February 12, 2026

To Shareholders,

Company Name: Renascience Inc.
Representative: Toshio Miyata, Chairman and CEO
(Code: 4889 TSE Growth)
For inquiries, please contact Administration Dept.

Announcement of Additional Grant for Program for Promoting Platform for Drug Discovery and Medical Technology Development - Project for Implementation of Innovative Cancer Medicine from the Japan Agency for Medical Research and Innovation

In March 2022, we began conducting a Phase III clinical trial (investigator-initiated clinical trial) with support from the Japan Agency for Medical Research and Development (AMED)'s "Promoting Platform for Drug Discovery and Medical Technology Development - Project for Implementation of Innovative Cancer Medicine" (representative institution: Tohoku University, with our company as a contributing institution). The trial began on August 3, 2022 and 57 cases had been enrolled by the end of December 2023, exceeding the number of cases required for analysis. As a result of the final year evaluation of AMED in December 2024, an extension of the grant period was approved, as the target number of cases for the Phase III trial had been enrolled (disclosed on December 3, 2024). We are pleased to announce that, as the trial is progressing smoothly as planned, we have been selected to receive additional funding from the AMED for the fiscal year ending March 2027.

[AMED Project]

Project Name: Promoting Platform for Drug Discovery and Medical Technology Development - Project for Implementation of Innovative Cancer Medicine
Area: 3-2 Investigator-Initiated Clinical Trial Aiming for the Development and Regulatory Approval of Innovative Cancer Treatment Drugs
Research Project Name: Phase III Study to Evaluate the Safety and Efficacy of TM5614 (RS5614) in Long-Term Combination with TKIs in Chronic Myeloid Leukemia (Principal Investigator: Professor Hideo Harigae, Tohoku University)
Grant Amount: 36 million yen (excluding indirect expenses)

[Challenges of Chronic Myeloid Leukemia]

Chronic myeloid leukemia (CML), a type of blood cancer, develops when genetic abnormalities occur in the cells that give rise to blood cells (hematopoietic stem cells), leading to the

uncontrolled proliferation of cancerous leukemia cells (CML cells). The mainstay of treatment for CML is molecularly targeted tyrosine kinase inhibitors (TKIs), such as imatinib. The development of TKIs has significantly improved the survival rate of CML patients. However, while TKIs act on CML cells, they do not affect the cells that give rise to CML cells (i.e., CML stem cells) that reside in a region of the bone marrow known as the "bone marrow niche." Therefore, discontinuing TKI therapy can result in relapse by transforming into CML cells. Therefore, long-term, expensive TKI therapy is required to cure CML, and side effects are also problematic. Therefore, it is important to achieve a cure that does not require TKI therapy as soon as possible.

Recently, it has been shown that CML patients who maintain a deep molecular response (DMR)[1)] for a certain period of time can achieve treatment-free remission even after discontinuing TKI therapy. However, only 5-10% of patients achieve treatment-free remission within the minimum treatment period of three years. To achieve treatment-free remission, DMR must be maintained for at least two years after achieving DMR. Our plasminogen activator inhibitor (PAI)-1 inhibitor (RS5614) acts on CML stem cells, causing them to be released from the bone marrow niche. The released CML stem cells are then affected by TKIs, eliminating them from the bone marrow niche, demonstrating the potential for a complete cure of CML. In fact, when RS5614 was administered in combination with a TKI in CML model mice, the number of CML stem cells remaining in the bone marrow was significantly reduced compared to administration of the TKI alone, significantly improving survival rates.

When used in combination with a TKI, RS5614 is expected to be a safe drug with a new mechanism of action that will quickly lead to a complete cure for many CML patients, eliminating the need for drug treatment.

[Clinical Trial of PAI-1 Inhibitors]

Late-stage Phase II Trial

In combination with TKIs, RS5614 achieved a DMR rate of 33.3% (11 of 33 patients achieved DMR) at 48 weeks after the start of treatment, a significant increase compared to historical controls using TKIs alone (8-12%). Notably, the cumulative DMR rate reached 50.0% in patients with a TKI treatment duration of 3 to 5 years. Furthermore, no serious adverse events causally related to the treatment were observed even after one year of long-term administration of RS5614. These trial results were published in the scientific journal ‘Cancer Medicine’.

Phase III Trial

In collaboration with 12 universities and medical institutions, including Tohoku University, Tokai University, and Akita University, we are currently conducting a placebo-controlled, double-blind,

investigator-initiated Phase III clinical trial to evaluate the efficacy of the combination of TKIs and RS5614 in patients with chronic phase CML. A clinical trial plan was submitted to the PMDA in May 2022, and a multicenter Phase III trial has begun. The trial enrolled 60 patients with chronic phase CML who had been on TKI treatment for at least three but less than six years. The trial is verifying whether the combination therapy with the investigational drug RS5614 significantly increased the rate of DMR maintenance for at least two years, an indicator of treatment-free remission, compared with the TKI monotherapy group.

[Impact on Financial Results]

We plan to incorporate this event into our full-year financial forecast for the fiscal year ending March 2027.

1) DMR

Deep molecular response (DMR) is a status which the causative gene for the cancer is no longer detectable. Current chronic phase CML treatment requires lifelong administration of expensive TKIs. However, recent evidence has shown that some patients who achieve and maintain DMR for a certain period of time are able to maintain treatment-free remission even after discontinuing TKIs.

2) Double-blind trial: This is a clinical trial method in which patients are randomly assigned to one group receiving the investigational drug and the other receiving a control drug (placebo), and both groups are simultaneously administered the drug without the doctor or patient knowing which group they are receiving. This trial method is intended to avoid the risk of doctors intentionally administering the investigational drug to patients in whom the drug is expected to be effective, the possibility of preconceived notions that the drug should be effective being reflected in the evaluation, and the possibility that patients' reactions to the treatment or their evaluations may be affected if they become aware of the treatment.