminib at 18 months (Cross N et al) (ID 5553) • Resistance to imatinib of leukemia growth in non-marrow organs and eradication by dasatinib: Published cases and gene clues (Cunningham I et al) (ID 5554) • Genetic evolution in CML patients from diagnosis to TKI failure: An analysis of 121 paired diagnosis and treatment failure samples (Huang YJ et al) (ID 5555) • Long-term outcomes of CML patients undergoing hematopoietic stem cell transplantation in the era of tyrosine kinase inhibitors (Raychaudhuri S et al) (ID 5556) • SETD2 deficiency in chronic myeloid leukemia (CML) contributes to tyrosine kinase inhibitor (TKI) resistance and disease acceleration by enhancing genetic instability and rewiring cellular metabolism and might be a novel biomarker of high risk disease since diagnosis (Mancini M et al) (ID 5557) • Intronic DNA variants in EZH2 and GATA2 predict treatment-free remission in chronic myeloid leukemia patients from the destiny trial (Patterson S et al) (ID 5558) • Next generation sequencing from dried blood spots: “Spot on CML” results from low- and middle-income countries (Oehler V et al) (ID 5559) • An open label randomized Study of low dose dasatinib (50 mg OD) vs standard dose dasatinib (100 mg OD) vs standard dose imatinib (400 mg OD) as upfront therapy in patients with newly diagnosed chronic myeloid leukemia (CML) - chronic Phase (CP) (Nayan N et al) (ID 5560) • Clinical and molecular features associated with glucolipid metabolic disorders and cardio-/cerebro-vascular adverse events in CML patients receiving olverembatinib therapy (Zhang X et al) (ID 5561)

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	<ul style="list-style-type: none"> • Early Results of comparison of low-dose generic dasatinib (50 MG/Day) versus imatinib (400 MG/Day) in newly diagnosed chronic Phase chronic myeloid leukemia: Real-world evidence from a single-center, randomized controlled study from India (Sharma S et al) (ID 5562) • ELN milestone-driven TKI switching improves deep molecular response rates in chronic myeloid leukemia (Huang L et al) (ID 5563) • Outcomes of patients with chronic myeloid leukemia receiving second-line therapy after failure of frontline second-generation tyrosine kinase inhibitor (Haddad F et al) (ID 5564) • Asciminib for newly diagnosed chronic myeloid leukemia: Results from A phase II trial (Haddad F et al) (ID 5565) • Asciminib and pregnancy in CML: Preliminary human data and clinical implications from 47 reported outcomes (Abruzzese E et al) (ID 5566) • Discontinuation of tyrosine kinase inhibitors in chronic myeloid leukemia. a randomized national fi-LMC comparative trial of two therapeutic strategies (Cayssials E et al) (ID 5567)
<p>Dec 8 (Monday) 6.00 – 8.00 p.m. OCCC - West Halls B3-B4</p>	<p>Poster Abstract Session: Myeloproliferative Syndromes and Chronic Myeloid Leukemia: Basic and Translational: Poster III</p> <p>Poster:</p> <ul style="list-style-type: none"> • Dynamic mutational evolution and transcriptomic remodeling on 3rd-generation TKI therapy in TKI-resistant patients with chronic myeloid leukemia (Zhang X et al) (ID 5540)