Biomarker	Suggested website label
CDKN2A	Cyclin-dependent kinase inhibitor 2A (CDKN2A)
Ki67	Antigen Ki67
P53	P53
RB	Retinoblastoma protein (RB)
ATIC	ATIC
BCL2	BCL2
BCL6	BCL6
BCL9	BCL9
CCND2	CCND2

CCND3	CCND3
EIF4A2	EIF4A2
IDO1	IDO1
MUC1	MUC1
NPM1	nucleophosmin (NPM1)
PCSK7	PCSK7
PTPN1	PTPN1

PTPRJ	PTPRJ
PTPRK	PTPRK
RHOH	RHOH
RXRB	RXRB
SH2D1A	SH2D1A
tumor necrosis factor superfamily, member 8 (TNFRSF8), also known as CD30	TNFRSF8
Tumor necrosis factor-alpha (TNF-α)	TNF-α
Tumor necrosis factor-beta (TNF-β)	TNF-β

TSPYL2	TSPYL2
AKR1C1	AKR1C1
CXCR5	CXCR5
CYBA	CYBA
FCGR2A	FCGR2A
FCGR3A	FCGR3A
IGH	IGH

IGL	Immunoglobulin lamda locus
IL12A	Interleukin 12A (IL12A)
IL4	Interleukin 4
SHMT1	SHMT1
UCHL1	UCHL1
BCR-ABL1 fusion gene	BCR-ABL1
Philadelphia chromosome	Philadelphia chromosome
Sokal score	Sokal score

Hasford (EURO) score	Hasford (EURO) score
European Treatment and Outcome Study (EUTOS) score	EUTOS score
EUTOS Long-Term Survival (ELTS) score	ELTS score
Heparanase	Heparanase
VEGF	Vascular Endothelial Growth Factor (VEGF)
CD68+ cells	CD68+ Cells
CD163+ cells	CD163+ Cells
CD83	CD83

PD-1/PD-L1	PD-1/PD-L1
FOXP3	FOXP3
CD21	CD21
CD117	CD117
LDH	Serum lactate dehydrogenase (LDH)
CD23	CD23
Thymidine kinase	Thymidine kinase
Beta 2 microglobulin	Beta 2 microglobulin
Zeta associated protein (ZAP70)	ZAP70
CD38	CD38

13q	13q
11q- (ATM)	11q- (ATM)
Trisomy 12	Trisomy 12
6q-	6q-
IgHV mutation analysis	IgHV mutation analysis
FLT3	FLT3-ITD, FLT3-TKD
CEBPA	CEBPA
MLLT3-KMT2A t(9;11)(p22;q23)	MLLT3-KMT2A t(9;11)(p22;q23)
RUNX1-RUNX1T1 t(8;21)	RUNX1-RUNX1T1 t(8;21)

CBFB-MYH11 inv(16); t(16;16)	CBFB-MYH11 inv(16); t(16;16)
inv(3); t(3;3) RPN1/MECOM	inv(3); t(3;3) RPN1/MECOM
RUNX1	RUNX1
ASXL1	ASXL1
KIT	KIT
t(6;9)	t(6;9) DEK-CAN
t(9;22)	t(9;22)
monosomal karyotype	monosomal karyotype -5; -7
CD138-selected bone marrow plasma cells	CD138
t(4;14)	t(4;14)
T(14;16)	T(14;16)

1q gain; 1q21 amplification	1q gain; 1q21 amplification
del(1p)	del(1p)
ETV6-RUNX1 gene fusion	ETV6-RUNX1
ABL gene fusions	ABL
ABL2 gene fusions	ABL2
CRLF2 gene fusions	CRLF2
CSF1R gene fusions	CSF1R
EPOR gene fusions	EPOR

JAK2 gene fusions	JAK2
PDGFRB gene fusions	PDGFRB
IL7R gene mutations	IL7R
SH2B3 gene mutations	SH2B3
JAK1 gene mutations	JAK1
JAK2 gene mutations	JAK2

JAK3 gene mutations	JAK3

Description	Cancer type
Cyclin-dependent kinase inhibitor 2A (*CDKN2A*) is a gene that makes several proteins, including p16 and p14arf. These two proteins suppress tumours, so mutations in the *CDKN2A* gene can lead to the development of cancers (e.g. melanoma and pancreatic cancer).	
Antigen Ki67 is a protein that plays a key role in cell proliferation. Ki67 is used as a marker to assess the rate at which cancer cells divide and form new cells. High levels of Ki67 are often correlated with a poor prognosis.	Breast, Lung, Haem
P53 is a protein that suppresses tumours and stops normal cells turning into cancerous cells via numerous methods. The *TP53* gene that controls the P53 protein is the most frequently mutated gene in human cancers.	Lung, Haem, Myeloma
Retinoblastoma protein (RB) is a protein that suppresses tumours and stops excessive cell growth. It has been shown not to work properly in several types of cancers.	
This gene encodes a protein that catalyzes the final two steps of the pathway responsible for purine synthesis. Genetic translocation involving this gene can result in the formation of an ATIC-ALK fusion protein, seen in cases of anaplasti large cell lymphoma (ALCL).	Haem (NHL)
The *BCL2* gene encodes a transmembrane protein in mitochondria that inhibtis apoptosis in lymphocyes. Genetic translocations, such as to the immunoglobulin heavy chain locus, can cause constitutive expression of the BCL2 protein and are thought to be the cause of follicular lymphoma.	Haem (NHL, HL)
BCL6 encodes a zinc finger transcription factor that represses transcription. Genetic hypermutations and translocations in this gene are associated with diffuse large cell lymphomas (DLCL). This protein may also be associated with the way DLCL develops.	Haem (NHL)
The function of BCL9 is unknown, however, expression of this protein is associated with B-cell malignancies including lymphomas.	Haem (NHL)
The *CCND2* gene encodes the protein Cyclin D2 which regulates the kinases CDK4 or CDK6 required for cell cycle G1 to S phase transition. Cyclin D3 has also been shown to phosphorylate the tumor supressor protein Rb. Rearrangemnts in the *CCDN2* gene are the most frequent in Cyclin D1 negative mantle cell lymphoma.	Haem (NHL)

The *CCND3* gene encodes the protein Cyclin D3 which regulates the kinases CDK4 or CDK6 required for ell cycle G1 to S phase transition. Cyclin D3 has also been shown to phosphorylate the tumor supressor protein Rb. Oncogenic mutations in the *CCND3* gene have been shown to generate highly stable Cyclin D3 proteins that can strongly drive cell cycle progression. This biomarker is used to help diagnose non-Hodgkin lymphoma.	Haem (NHL)
This gene encodes a protein involved in the intiation of protein translation. The translocation of *EFI4A2* with the *BCL6* gene has been observed in cases of follicular lymphoma and may indicate transformation into a more aggressive form of the disease.	Haem (NHL)
This gene encodes a protein responsible for the rate-limiting step in tryptophan catabolsim. The IDO protein is associated with suppressing immune responses and facilitating immunological tolerance. IDO expression is associated with several hematological malignancies including lymphoma.	Haem (NHL)
The MUC1 protein regulates tissue metabolism and gene expression, including inhibition of tumor suppressor genes. The MUC1 gene has been shown to be amplified or rearranged in some cases of B-cell lymphomas.	Haem (NHL)
Nucleophosmin is a posphoprotein found within cells that is thought to regulate the ARF-p53 tumor suppressor pathway. Translocation with the gene that encodes ALK can generate an unusual protein present in some blood cancers.	Haem (NHL, AML)
This gene is involved with the gene expression of "house keeping" proteins, however, unusual translocations of this gene are associated with B-cell lymphomas.	Haem (NHL)
PTPN1 gene encodes a protein that is a member of the protein tyrosine phosphatase family known to regulate cell growth, differentiation, mitotic cycle, and oncogenic transformation. Mutations in the *PTPN1* gene have been associated with increased phosphorylation of proteins in the JAK/STAT pathway and are implicated to drive lymphomagenesis.	Haem (NHL)

PTPRJ gene encodes a protein that is a member of the protein tyrosine phosphatase family known to regulate cell growth, differentiation, mitotic cycle, and oncogenic transformation. PTPRJ has been found to be expressed in a large number of lymphoma cases.	Haem (NHL)
PTPRK gene encodes a protein that is a member of the protein tyrosine phosphatase family known to regulate cell growth, differentiation, mitotic cycle, and oncogenic transformation. PTPRK has been identified as the major tumor suppressor gene that is commonly deleted in cases of primary central nervous system lymphomas.	Haem (NHL)
The RHOH gene encodes a protein that is a member of the RAS family and is expressed in hematopoietic stem cells. This protein functions as a negative regulator of cell proliferation and survival, and genetic mutations or translocations are associated with different leukemias and lymphomas. Translocations with BCL6 have been identified in non-Hodgkins lymphoma.	Haem (NHL)
The RXRB gene encodes a protein that is a member of the retinoid X receptor family, and heterodimerizes with other receptors to modulate gene expression. Single nuclotide polymorphisms within this gene have been associated with certain sub-types of lymphoma.	Haem (NHL)
This protein has been shown to play a major role in T and B-cell signaling, and mutations within this gene have been associated with malignant lymphomas.	Haem (NHL)
The TNFRSF8 receptor is expressed by activated T cells and can mediate signal transduction that leads to NF-kappaB activation. This receptor has been demonstrated to positively regulate apoptosis. Overexpression of TNFRSF8 on the cell surface can define a number of lymphoproliferative disorders including primary cutaneous anaplastic large-cell lymphoma.	Haem (NHL)
TNF-α is a proinflammatory cytokine that is integral to the disease course of rheumatoid arthritis. Gene variants have been identified that may increase the risk of non-Hodgkins lymphoma.	Haem (NHL)
TNF-β is a cytokine produced by lymphocytes and is inolved in many immune responses as well as apoptosis. Genetic mutations in this gene are associated with non-Hodgkin's lymphoma among other diseases.	Haem (NHL)

The *TSPYL2* gene encodes a protein localized to the nucleolus and is involved in chromatin remodeling to negatively regulate cell-cycle progression. Cases of genetic translocation with the ALK gene have been demonstrated in diffuse large B-cell lymphoma.	Haem (NHL)
This gene encodes an enzyme that catalyzes the breakdown of progesterone to the inactive form 20-alpha-hydroxy-progesterone. Certain genetic variants are associated with an increase in oxidative species and therefore an increase in risk for certain subtypes of lymphoma.	Haem (NHL)
The *CXCR5* gene encodes the CXCR5 chemokine receptor involved in T-cell migration as well as signaling B cells into the B-cell zones of secondary lymphoid organs. CXCR5 is specifically expressed in Burkitt's lymphoma. Polymorphisms of CXCR5 may also be related to risk and prognoses of certain suptypes of lymphoma.	Haem (NHL)
The *CYBA* gene plays an important role in processes within the cell. Certain genetic variations may be associated with an increased risk in non-Hodgkins lymphoma subtypes or how the disease progresses.	Haem (NHL)
FCGR2A encodes the cell surface receptor present on several different immune cells that binds the Fc portion of the immunoglobulin class IgG_2. It has been thought that single nucleotide polymorphisms in this gene can lead to alterations in affinity of the receptor for monoclonal antibody-based therapies such as rituximab and therefore impact patient outcomes. Additionally, mutations in this gene can make patients more susceptible to recurrent infections.	Haem (NHL)
FCGR3A encodes the cell surface receptor present on several different immune cells that can bind the Fc portion of the immunoglobulin class IgG_3. Single nucleotide polymorphisms in this gene may lead to alterations in affinity of the receptor for monoclonal antibody-based therapies such as rituximab, and this could impact patient outcomes. Additionally, mutations in this gene can make patients more likely to have repeat infections.	Haem (NHL)
Analysis of the *IGH* gene is a common diagnostic to detect B-cell malignacies such as lymphoma, as the IGH gene has undergone rearrangement even in precursor lymphoblasts and therefore can be detected as malignant clones expand.	Haem (NHL)

The *IGL* gene encodes the lamda class of the heavy chain domain of an immunoglobulin or antibody. Antibodies can recognize foreign antigens and mediate adaptive immune responses. Genetic translocationof the *IGL* gene are rare but can be found in certain kinds of lymphoma.	Haem (NHL)
The *IL12A* encodes the alpha subunit of the interleukin-12 cytokine that mediates important inflammatory responses in both innate and adaptive immunity. Genetic variants of the *IL12A* gene have been associated with an increased risk of certain lymphoma subtypes.	Haem (NHL, HL)
The *IL4* gene encodes a cytokine produced by T cells and mediates differentiation of T-helper cells. Increased levels of IL4 have been seen in certain subtypes of lymphoma and may make it easier for tumor cells to survive.	Haem (NHL)
This protein plays an important role in the metabolic folate pathway, and polymorphisms within this gene contribute to an increase risk of non-Hodgkin's lymphoma.	Haem (NHL)
The *UCHL1* gene encodes a ubiquitin hydrolase and has been shown to be an oncogenic biomarker of aggressive germinal center diffuse large B-cell lymphoma.	Haem (NHL)
The BCR-ABL1 fusion gene, which results from a translocation between chromosomes 9 and 22, is a marker of CML and ALL. By finding out if its there, and how much of it there is, healthcare professionals can diagnose CML or ALL and determine how well a patient is responding to treatment.	Haem (CML, ALL)
The Philadelphia chromosome is produced by the translocation between chromosomes 9 and 22 and contains the BCR-ABL1 fusion gene. By finding out if its there, and how much of it there is, healthcare professionals can diagnose CML or ALL and determine how well a patient is responding to treatment.	Haem (CML, ALL)
A prognostic score used by healthcare professionals to guide treatment strategies. The Sokal score is based on a patient's age, spleen size, and the amount of certain types of blood cells before treatment.	Haem (CML)

A prognostic score used by healthcare professionals to guide treatment strategies. The Hasford (EURO) score is based on a patient's age, spleen size, and the amount of certain types of blood cells before treatment.	Haem (CML)
A prognostic score used by healthcare professionals to guide treatment strategies. The EUTOS score is based on a patient's spleen size and the amount of a certain type of blood cell before treatment.	Haem (CML)
A prognostic score used by healthcare professionals to guide treatment strategies. The ELTS score is based on a patient's age, spleen size, and the amount of certain types of blood cells before treatment.	Haem (CML)
Heparanase is a protein in the body that works to break down carbohydrates in the wall of certain cells, and may be a signal of the spreading of cancer to other organs.	Haem (HL)
Vascular endothelial growth factor (VEGF) is a substance produced by cancer cells that can signal tumour growth and the spread of cancer to other organs in the body. Detection of VEGF may provide information about the possible long-term outlook for a patient with cancer.	Haem (HL)
Detection of increased levels of CD68+ might be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma. However, results of CD163+ might provide more reliable information.	Haem (HL)
Detection of increased levels of CD163+ might be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma, and may provide more reliable information than CD68+.	Haem (HL)
CD83 is a protein that may be seen in high levels in patients with Hodgkin lymphoma. Detection of high levels of CD83 may be helpful for the diagnosis of Hodgkin lymphoma, may also be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma.	Haem (HL)

PD-1 and PD-L1 are proteins that may be a signal cancer cells are present. Detection of high levels of PD-1 may be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma. Detection of PD-L1 may provide information about a patient's long-term outlook, and their response to certain types of treatments.	Haem (HL)
FOXP3 is a type of immune cell that may invade cancer cells to prevent further growth. Detection of increased levels of FOXP3 may be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma.	Haem (HL)
Detection of very low levels of CD21 may be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma.	Haem (HL)
Detection of increased levels of CD117 might be helpful for determining the long-term outlook for a patient with Hodgkin lymphoma.	Haem (HL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). Lactate dehydrogenase is an enzyme requred for turning sugar into energy in the body. High levels can be an indicator of tissue damage.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). CD23 is a protein found on the surface of certain types of B cells.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). Can be used as a surrogate for IgHV mutation.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL) and Multiple Myeloma. Beta 2 microglobulin is a component of MHC class I molecules which are present on all nucleated cells (except for red blood cells).	Haem (CLL), Myeloma
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). ZAP70 is a tyrosine kinase protein normally expressed near the surface membrane of T cells and natural killer cells.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). CD38 is a glycoprotein found on the surface of many immune cells include CD4+, CD8+, B lymphocytes and natural killer cells.	Haem (CLL)

A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). Tends to respond better to alkylating agents (fludarabine and chlorambucil)	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). Typically occur as monoallelic loss with and without mutation of the remaining ATM allele.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). This means the lymphocytes contain an extra chromosome 12.	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). Part of chromosome 6 is missing in the lymphocytes	Haem (CLL)
A prognostic marker for patients with Chronic Lymphocytic Leukaemia (CLL). IgHV is the immunoglobulin heavy chain variable region genes. In B-cell neoplastms, mutations of IgHV are associated with better responses to some treatments	Haem (CLL)
FLT3 is a receptor tyrosine kinase that is expressed by immature hematopoietic cells. Mutations in FLT3 are found in roughly 30% of patients with AML. It is also found in some people with ALL. FLT3-ITD mutations are typically associated with poorer outcomes (depends on allelic ratio). The impact of FLT3-TKD mutations is still debated.	Haem (AML, ALL)
CEBPA is responsible for myeloid differentiation and hindering cellular growth. Biallelic mutations of CEBPA are associated with a more favourable outcome.	Haem (AML)
This fusion t(9;11)(p22;q23) results in a protein that disrupts normal maturation of hematopoietic cells. This fusion is associated with an intermediate prognosis.	Haem (AML, ALL)
This gene is affected by a genetic rearrangement known as translocation. The most common in AML is t(8;21) whereby RUNX1 gene on chromosome 21 fuses to RUNX1T1 on chromosome 8. This abnormailities is associated with a favourable prognosis.	Haem (AML)

This genetic rearrangement, an inversion, involves the fusion of CBFB and MYH11 (inv(16)) genes on chromosome 16. Less common is a translocation between these two genes t(16;16). The fusion prevents CBF from controlling gene activity and is associated with a favourable prognosis.	Haem (AML)
Inversions and translocations of these two genes are seen in roughly 1.4% and 1.6% of patients with AML. The prognosis in these patients is particularly poor.	Haem (AML)
This gene is associated with development of hematopoietic stem cells. These are 'immature' cells that will develop into different types of blood cell. Mutations in this gene are usually linked to a poor prognosis.	Haem (AML)
Gain of function mutation results in poor regulation of gene activity and is associated with a poor prognosis.	Haem (AML)
KIT mutations are found in 46% of patients who have core binding factor leukemia (most frequently t(8;21) and inv(16)). Together this is associated with an intermediate prognosis in AML.	Haem (AML)
Translocation in a gene named Cain is found mostly in young adult patients with AML. It is usually linked to a poor prognosis.	Haem (AML)
The formation of this fusion gene involves BCR-ABL protein and results in enhanced tyrosine kinase activity. This translocation is associated with poor prognosis.	Haem (AML)
Loss of a single chromosome, most commonly chromosome 7 in AML. This is usually linked to a poor diagnosis.	Haem (AML)
Decreased expression of CD138 is frequently observed in plasma cells of myeloma patients and relates to poor pronosis.	Myeloma
Multiple myeloma is characterized by a high occurrence of chromosomal aberrations. Cytogenetic abnormalities are detected by fluorescent in situ hybridization (FISH).	Myeloma
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Multiple myeloma is characterized by a high occurrence of chromosomal aberrations. Cytogenetic abnormalities are detected by fluorescent in situ hybridization (FISH).	Myeloma
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. The ETV6-RUNX1 fusion gene, which results from a fusion of chromosomes 12 and 21, is also known as "TEL/AML". The fusion gene promotes cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the ABL gene are known to promote cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the ABL2 gene are known to promote cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the CRLF2 gene are known to promote cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the CSF1R gene are known to promote cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the EPOR gene are known to promote cancer development.	ALL

A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the JAK2 gene are known to promote cancer development.	ALL
A fusion gene forms when two separate genes are joined together. The presence of fusion genes can help determine which treatment is best for each patient. Fusion genes involving the PDGFRB gene are known to promote cancer development.	ALL
A mutation is a permanent change in the DNA sequence that makes up a gene. Mutations in some genes are known to be associated with ALL. The presence of specific gene mutations can help determine which treatment is best for each patient. Mutations in IL7R are known to o activate cancer promoting pathways in blood cells.	ALL
A mutation is a permanent change in the DNA sequence that makes up a gene. The presence of specific gene mutations can help determine which treatment is best for each patient. SH2B3 is known to regulate the normal formation of blood cells and mutations in the gene are associated with ALL.	ALL
A mutation is a permanent change in the DNA sequence that makes up a gene. Mutations in some genes are known to be associated with ALL. The presence of specific gene mutations can help determine which treatment is best for each patient. Mutations in JAK1 are known to activate cancer promoting pathways in blood cells.	ALL
A mutation is a permanent change in the DNA sequence that makes up a gene. Mutations in some genes are known to be associated with ALL. The presence of specific gene mutations can help determine which treatment is best for each patient. Mutations in JAK2 are known to activate cancer promoting pathways in blood cells.	ALL

A mutation is a permanent change in the DNA sequence that makes up a gene. Mutations in some genes are known to be associated with ALL. The presence of specific gene mutations can help determine which treatment is best for each patient. Mutations in JAK3 are known to activate cancer promoting pathways in blood cells.	ALL

MKUH Response
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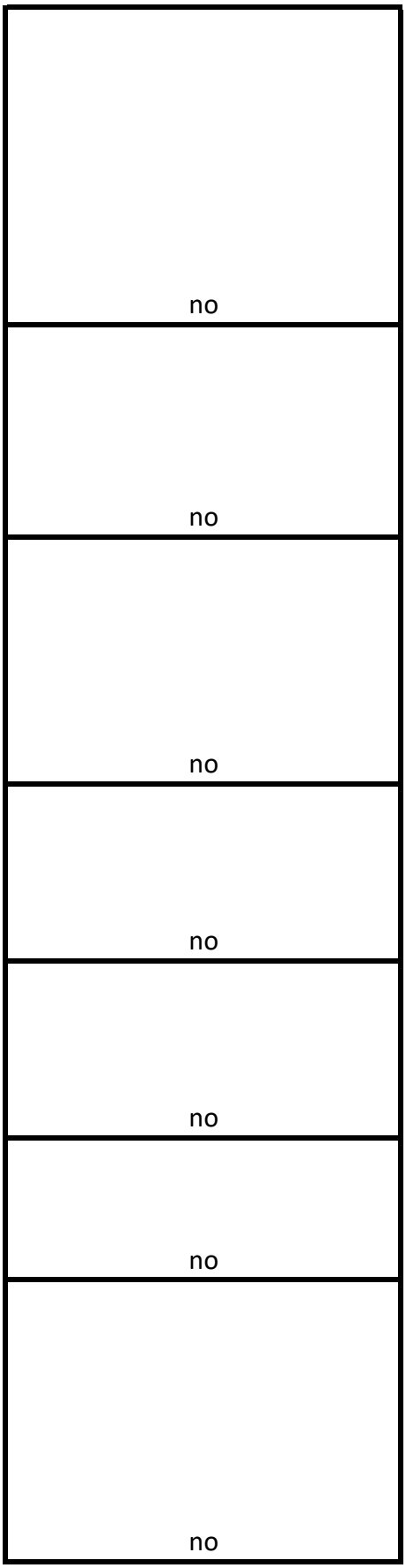
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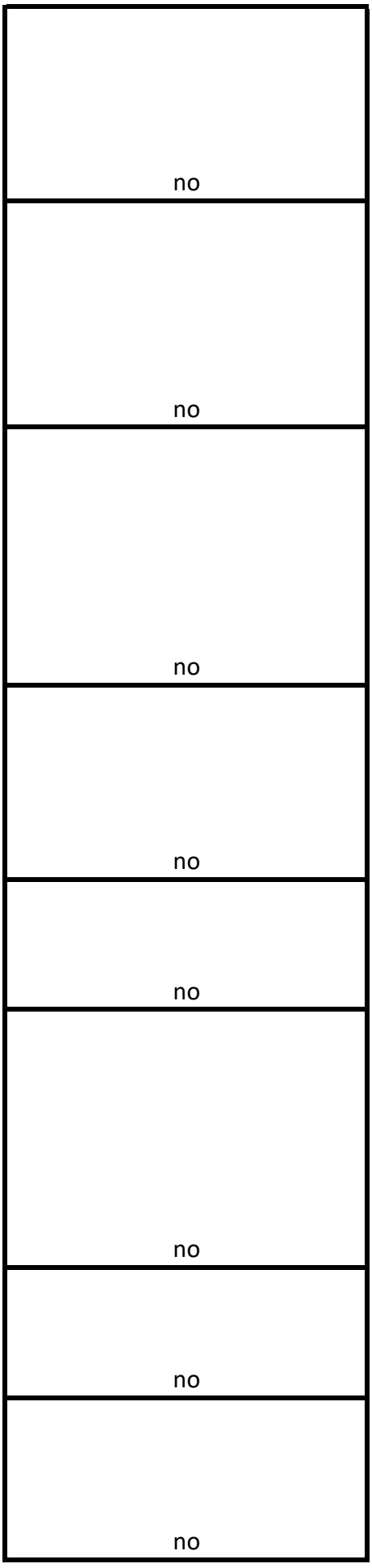
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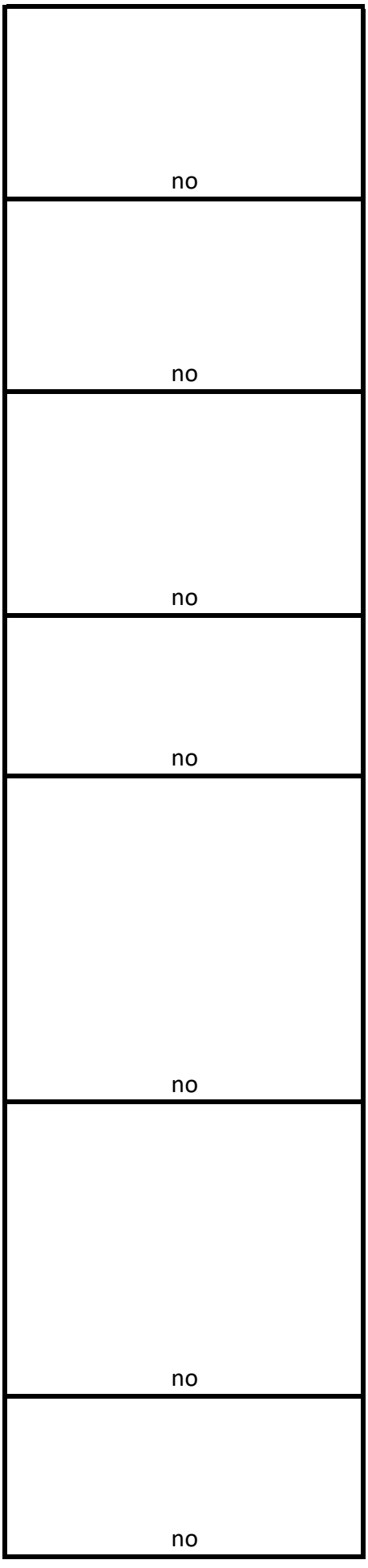
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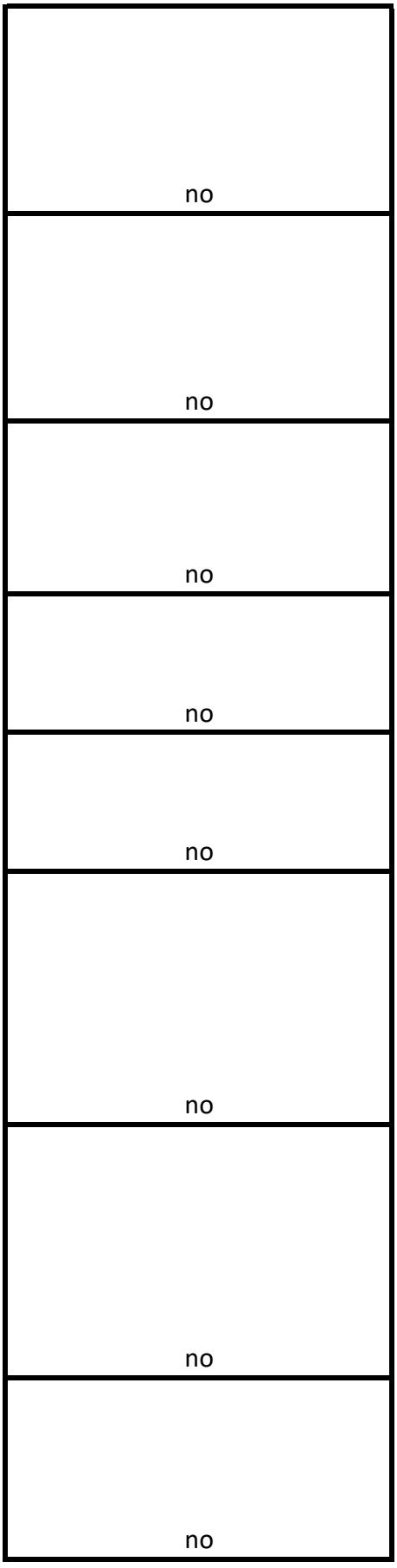
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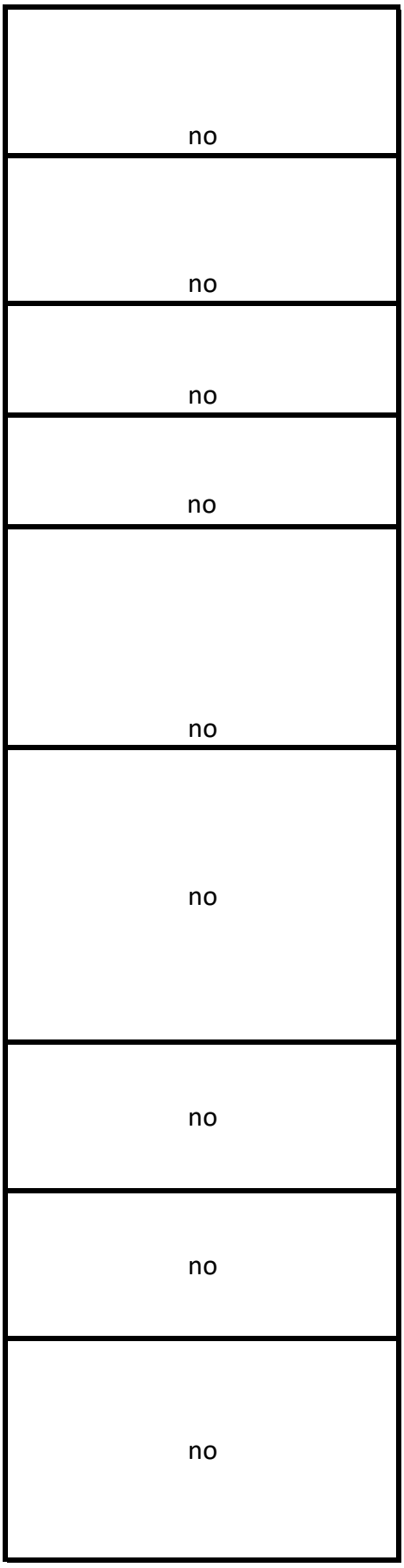
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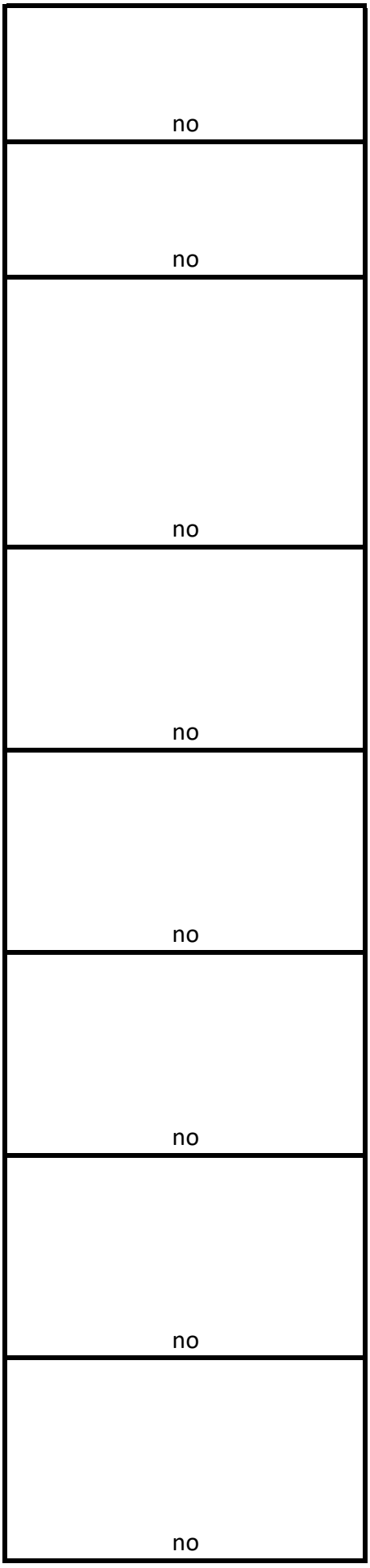
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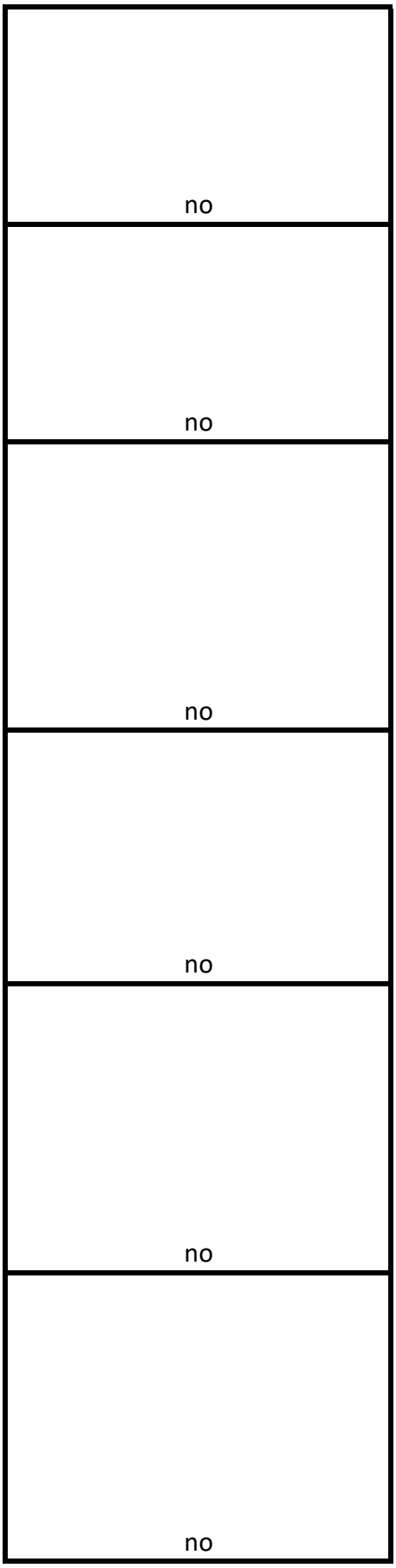
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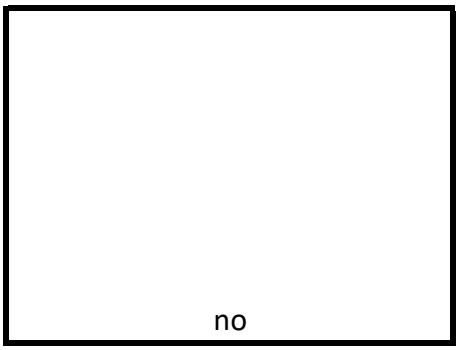
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