



Alaunos Reports Preclinical ALN1003 Data Showing Effects on Insulin-Resistance-Related Biomarkers and Liver Histology in Diet-Induced Obesity Models

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Findings from non-GLP DIO mouse studies show effects on insulin-resistance-related biomarkers, adipose endocrine signaling, and qualitative liver histology findings consistent with lower hepatic steatosis

FORT LAUDERDALE, Fla., May 18, 2026 (GLOBE NEWSWIRE) -- **Alaunos Therapeutics, Inc. (Nasdaq: TCRT)** today announced updated preclinical data from non-GLP diet-induced obesity (DIO) mouse studies evaluating ALN1003, the Company's investigational oral metabolic therapeutic candidate. Across selected biomarker and liver histology assessments, ALN1003 showed effects on insulin-resistance-related measures, adipose endocrine markers, and qualitative liver pathology findings relevant to metabolic syndrome biology. These findings support continued preclinical development of ALN1003 in metabolic syndrome and related conditions, including obesity, metabolic dysfunction-associated steatotic liver disease (MASLD) and insulin resistance.

Metabolic syndrome is increasingly understood not as a collection of independent risk factors, but as a multi-organ, adiposity-associated disease state characterized by insulin resistance, dysfunctional adipose tissue, chronic low-grade inflammation, and hepatic lipid accumulation. Emerging research suggests that the biological quality and endocrine function of adipose tissue, rather than fat mass alone, plays a central role in disease progression. Within this framework, the preclinical profile of ALN1003 is notable in that it appears to engage several of these interconnected systems simultaneously.

In the longer-duration 48-day DIO model, ALN1003 was associated with significantly lower fasting insulin and significantly lower Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), a calculated index derived from fasting glucose and insulin. ALN1003-treated animals had significantly lower HOMA-IR scores compared with controls after adjustment for percentage body fat in a standard ANCOVA analysis ($p=0.0006$), with the same conclusion confirmed using heteroscedasticity-robust HC3 standard errors as a sensitivity analysis ($p=0.0014$). ALN1003 was also associated with numerically lower leptin, significantly higher adiponectin, and a significantly higher adiponectin-to-leptin ratio. Together, these findings are consistent with improved insulin-resistance-related biomarkers and favorable adipose endocrine signaling in this preclinical model.

Liver histology findings provided additional supportive evidence relevant to the liver component of metabolic syndrome. In a blinded pathology review of selected liver samples from the 48-day and 18-day DIO studies (3 control and 3 treatment samples from each study, selecting the high dose group from the 18-day study), H&E-stained whole-slide images were evaluated for steatosis, lobular inflammation, ballooning degeneration, and NAS components, while Masson's trichrome-stained sections were used to assess fibrosis. ALN1003-treated animals showed qualitative findings consistent with lower hepatic steatosis relative to controls, along with lower mean NAS scores. Control samples evaluated in the selected pathology set had NAS scores of 5, consistent with more active steatotic liver disease-like histology, while ALN1003-treated samples had lower mean NAS scores of 2.7 and 1.3 in the 48-day and 18-day studies, respectively. These limited pilot pathology findings do not establish MASLD resolution, fibrosis reversal, inflammation improvement, or clinical efficacy, but are consistent with a qualitative anti-steatotic effect in this preclinical model.

Taken together, these findings suggest that ALN1003 may influence multiple components of metabolic syndrome biology in DIO mouse models, including insulin-resistance-related biomarkers, adipose endocrine signaling, and hepatic lipid accumulation. This multi-axis preclinical profile may be relevant to a therapeutic landscape increasingly focused on metabolic health beyond body weight alone, including liver health, cardiometabolic risk, and preservation of lean mass.

"We are encouraged by the consistency of signals across insulin-resistance-related biomarkers, adipose endocrine markers, and liver histology while acknowledging these findings are early and preclinical," said Holger Weis, CEO of Alaunos. "The data support continued investigation of ALN1003 as a potential oral, non-hormonal approach for metabolic syndrome and related conditions." The Company noted that the current treatment landscape for metabolic syndrome is evolving rapidly beyond weight loss alone. The Company believes that the emerging target product framework increasingly emphasizes liver health, metabolic stability, cardiovascular risk reduction, convenience, and preservation of lean mass, alongside weight and glucose control. The Company believes ALN1003's early preclinical profile may align with this broader therapeutic direction.

The Company plans to continue advancing ALN1003 through additional preclinical studies focused on dose optimization, exposure-response relationships, expanded metabolic phenotyping, and further characterization of liver pathology and underlying mechanisms.

These findings are based on non-GLP preclinical studies and should be interpreted with appropriate caution. Limitations include limited sample sizes, histological analysis limited to a sample of available livers, single-timepoint biomarker assessments, known constraints of HOMA-IR interpretation in rodent models, and qualitative/semi-quantitative pathology scoring. ALN1003 has not been evaluated in human clinical trials, and its safety and efficacy in humans has not been established.

As of March 31, 2026, the Company had cash and cash equivalents of approximately \$0.354 million. The Company's current cash runway extends into the second quarter of 2026. The Company intends to pursue additional financing to support continued operations and advancement of its preclinical obesity and metabolic disorders program.

About ALN1003

ALN1003 is an investigational oral metabolic therapeutic being developed to target multiple drivers of metabolic dysfunction, including insulin resistance, adipose tissue signaling, and hepatic lipid metabolism. Preclinical studies to date suggest potential relevance across metabolic syndrome and related conditions.

About Alaunos Therapeutics

Alaunos Therapeutics is a biotechnology company focused on developing novel therapeutics. The Company's obesity and metabolic disorders program is advancing ALN1003, an oral small-molecule candidate designed to offer a differentiated, non-hormonal approach compared with currently available therapies.

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements. Forward-looking statements are statements that are not historical facts, and in some cases can be identified by terms such as "may," "will," "could," "expects," "plans," "anticipates," "believes" or other words or terms of similar meaning. These statements include, but are not limited to, statements regarding the Alaunos Therapeutics, Inc.'s ("Alaunos" or "the Company") business and strategic plans, the timing of the Company's research and development programs, including potential data read out dates as well as any potential patent filings for the Company's obesity program.

These forward-looking statements are based on current expectations and assumptions that are subject to risks and uncertainties, which could cause actual results to differ materially. Important factors that could cause actual results to differ materially include, but are not limited to: changes in the Company's operating plans that may impact its cash expenditures; uncertainties built into research and development such as preclinical mouse data not translating to human trials, or challenges in scaling up formulations, including the risk that early non-GLP study results may not be replicated in confirmatory studies or pose safety concerns in IND-enabling studies; delays or failures in future studies; whether Alaunos' product candidates will advance further in the clinical trial process, including getting approval by the U.S. Food and Drug Administration (FDA) or other foreign health authority to conduct clinical trials and whether and when, if at all, they will receive final approval from the FDA or equivalent foreign regulatory agencies and for which uses; challenges to the strength and enforceability of Alaunos' intellectual property rights (such as patent disputes); competition from other pharmaceutical and biotechnology companies (including in the crowded obesity treatment market); funding shortages or market changes affecting our cash needs; tolerability issues from drug administration; the inherent uncertainties in drug development, including potential failures optimizing formulations, mechanistic studies, or large-animal pharmacokinetics that could delay IND-enabling activities; manufacturing and supply chain disruptions related to CMC work; and other factors discussed in our latest Form 10-Q and Form 10-K filed with the Securities and Exchange Commission (SEC). Forward-looking statements may also be protected if they are immaterial.

We caution you not to place undue reliance on these forward-looking statements, which speak only as of the date of this press release. Except as required by law, Alaunos undertakes no obligation to update these statements to reflect events that occur or circumstances that exist after the date hereof.

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