MANUKA & WOUND SCIENCE: PODCAST TRANSCRIPT

Methylglyoxal: The Antibacterial Science Behind Medical-Grade Manuka

MGO Mechanism of Action, Clinical Grading, Biofilm Disruption, and Bedside Application Protocol

Series: Wound Care & Healing | Speakers: Jordan & Quinn | Runtime: 19:10

SESSION OVERVIEW

This session examines the precise biochemistry that separates Manuka honey from every other honey on the shelf. Jordan, an apiology and melittology researcher, and Quinn, a wound care nurse with 14 years of clinical experience in a regional burn center, bridge the gap between laboratory data and bedside application. The discussion begins with the fundamental limitation of standard honey, specifically the rapid neutralization of hydrogen peroxide by tissue catalase enzymes in the wound bed, and establishes why methylglyoxal (MGO) operates on an entirely different and more resilient antibacterial pathway. The biosynthetic origin of MGO is traced from dihydroxyacetone (DHA) in raw Manuka nectar through the non-enzymatic hive conversion process. The dual mechanism of action is detailed: simultaneous protein glycation via advanced glycation end products (AGEs) and direct DNA crosslinking at guanine bases, explaining why bacteria cannot easily develop resistance. The session concludes with a clinical grading breakdown (MGO 100+, MGO 400+, MGO 800+), the UMF vs. MGO labeling distinction, the dilution challenge posed by wound exudate, and the rigorous dressing protocols required for therapeutic efficacy. All source data is available at manukawoundscience.org/methylglyoxal-antibacterial-science.

CRITICAL DATA SUMMARY

MGO MECHANISM OF ACTION	WHY BACTERIA CANNOT RESIST IT
Standard honey antibacterial route: Glucose oxidase enzyme produces hydrogen peroxide. Destroyed instantly by tissue catalase enzymes in the wound bed. No clinical value against active infection.	**Single-target antibiotics** (beta-lactams, macrolides) attack one receptor. Bacteria mutate one gene and develop a workaround. Resistance evolves rapidly.
MGO route: Non-peroxide antibacterial activity. Remains stable and active when diluted by wound fluids and blood. Human tissue enzymes cannot break it down.	**MGO attacks simultaneously:** Protein glycation (AGEs) disables structural proteins and enzyme function. DNA crosslinking at guanine bases halts replication. Two independent systems fail at once.
Protein glycation: MGO binds to lysine and arginine residues, forming advanced glycation end products (AGEs). Cell wall proteins misfold. Membrane integrity collapses.	**To resist MGO,** a bacterium must simultaneously evolve protection for its structural proteins AND its DNA blueprint against a highly reactive, constantly binding carbonyl compound. Evolutionarily near-impossible.

DNA crosslinking: MGO forms covalent bonds with guanine bases on the DNA double helix. Helicase enzymes cannot unzip the strand. Transcription and replication halt entirely.	**Effective against:** MRSA (Methicillin-Resistant Staphylococcus aureus), Pseudomonas aeruginosa, multidrug-resistant organisms with efflux pumps and beta-lactamases.

MGO TIER	CLASSIFICATION	CLINICAL INDICATION
MGO 100+	**Low Activity**	General dietary wellness only. Zero clinical value for wound care. Do not apply to compromised tissue under any circumstances.
MGO 400+	**Clinical Threshold**	Minimum concentration for medical use. Effective against most common wound pathogens in laboratory and clinical trial settings. Baseline for FDA-cleared wound care products.
MGO 800+	**Premium Medical Grade**	Reserved for chronic stalled wounds, deep biofilm infections with polysaccharide shielding, and antibiotic-resistant strains including MRSA and Pseudomonas. Provides therapeutic margin against heavy exudate dilution.

EXUDATE DILUTION CHALLENGE	BEDSIDE PROTOCOL REQUIREMENTS
The problem: Active infected wounds continuously weep plasma, white blood cells, dead tissue, and proteolytic enzymes. This fluid dilutes the applied honey within hours.	**Dressing change frequency** must increase in proportion to exudate volume to replenish MGO concentration at the tissue interface.
MGO 400 applied to a highly exudative venous leg ulcer can dilute to an effective concentration of MGO 50.100 within hours, dropping below the minimum inhibitory concentration.	**Deep cavity wounds:** Pack with calcium alginate ropes impregnated with Manuka honey under a highly absorbent secondary dressing to maintain contact time.
Therapeutic margin rule: Start with an overwhelmingly high MGO concentration (800+) so that after fluid dilution by 50% or more, the residual concentration at the cellular membrane remains lethal.	**Continuous undiluted contact time** between MGO and the bioburden is as critical as the MGO number on the tube. If it is not physically touching the bacteria, glycation and DNA crosslinking stop.
UMF vs. MGO: UMF (Unique Manuka Factor) measures MGO, DHA, and leptosperin (origin marker). MGO is the direct milligrams-per-kilogram measurement of the active compound. UMF 20+ correlates to approximately MGO 800+.	**For clinical procurement:** Specify MGO concentration, not UMF, in formulary orders. MGO directly quantifies the antibacterial weapon being deployed at the wound bed.

TRANSCRIPT

[Jordan] 0:00

Hi. I am Jordan and I am Quinn, and today we are talking about the one compound that separates Manuka honey from every other honey on the shelf.

[Quinn] 0:08

Right. And before we really get into the weeds today, I want to let you know that you can find the complete page we are discussing.

[Jordan] 0:14

The methylglyoxal, the antibacterial compound behind medical-grade Manuka page.

[Quinn] 0:20

Exactly. You can access all of this by visiting manukawoundscience.org. Our goal today is to really unpack the clinical data and figure out exactly why this specific honey is literally saving limbs.

[Jordan] 0:35

The cellular mechanics alone are absolutely fascinating to look at, especially when we bridge that gap between the in vitro laboratory data and actual clinical application.

[Quinn] 0:45

And that transition from the lab to the bedside is where my entire career lives. I spent 14 years as a wound care nurse in a regional burn center.

[Jordan] 0:53

Which is a completely different world from a controlled lab.

[Quinn] 0:55

Totally. And for anyone listening who works in critical care, you know exactly what it feels like to hit a wall with a patient. You have someone with a severe third-degree burn, highly compromised, and the wound bed is completely stalled. It's just not granulating. It is covered in slough.

[Jordan] 1:14

And the standard treatments just aren't working.

[Quinn] 1:16

Nothing. The standard courses of broad-spectrum IV antibiotics are doing absolutely nothing. You are going in every single shift for a dressing change, doing sharp debridement right there at the bedside, and you're just watching this multi-drug resistant infection hold its ground week after week.

[Jordan] 1:33

That has to be incredibly frustrating.

[Quinn] 1:34

It's exhausting. But then one day the attending physician walks in, reviews the chart, and orders a Manuka dressing.

[Jordan] 1:41

And the friction there must be incredible. You're operating in this modern, highly sterile, technologically advanced hospital environment, and suddenly there's an order for a biological product.

[Quinn] 1:51

The skepticism was completely off the charts. You're standing there in your sterile gown in this heavily sanitized room, and you're essentially being told to take a substance that most of us associate with a piece of toast and a cup of tea, and apply it directly onto an open, highly vulnerable wound bed.

[Jordan] 2:07

It sounds totally counterintuitive.

[Quinn] 2:09

It feels almost reckless if you haven't read the biochemistry. But you follow the order, you do the dressing change, and 48 hours later, you pull back that impregnated gauze, and you see the clinical reality.

[Jordan] 2:22

Which is what, exactly?

[Quinn] 2:23

The purulent exudate has cleared up, the bioburden is visibly reduced, the tissue is actually moving from a pale, angry gray to a healthy, vascularized, beefy red.

[Jordan] 2:35

Wow.

[Quinn] 2:35

The wound bed is finally actually improving. It's one of those paradigm-shifting moments that just forces you to respect the clinical science.

[Jordan] 2:43

Well, the biochemistry explains that exact clinical turnaround, because the skepticism you felt was completely valid based on what we know about standard honey.

[Quinn] 2:51

Right, because of the baseline properties.

[Jordan] 2:54

Exactly. It's crucial to understand that all honey, literally any floral honey you pull off a grocery store shelf, has some baseline antibacterial properties. And that happens primarily because of an enzyme called glucose oxidase.

[Quinn] 3:06

Which the bees add to the nectar, right?

[Jordan] 3:09

Right. When that enzyme comes into contact with moisture, it naturally produces hydrogen peroxide. So historically, honey has been used to keep basic bacterial colonization at bay because of that peroxide activity.

[Quinn] 3:21

Why does MGO matter when hydrogen peroxide already kills bacteria?

[Jordan] 3:25

That brings us directly to the hostile environment of a human wound. Hydrogen peroxide is great in a petri dish, but when standard honey touches actively granulating or infected human tissue, our own bodies fight it.

[Quinn] 3:38

We destroy it ourselves.

[Jordan] 3:39

Yes. We have tissue enzymes, specifically catalase, present in our cells and blood. The biological purpose of catalase is to protect our own cells from oxidative stress, so it instantly breaks down hydrogen peroxide into water and oxygen.

[Quinn] 3:52

So the moment standard honey hits a wound bed, our catalase enzymes destroy its primary antibacterial weapon.

[Jordan] 3:58

Exactly. But Manuka honey has a secondary, completely distinct antibacterial system, and it is powered by methylglyoxal, or MGO.

[Quinn] 4:07

And that is the non-peroxide activity we are always looking for in the clinical guideline. Because the critical difference for patients is that MGO remains entirely stable and active, even when it's highly diluted by wound fluids and blood.

[Jordan] 4:18

Right. Human enzymes simply don't recognize it.

[Quinn] 4:21

Our tissue enzymes cannot break down MGO. It survives the chaotic battlefield of the wound bed where hydrogen peroxide just gets instantly neutralized.

[Jordan] 4:30

That resilience is the cornerstone of its medical value. MGO provides this non-peroxide antibacterial activity through a completely different chemical pathway.

[Quinn] 4:41

Which is why medical-grade Manuka works when a standard clover honey would completely fail and, honestly, likely just feed the infection. It isn't magic. It's just a much more structurally resilient molecule. But where this gets really wild for me, looking at the botanical data, is where this MGO actually comes from. Because the logical assumption is that the bees are just out there collecting MGO directly from the flowers.

[Jordan] 5:03

Biology loves a good plot twist. Methylglyoxal does not actually exist in the flower nectar of the Leptospermum scoparium plant, the Manuka bush.

[Quinn] 5:10

Which is crazy to think about.

[Jordan] 5:12

It really is. If you analyze the raw nectar straight from the blossom in New Zealand or Australia, you will find zero MGO.

[Quinn] 5:19

Zero. Even after reading the studies, that still blows my mind. The plant doesn't even make the active drug.

[Jordan] 5:25

Instead, what that nectar contains are incredibly high concentrations of a very simple sugar molecule called dihydroxyacetone, or DHA. And the amounts can be staggering.

[Quinn] 5:36

Like, how much are we talking?

[Jordan] 5:37

Depending on the specific plant, a single tiny blossom produces nectar with DHA concentrations frequently exceeding 2,000 milligrams per kilogram.

[Quinn] 5:47

And the research indicates that the amount of DHA the plant produces isn't random. It's highly dependent on the environmental stressors the plant faces.

[Jordan] 5:55

Yes. There is a rapidly expanding field of study around MGO terroir mapping.

[Quinn] 6:00

Like with wine.

[Jordan] 6:01

Exactly like wine. Just as viticulturists map out how vineyard soil dictates a flavor profile, researchers are mapping out how initial DHA levels in Manuka nectar are dictated by specific soil chemistry, trace mineral profiles, and regional climate patterns.

[Quinn] 6:14

It's a defense mechanism.

[Jordan] 6:16

Right. The plant often upregulates DHA production as a biological stress response. Harsh winds, specific UV indexes, and the mineral density of the local dirt literally determine the potential potency of the final harvest.

[Quinn] 6:29

So the plant makes the precursor, the DHA, but we need MGO at the bedside. The transition from DHA to MGO happens inside the hive, doesn't it?

[Jordan] 6:39

That is where the hive chemistry takes over. The bees collect this DHA-rich nectar and store it in the honeycomb. Inside the hive, you have a very specific controlled environment. The temperature hovers around 35 degrees Celsius and the pH is highly acidic.

[Quinn] 6:53

And that sparks a reaction.

[Jordan] 6:54

Over time, yes. Under those specific conditions of ambient heat and acidity, the DHA undergoes a non-enzymatic chemical reaction. Specifically, it's a slow, Maillard-like dehydration process.

[Quinn] 7:05

So it's losing water.

[Jordan] 7:06

Literally losing a water molecule. And through that structural loss, it converts into methylglyoxal, MGO. This happens slowly, over months of storage and maturation. It can reach MGO levels anywhere between 100 and well over 1,000 milligrams per kilogram, entirely depending on storage conditions and time.

[Quinn] 7:23

So the bees are not making the medicine. The honey is making itself.

[Jordan] 7:27

It's a naturally occurring chemical reaction happening inside the physical matrix of the honey over time. Basic organic chemistry just does the heavy lifting.

[Quinn] 7:36

Let's get down to the mechanism of action. Because as a clinician, I am frequently looking at a wound bed colonized by multidrug-resistant organisms.

[Jordan] 7:44

The really nasty stuff.

[Quinn] 7:46

We're talking about Pseudomonas aeruginosa, MRSA, pathogens that have evolved sophisticated efflux pumps and beta-lactamases to survive our strongest synthesized antibiotics.

[Jordan] 7:57

They are incredibly adaptive.

[Quinn] 7:59

For those of you listening who track resistance trends, you already know how fast these biofilms mutate. So when you see MGO bypassing those defenses, we need to know exactly how this molecule is taking them down.

[Jordan] 8:09

MGO proves itself as an absolute powerhouse here because it is a highly reactive carbonyl compound. It doesn't just inhibit bacterial growth. It physically damages the bacteria through a brutal process called glycation.

[Quinn] 8:22

Unlike standard antibiotics, which usually just have a single target.

[Jordan] 8:26

Right. A beta-lactam attacks the bacterial cell wall, or a macrolide targets one specific ribosomal subunit. Bacteria can just mutate a single receptor and figure out a workaround for a single point of attack. But MGO attacks the bacteria on multiple fronts simultaneously.

[Quinn] 8:41

We are looking at a coordinated multi-system failure for the bacteria. The first attack route is protein damage, correct?

[Jordan] 8:47

Yes. The clinical data shows MGO physically binding to specific amino acids, primarily lysine and arginine residues inside the bacterial proteins. When it does this, it creates advanced glycation end products, or AGEs.

[Quinn] 9:01

And what do those AGEs actually do to the bacteria?

[Jordan] 9:04

By creating those AGEs, the MGO causes the bacterial proteins to physically misfold. It completely disrupts their enzyme function and compromises their cellular membrane integrity.

[Quinn] 9:15

So it wrecks their structure.

[Jordan] 9:16

Exactly. If the bacteria relies on a specific protein to maintain its cell wall or to pump toxins out of its system, MGO alters the shape of that protein so it can no longer function. It seizes up their basic survival machinery.

[Quinn] 9:28

So their internal machinery is destroyed and their outer wall is compromised. And then we have the second route, which goes straight for the genetic blueprint: the DNA.

[Jordan] 9:37

MGO physically reacts with the guanine bases in the bacterial DNA itself. It forms covalent bonds, permanent chemical crosslinks right on the DNA double helix. Breaking the code. By crosslinking those guanine bases, it's effectively stopping the bacteria's ability to transcribe its genetic code. The helicase enzymes can't unzip the DNA for replication. Reproduction just halts.

[Quinn] 10:03

Even if it survives the membrane damage.

[Jordan] 10:05

Right. Even if a bacterium somehow survives the massive protein misfolding, it cannot divide and spread the infection.

[Quinn] 10:11

So it is hitting them from two directions at once, which is why bacteria have such a hard time building resistance.

[Jordan] 10:17

To develop resistance to Manuka honey, a bacteria would have to simultaneously evolve a completely new way to protect both its structural proteins and its DNA blueprint from a highly reactive, constantly binding chemical.

[Quinn] 10:29

Which is an incredibly difficult hurdle for a single-celled organism to clear.

[Jordan] 10:34

Evolutionarily speaking, it's almost impossible.

[Quinn] 10:36

Which is exactly why we see such profound, rapid results in the burn unit. But here is the critical warning I need to give to anyone listening right now, especially if you manage clinical supplies or treat patients.

[Jordan] 10:48

The grading issue.

[Quinn] 10:49

Yes. Because of how effective this mechanism is, the market has exploded with products labeled Manuka. And from a clinical standpoint, it is terrifying because a massive amount of the Manuka on the retail shelf is absolutely not medical grade.

[Jordan] 11:03

Not even close.

[Quinn] 11:04

If you put a low-grade wellness honey on a serious diabetic foot ulcer, you are just feeding sugar to an active infection. And that is a fast track to an amputation. We have to break down the MGO rating guide.

[Jordan] 11:16

Right. Because not all Manuka honey has enough MGO to be therapeutically useful. The laboratory data shows us a very clear dose-response curve. Higher MGO concentrations kill bacteria faster, penetrate deeper into tissues, and destroy biofilms more completely.

[Quinn] 11:33

And there are three distinct tiers we track. Let's start with the first tier, MGO 100+.

[Jordan] 11:37

This is categorized as low activity. It has a very minimal antibacterial effect. It's perfectly fine for general dietary wellness, maybe stirring into your oatmeal. But it has absolutely zero clinical value for wounds.

[Quinn] 11:50

Do not bring this near a compromised patient.

[Jordan] 11:53

Absolutely not.

[Quinn] 11:54

The next tier is MGO 400+. This is the clinical threshold. It is the absolute minimum concentration required for actual medical use.

[Jordan] 12:03

That's where the real work begins.

[Quinn] 12:04

Right. At 400 milligrams per kilogram, laboratory testing and clinical trials show it is highly effective against most common wound pathogens. If you are looking at FDA-cleared wound care products, the baseline active ingredient usually begins around this concentration.

[Jordan] 12:20

And then there is the maximum potency tier: MGO 800+. This is the premium medical grade classification.

[Quinn] 12:26

Heavy hitter.

[Jordan] 12:27

Exactly. In the hospital, we reserve this for severe cases. Chronic wounds that have stalled for months, deep biofilm infections where the bacteria have built a protective polysaccharide shield, and those terrifying antibiotic-resistant strains we discussed earlier.

[Quinn] 12:41

And we should probably address the labeling confusion here.

[Jordan] 12:44

Yes. For those of you managing hospital formularies or ordering supplies, you often look at a tube of medical honey and you don't actually see MGO printed on the front. You see UMF. I want to clarify that difference because it causes a lot of confusion at the procurement level.

[Quinn] 12:59

UMF stands for Unique Manuka Factor.

[Jordan] 13:01

It's a composite rating system used to guarantee purity and origin. It measures MGO, but it also measures the precursor DHA and another chemical marker called leptosperin, which proves the honey actually came from the Manuka bush.

[Quinn] 13:14

UMF is a fantastic grading system for the overall authenticity of the honey. But MGO is the direct literal chemical measurement of the antibacterial compound itself in milligrams per kilogram.

[Jordan] 13:27

So it's the more relevant metric for clinical use.

[Quinn] 13:29

Exactly. For clinical wound care protocols, MGO is the metric you want because it directly quantifies the active weapon we're deploying against the bacteria. For context, a UMF 20+ rating typically correlates to an MGO 800+ concentration.

[Jordan] 13:42

That distinction is vital and it leads directly into a major translation challenge that we deal with at the bedside. You can read these brilliant laboratory studies where a concentration of MGO 400 looks incredibly effective at destroying a Pseudomonas colony in a controlled petri dish.

[Quinn] 13:58

But then it fails in practice.

[Jordan] 14:00

Right. You take a product with that exact same concentration, apply it to an actual human wound bed, and it completely fails to clear the infection. The primary reason for that failure is exudate.

[Quinn] 14:12

Wound fluid.

[Jordan] 14:13

Yes. An active infected wound is constantly weeping fluid. It's a mixture of plasma, white blood cells, dead tissue, and proteolytic enzymes. This exudate constantly washes over the wound bed.

[Quinn] 14:24

So it acts as a diluent.

[Jordan] 14:26

Exactly. If you apply an MGO 400 honey to a highly exudative venous leg ulcer, within a matter of hours that fluid has diluted the honey so thoroughly that the actual concentration of MGO sitting on the bacteria is nowhere near 400 anymore.

[Quinn] 14:40

It drops significantly.

[Jordan] 14:41

It might drop to 100 or 50, at which point it's no longer lethal to the pathogens.

[Quinn] 14:46

And that bedside observation is perfectly validated by the pharmacokinetic lab data. This dilution effect is the exact reason why true medical-grade products require much higher MGO concentrations than what we call minimum inhibitory concentrations, or MIC, in the lab.

[Jordan] 15:02

Because the lab doesn't account for the fluid wash.

[Quinn] 15:05

Exactly. In a sterile dish, a very low MIC might arrest bacterial growth. But in the real world, to penetrate thick bacterial biofilms and counter that constant heavy dilution from wound exudate, you need what we call a massive therapeutic margin.

[Jordan] 15:20

You have to overshoot the target.

[Quinn] 15:21

You have to start with an overwhelmingly high concentration of MGO, like an 800+, so that even after the wound fluid dilutes it by half or more, the remaining concentration sitting on the cellular membrane is still lethal to the bacteria.

[Jordan] 15:34

That therapeutic margin is why wound care is an active, highly specialized discipline. It isn't just about slapping a tube of medical honey onto a patient's leg and walking away for a week. It requires strict, rigorous dressing protocol.

[Quinn] 15:47

It is a dynamic treatment.

[Jordan] 15:48

Very dynamic. If a wound is producing a heavy volume of exudate, the frequency of dressing changes has to increase to physically replenish that MGO concentration. The physical quantity of the honey matters, too.

[Quinn] 16:01

You've got to assess the cavity depth, right?

[Jordan] 16:03

Exactly. We have to decide whether to pack a deep cavity wound with calcium alginate ropes impregnated with Manuka, or apply a thick layer of 100 percent Manuka gel under a secondary, highly absorbent dressing. Maintaining continuous, undiluted contact time between the MGO and the tissue is just as important as the big MGO number printed on the tube.

[Quinn] 16:24

Because if it's not touching the bacteria, it's not working.

[Jordan] 16:27

Exactly. If the honey gets washed away by fluid or isn't in direct physical contact with the bioburden, the glycation and DNA crosslinking simply stop happening.

[Quinn] 16:37

The intersection of biochemistry and clinical protocol is everything here. You can have the most fascinating, multi-targeted antibacterial molecule in the natural world. But if the application methodology doesn't account for fluid dynamics and therapeutic margins, the patient doesn't heal.

[Jordan] 16:51

To wrap this all up: MGO, methylglyoxal, is the specific, scientifically proven compound that gives Manuka honey its unique non-peroxide clinical power. It is the highly resilient molecule that allows it to bypass traditional antibiotic resistance by simultaneously destroying proteins and crosslinking DNA in severe pathogens.

[Quinn] 17:15

And understanding the science also means respecting the clinical limitations. High potency ratings, knowing the distinct difference between an MGO 100 wellness product and an MGO 800 clinical powerhouse, and utilizing rigorous bedside application protocols are what truly separate a limb-saving medical treatment from what is otherwise just a very expensive jar of sweet honey.

[Jordan] 17:36

As a final reminder, you can explore all the biochemical data, the terroir mapping, and the clinical application studies we referenced today by visiting manukawoundscience.org. Look for the methylglyoxal, the antibacterial compound behind medical-grade Manuka page.

[Quinn] 17:50

And definitely check out the live citation tracker there. It pulls real-time research impact data, showing you exactly how these foundational MGO papers are currently influencing global infection control protocols.

[Jordan] 18:00

It is an incredible resource for staying current on the literature. Before we sign off, I want to leave you with a thought that connects the microscopic cellular mechanics right back to our macro environment.

[Quinn] 18:10

This goes back to the terroir mapping.

[Jordan] 18:12

Exactly. We explored MGO terroir mapping earlier and how specific soil chemistry, UV indexes, and regional weather patterns in New Zealand and Australia dictate how much of that precursor DHA the plants actually produce as a stress response.

[Quinn] 18:27

The local environment physically dictates the final medical potency.

[Jordan] 18:32

Right. So here is the question to ponder. If the strength and availability of our most powerful natural antibiotic-resistant wound treatment is entirely dependent on very specific regional climate conditions, how might shifting global climates, rising baseline temperatures, and drastically altered rainfall patterns impact our future supply of this crucial infection-fighting resource?

[Quinn] 18:54

That's a profound thought.

[Jordan] 18:55

If the terroir fundamentally changes, does the medicine change with it?

[Quinn] 18:58

Now that is a question worth considering as we look at the future of global supply chains and antimicrobial resistance. Thank you all for joining us on this exploration of the science. Take care and we will catch you next time.

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