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cycle 1 but initiated FUL during cycle 2. PAL was initiated at 125 mg in 13 pts (86.7%) and 100 mg in 2 pts (13.3%). One pt had a dose modification (interruption due to pt decision).

**Conclusions:** This is one of the first prospective trials to report pt characteristics and Tx patterns among male pts with HR+/HER2— ABC receiving PAL+ET. In this real-world population with a heavy disease burden, PAL was well tolerated with only 1 pt requiring dose modification. Most men received PAL+ET as 2L therapy and initiated PAL at the recommended dose of 125 mg. Further evaluation of Tx patterns in this population is warranted.

**Clinical trial identification:** Pfizer; NCT03280303.

**Editorial acknowledgement:** Editorial support was provided by Jill Shults, PhD, of ICON plc, and was funded by Pfizer Inc.

**Legal entity responsible for the study:** Pfizer Inc.

**Funding:** Pfizer, Inc.

**Disclosure:** J.L. Blum: Advisory/Consultancy: Pfizer Inc; Advisory/Consultancy; AstraZeneca; Advisory/Consultancy: Novartis; Advisory/Consultancy; Puma Biotechnology; Advisory/Consultancy: Immunogenetics Inc; Advisory/Consultancy: Research to Practice. C. Dicristo: Full/Part-time employment, Oncology Specialist: Novant Health. Z. Zhang, Y. Wang: Shareholder/Stockholder/Stock options, Full/Part-time employment, Pfizer Inc. D. Tripathy: Advisory/Consultancy, Research grant/ Funding (self); Advisory/Consultancy, Research grant/Funding (self). All authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2021.03.120

**107P**

**CDK4/6i adjustment & tumour response in the COVID-19 era**

Z. Tippay, C. Ryan, C. Harper-Wyne

Medical Oncology Department, Maidstone Hospital Maidstone & Tunbridge NHS Trust, Maidstone, UK

**Background:** The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) presents a proven risk for adverse outcome for patients with cancer and on SACT. Following national guidance, treatment modifications of SACT including for CDK4/6i were initiated across Kent Oncology Centre (KOC) hospitals. We describe the characteristics and outcomes in those who had treatment suspended.

**Methods:** Retrospective data was extracted for all patients receiving CDK4/6i therapy across three NHS trusts between January to May 2020. Clinician discretion determined need for dose interruption or reduction (considering disease history, age, comorbidities and prior cytopenias).

**Results:** A total of 196 patients receiving Palbociclib (n = 113), Abemaciclib (n = 19) & Ribociclib (n = 4) were included. Concurrent endocrine therapy (Fulvestrant n = 32; Letrozole n = 164) was continued for all patients. If receiving Denosumab, this was stopped in 72.4% (n = 92). 60.2% (n = 118) had their CDK4/6i interrupted, with a further 1.53% (n = 3), having planned, non-toxicity related, dose reductions. Median cycle number at interruption was 10 (1–34), with the median duration of interruption 84 [6–133] days. The treatment interruption group were significantly older (71.4 vs. 59.4 years; p < 0.01), with 40% (n = 48) having bone only disease. In those who had treatment interrupted, 6.8% (n = 8) had radiological progression. 6 had progression at sites of established visceral disease. In those who had progressed, 6 were rechallenged with a CDK4/6i, with 4 having stable disease on a subsequent response assessment. There was no significant difference in the number of cycles received at interruption for those who had progressive vs. stable disease (13.9 vs. 12.7; p > 0.05). At the time of radiological progression, those who had progressed had a significantly longer time off drug (107.8 vs. 81.2 days; p < 0.001).

**Conclusions:** A short interruption of CDK4/6i and continuation of endocrine treatment alone, does not appear to adversely affect tumour response from this data. Continued monitoring of this approach during subsequent pandemic waves is required together with specific outcomes of SARS-CoV-2 in the metastatic breast cancer population to ensure evidence based decision making.

**Legal entity responsible for the study:** The authors.

**Funding:** Has not received any funding.

**Disclosure:** C. Harper-Wyne: Advisory/Consultancy; Pfizer; Advisory/Consultancy; Uly; Advisory/Consultancy: Novartis; Advisory/Consultancy: AstraZeneca; Advisory/Consultancy: Roche; Advisory/Consultancy: Genomic Health; Advisory/Consultancy: Myriad; Advisory/Consultancy: Everything Genetic. All other authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2021.03.121

**108P**

**Epigenetic regulation of the putative breast cancer metastasis suppressor gene SCN4B**

L. Hilberg1, A. Hartmann2, R.A. Fasching3, S. Viltwock1, L. Mull1, S. Roger1, R. Knüchel-Clarke1, F. Steib1, E.D. Dahl1

1Department of Pathology, Universitätsklinikum Aachen, Aachen, Germany; 2Department of Pathology, Universitätsklinikum Erlangen, Erlangen, Germany; 3Department of Gynecology and Obstetrics, Universitätsklinikum Erlangen, Erlangen, Germany; 4Department of Animal Physiology, Université de Tours, Tours, France

**Background:** Breast cancer is still the leading cause of cancer deaths in women worldwide. While SCN4B is considered to be a novel metastasis suppressor gene in breast cancer, very little is known about its epigenetic regulation — in particular its downregulation in cancer tissue. In this study we explored SCN4B epigenetic regulation by promoter methylation and histone deacetylation and the clinical properties of breast tumors showing loss of SCN4B.

**Methods:** After performing methylation analysis of the SCN4B promoter region with data obtained from the TCGA data base, SCN4B methylation and expression levels were investigated in breast cancer cell lines by pyrosequencing and qPCR, respectively. Next, cell lines were treated with methyltransferase inhibitor 5-aza-cytidine (AZA) and histone deacetylase inhibitor trichostatin A (TSA) to investigate possible re-expression of SCN4B. To study clinical properties, a tissue micro array (TMA) containing 420 breast cancer samples (Bavarian breast cancer cohort) was stained with a SCN4B antibody and evaluated using IRS classification.

**Results:** TCGA data clearly showed higher SCN4B methylation levels in breast cancer tissues compared to normal tissue (p < 0.001) as well as lower SCN4B mRNA expression (p < 0.001) in cancer — with moderate correlation between the two. Concordantly, breast cancer cell lines showed high promoter methylation levels. After AZA and TSA treatment, cell lines exhibited decreased SCN4B mRNA expression up to 200-fold. TMA staining showed significantly longer metastasis- and local recurrence-free survival for tumors retaining SCN4B protein expression, as well as correlations between SCN4B protein expression and several clinicopathological parameters such as molecular subtype where loss of SCN4B was particularly substantial in triple negative breast cancer (TNBC). Interestingly, Ki67 was inversely correlated (p < 0.01) with SCN4B expression.

**Conclusions:** Our findings indicate that SCN4B is a novel metastasis suppressor gene that is epigenetically downregulated in breast cancer, especially in TNBC. Interfering with the cellular signaling pathways normally suppressed by SCN4B may open new avenues to treat triple negative breast cancer.

**Legal entity responsible for the study:** The authors.

**Funding:** European Union (Horizon 2020).

**Disclosure:** All authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2021.03.122

**109P**

**Subsequent therapies after progressing to CDK4/6 inhibition (CDK4/6i) in hormone receptor positive/HER2 negative (HR+/HER2-) advanced breast cancer (ABC)**

J.C. Laguna1, F. Braso-Maristany, T. Pasquali1, A. Rodriguez Hernandez1, M. Chic1, F. Schettin1, E. Sanfeliz Torres1, B. Gonzalez-Parrelle1, D. Martínez1, P. Galván1, V. Diez-Guardia, B. Adamo, M. Vidal1, M.C. Guillén Sacoto1, R. Moreno1, A. Prat1, M. Muñoz1, O. Martínez-Sáez1

1Medical Oncology Department, Hospital Clinic y Provincial de Barcelona, Barcelona, Spain; 2Oncology Department, IDIBAPS - Institut d’Investigacions Biomèdiques August Pi i Sunyer; Barcelona, Spain; 3Scientific Department, SOLTI Breast Cancer Research Group, Barcelona, Spain; 4Department of Pathology, Hospital Clinic y Provincial de Barcelona, Barcelona, Spain

**Background:** There is limited data in the real world setting regarding the effectiveness of subsequent lines of treatment after progressing to CDK4/6i. The optimal thera-peutic sequence is still unknown and predictors are needed.

**Methods:** This is a retrospective single-center study of 99 consecutive patients (pts) with HR+/HER2- ABC who progressed to CDK4/6i + endocrine therapy (ET) in the 1st or 2nd line setting between 05/2015-01/2021. Research-based PAM50 subtyping using the nCounter platform was performed in tumor samples collected before CDK4/6i. Median progression free survival (mPFS) and overall survival (mOS) were calculated using the Kaplan Meier method.

**Results:** mPFS with CDK4/6i in 1st line (59%) was 10.4 months (m) and 11.7m in 2nd line (41%). At the time of the analysis, 71% of patients had progressed to the subsequent line. mPFS and mOS after CDK4/6i were 5.3 and 13.6m, respectively. No correlation was observed between previous PFS on CDK4/6i and mPFS (p=0.74); mPFS with chemotherapy (CT) (45%) was 6.4m; with ET alone (18%), 2.9m; with ET + everolimus (eve) [10%], 5.1m; with PIK3CA inhibitors + ET (9%), 5.4m; and with other CDK4/6i + ET (38%), 9.1m. Fourteen percent of pts did not receive any subsequent treatment. Responses were only observed with CT (12/44), eve + ET (4/110) and CDK4/6i re-treatment (2/3). PAM50 data was available for 75 pts (75%). Luminal A (30%) showed a mPFS of 6.4m and mOS was not reached; Luminal B (33%), 7.6 and 42.1m; HER2-enriched (19%), 1.8 and 11.6m; Basal-like (10%), 7.9 and 11.5m. Luminal vs.