UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): **December 6, 2019**

CARDAX, INC.
(Exact name of registrant as specified in its charter)

Delaware	**333-181719**	**45-4484428**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

2800 Woodlawn Drive, Suite 129, Honolulu, Hawaii 96822
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: **(808) 457-1400**

Not applicable
(Former name or former address, if changed since last report)

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol	Name of each exchange on which registered
Common Stock, par value $0.001 per share	CDXI	**OTCQB**

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

[ ] Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

[ ] Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a -12)

[ ] Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d -2(b))

[ ] Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e -4(c))

Item 7.01 Regulation FD Disclosure

On December 6, 2019, Cardax, Inc., a Delaware corporation (the "Company"), made available a corporation presentation, which is attached hereto as Exhibit 99.1. A copy of this presentation has been made available on the Company's corporate website at www.cardaxpharma.com on the date of this report.

In accordance with General Instruction B.2 of Form 8-K, the information set forth herein and in the presentation is deemed to be "furnished" and shall not be deemed to be "filed" for purposes of the Exchange Act. The information set forth in Item 7.01 of this Current Report on Form 8-K shall not be deemed an admission as to the materiality of any information in this Current Report on Form 8-K that is required to be disclosed solely to satisfy the requirements of Regulation FD.

Forward-Looking Statements

This filing includes statements that are not historical facts. These "forward-looking statements" can be identified by use of terminology such as "anticipate," "believe," "estimate," "expect," "hope," "intend," "may," "plan," "positioned," "project," "propose," "should," "strategy," "will," or any similar expressions. You should be aware that these forward-looking statements are subject to risks and uncertainties that are beyond our control. Although we believe that our assumptions underlying such forward-looking statements are reasonable, we do not guarantee our future performance, and our actual results may differ materially from those contemplated by these forward-looking statements. Our assumptions used for the purposes of the forward-looking statements specified in the following information represent estimates of future events and are subject to uncertainty as to possible changes in economic, legislative, industry, and other circumstances, including the development, acceptance and sales of our products and our ability to raise additional funding sufficient to implement our strategy. As a result, the identification and interpretation of data and other information and their use in developing and selecting assumptions from and among reasonable alternatives require the exercise of judgment. In light of these numerous risks and uncertainties, we cannot provide any assurance that the results and events contemplated by our forward-looking statements will in fact transpire. These forward-looking statements are not guarantees of future performance. You are cautioned to not place undue reliance on these forward-looking statements, which speak only as of their dates. We do not undertake any obligation to update or revise any forward-looking statements.

Item 9.01. Financial Statements and Exhibits

Exhibit No.	Description
99.1	Corporate Presentation

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the Company has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: December 6, 2019

CARDAX, INC.

By: */s/ David G. Watumull*
David G. Watumull
Chief Executive Officer and President

CORPORATE PRESENTATION

December 2019

CERTAIN DISCLAIMERS

There are statements in this presentation that are not historical facts. These "forward-looking statements" can be identified by use of terminology such as "anticipate," "believe," "estimate," "expect," "hope," "intend," "may," "plan," "positioned," "project," "propose," "should," "strategy," "will," "would," or any similar expressions. Forward-looking statements also include, but are not limited to, statements regarding the development and potential applications of our products and product candidates, including our discussion of clinical trial interim results, which are not necessarily indicative of final results. The interim results described herein do not ensure that the final results of the CHASE clinical trial or other clinical trials will be positive or statistically significant or clinically meaningful. Our characterizations of the CHASE clinical trial interim results are forward-looking statements and may not be replicated by the final results of the CHASE clinical trial or other clinical trials. The p-values for the CHASE clinical trial interim results reported herein are nominal p-values from non-parametric comparisons of the median between each group and placebo and no adjustments for multiple comparisons were made. There can be no assurance that we will successfully develop or commercialize our products or product candidates or that the results described herein will adequately support additional intellectual property protection. You should be aware that these forward-looking statements are subject to risks and uncertainties that are beyond our control. For a discussion of these risks and their potential impact to the information provided in this presentation, you should read the information that we have filed with the Securities and Exchange Commission, including the reports filed pursuant to the Securities Exchange Act of 1934, as amended, especially the risks discussed under the section entitled "Risk Factors" included in such reports. In light of these numerous risks and uncertainties, we cannot provide any assurance that the results and events contemplated by our forward-looking statements contained in this presentation will in fact transpire. You are cautioned to not place undue reliance on these forward-looking statements, which speak only as of their dates. We do not undertake any obligation to update or revise any forward-looking statements.

Unless otherwise indicated, information contained in this presentation concerning our company, our business, the services we provide and intend to provide, our industry and our general expectations concerning our industry are based on management estimates. Such estimates are derived from publicly available information released by third party sources, as well as data from our internal research, and reflect assumptions made by us based on such data and our knowledge of the industry, which we believe to be reasonable. However, we cannot provide any assurance that these assumptions or estimates will be accurate or events that we expect will in fact transpire.

Various third-party brands and logos depicted in this presentation are registered and common law trademarks of their respective owners and are used solely for illustrative and comparative purposes. The Company and its products have not been approved, sponsored, or endorsed by these entities, with which the Company has no affiliation, relationship, or arrangement. Quotes used in this presentation by unaffiliated persons do not indicate any endorsement or recommendation by any such person.

No securities are being offered by Cardax through this presentation. This presentation does not contain any material nonpublic information about or regarding Cardax. If you would like additional information, please contact Cardax.

CARDAX OVERVIEW

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- Cardax is a development stage biopharmaceutical company founded in 2006 (OTCQB:CDXI)
- Focused on development of pharmaceuticals to safely address one of the major underlying causes of many chronic diseases – inflammation
- Innovative product platform based on xanthophyll carotenoids (astaxanthin and zeaxanthin) – powerful anti-inflammatory agents with pleiotropic effects and excellent safety profiles
- Lead pharmaceutical candidate (CDX-101) in pre-clinical development for treatment of cardiovascular inflammation and dyslipidemia, with initial indication of severe hypertriglyceridemia (similar to Amarin's clinical pathway for Vascepa)
- Commercial business unit markets ZanthoSyn®, a physician recommended dietary supplement for inflammatory health

MANAGEMENT TEAM

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- **David G Watumull** – *Chief Executive Officer*
  Co-founder of Cardax and co-inventor of technology. Experienced biotech executive, former biotech analyst and investment banker.
- **David M Watumull** – *Chief Operating Officer*
  Two decades of experience in astaxanthin product development, commercialization, and business management.
- **John Russell, CPA** – *Chief Financial Officer*
  Accounting, finance, operations, and SEC reporting professional with over 20 years experience. Formerly with Grant Thornton and PwC.
- **Paresh Soni, MD, PhD** – *Chief Clinical and Regulatory Strategist*
  Former Senior Vice President and Head of Development at Amarin. Led development and regulatory approval for Vascepa.
- **Gilbert Rishton, PhD** – *Chief Science Officer*
  Built Amgen's Small Molecule Drug Discovery Group and served as chemistry manager for Sensipar development program.
- **Jon Ruckle, MD** – *Chief Medical Officer*
  PI of more than 350 clinical trials. Former Medical Director at Covance.
- **Timothy King, PhD** – *Vice President, Research*
  Expert on MOA and biological applications of astaxanthin.
  Former staff scientist at Fred Hutchinson Cancer Research Center.
- **Randall Mau** – *Vice President, Medical & Business Relations*
  Former Account Manager at Pfizer; grew market share and revenues.
- **Gilbert Shin** – *Vice President, Retail Sales & Marketing*
  Former Regional Sales Director of top performing GNC region in US.

BOARD OF DIRECTORS

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- **George W Bickerstaff** – *Chairman*
  Former Chief Financial Officer of Novartis Pharma.
- **David G Watumull** – *Director*
  Chief Executive Officer of Cardax.
- **Terence A Kelly, PhD** – *Director*
  Former research executive with Boehringer Ingelheim.
- **Michele Galen** – *Director*
  Former communications executive with Shire and Novartis.
- **Makarand Jawadekar, PhD** – *Director*
  Former research executive with Pfizer.
- **Elona Kogan** – *Director*
  Biotech business executive. Formerly with Ariad and Avanir.

SCIENTIFIC ADVISORY BOARD

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- **Deepak Bhatt, MD, MPH** – *SAB Chairman*
  Executive Director of Interventional Cardiovascular Programs at Brigham and Women's Hospital. Professor of Medicine at Harvard Medical School. Chair of REDUCE-IT clinical trial.

- **Paresh Soni, MD, PhD** – *SAB Member*
  Cardax Chief Clinical and Regulatory Strategist. Former Senior Vice President and Head of Development at Amarin. Led development and regulatory approval for Vascepa.

- **R Preston Mason, PhD** – *SAB Member*
  Harvard Medical School / Brigham and Women's Hospital. Expert on MOA of astaxanthin and fish oils, including Vascepa.


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KEY OPINION LEADERS AGREE
Inflammation = Major Unmet Medical Need in Cardiovascular Disease (CVD)
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IMPORTANT EXAMPLE

10,061 Patient CANTOS Trial (Novartis)

⬇️ Inflammation = ⬇️ CV Events

VALIDATING THE INFLAMMATORY HYPOTHESIS

TARGETING INFLAMMATION
TO REDUCE CVD RISK

Ridker et al. New Engl J Med, 377:1119-1131, 2017

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CANTOS Trial

Canakinumab ANti-inflammatory Thrombosis Outcome Study

- **Randomized, double-blind, placebo controlled**
- **Subjects**:
  - 10,061 cardiovascular patients, 39 countries
  - Standard of care (including statins)
  - Elevated inflammation (CRP > 2 mg/L)
- **Agent**: Canakinumab (anti-inflammatory drug, Novartis)
- **Duration**: 4 years
- **Results**:
  - No change in lipids
  - **REDUCTION OF INFLAMMATION (CRP < 2 mg/L) =**
    - Heart attacks & strokes ⬇ 25%
    - Cardiovascular death ⬇ 31%
    - All-cause mortality ⬇ 31%

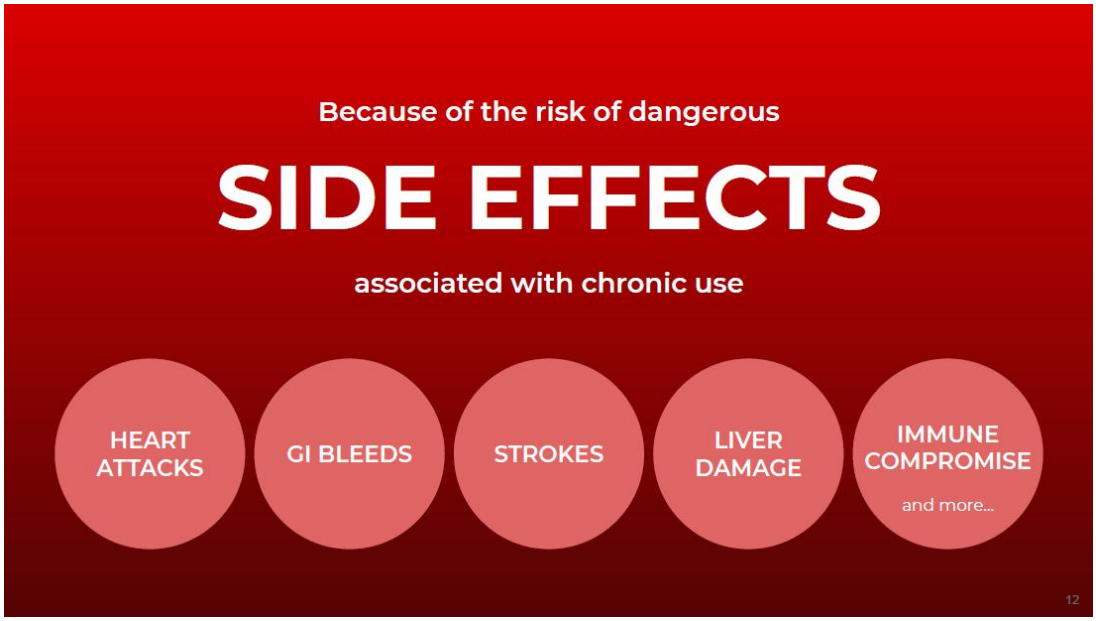
Response to **The CANTOS Trial:**

"It (this study) opens up an entirely new vista for the treatment of heart disease, because now *everybody on the planet*—in the pharmaceutical industry and in research institutions like ours and at the National Institutes of Health—*are going to be looking to find anti-inflammatory therapies*."

- Steve Nissen, MD
Chairman of Cardiovascular Medicine
Cleveland Clinic

Washington Post, August 27, 2017

Why not manage *chronic* inflammation with other leading anti-inflammatories?


Because of the risk of dangerous
SIDE EFFECTS
associated with chronic use
HEART ATTACKS
GI BLEEDS
STROKES
LIVER DAMAGE
IMMUNE COMPROMISE
and more...
12

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KEY OPINION LEADERS AGREE

***Pleiotropic Effects* on CVD Markers**
= Major CVD Benefit

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IMPORTANT EXAMPLE

8,179 Patient REDUCE-IT Trial (Amarin)

⬇️ CVD Markers = ⬇️ CV Events

PLEIOTROPIC BENEFITS REDUCE CVD RISK

Bhatt et al. New Engl J Med, 380:11-22, 2019

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REDUCE-IT Trial

Reduction of Cardiovascular Events–Intervention Trial

- **Randomized, double-blind, placebo controlled**
- **Subjects**:
  - 8,179 statin treated patients with elevated CV risk
  - Elevated triglycerides (median baseline 216 mg/dL)
- **Agent**: Vascepa (fish oil derived drug, Amarin), 4 g/day
- **Pleiotropic Mechanism of Action**:
  - Cellular functions related to atherosclerosis & CV events
  - Lipids, lipoproteins
  - Inflammation
- **Duration**: 5 years
- **Results**:
  - **Major adverse cardiovascular events ⬇ 25%**
  - Robust efficacy across multiple secondary endpoints
  - Well tolerated

Why not manage *chronic* inflammation and dyslipidemia with prescription fish oils?

DISADVANTAGES OF FISH OIL DRUGS

DOSING CHALLENGES	Oral dosing of large fish oil capsules is problematic
SCALABILITY LIMITATIONS	Fish oil manufacturing is limited by the declining global fish supply
SAFETY RISKS	Fish oils have certain safety risks*

*Safety risks of prescription fish oils: Lovaza and other DHA, EPA combination fish oil drugs have risks of side effects including back pain, eructation, dysgeusia, and increases in LDL cholesterol; Vascepa has risks of side effects including arthralgia, atrial fibrillation, and increased bleeding.

THE SOLUTION REQUIRES A UNIQUE COMBINATION OF BENEFITS

CARDAX PRODUCT PLATFORM

COMPETITIVE ADVANTAGES

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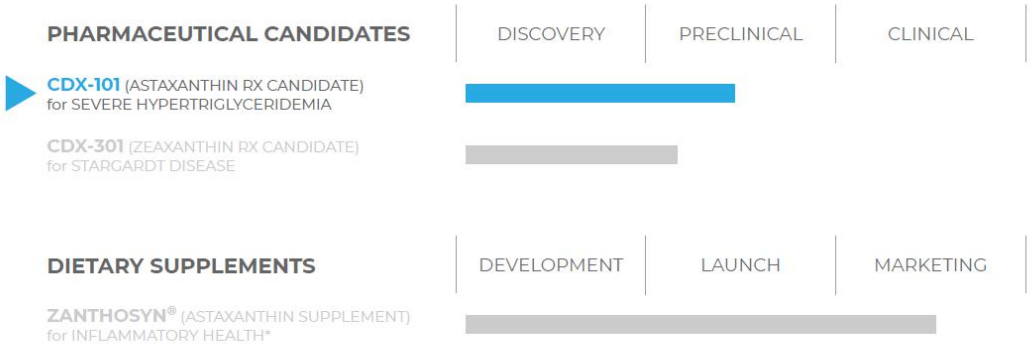
UNIQUE COMBINATION OF BENEFITS:

- ✓ **Excellent safety profile** that supports chronic use
- ✓ **Broad anti-inflammatory** activity & pleiotropic effects
- ✓ **Oral dosing** convenience & ease of administration
- ✓ **Scalable** manufacturing
- ✓ **Economical** pricing

CARDAX PRODUCT PLATFORM

CARDAX PRODUCT PLATFORM



CDX-101

ASTAXANTHIN PHARMACEUTICAL CANDIDATE

Potential applications include:

- Cardiovascular Disease
- Metabolic Disease
- Liver Disease
- Arthritis
- Aging

**Including earlier generations of CDX-101 (same active ingredient) and interim results from Cardax CHASE clinical trial*

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- **CDX-101:** Proprietary prodrug of astaxanthin with broad anti-inflammatory activity, pleiotropic effects, excellent safety
- **Primary Therapeutic Area:** Cardiovascular disease (cardiovascular inflammation and mixed dyslipidemia)
- **Proof of Concept**: Human & animal studies with astaxanthin,* which we believe provide mechanistic support (reduced inflammation & lipids) and support excellent safety profile
- **Initial Indication**: Severe hypertriglyceridemia (SHTG)
  - Efficient clinical pathway to drug approval for CDX-101; similar to Amarin's clinical pathway for Vascepa
  - 3.4 million Americans with SHTG; prescription fish oils approved for SHTG have global market near $2 billion
- **Competitive Advantages**: Excellent safety profile, ease of dose administration, and manufacturing scalability
- **Intellectual Property**
  - Patents issued (legacy): composition of matter and pharmaceutical use through mid-2020s
  - Patents pending (new): composition of matter and pharmaceutical use through 2039-2040
- **Development Stage**: Pre-clinical (target: IND Q4 2020 / Q1 2021)

WHAT IS ASTAXANTHIN?

Astaxanthin is a **naturally occurring marine carotenoid** found in salmon, microalgae, krill, lobster, and crab.

Carotenoids are natural pigments that impart coloration and support animal health and vitality.

Astaxanthin is responsible for turning salmon and shellfish pink.


WITHOUT ASTAXANTHIN, SALMON ARE:
Grey
Small
Have reproductive problems
Prone to infections
Too weak to swim upstream
24

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ASTAXANTHIN SAFETY

No significant side effects reported in published human studies (over 1,800 subjects)

- Long history of use in humans and animals
- Extensive safety testing (see table on next slide)

Source: ncbi.nlm.nih.gov

ASTAXANTHIN SAFETY

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No clinically meaningful safety issues found even at extremely high doses:

TYPE OF STUDY	MAXIMUM DOSING (mg/kg)
Acute Toxicity	>8,000 (mouse, rat), 2,000 (non-human primates)
Sub-Chronic Toxicity	1,240 (rat), 160 (dog)
1 Year Chronic Toxicity/Carcinogenicity	1,000 (rat), 1,400 (mouse), 200 (dog)
2 Year Carcinogenicity	1,000 (rat)
Genotoxicity/Mutagenicity	2,000 (mouse)
Teratogenicity	1,000 (rat), 400 (rabbit)

ASTAXANTHIN MECHANISM OF ACTION

- Astaxanthin spans and stabilizes cellular and mitochondrial membrane
  (see figures on this slide and next slide)
- Reduces pathological activation of inflammatory pathways by modulating oxidative stress in cells and mitochondria
- Does not inhibit normal function (supports excellent safety profile)

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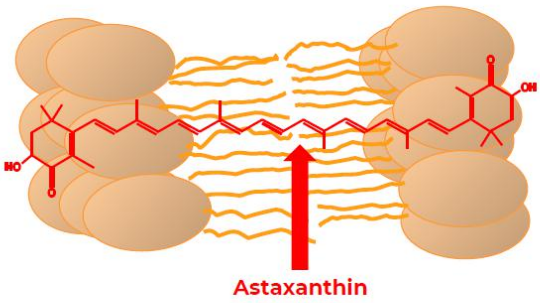
CELLULAR AND MITOCHONDRIAL LOCALIZATION AND FUNCTIONALITY



Astaxanthin

ASTAXANTHIN MECHANISM OF ACTION

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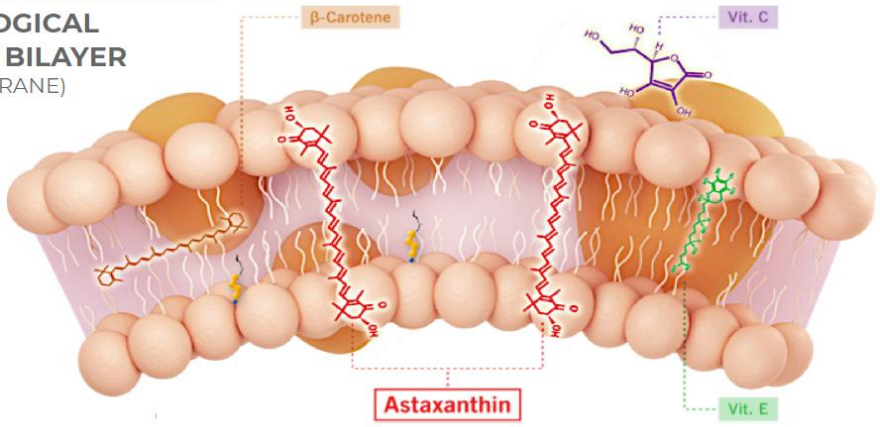
SPANS & STABILIZES BIOLOGICAL LIPID BILAYER
(MEMBRANE)



ASTAXANTHIN MECHANISM OF ACTION

Astaxanthin demonstrates positive and quantifiable pleiotropic effects on many inflammatory cytokines and drug targets:

- IL-1β (canakinumab)
- COX-2 (Celebrex)
- PGE-2 (aspirin)
- TNF-α (Humira, Remicade, Enbrel)
- NF-kβ (steroids)

In human proof-of-concept "pilot" studies and animal studies conducted by third parties, astaxanthin statistically significantly decreased inflammation and oxidative stress.

REDUCTION OF INFLAMMATION

HUMAN STUDIES

- **TNF-α decreased** (-30%, p=0.0022)
- **CRP decreased** (-20%, p<0.05, two studies)
- **Oxidative stress decreased** (MDA, IsoP, SOD, TAC increased)

ANIMAL STUDIES

- **Inflammatory markers decreased** in various model systems:
  - **TNF-α, IL-1β, IL-6, CRP, NF-κB, PGE-2, iNOS, MCP-1, MPO, ERK, JNK, COX-2**
  - **TNF-α decreased** equivalent to equal dose of prednisolone
- **Oxidative stress decreased** in mitochondria

ASTAXANTHIN MECHANISM OF ACTION

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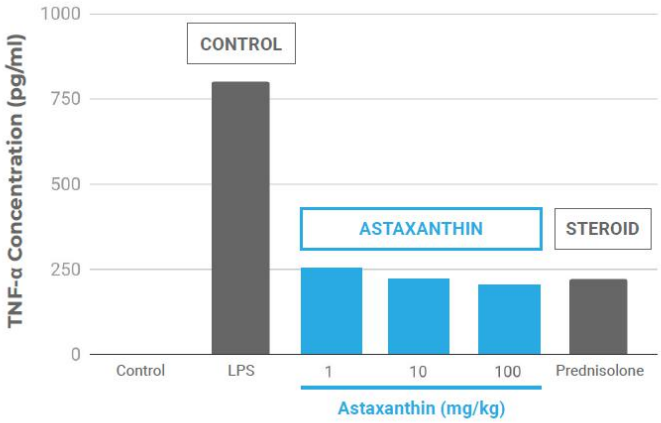
ASTAXANTHIN REDUCED TNF-α IN INFLAMMATORY ANIMAL MODEL = PREDNISOLONE

Effect of astaxanthin on TNF-α concentrations in aqueous humor. The aqueous humor was collected 24 hours after lipopolysaccharide (LPS) treatment. Each value represents mean ± SD (n=8). The dose of prednisolone was 10 mg/kg. p<0.01, compared with the LPS group.



Ohgami et al. Ophth. Invest. Vis. Sci. 44(6):2694-2701, 2003

ASTAXANTHIN RESEARCH

2,000+ peer reviewed papers

More than 50 peer reviewed papers published by Cardax team members

50+ pilot human clinical trials

20+ randomized, double-blind, placebo-controlled human proof-of-concept studies

ASTAXANTHIN THERAPEUTIC AREAS*

COMMONALITIES

- Inflammation
- Oxidative Stress

*Potential therapeutic areas for CDX-101 pharmaceutical development

INITIAL FOCUS

ASTAXANTHIN & CARDIOVASCULAR DISEASE

In human proof-of-concept "pilot" studies conducted by third parties, astaxanthin statistically significantly decreased inflammation, triglycerides, LDL cholesterol, and blood pressure.

In animal studies conducted by third parties and us, astaxanthin demonstrated statistically significant improvements in models of cardiovascular disease.

**Does not include Cardax CHASE clinical trial interim results, which are described on later slides.*

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Human Studies*

- **CRP decreased** (-20%, p<0.05, two studies)
- **Triglycerides decreased** (-25.8%, p<0.05)
- **LDL-C decreased** (-10.4%, p<0.05)
- **HDL-C increased** (+14.5%, p<0.01)
- **Apolipoprotein B decreased** (-7.5%, p<0.01)
- **Adiponectin increased**
  - Three studies: +26% (p<0.01), +14% (p=0.0053), +30% (p=0.01)
- **Blood pressure decreased** (two studies)
  - SBP -4.6% (p=0.021), DBP -6.9% (p<0.001)
- **Blood flow velocity increased**
  - Choroidal (p=0.018), blood transit time (p<0.01)

Animal Studies

- **CRP and IL-6 decreased**
- **Triglycerides decreased** (plasma, hepatic)
- **Re-thrombosis decreased**
- **Atherosclerosis decreased** (aortic arch plaque)
- **Cholesterol decreased**
- **Blood pressure decreased**
- **Nitric oxide production increased**

ASTAXANTHIN & CARDIOVASCULAR DISEASE

CARDAX STUDIES

provide mechanistic support
for pharmaceutical development

Cardax studies presented herein utilized Cardax synthetic astaxanthin (ZanthoSyn®) or related prodrugs (i.e., earlier generations of CDX-101, same active ingredient).

Cardax CHASE clinical trial interim results displayed as median percentage changes from baseline to week 12.

**p<0.05 **p<0.01*

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- **Cardax CHASE Clinical Trial Interim Results (9/23/19)**

Interim Results (40 subjects, 12 weeks)	**High Dose** (96 mg/day)	**Low Dose** (24 mg/day)	**Placebo** (0 mg/day)
CRP	↓ 28%	↓ 32%	↓ 5%
LDL-C	↓ 12% **	↓ 7%	↑ 5%
Total cholesterol	↓ 8% *	↓ 5%	↑ 4%
Triglycerides	↓ 16%	↓ 13%	↑ 6%
Oxidized LDL	↓ 10% *	↑ 3%	↑ 4%
Blood pressure	↓ 5% *	↓ 4% *	↑ 6%

- **Cardax Animal Studies**
  - **Reduced triglycerides** 72% in ApoE(-/-) mice
  - **Reduced re-thrombosis** 84% in dogs
  - **Reduced atherosclerosis** in LDLR(-/-) mice

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ASTAXANTHIN & CARDIOVASCULAR DISEASE

KEY POINTS

- Reduces inflammation
- Improves lipid profiles
- Lowers blood pressure
- Decreases artery plaque formation in animals

ASTAXANTHIN & METABOLIC DISEASE

In human proof-of-concept "pilot" studies conducted by third parties, astaxanthin statistically significantly increased adiponectin and decreased TNF-α and oxidative stress.

In animal studies conducted by third parties, astaxanthin demonstrated statistically significant improvements in models of metabolic disease.

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Human Studies

- **Adiponectin increased**
  - Three studies: +26% (p<0.01), +14% (p=0.0053), +30% (p=0.01)
- **TNF-α decreased** (-30%, p=0.0022)
- **Oxidative stress decreased** (MDA, IsoP, SOD, TAC increased)

Animal Studies

- **Fasting blood glucose levels decreased**
- **Insulin levels & sensitivity** (HOMA-IR, QUICK) **increased**
- **Insulin signaling** (PI3K-AKT, IRS-1p) **increased**
- **Adiponectin levels increased**
- **Insulin response and glucose tolerance** (ipGTT) increased
- **GLUT-4 translocation increased**
- **JNK, ERK-1 levels decreased**
- **Nitric oxide production increased**

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ASTAXANTHIN & METABOLIC DISEASE

KEY POINTS

- Reduces inflammation
- Increases adiponectin levels
- Improves blood glucose & insulin levels in animals
- Increases insulin signaling/response in animals

ASTAXANTHIN & LIVER DISEASE

In human proof-of-concept "pilot" studies conducted by third parties, astaxanthin statistically significantly decreased fat accumulation in biopsy-diagnosed NASH patients, decreased TNF-α, improved lipid profile parameters, and decreased oxidative stress.

In animal studies conducted by third parties and us, astaxanthin statistically significantly decreased elevated liver enzymes, lipids, insulin resistance, steatosis, and fibrosis.

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Human Studies

- **NASH disease markers decreased in patients**
  - Steatosis: $p<0.05$
  - NAFLD Activity Score (NAS): $p<0.08$
  - Lobular inflammation decreased: trend
- **TNF-α decreased** (-30%, $p=0.0022$)
- **Lipid profile parameters improved** (LDL, HDL, ApoB, TG)
- **Oxidative stress decreased** (MDA, IsoP, SOD, TAC increased)

Animal Studies

- **Elevated liver enzyme levels decreased**
- **Steatosis decreased**
- **Fibrosis and induced acute hepatitis decreased**
- **Insulin levels & sensitivity** (HOMA-IR, QUICK) **increased**
- **Insulin signaling** (PI3K-AKT, IRS-1p) **increased**
- **Adiponectin levels increased**

ASTAXANTHIN & LIVER DISEASE

KEY POINTS

- Decreases liver fat (steatosis)
- Reduces inflammation and oxidative stress
- Decreases elevated liver enzyme levels in animals
- Improves fibrosis and insulin response in animals

ASTAXANTHIN & ARTHRITIS

In human proof-of-concept "pilot" non-arthritis studies conducted by third parties, astaxanthin statistically significantly decreased markers of inflammation of relevance to arthritis, including TNF-α and CRP.

In animal studies conducted by third parties, astaxanthin statistically significantly decreased inflammation, oxidative stress, and joint degeneration.

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Human Studies

- **TNF-α decreased** (-30%, p=0.0022)
- **CRP decreased** (-20%, p<0.05, two studies)
- **Adiponectin increased**
  - Three studies: +26% (p<0.01), +14% (p=0.0053), +30% (p=0.01)
- **Oxidative stress decreased** (MDA, IsoP, SOD, TAC increased)

Animal Studies

- **Inflammatory markers decreased** in various model systems:
  - **TNF-α, IL-1ß, IL-6, CRP, NF-kB, PGE-2, iNOS, MCP-1, MPO, ERK, JNK, COX-2**
  - **TNF-α decreased equivalent to equal dose of prednisolone**
- **Oxidative stress decreased** in mitochondria
- **Cartilage degradation decreased** (Mankin score)
  - Surgically-induced model of OA (ACLT, rabbit)

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ASTAXANTHIN & ARTHRITIS

KEY POINTS

- Reduces inflammation
- Lowers oxidative stress
- Decreases joint degeneration in animal OA model
- Reduces major inflammatory markers in animals

ASTAXANTHIN & AGING

In human studies conducted by third parties, activation of the FOXO3 gene has been linked to decreased inflammation and aging.

In animal studies conducted by third parties and us, astaxanthin statistically significantly activated the FOXO3 gene and extended lifespan.

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Background

- Activation of anti-inflammatory, anti-aging gene FOXO3 promotes longevity in humans
  - Replicated in >20 independent studies
  - Confers CVD protective benefit (p=0.001)
  - Decreases inflammation (CRP, trend; TNF-α, p=0.018)

Animal Studies

- **FOXO3 mRNA levels increased** in mice by 90% (p=0.024)
- **Lifespan extended** by up to 30% via FOXO3 ortholog *DAF16* in roundworms

CDX-101

ASTAXANTHIN PHARMACEUTICAL CANDIDATE

In Summary

**Including earlier generations of CDX-101 (same active ingredient)*

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- **CDX-101:** Proprietary prodrug of astaxanthin with broad anti-inflammatory activity, pleiotropic effects, excellent safety
- **Primary Therapeutic Area:** Cardiovascular disease (cardiovascular inflammation and mixed dyslipidemia)
- **Proof of Concept**: Human & animal studies with astaxanthin,* which we believe provide mechanistic support (reduced inflammation & lipids) and support excellent safety profile
- **Initial Indication**: Severe hypertriglyceridemia (SHTG) provides efficient clinical pathway to drug approval for CDX-101, similar to Amarin's clinical pathway for Vascepa
- **Competitive Advantages**: Excellent safety profile, ease of dose administration, manufacturing scalability
- **Intellectual Property:** Patents pending for composition of matter and pharmaceutical use through 2039-2040
- **Development Stage**: Pre-clinical (target: IND Q4 2020 / Q1 2021)

CARDAX PRODUCT PLATFORM

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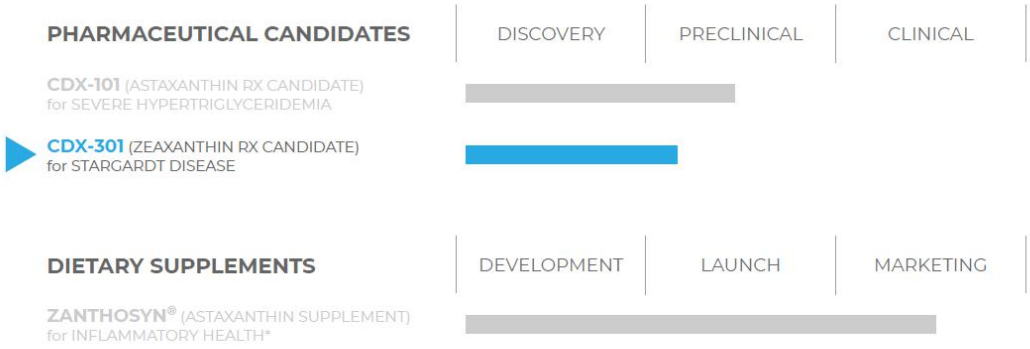

CDX-301

ZEAXANTHIN PHARMACEUTICAL CANDIDATE

Potential applications include:

- Stargardt Disease
- Age-Related Macular Degeneration

**Including earlier generation of CDX-301 (same active ingredient)*

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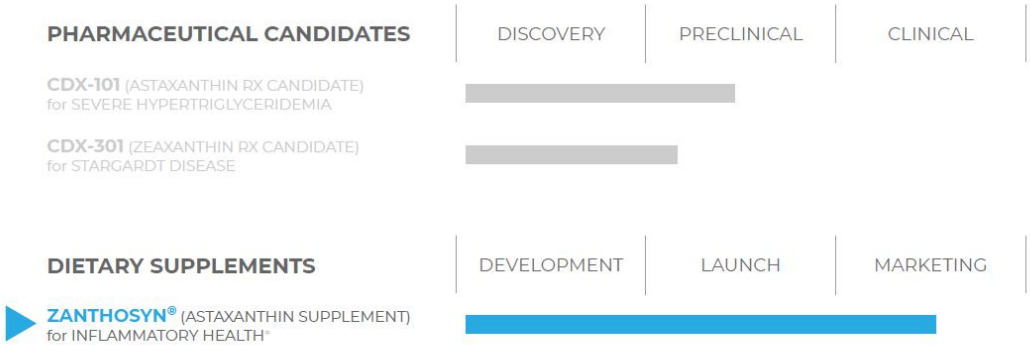
- **CDX-301:** Zeaxanthin pharmaceutical candidate
- **Mechanism of Action:** Zeaxanthin accumulates in human eye via retinal receptor and provides protection against blue light, oxidative damage, and inflammation that occurs in macular degeneration
- **Therapeutic Area:** Macular degeneration
- **Proof of Concept:** Human and animal studies with zeaxanthin* provide mechanistic support for treatment of macular disorders and support excellent safety profile
- **Initial Indication**: Stargardt disease (STGD), a juvenile form of macular degeneration
  - Efficient clinical pathway to drug approval for CDX-301
  - Potential orphan drug designation (≤42,000 in US with STGD)
- **Second Indication**: Age-related macular degeneration (AMD)
  - Large market opportunity (>3 million in US with AMD) but with increased competition
- **Development Stage**: Pre-clinical

CARDAX PRODUCT PLATFORM



ZANTHOSYN® OVERVIEW

Superior Absorption
- 2.85x better absorption vs. ordinary astaxanthin

Superior Purity
- Precision & purity (cGMP)
- No aftertaste or smell

Superior Safety
- Generally Recognized as Safe according to FDA regulations

Health applications include:
- Cardiovascular Health*
- Metabolic Health*
- Liver Health*
- Joint Health*
- Longevity*
- Fitness*

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ZanthoSyn® is a physician recommended astaxanthin dietary supplement for inflammatory health*

* These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.

ZANTHOSYN® ASTAXANTHIN ABSORPTION

- AUC: 2.85-fold greater
  - p=0.013
- C_{max}= 3.0-fold greater
  - p=0.013
- Coefficient of variation
  - ZanthoSyn = 27%
  - Ordinary asta = 62%
- T_{max}= 6 hours
- No adverse events

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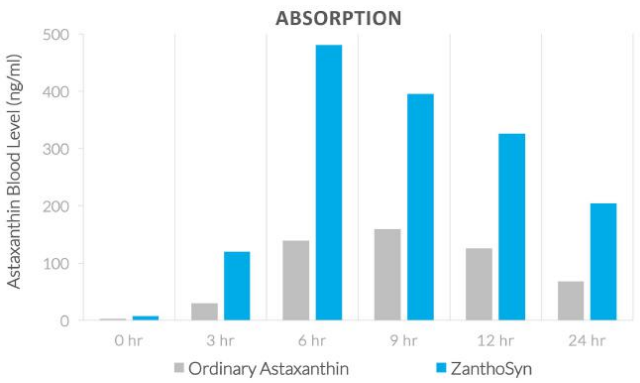

ZANTHOSYN® CLINICAL TRIAL (ongoing)

TARGETING CV HEALTH

Interim results provide:

- Mechanistic support for Rx
- Basis for additional patent filings
- Support for ZanthoSyn® marketing

*Interim results announced 9/23/2019 displayed as median percentage changes from baseline to week 12. *p<0.05 **p<0.01*

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CHASE Clinical Trial

Cardiovascular Health Astaxanthin Supplement Evaluation

- **Randomized, double-blind, placebo controlled, IRB approved**
- **Subjects:** Up to 120 subjects with CV risk factors and CRP > 2 mg/L
- **Primary Endpoint**: Cardiovascular health as measured by CRP
- **Other Endpoints**: Pre-specified secondary cardiovascular and inflammatory health markers, safety parameters, exploratory endpoints
- **Duration**: 12 weeks with open-label extension through 48 weeks

Interim Results (40 subjects, 12 weeks)	**High Dose** (96 mg/day)	**Low Dose** (24 mg/day)	**Placebo** (0 mg/day)
CRP	↓ 28%	↓ 32%	↓ 5%
LDL-C	↓ 12% **	↓ 7%	↑ 5%
Total cholesterol	↓ 8% *	↓ 5%	↑ 4%
Triglycerides	↓ 16%	↓ 13%	↑ 6%
Oxidized LDL	↓ 10% *	↑ 3%	↑ 4%
Blood pressure	↓ 5% *	↓ 4% *	↑ 6%

ZANTHOSYN® MARKETING

ZanthoSyn® provides a combination of safety, purity, manufacturing rigor, bioavailability, and scientific support not often present in other supplements.

ZanthoSyn® is well-accepted at medical conferences where crowds of physicians and other healthcare professionals receive samples after seminars.

ZanthoSyn® is the top selling product at GNC stores in Hawaii and the top selling product in the antioxidant category at GNC stores nationwide.

E-commerce offers convenient fulfillment with recurring shipment functionality and targeted marketing opportunities.

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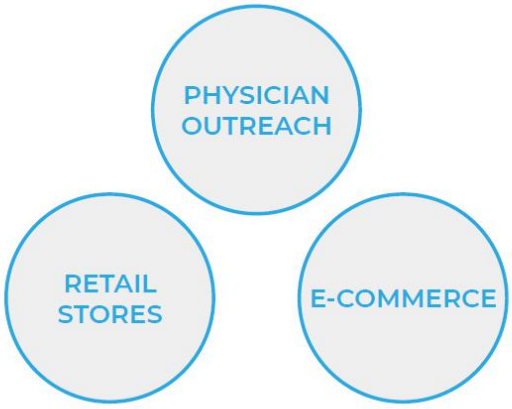
MULTI-PRONGED APPROACH



ZANTHOSYN®

In Summary

- ✓ Clinically Studied
- ✓ Superior Absorption
- ✓ Superior Purity
- ✓ Superior Safety
- ✓ Many Health Applications
- ✓ Multi-Pronged Marketing

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ZanthoSyn® is a physician recommended astaxanthin dietary supplement for inflammatory health*

* These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.

CDX-101 vs. ZANTHOSYN®

While both deliver astaxanthin to the bloodstream, we believe the unique molecular structure of CDX-101 and its pharmaceutical pathway will provide substantial differentiation.

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	CDX-101	ZANTHOSYN®
COMPOSITION	Synthetic Astaxanthin Prodrug (NCE)	Synthetic Astaxanthin Formulation
INTELLECTUAL PROPERTY	Composition of Matter and Use (issued & pending)	Use (pending)
PRODUCT TYPE	Rx Candidate	Dietary Supplement
CHANNEL	Doctor Prescription	Retail & E-Commerce
ECONOMICS	Insurance Coverage	Out of Pocket
DOSAGE	High Dose	Low Dose

INTELLECTUAL PROPERTY

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- **Cardax IP consists of 29 issued patents and 5 patent applications**:
  - 14 patents issued in the United States
  - 15 patents issued in Europe, Canada, China, India, Japan, Hong Kong, and Brazil
  - 4 patent applications pending in United States, Europe, and PCT countries
- **Cardax IP includes**:
  - **Patents issued for composition of matter** covering thousands of carotenoid derivatives
  - **Patents issued for pharmaceutical uses** covering hundreds of indications
  - **Patents pending for CDX-101** composition of matter (2040) and use (2039)

UPCOMING MILESTONES

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- **CDX-101** (astaxanthin Rx candidate)
  - IND filing: Q4 2020 / Q1 2021
- **CHASE Clinical Trial** (astaxanthin supplement study)
  - Final results: 2020

The milestones presented above are our best estimates and subject to adjustment.
There is no assurance such milestones will be achieved upon the specified timelines or at all.

IN SUMMARY

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- Cardax is focused on development of pharmaceuticals to safely address one of the major underlying causes of many chronic diseases – inflammation
- Innovative product platform based on xanthophyll carotenoids (astaxanthin and zeaxanthin) – powerful anti-inflammatory agents with pleiotropic effects, excellent safety profiles, oral dosing convenience, scalable manufacturing, and economical pricing
- Clinical pathway for lead pharmaceutical candidate (CDX-101) similar to Amarin's pathway for Vascepa
- Strong team, intellectual property, clinical results, pre-clinical data, and efficient development pathways support transformative market opportunities
- Commercial business unit markets leading dietary supplement for inflammatory health

THANK YOU