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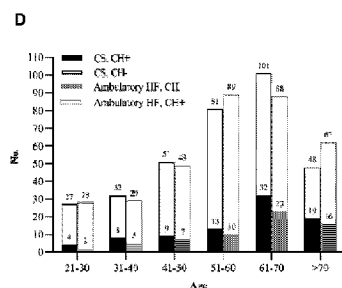


Figure 2

(57) Abstract: There is described herein methods predicting the risk of various disease condition by measuring clonal hematopoiesis in a patient, probes used to make such measurement and methods for treatment or preventive treatment of the disease condition.

CLONAL HAEMATOPOIESIS AS A BIOMARKER

RELATED APPLICATIONS

This application claims priority to U.S. Provisional Application No. 63/333,628 filed on
5 April 22, 2022, which is hereby incorporated by reference in its entirety.

FIELD OF THE INVENTION

The invention relates to the measurement of clonal haematopoiesis for the purposes of
assessing the risk associated with various disease states, particularly relating to solid
10 organs and/or cancer.

BACKGROUND OF THE INVENTION

Clonal haematopoiesis (CH) is the acquisition of mutations in hematopoietic stem cells
that results in selective clonal expansion leading to enhanced systemic inflammation.⁵
15 While early studies had shown that at least 10% of individuals harbour CH mutations in
their seventies,⁵ more sensitive techniques have demonstrated that CH mutations are
ubiquitous in healthy middle-age adults.^{6,7} Despite these mutations increasing the risk
of haematological cancer development, progression to malignancy is low.⁸

Interestingly, CH is also associated with elevated cardiovascular risk;^{5,9,10} more than
20 most traditional risk factors.¹¹ The presence of CH mutations is linked to a 25%
increase in the incidence of de novo heart failure (HF) and a 2-fold increase in
mortality in patients with pre-existing HF.^{9,12} In animal models of HF, mice harbouring
CH mutations developed adverse left ventricular (LV) remodelling with fibrosis and a
concomitant increase in IL-6 and IL-1 β .¹³

25 However, the incidence of CH in more specific cardiac diseases, specific cancers and
diseases in other organs, and its impact in clinical outcomes, remains unknown.

For example, cardiogenic shock (CS) is a life-threatening condition with markedly reduced cardiac output resulting in multi-organ failure.¹ Despite increasing recognition of CS and implementation of intensive therapies, morbidity and mortality remain exceedingly high.² The evolving epidemiological descriptions of CS patients illustrate that there is a large variability in the underlying aetiology, response to medical therapy, and outcomes.^{1,3} The association of a systemic inflammatory response syndrome with worse outcomes in CS³ could be a key to understanding the heterogeneity in the natural history of CS and uncover new pathways to target for treatment.⁴

Further, orthotopic heart transplant (OHT) is the treatment of choice for patients with advanced heart failure to improve quality of life and survival.^{s1} In the United States, over 80,000 HT were performed in the past three decades and over 5,000 yearly worldwide.^{s2, s3} However, despite the improvement in donor and recipient pre-transplant care, post-transplant survival is affected by a number of complications including infections due to long-term immunosuppression therapy, cancer development and cardiac allograft vasculopathy (CAV).^{s4, s5} OHT recipients require extensive monitoring after transplantation with endomyocardial biopsies (EMB) for rejection vigilance, viral detection for opportunistic infection, echocardiograms and coronary angiography for allograft function and CAV assessment to instill early treatment and improve survival.⁴ Despite this intensive care, new non-invasive surveillance methods that could improve morbidity and reduce mortality are still warranted.^{s6} The increased inflammatory cytokine/chemokine production linked to CH could also affect outcomes in OHT recipients, but this association has never been evaluated.^{s8, s15}

There remains a need to study the link between CH and various disease states, including those listed above.

SUMMARY OF THE INVENTION

We hypothesized that the enhanced inflammatory environment associated with CH may play a significant role in various disease outcomes and its related complications. Therefore, we evaluated the prevalence of CH in patients with various cardiac diseases, solid organ diseases and specific cancers.

In an aspect, there is provided a method of predicting the risk of a disease condition of a solid organ in a patient, the method comprising: receiving a sample from the patient containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis; comparing the degree of clonal hematopoiesis in the patient to a control degree; and determining the patient is at an elevated risk of the disease condition if the degree of clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner.

In an aspect, there is provided a kit comprising a library of probes library comprising at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.

10 In an aspect, there is provided a method of predicting the benefit of immunotherapy in a patient with melanoma, the method comprising: receiving a sample from the patient containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis in TET2; comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and determining the patient would benefit from immunotherapy if the degree of TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner. Preferably, the method further comprises treating the patient with immunotherapy.

20 In an aspect, there is provided a method of predicting metastatic risk in a patient with a non-hematological cancer, the method comprising: receiving a sample from the patient containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis in TET2; comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and determining the patient is at a lower risk of metastasis if the degree of TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner. Preferably, the method further comprises treating the patient with a treatment and monitoring regimen reflective of a low risk of metastasis.

BRIEF DESCRIPTION OF FIGURES

30 These and other features of the preferred embodiments of the invention will become more apparent in the following detailed description in which reference is made to the appended drawings wherein:

Figure 1. Study design. Cardiogenic shock patients were screened for eligibility and those with available biospecimen and consent were included. Ambulatory heart failure patients were screened on a digital database and those with complete data for age, sex, ejection fraction and aetiology of heart failure, available biospecimen and consent were included. Cardiogenic shock and ambulatory heart failure patients were matched on a 1:1 ratio for age, sex, ejection fraction, and aetiology. Ambulatory heart failure and cardiogenic shock patients were sequenced for clonal haematopoiesis related genes and its prevalence was estimated. A survival analysis was performed in each group. A survival analysis for specific genes mutations and a cytokine profile were assessed in cardiogenic shock patients.

Figure 2. Somatic variant characteristics in cardiogenic shock cohorts and ambulatory heart failure. Panel A shows the number of mutations for the 10 most frequently mutated genes according to each cohort. Panel B shows the number of mutations per patient in those where a clonal haematopoiesis mutation was identified. Panel C shows the variant allele frequency boxplot for patients with a mutation with a VAF $\geq 2\%$. No differences were observed between cohorts ($P=0.87$). Panel D shows the number of patients with CH mutation according to age in both cohorts. Filled columns represent those with CH mutations, and unfilled those without.

Figure 3. Survival according to clonal haematopoiesis and specific gene mutations. Panels A, B and C shows the reduced survival in CS patients with CH mutations in 30-day, 90-day and 3-year survival respectively. Panels D, E and F shows the reduced survival in CS patients according to specific CH-related mutations (*DNMT3A*, *ASXL1* and *TET2*) in 30-day, 90-day and 3-year survival respectively. All figures represents VAD/OHT censored survival analysis.

Figure 4. Inflammatory cytokines in CS patients with *TET2* and *ASXL1* mutations. Panel A shows the differences in SCD40L, IFN γ , IL-4, TNF- α and abundance in plasma of patients with or without *TET2* mutations of variant allele frequency $\geq 2\%$. Panel B shows the differences in CCL7 in patients with or without *ASXL1* mutations with a variant allele frequency $\geq 2\%$ ($P=0.03$).

Figure 5. Somatic clonal hematopoiesis gene mutations prevalence and characteristics in orthotopic heart transplant recipients. Panel A shows the frequency of CH in OHT recipients. Panel B shows the number of mutations according to genes related to CH. Panel C shows the number of patients with one, two, three, five and six

CH mutations. Panel D shows the number of patients with CH mutations according to age. (CH, clonal hematopoiesis; OHT, orthotopic heart transplant).

Figure 6. Survival analysis in orthotopic heart transplant recipients according to the presence of clonal hematopoiesis gene mutations. (CH, clonal hematopoiesis)

5 **Figure 7.** Somatic mutations related to clonal hematopoiesis in patients with hypertrophic cardiomyopathy. Panel A shows the number of mutations in the most common affected genes in the cohort. Panel B shows the number of mutations per patients in those with clonal hematopoiesis. Panel C shows the number of patients with clonal hematopoiesis according to the decade of the assessment. (CH, clonal
10 hematopoiesis; HCM, hypertrophic cardiomyopathy).

Figure 8. Survival in patients with hypertrophic cardiomyopathy stratified according to the presence of clonal hematopoiesis. Panel A shows the survival according to the presence of clonal hematopoiesis among HCM patients. Panel B shows the survival according to the presence of clonal hematopoiesis in the *DNMT3A*, *TET2*, and *ASXL1*
15 genes among HCM patients. Panel C shows the survival according to the presence of clonal hematopoiesis among HCM patients with sarcomeric mutations. Panel D shows the survival according to the presence of clonal hematopoiesis in the *DNMT3A*, *TET2*, and *ASXL1* genes among HCM patients with sarcomeric mutations. (CH, clonal hematopoiesis; HCM, hypertrophic cardiomyopathy).

20 **Figure 9.** Troponin I, cytokines and chemokines levels among HCM patients with sarcomeric mutations according to the presence of clonal hematopoiesis. (CH, clonal hematopoiesis; HCM, hypertrophic cardiomyopathy).

Figure 10. TET2-mutant clonal hematopoiesis is associated with clinical benefit from immunotherapy in melanoma. A) 569 patients with melanoma, bladder cancer, renal
25 cell carcinoma (RCC), or non-small cell lung cancer (NSCLC) treated with immune-checkpoint blockade were screened for clonal hematopoiesis using publicly-available exome sequencing, with at least one mutation of variant allele frequency (VAF) >0.02 detected in 74 patients (datasets EGAD00001006632, SRP064805, SRP067938, SRP072934, SRP090294, SRP095809, SRP115658, SRP128156). B) TET2-mutant
30 clonal hematopoiesis is associated with significantly higher odds of clinical benefit 6 months after receiving immunotherapy in melanoma patients (Odds Ratio = 5.98), but not bladder cancer, renal cell carcinoma, or non-small cell lung cancer patients (p

value from Firth's multivariate logistic regression adjusted for patient age, sex, study, and immune checkpoint). C) In an animal model of immunotherapy, mice with Tet2-mutant hematopoiesis – mimicking TET2-mutant clonal hematopoiesis – show enhanced response to PD-1 immune checkpoint blockade, while isotype control (ISO) treated tumours show identical growth kinetics (* $p < 0.05$ by Mann-Whitney Test).

Figure 11. TET2-mutant clonal hematopoiesis is associated with lower risk of metastatic disease in patients with non-hematological cancers. A) 16,744 patients with metastatic or non-metastatic solid tumours from Nguyen et al. Cell, 2022 were tested for clonal hematopoiesis in Bolton et al. Nature Genetics, 2020 using the MSK-IMPACT targeted sequencing panel. CH mutations with variant allele frequency of at least 0.02 were detected in 19.5% of patients. B) The 5 most commonly detected clonal hematopoiesis mutations from A) are shown. C) Exposure to TET2-mutant CH is associated with lower risk of having metastatic cancer in patients from A) as assessed by multivariate logistic regression adjusted for age, sex, smoking status, and chemotherapy treatment. D) Exposure to TET2-CH is associated with significantly lower risk of having metastases in patients from A) with Non-Small Cell Lung and Breast Cancer, with a trend towards lower risk of metastases in colorectal and bladder cancer, as assessed by multivariate logistic regression adjusted for age, sex, smoking status, and chemotherapy treatment. Patients in A) with TET2-mutant CH have significantly few metastases detected on clinical imaging (E) and have fewer overall sites / organs involved with metastatic disease (F), p values from Wilcoxon Rank Sum Test.

Figure 12. Clonal hematopoiesis prevalence among the 782 transplant recipients.

Figure 13. Clonal hematopoiesis prevalence according to transplanted organ..

Figure 14. Clonal hematopoiesis mutated genes among the 782 transplant recipients.

Figure 15. Clonal hematopoiesis mutated genes among the 127 heart transplant recipients.

Figure 16. Clonal hematopoiesis mutated genes among the 90 lung transplant recipients.

Figure 17. Clonal hematopoiesis mutated genes among the 189 kidney transplant recipients.

Figure 18. Clonal hematopoiesis mutated genes among the 374 liver transplant recipients.

Figure 19. Mortality according to the transplanted organ.

Figure 20. Mortality according to clonal hematopoiesis.

5 **Figure 21.** Mortality according to clonal hematopoiesis by transplanted recipients.

DETAILED DESCRIPTION

In the following description, numerous specific details are set forth to provide a thorough understanding of the invention. However, it is understood that the invention
10 may be practiced without these specific details.

As noted above, we investigated the role that the enhanced inflammatory environment associated with CH may play in various disease outcomes and its related complications.

In one example, we studied cardiogenic shock (CS) and its association with variable
15 systemic inflammation and whether it may be responsible for the patient heterogeneity and the exceedingly high mortality rate. Cardiovascular events have been associated with clonal haematopoiesis (CH) where specific gene mutations in hematopoietic stem cells lead to clonal expansion and the development of inflammation. This study aims to assess the prevalence of CH and its association with survival in a population of CS
20 patients in a quaternary centre.

We compared the frequency of CH mutations among 341 CS patients and 345 ambulatory heart failure (HF) matched for age, sex, ejection fraction, and HF aetiology. The association of CH with survival and levels of circulating inflammatory cytokines was analysed. We detected 266 CH mutations in 149 of 686 (22%) patients. CS
25 patients had a higher prevalence of CH-related mutations than HF patients (OR 1.5; 95% CI 1.0-2.1, $P=0.02$) and was associated with decreased survival (30-days: HR 2.7; 95% CI 1.3-5.7, $P=0.006$; 90-days: HR 2.2; 95% CI 1.3-3.9, $P=0.003$; and 3-years: HR 1.7; 95% CI 1.1-2.8, $P=0.01$). *TET2* or *ASXL1* mutations were associated with lower survival in CS patients at all-time points ($P\leq 0.03$). CS patients with *TET2*

mutations had higher circulating levels of SCD40L, IFN γ , IL-4, and TNF α ($P \leq 0.04$), while those with *ASXL1* mutations had decreased levels of CCL7 ($P = 0.03$).

CS patients have high frequency of CH, notably mutations in *TET2* and *ASXL1*. This was associated with reduced survival and dysregulation of circulating inflammatory
5 cytokines in those CS patients with CH.

Novel risk stratification and non-invasive surveillance methods are also needed in orthotopic heart transplant (OHT) to reduce morbidity and mortality post-transplant, and this was thus the focus of another example. The purpose of this study was to investigate the association between CH and OHT. Blood samples were collected from
10 127 OHT recipients. Error-corrected sequencing was used to detect CH-associated mutations. We evaluated the association between CH and acute cellular rejection, CMV infection, cardiac allograft vasculopathy (CAV), malignancies, and survival. CH mutations were detected in 26 (20.5%) patients, mostly in *DNMT3A*, *ASXL1*, and *TET2*. Patients with CH showed a higher frequency of CAV grade 2 or 3 (0% vs. 18%,
15 $P < 0.001$). Moreover, a higher mortality rate was observed in patients with CH [11 (42%) vs. 15 (15%), $P = 0.008$] with an adjusted hazard ratio of 2.9 (95% CI, 1.4-6.3; $P = 0.003$). CH was not associated with acute cellular rejection, CMV infection or malignancies. The prevalence of CH in OHT recipients is higher than previously reported for the general population of the same age group, with an associated higher
20 prevalence of CAV and mortality.

Similar studies were conducted with respect to hypertrophic cardiomyopathy, lung transplant, immunotherapy benefit in myeloma and metastatic risk of a non-hematological cancer.

Accordingly, in an aspect, there is provided a method of predicting the risk of a disease
25 condition of a solid organ in a patient, the method comprising: receiving a sample from the patient containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis; comparing the degree of clonal hematopoiesis in the patient to a control degree; and determining the patient is at an elevated risk of the disease condition if the degree of clonal hematopoiesis in the patient is higher than the
30 control degree in a statistically significant manner.

As used herein, the term "control" refers to a specific value or dataset that can be used as a reference to classify a measured value e.g. the wild type or frequency of

mutations in a cohort. A person skilled in the art will appreciate that the comparison between the measurement in the test sample and the reference values in the control will depend on the control used.

As used herein "hematopoietic stem cell" refers to cells capable of developing into any blood cell, including mature myeloid and/or lymphoid cells. These cells are typically bone marrow, liver, spleen or cord blood in origin. Myeloid and lymphoid lineages both are involved in dendritic cell formation. Myeloid cells include monocytes, macrophages, neutrophils, basophils, eosinophils, erythrocytes, and megakaryocytes to platelets. Lymphoid cells include T cells, B cells, natural killer cells, and innate lymphoid cells.

The term "sample" as used herein refers to any fluid, cell or tissue sample from a subject that can be assayed for the mutations in hematopoietic stem cells described herein..

In some embodiments, the degree of clonal hematopoiesis is measured using a variant allele frequency of mutations determined to be associated with clonal hematopoiesis. Preferably, the variant allele frequency (VAF) is $\geq 2\%$. Further preferably, the VAF is $\geq 5\%$.

In some embodiments, the following genes are sequenced in the sample: TET2, DNMT3A, and ASXL1, and optionally one or more of, but preferably all of, BCOR, BRAF, CALR, CBL, CEBPA, EZH2, FLT3A, GATA1, GATA2, GNAS, IDH1, IDH2, JAK2, KIT, KRAS, MPL, NRAS, PHF6, PPM1D, PTPN11, RAD21, RUNX1, SETBP1, SF3B1, SMC1A, SMC3, SRSF2, STAG2, TP53, U2AF1, WT1, and ZRSR2.

In some embodiments, the sequencing is performed using single-molecule molecular inversion probes (smMIPs). The smMIPs technique is an assay that combines single molecule tagging with multiplex targeted capture to enable practical and highly sensitive detection of low-frequency or subclonal variation.

In some embodiments, the mutations associated with clonal hematopoiesis are detectable by the probes listed in Table A.

In some embodiments, the mutations associated with clonal hematopoiesis are detected using a library comprising at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.

In some embodiments, the mutations associated with clonal hematopoiesis are detected using a library consisting of at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.

5 In some embodiments, the mutations associated with clonal hematopoiesis are detected using a library consisting of substantially all of the probes listed in Table A.

In some embodiments, the mutations associated with clonal hematopoiesis are detected using a library consisting of the probes listed in Table A.

In some embodiments, the solid organ is a heart.

10 In some embodiments, the disease condition is cardiogenic shock. In some embodiments, an elevated risk of cardiogenic shock is associated with an elevated risk of death. Preferably, the method further comprises treating or preventatively treating the patient for cardiogenic shock.

15 In some embodiments, the disease condition is an adverse outcome after orthotopic heart transplant (OHT). In some embodiments, the adverse outcome is an elevated risk of mortality and/or elevated risk of cardiac allograft vasculopathy. Preferably, the method further comprises treating or preventatively treating the patient for cardiac allograft vasculopathy.

20 In some embodiments, the disease condition is hypertrophic cardiomyopathy. Preferably, the method further comprises treating or preventatively treating the patient for hypertrophic cardiomyopathy.

In some embodiments, the solid organ is a lung. In some embodiments, the disease condition is an adverse outcome, including death, after lung transplant. Preferably, the method further comprises treating or preventatively treating the patient for the adverse outcome after lung transplant.

25 In an aspect, there is provided a kit comprising a library of probes library comprising at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.

In some embodiments, the kit comprises a library consisting of at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.

In some embodiments, the kit comprises a library consisting of substantially all of the probes listed in Table A.

In some embodiments, the kit comprises a library consisting of the probes listed in Table A.

- 5 In an aspect, there is provided a method of predicting the benefit of immunotherapy in a patient with melanoma, the method comprising: receiving a sample from the patient containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis in TET2; comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and determining the patient would benefit from
10 immunotherapy if the degree of TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner. Preferably, the method further comprises treating the patient with immunotherapy.

- In an aspect, there is provided a method of predicting metastatic risk in a patient with a non-hematological cancer, the method comprising: receiving a sample from the patient
15 containing hematopoietic stem cells; sequencing the sample to detect a degree of clonal hematopoiesis in TET2; comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and determining the patient is at a lower risk of metastasis if the degree of TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner. Preferably, the method further
20 comprises treating the patient with a treatment and monitoring regimen reflective of a low risk of metastasis.

- The advantages of the present invention are further illustrated by the following examples. The examples and their particular details set forth herein are presented for illustration only and should not be construed as a limitation on the claims of the
25 present invention.

EXAMPLE 1

MATERIALS AND METHODS

Patient selection

We performed a retrospective study to evaluate the prevalence of CH in CS patients and compared it to matched stable ambulatory HF patients. Stable ambulatory HF patients were chosen as controls as there is a known relationship with CH. We screened 1,315 consecutive patient admission with CS admitted to the cardiac intensive care unit (CICU) at the Toronto General Hospital (Ontario, Canada) from January 2014 to December 2020, identifying 341 patients with suitable biospecimens and consent. CS was diagnosed based on international consensus.¹⁴ Only the first CICU admission was considered for inclusion into the study. We also screened 9,485 ambulatory HF patients with no previous history of CS, orthotopic heart transplant (OHT), durable ventricular assist device (VAD), or HF admission in the 12 months prior to biospecimen collection and clinical evaluation (N=583). We included all patients aged ≥ 18 years, with biospecimens stored in the Peter Munk Cardiac Centre Cardiovascular Biobank. We excluded patients with incomplete digital records, those who withdrew consent during follow-up, or low biospecimen quality (Figure 1).

Definition of clinical states of enrolled patients

Cardiogenic shock diagnosis was based on international consensus criteria¹⁴ which required a systolic blood pressure ≤ 90 mmHg for more than 30 minutes, or the need for inotrope/vasopressor support, signs of end-organ failure (clammy skin, capillary filling time > 3 seconds, urine output < 0.5 mL/kg/h, lactate level > 4 mmol/L), or a low cardiac output (< 2.2 L/min/m² if receiving inotropes/vasopressors or < 1.8 L/min/m² without inotropes/vasopressors). In contrast, we defined stable ambulatory HF as patients with HF, with no history of OHT or VAD, or HF admission in the 12 months up to the time-point of biospecimen collection.

Data collection

Clinical and laboratory data for CS patients were collected within the first 24 hours of CICU admission. The Society for Cardiovascular Angiography and Intervention (SCAI) CS stage¹⁴ was calculated at 24 hours after CICU admission. Data collected after this time were related to in-hospital outcomes such as use of mechanical circulatory

support (MCS), renal replacement therapy, mechanical ventilation and death. Data for ambulatory HF patients were collected as close to the biospecimen collection date as possible. All study data were collected from electronic records and prior to CH sequencing.

5 *Follow-up and Outcomes*

All CS patients were followed until death or their last visit to our institution after hospital discharge. Follow-up time for the ambulatory HF group was defined from the time of biospecimen collection until the last visit to our institution or notice of death. As OHT and VAD substantially increase survival, a patient was right censored at the time of VAD, OHT or last follow-up, whichever was earlier. We reported event-free survival at 30-day, 90-day and 3-year. Similar to other studies,⁹ we defined CH-related mutations at a VAF cut-off $\geq 2\%$.

Biospecimens and analysis

Biospecimens were collected from patients during their admission to the CICU unit of the Toronto General Hospital with deferred consent. In the case that a patient, or their substitute decision-maker, denied consent at a later time point, the biospecimens were discarded. Biospecimens from ambulatory HF patients were obtained during routine heart function clinic visits at our institution. All samples were stored at -80°C at the Peter Munk Cardiac Centre biobank.

For patients with CS, biospecimens were collected within a median of 10 (interquartile range, IQR 67) days from CS admission. In the ambulatory HF patient group, 89% of biospecimens were collected on the same day as clinical evaluation. We felt the timing of biospecimen collection was appropriate as changes in clonal haematopoiesis do not occur over days, but rather over years.

25 *Genetic Sequencing Procedures*

Next-generation sequencing library construction was conducted with smMIPs.¹⁵

Cytokine Analysis

To define whether cytokine levels can be potential markers of inflammation, the analysis was restricted to those samples obtained during the admission to the CICU.

Statistical analysis

All analyses on the frequency and associations with CH were performed with a variant allele frequency (VAF) cut-off $\geq 2\%$. For specific gene analysis, we compared patients with no mutation with those with a mutation in one of the three more common mutations (*DNMT3A*, *TET2*, or *ASXL1*) with a VAF $\geq 2\%$ but with no overlap mutations. We also transformed VAF and cytokines levels using the logarithm function to reduce the distribution skewness.

A sample size of 345 patients for each group would detect a ratio of 1.5 in CH frequency between CS and ambulatory HF groups with a power of 87.5% and alpha of 0.05. There were 345 CS patients and 583 stable HF patients selected after initial screening. The CS and stable HF groups were one-to-one matched by age, sex, aetiology of HF, and ejection fraction. A nearest neighbour method was applied without replacement. After matching, there were 345 patients in each group. Figure 1 summarizes sample selection.

All variables were tested for normality with histogram analysis and Shapiro-Wilk test. Normal and non-normal continuous variables are presented as mean $\pm$ standard deviation or median (interquartile range), and categorical variables with frequencies. Between-cohort differences in continuous variables were evaluated with a Student's t-test or Mann-Whitney accordingly. Between-cohort differences in categorical variables were evaluated with a chi-square test. Logistic regression was performed to quantify the association of the prevalence of CH between CS and ambulatory heart failure patients in terms of odds ratios. Next, we separately characterized the 30-day, 90-day and 3-year event-free survival for each cohort using the Kaplan-Meier survival method. Furthermore, within each cohort, we contrasted event-free survival in patients with and without CH in terms of hazard ratios (HR) using Cox proportional hazards regression and evaluated the survival differences using log-rank tests. The HRs in this descriptive analysis were not adjusted for covariates. Subsequently, we also quantified the association between CS and mortality both with age only and with other clinically relevant covariates (i.e., age, sex, aetiology, use of mechanical circulatory support, creatinine, sodium, and white blood count). The model was validated by the proportional hazard assumption test based on Schoenfeld residual. Finally, as a sensitivity analysis, we repeated the previous analyses on all-cause mortality without right censoring patients at the time of OHT and VAD implantation. We described and

explored the event-free survival and the levels of 48 cytokines in patients with a single gene mutation in the three most commonly mutated genes to patients without any mutations. We used a statistical significance of 0.05 for all analyses and a two-sided p-value. All analyses were performed using SPSS, version 25.0 (SPSS Inc., NY, USA).

5 RESULTS AND DISCUSSION

In this study, 341 patients were included in the CS group and 345 in the ambulatory HF group. Four (1%) patients were excluded from the CS group due to poor biospecimen quality (Figure 1). All characteristics included in the case-control study were similar between groups (Table 1). Our study population consisted mostly of
10 males with a predominance of non-ischemic cardiomyopathy. Patients with CS, however, had a lower body mass index, more frequent history of smoking, lower incidence of pre-existing chronic HF, higher admission serum creatinine and higher BNP when compared with ambulatory HF patients.

In the CS group, the most common aetiology was acute decompensation of chronic HF
15 in 225 (66%) patients, followed by new onset HF in 93 (27%), myocardial infarction in 10 (3%) and myocarditis in 11 (3%). The majority were classified at SCAI stage D (285 patients, 83%) and pulmonary artery catheter was used in 178 (52%) patients confirming disease severity. Fifty-two (15%) patients required temporary MCS with intra-aortic balloon pump being the most commonly used (n=26, 8%), followed by
20 extra-corporeal life support (n=18, 5%), and Impella device® (Abiomed Inc., Massachusetts, USA) (n=8, 2%). Mechanical ventilation was needed in 63 (18%) patients and renal replacement therapy in 35 (10%). Clinical characteristics are summarized for both CS patients (Table 2) and ambulatory HF (data not shown). CS patients were followed for a median of 676 (1289) days, while ambulatory HF patients
25 were followed for 1246 (884) days.

In the CS group, 104 (30%) underwent OHT, 79 (23%) required durable VAD, 132 (39%) died during follow-up with 59 (17%) patients dying during the index hospitalization and 73 (21%) after hospital discharge. In the ambulatory HF group, 19 (6%) patients had OHT, seven (2%) were referred for durable VAD, and 35 (10%)
30 patients died.

Prevalence and distribution of CH mutations in the study groups

Overall, 422 mutations were identified in 28 genes, of which 266 (63%) mutations had a VAF $\geq 2\%$ and were included in the analysis (data not shown), affecting 149 (22%) of the 686 patients. The most common mutation type was missense, followed by frameshift, nonsense and splice site (data not shown). The most mutated gene was *DNMT3A* in 84 (32%), followed by *TET2* in 48 (18%), and *ASXL1* in 30 (11%) patients, with a similar distribution in mutations observed in both the CS and ambulatory HF groups (Figure 2A). Among all patients with CH, 83 (56%) had a single mutation, 35 (23%) had two and 31 (21%) had three or more (Figure 2B). The VAF distribution was similar in both populations (Figure 2C). As expected, an age-associated increase in CH prevalence (Figure 2D) was observed in both groups; however, the overall frequency of CH was significantly higher in CS patients, as compared with ambulatory HF patients (25.2% versus 18.3%; odds ratio, 1.5; 95% CI 1.0-2.1, $P=0.02$). CS patients with CH were older than those without mutations, and had a higher frequency of dyslipidaemia, lower sodium, and white blood count (Table 2). The frequency of CH in CS patients with previous HF was similar than those without out [24 (26%) vs. 24 (23%), $P=0.54$]. Ambulatory HF patients with CH were older and more likely to have ischemic cardiomyopathy (data not shown).

Association between CH and survival

The presence of CH in the CS group was associated with an increased risk of death in a multivariable adjusted analysis for age, sex, aetiology of CS, use of mechanical circulatory support, creatinine, sodium, and white blood count and censored for VAD/OHT: 17 patients (20%) with CH died in the first 30-days compared with only 17 (7%) without mutations (hazard ratio, (HR), 2.7; 95% CI 1.3 to 5.7, $P=0.006$; Figure 3A). A similar ratio was seen at 90-days (HR, 2.2; 95% CI 1.3 to 3.9, $P=0.003$; Figure 3B). At 3 years, 36 patients (42%) with CH had died compared with 47 (18%) without detectable CH at initial admission for CS (HR, 1.7, 95% CI 1.1 to 2.8, $P=0.01$; Figure 3C). Similar results were observed with the unadjusted and age-adjusted analysis for both uncensored and VAD/OHT censored (data not shown). There were no differences in survival between CH and non-CH groups in the ambulatory HF patients uncensored or VAD/OHT censored (data not shown). In addition, when considering VAF as a continuous variable, there was no association with death. However, analysis of VAF categorized as ≥ 2 to $<5\%$ and $\geq 5\%$ was associated with a higher risk of death in those

with VAF $\geq 5\%$, at 90-days and 3-years. Increasing number of mutated genes was not associated with death (data not shown).

We then stratified the CS group according to the three most mutated genes (*DNMT3A*, *TET2*, and *ASXL1*, Figures 3D, 3E, 3F) and found an association for *TET2* and *ASXL1* mutations with decreased 30-day, 90-day, and 3-year survival compared with patients who had no CH mutations. Results for survival censored for VAD/OHT had similar results. *DNMT3A* mutations had no effect on survival at 30-day, 90-day or 3-year follow-up.

Cytokine expression in cardiogenic shock

To assess changes in the inflammatory milieu associated with CH, we compared 48 cytokines in the plasma of CS patients with mutations in the three most mutated genes, to those without CH. CS patients with *TET2* mutations exhibited higher circulating levels of SCD40L, IFN γ , IL-4, and TNF α (Figure 4A, 4B, 4C and 4D), and those with *ASXL1* mutations had lower levels of CCL7 (Figure 4E) compared with patients lacking CH mutations. There were no differences in the cytokine profiles of patients with *DNMT3A* mutations (data not shown).

Discussion

CH mutations are associated with increased risk for adverse cardiovascular events in specific populations of patients with atherosclerosis and heart failure.^{5,9,10} We found that patients with CS had a 1.5-fold higher prevalence of CH as compared to ambulatory HF patients, with an associated decrease in 30-day, 90-day, and 3-year survival. The observed prevalence of CH in ambulatory HF patients (18.3%) is consistent with other reported findings, emphasizing the comparability of the methodology applied.^{9,16} Importantly, our data shows that CH may be considered as risk factor for CS admission regardless of prior HF. Of note, mutations specifically in *TET2* and *ASXL1* impacted the prognosis of CS and were associated with distinct circulating inflammatory cytokine profiles, compared with those patients without CH mutations. While the retrospective nature of this study may have introduced a survival bias, as CS patients who did not survive the first hours of presentation were less likely to have biospecimens taken, our study likely underestimates the prevalence of CH in CS. To our knowledge, this is the first study to show the higher prevalence of CH and lower survival in patients admitted with CS.

DNMT3A was the most common mutated gene in both groups, followed by *TET2* and *ASXL1*. These genes are epigenetic regulators with a role in altering DNA methylation to potentially promote stem cell self-renewal and clonal expansion.¹⁷ While *DNMT3A* is responsible for *de novo* DNA methylation, *TET2* promotes demethylation.¹¹ *ASXL1* has a role in chromatin regulation, promoting myeloid leukemogenesis.¹⁸ Although the mechanisms ascribed to these genes are still poorly understood, they likely boost systemic inflammation which may have an impact in CS.^{10,13,17} *TET2* mutations were associated with a significant decrease in both short- and long-term survival in CS patients. These findings mirror previous work showing higher pathogenicity of mutations in *TET2*, compared to *DNMT3A* in the broader setting of HF.^{9,10,16,19} Additionally, *ASXL1* mutations have been related to increased risk for myocardial infarction¹⁰ but its role in HF was unknown prior to our study. Here, we report a decrease of short- and long-term survival in *ASXL1* carriers admitted with CS. *DNMT3A* mutations have been associated with HF hospitalization and death¹⁶, but this was not seen in our analysis. Mutations in *DNMT3A* may have lower pathogenicity in CS, explaining their higher frequency in this population.

CS is associated with acute systemic inflammation, which has been shown to increase mortality.^{4,20} Augmentation of this inflammatory state could explain the increased risk of death seen in CS patients with CH mutations. In our study, patients with CS harbouring *TET2* mutations had elevated circulating levels of SCD40L, IFN γ , IL-4, and TNF- α . SCD40L has a pro-inflammatory, pro-coagulant function associated with cardiovascular events related to atherosclerosis.^{21,22} IL-4 leads to tissue macrophage accumulation,²³ and increases IFN γ expression, which has a key role in the adaptive immune response²⁴, and promotes myelopoiesis in response to inflammation^{24,25}. TNF- α is primarily produced by macrophages and can induce apoptosis in hematopoietic cells.^{26,27} Additionally, *ASXL1* mutations in CS patients were associated with lower circulating levels of CCL7, a chemokine that is a potent chemo-attractant for myeloid cells.²⁸ The differential regulation of cytokines promoted by specific CH mutation could enhance the immune response leading to reduced survival in *TET2* and *ASXL1* mutation carriers. The lack of any dysregulation of circulating cytokines with *DNMT3A* mutations offers an explanation why survival was not affected in our CS patients.

Clinically, the increasing incidence of CS remains a major limitation in patient management.² The association of CH mutations with higher mortality could provide a new biomarker to help identify patients at elevated risk, establishing a new paradigm of

risk assessment in CS. The fact that CH patients were older does not explain the dramatic increase in mortality in CH patients, as the age-adjusted analysis showed similar results. Also, similar age differences between study populations were described in previous studies showing the impact of CH in cardiovascular outcomes.^{9,19}

5 Mechanistically, the increased risk due to CH likely occurs through augmentation of the acute inflammatory state in CS. The altered cytokine profile may provide potential therapeutic targets in these patients. Prior studies have shown that decreased IL-6 receptor activity improved outcomes in patients with CH.²⁹ The development of smMIP-sq for CH assessment in our study should overcome the barrier to clinical
10 implementation of other sequencing methods.^{16,19} Furthermore, the 2% VAF cut-off remains controversial. VAF reflects the size of the expanded clone evaluated in the peripheral blood and it is reasonable to hypothesize that risk increases with an increase in VAF.^{11,16,17} However, we did not find a difference in the VAF between CS patients and ambulatory HF, and its use as a continuous variable was not associated
15 with increased mortality in CS patients. We did find that CH-associated risk increased with a categorized higher VAF cutoff.¹⁶ This may be explained by the highly-skewed distribution of VAF.

CS patients had a 50% higher prevalence of CH mutations than stable ambulatory HF patients. These mutations were associated with a 2-fold reduction in survival of CS
20 patients. Specifically, mutations in *TET2* and *ASXL1* genes were shown to be more lethal than *DNMT3A* in this context and were associated with an altered profile of circulating inflammatory cytokines that may suggest a mechanism for CH to affect patient outcomes.

25

EXAMPLE 2

MATERIALS AND METHODS

Study population

We performed a retrospective study of 127 patients that underwent OHT in the Toronto General Hospital from 2005 to 2021. Only patients older than 18 with available
30 biospecimens and complete clinical evaluation were included. No patients with active

malignancies were included because it is considered a criteria for heart transplant candidacy.

Data collection

The clinical chart and pre-transplant assessment of OHT recipients were reviewed
5 from the patients' digital health records. We collected demographic data, medical
history, laboratory assessment, date of OHT, transplant-related treatment at the time
of the procedure and the immunosuppressive regimen at 1-year post-OHT, the
occurrence and grade of acute cellular rejection episodes, CMV infection, CAV, any
malignancy and death. All baseline characteristics regarding demographic and
10 comorbidities were collected as close as possible to the biospecimens collection date
because of its relationship with CH mutations.

Follow-up and outcomes

Patients were followed from the time of OHT procedure to the last visit at our institution
or death. The patients were evaluated for the following outcomes: the first occurrence
15 of acute cellular rejection, CMV infection, de novo post-transplant malignancy, CAV,
and death.

Cellular rejection episode was diagnosed and classified according to the International
Society for Heart and Lung Transplantation (ISHLT).^{s4} A positive cellular rejection
episode was defined as the occurrence of 2R and 3R classification in EMB performed
20 routinely or due to clinical suspicion of cellular rejection. As a routine, patients are
routinely evaluated for rejection in weeks 1-4, 6, 8, 10 and months 3-6, 9, 12, 18, 24.
Gene-expression profiling was also used for rejection surveillance in low-risk patients
at our center, with a gene-expression derived high-risk score verified by confirmatory
EMB. CMV assessment was performed when infection was suspected or when
25 prophylaxis was discontinued. CMV infection was defined as positive PCR in
peripheral blood regardless of clinical symptoms. Post-OHT malignancy vigilance is
performed routinely by our centre and the diagnosis is based on tissue biopsy showing
malignant neoplastic cells. OHT recipients have coronary angiography performed with
intravascular ultrasound at 3 months, 12 months, 1-year and 5-years post OHT,
30 though this may be deferred if intercurrent illness or significant kidney disease is
present. The findings are graded according to the ISHLT criteria for CAV and

considered positive in the presence of CAV2 or CAV3.^{s4} In the survival analysis, we considered death from any cause in the end of follow-up.

Genetic Sequencing Procedures

Peripheral blood samples were collected with patient consent during clinical visits to
5 Toronto General Hospital and were stored in the Peter Munk Cardiac Centre biobank. 100ng of DNA was used to construct sequencing libraries using single molecule Molecular Inversion Probes (smMIP) as previously described.^{s16} Paired-end 150bp sequencing reads were generated using the Illumina Novaseq platform (Illumina Inc., California, USA).

10 *Statistical analysis*

All variables were tested for normality using the Shapiro-Wilk test. Normal and non-normal continuous variables are presented as mean $\pm$ standard deviation or median (interquartile range). Difference among groups were evaluated with a Student's t-test or Mann-Whitney accordingly. Group differences in categorical variables were
15 evaluated with a chi-square test or Fisher's exact test as appropriate. The hazard ratios (HR) for the event-free survival for OHT patients with or without CH was calculated using the Cox proportional hazards regression. An adjusted Cox proportional hazards regression including age, sex, primary heart failure diagnosis (ischemic, non-ischemic and congenital) and chronic kidney disease was also
20 calculated to estimate the impact of CH on survival. The model was ascertained by the proportional hazard assumption test based on Schoenfeld residual and found to be valid. The survival function is represented graphically using Kaplan-Meier curves and compared according to the presence of CH mutations using log-rank test. We used a statistical significance of 0.05 for all analyses and a two-sided p-value. All analyses
25 were performed using SPSS, version 25.0 (SPSS Inc., NY, USA).

RESULTS AND DISCUSSION

Study participants

Between 2005 and 2021, 589 patients underwent OHT at our institution. 127 patients (21.5%) were included in this study. 97 males and 30 females. Their mean age was
30 49 $\pm$ 14 years. The most common comorbidities were prior smoking (n=34, 27%), hypertension (n=33, 26%), dyslipidemia (n=33, 26%) and type 2 diabetes (n=28, 22%).

90 patients (71%) were diagnosed with underlying non-ischemic HF prior to OHT while 28 patients (22%) had ischemic heart disease. Transplantation was conducted in 44 (35%) patients with a ventricular assist devices (VAD). All patients received induction therapy, with the vast majority (n=123, 97%) receiving rabbit anti-thymocyte globulin.

- 5 The most common immunosuppressive therapy regimen at hospital discharge included tacrolimus (n=119, 94%), sodium mycophenolate (n=123, 97%) and corticosteroids (n=127, 100%). Table 3 summarizes the patient's baseline characteristics.

Evaluation of CH in OHT recipients

- The targeted sequencing of CH genes was successfully performed in all patients. In 87 (68%) cases, samples were collected before the procedure with a median time from sample to procedure of 200 (IQR 428) days, and in 40 (32%) cases the sample was collected after the transplant, with a median time from the procedure to the sample collection of 114 (IQR 624) days. We observed 46 CH mutations in 26 (20%) OHT recipients (Figure 1A) with a median variant allele frequency (VAF) of 8.6% (3.2-35.9).
- 15 The most commonly affected genes were *DNMT3A* in 11 (9%), *ASXL1* in 5 (4%), and *TET2* in 4 (3%). *TP53*, *PPM1D*, and *CEBPA* affected two (2%) patients each, while *JAK2*, *IDH2*, *SF3B1* and *SMC1A* were found in one (1%) patient each (Figure 2B). Most patients (N=16, 62%) with CH had only one mutation (Figure 3C). Clinical characteristics of patients with specific CH mutations were summarized (data not shown).
- 20 The list of variants detected can be found in Table 5. Gene mutations and the VAF according to immunosuppressive regimen at hospital discharge were summarized (data not shown).

- OHT patients with CH were older than those without mutation (54 ± 13 vs 48 ± 14 years, $P=0.04$) and were followed for a longer period after OHT (4.3 ± 3.9 vs. 2.9 ± 2.1 , $P=0.01$).
- 25 CH carriers also received an organ from EBV+ donor less frequently than those without CH [16 (62%) vs. 81 (80%), $P=0.02$]. At hospital discharge, CH patients were less likely to receive tacrolimus [22 (85%) vs. 97 (96%) $P=0.03$], and sodium mycophenolate [23 (89%) vs. 100 (99%), $P=0.006$]. There were no statistically significant differences in the prevalence of cardiovascular risk factors (diabetes,
- 30 hypertension, BMI, smoking, dyslipidemia) or pre-OHT HF etiology between patients with and without CH (Table 3).

CH is associated with increase mortality in the setting of OHT

The mean follow-up post-OHT was 3.2 ± 2.6 years. Primary graft dysfunction occurred in 19 (15%) with an in-hospital mortality post-OHT of 9%. During follow-up, 59 (46%) patients had CMV infection, 69 (54%) had at least one 2/3R acute cellular rejection episode, 11 (9%) had post-transplant malignancy, 4 (3%) CAV grade 2 or 3, and 26 (20%) died.

All four CAV diagnosis occurred OHT recipients with CH (18%; $P < 0.001$), but no CAV was found among those without CH. The number of patients assessed for CAV with coronary angiogram was similar between patients with CH and those without CH [17 (65%). Vs. 68 (67%), $P = 0.85$], and also time from OHT to first coronary angiogram 441 (1192) days vs. 399 (107) days, $P = 0.19$. Patients with CH showed a higher mortality rate than those without CH [11 (42%) vs. 15 (15%), $P = 0.008$], with an unadjusted hazard ratio (HR) of 3.1 (95% CI 1.4-6.7), $P = 0.005$, and an adjusted HR of 2.9 (95% CI 1.4-6.3; $P = 0.003$). The clinical characteristics and cause of death of patients with or without CH were summarized (data not shown). Because CH patients had a trend towards more Cyclosporine prescription at discharge and at the end of the first year after OHT, we performed a sensitivity analysis with age and type of immunosuppressive drugs in the 1st year. The adjusted model showed that CH was associated with increased mortality with an HR of 5.9 (95% CI 1.7-19.9), $P = 0.004$. Figure 2 shows the Kaplan-Meier survival analysis with an increased mortality in those with CH (P log-rank = 0.003). The evaluation of outcomes according to specific gene mutation showed that more acute cellular rejection 2/3R were observed in those with *DNMT3A*, more CAV grade 2/3 in patients with *DNMT3A* and with *TET2*, and higher mortality in those with *TET2* gene mutation (data not shown). We did not observe a higher mortality in patients with a $VAF \geq 10\%$ ($n = 14$) in comparison to those with $VAF < 10\%$ ($N = 113$) [5 (35.7%) vs. 21 (18.6%), $P = 0.31$]. Considering our results, we undertook a post-hoc power calculation. With a sample size of 127 patients and a hazard ratio of 3.1 for OHT survival, given an alpha of 0.05, we would have a power of 99.2% of detecting such a difference. No differences were observed between OHT patients with or without CH regarding CMV infection, 2/3R acute cellular rejection or post-transplant malignancy. Both cases of post-OHT malignancy in patients with CH were diagnosed with squamous cell carcinoma of the skin, and one also developed lung carcinoma. No patients developed PTLD. Table 4 summarizes outcomes in OHT recipients according to CH.

Discussion

CH has been linked to inflammatory conditions, increased mortality and incidence of cardiovascular diseases, but data in the setting of solid organ transplantation is scarce.

^{s8, s17} We showed that the CH prevalence in OHT recipients is higher than expected for the same age in the general population. CH mutations are associated CAV and a 3-fold increase in mortality after OHT. These findings suggests a new biomarker in post-transplantation risk assessment.

The detection of CH is an exceedingly common feature of aging, with a prevalence of 10% by the 7th decade of life and an overall prevalence of 4-5%.^{s7} In this study, 20% of OHT recipients were found to have a CH mutation in a younger population, mainly in *DNMT3A*, *ASXL1* and *TET2* genes, with most patients harboring a single mutation. Previously, HF patients were shown to have an 18.5% frequency in CH mutations at a median age of 65 years.^{s10, s14} Similarly, the median VAF in our cohort was 12%, which is much higher than the reported $\approx$ 4-6% in non-advanced HF cohorts.^{s10, s14}

Additionally, CH patients have been consistently reported to be older than those without these mutation, similar to our study. While concerns can be raised about CH being a risk marker of aging instead of a disease-driving factor, several studies have shown that CH is independently associated with increased cardiovascular risk after adjusting for age.^{s10, s14} CH has also been shown to have a causal effect in mice harboring either *Dnmt3a*, *Tet2*, *Jak2*, *Tp53*, and *Ppm1d* mutations with adverse LV remodeling, lower LV ejection fraction and worsening degree of fibrosis post-myocardial infarction.^{s15, s18–s21}

CH mutations have been shown to be associated with atherosclerosis, myocardial infarction, stroke and HF.^{s8–s10, s14} A similar background of inflammation driving clinical outcomes would be expected in OHT recipients. In our study, CH was associated CAV grade 3. Experimental models have shown that *Tet2*-deficient mice have larger atherosclerotic plaque size and increased levels of several inflammatory cytokines.^{s8, s22} Higher levels of coronary artery disease have been observed in patients with CH.^{s8} The pathways involved in atherosclerosis development overlap with the inflammatory background of CH, but the pathophysiology of CAV comprises a complex interaction of immune and non-immune factors which contribute to a pro-inflammatory state and ultimately result in endothelial injury, vascular cell proliferation, fibrosis, and remodeling.^{s23–s25} The increased inflammatory cytokines expressed in patients with

CH could have a role in CAV development, but further studies are needed to confirm this hypothesis, especially due to the low number of CAV diagnosis in the sample.

The complex relationship of immune phenomena and inflammation promoted by CH could be responsible for other outcomes in OHT recipients. The innate immune system can be activated by several different cytokines resulting in rejection episode ^{s26, s27}, and activation of IL-1R pathway has a central role in ischemic reperfusion injury. ^{s28} However, despite the overall higher proportion of CH patients experiencing acute cellular rejection, it failed to reach statistical significance. One possibility is that based on the 16% absolute increase observed, a sample size of over 400 patients would be required to reach statistical significance. CH has also been associated with the occurrence of infections and malignancies. ^{s11, s12} We observed only a small number of such events which may explain the lack of association of CH with these outcomes. Nevertheless, CH was associated with a 3-fold increase in mortality in OHT recipients even after adjustment for confounding factors. Cardiovascular events in CH patients have been shown to be related with ischemic events and progression to heart failure, but these are not common in OHT recipients. ^{s8, s10, s14} The cause of death in this study was related to allograft dysfunction, acute rejection episode, CAV, and septic shock. The mechanisms responsible for the observed high mortality rate remains unclear, yet previous work have shown that non-survivors after OHT were more likely to present with sustained inflammation. ^{s29} Nonetheless, patients with CH were older, and, despite not being statistically significant, also had higher frequency of hypertension, dyslipidemia and smoking that could have an impact in our results.

In summary, we demonstrated that CH is associated with, and a potential risk factor, for CAV and mortality in OHT recipients. The complex interaction of the inflammatory cytokines promoted by CH and the immune system could drive several other potential outcomes such as rejection, infections and malignancies. A prior case series of graft versus host disease in transplanted liver patients showed that 71% had CH, highlighting the possible association with these somatic mutations to adverse outcomes. ^{s30}

EXAMPLE 3

METHODS AND MATERIALS

We investigated a cohort of patients with hypertrophic cardiomyopathy (HCM) submitted to targeted sequencing for detecting CH. We included 799 patients who
5 were ≥ 18 years of age, with a clinical diagnosis of HCM by current guidelines²², cardiac magnetic resonance imaging (MRI) and available biospecimens from the PMCC Biobank. We excluded patients with incomplete records, those that withdraw consent, or low biospecimen quality.

Among the 799 included patients, CH was found in 183 (22.9%). HCM patients with
10 genotype-positive and CH were found to be more symptomatic and with a higher burden of fibrosis. CH was associated with major cardiovascular event (MACE) in HCM patients [adjusted HR of 3.46 (95% CI 1.25-9.52; $p=0.016$)], with the highest risk among genotype-positive and *DNMT3A*, *TET2* and *ASXL1* mutated genes [adjusted HR of 7.23 (95% CI 1.79-29.13) $p=0.005$]. Several cytokine and chemokines (IL-1ra,
15 IL-6, IL-17F, TGF α , CCL21, CCL1, CCL8, and CCL17), and also troponin I were upregulated in those genotype-positive with CH. Fibrosis, a hallmark of HCM, was found to be increased in those with CH, as well as ABPR at exercise. CH was also associated with a higher mortality and major cardiovascular events (MACE). These results indicate that CH is frequent among HCM patients and associated with a worse
20 clinical phenotype and outcomes.

Definitions

HCM was defined as the presence of maximal LV wall thickness (MLVWT) ≥ 15 mm. Also included were patients with MLVWT ≥ 13 mm and a P/LP genetic variant or a family history of HCM in the absence of other causes for hypertrophy. MLVWT was
25 defined as the higher LV wall measure on echocardiogram or cardiac MRI. The assessment of P/LP variants was conducted using a previously published strategy by our group²³. Evaluation for non-sustained ventricular tachycardia (NSVT) or abnormal blood pressure response (ABPR) at exercise were performed according to the attending clinician discretion. All patients with HCM underwent cardiac MRI with late
30 gadolinium enhancement (LGE) for quantification of fibrosis. The LGE was assessed visually and quantified manually as previously validated⁸. LGE extent was defined as the LGE mass percentage of the total LV mass. We were able to quantify LGE in

(84.1%) of patients with MRI. In the remaining 127 (15.9%) patients, we only included the qualitative measure of LGE as present or not.

Clonal hematopoiesis and cytokine assessment

The full method on CH and cytokines evaluation was performed substantially as described in earlier examples. Briefly, we used a single molecule molecular inversion probe (smMIP) method including 35 myeloid genes related to CH and smMIP-tools to call mutations and reported those with variant allele frequency (VAF), a measure of clone size, $\geq 2\%$ for all analysis. The cytokine analysis was performed using a human cytokine/chemokine 71-plex assay with the Luminex™ 200 system by Eve Technologies Corp. (Alberta, Canada) and included brain natriuretic peptide (BNP) and cardiac troponin I (cTnI).

RESULTS AND DISCUSSION

Clonal hematopoiesis prevalence and characteristics

Overall characteristic of cohort is summarized in Table 7. The median time from biospecimen collection to the echocardiogram was 0 (0-16) days and median time from biospecimen to MRI assessment was 2.2 (0.2-5.6) years.

All patients had an assessment of CH performed and all samples passed quality control specifications. CH mutations were observed in 183 (22.9%) patients with a median VAF of 6.7 (2.8-40.8)%, being 136 (17.0%) in the three most common genes: *DNMT3A* in 70 (8.8%), *TET2* in 51 (6.3%), and *ASXL1* in 24 (3.0%), comprising 73.8% of all CH mutations. All other genes included in the smMIP panel were present in less than 1% of patients. Most patients (158, 19.8%) harbored a single mutation, while two mutations were found in 18 (2.3%) patients, 4 (0.5%) three mutations, and 3 (0.3%) four or more mutations. Among the 183 patients with CH, 135 (73.7%) were over 50 years old. However, among the 54 patients <30 years, CH was present in 14 (25.9%). In relation to the age of HCM diagnosis, 27 (14.8%) patients with CH were diagnosis before 30 years of age, while 62 (33.8%) within 31-50 years, and the majority, 94 (51.3%), were diagnosed after 50 years old. Figure 7 summarizes CH mutations characteristics. Overall clinical characteristics of CH patients were similar to those without CH (Table 7).

We then analyzed the association of CH with the HCM phenotype. No differences were found in those with or without CH on echocardiographic parameter such as MLVWT (18.3 ± 4.2 mm vs. 18.8 ± 5.1 mm, $p=0.256$), left atrium diameter (36.7 ± 14.5 mm vs. 38.1 ± 12.7 mm, $p=0.208$) and LVOT gradient (24.1 ± 36.2 mmHg vs. 22.1 ± 34.6 mmHg, $p=0.491$). Cardiac MRI parameters, including the presence of fibrosis evaluated by LGE, as well as other HCM features such as syncope, ABPR at exercise, among others were also similar between groups. However, apical aneurysm [33 (5.4%) vs. 14 (7.7)%, $p=0.004$] and death or need for orthotopic heart transplant was higher among patients with CH [4 (0.6%) vs. 6 (3.3%), $p=0.005$]. MACE was also more frequent among patients with CH [12 (2.0%) vs. 9 (5.2%), $p=0.026$], with an unadjusted HR of 2.72 (95% CI 1.14-6.49; $p=0.023$), and adjusted HR of 3.46 (95% CI 1.25-9.52; $p=0.016$). CH patients showed a worse survival in comparison to those without CH as shown in the Kaplan-Meier curve (Log-Rank $P=0.018$) (Figure 8A).

Clonal hematopoiesis related to DNMT3A, TET2 and ASXL1

We evaluated whether somatic mutations in 3 genes (DNMT3A, TET2 and ASXL1) alone are sufficient to drive poor HCM patient outcome. We found that patients harboring *DNMT3A*, *TET2* and *ASXL1* mutations were older than their counterparts [55.3 (14.5%) vs. 58.1 (14.5%), $p=0.042$] and were more likely to have a family history of SCD [28 (4.2%) vs. 9 (6.7%), $p=0.038$]. As we noted in the wider CH analysis, these patients were more likely to have hypertension, a P/LP germline HCM related mutation, a pacemaker and treated with a non-dihydropyridine calcium channel blocker (Table 6).

While patients with *DNMT3A*, *TET2* and *ASXL1* CH mutations, exhibited a similar HCM phenotype to those without CH, patients with CH showed a higher mortality or need for orthotopic heart transplant [4 (0.6%) vs. 6 (4.4%), $p<0.0001$] and MACE [13 (2.0%) vs. 8 (6.2%), $p=0.008$]. The unadjusted HR for MACE was 3.19 (95% CI 1.32-7.73; $p=0.010$) and adjusted HR 3.97 (95% CI 1.39-11.28; $p=0.010$). Figure 8B shows the Kaplan-Meier curve with worse survival for patients with CH (Log-Rank $p=0.006$).

Clonal hematopoiesis in HCM patients with a sarcomeric mutation

HCM patients harboring a sarcomeric gene mutation are associated with early age at diagnosis, higher MLVWT and worse outcomes²⁵. Because this subtype of HCM patients have a distinct clinical profile, we sought to investigate if there could be a

distinct interaction with CH. The overall characteristics shown among those with or without CH were recorded (data not shown). A trend of a higher burden of symptoms (NYHA class II to IV) [15 (40.5%) versus 47 (28.0%), $p=0.132$] among those with CH, but it was not statistically significant. However, CH was associated with a higher

5 burden of fibrosis as reflected by LGE in $>15\%$ of the LV mass [11 (29.7%) versus 26 (15.3%), $p=0.044$], with an odds ratio of 2.32 (95% CI 1.00-5.38, $p=0.048$), there was a trend towards a higher amount of fibrosis in the LV of those with CH [15.6 % (10.4-24.6)] in comparison to those without CH [12.2 % (7.7-17.6)], $p=0.068$. CH patients also showed a higher frequency of ABPR at exercise [25 (29.8%) vs. 10 (62.5%),

10 $p=0.012$]. Finally, CH was associated with worse outcomes with higher mortality or orthotopic heart transplant [3 (8.1%) versus 2 (1.2%), ($p=0.013$)]. This was also observed for MACE in patients with CH [5 (13.5%) versus 5 (3.0%), $p=0.008$], with an unadjusted HR of 5.28 (95% CI 1.51-18.4, $p=0.009$), and adjusted HR of 6.89 (95% CI 1.78-26.6, $p=0.005$). Figure 8C shows the Kaplan-Meier survival curve showing a

15 worse survival for those with CH (log-rank $p=0.003$).

We then stratified the HCM phenotype and clinical outcomes in those with sarcomeric mutations and specifically the presence of *DNMT3A*, *TET2*, and *ASXL1* mutations. Baseline characteristics was similar between groups. The amount of fibrosis assessed by LGE was higher in those with CH [21.2% (13.7-41.8) versus 12.2% (7.7-17.5),

20 $p=0.014$], whereas LGE $\geq 15\%$ was not statistically different in HCM patients with and without CH [7 (35.0%) vs. 30 (18.3%), $p=0.09$]. However, CH patients showed a higher frequency of abnormal blood pressure response (ABPR) with exercise than those without CH [8 (66.7%) vs. 27 (30.7%), $p=0.014$]. Finally, mortality or orthotopic heart transplant was more common in the CH group than in those without CH [3 (13.0%) vs.

25 2 (1.1%), $p<0.0001$], as well as MACE [4 (17.4%) vs. 6 (3.2%), $p=0.004$]. MACE was associated with CH with an unadjusted HR of 5.04 (95%CI 1.39-18.28) $p=0.014$ and adjusted HR of 7.23 (95% CI 1.79-29.13) $p=0.005$. Figure 8D shows the Kaplan-Meier survival curve showing a worse survival for those with CH (log-rank $p=0.007$).

Biomarkers and cytokines/chemokines

30 A panel of 71 cytokines and chemokines, BNP and cardiac troponin I were evaluated in patients with a sarcomeric mutation and levels were compared among those with (N=37) or without CH (N=169). It was observed that troponin I ($p=0.008$), IL-1ra ($p=0.037$), IL-6 ($p=0.028$), IL-17F ($p=0.006$), TGFa ($p=0.005$), CCL21 ($p=0.036$), CCL1

($p=0.002$), CCL8 ($p=0.036$), and CCL17 ($p=0.047$) were all upregulated in patients with CH. Figure 9 shows levels measured among those with and without CH. We then sought to investigate if specific CH mutations were associated with specific cytokines/chemokines. In HCM patients with sarcomeric mutations, those with CH due to *DNMT3A* (N=16) mutations showed higher levels of IL-9 ($p=0.031$) and CXCL12 ($p=0.046$). *TET2* CH carriers (N=6) showed higher levels of troponin I ($p=0.013$), IL-5 ($p=0.012$), IL-10 ($p=0.010$), CXCL10 ($p=0.001$), CXCL9 ($p=0.012$), CCL4 ($p=0.037$), VEGF-A ($p=0.041$), CCL21 ($p=0.013$), CXCL13 (BCAp=0.012) and CCL1 ($p=0.007$). Finally, CH due to *ASXL1* (N=2) did not show any differences in comparison to those without CH..

Discussion

In this study, we showed that CH prevalence among HCM patients is higher than described in the general population. Fibrosis, a known SCD risk factor, was more prevalent among CH patients with HCM sarcomeric gene mutations. We also observed higher burden of symptoms and higher frequency of ABPR during exercise among CH patients. Finally, CH was associated with higher mortality or orthotopic heart transplant and MACE. Several cytokines, chemokines and troponin were differently expressed in HCM patients with CH, highlighting a plausible causal relationship with the phenotype and outcomes. This is the first cohort in which there is an association between CH prevalence, HCM phenotype and clinical outcomes.

To our knowledge, this is the first study to evaluate the prevalence of CH in patients with HCM. In this study, we found that 22.9% of HCM patients harbored CH.

When restricting analysis to the three specific genes *DNMT3A*, *TET2* and *ASXL1*, we observed that 17% of HCM patients harbored CH mutations.

Fibrosis was more common in those patients with HCM who have a sarcomeric mutation and specific *DNMT3A*, *TET2* and *ASXL1* mutations. This is an important finding as fibrosis is linked to SCD in patients with HCM⁴⁰. In addition, ABPR at exercise, a known marker of SCD risk and worse outcomes^{41–43}, was also more frequent among those with CH and HCM with sarcomeric mutations. This result potentially illustrates that CH could affect the HCM phenotype and promote adverse outcomes in patients with HCM.

We showed that, in subsets of HCM patients and evaluating for overall CH or specific CH mutations, the mortality or need for orthotopic heart transplant is increased among CH patients even when adjusting for confounding factors. Moreover, condensing important major cardiovascular events in HCM, such as stroke, sudden cardiac death, appropriate ICD shock, death or orthotopic heart transplant, CH increases its risk in all subsets of patients, reaching the highest risk among those with sarcomeric mutations and with specific CH genes. Our results show that CH is a new risk factor among HCM patients. In fact, HCM patients with sarcomeric mutations with CH on the most common genes showed the worse survival (17%), contrasting to recent cohorts showing that HCM patients have a low mortality.⁹ Our results do not show a clear evidence of which mechanisms CH increase mortality in HCM, but its strong association with ageing¹³ leads us to hypothesize that the epigenic ageing and the inflammatory milieu may have a causal relationship with the reduced survival.

Among HCM with sarcomeric mutations, CH was associated with several inflammatory cytokines and chemokines such as IL-1ra, IL-6, IL-17F, TGFa, CCL21, CCL1, CCL8, and CCL17, but also with troponin I, a marker of myocyte injury that is associated with clinical outcomes in HCM.⁴⁷ However, specific CH mutations may have distinct prognosis and inflammatory profiles.^{14,34} We observed that *DNMT3A* driven CH was associated with IL-9 and CXCL12. *TET2* driven CH was associated with the higher number of differently expressed cytokines/chemokines including troponin I among all tested genes. *DNMT3A* was associated with LV remodeling and worse outcomes, but the high frequency CH mediated by this gene in the population could be explained by its low lethality and lower burden of inflammation, introducing a survival bias.^{11,19,28,29,34} Cardiac aging processes mediated by *TET2* have shown to produce an enhanced inflammatory background that could be related to hypertrophy and fibrosis, but the worse prognosis associated with *TET2* could also explain its lower frequency in studies.^{11,17,29} *ASXL1* carriers did not show different levels of expressed cytokines, chemokines, BNP or Troponin I. We believe that the low number of patients with this specific mutation could explain the results.

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EXAMPLE 4

We also investigated *TET2* mutant clonal hematopoiesis and its association with the benefit of immunotherapy, as well as metastasis of non-hematological cancers.

Referring to Figure 10, TET2-mutant clonal hematopoiesis is associated with clinical benefit from immunotherapy in melanoma. Figure 10A shows 569 patients with melanoma, bladder cancer, renal cell carcinoma (RCC), or non-small cell lung cancer (NSCLC) treated with immune-checkpoint blockade were screened for clonal hematopoiesis using publicly-available exome sequencing, with at least one mutation of variant allele frequency (VAF) >0.02 detected in 74 patients (datasets EGAD00001006632, SRP064805, SRP067938, SRP072934, SRP090294, SRP095809, SRP115658, SRP128156). Figure 10B shows TET2-mutant clonal hematopoiesis is associated with significantly higher odds of clinical benefit 6 months after receiving immunotherapy in melanoma patients (Odds Ratio = 5.98), but not bladder cancer, renal cell carcinoma, or non-small cell lung cancer patients (p value from Firth's multivariate logistic regression adjusted for patient age, sex, study, and immune checkpoint). Figure 10C shows in an animal model of immunotherapy, mice with Tet2-mutant hematopoiesis – mimicking TET2-mutant clonal hematopoiesis – show enhanced response to PD-1 immune checkpoint blockade, while isotype control (ISO) treated tumours show identical growth kinetics (*p<0.05 by Mann-Whitney Test).

Referring to Figure 11, TET2-mutant clonal hematopoiesis is associated with lower risk of metastatic disease in patients with non-hematological cancers. Figure 11A shows 16,744 patients with metastatic or non-metastatic solid tumours from Nguyen et al. Cell, 2022 were tested for clonal hematopoiesis in Bolton et al. Nature Genetics, 2020 using the MSK-IMPACT targeted sequencing panel. CH mutations with variant allele frequency of at least 0.02 were detected in 19.5% of patients. Figure 11B shows the 5 most commonly detected clonal hematopoiesis mutations from Figure 11A are shown. Figure 11C shows exposure to TET2-mutant CH is associated with lower risk of having metastatic cancer in patients from Figure 11A as assessed by multivariate logistic regression adjusted for age, sex, smoking status, and chemotherapy treatment. Figure 10D shows exposure to TET2-CH is associated with significantly lower risk of having metastases in patients from Figure 11A with Non-Small Cell Lung and Breast Cancer, with a trend towards lower risk of metastases in colorectal and bladder cancer, as assessed by multivariate logistic regression adjusted for age, sex, smoking status, and chemotherapy treatment. Patients in Figure 11A with TET2-mutant CH have significantly few metastases detected on clinical imaging (Figure 11E) and have fewer overall sites / organs involved with metastatic disease (Figure 11F), p values from Wilcoxon Rank Sum Test.

EXAMPLE 5

Applicant further sought to investigate the prevalence of CH among solid organ transplant (SOT) recipients (heart, lung, liver and kidney), study the association with specific CH-related genes and the impact on outcomes.

5 METHODS AND MATERIALS

Study Population

We conducted a retrospective study of 1,500 patients who underwent SOT at the Toronto General Hospital between 2005 and 2021. The inclusion criteria were patients aged 18 years or older with available biospecimens and complete clinical evaluation.

- 10 Patients with active malignancies were excluded, as it is a criterion for solid organ transplant candidacy.

Data Collection

- 15 The electronic health records of the SOT recipients were reviewed to collect demographic data, medical history, laboratory assessments, date of SOT, transplant-related treatment at the time of the procedure, and the immunosuppressive regimen at one year post-SOT. Baseline characteristics, including demographic data and comorbidities, were collected as close as possible to the biospecimens collection date due to their relationship with CH mutations.

Follow-up and Outcomes

- 20 Patients were followed from the time of SOT procedure until the last visit at our institution or death. The following outcomes were evaluated: the first occurrence of acute cellular rejection, CMV infection, de novo post-transplant malignancy, CAV, and death.

Genetic Sequencing

- 25 Peripheral blood samples from patients who had provided consent during their visits to Toronto General Hospital. These samples were stored in the biobank of the Peter Munk Cardiac Centre. To construct the sequencing libraries, 100ng of DNA was utilized, and single molecule Molecular Inversion Probes (smMIP) were employed. The smMIPs were utilized to capture 35 genes known to be recurrently mutated in myeloid

neoplasms. Sequencing was performed using the Illumina Novaseq platform, and an in-house computational pipeline was used to reduce artifacts and false-positive mutation calls. Alleles were filtered based on a P-value cut-off of 0.05 and were manually inspected based on several criteria, including base-pair change, annotation
5 in COSMIC, minor allele frequency, and number of reads supporting the alternative allele. The resulting variants were analyzed and manually inspected to avoid selecting false positives.

Statistical analysis

The normality of all variables was assessed using the Shapiro-Wilk test, and both
10 normal and non-normal continuous variables are reported as mean $\pm$ standard deviation or median (interquartile range). Student's t-test or Mann-Whitney test were used to evaluate differences among groups depending on the normality of the data. Categorical variables were assessed using chi-square test or Fisher's exact test. All analyses were performed using SPSS, version 25.0 (SPSS Inc., NY, USA), with a
15 statistical significance level of 0.05 and a two-sided p-value.

RESULTS AND DISCUSSION

Out of the 1,500 patients enrolled, 782 have been sequenced as of the current date. Among these patients, 127 (16.2%) underwent heart transplant, 90 (11.5%) underwent lung transplant, 189 (24.2%) underwent kidney transplant, 374 (47.8%) underwent liver
20 transplant, and 2 (0.3%) underwent lung-liver transplant. The average age of the entire cohort was 54.2 ± 12.7 years, with a majority of male patients (52.9%). The age distribution varied significantly across organ groups ($p < 0.0001$), with lung-liver transplant patients being the youngest (47 ± 12.7), followed by heart (49.2 ± 14.0), kidney (53.7 ± 13.3), lung (54.4 ± 15.1) and liver (56.1 ± 10.8) transplant patients. Sex also
25 differed among the organ groups ($p = 0.05$), with all lung-liver transplant recipients being male, and a majority of heart (76.4%), kidney (76.2%), liver (66.5%), and lung (58.9%) transplant recipients being male. For a summary of the overall cohort characteristics, refer to Table 8.

CH was observed in 123 (15.7%) patients. CH prevalence was similar across the
30 organ groups (Figure 12), except for kidney (8%) that was lower than heart (20%), lung (19%), and (liver 18%), $p = 0.007$. Lung-liver showed a 50% of CH, but the low number of patients may explain this result.

We then analyzed the specific mutated genes among patients with CH. In the literature, *DNMT3A* is the most common mutated gene in the general population, followed by *TET2*.^[13] In our cohort, *DNMT3A* was the most commonly mutated gene (n=45, 5.7%), followed by *TET2* (n=40, 5.1%) and *ASXL1* (n=20, 2.5%). Notably, *DNMT3A* was the most commonly mutated gene among heart transplant recipients (Figure 15) and among kidney recipients (Figure 17), while *TET2* was the most common mutated gene among lung (Figure 16) and liver (Figure 18) recipients. These findings suggest that *TET2* may play a role in lung and liver end-stage disease, but further clinical validation is required.

- 10 The overall mortality rate in our cohort was 18.3%. Mortality differed across the different SOT ($p < 0.0001$), with the highest mortality observed in lung-liver recipients (50%), followed by lung (43.3%), heart (20.5%), liver (14.2%), and kidney (12.7%) recipients (Figure 19). We observed a trend in mortality according to the presence of CH (17.4% vs. 23.4%) (Figure 20). Among solid organ transplant recipients, those who
- 15 received a heart transplant showed a higher mortality rate when developing CH (14% vs. 42%, $p = 0.002$) with a trending shown for lung. CH did not affect the mortality of recipients of kidney or liver (Figure 21).

- The study partial results shows that CH was observed in 15.7% of patients, with similar prevalence across most organ groups except for kidney, which had a lower
- 20 prevalence. *DNMT3A* was the most commonly mutated gene among heart and kidney transplant recipients, while *TET2* was the most common in lung and liver recipients. Heart transplant recipients had a higher mortality rate when developing CH.

- Although preferred embodiments of the invention have been described herein, it will be understood by those skilled in the art that variations may be made thereto without
- 25 departing from the spirit of the invention or the scope of the appended claims. All documents disclosed herein, including those in the following reference list, are incorporated by reference.

TABLE 1. CHARACTERISTICS OF THE PATIENTS AT BASELINE^A

	Cardiogenic Shock (N=341)	Ambulatory HF (N=345)	p value
Matched variables			
Age (years)	55±15	57±15	0.25
- median (range)	58 (19)	58 (19)	
Male sex	249 (73%)	247 (72%)	0.64
Heart failure aetiology			
Ischemic cardiomyopathy	107 (31%)	106 (31%)	0.85
Non-ischemic cardiomyopathy	221 (65%)	225 (65%)	0.91
Congenital disease	13 (4%)	14 (4%)	0.86
Ejection fraction (%)	26±12	27±10	0.09
Other baseline characteristics			
Body mass index (kg/m ²) ^b	26±6	29±6	<0.001
Hypertension	125 (37%)	109 (32%)	0.16
Dyslipidaemia	115 (34%)	122 (35%)	0.65
Diabetes	99 (29%)	78 (23%)	0.05
Prior/current smoker	101 (30%)	82 (24%)	<0.001
Prior history of cancer	33 (10%)	41 (12%)	0.35
Prior coronary revascularization	69 (20%)	84 (24%)	0.19
Previous cerebrovascular disease/transient ischemic attack	39 (11%)	33 (10%)	0.42
Atrial Fibrillation/Flutter	122 (36%)	101 (29%)	0.06
Chronic Heart Failure	237 (70%)	345 (100%)	<0.001
Implantable cardioverter-defibrillator	85 (25%)	106 (31%)	0.09
Cardiac resynchronization therapy	69 (20%)	70 (20%)	0.98
White blood count (x10 ⁹ /L)	10.7±6.6	7.3±2.4	<0.001
Creatinine (µmol/L)	179±128	110±73	<0.001
Sodium (mmol/L)	135±6	138±3	<0.001
B-type natriuretic peptide (pg/mL)	1,428 (1730)	168 (337)	<0.001

^aPlus-minus are means±SD.^bThe body-mass index is the weight in kilograms divided by the square of the height in meters.

Table 2. Characteristics of Cardiogenic Shock Patients^a

	All cardiogenic shock (N=341)	No clonal haematopoiesis (N=256)	Clonal haematopoiesis (N=85)	P value
Age (year)	55±15	54±15	60±14	0.002
Male sex	249 (73%)	188 (73%)	61 (72%)	0.76
Body mass index (kg/m ²) ^b	26±6	26±6	26±6	0.50
Hypertension	125 (37%)	87 (34%)	38 (44%)	0.09
Dyslipidaemia	115 (34%)	78 (30%)	37 (44%)	0.03
Diabetes	99 (29%)	72 (28%)	27 (31%)	0.57
Prior/current smoker	101 (30%)	75 (29%)	25 (29%)	0.89
Prior history of cancer	33 (10%)	25 (10%)	9 (11%)	0.86
Prior coronary revascularization	69 (20%)	52 (20%)	17 (20%)	0.90
Previous cerebrovascular disease/transient ischemic attack	39 (11%)	27 (11%)	12 (14%)	0.39
Cardiogenic shock aetiology				
Acute decompensation of chronic heart failure	225 (66%)	168 (66%)	57 (67%)	0.94
New onset of heart failure	93 (27%)	72 (28%)	21 (25%)	0.27
Myocardial infarction	10 (3%)	6 (2%)	4 (5%)	0.87
Myocarditis	11 (3%)	8 (3%)	3 (4%)	0.58
SCAI classification ^c				
B	27 (8%)	21 (8%)	6 (7%)	0.73
C	27 (8%)	20 (8%)	7 (8%)	0.90
D	285 (83%)	213 (83%)	72 (85%)	0.74
E	2 (1%)	2 (1%)	0	0.41
Number of inotropes or vasopressors	1.7±1.0	1.6±1.0	1.8±1.0	0.24
Pulmonary artery catheter	178 (52%)	134 (52%)	44 (52%)	0.92
Temporary mechanical				

circulatory support				
Intra-aortic balloon pump	26 (8%)	20 (8%)	6 (7%)	0.50
Impella device®	8 (2%)	8 (3%)	0	0.09
Extra-corporeal life support	18 (5%)	16 (6%)	3 (4%)	0.59
Mechanical ventilation	63 (18%)	45 (18%)	18 (21%)	0.45
Renal replacement therapy	35 (10%)	24 (9%)	11 (13%)	0.34
White blood count (x10 ⁹ /L)	7.3±2.4	11.1±7.2	9.4±4.3	0.01
Creatinine (μmol/L)	110±73	176±129	188±124	0.44
Sodium (mmol/L)	138±3	135±6	133±5	0.02
Peak lactate (mmol/L)	3.4±3.2	3.4±3.1	3.4±3.1	0.86
B-type natriuretic peptide (pg/mL)	1428(1730)	1446(1712)	1350(1737)	0.42

^aPlus-minus are mean ± SD.

^bThe body-mass index is the weight in kilograms divided by the square of the height in metres.

- 5 ^cPatients were classified according to the Society for Cardiovascular Angiography & Interventions. Briefly, Class B are patients at beginning of shock; Class C is classic cardiogenic shock; Class D is deteriorating and failure to respond to initial interventions; and Class E is extreme shock on patients supported by multiple interventions who may be experiencing cardiac arrest and/or extracorporeal life support.

Table 3. Clinical characteristics of OHT recipients and according to CH status.

Characteristics	All (N=127)	No CH (N=101)	CH (N=26)	P value
Age - year	49±14	48±14	54±13	0.04
Median (range)	53 (23)	52 (24)	58 (21)	0.03
Male sex – no. (%)	97 (77%)	79 (78%)	18 (69%)	0.33
Body mass index - kg/m ² †	25±6	25±6	25±5	0.97
Hypertension – no. (%)	33 (26%)	25 (25%)	8 (31%)	0.53
Dyslipidemia – no. (%)	33 (26%)	24 (24%)	9 (35%)	0.26
Diabetes – no. (%)	28 (22%)	23 (23%)	5 (19%)	0.69
Prior smoker – no. (%)	34 (27%)	25 (25%)	9 (35%)	0.31
Prior neoplasia– no. (%)	8 (6%)	8 (8%)	None	0.20
Heart failure etiology– no. (%)				0.41
Ischemic cardiomyopathy	28 (22%)	20 (20%)	8 (31%)	
Non-ischemic cardiomyopathy– no. (%)	90 (71%)	73 (72%)	17 (65%)	
Congenital disease– no. (%)	9 (7%)	8 (8%)	1 (4%)	
Previous VAD - no. (%)	44 (35%)	37 (37%)	7 (27%)	0.35
White blood count (x10 ⁹ /L)	8.8±4.4	7.9±4.5	6.9±3.5	0.11
Sodium (mmol/L)	134±5	136±5	135±6	0.53
Creatinine (mmol/L)	117 (92)	122 (86)	116 (127)	0.65
Induction therapy				
Thymoglobulin	123 (97%)	97 (96%)	26 (100%)	0.30
Basiliximab	4 (3%)	4 (4%)	None	
Primary graft dysfunction	19 (15%)	17 (17%)	2 (8%)	0.24

In-hospital death post OHT	12 (9%)	10 (10%)	2 (8%)	0.73
MCS post-OHT	18 (14%)	17 (17%)	1 (4%)	0.09
Follow-up from OHT, years	3.2±2.6	2.9±2.1	4.3±3.9	0.01
Donor CMV+	57 (46%)	45 (46%)	12 (46%)	0.98
Recipient CMV+	80 (65%)	63 (64%)	17 (65%)	0.91
Donor EBV+	97 (14%)	81 (80%)	16 (62%)	0.02
Recipient EBV+	115 (93%)	89 (91%)	26 (100%)	0.10
Immunosuppressive therapy at discharge post-OHT				
Tacrolimus	119 (94%)	97 (96%)	22 (85%)	0.03
Sodium mycophenolate	123 (97%)	100 (99%)	23 (89%)	0.006
Steroids	127 (100%)	101 (100%)	26 (100%)	-
MTOR inhibitor	1 (1%)	None	1 (4%)	0.04
Cyclosporine	6 (5%)	3 (3%)	3 (12%)	0.06
Azathioprine	1 (1%)	None	1 (4%)	0.04
Immunosuppressive therapy at 1 year after OHT				
Tacrolimus	80 (86%)	64 (87%)	16 (84%)	0.79
Sodium mycophenolate	71 (77%)	58 (80%)	13 (68%)	0.30
Steroids	87 (94%)	68 (82%)	19 (100%)	0.19
MTOR inhibitor	17 (19%)	11 (15%)	6 (32%)	0.09
Cyclosporine	8 (9%)	5 (7%)	3 (16%)	0.21
Azathioprine	2 (2%)	1 (1%)	1 (5%)	0.30

CH, clonal hematopoiesis; CMV, cytomegalovirus; EBV, Epstein-barr virus; HF, heart failure MCS, mechanical circulatory support; OHT, orthotopic heart transplant; VAD, ventricular assist device; VAF, variant allele frequency;

Table 4. Clinical outcomes in patients with OHT according to CH mutations				
	All (N=127)	No CH (N=101)	CH (N=26)	P- value
CMV infection	59 (46%)	48 (47.5%)	11 (42.3%)	0.63
Acute cellular rejection 2/3R	69 (54%)	51 (53%)	18 (69%)	0.14
Post-transplant malignancy	11 (9%)	9 (9%)	2 (8%)	0.84
CAV grade 2 or 3	4 (3%)	None	4 (18%)	<0.001
Mortality	26 (20%)	15 (15%)	11 (42%)	0.008

CAV, cardiac allograft vasculopathy; CH, clonal hematopoiesis; CMV, cytomegalovirus

ble 5. List of clonal hematopoiesis mutations detected among orthotopic heart transplant recipients.

Sample ID	Gene	Chromosome	Start position	End position	Variant type	Variant type	Ref. Allele	Alt. Allele	Protein change	Alt. reads	Ref. reads	VAF
:CHCVD_0004	CEBPA	chr19	33792312	33792312	Missense_Variant	SNV	T	A	p.Thr337Ser	442	656	0.67378049
:CHCVD_0006A	ASXL1	chr20	31023894	31023894	Missense_Variant	SNV	G	A	p.Asp1127Asn	2559	13221	0.19355571
:CHCVD_0010B	DNMT3A	chr2	25463562	25463562	Frame_Shift_Del	indel	C	-	p.Gly707fs	791	32884	0.02405425
:CHCVD_0010B	DNMT3A	chr2	25467086	25467086	Frame_Shift_Del	indel	G	-	p.Arg597fs	277	92757	0.0029863
:CHCVD_0010B	FLT3	chr13	28608064	28608064	Missense_Variant	SNV	T	A	p.Lys634Asn	19623	42835	0.45810669
:CHCVD_0011	DNMT3A	chr2	25463187	25463187	Missense_Variant	SNV	A	G	p.Ile769Thr	496	16037	0.03092848
:CHCVD_0014	ASXL1	chr20	31024221	31024221	Missense_Variant	SNV	T	G	p.Ser1236Ala	6179	37380	0.1653023
:CHCVD_0017	ASXL1	chr20	31022344	31022344	Missense_Variant	SNV	G	A	p.Gly610Asp	1265	7311	0.17302695
:CHCVD_0017	BCOR	chrX	39933074	39933074	Missense_Variant	SNV	A	G	p.Trp509Arg	745	46356	0.01607127
:HCVD_0017	DNMT3A	chr2	25470612	25470612	Missense_Variant	SNV	G	A	p.Arg288Trp	1350	4998	0.27010804
CVD_0017	SF3B1	chr2	198266552	198266552	Missense_Variant	SNV	C	T	p.Ala762Thr	734	24252	0.03026555
:HCVD_0017	TP53	chr17	7577127	7577127	Stop_Gained	SNV	C	A	p.Glu271*	619	6642	0.09319482
:HCVD_0029	DNMT3A	chr2	25469086	25469086	Missense_Variant	SNV	G	A	p.Arg458Trp	416	4097	0.10153771
:CHCVD_0038	DNMT3A	chr2	25468174	25468174	Missense_Variant	SNV	T	C	p.Asn501Ser	9671	29430	0.32861026
:CHCVD_0038	TP53	chr17	7578371	7578371	Missense_Variant	SNV	C	G	p.Gly187Arg	133	17741	0.00749676
ARCHCVD_0038	TP53	chr17	7578380	7578380	Missense_Variant	SNV	C	G	p.Asp184His	133	17740	0.00749718
ARCHCVD_0041	TP53	chr17	7578380	7578380	Missense_Variant	SNV	C	G	p.Asp184His	142	20782	0.00683284
ARCHCVD_0043	IDH2	chr15	90631946	90631946	Missense_Variant	SNV	T	C	p.Asn136Ser	2076	5343	0.38854576
ARCHCVD_0046	DNMT3A	chr2	25470590	25470590	Missense_Variant	SNV	A	T	p.Leu295Gln	570	8500	0.06705882
ARCHCVD_0055	TET2	chr4	106158134	106158134	Missense_Variant	SNV	C	T	p.Pro1012Leu	3565	9098	0.39184436
ARCHCVD_0060	ASXL1	chr20	31023667	31023667	Missense_Variant	SNV	G	A	p.Arg1051His	841	9827	0.08558054
ARCHCVD_0060	TET2	chr4	106158560	106158560	Missense_Variant	SNV	A	G	p.Asn1154Ser	2104	7665	0.27449446
ARCHCVD_0096	TET2	chr4	106157326	106157326	Stop_Gained	SNV	C	T	p.Gln743*	200	28409	0.00704002
ARCHCVD_0100	DNMT3A	chr2	25463568	25463568	Missense_Variant	SNV	A	G	p.Ile705Thr	375	12290	0.03051261
ARCHCVD_0100	PTPN11	chr12	112910785	112910785	Missense_Variant	SNV	G	A	p.Arg265Gln	238	21688	0.01097381
ARCHCVD_0114	DNMT3A	chr2	25467491	25467491	Missense_Variant	SNV	C	T	p.Asp529Asn	170	10755	0.0158066

.CHCVD_0117	DNMT3A	chr2	25467124	25467124	Frame_Shift_Ins	indel	T	+	p.Tyr584fs	1599	36285	0.0440678
.CHCVD_0117	PPM1D	chr17	58740549	58740549	Frame_Shift_Del	indel	G	-	p.Ile486fs	77	24796	0.00310534
.CHCVD_0117	PPM1D	chr17	58740604	58740607	Frame_Shift_Del	indel	AACA	-	p.Asn505fs	70	29465	0.0023757
.CHCVD_0117	PPM1D	chr17	58740714	58740714	Frame_Shift_Del	indel	A	-	p.Glu540fs	362	22820	0.01586328
.CHCVD_0117	PPM1D	chr17	58740732	58740732	Frame_Shift_Del	indel	T	-	p.Leu546fs	262	22819	0.01148166
.CHCVD_0117	TP53	chr17	7577526	7577526	Missense_Variant	SNV	A	T	p.Leu252His	278	15061	0.01845827
.CHCVD_0128	DNMT3A	chr2	25469633	25469633	Missense_Variant	SNV	G	A	p.Arg379Cys	4474	62094	0.07205205
.CHCVD_0128	TP53	chr17	7577556	7577556	Missense_Variant	SNV	C	T	p.Cys242Tyr	422	40218	0.01049281
.CHCVD_0147	ASXL1	chr20	31023190	31023190	Missense_Variant	SNV	C	A	p.Ser892Tyr	228	36353	0.00627183
.CHCVD_0149	DNMT3A	chr2	25463196	25463197	Frame_Shift_Del	indel	TT	-	p.Lys766fs	1027	37205	0.02760382
.CHCVD_0165	TP53	chr17	7577120	7577120	Missense_Variant	SNV	C	G	p.Arg273Pro	4705	12830	0.36671863
.CHCVD_0166	DNMT3A	chr2	25463236	25463238	Frame_Shift_Del	indel	A	-	p.Trp753fs	1226	55482	0.02209726
.HCVD_0166	DNMT3A	chr2	25463586	25463586	Missense_Variant	SNV	C	T	p.Gly699Asp	658	13363	0.04924044
.HCVD_0166	DNMT3A	chr2	25464455	25464455	Missense_Variant	SNV	G	C	p.Asp886Glu	145	12401	0.01169261
.HCVD_0170	DNMT3A	chr2	25463583	25463583	Missense_Variant	SNV	G	A	p.Pro700Leu	172	15467	0.01112045
.HCVD_0200	PHF6	chrX	133549136	133549136	Stop_Gained	SNV	C	T	p.Arg274*	895	62494	0.01432137
.CHCVD_0210	SMC1A	chrX	53431967	53431967	Stop_Gained	SNV	G	A	p.Arg725*	697	13878	0.05022338
.CHCVD_0227	DNMT3A	chr2	25462078	25462078	Missense_Variant	SNV	G	A	p.Pro777Ser	547	19125	0.02860131
ARCHCVD_0227	PPM1D	chr17	58740522	58740522	Frame_Shift_Del	indel	A	-	p.Asn477fs	728	13283	0.0548069
ARCHCVD_0241	RUNX1	chr21	36252975	36252975	Missense_Variant	SNV	C	T	p.Leu129Leu	203	40261	0.0050421
ARCHCVD_0296	DNMT3A	chr2	25458637	25458637	Stop_Gained	SNV	G	A	p.Gln846*	196	7986	0.02454295
ARCHCVD_0331	DNMT3A	chr2	25470583	25470583	Stop_Gained	SNV	C	T	p.Trp297*	152	14952	0.01016586
ARCHCVD_0398	ASXL1	chr20	31022341	31022341	Missense_Variant	SNV	C	T	p.Thr609Ile	5571	12858	0.43327112
ARCHCVD_0456	GATA2	chr3	128200154	128200154	Missense_Variant	SNV	C	G	p.Arg384Thr	127	47555	0.00267059
ARCHCVD_0492	TET2	chr4	106157803	106157810	Frame_Shift_Del	indel	AGAA ACCA	-	p.Asn903fs	240	70735	0.00339295
ARCHCVD_0563	BCOR	chrX	39933121	39933121	Missense_Variant	SNV	C	T	p.Gly493Asp	58	29158	0.00198916
ARCHCVD_0563	EZH2	chr7	148506185	148506185	Missense_Variant	SNV	C	G	p.Glu725Gln	59	24362	0.0024218
ARCHCVD_0568	EZH2	chr7	148504787	148504787	Missense_Variant	SNV	G	C	p.Ala736Gly	134	25452	0.00526481
ARCHCVD_0568	STAG2	chrX	123220456	123220456	Missense_Variant	SNV	G	C	p.Trp1038Leu	99	40520	0.00244324

:CHCVD_0585	TP53	chr17	7578278	7578278	Missense_Variant	SNV	G	C	p.Pro191Ala	118	31392	0.00375892
:CHCVD_0603	JAK2	chr9	5073739	5073739	Missense_Variant	SNV	C	A	p.His606Gln	9294	25742	0.36104421
:CHCVD_0614	TET2	chr4	106197039	106197039	Missense_Variant	SNV	C	G	p.Ser1791Cys	146	29756	0.00490657
:CHCVD_0629	CEBPA	chr19	33792987	33792987	Missense_Variant	SNV	G	T	p.Pro112Thr	128	130	0.98461538
:CHCVD_0636	DNMT3A	chr2	25457243	25457243	Missense_Variant	SNV	G	A	p.Arg882Cys	6936	28768	0.02411012
:CHCVD_0636	DNMT3A	chr2	25469096	25469096	Frame_Shift_Del	indel	G	-	p.Lys456fs	659	10067 ₀	0.00654601
:CHCVD_0636	DNMT3A	chr2	25469162	25469162	Stop_Gained	SNV	G	C	p.Tyr432*	897	91092 ₂	0.00984719
:CHCVD_0644	TET2	chr4	106155429	106155429	Missense_Variant	SNV	G	T	p.Lys110Asn	476	19776	0.02406958
:CHCVD_0657	BCOR	chrX	39933039	39933039	Frame_Shift_Del	indel	G	-	p.Asn520fs	461	41910	0.01099976
:CHCVD_0673	PPM1D	chr17	58740698	58740698	Stop_Gained	SNV	A	T	p.Lys535*	3230	36839	0.08767882
:CHCVD_0680	ASXL1	chr20	31021109	31021109	Missense_Variant	SNV	T	A	p.Ser370Thr	12555	20108	0.62437836

	All-CH			DNMT3A, TET2, ASXL1		
	No CH (N=169)	CH (N=37)	p	No CH (N=182)	CH (N=23)	p
Echocardiogram						
MLVWT, mean (SD), mm	17.3 (4.6)	17.6 (5.1)	0.765	17.4 (4.7)	17.5 (4.9)	0.874
LA diameter, mm	3.9 (1.2)	3.8 (1.1)	0.651	3.9 (1.2)	3.7 (1.4)	0.642
LAVi, ml/m ²	38.5 (30.8)	41.3 (20.7)	0.599	39.5 (30.2)	35.6 (20.3)	0.547
LV EF, %	58.2 (15.3)	62.4 (6.2)	0.106	58.5 (14.8)	63.0 (7.3)	0.156
LVOT maximal gradient, mm Hg	17.2 (26.6)	22.5 (40.3)	0.316	17.6 (27.4)	22.6 (43.5)	0.442
SAM, No. (%)	76 (45.0)	21 (56.8)	0.193	84 (45.9)	13 (56.5)	0.336
Moderate-severe MR, No. (%)	7 (4.1)	1 (2.7)	0.681	8 (4.4)	0	0.306
Cardiac MRI						
LV mass, g	137.3 (52.9)	143.1 (61.4)	0.601	137.8 (52.9)	142.3 (65.5)	0.728
LV mass index, g/m ²	72.1 (25.5)	74.7 (27.2)	0.622	72.5 (25.3)	73.1 (30.2)	0.920
MLVWT, mm	18.9 (6.4)	19.2 (5.6)	0.814	19.0 (6.4)	19.0 (5.8)	0.980
LV EF, %	60.8 (8.3)	62.1 (6.2)	0.390	61.0 (8.2)	61.1 (5.8)	0.979
LGE, No (%)	134 (79.3)	27 (73.0)	0.400	144 (78.7)	17 (73.9)	0.601
LGE >15%, No (%)	26 (17.7)	11 (33.3)	0.044	30 (18.8)	7 (35.0)	0.090
LV mass % of LGE ¹ , %	12.2 (7.7-17.6)	15.6 (10.4-24.6)	0.068	12.2 (7.7-17.5)	21.2 (13.7-41.8)	0.014
Apical Aneurysm, No (%)	1 (0.6)	1 (2.7)	0.145	2 (1.1)	0	0.613
Syncope, No (%)	6 (3.6)	3 (8.1)	0.219	7 (3.8)	2 (8.7)	0.281
ABPR at exercise, No. (%) (85/15)	25 (29.8)	10 (62.5)	0.012	27 (30.7)	8 (66.7)	0.014
NSVT, No. (%) (145/21)	52 (37.7)	11 (39.3)	0.873	56 (37.6)	7 (41.2)	0.772
Stroke, No. (%)	2 (1.2)	2 (5.4)	0.092	3 (1.6)	1 (4.3)	0.375
Appropriate ICD shock, No. (%)	2 (1.2)	0	0.506	2 (1.1)	0	0.614
Cardiac arrest, No. (%)	0	0	n/a	0	0	n/a
Death or orthotopic heart transplant, No. (%)	2 (1.2)	3 (8.1)	0.013	2 (1.1)	3 (13.0)	<0.0001
MACE, No. (%)	5 (3.0)	5 (13.5)	0.008	6 (3.4)	4 (17.4)	.004

¹Quantified LGE>5%.

ABPR, abnormal blood pressure response; EF, ejection fraction; LA, left atrium diameter; LAVi, left atrium volume index; ICD, implantable cardioverter defibrillator; LGE, late gadolinium enhancement; LV, left ventricle; LVOT, left ventricular outflow tract; MACE, major cardiovascular events; MLVWT, maximal left ventricular wall thickness; MR, mitral regurgitation; MRI, magnetic resonance imaging; NSVT, non-sustained ventricular tachycardia; SAM, systolic anterior motion.

Table 7. Overall characteristics of the HCM cohort and between those with or without CH.				
	All (N=799)	No CH (N=616)	CH (N=183)	P
Age, mean (SD), years	55.7 (14.6)	55.3 (14.5)	57.2 (14.8)	0.126
Age at diagnosis, mean (SD), years	47.8 (15.8)	47.3 (15.6)	49.6 (16.3)	0.089
Male sex, No. (%)	547 (68.5)	424 (68.8)	123 (67.2)	0.679
Body mass index ¹ , mean (SD), kg/m ²	29.0 (6.9)	29.0 (7.1)	29.2 (6.2)	0.647
Hypertension, No. (%)	315 (39.4)	233 (37.8)	82 (44.8)	0.093
Diabetes, No. (%)	104 (13.0)	78 (12.6)	26 (14.2)	0.863
Prior/current smoker, No. (%)	155 (19.3)	119 (19.3)	36 (19.6)	0.637
Coronary artery disease, No. (%)	73 (9.1)	61 (9.9)	12 (6.5)	0.166
Atrial fibrillation, No. (%)	143 (17.8)	107 (17.3)	36 (19.6)	0.493
Genetic testing, No. (%)	712 (89.1)	554 (89.9)	158 (86.3)	0.170
Pathogenic/Likely pathogenic variant, No. (%)	206 (25.8)	169 (30.5)	37 (23.4)	0.083
<i>MYH7</i> , No. (%)	53 (25.7)	43 (25.6)	10 (27.0)	0.664
<i>MYBPC3</i> , No. (%)	127 (61.6)	106 (63.1)	21 (56.8)	
Other, No. (%)	26 (12.6)	20 (11.8)	6 (16.2)	
Family history of HCM, No. (%)	251 (31.4)	194 (31.5)	57 (31.1)	0.087
Family history of SCD, No. (%)	37 (4.6)	26 (4.2)	11 (6.0)	0.048
Implantable cardioverter defibrillator, No. (%)	111 (13.9)	89 (14.4)	22 (12.0)	0.405
Pacemaker, No. (%)	13 (1.6)	6 (1.0)	7 (3.8)	0.007
NYHA II-IV, No. (%)	235 (29.4)	182 (29.5)	53 (28.9)	0.803
Beta-blocker, No. (%)	421 (52.7)	321 (52.1)	100 (54.6)	0.547
Non-dihydropyridine calcium channel blocker, No. (%)	116 (14.5)	77 (12.5)	39 (21.3)	0.003
Disopyridine, No. (%)	67 (8.4)	51 (8.3)	16 (8.7)	0.842
Diuretic, No. (%)	81 (10.1)	59 (9.5)	22 (12.0)	0.303
Septal reduction therapy, No. (%)	76 (9.5)	53 (8.6)	23 (12.5)	0.130
Abbreviations: CH, clonal hematopoiesis; HCM, hypertrophic cardiomyopathy; SCD, sudden cardiac death				
¹ Body mass index calculated as weigh (kg)/height ² (m)				

Table 8. Clinical characteristics of OHT recipients and according to CH status.				
Characteristics	All (N=782)	No CH (N=659)	CH (N=123)	P value
Age - year	54.2±12.7	54.1±12.8	54.4±12.3	0.795
Median (range)		57 (48-63)	58 (46-63)	
Male sex	52.9%	68%	67.3%	0.882
Organ				
Heart	127 (16.2%)	101 (15.3%)	26 (21.1%)	0.007
Lung	90 (11.5%)	73 (11.1%)	17 (13.8%)	
Kidney	189 (24.2%)	174 (26.4%)	15 (12.2%)	
Liver	374 (47.8%)	310 (47.0%)	64 (52%)	
Lung-Liver	2 (0.3%)	1 (0.2%)	1 (0.8%)	

Table A - SmMIP Probe List

>mip_key	logistic_scor	chr	art	ext_probe_st	ext_probe_st	ext_pr	ext_probe_sequence	SEQ ID	lig_probe_st	lig_probe_st	lig_probe_st
	e			op	obe_c	op		ID	op	op	_copy
1:43814899-43815068/26,18/-	0.78875	1	43815043	43815068	1	1	TGGTCCACGCCAGTCTCTGCGCTGG	1	43814916	1	1
1:115256459-115256628/21,23/+	0.981109	1	115256459	115256479	1	1	GATGGCAATACACAGAGAA	2	115256628	1	1
1:115258635-115258804/21,23/-	0.951804	1	115258784	115258804	1	1	CAACAGTTCTTGTGCTGTG	3	115258657	1	1
2:25457124-25457293/22,22/+	0.833844	2	25457124	25457145	2	1	TACCTCAGTTTGCCCCCATGTC	4	25457293	1	1
2:25457152-25457321/20,24/-	0.839643	2	25457302	25457321	2	1	TCACCTGCGCTCTCTGCGCT	5	25457175	1	1
2:25458549-25458718/20,24/-	0.96996	2	25458699	25458718	2	1	TCTCTTTTCTCTCTTTCAT	6	25458572	1	1
2:25459749-25459918/21,23/-	0.860238	2	25459898	25459918	1	1	GTATCCAGGTTTCTGTGTT	7	25459771	1	1
2:25461967-25462136/22,22/-	0.90969	2	25462115	25462136	1	1	GCTGTTTCACTGCTCTCTTGG	8	25461988	1	1
2:25463123-25463292/20,24/+	0.881523	2	25463123	25463142	1	1	AGGTAGAAAGCCATTAGTGAG	9	25463292	1	1
2:25463185-25463354/20,24/-	0.871458	2	25463335	25463354	2	1	GCCCCAGCTGATGCTTTCT	10	25463208	1	1
2:25463484-25463653/20,24/+	0.917457	2	25463484	25463503	1	1	GTGGAGGGGACAGGATGGTA	11	25463653	1	1
2:25464510-25464629/20,24/+	0.808999	2	25464510	25464529	1	1	TCCTCACACACCTCCGAGGC	12	25464629	1	1
2:25464407-25464566/24,20/-	0.825581	2	25464543	25464566	1	1	GCTGAAAGGACTTGGCATTCAGGT	13	25464426	1	1
2:25466743-25466912/20,24/-	0.909729	2	25466893	25466912	1	1	GCTGGTGGGAGCTTGGGAC	14	25466766	1	1
2:25467012-25467163/20,24/+	0.81207	2	25467012	25467031	1	1	CCAGCACTCACAAATTCCT	15	25467163	1	1
2:25466991-25467110/22,22/-	0.807386	2	25467089	25467110	1	1	AAGGGTACCTACGGGCTGCTGC	16	25467012	1	1
2:25467117-25467237/22,22/-	0.81286	2	25467216	25467237	1	1	GTCTCTCTGCTCAGTGGTCT	17	25467138	1	1
2:25467383-25467552/23,21/-	0.896693	2	25467530	25467552	1	1	GTGGTTTCTGACCTTCCGCTG	18	25467403	1	1
2:25468100-25468249/20,24/+	0.927609	2	25468100	25468119	1	1	TCCTGGGTGGGTGCTCTCT	19	25468249	1	1
2:25468075-25468224/23,21/-	0.855948	2	25468202	25468224	1	1	CATCCCTCCCTGCTTCCAG	20	25468095	1	1
2:25468817-25468986/24,20/+	0.721162	2	25468817	25468840	1	1	CTCTAAGTGCTCTGCTACTCT	21	25468986	1	1
2:25468993-25469162/20,24/+	0.826636	2	25468993	25469012	2	1	TCCTGGTGCCACCTCTCCA	22	25469162	1	1
2:25469087-25469256/24,20/+	0.953003	2	25469087	25469110	1	1	GGGCTTTTGGCTGGTGAGGTGG	23	25469256	1	1
2:25469510-25469676/20,24/+	0.700311	2	25469510	25469529	1	1	TAGGGCCAGAAAGGCTGGAAG	24	25469676	1	1
2:25469385-25469551/22,22/-	0.787747	2	25469530	25469551	1	1	TGATTGAATGGGCTGGGGGG	25	25469406	1	1
2:25469926-25470095/23,21/+	0.871049	2	25469926	25469948	1	1	GACCTCGTAGATGGCTTTCGCT	26	25470095	1	1
2:25469859-25470028/22,22/-	0.898607	2	25470007	25470028	1	1	GGTGTGTTTGAGAAAGCTGATG	27	25469880	1	1
2:25470436-25470599/22,22/+	0.833016	2	25470436	25470457	1	1	CTACTGCCAAACCCCAACTT	28	25470599	1	1
2:25470523-25470685/23,21/-	0.911389	2	25470663	25470685	1	1	ATGGGGGATCAGGGTGGCAGGG	29	25470543	1	1
2:25470913-25471078/26,18/+	0.711861	2	25470913	25470938	1	1	TCTGGCTGTCATCGCTGCTTTGGT	30	25471078	1	1
2:198266544-198266713/20,24/+	0.804959	2	198266544	198266563	1	1	TGATTTGGCATATTTCTGCAT	31	198266713	1	1
2:198266417-198266586/23,21/-	0.890112	2	198266564	198266586	1	1	TGGGTAICTTATTCCTTTATGG	32	198266437	1	1
2:198266730-198266899/20,24/-	0.956695	2	198266880	198266899	1	1	TAGGTAATGTTGGGGCATAG	33	198266753	1	1
2:198267419-198267588/22,22/+	0.934101	2	198267419	198267440	1	1	GGGCAATAAAGAGGAATGCC	34	198267588	1	1
2:198267315-198267484/21,23/-	0.947509	2	198267464	198267484	1	1	CGTAACACAAACAGCTAGACT	35	198267337	1	1
2:209113063-209113232/24,20/+	0.895571	2	209113063	209113086	1	1	CAAAATCACATTATTGCCAACATG	36	209113232	1	1
3:128200021-128200186/21,23/-	0.950888	3	128200166	128200186	1	1	TCTGAAACTGGTGGTTCCT	37	128200043	1	1
3:128200661-128200783/24,20/+	0.899098	3	128200661	128200684	1	1	CATTGTGCAGCTTGTAGTAGAGGC	38	128200783	1	1
3:128202705-128202871/20,24/-	0.848532	3	128202852	128202871	2	1	TGCTCTCCCTGTTCCCTCG	39	128202728	1	1
4:55589697-55589866/23,21/+	0.941587	4	55589697	55589719	1	1	GTTGCTGAGGTTTCCAGCACTC	40	55589866	1	1

logistic_score	chr	art	ext_probe_st		ext_pr obe_c	ext_probe_sequence	seq ID	lig_probe_st op	lig_probe_st _copy
			op	op					
>mip_key									
4:55599221-55599390/20,24/-	4	55599371	55599390	1	GCAGGACTGTCAAGCAGAGA	41	55599244	1	
4:106155115-106155234/21,23/+	4	106155115	106155135	1	ACCAACCATGTTGAGGGCAAC	42	106155234	1	
4:106155243-106155367/20,24/+	4	106155243	106155262	1	AAATGGAGACACCAAGTGCC	43	106155367	1	
4:106155161-106155308/20,24/-	4	106155289	106155308	1	TGGCTCCCTTCATACAGG	44	106155184	1	
4:106155408-106155577/22,22/+	4	106155408	106155429	1	CTCTGGGCTCCTTCAGATCAAG	45	106155577	1	
4:106155297-106155466/23,21/-	4	106155444	106155466	1	GTCCTTCCTCATTAGCCTTTGG	46	106155317	1	
4:106155628-106155777/18,26/+	4	106155628	106155645	1	CCAGAGCTTCAGATTCTG	47	106155777	1	
4:106155497-106155666/20,24/-	4	106155647	106155666	1	ACTTTCCCTCCTGCTCAT	48	106155520	1	
4:106155827-106155996/20,24/+	4	106155827	106155846	2	AGAAAACACATCTCACATA	49	106155996	1	
4:106155708-106155877/20,24/-	4	106155858	106155877	1	ACTCATTAGTAGCCTGACTG	50	106155731	1	
4:106156035-106156184/25,19/+	4	106156035	106156059	1	TACCTGTTCTTTCAGAAACAGAA	51	106156184	2	
4:106155947-106156116/22,22/-	4	106156095	106156116	1	GTTATTTCTGCAGGAGATGGG	52	106155968	1	
4:106156251-106156420/22,22/+	4	106156251	106156272	1	CTCAGTGTTCACTAAGATTCC	53	106156420	1	
4:106156143-106156312/25,19/-	4	106156288	106156312	1	GCAATTGTGATGGTGGTGGTGT	54	106156161	1	
4:106156470-106156639/22,22/+	4	106156470	106156491	1	ACCACCTTCCAGAGATCCTAAT	55	106156639	1	
4:106156348-106156517/20,24/-	4	106156498	106156517	1	GAAGGCTGCATACATGTGT	56	106156371	1	
4:106156694-106156863/20,24/+	4	106156694	106156713	1	TGATGAGAAACAAAGAGCAA	57	106156863	1	
:106156579-106156748/20,24/-	4	106156729	106156748	1	CGTGTTCCTCCTGTCTCG	58	106156602	1	
4:106156916-106157085/23,21/+	4	106156916	106156938	1	CTGGAATTCACATGCCTGGG	59	106157085	1	
4:106156809-106156978/21,23/-	4	106156958	106156978	1	GCTGTGTGTTTCTGGGTGT	60	106156831	1	
4:106157149-106157318/21,23/+	4	106157149	106157169	1	CAACAAAGAGCAGATTCCAA	61	106157318	1	
4:106157036-106157205/20,24/-	4	106157186	106157205	1	GTGCTGTTTCAACACTGGGG	62	106157059	1	
4:106157399-106157568/18,26/+	4	106157399	106157416	1	ATGATCAGCAAAAGAGAAG	63	106157568	1	
4:106157269-106157438/18,26/-	4	106157421	106157438	1	TTAGTCTGGCCAAAGAAT	64	106157294	1	
4:106157597-106157766/21,23/+	4	106157597	106157617	1	CTGTTCACAAACATACACCC	65	106157766	1	
4:106157482-106157651/20,24/-	4	106157632	106157651	1	GGATGTGTAGTCTGTCTTT	66	106157505	1	
4:106157839-106158008/21,23/+	4	106157839	106157859	1	CTTGCTCAGCAAAAGGTACTTG	67	106158008	1	
4:106157718-106157892/22,22/-	4	106157871	106157892	1	AGGCACAGGAAACATTGCA	68	106157739	1	
4:106158007-106158176/18,26/+	4	106158007	106158024	1	ACTGAGTCTGCCATAGT	69	106158176	1	
4:106157931-106158100/23,21/-	4	106158078	106158100	1	TGTTTTCTGGTGGTGTGTGTC	70	106157951	1	
4:106158223-106158378/21,23/+	4	106158223	106158243	1	GACCATAAGGCTCTTACTCTC	71	106158378	1	
4:106158127-106158296/22,22/-	4	106158275	106158296	1	GTCAAACTGTGACTGGCCCTG	72	106158148	1	
4:106158415-106158589/20,24/+	4	106158415	106158434	1	GAGTCACCTCCAAATTACT	73	106158589	1	
4:106158315-106158478/20,24/-	4	106158459	106158478	1	GAGCTTGACAGGTGTATCC	74	106158338	1	
4:106162450-106162619/20,24/+	4	106162450	106162469	1	TAGTATAATTGAGGTCTAAA	75	106162619	1	
4:106163956-106164125/22,22/+	4	106163956	106163977	4	TGTATGTGTGTGTGTTTCTGT	76	106164125	1	
4:106164761-106164930/21,23/+	4	106164761	106164781	1	TGTGTTTGGCGGAGCGAG	77	106164930	1	
4:106164674-106164843/20,24/-	4	106164824	106164843	1	CGGGATTCTTCCACACCA	78	106164697	1	
4:106164852-106165021/21,23/-	4	106165001	106165021	1	CACGCTGAACCTCTTCTCTTT	79	106164874	1	
4:106180720-106180889/24,20/+	4	106180720	106180743	1	TGCACAGCCTATATAATGCTATCC	80	106180889	1	
4:106180786-106180955/20,24/-	4	106180936	106180955	1	AGCGATTATACATCAGGAAG	81	106180809	1	

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4:106182895-106183049/19,25/+	0.697024	4	106182895	106182913	1	AGAAATATTCACCTTATAC	82	106183049	1	1
4:106190771-106190940/20,24/+	0.930131	4	106190771	106190790	1	AATATGAACACAGACACCA	83	106190940	1	1
4:106190690-106190859/21,23/-	0.810744	4	106190839	106190859	1	AGCACAGAAGTCCAAACATGC	84	106190712	1	1
4:106193771-106193940/22,22/+	0.917879	4	106193771	106193792	1	GGATGAGCAGCTTCACGTTCTG	85	106193940	1	1
4:106193686-106193855/20,24/-	0.956667	4	106193836	106193855	3	TTTTTCTCTCTGAGCTT	86	106193709	1	1
4:106193946-106194115/20,24/+	0.908902	4	106193946	106193965	1	GAAGCCAAAGAAAGCTGCAGC	87	106194115	1	1
4:106193871-106194040/22,22/-	0.986116	4	106194019	106194040	1	TGTTTGTACGTGATGGGGCTG	88	106193892	1	1
4:106196177-106196316/24,20/+	0.915559	4	106196177	106196200	1	TTCTGTTCTCTTACCCTGTCC	89	106196316	1	1
4:106196361-106196530/21,23/+	0.913618	4	106196361	106196381	1	CCACCAATCATACATGAGAC	90	106196530	1	1
4:106196262-106196431/20,24/-	0.95844	4	106196412	106196431	1	GATATCTGAAGTGTGTGAAG	91	106196285	1	1
4:106196561-106196730/20,24/+	0.846784	4	106196561	106196580	1	CAATGCAATGGAAACCTATC	92	106196730	1	1
4:106196462-106196631/21,23/-	0.977129	4	106196611	106196631	1	GGTGAGACTGGGGAGAATAG	93	106196484	1	1
4:106196781-106196950/20,24/+	0.893805	4	106196781	106196800	1	GTTTCAGCAGTTGTACCAT	94	106196950	1	1
4:106196677-106196846/21,23/-	0.987067	4	106196826	106196846	1	GAGTGGGATAAGGAGGCAATT	95	106196699	1	1
4:106196887-106197056/21,23/+	0.931081	4	106196887	106196907	1	ACCACCAATCTGAGCAATCC	96	106197056	1	1
4:106196980-106197149/20,24/+	0.893803	4	106196980	106196999	1	CATGTTCAACAGCTCTTTC	97	106197149	1	1
4:106197193-106197362/24,20/+	0.908112	4	106197193	106197216	1	CAACGATAGGCTCTGTCAGACAG	98	106197362	1	1
4:106197071-106197240/18,26/-	0.907301	4	106197223	106197240	1	TCAGGATCCAGAAAGCTC	99	106197096	1	1
4:106197400-106197569/20,24/+	0.975927	4	106197400	106197419	1	ACATGGCTTGGCTCTTTGGG	100	106197569	1	1
4:106197289-106197458/24,20/-	0.959564	4	106197435	106197458	1	CTTCTCTTTCTCACGGGCTTTT	101	106197308	1	1
4:106197523-106197692/20,24/-	0.923275	4	106197673	106197692	1	ACAAAAGGGGTGATATCAT	102	106197546	1	1
5:170837475-170837644/22,22/-	0.943775	5	170837623	170837644	17	GGACAGCCAGATATCAACTGT	103	170837496	1	1
7:140453073-140453242/20,24/+	0.916227	7	140453073	140453092	1	ACCATCCACAAAATGGATCC	104	140453242	1	1
7:148504716-148504885/20,24/+	0.944239	7	148504716	148504735	1	AGGGGGAGGAGGTAGCAGA	105	148504885	1	1
7:148506108-148506277/22,22/-	0.949232	7	148506256	148506277	1	GCTCACTGACACCAAGTGTCT	106	148506129	1	1
7:148506397-148506516/24,20/+	0.969517	7	148506397	148506420	1	CCTACCTTTGCATAGCAGTTTGG	107	148506516	1	1
7:148507392-148507561/23,21/-	0.965315	7	148507539	148507561	1	GTCAAGCTTGATCACCTTTATCC	108	148507412	1	1
7:148508689-148508858/20,24/-	0.923191	7	148508839	148508858	2	TTTTTACCCTCTCTTTTGA	109	148508712	1	1
7:148511097-148511266/23,21/+	0.841123	7	148511097	148511119	1	TACTGTCCCAATGGTCAGCGGC	110	148511266	1	1
7:148510995-148511164/20,24/-	0.869647	7	148511145	148511164	1	CTGGCTGTCGAGAGTGTGA	111	148511018	1	1
7:148511955-148512124/20,24/-	0.889269	7	148512105	148512124	1	CTCTAACCATGTTTACAAC	112	148511978	1	1
7:148512532-148512701/22,22/-	0.917806	7	148512680	148512701	1	GCTCTCTGTGGATTGTAGCT	113	148512553	1	1
7:148516641-148516810/20,24/-	0.763635	7	148516791	148516810	1	TATTAGATCTTTGTTTCAT	114	148516664	1	1
7:148523639-148523808/20,24/+	0.843345	7	148523639	148523658	1	CAGATTAGCATTTGGTCCA	115	148523808	1	1
7:148523519-148523688/20,24/-	0.900594	7	148523669	148523688	1	ACTTCCTCTGAATGTACCC	116	148523542	1	1
7:148526795-148526934/21,23/-	0.896884	7	148526934	148526954	1	TTTTTTCTTTTAGTGGGAAG	117	148526817	1	1
8:117864762-117864931/20,24/-	0.909931	8	117864912	117864931	1	TTACACCGCTGTACACAGAA	118	117864785	1	1
8:117864807-117864976/23,21/-	0.857987	8	117864954	117864976	17	GAATTTTTCTTTTTTTTTTT	119	117864827	1	1
8:117866456-117866633/21,23/+	0.779265	8	117866456	117866476	1	GAAGTAAAAATCTGCAAACT	120	117866633	1	1
8:117866573-117866742/24,20/-	0.877384	8	117866719	117866742	1	CAGGAAAAATGATCTATTATTTAT	121	117866592	1	1
8:117868372-117868549/24,20/-	0.808009	8	117868526	117868549	31	TTTTTTTTTTTTTTTTTACAGTG	122	117868391	1	1

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9:5069922-5070091/20,24/-	0.88135	9	5070072	5070091	1	AAGGACAAAAAGACAGTAA	1	5069945	1	123	5069945	1
9:5073712-5073881/20,24/+	0.965559	9	5073712	5073731	1	AGCAAGTATGATGAGCAAGC	1	5073881	1	124	5073881	1
10:112350147-112350316/21,23/+	0.883234	10	112350147	112350167	1	TTTTTCATTTCTGACAACTTAC	1	112350316	1	125	112350316	1
10:112350217-112350386/21,23/-	0.943637	10	112350366	112350386	1	TAAGCTATTCTTATTCCTCT	1	112350239	1	126	112350239	1
10:112350706-112350875/22,22/+	0.922256	10	112350706	112350727	1	CCTGAGTACCAATAAAGATTGG	1	112350875	1	127	112350875	1
10:112350813-112350982/20,24/-	0.959726	10	112350963	112350982	1	GTGTATAGTGCCTGGGCACT	1	112350836	1	128	112350836	1
10:112352791-112352960/22,22/+	0.980092	10	112352791	112352812	1	CTGTGTGTGTGATCTCTCTGTG	1	112352960	1	129	112352960	1
10:112352887-112353056/21,23/-	0.892519	10	112353036	112353056	1	CGCCAGCCAGACATATATGTT	1	112352909	1	130	112352909	1
10:112356126-112356295/20,24/+	0.893075	10	112356126	112356145	1	ACAGACCTATTACATATGTT	1	112356295	1	131	112356295	1
11:32413487-32413656/20,24/+	0.897861	11	32413487	32413506	1	GAAAAATAAATGTGAAGAAA	1	32413656	1	132	32413656	1
11:32414159-32414328/20,24/-	0.930567	11	32414309	32414328	1	TCAAATAGAAATATGTGTCTT	1	32414182	1	133	32414182	1
11:32417866-32418035/20,24/+	0.938676	11	32417866	32417885	1	GGTAAGCACACATGAAGGGG	1	32418035	1	134	32418035	1
11:32417752-32417921/20,24/-	0.91638	11	32417902	32417921	1	TCTTGACGGTCGGCATCTG	1	32417775	1	135	32417775	1
11:32421472-32421637/20,24/+	0.93191	11	32421472	32421491	1	TGGGGCTGTCTGTGTGCTC	1	32421637	1	136	32421637	1
11:119148811-119148980/23,21/+	0.851218	11	119148811	119148833	1	CTGTTAACATTTTATAATTGCAGT	1	119148980	1	137	119148980	1
11:119148895-119149064/20,24/-	0.92696	11	119149045	119149064	1	TACCGAATTTTCAAGGTTA	1	119148918	1	138	119148918	1
11:119149175-119149344/20,24/+	0.895932	11	119149175	119149194	2	AGTATTTTCAGATGCATCTG	2	119149344	1	139	119149344	1
2:25378504-25378673/20,24/-	0.911124	12	25378654	25378673	1	GTACCTATGGTCCTAGTAGG	1	25378527	1	140	25378527	1
12:25380216-25380385/20,24/-	0.967652	12	25380366	25380385	1	TGCACTGTAATAATATCCAGAC	1	25380239	1	141	25380239	1
12:25398165-25398334/20,24/-	0.933725	12	25398315	25398334	1	ATAAGGCCTGCTGAAAAATGA	1	25398188	1	142	25398188	1
12:112888093-112888262/23,21/-	0.916866	12	112888240	112888262	3	CAATCTCTCTTTTAAATGGCC	3	112888113	1	143	112888113	1
12:112910715-112910880/22,22/+	0.894139	12	112910715	112910736	1	CACGTAATAATATTGACTTTTC	1	112910880	1	144	112910880	1
12:112915411-112915580/20,24/-	0.966125	12	112915561	112915580	1	GGCTAGAAATGTATGGTCAG	1	112915434	1	145	112915434	1
12:112926198-112926363/20,24/-	0.918085	12	112926344	112926363	2	CATAAACTAAAAACAGAAAC	2	112926221	1	146	112926221	1
12:112926812-112926981/20,24/+	0.957582	12	112926812	112926831	1	GATGTTTCTTCGTAGGTGT	1	112926981	1	147	112926981	1
13:28592560-28592729/20,24/+	0.919768	13	28592560	28592579	1	CACAAAATAGCCGTATAAAA	1	28592729	1	148	28592729	1
13:28602295-28602464/16,29/+	0.881258	13	28602295	28602310	2	TTTTCTGGAAGTGGG	2	28602464	1	149	28602464	1
13:28607990-28608155/20,24/-	0.927129	13	28608136	28608155	1	GCACGTACTCACCATTTTGTCT	1	28608013	1	150	28608013	1
13:28608205-28608374/20,24/+	0.894916	13	28608205	28608224	1	CATTCCATTTTACCAAACT	1	28608374	1	151	28608374	1
13:28608170-28608339/20,24/-	0.920908	13	28608320	28608339	1	GAAAGCCAGCTACAGATGGT	1	28608193	1	152	28608193	1
13:28608401-28608570/20,24/+	0.891863	13	28608401	28608420	1	AGAGGAAAGAATAATGAATT	1	28608570	1	153	28608570	1
13:28609586-28609755/21,23/+	0.98033	13	28609586	28609606	1	GTGGGGAAATTCCTGATGGTGG	1	28609755	1	154	28609755	1
13:28609701-28609870/20,24/+	0.946119	13	28609701	28609720	1	ACCTTTTATGGCTTCACTC	1	28609870	1	155	28609870	1
13:28610048-28610217/20,24/+	0.916878	13	28610048	28610067	1	AAGAGGCATCAATGTCCTTA	1	28610217	1	156	28610217	1
15:90631745-90631914/20,24/+	0.801172	15	90631745	90631764	1	AAGAGGATGGCTAGGCGAGG	1	90631914	1	157	90631914	1
15:90631845-90632014/20,24/+	0.93551	15	90631845	90631864	1	TGGTGATGGCTGTGCTCCAG	1	90632014	1	158	90632014	1
17:7572870-7573039/21,23/+	0.981202	17	7572870	7572890	1	AGGGGAGGAGAGATGGGGGT	1	7573039	1	159	7573039	1
17:7573943-7574112/22,22/-	0.910933	17	7574091	7574112	1	CAACAAATTTGTAATTTGAACCA	1	7573964	1	160	7573964	1
17:7576792-7576961/24,20/-	0.847926	17	7576938	7576961	1	TTCACTTTTATCACCTTTCCTTGC	1	7576811	1	161	7576811	1
17:7577075-7577244/24,20/+	0.980151	17	7577075	7577098	1	TTCTCTTCCTCTGTGCGCGGTCT	1	7577244	1	162	7577244	1
17:7576963-7577132/21,23/-	0.924868	17	7577112	7577132	1	GCTTTGAGGTGCGTGTGTTGTG	1	7576985	1	163	7576985	1

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17:7577438-7577602/20,24/+	0.897643	17	7577438	7577457	1	AGAGGCAAGCAGAGGCTGGG	1	164	7577602	1		1
17:7577529-7577693/22,22/-	0.959787	17	7577672	7577693	14	AAAAAAAAAAAAAAAAAGGCCTCC	14	165	7577550	1		1
17:7578169-7578338/23,21/+	0.893158	17	7578169	7578191	1	AACACACCTCAGCGGCTCATA	1	166	7578338	1		1
17:7578093-7578262/23,21/-	0.937736	17	7578240	7578262	1	GAGTGAAGGAAATTGCGTGTG	1	167	7578113	1		1
17:7578393-7578562/18,26/+	0.843508	17	7578393	7578410	1	ATGGTGGGGCAGCGCCT	1	168	7578562	1		1
17:7578334-7578453/18,26/-	0.833295	17	7578436	7578453	1	CATGGCCATCTACAAGCA	1	169	7578359	1		1
17:7578469-7578628/26,18/-	0.890436	17	7578603	7578628	1	CTGCCGCTTCCAGTTGCTTTATCTG	1	170	7578486	1		1
17:7579238-7579407/20,24/+	0.809488	17	7579238	7579257	1	CAACAAAAGAAATGCAGGG	1	171	7579407	1		1
17:7579298-7579467/21,23/+	0.74921	17	7579298	7579318	1	TCAGGGCACTGACCGTGCAA	1	172	7579467	1		1
17:7579416-7579577/20,24/+	0.784204	17	7579416	7579435	2	AGGAGGGGCTGGTGACGGG	2	173	7579577	1		1
17:7579495-7579664/21,23/-	0.900055	17	7579644	7579664	2	TGGGACCTGGAGGGCTGGGG	2	174	7579517	1		1
17:7579802-7579971/24,20/-	0.864469	17	7579948	7579971	1	TCATGCTGGATCCCACTTTCT	1	175	7579821	1		1
17:58740425-58740594/22,22/+	0.952631	17	58740425	58740446	1	GCCTTCTCAGAGAAATTTTATG	1	176	58740594	1		1
17:58740303-58740472/20,24/-	0.827005	17	58740453	58740472	1	CTCTCGAGCTATCTCAGCTG	1	177	58740326	1		1
17:58740633-58740802/21,23/+	0.979392	17	58740633	58740653	1	TGAAGATGTCAACTCTCTGGCC	1	178	58740802	1		1
17:58740506-58740675/21,23/-	0.853213	17	58740655	58740675	1	TCAATTTCTGGGCTTTTCAIT	1	179	58740528	1		1
17:58740833-58741002/20,24/+	0.902941	17	58740833	58740852	1	ATGCGACGACACTATAGGGG	1	180	58741002	1		1
7:58740728-58740897/21,23/-	0.938281	17	58740877	58740897	1	GTTTTCTGTGTGTAAGT	1	181	58740750	1		1
17:74732929-74733008/24,20/+	0.924881	17	74732929	74732952	1	GGTCCCGCGGCTGTGTGTGAG	1	182	74733008	1		1
18:42531843-42532012/21,23/+	0.911291	18	42531843	42531863	1	CTCCCTAAAGGAAATCAGCT	1	183	42532012	1		1
19:13054585-13054754/20,24/+	0.937952	19	13054585	13054604	1	AAGAAGACAAAGAAACGCAA	1	184	13054754	1		1
19:13054480-13054649/24,20/-	0.794174	19	13054626	13054649	1	ATCTTTGTCTCATCTCTCTT	1	185	13054499	1		1
19:33792231-33792385/16,27/-	0.810719	19	33792370	33792385	12	GAAAGTGCTGGAGCTG	12	186	33792257	1		1
19:33792335-33792504/20,24/-	0.830437	19	33792485	33792504	1	AAGGCCAAGAAAGTCGGTGA	1	187	33792358	1		1
19:33792732-33792851/16,26/+	0.652911	19	33792732	33792747	19	GCGGGTGGGGTGCGG	19	188	33792851	1		1
19:33792942-33793051/16,24/-	0.849634	19	33793036	33793051	2	GCCCAAGCGGCCGCTG	2	189	33792965	1		1
19:33793045-33793154/16,29/+	0.8992	19	33793045	33793060	10	CTTGGCTTCTCTCTG	10	190	33793154	1		1
19:33793184-33793288/16,26/+	0.876116	19	33793184	33793199	12	GGTGGGCGGGAGGCT	12	191	33793288	1		1
20:31021052-31021221/21,23/+	0.922229	20	31021052	31021072	1	GGCTGAAACTGATGGCTGTG	1	192	31021221	1		1
20:31021242-31021411/22,22/+	0.940116	20	31021242	31021263	1	CAGATCTCCGAACAGAGCCAG	1	193	31021411	1		1
20:31021151-31021320/24,20/-	0.925719	20	31021297	31021320	1	TTTGATCCTTAGCAACCCCTGCT	1	194	31021170	1		1
20:31021437-31021606/20,24/+	0.888392	20	31021437	31021456	1	CGAATTCACAGTTGAGTCT	1	195	31021606	1		1
20:31021362-31021531/23,21/-	0.82328	20	31021509	31021531	1	CAGGTAGGAATCTGTCTGGAG	1	196	31021382	1		1
20:31021534-31021703/20,24/+	0.939897	20	31021534	31021553	1	TCAGGAAACTGTGGATCAG	1	197	31021703	1		1
20:31022183-31022352/22,22/+	0.894333	20	31022183	31022204	1	TAGGTGAGATCACCCAGTCAGT	1	198	31022352	1		1
20:31022389-31022532/16,25/+	0.740488	20	31022389	31022404	2	GAGGGGCGAGAGGTCA	2	199	31022533	1		1
20:31022292-31022441/20,24/-	0.670733	20	31022422	31022441	1	TCCGATGGCAGTGTGGCCG	1	200	31022315	1		1
20:31022643-31022812/21,23/+	0.806603	20	31022643	31022663	1	GGAAGTCCCATGTCCAGAGCT	1	201	31022812	1		1
20:31022555-31022724/20,24/-	0.907715	20	31022705	31022724	1	CTGTAGCCCTCTGTAGTAGG	1	202	31022578	1		1
20:31022881-31023050/22,22/+	0.931489	20	31022881	31022902	1	GTGAGTCTGGCACCACCTTCTG	1	203	31023050	1		1
20:31022782-31022951/23,21/-	0.91537	20	31022929	31022951	1	ACCATTTGTCGAGGAACGGTGG	1	204	31022802	1		1

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20:31023086-31023255/21,23/+	0.933626	20	31023086	31023106	1	GCAGAACAGACGATTTGATGA	205	31023255	1	
20:31022984-31023153/23,21/-	0.932364	20	31023131	31023153	1	TATCACTTTCCCTCATAGAGGG	206	31023004	1	
20:31023328-31023497/20,24/+	0.949461	20	31023328	31023347	1	CTGCATTGCTGGGGATTGG	207	31023497	1	
20:31023202-31023371/21,23/-	0.881999	20	31023351	31023371	1	AGGATCTAGACCTCTCTCAGC	208	31023224	1	
20:31023421-31023590/20,24/-	0.918557	20	31023571	31023590	1	CTTTGTCACTGCAGCTTCTC	209	31023444	1	
20:31023517-31023686/20,24/-	0.878099	20	31023667	31023686	1	ATCTGTCTCTTGAACACGAC	210	31023540	1	
20:31023708-31023877/22,22/+	0.887733	20	31023708	31023729	1	TGGGTGTCTCGAGTATGTGCGG	211	31023877	1	
20:31023619-31023788/20,24/-	0.904275	20	31023769	31023788	1	GGCTCTTGGCTGGTACTCAG	212	31023642	1	
20:31023826-31023995/20,24/-	0.897669	20	31023976	31023995	1	GCCACTGTTTTTCCAAAGC	213	31023849	1	
20:31024004-31024173/20,24/+	0.889572	20	31024004	31024023	1	TGGAAGCAGCCCCAGTTCTT	214	31024173	1	
20:31024067-31024236/21,23/+	0.894101	20	31024067	31024087	1	AGGCACTGGTCTTGCCAGGAT	215	31024236	1	
20:31023931-31024100/20,24/-	0.934792	20	31024081	31024100	1	CTGGTGGCTCAATCTCTGG	216	31023954	1	
20:31024183-31024352/20,24/+	0.897143	20	31024183	31024202	1	CTAGGAACTGGAGAAATG	217	31024352	1	
20:31024301-31024470/24,20/+	0.881533	20	31024301	31024324	1	CCCAGGAGATCTTACTCTCGAG	218	31024470	1	
20:31024409-31024578/20,24/+	0.868986	20	31024409	31024428	2	AGGCCAAGGGAAGAGCTTT	219	31024578	1	
20:31024513-31024682/20,24/+	0.949413	20	31024513	31024532	1	TCCCAGTGGGAAGTTGGGA	220	31024682	1	
20:31024623-31024792/20,24/+	0.95695	20	31024623	31024642	1	GAGAAAGCTTTTGTGGGGGG	221	31024792	1	
0:31024736-31024905/23,21/+	0.924311	20	31024736	31024758	1	CTTGCCCTTCTGAAATACCCC	222	31024905	1	
20:31024855-31025024/21,23/+	0.905752	20	31024855	31024875	1	TCCAACTGAGTTCCACAGCT	223	31025024	1	
20:31024965-31025134/20,24/+	0.950131	20	31024965	31024984	1	CACAGTGCATCACTTTCTCT	224	31025134	1	
20:57484366-57484525/18,26/-	0.944333	20	57484508	57484525	1	CAAGAAACCATGATCTCT	225	57484391	1	
21:36164530-36164685/20,24/+	0.734511	21	36164530	36164549	1	GGTTCGGAGGCTGGGGTTG	226	36164685	1	
21:36164619-36164747/21,23/+	0.781897	21	36164619	36164639	2	ACCATTGGAGAACTGGTAGGAG	227	36164747	1	
21:36164705-36164815/20,24/+	0.711885	21	36164705	36164724	1	TTCGACGAGCCGGGGTAGG	228	36164815	1	
21:36164717-36164857/23,21/+	0.828908	21	36164717	36164739	1	GGGTAGGGCGGGCAGGTAGG	229	36164857	1	
21:36164802-36164956/19,25/+	0.794232	21	36164802	36164820	1	GTCGGGGAGTAGGTGAAGG	230	36164956	1	
21:36171528-36171697/21,23/-	0.841039	21	36171677	36171697	1	TCCATTGCCTCTCTCTCTGTG	231	36171550	1	
21:36171646-36171815/22,22/-	0.879815	21	36171794	36171815	1	CATTTTTAAATCCACCCAC	232	36171667	1	
21:36206622-36206777/23,21/+	0.865415	21	36206622	36206644	1	GGGAAGGTGTGTGCATGGGGG	233	36206777	1	
21:36206730-36206885/21,23/+	0.735902	21	36206730	36206750	1	GGGTAAAGGAGTAGGTGAAGG	234	36206885	1	
21:36206799-36206954/23,21/-	0.791705	21	36206932	36206954	15	TCTCCCTCCCTCTCTCTCTCCC	235	36206819	1	
21:36231732-36231901/22,22/-	0.873923	21	36231880	36231901	1	TTTTTGTCTCTATCGTGTCCCC	236	36231753	1	
21:36252801-36252970/23,21/+	0.943161	21	36252801	36252823	1	TGGGTTTGTGGCATGAAACGTG	237	36252970	1	
21:36252916-36253085/21,23/-	0.94378	21	36253065	36253085	1	ATCACTACACAAATGCCCTAA	238	36252938	1	
21:36259108-36259222/16,29/+	0.817091	21	36259108	36259123	15	CTGCTCTCCACCCAC	239	36259222	2	
21:36259159-36259263/22,22/+	0.706198	21	36259159	36259180	3	GTCTTTGTGTCAGCGCCAGTGG	240	36259263	1	
21:44514729-44514898/22,22/-	0.933507	21	44514877	44514898	2	TTTCGCGGTGAGGAAGATGCGG	241	44514750	1	
21:44524416-44524585/20,24/+	0.94191	21	44524416	44524435	1	CAAAACAACTGGCTAAACG	242	44524585	1	
X:15833872-15834041/20,24/+	0.979296 X		15833872	15833891	2	AATGGAGCAGTGCAGGAGGG	243	15834041	1	
X:15833745-15833914/20,24/-	0.924197 X		15833895	15833914	2	GCTTGGCTCAGGGTCATAGT	244	15833768	1	
X:15836657-15836826/21,23/-	0.93315 X		15836806	15836826	1	AACAGAAACAGAACAAAC	245	15836679	1	

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X:15838303-15838483/23,21/+	0.712992 X	15838303	15838325	1	GCATATCATTTGATTTTGGTTT	246	15838483	1					
X:39911340-39911509/20,24/+	0.940705 X	39911340	39911359	1	GTACATGGTGGGTCCAGCTT	247	39911509	1					
X:39911542-39911711/21,23/+	0.932675 X	39911542	39911562	1	TTCGCTCTGCAATGGTGAC	248	39911711	1					
X:39911448-39911617/20,24/-	0.941736 X	39911598	39911617	1	GAATTTGAAATGTCTCTCC	249	39911471	1					
X:39914595-39914764/20,24/+	0.919934 X	39914595	39914614	1	AGACCATTTCTTGAACCTTG	250	39914764	1					
X:39914691-39914860/24,20/-	0.966337 X	39914837	39914860	1	TGCCAGCTTGCCTGTGCTTTT	251	39914710	1					
X:39932843-39933012/24,20/+	0.82704 X	39932843	39932866	1	TCTGCTGTTTGTGGCCATCTG	252	39933012	1					
X:39933079-39933248/20,24/+	0.945629 X	39933079	39933098	1	GAGGGAGCAGTGTCTGATGAT	253	39933248	1					
X:39932964-39933133/20,24/-	0.933519 X	39933114	39933133	1	CTCCAGGAAATGTTGTGCT	254	39932987	1					
X:48649464-48649633/16,29/+	0.856993 X	48649464	48649479	14	TTCTGTGTCTGAGGA	255	48649633	1					
X:48649695-48649864/21,23/+	0.897556 X	48649695	48649715	1	TGGCTACTACAGGACGCTG	256	48649864	1					
X:48649576-48649745/20,24/-	0.823488 X	48649726	48649745	1	TGGAGTTACTGGGAGTGT	257	48649599	1					
X:53431899-53432068/20,24/+	0.892295 X	53431899	53431918	1	TGTGTGATGGCCTTTGGAG	258	53432068	1					
X:53432011-53432180/22,22/+	0.897516 X	53432011	53432032	1	ATCTGCAGTCCATGGCCTGAG	259	53432180	1					
X:53432104-53432273/23,21/+	0.897847 X	53432104	53432126	1	GCTAGGTGTAAGGTGGTGCTG	260	53432273	1					
X:53432228-53432397/24,20/+	0.904552 X	53432228	53432251	1	TTTCTATCCAGCGCCGTGCCTT	261	53432397	1					
X:53432336-53432505/20,24/+	0.904126 X	53432336	53432355	1	GGGGAGAGAGAAAGAGGGG	262	53432505	1					
:53432548-53432717/23,21/+	0.873273 X	53432548	53432570	1	ATCAATCACTAGCTTGGCCCTT	263	53432717	1					
X:53432438-53432607/22,22/-	0.912547 X	53432586	53432607	1	CAGGTGAAGCCTACAGATTGAGA	264	53432459	1					
X:53432775-53432944/23,21/+	0.945673 X	53432775	53432797	1	GCCTGTCTTCTCCAGTCCACAA	265	53432944	1					
X:53432662-53432831/22,22/-	0.898153 X	53432810	53432831	1	ACCAAGTTTTGGGCAAGAACA	266	53432683	1					
X:123181234-123181403/20,24/+	0.95047 X	123181234	123181253	1	TGGCACTAAATCTTAGCATT	267	123181403	1					
X:123181157-123181326/20,24/-	0.9185 X	123181307	123181326	1	GCCTCTCATTTGGCTCGTTT	268	123181180	1					
X:123182833-123183002/20,24/+	0.820783 X	123182833	123182852	1	GAATGATGTGTTTTTTTAC	269	123183002	1					
X:123184006-123184184/21,23/+	0.907704 X	123184006	123184026	1	TTATGCATCGTTTTCTCTCC	270	123184184	1					
X:123184910-123185079/20,24/+	0.833242 X	123184910	123184929	1	GAAGTTGAAAATACATAGAG	271	123185079	1					
X:123185026-123185195/22,22/+	0.858971 X	123185026	123185047	1	AAGAGCTTAATCCAAACTGGA	272	123185195	1					
X:123185116-123185285/20,24/+	0.932372 X	123185116	123185135	5	CTTTTCTTCTGCTTTCTTTT	273	123185285	1					
X:123189951-123190120/21,23/-	0.874373 X	123190100	123190120	1	GCCTTAGAAAATAGATAACAG	274	123189973	1					
X:123220363-123220532/20,24/+	0.871879 X	123220363	123220382	1	GTATCAAGCTAACAGTTTC	275	123220532	1					
X:123220475-123220644/20,24/-	0.7772 X	123220625	123220644	1	AAAGAGAAATAAATATATATCC	276	123220498	1					
X:133511649-133511818/20,24/+	0.966325 X	133511649	133511668	1	TGTCAGCTCAGTTTGAACAG	277	133511818	1					
X:133511601-133511770/20,24/-	0.96135 X	133511751	133511770	1	CGTCGCACCTCTGCTTTT	278	133511624	1					
X:133527602-133527771/20,24/+	0.867927 X	133527602	133527621	1	CTACCACTGTGCATTGCATG	279	133527771	1					
X:133527477-133527646/20,24/-	0.939432 X	133527627	133527646	1	GGTTTCTCTCGTATTGAGC	280	133527500	1					
X:133527898-133528017/22,22/+	0.864925 X	133527898	133527919	1	TTAGTTTCTTACTAATTTTGG	281	133528017	1					
X:133547773-133547942/20,24/+	0.896119 X	133547773	133547792	1	GAATGTTAAGTAAGCTTGA	282	133547942	1					
X:133547875-133548044/23,21/-	0.984411 X	133548022	133548044	17	AAACAACAACAAAAACAAAA	283	133547895	1					
X:133549006-133549180/27,27/-	0.875443 X	133549154	133549180	1	TGTTGCATAACAAATATAAAACTACA	284	133549032	1					
X:133551196-133551365/22,22/-	0.876924 X	133551344	133551365	1	GTTTAGGGAAATAGACAACCTGT	285	133551217	1					

SEQ.ID	lig_probe_sequence	mip_scan_start	mip_name
286	TCAGACTTCGGCCACCC	43814917	MPL_001_Myeloid_Panel
287	GTGTGGAGGGTAAGGGGGCAGGG	115256480	NRAS_001_Myeloid_Panel
288	GTAGCCCGCTGACCTGATCTGT	115258658	NRAS_002_Myeloid_Panel
289	ACTGGGAACCAATACCTGG	25457146	DNMT3A_001_Myeloid_Panel
290	GCTGAAGGAGTATTTTGGTGTTGT	25457176	DNMT3A_002_Myeloid_Panel
291	CCGGGTTGTGCTGGCATCTGGCT	25458573	DNMT3A_003_Myeloid_Panel
292	GCAGGGAGAGGAAGGGCAGGAT	25459772	DNMT3A_004_Myeloid_Panel
293	GCTCTGGGCTGGGGGCTGT	25461989	DNMT3A_005_Myeloid_Panel
294	GGCATCATGCAGAGGGCGGTAGA	25463143	DNMT3A_006_Myeloid_Panel
295	GCGTTAGTGACAAAGGGGACATCT	25463209	DNMT3A_007_Myeloid_Panel
296	GCTGTCAGGGACAGAGGCAGACA	25463504	DNMT3A_008_Myeloid_Panel
297	TTGGAGCATCTCCTGGCACCT	25464530	DNMT3A_009_Myeloid_Panel
298	GTCCATGCTGTGGGCGCAG	25464427	DNMT3A_010_Myeloid_Panel
299	GTGAGGGGTGCAGGCCCAAGAGGT	25466767	DNMT3A_011_Myeloid_Panel
300	CTTCCTTAATGGCTGCCTGGGCAG	25467032	DNMT3A_012_Myeloid_Panel
301	GCCTGGGGCGGTCTCGAGCT	25467013	DNMT3A_013_Myeloid_Panel
302	:CCCTGGAACGTACATGTGC	25467139	DNMT3A_014_Myeloid_Panel
303	GCTGTTGTGGCCTCCAGTGTT	25467404	DNMT3A_015_Myeloid_Panel
304	GGTGAGTACCAACGAAGGGCCTCT	25468120	DNMT3A_016_Myeloid_Panel
305	TGCCGTTGAGCGGGCCCTTC	25468096	DNMT3A_017_Myeloid_Panel
306	CCGGCCCTGCTCTCGGATG	25468841	DNMT3A_018_Myeloid_Panel
307	CACATGTCGCTGTACACTTCTTG	25469013	DNMT3A_019_Myeloid_Panel
308	TTCCCCACCCTCTTACAG	25469111	DNMT3A_020_Myeloid_Panel
309	GTGACACGCCAGGGTTGGGGTTGT	25469530	DNMT3A_021_Myeloid_Panel
310	GGCTGCCAAGGCTCCACAGAG	25469407	DNMT3A_022_Myeloid_Panel
311	CACCCACCCCATGCCCTGCAA	25469949	DNMT3A_023_Myeloid_Panel
312	GCACTAGGAGGCCCTGGAAGTTG	25469881	DNMT3A_024_Myeloid_Panel
313	TTCCCCACACAGCTCCCAA	25470458	DNMT3A_025_Myeloid_Panel
314	GTGCTTGGTGGATGACGGGC	25470544	DNMT3A_026_Myeloid_Panel
315	CGGGCCCTGGTTTCTT	25470939	DNMT3A_027_Myeloid_Panel
316	AGGTAATTGGTGGAATTACCTTC	198266564	SF3B1_001_Myeloid_Panel
317	CTGGATATGTTTCATGGTTCT	198266438	SF3B1_002_Myeloid_Panel
318	GTGTTAAAGCCTTTATGGAAGGT	198266754	SF3B1_003_Myeloid_Panel
319	GAAAGGACAGTCATGAGTTGTT	198267441	SF3B1_004_Myeloid_Panel
320	CTATGGGCTGTGCCATCTTGCC	198267338	SF3B1_005_Myeloid_Panel
321	GGTGCCATTGGTGATTTC	209113087	IDH1_001_Myeloid_Panel
322	TGCAGGAGAAGTCATCCCTTC	128200044	GATA2_001_Myeloid_Panel
323	GGTGCCGCTCTCTGGCGG	128200685	GATA2_002_Myeloid_Panel
324	CACATCAAGCCCAAGCAAGAC	128202729	GATA2_003_Myeloid_Panel
325	GTCAGGAACTGAGCAGGT	55589720	KIT_001_Myeloid_Panel

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GCCCACTGCCTGAGAGAGCTCAT	327	106155136	TET2_001_Myeloid_Panel	
GTAGAGGGTATTCCAAGTGTITGC	328	106155263	TET2_002_Myeloid_Panel	
GTTCTGTGGCAAAATGGAGGTG	329	106155185	TET2_003_Myeloid_Panel	
TCTGTAGCCCAAGAAAATGCAG	330	106155430	TET2_004_Myeloid_Panel	
CGACTATTCTGGCTTCCTTC	331	106155318	TET2_005_Myeloid_Panel	
TGGAACACACACATGGTGAACCTCTG	332	106155646	TET2_006_Myeloid_Panel	
CATTGGTGACTGCTTACCTG	333	106155521	TET2_007_Myeloid_Panel	
GTGAGTAGGCCGTGTGATGCTGAT	334	106155847	TET2_008_Myeloid_Panel	
GCACCATTAGGCATTAGCACTGCC	335	106155732	TET2_009_Myeloid_Panel	
CCAGCAGCAATTTGCAAGC	336	106156060	TET2_010_Myeloid_Panel	
GCAGCTGGCTTTGGAGGCAGCT	337	106155969	TET2_011_Myeloid_Panel	
CACCACCACTACCCCAACCAAA	338	106156273	TET2_012_Myeloid_Panel	
TGAACAGAAATTCACCA	339	106156162	TET2_013_Myeloid_Panel	
CCAAATGTCAGAACACTCAAGC	340	106156492	TET2_014_Myeloid_Panel	
GCTTTTCTCTGAAGGAAGCTG	341	106156372	TET2_015_Myeloid_Panel	
CGTAATGAGGCATCACTGCCATCA	342	106156714	TET2_016_Myeloid_Panel	
.CACAATGGAACAGTCATTGTCCC	343	106156603	TET2_017_Myeloid_Panel	
CCCTCACACCAGGTGCAC TTC	344	106156939	TET2_018_Myeloid_Panel	
GGGATTCCGTTGGTGAAAACGA	345	106156832	TET2_019_Myeloid_Panel	
TCCCAGAGTTCACATCTCCCTCA	346	106157170	TET2_020_Myeloid_Panel	
GGAAC TGGAGATGTTGGTCCACTG	347	106157060	TET2_021_Myeloid_Panel	
GAAATTCCTTATAGTCAGACCATG	348	106157417	TET2_022_Myeloid_Panel	
GGTTGTGTTTGCTGCGCTGTTTATG	349	106157295	TET2_023_Myeloid_Panel	
GCTTTCAGAAAGAGGAGCAGAAG	350	106157618	TET2_024_Myeloid_Panel	
GACATTATGAGTCTCGAATCGCT	351	106157506	TET2_025_Myeloid_Panel	
CAGCAACACACAGCAACCCCAAC	352	106157860	TET2_026_Myeloid_Panel	
GTGAAGAAGATCTTGCTTTGGG	353	106157740	TET2_027_Myeloid_Panel	
AATGTGCAGCAAAAGAGCATCATTGA	354	106158025	TET2_028_Myeloid_Panel	
GCAGCATGCTTTGAGTGTCC	355	106157952	TET2_029_Myeloid_Panel	
CTTCTTCAGAAAAGACACCAAC	356	106158244	TET2_030_Myeloid_Panel	
CACAGCTTGACGGTGGAITCTC	357	106158149	TET2_031_Myeloid_Panel	
CTTCAGATATGGGATTTCCTTCT	358	106158435	TET2_032_Myeloid_Panel	
GCTGGGGTGTGGCTATCAAGTTCT	359	106158339	TET2_033_Myeloid_Panel	
GCAAGGGCACAGGCAGATTAAACG	360	106162470	TET2_034_Myeloid_Panel	
GCCTTTGGTCTTAAATCTTGGG	361	106163978	TET2_035_Myeloid_Panel	
CCAAATCGCGGTGTGCTTGAAT	362	106164782	TET2_036_Myeloid_Panel	
CCCCCACCACCAACCAACAAAAA	363	106164698	TET2_037_Myeloid_Panel	
GCTCCGAGTAGAGTTTGTCAAGC	364	106164875	TET2_038_Myeloid_Panel	
TTTGCCAGGAAGCAAGATCCC	365	106180744	TET2_039_Myeloid_Panel	
TTCTGGATCCAGCCCTGACAGGC	366	106180810	TET2_040_Myeloid_Panel	

lig_probe_sequence	SEQ.ID	mip_scan_start	mip_position	mip_name
GGCAGCAATTGTAACAACTTACTTG	367	106182914	TET2_041_Myeloid_Panel	
GAGGACAGCTTAGCAGCTGTTGAG	368	106190791	TET2_042_Myeloid_Panel	
GAAAACTCACTAGTATTAGACC	369	106190713	TET2_043_Myeloid_Panel	
GTCAGACTTGCCGACAAAGGA	370	106193793	TET2_044_Myeloid_Panel	
GTAAGACATTACAGCTCAACTAC	371	106193710	TET2_045_Myeloid_Panel	
GCATTTGTAGATAAATGTTGTG	372	106193966	TET2_046_Myeloid_Panel	
GCCGAAAAAGAACTCAGTACCTG	373	106193893	TET2_047_Myeloid_Panel	
CCCGCAGCAGCAGCCACA	374	106196201	TET2_048_Myeloid_Panel	
CCATGAACCCTTACCTGGGCTT	375	106196382	TET2_049_Myeloid_Panel	
GCTGGGCTGTGGTGGCTGCTCT	376	106196286	TET2_050_Myeloid_Panel	
GCCAAAGTTTGAAATAGCCAGAG	377	106196581	TET2_051_Myeloid_Panel	
CCTGCAGCTTGAGATGAGGTGGA	378	106196485	TET2_052_Myeloid_Panel	
GGTGAACATCTTACCTTCTCAC	379	106196801	TET2_053_Myeloid_Panel	
GTGTATGGATGGTGGTAGACTG	380	106196700	TET2_054_Myeloid_Panel	
GCTTCCACACAGCTAATGGT	381	106196908	TET2_055_Myeloid_Panel	
GTGATGCTAATGGTCAGGAAAGC	382	106197000	TET2_056_Myeloid_Panel	
TCACCCACCAGGATCTCCC	383	106197217	TET2_057_Myeloid_Panel	
ICAGTTCTATCGTTAAAGGCTGG	384	106197097	TET2_058_Myeloid_Panel	
CAGAGCCCACTTACCTGCGTTTCA	385	106197420	TET2_059_Myeloid_Panel	
GCAGCTCAGCTTTGCACAC	386	106197309	TET2_060_Myeloid_Panel	
GAAGTTTCATGTGGCTCAGCAGGC	387	106197547	TET2_061_Myeloid_Panel	
CCACAACACTTCATAGACATCA	388	170837497	NPM1_001_Myeloid_Panel	
CAGTAGATCTCATTTTCTATCAG	389	140453093	BRAF_001_Myeloid_Panel	
CACAACAAAGCCTGCTGAAGATAG	390	148504736	EZH2_001_Myeloid_Panel	
GGGCTTTTCTACTGGATTGTG	391	148506130	EZH2_002_Myeloid_Panel	
GTAAGCACAGCCCAAGTGAAT	392	148506421	EZH2_003_Myeloid_Panel	
GCTGGGAGGCAGTGAGTTCT	393	148507413	EZH2_004_Myeloid_Panel	
GGCACTGATAACCTGTATTGAGGT	394	148508713	EZH2_005_Myeloid_Panel	
GGAGGTTCTTCACATCACC	395	148511120	EZH2_006_Myeloid_Panel	
GTAGTTAGCTATTAGTGATGCAA	396	148511019	EZH2_007_Myeloid_Panel	
GCATGAGAACTAAATAGGCTTTG	397	148511979	EZH2_008_Myeloid_Panel	
GCTTTGTTTCATTTGTTTTAG	398	148512554	EZH2_009_Myeloid_Panel	
GCAGAGGGTACTTGAGAGGACTTT	399	148516665	EZH2_010_Myeloid_Panel	
GTTTCTAAAAGGTTTCCATGTGT	400	148523659	EZH2_011_Myeloid_Panel	
CGTTTTCATTTTCTATCTTTGTG	401	148523543	EZH2_012_Myeloid_Panel	
GAGCCATATGCTTCTTCTCTGG	402	148526818	EZH2_013_Myeloid_Panel	
TTGTTCTGTGGAATAGTTCCTTG	403	117864786	RAD21_001_Myeloid_Panel	
GAGGACCAGCAACAGCAGCAT	404	117864828	RAD21_002_Myeloid_Panel	
GAATAATCACTAAGTTGGGCTCT	405	117866477	RAD21_003_Myeloid_Panel	
GGATCTGGCAGCCGCCACCA	406	117866593	RAD21_004_Myeloid_Panel	
CCTTAGATTATACAGAT	407	117868392	RAD21_005_Myeloid_Panel	

lig_probe_sequence	SEQ ID	mip_scan_start	mip_position	mip_name
AGACTAGAGGTTTGATTATCTG	408		5069946	JAK2_001_Myeloid_Panel
CAGGATCACAGCTAGGTGTCACTG	409		5073732	JAK2_002_Myeloid_Panel
CTTTTACACATCGTGAAGTC	410		112350168	SMC3_001_Myeloid_Panel
GCTGGTTTATCTTTTCGACGG	411		112350240	SMC3_002_Myeloid_Panel
GTTAGATGTCAGGTATACAGCC	412		112350728	SMC3_003_Myeloid_Panel
AGTAACTCTCCAGGAAGATTCTAT	413		112350837	SMC3_004_Myeloid_Panel
CAGCTGGCCCGTCTTTCACATA	414		112352813	SMC3_005_Myeloid_Panel
GAGCTTTTCCAAACACATGTTTG	415		112352910	SMC3_006_Myeloid_Panel
GCAAAGCTCAATGAAACCTGCGC	416		112356146	SMC3_007_Myeloid_Panel
GCCTCGCCCTAACAAATGTGGCA	417		32413507	WT1_001_Myeloid_Panel
GCTGTGTTCCCTTGGCTAGGGTT	418		32414183	WT1_002_Myeloid_Panel
GTCTGAGGGAGGTGAGCACTGG	419		32417886	WT1_003_Myeloid_Panel
GCAAACATGGTTCAAGAGCTCTT	420		32417776	WT1_004_Myeloid_Panel
AGGCTCAGTGTGGCTCACAGTCGC	421		32421492	WT1_005_Myeloid_Panel
GAGCCTGTGGACACCTCATG	422		119148834	CBL_001_Myeloid_Panel
GTTGGAATGTGGAGCCCATCTCAC	423		119148919	CBL_002_Myeloid_Panel
GAGCAAGGAGCAGAGGGAGCTCC	424		119149195	CBL_003_Myeloid_Panel
'ACAGATCTGTTTCTGCAAAATC	425		25378528	KRAS_001_Myeloid_Panel
GACTGGGAGGGCTTCTTTGTGT	426		25380240	KRAS_002_Myeloid_Panel
GCATATTACTGGTGCAGGACCATT	427		25398189	KRAS_003_Myeloid_Panel
GTCCATTGGAAGGGAGGCAA	428		112888114	PTPN11_001_Myeloid_Panel
TCCGCTCAGTAATAGTCACTCT	429		112910737	PTPN11_002_Myeloid_Panel
GGTCACATAAGTCTGGACTGCTT	430		112915435	PTPN11_003_Myeloid_Panel
GGACAACAAGATCATTCATGGGGG	431		112926222	PTPN11_004_Myeloid_Panel
GGCAGGATTGAAGAAGAGCAGGT	432		112926832	PTPN11_005_Myeloid_Panel
GGCCAGGTCTCTGTGAACACACTG	433		28592580	FLT3_001_Myeloid_Panel
AAGGAGCATTAATAATGTAAACTCAAGT	434		28602311	FLT3_002_Myeloid_Panel
GTGGAAGGACAGCAACAAAGATGC	435		28608014	FLT3_003_Myeloid_Panel
GCTTCAGAGATGAATGATGAGTC	436		28608225	FLT3_004_Myeloid_Panel
TCTGCAGCATTTCTTTCCATTGG	437		28608194	FLT3_005_Myeloid_Panel
GCAACAAAAGAGTGTCACTCAGCG	438		28608421	FLT3_006_Myeloid_Panel
CTGCTGACACCCACTGTCCAAA	439		28609607	FLT3_007_Myeloid_Panel
GCTGTATCATGATTGGAAGTTAGG	440		28609721	FLT3_008_Myeloid_Panel
CGTGTGAAATAAGCTCACTGGCTGG	441		28610068	FLT3_009_Myeloid_Panel
GCAGATGATGGGCTCCCGGAAGAC	442		90631765	IDH2_001_Myeloid_Panel
GAGATAATAGTGTCCCACTGCAG	443		90631865	IDH2_002_Myeloid_Panel
GACAAGCAGGGAGGAGAGATG	444		7572891	TP53_001_Myeloid_Panel
GCTGGGAGGAGCAGGGGGGA	445		7573965	TP53_002_Myeloid_Panel
GTCTAACACTCAAAATGCCG	446		7576812	TP53_003_Myeloid_Panel
ACCAAGCTCTCATCTACTCCC	447		7577099	TP53_004_Myeloid_Panel
CCAAGGGTGCAGTTATGCCTCAG	448		7576986	TP53_005_Myeloid_Panel

lig_probe_sequence	SEQ ID	mip_scan_start	mip_position	mip_name
GTAGTGGATGGTGGTACAGTCAGA	449		7577458	TP53_006_Myeloid_Panel
GCGGCATGAACCGGAGGCCCAT	450		7577551	TP53_007_Myeloid_Panel
CCTGGGACCTCTGGGCAACCA	451		7578192	TP53_008_Myeloid_Panel
GTAGCAGTAGGGGGGCTTTC	452		7578114	TP53_009_Myeloid_Panel
GTTGAGGCGAGGGAGTACTGTAGGA	453		7578411	TP53_010_Myeloid_Panel
GGGCTGGAGAGACGACAGGGCTGGTT	454		7578360	TP53_011_Myeloid_Panel
TTCCACACCCCGCCCG	455		7578487	TP53_012_Myeloid_Panel
TTTCTGGGAAGGACAGAAGATGA	456		7579258	TP53_013_Myeloid_Panel
GTAGAGCTGCTGGTCAGGGGC	457		7579319	TP53_014_Myeloid_Panel
GCATCAATCATCATTGCTTGGG	458		7579436	TP53_015_Myeloid_Panel
ACCCAGTCCAGATGAAGCTCCC	459		7579518	TP53_016_Myeloid_Panel
AAGGCGGCCCAACACCCC	460		7579822	TP53_017_Myeloid_Panel
GCCTTCAATGGCCTTGTGCC	461		58740447	PPM1D_001_Myeloid_Panel
AAAAAATTTATCCAGAACTCAAC	462		58740327	PPM1D_002_Myeloid_Panel
AGCCTGCAAGTCTCCCCAACAC	463		58740654	PPM1D_003_Myeloid_Panel
CAATTTTCTCAAGTGTTCTGG	464		58740529	PPM1D_004_Myeloid_Panel
GATGTGAACTTTTTTAAGGGGA	465		58740853	PPM1D_005_Myeloid_Panel
GTCTATGCTTCTTCATCAGGGG	466		58740751	PPM1D_006_Myeloid_Panel
AGCTCGGGCGCTCCAGCAC	467		74732953	SRSF2_001_Myeloid_Panel
TTTCTGCTCCTGGACAAACCCGG	468		42531864	SETBP1_001_Myeloid_Panel
GGCTGCTCCAGGGCTGGACTGA	469		13054605	CALR_001_Myeloid_Panel
CTTGCCCCCTGCCAGCCCTG	470		13054500	CALR_002_Myeloid_Panel
GCAACTGCGGTGAGGCGCGGGCTGT	471		33792258	CEBPA_001_Myeloid_Panel
TGACCGCTCGCAAGCGGGTGGA	472		33792359	CEBPA_002_Myeloid_Panel
GCGGGGCTCTGCTTGATCACCAGCG	473		33792748	CEBPA_003_Myeloid_Panel
TCATGCCCGGGGAGCGCACGGGC	474		33792966	CEBPA_004_Myeloid_Panel
GCTGATGTCGATGGACGCTCTGTGCTCGC	475		33793061	CEBPA_005_Myeloid_Panel
GCAAGTGCTGCTCATCGGGGGCCGC	476		33793200	CEBPA_006_Myeloid_Panel
GCCACCCGACGGAGATGGGA	477		31021073	ASXL1_001_Myeloid_Panel
TCCCCATCTGCCAGGCACATCC	478		31021264	ASXL1_002_Myeloid_Panel
CCTGGGACACACAAGCCACT	479		31021171	ASXL1_003_Myeloid_Panel
GAGCAGGGCGCTCTGCATCCTTT	480		31021457	ASXL1_004_Myeloid_Panel
GCCTGCTGGGTAGTCTTAG	481		31021383	ASXL1_005_Myeloid_Panel
CACAGCCCACTAAAGAGAGGCCCA	482		31021554	ASXL1_006_Myeloid_Panel
CGGGTTGGACTGGCGCCAGGA	483		31022205	ASXL1_007_Myeloid_Panel
GTGGTGATGGTGGTGGAGCCTGTGG	484		31022405	ASXL1_008_Myeloid_Panel
GGTGGGGATATCCGGGGGCATAT	485		31022316	ASXL1_009_Myeloid_Panel
TGCCAGGCTTGCCTTACTGTC	486		31022664	ASXL1_010_Myeloid_Panel
GTACACTTTCAGGGGTGCTCGGG	487		31022579	ASXL1_011_Myeloid_Panel
GGATCCTGTAAATGTGACCCCC	488		31022903	ASXL1_012_Myeloid_Panel
GCAAGGCTGGCATGGCTGTT	489		31022803	ASXL1_013_Myeloid_Panel

lig_probe_sequence	SEQ ID	mip_scan_start	mip_position	mip_name
GGTAGTAAACACGCCCAACACG	490		31023107	ASXL1_014_Myeloid_Panel
GCTTGCCAGTTCCTTTCTCT	491		31023005	ASXL1_015_Myeloid_Panel
GGAGACTCTGAAGCACTGAGTCCT	492		31023348	ASXL1_016_Myeloid_Panel
CGATGGGATGGGTATCAATGCA	493		31023225	ASXL1_017_Myeloid_Panel
GTTGACAGTAAGTGCATTGCTGT	494		31023445	ASXL1_018_Myeloid_Panel
GGTGACCTTCAAGTCAGAGGCTG	495		31023541	ASXL1_019_Myeloid_Panel
GTAGCTTGCCCTAGAGAAAGT	496		31023730	ASXL1_020_Myeloid_Panel
GTGGGCGAGATTGGTTCCAATTGG	497		31023643	ASXL1_021_Myeloid_Panel
GCAACTGCATCACAAAGTGGGTAG	498		31023850	ASXL1_022_Myeloid_Panel
CCCTCCATCCAGTGACAAATCCA	499		31024024	ASXL1_023_Myeloid_Panel
GCAGTTCTCTCTTTAGTTGTG	500		31024088	ASXL1_024_Myeloid_Panel
GCAGGCCATGGCTCTGGTCTTTTG	501		31023955	ASXL1_025_Myeloid_Panel
CCTGTTTCTCATCTCAAAATGTG	502		31024203	ASXL1_026_Myeloid_Panel
AACCTTCAGCGCCCGAGGC	503		31024325	ASXL1_027_Myeloid_Panel
GTGGGTACAGACTCCAAGGGAAG	504		31024429	ASXL1_028_Myeloid_Panel
GCCGAGAACAGGAAAGTACTGGG	505		31024533	ASXL1_029_Myeloid_Panel
GAGGGGCTCAGTGAGCCTCTGGA	506		31024643	ASXL1_030_Myeloid_Panel
:CTTAGCTCTCCACCTTC	507		31024759	ASXL1_031_Myeloid_Panel
GCAGCACGGTGAAAGCATCTCG	508		31024876	ASXL1_032_Myeloid_Panel
GCTCTGTGTAITGTGCCTTGTGGT	509		31024985	ASXL1_033_Myeloid_Panel
GAGTCAATGGATCTCACCACCAAGCA	510		57484392	GNAS_001_Myeloid_Panel
GGTGGTAGGAGGGCGAGCTGGCTT	511		36164550	RUNX1_001_Myeloid_Panel
GCGCGGCAGAGTAGGTGTGGTAG	512		36164640	RUNX1_002_Myeloid_Panel
GGTGACCGCGCTCGGGAGTAGGT	513		36164725	RUNX1_003_Myeloid_Panel
GCGGGGGTCGGAGATGGAGGG	514		36164740	RUNX1_004_Myeloid_Panel
GCGGAAGTGAGTAGGAGGTTGCGGA	515		36164821	RUNX1_005_Myeloid_Panel
GTCCAGGAGACTAGAGGTGCATG	516		36171551	RUNX1_006_Myeloid_Panel
ACGCCATTTCACCTGGACGTG	517		36171668	RUNX1_007_Myeloid_Panel
GAGCACAGAGGGTTGGCGTG	518		36206645	RUNX1_008_Myeloid_Panel
CGGCTTGGCTGATCATCTAGT	519		36206751	RUNX1_009_Myeloid_Panel
GCGGCGCACAGCCATGAGGGT	520		36206820	RUNX1_010_Myeloid_Panel
GGGCTGGTACACCTCCAGGCT	521		36231754	RUNX1_011_Myeloid_Panel
CATCTTGCAGCCATCACAG	522		36252824	RUNX1_012_Myeloid_Panel
GCTGAGCTGAGAAATGCTACCGC	523		36252939	RUNX1_013_Myeloid_Panel
GCAGAGGAAGTTGGGGCTGTCGGTGC	524		36259124	RUNX1_014_Myeloid_Panel
GGCCAGACCTCCACCATGCTG	525		36259181	RUNX1_015_Myeloid_Panel
GCCAGTGACGTGACTGAGCACA	526		44514751	U2AF1_001_Myeloid_Panel
CATGGAATATGTCAGCAGCATGAC	527		44524436	U2AF1_002_Myeloid_Panel
GCATGCGGTGGAGGAGGGGACTG	528		15833892	ZRSR2_001_Myeloid_Panel
GGCCAATAGTTGAAAATTACTAC	529		15833769	ZRSR2_002_Myeloid_Panel
AAATTCAGGAAAGAAACACGCG	530		15836680	ZRSR2_003_Myeloid_Panel

lig_probe_sequence	SEQ.ID	mip_scan_start	mip_position	mip_name
TTTCCTCAATTGTTCCACTGC	531	15838326	ZRSR2_004_Myeloid_Panel	
GCTTCCAGGTCTTTGGAGCAAGAG	532	39911360	BCOR_001_Myeloid_Panel	
GTAATAATGAAAGTGCGCCCAAC	533	39911563	BCOR_002_Myeloid_Panel	
GTAAGGAGCTGTAGATCTGGTGG	534	39911472	BCOR_003_Myeloid_Panel	
CGTTCTCAACAGCATGTGCAGAG	535	39914615	BCOR_004_Myeloid_Panel	
CTTATGGTGTGACCCACC	536	39914711	BCOR_005_Myeloid_Panel	
TCGCCAGTCCAATGCCTTG	537	39932867	BCOR_006_Myeloid_Panel	
CATGTGTGACGCTTGGAAAGCATC	538	39933099	BCOR_007_Myeloid_Panel	
CAGCAGCGAGTTTCATCATGCCCG	539	39932988	BCOR_008_Myeloid_Panel	
CAGGGTTTTCTTCCCTCTGGCCTGAG	540	48649480	GATA1_001_Myeloid_Panel	
GCTGGAAGCTTCTCAAATGGATG	541	48649716	GATA1_002_Myeloid_Panel	
GGTGTGGAGGACACAGAGCAGGA	542	48649600	GATA1_003_Myeloid_Panel	
GCAGCTGCCTCTTTCCGTTTTG	543	53431919	SMC1A_001_Myeloid_Panel	
GCACTGCTGTGGCTTACTTTC	544	53432033	SMC1A_002_Myeloid_Panel	
GCCTTCAGGTCACTGGCCCCA	545	53432127	SMC1A_003_Myeloid_Panel	
CCAGTACTGAGCCTGTCCAG	546	53432252	SMC1A_004_Myeloid_Panel	
GACAAGGCATTGCCACAAGCAT	547	53432356	SMC1A_005_Myeloid_Panel	
:CTCACTCCAGGTAGTCAAG	548	53432571	SMC1A_006_Myeloid_Panel	
GCCGATTGCCCTTTGGAGGCCA	549	53432460	SMC1A_007_Myeloid_Panel	
GCTTTTGGAACTGGCTCAGG	550	53432798	SMC1A_008_Myeloid_Panel	
GGGTCAGGCCAGTGTTCAGGG	551	53432684	SMC1A_009_Myeloid_Panel	
GGCTATTGTGTGACCAACTTGGTC	552	123181254	STAG2_001_Myeloid_Panel	
GGTGAAACTAATCTAACAGACACA	553	123181181	STAG2_002_Myeloid_Panel	
GCAAGTTTGCATATTTCTGGTGT	554	123182853	STAG2_003_Myeloid_Panel	
AAGATGTGCCCTTCAGACTGCTT	555	123184027	STAG2_004_Myeloid_Panel	
CCAGTCGGTTCAAGGTAGTATTA	556	123184930	STAG2_005_Myeloid_Panel	
GTGTCTATGACCCCTTGACAAAG	557	123185048	STAG2_006_Myeloid_Panel	
GCATATTTGCACTAATGTTTCAGAT	558	123185136	STAG2_007_Myeloid_Panel	
GAAATAAGCAGTAACAGGTGCTT	559	123189974	STAG2_008_Myeloid_Panel	
ACCATGTCACTCATTAGTGAATC	560	123220383	STAG2_009_Myeloid_Panel	
CCACCAGCTAGCAAAAGAAATTCGG	561	123220499	STAG2_010_Myeloid_Panel	
CCGGCAGCAACAGAGACCTTGAA	562	133511669	PHF6_001_Myeloid_Panel	
GCCACTTTAAGTCTCAAGAAATGC	563	133511625	PHF6_002_Myeloid_Panel	
GCATTTTCATCATCATATAAGGG	564	133527622	PHF6_003_Myeloid_Panel	
GTATGTGACTTCTTAAGGCTGTAT	565	133527501	PHF6_004_Myeloid_Panel	
CCACGTTTCAGCCACTTTTCAG	566	133527920	PHF6_005_Myeloid_Panel	
GGGGAGGAAGAAATGAAGCACGA	567	133547793	PHF6_006_Myeloid_Panel	
GCCTGGTGTCACTAGGGCTGC	568	133547896	PHF6_007_Myeloid_Panel	
ATGCAGGAAAAATTAACATTCAGAAATCC	569	133549033	PHF6_008_Myeloid_Panel	
GACTGCAAGGTGTACATTCTG	570	133551218	PHF6_009_Myeloid_Panel	

SEQ ID	scan_target_sequence
571	CGGGGCGGTACCTGTGTCAGGAACTGCCACCTCAGCAGCAGCAGGCGCCAGGACGCGGCTGAGGCGCCAGCACTAGATGCAGAGCGGTCCACAAAGGAGATCCAGGGCTAGGAGACAAAAAGGG
572	GCCTTCGCTGTCTCATGTATATTGGTCTCTCATGGCACTGTACTCTTTGTCCAGCTGTATCCAGTGTATCCAAACAAAGGTTTCCACCATCTATAACCACTGTTTTCTGTAAGAATCCTGGGG
573	AAATGACTGATGACAACTGGTGGTGGTGGAGCAGGTGGTGTGGGAAAGCGCACTGACAATCCAGAACTAATCCAGAACTAATGATGAATATGATCCCACTAGAGGTGAGGCCCAAGTG
574	CCTTACACACGCAAAATACTCTTCAGCGGAGGAGAGGTGGCGGATGCTGCGACCTCATATCCGCGCCAGCACTCTCTGCTCGCAAGCGGCTCATGTTGGAGACGTACGTATAGTGG
575	TTTCTCCCAAGGTTATTTGGTTTCCAGTCCACTATACGTACGCTCCTCAACATGAGCGCTTGGCGAGGAGAGACTGCTGGGCGGTGAGCGGTGCGAGTCCATCCGCCACCTCTTCGCTCC
576	CTAGTTCAGAAAGTGAGGACATTACTACGAGGTCAAACTCAAAAGAGGCAAGACAGCATTTTCTGCTTCTAGAAAGAGAAAGGACATCTATGTCGACTGAAATGGAAGAGTGA
577	ACATGCTCTCTGCTCCTAGGCGGTTGGCATCCACTGTGAAGTGAAGTGCAGGAGTGTCTGGAGATGAGCAAGTGCAGCTCCAGCGCTAGAACCTCTGCTGGGG
578	CATCTTTCAACCGCTCTGTTTGTAGTCCAAACCTGTGATGATTGATGCCAAAGAGTGTAGCTGCACAGGCGCCGCTACTCTGGGGTAACTTTCCCGGTATGAACAGGTTGTGAAAA
579	CTGGCCAAACCAAGGTTGCTGGCTATACCTCGAGAAATCGGAGAAATGCTCTTGTCACTAACGCCATGCGCCACCACTTCTCAAAGAGCCAGAAGAGGCGCATCTCCTCTCTTGGGCC
580	CTTCCGACCTCTCAGAGGCACTGGCGGCTCTCTTTGAGTTCTACCGCTCTCTGATGCGGCGCCAAAGAGGAGATGATCGCCCTTCTTCTGGCTCTTTGAGAAATGTTGGTGGCCATGG
581	CCTACCTAGAGGCCCTTCGAGCAGGGTTGACGATGGAGAGTCAATGAGGGACTGCCCAATCACAGATCGAAATGGGCCCCACTCTGGATCTGGAGGATAAAGGCAACGTATGGGCCT
582	AATGTAGGGGTCCACTGAATGCCCAAGTCTTCAGCACAGGAGGCCCTGCACACGCCAGCAGACAGCACCGTTAC
583	GGACCGCTACATTGCTCGAGGTGTGTGAGGACTCCATCACGGTGGGATGTTGCGGCACCAAGGGAAGATCATGTACGTCGGGGACGTCCTCGCAGCGTCCACAGAAGCATGTAT
584	ACCGCTGGGCTGCATCTGACCTGTTGTGCTCACTGCTTAGGACCTCTCAAAGGTTTACCCACCTGTCCCACTGAGAAGAGGAGCCCATCCGGGTGCTGCTCTCTTTGATGGAATCGCTACAG
585	GGTCGTGTTATTAGCGAAGAACTCTGGAGCGGGAGGGCCAGTCTCTTCGCCCGCAGCAGCCGCTAGGTACCCCTTGTGCCCCACATGTAGCATGTTCCAGGGGT
586	GGGGCGAGAGGACTGGCCCTCCAGATGTTCTTCGCTAATAACACAGCACAGGAATTTGTGAGTGTGG
587	:CTTCAGGTGCTTTTGGTGGAGTGTGTGGACCTCTTGTGGGGCGGGGCTGCCAGCGCCATTAAAGGAAGA
588	CTGTGTAGAACTGCTTTCTGAGTGTGCGTACCACTACGACGACGGCTACCACTCTACTCTACCACTCTGCTGTGGGGCGGTGAGGTCTCATGTGCGGAAAAACAACAACTGCTGCAGGTGAG
589	ACCTTGCACTTTTGGCACATCTCTCAACGAAGAGGGGGTGTTCAGGGTAACATTGAGGCTCCACAGAGATGCAGATGTCTGTGAAAGCAGAGGGAGGGGATGG
590	ACATCTGCATCTCTGTGGAGCTCAATGTTACCTTGGAACCCCTCTTCGTGGAGAAATGTGCCAAACTGCAAGGTAGGAGCACACCCACCAGGAGAGG
591	GCCCCATGCCACTAGGAGTGCAGAGTCCCAGSCAACAACTTACCTCAATGTTCCGGCACTTCTGCCGACCTGTACACCAGCGCTCTGCAAGSGGGAGGAGAGCTGGCGTCAAGAGGAG
592	GAAAGCGGCCAATCTCTGTGGCTCATTAATCTCTGACCTTGGCTTCTCCGCTGTCTCTTCGGGGCTTTTGGCTGTGGAGGTGTCGTAGGCACTGCTCAGGTGTTCCACC
593	TGCGTAGCAGCTGCTCAGGTTCCACCATGTCCTGTACACTTCTTTGAGGATTTCTCTCTTCGGAGGAGAAAGAGTGCACAGGTCAAGTCAAGGCTGACAGGAACTCCAGCCCC
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817	GCTCACCCAGTAGTTGTCTGAGGCCAGATCACTGGGTGGAGCCACTCTACAGAGAGCCAGCAGAGCTGAATTTCTGTCGTGAATTCACAGATCTAACACAGCTCCTTACTTTTCAGGGTTGAAG
818	AATTTCCACGTTTGGAAAAATGCGAGGAAATATGCGGAGGACATTTTCAATTTCTTAAGGACATCCGAAGCAGTAGCAGTTTCTGGCTTACAAAACAGAAAGGAAAAATGCTTTGAGTTTCCA
819	GCATATTCGCTGCAATTTTCCAAAGCTGGAAATTTGCACATTCGACAGGCAAGATTTATTCGGCAGGTTTCTGCAAGTCTCTTGTCTCTGCTCCAAAGACCTGGAAGCCTTCAACCTGAAA
820	TCATACTGTTAAGAACTTTCCATAAGTTCACTGTGGGTCAITTTTCATGATGGTTTACCTGAGTAGCAAGGTGGGTGACGACCATAGAGAGAAAGTAGTCGGACAATTTTCAAGTGAT
821	AAAGGAGACAACCTGCTTTAAGCAAGTGTGGCTTGCTGAGCATAATTTCTATTCGCTCTCTCTAGGCTCTGACGATGCTGTTGAGAACGATCACTTGGAAATTTGCCGACTACTCTCT
822	CATTGGCATTTGGGGCGGGTGTGCGGAGGCTGGCGGCTGCACTGCACATGACCTGAAACCTTAGTGATGACAGATCGGTGCCCGCATGCGGCGCATGATGAATCCGCTGCTGTGGTA
823	TTGAGATCTAGATAGCACAACTTTCTCGAGGAGATAGTGTCTTTTCGGAACTCTCACTTCGAGAGACATAAGCCACTTCAGCCCTCTGCTGTGAACAGGACCTGGGAGCCATCTTTTT
824	ATCTATAGATCTGAATCATCAGCACTGCTCCCTCATCTGGGTGGTCCCGGCGCAAGCTCTAGGAAGACAAATGGCAAAAGCATGTGCTGAAAAACAAAGGCAATGGAGCTGGCGGATACCA
825	CCCCTTCTGCTCGCAGGTTAATCCCCAGAGCTCCATGGAGTTCCCTGGCTGGGTCCCTGAGGCCCTACAGGCCCTCCCCAGTTTGTGAGTCTGCTCTGGTGCTCCACACCGAAT
826	AGGCTACAGACACTCCCGAGGTAACCTCATTGAGTGGCTGCTTGGCATGGAGATCCTTGGGTTGCCATGGAGATCCTTGGCTAGGTCAGAAATACCACCTGTGAGGATATCTCAGAAATG
827	CTGTAGGCTCAGCGTCCCTGTAGGCCAGTCCGCACTGCAGCGTGGCTGTGCTCGGGCAGTGGAGGAAGCTGCTGCATCCAGCCCTCAGGCCACAGGGGGAAGAAACCCCTGATTTCT
828	AACAGGGCTGAAGGCCAGGCCACTGCAGATTTCAGGGCTAGATGCTGTCTTGGTCTGTCTAGTCACTCTGGGAGTACTTGGCCGATCTGCAGTCCATGGGCTGAGACTGCACCTGAC
829	ACTGCACCTGACGAGCTCTGCCCTTCCGTTTGGCTTCACTGCTCCTGAAGGGAAACAAGAAAGGGCTAGGTGTTAAGGTGGTGGCTGACCTAGGCTTAGGACTCCCACTGCTAAAAACA
830	ACCTAGGCTTAGGACTCCCACTGCTAAAAACAGCACTGCTGTGGCTTACTTTTTCAGCGCTCTCTCTCTTCAACTTGTCTACTGCTTCTCATCCAGCGCCGCTGGCTTG
831	GGCCTTCAGGTCACCTGGCCCCACAGAGATCACTCTGACTTTCTGGAATAGGGTTTCCATCCAGTGCCTGTCTACACAGCAGGGGGAAGAGAAAGGGGGAAGAACTGAAACAAATGAAATCT
832	GAGAAGCTGAACAAATGAATCTCAGTACTGAGCTGTCCAGCTCCAGCTGGGCAAGGGAATCCACACTTTGGGGCTGTGGCTCCAAAGGCAATGCGGCGGGCATCTCCACGTTGTACA
833	CAGTCCCGGAGTTTCTCATCTGTAGGCTTCACTGTGGGAGAACTCAGTCAGTGGCAGAACACACAGGAGTACTAGAGAGGGGCCCTTGAACACTGGCTGACCCAACTCCCAACAAG
834	AACTCCGGGAGCTGAAGGGGGCAAGCTAGTATTGATTGCTATGAGCCACCTATATCAAAAGGCCCTGCAGTATGCTGTGGCAATGCCCTTGTCTGTGACAACTGGAAGATGCC
835	TAATGGCATCATGTTCTTGGCCAAAACCTTGGTTACAGCAATCTGATACTTTTGTGTGGCTGGCATAGTCAATGAGGCGGCCCTACTGAGTAAAGTAGGAAGGAAAACTGAAGATGAA
836	TGGATGCCATTATTTGTGGACTCGAGAAAGACAGCGCGGACTGTATTCAGTATATCAAGAGCAGCGTGGGAGCCTGAGACCTTCTGCTCTTGACTCTGGAGGTGAGGCTTGTGGGATTT
837	AATATGGATAATACAAAGACAATATGAAGCAGACGGAATAAAATGATTGAAAACAGGCAATGAGAGCTGAGAACTCTGCTACAAAAGCGGAAAGAGTAAACTTTTATTTGAATATTTA
838	CCAAATCATTTTATTCGCTCTGCTTTCATATTTGTTGTTATTCATATTAATGCTAAGATTAGTGCCCATTCACCAAGCTGTCACTCACTTCTTAAAGAGTAAAAAGTAT
839	AGCTTCAGGAAATCAAGATGAAATAGAAATATGATGAATGCAATTTTAAAGGAGTGTGTGTACATAGATACCGGTAAAGTTGTGACAGTTTTTTTCATAAATAGCATTATGTAAATTTCTACTCA
840	CCCATTCAGTGATGGGATAGCTGAAATTCGAGCTATTTGCAATGAAGAGATTGGCATTTGGATGAAGATGTATAGTAGTGCCTTCTTAAAGACATTTTAAAAATATGTTGGTTGGACTATGCATGAAGGT
841	TTTTAATGCAATGCTCATCTTTTTTTTTTTTTTTTATAGCAAGGTGAAGTAAGACTCAATGTCTTACTGCTACAAAGGGCTTTATATACAAAGAGCTTAATCCAACTGGAACTTTTTA
842	ACTTTTACCAGTCCGTTCAAGGTTAGTATTAAAGAAATTTACAAATAACTTGTCAATTTGCAACACTTTTCTCTGCTTCTTTTAAAAATATTTTAAATTTTGTCTTAGGATAGAATT
843	TAAAAATATTTTAAATTTTTTGTCTTAGGATAGAAATTTGTCTAGCCCTTGACAAAGAAATATGATTTGCAGTACAAAGCAATAAAAATTACTCACTCTTGTTTTACAGTAAGTATGTATTTGTT
844	ATATATAGATTTACTTTTGTAGAGAAATCTCCAGCTGCTACTGCTGCTGAGCTGAATTAACACAGATGATAGACATTTTCACAATCTCTGCACTGAGAACTTCTTCACTCTCTTAA
845	GATTTCTTTTCAAGGATGTTTACTTGGAAAAGTTTCAAGCTTTCAGATGCTACTCCGAAAGAGGATGTGGCTTCCACTGATGCTTACCAGAAATCTTGTGCTAGCTGGTGGTGAATGAC
846	TTACTGAGTGAAGGCTGCATGCCCTCAACCACCTTCCGTTTCTGATGATGTTTGAATTTTACTCCGTTACTGTTGACCCCGGCTGCTGATTCCTAAATGACTGACATGGTGCATCATCA
847	AAAAAAGGCTTACAGACAGCGCAAAATGTGGCTTTTGAAGTCAAAATAGAGACAGGAATGTGGACAGTTACTAATATCTGAAAACAGGAAGTGGCAGCGCACTAAAGTGCATGGTAAAGTATA
848	CAGATATGATGTGCCATTCCTGTCTATTTGACTTACAAAAGCCATTGCGCTGTCTGTAGGCCCTTTTCTGTTCAACTGAGCTGACATGATTTTAAATTCGCTTTAGAAT
849	ATAAGCTCAAAACAGAGAAACCTTCAAGGAAATTTACATGTTAATTTTAACTTCTTAAAGTTTCTTAACTAATACTTCTTGGGCTTTGTAAAAATTTTTCCTCATTTTCAAA
850	TTTATCATGCAATGCACAGTGTAGTGGTATGCTGTGACATGTATTCACATCAACCAATGTGCTCCAGCAAGTGGCACAAGAACACATCTACAGCAAAACGACAAAAATAATATTATA
851	ATTTCTCATTTTTTATAGGGTCTATTTGGGAAAACACAAAGAAACTGCACATACTCCGAAGGTACATCATTTAG
852	AATACCGATAGATATTTTTCATGATTTTTTAAAGTCTGTTTTTCTTTTACATTTGCAGAGATAGGCTCCACAGAAAGCAGCCCTAGTGACACCGGCTAAATGTGGATTTTGGCCATGTA
853	TTGGGCTTAAAGAACCATGCTTACCATGCATTTAATGGCAGCTGCCTCTTGGCATTAAATATAGAGTTTCTCTGCTTCACTTTTCTTCCCTACATGGCAAAATCCACATTTAG
854	GACCATTTCTTTTCTGTTAATCTCTGAAGTACAGTTTAAATCAAAAGCTCCAAATTCCTGCTCTGATGTTGTGTGAGCTGGACTGTGCCAGAAAGAAACAACTAGAGAAAGAAA
855	TGTTTTCTACTGTAAATTCCTCGTGACATATTTTCAATGTATTAGCTTTGTCTGTACTCCACAGTGGTAAAGTTTAAACACAGGCTTTTATTTTCAACATCCCAATAGTAGCACCAGGCT

SEQ ID	mip_sequence	feature_start	feature_end	position	strand	probe_failure
856	TCAGACTTCGGCCACCCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNNTGGTCCACCGCCAGTCTCTCGCTCG	43814931	43815035	-		0
857	GTGTGAGGGTAAGGGGCGAGGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNINGATGCAAAATACACAGAGAA	115256486	115256577	+		0
858	GTAGCCCGTACCTGATCTGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNCAACAGGTTCTTGTGGTGTG	115258743	115258748	-		0
859	ACTGGAAACCAAACTCCGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTACCTCAGTTTGCCCCCATGTC	25457148	25457289	+		0
860	GCTGAAGGAGTATTTGCGTGTGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTACCCCTGCTCTGCT	25457144	25457294	-		0
861	CCGGGTTGTCTGGCATCTGGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTCTCTTTCTCTCTTCAT	25458573	25458698	-		0
862	GCAGGAGAGGAGGCGAGATNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGTTATCCAGGTTCTTGTGTT	25459792	25459882	-		0
863	GCTCTGGGCTGGGGGCTGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNGTGTTCATGCTCTCTCTGG	25461992	25462092	-		0
864	GGCATATGACAGAGGCGGTAGANNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNAGGTAGAAGCCATTAGAG	25463166	25463483	+		0
865	GCGTTAGTGAAGAGGACATCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGCCAGCTGATGGCTTCT	25463166	25463483	-		0
866	GCTGTCAGGACAGAGCGACANNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGTGGAGGGACAGGATGGTA	25463503	25463605	+		0
867	TTGGAGCATCTCTGGACCCCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTCTCACACACCTCCAGGC	25464533	25464580	+		0
868	GTCCATGCTGTGGGCGCAGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNGCTGAAGGACTTGGGCATTCAGT	25464429	25464508	-		0
869	GTAGGGGTGCAGGCCAAGAGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGCTGGGTGGGAGCTTGGGAC	25466760	25466845	-		0
870	CTTCTTAATGGCTGTGGGCGAGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNCCAGCACTCACAAATTCCT	25467088	25467139	+		0
871	GCCTGGGCGGCTCTGAGCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNAAGGCTACCTAGGGCTGTGC	25467018	25467212	-		0
872	:CCCTGGAACCTGTACATGTGCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGTCTCTCTGCTACTGGTCT	25467018	25467212	-		0
873	GCTGTGTGGCTCCAGTGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGTGTTCGTGACCTTCCCGCTG	25467407	25467525	-		0
874	GGTAGTACCAAGAGGCTCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTCTGGGTGGGTGTGCTCT	25468119	25468204	+		0
875	TGCGTTGAGCGGGCTCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTCACTCCCTCTCTGTTTCCAG	25468119	25468204	-		0
876	CCGGCTGTCTCTGGATGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNCTTCTAAGTGCCTCTGCTACTCT	25468879	25468940	+		0
877	CACATGTCGTGTACACTTCTTGNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTCTGGTGCACCTCTCCA	25469025	25469183	+		0
878	TTCCCCACCCTCTTACAGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGGCTTTTGGCTGGAGGTGG	25469025	25469183	+		0
879	GTACACGCCAGGTTGGGGTTGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTAGGGCCAGAGGCTGGAAG	25469487	25469650	+		0
880	GGTGCCAAAGCCTCCACAGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTGATTGAATGGGCCCTGGGGGG	25469487	25469650	-		0
881	CACCCACCCCTGCTTCAANNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNGACCTCGTAGATGGCTTTGCGGT	25469917	25470030	+		0
882	GCATAGAGGCTGGAAGTTGNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNGTGTGTGTGAGAAAGCTGATG	25469917	25470030	-		0
883	TTCCCCACACAGCTCCCAANNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNCTACTGCCAAACCCACAACCTT	25470458	25470621	+		0
884	GTGCTTGTGATGACGGGCGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNATGGGGGATCAGGGTGGCAGGG	25470458	25470621	-		0
885	CGGGCCCTGGTTTCTTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTCTGGCTCGTCATCGCTCTTGGT	25470974	25471055	+		0
886	AGTAATTTGTGATTTACCTTTCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTAGTTGGCATATTTCTGCAT	198266442	198266624	+		0
887	CTGGATGTTTCTATGTTCTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTGGGTATCTTCTCTTATGG	198266442	198266624	-		0
888	GTGTTAAAGCCTTTATGGAAGGTTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTAGTAATGTTGGGGCATAG	198266810	198266849	-		0
889	GAAAGGACATCATGATGTGTTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNGGCAATAAAGAAAGGAATGCC	198267481	198267491	+		0
890	CTTATGGGCTGTGCATCTTGCCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNCGTAAACAACAGCTAGAGCT	198267342	198267373	-		0
891	GGTGCCATTTGGTATTTCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNCAAAATCACATTATGCCAACATG	209113112	209113210	+		0
892	TGCAGGAGAAGTATCCCCCTTCNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTCTGAAAACCTGGTGTGCT	128200060	128200163	-		0
893	GGTCCGGCTCTCTGGCGGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNCA TTGTCAGCTTGTAGTAGAGGC	128200661	128200789	+		0
894	CACATCAAGCCCAAGCAAGNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNTGTCTCTCCCTGTCCCTCG	128202700	128202851	-		0
895	GTCCAGGAACCTGAGCAGAGGTNNNNCTTACGCTTCCGATATCCGACGGTAGTGTNNNNTTGTCTGAGGTTTTCCAGCACTC	55589745	55589869	+		0

SEQ ID	mip_sequence	feature_start	feature_end	position	strand	failure_flags
896	GTGAATACACATATTAGGTTGGAGGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGCAGGACTGTCAAGCAGAGA	55599285	55599349	-	0	0
897	GCCCACTGCCTGAGAGAGCTCATNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNACCAACCATGTTGAGGGCAAC	106155099	106155185	+	0	0
898	GTAGAGGGTATTCCAAGTGTTCGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNAAATGGAGACCAACGAAGTGGC	106155144	106155319	+	0	0
899	GTTCTGTGGCAATGGAGGTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGCTTCCCTTCATACAGGG	106155144	106155319	-	0	0
900	TCTGAGCCCAAGAAATCGAGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNCTCTGGGCTCTTCAGATCAAG	106155044	106158550	+	0	0
901	CGACTATTCTGGCTTCCCTCCTNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNGTCTTCTCCATTAGCCCTTTGG	106155044	106158550	-	0	0
902	TGGAACACACATGTTGAATCTCTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCCAGAGCTTCAGATTCTG	106155646	106155733	+	0	0
903	CATTGGTTGAGCTTTCAGCTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNACTTTTCCCTCTCCTGCTCAT	106155044	106158550	-	0	0
904	GTGAGTGAGCCCTGTGATGTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNAGAAACCACTCATACATA	106155044	106158550	+	0	0
905	GCACATTAGGCATTAGCACTGCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNACTCATTAGAGCCTGACTG	106155044	106158550	-	0	0
906	CCAGCAGCAATTTGAAGCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNFACCTGTTCTTCAGAAACCAAGAA	106156091	106156165	+	0	0
907	GCACTGGCTTTGGAGGCAGCTNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGTATTTCTCGAGGAGATGGG	106155044	106158550	-	0	0
908	CACCACACTACCCCAACAAANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNCTCAGTGTTCACATAAGGATTCC	106155044	106158550	+	0	0
909	TGAACAGAAATTCACCANNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNGCAATTGTGATGGTGGTGGTGT	106156165	106156274	-	0	0
910	CCAAATGTCAGAACACCTCAAGCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNACCACTTCCCAAGAGTCTAAT	106155044	106158550	+	0	0
911	GCTTTTCTTGAAGGAAGCTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGAGGGCTGCATACATGTGT	106155044	106158550	-	0	0
912	GCTAATGAGGCATCACTGCATCANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNTGATGAGAAACAAGAGCAA	106155044	106158550	+	0	0
913	.CAAAATGGAACAGTCAATGTCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCGTGTTCCTCTGCTCG	106155044	106158550	-	0	0
914	CCCTACACCAGGTGCACTTCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCTGAAATTCCAACACTGCTGGG	106155044	106158550	+	0	0
915	GGGATTCGCTTGGTGAAGAACGANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGCTGTGTTTCTGGGTGT	106155044	106158550	-	0	0
916	TCCAGAGTTACATCTCCCTCANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCAACAAGACAGATTCCCAA	106155044	106158550	+	0	0
917	GGAATGAGAGATGTTGGTCCACTGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGTCTGTTTCAACACTGGG	106155044	106158550	-	0	0
918	GAAATCCCTTATAGTCAGACCATGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNATGATCAACAAGAGAGAAG	106157293	106157510	+	0	0
919	GGTTGTGTTGTGTCGCTGTTTATGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNTAGTCTGGCCAAAGAAT	106157294	106157418	-	0	0
920	GCTTTCAGAGACAGGACAGAGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNCTGTTTCAACAACATACACACC	106155044	106158550	+	0	0
921	GACATTATGAGTCTCGAATCGCTNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGATGTGATGCTGTTCTTT	106155044	106158550	-	0	0
922	CAGCAACACAGCAACCCCAACNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNCTGCTCAGCAAAAGGTACTTG	106155044	106158550	+	0	0
923	GTGAAGAAGATCTTGTTTGGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNAGCAGAGGAAAAACATTTGCA	106157742	106157861	-	0	0
924	AATGTGCAGCAAAAGAGCATATTGANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNACTGATCTTGCCATAGT	106158077	106158150	+	0	0
925	GCAGCATGCTTTGAGTGTCCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNTGTTTCTGGTGGTCTGTGTC	106155044	106158550	-	0	0
926	CTTCTTCAGAAAAGACACCAACNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNGACCATAAAGGCTCTACTCTC	106158273	106158480	+	0	0
927	CACAGCTTGACGGTGGATTCTCANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGTCAAAACTGTGACTGCCCTTG	106155044	106158550	-	0	0
928	CTTCAGATATGGGATTTCTCTNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNAGACTCACCTTCCAAATTAAT	106158273	106158480	+	0	0
929	GCTGGGGTGTGGCTATCAAGTCTCANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNGAGTCTTGACAGGTATCC	106158273	106158480	-	0	0
930	GCAAGGACACAGGCGAGATAACGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNTAGTATAATTGAGGTCTAA	106162468	106162605	+	0	0
931	GCCTTTGGTCTTAAATCTTGGNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNTGATGTGTGTGTGTTCTGT	106163988	106164088	+	0	0
932	CCAAATCCGGGTGTGCTTGAATNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNTGTGTTGGTGGGAGCGAG	106164823	106164875	+	0	0
933	CCCCCAACCCCAACAAAAANNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCGGATTCCTTCCACACCA	106164725	106164940	-	0	0
934	GCTCCGAGTAGAGTTGTGAGCCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNCACGCTGAACTCTCTCTCTT	106164725	106164940	-	0	0
935	TTTGCAAGAAGCAAGATCCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNNTGCACAGCTATATAATGCTATCC	106180773	106180933	+	0	0
936	TTCTGGATCCAGCCCCTGACAGCNNNNCTTCAGCTTCCGATATCCACGGTAGTGTNNNAGCATTATACATCAGGAAG	106180773	106180933	-	0	0

SEQ ID	mip_sequence	feature_start	feature_stop	position	strand	probe_failure
937	GGCAGCAATTGTAACAACTTACTTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAGAAATATTTCACCTTTATAC	106182914	106183008	+	0	0
938	GAGGACAGCTTAGCAGCTGTTGAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAAATATGAACACAGACGACCA	106190761	106190911	+	0	0
939	GAAACTCACTAGTATTTAGACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNAGCAGAGATCCAAACATGC	106190761	106190911	-	0	0
940	GTCAGACTGCGCAAGAGANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGGATGAGCAGCTTCAGTTCTG	106193714	106194080	+	0	0
941	GTAAGACATTACAGCTCAACTACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTTCTCTCCTGAGCTT	106193714	106194080	-	0	0
942	GCATTTGTAGATAAATGTTGTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGAAGCAAGAAAGCTCGACG	106193714	106194080	+	0	0
943	GCCGAAAGAACTCAGTACCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTGTTTGTACGTGATGGGCTG	106193918	106193967	-	0	0
944	CCCAGCAGCAGCAGCAGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTCTCTCTCTACCTGTGCC	106196203	106196292	+	0	0
945	CCATGAACCTTACCTTCCGCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCCACCAATCCATCAGATGAGAC	106196203	106197683	+	0	0
946	GCTGGGCTGTGGTGGCTGCTTCTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNGATATCTGAAGTGTGGAAG	106196203	106197683	-	0	0
947	GCCAAAGTTTGAAATAGCCAGAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCAATGCAATGGAAACCTATC	106196203	106197683	+	0	0
948	CCTGCAGCTTGAGATGAGGTGGANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGGCTGAGACTGGGGAGAATAG	106196203	106197683	-	0	0
949	GGTGAACATCTTACCTTCTCACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGTTTCAGCAGTTGTACCAAT	106196203	106197683	+	0	0
950	GTGTATGGATGGGTGATAGCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGAGTGGGTAAGGAGGCAATT	106196203	106197683	-	10	0
951	GCTTCCACACAGCTAATGGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNACCACCAATCTGAGCAATCC	106196203	106197683	+	0	0
952	GTGATGTAATGGTCAGGAAAGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCAATGTTCAACAGCTCTCTTC	106196203	106197683	+	0	0
953	TCACCCACAGGATCTCCNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCAACGATGAGTCTGGTCAGACAG	106196203	106197683	+	0	0
954	TCAGTTCTATCATGGTTAAGAGCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTAGGATCCAGAAAGCTC	106197125	106197218	-	0	0
955	CAGAGCCACTTACCTGCTTTCANNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNACATGCTGGCTCTTGGG	106196203	106197683	+	0	0
956	GCAGCTCAGCTTTGCACACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTTCTCTTCTCACGGGCTTTT	106196203	106197683	-	0	0
957	GAAGTTTCATGTGGCTCAGAGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNACAAAAGGGGTGATATCAT	106196203	106197683	-	0	0
958	CCACAACCTTCATAGACATCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNGACAGCCAGATATCAACTGTT	170837531	170837568	-	0	0
959	CAGTAGATCTCATTTCTATCAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNACCATCCACAAAATGGATCC	140453092	140453191	+	0	0
960	CACAACAAAGCCTGCTGAAGATAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNAGGGGGAGGAGGTAGCAGA	148504736	148504801	+	10	0
961	GGGCTTTTCTACTGGATTGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGCTCACTGACACCAAGTGTGTCT	148506160	148506251	-	0	0
962	GTAAGCACAGCCAGTGAATNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCTACCTTTTGATAGCAGTTTGG	148506400	148506485	+	0	0
963	GCTGGGAGGCGAGTGTCTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNGTCAGGCTTGATCACCCTTATCC	148507415	148507487	-	0	0
964	GGCACTGATAACCTGTATTGAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTTCACCCCTCTTTTGA	148508716	148508816	-	0	0
965	GGAGTTCTTCACATCACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTACTGTCCCAATGGTCAGCGGC	148511048	148511235	+	0	0
966	GTAGTTAGCTATTTAGTGATCAANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCTGGCTGCCGAGAGTGTGA	148511048	148511235	-	0	0
967	GCATGAGAACTAAATAGGCTTTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCTTAACCACTGTTTACAAC	148511999	148512138	-	0	0
968	GCTTTGTTTCTTTGTTAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGCTCTCTGTGATTTGTAGCT	148512570	148512669	-	0	0
969	GCAAGGGTACTTGAGAGGCTTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTATAGATTCTTTGTTTCAT	148516685	148516783	-	0	0
970	GTTTCTAAAGGTTTCCAGTGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCAGATTAGCATTTGGTCCA	148523544	148523730	+	0	0
971	CGTTTCTATTTCTATCTTTGTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNACTTCTCTGAAATGTAACC	148523544	148523730	-	0	0
972	GAGCCATATGCTTCTCTTGGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTTCTTTAGTGGAAAG	148526828	148526931	-	0	0
973	TTGTTCTGTGGAATAGTCTCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTACACCGCTGTACAGAA	117864785	117864951	-	0	0
974	GAGGACCAACAGCAGCATNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGAAATTTTCTTTTTTTTTT	117864785	117864951	-	0	0
975	GAATAATCACTAAGTTGGCTCTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGAAGTAAAAATTCGCAACT	117866482	117866595	+	0	0
976	GGATCTGCACCGCCACANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCAAGAAAAATGATCTTATTTTAT	117866604	117866713	-	0	0
977	CCTTTAGATTTATACAGCATNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTTTTTTTTTTTTACAGTG	117868403	117868530	-	0	0

SEQ ID	mip_sequence	feature_start	feature_end	position	strand	probe_failure_flags
978	AGACTAGAAAGTTTGATTATCTGNNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNNAGGACAAAAACACAGTAA	5069950	5070055	-	0	0
979	CAGGATCACAGCTAGGTGTGAGTGNNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAGCAAGTAGTATGAGCAAGC	5073739	5073781	+	0	0
980	CTTTTACACATCGTGAAGTGNNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTCATTTCTGACAACTTAC	112350168	112350333	+	0	0
981	GCTGGTTATCTCTTCGACGGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTAAGCTATTCTTATCCCTCT	112350168	112350333	-	0	0
982	GTTAGATGTACAGGATACAGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTGAGTACCAATAAAGATTGG	112350747	112350893	+	0	0
983	AGTAACTCTCCAGGAAGATTTCATNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGTATAGTGCCTGGCACT	112350747	112350893	-	0	0
984	CAGCTGGCCCTGCTTCACTANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTGTGTGATCTCTCTGTTG	112352828	112352984	+	0	0
985	GAGCTTTCAAAACACATGTTTGNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCGCCACGACCAATTATATGTT	112352828	112352984	-	0	0
986	GAAAGCTAATGAAACCTCGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNACAGCACTTATACATATGTT	112356153	112356245	+	0	0
987	GCCTGGCCCTTAACAAATGTGGGCAANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNGAAAAATAATGTGAAGAAA	32413516	32413615	+	0	0
988	GCTGTGTTCCCTGGGCTAGGTTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTCAAAATAGAATATGTGCTT	32414197	32414308	-	0	0
989	GTCTGAGGGAGGTGACACTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGGTAAGCACACATGAAGGGG	32417797	32417956	+	0	0
990	GAAACATGTTCAAGAGCTCTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTTGTACGGTGGCATCTG	32417797	32417956	-	0	0
991	AGGCTCAGTGTGGCTCACAGTCGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNTGGGCGCTGTCTGTGTGCTC	32421491	32421591	+	0	0
992	GAGCCCTGTGGACACCTCATNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCTGTTAAACATTTATAATTGCAGT	119148874	119149011	+	0	0
993	GTTGGAATGTGGAGCCCATCTACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNACCGAAATTTTCCAAAGTTTA	119148874	119149011	-	0	0
994	GAGGACGAGAGAGGAGCTCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAGTATTTTCAGATGCACTG	119149218	119149335	+	0	0
995	'ACAGATCTGTTTCTGCAAAATCNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGTACCTATGCTCTAGTAGG	25378562	25378647	-	0	0
996	GACTGGGAGGGCTTTCTGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNTGCACTGTAATAATCCAGAC	25380275	25380285	-	0	0
997	GCATATTACTGTGCAGGACATTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNATAAGGCTGCTGAAAAATGA	25398211	25398286	-	0	0
998	GTCAATTGAAAGGGAGCAANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCCATTCTCTTTTAAATGGCC	112888139	112888229	-	0	0
999	TCCGCTCAGTAAATAGTCACTCTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNCACGTAATAATATTGACTTTC	112910747	112910847	+	0	0
1000	GGTCACATAAGTCTGGACTGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGGCTAGAAATGTATGGTCTAG	112915440	112915540	-	10	0
1001	GGACAACAATCATTTCTGGGGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCATAACTAAAAACAGAAAC	112926228	112926328	-	0	0
1002	GGCAGGATTGAAGAAGACAGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAGTGTTCCTTCGTAGGTGT	112926848	112926919	+	0	0
1003	GGCCAGGTCTGTGAACACACTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNACAAAAAGCCGTATAAAA	28592612	28592705	+	0	0
1004	AAGGAGCATTAATAAATGTAAAACTCAAGTNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTTTCGTGGAAGTGGG	28602314	28602426	+	0	0
1005	GTGGAAGGACAGCAAAAGATGCNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGCACGTACTCACCATTITGTC	28608022	28608130	-	0	0
1006	GCTTCAGAGATGAATGATGATCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCATTCATTACCAAAC	28608200	28608360	+	0	0
1007	TCTGCACATTTCTTTCCATTGGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNGAAAGCCAGCTACAGATGGT	28608200	28608360	-	0	0
1008	GCAACAAAAGAGTGTCACTCAGCGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAGAGGAAAGAAATAATGAATT	28608437	28608546	+	0	0
1009	CTGCTGACACCCACTGTCAAANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGTGGGGAATTCCTGTGGTGG	28609629	28609815	+	0	0
1010	GCTGTCATAGATTGGAATAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNACCCCTTTATGGCTTCACTC	28609629	28609815	+	0	0
1011	CGTGTGAATAAGCTCACTGGCTGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAAGAGGCATCAATGCTCTTA	28610070	28610185	+	0	0
1012	GCAGATGATGGGCTCCGGGAAGACNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNAAGAGGATGGCTAGCGGAGG	90631835	90631946	+	0	0
1013	GAGATAATAGTGGTCCCACTGCANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNTGTGATGGGCTTGGTCCAG	90631835	90631946	+	0	0
1014	GACAAGCAGGAGAGAGATGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNAGGGGAGGAGAGATGGGGGT	7572925	7573012	+	0	0
1015	GCTGGGAAGGAGCAGGGGANNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNCAACAAATGTAACCTGAACCA	7573973	7574036	-	0	0
1016	GTCTAACACTCAAAATGCCNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTCACTTTTATCACCTTCTCTTG	7576850	7576934	-	0	0
1017	ACCAAGGCTCACTACTCCNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNTTCTCTCTCTGCGCGGCTC	7577014	7577158	+	0	0
1018	CCAAGGGTGCAGTTATGCCTCAGNNNNCTTCAGCTTCCCGATATCCGACGGTAGTGTNNNNGCTTTGAGGTGCGGTTTGTG	7577014	7577158	-	0	0

SEQ ID	mip_sequence	feature_start_position	feature_stop_position	strand	failure_flags
1019	GTAAGGATGGTGTACAGTACAGANNNNCTTCAGCTTCCGATATCCAGCGGTAGTGTNNNNNAGAGGCAAGCAGAGGCTGGG	7577495	7577613	+	0
1020	GCGGCATGAACCGGAGGCCCATNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAAAAAAGGCTCC	7577495	7577613	-	0
1021	CTGGGACCTCTGGCAACANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNAACCACTCAGGCGGCTCATAT	7578168	7578295	+	0
1022	GTAGCAGTAGGGGGCTTTCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAGGTGGAAGAAATTTGCGTGTG	7578115	7578211	-	0
1023	GTTAGGGCAGGGAGTACTGTAGGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATGTGGGGCAGCGCTT	7578364	7578560	+	0
1024	GGGCTGGAGACGACAGGGCTGGTTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCATGSCCATCTACAAGCA	7578364	7578560	-	0
1025	TTCCACCCCGCCGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTGCCGCTTCACAGTTGCTTTATCTG	7578364	7578560	-	0
1026	TTTCTGGGAAGGACAGAAGATGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCAACAAAAAATTCAGGG	7579308	7579600	+	0
1027	GTAGAGCTGCTGGTGCAGGGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATGAGGCAACTACCGTGC	7579308	7579600	+	0
1028	GCATCAAAATCATCAATGCTTGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNAGGAGGGGCTGGTGACGG	7579443	7579523	+	0
1029	ACCCAGTCCAGATGAAGCTCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTGAGGACCTGGAGGCTGGG	7579308	7579600	-	0
1030	AAGGACGCCCAACCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTCATGCTGGATCCCACTTTCTCT	7579812	7579915	-	0
1031	GCCTTCAATTTGGCTTGTGCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCCTTCTCAGAGAAATTTTATG	58740352	58740920	+	0
1032	AAAAAATTTATCCAGAACTCAACNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCTCGAGCTATCTCAGCTG	58740352	58740920	-	0
1033	AGCCTGCAAGTCTCCCAACCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTGAAGATGTCAACTCTCGGCC	58740352	58740920	+	0
1034	CAATTTTCTCAAGTGGTCTGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCAATTTCTTGGCTTTTCAAT	58740352	58740920	-	0
1035	GATGTGAACTTTTTAAGGGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATGCGACGACAGCTAGGGG	58740352	58740920	+	0
1036	GTCTATGCTTCTTCAGGGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTTTTCTGTGTTGATGAAGT	58740352	58740920	-	0
1037	AGCTCGGGCGCTCCAGCANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTCCCGGGCGCTGGTGTGAG	74732956	74732961	+	0
1038	TTTTCTCTCTGGACAAACCCGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCCCTAAAGAAATCACGCT	42531868	42531969	+	0
1039	GGCTGCTCCAGGGCTGACTGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAAGAAAGAAAGGCAAA	13054525	13054730	+	0
1040	CTTGCCCTCTCCAGCCCTGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATCTTTGTCTCATCATCTCTCT	13054525	13054730	-	0
1041	GAACTCGCGTGAGGCGCGGCTGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAAGGTGCTGGAGCTG	33792300	33792469	-	0
1042	TGACCGCTCGGCAAGGGGTGGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAAGGCCAAGAGTCGGTGA	33792244	33793326	-	0
1043	GCGGGCTCTGCTTGATCACACGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCGGGTGC	33792674	33792865	+	0
1044	TCATGCCCGGGGAGCGACGCGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCCAAAGCGCCGCTG	33792970	33793010	-	0
1045	GCTGATGTCGATGGACGCTCGTGCTCGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTTGGCTTCTCTGCT	33793025	33793090	+	0
1046	GCAAGTGGCTGCTCATCGGGGCGCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTGGGGCGGAGGCT	33793243	33793300	+	0
1047	GCCACCGACAGGAGATGGGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGGCTGAAACTGATGGCTGTG	31021087	31021727	+	0
1048	TCCCCATCTGCCAGGCACATCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCAGATCTCCGAACACAGCCAG	31021087	31021727	+	0
1049	CCTGGGACACAAAGCCACTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTTGCATCTCTAGCAACCCCTGCT	31021087	31021727	-	0
1050	GAGCAGGCGGCTCTGCATCTTTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCCGAATTCACGATGAGTCT	31021087	31021727	+	0
1051	GCCTCTGGGTGCTCTTAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCAAGGCTAGGAAATCTGTCTGGAG	31021087	31021727	-	0
1052	CACAGCCACTAAAGAGACCCANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTCAGGAAACTGTGGATCAGG	31021087	31021727	+	0
1053	CGGGTTGGAAGTGGCGCAGGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNAGTGCAGATCACCCACTCAGT	31022233	31025152	+	0
1054	GTGTGATGTGTGGTAGGCTGTGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAGGGCGAGAGTCA	31022435	31022452	+	0
1055	GGTGGGGATGATCCGGGGCATATNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCCGATGGCAGTGGTGGCGG	31022322	31022412	-	0
1056	TGCCAGGCTTGCCTTACTGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGGAACTGCCATGTCCAGAGCT	31022233	31025152	+	0
1057	GTACACTTTCCAGGGTGTCTCGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTGTAGCCCTCTGTAGTAGG	31022233	31025152	-	0
1058	GGATCTGTAAATGTACCCCNNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTAGTCTGGCACCACTTCTG	31022233	31025152	+	0
1059	GCAAGGCTGGCATGGCTGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNACCATTTGCTGCGAAGAACGGTGG	31022233	31025152	-	0

seq_id	feature_start	feature_end	strand	probe	failure
1060	31022233	31025152	+		0
1061	31022233	31025152	-		0
1062	31022233	31025152	+		0
1063	31022233	31025152	-		0
1064	31022233	31025152	-		0
1065	31022233	31025152	-		0
1066	31022233	31025152	+		0
1067	31022233	31025152	-		0
1068	31022233	31025152	-		0
1069	31022233	31025152	+		10
1070	31022233	31025152	+		0
1071	31022233	31025152	-		0
1072	31022233	31025152	+		0
1073	31022233	31025152	+		0
1074	31022233	31025152	+		0
1075	31022233	31025152	+		0
1076	31022233	31025152	+		0
1077	31022233	31025152	+		0
1078	31022233	31025152	+		0
1079	31022233	31025152	+		0
1080	57484402	57484481	-		0
1081	36164474	36164925	+		0
1082	36164474	36164925	+		0
1083	36164474	36164925	+		0
1084	36164474	36164925	+		0
1085	36164474	36164925	+		0
1086	36171591	36171765	-		0
1087	36171591	36171765	-		0
1088	36206704	36206900	+		0
1089	36206704	36206900	+		0
1090	36206704	36206900	-		0
1091	36231767	36231880	-		0
1092	36252844	36253026	+		0
1093	36252844	36253026	-		0
1094	36259157	36259187	+		0
1095	36259192	36259400	+		0
1096	44514777	44514876	-		0
1097	44524444	44524480	+		0
1098	15833798	15834016	+		0
1099	15833798	15834016	-		0
1100	15836703	15836772	-		0

mip_sequence

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CATATTGCCAGCCATGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTGGGTTGTGCTGCAATGAACGTG
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GCAGAGGAAGTTGGGGCTGCGGCGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTGCTCTCCACCCAC
GGCCAGACCTCCACCATCTGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTTGTGTCGACGCCATGCTG
GCCAGTACGTGACTGAGCAANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTTCCGCTGAGGAAGATGCGG
CATGGAATATGTCAGCAGCATGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCAAAACAACTGGCTAAACG
GCATGCGGTGGAGGAGGGAGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATGGAGCAGTGCAGGAGGG
GGCCAATAGTTGAAAATTAACNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTTGGTCAAGGGTCTATAGT
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SEQ ID	mip_sequence	feature_start_position	feature_end_position	strand	probe_strand	failure_flags
1101	TTTCTCAATTGTTCCACTGCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCATATCATTTGATTTTTGGTTT	15838328	15838442	+	+	0
1102	GCTTCAGGCTTTGGAGCAAGAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTACATGGTGGTCCAGCTT	39911360	39911656	+	+	0
1103	GTAATAATGAAAGTGGCCCAACNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTTCTGCCTCTGCAATGGTGAC	39911360	39911656	+	+	0
1104	GTAAGGAGCTGTAGATCTGTGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNGAAATGAAATGTCTCC	39911360	39911656	-	-	0
1105	CGTTCTCAACAGCATGTGCAGAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNAGACCATTCTTGAACCTTG	39914617	39914770	+	+	0
1106	CTTATGGTGTGACCCCAACNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTGCCAGCTTTCGCTGTGCTTTT	39914617	39914770	-	-	0
1107	TCGCCATCCAATGCTTGNNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCTGCTGTTTGGTGCCATCTG	39931589	39934460	+	+	0
1108	CATGTGGTGCAGCTTGGAAAGCATNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAGGAGCAAGCTGTATGAT	39931589	39934460	+	+	0
1109	CAGCAGCGAGTTCATATGCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTCAGGAAATGGTTGTGCT	39931589	39934460	-	-	0
1110	CAGGGTTTTCTTCCCTCTGGGCTGAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTTCTGTCTGAGGA	48649515	48649604	+	+	0
1111	GCTGGAAGCTCTCAATGGATGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTGCCCTACTACAGGGACGCTG	48649516	48649738	+	+	0
1112	GGTGTGAGGACACAGACAGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTGAGATTACCTGGGGAGTGT	48649516	48649738	-	-	0
1113	GCACTCTGCCTTTTCGTTTTGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNTGTGGATGGCCTTTGGAG	53431940	53432902	+	+	0
1114	GCACTGCTGTGGCTTACTTTCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATCTGCAGTCCATGGGCTGAG	53431940	53432902	+	+	0
1115	GCCTTCAGGTCACCTGGCCCAANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCTAGGTGTAAGGTGGTGCTG	53431940	53432902	+	+	0
1116	CCAGTACTGAGCTGTCCAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTTCTCATCCACGCCCTGCCTT	53431940	53432902	+	+	0
1117	GACAAGGATTCGCAAGCATANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGGGGAGAGAGAGAGGGG	53431940	53432902	+	+	0
1118	:CTCACTCCAGGTAGTCAAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNATCAATCACTAGCTTGGCCCTT	53431940	53432902	+	+	0
1119	GCCGATTTGCTTTGGAGGCANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCCAGGTGAAGCCTACAGATGAGA	53431940	53432902	-	-	0
1120	GCTTTTGAAGCTGGCTCAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCCTGTCTCTCCGAGTCCACAA	53431940	53432902	+	+	0
1121	GGGTGACGGCAGTGTTCAGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNACCAAGTTTTGGGCAAGAACA	53431940	53432902	-	-	0
1122	GGCTATTGTGACCAACTTGTGTCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNTGGCCTAAATCTTAGCAT	123181202	123181359	+	+	0
1123	GGTGAAACTAATCTAACAGACANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCGCTCTCATTTGGCTCGTTTT	123181202	123181359	-	-	0
1124	GCAAGTTTGCATATTTCTGGTGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAAATGATGTGTTTTTAC	123182853	123182930	+	+	0
1125	AAGATGTGCCCTTCAGACTGTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNTTATGATCGTTTTTCCCTCC	123184036	123184161	+	+	0
1126	CCAGTCGGTTCAAGGTAGTATTANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAAGTTGAAATACATAGAG	123184969	123185250	+	+	0
1127	GTGCTATGACCCCTTGACAAAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAAGAGCTTAATCCAAACTGGA	123184969	123185250	+	+	0
1128	GCATATTTGCAATAATGTTACATNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNCTTTCTCTGCTTTCCTTT	123184969	123185250	+	+	0
1129	GAAATAGCAGTAACAGGTGCTNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGCCTTAGAAAATGAGTAACAG	123189975	123190088	-	-	0
1130	ACCATGTCAGTATTAGTGAATCANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTATCAAAGCTAACAGTTTC	123220395	123220624	+	+	0
1131	CCACCAGCTAGCAAGAAATTCGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNAAAGAGATAAATATATCC	123220395	123220624	-	-	0
1132	CCGCGCAGCAAGAGACTTGAANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNITGCAAGCTCAGTTGAACAG	133511647	133511790	+	+	0
1133	GCCATTTAAGTCTAGAAAATGCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCGCTGCCACCTTCGTGTTTT	133511647	133511790	-	-	0
1134	GCCTTTTCATCATATAAAGGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNCTACCACTGTGCATTGCATG	133527529	133527670	+	+	0
1135	GTATGTGACTTCTAAGGCTGTATNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTTTCTCTCGTATTTGAGC	133527529	133527670	-	-	0
1136	CCAGTTTCAGCCACTTTTCAGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNITAGTTGTTACTAATTTTG	133527932	133527989	+	+	0
1137	GGGGAGGAGAAAATGAAGACGANNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGAAATGTTAAGTAACTTGA	133547847	133548010	+	+	0
1138	GCCTGGTGTCACTAGGGCTGCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNAACAAACAAACAAAAACAAA	133547847	133548010	-	-	0
1139	ATGCAGGAAAAATTAACATTAGAAATCCNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTTGTCATAACAAATATAAAAACTACA	133549044	133549153	-	-	0
1140	GACTGCAAGGTGTACATTTCTGNNNNCTTCAGCTTCCGATATCCGACGGTAGTGTNNNNGTTTAGGGAAATAGACAACTGT	133551219	133551319	-	-	0

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CLAIMS:

1. A method of predicting the risk of a disease condition of a solid organ in a patient, the method comprising:
 - a) receiving a sample from the patient containing hematopoietic stem cells;
 - 5 b) sequencing the sample to detect a degree of clonal hematopoiesis;
 - c) comparing the degree of clonal hematopoiesis in the patient to a control degree; and
 - 10 d) determining the patient is at an elevated risk of the disease condition if the degree of clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner.
2. The method of claim 1, wherein the degree of clonal hematopoiesis is measured using a variant allele frequency of mutations determined to be associated with clonal hematopoiesis.
3. The method of claim 2, wherein the variant allele frequency (VAF) is $\geq 2\%$.
- 15 4. The method of claim 3 wherein the VAF is $\geq 5\%$.
5. The method of any one of claims 1-4, wherein the following genes are sequenced in the sample: TET2, DNMT3A, and ASXL1, and optionally one or more of, but preferably all of, BCOR, BRAF, CALR, CBL, CEBPA, EZH2, FLT3A, GATA1, GATA2, GNAS, IDH1, IDH2, JAK2, KIT, KRAS, MPL, NRAS,
20 PHF6, PPM1D, PTPN11, RAD21, RUNX1, SETBP1, SF3B1, SMC1A, SMC3, SRSF2, STAG2, TP53, U2AF1, WT1, and ZRSR2.
6. The method of any one of claims 1-5, wherein the sequencing is performed using single-molecule molecular inversion probes (smMIPs).
7. The method of any one of claims 2-4, wherein the mutations associated with
25 clonal hematopoiesis are detectable by the probes listed in Table A.

8. The method of any one of claims 2-4, wherein the mutations associated with clonal hematopoiesis are detected using a library comprising at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.
- 5 9. The method of any one of claims 2-4, wherein the mutations associated with clonal hematopoiesis are detected using a library consisting of at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.
- 10 10. The method of any one of claims 2-4, wherein the mutations associated with clonal hematopoiesis are detected using a library consisting of substantially all of the probes listed in Table A.
- 11 11. The method of any one of claims 2-4, wherein the mutations associated with clonal hematopoiesis are detected using a library consisting of the probes listed in Table A.
12. The method of any one of claims 1-11, wherein the solid organ is a heart.
13. The method of claim 12, wherein the disease condition is cardiogenic shock.
- 15 14. The method of claim 13, wherein an elevated risk of cardiogenic shock is associated with an elevated risk of death.
15. The method of claim 13 or 14, further comprising treating or preventatively treating the patient for cardiogenic shock.
- 20 16. The method of claim 12, wherein the disease condition is an adverse outcome after orthotopic heart transplant (OHT).
17. The method of claim 16, wherein the adverse outcome is an elevated risk of mortality and/or elevated risk of cardiac allograft vasculopathy.
18. The method of claim 17, further comprising treating or preventatively treating the patient for cardiac allograft vasculopathy.
- 25 19. The method of claim 12, wherein the disease condition is hypertrophic cardiomyopathy.

20. The method of claim 19, further comprising treating or preventatively treating the patient for hypertrophic cardiomyopathy.
21. The method of any one of claims 1-11, wherein the solid organ is a lung.
22. The method of claim 21, wherein the disease condition is an adverse outcome,
5 including death, after lung transplant.
23. The method of claim 22, further comprising treating or preventatively treating the patient for the adverse outcome after lung transplant.
24. A kit comprising a library of probes library comprising at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.
- 10 25. The kit of claim 24, comprising a library consisting of at least 50%, 60%, 70%, 80%, 90%, 95%, 98% or 99% of the probes listed in Table A.
26. The kit of claim 24, comprising a library consisting of substantially all of the probes listed in Table A.
27. The method of any one of claims 2-4, comprising a library consisting of the
15 probes listed in Table A.
28. A method of predicting the benefit of immunotherapy in a patient with melanoma, the method comprising:
- a) receiving a sample from the patient containing hematopoietic stem cells;
- b) sequencing the sample to detect a degree of clonal hematopoiesis in
20 TET2;
- c) comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and
- d) determining the patient would benefit from immunotherapy if the degree of
25 TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner.
29. The method of claim 28, further comprising treating the patient with immunotherapy.

30. A method of predicting metastatic risk in a patient with a non-hematological cancer, the method comprising:
- a) receiving a sample from the patient containing hematopoietic stem cells;
 - b) sequencing the sample to detect a degree of clonal hematopoiesis in TET2;
 - 5 c) comparing the degree of TET2 clonal hematopoiesis in the patient to a control degree; and
 - d) determining the patient is at a lower risk of metastasis if the degree of TET2 clonal hematopoiesis in the patient is higher than the control degree in a statistically significant manner.
- 10 31. The method of claim 30, further comprising treating the patient with a treatment and monitoring regimen reflective of a low risk of metastasis.

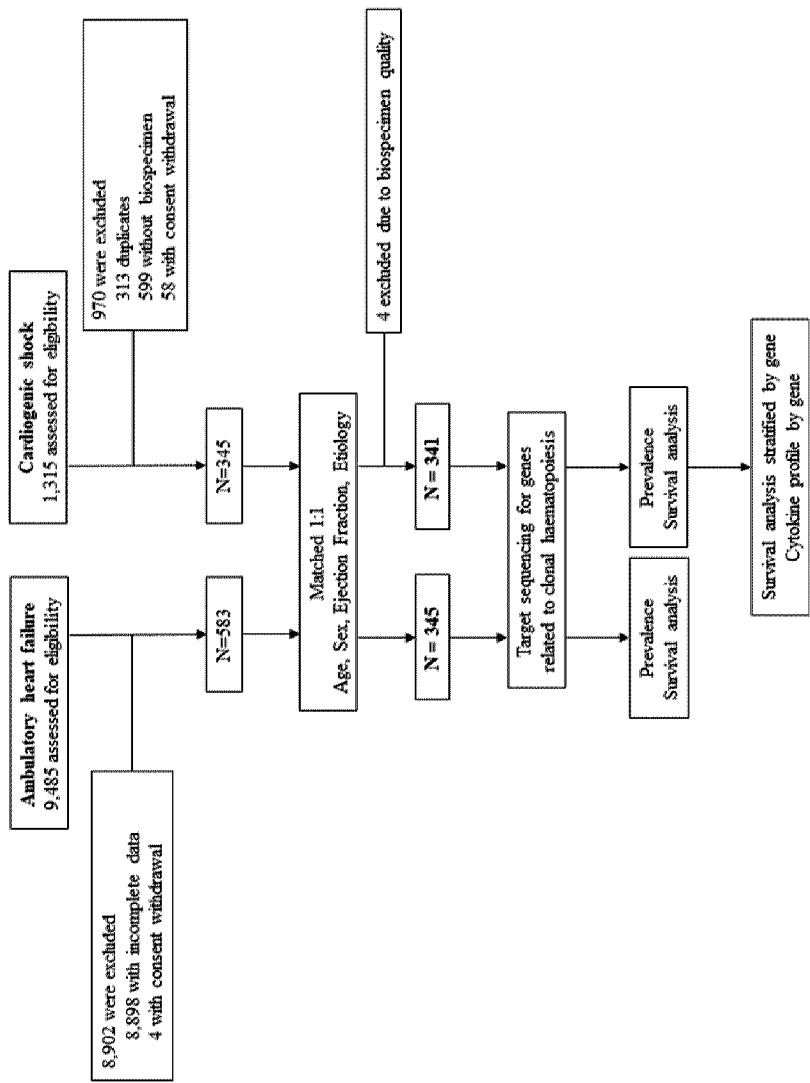


Figure 1

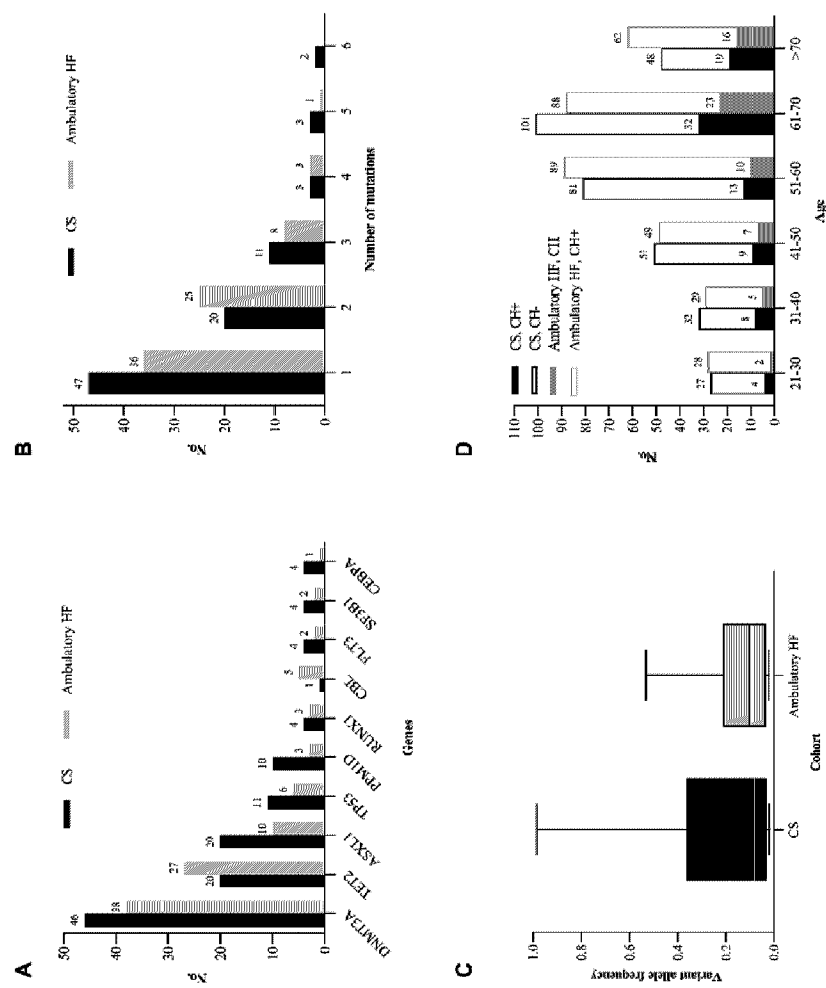


Figure 2

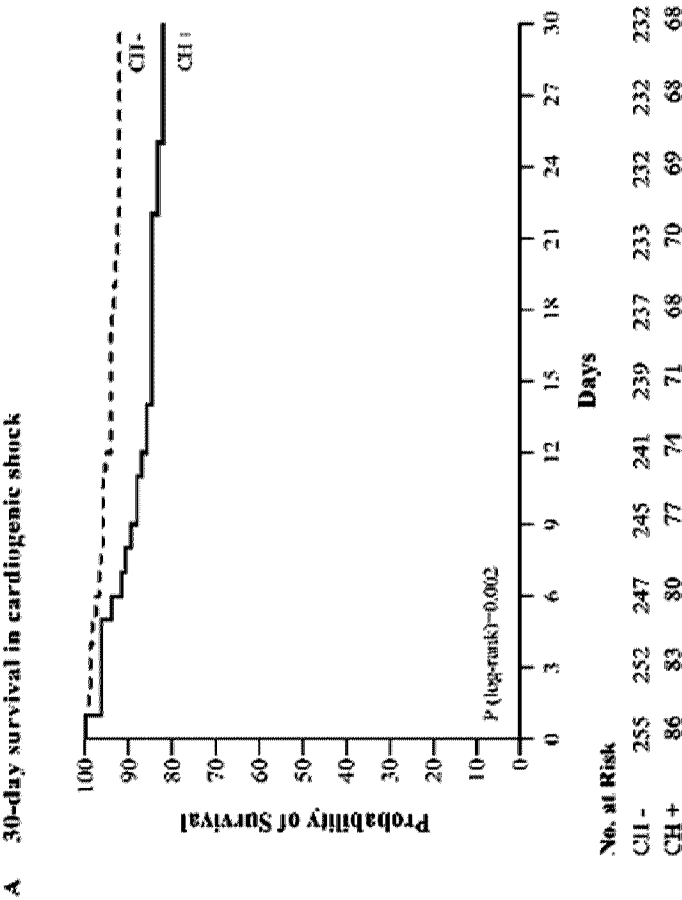


Figure 3

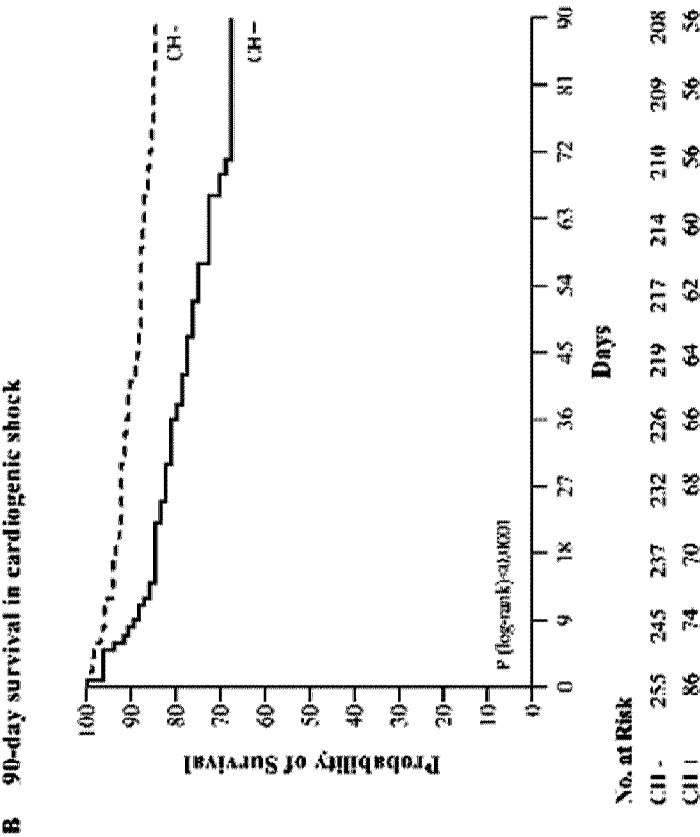


Figure 3

C 3-year survival in cardiogenic shock

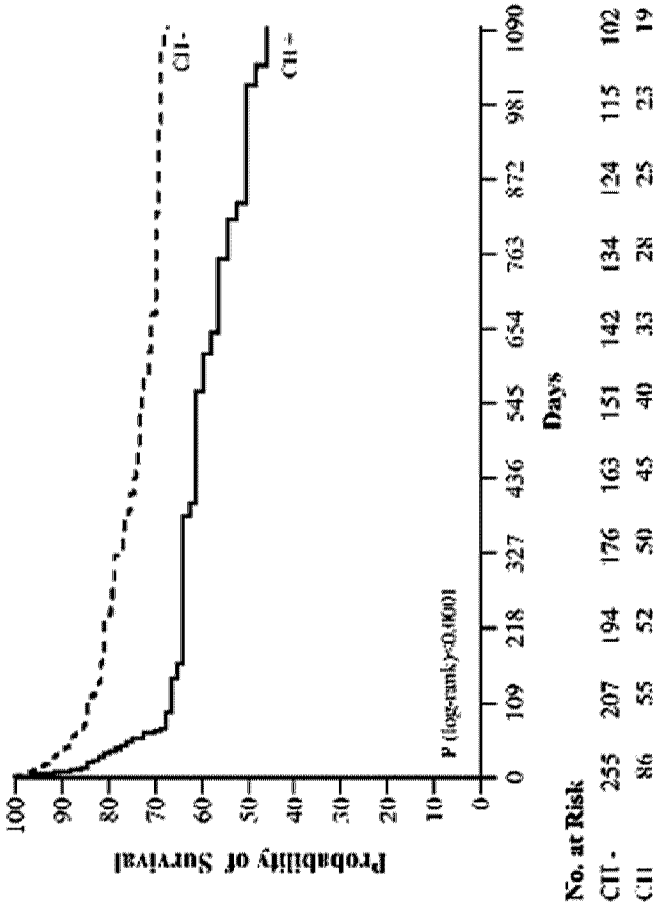


Figure 3

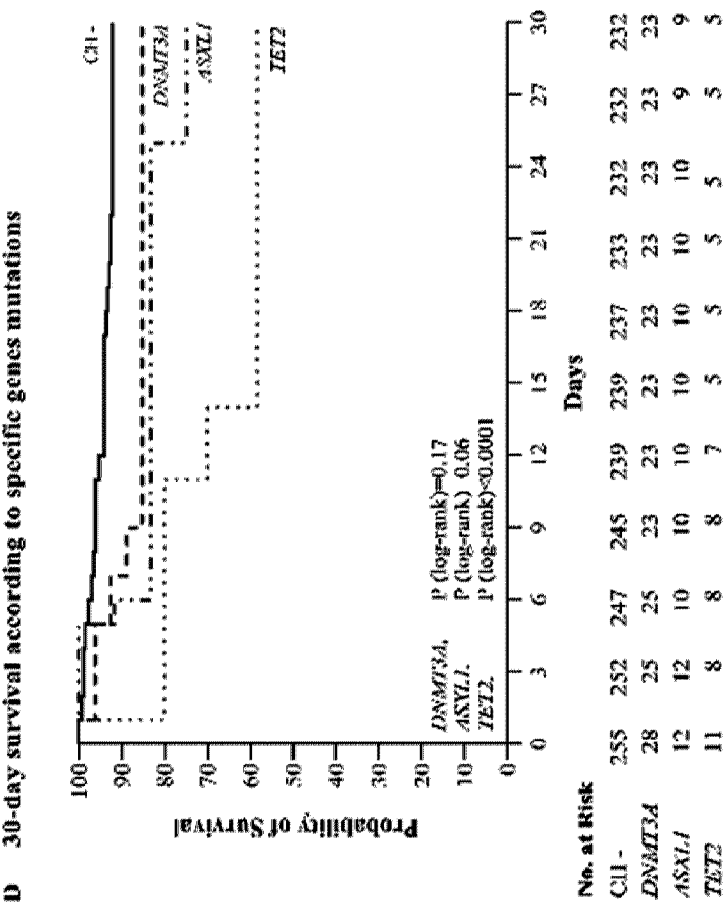


Figure 3

E 90-day survival according to specific genes mutations

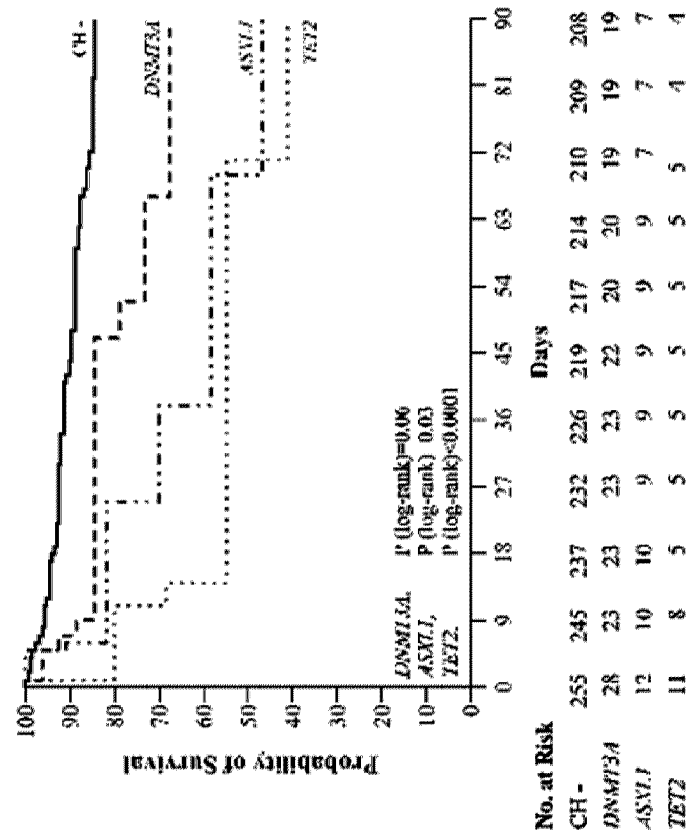


Figure 3

F 3-year survival according to specific genes mutations

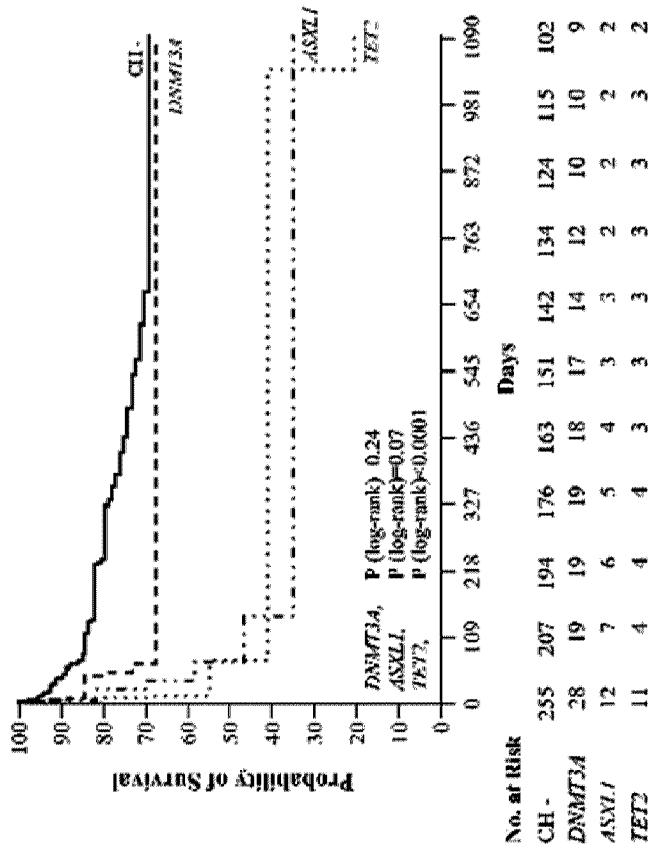


Figure 3

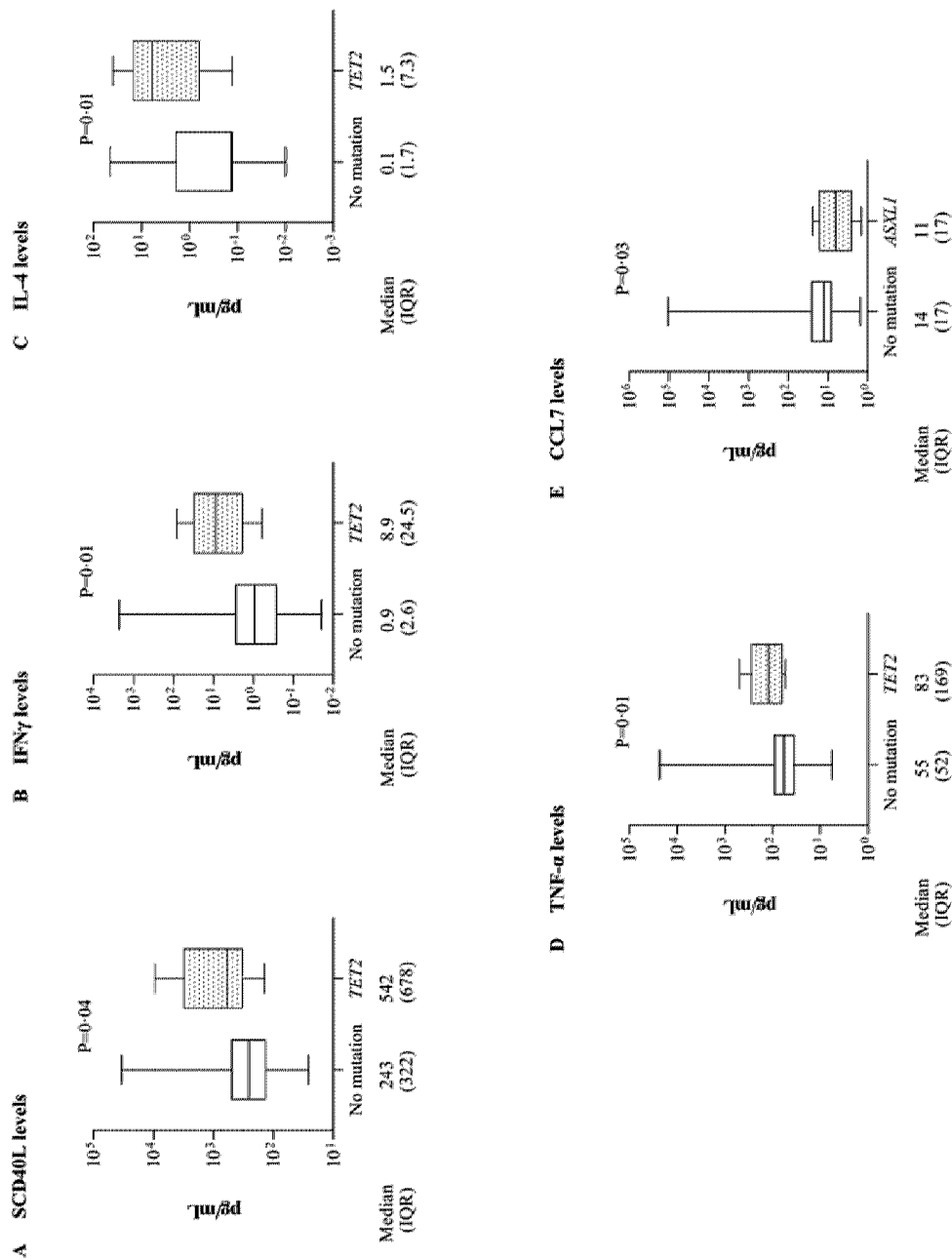


Figure 4

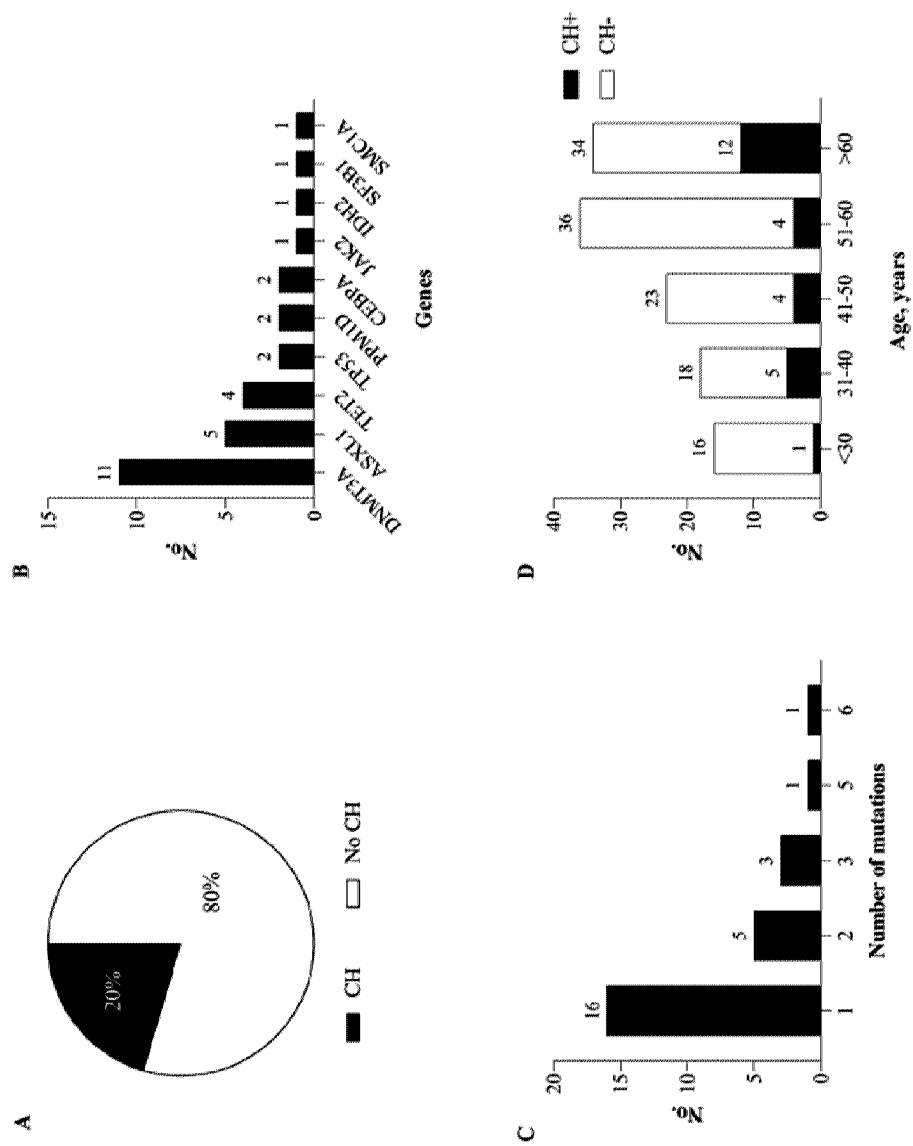


Figure 5

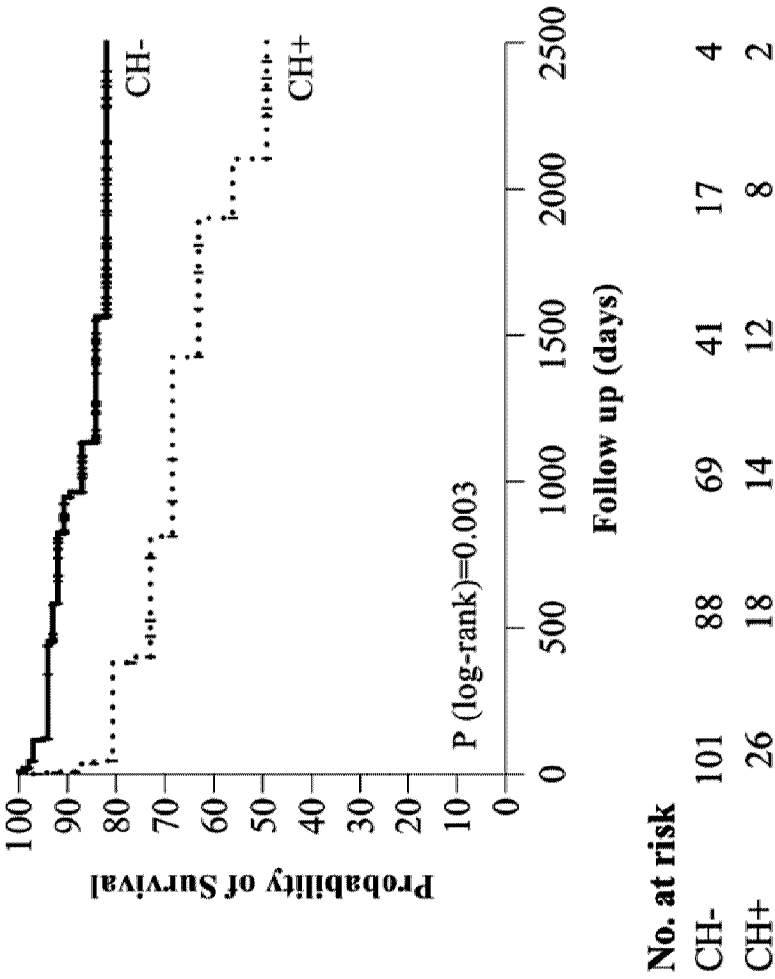


Figure 6

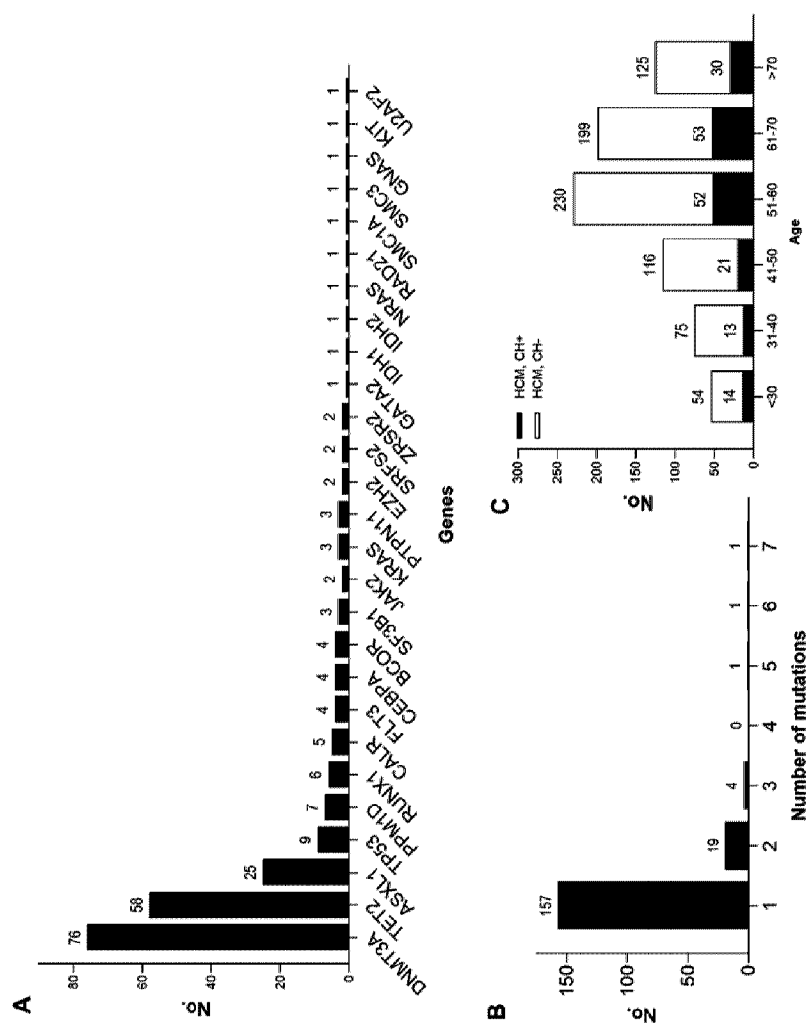


Figure 7

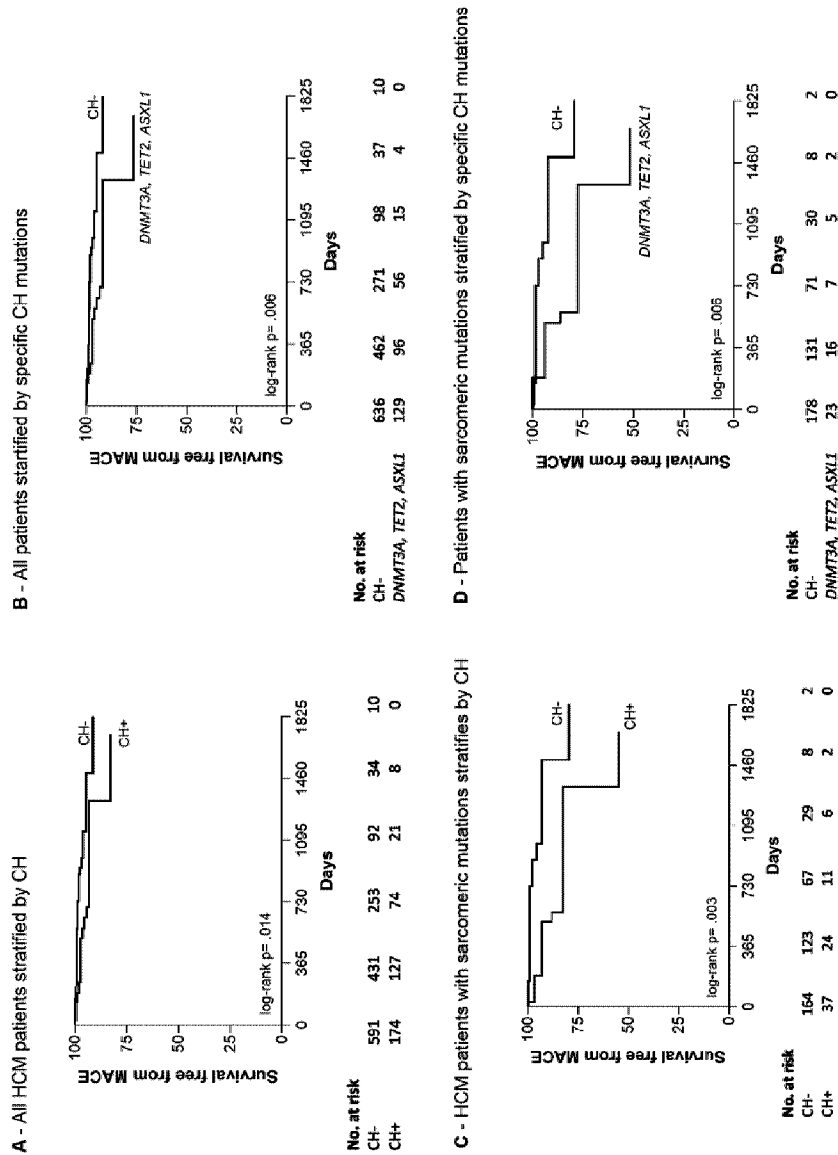


Figure 8

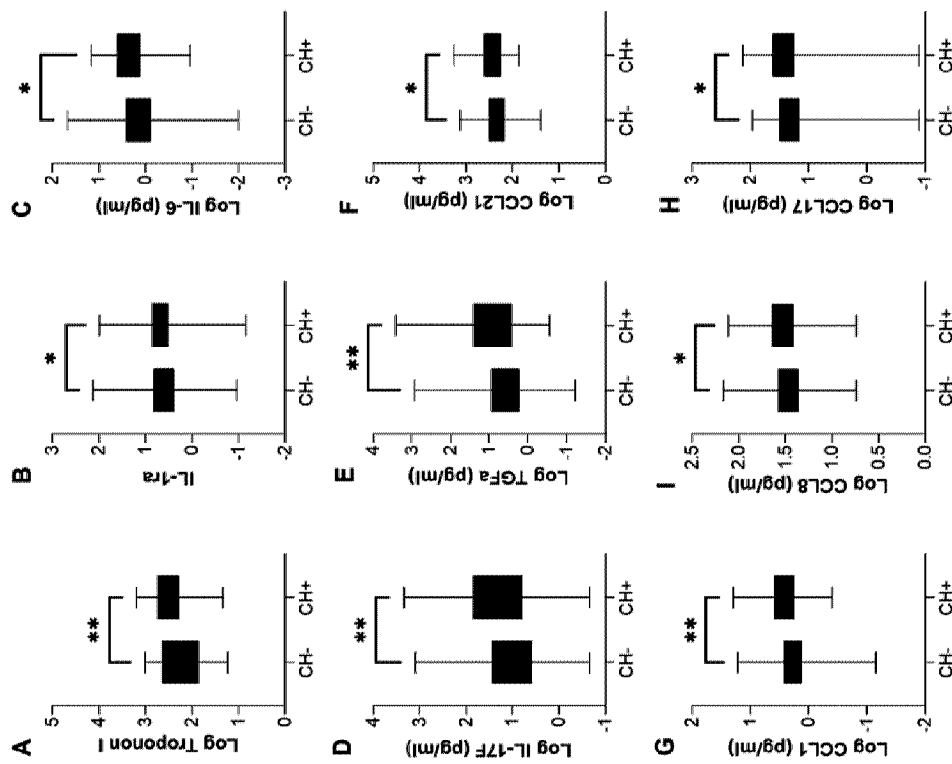


Figure 9

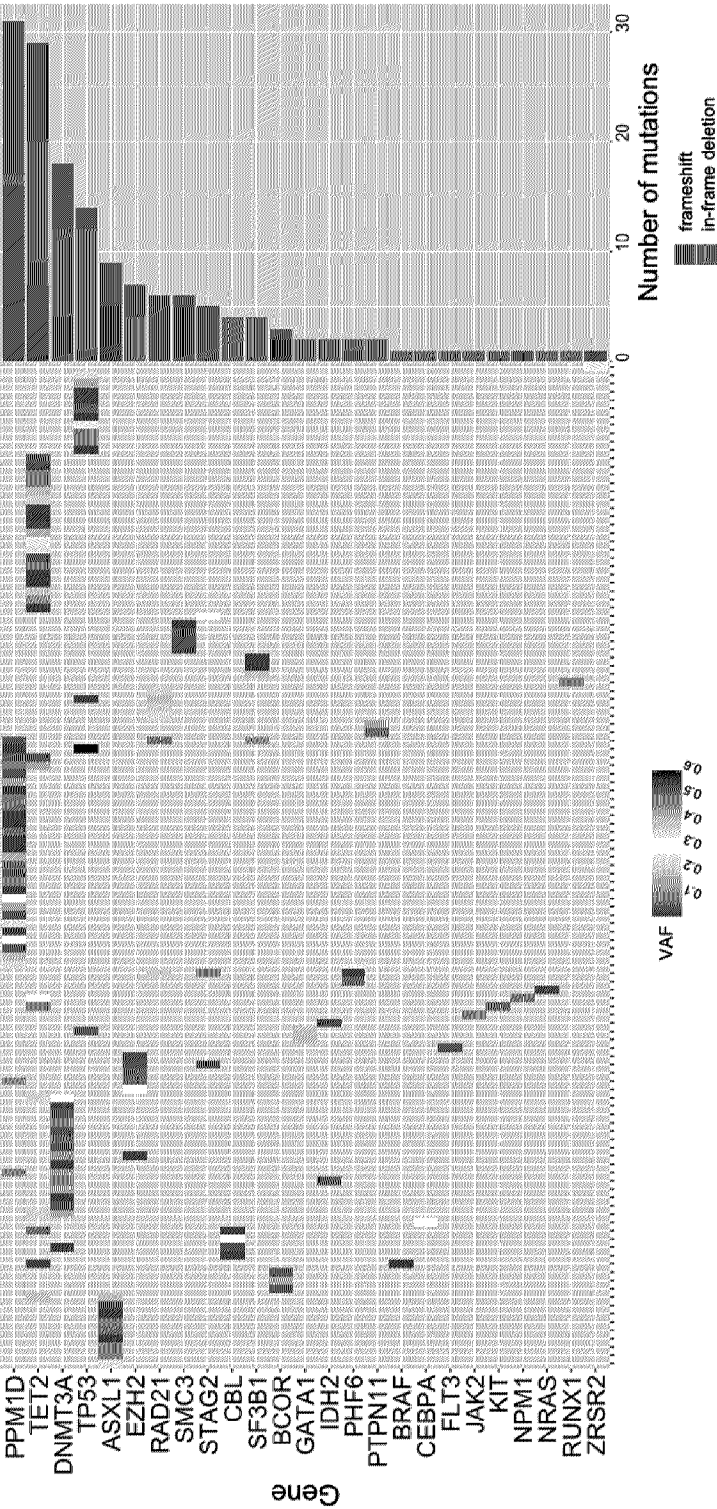


Figure 10A

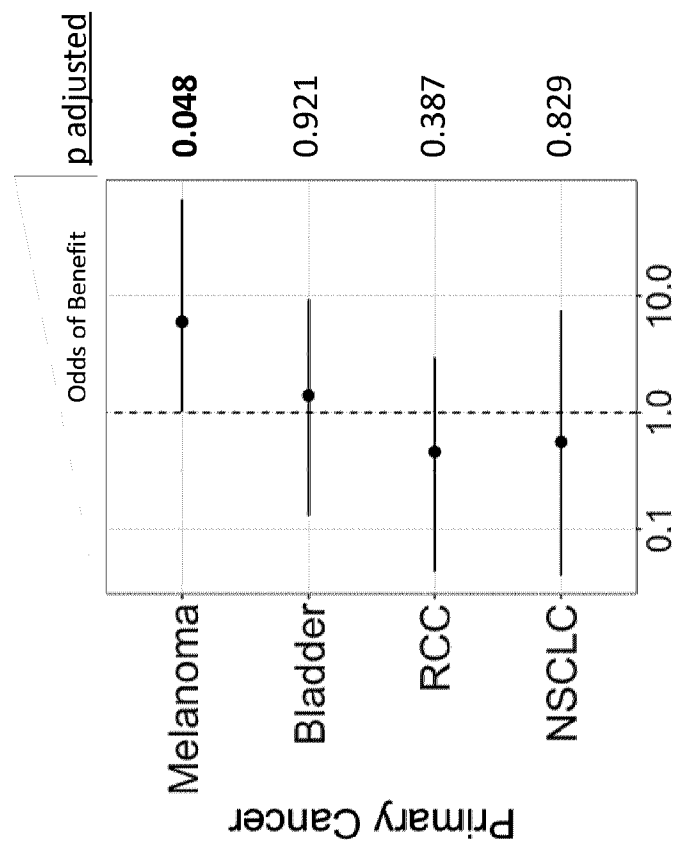


Figure 10B

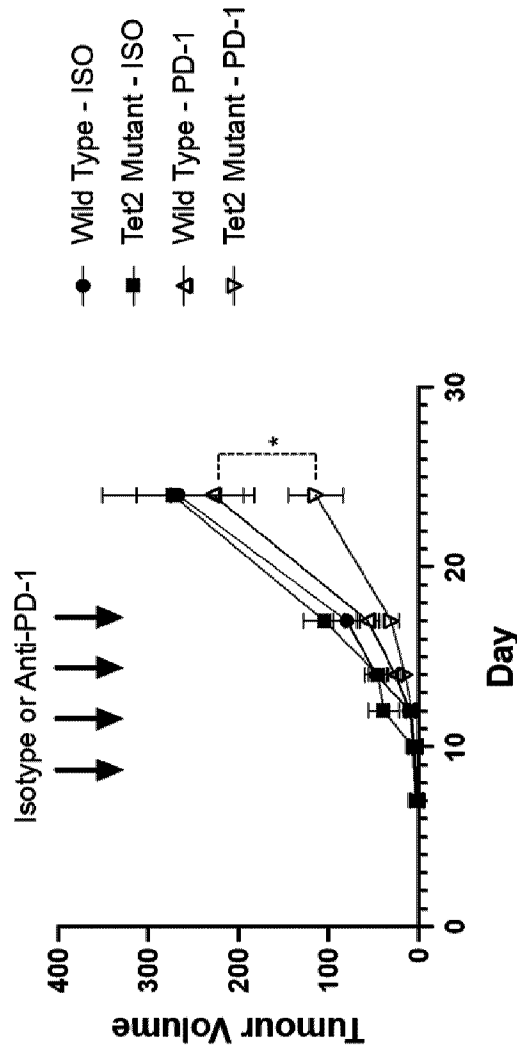


Figure 10C

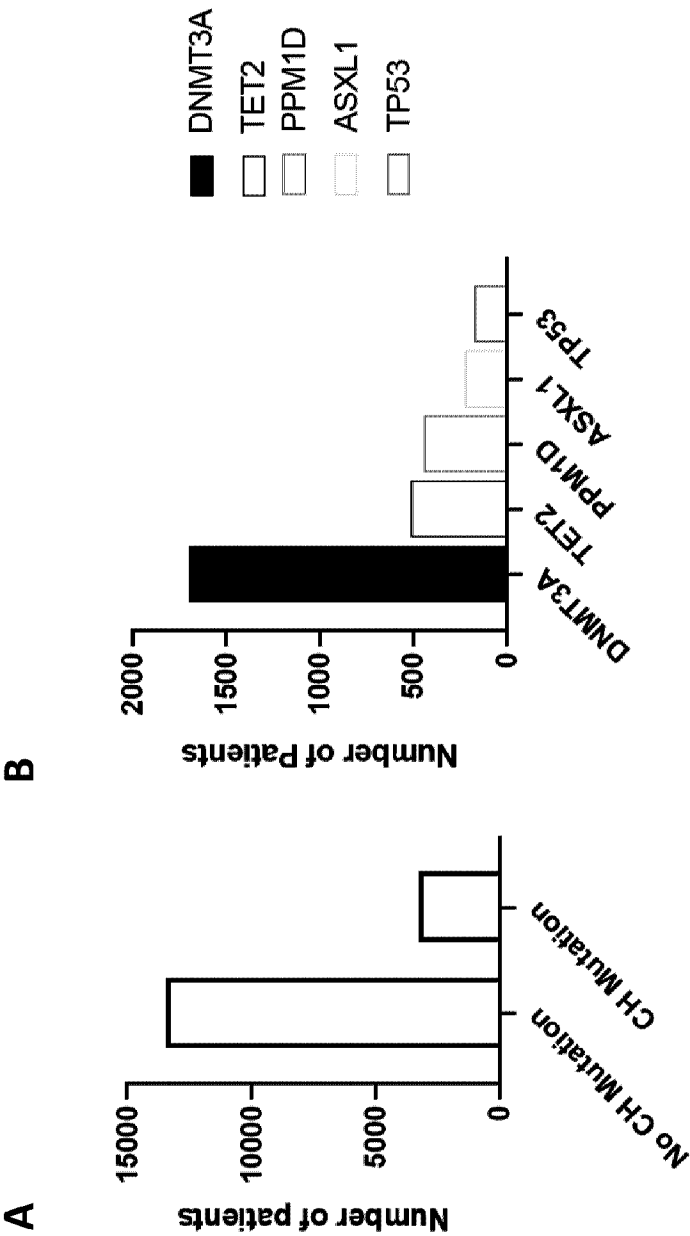


Figure 11

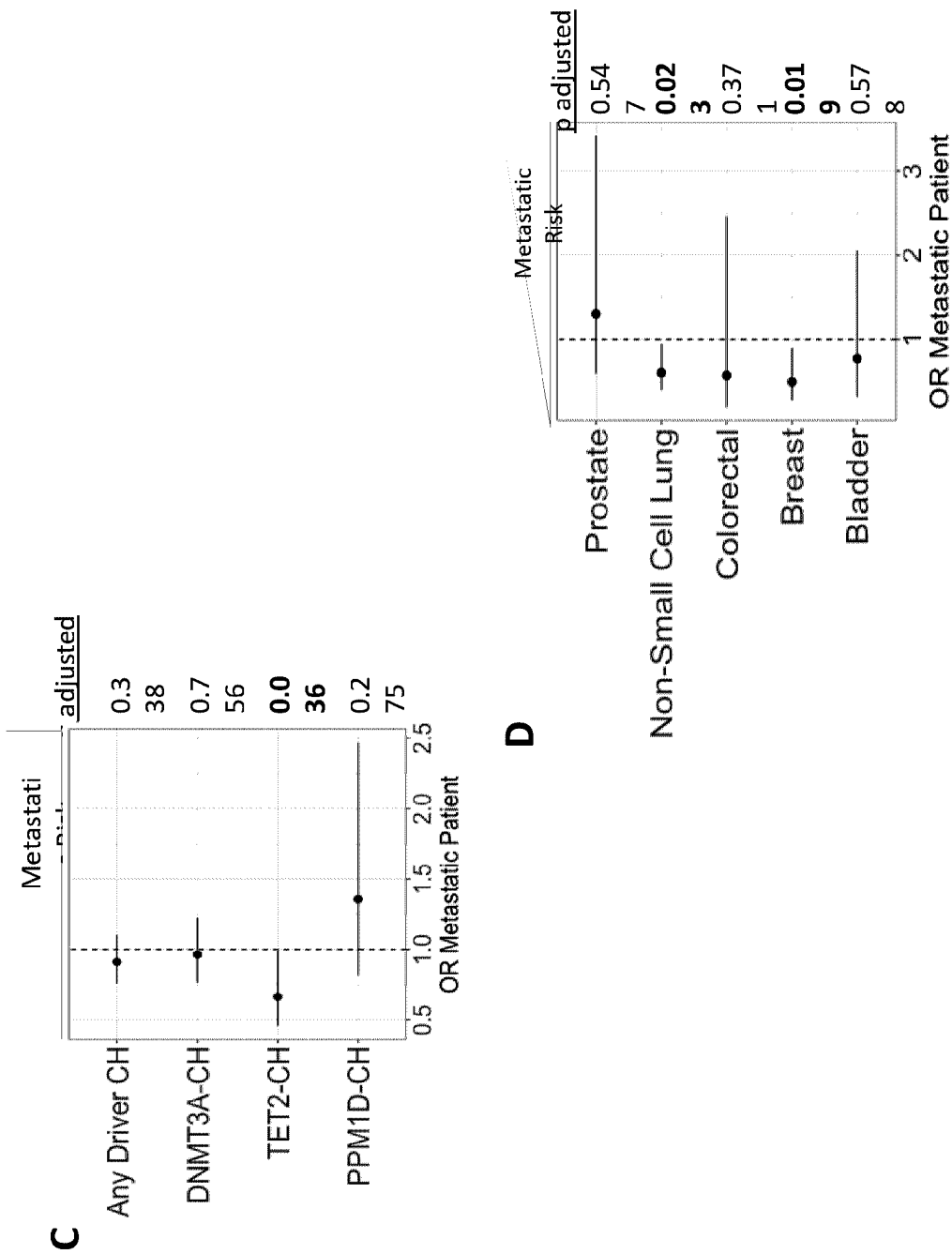


Figure 11

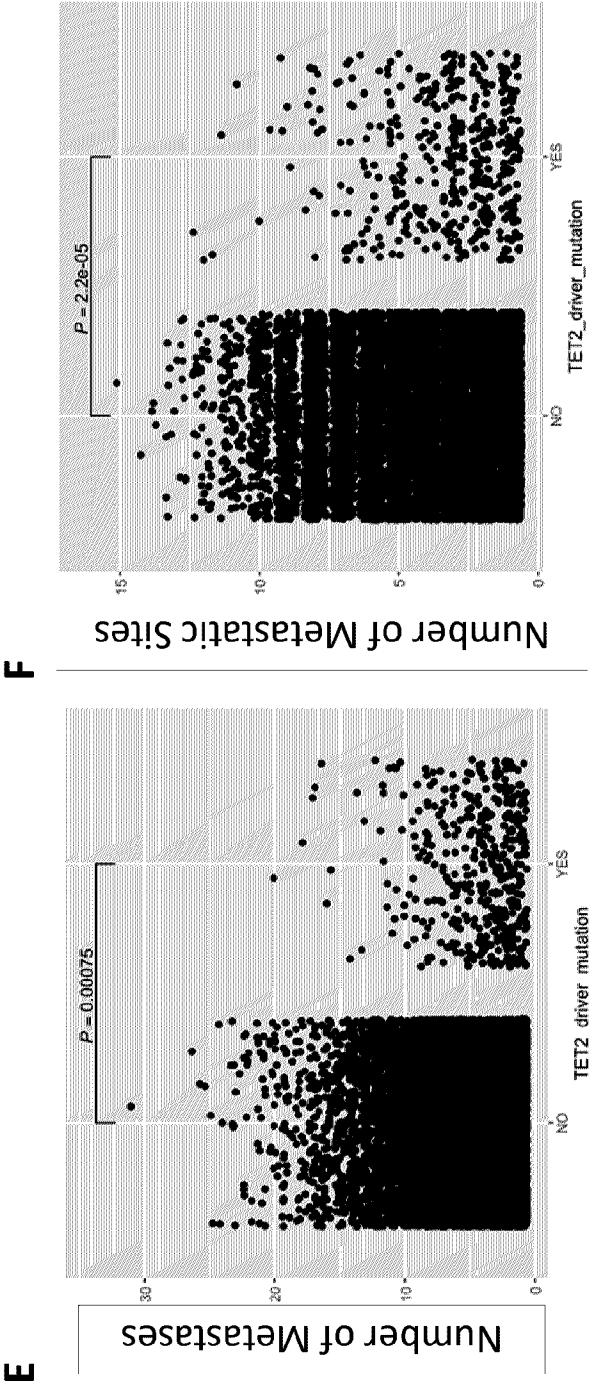


Figure 11

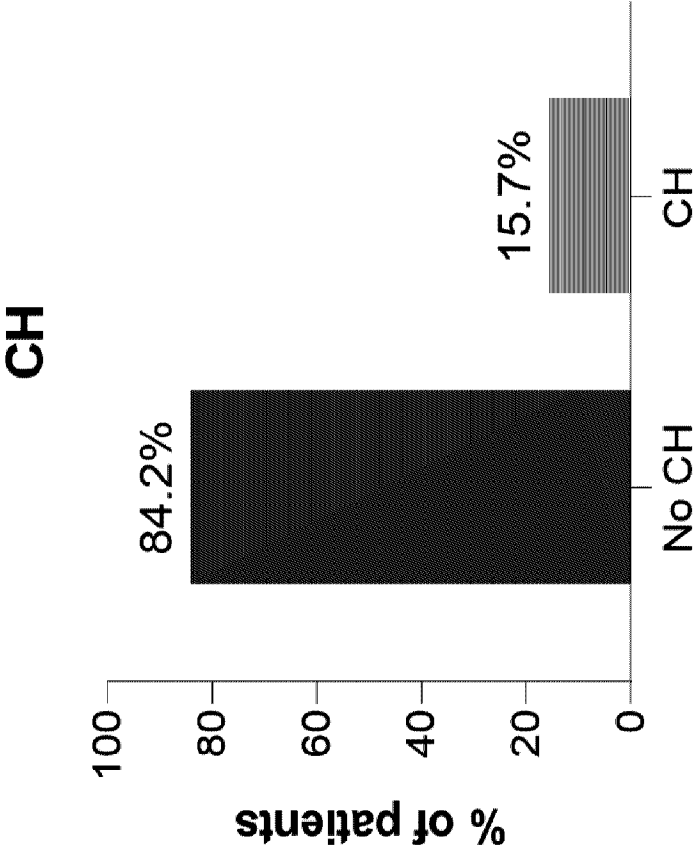


Figure 12

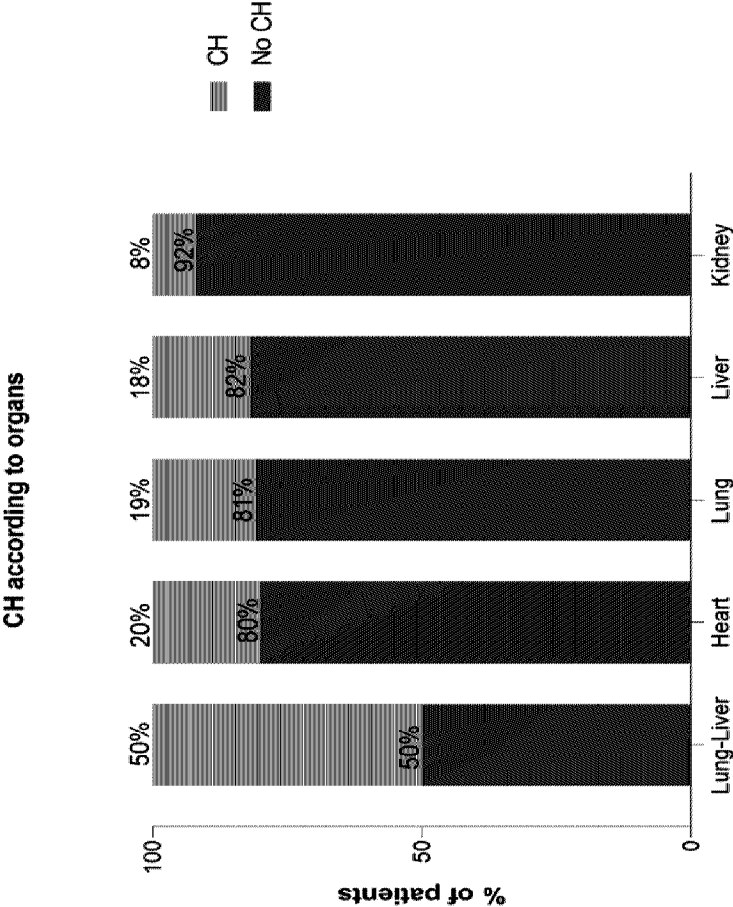


Figure 13

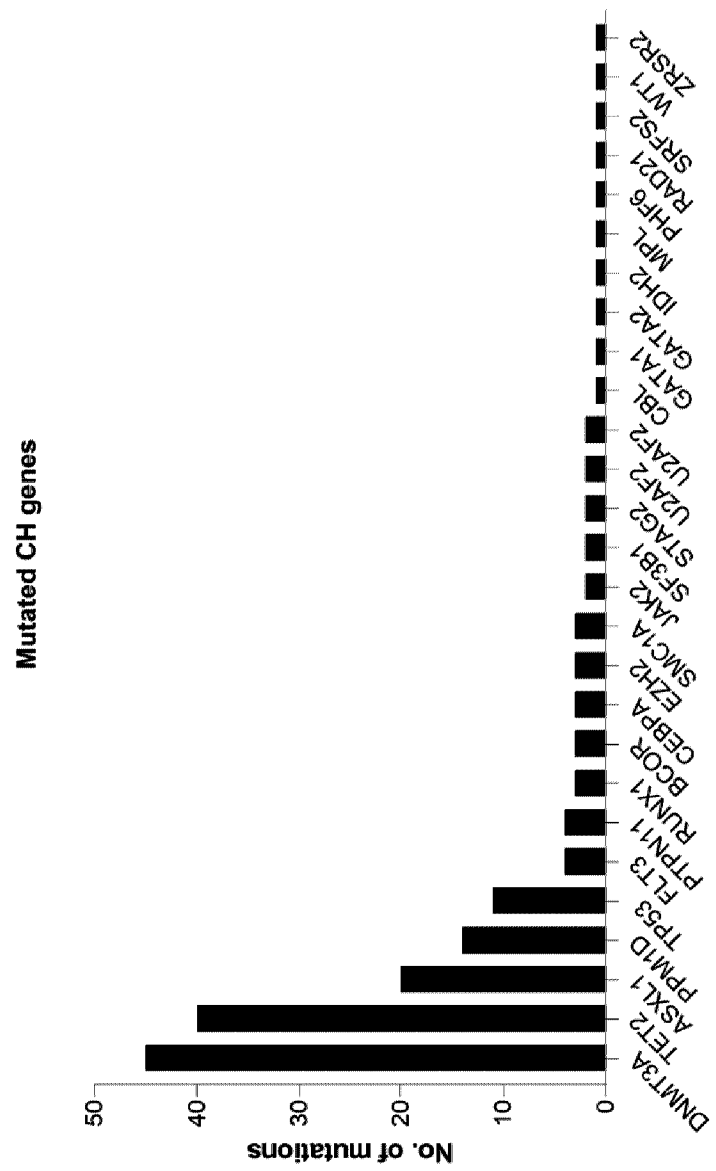


Figure 14

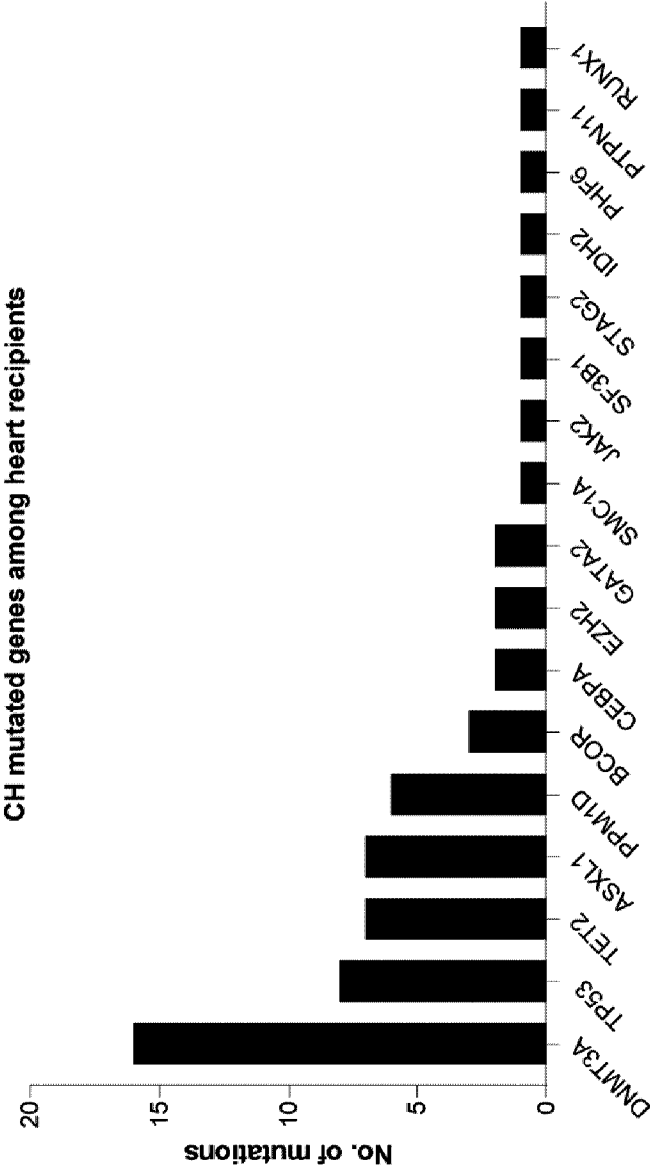


Figure 15

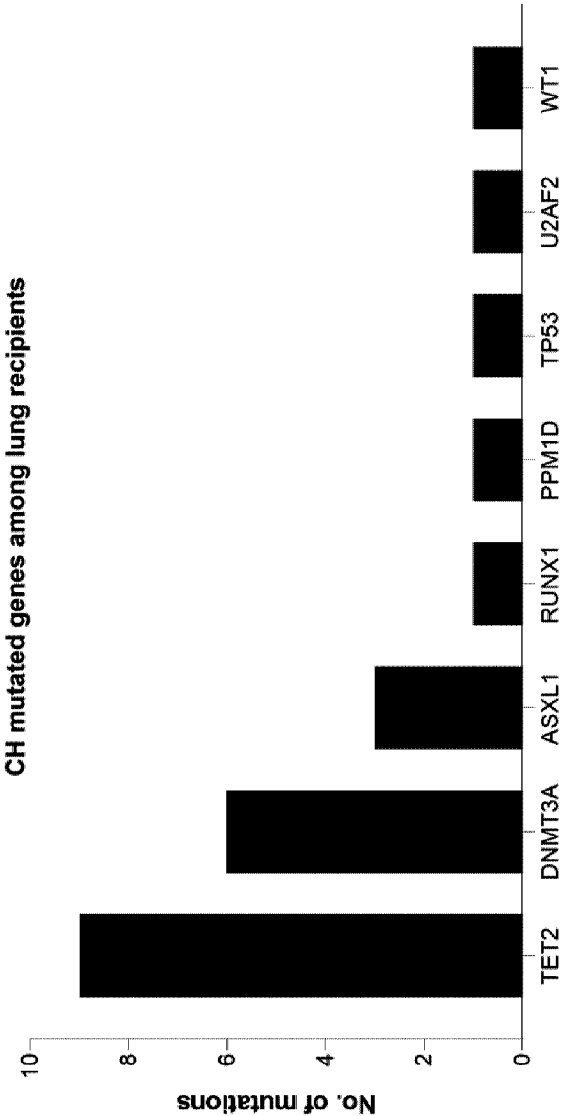


Figure 16

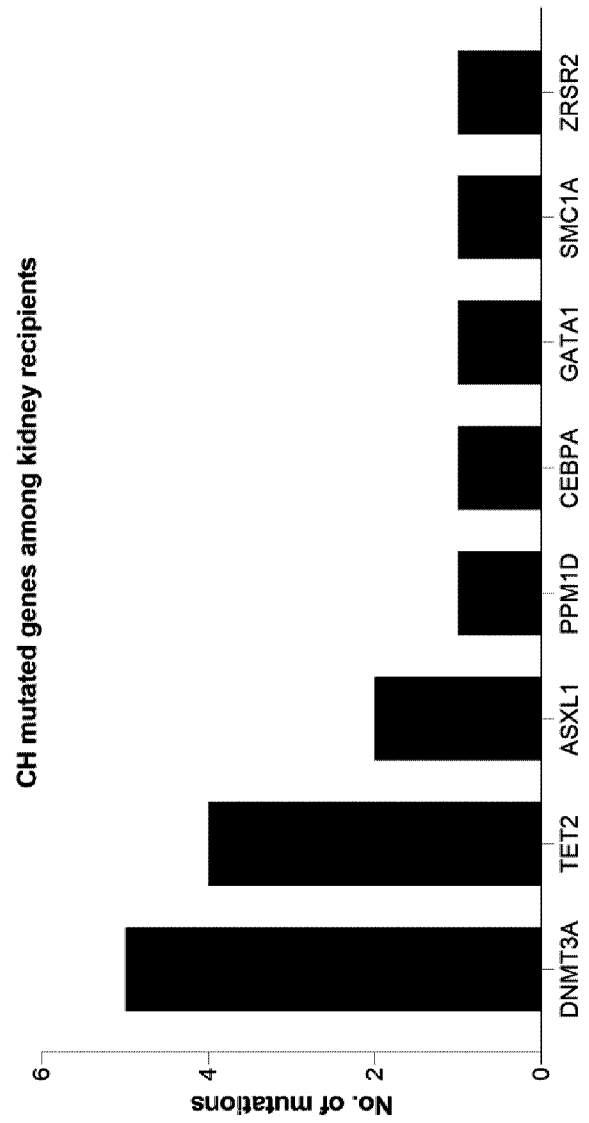


Figure 17

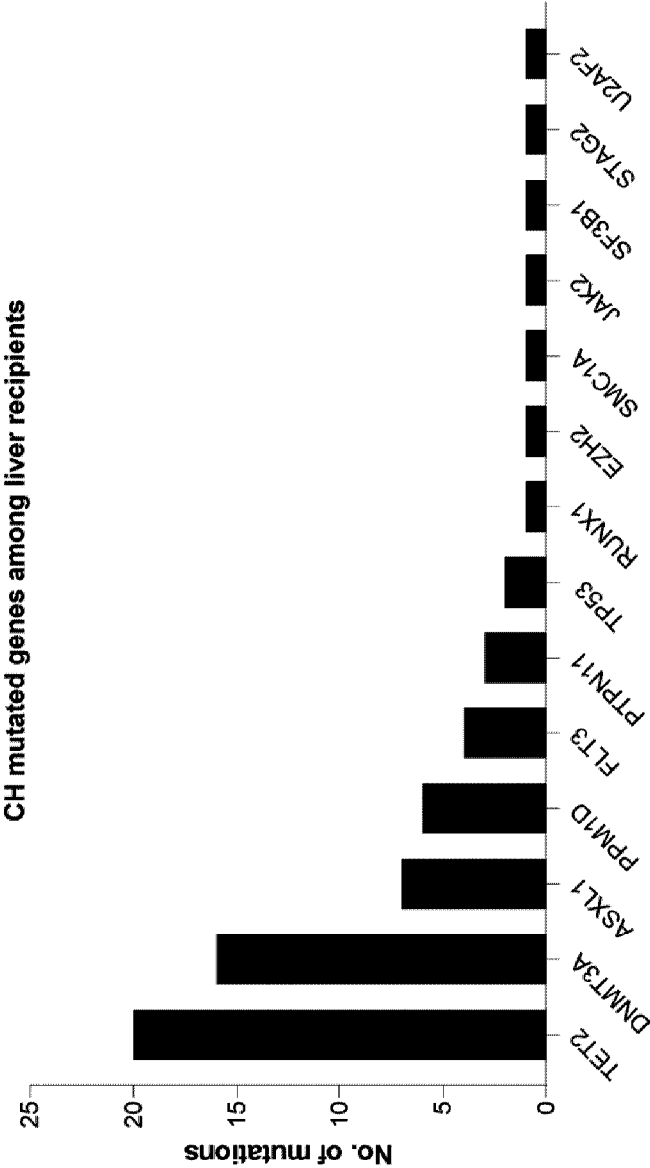


Figure 18

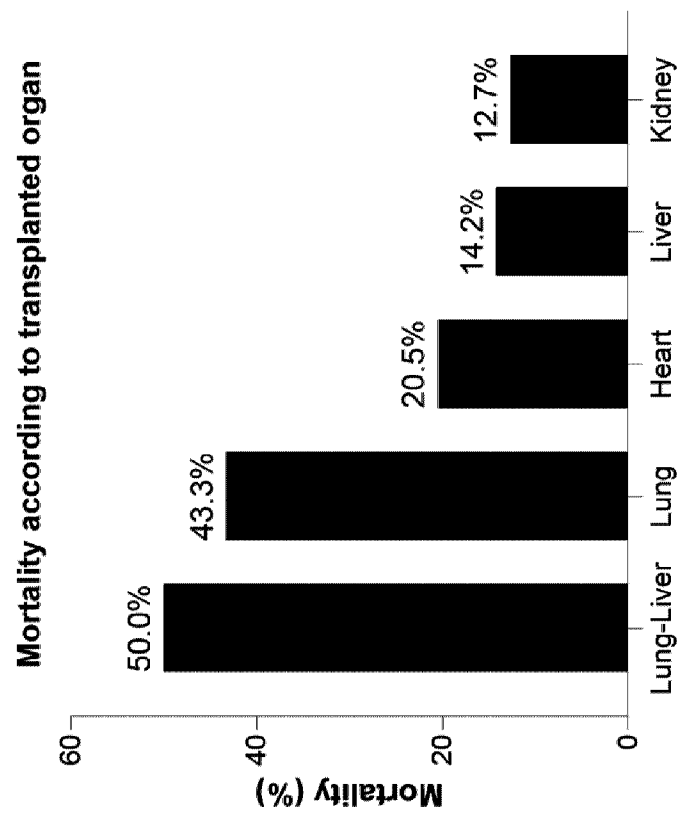


Figure 19

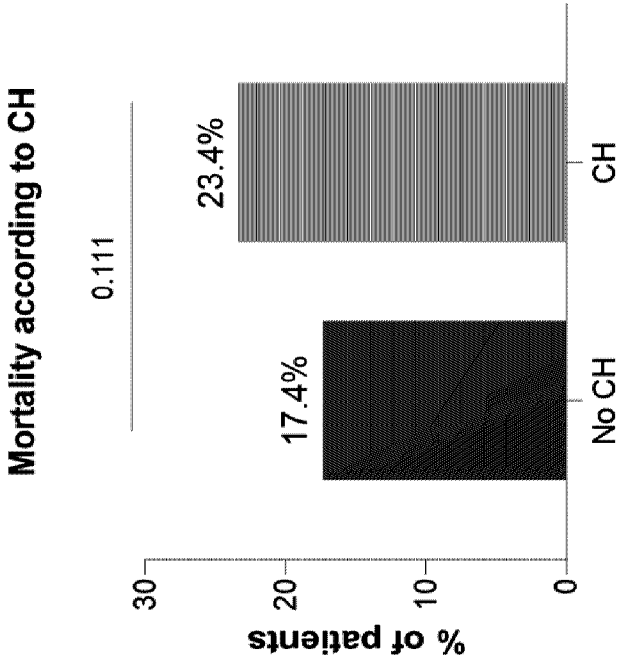


Figure 20

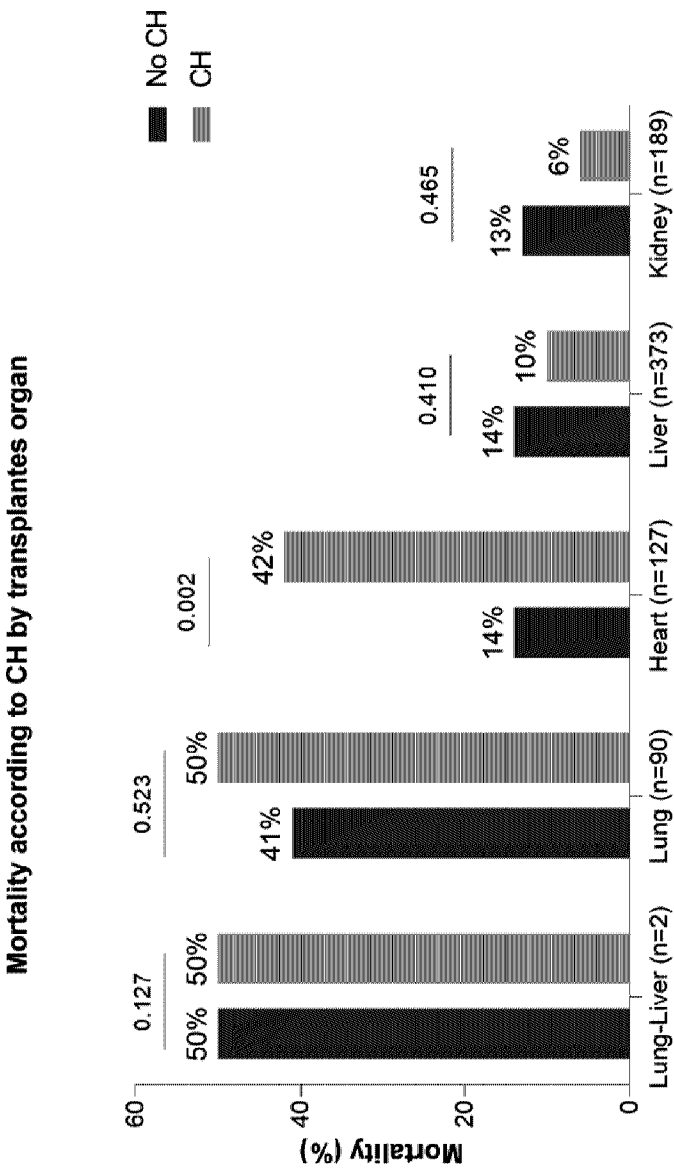


Figure 21

INTERNATIONAL SEARCH REPORT

 International application No.
PCT/CA2023/050553

A. CLASSIFICATION OF SUBJECT MATTER

 IPC: *C12Q 1/6809* (2018.01), *C12Q 1/6883* (2018.01), *C40B 30/04* (2006.01), *G01N 33/50* (2006.01),
G16B 20/00 (2019.01)

 CPC: , *C12Q 1/6809* (2020.01), *C12Q 1/6883* (2020.01), *C40B 30/04* (2020.01),
G01N 33/5091 (2020.01), *G16B 20/00* (2022.02)

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

 Minimum documentation searched (classification system followed by classification symbols)
 Keywords used across the whole IPC and CPC.

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic database(s) consulted during the international search (name of database(s) and, where practicable, search terms used)

 PubMed, Questel Orbit, Scopus, Canadian Patent Database, Genome Quest, STN: BIOSIS, BIOTECHABS:
 clonal hematopoiesis, SmMIP, cardiogenic shock, orthotopic heart transplant, hypertrophic cardiomyopathy, lung transplant, probe mutations
 (Table A), melanoma immunotherapy, non-hematological cancer metastasis

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
P, X	TAGUE LK, OETJEN KA, MAHADEV A, WALTER MJ, ANTHONY H, KREISEL D, LINK DC, GELMAN AE, "Increased clonal hematopoiesis involving DNA damage response genes in patients undergoing lung transplantation". JCI Insight, 10 April 2023 (10-04-2023), Vol. 8(7), pp. e165609, (whole document).	21-23
X Y	TAGUE, LANESHIA; OETJEN, KAROLYN A.; MAHADEV, ANIRUDH; LINK, DANIEL C.; GELMAN, ANDREW E, "Increased Incidence of Clonal Hematopoiesis in Lung Transplant Recipients Involves DNA Damage Response Genes". Blood, 5 November 2021 (05-11-2021), Vol. 138(S1), pp. 2163, (whole document).	1-4, 21-23 5-11, 27

☒ Further documents are listed in the continuation of Box C.

☐ See patent family annex.

* "A" "D" "E" "L" "O" "P"	Special categories of cited documents: document defining the general state of the art which is not considered to be of particular relevance document cited by the applicant in the international application earlier application or patent but published on or after the international filing date document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified) document referring to an oral disclosure, use, exhibition or other means document published prior to the international filing date but later than the priority date claimed	"T" "X" "Y" "&"	later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art document member of the same patent family
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 Date of the actual completion of the international search
 06 July 2023 (06-07-2023)

 Date of mailing of the international search report
 06 July 2023 (06-07-2023)

 Name and mailing address of the ISA/CA
 Canadian Intellectual Property Office
 Place du Portage I, C114 - 1st Floor, Box PCT
 50 Victoria Street
 Gatineau, Quebec K1A 0C9
 Facsimile No.: 819-953-2476

 Authorized officer

 Antonio Candeliere (819) 639-7769

INTERNATIONAL SEARCH REPORT

International application No.

PCT/CA2023/050553

C (Continuation). DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X Y	MITCHELL SR, GOPAKUMAR J, JAISWAL S, " <i>Insights into clonal hematopoiesis and its relation to cancer risk</i> ". Curr Opin Genet Dev, February 2021 (02-2021), Vol. 66, pp. 63-69, ("CHIP in solid tumors" section).	1-4 5-11, 27
X Y	BOLTON KL, PTASHKIN RN, GAO T, BRAUNSTEIN L, DEVLIN SM, KELLY D, PATEL M, BERTHON A, SYED A, YABE M, COOMBS CC, CALTABELLOTTA NM, WALSH M, OFFIT K, STADLER Z, MANDELKER D, SCHULMAN J, PATEL A, PHILIP J, BERNARD E, GUNDEM G, OSSA JEA, LEVINE M, MARTINEZ JSM, FARNOUD N, GLODZIK D, LI S, ROBSON ME, LEE C, PHAROAH PDP, STOPSACK KH, SPITZER B, MANTHA S, FAGIN J, BOUCAI L, GIBSON CJ, EBERT BL, YOUNG AL, DRULEY T, TAKAHASHI K, GILLIS N, BALL M, PADRON E, HYMAN DM, BASELGA J, NORTON L, GARDOS S, KLIMEK VM, SCHER H, BAJORIN D, PARAISO E, BENAYED R, ARCILA ME, LADANYI M, SOLIT DB, BERGER MF, TALLMAN M, GARCIA-CLOSAS M, CHATTERJEE N, DIAZ LA JR, LEVINE RL, MORTON LM, ZEHIR A, PAPAEMMANUIL E, " <i>Cancer therapy shapes the fitness landscape of clonal hematopoiesis</i> ". Nat Genet, November 2020 (11-2020), Vol. 52(11), pp. 1219-1226, (whole document).	1-4 5-11, 27
X Y	ARENDS CM, DIMITRIOU S, STAHLER A, HABLESREITER R, STRZELECKA PM, STEIN CM, TILGNER M, SAIKI R, OGAWA S, BULLINGER L, MODEST DP, STINTZING S, HEINEMANN V, DAMM F, " <i>Clonal hematopoiesis is associated with improved survival in patients with metastatic colorectal cancer from the FIRE-3 trial</i> ". Blood, 10 March 2022 (10-03-2022), Vol. 139(10), pp. 1593-1597, (whole document).	1-4 5-11
X Y	MEDEIROS JF, CAPO-CHICHI JM, SHLUSH LI, DICK JE, ARRUDA A, MINDEN MD, ABELSON S, " <i>SmMIP-tools: a computational toolset for processing and analysis of single-molecule molecular inversion probes-derived data</i> ". Bioinformatics, 12 February 2022 (12-02-2022), Vol. 38(8), pp. 2088-2095, (discussion and supplementary data).	24-26 5-11, 27

INTERNATIONAL SEARCH REPORT

International application No.

PCT/CA2023/050553**Box No. I****Nucleotide and/or amino acid sequence(s) (Continuation of item 1.c of the first sheet)**

1. With regard to any nucleotide and/or amino acid sequence disclosed in the international application, the international search was carried out on the basis of a sequence listing:

- a. ☐ forming part of the international application as filed.
- b. ☒ furnished subsequent to the international filing date for the purposes of international search (Rule 13*ter*.1(a)),
☒ accompanied by a statement to the effect that the sequence listing does not go beyond the disclosure in the international application as filed.

2. ☐ With regard to any nucleotide and/or amino acid sequence disclosed in the international application, this report has been established to the extent that a meaningful search could be carried out without a WIPO Standard ST.26 compliant sequence listing.

3. Additional comments:

INTERNATIONAL SEARCH REPORT

International application No.
PCT/CA2023/050553

Box No. II Observations where certain claims were found unsearchable (Continuation of item 2 of the first sheet)

This international search report has not been established in respect of certain claims under Article 17(2)(a) for the following reasons:

1. ☐ Claim Nos.:
because they relate to subject matter not required to be searched by this Authority, namely:

2. ☐ Claim Nos.:
because they relate to parts of the international application that do not comply with the prescribed requirements to such an extent that no meaningful international search can be carried out, specifically:

3. ☐ Claim Nos.:
because they are dependent claims and are not drafted in accordance with the second and third sentences of Rule 6.4(a).

Box No. III Observations where unity of invention is lacking (Continuation of item 3 of first sheet)

This International Searching Authority found multiple inventions in this international application, as follows:

The claims are directed to a plurality of inventive concepts as follows:

- Group A** - Claims 13 - 15 (completely) 1 – 12, 27 (partly) are directed to predicting the risk of cardiogenic shock;
- Group B** - Claims 16 - 18 (completely) 1 – 12, 27 (partly) are directed to predicting the risk of an adverse outcome after orthotopic heart transplant;
- Group C** - Claims 19, 20 (completely) 1 – 12, 27 (partly) are directed to predicting the risk of hypertrophic cardiomyopathy;
- Group D** - Claims 21 - 23 (completely) 1 – 11, 27 (partly) are directed to predicting the risk of an adverse outcome after lung transplant;

.../continued on "extra sheet" below.

1. ☐ As all required additional search fees were timely paid by the applicant, this international search report covers all searchable claims.
2. ☒ As all searchable claims could be searched without effort justifying additional fees, this Authority did not invite payment of additional fees.
3. ☐ As only some of the required additional search fees were timely paid by the applicant, this international search report covers only those claims for which fees were paid, specifically claim Nos.:

4. ☐ No required additional search fees were timely paid by the applicant. Consequently, this international search report is restricted to the invention first mentioned in the claims; it is covered by claim Nos.:

Remark on Protest

- ☐ The additional search fees were accompanied by the applicant's protest and, where applicable, the payment of a protest fee.
- ☐ The additional search fees were accompanied by the applicant's protest but the applicable protest fee was not paid within the time limit specified in the invitation.
- ☐ No protest accompanied the payment of additional search fees.

.../continued from Box III above:

Group E - Claims 24 - 26 (completely) are directed to clonal hematopoiesis SmMIP probes;

Group F - Claims 28, 29 (completely) are directed to predicting the benefit of melanoma immunotherapy; and

Group G - Claims 30, 31 (completely) are directed to predicting the metastatic risk in a non-hematological cancer.

The common set of elements linking the subject-matter of claims 1 to 23 and 27 is prediction of the risk of a disease condition of a solid organ.

The common set of elements is known from any one of D2 to D5; therefore the common set of elements does not involve a same or corresponding special technical feature. D2 teaches evidence of increased clonal hematopoiesis in a solid organ transplant population. D3 teaches the presence of Clonal Hematopoiesis of Indeterminate Potential (CHIP) that is associated with increased mortality rate in patients with solid cancers. D4 teaches the mechanisms that drive the transition of a normal HSPC to a cell with a considerably stronger proliferation advantage, and study how the ensuing trajectories are shaped by host and environmental exposures including age, ethnicity, smoking and solid cancer therapy. D5 teaches that in metastatic colon cancer, presence of DNMT3A clonal hematopoiesis was associated with improved overall survival, possibly due to enhanced anti-tumor immunosuppressive activity following cytotoxic chemotherapy. Thus, due to the lack of a common set of elements, the above-mentioned groups lack unity. Therefore the claims cannot be considered to be one invention in the sense of being linked in such a manner that they form a single general inventive concept.

Further, claims 24 to 26 and 28 to 31 cannot be considered to be one invention in the sense of being so linked to form a single general inventive concept because each of the groups E, F and G possess different special technical features and these features do not share any technical relationship.

The claims must be limited to one inventive concept as set out in PCT Rule 13.