Exhibit 19

From: Ryan Dellinger <RyanD@chromadex.com>
Sent: Fri, 7 Aug 2015 17:53:02 +0000 (UTC)
To: Claire Kruger <ClaireK@chromadex.com>; Dietrich Conze<DietrichC@chromadex.com>; Juan Crespo-Barreto <JuanC@chromadex.com>
Cc: Troy Rhonemus <TroyR@chromadex.com>
Subject: NR posters
Attachments: NIAGENposter2015-final_p.pdf;NRposter0815_brenner_verticle.pdf

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Here are the 2 NR posters.

NIAGEN poster 2015-final was presented at the FASEB meeting on Mitochondrial Biogenesis and Dynamics in Health, Disease and Aging in West Palm Beach Florida MAY 17-22.

The Brenner poster will be presented at the FASEB NAD+ metabolism and signaling meeting in Timmendorfer Starnd, Germany August 9-14. This poster includes his n=1 data and a new mouse study.

Ryan

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ChromaDex®

Nicotinamide Riboside is an NAD+ precursor in humans; results from the first-in-man study.

Ryan W Dellinger[1], Mark S Schmidt[2], Nisrine Zakaria[1], Andrew Charrette[1], Mal Evans[3], Charles Brenner[2] and Frank L Jaksch[1]
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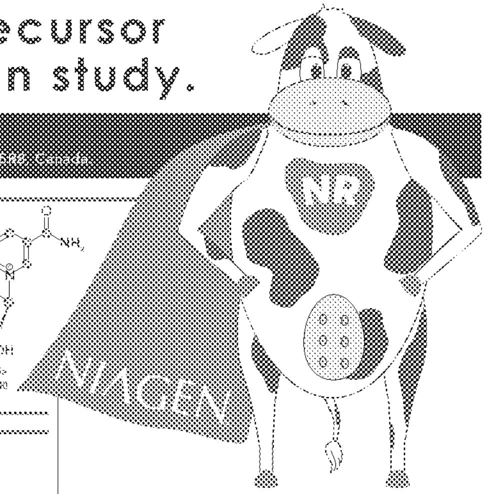


NR IS NATURALLY FOUND IN MILK

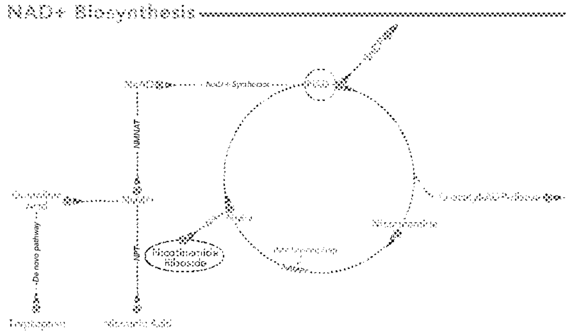
NIAGEN® is a commercially-available form of nicotinamide riboside (NR), a single chemical moiety containing nicotinamide and ribose. NR is a form of vitamin B3 found naturally occurring in milk. Recent research has shown that NR is an efficient NAD+ precursor in animals since NR bypasses a key rate limiting step in NAD+ biosynthesis. In one study, prolonged NR-supplementation in mice being fed a high fat diet caused animals to gain weight at a slower rate, improved insulin sensitivity, increased endurance, and increased muscle and liver NAD+ concentrations. Moreover, NR was shown to induce mitochondrial biogenesis.

However, despite the wealth of pre-clinical studies supporting NR supplementation in animals, there is no information on safety and pharmacokinetics of NR in humans. This randomized, double-blind three-arm crossover, 24 hr single-dose pharmacokinetic study has been conducted to evaluate the effects of orally administered NIAGEN® on the pharmacokinetics of NR and NR metabolites in humans. Participants served as their own control and received a single dose of 100, 300 or 1000 mg of NIAGEN® on three test days, separated by a 7 day washout period. NR and NR metabolites were analyzed in urine, blood plasma, and white blood cells. For urine, they were analyzed at pre-dose, 0-6h, 6-12h, and 12-24h collection intervals and for blood plasma and white blood cells, they were analyzed at pre-dose, 1h, 2h, 4h, 8h, 12h, and 24h post-dose.

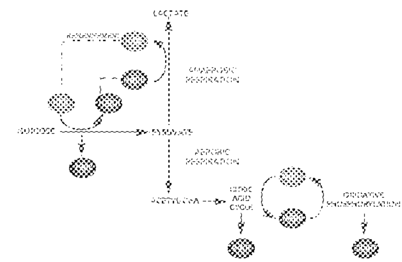
The pharmacokinetics of NIAGEN® followed a typical dose-response pattern with respect to the detection of NR metabolites in the urine, white blood cells and blood plasma of participants. Importantly, a single administration of NR increases NAD+ levels in the blood of humans. This is the first report confirming that NR is an NAD+ precursor in humans. Furthermore, no serious adverse events were observed in this study.

In summary, the current study provides evidence that NIAGEN® supplementation is measurable, safe, and well tolerated in healthy participants.

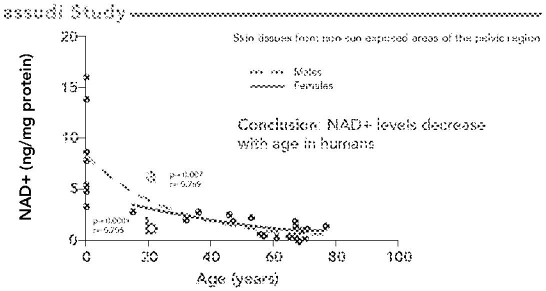
NAD+ Biosynthesis



NAD+ and ATP Production

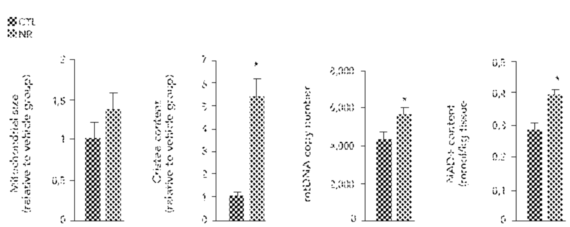


Massudi Study



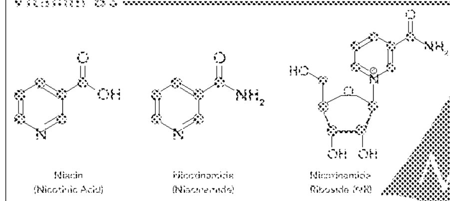
Canto Study

Muscle tissue from Mice

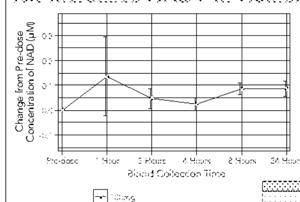


Conclusion: NR is a NAD+ precursor in mice

Vitamin B3



NR Increases NAD+ in Humans



Mean (SD) blood plasma NAD concentration for the pre-dose, 1h, 2h, 4h, 8h, and 24h blood collection times following administration of 100mg NR to Healthy Adults (N=12).

The blood plasma concentration of NAD increased non-significantly, relative to pre-dose, at 1 hour, 2 hours, and 4 hours post-dose and reached significance at 8 hours (p = 0.002), and 24 hours (p = 0.014) post-dose for the 100mg NR group. The peak equal height peak at 8 and 24 hours post-dose resulted in an increased concentration of 33% over pre-dose on average.

NAD Concentration (μM)	NR 100mg Mean ± SD (N) Within Group P Values*
Pre-dose	0.25 ± 0.21 (12)
1 Hour	0.40 ± 0.67 (11)
2 Hour	0.30 ± 0.26 (12)
4 Hour	0.222 ± 0.193 (12)
8 Hour	0.34 ± 0.18 (12)
24 Hour	0.342 ± 0.269 (12)
Change from Pre-dose to 1 Hour	0.13 ± 0.53 (11) p = 0.410
Change from Pre-dose to 2 Hour	0.05 ± 0.13 (12) p = 0.257
Change from Pre-dose to 4 Hour	-0.028 ± 0.090 (12) p = 0.307
Change from Pre-dose to 8 Hour	0.085 ± 0.076 (12) p = 0.002
Change from Pre-dose to 24 Hour	0.095 ± 0.112 (12) p = 0.014

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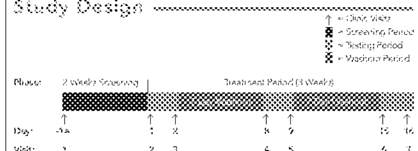
Study Design



Demographics & Characteristics

Demographics and Characteristics of All Participants Completing the Study (N=12)

	Mean ± SD (N)	SEM	Median (Min, Max)
Age	45.8 ± 6.6 (12)	1.9	46 (33 – 54)
Weight (kg)	74.5 ± 12.1 (12)	3.5	74.6 (49.8 – 90.4)
BMI (kg/m2)	25.85 ± 2.49 (12)	0.72	25.97 (21.35 – 29.88)
Mean Systolic Blood Pressure (mmHg)	115.9 ± 9.7 (12)	2.8	113.3 (106.7 – 131.0)
Mean Diastolic Blood Pressure (mmHg)	75.5 ± 8.3 (12)	2.4	74.7 (66 – 95.0)
Mean Heart Rate (bpm)	67.9 ± 10.7 (12)	3.1	64.3 (53.5 – 91.4)
	N (%)		
Gender			
Female	6/12 (50%)		
Male	6/12 (50%)		
Ethnicity			
Hispanic or Latino	2/12 (17%)		
Not Hispanic or Latino	10/12 (83%)		

Conclusions

- We have successfully completed the first human clinical trial for nicotinamide riboside
- No serious adverse events were observed and NR was shown to be safe up to 1000 mg single administration
- We demonstrate that NR is an NAD+ precursor in humans

Safety Summary

Sixteen healthy participants were enrolled in the study and randomized to a three treatment sequence after screening and passing eligibility criteria. Twelve participants were able to complete the study. Participants completing the study had a mean age of 45.8 years with an average BMI of 25.85 ± 2.49.

With respect to product safety, evaluation of vital sign measures showed that there were no differences between 100mg, 300mg or 1000mg dosages of NIAGEN® in SBP, DBP or HR. All values for blood pressure and heart rate were at a normal and acceptable range for healthy adults.

There were no between group differences in hemoglobin, hematocrit, WBC, RBC, MCV, MCH, MCHC, RDW, platelets, lymphocytes, monocytes in 100mg, 300mg and the 1000mg dosages of NIAGEN®. Liver function tests: AST, ALT and GGT and Total bilirubin; Kidney function tests: creatinine and eGFR and electrolytes: sodium, potassium and chloride were not different 24h post-dose between the 100mg, 300mg or the 1000mg dosage groups of NIAGEN®.

NR was not observed in urine, although it metabolites; Nam, MeNam, Py_4, Py_2 were observed. This indicates that nicotinamide riboside is eliminated from the body via methylation of the nicotinamide ring after generating NAD+ (unless retained by the salvage pathway). Other metabolites including NA, NAD+, NADP, and NMN, did not display significant changes in their relative urine concentrations with NIAGEN® treatment, irrespective of dose.

Full results of this first human study will be revealed in the near future, plus other human studies are ongoing.

References

1. Canto C, Houtkooper RH, Pirinen E, Youn DY, Oosterveer MH, Cen Y, Fernandez-Marcos PJ, Yamamoto H, Andreux PA, Cettour-Rose P, Gademann K, Rinsch C, Schoonjans K, Sauve AA and Auwerx J. The NAD+ precursor nicotinamide riboside enhances oxidative metabolism and protects against high-fat diet-induced obesity. (2012) Cell Metab. 15(6):838-47
2. Massudi H, Grant R, Braidy N, Guest J, Farnsworth B and Guillemin GJ. Age-associated changes in oxidative stress and NAD+ metabolism in human tissue. (2012) 7:e42357

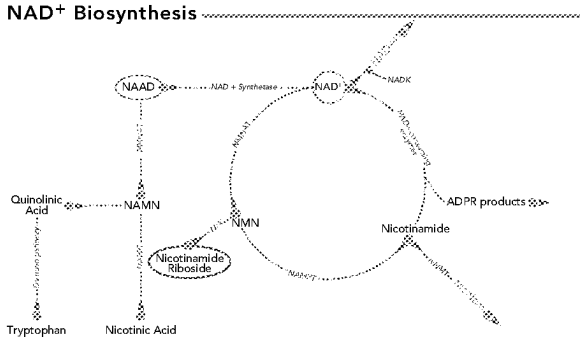
Dose-Dependent Elevation of the Blood NAD Metabolome by NR in Healthy Human Beings: *Clinical Efficacy and Novel Diagnostic Biomarkers*

Charles Brenner[1], Samuel AJ Trammell[1], Mark S Schmidt[1], Benjamin J Wardenaar[1], Philip Redpath[2], Marie E Migaud[2], Frank Jaksch[3] & Ryan W Bellinger[3]
[1]University of Iowa, USA, [2]Queens University Belfast, Northern Ireland, and [3]ChromaDex, Inc, USA

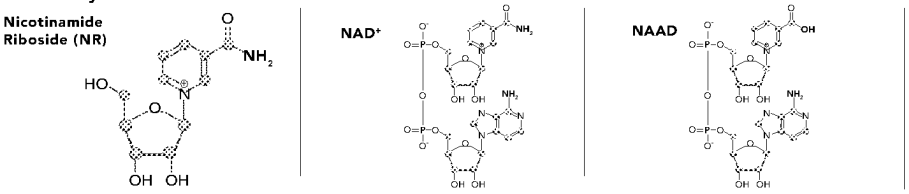
Abstract

Nicotinamide riboside chloride (NR) is in wide use as an orally available NAD precursor vitamin. Here we conducted three experiments to determine the time and dose-dependent effects of NR on blood and liver NAD metabolomes in people and in mice, respectively. We report that human blood cell NAD+ can rise as much as 2.7-fold with a single dose of NR, that NR elevates mouse hepatic NAD+ with distinct and superior kinetics to those of nicotinic acid (NA) and nicotinamide (Nam), and that single oral doses of 100 mg, 300 mg and 1 gram of NR provide dose-dependent increases in the blood cell NAD metabolome in the first controlled clinical study of NR pharmacokinetics. We also report that nicotinic acid adenine dinucleotide (NAAD), which is not thought to be en route for conversion of NR to NAD, is a highly sensitive biomarker of effective NAD supplementation.

NAD+ Biosynthesis



NR & Key Metabolites



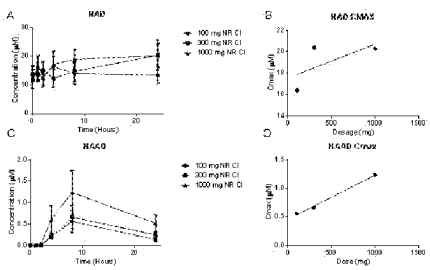
Blood cell NAD metabolites in a 52 year old male who took 1g NR chloride for 7 consecutive days

Time (hr)	NAD+	NAAD	Nam	Me4PY
0	6.0	<0.002	0.80	0.05
0.3	13	<0.002	1.1	0.11
0.6	8.5	<0.002	1.8	0.14
1	12	<0.002	0.80	0.15
1.4	9.3	<0.002	0.93	0.16
2.7	6.0	<0.002	0.76	0.13
4.1	21	<0.002	1.2	0.47
7.7	22	0.26	1.5	0.38
8.1	25	0.45	1.4	1.1
23.8	16	0.17	0.66	0.48
167.6	19	0.22	0.80	0.62

Human n=1 experiment

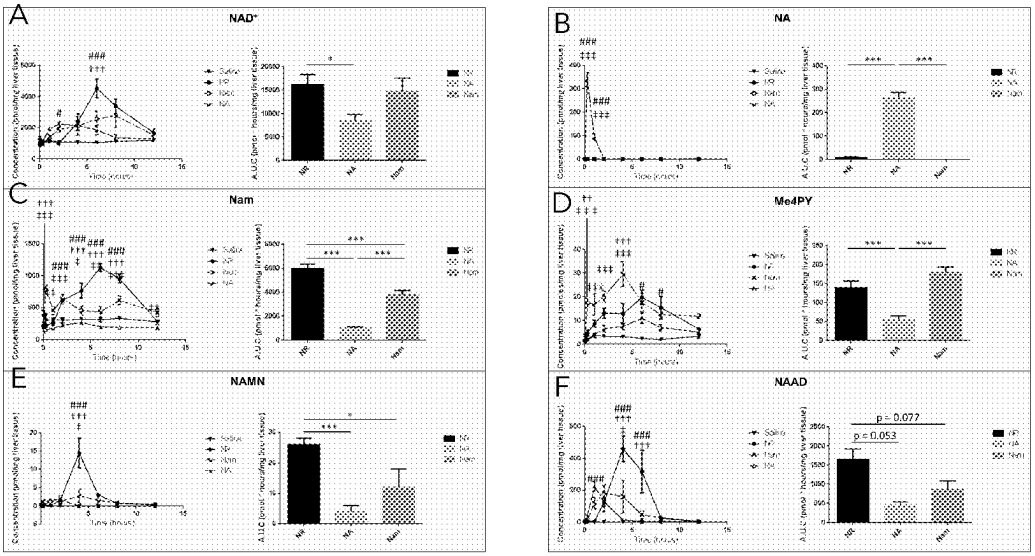
A healthy 65 kg, 52 year old male took 1g of NR orally at 8 AM for 7 consecutive days (15 mg/kg). His blood was taken just prior to ingestion on days 0, 1 and 7, and was taken at 8 time points in the first 8.1 hours after ingestion. Blood was separated into a buffy coat (cellular) fraction and a plasma fraction. Urine was also collected. Table 1 shows the most informative components of the blood cell NAD metabolome on a common scale (1). NAD+ remained constant at ~9.1 microM for at least 2.7 hrs but rose to 2-3 fold basal levels at 4 to 24 hrs after oral doses. Peak NAD+ was at 8.1 hrs and was temporally coincident with peak Nam and N-methyl-4-pyridone-5-carboxamide (Me4PY), a nonsalvageable waste product. Surprisingly, NAAD rose from below 2 nM to above 400 nM. These data suggested that the rise in NAAD might be a 80-fold more sensitive biomarker of effective NAD+ supplementation than the rise in NAD+ itself. (CB and SAJT)

Clinical Trial



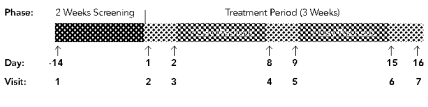
6 healthy male and 6 healthy female volunteers were recruited to take 100 mg, 300 mg and 1 gram doses of NR orally on 3 test dates separated by 7 day washout periods. Plasma, blood cell and urinary NAD metabolomes were determined as a function of time and dose. Blood cell NAD+ and NAAD excursions and Cmax values are shown for the three doses of NR. As expected, blood cell NAD+ levels are high and variable in human populations and changes in blood cell NAD+ are likely affected by diet, circadian oscillation and other factors. Nonetheless, comparing the 24 hr blood cellular NAD+ level for all participants at all doses to the predose NAD+ level , NR elevated NAD+ ($P < 0.05$) and higher doses of NR tended to have greater cellular NAD+ AUCs not because of higher NAD+ Cmax values but because of more temporally extended NAD+ excursions. NAAD exhibited reliable and sensitive dose-dependent excursions with a clear peak at 8 hrs after each dose. (CB, MSS, BJW, FJ and RWD)

Mouse Gavage Experiment



NAD metabolites undergo circadian oscillation(2,3). To eliminate circadian oscillation as an experimental confounder, we developed an oral gavage protocol in which male C57BL/6 mice were given single oral doses of NR at 185 mg/kg 20', 1 hr, 2 hr, 4 hr, 6 hr, 8 hr and 12 hr prior to sacrifice, which was always performed at ~ 2 pm. In addition, mice were dosed with equimole amounts of NA, Nam and a control saline solution, and mice were sacrificed at 2 pm without gavage. Each experimental point included 4 mice (128 mice in total), all of which were sacrificed with freeze-clamping of the liver followed by quantitative targeted NAD metabolomic analysis(1). Six NAD metabolites are shown for all three NAD precursor vitamins plus saline gavage. In the left panels are concentration vs time excursions for the metabolites. In the right panels are baseline-subtracted areas under the curve (AUCs) for the corresponding metabolites. Figure 1A shows that the pharmacokinetics of NR, Nam and NA are distinct with NR having the slowest and greatest degree of hepatic NAD+ formation. Figure 1B shows that NA peaks in the liver 20' after NA gavage and neither NR nor Nam produce a peak of NA at any time. Figure 1C shows that Nam gavage produces a 20' peak of hepatic Nam in addition to a lengthy peak of elevated Nam that corresponds to the hepatic NAD+ excursion. In contrast, NA and NR gavage produce hepatic Nam with kinetics that exclusively correspond to elevated NAD+, suggesting that NR is a hepatic NAD+ precursor through the NR kinase pathway rather than Nam salvage. Surprisingly, Figures 1E and 1F indicate that all three NAD+ precursor vitamins elevate NAMN and NAAD. Though neither metabolite is considered on-pathway for NR(4), NR elevated NAMN and NAAD to a much greater extent than did NA or Nam with a peak at 4 hrs post-gavage. Preliminary experiments with double stable isotope-labeled NR indicate that both the Nam and ribosyl moieties of NR are incorporated into both metabolites. (CB, SAJT, BJW, PR and MEM).

Study Design



Conclusions

Though NAD+ is a highly abundant and variable metabolite in human blood, it is effectively elevated by single 100 mg, 300 mg and 1g oral doses of NR and this can be seen in small, controlled clinical studies. NAAD is a sensitive, novel biomarker of effective NAD supplementation.

References

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2. Ramsey et al, Science 324, p 651, 2009
3. Nakahata et al, Science 324, p 654, 2009
4. Bogan & Brenner, Ann Rev Nutr 28, p 115, 2008