Transactions on Materials, Biotechnology and Life Sciences
ISSN: 2960-1932, eISSN: 2960-2319 | Volume 9
FENBC 2026

Daraxonrasib Treatment in RAS-Mutated Pancreatic Cancer: Mechanisms, Clinical Evidence and Future Perspectives

Zhouyu Song

Shenzhen College of International Education, Shenzhen, Guangdong, 518040, China

zsong3322@gmail.com

Abstract. Pancreatic ductal adenocarcinoma (PDAC) ranks among the deadliest malignancies worldwide, with its poor prognosis stemming from aggressive progression, delayed detection, and limited treatment responses. While KRAS has attracted substantial interest as a therapeutic target, its molecular architecture long defied direct pharmacological intervention. The recent introduction of daraxonrasib (RMC-6236)—a multiselective RAS(ON) inhibitor capable of engaging multiple active RAS variants—marks a notable shift in this landscape. By targeting a broader range of mutations rather than a single subtype, this agent holds promise for treating a wider patient population. Preclinical investigations have demonstrated robust suppression of RAS signalling and notable tumour regression, while clinical studies have yielded encouraging patient outcomes. The Phase III RASolute 302 trial, in particular, reported substantial gains in overall survival, progression-free survival, and objective response rates relative to standard second-line chemotherapy. This review explores the pathogenic role of KRAS mutations in pancreatic cancer, traces the development and mechanism of daraxonrasib, evaluates supporting clinical data, and discusses its strengths, limitations, and future positioning in managing RAS-mutated PDAC.

Keywords: Ductal adenocarcinoma; daraxonrasib; RAS-mutated pancreatic cancer.

1. Introduction

The most prevalent form of pancreatic cancer is pancreatic ductal adenocarcinoma (PDAC) which makes up about 90% of all pancreatic malignancies [1]. Although PDAC is a relatively rare type of cancer globally, it accounts for a significant percentage of cancer deaths, due in part to its aggressive nature and high mortality. The disease is sometimes referred to as a ‘silent cancer' as the symptoms of the disease – including abdominal pain, weight loss, jaundice and fatigue – tend to only occur when the tumour is at a more advanced stage. Therefore, when a patient finally becomes diagnosed, there is often little opportunity for long-term survival because the patient has become too far advanced for the surgery to be performed.

The treatment for pancreatic cancer depends on the stage of the disease. Surgical resection plus chemotherapy provides the greatest chance of cure for those patients who have localised tumours. However, surgery is only an option for a few at diagnosis. The majority of patients have locally advanced or metastatic disease and receive systemic chemotherapy (e.g. FOLFIRINOX or gemcitabine + nab-paclitaxel). Many patients will become resistant to these treatments and although they may slow the progress of tumour and provide relief from symptoms for a while, the effects of these treatments are not long lasting. Thus, the 5-year overall survival rate for patients with PDAC remains low, highlighting the need for more effective therapeutic approaches [1].

Pancreatic cancer is a complex disease for which it is difficult to treat patients successfully. The PDAC is defined by its aggressive nature with features of tumor growth, intense fibrosis around the tumor, early invasion of neighboring tissue and highly immunosuppressive tumor microenvironment. These properties will accelerate the disease process but also hamper the efficacy of chemotherapy and numerous types of immunotherapy. As a result the research has been mainly directed towards understanding the molecular pathways that are involved in tumour initiation and progression in order to find more precise and targeted therapy [1,2].

There are many mutations found in the genes of people with pancreatic cancer, but the most common is a mutation in the KRAS gene. The activating KRAS mutation is one of the earliest and most important events in pancreatic tumour development, and is found in ~90-95% of PDAC tumours. The KRAS protein is a member of a family of small guanosine triphosphatase (GTPase) proteins that play a key role in controlling a variety of critical cellular functions such as proliferation, differentiation, metabolism and survival. In normal physiology, KRAS switches between an inactive guanosine diphosphate (GDP)-bound form and an active guanosine triphosphate (GTP)-bound form, depending on signals received from cell surface receptors.

Mutations in KRAS upset this well-balanced cycle by disrupting the ability of the protein to efficiently hydrolyse GTP to GDP. This leaves KRAS stuck in an active state all the time, constantly on and stimulating other pathways downstream of it, such as the RAF–MEK–ERK pathway and the PI3K–AKT–mTOR pathway. These pathways are constantly activated and lead to uncontrolled cell growth, inhibition of programmed cell death, metabolic reprogramming and tumour initiation and progression. Furthermore, mutant KRAS can modify the tumour microenvironment by promoting inflammation, fibrosis and immune evasion, which are aspects of pancreatic cancer's overwhelming treatment resistance [3,4].

KRAS has long been known as a good target for drug development, but developing drugs to directly inhibit the protein has been extremely challenging. KRAS does not have the same deep binding pockets that are normally targeted by small molecule drugs, so it was dubbed 'undruggable' for many years. More recently, KRAS G12C specific inhibitors have been shown to have clinical efficacy in lung cancer. These mutations of G12C are not often found in pancreatic cancer, however, in pancreatic cancer G12D, G12V, and G12R mutations are much more common. Thus, there is an unmet need for therapies that are able to recognize the multiple KRAS mutation variants instead of one particular mutation [5,6].

The development of targeted drugs against RAS has come a long way in recent years and the first-in-class multiselective RAS(ON) inhibitor daraxonrasib (RMC-6236) was recently discovered to suppress RAS(ON) signalling from multiple active RAS variants. Unlike previous mutation-specific inhibitors, daraxonrasib could be a tool for a wider patient population with pancreatic cancer that is RAS-mutated. While there were some promising early laboratory evidence and clinical trials, the largest and most important trial was the Phase III RASolute 302 that showed substantial improvements in survival rates over standard chemotherapy. This review explores the biology of KRAS-induced pancreatic cancer, the mechanism of action of daraxonrasib, the current clinical data for its use, and the future prospects for its involvement in the treatment of RAS-mutated pancreatic cancer.

2. Daraxonrasib

2.1. Development of Daraxonrasib

The targeting of KRAS has been one of the biggest hurdles in the development of cancer drugs for many years. KRAS is a relatively smooth molecule with very few pocket sites that would be good targets for existing small molecule drugs, unlike many proteins that are involved in cancer progression. Moreover, KRAS has a very tight binding to guanosine triphosphate (GTP) which makes it challenging for inhibitors to compete with the natural ligand. So, KRAS was considered an 'undruggable' protein and researchers turned their attention to the downstream signalling pathways (such as RAF, MEK and PI3K). These strategies were seen to have some efficacy in lab studies but in general had only a limited clinical effect as the cancer cells could discover alternate signalling pathways and build resistance to them.

Targeted cancer therapy was given a significant boost with the development of mutation-specific inhibitors called sotorasib and adagrasib. These agents are selective KRAS G12C inhibitors, and have shown clinical activity in non-small cell lung cancer. However, mutations other than KRAS G12C,

like G12D, G12V, and G12R, occur much more frequently, and cause the majority of pancreatic cancers. Thus, these mutation-specific drugs do not work for most patients who have PDAC. The restriction led the researchers to look for alternative strategies to target multiple RAS mutations instead of targeting one genetic subtype.

This is where a new system, one that is designed to overcome this challenge, comes in: Daraxonrasib (RMC-6236). Daraxonrasib was not intended to target a particular mutation in the KRAS gene but rather target multiple RAS proteins that are active. After a long process of medicinal chemistry optimisation, the compound was found by Cregg et al.to have improved selectivity and potency, as well as to be orally bioavailable and to have an improved pharmacokinetic profile [7]. These properties enable the drug to be effective in the body and minimize unwanted side effects. Other benefits associated with daraxonrasib are: It is an oral therapy, which allows for convenient patient access to treatment, as opposed to some intravenous therapies that require more frequent hospital visits.

2.2. The mechanism of Daraxonrasib

Daraxonrasib is a therapeutic agent that binds to the GTP-bound (active) state of a mutated RAS protein, called RAS(ON), which is different from the GTP-bound RAS proteins that were targeted by other RAS inhibitors. This difference is especially significant as many oncogenic KRAS mutations are locked in an active state and are constantly sending growth signals to encourage tumour growth. Daraxonrasib directly inhibits active RAS proteins, allowing it to inhibit signalling in multiple KRAS mutation subtypes.

Daraxonrasib does this by virtue of forming a ternary complex between the drug, cyclophilin A and the active form of the RAS protein. The cyclophilin A is a natural, intracellular binding protein which plays a molecular partner role in the binding process of the drug. Instead, daraxonrasib doesn't block the RAS protein itself; it first binds to the cyclophilin A protein, which then binds to active RAS. This interaction keeps RAS unable to bind to its effector proteins downstream of RAS, such as the RAF kinases, which disrupts signalling through the MAPK pathway. In parallel, blocking the activity of RAS also inhibits activation of the PI3K–AKT–mTOR pathway, a key pathway for cell survival and metabolism. Once these signalling pathways are turned off, the cancer cells will no longer grow efficiently and tumour growth will be much harder.

The biggest benefit of this process is that it does not depend on the type of KRAS mutation occurring in the tumour. Daraxonrasib is active against cancers with KRAS mutations G12D, G12V, G12R and G13 as well as various NRAS and HRAS mutations in preclinical studies due to its ability to target the active conformation shared by multiple RAS variants. This is a wider activity compared to approved mutation-specific inhibitors, and vastly increases the number of patients that could potentially receive targeted RAS therapy.

2.3. Potential Advantages

Daraxonrasib heralds a new era in the therapy of RAS mutated cancers as it is able to overcome several drawbacks of conventional therapies. Chemotherapy is the traditional treatment for advanced pancreatic cancer, but it is toxic to healthy and cancerous cells, and offers only slight increases in survival. In addition, chemotherapy does not specifically target the genetic defects that promote the growth of tumour cells and resistant cells can survive and then lead to the disease worsening.

Direct targeting of KRAS has been shown to be clinically feasible with mutation specific inhibitors, but their application to pancreatic cancer is restricted as they are only effective in specific mutations, primarily KRAS G12C. In contrast, daraxonrasib is mutation agnostic, blocking the active form of multiple RAS proteins. The increased breadth of activity could also have a positive impact on a much larger subset of patients who have pancreatic cancer, in which non-G12C mutations are more common. Preclinical studies have also indicated that multiselective RAS inhibition may decrease the risk of

escaping tumour cells by alternative RAS signalling pathways, but long-term clinical studies are necessary to confirm the potential possibility of this [7,8].

Though daraxonrasib is a promising technology, it should not be considered as a cure for pancreatic cancer. Like all targeted therapies resistance can develop over time, perhaps from other genetic changes or with the activation of another compensatory signalling pathway. For this reason, many scientists surmise that daraxonrasib could best be used in conjunction with some of these other targeted drugs, chemotherapies, or immunotherapies. However, due to its novel mechanism of action and promising early clinical results, daraxonrasib stands out as one of the most promising advances in the treatment of pancreatic cancer in recent years.

3. Clinical Evidence

3.1. Early Research

Before testing in patients, researchers had to find out if it could also inhibit the RAS signalling pathway and inhibit the growth of tumours in laboratory models. Preclinical studies are crucial during the development of a drug as they give evidence of the biological activity, safety and potential therapeutic benefits of a drug prior to the first man or woman trial. These studies were on its inhibition of multiple RAS mutations that are prevalent in pancreatic and other solid cancers, which is what is important for daraxonrasib.

Laboratory studies with cultured tumour cells indicated that daraxonrasib is able to inhibit the activation of both the RAF–MEK–ERK and the PI3K–AKT–mTOR pathways, both of which are important in tumour growth, in a highly effective manner. As these signalling pathways got turned off, the proliferation of cancer cells slowed down and programmed cell death accelerated. Importantly, the drug was active not just in one type of KRAS mutation but in multiple subtypes; allowing it to be considered for development as a “broad-spectrum RAS inhibitor”.

Encouraging results also were observed in animal studies. Daraxonrasib significantly reduced tumour growth and, in some mice, resulted in tumour regression, in mouse models with RAS-mutated tumours. Such responses were seen in various forms of cancer, including pancreatic cancer, and could thus be used as a therapeutic strategy in a number of tumour types. They further discovered that daraxonrasib possessed favourable pharmacokinetic parameters: it was well absorbed from the gut and maintained high drug exposures for a long time, allowing for effective inhibition of RAS signalling over time. All of these studies showed that daraxonrasib possessed enough biological activity and safety to warrant clinical trials in humans.

3.2. Phase III Clinical Trial

Daraxonrasib was taken into clinical development after successful pre-clinical studies to assess its safety and efficacy in patients with advanced RAS-mutated cancers. The main goal of the early Phase I studies was to establish a safe dose level and to track any adverse effects from the treatment and assess early anti-tumour activity. A large number of patients who participated in these trials had already tried numerous other treatments and had very little other treatment options left. In spite of this tough patient population, daraxonrasib showed promising clinical activity with a significant fraction of patients experiencing either tumour shrinkage or disease stability. Initial results indicated clinical value of multiselective inhibition of active RAS and informed the continuation of the trials to larger randomized trials.

The best proof for daraxonrasib currently is from the international Phase III RASolute 302 trial by O'Reilly [6]. This open-label, randomised study involved patients with metastatic PDAC with RAS mutations who had previously received chemotherapy, and compared daraxonrasib with investigator's choice of standard chemotherapy. Participants had already progressed the disease after the first line of treatment, which means they were a patient population who had few treatment options and in general poor prognosis.

The trial showed adherence to treatment to be significantly better on all key measures of treatment effectiveness. The median overall survival for patients who received daraxonrasib was 13.2 months, nearly double the median overall survival for patients who received chemotherapy (6.7 months). Median progression-free survival was significantly longer with daraxonrasib than with chemotherapy (7.2 months vs. 3.6 months, respectively; hazard ratio, 0.49; 95% CI, 0.38–0.63; P<0.001). Moreover, there was a higher objective response (31.6% vs 11.2% for chemotherapy) suggesting a greater tumour burden reduction effect of daraxonrasib. Importantly, these benefits were seen in multiple subsets of patients with different KRAS mutations, supporting the drug's activity in a broad population of patients with various mutations.

In addition to increasing patients survival, daraxonrasib also slowed the progression of the disease and allowed patients to maintain their quality of life. People taking the drug had a longer delay before any cancer symptoms, such as pain, became worse than they were when they first started taking the drug, compared with those taking chemotherapy. This discovery is particularly significant because at times symptoms of advanced pancreatic cancer are very intense, having a significant impact on daily life and well-being. Thus, the advantages of daraxonrasib are not only survival, but also patient-centred outcomes which are very relevant in the clinical settings.

Overall, the findings from the RASolute 302 study are considered one of the biggest breakthroughs in the treatment of pancreatic cancer in recent years. Treatment with second-line chemotherapy has offered only marginal advances in overall survival, and has been associated with significant treatment toxicities. Daraxonrasib, on the other hand, had improved clinical responses without a high incidence of adverse effects, and thus multiselective inhibition of active RAS may be a key strategy to treat patients with metastatic pancreatic cancer in the near future.

3.3. Safety Evaluation

A key question that arises when a new anti-cancer drug is developed is whether the drug's pros outweigh its cons. Daraxonrasib was found to be associated with adverse events related to the treatment, but the safety profile seen during clinical trials was deemed manageable. Common side-effects were rash, diarrhoea, stomatitis, nausea and vomiting. The overall severity of these events was mostly mild to moderate, and supportive care, temporary dose interruption or dose reduction often successfully managed these events. These side effects are also consistent with the mechanism of action of the drug, which is also involved in normal cell function, especially of the gastrointestinal tract and the skin.

Daraxonrasib showed a number of important safety benefits over conventional chemotherapy. Severe haematological toxicity (neutropenia and thrombocytopenia) was less common than amongst chemotherapy-treated patients. Moreover, though patients were on treatment for a longer period of time, relatively few withdrew from treatment due to adverse events. Overall, the results indicated that daraxonrasib was well tolerated and patients were able to treat themselves for long enough to gain any clinical benefit.

Although these encouraging results are derived, there are certain limitations. Phase III trial participants most commonly had KRAS G12 mutations and further research will be needed to determine if similar results can be seen in those with less prevalent RAS mutations. In addition, the long-term safety of daraxonrasib is still not well established as it is a relatively new drug. Like all targeted drugs, resistance can also develop over time, via further genetic changes or via other signalling pathways being activated. Further studies should thus explore combination treatment approaches and determine biomarkers which can be used to identify patients who are most likely to benefit from treatment [9,10].

4. Discussion

4.1. Comparison with the Traditional Treatments

The results of the comparisons with the traditional treatments described below are presented in 4.1.

Systemic chemotherapy has been the mainstay of therapy for many years in the case of advanced PDAC. First line treatments such as FOLFIRINOX and gemcitabine plus nab-paclitaxel may extend survival and improve symptoms, but may not be effective long-term as most tumours become resistant to treatment. Chemotherapy also damages healthy cells in the body, which can cause tiredness, make it easy to get sick, make bones weak, cause peripheral neuropathy (a loss of sensation in the hands and feet), and lower resistance against infections. These toxicities can impact patient's quality of life, and do frequently, if not routinely, necessitate dose adjustment or treatment cessation.

The identification of the first mutation-specific KRAS inhibitors (sotorasib and adagrasib) was a significant step forward in targeted therapy, showing that it is possible to directly target and inhibit KRAS. These medications, however, are targeted toward the KRAS G12C mutation which is prevalent in non-small cell lung cancer but is rarely found in pancreatic cancer. Thus, only a minority of patients with PDAC can benefit from these therapies.

A major drawback to this is that it only targets an individual form of multiple RAS proteins, whereas Daraxonrasib targets multiple RAS proteins. As mentioned in the previous sections, the Phase III RASolute 302 trial has shown that it gave better results in terms of overall survival, progression-free survival, tumour response and quality of life than conventional second-line chemotherapy. The results indicate that combination therapy with multi-selective RAS inhibitors could be a better treatment approach for most pancreatic cancer patients with RAS mutations. However, chemotherapy is likely to continue to play a key role in treatment as it can be used in combination with targeted treatment to achieve the best therapeutic results [6,7].

4.2. Limitation

The clinical results achieved with daraxonrasib are promising, but there are some restrictions that need to be taken into account in interpreting the data presented so far. Firstly, since Daraxonrasib is a relatively new drug, long-term follow-up data are limited. The Phase III trial showed there were definite survival benefits, but further trials are required to find out if these can be sustained over the longer term, and if there are any late effects to be expected during long-term treatment.

Secondly, most of the patients who entered the clinical trials in the current study were the ones with KRAS G12 mutations. It is therefore unclear if it is effective in patients who have less common RAS variants. Future, larger studies of greater genetic diversity among patients will be needed to see if the clinical benefits seen are applicable to all RAS-mutated cancers.

One of the big issues is the development of acquired drug resistance. The genetic changes that occur in cancer cells as a consequence of treatment are ongoing, and may even result in further genetic mutations that re-activate downstream signalling pathways or completely overcome the effect of RAS inhibition. Many targeted therapies have been seen to become resistant and it is likely this will be a major challenge for daraxonrasib. To be able to improve long-term treatment outcomes and to develop more effective therapeutic strategies, knowledge of these molecular mechanisms will be important.

4.3. Future Perspectives

Daraxonrasib has now shown some fruitful avenues of future research. Combination therapy is one of the salient points. The combination of daraxonrasib with chemotherapy, immunotherapy or inhibitors of parallel signalling pathways may enhance the effectiveness of treatment and help to prevent the emergence of drug resistance, as pancreatic cancer involves multiple signalling pathways and has a very complex microenvironment.

An early use of daraxonrasib in the course of disease is another promising direction. One of the main type of current clinical evidence are patients with metastatic pancreatic cancer who had been treated with a previous systemic therapy. Further studies in the future are warranted to examine if a first-line treatment with daraxonrasib or treatment as adjunct therapy after surgical resection of the tumor may further improve survival outcomes.

Aside from pancreatic cancer, mutations in RAS are commonly found in other solid tumours, such as colorectal cancer, non-small cell lung cancer, melanoma and many others. Due to its specific action on multiple activated RAS variants, daraxonrasib could be applicable to other types of cancer. Routine and future clinical trials will further help to identify the patients who might benefit from the drug treatment and its efficiency in various tumour types.

Daraxonrasib is more than a new anti-cancer product, it is a change. This shows that targeting active RAS proteins is a therapeutic approach that can be done and adds to the evidence of precision medicine's continued progress. New drugs, like daraxonrasib, which are multiselective inhibitors of RAS, are likely to play a major part in the treatment of cancer in the near future as scientists better understand the biology of the tumour and the mechanisms of resistance to existing drugs.

5. Conclusion

The difficult nature of the disease, combined with late diagnosis, the frequent presence of KRAS mutations and the aggressiveness of the disease, make PDAC one of the most difficult cancers to treat and manage. The RAS protein's unique biological properties for decades made the development of effective therapies for KRAS a challenge. The advent of daraxonrasib has altered this situation by providing a multiselective RAS(ON) inhibitor that is able to target multiple activated RAS variants, not just a single mutation subtype.

Laboratory studies have provided evidence of daraxonrasib's ability to inhibit key tumour growth pathways, and clinical trials have demonstrated that daraxonrasib has an impact on patient quality of life and significantly improved overall survival, progression-free survival and tumour response compared to standard second line chemotherapy. Based on these results, daraxonrasib is one of the most promising novel targeted therapeutics in the past few years for Ras mutated pancreatic cancer.

While there are still challenges to address, including the issue of acquired drug resistance and the lack of long-term clinical data, the available evidence suggests that daraxonrasib holds promise for revolutionizing the treatment of advanced pancreatic cancer. Its potential full clinical value will be further clarified through future studies of combination therapies, earlier treatment strategies and other cancers with RAS mutations. Overall, daraxonrasib is a promising advancement towards precision medicine in modern oncology and more effective and precise treatment of pancreatic cancer patients.

References

[1] J.P. Neoptolemos, J. Kleeff, P. Michl, E. Costello, W. Greenhalf, D.H. Palmer, Therapeutic developments in pancreatic cancer: current and future perspectives, Nat. Rev. Gastroenterol. Hepatol. 15 (2018) 333–348. https://doi.org/10.1038/s41575-018-0005-x.

[2] R.L. Siegel, T.B. Kratzer, A.N. Giaquinto, H. Sung, A. Jemal, Cancer statistics, 2025, CA Cancer J. Clin. 75 (2025) 10–45. https://doi.org/10.3322/caac.21871.

[3] M.B. Ryan, R.B. Corcoran, Therapeutic strategies to target RAS-mutant cancers, Nat. Rev. Clin. Oncol. 15 (2018) 709–720. https://doi.org/10.1038/s41571-018-0105-0.

[4] A.M. Waters, C.J. Der, KRAS: The critical driver and therapeutic target for pancreatic cancer, Cold Spring Harb. Perspect. Med. 8 (2018) a031435. https://doi.org/10.1101/cshperspect.a031435.

[5] M. Holderfield, B.J. Lee, J. Jiang, A. Tomlinson, K.J. Seamon, A. Mira, E. Patrucco, G. Goodhart, J. Dilly, Y. Gindin, N. Dinglasan, Y. Wang, L.P. Lai, S. Cai, L. Jiang, N. Nasholm, N. Shifrin, C. Blaj, H. Shah, M. Singh, Concurrent inhibition of oncogenic and wild-type RAS-GTP for cancer therapy, Nature 629 (2024) 919–926. https://doi.org/10.1038/s41586-024-07205-6.

[6] E.M. O'Reilly, Z.A. Wainberg, A.E. Hendifar, M.J. Borad, F. Pietrantonio, S. Pant, P. Hammel, C. Cremolini, G.A. Manji, P.E. Oberstein, I. Garrido-Laguna, C. Springfeld, N.S. Azad, M. Ueno, S.Y. Chui, Y. Zhang, H. Patel, Y. Lee, Z. Salman, B.M. Wolpin, Daraxonrasib or chemotherapy in previously treated metastatic pancreatic cancer, N. Engl. J. Med. (2026). https://doi.org/10.1056/NEJMoa2605555.

[7] J. Cregg, A.V. Edwards, S. Chang, B.J. Lee, J.E. Knox, A.C.A. Tomlinson, A. Marquez, Y. Liu, R. Freilich, N. Aay, Y. Wang, L. Jiang, J. Jiang, Z. Wang, M. Flagella, D. Wildes, J.A. Smith, M. Singh, Z. Wang, A.L. Gill, Discovery of Daraxonrasib (RMC-6236), a potent and orally bioavailable RAS(ON) multi-selective, noncovalent tri-complex inhibitor for the treatment of patients with multiple RAS-addicted cancers, J. Med. Chem. 68 (2025) 6064–6083. https://doi.org/10.1021/acs.jmedchem.4c02314.

[8] J. Jiang, L. Jiang, B.J. Maldonato, Y. Wang, M. Holderfield, I. Aronchik, I.P. Winters, Z. Salman, C. Blaj, M. Menard, J. Brodbeck, Z. Chen, X. Wei, M.J. Rosen, Y. Gindin, B.J. Lee, J.W. Evans, S. Chang, Z. Wang, M. Singh, Translational and therapeutic evaluation of RAS-GTP inhibition by RMC-6236 in RAS-driven cancers, Cancer Discov. 14 (2024) 994–1017. https://doi.org/10.1158/2159-8290.CD-24-0027.

[9] J. Lokhandwala, T.B. Smalley, T.H. Tran, Structural perspectives on recent breakthrough efforts toward direct drugging of RAS and acquired resistance, Front. Oncol. 14 (2024) 1394702. https://doi.org/10.3389/fonc.2024.1394702.

[10] A. Petzold, T. Steeb, A. Wessely, E.A.T. Koch, J. Vera, C. Berking, M.V. Heppt, Is tebentafusp superior to combined immune checkpoint blockade and other systemic treatments in metastatic uveal melanoma? A comparative efficacy analysis with population adjustment, Cancer Treat. Rev. 115 (2023) 102543. https://doi.org/10.1016/j.ctrv.2023.102543.