

NEWS RELEASE

Newly Published Preclinical Study Demonstrates CD38 as a Driver of Age-Related NAD⁺ Decline, Older Mothers Have Lower NAD⁺ Levels in Placental Tissue, and NAD⁺ Restoration with Nicotinamide Riboside (NR) Improves Pregnancy Outcomes

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Preclinical study results showcase that CD38, an NAD-consuming enzyme more active with age and inflammation, leads to NAD⁺ depletion in placental tissue, while restoring NAD⁺ with the precursor NR improved placental, fetal, and offspring outcomes in aged mice

LOS ANGELES--(BUSINESS WIRE)-- **Niagen Bioscience, Inc.** (NASDAQ: NAGE), the global authority on NAD⁺ (nicotinamide adenine dinucleotide) with a focus on the science of healthy aging, today announces the publication of independent preclinical research in **Nature Communications** led by a team at Sichuan Agricultural University identifying the CD38 enzyme as a primary driver of NAD⁺ decline in aging tissue, and that nicotinamide riboside (NR) restored NAD⁺ and improved placental, fetal, and offspring outcomes in aged mice despite elevated CD38.

The researchers measured NAD⁺ and related metabolites in human placental tissue from younger and older mothers and found older placentas held less NAD⁺, NADH, and NADP⁺. Higher placental levels of the NAD⁺, NADH, and NADP⁺ metabolites were associated with higher infant birth weight. Mice and pigs showed the same age-related decline alongside poorer pregnancy outcomes, suggesting a broader feature of reproductive aging rather than specific to one species.

Andrew Shao, PhD, Senior Vice President, Global Scientific & Regulatory Affairs at Niagen Bioscience stated, "This paper demonstrated age-related decline in placental tissue NAD⁺, reaffirmed that CD38 consumes both NAD⁺ and

NMN, and showed the positive reproductive effects of increasing NAD⁺ with NR.”

Niagen Bioscience did not conduct, fund, or design this study, and the NR used was not the Company’s Niagen ingredient. The Company is highlighting the work because the results speak to the mechanism behind NAD⁺ decline and how different NAD⁺ precursors behave in tissue where that mechanism is most active.

The demand side of NAD⁺ decline

This research provides insight into how NAD⁺ consumption increases with age by overactivation of CD38. CD38 is a multifunctional ectoenzyme, and its expression increases with age in multiple cell types, particularly on the surface of immune cells, contributing to metabolic changes and age-related inflammation. In addition, CD38 degrades another precursor, nicotinamide mononucleotide (NMN). This CD38 action exacerbates inflammation, which drives CD38 activity, and so on.

The authors traced the source to CD38-expressing macrophages accumulating in aged placental tissue and depleting NAD⁺ in neighboring decidual stromal cells. Across several experiments, the authors showed that the CD38 enzyme acts on NAD⁺ and NMN outside the cell, before they can cross the cell and be used.

CD38 consumes NMN, while previous research shows NR inhibits CD38

The analysis examined how CD38 inhibition vs. NAD⁺ boosting with NR affects NAD⁺ in placental tissues and reproductive outcomes. Consistent with previous findings, the study also demonstrated NMN is also a substrate for CD38. NMN cannot enter the cell directly because it carries a phosphate group, which prevents it from crossing the cell membrane intact. Because NMN must be converted first, it is left outside the cell and exposed. The authors observed that CD38 breaks down NMN, while previously published research demonstrates NR actually inhibits CD38 activity (**Kao et al., 2024**). This suggests that in tissues with high CD38 activity, NR is more effective than NMN because NMN may not survive long enough to be used by the cell.

Preclinical study overview

The investigators analyzed human placental tissue from younger and older mothers in mouse and pig models. They profiled NAD⁺ and related metabolites, identified the cell population responsible for the decline, and then tested whether restoring NAD⁺ changed outcomes. They attempted restoration in two ways: oral NR and CD38 inhibitor(78c).

Key findings

- Across humans, mice, and pigs, placental aging was consistently associated with lower NAD⁺, NADH, NADP⁺, identifying a conserved metabolic signature of reproductive aging.
- Higher placental levels of these metabolites were associated with higher infant birth weight.
- Aged mice and pigs showed the same decline in placental NAD⁺ alongside poorer pregnancy outcomes.
- Mechanistic studies showed that CD38 macrophages depleted NAD⁺ in neighboring stromal cells
- In aged pregnant mice, NR administration or CD38 inhibitor restored placental NAD⁺, improved fetal growth, and shifted placental gene expression toward a younger profile.
- CD38 degraded NMN outside the cell, blocking its ability to elevate NAD⁺ levels. Previously published research shows that NR inhibits CD38, suggesting NR may be more effective in tissues where CD38 activity is high (**Kao et al., 2024**).

Relevance

While additional clinical research is needed to confirm these findings, this study shows that CD38 is a key contributor to age-related NAD⁺ decline in placental tissues and that, unlike NMN, NR is not consumed by CD38 outside the cell.

Additional insights on this study can be found on AboutNAD, at **www.aboutNAD**.

This preclinical study adds to a growing body of preclinical and ex vivo evidence examining NR across reproductive endpoints, including oocyte quality in aged animals, ovarian function in models of polycystic ovary syndrome, placental function in models of preeclampsia, fetal growth under metabolic and oxygen stress, semen quality, and lactation and offspring development, as demonstrated by Table 1 below.

For additional insights, read **“Placental NAD⁺ Declines with Maternal Age: What New Research Reveals About CD38, NR, and NMN”** at **www.aboutNAD.com**.

Table 1. Preclinical and ex vivo research on NR and reproductive endpoints:

Human Ex Vivo			
Publication	Intervention	Objective	Key Outcomes
Wang et al., 2021 NAD ⁺ Deficiency and Mitochondrial Dysfunction in Granulosa Cells of Women with Polycystic Ovary Syndrome	Nicotinamide Riboside	To investigate the relationship between NAD ⁺ and inflammation in polycystic ovary syndrome (PCOS) patients. PCOS is an endocrine disorder characterized by ovulation dysfunction. Granulosa cells (GCs) support and create the microenvironment necessary for oocyte (egg cell) development.	In the GCs of PCOS patients, NAD ⁺ levels were decreased, accompanied by increased inflammation, oxidative stress, and mitochondrial dysfunction. NR administration restored NAD ⁺ levels, reduced oxidative stress, and improved mitochondrial function in GCs.



Clinical Studies			
Publication	Intervention	Objective	Key Outcomes
Huang et al., 2025 NAD ⁺ Repletion Enhances Mammary Lactogenesis and Improves Offspring Development in a Sow Model	Nicotinamide Riboside	To evaluate the impact of NAD ⁺ metabolism, through NR supplementation, on lactation in sows.	NR improved the litter's weaning weight while reducing hypothermia and gut barrier dysfunction. NR improved mammary and milk NAD ⁺ levels, milk yield, mitochondrial number and biogenesis, size of the milk-producing spaces (alveolar lumen area) in the mammary gland, and fat and carbohydrate metabolism.
Huang et al., 2026 Effects of Nicotinamide Riboside Supplementation During Late Gestation and Lactation on Sow Performance, Milk Metabolome, and Gut Microbiome	Nicotinamide Riboside	To evaluate the effects of NR supplementation during late gestation and lactation on sow performance, milk composition, and the gut microbiome of sows and their offspring.	NR supplementation shortened the duration of giving birth and reduced the number of late-gestation mummies while tending to reduce fetal growth restriction. NR improved litter weaning weight and average daily gain, while tending to produce more consistent birth weights within litters. In sows, NR improved antioxidant and inflammation biomarkers, while increasing estimated milk production and levels of protein, fat, NAD ⁺ , and related metabolites in milk. Certain potentially beneficial gut bacteria were enriched in NR-supplemented sows and their offspring.

Preclinical Studies			
Publication	Intervention	Objective	Key Outcomes
Ear et al., 2019 Maternal Nicotinamide Riboside Enhances Postpartum Weight Loss, Juvenile Offspring Development, and Neurogenesis of Adult Offspring	Niagen® Nicotinamide Riboside	To investigate whether postpartum might dysregulate maternal NAD ⁺ and whether increasing systemic NAD ⁺ with NR supplementation could benefit rodent mothers and their offspring.	- NR increased prolactin production, boosting lactation, with peak lactation occurring on day 14. - NR caused a more than 20-fold increase in the mammary NAD metabolome in postpartum rodents. - NR-supplemented mothers had higher levels of BDNF (brain-derived neurotrophic factor) in their milk. - The offspring of NR-supplemented mothers had higher BDNF levels in their hindbrain and exhibited qualities that reached adulthood, including reduced anxiety, increased strength, better endurance in swim tests, improved performance on balance tests, better spatial memory, increased hippocampal neurogenesis, and leaner body composition.
Yang et al., 2020 Increasing Ovarian NAD ⁺ Levels Improve Mitochondrial Functions and Reverse Ovarian Aging	Nicotinamide Riboside	To investigate whether NR supplementation in aged mice could increase ovarian NAD ⁺ levels in the context of age-dependent decreases in ovarian fertility.	- NR increased ovarian NAD⁺ levels, leading to an increased number of ovarian follicles and ovulatory potential, as well as an increased live birth rate. - NR also reduced levels of reactive oxygen species and abnormalities in aging mouse oocytes. - NR improved ovarian mitochondrial energy metabolism.
Li et al., 2022 Nicotinamide Riboside Supplementation Ameliorated Postovulatory Oocyte Quality Decline	Nicotinamide Riboside	To investigate the effects of NR treatment on maintaining mouse oocyte quality in vitro.	- NAD⁺ levels naturally declined in postovulatory mouse oocytes over time, but this decline was reversed with NR treatment. - NR increased NAD⁺ levels and reduced the occurrence of abnormal oocytes by 9%. - NR improved oocyte quality by reducing abnormal spindle

			formations and enhancing fertilization potential. NR helped prevent age-related mitochondrial dysfunction in oocytes, lowering levels of reactive oxygen species, DNA damage, and cell death.
Yang et al., 2022 Metabolic and Epigenetic Dysfunctions Underlie the Arrest of In Vitro Fertilized Human Embryos in a Senescent-Like State	Nicotinamide Riboside	To investigate why many human embryos halt developing during in vitro fertilization (IVF) and to explore possible treatments to prevent this, such as NR treatment.	- Arrested human embryos entered a senescent-like state and had a reduction in key proteins. - NR partially rescued the arrested embryos, allowing them to develop into the morula stage, where the embryo is a solid ball of cells, and the early blastocyst stage, where a fluid-filled cavity begins to form.
Lee et al., 2023 Dietary Supplementation with Nicotinamide Riboside Improves Fetal Growth Under Hypoglycemia	Nicotinamide Riboside	To assess the protective effects of NR supplementation on gestational hypoglycemia in female mice. Gestational hypoglycemia occurs when a woman develops low blood sugar during pregnancy.	- In control mice, NR enhanced resistance to energy deprivation and boosted gluconeogenesis, the process of producing glucose from non-carbohydrate sources. - In ovariectomized mice (mice without ovaries), NR reduced high blood sugar levels and induced gluconeogenesis. - In pregnant mice, NR prevented maternal hypoglycemia by increasing blood sugar levels. - Hypoglycemic mice supplemented with NR had higher fetal birth weights.
Li et al., 2023 Characterization of Long-Term Ex Vivo Expansion of Tree Shrew Spermatogonial Stem Cells	Nicotinamide Riboside	To investigate the effects of repeated cell culture transfer on the sperm-producing capabilities of the spermatogonial stem cells of tree shrews, and assess the potential protective effects of boosting NAD+ through treatment with NR. Tree shrews, closely related to primates, offer a valuable model for studying human-relevant spermatogenesis processes.	NR increased NAD+ levels, decreased oxidative stress, and alleviated deficiencies in mitochondrial function, DNA damage, and ATP production in spermatogonia—precursor sperm cells. However, short-term NR administration did not increase the spermatogonial stem cell population.
Ni et al., 2023 BNC1 Deficiency Induces Mitochondrial Dysfunction-Triggered Spermatogonia Apoptosis Through the CREB/SIRT1/FOXO3 Pathway: The Therapeutic Potential of Nicotinamide Riboside and Metformin	Nicotinamide Riboside	To assess the therapeutic potential of NR and metformin administration in ameliorating spermatogenic failure and subfertility in mice with a Basoonclin 1 (BNC1) gene mutation, which is linked to subfertility in males and females.	- In mice with the BNC1 mutation, NR and metformin alone effectively reversed mitochondrial dysfunction, inhibited spermatogonia death, and improved testicular architecture. - NR and metformin alone also increased sperm count, motile sperm count, and progressive sperm count, improving overall sperm quality and quantity.
Selli et al., 2023 Nicotinamide Riboside Preserves Ovarian Injury in Experimental Sepsis Model in Rats	Nicotinamide Riboside	To assess the protective effects of NR administration on ovarian damage in a rat model of intestinal damage-induced sepsis.	- High-dose NR reduced inflammatory stress markers and ovarian tissue damage caused by sepsis in rats. - NR mitigated inflammation by decreasing white blood cell infiltration, markers of cell damage, and follicular cell death, which is crucial for oocyte maturation. - NR increased antioxidant enzyme activity in the ovaries.
Thompson et al., 2023 Nicotinamide Riboside, an NAD + Precursor, Protects Against Cardiac Mitochondrial Dysfunction in Fetal Guinea Pigs Exposed to Gestational Hypoxia	Niagen® Nicotinamide Riboside	To evaluate whether NR supplementation could reverse mitochondrial dysfunction in fetal guinea pig hearts caused by gestational hypoxia, a condition that reduces oxygen supply to the fetus and disrupts heart and placental mitochondrial function, impairing growth and organ development.	- Hypoxia reduced fetal body weight and placental NAD+ levels, while increasing placental weight and mitochondrial dysfunction. - NR restored body weight, mitochondrial function, and placental NAD+ levels in hypoxic fetuses, and also elevated liver NAD+ levels in both normoxic (normal oxygen) and hypoxic fetuses. - NR also prevented the reduction in fetal heart weight in males and normalized the increase in fetal brain weight in females.

Warren et al., 2023 The NAD Salvage Pathway in Mesenchymal Cells is Indispensable for Skeletal Development in Mice	Niagen® Nicotinamide Riboside	To investigate the role of NAD ⁺ in bone development using mice lacking the NAMPT gene in all mesenchymal cells (stem cells that differentiate into bone and cartilage) of the limb, and to assess whether oral NR supplementation during pregnancy can mitigate bone formation defects in offspring.	- Mice lacking the NAMPT enzyme exhibited severe limb and sternum shortening at birth, likely due to NAD⁺ deficiency, and died a few days after birth for reasons that were unclear. - To determine if the NAD⁺ deficiency was responsible for these defects, NR was administered orally to the mice during pregnancy. Prenatal NR supplementation reduced the severity of skeletal abnormalities, preventing joint malformations and promoting better development of the forelimbs, though the defects were not fully corrected. - The partial rescue of prenatal skeletal defects by NR allowed the NAMPT-knockout mice to survive and ambulate after birth.
Yang et al., 2023 Deletion of Enzymes for De Novo NAD ⁺ Biosynthesis Accelerated Ovarian Aging	Nicotinamide Riboside	To investigate how reduced NAD ⁺ levels impact ovarian aging and fertility, and whether NR supplementation could enhance fertility and egg quality in middle-aged mice with genetic mutations.	- Middle-aged mutant mice showed lower NAD⁺ levels, leading to reduced fertility and dysfunction in both the ovaries and mitochondria. - NR increased ovarian NAD⁺ levels, partially restoring fertility in the mutant mice, as evidenced by an increased litter size. - NR also increased ovarian reserve and improved the quality of oocytes in the mutant mice.
Arslan et al., 2024 Nicotinamide Mononucleotide and Nicotinamide Riboside Reverse Ovarian Aging in Rats Via Rebalancing Mitochondrial Fission and Fusion Mechanisms	Nicotinamide Riboside & Nicotinamide Mononucleotide	To evaluate the effectiveness of NR supplementation and nicotinamide mononucleotide (NMN) administration alone in reversing ovarian aging and improving egg production by restoring mitochondrial function in middle-aged rats.	- NR and NMN treatment enhanced both the quantity and quality of follicles, as well as the corpus luteum, which is vital for fertility hormone production. - NR and NMN treatment in middle-aged rats boosted gene expression related to mitochondrial fusion, which is essential for protecting oocytes and follicles. - NR and NMN supplementation raised SIRT1 levels in the ovaries, helping to delay ovarian aging by promoting key enzymes. - NR and NMN restored the balance of luteinizing and follicle stimulating hormones, which are often disrupted during ovarian aging.
Jahan et al., 2024 NAD ⁺ Depletion is Central to Placental Dysfunction in an Inflammatory Subclass of Preeclampsia	Nicotinamide Riboside	To investigate the role of NAD ⁺ depletion in placental function and inflammation-driven preeclampsia (PE), and to evaluate whether boosting NAD ⁺ with NR administration can mitigate the harmful effects of inflammation in PE, using human placenta, a human placental precursor (trophoblast) cell model of inflammation-driven PE, and a rat model of PE.	- In both human and rat placentas affected by PE, NAD⁺ levels were lower, and the activity of NAD-consuming enzymes was elevated, resulting in mitochondrial dysfunction and increased oxidative damage. - In a human trophoblast cell model, NR treatment restored NAD⁺ levels, reduced protein damage, and improved mitochondrial function. - In a rat model of PE, NR treatment reduced blood pressure, decreased placental inflammation, and improved both placental and fetal weight, leading to increased fetal survival.
Li et al., 2024 The NAD ⁺ Precursor Nicotinamide Riboside Protects Against Postovulatory Aging In Vitro	Nicotinamide Riboside	To determine if NR administration could prevent postovulatory aging (POA) of oocytes during in vitro culture and improve the success rates of artificial reproductive technology (ART). POA is a significant challenge, especially for older women, as it reduces egg quality and lowers the chances of successful pregnancies through ART.	- NR effectively reduced signs of aging-related decline in oocyte quality by lowering levels of reactive oxygen species, enhancing mitochondrial function (demonstrated by increased ATP production), and correcting the positioning of essential cellular components known as cortical granules. - NR also helped restore the proper arrangement of spindles and the

			alignment of chromosomes, while lowering DNA damage in the oocytes.
Liu et al., 2024 Maternal Administration of Acetaminophen Affects Meiosis Through its Metabolite NAPQI Targeting SIRT7 in Fetal Oocytes	Nicotinamide Riboside & Nicotinamide	To assess the potential effects of maternal administration of acetaminophen on fetal oocyte development in mice and assess the potential of NR and nicotinamide (NAM) supplementation to reverse these adverse effects.	- Acetaminophen caused a dose-dependent delay in chromosomal development of fetal oocytes, disrupted DNA damage repair in oocytes, and led to a significant reduction in NAD⁺ levels in the ovaries. - Both NR and NAM effectively reversed the acetaminophen-induced delay in chromosomal development in the ovaries of mice. However, NR was slightly more effective than NAM, as it better rescued genes crucial for meiosis (cell division).
Shen et al., 2025 Nicotinamide Riboside Supplementation Alleviates Heat Stress-Induced Testicular Damage and Improves Semen Quality in Boars	Nicotinamide Riboside	To investigate the impact of heat stress (HS) on testicular NAD ⁺ metabolism and assess the potential of NR supplementation in alleviating HS-induced reductions in sperm quality in boars.	- Testicular NAD⁺ deficiency was identified as a hallmark of testicular damage under HS. - In HS-exposed boars, NR restored testicular NAD⁺ levels, improved semen quality, reduced defects during spermatogenesis—the process of sperm production—and attenuated oxidative stress and inflammation.
Wei et al., 2025 Nicotinamide Riboside Supplementation Protects Against Maternally Diabetes-Associated Decline in Oocyte Quality	Nicotinamide Riboside	To investigate the effects of NR supplementation on diabetes-associated oocyte quality decline in mice.	- In diabetic mice, NR protected against NAD⁺ depletion and disruption to the NAD⁺/NADH ratio in the ovaries and oocytes. - NR alleviated diabetes-induced reductions in ovarian weight relative to body weight, enhanced superovulation—the hormone-driven production of more oocytes than usual during ovulation—and improved oocyte quality and embryonic development potential, as indicated by increased ovulations and higher fertilization rates. - NR preserved oocyte structure, improved mitochondrial function, and reduced oxidative stress in diabetic mice.
Xu et al., 2025 Nicotinamide Riboside Supplementation Alleviates Testicular Aging Induced by Disruption of Qprt-Dependent NAD ⁺ De Novo Synthesis in Mice	Nicotinamide Riboside	To investigate the role of NAD ⁺ in sperm production in mice lacking the quinolinate phosphoribosyl transferase (Qprt) gene, which is essential for NAD ⁺ production in spermatocytes (cells in the testes that produce sperm).	- In mice lacking the Qprt gene, NR treatment increased NAD⁺ levels in immature spermatocytes and the testes. - NR also increased testicular weight and sperm count, reduced programmed cell death, protected the development of sperm cells, and altered the expression of X and Y sex chromosomes.
Zhu et al., 2025 Nicotinamide Riboside Supplementation Ameliorates Ovarian Dysfunction in a PCOS Mouse Model	Nicotinamide Riboside	To investigate the impact of NR supplementation on ovarian function in a mouse model of PCOS.	- NR prevented a decrease in ovarian NAD⁺ levels, normalized estrous cycle irregularities, and improved ovulation potential in PCOS mice. - NR protected against ovarian fibrosis and improved mitochondrial function in stromal cells (connective tissue cells) within the ovaries of PCOS mice. - NR improved oocyte quality by increasing mitochondrial function, supporting early embryonic development, and reducing mitochondrial clustering, oxidative stress, and spindle abnormalities in fertilized oocytes from PCOS mice.
Hua et al., 2026 CD38-Activated Macrophages Drive Age-related Placental Senescence by Depleting	Nicotinamide Riboside	To investigate age-related placental senescence across humans, pigs, and mice, focusing on CD38—an NAD ⁺ -consuming enzyme whose levels increase with age.	- In placental tissues of humans, pigs, and mice, aging was associated with decreased NAD⁺ and increased levels of NAD⁺-consuming enzymes, including CD38. - In mice, age-related inflammation

Nad+ in Decidual Stromal Cells			...elevated CD38, depleting NAD+ in placental cells and driving further inflammation—a vicious self-reinforcing cycle. • In mouse cells, CD38 blocked NMN's ability to elevate NAD+ levels. • In mice, restoring or preserving NAD+ through NR or a CD38 inhibitor improved pregnancy outcomes and offspring metabolic health, with no detectable signs of maternal toxicity.
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About Niagen Bioscience

Niagen Bioscience, Inc. (NASDAQ: NAGE) is the global authority in healthy aging and NAD+ (nicotinamide adenine dinucleotide) science. As a trusted pioneer of NAD+ discoveries, Niagen Bioscience™ is dedicated to advancing healthspan through precision science and innovative NAD+-boosting solutions.

The Niagen Bioscience team, composed of world-renowned scientists, works with independent investigators from esteemed universities and research institutions around the globe to uncover the full potential of NAD+. A vital coenzyme found in every cell of the human body, NAD+ declines with age and exposure to everyday lifestyle stressors. NAD+ depletion is a key contributor to age-related changes in health and vitality.

Distinguished by state-of-the-art laboratories, rigorous scientific and quality protocols, and collaborations with leading research institutions worldwide, Niagen Bioscience sets the gold standard for research, quality, and innovation. There's a better way to age.

At the heart of its clinically tested product portfolio is Niagen® (patented nicotinamide riboside, or NR), the most efficient, well-researched, and high-quality NAD+ booster available. Niagen powers the Company's consumer supplement, Tru Niagen®, the number one NAD+ boosting oral supplement in the United States† (available at www.truniagen.com), and Niagen™ Plus, featuring pharmaceutical-grade intravenous (IV) and injectable Niagen products (www.niagenplus.com). Pharmaceutical-grade Niagen IV and injections are compounded and distributed by U.S. FDA-registered 503B outsourcing facilities and are available exclusively at clinics with a prescription. NAD Pharmaceuticals Corp., the Company's wholly owned subsidiary focused on developing therapies for accelerated aging and rare genetic diseases, is conducting research on NB4168, a differentiated small molecule.

Niagen Bioscience's robust patent portfolio protects NR and other NAD+ precursors. Niagen Bioscience maintains a website at www.niagenbioscience.com, where copies of press releases, news, and financial information are regularly published.

† Based on revenue per largest U.S. e-commerce marketplace (Jan. 2025 – Dec. 2025)

Forward Looking Statements:

This release contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, and Section 21E of the Securities Exchange Act of 1934. Statements that are not a description of historical facts constitute forward-looking statements and may often, but not always, be identified by the use of such words as “expects,” “anticipates,” “intends,” “estimates,” “plans,” “potential,” “possible,” “probable,” “believes,” “seeks,” “may,” “will,” “should,” “could,” “predicts,” “projects,” “continue,” “would” or the negative of such terms or other similar expressions.

Forward-looking statements are based on current expectations and assumptions and are subject to risks and uncertainties that could cause actual results to differ materially from those described. These risks and uncertainties include, but are not limited to, statements regarding Niagen Bioscience's NB4168 pharmaceutical development program; planned preclinical, IND-enabling and clinical development activities; the potential timing of an IND submission or first-in-human study; the potential bioavailability, exposure, safety, tolerability, efficacy, pharmacodynamic or clinical profile of NB4168; and the Company's ability to translate its NAD+ platform into pharmaceutical products; inflationary conditions and adverse economic conditions; our history of operating losses; the growth and profitability of our product sales; our ability to maintain and grow sales, marketing and distribution capabilities; changing consumer perceptions of our products; our reliance on a single or limited number of third-party suppliers; risks of conducting business in China; including unanticipated developments in and risks related to the Company's ability to secure adequate quantities of pharmaceutical-grade Niagen in a timely manner; the Company's ability to obtain appropriate contracts and arrangements with U.S. FDA-registered 503B outsourcing facilities required to compound and distribute pharmaceutical-grade Niagen to clinics; the Company's ability to remain on the U.S. FDA Bulk Drug Substances Nominated for Use in Compounding Under Section 503B of the Federal Food, Drug, and Cosmetic Act Category 1 list; the Company's ability to maintain and enforce the Company's existing intellectual property and obtain new patents; whether the potential benefits of NRC can be further supported; further research and development and the results of clinical trials possibly being unsuccessful or insufficient to meet applicable regulatory standards or warrant continued development; the ability to enroll sufficient numbers of subjects in clinical trials; determinations made by the FDA and other governmental authorities, including with respect to products seeking to compete in our market; mislabeling or other misleading marketing practices by competitors; economic and market instability, including as a result of tariffs or trade conflicts; and the risks and uncertainties associated with our business and financial condition in general, described in our filings with the Securities and Exchange Commission (SEC), including, without limitation, our most recent Annual Report on Form 10-K and Quarterly Report on Form 10-Q as filed with the SEC.

Readers are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and actual results may differ materially from those suggested by these forward-looking statements. All

forward-looking statements are qualified in their entirety by this cautionary statement and Niagen Bioscience undertakes no obligation to revise or update this release to reflect events or circumstances after the date hereof.

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