**Supplementary Table S1a: PRISMA 2020 Checklist**

| **Section and Topic**  | **Item #** | **Checklist item**  | **Location where item is reported**  |
| --- | --- | --- | --- |
| **TITLE**  |  |
| Title  | 1 | Identify the report as a systematic review. | 1 |
| **ABSTRACT**  |  |
| Abstract  | 2 | See the PRISMA 2020 for Abstracts checklist. |  |
| **INTRODUCTION**  |  |
| Rationale  | 3 | Describe the rationale for the review in the context of existing knowledge. | 4 |
| Objectives  | 4 | Provide an explicit statement of the objective(s) or question(s) the review addresses. | 4 |
| **METHODS**  |  |
| Eligibility criteria  | 5 | Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses. | 5,6 |
| Information sources  | 6 | Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted. | 5 |
| Search strategy | 7 | Present the full search strategies for all databases, registers and websites, including any filters and limits used. | 5 |
| Selection process | 8 | Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process. | 6 |
| Data collection process  | 9 | Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process. | 6 |
| Data items  | 10a | List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect. | 7 |
| 10b | List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information. | 7 |
| Study risk of bias assessment | 11 | Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process. | 6 |
| Effect measures  | 12 | Specify for each outcome the effect measure(s) (e.g., risk ratio, mean difference) used in the synthesis or presentation of results. | 7 |
| Synthesis methods | 13a | Describe the processes used to decide which studies were eligible for each synthesis (e.g., tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)). | 6, 7 |
| 13b | Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions. | Not applicable |
| 13c | Describe any methods used to tabulate or visually display results of individual studies and syntheses. | Not applicable |
| 13d | Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used. | 7 |
| 13e | Describe any methods used to explore possible causes of heterogeneity among study results (e.g., subgroup analysis, meta-regression). | Not applicable |
| 13f | Describe any sensitivity analyses conducted to assess robustness of the synthesized results. | Not applicable |
| Reporting bias assessment | 14 | Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases). | Not applicable |
| Certainty assessment | 15 | Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome. | 8 |
| **RESULTS**  |  |
| Study selection  | 16a | Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram. | 8 |
| 16b | Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded. | 8 |
| Study characteristics  | 17 | Cite each included study and present its characteristics. | 8 |
| Risk of bias in studies  | 18 | Present assessments of risk of bias for each included study. | 9 |
| Results of individual studies  | 19 | For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots. | 9 |
| Results of syntheses | 20a | For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies. | 8,9 |
| 20b | Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g., confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect. | 9 |
| 20c | Present results of all investigations of possible causes of heterogeneity among study results. | Not applicable |
| 20d | Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results. | Not applicable |
| Reporting biases | 21 | Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed. | Not applicable |
| Certainty of evidence  | 22 | Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed. | 9 |
| **DISCUSSION**  |  |
| Discussion  | 23a | Provide a general interpretation of the results in the context of other evidence. | 10, 11,12 |
| 23b | Discuss any limitations of the evidence included in the review. | 12 |
| 23c | Discuss any limitations of the review processes used. | Not applicable |
| 23d | Discuss the implications of the results for practice, policy, and future research. | 12 |
| **OTHER INFORMATION** |  |
| Registration and protocol | 24a | Provide registration information for the review, including register name and registration number, or state that the review was not registered. | 5 |
| 24b | Indicate where the review protocol can be accessed, or state that a protocol was not prepared. | 5 |
| 24c | Describe and explain any amendments to information provided at registration or in the protocol. | Not applicable |
| Support | 25 | Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review. | Nil |
| Competing interests | 26 | Declare any competing interests of review authors. | Nil |
| Availability of data, code and other materials | 27 | Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review. | Not applicable |

**Table S1b: PRISMA 2020 Abstract Checklist**

| **Section and Topic**  | **Item #** | **Checklist item**  | **Reported (Yes/No)**  |
| --- | --- | --- | --- |
| **TITLE**  |  |
| Title  | 1 | Identify the report as a systematic review. | Yes |
| **BACKGROUND**  |  |
| Objectives  | 2 | Provide an explicit statement of the main objective(s) or question(s) the review addresses. | Yes |
| **METHODS**  |  |
| Eligibility criteria  | 3 | Specify the inclusion and exclusion criteria for the review. | Yes |
| Information sources  | 4 | Specify the information sources (e.g. databases, registers) used to identify studies and the date when each was last searched. | Yes |
| Risk of bias | 5 | Specify the methods used to assess risk of bias in the included studies. | Yes |
| Synthesis of results  | 6 | Specify the methods used to present and synthesise results. | Yes |
| **RESULTS**  |  |
| Included studies  | 7 | Give the total number of included studies and participants and summarise relevant characteristics of studies. | Yes |
| Synthesis of results  | 8 | Present results for main outcomes, preferably indicating the number of included studies and participants for each. If meta-analysis was done, report the summary estimate and confidence/credible interval. If comparing groups, indicate the direction of the effect (i.e. which group is favoured). | Yes |
| **DISCUSSION**  |  |
| Limitations of evidence | 9 | Provide a brief summary of the limitations of the evidence included in the review (e.g. study risk of bias, inconsistency and imprecision). | Yes |
| Interpretation | 10 | Provide a general interpretation of the results and important implications. | Yes |
| **OTHER**  |  |
| Funding | 11 | Specify the primary source of funding for the review. | No |
| Registration | 12 | Provide the register name and registration number. | No |

**Table S2: Search strategy methods with MESH terms**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **S.****No** | **Domain** | **Search details****Searched till February 2024** | **PubMed** | **Scopus** | **Embase** | **OVID** |
| 1. | “Sphingosine kinase 1” AND Lymphoma | "Sphingosine kinase 1"[All Fields] AND ("lymphoma"[MeSH Terms] OR "lymphoma"[All Fields] OR "lymphomas"[All Fields] OR "lymphoma s"[All Fields]) | 18 | 32 | 38 | 148 |
| 2. | SphK1 AND Lymphoma | "SphK1"[All Fields] AND ("lymphoma"[MeSH Terms] OR "lymphoma"[All Fields] OR "lymphomas"[All Fields] OR "lymphoma s"[All Fields]) | 13 | 17 | 21 | 156 |
| 3. | SK1 AND Lymphoma | "SK1"[All Fields] AND ("lymphoma"[MeSH Terms] OR "lymphoma"[All Fields] OR "lymphomas"[All Fields] OR "lymphoma s"[All Fields]) | 9 | 9 | 14 | 230 |
| 4. | “Sphingosine kinase 1” AND Leukemia | "Sphingosine kinase 1"[All Fields] AND ("leukaemia"[All Fields] OR "leukemia"[MeSH Terms] OR "leukemia"[All Fields] OR "leukaemias"[All Fields] OR "leukemias"[All Fields] OR "leukemia s"[All Fields]) | 57 | 110 | 156 | 219 |
| 5. | SphK1 AND Leukemia | "SphK1"[All Fields] AND ("leukaemia"[All Fields] OR "leukemia"[MeSH Terms] OR "leukemia"[All Fields] OR "leukaemias"[All Fields] OR "leukemias"[All Fields] OR "leukemia s"[All Fields]) | 42 | 48 | 67 | 201 |
| 6. | SK1 AND Leukemia | "SK1"[All Fields] AND ("leukaemia"[All Fields] OR "leukemia"[MeSH Terms] OR "leukemia"[All Fields] OR "leukaemias"[All Fields] OR "leukemias"[All Fields] OR "leukemia s"[All Fields]) | 17 | 17 | 64 | 244 |
| 7. | “Sphingosine kinase 1” AND“Multiple myeloma”  |

|  |  |
| --- | --- |
| "Sphingosine kinase 1"[All Fields] AND "multiple myeloma"[All Fields]  |  |

 | 2 | 18 | 31 | 49 |
| 8. | SphK1 AND “Multiple myeloma” | "SphK1"[All Fields] AND "multiple myeloma"[All Fields] | 4 | 8 | 21 | 53 |
| 9. | SK1 AND “Multiple myeloma” | "SK1"[All Fields] AND "multiple myeloma"[All Fields] | 2 | 3 | 10 | 61 |
|  | **Total** | **164** | **262** | **422** | **1361** |

**Table S3: Excluded studies after screening the full-length paper**

|  |  |  |
| --- | --- | --- |
| S. No. | Excluded study | Reason of exclusion |
| 1 | Gencer EB, Ural AU, Avcu F, Baran Y. A novel mechanism of dasatinib-induced apoptosis in chronic myeloid leukemia; ceramide synthase and ceramide clearance genes. Ann Hematol. 2011 Nov;90(11):1265–75. | Cell line study |
| 2 | Paugh SW, Paugh BS, Rahmani M, Kapitonov D, Almenara JA, Kordula T, et al. A selective sphingosine kinase 1 inhibitor integrates multiple molecular therapeutic targets in human leukemia. Blood. 2008 Aug 15;112(4):1382–91. | SphK1 activity is not reported from patient’s sample |
| 3 | Li Q-F, Wu C-T, Duan H-F, Sun H-Y, Wang H, Lu Z-Z, et al. Activation of sphingosine kinase mediates suppressive effect of interleukin-6 on human multiple myeloma cell apoptosis. Br J Haematol. 2007 Sep;138(5):632–9. | No comparison with healthy participants |
| 4 | Evangelisti C, Evangelisti C, Teti G, Falconi M, Cappellini A, Chiarini F, et al. Assessment of the effect of sphingosine kinase inhibitors on apoptosis, unfolded protein response and autophagy of T-acute lymphoblastic leukemia cells: Indications for novel therapeutics. Haematologica. 2014;99((Evangelisti C.; Teti G.; Falconi M.; Cappellini A.; Chiarini F.; Buontempo F.; Bressanin D.; Lonetti A.; Spartà A.; Martelli A.M.) Department of Biomedical and Neuromotor Sciences (DIBINEM), University of Bologna, Bologna, Italy):S83. | SphK1 activity is not reported |
| 5 | Baran Y, Kartal M, Cakir Z, Saydam G, Sahin F. Ceramides regulate resveratrol-induced apoptosis in human acute promyelocytic and chronic myeloid leukemia cells. Haematologica. 2010;95((Baran Y.; Cakir Z.) Izmir Institute of Technology, Faculty of Science, Dept. of Mol. Biol. and Genetics, Urla Izmir, Turkey):686–7. | Full text paper not available |
| 6 | Li Y, Gao Y, Liang B, Nie W, Zhao L, Wang L. Combined effects on leukemia cell growth by targeting sphingosine kinase 1 and sirtuin 1 signaling. Exp Ther Med. 2020 Dec;20(6):262. | Cell line study |
| 7 | Xu L, Zhang Y, Gao M, Wang G, Fu Y. Concurrent targeting Akt and sphingosine kinase 1 by A-674563 in acute myeloid leukemia cells. Biochem Biophys Res Commun. 2016 Apr 15;472(4):662–8. | SphK1 activity is not reported (is reported after the treatment of drugs) |
| 8 | Beider K, Rosenberg E, Bitner H, Leiba M, Koren-Michowitz M, Abraham M, et al. Cross-talk between sphingosine 1-phosphate (S1P) and CXCR4/CXCL12 regulates viability, supports CXCR4-dependent cell motility and defines the sensitivity to FTY720 in multiple myeloma. Haematologica. 2014;99((Beider K.; Rosenberg E.; Bitner H.; Leiba M.; Koren-Michowitz M.; Shimoni A.; Nagler A.) Hematology Division, Bone Marrow Transplantation, Guy Weinshtock Multiple Myeloma Foundation, Sheba Medical Center, Ramat-Gan, Israel):248. | Full text paper not available |
| 9 | KartalYandim M, Kozanoglu I, Ozdogu H, Piskin O, Ozcan MA, Saydam G, et al. Discovering alternative targets in chronic myeloid leukemia (CML): Determination of expression levels of bioactive sphingolipid genes in newly diagnosed and drug-resistant CML patients. Haematologica. 2015;100((KartalYandim M.; Baran Y.) Molecular Biology and Genetics, Izmir Institute of Technology, Izmir, Turkey):432. | Conference Abstract |
| 10 | Lewis AC, D’Andrea R, Powell JA, Pitson SM. Dual sphingosine kinase and BCL-2 inhibition exhibits synergistic cell death in acute myeloid leukemia. Blood [Internet]. 2017;130((Lewis A.C.) Centre for Cancer Biology, SA Pathology, University of South Australia, Australia). Available from: <https://www.embase.com/search/results?subaction=viewrecord&id=L620385243&from=export> | Poster  |
| 11 | Brown TJ, Barth B, Claxton DF. Enhancing ceramide cytotoxicity in acute Myelogenous Leukemia. Blood [Internet]. 2012;120(21).Available from: <https://www.embase.com/search/results?subaction=viewrecord&id=L70961817&from=export> | Full text paper not available |
| 12 | Wallington-Beddoe CT, Pitson SM, Powell JA, Bradstock KF, Bendall LJ. Evaluation of sphingosine kinase 1 as a therapeutic target in B-lineage acute lymphoblastic leukemia. Blood [Internet]. 2013;122(21). Available from: <https://www.embase.com/search/results?subaction=viewrecord&id=L71265032&from=export> | Poster  |
| 13 | KartalYandim M, Kozanoglu I, Ozdogu H, Piskin O, Ozcan MA, Saydam G, et al. Expression levels of ceramide-generating and clearance genes in newly diagnosed and tyrosine kinase inhibitor resistant chronic myeloid leukemia patients: An attempt to find novel targets. Haematologica. 2014;99((KartalYandim M.; Baran Y.) Molecular Biology and Genetics, Izmir Institute of Technology, Izmir, Turkey):69–70. | Full text paper not available  |
| 14 | KartalYandim M, Kozanoglu I, Ozdogu H, Piskin O, Ozcan M, Saydam G, et al. Expression levels of ceramide-metabolising genes in newly diagnosed and tyrosine kinase inhibitor-resistant chronic myeloid leukemia (CML) patients: The discovery of novel targets in CML. Leuk Res. 2014;38((KartalYandim M.; Baran Y.) Izmir Institute of Technology, Department of Molecular Biology and Genetics, Izmir, Turkey):S47. | Duplication of study no. 13 |
| 15 |  LeBlanc FR, Wang H-G, Feith DJ, Loughran Jr. TP. FTY720 (fingolimod) targets the sphingolipid pathway and induces autophagy-related apoptosis in human natural killer large granular lymphocyte leukemia. Blood. 2015;126(23):1288. | Poster |
| 16 | Petrusca DN, Park C, Crean C, Toscani D, Anderson J, Silbermann R, et al. Gfi1 modulation of SphK1 maintains growth and survival of myeloma cells. J Bone Miner Res. 2017;32((Petrusca D.N.) Indiana University, Department of Medicine/Hematology-Oncology, United States): S65. | Abstract presentation at 2017 Annual Meeting of theAmerican Society for Bone and Mineral Research |
| 17 | Toscani D, Park C, Wang F, Anderson J, Giuliani N, David Roodman G, et al. Gfi1 transcription factor is a pro-survival molecule in multiple myeloma cells. Blood [Internet]. 2016;128(22). Available from: <https://www.embase.com/search/results?subaction=viewrecord&id=L614309298&from=export> | Poster |
| 18 | Petrusca DN, Berdyshev E, Crean CD, Anderson JL, Roodman GD. GFI1-Dependent SGPP1 Repression Promotes Growth and Survival of Myeloma Cells. Blood. 2019;134((Petrusca D.N.; Crean C.D.; Anderson J.L.) Medicine/Hematology and Oncology, Indiana University School of Medicine, Indianapolis, IN, United States):4387 | Annual meeting abstract |
| 19 | Gao S, Guo T, Luo S, Zhang Y, Ren Z, Lang X, et al. Growth Inhibitory and Pro-Apoptotic Effects of Hirsuteine in Chronic Myeloid Leukemia Cells through Targeting Sphingosine Kinase 1. Biomol Ther. 2022;30(6):553–61. | Cell line study |
| 20 | Sobue S, Nemoto S, Murakami M, Ito H, Kimura A, Gao S, et al. Implications of sphingosine kinase 1 expression level for the cellular sphingolipid rheostat: relevance as a marker for daunorubicin sensitivity of leukemia cells. Int J Hematol. 2008 Apr;87(3):266–75. | Cell line study |
| 21 | Ricci C, Onida F, Servida F, Radaelli F, Saporiti G, Todoerti K, et al. In vitro anti-leukaemia activity of sphingosine kinase inhibitor. Br J Haematol. 2009 Feb;144(3):350–7. | SphK1 activity is not reported from patient’s sample |
| 22 | Camgoz A, Gencer EB, Ural AU, Baran Y. Mechanisms responsible for nilotinib resistance in human chronic myeloid leukemia cells and reversal of resistance. Leuk Lymphoma. 2013 Jun;54(6):1279–87. | Cell line study |
| 23 | Yan J, Li Q-F, Wang L-S, Wang H, Xiao F-J, Yang Y-F, et al. Methyl-β-cyclodextrin induces programmed cell death in chronic myeloid leukemia cells and, combined with imatinib, produces a synergistic downregulation of ERK/SPK1 signaling. Anticancer Drugs. 2012 Jan;23(1):22–31. | SphK1 activity is not reported from patient sample  |
| 24 | Morad SAF, Tan S-F, Feith DJ, Kester M, Claxton DF, Loughran TPJ, et al. Modification of sphingolipid metabolism by tamoxifen and N-desmethyltamoxifen in acute myelogenous leukemia--Impact on enzyme activity and response to cytotoxics. Biochim Biophys Acta. 2015 Jul;1851(7):919–28. | SphK1 activity is not reported from patient’s sample |
| 25 | Baran Y, Camgoz A, Ural A, Avcu F. Molecular mechanisms of nilotinib resistance and reversal of resistance in chronic myeloid leukemia cells. Haematologica. 2011;96((Baran Y.; Camgoz A.) Izmir Institute of Technology, Izmir, Turkey):88–9. | Cell line study |
| 26 | Bonhoure E, Pchejetski D, Aouali N, Morjani H, Levade T, Kohama T, et al. Overcoming MDR-associated chemoresistance in HL-60 acute myeloid leukemia cells by targeting sphingosine kinase-1. Leukemia. 2006 Jan;20(1):95–102. | Cell line Study |
| 27 | Le Scolan E, Pchejetski D, Banno Y, Denis N, Mayeux P, Vainchenker W, et al. Overexpression of sphingosine kinase 1 is an oncogenic event in erythroleukemic progression. Blood. 2005 Sep 1;106(5):1808–16. | Cell line Study |
| 28 | Li Q-F, Zhu H-Y, Yang Y-F, Liu J, Xiao F-J, Zhang Q-W, et al. Prokineticin-1/endocrine gland-derived vascular endothelial growth factor is a survival factor for human multiple myeloma cells. Leukemia & Lymphoma. 2010 Oct;51(10):1902–12. | SphK1 activity is not reported from patient’s sample |
| 29 | Sobue S, Iwasaki T, Sugisaki C, Nagata K, Kikuchi R, Murakami M, et al. Quantitative RT-PCR analysis of sphingolipid metabolic enzymes in acute leukemia and myelodysplastic syndromes [4]. Leukemia. 2006;20(11):2042–6. | No comparison with healthy participants |
| 30 | Petrusca DN, Park C, Wang F, Anderson J, Roodman DG. Role of Gfi1 transcription factor in myeloma cells growth and survival. FASEB J [Internet]. 2016;30((Petrusca D.N.; Park C.; Wang F.; Anderson J.; Roodman D.G.) Medicine-Hematology/Oncology, Indiana University, Indianapolis, IN, United States). Available from: <https://www.embase.com/search/results?subaction=viewrecord&id=L72319287&from=export> | Abstract |
| 31 | Beider K, Rosenberg E, Bitner H, Leiba M, Koren-Michowitz M, Abraham M, et al. S1P modulator FTY720 targets multiple myeloma cell proliferation and stromal interactions via CXCR4/CXCL12 and mTOR pathways. Blood [Internet]. 2014;124(21).  | Poster |
| 32 | Vrzalikova K, Ibrahim M, Vockerodt M, Perry T, Margielewska S, Lupino L, et al. S1PR1 drives a feedforwardsignalling loop to regulate BATF3 and the transcriptional programme of Hodgkin lymphoma cells. Leukemia. 2018;32(1):214–23. | No comparison with healthy participants |
| 33 | Bayerl MG, Bruggeman RD, Conroy EJ, Hengst JA, King TS, Jimenez M, et al. Sphingosine kinase 1 protein and mRNA are overexpressed in non-Hodgkin lymphomas and are attractive targets for novel pharmacological interventions. Leuk Lymphoma. 2008 May;49(5):948–54. | No comparison with healthy participants |
| 34 | Limaye V, Li X, Hahn C, Xia P, Berndt MC, Vadas MA, et al. Sphingosine kinase-1 enhances endothelial cell survival through a PECAM-1-dependent activation of PI-3K/Akt and regulation of Bcl-2 family members. Blood. 2005 Apr 15;105(8):3169–77. | Not relevant |
| 35 | Bonhoure E, Lauret A, Barnes DJ, Martin C, Malavaud B, Kohama T, et al. Sphingosine kinase-1 is a downstream regulator of imatinib-induced apoptosis in chronic myeloid leukemia cells. Leukemia. 2008 May;22(5):971–9 | SphK1 activity is not reported from patient’s sample |
| 36 | Li Q-F, Huang W-R, Duan H-F, Wang H, Wu C-T, Wang L-S. Sphingosine kinase-1 mediates BCR/ABL-induced upregulation of Mcl-1 in chronic myeloid leukemia cells. Oncogene. 2007 Dec 13;26(57):7904–8. | Detection method is not mentioned |
| 37 | Almejun MB, Borge M, Colado A, Podaza E, Risnik D, DeBrasi CD, et al. Sphingosine kinases (SK): Key molecules associated with the activation, proliferation and ibrutinib-induced cell death of chronic lympocytic leukemia cells. Blood. 2015;126(23):1714. | Poster |
| 38 | Wallington-Beddoe C, Bradstock K, Bendall L. Sphingosine kinases are important therapeutic targets in precursor B-cell acute lymphoblastic leukemia. Exp Hematol. 2012;40(8):S81–2. | Full text paper not available |
| 39 | Lupino L, Perry T, Margielewska S, Hollows R, Ibrahim M, Care M, et al. Sphingosine-1-phosphate signalling drives an angiogenic transcriptional programme in diffuse large B cell lymphoma. Leukemia. 2019 Dec;33(12):2884–97 | No comparison with healthy participants |
| 40 | Huang W-R, Wang L-S, Wang H, Duan H-F, Li Q-F, Gao C-J, et al. [SphK-1/S1P signal pathway in CML cells]. Zhongguo Shi Yan Xue Ye Xue ZaZhi. 2008 Aug;16(4):730–3. | Not in English language |
| 41 | Liu X-Y, Yang Y-F, Wu C-T, Xiao F-J, Zhang Q-W, Ma X-N, et al. Spred2 is involved in imatinib-induced cytotoxicity in chronic myeloid leukemia cells. Biochem Biophys Res Commun. 2010 Mar 19;393(4):637–42. | SphK1 activity is not reported |
| 42 | Powell J, Pitman M, Coolen C, Zhu W, Lewis I, D’Andrea R, et al. Targeting sphingosine kinase as atherapy for acute myeloid leukaemia. Exp Hematol. 2013;41(8):S54. | Poster |
| 43 | Petrusca DN, Berdyshev E, Mulcrone P, Crean CD, Anderson JL, Sun Q, et al. The Gfi1-SphK1 axis regulates the growth and survival of myeloma cells. Blood [Internet]. 2018;132((Petrusca D.N.; Mulcrone P.; Crean C.D.; Anderson J.L.; Roodman G.D.) Department of Medicine/Hematology-Oncology, Indiana University School of Medicine, Indianapolis, IN, United States)  | Poster presentation at 60th ASH Annual Meeting  |
| 44 | Baran Y, Gencer E, Ural A, Avcu F. The roles of ceramide generating and ceramide clearence genes in apoptosis in chronic myeloid Leukemia cells exposed to dasatinib. Haematologica. 2010;95((Baran Y.) Izmir Institute of Technology, Faculty of Science, Dept.of Mol.Biol. and Genetics, Urla Izmir, Turkey):335–6. | Full text paper not available |