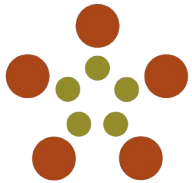

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CORPORATE PRESENTATION

December 2019

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CARDAX OVERVIEW

- 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

- **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

- **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

- **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.

KEY OPINION LEADERS AGREE

Inflammation **= Major Unmet Medical Need in Cardiovascular Disease (CVD)**

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

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...

KEY OPINION LEADERS AGREE

Pleiotropic Effects on CVD Markers = Major CVD Benefit

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

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

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PHARMACEUTICAL CANDIDATES

DISCOVERY | PRECLINICAL | CLINICAL

CDX-101 (ASTAXANTHIN RX CANDIDATE)
for SEVERE HYPERTRIGLYCERIDEMIA

CDX-301 (ZEAXANTHIN RX CANDIDATE)
for STARGARDT DISEASE

DIETARY SUPPLEMENTS

DEVELOPMENT | LAUNCH | MARKETING

ZANTHOSYN® (ASTAXANTHIN SUPPLEMENT)
for INFLAMMATORY HEALTH*

CARDAX PRODUCT PLATFORM

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PHARMACEUTICAL CANDIDATES

DISCOVERY | PRECLINICAL | CLINICAL

CDX-101 (ASTAXANTHIN RX CANDIDATE)
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DIETARY SUPPLEMENTS

DEVELOPMENT | LAUNCH | MARKETING

ZANTHOSYN® (ASTAXANTHIN SUPPLEMENT)
for INFLAMMATORY HEALTH*

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*

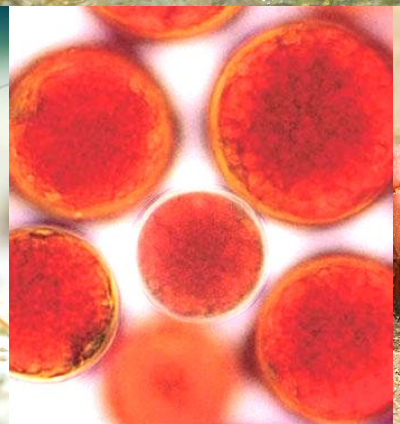
- **CDX-101:** Proprietary prodrug of astaxanthin with broad anti-inflammatory activity, pleiotropic effects, excellent safety
- **Primary Therapeutic Area:** Cardiovascular disease (cardiovascular inflammation and mixed dyslipidemia)
- **Proof of Concept**: Human & animal studies with astaxanthin,* 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

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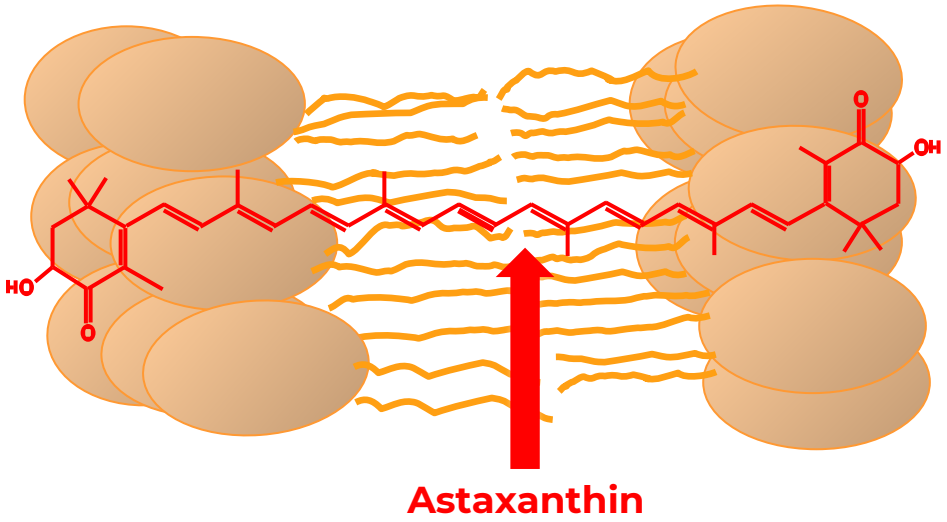
No clinically meaningful safety issues found even at extremely high doses:

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)

CELLULAR AND MITOCHONDRIAL LOCALIZATION AND FUNCTIONALITY



ASTAXANTHIN MECHANISM OF ACTION

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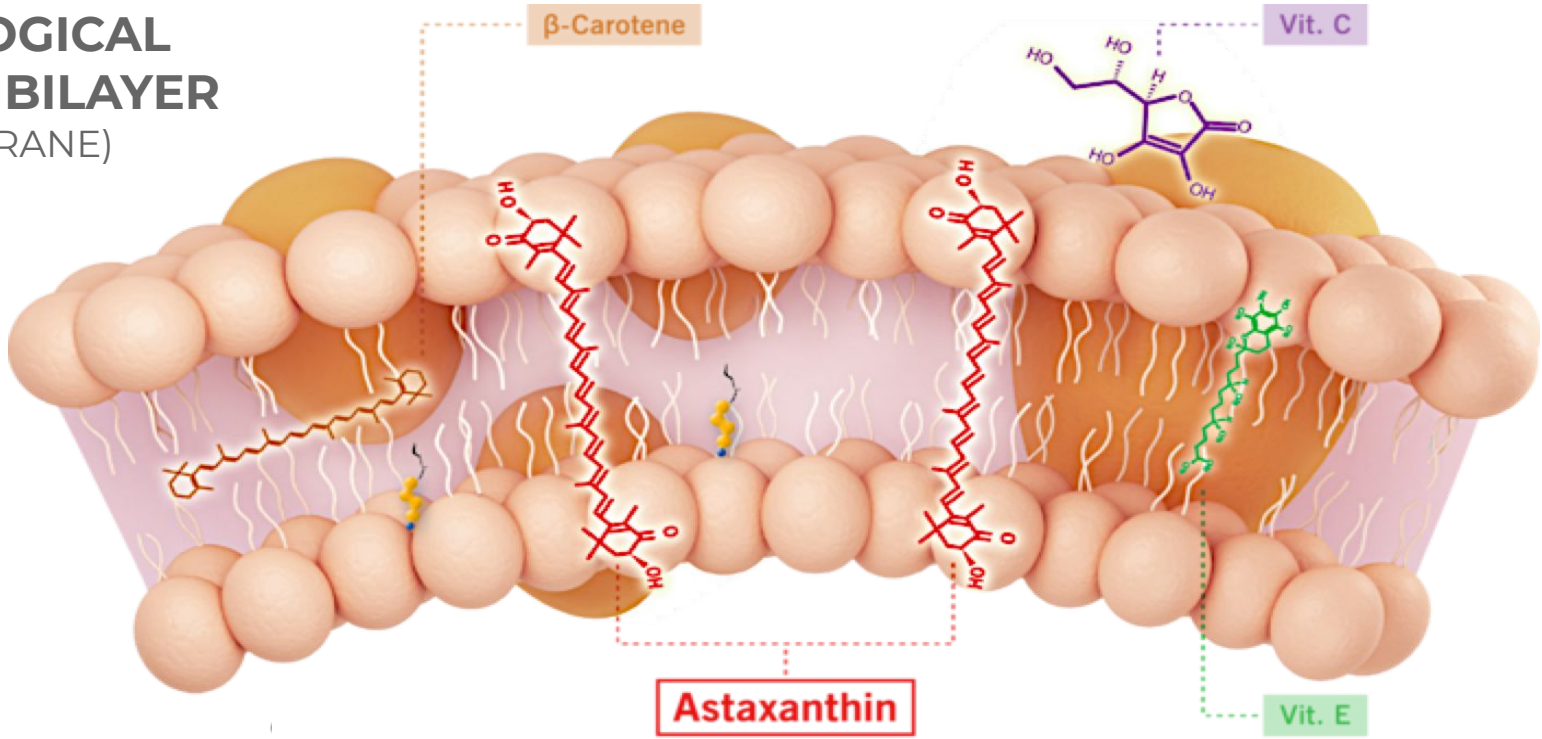
SPANS & STABILIZES BIOLOGICAL LIPID BILAYER
(MEMBRANE)



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-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

ASTAXANTHIN MECHANISM OF ACTION

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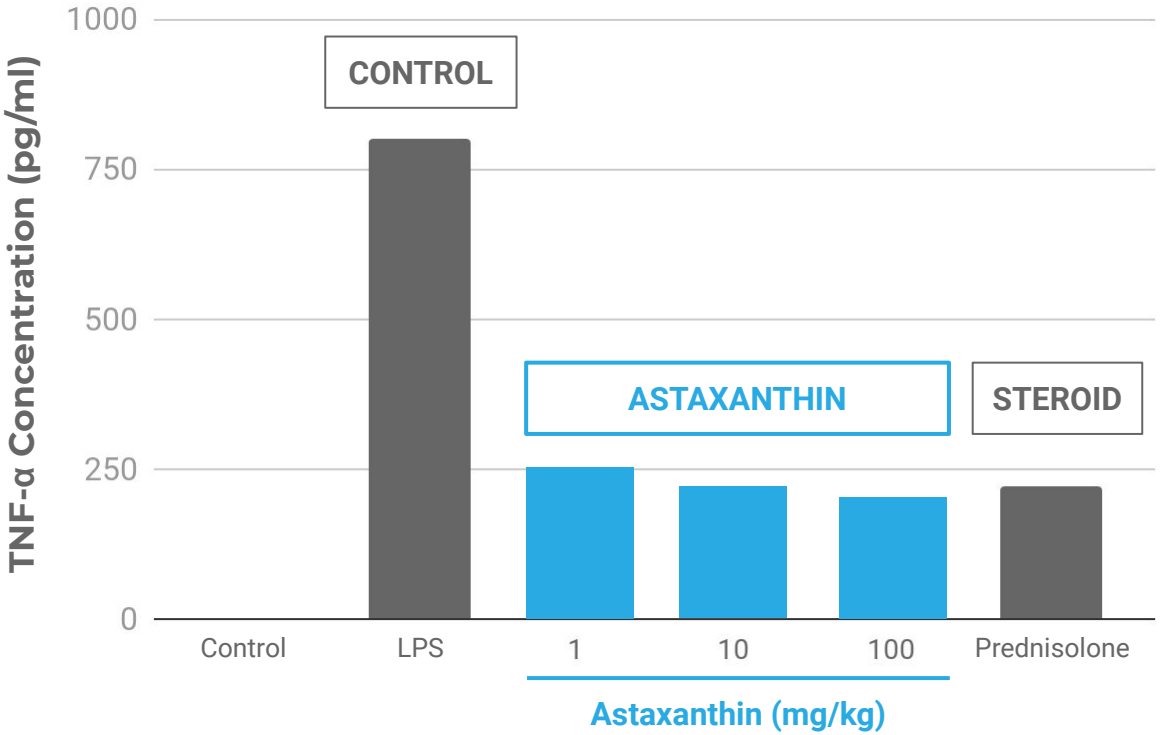
ASTAXANTHIN REDUCED TNF-α IN INFLAMMATORY ANIMAL MODEL = PREDNISOLONE

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: A Novel Potential Treatment for Oxidative Stress and Inflammation in Cardiovascular Disease

Fredric J. Pashkow, MD,[a,b,*] David G. Watumull,[b] and Charles L. Campbell, MD[c]

Oxidative stress and inflammation are implicated in several different manifestations of cardiovascular disease (CVD). They are generated, in part, from the overproduction of reactive oxygen species (ROS) and reactive nitrogen species (RNS) that activate transcriptional messengers, such as nuclear factor–κB, tangibly contributing to endothelial dysfunction, the initiation and progression of atherosclerosis, irreversible damage after ischemic reperfusion, and even arrhythmia, such as atrial fibrillation. Despite this connection between oxidative stress and CVD, there are currently no recognized therapeutic interventions to address this important unmet need. Antioxidants that provide a broad, "upstream" approach via ROS/RNS quenching or free radical chain breaking seem an appropriate therapeutic option based on epidemiologic, dietary, and in vivo animal model data. However, human clinical trials with several different well-known agents, such as vitamin E and β-carotene, have been disappointing. Does this mean antioxidants as a class are ineffective, or rather that the "right" compound(s) have yet to be found, their mechanisms of action understood, and their appropriate targeting and dosages determined? A large class of potent naturally-occurring antioxidants exploited by nature—the oxygenated carotenoids (xanthophylls)—have demonstrated utility in their natural form but have eluded development as successful targeted therapeutic agents up to the present time. This article characterizes the mechanism by which this novel group of antioxidants function and reviews their preclinical development. Results from multiple species support the antioxidant/anti-inflammatory properties of the prototype compound, astaxanthin, establishing it as an appropriate candidate for development as a therapeutic agent for cardiovascular oxidative stress and inflammation. © 2008 Elsevier Inc. All rights reserved. (Am J Cardiol 2008;101[suppl]:58D–68D)

Atherosclerosis is an inflammatory disease of the arterial wall that remains a principal cause of death and disability, despite the application of statin and antiplatelet therapies. The severe clinical manifestations of the disease—myocardial infarction (MI) and stroke—are mainly caused by the abrupt obstruction of the vessel lumen by a thrombus triggered by the rupture or erosion of an atherosclerotic plaque.[1] Existing data strongly suggest that immunoinflammatory–related mechanisms are the major determinants of these atherothrombotic plaque sequelae.[2] Thus, most of the important advances in the comprehension of the mechanisms of atherothrombosis come from studies of the critical components involved in the modulation of the immunoinflammatory balance within the plaque. Despite an increasing understanding of these processes, there have been no approved therapeutic interventions in vascular biology that fully incorporate the current understanding of oxidative stress and inflammation.[3]

Role of Reactive Oxygen and Nitrogen Species in Cardiovascular Inflammation

Reactive oxygen species (ROS) and reactive nitrogen species (RNS) are well recognized for functioning as both potentially harmful and beneficial cell-signaling molecules. Normally generated by tightly regulated enzymes, such as nicotinamide adenine dinucleotide phosphate oxidase (NADPH) and nitric oxide synthase (NOS), excessive and/or chronic overproduction of ROS/RNS either from the mitochondrial electron transport chain or various other ROS/RNS–generating enzymes, NADPH or NOS, results in oxidative stress, a harmful process that can be an important source of damage to cellular components, including lipids, proteins, and DNA. In contrast, beneficial effects of ROS/RNS (eg, superoxide radical and nitric oxide) occur transiently at low-to-moderate concentrations and mediate physiologic roles in cellular responses to oxygen deprivation: defense against infectious agents, modulation of cellular signaling pathways, and the induction of cellular proliferation. Paradoxically, various ROS-mediated actions, in

[a]John A. Burns School of Medicine, University of Hawaii, Honolulu, Hawaii, USA; [b]Cardax Pharmaceuticals, Inc., Aiea, Hawaii, USA; and [c]Gill Heart Institute, University of Kentucky, Lexington, Kentucky, USA.

Statement of author disclosure: Please see the Author Disclosures section at the end of this article.

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www.AJConline.org

MODERN RHEUMATOLOGY

ORIGINAL ARTICLE

Astaxanthin ameliorates

Thrombosis Research

Contents lists available at ScienceDirect

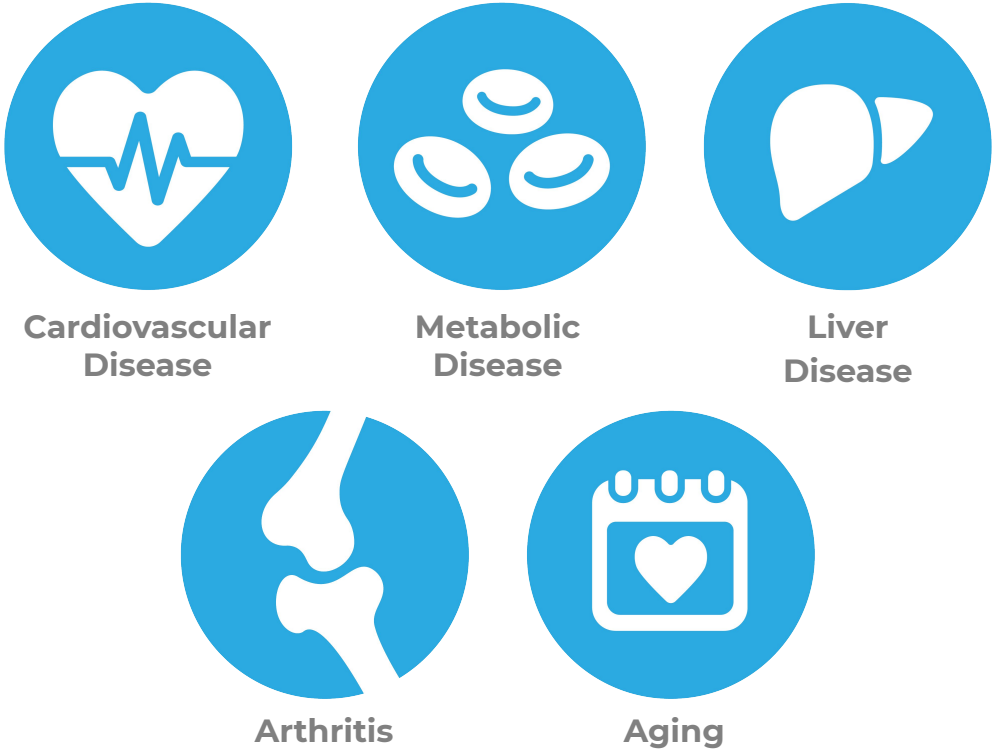
homepage: www.elsevier.com/locate/thromres

DX-085) attenuates thrombosis in a mouse model

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 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

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.

*$p<0.05$ **$p<0.01$

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.

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**

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.

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.

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-1β, 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)

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

- **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)

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

CARDAX PRODUCT PLATFORM

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PHARMACEUTICAL CANDIDATES

DISCOVERY | PRECLINICAL | CLINICAL

CDX-101 (ASTAXANTHIN RX CANDIDATE)
for SEVERE HYPERTRIGLYCERIDEMIA

CDX-301 (ZEAXANTHIN RX CANDIDATE)
for STARGARDT DISEASE

DIETARY SUPPLEMENTS

DEVELOPMENT | LAUNCH | MARKETING

ZANTHOSYN® (ASTAXANTHIN SUPPLEMENT)
for INFLAMMATORY HEALTH*

CDX-301

ZEAXANTHIN PHARMACEUTICAL CANDIDATE

Potential applications include:

- Stargardt Disease
- Age-Related Macular Degeneration

- **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

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

CARDAX PRODUCT PLATFORM

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PHARMACEUTICAL CANDIDATES

DISCOVERY | PRECLINICAL | CLINICAL

CDX-101 (ASTAXANTHIN RX CANDIDATE)
for SEVERE HYPERTRIGLYCERIDEMIA

CDX-301 (ZEAXANTHIN RX CANDIDATE)
for STARGARDT DISEASE

DIETARY SUPPLEMENTS

DEVELOPMENT | LAUNCH | MARKETING

ZANTHOSYN® (ASTAXANTHIN SUPPLEMENT)
for INFLAMMATORY HEALTH*

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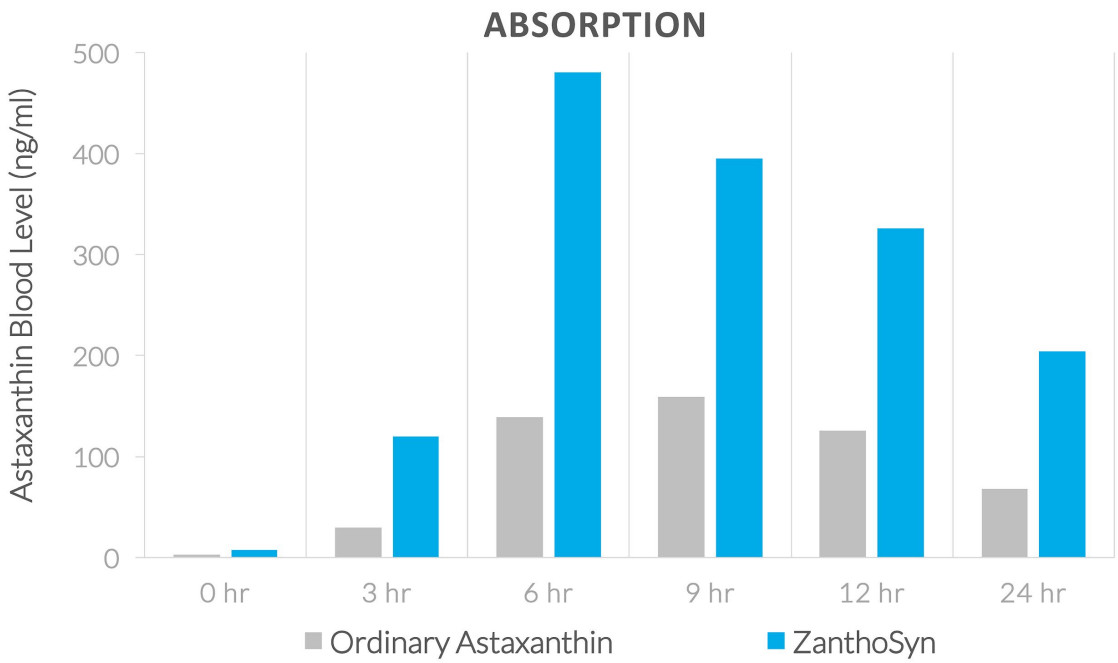
ZanthoSyn® is a physician recommended astaxanthin dietary supplement for inflammatory health*

* 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



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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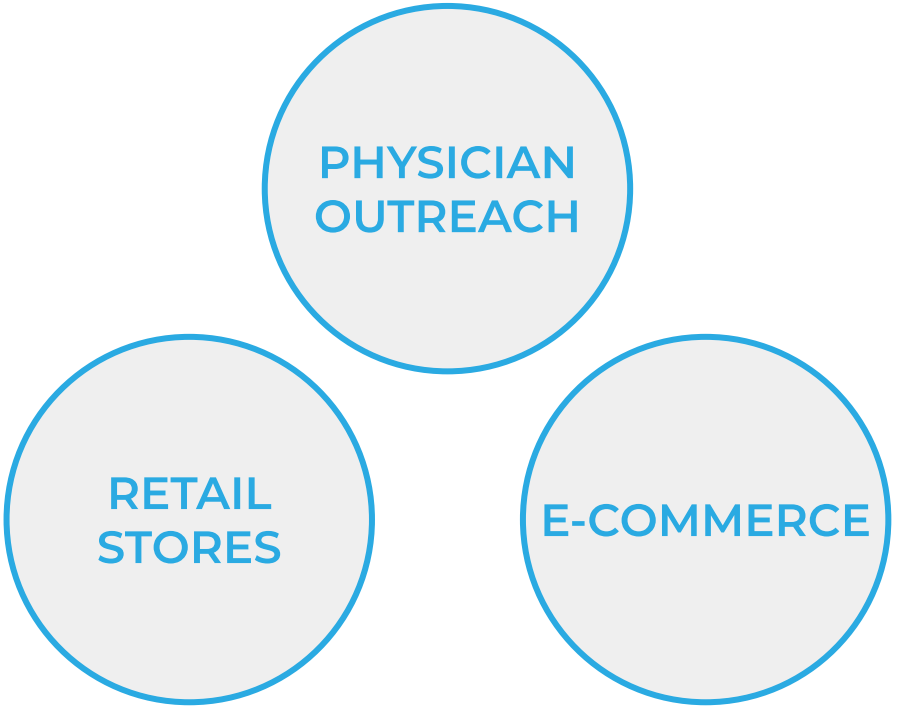
MULTI-PRONGED APPROACH



ZANTHOSYN®

In Summary

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

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.

	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

- **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

- **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

- 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