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**GenFleet Therapeutics (Shanghai) Inc.**

**劲方医药科技(上海)股份有限公司**

*(A joint stock company incorporated in the People's Republic of China with limited liability)*

**(Stock Code: 2595)**

**VOLUNTARY ANNOUNCEMENT**

**Initial Clinical Readout of GFH276 (an Oral Molecular-glue Pan RAS Inhibitor) Presented as Late-breaking Abstract (LBA) at 2026 ESMO Annual Meeting, Demonstrating Promising Clinical Potential and Favorable Safety in Pretreated RAS-mutant Solid Tumors**

This announcement is made by GenFleet Therapeutics (Shanghai) Inc. (the “**Company**” or “**GenFleet**”, together with its subsidiaries, the “**Group**”) on a voluntary basis to inform the shareholders and potential investors of the Company about the latest business updates of the Group.

The board of directors of the Company (the “**Board**”) is pleased to announce that the preliminary study data of GFH276 (an oral molecular-glue Pan RAS inhibitor) in pretreated RAS-mutant solid tumors were featured in a late-breaking abstract through an on-site oral presentation at ESMO (European Society for Medical Oncology) annual meeting on October 25 (local time). GFH276 already demonstrated encouraging preliminary efficacy and a favorable safety profile in dose-escalation, with partial responses (PR) of patients observed starting from the 40 mg QD dose level; no dose-limiting toxicities (DLTs) were observed across 40-160 mg QD dose levels when patients did not receive prophylactic medications.

72.6% of pancreatic ductal adenocarcinoma (PDAC) patients and 61.1% of non-small cell lung cancer (NSCLC) patients received GFH276 monotherapy as the third- or later-line of therapy post enrollment in phase Ia trial. As of data cutoff date, the overall objective response rate (ORR) was 41.1% among 61 evaluable PDAC patients (40-160 mg QD), and 43.1% among RAS inhibitor naive PDAC patients; the overall ORR was 45.5% among 11 evaluable NSCLC patients (80-160 mg QD), and 55.6% among RAS inhibitor naive NSCLC patients.

“This is the initial readout of GFH276’s first-in-human study data presented at a global academic conference. The preliminary result demonstrated robust efficacy and a wide

therapeutic window, supporting the advancement of mid-to-late-stage clinical studies of GFH276 as monotherapy and in combination therapies, including the combination with an EGFR antibody or with chemotherapy. The approval of the overseas product addressing the same target validates the clinical value and market potential of Pan RAS inhibitors. Our pipeline comprises a launched KRAS G12C inhibitor, a KRAS G12D inhibitor with leading clinical progress as a monotherapy in global landscape, and a RAS-inhibiting matrix spanning diverse targets, molecular modalities, and regimens. We look forward to bringing more treatment options to patients with RAS-mutant tumors through innovative monotherapy and combination regimens.” stated Yu Wang, M.D., Ph.D., Chief Medical Officer of GenFleet.

**LBA33: Safety and Efficacy of GFH276 Monotherapy in Previously Treated RAS-mutated Advanced Solid Tumors**

**Presenter: Dr. Yuxiang Ma, Sun Yat-sen University Cancer Center**

The phase Ia dose-escalation trial of GFH276 monotherapy study (NCT07198321) enrolled patients across diverse RAS mutation subtypes, with major tumor types covering PDAC, NSCLC, and colorectal cancer (CRC), across dose levels of 5-320 mg QD of monotherapy. As of data cutoff date, 78.1% of PDAC patients had liver metastases, 31.5% had peritoneal metastases, and 16.4% had lung metastases upon enrollment; 77.8% of NSCLC patients had intrapulmonary metastases, 72.2% had bone metastases, and 38.9% had brain metastases upon enrollment. More than 90% of PDAC and NSCLC patients, respectively, had received chemotherapy prior to enrollment; over 80% of NSCLC patients had received immunotherapy prior to enrollment.

| Baseline characteristics of patients enrolled as of data cutoff date |                     |      |            |                     |                               |                |
|----------------------------------------------------------------------|---------------------|------|------------|---------------------|-------------------------------|----------------|
| Cancer types                                                         | KRAS G12X mutations |      |            | Other RAS mutations | Therapies prior to enrollment |                |
|                                                                      | G12D                | G12V | Other G12X |                     | Chemo-therapy                 | Immuno-therapy |
| PDAC (73 patients)                                                   | 40%                 | 35%  | G12R 11%   | 14%                 | 98.6%                         | 32.9%          |
| NSCLC (18 patients)                                                  | 34%                 | 22%  | G12C 11%   | 33%                 | 94.4%                         | 83.3%          |

As of September 15, 2026, 61 PDAC patients (40-160 mg QD) had completed at least one post-treatment tumor assessment, with an overall ORR of 41.1% and a disease control rate (DCR) of 91.8%; 11 NSCLC patients (80-160 mg QD) had completed at least one post-treatment tumor assessment, with an overall ORR of 45.5% and a DCR of 72.7%. The study data demonstrated favorable pharmacokinetic characteristics of GFH276, with no significant accumulation observed, thereby showing preliminary clinical efficacy consistent with the anti-tumor activity in preclinical single-agent animal tests. Additionally, clinical data demonstrated wide therapeutic window of

GFH276, which was also consistent with preclinical findings - animal models across tumor types indicated synergistic effects in combination of GFH276 with standard-of-care regimens (including EGFR antibody, chemotherapy or immunotherapy), thus providing rationale for clinical combinational strategies.

As of July 15, 2026, a total of 80 patients treated with GFH276 monotherapy at 40-160 mg QD showed a favorable safety/tolerability profile: most treatment-related adverse events (TRAEs) were grade 1-2, and the most common TRAEs included the category of rash (including rash, dermatitis acneiform, rash maculopapular, erythema nodosum, dermatitis exfoliative generalized, rash pustular, drug eruption and genital rash), anemia, stomatitis (including stomatitis and mouth ulceration) and diarrhea. Grade 3 TRAEs mainly fell into the category of rash (7.5%), and each of other grade 3 TRAEs had an incidence of below 4%. TRAEs were generally manageable and there were no grade 4-5 TRAEs. GFH276 has been granted clinical trial approvals for programs to investigate various combinational regimens across indications including PDAC, CRC, and NSCLC in first-line or late-line settings.

### **About RAS Proteins and GFH276**

RAS proteins, in active GTP-bound or inactive GDP-bound form, are binary molecular switches controlling cellular responses in signaling pathways including RAF-MEK-ERK and PI3K-AKT mTOR. Three RAS genes encode for protein isoforms, namely Kirsten Ras (KRAS), Harvey Ras (HRAS) and Neuroblastoma Ras (NRAS), and KRAS is the most frequent RAS mutation in humans.

GFH276 is an oral novel small-molecule Pan RAS (ON) inhibitor employing the tri-complex mechanism (Cyp A-GFH276-RAS) to target active RAS proteins of most wild/mutant subtypes, including most commonly found KRAS mutant (G12C, G12D, G12V, etc.) proteins, as well as NRAS and HRAS isoforms. Preclinical research of GFH276 demonstrates dose-dependent anti-tumor activity and drives tumor regression in multiple KRAS mutant tumor models. GFH276 also holds the potential to outperform SIIP (switch II pocket)-based KRAS inhibitors in overcoming adaptive and acquired resistance

### **Forward-looking Statements**

Specific information in this announcement may contain or constitute forward-looking words that are not historical facts. They can be identified by using forward-looking terminology, such as “predict”, “believe”, “plan”, “forecast”, “expect”, “will”, “may”, “should” and other words of similar meanings. Based on the management’s current beliefs, plans, estimates and expectations of the Company’s operation and market trends subject to changes beyond control, the forward-looking terminology reflects the Company’s beliefs, plans, estimates and expectations of future development. Actual outcome in the future may differ significantly from forward-looking words owing to market, policy, and R&D uncertainties, among others. Subject to the above-mentioned

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## **GLOSSARY OF TECHNICAL TERMS**

|         |                                                                                                                                                                                                                             |
|---------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| “AKT”   | a serine/threonine protein kinase with three isoforms (AKT1, AKT2 and AKT3) that participate in multiple pathways regulating several cellular processes, including survival, proliferation, tissue invasion, and metabolism |
| “CRC”   | colorectal cancer                                                                                                                                                                                                           |
| “Cyp A” | cyclophilin A is a type of peptidyl prolyl cis-trans isomerase and is broadly expressed and abundant inside cells                                                                                                           |
| “DCR”   | disease control rate                                                                                                                                                                                                        |
| “DLT”   | dose-limiting toxicities                                                                                                                                                                                                    |
| “ERK”   | extracellular signal-regulated kinase, a key protein in the mitogen-activated protein kinase signaling pathway                                                                                                              |
| “EGFR”  | epidermal growth factor receptor                                                                                                                                                                                            |
| “GDP”   | guanosine diphosphate is the precursor or resulted from hydrolysis of GTP and is involved in cell metabolism and signal transduction (e.g., inactivating downstream pathway via binding to RAS proteins)                    |
| “GTP”   | guanosine triphosphate serves as an energy carrier and is involved in protein synthesis and signal transduction upon                                                                                                        |

its hydrolysis and binding (e.g., activating downstream pathway by binding to RAS

|           |                                                                                                                                                                       |
|-----------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| “HRAS”    | Harvey RAS, a member of the RAS family proteins                                                                                                                       |
| “KRAS”    | Kirsten RAS, a member of the RAS family of proteins                                                                                                                   |
| “MEK”     | MAPK/ERK, a key protein kinase that plays a crucial role in the MAPK signaling pathway                                                                                |
| “mTOR”    | a protein kinase that controls cellular metabolism, catabolism, immune responses, autophagy, survival, proliferation, and migration, to maintain cellular homeostasis |
| “NRAS”    | neuroblastoma RAS, a member of the RAS family proteins                                                                                                                |
| “NSCLC”   | non-small-cell lung carcinoma                                                                                                                                         |
| “ORR”     | objective response rate                                                                                                                                               |
| “Pan RAS” | forms of RAS proteins across wild-type and mutant variants                                                                                                            |
| “PDAC”    | pancreatic ductal adenocarcinoma                                                                                                                                      |
| “PI3K”    | phosphoinositide 3 kinase, an important signaling pathway for many cellular functions such as growth control, metabolism and translation                              |
| “PR”      | partial response                                                                                                                                                      |
| “QD”      | quaque die                                                                                                                                                            |

|         |                                                                                                                                           |
|---------|-------------------------------------------------------------------------------------------------------------------------------------------|
| “RAF”   | rapidly accelerated fibrosarcoma, a group of kinases that play a crucial role in the MAPK/ERK signaling pathway                           |
| “RAS”   | rat sarcoma, a family of proteins that are critical regulators of cellular signaling pathways; it primarily includes HRAS, KRAS, and NRAS |
| “TRAEs” | treatment-related adverse events                                                                                                          |

**Cautionary Statement required by Rule 18A.05 of the Rules Governing the Listing of Securities on The Stock Exchange of Hong Kong Limited:** The Company cannot guarantee that it will be able to develop, or ultimately market GFH276 successfully. Shareholders and potential investors of the Company are advised to exercise caution when dealing in the shares of the Company.

By order of the Board  
**GenFleet Therapeutics (Shanghai) Inc.**  
**Dr. Qiang LU**  
*Chairman and Executive Director*

Hong Kong, October 26, 2026

*As at the date of this announcement, the Board of the Company comprises: (i) Dr. Qiang LU, Dr. Jiong LAN and Ms. ZHANG Wei as executive directors; (ii) Mr. ZHU Jingyang as non-executive director; (iii) Ms. Christine Shaohua LU-WONG, Dr. ZHOU Demin and Mr. LI Bo as independent non-executive directors*