

OEHHA SYNTHETIC TURF STUDY

Synthetic Turf Scientific Advisory Panel Meeting
April 28, 2025

Certified Transcript

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(Pages 1-121)

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APPEARANCES

PANEL MEMBERS:

John Balmes, MD, Chairperson

Deborah Bennett, Ph.D.

Ed Avol, MS

Sandy Eckel, Ph.D.

Amy Kyle, Ph.D.

Tom McKone, Ph.D.
(virtually)

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT:

Dave Edwards, Ph.D., Acting Director

Jocelyn Claude, Ph.D., Staff Toxicologist

Kannan Krishnan, Ph.D., Assistant Deputy Director

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Public Comment:

Robina Suwol

PROCEEDINGS FROM MONDAY, APRIL 28, 2025

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DR. EDWARDS: Good morning, everyone.

Welcome to our Synthetic Turf Scientific Advisory Panel meeting to go over our draft synthetic turf study. My name is Dave Edwards. I'm the acting director here at OEHHA. I just wanted to let you know that we'll be starting shortly, probably at about 10:07. So we'll be back on at 10:07. Thank you.

(Recess.)

DR. EDWARDS: Good morning, everyone, again.

Once again, my name is Dave Edwards, and I am the acting director at OEHHA. So welcome, everyone, today in the room and online to our Synthetic Turf Scientific Advisory Panel meeting.

Just to let you know that we have two of our members had a little transportation hiccup, so they will be joining shortly. But we will go ahead and start for the sake of time.

Just a little bit of background on OEHHA. We are an office within the California Environmental Protection Agency and the lead state agency for the assessment of health risks posed by

1 environmental contaminants.

2 First off, I would like to welcome our
3 panel members to this fifth meeting of the Scientific
4 Turf Advisory Panel. Let me start out by calling on
5 panel members to introduce and provide their
6 affiliation, if any, at this point.

7 We'll start with Dr. Debbie Bennett.

8 DR. BENNETT: (No audio.)

9 DR. EDWARDS: Thank you. Mr. Ed Avol.

10 MR. AVOL: (No audio.)

11 DR. EDWARDS: And then Dr. Sandy Eckel.

12 DR. ECKEL: Hi. I'm Dr. Sandy Eckel. I am
13 an associate professor in the Department of Population
14 and Public Health Sciences at USC.

15 DR. EDWARDS: Online we have Dr. Tom McKone.

16 DR. MCKONE: Hello. I'm Tom McKone,
17 professor emeritus at the University of California at
18 Berkeley School of Public Health.

19 DR. EDWARDS: Thank you. And our chair,
20 Dr. Balmes, and Dr. Amy Kyle, another member, they're
21 on their way, so they will introduce themselves when
22 they arrive.

23 I would now like to introduce you to
24 the OEHHA team. So if you can, just raise your hands
25 in the room so everyone on the panel gets to know who

1 you all are.

2 We first off have assistant deputy
3 director for scientific programs, Dr. Kannan Krishnan;
4 our acting chief counsel, Ryan Mahoney.

5 And as far as our staff goes, we have
6 Dr. Jocelyn Claude, Mrs. Allison Lensing, and
7 Ms. Kiana Vaghefi and Chris Sawders. So thanks,
8 everyone, for helping on getting this day ready.

9 Before we begin, there's a few
10 housekeeping items. So first off, the drinking
11 fountains and the restrooms are located out the back
12 door and left, and to your left down the hall, and
13 then they're located on the right side of that
14 hallway.

15 In the event of a fire alarm or any
16 other reason to evacuate this room, please leave by
17 those lighted exit doors, here or here. And then take
18 the steps down, and outside we will gather across the
19 street in Cesar Chavez Park.

20 It is a day-long meeting, so we are
21 planning to take a 75-minute break for lunch around
22 noon, and a 15-minute break at 2:30 in the afternoon,
23 for those of you online just for planning purposes.

24 If members of the public have digital
25 media that they want to show during their three-minute

1 comment period, please bring the external devices to
2 the OEHHA staff to upload the files before the lunch
3 break.

4 This meeting is being recorded and
5 transcribed. Remind people to identify themselves and
6 speak clearly into the microphones when you are giving
7 public comment.

8 The transcript will be posted on
9 OEHHA's website.

10 A little bit of background on the draft
11 itself. The draft report of this OEHHA study on
12 synthetic turf was released for public comments on
13 March 13th. The goal of the study was to assess the
14 potential health impacts associated with the use of
15 synthetic turf and playground mats made of crumb
16 rubber.

17 The Scientific Advisory Panel has held
18 four previous meetings to advise OEHHA on study plans,
19 data interpretation, and reporting of study results.
20 Now, here we are after the publication of the draft
21 report and looking forward to your advice and comments
22 on the draft report.

23 This report has been a long time
24 coming, six years since our last panel meeting, and
25 several folks have retired that have been involved

1 with this. So before we go forward, I'd like to
2 recognize their contributions. First off, starting
3 with Scientific Advisory Panel member Dr. Linda
4 Sheldon; lab members Randy Maddalena, Hugo
5 Destailats, and Marion Russell; and OEHHA staff
6 retirees and former project staff Patty Wong, Lori
7 Lim, Dave Siegel, David Ting, and Rebecca Belloso.

8 So with that, I'd now like to do a
9 little bit more logistics regarding public comment.
10 There will be an opportunity to provide oral public
11 comment in the afternoon. The public comment will be
12 limited to three minutes per commenter. For those of
13 you that are in person, you should have received a
14 comment card at check-in. Otherwise, cards are
15 available on the back table. Please fill one out if
16 you would like to speak, and then turn it in to the
17 OEHHA staff.

18 For those of you watching by CalEPA
19 webcast, you'll be able to watch the meeting but
20 you'll need to join the meeting by Zoom to speak or
21 you may send the comments via e-mail to
22 SyntheticTurf@oehha.ca.gov. Staff will read the
23 comments allowed up to three minutes each as time
24 allows.

25 For folks who are joining virtually and

1 wish to comment, are asked to join the Zoom webinar.
2 You will receive a link to join the webinar at the end
3 of the registration process. If you have provided a
4 working e-mail address, you will also receive an
5 e-mail with a link. When requested by the chair,
6 individuals may queue to provide oral comment by using
7 the raise hand function.

8 When your name is called to speak, you
9 will unmute yourself and comment. If you would like
10 to present slides and have not already sent them,
11 please e-mail them now to SyntheticTurf@oehha.ca.gov.

12 With that, I'm going to turn it over to
13 Dr. Kannan Krishnan to give a brief introduction and
14 overview presentation.

15 Kannan.

16 DR. KRISHNAN: Let me start today's
17 scientific presentation by OEHHA with this brief
18 introduction.

19 The goal of the OEHHA Synthetic Turf
20 Study (no audio).

21 This study was designed with the
22 consultations and input from the general public,
23 player support groups, parents, tire industry, and
24 field owners, and we're thankful for that. And a
25 study of this complexity was only possible due to

1 collaborations and partnerships that included Lawrence
2 Berkeley National Laboratory and California Institute
3 of Quantitative Sciences at UCB, UCB Center for
4 Environmental Research and Children's Health, and the
5 University of Arizona.

6 Since the last SAP meeting in 2019,
7 thanks to the panel input and advice on the design and
8 implementation of the study, now we have completed the
9 data analysis, the exposure assessment, hazard and
10 risk assessment and have released the draft report for
11 public and SAP review, as Dr. Edwards mentioned
12 earlier, on March 13th, with the public comment period
13 ending today.

14 This particular OEHHA study differs
15 from other studies in terms of its uniqueness of the
16 field characterization, exposure characterization, and
17 the human health risk assessment component.

18 In terms of the field characterization
19 or to understand what are on the fields, we collected
20 air samples right next to soccer activities, important
21 for characterizing inhalation exposure of athletes,
22 coaches, and referees. We also conducted nontarget
23 analysis to identify chemicals that may be released
24 from crumb rubber and present in the air on the
25 fields.

1 In terms of exposure characterization
2 to be able to get better estimate of exposure of
3 athletes, we collected both exposure and activity data
4 directly from athletes, as Dr. Claude will be
5 referring to in detail momentarily.

6 In order to better evaluate the risks
7 from exposure to tire-related chemicals, we compiled
8 and derived toxicity criteria of selected tire-related
9 chemicals, and we conducted exposure assessment and
10 risk characterization for athletes, coaches, referees,
11 and spectators, both on average and at individual
12 field level.

13 On average, it reflects exposure across
14 all fields indicative of players, referees, and
15 coaches traveling to many fields. But as the
16 individual field assessment reflects, playing at the
17 same local field or a home field primarily or only.
18 These are the scenarios for which we've conducted the
19 exposure and risk characterizations.

20 These aspects will be covered in detail
21 in the upcoming presentations. First of all,
22 Dr. Jocelyn Claude on field characterization and
23 exposure characterization, and then in the afternoon
24 by myself on toxicity evaluation and risk
25 characterization.

1 With that brief intro, I was going to
2 turn it over to the chair.

3 DR. EDWARDS: Thank you, Kannan. Just for
4 interest of time, we're going to just go on to
5 Jocelyn's presentation, and then after Jocelyn's
6 presentation, we'll have a time for clarifying
7 questions and panel discussion.

8 All right. Jocelyn.

9 DR. CLAUDE: Good morning. My name is
10 Jocelyn Claude. I'm a staff toxicologist here at
11 OEHHA, and I have been working on the synthetic turf
12 project over the past ten years. Happy to be here to
13 kind of sum up what we've done.

14 I'm going to be talking about the field
15 characterization and exposure characterizations we did
16 for this study. As evidenced by our report, these two
17 topics encompass quite a bit of information. Since
18 we've talked a lot about several aspects of these in
19 prior meetings, I'm going to be providing mostly an
20 overview, but I will go into detail in some areas that
21 we haven't previously discussed before.

22 Here is our outline. I'll give just a
23 brief introduction to kind of refresh our memories on
24 a synthetic turf field and the various components.
25 Then I'll talk about the field characterization,

1 exposure characterization, and I'll sum up with a
2 short summary before I'll turn it over to Dave and
3 we'll have discussion then.

4 First, I just want to give a brief
5 reminder of what a turf field is since it's been a
6 while since our last meeting. These are fields that
7 are typically used for recreation. You can see them
8 in residential and commercial areas, and they're
9 designed to look like natural grass fields. So we
10 focus specifically on synthetic turf athletic fields
11 which are popular due to reduced need for water and
12 maintenance.

13 And they've also got improved
14 playability, which means they need very little or no
15 rest between use so they can be used all year around.

16 They're comprised of three basic parts.
17 They've got synthetic grass blades, which look like
18 natural grass blades. They're attached to backing
19 material, which helps to provide support for those
20 blades. And then the fields contain an in-fill, which
21 helps support the blades and provides cushion for the
22 fields.

23 And there are many types of in-fill out
24 there. You can have crumb rubber, which I'll talk
25 about a little bit. Also, you can have organic

1 materials like husk or coconut. Our study focused on
2 specifically the crumb rubber in-fill, and this is
3 made from recycled automobile and light truck tires.
4 And after these tires are recycled, they're ground up
5 into very small pieces and they're used on this field
6 to provide cushioning.

7 So these in-fill particles are the
8 focus of our study. We did not specifically look at
9 blades or the backing materials on these fields.

10 So now for the field characterization.
11 So this was one of the primary tasks of our study,
12 which was to examine and identify the chemicals that
13 could potentially be released from the in-fill used on
14 these fields.

15 So to do that we sampled 35 fields
16 across California. We had a database of roughly 900
17 fields to start with, and we used a random stratified
18 sampling method to ensure that we got representative
19 samples across the state. The specific details of
20 this can be found in Chapter 2 of the report. I won't
21 get into too many of those right now.

22 But we did stratify by both climate
23 zone and region as well as age, since these factors
24 can affect how the crumb rubber itself gets weathered
25 and ages.

1 So you can see here on the map we had
2 five regions based on climate zones. Regions 4 and 5
3 were combined mainly due to the fewer number of fields
4 that were located in those areas. And within itself
5 California has quite a diverse climate. You have
6 desert, mountain areas. You've also got locales where
7 they have mild temperatures all year round and some
8 areas where they have very hot summers and very cold
9 winters. So all of these types of things can affect
10 how crumb rubber gets broken down and weathered and
11 aged.

12 So for age we characterized fields
13 based on whether they were less than nine years old as
14 new, and older than nine years old as old. And we
15 made this cutoff based on discussions with field
16 owners and managers who told us that typically
17 warranties on the fields run out about eight to ten
18 years, and at that point fields may be replaced.

19 As I mentioned, our goal was to collect
20 the samples to quantify and characterize the
21 chemicals. Our collaborators at the Lawrence Berkeley
22 National Lab were instrumental in helping us achieve
23 this goal. They designed an experimental unit that
24 consists of a regulation soccer goal and the depth of
25 the penalty box, as you can see here in this pictures.

1 So the goal net was used as a backstop,
2 and we had air-sampling packages on both sides as well
3 as behind the net. We used volunteers to kind of kick
4 the ball around on the field to simulate activity on
5 the field and we also used a soccer ball kicking
6 machine, as you can see on the picture on the right
7 here, to kick a ball into the goal area to kind of
8 simulate activity within that area as samples were
9 being taken.

10 Now, the top two pictures here show
11 more details about what the sampling packages look
12 like. So in addition to the equipment for air
13 sampling, these sampling packages also contained
14 equipment to measure temperature, humidity, wind
15 speed, direction, as well as particle collection.

16 So we sampled air on the field and at a
17 nearby off-field location so that way we could
18 characterize differences in chemical exposure. We
19 used targeted chemical lists that we derived from
20 literature research, solvent extractions of crumb
21 rubber, as well as chamber emission and thermal
22 desorption studies.

23 And our samples were analyzed for
24 volatile organic chemicals, VOCs; semi-volatile
25 organic chemicals, SVOCs; and carbonyls, aldehydes and

1 ketones.

2 We also did stratified sampling where
3 we collected samples at different heights above the
4 field so that way we could differentiate the source of
5 the chemicals which I'll --

6 (Lost audio.)

7 DR. CLAUDE: I'll restate that. That table
8 at the top there shows the total number of samples we
9 collected across the 35 fields. And the bottom table
10 shows how many were detected.

11 So out of targeted 55 VOCs, we detected
12 46 chemicals on-field and 45 off-field.

13 Out of 13 targeted carbonyls, we
14 detected 11 on-field and three off-field.

15 For semi-volatiles we targeted 70 and
16 detected 62 and 60 chemicals, respectively, on- and
17 off-field.

18 Now, in our analysis of the crumb
19 rubber samples, we collected several hundred crumb
20 rubber samples across all the fields, and they were
21 analyzed with artificial biofluids to simulate dermal
22 and ingestion exposures. These were analyzed and we
23 identified chemicals and calculated bioaccessible
24 concentrations. So these are concentrations that
25 would be releasable into the human body for absorption

1 from the GI tract or the skin.

2 So using our gastrointestinal or GI
3 fluids, as indicated on the slide, we detected 30
4 metals and 76 organic chemicals for dermal biofluids,
5 which was comprised of mainly sweat. We detected 75
6 organic chemicals.

7 All together, 81 of the 86 targeted
8 chemicals were detected with 70 chemicals being
9 detected in both biofluids.

10 Some of our organic classes that we
11 detected include aldehydes, phthalates, and PAHs. We
12 did not detect any PFAS in our analysis, which
13 included nontarget analysis on both the air and the
14 crumb rubber samples.

15 You may note here in the slide, metals
16 were only analyzed in our GI fluids. There is data
17 that shows that the bioaccessibility of metals in
18 crumb rubber and released into the air is very low, so
19 considering this, inhalation and dermal exposure to
20 metals was very unlikely and was not assessed in this
21 study.

22 So our chair has joined. Do we want to
23 take a moment to give an introduction?

24 DR. BALMES: Thank you. I don't know if you
25 heard. I took Capital Corridor Amtrak California

1 train from Berkeley this morning, and we made it on
2 time to Sacramento, except that we were a few yards
3 outside of the station and there was a signal problem
4 and we literally sat there for 40 minutes. So much
5 for taking public transportation.

6 Anyway, I'm pleased that we're finally
7 having what I think will be the final public workshop
8 or meeting of this advisory committee. It's been ten
9 years. It's a long time. The pandemic didn't help.
10 But I'm actually very pleased that we're here to
11 discuss the report, and I think it's, if I might say,
12 well-written. It's actually understandable, I would
13 think, for many people even without a scientific
14 background.

15 So I'm sorry I'm late, but I'm pleased
16 to be here, and I look forward to the rest of the day.
17 Thank you.

18 DR. CLAUDE: Thank you. To continue, as
19 part of our chemical characterization process, we also
20 used the data to help determine each chemical's
21 primary source. So we used this to help differentiate
22 which chemicals might be coming from the field versus
23 which chemicals might be coming from other
24 environmental or human sources.

25 For chemicals that would be coming from

1 the field, we designated them as field-related, as
2 indicated here on the slide. And from those which
3 might be coming from another source, we designated
4 them as non-field-related.

5 So for all metals and SVOCs, our target
6 lists for those chemicals were derived from direct
7 analysis of crumb rubber itself, so we presumed all of
8 those to be field-related.

9 For VOCs, we used those stratified
10 samples that I mentioned a few minutes ago to help
11 differentiate their source. So as the VOCs get
12 released from the crumb rubber, they're going to enter
13 the air and become less concentrated as they rise from
14 the surface and mix with the ambient air.

15 So for VOCs that had statistically
16 significant higher concentrations closer to the field
17 and followed a trend of decreasing concentrations with
18 increasing height, we designated those as
19 field-related. For all chemicals that lacked that
20 gradient, we designated them as non-field-related.

21 And the specifics or analysis for this
22 can be found in Chapter 3 as well Appendix D in the
23 report.

24 So for aldehydes and ketones, for
25 chemicals where there was stratified data available,

1 we used that to make the designation. For chemicals
2 that didn't have stratified data, we used the South
3 Coast Air Quality Management District's Multiple Air
4 Toxics Exposure Study, MATES, exposure as a point of
5 comparison for our study data from fields in the same
6 district to make our designation, if there was
7 chemical data available.

8 So if our data was higher than what was
9 reported by MATES to be the ambient concentration, we
10 designated it as field-related.

11 And then for chemicals where there was
12 no tower data and no MATES data available, we presumed
13 that it was field-related.

14 So for the last slide for our field
15 characterization, we collected environmental data at
16 each field, and these data were collected for one hour
17 before, three hours during, and one hour following
18 field activity. So we collected temperature data
19 continuously at various heights.

20 The air temperatures decreased as the
21 height increased. And the average surface
22 temperatures increased as the ambient temperatures
23 increased.

24 In general, our surface temperatures
25 were about 20 degrees higher than the ambient

1 temperatures, and as you can see on the slide, the
2 maximum reported surface temperature was 131 degrees.

3 We also measured ozone, which is known
4 to affect the aging of crumb rubber. It's naturally
5 occurring, but it also is formed near the ground
6 surface from photochemical reactions. So all measured
7 concentrations were within the California ambient
8 zone's one-hour exposure standard of 90 parts per
9 billion.

10 There were three exceedances of the
11 eight-hour standard of 70 parts per billion with a max
12 value of 87 parts per billion.

13 We collected PM2.5 on and off the
14 field, and levels observed on and off the field were
15 similar and activity did not appear to increase the
16 particle concentrations that were observed.

17 Moving to the exposure
18 characterization, our goal here was to characterize
19 the exposure of soccer players and other related users
20 of synthetic turf fields. So we focused on soccer due
21 to its popularity in both males and females. Players
22 tended to start from a young age and continued all the
23 way to adulthood.

24 And we looked at four main groups
25 including athletes, soccer coaches, referees, as well

1 as spectators. So we evaluated exposure on-field for
2 all these groups as well as off-field for spectators
3 who might sit on the sidelines or stand on the
4 sidelines.

5 These groups have a wide range of ages
6 that could be associated with them. So those ages
7 represent different stages of development, activity
8 patterns, as well as susceptibility to exposure. So
9 to account for this, we broke our receptor age groups
10 into these various groupings, as you can see here on
11 the slide.

12 Each check mark indicates that we did
13 an assessment for that age group for that receptor.
14 So for athletes, we evaluated ages 2 to 70. Coaches
15 and referees, we evaluated ages 16 to 70. And for
16 spectators, we evaluated the third-trimester fetus all
17 the way to age 70.

18 This slide here shows our exposure
19 model. So it describes the potential pathways that
20 receptors can be exposed on synthetic turf fields.
21 So, for example, here at the top it illustrates how
22 gases or vapors can be released from the field and
23 available for inhalation by all receptor groups.

24 Our model also shows which pathways we
25 deemed were either incomplete, negligible, or not

1 dominant, such as dermal exposure of vapor and
2 suspended particles; and thus, we excluded those from
3 our analysis.

4 So for us to evaluate the exposure of
5 soccer players and the other exposure groups, we
6 needed to characterize their activities and exposure
7 patterns. To do this, we found no information in the
8 literature that was relevant to help us, so we
9 developed three exposure studies in collaboration with
10 both UC Berkeley and the University of Arizona, and we
11 conducted three time-activity studies, as we call
12 them.

13 We did a survey, a field observational
14 study, as well as an archived recording study, and the
15 study reports and data for each of these can be found
16 in the supplemental materials for the draft report.

17 But briefly, the survey captured soccer
18 players' activity patterns and behaviors on the
19 fields. We also collected data online and in person.
20 We collected demographic data. We were able to look
21 at what kind of activities they did during practices
22 like diving or sliding, and the types of direct
23 contact they might have with the field.

24 We also collected information on the
25 frequency of practices and games, as well as what

1 types of uniforms they wore, so shorts and long sleeve
2 versus long-sleeve shirts; personal hygiene practices;
3 as well as soccer history. And overall we received
4 1,029 responses online and 40 in-person responses.

5 And for those 40 in-person responses,
6 those players were also videotaped at either a
7 practice or a game. So University of Arizona analyzed
8 the videotape that we collected for micro-level
9 activity data, and so this data tells you the types
10 and behaviors that occur on field with very precise
11 detail, like for how long, how many contacts.

12 For the archived video recording study,
13 the University of Arizona translated archived footage
14 of children playing on synthetic turf or in playground
15 settings. And these data were helpful in developing
16 mouthing frequency parameters specifically for the
17 infant spectators on the field.

18 All together, these three exposure
19 studies were used to help us evaluate inhalation,
20 dermal, and ingestion exposure on the fields for all
21 the receptors. You can see in this table for each
22 pathway some of the pathway-specific parameters that
23 we developed.

24 One of them is the time-weighted
25 breathing rate during activity. So we estimated these

1 using reported exertion levels from the survey data,
2 as well as we looked into the literature to develop
3 some breathing rates that would be relevant for
4 trained athletes.

5 We developed dermal loading onto the
6 skin, which we used body metric data that was provided
7 to us along with kind of estimates of what body parts
8 and to what extent they might be exposed based on how
9 soccer players report their dress during activities.

10 We also developed ingestion rates by
11 combining the amount of crumb rubber that might be
12 incidentally or accidentally ingested as well as what
13 might be indirectly ingested from pathways including
14 hand to mouth, hand to object to mouth, and object to
15 mouth.

16 So the details of how we developed all
17 these parameters can be found in Appendix B in the
18 draft report.

19 We were also -- we are also able to
20 develop body weight and exposure duration parameters
21 specific to athletes for the soccer scenario.

22 Though we did not collect survey data
23 specifically for coaches, referees, or spectators, we
24 borrowed parameters from athletes based on some
25 assumptions that are detailed in the report.

1 For instance, for coaches, since
2 they're the team leaders, we presume that they're
3 going to be on the field when athletes are on the
4 field, so we used similar parameters for coaches as
5 athletes.

6 And we used mean values for athletes,
7 so for coaches, receptors, and spectators, we used the
8 higher mean estimates, so we took all the means and we
9 used the highest value to make sure that we would get
10 a protective value.

11 Now, for the remaining slides, I'm
12 going to talk about our exposure estimation. So this
13 is where we estimate exposure concentrations and
14 exposure doses, which are the dose that a receptor
15 would be exposed to on the field.

16 So we evaluated five specific scenarios
17 that differ based on different types of toxicity. So
18 the first is an acute one-hour inhalation scenario to
19 these 11 chemicals shown here on the screen. So we
20 did not assess acute dermal or oral exposures, which
21 Dr. Krishnan will talk about a little bit later in the
22 afternoon.

23 So these short-term, high-level
24 exposures can lead to adverse health effects on the
25 respiratory and circulatory system.

1 So we used acute exposure
2 concentrations that are equal to the maximum air
3 concentration that was detected at any time point
4 across any of the 35 fields in order to get a
5 health-protective estimate.

6 Now, before I continue, Dr. Krishnan
7 alluded to this a little bit in his introduction. So
8 for the data that we collected for both air samples
9 and in our crumb rubber samples, we have two different
10 options to utilize the data.

11 One option represents individual field
12 data where we can use values that might represent a
13 single home field or home practice field. And these
14 would be relevant for acute effects as well as
15 developmental and reproductive effects or DART, as
16 indicated here on the slides.

17 And DART effects are assumed to have
18 the potential to occur after a single exposure. So
19 these individual field data, since they're collected
20 on a single day, they're a good surrogate for that
21 one-time, single-event exposure.

22 The second option would be to use the
23 average across all the 35 fields, which would reflect
24 players traveling for games or tournaments across the
25 state during the soccer season. And we use this data

1 for all other relevant health end points.

2 For the one-day exposures I just
3 mentioned, we used individual field data to evaluate
4 these chemical end points. And so we did evaluate all
5 three pathways. We did inhalation, dermal, and
6 ingestion. So these exposures focus on a single day
7 or event.

8 And so for inhalation we assessed 18
9 chemicals. The top ten chemicals that contributed to
10 exposure are listed here on the slide. And so for the
11 inhalation exposure concentration, we used the average
12 air concentrations for an individual field, and we
13 used an adjustment factor that adjusted by a
14 receptor's breathing rate and the duration of
15 exposure.

16 For the dermal pathway, we used
17 exposure doses that are represented by the dermal load
18 times the average individual field concentrations in
19 crumb rubber, divided by the body weight. And so for
20 dermal we assessed 19 chemicals. And the top ten
21 contributors to exposure are listed here on the slide.

22 For ingestion, the ingestion dose is
23 represented by the one-day ingestion rate, which
24 assumes a single-event exposure per day, times the
25 average of the individual bioaccessible concentration

1 in crumb rubber all over body weight. So we assessed
2 20 chemicals for this pathway. And again, here the
3 top ten exposure contributors are listed here in the
4 table.

5 So one other end point we looked at was
6 sensory irritants, and these are chemicals that cause
7 irritation to the eye and respiratory system. So
8 there were three chemicals we assessed by this
9 pathway, and the exposure concentration for these was
10 just the average air concentrations since it's
11 concentration-dependent mediated effect.

12 So now for chemicals that have all
13 other end points other than acute, sensory irritation
14 or developmental end points, we evaluated chronic
15 exposure. We did inhalation, ingestion, and dermal
16 exposures.

17 So for inhalation, the exposure
18 concentration is equal to the average air
19 concentration times that adjustment factor, once
20 again, for breathing rate as well as exposure
21 duration. The top ten contributors here are listed on
22 the slide for spectators ages zero to two.

23 For the dermal pathway, the daily dose
24 is equal to the dermal load times the average crumb
25 rubber bioaccessible concentration times the events

1 per year, all over the body weight. And the top ten
2 contributors are listed here on the slide.

3 For ingestion, we assessed 69
4 chemicals, and the exposure dose is equal to the daily
5 ingestion rate times the crumb rubber bioaccessible
6 concentration over the body weight. And for all of
7 these chronic exposures, we did the calculations using
8 both individual field data as well as the average
9 across all fields.

10 For chemicals that are known
11 carcinogens, we calculated lifetime average daily
12 doses for those. So we assessed each life stage from
13 the third trimester of pregnancy all the way to 70
14 years old, and these doses measured the potential
15 lifetime exposure to a chemical.

16 In order to do those calculations, we
17 used average daily doses. So for dermal ingestion,
18 the doses are the same as I just described a couple
19 slides ago. But I do want to highlight here the
20 average inhalation daily dose. So unlike for acute,
21 one-day and chronic where we use an exposure
22 concentration, here we use an inhalation daily dose
23 that's equal to the air concentration times a
24 breathing rate over the body weight.

25 And those average daily doses are used

1 to calculate the lifetime average daily dose by
2 multiplying by an age-sensitivity factor, which is a
3 time-weighting factor that accounts for increased
4 susceptibility to chemical exposure earlier in life.
5 So it modifies the chemical's cancer potency for each
6 life stage presuming an increased sensitivity early in
7 life. So it decreases with age from a factor of 10
8 for infants to 1 for adults.

9 We also include an exposure duration
10 that represents the length of exposure, and all of
11 this is divided by the averaging time, which
12 represents an average lifetime of 70 years.

13 Shown here are ten chemicals. These
14 ten chemicals were evaluated across all three
15 pathways. So on this slide you can see how -- and all
16 these are for spectators zero to two. So you can see
17 how the lifetime average daily dose can vary across
18 the pathways and how a pathway with the largest
19 contribution can vary for each chemical.

20 So this afternoon's presentation will
21 look more into how we assess chemicals that were
22 detected across these multi-routes.

23 So just to wrap up, so we sampled 35
24 fields across California. We collected air and crumb
25 rubber samples. We identified chemicals that

1 potentially could be released from them. So we
2 identified 119 chemicals in the air above the turf
3 fields as well as 75 chemicals in the dermal extracts.
4 We identified 76 organic chemicals and 30 metals in
5 the gastric extracts of crumb rubber. And we
6 estimated exposures to these chemicals using
7 California soccer-specific exposure parameters that
8 were developed from three time-activity studies we
9 conducted.

10 So for all of our calculations where I
11 indicated previously we utilized either individual
12 field data or we utilized the average across all the
13 fields, here I only presented a small fraction of the
14 results. So for any more results or details, I
15 encourage you to look at the report, both the main
16 report as well as the appendices and supplemental
17 materials as they contain a lot more on the sampling
18 methods and the specific sampling results.

19 Now, with that, I'd like to turn it
20 back over to Dr. Balmes for clarifying questions and
21 panel discussions and also we want to introduce
22 Dr. Amy Kyle who has joined us.

23 DR. BALMES: Dr. Kyle, do you want to say
24 hi?

25 DR. KYLE: I'm happy to be here and happy to

1 see you all again, and while I have the floor for just
2 a moment, I want to say I thought you guys did an
3 amazing job explaining this in the report. Truly
4 amazing. Blue ribbon. I do have some questions, but
5 I just wanted to say that first. So thank you.

6 DR. BALMES: And I will say, because I've
7 worked with Dr. Kyle for a long time, that's a major
8 compliment. And I would like to compliment you on
9 your presentation just now, the part of it I caught.
10 Because it was also very clear. Good slides.

11 Now, the panel may ask clarifying
12 questions or make comments.

13 Is your mic on?

14 DR. BENNETT: Again, I agree, this was a
15 great report. You guys did a great job. And I feel
16 like some of my clarifying questions are super
17 detailed because those were the only questions I had
18 to come up with, but some of them are bigger.

19 So on the time activity for the one-day
20 exposure, I wasn't quite clear on the decision to only
21 use the mean exposure duration for the athletes,
22 because especially I did not -- I will admit I did not
23 dig into your appendices. But it looked like the mean
24 was on the order of the length of one game, and I was
25 thinking for that one-day exposure, you would want to

1 be reflective of kind of a day where they're playing a
2 tournament on a field. And I wasn't quite sure why --
3 and maybe I misunderstood why the one-day exposure
4 seemed to be on the order of only I think it said like
5 two and a half hours.

6 DR. CLAUDE: So for some of the parameters
7 from the time-activity studies, some of them we asked
8 them how many days per week do you practice, how many
9 games per week do you practice or play. So for some
10 of them, when we calculated, we calculated only using
11 individual athlete's data, so we didn't calculate a
12 mean across all of them.

13 And for some of them, we did exclude
14 data because we don't know if it's just
15 misunderstanding the question. We did have some
16 people report that they used like maybe 16 hours a day
17 they practiced on the field or 20 hours a day they
18 practiced on the field. So some of those higher end
19 values did wind up getting excluded just because we
20 weren't sure about what the data actually looked like,
21 and we didn't want to potentially overestimate too
22 much, telling people six hours a day on the field or
23 something like that.

24 So we went with the mean to kind of
25 just look at what the average scenario would be.

1 DR. BENNETT: But I think a lot of them do
2 spend six hours a day on a field on a tournament.

3 DR. CLAUDE: Some of these were like for
4 little kids, you know like six-year-olds or
5 12-year-olds. So we're not sure if there was just
6 misinterpretation of the question. Like for some
7 older athletes, we did have some professionals who
8 answered the survey, and for sure, they might spend
9 all day on the field practicing or playing different
10 types of games and stuff.

11 DR. BENNETT: I saw the thing about the 16
12 hours a day. That did seem a little excessive, but I
13 feel like there's data that clearly was a
14 misunderstanding of the question. But then to go all
15 the way from those maximum ones that misunderstood
16 down to the average, and basically effectively exclude
17 on your one-day exposure anyone playing a tournament,
18 seemed like an odd choice to me.

19 DR. CLAUDE: And a lot of those higher end
20 values, they were definitely the high end. There was
21 a clear separation too as well between those kind of
22 values where we were like this might be
23 misunderstanding versus the mean. So we can revisit
24 it to kind of see what -- when we take out what that
25 kind of high-end mean looks like to see if maybe we

1 can use something to better represent if there was a
2 tournament several days.

3 DR. BENNETT: Yeah, because I mean, because
4 if the DART is a health end point applicable to a
5 one-day exposure, there's a lot of kids that play in
6 tournaments. I don't have kids that played soccer
7 tournaments, but from my friends that did, they would
8 be at Stockton at 8:00 a.m. and they would be there on
9 the field until 5:00 p.m. and back the next day for a
10 repeat encore. I just wasn't sure about that. So
11 that's my first question.

12 My second comment was, you talked about
13 how you did not sum athletes -- people being different
14 positions, but I think it's -- actually, I'm going to
15 skip that one.

16 So my next question is on the lifetime
17 exposure. I wasn't quite clear, when you summed up,
18 were you summing up assuming a kid went from being a
19 spectator to a player and then through the years or
20 were you just summing the total over each given stage?

21 DR. CLAUDE: Each given stage. So we did
22 calculations for the incremental risk, so we didn't
23 sum any of the age groups together. We presented
24 values for each age group separately.

25 We didn't assume you'd be a spectator

1 during pregnancy and then zero to two and then you
2 might transition to an athlete and then to a coach or
3 receptor. We didn't do any summations like that. We
4 just strictly calculated for each specific age group
5 for each receptor.

6 DR. BENNETT: And so if you wanted to get
7 the lifetime risk, somebody would need to sum the --

8 DR. CLAUDE: Yeah, you would have to sum the
9 relevant categories.

10 DR. BENNETT: You just might want to make
11 that a little bit clearer in the text because that
12 wasn't super clear.

13 And then on the field selection, it
14 appeared you only had one field that was fairly new,
15 less than a year old. And it seemed to me that was
16 one with the non-crumb rubber. And I just didn't know
17 if -- it did look like you had three fields that were
18 a year old. And I just didn't know if you had done
19 any analysis to see if when those fields were very
20 new, if there was any increase in the VOCs, just
21 because like intuitively you kind of think, huh, that
22 first year they'd have the highest levels. And I
23 noticed that there was very little measured in that
24 initial time frame. And I just wanted to hear your
25 thoughts on that.

1 DR. CLAUDE: There is data that shows that
2 once you know those new fields, that first year is
3 when you get the largest off-gas of those volatile
4 chemicals. So if I recall, we didn't see any specific
5 increase compared to the other fields for those
6 fields --

7 DR. BENNETT: Because you didn't measure any
8 the first year.

9 DR. CLAUDE: -- either for the air or the
10 crumb rubber because, like I said, we only had a
11 couple out of the whole 35 that we see. We didn't see
12 very much difference. As well as other fields too,
13 sometimes the -- sometimes the crumb rubber, it will
14 get in the shoes so it will get taken off the field.
15 So some other fields do replenish the crumb rubber as
16 the season goes along, so the field might not be new
17 but they might still have new areas of crumb rubber
18 that have been on there too, so that might dilute some
19 of that kind of analysis as well.

20 DR. BENNETT: Do you think it would make
21 sense to know that as like a potential limitation if
22 there weren't really very many? There wasn't really
23 anything that was less than a year old except for that
24 one, if I remember.

25 I have other questions but I'll let

1 somebody else take a turn for a while.

2 DR. BALMES: I'd like to recognize
3 Dr. McKone, who is remote.

4 DR. MCKONE: Thank you. Sorry I couldn't be
5 there in person, but I guess the technology I chose
6 was a little bit more reliable than the train today.
7 It would have been three of us that would have been
8 late, but I made it on time.

9 First of all, I want to add my
10 compliment. This is a just such a remarkable study,
11 and of course, we've been following it for years. But
12 for anyone who has questions about -- I mean, I think
13 for the general audience and the scientific audience,
14 just the detailed efforts to not make assumptions but
15 actually make so many measurements. You worked with
16 some really good scientists. I'm biased because some
17 of them worked with me when I wasn't retired and they
18 weren't. But it was really clever.

19 And I think it's compelling that this
20 is not just a hand-waving exercise. It's really
21 literally in the dirt or in the crumb rubber and
22 really looking.

23 So I had kind of a comment or a
24 question, something I wanted to explore a bit that you
25 alluded to, I think early on about, if you look at

1 this list of chemicals, many of these, probably most
2 of them are chemicals people are exposed to from other
3 sources. There are toxic air contaminants or they're
4 in consumer products.

5 I know you tried to mention -- or you
6 mentioned something about looking at the relative
7 exposure from people associated with soccer events,
8 either on-field or off-field. And I was wondering the
9 extent to which you were really able to put it in
10 context in terms of cumulative exposure to toxic air
11 contaminants from all other sources.

12 Again, that's highly variable, so
13 that's hard to do. Some of the communities that
14 people live in are quite clean and others are coming
15 from communities where I'm guessing a substantial
16 amount of their exposure to these substances may be in
17 their backyard and not on their soccer field.

18 But I don't know if you could just
19 comment a little bit about how -- I guess how you
20 would address people's question about, is this big
21 relative to their cumulative exposure to these
22 substances from other sources over the lifetime, and
23 if so, how might we communicate that?

24 DR. CLAUDE: That's a good question. So
25 yes, we really didn't compare to other sources, like

1 for the chemicals that -- like for example, we
2 detected all the BTEX chemicals. And when we sampled,
3 we sampled in the morning during high periods of
4 traffic and people traveling to work and school and
5 other places.

6 So we did see fluctuations of those
7 types of chemicals with the time pattern of the day.
8 So it kind of increased in the morning, and then it
9 kind of flattened out in the afternoon. So we did see
10 those kinds of patterns for certain chemicals that we
11 presume you'll have other sources of exposure to them.

12 So we did do hazard and risk. We did
13 separate out our calculations for what we presumed to
14 be field-related versus what we might presume to be
15 non-field-related. And so we did not compare those
16 kind of non-field-related exposures to anything that
17 might be in ambient air or any types of other
18 cumulative exposures from those types of things.

19 But we did see predominantly that a lot
20 of the exposures were driven by those
21 non-field-related chemicals. The contribution from
22 the field-related chemicals tended to be much less
23 than the field-related exposures. So it is a
24 likelihood that a lot of those exposures could be
25 coming from other sources, traffic or some fields are

1 located where the area is industrial areas or just
2 high areas of traffic.

3 DR. MCKONE: If I can follow up, it seems,
4 though this is kind of getting into the next topic,
5 but mostly the concern is acute exposures, right, or
6 maybe chronic over a period when someone is in soccer.

7 So I guess the question I'm asking is
8 probably only relevant for chemicals that give rise to
9 chronic -- long-term chronic exposures and lifetime
10 burdens of disease, whereas I think the bigger issue
11 was acute for a lot of these. I guess we can take
12 this up this afternoon. But I think in kind of
13 supporting your response, because the goal was to look
14 at short-term typically acute exposures during a day
15 or during a soccer game.

16 Then the other non-field-related
17 exposures probably wouldn't matter that much unless
18 they just happened to be near a facility that was
19 having an off-normal event and releasing chemicals,
20 which I don't think that's part of your purview to go
21 into a rare event like that. But anyway, thank you.

22 DR. BALMES: Thanks, Dr. McKone. And if I
23 could just jump in a little bit, Jocelyn. I think
24 that exchange with Dr. McKone, it might be worth
25 considering adding some wording about how -- even

1 though we don't know how the synthetic turf exposures
2 compare to exposures to, for example, BTEX chemicals
3 from other sources, I think it's probably worth saying
4 that it's not likely to be an overwhelming exposure.
5 Don't use that word.

6 But I think somebody reading this might
7 have that same kind of question that Dr. McKone just
8 dealt with trying to put it in context. I know you're
9 being very careful about on-field and off-field and
10 not exceeding your purview. But I think putting that
11 in context for the public would be helpful.

12 DR. CLAUDE: Okay.

13 DR. BALMES: So I'm going to turn to
14 Dr. Eckel next.

15 DR. ECKEL: I would like to echo the rest of
16 the panel and say that this is a really great study.
17 I really enjoyed reading it, and I've enjoyed seeing
18 the development over time, and so my comments are
19 coming from a statistician point of view. So I really
20 appreciated the rigor that I saw and the sampling of
21 the fields, that stratified random sampling to really
22 characterize the different ages and the different
23 climate areas of California. I thought that was
24 great.

25 And I also really appreciated the

1 characterization of the on-field based on the height
2 of the VOCs, for example, using that linear mix model.
3 I thought that was great to see that -- the state of
4 the science on statistical methodology.

5 I did have one question about the
6 exposure characterization, and I saw looking at like
7 individual fields versus looking at averages across
8 all the fields of exposures to chemicals, and I was
9 wondering, did the approach that you take -- when I
10 was reading through and seeing your presentation, it
11 looked like for shorter exposures, like maybe one-day
12 exposures, you looked at individual fields, but for
13 longer exposures it was more of an average across all
14 the fields.

15 But I was wondering if maybe that
16 didn't capture individuals who maybe are playing on
17 the same home field over a period of years because an
18 average does attenuate a little bit the extremes.

19 DR. CLAUDE: And we did. So for those ones
20 like the chronic exposure, we did do calculations with
21 the average across all the fields, assuming people
22 will travel, but we also did those individual fields
23 assuming people might have a home base kind of field
24 for practice and/or games. So we do have both those
25 data.

1 DR. ECKEL: Oh, great.

2 DR. CLAUDE: So it's both in the appendix.

3 In the main report we just have the individual data,
4 but we did present the data for both.

5 And generally, when we look at the
6 distribution of the individual fields as well, the
7 average value tended to fall right around where the
8 average across all the fields looked at. So we
9 thought that was -- we were like that's nice-looking
10 data, right, that were our average. So we did kind of
11 look at both of those situations to kind of see what
12 it would look like, especially for like younger kids.
13 They're probably most likely not going to travel
14 across the state. They'll probably have that one
15 field that they might look at.

16 So we did look at both of those
17 scenarios for the chronic exposures.

18 DR. ECKEL: Thank you.

19 DR. BALMES: I would be careful in saying
20 that they wouldn't travel across the state. I had a
21 kid who was on a traveling team, and he traveled all
22 over the state.

23 Dr. Avol.

24 MR. AVOL: I just have a couple of questions
25 and I might ask you a question much larger than this,

1 which is, I know you focused on exposure
2 characterization, and this afternoon we'll hear more
3 about the health assessment, but some of your summary
4 and questions did get -- had some of the health. So I
5 don't know whether to hold this and expect that we'll
6 hear more in the afternoon or ask it now.

7 DR. CLAUDE: I would say ask now because the
8 topics do very much kind of intertwine with each
9 other. So I would say ask away, and if there will be
10 more on it this afternoon, we can talk about --

11 MR. AVOL: I only have maybe one or two
12 additional comments over and above my colleagues. And
13 parenthetically, I will say that as a parent of
14 children who did play lots of soccer on lots of fields
15 and did play both local AYSO and traveling squad team,
16 Dr. Balmes is exactly right. We were all over the
17 state.

18 But more importantly, I think for many
19 hours a day, particularly at tournaments, as
20 Dr. Bennett suggested, the teams will stay at or near
21 the fields and be there for hours of the day. So I
22 think you may have thought about it may be only
23 minimal exposures that you consider in the scheme of
24 what teams actually do in these tournaments. We were
25 there for a weekend.

1 So I think whether that comes up in the
2 limitations or discussions or commentary, I think it's
3 worth thinking about how you sort of address that.
4 Because I know that's an issue for many parents who
5 are going to look at this and say, well, wait a
6 second. That's not my experience.

7 I have two questions related to what
8 you presented and then your summary. But you talked
9 about this sort of defining point, older or younger
10 than, I guess, nine years of field life. But when you
11 summarized your data, you didn't say anything about
12 whether there was any appreciable difference you
13 wanted to call attention to, or just say that you
14 really didn't see much difference.

15 Again, Dr. Bennett alluded to this
16 question of the very new fields, which is certainly an
17 issue. But because there's so many fields in use
18 across California and the country, I was wondering if
19 you did have any insights or whether there will be
20 more said in the final report about this dichotomy --
21 if there is such a dichotomy.

22 DR. CLAUDE: We really didn't see much of
23 that kind of separation between like the old versus
24 the new because, like I said, some of those older
25 fields as the years go on, they do replenish the

1 crumb. So when your field gets to be nine years old,
2 your crumb is likely not going to be nine years old.
3 You will have replenished either certain areas or the
4 whole field by that time point.

5 But we could make a note in the report
6 to address that we didn't see that kind of breakdown,
7 that old versus new kind of difference in either the
8 VOCs, the difference in the release or the crumb
9 rubber, the presence of those chemicals.

10 MR. AVOL: I think it also would be useful
11 for the public to understand better through OEHHA's
12 sort of guided expertise eyes to say something about
13 the mean versus the max in terms of what you've seen
14 in the range of these fields. Because, again, you
15 talked about a number of fields. You talked about a
16 number of concentration, but then you sort of focused
17 in on this is the average exposure. This is the
18 average thing.

19 I understand why we do that, but I
20 think there's still some concerns about these outlier
21 conditions which do -- may not be so much outlier for
22 a number of people.

23 DR. CLAUDE: Yes. Thank you.

24 DR. BALMES: Dr. Kyle.

25 DR. KYLE: Thank you. I'm thinking about

1 not only the details of the study and the methods but
2 also what did we learn from this is kind of the main
3 thing on my mind because of some of the reasons people
4 mentioned of the depth of the analysis that you did,
5 the creativity of it, and the fact that we looked at
6 this in a different way than we often do.

7 And so this is the spirit in which I'm
8 asking this question. I don't know if it's germane,
9 really, to this part, but it's from the beginning.
10 I'm trying to understand what we thought we'd find and
11 then what we found in terms of the substances in this
12 material. And I'm very interested in how that
13 distributed about between the air, dermal, and the
14 ingestion-related one.

15 I think that that pattern is really
16 interesting. But my first question for you is,
17 remember the ones that fell out because we couldn't
18 find an analytic standard for them? Did they ever
19 reappear anywhere? And I understand why you took them
20 out, I think. But I also would like to figure out
21 what they -- I'd like to commemorate that there was
22 some number between 10 and 100, I think.

23 DR. CLAUDE: I forget the exact number, but
24 yeah, so if it disappeared before we could do -- find
25 the standard. So we didn't look for it after that.

1 Because when we did our analysis, we had those
2 specific targeted lists that we used. So if it fell
3 out before it got to that point, we didn't look for it
4 again in the rest of the analysis.

5 DR. KYLE: But can we find that again?

6 DR. CLAUDE: We can pull out those lists of
7 chemicals.

8 DR. KYLE: Because I would just like to see
9 what was left there and the reasons it was left there.
10 And I'm not saying you made the wrong decision, but in
11 terms of what we learned from this, there's something
12 we learned about that piece of it too, and the issue
13 of standards and why they're not available and what is
14 it we don't have standards for at all. And I'm
15 running into this in other contexts as well. But
16 there are whole areas of inquiry that are limited
17 because, oh, gee, we don't have a standard, which in
18 the 21st century seems like kind of a lame answer,
19 doesn't it?

20 So I would like to figure out the right
21 way to ask about that. So I guess that's the main
22 thing I'm going to ask right now. I have some others
23 for later, but thank you.

24 DR. BENNETT: I had some other questions on
25 that, because I got a little confused on some of the

1 non-targeted stuff. So the non-targeted wasn't done
2 by LBNL? It was done somewhere on campus by different
3 people?

4 DR. CLAUDE: LBNL analyzed the samples, they
5 got the extracts, and the sample extracts were
6 analyzed by UC Berkeley's QCB lab because LBNL did not
7 have a liquid gas -- a liquid chromatography machine.
8 So LBNL did all the GC/MS, and then the QCB lab at
9 Berkeley did all the liquid chromatography.

10 DR. BENNETT: And that was only on the pilot
11 samples then?

12 DR. CLAUDE: It was the pilot and then -- we
13 did pilot and then we did a second phase where we --
14 pilot we did four and we did the manufacturing
15 samples, and then we expanded a little bit for the
16 second phase and we did some of those.

17 DR. BENNETT: And then in addition to the
18 ones that you couldn't buy the standard for, were
19 there other ones that you just couldn't identify for
20 sure what the compound was? The nontarget world, they
21 come up with so much stuff, and I also felt a little
22 bit like ...

23 DR. CLAUDE: And I think a couple meetings
24 ago we showed some of the chromatograms, and you could
25 see there's so much stuff in there. So there was some

1 stuff, I think, that we weren't able to identify. I
2 don't know off the top of my head the magnitude of how
3 many that was, but there was some stuff that was just
4 kind of out that we were not able to identify.

5 DR. BENNETT: It said they didn't find any
6 of the PFAS in that. So did they look -- they ran a
7 library of PFAS versus the nontarget?

8 DR. CLAUDE: Yeah, so in our nontarget
9 analysis we didn't see any PFAS, but also we looked at
10 the crumb rubber, so we didn't look at any of the
11 blades or the backing which is likely where the PFAS
12 would be present. And in the air samples PFAS are
13 likely not going to volatilize.

14 DR. BALMES: And that's in the report.

15 DR. CLAUDE: Yes, and we did put that in the
16 report.

17 DR. BENNETT: And you didn't really look for
18 any OPEs either, the organophosphate ones?

19 DR. CLAUDE: No.

20 DR. BENNETT: I was also confused on the
21 volatile sulfur compounds because it said they did
22 something with the volatile sulfur compounds in the
23 pilot, and then that seemed to go away. I didn't see
24 where there was any reference to like "we just didn't
25 see anything."

1 DR. CLAUDE: So those volatiles, that was
2 for the benzothiazole that we wanted to make sure if
3 it was there, we captured it; as well as the
4 2-Mercaptobenzothiazole. So when we were doing the
5 analysis, they came up in the regular VOC analysis, so
6 we wound up not needing to do the additional
7 specialized sampling and analysis for those volatile
8 sulfur compounds.

9 So that was mainly to make sure that we
10 saw the benzothiazole.

11 DR. BENNETT: Okay. I missed that, so you
12 might just want to put -- I mean, maybe it was in
13 there but ...

14 Then I have one more question on the
15 chemical analysis. So I did notice that you guys did
16 find a few fields where the maximum was just really
17 high for a few compounds. I didn't ever find anything
18 to get a sense as to whether or not those really high
19 levels were scattered across different fields or if it
20 was just like a limited number of fields that tended
21 to be the outlier fields for all of the different
22 concentrations.

23 I didn't know if you did anything to
24 look at that or not, to just get a sense of is it one
25 chemical high here and another somewhere else or is it

1 just some fields that were just high.

2 DR. CLAUDE: We didn't look to see if -- you
3 know, in the high end, those fields, it was all one
4 field. We did note some fields did tend to have
5 higher concentrations of chemicals. We measured wind
6 speed and all that kind of -- so for some fields we
7 did look to see if that kind of played into seeing
8 those higher levels.

9 And we did have some fields where we
10 had frequent changes in wind direction that we noted,
11 so that could be why we did see either higher or lower
12 concentrations of chemicals. But we didn't look to
13 see if those higher values all tended to congregate in
14 one field. We didn't look specifically at that kind
15 of individual analysis. But that's something we could
16 look at to see if it was localized into certain areas,
17 all those max values.

18 DR. BENNETT: I think that's everything I
19 had.

20 DR. BALMES: I think Dr. Avol has another
21 question.

22 MR. AVOL: I have one question to sort of
23 help place this into relevance. Given that the
24 sampling on this study, I mean, it was an excellent
25 study, tremendous detail has evolved -- been

1 appreciating. But given that the sampling was done
2 almost ten years ago, and I think it's fair for public
3 to ask a question of relevance in terms of current
4 fields.

5 So I don't know if there's information
6 that you have access to or if you looked at or even
7 thought about as to whether in today's fields in the
8 last five years, the fields that are being used now,
9 if crumb rubber from passenger and light-duty vehicle
10 trucks is still the primary in-fill or if there has
11 been a move by the industry to go to coconut or
12 something else, that is, is all your hard work
13 still --

14 DR. CLAUDE: Still relevant? Yeah. And
15 when we did the study ten years ago, there was already
16 that you started seeing kind of people moving towards
17 the alternatives because the senate, they tried to
18 push for that moratorium to stop installing fields and
19 parents became more, "Are we going to put this? Is
20 there an alternative?" That's why we did see those
21 organic, corn husks, coconut husks, you've got sand,
22 and I forget Nike, they had their recycled shoe rubber
23 kind of in-fill.

24 So there have been quite a few in-fills
25 that have been pushed in the past ten years as well.

1 And we haven't updated our database, so we don't know
2 of the 900 that were in California how many might
3 still be around or how many might have switched to an
4 alternative or gone back to natural grass. So we
5 don't have that information at this time, but yeah.

6 DR. BALMES: Anybody else have another
7 question?

8 So I have one issue, and it may not be
9 the right time. I know we have public comment this
10 afternoon. But the report came out in March. Have
11 there been written comments from the public, from
12 scientists, that you can tell us how you responded to?

13 DR. CLAUDE: So we received actually a
14 comment yesterday, a written one, to our e-mail -- so
15 we received a comment yesterday from the public that
16 obviously we haven't responded to yet, but it dealt
17 with the issues around the heat stress on the field
18 and the temperatures, so that issue where the surface
19 temperature, it gets hot. So people have complained
20 of getting burns and your shoes are melting.

21 So after the meeting we'll read into
22 that comment more and see how we address those issues
23 there.

24 DR. BALMES: Was that the only written
25 comment?

1 DR. CLAUDE: That's the only written
2 comment.

3 DR. BALMES: That actually dovetails to a
4 concern. Dave and I, when you posted the surface
5 temperature, I said "Whoa." This is only going to get
6 worse with the climate change -- dare I use that term?

7 And I have heard about when my son was
8 a soccer player, when it was really hot, how they felt
9 the heat in their shoes and didn't like to fall when
10 it was that hot.

11 DR. CLAUDE: Yeah. And some managers, you
12 don't need to water the fields, but some managers,
13 they do water the field to kind of help to alleviate
14 some of that temperature. It's only a temporary
15 remedy, but they do tend to water it before the
16 players go on to kind of help with that.

17 DR. BALMES: Do we think that those high
18 surface temperatures affected exposures to the various
19 toxicants that you have looked at?

20 DR. CLAUDE: On hot days you might have more
21 volatilization on those days.

22 DR. BALMES: But we didn't look at that
23 specifically? When I say "we," you.

24 DR. CLAUDE: No, because we collected -- it
25 was mainly the summertime when we collected. We had a

1 lot of those hot days. So we had very few fields that
2 we sampled in like the wintertime, and we didn't
3 sample fields like during both periods. So if we had
4 sampled fields during both periods, that would have
5 been a great way for us to help see.

6 DR. BALMES: I think, again, just a little
7 note in the discussion about how that's a potential
8 concern would be good.

9 Dr. Bennett. Your mic is off.

10 DR. BENNETT: One other quick thing. I just
11 thought on the ingestion, you guys give the mass that
12 you assume for the ingestion. It might be useful just
13 for readability for a non-science audience to convert
14 that into a volume measurement that would be in
15 something people would be familiar with.

16 DR. CLAUDE: Okay.

17 DR. BALMES: That said, I still think given
18 the complexity of the study and the multiple sections
19 of the report, it's in pretty public user-friendly
20 form. Much better than I expected, actually. As
21 scientists.

22 DR. CLAUDE: We did work hard on trying to
23 make sure --

24 DR. BALMES: As scientists we often are not
25 good at communicating with the public, but you did a

1 good job.

2 DR. CLAUDE: Many thanks. It was a group
3 effort. But thanks to all.

4 DR. BALMES: If there are no more questions
5 from the panel here or Dr. McKone virtually, then I
6 think we can adjourn early for our lunch break. But
7 then we'll come back early. We'll come back at 12:45
8 instead of 1:00.

9 (Recess.)

10 DR. BALMES: Our next speaker is
11 Dr. Krishnan. Kannan Krishnan.

12 Take it away.

13 DR. KRISHNAN: Good afternoon. Do you hear
14 me okay?

15 In this presentation I'll focus on
16 toxicity evaluation, risk characterization, and
17 present the conclusions of the draft report.

18 This study examined the non-cancer
19 hazards and cancer risks as the health outcome, as you
20 see in the first column, from exposure to chemicals
21 from crumb rubber in-fill via multiple routes, as
22 shown in the second column, inhalation, dermal,
23 ingestion, on the synthetic turf fields for four
24 receptor categories, athletes, coaches, referees, and
25 spectators and appropriate age groups in each one of

1 those categories.

2 Specifically, the toxicity evaluation
3 or the health outcome focused on acute inhalation
4 exposure to chemicals. That's the first line
5 combining the first two columns. And one-day
6 inhalation, oral, or dermal exposure to DARTs, or the
7 developmental reproductive toxicants, on chronic
8 inhalation exposure to sensory irritants, then chronic
9 inhalation, dermal, or ingestion exposure to general
10 toxicants.

11 What we refer to as general toxicants,
12 those that cause chronic effects other than DART or
13 sensory irritation, where the key critical effect is
14 other than sensory irritation and developmental
15 reproductive toxicity.

16 And then cancer outcome where we
17 calculated the lifetime risk from exposure to
18 carcinogens by inhalation, dermal, and ingestion
19 routes.

20 In applying these on-field or for
21 off-field exposures, the only difference is that the
22 turf materials are only present on-field, obviously.
23 That's the only difference when it comes to the
24 calculations on-field or off-field.

25 In terms of the toxicity evaluation of

1 these chemicals or the dose-response assessment of
2 these chemicals requires the knowledge of toxicity
3 criteria. The toxicity criteria are basically the
4 numerical value that quantitatively characterizes the
5 relationship between the exposure and the outcome such
6 that either reference exposure level is developed for
7 non-cancer end points or cancer slope factors are
8 developed for carcinogens.

9 In terms of developing toxicity
10 criteria, we had five complementary approaches.
11 Initially, toxicity criteria developed by OEHHA or
12 other governmental agencies were used. Then
13 extrapolation using toxic equivalency factors was
14 conducted, particularly using benzo[a]pyrene for the
15 PAHs, and then adopting toxicity criteria from
16 structurally similar chemicals, and then the conduct
17 of route-to-route extrapolation of toxicity criteria.
18 When a value is available for oral route, converting
19 it to an inhalation route and vice versa, particularly
20 for systemically acting chemicals.

21 Finally, development of de novo
22 toxicity criteria for chemicals based on toxicological
23 studies from the literature.

24 For chemicals with established toxicity
25 criteria, the most health-protective value based on

1 the most sensitive end point was used. For chemicals
2 without such established -- my picture keeps coming in
3 the middle of the slides so it's hard to read.

4 For chemicals without established
5 non-cancer toxicity criteria, as alluded to earlier,
6 new toxicity criteria were developed either de novo or
7 based on route-to-route extrapolation or based on
8 structural similarity.

9 For those without any toxicity criteria
10 or toxicity data, those weren't included in risk
11 characterization.

12 In terms of the data availability for
13 acute toxicity, there were 11 chemicals with tox
14 criteria from OEHHA or U.S. EPA for inhalation route.
15 So that was for the acute inhalation toxicity.

16 For those without toxic criteria for
17 the acute exposures, comparisons were made with the
18 subchronic health guidance values. Even though
19 subchronic is a longer duration with lower health
20 guidance values, those were used in this project.

21 Now, regarding DART, six chemicals had
22 toxic criteria from OEHHA or EPA. Two were developed
23 in this study and 12 were based on structural analogs.

24 And for sensory irritation there were
25 three chemicals, as we will see momentarily, and all

1 of them were from OEHHA. And in terms of the chronic
2 toxicity, for the inhalation route, 41 values were
3 available from OEHHA, EPA, and the ATSDR. 52 were
4 based on analog values, structural analogs, and 37
5 were developed based on route-to-route extrapolation.

6 In terms of the oral and dermal route,
7 the toxicity criteria, 18 of them were available from
8 OEHHA and EPA, and 36 were based on structural
9 analogy.

10 Four relatively nontoxic chemicals were
11 excluded from these calculations. The limonenes,
12 pinene and carene were those.

13 And in terms of the cancer slope
14 factor, out of the 23 identified carcinogens in the
15 study, 16 of them had chemical-specific cancer slope
16 factor, five of them had established potency
17 equivalency factor or toxic equivalency factor. Those
18 are the PAHs, polycyclic aromatic hydrocarbons. And
19 for two of them, we had derived specific values in
20 this study.

21 And of the 23 carcinogens, 13 were
22 identified in a single route with a chemical-specific
23 cancer slope factor, whereas ten of them were assessed
24 for multiple routes of exposures, inhalation, dermal,
25 and ingestion.

1 So in terms of risk characterization
2 combining the exposure assessment and toxicity
3 criteria, we have evaluated the acute and chronic
4 non-cancer hazards as well as the lifetime excess
5 cancer risks for each category; that is, athletes,
6 coaches, referees, and spectators, for a total of 33
7 groups, based on individual field data on chemical
8 concentrations as well as the average across fields,
9 as we had indicated.

10 And acute and chronic exposure
11 scenarios for both on-field and off-field were
12 considered in computing the cancer risks and
13 non-cancer hazards.

14 For non-cancer hazards, initially a
15 hazard quotient for each chemical was calculated. So
16 hazard quotient is basically the ratio of the exposure
17 metric to the toxicity criterion or the reference
18 value.

19 So the exposure metric is either an
20 exposure concentration or a dose that's calculated.
21 For inhalation exposures, it's an airborne
22 concentration, whereas for dermal and ingestion
23 exposures, it's an average daily dose.

24 And the toxicity criterion is a
25 chemical-specific numerical value that reflects the

1 potency of the chemical for the specific non-cancer
2 effect and the route of exposure.

3 An example would be the REL, or the
4 reference exposure levels developed by OEHHHA. So
5 these are concentrations at or below, which no adverse
6 health --

7 (No audio.)

8 DR. KRISHNAN: Thanks for bringing that up.

9 Now, for cancer risk, was calculated
10 using the general equation, lifetime average daily
11 dose times the cancer slope factor, CSF. LADD times
12 the CSF for the route of exposure here.

13 The cancer slope factor or CSF is a
14 95th percentile upper confidence limit on the slope of
15 the dose-response curve, which is based on continuous
16 lifetime exposure to a substance.

17 The risk calculations are conducted for
18 multiple chemicals via multiple routes for all age
19 groups. One in a million or one excess cancer in a
20 population of one million people over a lifetime is
21 considered a negligible risk or de minimus risk level.
22 So that's the benchmark that I will be referring to as
23 we go along.

24 So now let's start with some of the
25 results of risk characterization. First, for acute

1 toxicity, using the maximum one-hour concentration of
2 a chemical, detected at any time in any of the fields,
3 the acute hazard quotient for the inhalation route was
4 well below the benchmark of 1, both for on-field and
5 off-field exposures.

6 So there's a single field-related
7 chemical and ten non-field-related chemical, and this
8 table shows that the on-field acute hazard index was
9 well below 1 for the field-related chemicals and also
10 was below 1 for the non-field-related chemicals. And
11 same scenario for the off-field.

12 The styrene was the only field-related
13 chemical with toxicity criteria here, whereas
14 acetaldehyde, benzene, 2-butanone, 2-butoxyethanol,
15 formaldehyde, phenol, tetrachloroethylene, toluene and
16 xylenes were the non-field-related chemicals that were
17 included in this calculation. You have already seen
18 this during this morning's presentation.

19 So those were the chemicals that had
20 sources other than crumb rubber in-fill or the
21 synthetic turf field as the source.

22 For the field-related chemicals,
23 without toxicity criteria for acute exposures, we have
24 made comparisons of maximal air concentrations with
25 available subchronic health guidance values from other

1 sources. So there are three of them available from
2 U.S. EPA's PPRTV database and the ATSDR MRL. And in
3 all those cases, those concentrations were much lower
4 than the subchronic health guidance values as well.

5 Similarly, for one-day dermal and
6 ingestion exposures there were no one-day toxicity
7 criteria available, but there were no exceedances
8 based on comparisons with the subchronic health
9 guidance values for the dermal or oral route as well.

10 Now, after developmental and
11 reproductive toxicants, here on average, the hazard
12 index was less than 1 ranging from .01 to .58 for all
13 the receptor groups and age groups. That was based on
14 the 24 field-related chemicals. So it's adding the
15 hazard quotient for all the chemicals via all of the
16 routes yielding a hazard index which was well below 1
17 on average.

18 Looking at the individual values, the
19 maximal value we could find were -- there were cases
20 where it exceeded 1. That would be for athletes of 11
21 to 70 years old, when it ranged from 1.2 to 1.8.

22 The chemical driver in this case was
23 benzo[a]pyrene. Benzo[a]pyrene was one for which the
24 toxicity criteria was calculated using an uncertainty
25 factor of 3,000. This means the reference guidance

1 value for humans is 3,000 times less than the lowest
2 concentration at which adverse health effects were
3 observed in animals, just to put it in perspective in
4 terms of the data gaps for benzo[a]pyrene.

5 And it's also relevant to note that the
6 parameters and assumptions used for the exposure
7 assessment -- well, I'll come to that in a moment.

8 So here, this picture captures the
9 maximal hazard index. You know, the hazard index was
10 calculated for the 24 chemicals for each one of the
11 fields. I'm picking out the maximal numbers. One can
12 see that it ranged from 1.2 to 1.8 in athletes of 16
13 to 30 years old, whereas in all other cases it was
14 below 1.

15 All the average values were within 1,
16 the benchmark of 1, whereas the maximum individual
17 value that we had seen were in the range of that 1.2
18 to 1.8 in those three cases.

19 So this hazard indices, once again,
20 were calculated based on the 24 chemicals accounting
21 for inhalation exposure during about three hours
22 during each game and practice, ingestion of about
23 300 milligrams crumb rubber per event and dermal
24 exposure based on skin load of about 180 milligram
25 crumb rubber per event.

1 And the odds of all of these occurring
2 in these age groups were actually low. They're
3 indicating a low probability of observing a hazard of
4 1.2 to 1.8, which is well within the uncertainty
5 factor of 3,000 used for benzo[a]pyrene which is the
6 driver in this case.

7 Now on to chronic exposure to sensory
8 irritants. Here the hazard indices were well below 1,
9 based on either the field-related chemical, which was
10 styrene, and the two non-field-related sensory
11 toxicants which were formaldehyde and acetaldehyde.

12 So each individual field hazard was
13 assessed using the field's average concentration for
14 this. So each field, the sensory irritation hazard
15 indices were calculated based on values from each
16 field.

17 So these were below .01 for the
18 on-field exposures for all exposure groups, and the
19 off-field numbers were always below .01, and not shown
20 on this table.

21 So overall, only the field-related
22 chemical styrene evaluated as a sensory irritant did
23 not present a hazard on-field or off-field. However,
24 you do see the contributions from the
25 non-field-related chemicals in the second column as

1 the maximum numbers did exceed 1 in the case of
2 formaldehyde and acetaldehyde as the non-field-related
3 contributors.

4 Now on to the non-cancer hazards,
5 chronic effects.

6 The average across fields was below 1.
7 The hazard index was below 1, ranging from .03 to .5
8 for all receptors. This was based on 99 field-related
9 chemicals and 21 non-field-related chemicals.

10 Now, to the second scenario of looking
11 into the individual field values, the maximal hazard
12 index was below 1 for all receptors except in two
13 cases, or in 31 groups except for two cases. One is
14 the athletes of 16 to 30-year-old where the hazard
15 index was equal to 1. And the second was on-field
16 spectators, zero to two years old where the hazard
17 index was 1.2.

18 So the on-field spectators are children
19 going off of the spectator stands onto the play field,
20 sitting, playing, and eating in the field. Those are
21 the on-field infant spectators that are being referred
22 to here where there's a small exceedance compared to a
23 benchmark of 1.

24 In this case the driver was lead and
25 ingestion route was the main contributor as well.

1 Pictorially I'm just capturing those
2 chronic hazard indices for the field-related general
3 chemicals. In this case you see the values for
4 athletes and then coaches, referees, and finally the
5 on-field spectators. Where you see the infant group
6 all the way in the top, that's the blue circle that
7 you see just above the dotted line. That's the one
8 that I'm referring to.

9 So these are the maximum hazard indices
10 calculated. And this value for the on-field infant
11 spectators, as I mentioned, is driven by ingestion and
12 by lead basically.

13 And in all other cases it's well-below
14 the reference value of 1. And also very often you see
15 all of them congested together below. That's why you
16 don't see all of the colors in there. So they're all
17 kind of together in there at the bottom, on the bottom
18 and the lower levels.

19 So this pictorial shows the ingestion
20 contribution is the main driver of the hazard index in
21 the on-field spectators or the infants. Where you see
22 the distribution of hazard index that goes from below
23 .2 to .4 to .6 to .8 to 1, and above 1, that's the
24 last case where I referred to with the circle in the
25 previous picture. In all cases it's the ingestion

1 that's the driver.

2 Whereas in the athletes of 16 to 30
3 years, it's mainly inhalation that contributed, which
4 was a driver of the hazard index.

5 Finally, the cancer risk. Here, the
6 mean cancer risk for combined exposure to field and
7 non-field-related carcinogens was greater than one in
8 a million for all receptors. And it was primarily
9 driven by the non-field-related carcinogens. The
10 non-field-related carcinogens accounted for more than
11 90 percent of the cancer values. That's the mean.

12 And the mean cancer risk levels
13 associated with field-related chemicals in all the
14 receptor groups, athletes, coaches, referees, and
15 spectators, were below one in a million except for one
16 group, the on-field infant spectators for which it was
17 slightly above the benchmark which was at 1.1 in a
18 million.

19 So once again, the on-field spectators
20 are the ones who go from the bleachers onto the field,
21 sitting and playing and consuming.

22 Based on the individual field cancer
23 risk levels, the maximum, when you look at the
24 individual field cancer risk levels, the maximal value
25 exceeded -- was always greater than one in a million

1 in two groups. One, the on-field infant spectators
2 where the range was .3 to 2.7 in a million. So the
3 one in a million is encompassed in there or the slight
4 exceedance is encompassed in there. The same with the
5 athlete groups as well as I'll show in the next two
6 slides.

7 So here we see the range of the values,
8 the cancer risk levels in each one of the age groups
9 of the on-field spectators and off-field spectators
10 based on multi-route considerations and multi-chemical
11 considerations.

12 And I draw your attention to the first
13 bar graph that exceeds the dotted line. So here it
14 goes from .3 to 2.7. So those are the -- that's the
15 range of the values calculated for the on-field
16 spectators.

17 One of the considerations is the
18 assumptions and parameters of going into the exposure
19 assessment and categorizing the risk for the on-field
20 spectators, the infants that get on the field.

21 They're exposed to about three hours
22 per event. 161 events per year for the infants. And
23 these are numbers from the supplementary materials.
24 I'm just repeating them. Oral exposure to
25 153 milligrams of crumb rubber per event, per game,

1 that they'll go to, 153 milligrams during those 161
2 events.

3 And dermal exposure or a skin load of
4 about 50 milligrams of crumb rubber per event during
5 the 161 events on the children -- in the infants that
6 get onto the field.

7 So considering all of these worst-case
8 scenarios and parameters used, we conclude that these
9 do not raise to significant level of concern.

10 Also, there's the additional
11 application of an age sensitivity factor of 10 to
12 calculate the cancer risk for infants of zero to
13 two-year-old, calculations as Jocelyn had shown this
14 morning.

15 So considering that we would say,
16 considering that this risk level is of possible
17 concern, particularly because of the hand-to-mouth
18 activity and the ingestion of crumb rubber in-fill in
19 the turf field by the infants.

20 Similarly, these are the ones for the
21 athletes. And I draw your attention to the two bars
22 where they're slightly above the benchmark of 1. The
23 whole range goes from well below 1 to a little above
24 1.

25 So having presented these numbers for

1 the cancer and non-cancer risks, let me move on to
2 address the considerations of variability, uncertainty
3 and limitations of the study before concluding.

4 In terms of the variability was
5 addressed to the extent by considering -- to the
6 extent of considering the various subgroups, the 33
7 subgroups, seven athletes, four for coaches, four
8 referees, nine on-field spectators, and nine off-field
9 spectators.

10 Additional aspects that deserve mention
11 are the sample heterogeneity. Given that crumb rubber
12 in-fill is produced from a variety of sources, in
13 terms of automobile waste tires, different tire types,
14 models, brands, production years, age in traffic and
15 so forth, but we have considered this heterogeneity in
16 the analysis of crumb rubber variation within the
17 samples and within the fields in their analysis.

18 Then for time-activity and exposure
19 parameters, as Dr. Claude mentioned this morning, we
20 conducted a California specific time-activity survey
21 to derive athlete-specific physical parameters and
22 also soccer-specific exposure parameters to better
23 characterize exposures and better characterize the
24 variability there.

25 In terms of the athlete player

1 position, even though we did not consider the
2 variability among the various athlete positions, given
3 the concern for the high exposure of chemicals for
4 goalies due to frequent diving activity and
5 potentially ingestion of crumb rubber, the study used
6 a goalie-specific scenario to compute the risk values
7 for all the athlete exposure scenarios.

8 In terms of uncertainty, any risk
9 characterization should recognize the sources and
10 impact of uncertainty. In this regard I would bring
11 about three aspects here. One on the subject of
12 chemical characterization and source designations, a
13 critical concept and aspect of designating the
14 chemicals as being field-related or non-field-related.

15 You saw the methodologies that were
16 used to characterize them. But in this study,
17 however, we conducted and presented calculations for
18 both, both field-related and non-field-related.
19 That's the way to address that concern for
20 uncertainty.

21 And then in terms of exposure
22 assessment, the parameters for coaches, referees, and
23 spectators were based on the time-activity survey
24 collected in athletes. And we used the highest mean
25 values from athletes for all the other groups to be

1 health-protective.

2 This task survey did not provide any
3 information about the amount of crumb rubber ingested
4 during the course of soccer activities, so we either
5 used literature data to estimate the direct ingestion
6 amount and it is likely to be a conservative
7 overestimate in the exposure on risk assessments.

8 On the topic of dose-response
9 assessment, I think we have already touched upon that
10 for noncarcinogens for chemicals without established
11 values, for which we developed new values applying
12 appropriate factors. And for the DARTs considered as
13 single-exposure event as being sufficient to cause
14 developmental and reproductive health outcomes, so we
15 did not use the overall average or anything but we
16 considered just a single-day or single-event
17 concentrations there.

18 Also, the use of the toxic equivalency
19 factor or potency equivalency factor of BaP or
20 benzo[a]pyrene and other PAHs is certainly a source of
21 uncertainty to be kept in mind in this context.

22 That's what was used to derive the
23 toxicity criteria for benzanthrane,
24 benzo[fluoranthene, chrysene, indeno pyrene, and
25 cyclopenta pyrene.

1 In terms of the study boundaries and
2 limitations, I just want to emphasize that we
3 evaluated the crumb rubber in-fill, but not the
4 backing materials or the grass blade components. And
5 no analysis for metals and fine particulate matter
6 from the air was conducted due to logistics and
7 logical constraints.

8 However, we measured the bioaccessible
9 concentrations of metals and crumb rubber for
10 evaluating ingestion exposure, as you saw in this
11 morning's presentation.

12 Finally, the study conducted
13 assessments for each receptor category and not for
14 various scenarios of combined receptor roles, which
15 can be done given the -- like a person acting multiple
16 roles during a single event or on different roles
17 during a year or more than a year and so forth.

18 But a time-adjusted hazard index or a
19 combination of cancer risks can be calculated based on
20 what we have presented.

21 But given the sufficient number of
22 hours, exposure hours used per year in these
23 calculations in any given role, and given essentially
24 that all the risk values were below the -- or above
25 the acceptable benchmark, below the acceptable

1 benchmarks, the likelihood of significant health risks
2 arising from combined those scenarios is considered to
3 be low, and we indicate that in the report.

4 So to conclude, overall, based on the
5 available data, the methods used and the limits
6 described in this report, overall the study found no
7 significant health risks in terms of acute toxicity,
8 developmental and reproductive toxicity, sensory
9 irritation, general chronic toxicity and cancer risk
10 to players, coaches, referees, and spectators from
11 on-field or off-field exposures to field-related
12 chemicals in crumb rubber in-fill from synthetic
13 fields.

14 Based on the maximal values from the
15 individual field data, there were a few instances
16 associated with turf-field-related chemicals that are
17 of low odds of actually occurring that one would
18 consider as of low probability and of low concern.
19 And that would be three cases. DART in athletes aged
20 11 to 70, chronic toxicity in the on-field infant
21 spectators, and the excess cancer risk in on-field
22 infant spectators and athletes of 16 to 30 years.

23 And much of these calculations for the
24 on-field infant spectators would indicate that the
25 necessity or the emphasis to reduce the hand-to-mouth

1 activity and limiting the time on the turf field for
2 the infant spectators who get off the spectator stand
3 and play on the turf fields.

4 I would stop at that and address any
5 questions or clarifications either to me or to
6 Jocelyn.

7 DR. BALMES: Thank you, Dr. Krishnan.

8 So the floor is open for the panel.
9 The dais is open for the panel to comment.

10 Sandy?

11 DR. ECKEL: This is Sandy Eckel. I have a
12 clarification question about the elevated DART for
13 athletes aged 11 to 70. I think it was on Slide 15.
14 I was just trying to understand the heterogeneity on
15 Slide 15, the hazard index estimates for athletes.
16 And I noticed that -- I at least see three dots that
17 are above the 1, and one dot that's very close to 0,
18 and I was trying to understand what was the driver of
19 that heterogeneity in those estimates there?

20 Was it different breathing rates?
21 Because it seems like it's by age, these differences,
22 and I was trying to understand why there was such a
23 difference there.

24 DR. KRISHNAN: Do you want to start or do
25 you want me to start?

1 DR. CLAUDE: I can start. So a lot of those
2 groups that are -- so a lot of those groups that are
3 down there toward right on the X axis, so they look
4 like they're 0, but those are the values that are like
5 the less than .01. And for most of those groups are
6 the younger age groups, so their time on the field is
7 a lot less than those older age groups. And they may
8 not have as many practices. They might have one
9 practice per week, whereas some of the other groups
10 might have two or three practices and games.

11 So a lot of that is due to differences
12 in exposure frequency. You do get some of those
13 breathing rates in there because the younger kids
14 aren't going to have as high breathing rates and have
15 such high exertion levels as some of the higher, more
16 competitive athletes.

17 So a lot of what's driving that is
18 those types of differences and physiological
19 differences in breathing rate and then duration and
20 exposure frequencies.

21 DR. KRISHNAN: Thank you. These are maximal
22 values for any of the age groups. So if you picked
23 the one on the top, the 1.8, that's for the 16 to 30
24 years. So the average for that across the 35 fields
25 was .6. So from that distribution we just picked only

1 the maximal value to show here in each one of those
2 cases.

3 But all of the averages for all of the
4 age groups were well-below the reference index of 1.

5 DR. ECKEL: Thank you very much. I had one
6 other big-picture comment. My expertise is more in
7 epidemiology studies, and I noticed that it seems like
8 these risk assessments are all sort of chemical by
9 chemical. And it's very popular in epidemiological
10 studies these days of multiple exposures to look at
11 interactions between different exposures, and it seems
12 like this might not be possible in this kind of study
13 but it might be worth it to mention the limitations
14 that the elements were considered one by one rather
15 than potential joint effects of multiple chemical
16 exposures, if I understand correctly.

17 DR. KRISHNAN: We'll certainly take that
18 into account and mention that. One way this study has
19 addressed that is by adding the hazard or the risk
20 associated with each one of the chemicals. So it's
21 the additivity that's used in this particular case.
22 So that's the hazard index.

23 DR. BALMES: While we're on this Slide 15, I
24 actually like the way that age strata are shown here.
25 But in the conclusions, when you say DART for athletes

1 age 11 to 70, it's the 70 -- I'm 75, so I appreciate
2 you're worried about developmental reproductive
3 toxicity for old people, but it doesn't -- I don't
4 know for public, it just seems like too wide of a
5 range in terms of summarizing things.

6 Again, I don't have any problem the way
7 it's broken down on Slide 15 because you have each age
8 group, but it just sounds weird to have DART for
9 athletes aged 11 to 70. I know people are still
10 playing soccer at 70, but I don't think there's too
11 much concern about developmental reproductive toxicity
12 at that age. It just doesn't quite meet the test, to
13 me.

14 DR. BENNETT: And I had a quick question
15 about the slide too because I wasn't clear. I thought
16 you didn't calculate the DART for 6 to 11, but you did
17 calculate the DART for 6 to 11 and it was basically
18 zero? Isn't that one that's on the line, the 6 to 11?
19 What's the value of the 6 to 11 years? Isn't that
20 that dot that's basically athletes at zero? That
21 brown dot at zero?

22 DR. CLAUDE: The 6 to 11 for field-related
23 DARTs is less than .01. So it was very low. So it's
24 the 11 to 16 through 50 to 70 that encompassed those
25 three dots. And there's a little bit of overlap

1 between them for those five age groups.

2 DR. BENNETT: So the 6- to 11-year-olds had
3 basically like a hundred times less exposure?

4 DR. CLAUDE: Very low, yeah. Also one thing
5 to keep in mind, because these are individual field
6 calculations, so -- there's 35 fields, so not every
7 DART was tested and detected. Well, not tested, it
8 was not detected on each field. So some fields may
9 have less detection than others, so that also will
10 take into account in the average. You might have one
11 field that's only got three versus one that's got all
12 of them.

13 So some of that difference you're
14 seeing in the levels of the hazard might be due kind
15 of to that too just because some fields having fewer
16 chemicals detected on it as well.

17 DR. BENNETT: It's just surprising that the
18 exposure is 100 times greater for 11- to 16-year-olds
19 than 6- to 11-year-olds.

20 DR. BALMES: Did you have --

21 DR. BENNETT: I did have one other question.
22 Or do you want to go and then I can ask mine?

23 DR. BALMES: No, you have the floor. Go
24 ahead.

25 DR. BENNETT: I noticed on the zero- to

1 two-year-olds, you had lead exposure rates, but I know
2 on -- again, sort of from epidemiology and whatnot,
3 really seeing no level of lead exposure is safe. So I
4 felt like, yeah, you guys probably are taking a
5 published value for the levels of lead exposure that
6 were considered safe, but I feel like it would be
7 important to note that at this point we kind of
8 consider no exposure to lead as being acceptable.

9 I mean, I do agree that you've got, I
10 think, the 153 grams of field ingested is a lot.
11 Again, it would probably be good to convert that into
12 some sort of measurement we can understand. But even
13 so, I feel like you needed to say a little bit more
14 about any lead exposure being problematic.

15 It sounds like John might concur with
16 that thought.

17 DR. BALMES: Well, yes, I definitely agree
18 that no level of lead exposure is currently advised
19 for child development, cognitive development.

20 You may, Dr. Kyle.

21 DR. KYLE: I support your comment. If
22 you're adding lead to children's environment, that's a
23 health concern irregardless of these numbers.

24 But I was wondering what you used for
25 the hazard index for lead because we don't have a

1 hazard index for lead number because there's no safe
2 level of exposure. So it talks here as if a hazard
3 index calculation was used, and I was just wondering
4 how you did that. Because I don't think OEHHA even
5 has one, do they?

6 And if this is a look-up, you can
7 answer that later. I don't want to draw the meeting
8 to a close. This is a small question.

9 Do you understand my question?

10 DR. CLAUDE: Yes.

11 DR. KYLE: Thank you.

12 DR. BALMES: While they're working on that,
13 do you have any other comments, Dr. Kyle?

14 DR. KYLE: Yeah.

15 DR. BALMES: You may.

16 DR. KYLE: And this may be more a matter of
17 writing than the science, but the distinction between
18 the so-called field-related chemicals and the
19 so-called non-field-related chemicals is soft under
20 these methods because, as the report says, this is
21 what the report says somewhere else, that we're not
22 really sure whether what is measured on-field and
23 off-field are real differences between what comes from
24 the crumb rubber and what doesn't because of the
25 proximity of the areas, for one reason, the space

1 between what was measured on-field and off-field
2 wasn't necessarily that far away or free of wind
3 interference.

4 And also there's another reason. I'm
5 blanking on it right now.

6 So I guess I feel like this makes too
7 much of that distinction. The way that some of this
8 is done, it really makes it look like we know that
9 these are the ones that are, quote, on-field which
10 means from the crumb rubber, and these other ones
11 which are off-field, are therefore not from the crumb
12 rubber. And we really -- we're not as sure about that
13 as this sounds.

14 I remember the second reason now. The
15 second reason was because the crumb rubber also may
16 have blown around. It's not necessarily strictly
17 limited to the field or any specific proximity. It
18 gets tracked in and out so that could be another
19 reason.

20 So I'm not saying we shouldn't do this,
21 but I feel like that softness of that distinction
22 doesn't get drawn into the discussion about -- I don't
23 know if that's exactly uncertainty. It's a
24 limitation, I guess, or something like that. I'm not
25 sure what category to put that in, but I feel like

1 that was missing from this discussion.

2 And it's really quite important
3 whether -- and also probably unknowable without
4 spending a lot more money. Which is kind of part of
5 the, well, what did we learn from this? What kind of
6 studies can we do to figure out actually in the
7 environment to make these small differentiation
8 between what's actually on a field.

9 In my mind, then, does that mean that
10 when is this kind of research valuable or when should
11 we look at what the ingredients are and make decisions
12 and finding space on that? That's a policy question,
13 not a science question.

14 But again, I'm thinking about what did
15 we learn from this. And one thing we learned is, it's
16 hard to be clear in the environment about what's
17 ambient and what comes from where in a dispositive
18 way.

19 And I still commend your efforts. We
20 learned a lot from this. And so I'm not being
21 critical of doing it, but just that maybe the
22 conclusion is too -- the distinction is too strong.
23 As we think about what we can learn and where in this
24 sort of chain of use, et cetera, it's worthwhile to do
25 studies and how we can make decisions. It's on my

1 mind a little bit. When is this helpful?

2 It's related to the question that
3 someone raised about, well, are these facts still
4 true? When you have something that you know is an
5 unmanaged mix that's uncharacterized, it may change in
6 the form of the waste rubber, in ways that no one will
7 flag for you. So how long are findings like this good
8 for and what are the implications for that and should
9 we spend more time measuring upstream to see whether
10 the upcoming stream is changing.

11 So those are some thoughts on my mind.

12 DR. BALMES: Can I butt in for a second?

13 DR. KYLE: Yes, please, because I need to
14 look up my next point.

15 DR. BALMES: Because to be fair -- I don't
16 have -- I don't disagree with you that maybe the
17 softness of the field versus non-field exposures could
18 be highlighted, but Section 7.5.1 actually does talk
19 about the issue of field versus non-field being
20 difficult to characterize.

21 So it may not have been spelled out as
22 clearly as I think you'd like it to be. And I agree
23 with that. But it is in there.

24 DR. KYLE: And I said it was in there.

25 DR. BALMES: I'm sorry. I missed that.

1 DR. KYLE: I said I know you have it in
2 here. And that's where I learned about it. I'm
3 quoting back to them really their own text. That's
4 okay. Maybe it was confusing in my presentation. But
5 I think in the way this data is structured, it doesn't
6 reflect that. It makes it sound more like a real --

7 DR. BALMES: And I agree with you.

8 DR. KYLE: Thank you. So I will yield the
9 mic for now.

10 DR. BALMES: I wanted to see if Dr. McKone,
11 who is online, has any thoughts.

12 DR. MCKONE: I just have a couple of points.
13 One is a kind of a technical question, I guess. So
14 just to be clear, each of the hazard quotients that
15 were used to aggregate a hazard index, I think you
16 said the toxicity criteria came from either OEHHA or
17 probably a REL or EPA, right? Was the choice based on
18 which was most restrictive, would lead to the highest
19 hazard quotient? Or were they all OEHHA RELs?

20 I can't hear. I'm not hearing the
21 answer. Sorry. I'm not getting ...

22 DR. KRISHNAN: Okay.

23 DR. MCKONE: Now I can hear you.

24 DR. KRISHNAN: There's a schematic in the
25 Chapter 4 that's presented as to how the values were

1 chosen. It's the most health-protective value with
2 the way it has been the priority.

3 DR. MCKONE: That's what I thought. So it
4 wouldn't be preferenced to REL. It would be
5 preferenced to the most health-restrictive, right?
6 Which makes sense.

7 So the question I have, though, is, it
8 might be useful to look at the date that the toxicity
9 criteria was established. For example, I've done this
10 where we look at OEHHA REL, or we look at EPA,
11 reference dose, and sometimes the one that is most
12 restrictive is also the oldest, and may even be 20
13 years old.

14 And so one of the things that -- not
15 that it would really change things, but it might be
16 interesting to see if this is the most restrictive
17 value is based on more recent data or older data. And
18 I don't think -- it's not that you should change it,
19 but it's interesting to see, in terms of understanding
20 some of the uncertainties and other issues, it would
21 just be useful to have a date associated with the
22 toxicity criteria that was used in the hazard
23 quotient.

24 I don't know if that's possible, but
25 it's kind of useful because for an outsider to look at

1 it, "Oh, it was really restrictive," but it was like
2 1970 study and nobody has updated it.

3 Another comment, while people were
4 talking, I looked up OEHHA's website. There is no REL
5 for lead that I could find, but there is a Prop 65
6 MADL, maximum allowed daily --

7 DR. KRISHNAN: Which one are you referring
8 to?

9 DR. MCKONE: For lead. There's no safe dose
10 for lead, but Prop 65 actually has MADL.

11 DR. KRISHNAN: So for DART --

12 DR. MCKONE: Well, it's reproductive under
13 Prop 65. So that might have been what you were using
14 in the toxicity criteria.

15 DR. EDWARDS: It's our maximum allowable
16 daily level.

17 DR. KRISHNAN: That's the MADL. That's what
18 is indicated on page E8 of the index.

19 DR. MCKONE: You were saying there's no
20 standard. Well, there is no REL. And I'm not sure
21 about EPA, but Prop 65 does have a number, just for
22 information.

23 This is more of a broader comment and
24 it has to do -- and I think in your summary and
25 discussion I really welcome the fact that it kind of

1 gets into issues of confidence and how safe people
2 are. I get a little nervous when somebody says,
3 "Well, one in a million is de minimus, and we're all
4 safe." But if it goes above that -- I mean, I always
5 get a little uncomfortable when somebody says, "Well,
6 it doesn't meet the standard because it's two in a
7 million."

8 And I always ask, "Well, if somebody
9 made a bet with me and said you have a one in a
10 million chance of winning a million dollars, how much
11 would you pay?"

12 And then they say, "What if it goes to
13 2.7 in a million? Would you double your bet?"

14 No, not really, right? It's two in a
15 million. In the grand scheme of things, 2.7 in a
16 million, in the reality of likelihood and things is
17 not that -- so I think instead of focusing as much on
18 oh, the hazard index went slightly above 1 or the risk
19 was a little over one in a million, it might be -- I
20 mean, not to focus so much on that as the fact -- what
21 I see in this is a study that says, yes, there's
22 health issues but we have high confidence based on
23 everything we did, looking at exposures, looking at
24 toxicity factors. We're down in numbers that any
25 reasonable person would be comfortable with if you

1 trust the toxicity and the exposure assessment.

2 And, again, it's just how we discuss
3 our confidence or communicate the fact that the study
4 really points to the fact that the community is quite
5 safe. There's no reason to run out and put a ban on
6 all soccer playing on synthetic turf.

7 And, again, I mean, that's just a
8 little bit about crafting the language for
9 communities. So I'm not quite sure how to do that,
10 and others may have an idea, but I do get a little
11 nervous when somebody says, "Well, if one in a million
12 is acceptable, two in a million is over the limit so
13 it's not good."

14 That's the end of my comments.

15 DR. BALMES: Thank you, Dr. McKone.

16 DR. CLAUDE: Could I chip in real quick?

17 DR. BALMES: Go ahead, Jocelyn.

18 DR. CLAUDE: I just want to address the
19 toxicity criteria. So we did choose values that were
20 the most health-protective, but we also did put more
21 weight on more recent assessments. So in the case
22 where we did have some of those chemicals that had
23 criteria from the '80s, if there was a more recent
24 assessment if the value maybe wasn't as low, we did
25 still look at all the data and quite possibly choose

1 maybe a value that was higher because based on more
2 recent data of higher quality. So we did take that
3 into account.

4 In the appendix, I believe it's
5 Appendix E, all of the studies and all of the years
6 for all the criteria that are used are presented, so
7 that data should be in the appendix.

8 DR. MCKONE: That's wonderful.

9 DR. KRISHNAN: The workflow is captured in
10 Figure 4-1 in the main report. That shows not only
11 the most health-protective but also the most recent
12 value was sought for and used. Thank you.

13 DR. BALMES: Thank you for that
14 clarification, Jocelyn.

15 Ed, do you have any comments?

16 MR. AVOL: I'm still struggling a little bit
17 with, again, it's more in the sense of interpretation
18 and how this is going to be understood by the public.
19 So I'm looking at, I guess, Slide 15, the one-day
20 hazard index and that plot that showed the athletes
21 over a range and then the conclusion, Slide 27, I
22 guess, that sort of said the DART for the athletes age
23 11 to 70.

24 One comment just as a side issue for
25 those of us who are color-challenged when looking at a

1 chart, it would have helped me to somehow identify
2 what those four dots are under athletes to know who
3 we're talking about. So could you actually just tell
4 me once again so I can make sure that the comment I'm
5 about to make, makes sense? The top one.

6 DR. EDWARDS: The top one is 16 to 30 years.
7 The gray one is 50 to 70. And the third one down --

8 MR. AVOL: Which one is the gray one?

9 DR. EDWARDS: A little bit below 1.4. And
10 then the third one at 1.2 is 40 to 50.

11 DR. BALMES: And the one on the bottom is 6
12 to 11.

13 DR. KRISHNAN: Actually, there's a table in
14 Chapter 6, page 767. That's the table that has these
15 numbers. And we only tried to pull out just the
16 maximum numbers to show in this.

17 MR. AVOL: I appreciate that the report is
18 much more detailed. The numbers are there. But if
19 you're going to use a chart like this to sort of
20 summarize it so people can look at it, I think you
21 want to be sure that you get the right message across
22 that you plan to apply.

23 So then the question -- not to dredge
24 it up again, this 6 to 11 being almost at zero on the
25 sheet here, which was a little surprising given that

1 children sort of ages 7 to 11, are a large, if not
2 substantial, component of the soccer population, the
3 kids soccer population. They get per kilogram mass,
4 they get to pretty high ventilation rates when they're
5 running around, and they're close to the field, so
6 they're potentially in the right exposure range.

7 And based on what your previous comment
8 was in terms of, I guess, even a cancer risk, you said
9 it was from 10 to 1, you assigned some numbers with
10 the highest number being in the lowest ages. So it
11 seems like they would be in the higher category.

12 So it seems like -- I guess my
13 expectation is they're all pushing in the direction
14 that they would have been sort of highest in range and
15 it came out sort of just barely being measurable in
16 the chart.

17 And so I don't dispute the data, the
18 actual data. If that's what it says, that's what it
19 says. It's just a question of the interpretation, how
20 you explain that, because it seems like it's -- I
21 wouldn't say inconsistent, but it seems like it's
22 counterintuitive to what all the built-in
23 considerations were leading up to that calculation.

24 So it seems like it would be helpful to
25 have some sort of sentence or two that explains why

1 even children, who we are potentially most concerned
2 about being exposed on these fields, in this
3 calculations turn out to be those we need to be
4 concerned least about.

5 DR. BALMES: Dr. Bennett just pointed out --
6 I didn't notice this before -- the 11 to 16 is not
7 even showing up.

8 DR. BENNETT: And what's also curious is
9 when you read the table in the text, they don't list
10 the value for 6 to 10. They start at 11 to 16 in the
11 table. And now I've put my thing back, I don't
12 remember what table it was. So it didn't even show
13 that they calculated a 6 to 10.

14 DR. BALMES: But on Slide 15 there's no --

15 DR. BENNETT: It's probably under another
16 value, right?

17 DR. BALMES: It must be under the
18 athletes -- I mean, it must be under 6 to 11.

19 DR. CLAUDE: In the tables for the
20 individual exposure routes, if the value was below .1,
21 it wasn't put in the table. So those are the values
22 only that are above .1. And so for the field-related
23 DARTs too, inhalation was the primary driver of the
24 pathway, and those were very low for those age groups.
25 So that's why they're not in the inhalation table but

1 they are in the tables for the oral exposures.

2 DR. BENNETT: But they're chronic
3 inhalation -- if you look at Table 6-10, the 6- to
4 11-year-olds have very similar values to all of the
5 other age groups for inhalation.

6 DR. BALMES: The other thing, as Dr. Avol
7 just said, or Mr. Avol just said, the ventilation
8 rates of young kids relative to their body mass is
9 high.

10 DR. CLAUDE: Some uncertainty in that
11 aspect. So in the survey we did, we had a very small
12 number of participants aged two to six. So for that
13 age group, I think we had three six-year-olds who
14 filled out information.

15 So we did ask them information about
16 exertion levels. So their reported exertion levels
17 were lower than the athletes. They do say they're
18 running at high intensity for most of the practice,
19 whereas the borrowed six-year-old data, they didn't
20 report having --

21 (No audio.)

22 DR. BENNETT: And then throwing in that the
23 kids, also probably one of referees to really get at
24 kind of modeling a kid that soccer is their youth
25 sport and they do it all, you'd have a risk value if

1 you added those up. It is slightly over 1 times 10
2 minus 6. It would probably be like 5 or 6.

3 Just to do that and show, look, even if
4 we're really conservative and you were dragged to the
5 soccer field as an infant to watch your older sibling
6 and you then you played and then you kept playing and
7 then you were a referee, you know, you pointed out
8 that you can add these things up, but you just might
9 want to do a couple scenarios and say here's what it
10 is. It's still going to be relatively low in the
11 grand scheme of things, but then it just feels like
12 you've taken this extra step to being conservative on
13 the exposure side and it's still showing that the
14 risks are above de minimus, but they're still not that
15 high.

16 DR. BALMES: Ed?

17 MR. AVOL: So I have a larger question.
18 This morning we were the beneficiaries of an excellent
19 report on the characterization, this afternoon on one
20 of the toxicity and the risk characterization parts.
21 And so now I'm not sure if there's going to be -- I
22 don't think there's going to be a third presentation
23 on sort of the synthesis of what the whole total
24 picture is in the report.

25 So I guess I'm asking, put it all

1 together, and then you think about what we have. So
2 we have this dataset from 2015, and in the appendices
3 you did go back and you said, well, there have been a
4 number of studies since then and some additional newer
5 studies are cited and what the implications or the
6 reported results of those studies are, and how that
7 fits with your findings and how that's assimilated.

8 In some ways it's sort of like the
9 report is sort of a picture in time. And then in the
10 appendices there's sort of additional updates on where
11 we are. But in the discussion and summary conclusion
12 in the main body of the report, it seems like it would
13 be useful for the public, for the reader, for people
14 to be able to say, "Don't just think about this as
15 this is something we did in the past. We've looked at
16 what we did, which was exhaustively detailed. We've
17 looked at what's been done since then to see if there
18 had been any meaningful changes in it, and here is
19 where we sit now," which I think helps to frame the
20 study in a more contextually relevant and important
21 consideration than saying, "Well, all this was done
22 ten years ago. We better go look back and see where
23 we are now."

24 DR. BALMES: Any other thoughts from the
25 panel?

1 Dr. Kyle.

2 DR. KYLE: On the issue of the chemicals
3 that fell out because of lack of tox values like the
4 issue I raised in the first session about let's
5 identify where things fell out and building on your
6 comment, I think it would be good to identify that and
7 where it fell out and what.

8 I agree with all the comments that
9 everyone else has made here too. I guess the last
10 thing I wanted to say is that I agree our hair doesn't
11 need to be on fire about this issue. It's not an
12 emergency. I wouldn't evacuate playgrounds,
13 et cetera.

14 But if I were advising my friend on the
15 school board about this, I would say I would try not
16 to use this stuff because it has known toxic
17 chemicals. It's lead. We don't really know
18 everything about it and you don't necessarily have to.
19 The only context for this is not people coming in and
20 banning it. It's also an information thing about, are
21 we sure it's safe? And I would say I agree it's not a
22 huge problem or a crisis, but am I sure it's safe?
23 No.

24 So I think there's a range of questions
25 people ask. And there might be some way to

1 acknowledge that. And maybe there isn't because it's
2 OEHHA, and you're the government, and you're speaking
3 in kind of a government way. And someone might sue
4 you.

5 But I do think that the standard you're
6 setting here in a way is too high and that a lot of
7 people, they want to avoid things that might be
8 hazardous. They want to make sure their kids are
9 using and have safe food and products and stuff. And
10 so if you look at this -- and maybe no one wants to
11 answer that -- but would you say you're sure it's
12 safe? I would say I would probably pick something
13 else if I could, speaking solely for myself, but based
14 with having reviewed this. Thank you.

15 DR. BALMES: Dr. McKone, any other thoughts?

16 DR. MCKONE: No. I mean, I don't think a
17 government report can really say what Dr. Kyle did,
18 but I think that's kind of the reality of it, is this
19 is not something -- after years of study, we don't see
20 a smoking gun or we don't see anything where it's time
21 to call out or for the state to take regulatory action
22 or ban it.

23 But it is -- I mean, there's an
24 interesting question of when people say, "Well, what
25 would you do?" I'm kind of with Dr. Kyle. It's like,

1 well, if you could avoid it, who knows. If somebody
2 said, "Are we at risk?" I would say, "No, I don't
3 think you are."

4 But if you asked me, I would look
5 through the data and think about it. There's a lot of
6 things that are that way but I don't think the state
7 can write that in a report, but we can say that.

8 DR. BALMES: And I'm glad my kid mostly
9 played on grass.

10 Dr. Eckel, any other comments?

11 Well, I guess I would make one comment
12 and maybe let our OEHHA scientists respond. This
13 report took ten years. I think it's really an
14 excellent report, as we've all said. And I realize
15 there was a pandemic in there, and also a change in
16 leadership at the agency and such.

17 But anyways, a complicated study design
18 with lots of moving parts. But it really did take a
19 long time. And now we're saying, is this still
20 relevant, this 2015 snapshot? I'm not being critical
21 of any individual here, but sort of institutionally,
22 CARB, my agency, is part of CalEPA as well, but it
23 just seems like this took too long.

24 So in the future when OEHHA has a big
25 task like this -- there's no question it was big --

1 we'll probably need to have it better resourced. I
2 guess I'll put it that way. Again, not being critical
3 of anyone in the room because I think you worked hard
4 and have a good report to show for it.

5 So we finished the morning session
6 early, which I think is a testament to how well it was
7 presented. So we came back early. I don't think we
8 really need a break, if I can read the room, at least
9 my colleagues on the dais here. Do we need to have
10 our public comments right at 2:45?

11 Thank you.

12 DR. KYLE: I've been authorized to bring up
13 a detail, and that is, I was wondering if we could
14 look up how you did the PFAS testing on this because
15 of the reason that there aren't standards either for
16 very many PFAS chemicals. So I'm -- I'm just curious
17 about.

18 DR. BALMES: One of the more recent reports
19 did find PFAS.

20 DR. KYLE: I think from the grass part.

21 DR. BALMES: Yeah.

22 DR. KYLE: I'm just interested to know how
23 you did that because you said you didn't find any.
24 I'm wondering what was tested. What did you test for?
25 And I don't want need to know right now. But thank

1 you.

2 And I guess I would also say I guess we
3 should have looked at the grass too or the -- it's not
4 grass, the plastic stuff.

5 DR. BALMES: Synthetic fibers.

6 DR. KYLE: Yes, thank you so much, John.

7 DR. BALMES: Blades, yes.

8 We need the court reporter for the
9 public comments, right?

10 We'll take a five-minute break while
11 waiting to hear from the court reporter.

12 (Recess.)

13 MS. SUWOL: I'm Robina Suwol. I'm the
14 executive director of California Safe Schools. I want
15 to thank you very much for the opportunity to comment
16 both in writing and provide a short verbal comment
17 today on the OEHHA Synthetic Turf Study Report.

18 We greatly respect and appreciate the
19 significant time and effort and extensive details
20 contributed by staff and the OEHHA scientific panel in
21 preparing the report.

22 The report indicates 35 fields were
23 studied, with a focus of risk from tire crumb rubber.
24 Given the wide variation of materials, age,
25 maintenance, and usage of artificial turf across

1 California, the limited focus on tire crumb rubber
2 appears to be insufficient to represent the broader
3 landscape and variability of all chemicals in
4 artificial turf installations statewide and their
5 health impacts.

6 We would like to see comprehensive
7 health and safety assessment with real-time
8 biomonitoring of individuals actively using the
9 synthetic turf fields under normal playing conditions.

10 The current conclusions of health
11 impacts in the report appear to have been determined
12 by the responses of the 1,069 participants who
13 completed online or in-person surveys, designated
14 staff observing and noting 40 videotaped soccer
15 players during five practices and five games, and 35
16 field studies, and artificial biofluid studies, which
17 is a wonderful first step but not the same as
18 real-time biomonitoring.

19 We hope that future research will
20 evaluate all potential pathways including not only
21 tire crumb, but synthetic turf fibers, backing
22 materials, and in-fill materials, all of which may
23 contribute to health risk.

24 Without full chemical exposure
25 biomonitoring data, the health risk analysis remains

1 limited and speculative. And given the limitations
2 identified and the time frame that these studies were
3 performed in 2015, California Safe Schools
4 respectfully and strongly urges OEHHA to call for a
5 precautionary moratorium on installation of new
6 synthetic turf fields until full comprehensive
7 peer-reviewed health and environmental risk
8 assessments, including biomonitoring of active users,
9 are completed. The health and safety of our children,
10 athletes, community, and public deserve the highest
11 protection.

12 And, again, thank you to OEHHA for this
13 report. Thank you.

14 DR. BALMES: Thank you, Ms. Suwol.

15 Do we have any other public comments?

16 MS. VAGHEFI: I do not see any hands raised.

17 DR. BALMES: I guess we should keep the line
18 open until 2:45.

19 Moving on, our next discussion is the
20 final panel discussion and comments. We've been
21 giving our overall comments along the way.

22 Any big thoughts, Dr. Bennett?

23 DR. BENNETT: I've kind of given most of my
24 thoughts.

25 DR. BALMES: That's fine.

1 Dr. McKone, any sort of overriding
2 comments?

3 DR. McKONE: No. Just I guess it's a
4 thought that we probably brought up and discussed, but
5 I think the real challenge in trying to do all of this
6 is there is an expectation that people want quick
7 answers. And it's very difficult. I mean, this is
8 not -- it's just the nature of the issue that you
9 can't throw together a team, you can't throw together
10 protocols and measurements and deploy them within
11 months.

12 As you brought out early, Dr. Balmes,
13 it took longer for a number of factors, including the
14 pandemic. A broader thought is how do we deploy or
15 how does an agency like OEHHA deploy a rapid response?
16 I'm not sure they can. But it's something I think we
17 all need to think about it, is when communities have a
18 concern.

19 I mean, a similar issue that I've been
20 involved with but also is the same kind of problem as
21 Aliso Canyon, where communities really need answers
22 and it's just putting together the team and the
23 equipment and the protocols, just takes years. And I
24 don't know if we can get around that.

25 And I'm sure there are many others that

1 we have experience with. So that's kind of something
2 that bothers me at some level, is the failure of the
3 environmental health sciences community to engage in a
4 little more rapid response because our problem is we
5 want to get it right, and if you do a rapid response,
6 you usually get it wrong. And that doesn't serve
7 anyone well.

8 So it's kind of rambling, but I think
9 that's what this and a number of other issues bring
10 up.

11 DR. BALMES: Thank you, Dr. McKone.

12 Dr. Eckel, any last comments?

13 DR. ECKEL: I just wanted to reiterate that
14 I really enjoyed this experience being on this panel.
15 I thought it was a well-conducted study. I did want
16 to think more broadly. This study was narrow and it
17 was focused on the chemical exposures from the crumb
18 rubber. You had some really interesting data on
19 temperature, and I know that that will be of interest
20 to folks in the future.

21 I didn't look in all the appendices,
22 but if you could include the data, like a plot of the
23 ambient temperature versus the surface temperature,
24 that could be useful to people in the future because I
25 think that will be of growing concern.

1 Thank you very much.

2 DR. BALMES: Before I ask Dr. Kyle for her
3 final comments, I just would echo what you just said
4 about the heat. That is in some ways the most
5 striking thing in the report for me.

6 Dr. Kyle.

7 DR. KYLE: Thank you.

8 I also have enjoyed this and appreciate
9 the creativity that OEHHA brought to this in doing
10 some things that we don't usually do. I'm not sure
11 we've drawn the full lessons from those yet. I think
12 we will, and I appreciate that because sometimes we
13 aren't creative and we do the same thing we did the
14 last time.

15 I think about this from a little
16 broader point of view in that I think we're in
17 somewhat of a crisis with toxics because we're not
18 able to keep up generally with the identification of
19 toxic materials and substances, characterization,
20 testing, listing, all those things that OEHHA does and
21 other entities like EPA do too. We're not keeping up
22 with changes in use or anything. We're still in many
23 ways in 1990.

24 And so some of the methods and
25 explorations you did here I think would be informative

1 to ways to catch up. And so that's one reason I want
2 to look into some of these things somewhat.

3 But Dr. McKone kind of raised this, we
4 don't have a rapid response capability. I think it's
5 more than that. We don't have a range of methods that
6 we can use to do something rather than nothing instead
7 of the perfect thing.

8 And I'm working a lot on PFAS now and
9 we just got last year the mega reviews of two of the
10 legacy PFAS that were supposedly phased out 20 years
11 ago. And now we have a long major review that they
12 can use to set a drinking water standard.

13 Okay, then what about the 800 other
14 ones? We're nowhere on most of that. We don't have
15 standards for most of those things.

16 And this is an endemic problem that
17 we're not keeping up. So how do we deal with that?

18 Like I said, I think you all were
19 creative in trying to do some new things and there are
20 things to learn from that, and that's kind of what my
21 next spot is.

22 Thank you.

23 DR. BALMES: Thank you for those comments.

24 And I guess I would just say that while
25 I just express my sort of frustration about how long

1 it took, on the other hand I agree with Dr. Kyle.

2 This is a state agency report that actually was
3 innovative and looked at multiple -- looked at both
4 exposure and potential toxicity of the exposures in
5 creative ways that she said.

6 So I'm not in any way trying to devalue
7 what you've done. It's our job to look at it,
8 especially how it translates for future work and to
9 the public, and I think we've done that.

10 And I'll ask Dr. Bennett for her last
11 comment.

12 DR. BENNETT: I just had one more thought.
13 It's kind of related to not being able to keep up and
14 the rapid response thing. And I think partly a lot of
15 the problem is a lot of the compounds that we're
16 concerned about, we're exposed to every day, and so
17 it's hard to tease out how much came from this event
18 that happened or the fields, and if we go all the way
19 and look at biomonitoring, you can't differentiate out
20 what was already there versus what's new from the
21 event; and the fact that we're not keeping up to have
22 measurement techniques to look at all the new things
23 to see if maybe there is something new that came from
24 some event that happened or particular exposure. And
25 then, again, understanding how those go into the body

1 and what the changes in concentration are.

2 So it's just all very challenging, and
3 I do feel like you guys did the absolute best that you
4 could with the techniques and methods that we have
5 available.

6 DR. BALMES: And I guess I would say that
7 one of the main reasons we can't keep up is that new
8 chemicals and new uses of the chemicals are being put
9 forward on almost a daily basis, certainly a frequent
10 basis. And I think Dr. Kyle would probably agree with
11 me, we need to go upstream and assess exposures in
12 toxicity before there's wide market use of something.

13 I mean, crumb rubber use was supported
14 by the EPA because it was a good use of -- good way to
15 recycle tires, which when they're in a big dump they
16 catch on fire and they burn for months.

17 Europe has done a little bit better
18 than we have with their REACH efforts, but we really
19 need to be looking at toxicity before market --
20 widespread market use, which we're a long way from in
21 this country.

22 I think Mr. Avol has a comment or two.

23 MR. AVOL: So I just have one other
24 consideration. I think I look at this -- in some ways
25 I look at the reporting in two parts, one in terms of

1 the science, which is excellent. It was diverse. It
2 was creative. Thought about a lot of these pieces.
3 Many different groups contributed to it, and all
4 should be thanked as an accomplished piece of work.

5 And I think about how this is going to
6 be used and how it's described and contextualized. So
7 on that second point I would just say clearly -- well,
8 I don't want to put words in your mouth. But from my
9 perspective in 2014, 2013, there's a lot of concern
10 about the use of crumb rubber on these fields and that
11 was the focus.

12 And so it makes contextual complete
13 sense that we systematically went to understand what
14 it was in that rubber which was not meant to be used
15 for children's fields but rather for car tires and
16 truck tires and so forth, to see if there were any
17 health concerns. So I think that's completely
18 legitimate.

19 Now that we've looked at that and we
20 understand that, maybe if we could rewind and go back
21 and say, well, there's also the plastic fibers.
22 There's also the cushioning material. There's several
23 components to this, but clearly the crumb rubber was
24 the red flag in our face that we're trying to
25 understand, and the paints and the coverings and so

1 forth on the material.

2 So I don't think you need to be
3 particularly defensive about focusing on the rubber,
4 but I think, again, in the report, in the summary and
5 description, maybe in the introduction or rationale
6 for why this study was set up the way it was, I think
7 it would be appropriate to say something. Crumb
8 rubber is widely and generally used in all this and
9 was not made for a specific purpose. And so from a
10 public health standpoint, it was an important question
11 to ask because of the potential for exposure.

12 DR. BALMES: I think if the panel is
13 finished with final comments, I'll turn the mic over
14 to Dr. Edwards.

15 DR. EDWARDS: Thanks, John.

16 All right. So just wanted to really
17 thank the committee. Really appreciate all the
18 thoughtful comments from today. I thought the
19 discussion was excellent, and we really do appreciate
20 the feedback around the draft report and sort of
21 broadly where we can go in the future.

22 Just to kind of summarize some of the
23 main actions that I heard, I think from the afternoon
24 discussion to -- and also from the morning from
25 Dr. Bennett's comments around sort of the time

1 activity relationships, really look at those factors
2 to -- I guess my quotes here or my notes says "the
3 soccer is life youth," where there is a strong soccer
4 component to their everyday activities that we can
5 kind of look at and see where the values come from
6 that compared to what we had done.

7 The sort of second piece around -- and
8 this is just a broader categorization, but within the
9 limitations and the uncertainties to really be able to
10 highlight and provide context into some of the areas a
11 little bit more that we had not looked at before to
12 kind of see if we could tease out a little bit on the
13 first-year effects a little bit more. And then also
14 to maybe look at heat, if there's any inferences we
15 can make on the evaluation of the temperatures a
16 little bit more throughout the report.

17 The last item is sort of to kind of go
18 back and have that sort of revisit component to really
19 look at the chemicals that we initially started
20 looking at. This gets to Dr. Kyle's comments. And to
21 be able to maybe talk about why they weren't conducted
22 and sort of so that there is an expansion of the
23 discussion to have a broader awareness of these gaps
24 and that was not evaluated during this study.

25 As for next steps on this report, we're

1 going to be taking into consideration the oral
2 comments that we received today, the panel member
3 comments, any written comments that we've also
4 received, updating the current draft and finalizing
5 later this year. So we're excited to get to a sort of
6 closure on this work.

7 A couple of -- just to go off of
8 Dr. Kyle's comments on sort of we're behind a bit.
9 Just sort of stay tuned. We had a couple of workshops
10 last year. It's what Dr. Krishnan is leading a group
11 at OEHHA that just started a couple years ago
12 concerning computational toxicology and new approach
13 methodologies sort of really looking instead of trying
14 to identify everything with animal studies or an epi
15 study to be able to leverage existing information that
16 does have those types of studies, to looking at
17 chemical analogs and so forth from not only a
18 scientific and technical perspective, but also the
19 ability to use in a regulatory construct.

20 So I think the behindness part could
21 maybe sort of try to -- through this, can hopefully
22 maybe be able to get on par or catch up to some
23 extent. But I did want to highlight that to this
24 group so that you're aware that we are starting this
25 work and in the next couple years hope to have

1 something out in the near future.

2 DR. BALMES: I'm glad to hear that, Dave,
3 because in the academic world that's already been
4 going on for a few years, as I'm sure Dr. Krishnan
5 knows. But it's good to see OEHHA getting there,
6 getting up to speed.

7 DR. KYLE: Dr. Balmes, if I might, just for
8 a second. And it's not just you. Everyone is behind.
9 I didn't want that to sound like it was OEHHA. And I
10 listened in to some of those workshops and there's
11 some great stuff there.

12 DR. BALMES: I would just say this state is
13 blessed to have OEHHA. Most states don't have
14 anything like this agency.

15 DR. EDWARDS: So we're just waiting for four
16 more minutes. Okay. This is going to be the last
17 call for anyone that wants to have a public comment go
18 on the record.

19 I don't see anything in the room and
20 we're not seeing anything online.

21 So with that, I will go ahead and
22 adjourn the meeting. Once again, thank you for
23 everyone that attended online and in person. And the
24 panel.

25 Thank you.

(Meeting adjourned at 2:41 p.m..)

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1 STATE OF CALIFORNIA)
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2 COUNTY OF LOS ANGELES)

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