

Deborah L White

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

119
papers

5,139
citations

28
h-index

71
g-index

131
ext. papers

5,861
ext. citations

5.9
avg, IF

4.96
L-index

#	Paper	IF	Citations
119	Case Report: Precision Medicine Target Revealed by Modeling of Relapsed, Refractory Acute Lymphoblastic Leukemia From a Child With Neurofibromatosis.. <i>Frontiers in Oncology</i> , 2022 , 12, 851572	5.3	0
118	B-cell acute lymphoblastic leukaemia: recent discoveries in molecular pathology, their prognostic significance, and a review of the current classification. <i>British Journal of Haematology</i> , 2021 ,	4.5	3
117	modeling of TKI resistance in the high-risk B-cell acute lymphoblastic leukemia fusion gene - implications for targeted therapy. <i>Leukemia and Lymphoma</i> , 2021 , 62, 1157-1166	1.9	
116	Simvastatin enhances the efficacy of nilotinib in chronic myeloid leukaemia by post-translational modification and drug transporter modulation. <i>Anti-Cancer Drugs</i> , 2021 , 32, 526-536	2.4	1
115	Outcomes for Australian children with relapsed/refractory acute lymphoblastic leukaemia treated with blinatumomab. <i>Pediatric Blood and Cancer</i> , 2021 , 68, e28922	3	8
114	CKLF and IL1B transcript levels at diagnosis are predictive of relapse in children with pre-B-cell acute lymphoblastic leukaemia. <i>British Journal of Haematology</i> , 2021 , 193, 171-175	4.5	
113	Acquired JAK2 mutations confer resistance to JAK inhibitors in cell models of acute lymphoblastic leukemia. <i>Npj Precision Oncology</i> , 2021 , 5, 75	9.8	1
112	Constitutive JAK/STAT signaling is the primary mechanism of resistance to JAKi in TYK2-rearranged acute lymphoblastic leukemia. <i>Cancer Letters</i> , 2021 , 512, 28-37	9.9	2
111	Monocytoid switch in an adult with B-cell precursor acute lymphoblastic leukaemia characterised by the PAX5 P80R mutation.. <i>Pathology</i> , 2021 ,	1.6	0
110	The immunotoxicity, but not anti-tumor efficacy, of anti-CD40 and anti-CD137 immunotherapies is dependent on the gut microbiota.. <i>Cell Reports Medicine</i> , 2021 , 2, 100464	18	4
109	Two novel cases of NUTM1-rearranged B-cell acute lymphoblastic leukaemia presenting with high-risk features.. <i>British Journal of Haematology</i> , 2021 ,	4.5	1
108	Successful treatment-free remission in chronic myeloid leukaemia and its association with reduced immune suppressors and increased natural killer cells. <i>British Journal of Haematology</i> , 2020 , 191, 433-441	4.5	22
107	Next Generation Genomic Analyses in T-ALL Patients Identify Recurrent and Novel Genomic Abnormalities. <i>Blood</i> , 2020 , 136, 13-14	2.2	1
106	Persistent Activation of JAK/STAT Signaling Plays an Important Role in In Vitro JAKi Resistance in TYK2-rearranged B-Cell Acute Lymphoblastic Leukaemia. <i>Blood</i> , 2020 , 136, 3-3	2.2	
105	Acquired Mutations within the JAK2 Kinase Domain Confer Resistance to JAK Inhibitors in an in Vitro model of a High-Risk Acute Lymphoblastic Leukemia. <i>Blood</i> , 2020 , 136, 5-6	2.2	0
104	KMT2A rearranged acute lymphoblastic leukaemia: Unravelling the genomic complexity and heterogeneity of this high-risk disease. <i>Cancer Letters</i> , 2020 , 469, 410-418	9.9	10
103	The effect of co-occurring lesions on leukaemogenesis and drug response in T-ALL and ETP-ALL. <i>British Journal of Cancer</i> , 2020 , 122, 455-464	8.7	6

102	Lineage of measurable residual disease in patients with chronic myeloid leukemia in treatment-free remission. <i>Leukemia</i> , 2020 , 34, 1052-1061	10.7	23
101	DUX Hunting-Clinical Features and Diagnostic Challenges Associated with -Rearranged Leukaemia. <i>Cancers</i> , 2020 , 12,	6.6	4
100	MLLT10 rearranged acute leukemia: Incidence, prognosis, and possible therapeutic strategies. <i>Genes Chromosomes and Cancer</i> , 2020 , 59, 709	5	3
99	High-risk B-cell acute lymphoblastic leukaemia presenting with hypereosinophilia and acquiring a novel PAX5 fusion on relapse. <i>British Journal of Haematology</i> , 2020 , 191, 301-304	4.5	1
98	Widespread Aberrant Alternative Splicing despite Molecular Remission in Chronic Myeloid Leukaemia Patients. <i>Cancers</i> , 2020 , 12,	6.6	3
97	Lenalidomide maintenance treatment after imatinib discontinuation: results of a phase 1 clinical trial in chronic myeloid leukaemia. <i>British Journal of Haematology</i> , 2019 , 186, e56-e60	4.5	7
96	Azacytidine Sensitizes AML Cells for Effective Elimination By CD123 CAR T-Cells. <i>Blood</i> , 2019 , 134, 3904-3904		3
95	High Risk Genomic Alterations Identified at the Time of Diagnosis Are Strongly Associated with MRD and Subsequent Poor Outcomes in AYA ALL Patients Treated on a Pediatric Inspired Chemotherapy Regimen. <i>Blood</i> , 2019 , 134, 3949-3949	2.2	
94	A Novel Role for HMGN1 in Down Syndrome Acute Lymphoblastic Leukemia. <i>Blood</i> , 2019 , 134, 1462-1462.		
93	Gene expression signature that predicts early molecular response failure in chronic-phase CML patients on frontline imatinib. <i>Blood Advances</i> , 2019 , 3, 1610-1621	7.8	20
92	Patients with low OCT-1 activity and high ABCB1 fold rise have poor long-term outcomes in response to tyrosine kinase inhibitor therapy. <i>Leukemia</i> , 2018 , 32, 2288-2291	10.7	7
91	Pre-B acute lymphoblastic leukaemia recurrent fusion, EP300-ZNF384, is associated with a distinct gene expression. <i>British Journal of Cancer</i> , 2018 , 118, 1000-1004	8.7	14
90	Guidelines for whole genome bisulphite sequencing of intact and FFPE DNA on the Illumina HiSeq X Ten. <i>Epigenetics and Chromatin</i> , 2018 , 11, 24	5.8	27
89	genomic DNA PCR response kinetics during first-line imatinib treatment of chronic myeloid leukemia. <i>Haematologica</i> , 2018 , 103, 2026-2032	6.6	22
88	Precision medicine approaches may be the future for CRLF2 rearranged Down Syndrome Acute Lymphoblastic Leukaemia patients. <i>Cancer Letters</i> , 2018 , 432, 69-74	9.9	1
87	ABCC6 plays a significant role in the transport of nilotinib and dasatinib, and contributes to TKI resistance in vitro, in both cell lines and primary patient mononuclear cells. <i>PLoS ONE</i> , 2018 , 13, e0192187	3.7	13
86	Combination of Nilotinib and Pegylated Interferon Alfa-2b Results in High Molecular Response Rates in Chronic Phase CML: Interim Results of the ALLG CML 11 Pinnacle Study. <i>Blood</i> , 2018 , 132, 459-459	4.3	6
85	Modelling ponatinib resistance in tyrosine kinase inhibitor-naïve and dasatinib resistant + cell lines. <i>Oncotarget</i> , 2018 , 9, 34735-34747	3.3	6

84	Divergent Evolutionary Trajectories of Erk- and Stat5-Activating Lesions in Acute Lymphoblastic Leukemia. <i>Blood</i> , 2018 , 132, 568-568	2.2	
83	The new allosteric inhibitor asciminib is susceptible to resistance mediated by ABCB1 and ABCG2 overexpression. <i>Oncotarget</i> , 2018 , 9, 13423-13437	3.3	24
82	Long-term treatment-free remission of chronic myeloid leukemia with falling levels of residual leukemic cells. <i>Leukemia</i> , 2018 , 32, 2572-2579	10.7	37
81	Integrative genomic analysis reveals cancer-associated mutations at diagnosis of CML in patients with high-risk disease. <i>Blood</i> , 2018 , 132, 948-961	2.2	80
80	The clinical significance of ABCB1 overexpression in predicting outcome of CML patients undergoing first-line imatinib treatment. <i>Leukemia</i> , 2017 , 31, 75-82	10.7	44
79	CML patients with deep molecular responses to TKI have restored immune effectors and decreased PD-1 and immune suppressors. <i>Blood</i> , 2017 , 129, 1166-1176	2.2	95
78	Increased peroxisome proliferator-activated receptor α activity reduces imatinib uptake and efficacy in chronic myeloid leukemia mononuclear cells. <i>Haematologica</i> , 2017 , 102, 843-853	6.6	7
77	Response to overexpression of ABCB1 as prediction marker for CML: How close we are to translation into clinics? <i>Leukemia</i> , 2017 , 31, 769-770	10.7	
76	CYP2C8 Genotype Significantly Alters Imatinib Metabolism in Chronic Myeloid Leukaemia Patients. <i>Clinical Pharmacokinetics</i> , 2017 , 56, 977-985	6.2	8
75	High prevalence of relapse in children with Philadelphia-like acute lymphoblastic leukemia despite risk-adapted treatment. <i>Haematologica</i> , 2017 , 102, e490-e493	6.6	36
74	Modelling Predictors of Molecular Response to Frontline Imatinib for Patients with Chronic Myeloid Leukaemia. <i>PLoS ONE</i> , 2017 , 12, e0168947	3.7	2
73	Differential expression of MUC4, GPR110 and IL2RA defines two groups of CRLF2-rearranged acute lymphoblastic leukemia patients with distinct secondary lesions. <i>Cancer Letters</i> , 2017 , 408, 92-101	9.9	17
72	A Method for Next-Generation Sequencing of Paired Diagnostic and Remission Samples to Detect Mitochondrial DNA Mutations Associated with Leukemia. <i>Journal of Molecular Diagnostics</i> , 2017 , 19, 711-721	5.1	7
71	A novel somatic JAK2 kinase-domain mutation in pediatric acute lymphoblastic leukemia with rapid on-treatment development of LOH. <i>Cancer Genetics</i> , 2017 , 216-217, 86-90	2.3	8
70	TGF- β and IL-6 plasma levels selectively identify CML patients who fail to achieve an early molecular response or progress in the first year of therapy. <i>Leukemia</i> , 2016 , 30, 1263-72	10.7	21
69	Quantitative phosphotyrosine profiling of patient-derived xenografts identifies therapeutic targets in pediatric leukemia. <i>Cancer Research</i> , 2016 , 76, 2766-2777	10.1	14
68	COBL is a novel hotspot for IKZF1 deletions in childhood acute lymphoblastic leukemia. <i>Oncotarget</i> , 2016 , 7, 53064-53073	3.3	8
67	A Low Concentration of ABL001 Potentiates In Vitro TKI-Induced Bcr-Abl Kinase Inhibition in CML Cells. <i>Blood</i> , 2016 , 128, 1121-1121	2.2	1

66	ABCB1 Overexpression Is a Key Initiator of Resistance to Tyrosine Kinase Inhibitors in CML Cell Lines. <i>PLoS ONE</i> , 2016 , 11, e0161470	3.7	19
65	OCT1 and imatinib transport in CML: is it clinically relevant?. <i>Leukemia</i> , 2015 , 29, 1960-9	10.7	41
64	Ponatinib is not transported by ABCB1, ABCG2 or OCT-1 in CML cells. <i>Leukemia</i> , 2015 , 29, 1792-4	10.7	20
63	Relapse of BCR-ABL1-like ALL mediated by the ABL1 kinase domain mutation T315I following initial response to dasatinib treatment. <i>Leukemia</i> , 2015 , 29, 230-2	10.7	20
62	TIDEL-II: first-line use of imatinib in CML with early switch to nilotinib for failure to achieve time-dependent molecular targets. <i>Blood</i> , 2015 , 125, 915-23	2.2	65
61	Living with CML: is death no longer the end (point)?. <i>Blood</i> , 2015 , 126, 2-4	2.2	6
60	KIR2DL5B genotype predicts outcomes in CML patients treated with response-directed sequential imatinib/nilotinib strategy. <i>Blood</i> , 2015 , 126, 2720-3	2.2	18
59	A DNA real-time quantitative PCR method suitable for routine monitoring of low levels of minimal residual disease in chronic myeloid leukemia. <i>Journal of Molecular Diagnostics</i> , 2015 , 17, 185-92	5.1	19
58	Sustained inhibition of STAT5, but not JAK2, is essential for TKI-induced cell death in chronic myeloid leukemia. <i>Leukemia</i> , 2015 , 29, 76-85	10.7	27
57	The Clinical Significance of Early Imatinib Induced ABCB1 Overexpression in Chronic Phase CML Patients: A TIDEL II Sub-Study. <i>Blood</i> , 2015 , 126, 348-348	2.2	1
56	The Allosteric Inhibitor ABL001 Is Susceptible to Resistance in Vitro Mediated By Overexpression of the Drug Efflux Transporters ABCB1 and ABCG2. <i>Blood</i> , 2015 , 126, 4841-4841	2.2	2
55	A 20 Gene Expression Signature That Predicts Early Molecular Response Failure in Chronic Phase CML Patients Treated with Frontline Imatinib. <i>Blood</i> , 2015 , 126, 596-596	2.2	1
54	Targetable kinase-activating lesions in Ph-like acute lymphoblastic leukemia. <i>New England Journal of Medicine</i> , 2014 , 371, 1005-15	59.2	885
53	Interaction of the efflux transporters ABCB1 and ABCG2 with imatinib, nilotinib, and dasatinib. <i>Clinical Pharmacology and Therapeutics</i> , 2014 , 95, 294-306	6.1	54
52	Monoclonal antibody targeting of IL-3 receptor α with CSL362 effectively depletes CML progenitor and stem cells. <i>Blood</i> , 2014 , 123, 1218-28	2.2	74
51	Elevated PTPN2 expression is associated with inferior molecular response in de-novo chronic myeloid leukaemia patients. <i>Leukemia</i> , 2014 , 28, 702-5	10.7	4
50	Low GFI1 expression in white blood cells of CP-CML patients at diagnosis is strongly associated with subsequent blastic transformation. <i>Leukemia</i> , 2013 , 27, 1427-30	10.7	10
49	Clarithromycin enhances dasatinib-induced cell death in chronic myeloid leukemia cells, by inhibition of late stage autophagy. <i>Leukemia and Lymphoma</i> , 2013 , 54, 198-201	1.9	23

48	Dasatinib targets chronic myeloid leukemia-CD34+ progenitors as effectively as it targets mature cells. <i>Haematologica</i> , 2013 , 98, 896-900	6.6	11
47	Degree of kinase inhibition achieved in vitro by imatinib and nilotinib is decreased by high levels of ABCB1 but not ABCG2. <i>Leukemia and Lymphoma</i> , 2013 , 54, 569-78	1.9	21
46	NPM1 mutations occur rarely or not at all in chronic myeloid leukaemia patients in chronic phase or blast crisis. <i>Leukemia</i> , 2013 , 27, 489-90	10.7	9
45	Proton pump inhibitors significantly increase the intracellular concentration of nilotinib, but not imatinib in target CML cells. <i>Leukemia</i> , 2013 , 27, 1201-4	10.7	7
44	Safety and efficacy of imatinib cessation for CML patients with stable undetectable minimal residual disease: results from the TWISTER study. <i>Blood</i> , 2013 , 122, 515-22	2.2	519
43	Association between imatinib transporters and metabolizing enzymes genotype and response in newly diagnosed chronic myeloid leukemia patients receiving imatinib therapy. <i>Haematologica</i> , 2013 , 98, 193-200	6.6	83
42	MicroRNA Dysregulation in Newly Diagnosed Chronic Myeloid Leukaemia Patients. <i>Blood</i> , 2013 , 122, 4985-4985	2.2	
41	STAT5 Is a Critical Component Of The Time-Dependent Sensitivity Of CML Cells To TKI Treatment In a Bcr-Abl-Dependent, But JAK2-Independent Manner. <i>Blood</i> , 2013 , 122, 2705-2705	2.2	
40	Chronic phase chronic myeloid leukemia patients with low OCT-1 activity randomized to high-dose imatinib achieve better responses and have lower failure rates than those randomized to standard-dose imatinib. <i>Haematologica</i> , 2012 , 97, 907-14	6.6	48
39	Contrasting effects of diclofenac and ibuprofen on active imatinib uptake into leukaemic cells. <i>British Journal of Cancer</i> , 2012 , 106, 1772-8	8.7	16
38	Classification of patients with chronic myeloid leukemia on basis of BCR-ABL transcript level at 3 months fails to identify patients with low organic cation transporter-1 activity destined to have poor imatinib response. <i>Journal of Clinical Oncology</i> , 2012 , 30, 1144-5; author reply 1145-6	2.2	15
37	OCT-1 function varies with cell lineage but is not influenced by BCR-ABL. <i>Haematologica</i> , 2011 , 96, 213-206	12	
36	OCT-1 as a determinant of response to antileukemic treatment. <i>Clinical Pharmacology and Therapeutics</i> , 2011 , 89, 608-11	6.1	9
35	Predicting the response of CML patients to tyrosine kinase inhibitor therapy. <i>Current Hematologic Malignancy Reports</i> , 2011 , 6, 88-95	4.4	7
34	Tyrosine kinase inhibitor resistance in chronic myeloid leukemia cell lines: investigating resistance pathways. <i>Leukemia and Lymphoma</i> , 2011 , 52, 2139-47	1.9	48
33	Imatinib trough levels in chronic myelogenous leukemia: does one dose fit all?. <i>Leukemia and Lymphoma</i> , 2011 , 52, 165-7	1.9	
32	Optimizing the selection of kinase inhibitors for chronic myeloid leukemia patients. <i>Expert Review of Hematology</i> , 2011 , 4, 285-99	2.8	5
31	Upfront Imatinib Therapy in CML Patients with Rapid Switching to Nilotinib for Failure to Achieve Molecular Targets or Intolerance Achieves High Overall Rates of Molecular Response and a Low Risk of Progression - An Update of the TIDEL-II Trial. <i>Blood</i> , 2011 , 118, 451-451	2.2	9

30	ATP Dependent Efflux Transporters ABCB1 and ABCG2 Are Unlikely to Impact the Efficacy, or Mediate Resistance to the Tyrosine Kinase Inhibitor, Ponatinib. <i>Blood</i> , 2011 , 118, 2745-2745	2.2	2
29	Non-Steroidal Anti-Inflammatory Drugs and Imatinib; Drug Interactions That May Impact Efficacy,. <i>Blood</i> , 2011 , 118, 3501-3501	2.2	
28	Nilotinib-mediated inhibition of ABCB1 increases intracellular concentration of dasatinib in CML cells: implications for combination TKI therapy. <i>Leukemia</i> , 2010 , 24, 658-60	10.7	27
27	Blocking cytokine signaling along with intense Bcr-Abl kinase inhibition induces apoptosis in primary CML progenitors. <i>Leukemia</i> , 2010 , 24, 771-8	10.7	45
26	Chronic myeloid leukemia CD34+ cells have reduced uptake of imatinib due to low OCT-1 activity. <i>Leukemia</i> , 2010 , 24, 765-70	10.7	57
25	Nilotinib does not significantly reduce imatinib OCT-1 activity in either cell lines or primary CML cells. <i>Leukemia</i> , 2010 , 24, 855-7	10.7	10
24	Functional activity of the OCT-1 protein is predictive of long-term outcome in patients with chronic-phase chronic myeloid leukemia treated with imatinib. <i>Journal of Clinical Oncology</i> , 2010 , 28, 2761-7	2.2	153
23	OCT-1 activity measurement provides a superior imatinib response predictor than screening for single-nucleotide polymorphisms of OCT-1. <i>Leukemia</i> , 2010 , 24, 1962-5	10.7	36
22	Plasma adiponectin levels are markedly elevated in imatinib-treated chronic myeloid leukemia (CML) patients: a mechanism for improved insulin sensitivity in type 2 diabetic CML patients?. <i>Journal of Clinical Endocrinology and Metabolism</i> , 2010 , 95, 3763-7	5.6	43
21	The poor response to imatinib observed in CML patients with low OCT-1 activity is not attributable to lower uptake of imatinib into their CD34+ cells. <i>Blood</i> , 2010 , 116, 2776-8	2.2	18
20	Specific Drug Transporter Genotypes Are Significantly Associated with Increased Rates of Major and Complete Molecular Responses In Newly Diagnosed Chronic Myeloid Leukemia Patients Treated with Imatinib [A TOPS Correlative Substudy. <i>Blood</i> , 2010 , 116, 670-670	2.2	
19	Predicting the response of CML patients to tyrosine kinase inhibitor therapy. <i>Current Hematologic Malignancy Reports</i> , 2009 , 4, 59-65	4.4	6
18	Short-term intense Bcr-Abl kinase inhibition with nilotinib is adequate to trigger cell death in BCR-ABL(+) cells. <i>Leukemia</i> , 2009 , 23, 1205-6	10.7	13
17	BCR-ABL1 lymphoblastic leukaemia is characterized by the deletion of Ikaros. <i>Nature</i> , 2008 , 453, 110-4	50.4	835
16	Dasatinib cellular uptake and efflux in chronic myeloid leukemia cells: therapeutic implications. <i>Clinical Cancer Research</i> , 2008 , 14, 3881-8	12.9	157
15	Is telomerase a player in chronic phase chronic myeloid leukemia, disease progression and imatinib resistance?. <i>Leukemia and Lymphoma</i> , 2008 , 49, 1022-3	1.9	
14	Impact of early dose intensity on cytogenetic and molecular responses in chronic- phase CML patients receiving 600 mg/day of imatinib as initial therapy. <i>Blood</i> , 2008 , 112, 3965-73	2.2	151
13	Genome-Wide Analysis of Genetic Alterations in Chronic Myelogenous Leukemia.. <i>Blood</i> , 2008 , 112, 1089-1089	2.2	3

12	Reduced Activity of the OCT-1 Protein in Primitive CML Cells: A Likely Determinant of Stem Cell Resistance in Imatinib Treated CML Patients. <i>Blood</i> , 2008 , 112, 196-196	2.2	3
11	CML Patients with Low OCT-1 Activity Achieve Better Molecular Responses on High Dose Imatinib Than on Standard Dose. Those with High OCT-1 Activity Have Excellent Responses on Either Dose: A TOPS Correlative Study. <i>Blood</i> , 2008 , 112, 3187-3187	2.2	11
10	The IC50 Assay Is Predictive of Molecular Response, and Indicative of Optimal Dose in De-Novo CML Patients.. <i>Blood</i> , 2008 , 112, 1109-1109	2.2	
9	Measurement of in vivo BCR-ABL kinase inhibition to monitor imatinib-induced target blockade and predict response in chronic myeloid leukemia. <i>Journal of Clinical Oncology</i> , 2007 , 25, 4445-51	2.2	53
8	Imatinib increases the intracellular concentration of nilotinib, which may explain the observed synergy between these drugs. <i>Blood</i> , 2007 , 109, 3609-10	2.2	43
7	Most CML patients who have a suboptimal response to imatinib have low OCT-1 activity: higher doses of imatinib may overcome the negative impact of low OCT-1 activity. <i>Blood</i> , 2007 , 110, 4064-72	2.2	277
6	OCT-1-mediated influx is a key determinant of the intracellular uptake of imatinib but not nilotinib (AMN107): reduced OCT-1 activity is the cause of low in vitro sensitivity to imatinib. <i>Blood</i> , 2006 , 108, 697-704	2.2	370
5	Successful peripheral blood stem cell mobilisation with filgrastim in patients with chronic myeloid leukaemia achieving complete cytogenetic response with imatinib, without increasing disease burden as measured by quantitative real-time PCR. <i>Leukemia</i> , 2003 , 17, 821-8	10.7	39
4	Selected CD34 blood cell allografts for older patients: low transplant-related mortality, graft failure and acute GvHD. <i>Cytotherapy</i> , 2003 , 5, 534-41	4.8	3
3	Detection of minimal residual disease in an AML patient with trisomy 8 using interphase fish. <i>Pathology</i> , 1997 , 29, 289-93	1.6	7
2	Apoptosis regulatory gene NEDD2 maps to human chromosome segment 7q34-35, a region frequently affected in haematological neoplasms. <i>Human Genetics</i> , 1995 , 95, 641-4	6.3	33
1	The expression of mature myeloid cell differentiation markers in acute leukemia. <i>Pathology</i> , 1987 , 19, 137-42	1.6	6