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Stantec Consulting Services Inc.
55 Church Street Suite 601,
New Haven CT 06510-3014

Conceptual Design Study for Improvements to Nursery Field
Nursery Field, Rye NY

PROJECT SUMMARY

Introduction

Stantec Consulting Services Inc. has been retained by the City of Rye to prepare a feasibility study for the replacement of Nursery field either with a natural turf field or a synthetic turf field. The various options for each field, as well as the sub options within each field type were explored and opinions of probable cost were developed for each.

Nursery Field is currently a natural turf field in poor condition. Despite significant maintenance by the Parks Department, little desirable turf remains on the field. The demand for playing hours on this field regularly exceeds the typical recommended playing hours for a natural turf field, thus contributing to the deterioration of turf. Furthermore, Nursery Field is poorly drained and rain events will typically cause field closures, further exacerbating the over programming of the field.

Existing Conditions

A survey of the existing conditions at Nursery Field was performed by T.C. Merritts on May 17, 2019. This survey can be found within the attached *Appendix A*. The field slopes from the southeast corner (Elevation 14.72) diagonally to the northeast corner (Elevation 9.82). This is not typical of a well-drained, properly constructed field. A natural turf field should have a crown down the middle with a 2% pitch to the sidelines. This allows the water to run off the field rather than accumulate, which is likely what is currently occurring on site. The long slope across the diagonal of the field is leading to the poor turf conditions. The field is within the FEMA 100-year floodplain which has an elevation of 12.0. See *Appendix J*. for flood map. This flood elevation will inundate the field during a 100-year event. It is recommended that the field be raised above this elevation to protect it from flooding.

Geotechnical

On April 16, 2019, soil borings were conducted by SoilTesting Inc. to determine the nature of the underlying soils at the field. Infiltration rates were identified as very low. Geoprobes were drilled 6 feet below the surface of the field. In general, they revealed that the soil was a silty-sand and no groundwater was detected within 6 feet of the field surface. In the southeast corner, the probes hit refusal or cobbles at the bottom of the probe. The complete soil boring results can be found in the *Appendix G*.

On April 23, 2019, three (3) topsoil samples were taken and analyzed by Turf and Soil Diagnostics. Each sample was classified as gravelly sandy-loam per USDA textural classification. The soil had between 56% to 62% sand, 26%-34% silt and 10%-11% clay. The soil had a pH from 6.4 to 6.7 and approximately 4% – 5.6% organic matter. The infiltration rate was 0.1 inches per hour. According to the report, these soils would release water gradually and may be prone to excessive water retention. This confirms the reality of what is currently happening at the field. The complete results and summary can be found in *Appendix H*.

Natural Field

A natural turf field would consist of a root zone mix of 6 to 12 inches thick. This can be either amended natural soils or an imported mix. Below this root zone would be imported suitable soil to raise the grade to the proposed elevation. The existing subgrade would be compacted and stripped of existing vegetation.

Another option for the root zone would be a sand-based soil which would include 90 percent sand. This construction type would require significantly more maintenance than typical natural turf fields. Natural turf

fields can be developed from seed or from sod. Each has its own advantages and disadvantages. Refer to the Field Renovation presentation in *Appendix D.* for additional information.

Synthetic Turf Field

A synthetic turf field begins with a free draining stone layer below the field which is then covered with a finishing layer of finer stone. A shock pad is typically installed, then the turf is placed on top of that. The turf is then infilled with a variety of fill materials. Typically sand and SBR rubber are used, but there are a variety of other options available to the City. These infill options are summarized in the Infill Matrix in *Appendix E*. Also refer to the Field Renovation presentation in *Appendix D*.

Health & Environmental Concerns

In recent years, some concerns have been raised concerning the use of synthetic fields. There have been several studies which have investigated these matters. Copies of several of these studies are contained in *Appendix I*. There are also issues and concerns in regarding the use of pesticides, herbicides and fertilizers on natural turf fields that can be taken into consideration.

Construction Costs/Maintenance

A synthetic field costs more in initial construction than a natural turf field, however the annual maintenance costs are less for synthetic. A detailed estimate can be found in the Field Renovation presentation in *Appendix D*. Also, relevant is the information provided in *Appendix F.*, Stantec Synthetic Turf Field Projects.

Advantages/Disadvantages

There are various advantages/disadvantages for the use of each field type. These have been identified within *Appendix D*. The primary advantage of a synthetic turf field over natural grass field is synthetic fields are weather resistant, therefore allowing a significantly greater amount of playing time over the life of the field.

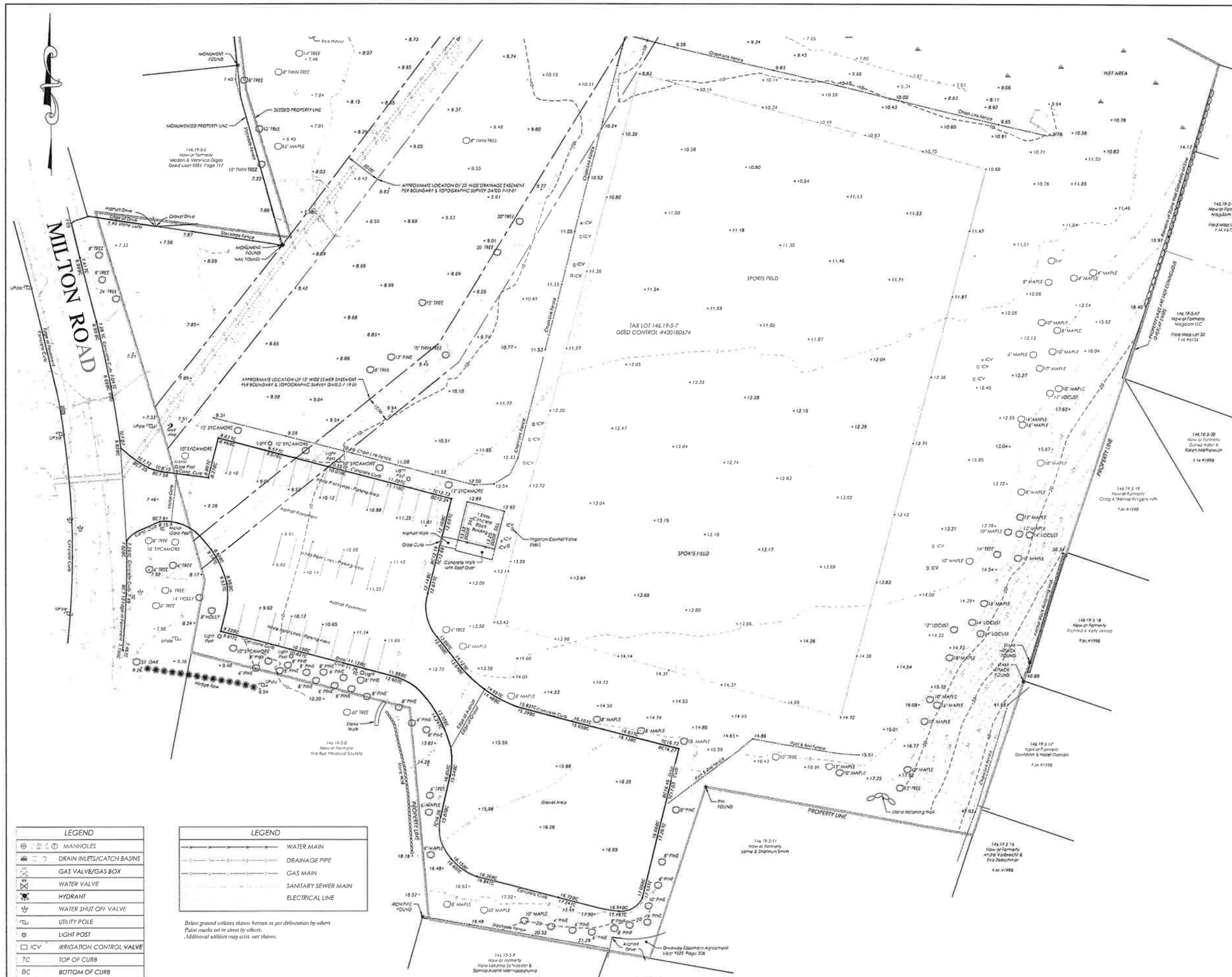
See the following attachments for additional information.

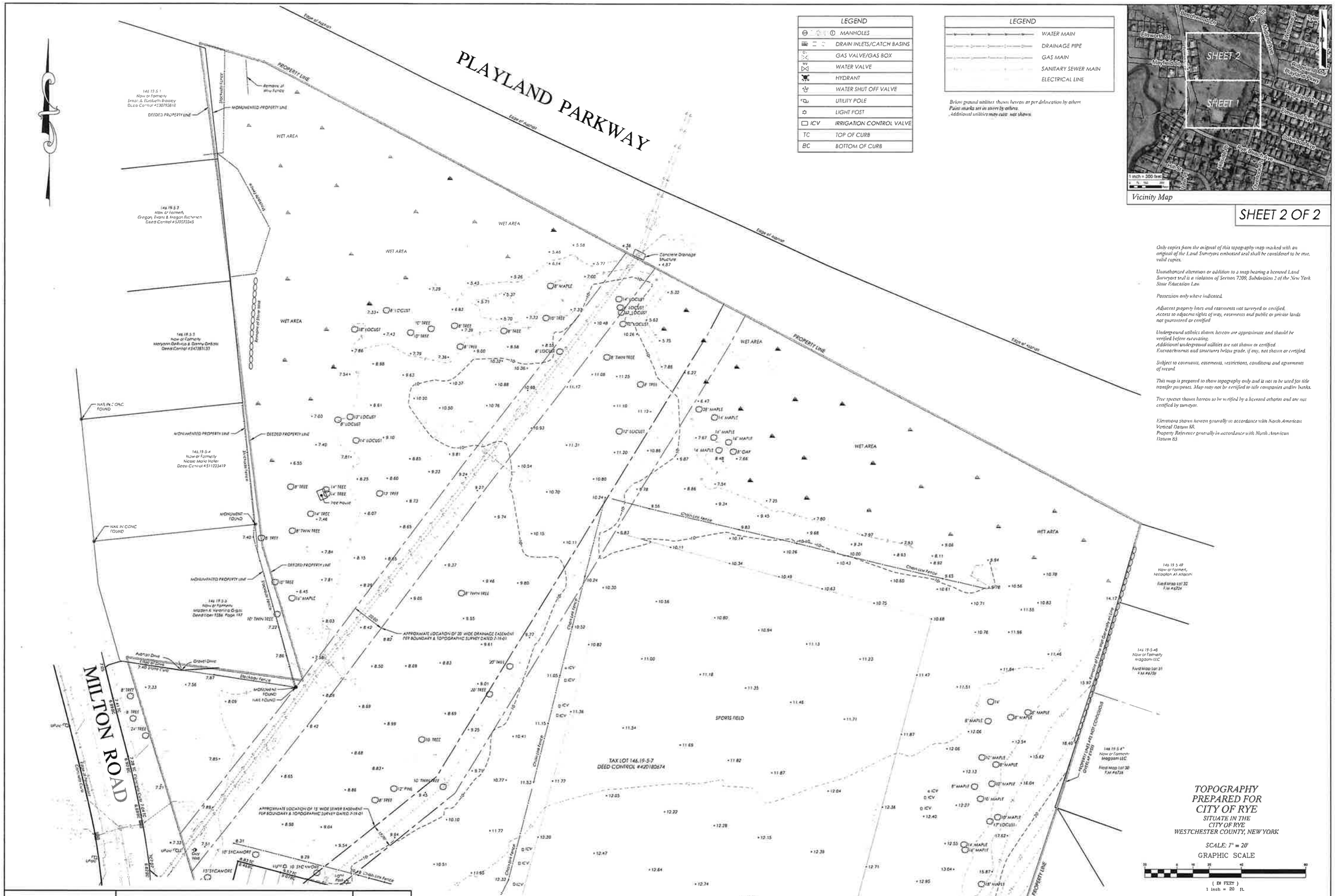
Appendix:

A. Site Survey
B. Field Layout
C. Field Sections
D. Field Renovation Options – PPT Presentation
E. Infill Matrix
F. Stantec Synthetic Turf Field Projects
G. Geotech Log Report
H. Soil Study Testing
I. Health Studies and the Environment
J. FEMA Flood Map

Appendix:

A. Site Survey


MILTON ROAD
TAX LOT 146.19-5-7
DEED CONTROL #201806574
SPORTS FIELD
WET AREA
PROPERTY LINE
Chain Link Fence
Stockade Fence
Asphalt Drive
Gravel Drive
Asphalt Parking Area
Concrete Walk
Gravel Area
Stone Wall
Stone Retaining Wall
Wooden Fence
Post & Rail Fence
Irrigation Control Valve
Driveway Easement Agreement
Liber 4325 Page 326
146.19-5-6
Now or Formerly
The Rye Historical Society
146.19-5-8
Now or Formerly
Hans Lorenzo Schroeder &
Samira Avanti Mamiaddaphung
146.19-5-11
Now or Formerly
Lorne & Stephanie Smith
146.19-5-17
Now or Formerly
Andre Valbrecht &
Eva Deutchman
F.M. #1998
146.19-5-18
Now or Formerly
Richard & Kelly Jenson
F.M. #1998
146.19-5-17
Now or Formerly
Davidson & Hazell Gorman
F.M. #1998
146.19-5-19
Now or Formerly
Craig & Melinda Kingsmith
F.M. #1998
146.19-5-20
Now or Formerly
Daniel Adler &
Karen Matthews Jr.
F.M. #1998
146.19-5-47
Now or Formerly
Magdom LLC
Filed Map Lot 32
F.M. #5726
146.19-5-48
Now or Formerly
Magdom LLC
Filed Map Lot 31
F.M. #5726
146.19-5-3
Now or Formerly
Dr. Ralph & Kathleen Ann Cordero
F.M. #1998
146.19-5-5
Now or Formerly
Wesley & Veronica Cagas
Deed Liber 9585 Page 157
146.19-5-13
Now or Formerly
Agnes Koch
MONUMENT FOUND
DEEDED PROPERTY LINE
MONUMENTED PROPERTY LINE
APPROXIMATE LOCATION OF 20' WIDE DRAINAGE EASEMENT PER BOUNDARY & TOPOGRAPHIC SURVEY DATED 7-19-01
APPROXIMATE LOCATION OF 15' WIDE SEWER EASEMENT PER BOUNDARY & TOPOGRAPHIC SURVEY DATED 7-19-01
PIN FOUND
IRON PIPE FOUND
NAIL FOUND
Hedge Row
Hedge Saw
Stone Arch Culvert
Concrete Curb
Asphalt Walk
Grab Curb
Light Post
Metal Gate Post
Concrete Block Building
Wooden Fence Posts
Wooden Fence Lines - Parking Areas
Asphalt Pavement
Gravel Drive
Stone Wall
Stone Arch Culvert
Stone Retaining Wall
Post & Rail Fence
Wooden Fence
Chain Link Fence
Concrete Curb
Asphalt Drive
Stone Arch Culvert
Stone Arch Culvert
Stone Retaining Wall
Stone Retaining Wall
Stone Retaining Wall



LEGEND	
	MANHOLES
	DRAIN INLETS/CATCH BASINS
	GAS VALVE/GAS BOX
	WATER VALVE
	HYDRANT
	WATER SHUT OFF VALVE
	UTILITY POLE
	LIGHT POST
ICV	IRRIGATION CONTROL VALVE
TC	TOP OF CURB
BC	BOTTOM OF CURB

LEGEND
WATER MAIN
DRAINAGE PIPE
GAS MAIN
SANITARY SEWER MAIN
ELECTRICAL LINE

Below ground utilities shown hereon as per delineation by others
Paint marks set in street by others.
Additional utilities may exist not shown.

Vicinity Map

SHEET 2 OF 2

Only copies from the original of this topography map marked with an original of the Land Surveyors embossed seal shall be considered to be true, valid copies.

Unauthorized alteration or addition to a map bearing a licensed Land Surveyors seal is a violation of Section 7209, Subdivision 2 of the New York State Education Law.

Possession only where indicated.

Adjacent property lines and easements not surveyed or certified.
Access to adjacent rights of way, easements and public or private lands not guaranteed or certified.

Underground utilities shown hereon are approximate and should be verified before excavating.
Additional underground utilities are not shown or certified.
Encroachments and structures below grade, if any, not shown or certified.

Subject to covenants, easements, restrictions, conditions and agreements of record.

This map is prepared to show topography only and is not to be used for title transfer purposes. Map may not be certified to title companies and/or banks.

Tree species shown hereon to be verified by a licensed arborist and are not certified by surveyor.

Elevations shown hereon generally in accordance with North American Vertical Datum 88.
Property Reference generally in accordance with North American Datum 83.

TOPOGRAPHY PREPARED FOR CITY OF RYE

SITUATE IN THE
CITY OF RYE
WESTCHESTER COUNTY, NEW YORK

SCALE: 1" = 20'

GRAPHIC SCALE

(IN FEET)
1 inch = 20 ft.

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(914) 769-8003 • (203) 622-8899

Surveyed: May 17, 2019
Map Prepared: May 24, 2019

By: David T. Merritt
New York State Licensed Land Surveyor No 050604

Project: 18-417	Field Survey By: BC/CR
Drawn By: DA	Checked By: DM

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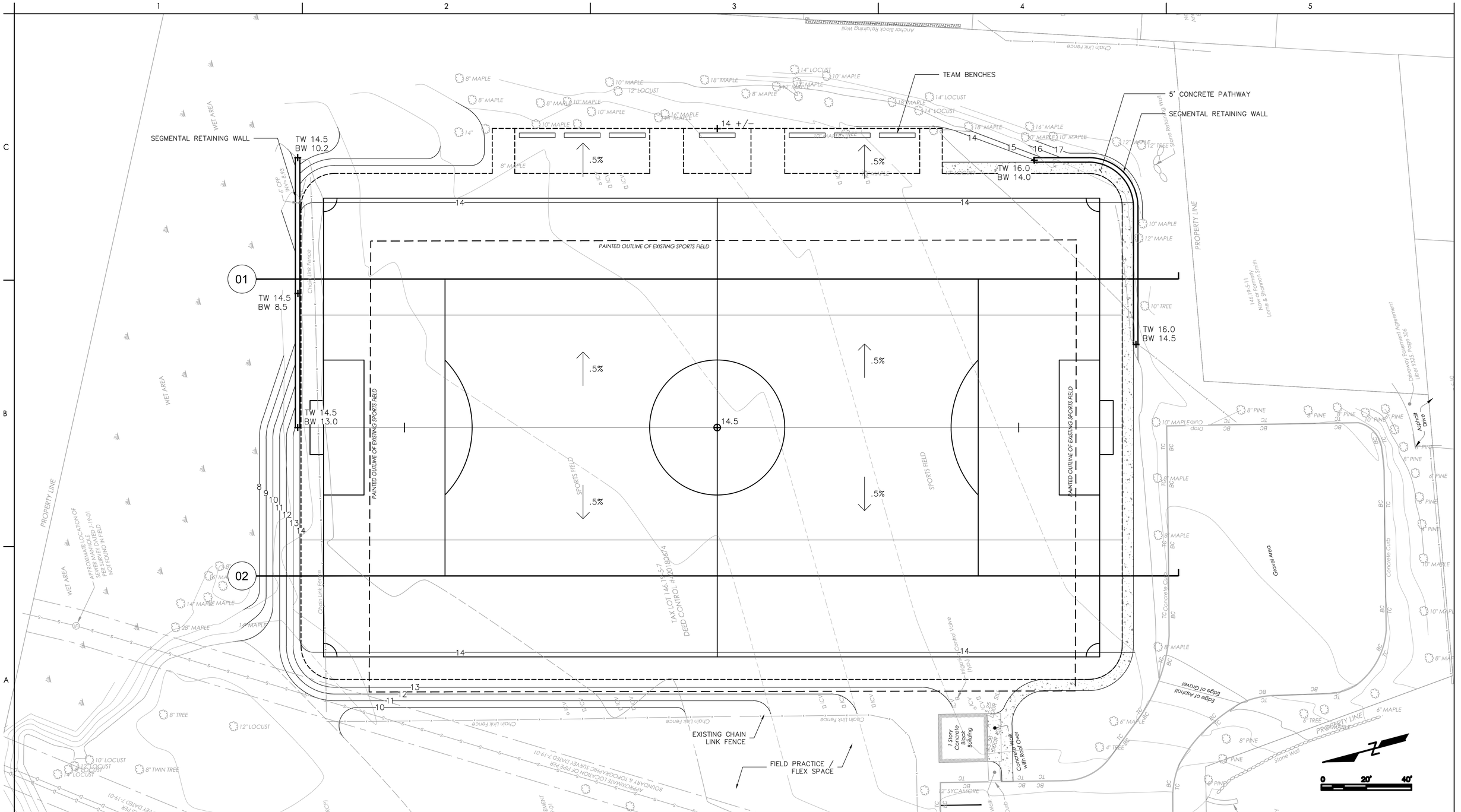
Appendix:

B. Field Layout



Revision	By	Appd	YYYY.MM.DD

Issued	By	Appd	YYYY.MM.DD

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Client/Project Logo

CITY OF RYE NY 1942 1660 1904

Client/Project

CONCEPTUAL DESIGN STUDY FOR IMPROVEMENTS TO NURSERY FIELD

RYE, NY

File Name: L-SITE-PLN+DTL	CH	CH	PK	2019.06.26
	Dwn.	Dsgn.	Chkd.	YYYY.MM.DD

Title
LAYOUT & GRADING PLAN

Project No.	Scale
192311021	1" = 20'
Revision	**Sheet** / **Drawing No.**
0	1 of 2 / L001

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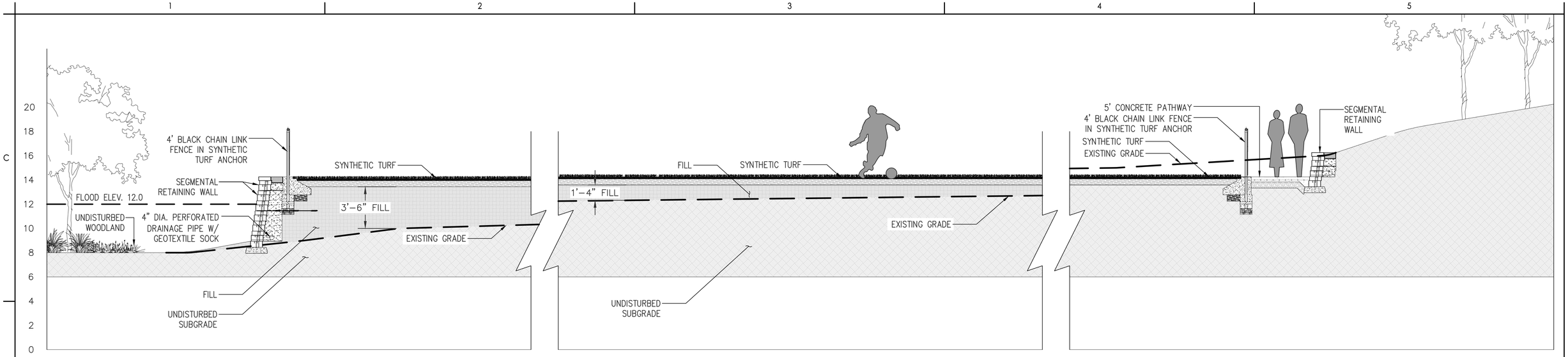
Appendix:

C. Field Sections



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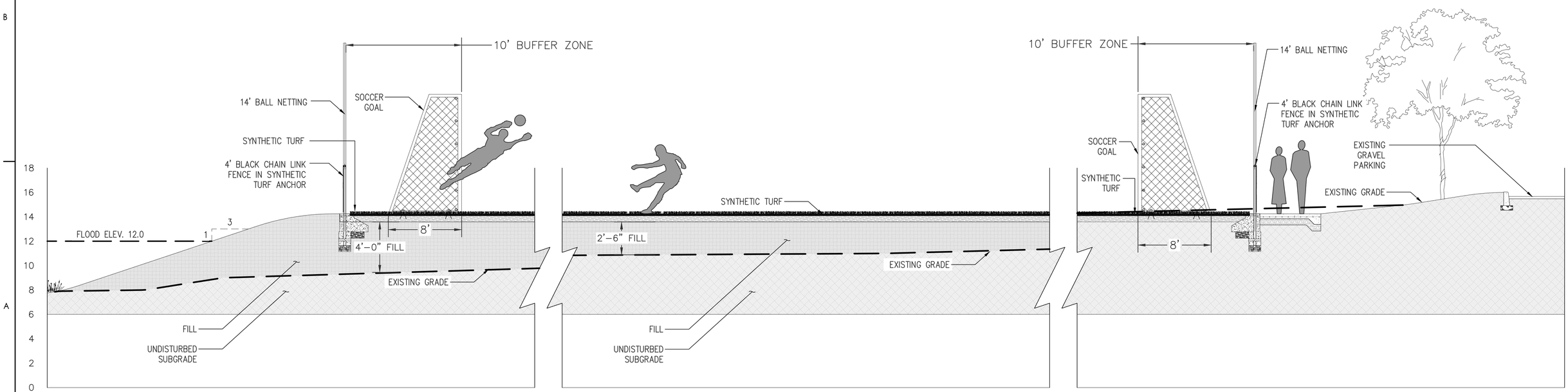
SCALE: 1/4" = 1'



02 SECTION B
SCALE: 1/4" = 1'

Revision	By	Appd	YYYY.MM.DD

Issued	By	Appd	YYYY.MM.DD

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CITY OF RYE NY 1942 1660 1904

Client/Project

CONCEPTUAL DESIGN STUDY FOR IMPROVEMENTS TO NURSERY FIELD

RYE, NY

File Name: L-SITE-PLN+DTL	CH	CH	PK	2019.06.26
	Dwn.	Dsgn.	Chkd.	YYYY.MM.DD

Title
SECTIONS

Project No.	Scale
192311021	1/4" = 1'

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Appendix:

D. Field Renovation Options – PPT Presentation

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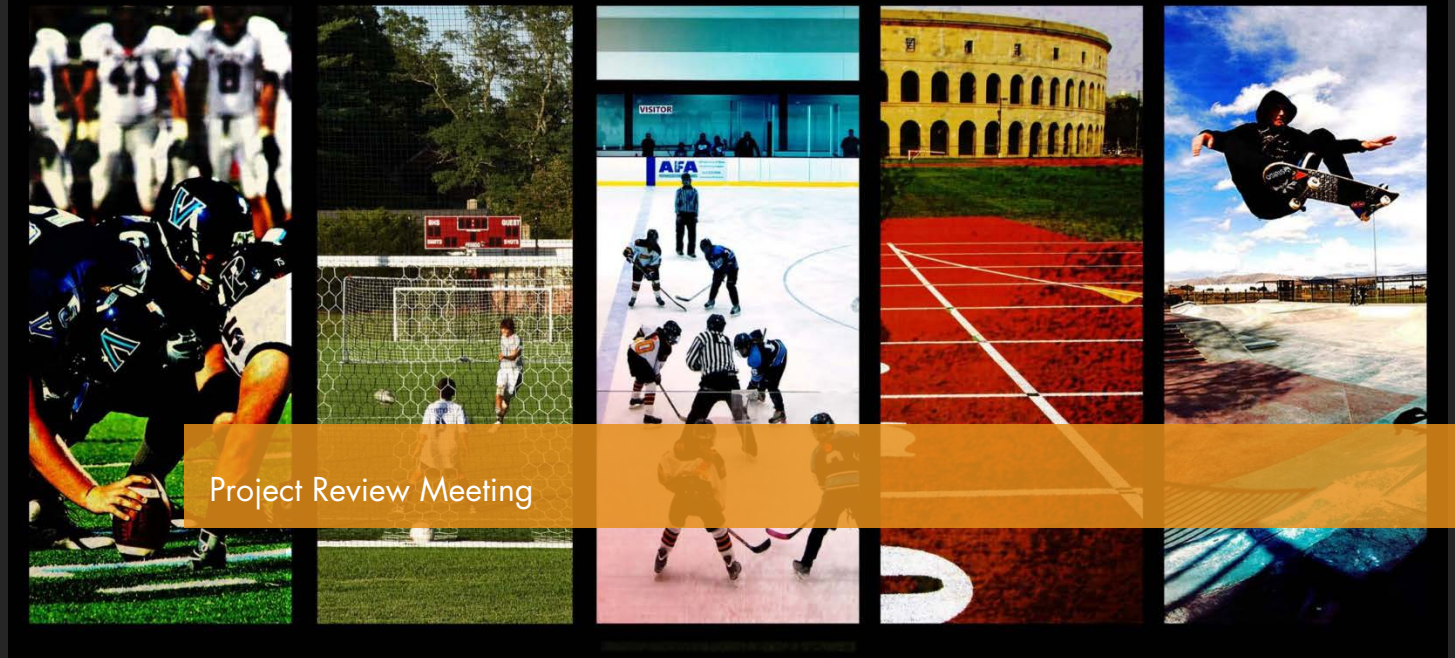

Project Review Meeting

15 May 2019

Field Renovation Options

Nursery Field | Rye, New York

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Agenda

1. Existing Conditions Review
2. Natural Grass Renovation
   a. Natural Grass System
   b. What the Reno Will Entail
3. Synthetic Turf Renovation
   a. Synthetic Turf Components
   b. What the Reno Will Entail
4. Renovation Differences, Pros and Cons Natural vs. Synthetic
5. Costs, Hours of Use
6. Other Questions/Concerns?

Existing Condition Review

North

Field Renovation Options

Nursery Field | Rye, New York

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Existing Condition Review

Field Renovation Options

Nursery Field | Rye, New York

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Natural Grass Renovation – Field Layout

Field Renovation Options

Nursery Field | Rye, New York

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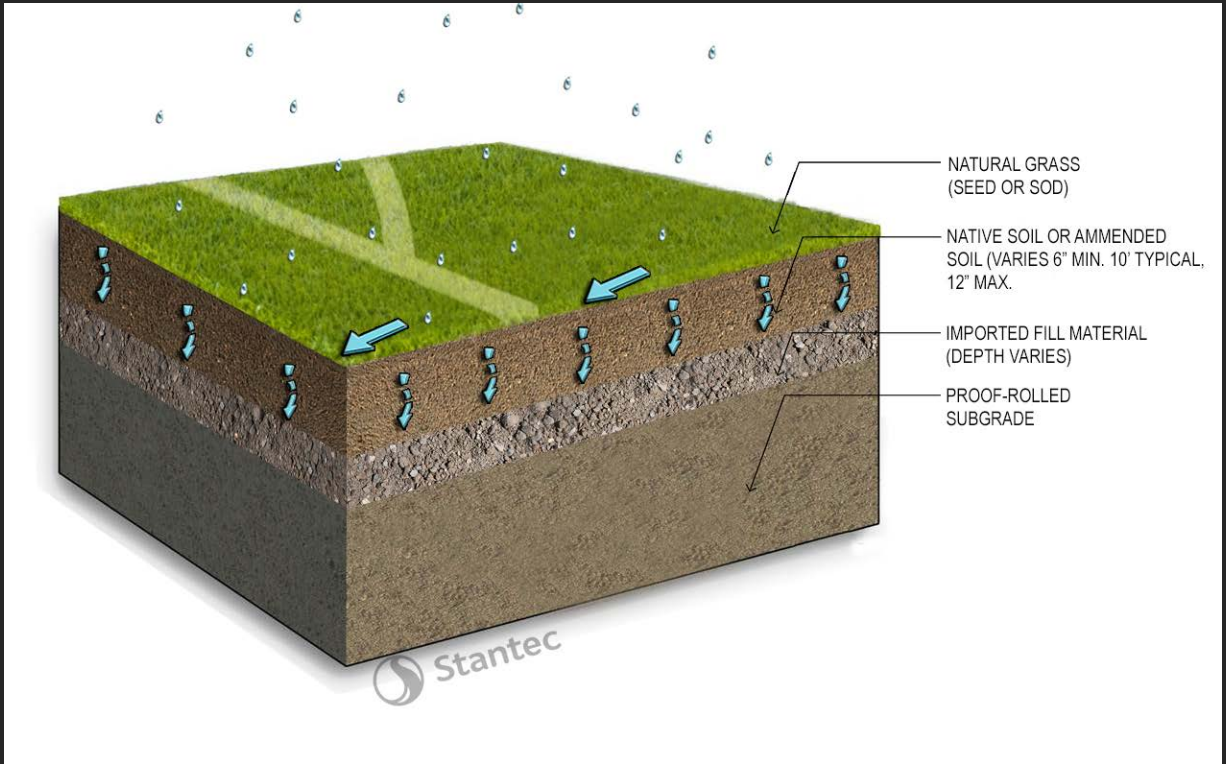
Natural Grass Renovation



Field Renovation Options

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Natural Grass Renovation : Seed vs Sod

» ADVANTAGES AND DISADVANAGES

	Seed	Sod
PROS....	▪ DEEP ROOTS SYSTEM ▪ LOW INITIAL COST	▪ IMMEDIATE GRASS COVERAGE ▪ HIGH INITIAL COST ▪ LESS MAINTENANCE DURING GROWN IN ▪ IF INSTALLED FALL 2020, READY FOR PLAY SPRING 2021
CONS....	▪ NOT IMMEDIATE GRASS COVERAGE ▪ ESTABLISHMENT PERIOD IS 2 GROWING SEASONS ▪ IF INSTALLED FALL 2020, READY FOR PLAY FALL 2021	▪ TEND TO HAVE SHALLOW/ WEAKER ROOTS ▪ MORE EXPENSIVE INITIAL COST

Synthetic Turf Renovation – Field Layout

Field Renovation Options

Nursery Field | Rye, New York

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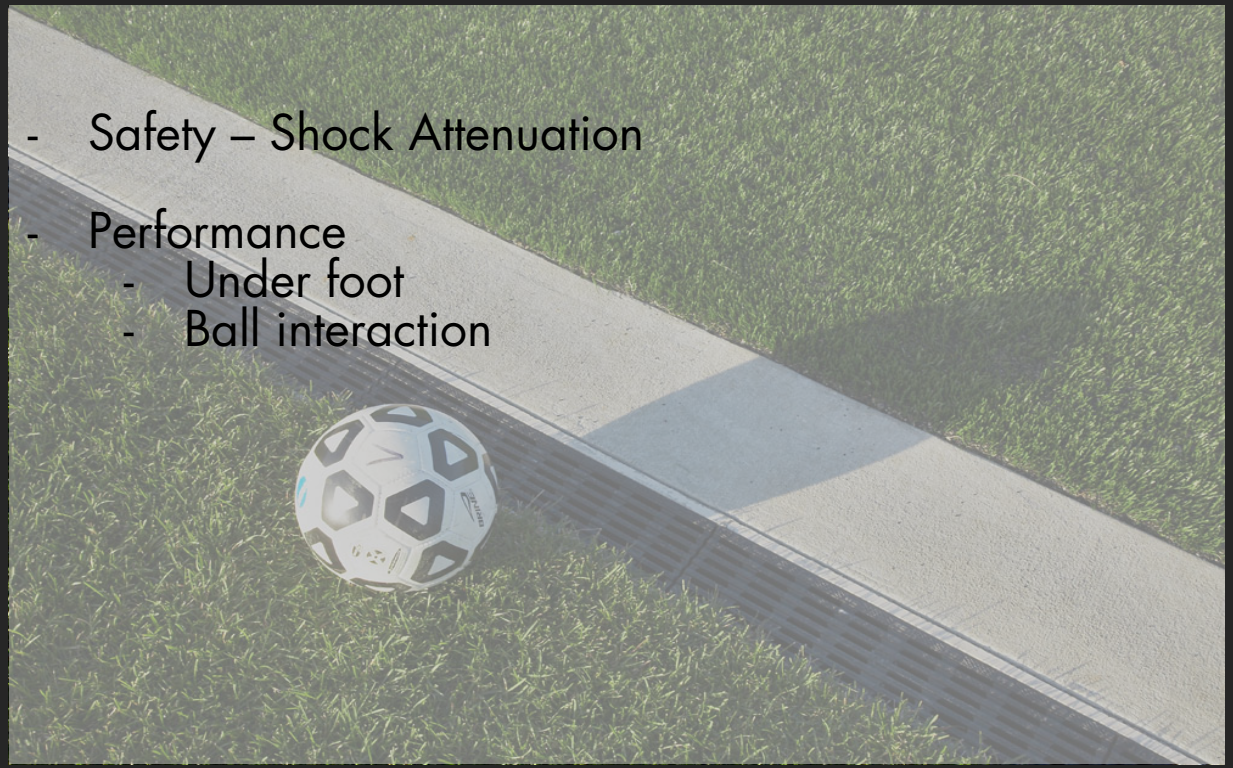
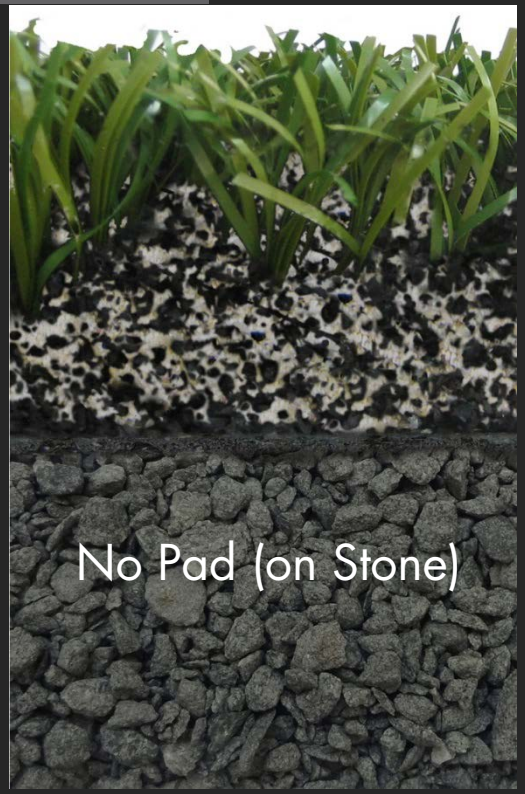
Synthetic Turf to Emulate Natural Grass

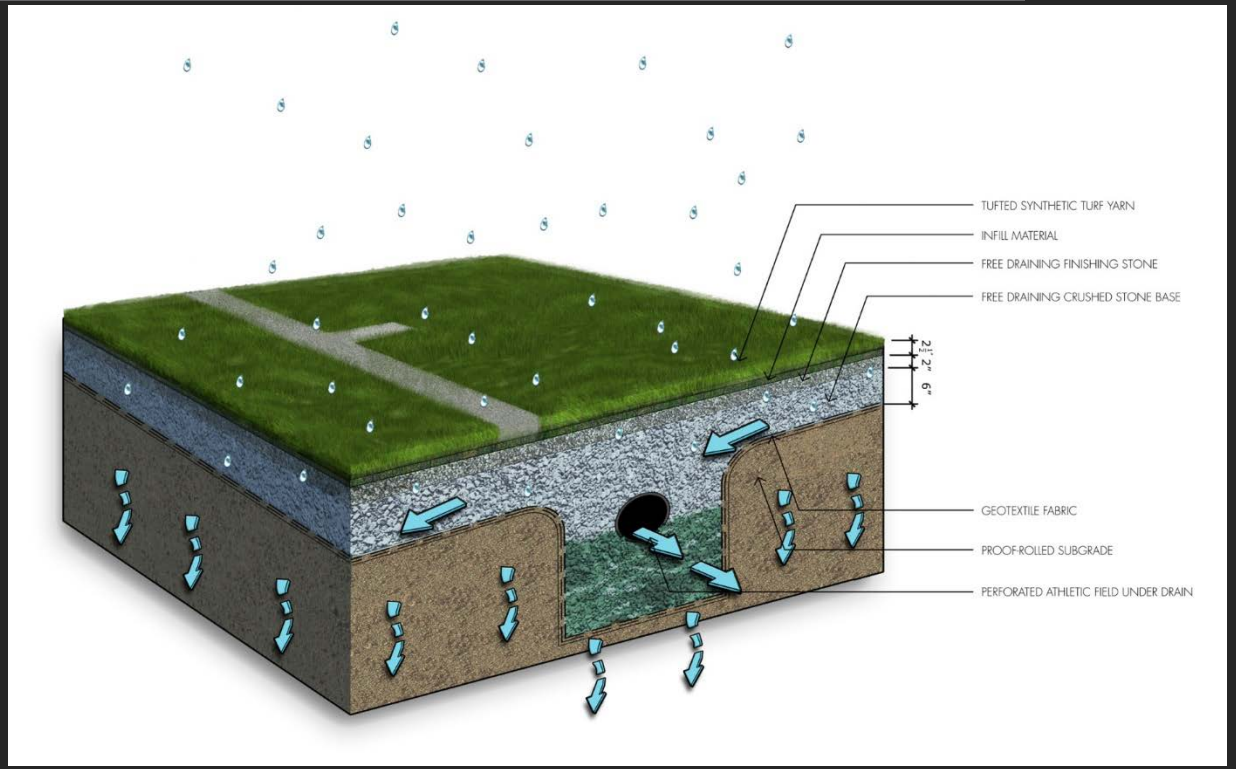


- Safety – Shock Attenuation
- Performance
  - Under foot
  - Ball interaction

Shock Pad System vs. Non Padded Systems



Turf Base Profile – *Conventional System*



Field Renovation Options

Nursery Field | Rye, New York

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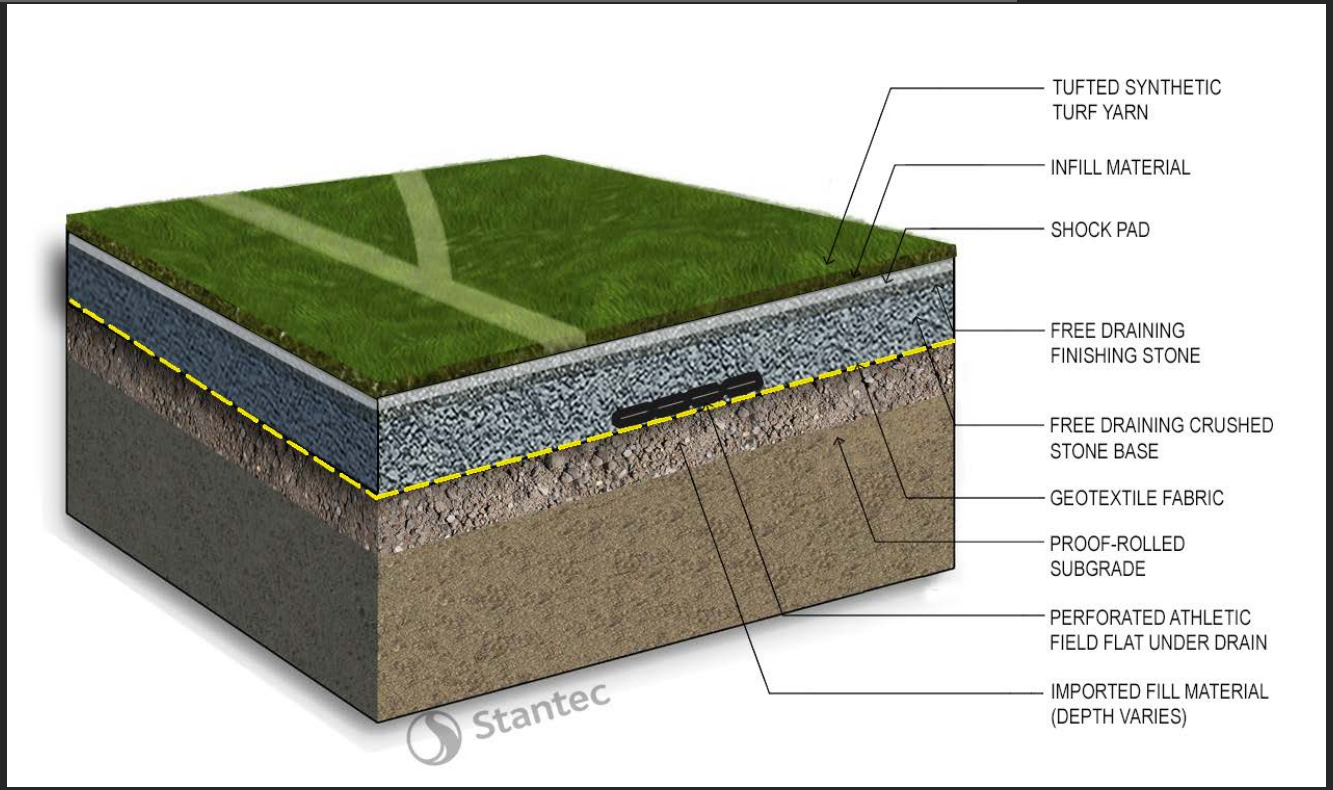
Turf Base Profile – Flat Drain System



Synthetic Turf System Components

Fiber – Traditional Slit Film

Fiber – Monofilament

Fiber – Parallel Slit Film

Fiber – Dual (Slit Film and Monofilament)

Components

Turf Fiber

Primary Backing

Secondary Backing

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Synthetic Turf System Components

Components

Infill

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Synthetic Turf System Components

Infill – Recycled / SBR

Infill – Natural

Infill – Sand(s)

Infill – TPE

Components

Infill

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Synthetic Turf Infill

	Recycled Rubber	TPE	Sand	Natural
MAINTENANCE	▪ LOW	▪ LOW	▪ LOW	▪ HIGH
COST	▪ $65K - $80K	▪ $310K -$320K	▪ $195K - $220K	▪ $150 - $210K
LIFE CYCLE	▪ 10 – 20+ YRS	▪ 20+ YRS	▪ 20+ YRS	▪ 8-10 YRS
ENVIRONMENTAL (PERCEIVED)	▪ MED	▪ LOW	▪ N.A.	▪ LOW

Synthetic Turf System Components

Pad – Panel/Roll

Pad – E-Layer

Components

Shock Pad

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Athletic Systems / Surfacing

» ADVANTAGES AND DISADVANAGES

	Natural Grass Systems	Synthetic Turf Systems
PROS….	▪ SURFACE TEMPERATURE ▪ MAINTAIN NATURAL SURFACE ▪ INITIAL COST (NATIVE)	▪ DURABILITY/HOURS OF USE ▪ COST PER HOUR OF USE ▪ WEATHER RESISTANCE ▪ LESS MAINTENANCE/ NO FERTILIZER/ NO WATER
CONS….	▪ MAINTENANCE/FERTILIZER/ WATER/$/$$$ ▪ DURABILITY/HOURS OF USE ▪ WEATHER/FIELD CLOSURES	▪ INITIAL COST ▪ SURFACE TEMPERATURE

Native Soil Natural Grass $

Athletic Systems / Surfacing

» HOURS OF USE

In order to maintain a high quality surface, natural systems are limited in the plan they can withstand and weather only impacts these limitations

- NATIVE SOIL NATURAL GRASS 10-15 HOURS
- SAND BASED NATURAL GRASS 15-20 HOURS
- SYNTHETIC TURF SYSTEMS 50+ (AND MINIMAL CANCELATIONS ETC.)

More available hours translates to more children and adults in the community have the chance to be active!

- CURRENT NURSERY FIELD PROGRAM 44.5 HOURS SPRING & 36.5 HOURS IN FALL

Cost Analysis

CAPITAL COSTS - FIELD FOOTPRINT ONLY

- AMENDED TOP SOIL FIELD
  - Remove and amend topsoil
  - Fill and Grade subgrade
  - Perimeter drainage
  - Spread and laser grade amended soil
  - Seed or sod, grow-in time

$550K - $700K

- SYNTHETIC TURF FIELD
  - Remove and dispose topsoil
  - Fill and Grade subgrade
  - Drainage layer and perimeter drainage
  - Finishing stone and laser grade
  - Turf system installation (shock pad, carpet, sand and rubber infill)

$1.2M - $1.5M

Cost Analysis

CAPITAL COSTS - TOTAL PROJECT

- AMENDED TOP SOIL FIELD
  - Natural Grass Field
  - Retaining wall - $75K +/-
  - Chain Link Fence and Netting - $180K +/-
  - Fill Material - $250K +/-
  - Score Board - $30K +/-
  - 100 Seat Portable Bleachers - 12K +/-
  - Goals and Benches - $24K +/-
  - Soft Costs - $110K - $150K +/-
  - Contingencies

$1.3M - $1.6M

- SYNTHETIC TURF FIELD
  - Synthetic Turf System
  - Retaining wall - $75K +/-
  - Chain Link Fence and Netting - $180K +/-
  - Fill Material - $200K +/-
  - Score Board - $30K +/-
  - 100 Seat Portable Bleachers - 12K +/-
  - Goals and Benches - $24K +/-
  - Soft Costs - $150K - $180K +/-
  - Contingencies

$1.8M - $2.2M

Cost Analysis

TYPICAL ANNUAL MAINTENANCE

» AMENDED TOP SOIL FIELD +/–$30,000

Mowing, fertilizer, water, over seeding, aeration, herbicide & pesticides

» SYNTHETIC TURF FIELD +/-$14,000

Grooming, sweeping, inspecting/adding infill, addressing goal areas, water (if needed for natural infill) etc.

*These include labor costs

Cost Analysis

FIELD LIFE CYCLE COSTS 20 YEARS	TOTAL	PER HOUR OF USE
» AMENDED TOP SOIL FIELD (Program as recommended, 600 hours/year)	$1.3 M	$109
» SYNTHETIC TURF FIELD (Program Up to 2000 hours)	$2.4 M	$60

* Depends of typical Town usage

Natural vs Synthetic

Factor	Natural Grass Systems	Synthetic Turf Systems
SURFACE TEMPERATURE	80°	130°+ (with SBR & Sand)
HOURS OF USE/WEEK	10-15	50+
MAINTENANCE ($)	+/-$30K	+/-$14K
INITIAL COST	$550K - $700K	$1.2M - $1.5M
ENVIRONMENTAL	FERTILIZERS, PESTICIDES	RUNOFF
SAFETY	VARIES, RUTS, COMPACTION	MORE CONSISTENT SHOCK ATTENUATION
CLOSURES	FREQUENT AFTER STORMS	NEVER

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Questions

Thank you for your time

Appendix:

E. Infill Matrix

Synthetic Turf Infill Matrix

6/14/2019

INFILL PRODUCT	DESCRIPTION	PROS	CONS	LIFE CYCLE	COST
Recycled Rubber Infill					
SBR Rubber	Styrene Butadiene Rubber. Recycled tires, referred to as "Crumb Rubber. Granular shape, the crumb rubber infill blend is derived from used car tires that are ground up and recycled. Crumb rubber is a durable, high-performing and low-cost infill that provides shock absorption, traction, and foot stability	Performs to the athletes advantage in terms of playability and shock absorption. Durable. Is a recycled material and reduces tires in landfill. Easily available. Very low cost.	Recent health concerns in media, though none of it proved scientifically. Suppliers vary and product quality control must be diligent.	10-20+ years	$65,000 - $80,000
Virgin Rubber Infill					
TPE	Thermoplastic Elastomer. Has Plastic and Elastic qualities. Extruded and cut pieces, making rounded pellet shape.	Good shock absorption. Raw materials means no product variations/unknowns. Can be recycled/melted down to re-use.	Suppliers vary and product quality control must be diligent. Low quality versions have been known to melt in field. Consistent shape/size so doesn't compact the way athletes want. Rounded shape may cause less traction on hard surfaces out of field. High cost.	20+ years	$310,000 - $320,000
Natural Infill					
Wood Chip (Brockfill)	This product is an engineered wood particle infill specifically designed for artificial turf. It's made here in the USA from a species of southern pine that is grown, harvested, and replanted in continuous cycles, making it ideal for a sustainable, renewable organic infill product.	No chemicals added, all natural. Successful in cool damp environments. Similar athlete play to natural grass.	Holds water, but during dry periods will need irrigation. Low shock absorption. Often times leaves a brown dust on the field due to breakdown. Compacts often, requires more maintenance than other infills. Product is sold to limited Turf Vendors. Material tends to float/migrate. Needs topping off of new material every season. High Cost.	20+ years	$80,000-$115,000
Coconut (Greenplay, Geofill, etc.)	Depending on product can be Coconut Husks, Cork, or Walnut shells. Shape varies from angular to shredded depending on product.	No chemicals added, all natural. Successful in cool damp environments. Similar athlete play to natural grass.	Holds water, but during dry periods will need irrigation. Low shock absorption. Often times leaves a brown dust on the field due to breakdown. Compacts often, requires more maintenance than other infills. Product is sold to limited Turf Vendors. Material tends to float/migrate. Needs topping off of new material every season. High Cost.	8-10 years (requires seasonal supplemental material added)	$150,000 - $210,000
Walnut Shell Blend	This is an organic blend of Black and English Walnut shells, one of the hardest nut shells on the planet. The shells are processed and follow FDA guidelines to remove the allergens that remain on the shells.	Temperature, Durability, Performance	Needs periodic topping off on new material	8-10 years	$110,000 - $125,000
Acrylic Coated Sand	Round quartz coated with colored acrylic.	Not as frequent infill migration, less need for topping off with added material. Antibacterial protection infused into manufacturing process to prevent bacteria and deterioration. Firm and fast, but doesn't compact.	Cost, Temperature	16+ years	$150,000 - $205,000

Assumptions / Comments / Potential Alternates:

1 Prices on infill are an average and may not reflect actual costs which vary depending on turf provider.
2 Assumes 50/50 Sand and Rubber infill ratio; For the purpose of this study 3.5 lbs sand/3.5lbs rubber(or alternate infill) was used. Depending on infill selection more or less will be used.
3 Product varies and limited turf manufacturers due to select companies only sell to select manufacturers.
4 All products require a rounded silica sand in the mix.
5 Assumes a field that is 85,000 s.f.

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Appendix:

F. Stantec Synthetic Turf Field Projects

Stantec Designed Synthetic Turf Soccer Field Projects

6/14/2019

NAME	LOCATION	TURF TYPE	INFILL	SHOCK PAD	INSTALLER	YEAR INSTALLED	CLIENT CONTACT	NOTES
Battery Park	NYC	Parallel Slit Film - UV Resistant Polyethylene	(1 to 1 to 1) Coconut to Peat to Silica	Brock Power Base	Astroturf	2008 and 2013	Joseph Ganci 212-417-4303	Battery Park City Authority: Installed coconut for first install. Flooded in Hurricane Sandy, installed TPE and Sand for second install.
Belmont Hill School - Soccer/Lax Field	Belmont, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(1 to 1) SBR to Silica	Brock YSR	Shaw	2019	Jay Bounty 617-933-5313	Private School
Belmont Hill School - Varsity Field	Belmont, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(1 to 1) SBR to Silica	Brock YSR	Astroturf	2014	Jay Bounty 617-933-5313	Private School
Brien McMahon Fields	Norwalk, CT	Monofilament - UV Resistant Ridged Polyethylene	(1 to 1) SBR to Silica	N/A	FieldTurf	2018	Ken Hughes 203-854-7725	Public Park: Multi-use field, including baseball
City Park - Fosina Field	New Rochelle, NY	Monofilament - UV Resistant Polyethylene	(1 to 1) SBR to Silica	N/A	Astroturf	2009 - 2014 Phased Project	William Zimmermann 914-654-2092	Public Park: natural grass football field converted to synthetic turf field
City Park - Multi Use Field	New Rochelle, NY	Monofilament - UV Resistant Polyethylene	(1 to 1) SBR to Silica	N/A	Astroturf	2009 - 2014 Phased Project	William Zimmermann 914-654-2092	Public Park: flooding conditions of natural grass field replaced with new synthetic turf construction including underground water storage (baseball & soccer field)
City Park - Skidelsky Field	New Rochelle, NY	Monofilament - UV Resistant Polyethylene	(1 to 1) SBR to Silica	N/A	Field Turf	2009 - 2014 Phased Project	William Zimmermann 914-654-2092	Public Park: flooding conditions of natural grass field replaced with new synthetic turf construction including underground water storage (soccer field)
Fessenden School	Newton, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(70 to 30) Silica to Coconut	Brock YSR	SprintTurf	2015	Mike Grossman 617-928-8863	Private School
Harding High School	Bridgeport, CT	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(70 to 30) Silica to SBR	Brock Power Base	Field Turf	2018	David C. Ferris 203-377-1300	High School (football & soccer field)
Harvard University	Boston, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(1 to 1) SBR to Silica	Brock Power Base	FieldTurf	2015	Tim Troville 617-384-8426	Collegiate (football & soccer field)
Haverhill High School	Haverhill, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(70 to 30) SBR to Silica	Brock YSR	FieldTurf	2019	Tom O'Brien 978-374-5732	High School
Malden High School	Malden, MA	Long Slit Film Parallel - UV Resistant Polyethylene	(1 to 1) SBR to Silica	N/A	SprintTurf	2012	Contact has left	High School
Roosevelt Elementary School	Bridgeport, CT	Monofilament - UV Resistant Ridged Polyethylene	(1 to 1) SBR to Silica	N/A	Field Turf	2015	David C. Ferris 203-377-1300	Elementary School (soccer field)
Town of Wilmington	Wilmington, MA	Dual Fiber Monofilament & Parallel Slit Film - UV Resistant	(70 to 30) SBR to Silica	Brock YSR	SprinTurf	2016	Paul Alunni 978-658-4499	High School
West Thames	NYC	Slit Film - UV Stable Tufted Polyethylene	(70 to 30) Silica to TPE	Brock YSR	Astroturf	2010	Joseph Ganci 212-417-4303	Battery Park City Authority

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Appendix:

G. Geotech Log Report

Phone
(203) 262-9328

Telefax
(203) 264-3414

WHITE PLAINS, N.Y.
(914) 946-4850

SOILTESTING, INC.

90 DONOVAN ROAD - OXFORD, CONN. 06478-1028

GEOTECHNICAL / ENVIRONMENTAL SUBSURFACE INVESTIGATIONS - Test Borings - Core Drilling
Monitoring Wells - Recovery Wells - Direct Push/Probe Sampling
UNDERPINNING - HELICAL PILES - SOIL NAILS

April 24, 2019

Stantec Consulting Services, Inc.
55 Church Street, Suite 601
New Haven, CT 06510
203-495-1645

Attn: Jennifer Waldron

Re: Nursery Field
Disbrow Park
Rye, NY

G60-1240-19

Dear Ms. Waldron

Enclosed are boring logs and location plan for the above referenced project site.

If you have any questions, please do not hesitate to contact us.

Very truly yours,
SOILTESTING, INC.

James A. DeAngelis
President

JAD:cl

SOILTESTING, INC.

TO: Stantec Consulting Services, Inc.
ADDRESS: 55 Church Street, Suite 601, New Haven, CT 06510
DATE: April 24, 2019
SITE LOCATION: Nursery Field, Disbrow Park, Rye, NY
REPORT SENT TO: Jennifer Waldron
SAMPLES SENT TO: Storage (Max. 60 days)

90 DONOVAN ROAD
Oxford, Connecticut 06478
(203) 262-9328

Branch Office:
White Plains, New York 10607
914-946-4850

JOB NO.
G60-1240-19

SOILTESTING, INC. 90 DONOVAN RD. OXFORD, CT 06478 CT (203) 262-9328 NY (914) 946-4850	CLIENT: Stantec Consulting Services	SHEET 1 OF 1 HOLE NO. B-1 & B-2
	PROJECT NO. G60-1240-19	
	PROJECT NAME Nursery Field	BORING LOCATIONS per Plan
FOREMAN - DRILLER MK/ao	LOCATION Disbrow Park, Rye, NY	
INSPECTOR	GeoProbe	OFFSET
GROUND WATER OBSERVATIONS AT 6' FT AFTER 0 HOURS AT __FT AFTER __HOURS	SAMPLER MC / I.D. 1.25	DATE START 4/16/19 DATE FINISH 4/16/19 SURFACE ELEV. GROUND WATER ELEV.

DEPTH	CASING BLOWS PER FOOT	SAMPLE NO	Type	PEN	REC.	DEPTH @ BOT	BLOWS PER 6 IN ON SAMPLER (FORCE ON TUBE) 0 - 6	6 - 12	12- 18	CORE TIME PER FT (MIN)	DENSITY OR CONSIST / MOIST	STRATA CHANGE DEPTH / ELEV	FIELD IDENTIFICATION OF SOIL REMARKS INCL. COLOR, LOSS OF WASH WATER, SEAMS IN ROCK, ETC.
												2'6"	6" Topsoil Brn FM SAND & FC GRAVEL, tr silt
5		1	mc	60"	40"	5'0"					dry	5'6"	Grey F SAND & SILT, tr F gravel Blk FMC SAND, sm FC gravel, tr silt
		2	mc	36"	30"	8'0"					wet	8'0"	Grey F SAND, lit silt Brn FMC SAND, tr F gravel
10													E.O.B 8'0"
15													

GROUND WATER OBSERVATIONS
AT 6' FT AFTER 0 HOURS
AT __FT AFTER __HOURS

DEPTH	CASING BLOWS PER FOOT	SAMPLE NO	Type	PEN	REC.	DEPTH @ BOT	BLOWS PER 6 IN ON SAMPLER (FORCE ON TUBE) 0 - 6	6 - 12	12- 18	CORE TIME PER FT (MIN)	DENSITY OR CONSIST / MOIST	STRATA CHANGE DEPTH / ELEV	FIELD IDENTIFICATION OF SOIL REMARKS INCL. COLOR, LOSS OF WASH WATER, SEAMS IN ROCK, ETC.
0	B-2												B-2
												2'6"	4" Topsoil Brn FM SAND, sm FC gravel, tr cobbles
												3'0"	Blk FMC SAND, sm FC gravel
												3'6"	Brn organic SILT
5		1	mc	60"	50"	5'0"					dry		Grey F SAND & SILT Grey F SAND, lit silt
		2	mc	36"	30"	8'0"					wet	8'0"	Brn F SAND, tr F gravel, tr silt
10													E.O.B 8'0"
15													
20													

NOTE: Subsoil conditions revealed by this investigation represent conditions at specific locations and may not represent conditions at other locations or times.

GROUND SURFACE TO _____FT. USED _____CASING THEN _____ CASING TO _____FT. **HOLE NO. B-1 & B-2**

A = AUGER UP = UNDISTURBED PISTON T = THINWALL V = VANE TEST

WOR = WEIGHT OF RODS WOH = WEIGHT OF HAMMER & RODS

SS = SPLIT TUBE SAMPLER H.S.A. = HOLLOW STEM AUGER

PROPORTIONS USED: TRACE = 0 - 10% LITTLE = 10 - 20% SOME = 20 - 35% AND =35 - 50%

C = COARSE
M = MEDIUM
F = FINE

SOILTESTING, INC. 90 DONOVAN RD. OXFORD, CT 06478 CT (203) 262-9328 NY (914) 946-4850	CLIENT: Stantec Consulting Services	SHEET 1 OF 1 HOLE NO. B-3 & B-4
	PROJECT NO. G60-1240-19	
	PROJECT NAME Nursery Field	BORING LOCATIONS per Plan
FOREMAN - DRILLER MK/ao	LOCATION Disbrow Park, Rye, NY	
INSPECTOR	GeoProbe	OFFSET
GROUND WATER OBSERVATIONS AT 3' FT AFTER 0 HOURS AT __FT AFTER __HOURS	SAMPLER MC / I.D. 1.25	DATE START 4/16/19 DATE FINISH 4/16/19 SURFACE ELEV. GROUND WATER ELEV.

DEPTH	CASING BLOWS PER FOOT	SAMPLE NO	Type	PEN	REC.	DEPTH @ BOT	BLOWS PER 6 IN ON SAMPLER (FORCE ON TUBE) 0 - 6	6 - 12	12- 18	CORE TIME PER FT (MIN)	DENSITY OR CONSIST / MOIST	STRATA CHANGE DEPTH / ELEV	FIELD IDENTIFICATION OF SOIL REMARKS INCL. COLOR, LOSS OF WASH WATER, SEAMS IN ROCK, ETC.
											dry		6" Topsoil
											wet	2'6"	Brn FM SAND & FC GRAVEL
		1	mc	48"	32"	4'0"					wet		GreyBrn F SAND, sm silt
5												5'0"	Grey FMC SAND, sm FC gravel
													Grey F SAND, sm silt
											wet		
		2	mc	48"	36"	8'0"					dry	8'0"	GreyBrn F SAND & SILT
10													E.O.B 8'0"
15													

GROUND WATER OBSERVATIONS
AT 6' FT AFTER 0 HOURS
AT __FT AFTER __HOURS

DEPTH	CASING BLOWS PER FOOT	NO	Type	PEN	REC.	DEPTH @ BOT	0 - 6	6 - 12	12- 18	CORE TIME PER FT (MIN)	DENSITY OR CONSIST / MOIST	STRATA CHANGE DEPTH / ELEV	FIELD IDENTIFICATION OF SOIL REMARKS INCL. COLOR, LOSS OF WASH WATER, SEAMS IN ROCK, ETC.
0	B-4												B-4
													4" Topsoil
													Brn FMC SAND & FC GRAVEL
		1	mc	48"	20"	4'0"					dry	4'0"	
5													
											moist		GreyBrn F SAND & SILT
		2	mc	48"	36"	8'0"					wet	8'0"	
10													E.O.B 8'0"
15													
20													

NOTE: Subsoil conditions revealed by this investigation represent conditions at specific locations and may not represent conditions at other locations or times.

GROUND SURFACE TO _____FT. USED _____CASING THEN _____ CASING TO _____FT. **HOLE NO. B-3 & B-4**

A = AUGER UP = UNDISTURBED PISTON T = THINWALL V = VANE TEST

WOR = WEIGHT OF RODS WOH = WEIGHT OF HAMMER & RODS

SS = SPLIT TUBE SAMPLER H.S.A. = HOLLOW STEM AUGER

PROPORTIONS USED: TRACE = 0 - 10% LITTLE = 10 - 20% SOME = 20 - 35% AND =35 - 50%

C = COARSE
M = MEDIUM
F = FINE

SOILTESTING, INC. 90 DONOVAN RD. OXFORD, CT 06478 CT (203) 262-9328 NY (914) 946-4850	CLIENT: Stantec Consulting Services	SHEET 1 OF 1 HOLE NO. B-5, B-6, B-7
	PROJECT NO. G60-1240-19	
	PROJECT NAME Nursery Field	BORING LOCATIONS per Plan
FOREMAN - DRILLER MK/ao	LOCATION Disbrow Park, Rye, NY	
INSPECTOR	GeoProbe	OFFSET
GROUND WATER OBSERVATIONS AT none FT AFTER 0 HOURS AT__FT AFTER__HOURS	SAMPLER MC / I.D. 1.25	DATE START 4/16/19 DATE FINISH 4/16/19 SURFACE ELEV. GROUND WATER ELEV.

DEPTH	CASING BLOWS PER FOOT	SAMPLE NO	Type	PEN	REC.	DEPTH @ BOT	BLOWS PER 6 IN ON SAMPLER (FORCE ON TUBE) 0 - 6	6 - 12	12- 18	CORE TIME PER FT (MIN)	DENSITY OR CONSIST / MOIST	STRATA CHANGE DEPTH / ELEV	FIELD IDENTIFICATION OF SOIL REMARKS INCL. COLOR, LOSS OF WASH WATER, SEAMS IN ROCK, ETC.
	B-5											1'0"	Topsoil
													Brn FMC SAND & FC GRAVEL, lit cobbles
		1	mc	48"	36"	4'0"					dry		SAME
5													
		2	mc	24"	5"	6'0"					dry	6'0"	Auger refusal
													E.O.B 6'0"
0	**B-6**												**B-6**
												1'0"	Topsoil
													Brn FMC SAND & FC GRAVEL
													Blk FMC SAND, sm FC gravel, tr silt (glass)
		1	mc	48"	32"	4'0"					dry	4'0"	Brn FMC SAND & FC GRAVEL, tr cobbles
5													
													Brn F SAND
													SAME
													cobbles @ 6'0"
		2	mc	48"	28"	8'0"					wet	8'0"	Brn FMC SAND, lit FC gravel
10													
													E.O.B 8'0"
0													
	B-7												**B-7**
													4" Topsoil
		1	mc	48"	38"	4'0"					moist		Blk FM SAND, tr silt
5													
		2	mc	24"	8"	6'0"					moist	6'0"	
													E.O.B 6'0"
10													

GROUND WATER OBSERVATIONS
AT 7' FT AFTER 0 HOURS
AT__FT AFTER__HOURS

GROUND WATER OBSERVATIONS
AT none FT AFTER 0 HOURS
AT__FT AFTER__HOURS

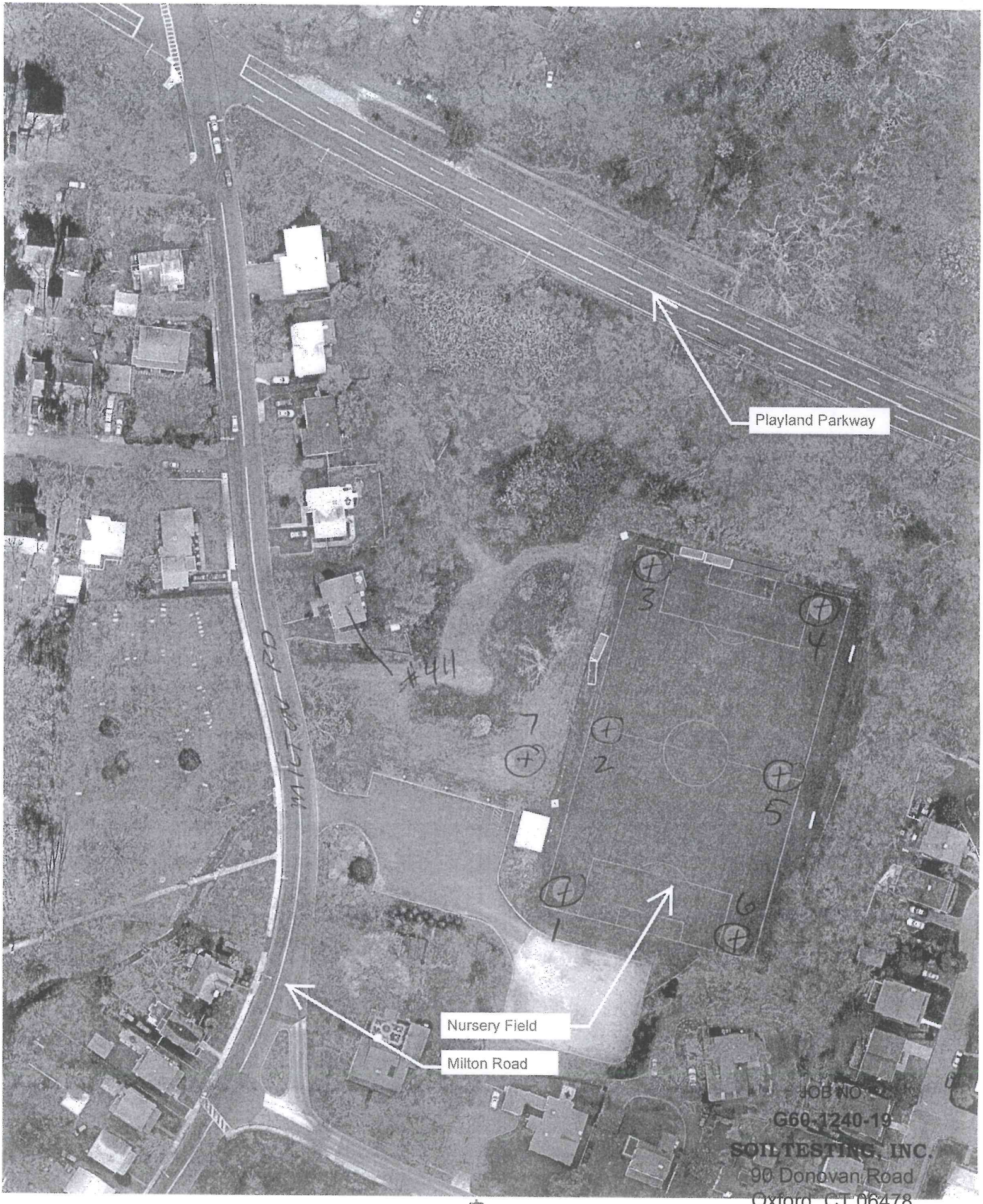
NOTE: Subsoil conditions revealed by this investigation represent conditions at specific locations and may not represent conditions at other locations or times.

GROUND SURFACE TO _____FT. USED _____CASING THEN _____ CASING TO _____FT.

HOLE NO. B-5, B-6, B-7

A = AUGER UP = UNDISTURBED PISTON T = THINWALL V = VANE TEST
WOR = WEIGHT OF RODS WOH = WEIGHT OF HAMMER & RODS
SS = SPLIT TUBE SAMPLER H.S.A. = HOLLOW STEM AUGER
PROPORTIONS USED: TRACE = 0 - 10% LITTLE = 10 - 20% SOME = 20 - 35% AND =35 - 50%

C = COARSE
M = MEDIUM
F = FINE



JOB NO.
G60-1240-19
SOILTESTING, INC.
90 Donovan Road
Oxford, CT 06478

4-15-19

Stantec

Appendix:

H. Soil Study Testing

Turf & Soil Diagnostics

ACCREDITED TESTING Cert #0797.02

MATERIALS TEST REPORT FOR
Conceptual Turf Plan - Nursery Field

REPORT TO: Stantec
Jennifer Waldron
55 Church Street
New Haven, CT 06510

DATE RECEIVED: Apr-15-2019
REPORT DATE: Apr-23-2019
CONDITION OF SAMPLE: Normal

PARTICLE SIZE (ASTM F1632)

		Gravel %			Soil Separate* %			Sieve Size / Sand Fraction Sand Particle Diameter % Retained				
Lab ID#	**Sample Name**	**1/4" 6.3 mm**	**No. 5 4.0 mm**	**No. 10 2.0 mm**	**Sand**	**Silt**	**Clay**	**No. 18 V. Coarse 1.0 mm**	**No. 35 Coarse 0.50 mm**	**No. 60 Medium 0.25 mm**	**No. 140 Fine 0.10 mm**	**No. 270 V. Fine 0.05 mm**
44739-1	#1	14.6	2.9	4.6	62.5	26.8	10.7	7.0	11.9	19.6	16.6	7.3
44739-2	#2	20.3	0.8	5.3	56.4	33.4	10.2	7.1	10.9	16.1	15.0	7.3
44739-3	#3	18.4	0.8	4.4	60.2	29.7	10.0	6.6	10.8	18.5	16.6	7.6

pH / ORGANIC MATTER / TEXTURAL CLASS

Lab ID#	Sample Name						pH[1] 1:1	% Organic Matter[2] Dry Weight	Textural Class
44739-1	#1						6.4	5.58	Gravelly Sandy Loam
44739-2	#2						6.7	4.04	Gravelly Sandy Loam
44739-3	#3						6.4	5.42	Gravelly Sandy Loam

*ASTM F1632 Method B
[1]ASTM D4972, method A, $CaCl_2$, 25 g sample used
[2] ASTM F1647 Method A

Data reported using USDA definitions of soil classification
Samples were tested as received and comments pertain only to the samples shown.
This report may not be reproduced except in full without written permission from Turf & Soil Diagnostics, Inc.
Samples were received with a transmittal letter.

Reviewed by Duane K Otto — Duane K Otto 2019-04-23 15:39:53

35 King Street, Trumansburg NY 14886 ■ Phone: 855-769-4231
E-mail: lab@turfdiag.com ■ Website: http://www.turfdiag.com

Turf & Soil Diagnostics

ACCREDITED TESTING
Cert #0797.02

MATERIALS TEST REPORT FOR
Conceptual Turf Plan - Nursery Field

REPORT TO: Stantec
Jennifer Waldron
55 Church Street
New Haven, CT 06510

DATE RECEIVED: Apr-15-2019
DATE REPORTED: Apr-23-2019
CONDITION OF SAMPLE: Normal

PHYSICAL PROPERITES*

Lab ID#	Sample Name	Particle Density** g/cc	Infiltration Rate in/hr	Bulk Density g/cc	Total Pore Space %
44739-1	#1	2.58	0.1	1.28	50.2
44739-2	#2	2.63	0.1	1.49	43.3
44739-3	#3	2.59	0.1	1.31	49.4

	#1			#2			#3		
Tension (cm)	Capillary Porosity	Non-Capillary Porosity	Degree of Saturation	Capillary Porosity	Non-Capillary Porosity	Degree of Saturation	Capillary Porosity	Non-Capillary Porosity	Degree of Saturation
	%	%	%	%	%	%	%	%	%
0	50.2	0.0	100.0	43.3	0.0	100.0	49.4	0.0	100.0
30	48.9	1.3	97.3	41.8	1.5	96.4	46.1	3.3	93.3
40	48.0	2.2	95.6	41.3	2.1	95.2	45.1	4.4	91.2
60	44.9	5.3	89.4	37.7	5.7	86.9	41.3	8.2	83.5

*ASTM F1815 with water release
**ASTM D5550

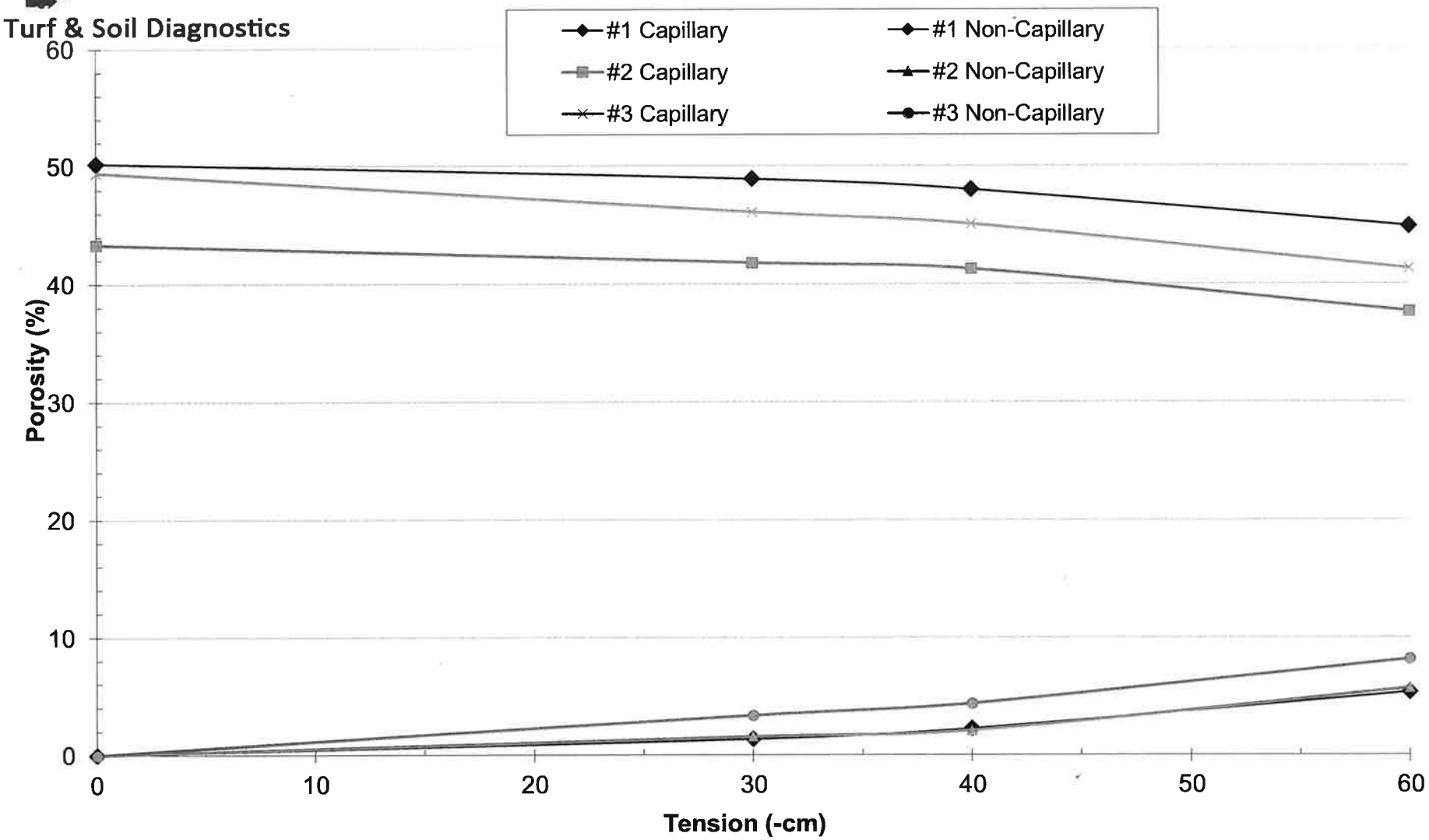
Samples were tested as received and comments pertain only to the samples shown.
This report may not be reproduced in part, but only in full.
Samples were received with a transmittal letter.

Reviewed by Duane K Otto Duane K Otto 2019-04-23 15:40:10

35 King St Trumansburg, NY 14886 ■ Phone: 855-769-4231
E-mail: lab@turfdiag.com ■ Website: http://www.turfdiag.com

Turf & Soil Diagnostics

Conceptual Turf Plan - Nursery Field
Water Release Curve
File #44739



35 King St. Trumansburg, NY 14886 ■ Phone: 855-769-4231
E-mail: lab@turfdiag.com ■ Website: http://www.turfdiag.com

Turf & Soil Diagnostics

April 23, 2019

Stantec
Conceptual Turf Plan – Nursery Field
TSD File #44739

Enclosed are the laboratory results of the three soil samples, which were tested as received. No specifications were provided for this project.

The results of the three samples are somewhat similar to each other. Each sample is classified as gravelly sandy loam per the USDA textural classification scheme. There is significant amount of gravel larger than ¼" in these samples. Sandy loam soils provide acceptable water and nutrient holding for most turf and landscapes.

The performance testing indicates that the soils have very low saturated hydraulic conductivities (infiltration rates). These results indicate these soils provide limited internal drainage and can be prone to excess compaction under heavy use. Areas should be crowned or properly slope to facilitate adequate surface drainage.

To further evaluate performance, the samples were subjected to water release characterization testing. Due to increasing hydraulic head or pressure, the greater the tension the greater the aeration porosity and the lower the capillary porosity. In this test, increasing levels of energy (increasing tension) are applied to the sample to extract water. The data from this evaluation provides information on soil performance at varying tensions.

The water release curve plots the aeration and capillary porosity values at different tensions. Aeration porosity is made up of relatively large pores that conduct water under saturated conditions. When drained, they are filled with air providing the oxygen necessary for root growth. Capillary porosity is made up of small pores that hold water against the force of gravity, retaining much of it for plant use. Ideally, a root zone mix would contain a nearly equal distribution of air and water filled pore space after free drainage.

The water release results indicate these soils slowly release water through the tested range of tensions. These results suggest these soils should gradually release water and may be prone to excessive water retention.

Please let us know if you have any questions or are in need of further assistance. Samples are generally kept on the premises for 45 days after report date. Thank you for using Turf & Soil Diagnostics, Inc.

Sincerely,

Duane K Otto Duane K Otto 2019-04-23 15:40:49

Duane K. Otto
Vice President

35 King Street, Trumansburg, NY 14886
Phone: 855-769-4231 Email: lab@turfdiag.com Web: www.turfdiag.com

Appendix:

I. Health Studies and the Environment

Synthetic Turf Fields, Crumb Rubber, and Concerns about Cancer

Archie Bleyer, MD*

In addition to a significant number of prior studies in the U.S. and Europe that do not identify any reason for concern around playing on synthetic turf fields with recycled rubber infill, there are three other reasons to be reassured that synthetic turf does not cause cancer.

1) While Chair of the world's largest pediatric cancer research organization during the 1990s, I was responsible for millions of dollars of research on what caused cancer in children, adolescents and young adults. **None of the studies that we conducted, nationally and in multistate surveys, within homes and with environmental sampling, of childhood and prenatal exposures, and of many other variables, showed evidence that an environmental factor caused these cancers. The cancers we studied included the lymphomas implicated in the crumb rubber controversy**

For middle and later adulthood, we know that cancer can be caused by cumulative exposure over many years to carcinogens like tobacco, radiation, asbestos, ultraviolet ray (sun and tanning machines) and alcohol. For cancer in youth, however, none of our extensive research efforts could "*identify environmental exposures that might explain more than a small fraction of the observed cases*".[1,2] We concluded that virtually all cancer in the young is a mistake of nature—*spontaneous mutation to malignancy* is the biologic term—and not due to an exogenous, potentially preventable cause. Since our studies, no valid scientifically-conducted research has been published that has uncovered external causes of cancer in children, adolescents or young adults.

2) **It's human nature to blame.** When unfortunate events occur for which there is no known cause, we want to assume that there has to be a reason that hopefully can lead to prevention. Blaming autism on vaccines is a recurrent example, and one that illustrates another human behavior: refusal to believe objective, scientific, irrefutable evidence.[3] This human need and attendant denial causes unnecessary alarms, especially when cancer is the event. The notion that synthetic turf fields cause cancer in the young is another example of need to attribute blame.

3) **The cancers that have been reported to occur in soccer players are precisely those cancers that are expected to occur in the age group that's being discussed.** Moreover, they are consistent with the race/ethnicity and socioeconomic status of those who have access to synthetic fields. When these factors are taken into consideration (click here or paste the URL[4] to review), the incidence of the implicated cancers in no higher than in those who do not have access to synthetic turf fields.

4) Regular physical activity has been clearly demonstrated to prevent cancer. Not participating in physical activity increases the risk of cancer and hence lack or removal of facilities that allow exercise increase the incidence of cancer.

In conclusion, we naturally have a need to find something to blame but it's not the crumb rubber or anything else in synthetic turf that caused the cancers. On the contrary, physical activity should be encouraged and promoted by year-round, weather-resistant fields to help prevent cancer and other chronic diseases. Limiting field development could in the long run actually increase cancer incidence.

Archie Bleyer, MD
Pediatric and Young Adult Oncologist

**Dr. Bleyer is Clinical Research Professor in Radiation Medicine at the Oregon Health and Science University, and founding member of the Critical Mass Young Adult Cancer Alliance and founder of DEFEATcancer.[5] Dr. Bleyer chaired the Children's Cancer Group for 10 years, then the world's largest pediatric cancer research organization, the Department and Division of Pediatrics at the University of Texas MD Anderson Cancer Center, and Community Oncology in the Department and Division of Medicine at the M.D. Anderson Cancer Center. At the University of Washington School of Medicine, he was the American Cancer Society Professor of Clinical Oncology and in charge of the cancer curriculum. Dr. Bleyer has been awarded research grants totaling more than $75 million as a principal investigator from the National Institutes of Health, the American Cancer Society, and the Leukemia Society of America. His research has been published in more than 300 peer-reviewed articles, chapters, and books. His current personal clinical research is dedicated to adolescents and young adult (AYA) oncology.*

---

[1] Buckley JD, Buckley CM, Breslow NE, et al. *Med Pediat Oncol* 26:223,1996.
[2] Olsen JH, Boice JD Jr, Seersholm N, Bautz A, Fraumeni JF. *N Engl J Med.* 333(24):1594-9,1995.
[3] The most recent study, performed because there are still doubters, by the University of Washington's Center on Human Development and Disability, the University of Texas Southwestern; and the Texas A&M Health Science Center & Central Texas Veterans Health Care System, documents no evidence for adverse behavioral effects in infant monkeys
administered the suspect vaccine (http://hsnewsbeat.uw.edu/sites/default/files/sites/default/file s/documents/PNAS-2015-Gadad-1500968112.pdf).
[4] http://comedsoc.org/index.php?m=47&s=486
[5] http://www.stcharleshealthcare.org/Our-Services/Cancer-Care/DEFEATCancer

STATE OF CONNECTICUT
DEPARTMENT OF PUBLIC HEALTH

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EHS Circular Letter #2015-02
(Follow up to Circular Letter #2014-26a)

DATE: January 20, 2015

TO: Local Health Departments and Districts

FROM: Brian Toal, Gary Ginsberg
Environmental and Occupational Health Assessment

RE: Recent News Concerning Artificial Turf Fields

Brief Video Clip for Local Health Departments – *Click Here* →

This letter and video clip are being sent to update you regarding the news story that has circulated since last spring regarding potential cancer risks at artificial turf fields. Various media outlets have continued to run this story and a number of local health departments have inquired as to its validity. Since many Connecticut towns have installed or are considering artificial turf fields an elevated cancer risk would be an important consideration. However, this news story is still based upon very preliminary information and does not change CTDPH's position that outdoor artificial turf fields do not represent an elevated health risk.

The Connecticut Department of Public Health has evaluated the potential exposures and risks from athletic use of artificial turf fields. Our study of 5 fields in Connecticut in 2010-2011 was a comprehensive investigation of releases from the fields during active play. This study was conducted as a joint project with the CT DEEP and the University of CT Health Center and was peer-reviewed by the Connecticut Academy of Science and Engineering. Our study did not find a large amount of vapor or particle release from the fields confirming prior reports from Europe and the US. We put these exposures into a public health context by performing a risk assessment. Our risk assessment did not find elevated cancer risk. These results have been published as a set of 3 articles in a peer review journal and are available on the DPH artificial turf webpage (http://www.ct.gov/dph/cwp/view.asp?a=3140&q=464068).

The news story suggests soccer players and especially goalies may have an elevated cancer risk from playing on artificial turf fields. This is based upon anecdotal observations of a university soccer coach (http://www.komonews.com/news/local/Soccer-coach-Could-field-turf-be-causing-cancer-259895701.html). Reportedly the coach is developing a list of soccer players who have contracted cancer. However, the types of cancer are undocumented and so it is impossible to say whether they

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represent a common effect and there has been no reporting on how long the goalies played on artificial turf fields to see if there was plausible exposure and latency. There are many reasons why someone collecting a list of cancer cases may appear to find a cluster including the fact that when you have a single-minded focus on finding cases you do not capture all the non-cases that would tend to disprove the cluster. Documentation of an increased rate in soccer players would require an epidemiological study in which the total number who play on turf fields in a given region was also known so that a cancer rate could be established and compared to those that do not play on artificial turf fields. The current news report does not constitute epidemiological evidence and thus is very preliminary.

Our risk assessment did cover carcinogens that are known to be in recycled tires and the crumb rubber used to cushion fields. Once again, we found there to be very little exposure of any substances, carcinogenic or not, in the vapors and dust that these fields generate under active use, summer conditions. Background levels of chemicals in urban and suburban air from heating sources and automobile traffic are much more significant sources of airborne carcinogens. The fact that we sampled 5 fields (4 outdoor and 1 indoor) of different ages and composition suggests that the results can be generalized to other fields, a conclusion supported by the fact that results were similar to what was found in California, USEPA and European studies. Our study did not evaluate ingestion of the crumb rubber itself as players are unlikely to ingest an entire rubber pellet. However, two studies, one in California and one at Rutgers University did evaluate the cancer risk if children ingested a mouthable chunk of playground rubber (10 gram), using laboratory extraction methods to estimate the amount of chemicals that might become available in the stomach and absorbed into the body. Both studies found very low cancer risk from this scenario (Cal OEHHA 2007; Pavilonis et al. 2014). Thus, CT DPH finds no scientific support for a finding of elevated cancer risk from inhalation or ingestion of chemicals derived from recycled tires used on artificial turf fields. US EPA has a similar position: "At this point, EPA does not believe that the field monitoring data collected provides evidence of an elevated health risk resulting from the use of recycled tire crumb in playgrounds or in synthetic turf athletic fields." (http://www.epa.gov/epawaste/conserve/materials/tires/health.htm)

In summary, federal and state authorities have taken seriously the concerns that artificial turf fields may present a health risk due to contaminants in recycled rubber. The best way to investigate these concerns is via an exposure investigation. Studies conducted in Connecticut and elsewhere have shown a very low exposure potential, less than from typical outdoor sources of air pollution. The current news reports of a list of soccer players with cancer does not constitute a correlation or causality and thus raises a concern that currently lacks scientific support. Thus, the CT DPH position expressed in 2011 at the conclusion of the Connecticut study, that outdoor artificial turf fields do not represent an elevated health risk, remains unchanged. For further information please contact Brian Toal or Gary Ginsberg at 860-509-7740.

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March 23, 2015

Stephanie Bacon, Health Agent
Office of Board of Health
155 Village Street
Medway, MA 02053

Dear Ms. Bacon:

Thank you for your letter of February 24, 2015, in which you requested that the Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH), evaluate health concerns related to the use of crumb rubber infill material for artificial turf fields in Medway, Massachusetts. As you are likely aware, our office had previously evaluated this issue in a series of letters to the Town of Needham Board of Health in 2008, 2011, and 2013.

In response, MDPH/BEH staff have evaluated more recent information on potential exposure opportunities to artificial turf components, including crumb rubber infill, and evaluated health concerns, including cancer, in relation to exposure to such turf. Recent media reports on soccer players, particularly goalies that have played on artificial turf, and the incidence of some cancers have been expressed. These reports raised concerns about the possible association between playing on crumb rubber fields and the development of cancers, notably, non-Hodgkin's lymphoma, Hodgkin Lymphoma, and osteosarcoma. We also evaluated information you provided on the content of the specific products used in Medway. Our review is summarized below.

Updated Literature Review

Our previous evaluations noted that crumb rubber infill has been found to contain chemicals, including polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), and metals. We further stated that although these chemicals are in the material itself, information available at that time did not suggest significant exposure opportunities to the chemicals in the materials such that we would expect health effects. We noted that the most relevant study on this topic at the time was a study conducted by the California Office of Environmental Health Hazard Assessment (CA OEHHA).

Since that time, the CA OEHHA conducted additional evaluations of chemical concentrations in air above crumb rubber turf fields under active use (CA OEHHA 2010). Air samples were taken above fields and analyzed for VOCs and metals. Results suggested that adverse health effects were unlikely to occur from inhalation of VOCs or metals in particulates above these fields. To assess the potential for skin infections due to bacteria or to skin abrasions on these fields, tests for bacterial contamination were performed and the frequency of skin abrasions was assessed. Researchers found fewer bacteria detected on the artificial turf compared to natural turf, suggesting that the risk of infection to athletes using these fields was actually lower. However, more skin abrasions were observed in athletes using artificial turf fields than natural turf fields, and the study authors made various recommendations to help prevent skin abrasions (e.g., protective equipment or clothing) and prompt treatment of skin abrasions.

In another study, the state of Connecticut conducted air sampling at four outdoor artificial turf fields with crumb rubber infills (most relevant to Medway) under summer conditions (Simcox et al. 2011). Air measurements were taken using stationary air sampling monitoring devices as well as personal samplers (placed on people using the fields). They concluded that exposure opportunities to turf contaminants were not associated with elevated health risks and suggested that their findings were consistent with other studies available at the time. A letter prepared by the Connecticut Department of Public Health reiterates these conclusions (CTDPH 2015).

A 2014 study by researchers at the Rutgers Robert Wood Johnson Medical School in New Jersey evaluated opportunities for exposures to PAHs, semivolatile organic compounds (SVOCs), and heavy metals from exposures to artificial turf fibers and crumb rubber infills by measuring these constituents in simulated body fluids (digestive fluids, lung fluids, sweat) that represented different routes of exposure (ingestion, inhalation, dermal). This bioaccessibility study aimed to provide a better measure of the actual amount of these contaminants that might be absorbed into the body after exposure. The researchers found that PAHs were routinely below the limit of detection and SVOCs that have environmental regulatory limits to use for comparison were identified at levels too low to quantify. Some metals were detected but at concentrations at which health risks were low, with the exception of lead from the field sample collected. That sample indicated lead at levels in the simulated digestive fluids that the authors reported could result in blood lead levels above the current U.S. Centers for Disease Control and Prevention (CDC) reference value for blood lead in children (5 ug/dL). It should be noted that the lead concentration of the materials used in this study included a sample of turf fiber with a lead concentration of 4,400 mg/kg. This level contrasts with information on the Medway artificial turf components, which reportedly either contained lead at 39 mg/kg (crumb rubber infill) or had no lead (turf fibers) (see discussion later in this letter). Based on the lead result from this one field sample, the authors suggested that components of artificial turf fields should be certified for low or no lead content prior to use. Overall, however, the authors concluded that opportunities

for exposure to constituents in these fluids presented very low risk among all populations that would use artificial turf fields (Pavilonis et al. 2014).

A study conducted in 2010 in the Netherlands assessed the exposure of soccer players to PAHs after playing sports on a rubber crumb field. Urine testing in participants indicated that uptake of PAHs by the participants following exposure to artificial turf with rubber crumb infill was minimal. If there is any exposure, the authors reported, uptake is minimal and within the normal range of uptake of PAHs from environmental sources and/or diet observed in healthy individuals (van Rooij and Jongeneelen 2010).

It is probably worthwhile to also note that MDPH/BEH reviewed testing data for artificial turf for the Town of Needham, as reported in our letters of 2011 and 2013 to the Needham Board of Health. The Town of Needham contracted with an environmental testing firm to conduct environmental tests including, air measurements of volatile organic compounds taken in the laboratory and heavy metals (arsenic, cadmium, chromium, lead, mercury, selenium, zinc) content of crumb rubber materials. Our review and conclusions for that testing, did not indicate exposures of health concern.

Material in Medway

MDPH/BEH reviewed available information provided by the Medway Board of Health regarding the specific materials used in the Medway fields. These included the APT Gridiron turf system and Liberty Tire Recycling 10+20 BM Rubber Crumb Brantford, ON. Among the materials provided for these products were statements or test results for various constituents in these products.

APT submitted a written statement dated October 29, 2014, that reported that the APT Gridiron turf systems (essentially the grass fibers of the artificial turf) are manufactured and installed without the use of any lead or heavy metals. They reported that this included all materials used for the turf fibers and backings. No other documentation about this product, including any testing results, was provided to support this statement.

With respect to the 10+20 BM Crumb Rubber infill product, laboratory testing results were provided for this product, although it is not clear whether the testing was for the materials specifically used in turf applied in Medway. Testing was conducted for metals content as well as emissions of volatile organic compounds (VOCs). It appears that testing included the following: (1) testing for VOCs emitted into a confined air space in the laboratory after heating the product to 73 degrees F; and (2) content testing for eight heavy metals, including lead. The laboratory compared results to criteria established by the Greenguard certification program, part of Underwriters Laboratory, that uses among its criteria for certification health-based levels derived by the CA OEHHA.

Testing results for metals content of the product indicated a lead concentration of 39 mg/kg, which is less than the current Consumer Product Safety Improvement Act (CPSIA) limit of 100 mg/kg for lead in children's products (Ulirsch et al. 2010). No other metals were detected.

Test results measuring emissions off-gassing from heated material were provided in measurements that cannot be compared to any health-based standards or guidelines and thus, MDPH/BEH did not further evaluate this information. Typically, when certain products raise health concerns, health agencies review Material Safety Data Sheets (MSDS). An MSDS provides information on health risks associated with use of the product. An industry group, Synthetic Turf Council, provides a sample template MSDS for crumb rubber infill material (Synthetic Turf Council 2014). Although this sample MSDS is not specific to any particular product, it appears to be applicable to crumb rubber infill in general. In the section under "Hazardous Ingredients," the MSDS notes that the product can contain fine fibers that may cause irritation symptoms (e.g., itching, irritation of mucous membranes, eye irritation). The MSDS notes that the crumb rubber material is generally thought to be a nuisance dust.

Concerns About Cancer Among Soccer Players

As noted earlier in this letter, some recent news reports suggested that the incidence of cancers among soccer players, particularly goaltenders exposed to artificial turf, might be atypical. These reports included many cancer types, but some focused specifically on NHL, Hodgkin Lymphoma, and osteosarcoma in three individuals. We thought it would be helpful to provide additional information on cancers in general and known risk factors for NHL, Hodgkin Lymphoma, and osteosarcoma.

Cancer in General

Understanding that cancer is not one disease, but a group of diseases, is very important. Research has shown that there are more than 100 different types of cancer, each with separate causes, risk factors, characteristics and patterns of survival. A risk factor is anything that increases a person's chance of developing cancer and can include hereditary conditions, medical conditions or treatments, infections, lifestyle factors, or environmental exposures. Although risk factors can influence the development of cancer, most do not directly cause cancer. An individual's risk for developing cancer may change over time due to many factors and it is likely that multiple risk factors influence the development of most cancers. In addition, an individual's risk may depend on a complex interaction between their genetic make-up and exposure to environmental agents, including infectious agents and/or chemicals. This may explain why some individuals have a fairly low risk of developing a particular type of cancer as a result of an environmental exposure, while others are more vulnerable.

Cancers in general have long latency or development periods that can range from 10 to 30 years in adults, particularly for solid tumors. In some cases, the latency period may be more than 40 to 50 years. It is important to note, however, that latency periods for children and adolescents are significantly shorter than for adults.

Hodgkin Lymphoma

Hodgkin Lymphoma is most common in young adults between the ages of 15 and 40, especially in individuals in their 20s. Among adolescents, it is the most common type of cancer.

Hodgkin Lymphoma occurs specifically in a type of B lymphocyte (or white blood cell) called the Reed-Sternberg cell while other lymphomas (non-Hodgkin's types) occur in different cells.

Established risk factors for Hodgkin Lymphoma include: exposure to the Epstein-Barr virus (EBV); a previous diagnosis of mononucleosis (mono is caused by the EBV); family history; and certain hereditary conditions (such as ataxia telangiectasia) associated with a weakened immune system. The Epstein-Barr virus is very prevalent in the general population. Even though most of us have been exposed to the virus (which remains latent in our bodies), most people do not develop mononucleosis or Hodgkin Lymphoma. EBV is thought to account for about 20% or 25% of the diagnoses of classical Hodgkin's in the US.

Higher socioeconomic status is also a possible risk factor. This is thought to be due to delayed infectious exposures in childhood.

Occupational exposures as risk factors have been studied extensively and none have emerged as established risk factors. Likewise, there is very little evidence linking the risk of Hodgkin Lymphoma to an environmental exposure, other than the EBV.

Non-Hodgkin Lymphoma (NHL)

NHL refers to a diverse group of cancers that are characterized by an increase in malignant cells of the immune system. Each subtype of NHL may have different risk factors associated with its development. The specific cause of NHL in most individuals is unknown.

Although some types of NHL are among the more common childhood cancers, more than 95% of diagnoses occur in adults. Incidence generally increases with age, and most diagnoses occur in people in their 60s or older.

Established risk factors for NHL include a weakened immune system, associated with various medical conditions, and exposure to various viruses. An increased risk is faced by individuals taking immunosuppressant drugs following organ transplants; individuals with autoimmune disorders, such as rheumatoid arthritis and lupus; and individuals who have taken certain chemotherapy drugs for other cancers. Several viruses have been shown to play a role in the development of NHL, including the human immunodeficiency virus (HIV), the human T-cell leukemia/lymphoma virus (HTLV-1), and the Epstein-Barr virus.

Exposure to high-dose radiation (for example, by survivors of atomic bombs and nuclear reactor accidents and possibly by patients who have received radiation therapy for a previous cancer) may pose an increased risk. Some studies have also suggested that exposure to chemicals such as benzene and certain herbicides and insecticides may be linked with an increased risk of NHL. Smoking has been associated in some studies with certain types of NHL.

Osteosarcoma

Osteosarcoma is a type of malignant bone cancer which accounts for about 2% of childhood cancers in the United States. It is the most common type of cancer that develops in bone and comprises about 66% of malignant bone tumors in children in Massachusetts. Most osteosarcomas occur in children and young adults between the ages of 10 and 30. Teenagers comprise the most commonly affected age group and are at the highest risk during their growth spurt. However, osteosarcoma can occur in people of any age, with about 10% of all osteosarcomas occurring in people over the age of 60.

Established risk factors for osteosarcoma include certain inherited syndromes (such as retinoblastoma, the Li-Fraumeni syndrome, and others) and certain bone diseases (such as Paget disease of the bone and hereditary multiple osteochondromas). Individuals with these syndromes and bone diseases have an increased risk of developing osteosarcoma. People who have received radiation treatment for a previous cancer may have a higher risk of later developing osteosarcoma in the area that was treated. Being treated at a younger age and with higher doses of radiation both increase the risk. Because the risk of osteosarcoma is highest between the ages of 10 and 30, especially during the teenage growth spurt, experts believe that there may be a link between rapid bone growth and the risk of a bone tumor. Children with osteosarcoma are often tall for their age, which supports the link with rapid bone growth. Other than radiation, there are no known lifestyle or environmental risk factors associated with osteosarcoma. Asides from these risk factors, the causes of most osteosarcomas are unknown.

Summary

In summary, the scientific literature continues to suggest that exposure opportunities to artificial turf fields are not generally expected to result in health effects. Testing results on the crumb rubber infill indicated lead content less than CPSIA statutory limits established for children's products. For the turf fibers, APT provided a statement that this material did not have lead used in its manufacture, but no additional documentation was provided.

With respect to cancer concerns reported in media stories, it is important to note that the reports of cancers were of a wide variety of different types, each with its own set of risk factors. In addition, our staff reviewed cancer incidence data for the Town of Medway. The Massachusetts Cancer Registry (MCR) is a population-based surveillance

system that began collecting information in 1982 on Massachusetts residents diagnosed with cancer in the state. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within six months of the date of diagnosis (MGL, c.111, s.111B). This information is kept in a confidential database and reviewed for accuracy and completeness.

Available information on the occurrence of cancers in children living in Medway indicates no diagnoses of Hodgkin Lymphoma, NHL, or osteosarcoma have been reported to the MCR in a search of their files from 2006 to the present. Although it is possible that a very recent diagnosis may not yet have been reported to the MCR, the fact that there are no reports of such cancers is reassuring.

Although available resources cannot support MDPH conducting environmental testing of this material, we would be happy to assist the Town of Medway in developing a sampling and analysis plan as well as provide technical support in interpreting results, similar to the assistance that we provided to the Town of Needham.

As we stated in our letters to Needham officials, while available information does not indicate exposure opportunities of health concern, MDPH/BEH continues to recommend common sense ways to minimize any potential exposure to chemicals that may be contained in synthetic turf fields made of crumb rubber. MDPH/BEH suggests washing hands after playing on the field and before eating, particularly for younger children with frequent hand-to-mouth activity, and taking off shoes before entering the house to prevent tracking in any crumb rubber particles. Also, there are studies that indicate heat levels on artificial turf fields may rise as outdoor temperatures increase (New York State 2009). Thus, for protection of the players, MDPH/BEH recommends increasing hydration, taking frequent breaks, and watering down the field to cool it on hot days to prevent the potential for burns or heat stress. Finally, based on recent work in California, MDPH/BEH recommends that steps be taken to minimize the potential for skin abrasions (e.g., protective equipment) and that skin abrasions be treated promptly to prevent potential infections.

We hope this information is helpful to you and Medway residents. If you have any questions, please feel free to contact us at 617-624-5757.

Sincerely,

Suzanne K. Condon, Associate Commissioner
Director, Bureau of Environmental Health

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Washington State Department of Health

Investigation of Reported Cancer among Soccer Players in Washington State

Revised April 2017

DOH Pub 210-091 Revised April 2017

Investigation of Reported Cancer among Soccer Players in Washington State

Revised April 2017

For more information or additional copies of this report contact the Washington State Department of Health:

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DOH Pub 210-091 Revised April 2017

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April 2017 Revisions

After publishing the report "Investigation of Reported Cancer among Soccer Players in Washington State" in January 2017, the Department of Health realized the purpose and conclusions from the investigation were not stated clearly enough in the report. A complete review of the data after publication also identified some minor errors in the data. The department published the following revision to ensure the data were accurate and the conclusions were not misinterpreted.

To clarify the purpose of the investigation and explain why we did not actively look for soccer players with cancer, we provided information on the cluster investigation process used by the Department of Health. Details from the Department of Health Guidelines for Investigating Clusters of Chronic Disease and Adverse Birth Outcomes (Guidelines) are provided below and can be found in the background section of the Executive Summary and main report. The full Guidelines are available upon request.

The Guidelines provide a standardized approach to investigating potential non-infectious disease clusters. Stage 1 of the investigation involves collecting information on the disease of concern and determining if criteria to continue the investigation have been met. These criteria include: at least 3 cases of the same or similar conditions, or a specific exposure of concern where a potential route of exposure is alleged as the cause of the cluster. Initial review of the cases reported by Coach Griffin met these criteria so we proceeded to Stage 2. Stage 2 involves providing an initial assessment of the magnitude of the reported cluster and whether there is enough concern to continue investigating. As part of Stage 2, we developed an initial case definition, validated the reported cancers, explored background rates of disease, conducted a literature review of childhood leukemia and lymphoma, and assessed the literature on crumb rubber and the potential for exposures that could result in elevated rates of cancer. The January 2017 report described methods and findings from Stage 2 of the protocol.

The Guidelines list the following criteria for moving to Stage 3:

- At least 3 cases of the same condition, an excess of cases that is at least marginally statistically significant, AND one or more of the following:
    - The disease is of known etiology and there is potential for exposure to the causal agent OR
    - Scientific literature supports an association between the reported exposure and the reported condition OR
    - The disease is of unknown etiology and there is potential for exposure to a rare or unusual substance (i.e., these exposures are unique to an area or an occupation and are not commonly found in other places in Washington or the United States) OR
    - The disease is extremely rare

Our investigation showed that we did not meet the criteria for moving beyond Stage 2 of the investigation. When we explored Coach Griffin's data, we noted that the people on her list were diagnosed over an extended period of time, across a broad range of ages, and included

a number of different cancer types. While we did have more than three soccer players with leukemia, Hodgkin lymphoma and Non-Hodgkin lymphoma, we did not meet any of the other conditions for continuing our investigation. Thus, we did **not** progress to Stage 3 which would have entailed efforts to identify all soccer players ages 6–24 years old diagnosed with cancer in the state during 2002–2015.

The purpose of our investigation was to explore whether the information from Coach Griffin's list warranted further public health response. **Our investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf.** Our findings do not support further public health response at this time. The available scientific literature suggests exposures to toxic chemicals from crumb rubber are very low, however, questions remain about potential toxicities and levels of exposure. Therefore, we will continue to monitor research in this area.

In addition to clarifying the purpose and conclusions of the report, this revised report corrects the following data errors.

- Executive summary: Review found that two-thirds of respondents were playing soccer at the time of getting cancer and one-third had stopped playing soccer. The original noted three-quarters and one quarter, respectively.
- Table 15: The years between ending soccer play and diagnosis for the 33 participants was changed from 0-13 to 0-9 years. The change was necessitated by gaps in play (e.g., playing for several years, stopping play for several years and then playing again) for several participants that had not been accounted for previously.
- Table 17: The median years of playing recreational level soccer for those meeting the case definition was changed from 5 to 5.5 due to an error in transferring from Microsoft Excel to Microsoft Word.
- Table 18: One person who met the case definition and played soccer in high school was erroneously characterized as playing soccer in high school and college. Correcting this error resulted in increasing the number of high school only players by one (becoming 8 for all participants and 5 for those meeting the case definition) and reducing the number of high school and college players by one (becoming 4 for all participants and 3 for those meeting the case definition).
- Table 19: The same error described for Table 18 resulted in reducing the number of college goalkeepers by one (becoming 4 for all participants and 3 for those meeting the case definition). There was also an unexplained error in the number of total participants playing goalie at any level, changing the count from 14 to 16.
- Table 20: A weighting error in parsing percentage of play on artificial turf into indoor and outdoor play resulted in moving one person meeting the case definition from the 25-<50 percent quartile to the 0-<25 percent quartile for indoor artificial turf (changing 2 to 1 and 22 to 23, respectively) and vice versa for percent of time on outdoor artificial turf (changing 8 to 9 and 11 to 10, respectively). These changes also required changes in the quartiles for all players, because "all players" include those meeting the case definition. The median percent of time on outdoor artificial turf

also increased from 25-29 percent to 30-34 percent for players meeting the case definition.

- Table 21: The same weighting error described for Table 20 affected Table 21. A programming error in the calculation of percent of total time spent on artificial turf compounded the weighting error. This resulted in changes for 6 of the 8 entries for quartiles of percent of time playing on grass or dirt and 5 of the 8 entries, as well as the medians, for playing on outdoor artificial turf. The medians for both players meeting the case definition and all players changed from 30-34 percent of time on outdoor artificial turf to 35-39 percent.

Executive Summary

Background

University of Washington Women's Associate Head Soccer Coach Amy Griffin became concerned about the amount of cancer among soccer players in Washington State and compiled a list of soccer players with cancer. Coach Griffin was especially concerned about the number of goalkeepers she identified with cancer and wondered whether exposure to crumb rubber infill in artificial turf might be causing it. The list included 53 people, most of whom played soccer. Due to heightened public concern and the large number of people on the list, public health officials at the Washington State Department of Health and researchers from the University of Washington School of Public Health formed a project team to investigate following the Department of Health Cluster Guidelines (see p. iv for additional detail). The overall purpose of the investigation was to explore whether the information from Coach Griffin's list warranted further public health response. The main goals of the investigation were to:

1) Compare the number of cancers among soccer players on the coach's list to the number that would be expected if rates of cancer among soccer players were the same as rates among all Washington residents of the same ages.
2) Describe individuals reported by the coach in terms of their demographics, factors related to cancer, and history of playing soccer and other sports.
   Note: The investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf.

To provide background for accomplishing the two main goals, we:

1) Compared cancer types, rates and changes in rates over time among Washington and U.S. residents, ages five to 24 years old. We did not find unusual patterns of cancer in Washington compared to the United States.
2) Reviewed the scientific and medical literature to understand factors that increase the likelihood of getting leukemia or lymphoma—the two types of cancer most frequently reported by the coach—as a child or adolescent. This review noted that leukemia and lymphoma are complex diseases that can only rarely be attributed to a single cause or exposure. Exposures during the prenatal and early postnatal periods may be particularly important, because they can disrupt processes that are important for normal health and development.
3) Reviewed research on the relationship of crumb rubber, recycled rubber products and artificial turf to human health. Crumb rubber is made from tires or other rubber products that are ground into small pieces to provide a soft infill for artificial turf fields. Crumb rubber has become popular because of its relatively low cost and long life. Seven review articles published in the last 10 years all concluded that playing on artificial turf fields is unlikely to expose children, adolescents or adults to sufficient levels of chemicals from the fields to significantly affect health. However, there are still unanswered questions due to limitations in existing research

Methods for Primary Goals

Comparing the observed and expected number of cancers

To compare the number of cancers observed among soccer players on Coach Griffin's list to the number that would be expected if rates among soccer players were the same as all Washington residents, we took several steps.

Tabulating the observed number of cancers. For this part of the investigation, we specified how we would count soccer players with cancer. We defined observed cancers as cancers among people who:

- Were diagnosed during 2002–2015.
- Were six to 24 years old at the time of the diagnosis.
- Played soccer while living in Washington State at some point before getting cancer.
- Began playing soccer at least 0.4 years before diagnosis.

We refer to people meeting the above criteria as people who "meet the case definition." We limited the case definition to people who were ages six to 24 years when diagnosed, because processes leading to the development of cancer are often different for children and adults.

Calculating the number of cancers expected among soccer players. To determine the number of cancers expected among soccer players if rates among soccer players were the same as rates among all Washington residents of the same ages, we needed to estimate the number of people ages six to 24 years old at any point during 2002–2015 who had played soccer while living in Washington. We used Washington Youth Soccer (WYS) enrollment information from 1983–2015 to estimate the number of people who had played soccer. This is a subset of all people ages six to 24 years during each year from 2002–2015. We needed information beginning in 1983, because a person diagnosed with cancer at age 24 in 2002 could have begun playing soccer at age six in 1983. People who played soccer and were ages six to 24 years during each year from 2002–2015 make up the population from which the observed cases of cancer come. For every year that a person could have developed cancer and been considered in the group of observed cases, they contribute one person-year at risk. The number of people who played soccer times the number of years over which they could have developed cancer from 2002–2015 is the total person-years at risk from which we can calculate the expected number of cancers. We multiplied the total person-years at risk in each year of age by Washington cancer rates for each year of age. We added the number of cancers expected at each age to get the total number of expected cancers.

Ratio of the number of cancers observed to the number expected. We computed ratios of the observed number of cancers diagnosed from 2002–2015 among soccer players on the coach's list who were six to 24 years at diagnosis to the expected number of cancers described above. We computed these ratios for all cancers combined among all players, players at the WYS-defined select and premier levels, goalkeepers and players grouped by age. We also computed these ratios for leukemia, non-Hodgkin lymphoma and Hodgkin lymphoma for the same groups of players.

Describing selected characteristics of people reported to the project team. We interviewed people or parents of people on the coach's list to obtain information on demographics, cancer-related factors, and history of playing soccer and other sports.

Results

Response rate. We obtained interviews for 35 of the 53 people on Coach Griffin's list (66 percent), including 25 of the 27 (93 percent) people who met the case definition.

Comparing the observed number of cancers among soccer players to the expected number. The 27 people meeting the case definition had 28 primary cancer diagnoses. The number of cancers expected to occur in Washington among comparable soccer players was 1,384. Thus, the number of cancer cases on the coach's list was about two percent of the number expected. The numbers of cancer cases on the coach's list for specific cancer types, as well as for goalkeepers and for select and premier players were also lower than expected.

Table 1. Observed cancers from coach's list and expected cancers: soccer players ages 6–24 years diagnosed during 2002–2015

	Observed cancers from coach's list	Expected cancers	Ratio of observed to expected	95 percent confidence interval
All soccer players				
All types of cancer	28	1,384	0.02	0.01-0.03
Leukemia	6	131	0.05	0.02-0.10
Hodgkin lymphoma	5	147	0.03	0.01-0.08
Non-Hodgkin lymphoma	6	89	0.07	0.02-0.14
Goalkeepers	14	153	0.09	0.05-0.15
Select/premier soccer players	15	284	0.05	0.03-0.09

Selected characteristics of people on Coach Griffin's list.

Demographics and types of cancer. Coach Griffin's list included more females and more people living in King County than we would expect given WYS enrollment and U.S. Census data. There were 55 primary cancers among the 53 people on the coach's list: 11 leukemias, 13 non-Hodgkin lymphomas, 11 Hodgkin lymphomas, four bone cancers, three soft-tissue sarcomas and three brain cancers, all of which had multiple subtypes. Ten people had eight other types of cancer. The 53 people ranged from three to 51 years old when they got cancer and all but three were diagnosed during 2002–2015.

Soccer. The shortest amount of soccer play was one season for one year, but about half of the interviews indicated soccer playing for at least 10 years before getting cancer. About half of the interviews showed soccer play beginning at four to six years old, two-thirds as playing soccer at the time of getting cancer, and one-third as having stopped playing soccer for between one and nine years before getting cancer. The proportion of select and premier players on the coach's list was higher than expected based on WYS estimates of the percent of players at these levels. Based on estimates of the percentage of all soccer players who are goalkeepers, there were also more goalkeepers than expected.

Interviews indicated players practiced on about 110 different soccer fields in Washington. Most fields were reported once or twice. Two fields were each reported in four interviews. All interviews with detail on types of artificial surfaces included exposure to crumb rubber through soccer or other activities. Most soccer play, however, was on grass. Averaged across all reported play, half of the soccer players practiced on grass or dirt 70–74 percent of the time; on outdoor artificial turf 20–24 percent of the time; and on indoor artificial turf zero to four percent of the time. Interview participants spent more time on artificial turf for games, but most games for most players were on grass.

Discussion

We found that the number of cancers among all soccer players reported by Coach Griffin was less than expected given rates of cancer in Washington residents. This was also true for the number of cancers among select and premier players and goalkeepers on the coach's list, even though the list included larger percentages of these players than percentages of these groups enrolled with WYS.

Although there were several instances where participants practiced on the same fields, overall participants reported a large number of different fields suggesting that no specific field was potentially problematic. Given the ubiquity of crumb rubber infill, it is not surprising that all participants reported exposure through soccer, other activities or both. For most participants, most soccer play was on grass. Averaging the amount of time spent on outdoor and indoor artificial turf across all years of play, however, can mask intense play on artificial turf for periods of time. This can occur, for example, when someone plays on grass for many years before beginning to play adult recreational soccer on a league that consists exclusively of indoor games on artificial turf.

These findings are subject to several limitations. The list from the coach likely did not include all soccer players ages six to 24 years old who developed cancer during 2002–2015. The coach primarily works with skilled female goalies, which might have resulted in the relatively high percentages of females, select and premier players, and goalkeepers on her list. The coach might also be most familiar with cancer cases among soccer players in King County due to her working primarily in that county. Other soccer players with cancer were likely missed. There are also potential errors in the computation of the expected number of cancers among soccer players resulting from the need to make several assumptions about the numbers and ages of players each year from 1983–2015. None of the limitations are substantial enough to affect our conclusions. The findings from the interviews, however, might best represent characteristics of females, WYS-defined select and premier soccer players, goalies and players from King County, rather than soccer players overall.

Conclusions and Recommendations

Our investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf. Rather, its purpose was to explore whether the information from Coach Griffin's list warranted further public health response.

This investigation found less cancer among the soccer players, select and premier players, and goalkeepers on the coach's list than expected based on rates of cancer among Washington residents of the same ages. In addition, the currently available research on the health effects of artificial turf does not suggest that artificial turf presents a significant public health risk. Assurances of the safety of artificial turf, however, are limited by lack of adequate information on potential toxicity and exposure.

The Washington State Department of Health recommends that people who enjoy soccer continue to play irrespective of the type of field surface. The Washington State Department of Health will continue to monitor new research that emerges on the health and environmental impacts of crumb rubber.

Background and Objectives

Background on Reports of Cancer among Soccer Players

Initial report and follow up. In late 2008 and early 2009, two parents, each of whom had a child who played soccer and got cancer, contacted the Washington State Department of Health with concerns about cancer in youth who play soccer and a possible connection to artificial turf and crumb rubber infill. At that time, the health department:

- Conducted a literature review of health risks for soccer players and health outcomes associated with artificial turf or recycled rubber products.
- Obtained information from a pediatric oncologist at Mary Bridge Children's Hospital—where both of the children had been seen—about all children with cancer who were seen at the hospital in the prior five years. Information included birth date, gender, type of cancer, date of diagnosis, residence at the time of diagnosis and any sports played. While sports information is not gathered routinely, it is often noted in medical records of children for whom sports are important.

The literature review and the information from Mary Bridge Hospital—including the types and numbers of cancers reported among children who played soccer and their residences at diagnosis—did not suggest a public health problem requiring additional investigation.

Recent Report and Renewed Attention. In 2009, University of Washington Women's Associate Head Soccer Coach Amy Griffin became concerned that several soccer goalies had developed blood cancers at around the same time. By 2014, Coach Griffin had compiled a list of soccer players with cancer and the issue began receiving widespread media attention, as for example, in the October 2014 NBC evening news report.[1] University of Washington School of Public Health researchers contacted the Washington State Department of Health to request an investigation and subsequently met with Coach Griffin to explore the information she had compiled. The initial information included about 30 current or former Washington residents who played soccer and developed a variety of cancer types between the mid-1990s and 2015. By the end of 2015, the list included a total of 51 people, not all of whom played soccer. Those who did not play soccer were reported as having contact with the same types of playing fields as soccer players. Two more people were added in 2016 for a total of 53 people.

Leukemia and lymphoma were the most frequently reported types of cancer. The coach was concerned because many of the players on her list were soccer goalies who played at elite levels, such as college teams and select or premier play as defined by the Washington Youth Soccer (WYS). She hypothesized that the rubber crumb from artificial turf could be contributing to the cancers. Players commonly find the crumb on their clothes, equipment and hair after play. All players have contact with crumb rubber, but Coach Griffin theorized that goalies might have the most contact due to close, repeated contact with the ground.

In light of this renewed concern and the large number of people reported by Coach Griffin, health officials at the Washington State Department of Health and researchers at the University of Washington School of Public Health formed a project team to investigate issues related to soccer playing and cancer following the Washington State Department of Health

Guidelines for Investigating Clusters of Chronic Disease and Adverse Birth Outcomes (see April 2017 Revisions for more details). Appendix A lists the members of the project team.

Objectives

The overall purpose of the investigation was to explore whether the information from Coach Griffin's list warranted further public health response. The primary goals of the investigation were to:

1) Determine whether the number of cancer diagnoses among the soccer players on the coach's list was higher than would be expected if rates of cancer among these soccer players were similar to rates among all Washington residents of the same ages (referred to as "observed to expected ratios" below).
2) Describe individuals from the coach's list in terms of their demographics, factors related to cancer and history of playing soccer and other sports (referred to as "descriptive epidemiology" below).

Note: The investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf

Notably, this investigation is not designed to add to our understanding of the risks or benefits of crumb rubber fields or to discover the causes of cancer among the people reported to the project team. These concerns could not be addressed due to the diversity of the types of cancer, the lack of known causes for most cancer in children and young adults, little information about the potential for chemicals in crumb rubber to cause toxic exposures, and no biological or environmental testing.

To provide a background for accomplishing the primary objectives, we also:

1) Compared cancer rates and changes in rates over time among children and young adults in Washington to those seen nationally, in order to consider whether cancer rates and trends in Washington are unusual.
2) Reviewed the scientific and medical literature to understand factors that increase the likelihood of developing leukemia or lymphoma.
3) Reviewed published papers related to crumb rubber and recycled rubber products to determine:
    a. Whether crumb rubber or components of crumb rubber have been associated with specific diseases or adverse health conditions.
    b. Whether and how athletes who play on crumb rubber fields may be exposed to hazardous levels of chemical components of crumb rubber.

Background on Rates of Cancer in Washington and the United States

State law (RCW 70.54.230) authorizes the Washington State Cancer Registry (referred to in this report as "cancer registry") to collect cancer-related information for Washington residents diagnosed and treated for cancer, including residents diagnosed and treated in other states. The cancer registry can be used to compute Washington's rates of cancer diagnoses (cancer incidence), compute changes in rates over time, and compare incidence

rates in Washington to rates in other places. For this report, we used the cancer registry's January 2016 data release that included complete information for 1992–2013.[2]

The Surveillance, Epidemiology and End Results (SEER) program of the National Cancer Institute provides information on cancer incidence nationally. For this report we used national cancer data from 13 SEER regions available from SEER*Stat for all analyses except for the most frequently diagnosed types of cancer. For that analysis, national data are from 18 SEER sites also available from SEER*Stat.[3] We used the Washington State Department of Health mortality files to assess death from cancer in Washington and mortality data available through SEER*Stat for national cancer deaths.[3,4]

Population counts needed for calculating the Washington State rates are from the Washington State Office of Financial Management. These include intercensal interpolations for 1992–1999 and 2001–2009, U.S. Census data for 2000 and 2010, and postcensal estimates for 2011–2013.[5] Population counts for calculating national rates are available through SEER*Stat.

SEER data are available by five-year age groups. To compare Washington and national rates, we focused on ages five to 24 years because these ages were the closest to the six- to 24-year age group in the case definition described in the Methods section below. We compared age-adjusted incidence rates for all cancers combined, leukemia, non-Hodgkin lymphoma and Hodgkin lymphoma. Age-adjustment allows us to compare rates among groups with different age distributions. It helps us to understand whether there are differences among groups independent of differences in numbers of people at older or younger ages. This is important when looking at cancer rates because cancer rates are higher for some age groups than for others.

Except for assessing changes over time, we computed age-adjusted rates for 2009–2013 combined so that random year-to-year fluctuations would be less likely to influence the findings. For assessing changes over time, we computed age-adjusted incidence rates for each year from 1992–2013. We used Joinpoint software to analyze changes in these rates over time.[6]

Appendix B provides additional detail of the methods used for these calculations.

Combining all types of cancer, Washington residents ages five to 24 years had an average of 439 cancers diagnosed and an average of 55 deaths from cancer each year during 2009–2013. Lymphoma, brain and other nervous system, and leukemia were the most frequently diagnosed cancers, followed by thyroid, melanoma of the skin, and testes. These were also the six most frequently diagnosed cancers among U.S. residents ages five to 24 years.

Table 2. Age-adjusted cancer incidence rates per 100,000 people ages 5–24 in Washington and the United States, 2009–2013

Type of Cancer	Washington State	United States
All types of cancer combined	23.6	22.4
Leukemia	3.1	3.3
Non-Hodgkin lymphoma	1.5	1.7
Hodgkin lymphoma	2.3	2.2

During 2009–2013, the age-adjusted cancer incidence rate for all types of cancer combined among people ages five to 24 years was statistically significantly higher in Washington than in the United States (23.6 and 22.4 per 100,000 people, respectively). Even though Washington's rate is statistically significantly higher than the rate in the United States, the difference is small—about one person per 100,000—and does not suggest an unusual amount of cancer in Washington. The age-adjusted death rates were similar (3.0 per 100,000 people in Washington and 2.9 per 100, 000 in the United States). Washington's age-adjusted rates for new diagnoses of leukemia, non-Hodgkin lymphoma and Hodgkin lymphoma were similar to the national rates, as was the age-adjusted rate for both types of lymphoma combined.

From 1992 to 2013, changes in age-adjusted incidence rates among people ages five to 24 years were slightly different in Washington and the United States. For all types of cancer combined, Washington's rates fluctuated more than did rates in the United States. Greater random variability in Washington than in the United States due to Washington's smaller population is likely the reason for this pattern.

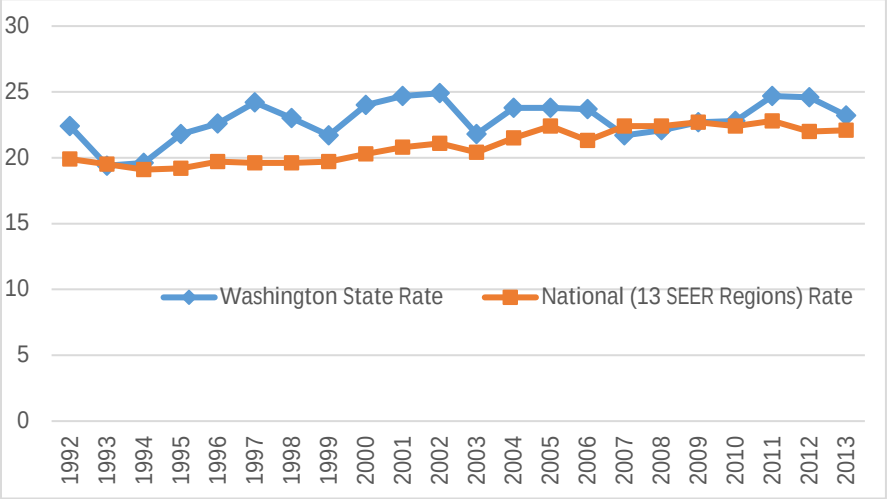

Figure 1. Age-adjusted cancer incidence (all types combined), ages 5–24, Washington and United States

After accounting for random variability, incidence rates for all cancers combined among Washington residents ages five to 24 years increased slightly from 1992 to 2013. In the United States, these rates did not change from 1992 to 1994; increased steadily at about three times the rate of the increase in Washington from 1994 to 2009; and leveled off again from 2009 to 2013. Incidence rates of leukemia and non-Hodgkin lymphoma stayed the same in Washington; nationally, the leukemia rate increased slightly and the rate for non-Hodgkin lymphoma increased steadily. The incidence rate of Hodgkin lymphoma decreased steadily in Washington and stayed the same in the United States. Overall, we concluded that changes in Washington were not unusual compared to changes seen nationally.

Table 3. Annual percent change in age-adjusted cancer incidence rates per 100,000 people ages 5–24 in Washington and the United States, 1992–2013

Type of Cancer	Washington State	United States
All types of cancer combined	0.4 percent increase per year	1.2 percent increase per year, 1994-2009; no change 1992-1994, 2009-2013
Leukemia	No change	0.9 percent increase per year
Non-Hodgkin lymphoma	No change	1.4 percent increase per year
Hodgkin lymphoma	1.3 percent decrease per year	No change

Background on Leukemia and Lymphoma

Leukemia. Leukemia, a cancer of the blood and bone marrow, is the most common type of childhood cancer in the United States. This diagnosis accounts for approximately 30 percent of all cancers among children less than 15 years old.[7] Leukemia is also one of the most common cancers among adolescents and young adults, ages 15–24.[8]

There are two primary subtypes of childhood leukemia, acute lymphocytic leukemia (ALL) and acute myelogenous leukemia (AML). ALL comprises about 75 percent of all childhood leukemia and is most common in children between two and four years old. By contrast, AML, which develops from a different type of cell than ALL, is often detected in children under age two, as well as during adolescence.[7,8]

Lymphoma. Lymphoma is a cancer of the white blood cells, called lymphocytes, that are part of the body's immune system. Lymphocytes are present in many parts of the body, such as in lymph nodes, the spleen, bone marrow and the digestive tract. Lymphoma is the third most common type of childhood cancer and also one of the most common cancers among adolescents and young adults, ages 15–24.[9]

The two primary lymphoma subtypes are non-Hodgkin lymphoma (NHL) and Hodgkin lymphoma (HL). NHL accounts for about five percent of all childhood cancer in the United States and most of the lymphoma diagnosed in children less than 14 years old. It is two to three times more common in males than females, and it is more common in white children than African-American children.[10] HL is the most commonly diagnosed cancer among adolescents and young adults but is much less common in young children.[11,12] In young children, HL is about five times more common among males, while in adolescents, HL is slightly more common among females.[12]

Risk factors for leukemia and lymphoma. Risk factors for a disease increase the chances of developing the disease. Leukemia and lymphoma are complex diseases that can only rarely be attributed to a single cause or exposure. Rather, both genetic and non-genetic factors, such as infections and toxic chemicals, likely play roles in disease development. The scientific and medical literature describes several factors that most scientific, medical and other cancer-related organizations accept as known risk factors for leukemia, lymphoma or both. Other risk factors are more controversial. Exposures during the prenatal and early postnatal periods may be particularly important, because they can disrupt processes that are important for normal health and development.

We investigated risk factors for cancer in children and adolescents through an online search of scientific and medical research using PubMed and Google Scholar. We included search terms such as "'child' and 'leukemia' and 'risk factor'" and "'child' and 'lymphoma' and 'risk factor.'" We focused on studies that followed groups of children over time who were exposed or not exposed to one risk factor (cohort studies); studies that compared children with and without cancer for exposure to a given risk factor (case-control studies); and studies that combined results from multiple cohort or case-control studies (meta-analyses). We also consulted online information from the American Cancer Society and the National Cancer Institute and a publication of the American Academy of Pediatrics (AAP).[13]

Below are tables that highlight several known or suspected risk factors for leukemia and lymphoma in children and adolescents. Some factors have been studied extensively and are well accepted as increasing the risk of developing leukemia, lymphoma or both. Others are less well documented or more controversial. The tables include the AAP's designation of factors as known or suggested risk factor and ACS' designations of known or possible risk factors.

Table 4. Risk Factors for Leukemia among Children and Adolescents

Risk Factor	AAP[a] and ACS[b] Designations or Other Evidence[c]	Brief Description/Notes	Selected References
Ionizing radiation	AAP: known ACS: known	In utero and early life exposure to ionizing radiation is a well-established risk factor for leukemia, particularly AML. Possible sources of ionizing radiation include x-rays and CT scans.	14,15,16,17,18
Family history	AAP: known ACS: known	Individuals are at higher risk of developing leukemia if they have siblings (especially a twin), parents, or other close relatives with a prior leukemia diagnosis.	14,19
Inherited genetic conditions	AAP: known ACS: known	Examples of specific genetic conditions that may increase risk of developing leukemia include: Down syndrome, Klinefelter syndrome, Faconi anemia, and Bloom syndrome. These conditions are commonly diagnosed at birth or in early childhood.	20,21
Birth weight	AAP: known	Birth weight greater than 4,000 grams has been linked to the development of ALL in some epidemiological studies.	14,22,23
Chemotherapy treatment	AAP: known ACS: known	Treatment with certain chemotherapy drugs for cancers lead to higher risk of developing other cancers. These cancers usually develop 5-10 years after the initial treatment.	7,24
Pesticide exposure	AAP: suggested ACS: possible	Studies report associations between preconception, fetal and/or early life exposure to pesticides and the development of leukemia. However, there are only a limited number of studies that have assessed this association and so further work is needed to provide definitive conclusions.	14,22,25,26,27,28

[a] AAP = American Academy of Pediatrics[13]
[b] ACS = American Cancer Society[29]
[c] Other evidence only for factors not included in AAP or ACS classifications

Table 4 (continued). Risk Factors for Leukemia among Children and Adolescents

Risk Factor	AAP[a] and ACS[b] Designations or Other Evidence[c]	Brief Description/Notes	Selected References
Solvent exposure	AAP: suggested ACS: possible	Preconception, fetal, and early postnatal exposure to solvents has been linked to the development of leukemia. Benzene is one solvent of particular concern, given that it is also an established cause of adult leukemia.	14,30,31,32
Maternal age	AAP: suggested ACS: possible	Maternal age older than 35 years during pregnancy has been associated with increased risk of leukemia development.	22,33
First-born child	AAP: suggested	There is conflicting evidence about the role of birth order in leukemia risk, but some studies suggest that first-born children are at higher risk. Some theorize that this increased risk is related to decreased early life immune stimulation. (See below.) Children with older siblings are likely to be exposed to communicable diseases from their older siblings, while firstborn children may have fewer extra exposures that help their systems develop.	34,35
Decreased early life immune stimulation	Epidemiological evidence	Some studies suggest that early life immune stimulation (ex: contact with farm animals and daycare attendance) can promote normal immune system development and prevent cancer. Other studies, however, report no association.	14,22,33,35,36
Electromag-netic fields	ACS: possible	A 2002 report from the International Agency for Research on Cancer (IARC) classified extremely low frequency electromagnetic fields as "possibly carcinogenic to humans."	37,38,39,40,41
Hazardous air pollutants/ traffic pollution exposure	Epidemiological evidence	Examples of air pollutants that may increase risk of developing leukemia include: polycyclic aromatic hydrocarbons (PAHs), diesel exhaust, and benzene.	31**Error! Bookmark not defined.**, 42,43, 44
Maternal alcohol use during pregnancy	ACS: possible	Fetal alcohol exposure has been associated with increased risk of leukemia, particularly AML.	14,15,22,45
Maternal infection during pregnancy	Epidemiological evidence	Studies suggest that maternal infection during pregnancy could provide an initial genetic disruption that increases the risk of childhood cancer.	34,46
Paint exposure	Epidemiological evidence	Studies report associations between preconception, fetal, or early life paint exposure and the development of leukemia.	30,47

[a] AAP = American Academy of Pediatrics[13]

[b] ACS = American Cancer Society[29]

[c] Other evidence only for factors not included in AAP or ACS classifications

Table 5. Risk Factors for Lymphoma among Children and Adolescents

Risk Factor	AAP[a] and ACS[b] designations or other evidence[c]	Brief Description/Notes	Selected References
Family history	AAP: known ACS: possible	Individuals are at higher risk of developing lymphoma if they have siblings (especially a twin), parents, or other close relatives with a prior lymphoma diagnosis.	48,49
Immune deficiency	AAP: known ACS: known	Weakened immune systems may result from inherited genetic conditions (ex: Wiskott-Aldrich syndrome, Ataxia-telandiectasia, and Bloom syndrome), HIV/AIDS, or specific drug treatments to suppress immune responses after organ transplants.	50,51
Viral infections	AAP: known ACS: known	Epstein-Barr virus (EBV) has been classified by the International Agency for Research on Cancer (IARC) as a known human carcinogen that increases risk of lymphoma.	52,53,54,55,56, 57,58,59
	ACS: known	IARC has also classified both Hepatitis B and C as known human carcinogens. Hepatitis C is listed as a causal factor for NHL, while Hepatitis B is listed as positively associated with NHL.	
Autoimmune diseases	Epidemiological evidence	Autoimmune diseases including Sjogren disease, celiac sprue, and systemic lupus erythematosus (SLE) increase risk of lymphoma.	51,60,61,62
Decreased early life immune stimulation	Epidemiological evidence	Some studies suggest that early life immune stimulation (ex: contact with farm animals and daycare attendance) can promote normal immune system development and prevent abnormal responses leading to cancer. Other studies, however, report no association.	35,63
Birth weight	Epidemiological evidence	Birth weight over 4,000 grams has been linked to the development of lymphoma in some, but not all, studies.	48,64
Hazardous air pollutants or pollution from traffic	Epidemiological evidence	Examples of relevant toxic air pollutants that may increase risk of developing lymphoma include: PAHs, diesel exhaust, and benzene.	31
Maternal smoking during pregnancy	Epidemiological evidence	Maternal smoking during pregnancy has been linked to increased risk of NHL.	65
Pesticide exposure	Epidemiological evidence	Some studies report associations between preconception, fetal and/or early life exposure to pesticides and the development of lymphoma, but overall results are inconsistent.	26,27,66,67
Solvent exposure	Epidemiological evidence	Preconception, fetal, and early postnatal exposure to solvents has been linked to the development of lymphoma.	31

[a]AAP = American Academy of Pediatrics[13]
[b]ACS = American Cancer Society[50,51]
[c] Other evidence only for factors not included in AAP or ACS classifications

Crumb Rubber

Artificial or synthetic turf fields were first introduced in the 1960s, under the brand "Astro-turf." Over the next several decades, artificial turf fields became very popular. Now, many different manufacturers produce their own brands of artificial turf. Because artificial turf fields are not cushioned like natural grass fields, manufacturers use an infill to adjust the

firmness of fields to make them safer for athletes. Originally, manufacturers used sand as infill. However, the sand infill was quite hard and quickly became even more compacted through normal athletic play. Additionally, the sand infill often produced dust during play.[68] Manufacturers looking for a softer and less dusty alternative soon turned to recycled rubber. Tires or other rubber products are processed into small pieces to provide a better infill for artificial turf fields. Although there are other types of infill—such as organic substances, virgin rubber, and coated sand—crumb rubber became very popular because of its relatively low cost and long life.[68] First introduced in the 1980s, crumb rubber now is used as an infill in 98 percent of all artificial turf fields.[69] Our understanding is that the first artificial turf fields with crumb rubber infill were installed in Washington in the late 1990s.[70]

Artificial turf may save water, avoid the need for fertilizers and pesticides, and provide a cushion to help prevent injuries from impact. Artificial turf provides a year-round playable surface, which may make it easier for youth and adults in Washington to be more physically active. However, in the past several years, people have voiced concerns about potential health effects from both artificial turf and crumb rubber infill. These include concerns about cancer, dehydration, and infection. If an artificial turf field is under direct sunlight, temperatures are often higher than natural turf temperatures[71] and this may contribute to more heat-associated illnesses, like heat stress or dehydration.[69] One study also found an association between "turf burns" from artificial turf and methicillin-resistant bacteria infections, commonly known as MRSA.[72] Other concerns are related to the chemicals in the crumb rubber infill. Rubber tires used as infill contain many different chemicals, some of which have been linked to cancer. For example, the benzene found in some samples of crumb rubber is known to cause cancer.[73,74] To understand if the chemicals in crumb rubber found in artificial turf fields may be linked to health risks, we reviewed recent publications about potential health effects from recycled rubber products and artificial turf fields.

Using Google Scholar and UWLibraries with search terms such as "'chemical' and 'artificial turf'" or "'health' and 'recycled tires' and 'hazard,'" we located review papers that were published in the last 10 years. Review papers are designed to examine the larger body of literature as a whole.

We found seven publications (Table 6), only one of which went through the scientific peer review process before publication.[68] The remaining papers were gray literature published either by government agencies or consulting firms hired by a person, company, or government agency. Gray literature includes material such as government documents, research or materials published by non-governmental organizations, working papers, and white papers. Gray literature does not go through the scientific peer review process that characterizes publications in scientific journals. Papers that do not go through the scientific peer review process may be less reliable than those that do. One paper[75] was published for the Rubber Manufacturers' Association raising questions about potential conflicts of interest.

All seven publications concluded that children, teenagers, and adults are unlikely to be exposed to sufficient levels of the chemicals found in artificial turf fields to significantly affect health. One report discussed that indoor facilities using crumb rubber and lacking proper

ventilation may experience higher chemical concentrations in the air,[76] but another paper contradicted this conclusion.[77]

Table 6. List of published reviews used to assess whether crumb rubber or components of crumb rubber have been associated with poor health

Title	Author	Published for	Year of Publication
Evaluation of Health Effects of Recycled Waste Tires in Playground and Track Products	Government - Office of Environmental Health Hazard Assessment	State of California	2007
A Review of the Potential Health and Safety Risks from Synthetic Turf Fields Containing Crumb Rubber Infill	Consulting firm – TRC	New York City Department of Health	2008
Initial Evaluation of Potential Human Health Risks Associated with Playing on Synthetic Turf Fields on Bainbridge Island	Consulting firm – Windward Environmental LLC	Bainbridge Island Metro Parks and Recreation District; Bainbridge Island School District	
Review of the Impacts of Crumb Rubber in Artificial Turf Applications	Academic Consulting – University of California Berkley	Manex	2010
Review of the Human Health and Ecological Safety of Exposure to Recycled Tire Rubber found at Playgrounds and Synthetic Turf Fields	Consulting firm – Cardno ChemRisk	Rubber Manufacturers' Association	2013
Environmental and Health Impacts of Artificial Turf: A Review[a]	Academic Researchers – H. Cheng, Y. Hu, and M. Reinhard	Journal – Environmental Health and Technology Sciences	2014
Evaluation of Human Health Risks for Synthetic Field Turf	Consulting firm – Gradient	Lynnwood School District	2015

[a] Peer-reviewed paper

Although these papers all reached similar conclusions, there are still unanswered questions about the health effects from these chemicals due to limitations in existing research. To determine if health effects are associated with artificial turf or crumb rubber exposure, researchers need to understand realistic routes of exposure and estimated doses for each route as illustrated in the following diagram. To determine potential health effects, each step of the diagram shown below needs to be measured and studied to identify chemical exposure concentrations, duration of exposure and the dose received.

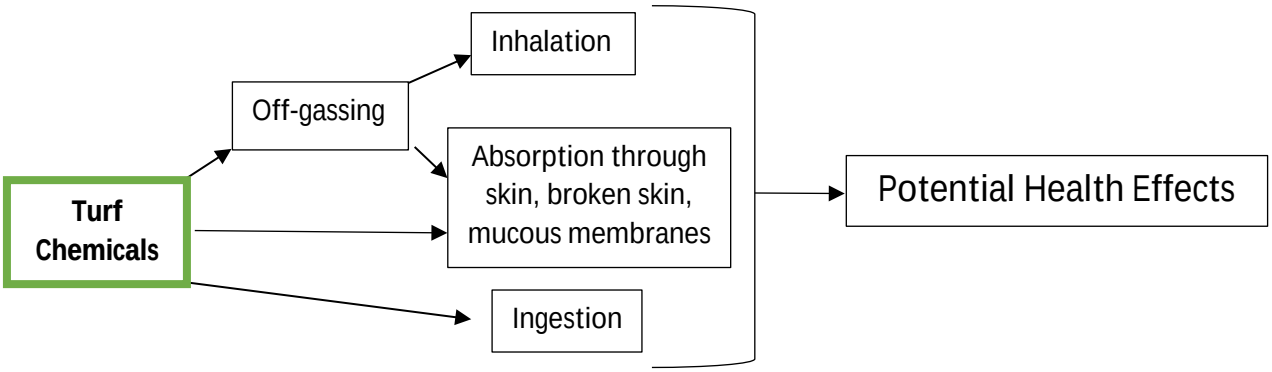

Figure 2. Diagram illustrating the possible routes of exposure from chemicals in artificial turf or crumb rubber

The routes of exposure show potential pathways chemicals travel from artificial turf infill into a human body. The most common routes of exposure to crumb rubber infill may be breathing (inhalation), eating (ingestion), and absorption through the skin, broken skin or cuts, or eyes. The potential dose describes exactly how much of a chemical that travels through a specific route of exposure ends up inside of the human body. The dose that ultimately ends up inside the body cannot be determined without information on the levels of chemicals in the exposure medium (for example, in the air or tire crumbs), as well as routes and durations of exposure. Without information on dose, scientists cannot determine the potential for chemicals to cause harm. Sometimes even when chemicals are present, the dose that enters the body is not large enough to cause harm. Most of the papers included in the reviews did not measure chemicals in the air or in crumb rubber or artificial turf, but rather estimated potential doses from information in other published reports. Thus, definitive conclusions about health risks cannot be made.

In addition to challenges from missing information on the route of exposure and dose, understanding any potential health effects from the chemicals in artificial turf or infill is difficult because the chemicals inside crumb rubber might differ depending on where the rubber came from and where and how it was recycled. A recent federal report by multiple agencies notes limited information on the variability of chemicals in crumb rubber, because most studies assessing the composition of the crumb rubber have been relatively small and restricted to a few fields or playgrounds.[78] Furthermore, crumb rubber and artificial turf degrade over time.[68] This means that the chemical exposures from a field that is one day old may be different from the exposures of the same field when it is one year old. These uncertainties make it difficult to understand the varying chemicals and potential amounts of chemicals in each field and crumb rubber infill and limit the ability of any paper to draw definite conclusions about potential health effects.

Thus, before we can more definitively understand the potential for artificial turf and crumb rubber infill to affect health, researchers need to describe routes of exposure, the specific chemical contents of turf fields in different areas and over different time periods, and doses. The recent federal report included an extensive literature review of information related to

the toxicity and human exposure to chemical constituents of crumb rubber. The report also detailed gaps in knowledge. Among other things, the report identified the need for greater understanding of exposures through the skin, eyes, and ingestion; identifying situations resulting in the highest exposures; monitoring levels of chemicals of concern in people; and assessing the feasibility of more in-depth epidemiologic study.[78]

Methods

Calculating Observed to Expected Ratios

Determining the number of observed individuals with cancer. One goal of the investigation was to determine whether the number of cancer cases on the coach's list (the "observed" number) was more than what we might expect based on cancer rates among people of the same ages (the "expected" number). To do this, we first developed a "case definition" that specified how we would count observed cancers.

For this investigation people met the case definition if they met all of the following conditions:

- Were diagnosed with cancer during 2002–2015.
- Were six to 24 years old at the time of the diagnosis.
- Played soccer while living in Washington State at some point before getting cancer. They did not need to be playing soccer or living in Washington when they were diagnosed.
- Began playing soccer at least 0.4 years before getting cancer.

We limited our time period for people meeting the case definition to 2002–2015, because reports of people with cancer were more likely to be complete during this time period than in earlier time periods. The only person who got cancer before 2002 was diagnosed in the mid-1990s before artificial turf fields were installed in Washington. Information for 2016 might not have been complete, because most of the work for this investigation was completed before the end of 2016. Two people were excluded from the case definition because they were diagnosed with cancer in 2016.

We limited the case definition to people who were ages six to 24 years when diagnosed, primarily because processes leading to the development of cancer are often different for children and adults. Most cancer investigations and research focus on children and adults separately, with children defined as under ages 15 or 20 years old. We opted to include people diagnosed up to age 24 years, because we wanted to include as many people as possible in the case definition without becoming so inclusive that we would lessen the potential to find a problem if one existed. This age group included about 70 percent of people reported to the project team.

We focused on soccer players, because this was the original group of concern, and most of the individuals reported to the project team played soccer. A relatively small number of people played a variety of other field sports or were exposed to crumb rubber through other activities.

Because cancer takes time to develop and be diagnosed, we also required a time lapse of about five months (0.4 years) between first playing soccer and diagnosis. The time between exposure to a cancer-causing agent and getting cancer is called the latency period. A 2012 publication on the Center for Disease Control and Prevention's website noted a latency period of 0.4 years for the development of leukemia after exposure to low-level ionizing radiation.[79] We used this minimum latency period to err on the side of including as many cancers as possible in the observed number of cancers. The time between beginning to play soccer and developing cancer was greater than 0.4 years for everyone reported to the project team. Thus, no one was excluded from meeting the case definition for this reason.

We limited our investigation to Washington residents because that is our jurisdictional authority, and information from the cancer registry was limited to Washington residents.

We used the cancer registry, reports to the project team and interviews (described below) to determine the number of people who met the case definition and the numbers and types of their primary cancers. Most of the demographic and cancer information from the cancer registry matched the information provided in interviews and from Coach Griffin. In the few instances where demographic or cancer information from these three sources did not match, we used information from the interviews where available, then information from the cancer registry, and finally information from the coach. Information about soccer playing was available from interviews and the coach. We relied on information from the interviews where possible and from Coach Griffin for people we did not interview.

Because of concerns about elite players and goalkeepers, we also tabulated the number of primary cancers among: 1) WYS-defined select and premier players and 2) goalkeepers. For this portion of the investigation, people were counted as goalkeepers if information from interviews indicated that they played goalkeeper at least 50 percent time at the WYS-defined select or premier levels or at least 25 percent time at the recreational level. We also included as goalies, one recreational level player who did not play the position for the first six years and then played goalie 30–50 percent of the time for three years and two people who were reported to the department as goalkeepers but were not interviewed. (See Descriptive Epidemiology in the Results section below for more detail.)

Calculation of expected number of cancer cases. The expected number of cancers is the number of cancers that would have occurred among soccer players if they experienced the same cancer rates as people of similar ages living in Washington. To compute the number of Washington soccer players ages six to 24 years expected to get cancer during 2002–2015, we needed to identify who would be "at risk" of developing cancer. The "at risk" population is the population from which the observed cancers come. People are included in the at-risk population if they were six to 24 years in any year of the study (2002–2015) and had played soccer in Washington for at least 0.4 years prior to the time during 2002-2015 they are considered to enter the at risk population. For every full year that they would have been included in the observed cases had they been diagnosed with cancer, they contribute one person-year at risk. "At-risk" refers only to the number of people times the number of years

over which they *could* get cancer. It does not refer to any theoretical risks from playing soccer or living in Washington.

To estimate the size of the at-risk population, we had to estimate the number of people who had ever played soccer who were ages six to 24 during 2002–2015. They did not have to play soccer during 2002–2015. For example, a person who began playing as a six-year-old in 1983, played for three years and was diagnosed with cancer before their 25th birthday in 2002 could be included as an observed case in this study. Thus, our count of the at-risk population needed to include people who had played from 1983 to 2015. We counted a person as contributing to the at-risk population if they were enrolled with the WYS between the ages of six and 15 (players ages 16 and older were assumed to have begun playing when they were 15 or younger) during 1983–2015. We calculated the total number of years lived by people during 2002–2015 while meeting these conditions: they were at least six years old and less than 25 years old and had started playing soccer at least 0.4 years previously. This is called the "person-years at risk." Appendix C includes a detailed description of this calculation.

To make a definitive calculation of the person-years at risk, we would have needed a roster of everyone who played soccer in Washington State at ages six to 24 from 1983 through 2015. However, because the WYS only provided a tabulation of the number of youth players by age from 2000–2015, we had to make several assumptions.

First, because we only had counts of the total number of players for 1983–1999,[80] we assumed that the age distribution of youth soccer players in each year during 1983–1999 was the same as in 2000. Second, we assumed a 10 percent turnover among players each year. If the previous year had fewer players than the current year, then we assumed that 90 percent of the previous year's players returned, and the difference was made up with new players. This was the case, for example, with the age seven enrollment in 2006 as less than the age eight enrollment in 2007. If the previous year had more players, then we assumed that the current year's players were 90 percent returnees and 10 percent new players. This was the case, for example, with the age 10 enrollment in 2006 being more than the age 11 enrollment in 2007. (See Table 2, Appendix C)

Third, we assumed that everyone who played on a soccer team at age 16 and older had also played at a younger age. We made this assumption because the WYS enrollment by age showed a big drop in the number of players at age 16. Fourth, we assumed that middle and high school players also played on a WYS team at some point.

Using these assumptions, we computed the person-years at risk at each year of age from six to 24 individually. To compute the expected number of cancer diagnoses among people who met the criteria for the case definition except for a known diagnosis of cancer, we multiplied the age-specific person-years at risk by Washington cancer rates from the cancer registry for 2002–2013 for each year of age. This calculation provided the number of cancer cases at each age that would be expected among all soccer players in Washington State if those players had the same cancer rates as all state residents of the same ages. We summed the

expected cancers at each age to determine the total number of cancer cases expected among soccer players ages six to 24 years from 2002-2015. We made similar calculations for WYS-defined select and premier players. WYS estimated that 20 percent of their athletes played at these levels.[81]

We made similar calculations to compute the person-years at risk for goalkeepers so that we could compute the expected numbers of cancer cases separately for them. Coach Griffin provided estimates of the numbers of goalkeepers at each age from ages 10–19. She compiled these estimates by talking with directors of coaching, club coaches or both from five of the larger clubs in Washington for both WYS-affiliated select and premier clubs. She also contacted a few smaller clubs to verify that their numbers were similar. Additionally, she talked with coaches at recreational clubs and noted that the number of goalkeepers on recreational teams was more difficult to assess, because players are not as specialized.[82] Thus, the case definition for goalkeepers required a cancer diagnosis between ages 10–24. We computed the person-years at risk for each year of age 10 to 24. As above, we multiplied the age-specific person-years at risk by age-specific cancer rates to get the number of cancers expected for each year of age. We added the number of cancers expected at each age to get the total number of cancers expected among goalkeepers.

We compared the observed number of cancer diagnoses (described above) to the expected number of diagnoses by computing the observed to expected ratio (observed/expected or O/E). If the O/E ratio is one, we conclude that the observed and expected numbers of cancer diagnoses are the same. If the O/E is more than one, there are more observed cancers than expected; if it is less than one, there are fewer cancers than expected. We used an R software[83] function to compute exact Poisson 95 percent confidence intervals for O/E. The 95 percent confidence interval provides an indication of the random variability of an estimate; wide confidence intervals indicate more random variability than narrow intervals. If the confidence interval for the O/E does not include one, we conclude that our observed number of cancers is statistically significantly different from the expected number. We computed O/E ratios for all cancers combined and O/Es individually for leukemia, non-Hodgkin lymphoma, and Hodgkin lymphoma. Details of these calculations are available in Appendix C.

Descriptive Epidemiology

Our primary purpose for the descriptive section of this report was to better understand similarities and differences of the people reported to the project team in terms of their histories of cancer and playing soccer. The information on playing soccer might also be useful to researchers designing risk and exposure assessments.

Questionnaire. We developed a questionnaire that allowed us to broadly describe characteristics of the individuals reported to the project team in terms of their demographics, factors related to their cancer diagnoses, and history of playing soccer and other sports. We included questions on race and Hispanic ethnicity in the section on demographics, because some types of cancer are more common in some racial and ethnic groups than in others. We focused on risk factors for leukemia and lymphoma because those

were the types of cancer most frequently reported. All participants were asked about risk factors specific to leukemia and lymphoma regardless of the types of cancer with which they were diagnosed.

Information related to playing soccer allowed us to describe the reported individuals in terms of the durations, types of play (such as recreational or select soccer), fields, turf types, and positions (goalkeeper) played. Because of the concern about crumb rubber, information about other sports focused on sports played on artificial turf only.

The questionnaire was conducted as a telephone interview. The interviewer read the questions to participants and recorded their answers in writing. One minor provided information with parental approval, and one parent provided partial information for an adult. Otherwise, people ages 18 and older provided their own information, and parents provided information for children under 18 and for people who had died. Throughout the remainder of this report, the term "participants" refers to people who provided their own information and to children and deceased adults whose parents provided information. Appendix D provides a copy of the questionnaire.

Contacting individuals. The list provided by Coach Griffin included names of individuals with cancer, parents' names for children and young adults, email addresses for most people with cancer or their parents and some telephone numbers. We initially used a secure website at Public Health – Seattle & King County to email information about the investigation and invite participation. We sent second emails to those who did not respond to the initial invitation. We then focused on calling potential participants who seemed to meet the case definition and either had not responded to the emails or whose contact information from Coach Griffin did not include email addresses. We focused on contacting this group because information about these soccer players was essential for determining whether there was more cancer among the soccer players reported to the project team than in the general population. We did not attempt to contact one person who potentially met the case definition, because Coach Griffin indicated the individual did not want to be contacted. We used a combination of telephone numbers provided by the coach and numbers available through Whitepages® at whitepages.com.

Approach to interviewing. Many questions on the questionnaire lent themselves to a structured interview in which the interviewer read questions in exactly the same way to everyone without diverging from the set script. Information for some questions, however, was more easily gathered using a semi-structured approach. A semi-structured approach allows the interviewer to tailor questions exploring specific topics to each respondent.

The semi-structured approach worked especially well for collecting information on soccer playing, because respondents often found these questions difficult to answer. For example, some people had difficulty recalling details about early soccer play that occurred many years ago. Often, individuals reporting for themselves had been very young children at their earliest play. Thus, they might not have been aware of details such as lengths of seasons, field names, and the exact years or ages they started playing. Conversely, once children become more independent in their teens, parents might not be aware of details of soccer play. Even for more recent play, some questions were challenging. For example, reporting on

the overall percent of time players practiced on crumb rubber fields or the overall percent of indoor play was challenging when participants practiced at multiple fields throughout the year or different fields over a period of years.

The semi-structured approach allowed the interviewer to explore these questions with respondents, allowing reporting in a manner that made most sense to them. For example, while the questionnaire specifies years of beginning and ending play, the semi-structured approach allowed reporting of ages or grades in school rather than calendar years. As another example, the semi-structured approach permitted reporting the percentage of total practice on each field with artificial turf, rather than estimating an overall percentage of practice on artificial turf. The interviewer used the information collected from the semi-structured approach to compute and summarize responses, as needed. For example, if a respondent provided a grade in school or age rather than a year of beginning play, the interviewer assigned the year of beginning play based on birthdate and other information provided. Appendix E provides detail on computations.

Descriptive statistics. For the descriptive statistics, we included ranges of responses and medians. The range is the span of responses from the lowest to highest values. The median is the value that lies at the midpoint of a range. Medians are often similar to averages, but extremely high or low values can skew the average so that it does not provide a good representation of the entire group. The median is generally not affected by extreme values. We used functions in Microsoft Excel to determine ranges and medians.

Results

Meeting the Case Definition

Confirming a diagnosis of cancer. Information from the cancer registry on types of cancer, birthdate, and age and year at diagnosis was available for 48 of the 53 people reported to the project team. The cancer registry only includes Washington residents. Interviews for two of the remaining five individuals confirmed that they were not Washington State residents when they got cancer. A third person, whom we did not interview, was also likely to have been a resident of another state based on information from Coach Griffin. One person was diagnosed in mid-2016 and so cancer registry information was not available at the time of this writing. There was no apparent reason why the cancer registry had no information on the final person, whom we were unable to reach. We did not include this person as meeting the case definition, because the information from Coach Griffin did not clearly indicate whether this person met the age criteria.

Overall 27 people met the case definition and 26 did not. People did not meet the case definition for the following reasons:

- Age at diagnosis: 15 people were diagnosed after age 24, one person was diagnosed before age six and one person (described in the previous paragraph) might have been diagnosed after age 24.
- Soccer: Five people did not play soccer.
- Year of diagnosis: Three people were diagnosed before 2002 or after 2015.

- Residence: One person likely never lived in Washington.

Response Rate

We sent emails inviting participation in the investigation to 41 people for whom we had email addresses. This resulted in seven interviews for people who met the case definition and 10 individuals who did not. Our efforts then focused on obtaining interviews for the remaining people who were most likely to meet the case definition. This resulted in 18 additional interviews for people who met the case definition. Thus, in total, we obtained interviews for 35 of the 53 (66 percent) people reported to the project team and for 25 of the 27 (93 percent) people meeting the case definition.

Observed to Expected Ratios

Tables 7–10 provide information on the numbers of primary cancer diagnoses among people meeting the case definition (observed), the expected number of cancer diagnoses among soccer players given rates for Washington residents of the same ages during the same time period (expected), ratios of the observed to expected (O/E) numbers, and the 95 percent confidence intervals around the O/Es. All of the O/Es are less than one and in no instance does the 95 percent confidence interval include one. Based on these O/E ratios, we conclude that the number of cancer cases on the coach's list is much less than was expected to occur among soccer players in Washington.

Table 7. Observed to expected ratios for cancer: all soccer players diagnosed during 2002–2015 at ages 6–24 years

Cancer type	Observed cancers from the coach's list	Expected cancers	Ratio of observed to expected	95 percent confidence interval
All	28	1,384	0.03	0.01-0.03
Leukemia	6	131	0.05	0.02-0.10
Hodgkin lymphoma	5	147	0.03	0.01-0.08
Non-Hodgkin lymphoma	6	89	0.07	0.03-0.15

Table 8. Observed to expected ratios for cancer: WYS-defined select and premier level players diagnosed during 2002–2015 at ages 6–24 years

Cancer type	Observed cancers from the coach's list	Expected cancers	Ratio of observed to expected	95 percent confidence interval
All	15	284	0.05	0.03-0.09
Leukemia	3	26	0.11	0.02-0.33
Hodgkin lymphoma	4	30	0.13	0.04-0.34
Non-Hodgkin lymphoma	4	18	0.22	0.06-0.56

Table 9. Observed to expected ratios for cancer: goalkeepers diagnosed during 2002–2015 at ages 10–24 years

Cancer type	Observed cancers from the coach's list	Expected cancers	Ratio of observed to expected	95 percent confidence interval
All	14	153	0.09	0.05-0.15
Leukemia	3	14	0.21	0.04-0.61
Hodgkin lymphoma	4	17	0.24	0.07-0.62
Non-Hodgkin lymphoma	2	10	0.20	0.02-0.73

Table 10. Observed to expected ratios for cancer by age group: all soccer players diagnosed during 2002–2015 at ages 6–24 years

Age at diagnosis	Observed cancers from the coach's list	Expected cancers	Ratio of observed to expected	95 percent confidence interval
All ages	28	1,384	0.03	0.01-0.03
6–9 years old	1	30	0.03	0.00-0.19
10–14 years old	12	180	0.07	0.03-0.12
15–19 years old	8	427	0.02	0.01-0.04
20–24 years old	7	747	0.01	0.00-0.02

Descriptive Epidemiology

For some factors in this portion of the report, we used information from the interviews, the cancer registry and reports to the project team. For other factors, however, the interviews provided the only source of information. In general, with all three sources of information, we could describe the 53 people reported to the project team. Interview information was available for 35 people.

Table 11. Sources of information for factors discussed in the descriptive epidemiology section

Factors with information from interviews, cancer registry and reports to the project team
Demographics
Birth year
Gender
Cancer diagnosis
Type of cancer
Year of diagnosis
Age at diagnosis
Primary sport played
Factors with information from interviews
Demographics
Race
Hispanic ethnicity
Residence
All medical, health-related and familial risk factors for cancer
All aspects of soccer play
Other sports played among soccer players

Demographic Characteristics

Birth years for the 53 people reported to the project team ranged from the 1960s to the 2000s. The range for people meeting the case definition was narrower—early 1980s to the mid-2000s—due to the case definition's criteria for age and year of diagnosis. During 1983–2010, about 28 percent of soccer players on WYS teams were female.[80] Thus, the number of females reported to the project team, 50 percent overall and 63 percent for those meeting the case definition, is greater than we would expect from random variation alone. In 2015, the 73 percent of state residents reported their race and ethnicity as non-Hispanic white.[84] The percentage of participants who classified themselves or their children as non-Hispanic white is similar to that of Washington residents overall after accounting for random variation.

Table 12. Demographic factors

	53 people reported to the project team[a]	**27 people who met the case definition**
Year of birth[a]		
Range	1961-2008	1980-2005
Median	1991	1994
Gender: number (percent in parentheses)[a]		
Female	26 (50)	17 (63)
Male	26 (50)	10 (37)
Race and Hispanic ethnicity: number (percent)		
	35 interviews	**25 people who met the case definition**
Non-Hispanic white	28 (80)	19 (76)
Other including more than one race	7 (20)	6 (24)

[a] Information on this factor was missing for one person.

Residence. The 35 interviews included many in- and out-of-state residences from birth to getting cancer, including living abroad. Only one participant, however, lived primarily in Eastern Washington before getting cancer. Considering only residences in Washington State, 20 participants lived exclusively or mostly in King County; eight in Pierce County; two each in Kitsap and Snohomish counties; and one each in Skagit, Thurston and Spokane counties.

Cancer Diagnoses

Types of cancer. Most of the 53 people had one primary type of cancer. Two people, however, had two primary cancers for a total of 55 cancers. The 10 cancers shown in Table 13 below as "other" include eight different types of cancer.

Table 13. Number of cancers among people on the coach's list by type of cancer

Type of cancer	55 total cancers among 53 individuals	28 cancers among 27 individuals meeting the case definition
Leukemia	11	6
Non-Hodgkin lymphoma	13	6
Hodgkin lymphoma	11	5
Bone (Ewing sarcoma or osteosarcoma)	4	3
Soft tissue sarcoma	3	2
Brain	3	1
Other	10	5

Years of diagnosis. Year of diagnosis was missing for one primary cancer. Figure 2 provides the years in which 54 cancers were diagnosed. The peaks in 2008 and 2014 are consistent with reports of healthcare providers mentioning to patients or their parents that there seemed to be a lot of cancer among soccer players and similar concerns arising from the public.

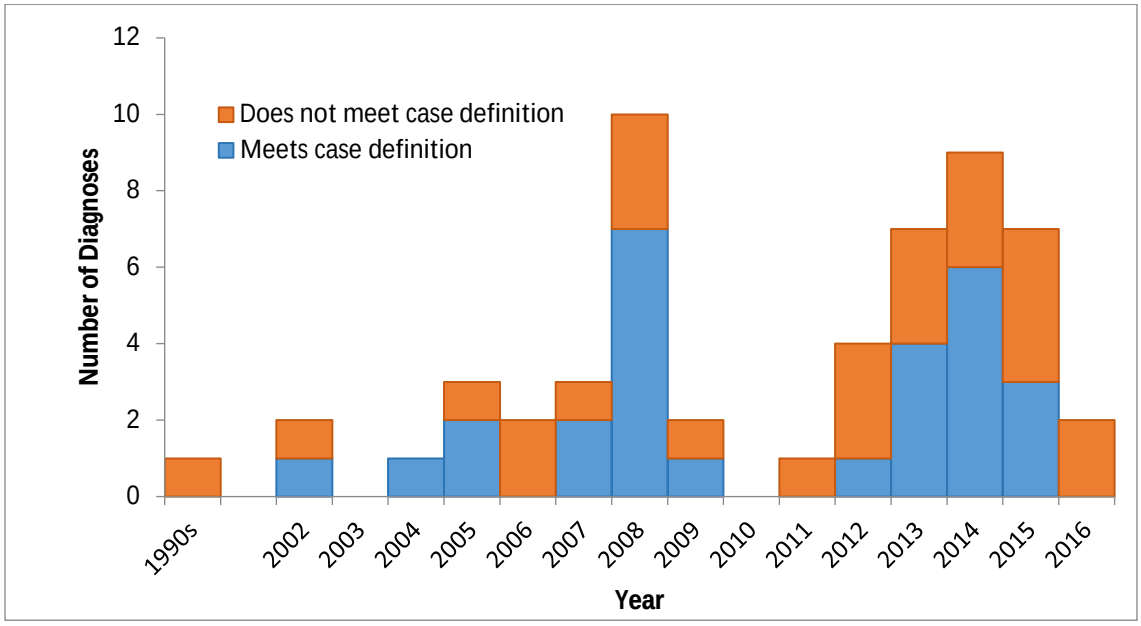

Figure 2. Year of cancer diagnosis among people on the coach's list

Ages at diagnosis. The 53 people ranged from under five to over 50 years old when first diagnosed with cancer. The median age was 18 years. Because the case definition included a diagnosis of cancer between six and 24 years old, the 27 people meeting the case definition had a narrower range of ages—nine to 23 years old—with a median of 15 years.

Known or Suspected Potential Risk Factors for Cancer

Nineteen of the 35 interviews contained no reports of potential risk factors for cancer. Nine participants had one potential risk factor: one report of infectious mononucleosis, one chronic condition, two CT scans not connected to the cancer diagnosis, and five biological parents who had cancer. Four participants had two or more potential risk factors. Three respondents were not sure about CT scans. Two of these respondents reported no other risk factors, and one was also unsure of autoimmune disease.

Biological parents of three participants had melanoma in their mid-20s. The remaining four parents had four different types of cancer in their 50s, 60s, or 70s. No interviews indicated cancer among biological sisters or brothers.

Table 14. Number of reports of risk factors for cancer

Cancer risk factor	**35 interviews**	**25 people who met the case definition**
History of smoking	2	1
Autoimmune disease[a]	0	0
Hepatitis B	0	0
Mononucleosis (Epstein Barr virus)	3	1
Human immunodeficiency virus (HIV)	0	0
Chronic disease[b]	4	4
Computer tomography (CT) scan[c]	5	4
Parent or sibling with cancer	7	4

[a] Two people were not sure and information was missing for one person.

[b] Information was missing for two people.

[c] Three participants, who each reported a possible CT scan, were either unsure that the scan was CT or unsure if the scan was done to diagnose cancer.

Soccer Playing

We present information on reported soccer play prior to initial diagnosis. Because the case definition included playing soccer, the 25 participants who met the case definition played soccer. Eight of the 10 participants who did not meet the case definition played soccer. Thus, information for soccer play was available for 33 people overall and 25 people who met the case definition.

Amount of Play. The shortest amount of soccer play reported was one season for one year, but about half of the players played soccer for at least 10 years before getting cancer. Two participants started playing soccer in preschool and one person did not play soccer until their 40s. Of the remaining 30 players, 17 began playing soccer at ages four to six, nine at ages seven to nine, and four at ages 10 to 12. About two-thirds of the soccer players were still playing when they got cancer, resulting in a median of zero years between ending soccer play and getting cancer. One-third had stopped playing soccer for about one to nine years before getting cancer.

Table 15. Years of soccer play in relation to cancer diagnosis

	33 interviews with reported soccer play	25 people meeting the case definition
Total years of playing soccer before diagnosis		
Range	1-30	1-17
Median	10	9
Years between first playing soccer and diagnosis		
Range	3-43	3-18
Median	10	9
Years between ending soccer play and diagnosis		
Range	0-9	0-9
Median	0	0

Washington Youth Soccer levels of play. The WYS defines recreational, select and premier soccer play. Most commonly, children first play on recreational teams and move to select or premier teams depending on their abilities and interest. In general, players on select or premier teams play soccer more days of the week and more months of the year than recreational-level players. Several participants reported playing with the Catholic Youth Organization (CYO). We classified CYO as recreational soccer, because they seem to follow the recreational season, with less total time throughout the year playing soccer. We included the Olympic Development Program as premier play based on information at the WYS website.[85]

Table 16. Washington Youth Soccer defined levels of soccer play

Level of Washington Youth Soccer play	33 interviews with reported soccer play	25 individuals meeting the case definition
Recreational only	11	10
Recreational and select	11	9
Recreational and premier	5	4
Recreational, premier and select	4	2
No recreational, select or premier play	2	0

Table 17. Selected characteristics of play by Washington Youth Soccer -defined levels of soccer play

Level	Number of players[a]	Median age of beginning play	Median years played	Median months of play per year
33 total soccer players				
Recreational	31	6	5	3
Select	15	11	3	9.5
Premier	9	12	4	9
25 who meet the case definition				
Recreational	25	6	5.5	3
Select	11	10	3.5	10
Premier	6	11	6	9

[a] Because information is provided by level of play, soccer players who played at more than one level are included more than once.

Overall, the 31 soccer players who played at WYS-defined levels played for a total of 276 years. This includes 176 years of recreational-level play and 100 years of play at select and premier levels. Thus, about 36 percent of the WYS play described in the interviews was at the select or premier levels.

School teams. Some participants' middle schools did not offer team soccer. Additionally, if participants got cancer before entering a given level of school, we did not ask about soccer playing for that level of school or schools with higher grades. For example, if someone got cancer in middle school, we did not ask about high school or college teams. To determine the number of participants potentially able to play on a school team, we used information directly from the interview when available. For example, someone might report getting cancer in middle school and so, for this investigation, we would not include play on high school or college teams. If participants did not provide grades in school when they got cancer, we considered players eligible to play middle school soccer pre-diagnosis if they got cancer after age 11, eligible for high school soccer if they got cancer after age 13, and eligible for college soccer if they got cancer after age 17. Overall, 29 participants were eligible to play on middle school teams, 20 on high school teams and 15 on college teams. Among people meeting the case definition 22 were eligible for middle school, 13 for high school and eight for college teams.

The season for most middle and high school soccer was about two to four months. Most of the school players also played WYS soccer either concurrently with school soccer or during the schools' off-seasons. College play ran from about nine or 10 months to 12 months of the year.

Table 18. Numbers of soccer players on middle school, high school and college teams.

Level of school soccer	29 participants overall[a]	22 participants meeting the case definition[a]
Middle school only	5	5
High school only	8	5
College only	1	0
Middle and high school	1	1
High school and college	4	3
Middle school, high school and college	2	2
No school-related play	8	6

[a] Overall, four people got cancer before middle school and so were not eligible for any school-related play pre-diagnosis. Three of these people met the case definition.

Other soccer play. Fifteen individuals—eight of whom met the case definition—got cancer at ages 18 or older. We considered them eligible to play organized adult recreational soccer, such as co-ed and indoor leagues with games only. Eight of these participants, including one who met the case definition, played organized adult recreational soccer. Four of the seven who did not play on adult recreational teams continued playing as adults on college teams.

Other types of play included two participants who played preschool soccer, five reports of summer camps or weekend workshops, and four reports of pick-up games. Five respondents noted a variety of other games, including one outdoor tournament and four who noted a

variety of indoor games that did not fit into other categories. Most soccer that was not part of WYS, school teams or organized adult recreational play was of relatively short duration, but several participants played pick-up games for seven to 12 months per year for two to six years.

Goalkeeper. For each level of play, respondents reported the amount of time participants played goalie. We classified select and premier players as goalies if they played the position at least 50 percent of the time, but most players classified as goalkeepers played the position for at least 90 percent of the time. Most select and premier players not classified as goalies played the position for less than 10 percent of the time.

The WYS rules state that there are no goalkeepers until "under 9" teams.[86] This means that for the most part goalkeeping begins at age eight. At age eight and older, however, recreational players often rotate through positions before becoming more specialized. Thus, the same overall percentage of time playing goalkeeper on WYS-defined recreational teams can have a variety of meanings. For example, it could mean that someone rotated through the position with limited goalkeeping practice. It could also mean that players never played the position in their early years of play, but primarily played and practiced as goalkeeper in later years. Thus, we classified people as playing goalkeeper at the recreational level if they played the position at least 25 percent of the time overall. We also included one person who did not play goalkeeper for the first six years, but played the position 30–50 percent of the time for the final three years of recreational play. Of the 10 participants classified as goalies at the recreational level, seven played the position at least 50 percent of the time and all played recreational level soccer at ages eight or older. Nineteen of the 20 recreational players not categorized as goalkeepers played the position at most 10 percent of the time overall; one participant played the position 20 percent overall. Information on goalkeepers was missing for one participant who played recreational level soccer.

Table 19. Number of goalkeepers by levels and types of play

Level of Play	32[a] participants with reported soccer play	24[a] players meeting the case definition
Any level of play	16	12
Recreational	10	9
Select, premier or both	10	8
Middle school	3	3
High school	9	6
College	4	3
Adult recreational	5	1

[a] Information on playing goalie was missing for one person. Players who played goalkeeper at more than one level are counted more than once.

Overall, about half of the participants were categorized as goalkeepers. Most goalkeepers played the position for many years for considerable proportions of their play. All of the players categorized as goalkeepers in middle and high school were also goalies at WYS-defined levels of play. College goalkeepers also played goalkeeper at WYS-defined levels of play, pre-college school teams, or both. The 31 soccer players, who played at the WYS-

defined levels of play, played for a total of about 218 years beginning at ages eight or older. Players spent about half of those years (112) playing goalkeeper.

Names of fields. One respondent did not provide names of fields. Thirty-two respondents provided the names of about 110 fields in Washington at which participants practiced soccer. They also noted 11 fields located out-of-state. Most fields were reported by one or two respondents only, but five practice fields were each reported for three participants and two practice fields were each reported for four participants. We did not collect field names for games, because home games are often played on the same fields as the practice fields and away games are played in many different locations.

Types of surfaces. The initial eight interviews—five for people who met the case definition—asked about playing soccer on artificial turf, but did not ask about the specific type of artificial surface. Nonetheless, two of the initial respondents provided information on the types of artificial surfaces. Interviews after the initial eight systematically collected this information.

If participants reported artificial turf, but did not know what type of artificial turf, the interviewer asked whether the surface resulted in their having little black balls in their shoes or clothes. Little black balls in shoes and clothes are commonly reported by soccer players who play on fields with crumb rubber infill. Artificial turf was classified as crumb rubber if participants answered "yes" and as non-crumb rubber if they answered "no."

An interview for one person did not contain any field-related information. Thus, information classifying fields as artificial turf, without specifying the type of turf, was available for 32 of the 33 soccer players, of whom 24 met the case definition. Due to the initial interviews not asking about the types of artificial surfaces, one person who did not know the types of surfaces and one person who played soccer only on grass, information on specific types of artificial turf was available for 24 participants overall and 18 who met the case definition.

Grass and artificial turf with crumb rubber infill were the most frequently reported field surfaces. Other surfaces included dirt, AstroTurf®, and red rock cinder. AstroTurf® is a brand of artificial turf, but the term is also used generically. The interview did not distinguish between the brand name and the generic use of the term. The earliest AstroTurf® fields did not include crumb rubber infill, but fields installed more recently are likely to have crumb rubber infill. Four participants reported playing on "astroturf" between 1982 and 2010.

Practicing or playing games for some portion of the time on fields with crumb rubber infill was reported for 22 of the 24 soccer players for whom at least partial detailed information was available. The two participants who did not report playing on crumb rubber participated in the early interviews and so were not specifically asked about the type of surface. They might have played on crumb rubber. These participants specified "astroturf" for some, but not all, artificial turf fields. This might imply that artificial turf not specified as "astroturf" was, in fact, crumb rubber.

Another eight soccer players played on artificial turf but participants either did not know or were not asked the specific type of turf. Seven of these eight players played on artificial turf

after 2005 and so it is likely that they played on crumb rubber. The eighth person might also have played on crumb rubber. This participant specified “non-crumb rubber” for some, but not all, artificial turf fields. This might imply that artificial turf not specified as “non-crumb rubber,” was, in fact, crumb rubber. Thus, it is likely that 31 of the 32 soccer players for whom field information was available, had some exposure to crumb rubber through soccer.

The one soccer player who played soccer only on grass fields, played another sport on crumb rubber for several years. The two participants who did not play soccer reported exposure to crumb rubber fields through other activities.

We calculated the percentage of time athletes played soccer on grass or dirt, outdoor artificial turf, and indoor artificial turf fields. We made separate calculations for practice and games. The percentages combine all types of artificial turf and all types of play: WYS-defined levels, school play, adult recreational play and other types of play. We calculated these percentages in five percentage point increments. Appendix E provides information about the calculations.

Tables 20 and 21 show the number of players who practiced and played games on grass or dirt, outdoor artificial turf and indoor artificial turf by specified percentages of time. The tables also provide the median percentage of time playing on each surface type.

Table 20. Field surfaces used for practice

Percentage of time spent on selected types of fields	Number (percent) of 31[a] players with reported soccer practice	Number (percent) of 24[a] players meeting the case definition
Grass or dirt		
75–100	14 (45%)	8 (33%)
50– <75	12 (39%)	11 (46%)
25– <50	3 (10%)	3 (13%)
0– <25	2 (6%)	2 (8%)
Median percent of time on grass	70-74	65-69
Outdoor artificial turf		
75–100	2 (6%)	2 (8%)
50– <75	3 (10%)	3 (13%)
25– <50	10 (32%)	9 (38%)
0– <25	16 (52%)	10 (42%)
Median percent of time on outdoor artificial turf	20-24	30-34
Indoor artificial turf		
75–100	0 (0%)	0 (0%)
50– <75	0 (0%)	0 (0%)
25– <50	1 (3%)	1 (4%)
0– <25	30 (97%)	23 (96%)
Median percent of time on indoor artificial turf	0-4	0-4

[a]Field-related information was missing for one person and one person reported all soccer playing as games only and thus, did not provide information for practice.

Table 21. Surfaces during games

Percentage of time spent on selected types of fields	Number (percent) of 31[a] players with reported soccer practice	Number (percent) of 23[a] players meeting the case definition
Grass or dirt		
75–100	4 (13%)	3 (13%)
50– <75	16 (52%)	12 (52%)
25– <50	7 (23%)	5 (22%)
0– <25	4 (13%)	3 (13%)
Median percent of time on grass field	60-64	60-64
Outdoor artificial turf		
75–100	3 (10%)	2 (9%)
50– <75	5 (16%)	4 (17%)
25– <50	15 (48%)	11 (48%)
0– <25	8 (26%)	6 (26%)
Median percent of time on outdoor artificial turf	35-39	35-39
Indoor artificial turf		
75–100	0 (0%)	0 (0%)
50– <75	1 (3%)	1 (4%)
25– <50	0 (0%)	0 (0%)
0– <25	30 (97%)	22 (96%)
Median percent of time on indoor artificial turf	0-4	0-4

[a]Field-related information was missing for one person; information on field surfaces for games was missing for one person.

The tables combine grass and dirt, but almost all of the participants played on grass with very little play on dirt. While all but one soccer player practiced, played games or both for some of the time on artificial turf, most players practiced and played games primarily on grass. Overall, half of the players practiced at least 70–74 percent of the time on grass or dirt, less than 25 percent of the time on outdoor artificial turf and zero to four percent of the time on indoor artificial turf. Games were more likely than practice to be on artificial turf, but most games were on grass.

Other Sports

Seven—including four meeting the case definition—of the 35 participants reported they or their children played other organized sports on artificial turf. Sports included fast pitch, softball, baseball, football, ultimate Frisbee and track. Most of this play was reported on fields with crumb rubber infill, but one person reported a clay surface.

Comments

Sixteen people responded to an open-ended question about whether there was anything that might be important in relation to their or their children's cancer that had not been covered in the interview. Most of these comments focused on crumb rubber including:

- The large quantity of little black balls or black pellets that were brought home in shoes, gloves, socks and clothes and tracked into the house.

- The large quantity of the pellets that got in the nose and mouth and were then inhaled or swallowed, especially for goalkeepers.
- Getting the pellets in the eyes and open cuts or embedded in the skin.
- The large amount of time spent on surfaces with crumb rubber infill, including spending time at very young ages during organized activities for toddlers, accompanying parents or siblings to sports fields, or during "free" play on surfaces with crumb rubber fill.
- Concerns about increased exposures during extreme heat.

Several participants mentioned concerns about exposure to pesticides from a variety of sources including playing on grass fields and international travel. Several also reported that other team members, coaches or parents of team members got cancer during the same time periods as they or their children.

A few participants mentioned non-crumb rubber potential causes for their or their children's cancers that were unique to their situation or from information given to them by their healthcare providers.

Discussion

Study Design

This investigation was prompted by perceptions of an unusually high number of soccer players, especially goalkeepers, with cancer. We did not find the number of cancers among soccer players, select and premier players, or goalkeepers on the coach's list to be higher than expected based on Washington cancer rates for people of the same ages.

The investigation was not designed to discover the causes of cancer among the people reported to the project team. One common type of study to find causes of cancer involves comparing people who already have cancer, as was the case in this investigation, to people who do not have cancer. These studies try to determine whether exposures or other factors are different among people who have and do not have cancer. These studies usually involve hundreds of people with the same type of cancer, as well as people who do not have cancer. They generally focus on relatively narrow age ranges, especially when searching for causes of cancer among children and adolescents. These types of studies often involve lengthy interviews, as well as biological measures, environmental measures or both. Because biological factors often change after the onset of cancer and both biological and environmental factors can change over time, studies that make these measurements usually include only people diagnosed relatively recently. Academic or research organizations specializing in cancer are generally best suited to conducting these types of study.

Thus, even narrowing the list of people reported to those meeting the case definition, we could not design this type of study. The reports included people with many different types of cancer who were diagnosed over a 14-year period beginning in 2002. Additionally, the number of soccer players with cancer, while perhaps large for a small, well-defined community, is still too small for a study aimed at investigating the causes of cancer.

This investigation was also not designed to add to our understanding of the risks or benefits of crumb rubber fields. Basic research looking at whether substances cause cancer often begins with laboratory studies using animals or cell-culture systems. Most laboratory toxicological studies are conducted on a single chemical at a time. Therefore, in situations where exposure to numerous chemicals is of concern, such as with artificial turf and crumb rubber, standard toxicological studies can rarely provide adequate information. We were unable to find any studies that investigated the effects of simultaneous exposure to the dozens of chemicals that have been identified in artificial turf crumb rubber. Additionally, potential exposures from crumb rubber likely vary depending on factors such as the source and processing of the rubber; the age of the crumb rubber; characteristics of the field, such as ambient temperature and the amount of ventilation; and the route of exposure, such as swallowing entire crumbs, having crumb rubber embedded under the skin, absorption through the skin or open cuts, rubbing the eyes, or inhaling substances that off-gas from the crumb rubber.

Our review of the scientific and medical literature found a number of risk factors that most scientific, medical and other cancer-related organizations accept as known risk factors for childhood leukemia, lymphoma or both. Studies also suggest possible additional risk factors, some of which may be related to chemicals found in artificial turf. For example, solvent exposures are associated with the development of leukemia, and benzene is a solvent that has been detected in crumb rubber infill. However, while benzene may be present in crumb rubber, not all crumb rubber necessarily contains benzene, and people can be exposed to benzene from a variety of common sources such as gas stations, industrial emissions, glues or paints. Further, even if benzene is present in crumb rubber, it might not be at a level sufficiently high to cause cancer. The International Agency for Research on Cancer has classified the rubber manufacturing process as causing leukemia and lymphoma and other types of cancer in people.[87] However, occupational exposures during manufacturing are usually different from exposures to a finished product. Thus, it is unlikely that athletes playing on crumb rubber infill are exposed to the same toxicants, at the same levels and by the same routes of exposure as workers in rubber manufacturing facilities.

Benzene is an example of one potential chemical exposure from crumb rubber. The recent literature review from the multi-agency federal status report identified research gaps related to numerous chemicals and potential exposure pathways. The report also described federal research that is being undertaken to better characterize the components of crumb rubber infill made from tires. This research includes samples from 40 synthetic turf fields across the United States. In addition, the report describes ongoing research to better understand how people may be exposed to chemicals from crumb rubber infill.[78] Researchers in California are also engaged in characterizing potential exposures from turf fields and playground mats under a variety of climate conditions and ages of field. The California research further seeks to understand potential exposures based on human activities and to develop methods for monitoring chemicals of interest in people exposed to crumb rubber.[88]

Observed to Expected Ratios

Our investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf. Rather, its

purpose was to explore whether the information from Coach Griffin's list warranted further public health response. We found that the number of cancers on the coach's list was less than was expected to occur among soccer players in Washington. This was true for soccer players on the coach's list playing at all WYS-defined levels combined, for those who played at select and premier levels and for those who played goalkeeper.

The list from Coach Griffin likely undercounted the number of Washington soccer players who got cancer during 2002–2015 at ages six to 24 years. That is, our observed number of people who met the case definition was likely fewer than the true number. Initially, we considered that we may have identified all or nearly all of the soccer players with cancer because of the breadth of the news reports and publicity in fall 2014. After the interviews, which showed disproportionate numbers of females, people from King County, select and premier players, and goalkeepers, we considered this less likely. However, with 28 cancers observed and 1,384 expected, we would need to have missed hundreds of people who met the case definition to change our conclusion.

Excluding people who did not meet the case definition from the observed number of cancers reduced the total number of observed cancers. These restrictions, however, also resulted in fewer expected cancers. For example, if we had included the one cancer diagnosed in the mid-1990s, we would have added one cancer to the observed number, while adding hundreds of thousands of person-years to the calculation of the expected number of cancers. Thus, expanding the case definition to include cancers among all people reported to the project team would have most likely resulted in even smaller O/Es than those computed.

Our computations for goalkeeper were restricted to players ages 10–24. Because all goalkeepers who played the position before age 10 also played at older ages, we did not exclude any of the cancers among goalkeepers from the observed number of cancers. If we had included goalkeepers beginning at age eight, our observed number of cancers would not have changed. The expected number of cancers, however, would have increased as we added person-years at risk for goalkeepers ages eight and nine. Thus, expanding the computations to goalkeepers beginning at age eight would have resulted in even smaller O/Es than those computed.

In addition to underestimating the observed number of cancers, this investigation may have over- or underestimated the number of cancers expected among soccer players based on cancer rates among Washington residents ages six to 24 years. Over- or underestimation could have occurred by not correctly defining the number of residents ages six to 24 years who play soccer. We used a readily available data source (WYS) to determine the number of Washington residents who play soccer. We recognize that this source likely does not account for all soccer players and may exclude those playing exclusively for Catholic Youth Organizations, summer camps, private instruction or other groups that do not require a youth soccer "player card" for participation.

Over- or underestimation of the number of soccer players expected to get cancer might also have occurred if our four assumptions about soccer players were not accurate. Two of these assumptions—everyone who played on a soccer team at age 16 and older had also played at a younger age and middle and high school players also played on a WYS team at some

point—are accurate for the 25 players with interviews who met the case definition. These assumptions, however, might not be accurate for all youth soccer players in Washington.

We have no direct way to check our assumption that the distribution of players by age was the same from 1983–1999 as in 2000. The WYS counts of players by age from 2000–2015 showed small changes in the distribution of players by age from year to year. For example, each year for all 16 years, about one to three percent of players were six years old, nine to 11 percent were 10 years old, and two to four percent were 16 years old. If this same pattern is true for earlier years, inaccuracies in the assumption about the distribution of players by age would not introduce large errors.

We assumed a 10 percent turnover in players each year. This assumption was needed to count how many people played soccer. We did not have a roster of individual players; we only had the total number of players registered each year. Assuming that some players stopped and new players started (that is, there is turnover) each year leads to a higher estimate of the number of different players than assuming no turnover. The larger the turnover, the larger the number of soccer players. Thus, if turnover was more than 10 percent, we underestimated the number of soccer players; if it is less than 10 percent, we overestimated the number.

The 1,384 expected cancers might seem high. From 2002–2013 there were almost 5,000 cancer diagnoses among Washington residents ages six to 24 years. Registry data for 2014 and 2015 are not yet complete, but there likely have been about 900 diagnoses in 2014–2015 given an average of 439 such diagnoses each year during 2009–2013. Thus, we estimate a total of about 5,900 cancers diagnosed among Washington residents ages six to 24 years during 2002–2015. The 1,384 expected cancers is about 23 percent of the total cancers. Although less than 23 percent of the population ages six to 24 years plays soccer each year, once someone begins playing soccer, they contribute to the person years at-risk whether or not they continue playing. Using the assumption of a 10 percent turnover each year, we can estimate the number of children in each age group who ever played soccer. For example, using the WYS enrollment data, we estimated that 22,827 of the children who turned age 15 in 2015 played soccer for at least one year between ages six and 15. The estimated number of 15-year-olds living in Washington in 2015 was 89,944.[89] Thus, about 25 percent of 15-year-olds played soccer at some point in their lives

It is not possible to determine the net effect of potential inaccuracies from these four assumptions. If we overestimated the number of soccer players in Washington, we would also overestimate the expected number of cancers and underestimate the O/E ratios. For example, we observed 28 cancers altogether and expected 1,384 based on our estimated number of players and cancer rates in Washington. The O/E ratio was 0.03. If there were only half the number of players (and assuming the same age distributions), we would expect half the number of cancers or 692. The O/E ratio would then be 28/692 or 0.04, which is still very small and does not indicate an increased risk of cancer among the soccer players on the coach's list. Even with half the expected number of soccer players, we would need hundreds of additional observed cancers for the number of cancers among soccer players to approach the expected number.

Conversely, if we underestimated the number of soccer players and there were really more soccer players than we estimated, the number of expected cancers would increase and the O/E would become even smaller. This would happen if, for example, there is more than 10 percent turnover each year.

We did not calculate observed to expected ratios for select and premier goalkeepers. We did not do this because we could not adequately compute the expected number of cancers among this population. We did not have information on the number of premier and select goalkeepers by age and year of play.

Descriptive Epidemiology

Demographics. The findings for all participants for whom we had interviews were similar to the findings for participants who met the case definition. This is not surprising given that 71 percent of the interviews were for participants who met the case definition. Unless otherwise noted, the information discussed below is for the group as a whole.

The interview information was collected to help determine who met the case definition, who played goalkeeper, who played at WYS-defined select and premier levels and whether players had commonalities that might be unusual. Soccer-related information might also help researchers design studies to measure potential exposures from artificial turf soccer fields. We were not able to present the descriptive information by type of cancer, because numbers of specific types of cancer were too small for this to be meaningful. Overall, there were 11 diagnoses of leukemia, 13 of non-Hodgkin lymphoma, 11 of Hodgkin lymphoma and 20 of 11 other types of cancer. Interviews with information on soccer playing were available for eight of the leukemias, six of the non-Hodgkin lymphomas, eight of the Hodgkin lymphomas and 10 of the other types of cancer.

The percentages of females and males reported to the project team and the percentages of people by race and Hispanic ethnicity were similar to those of Washington residents overall after accounting for random variation.[84] However, we do not know the percentages of people by race and Hispanic ethnicity who play soccer. If these differ from those of the general Washington population, we could have missed an unusual pattern.

During 1983–2010, about 28 percent of soccer players on WYS teams were female.[80] Thus, there were more females reported to the project team—overall and among those meeting the case definition—than we would expect due to random variation. This remains true even though rates of cancer during 2009–2013 were slightly higher among Washington's female population ages six to 24 years (24.6 per 100,000 females) than among males (22.6 per 100,000 males).[2] The disproportionate number of females reported to the project team might have resulted from missing male soccer players with cancer. Coach Griffin is a woman's soccer coach and so might have had more contact with female players. There might also be other reasons for the disproportionate number of females. For example, women might be more comfortable than men with sharing medical information. Thus, the descriptive portions of this investigation might reflect soccer play and other factors among females more accurately than among males.

While living in Washington, 34 of the 35 interviews (97 percent) indicated participants living primarily in the western part of the state. Only one person living in Eastern Washington might seem unusual given that the project team understood that reports were from all of Washington. The 97 percent, however, does not differ from what we would expect due to random variation. U.S. Census data for 2000 and 2010 show about 78 percent of the total state population and about 75 percent of the population ages five to 24 years living in counties west of the Cascades.[90]

The same U.S. Census data show about 29 percent of Washington residents and 26 percent of residents ages five to 24 years living in King County. Twenty of the 35 participants (57 percent) living primarily in King County is more than we would expect due to random variation. However, as with race and ethnicity, we do not know the overall proportions of soccer players by county. If a larger proportion of King County residents plays soccer compared to residents of other counties, the large proportion of participants from King County might not be usual. Alternatively, the large proportion of participants from King County might be due to other factors, such as the issue possibly receiving more media attention in King County compared to other counties or the coach being more likely to know of soccer players with cancer from King County. As with the disproportionate number of females, more participants than expected from King County is unlikely to affect our conclusion about the O/E ratios, but the descriptive portions of this report might better reflect soccer playing and other factors among residents of King County than among residents of other counties.

Cancer. The 53 people reported to the project team had about 13 different types of cancer depending on how cancers are categorized. For example, we could count lymphoma as one type of cancer with non-Hodgkin and Hodgkin being two subtypes, or we could count non-Hodgkin and Hodgkin as two separate types of cancer. Within the 13 types of cancer, there were many more distinct subtypes of cancer. That leukemia and lymphoma were the types of cancer most frequently diagnosed in this group is not surprising given that leukemia and lymphoma are among the most commonly diagnosed types of cancer in children and young adults in the United States.

Despite years of study, the causes of most cancers remain unknown. The interviews conducted for this investigation included some known risk factors for leukemia and lymphoma and some more general risk factors, such as smoking and family history of cancer. The Epstein-Barr virus and CT scans might have played roles in the development of some cancers, but it is difficult to know.

Epstein-Barr virus causes infectious mononucleosis and increases risk of lymphoma. Two participants who had infectious mononucleosis also developed Hodgkin lymphoma and another participant with infectious mononucleosis developed non-Hodgkin lymphoma. Developing any type of lymphoma after infection with Epstein-Barr virus is rare.[91] In the United States, Epstein-Barr virus is most closely associated with development of non-Hodgkin lymphoma among people who also have HIV.[92]

The National Cancer Institute reports that for every 10,000 children who receive a single CT scan, one will develop cancer at some point during their life. [93] Children who have multiple CT

scans before the age of 15 are at higher risk of developing CT-related cancers in the decade after the scans.[93] Of the eight people who had or might have had CT scans, only one had multiple scans before age 15 years. The remaining seven each had a single scan with three having had the scan when they were 15 years or older.

The remaining risk factors were unlikely to have increased risk for the specific cancers reported: people who smoked did not develop types of cancer known to be associated with smoking; types of cancer among biological parents were not related to the types of cancer among the participants; and the chronic diseases were not those related to development of cancer.

Soccer. The soccer information describes the experience of the 33 interviewed participants, all of whom got cancer. We do not know whether these experiences are unusual, because we do not have information from soccer players who did not get cancer. The cumulative amount of soccer play ranged from one season for one year to year-round for more than a decade. Most players played at least 10 years, but we do not know whether the cumulative amount of play is higher, lower or the same as for soccer players overall.

The 31 soccer players, who played at WYS-defined levels, played for a total of 276 years. This includes 176 years of recreational-level play and 100 years of play at select and premier levels. Thus, about 36 percent of the WYS play described in the interviews was at the select or premier levels. Given the WYS report of 20 percent of their enrollees playing at these levels,[81] select and premier players appear to be overrepresented in the reports to the project team. This is also true for goalkeepers. The 31 soccer players, who played at the WYS-defined levels of play, played for a total of about 218 years beginning at ages eight or older. Players spent about half of those years (112) playing goalkeeper. This proportion is high compared to the estimate of about 10 percent of players as goalies annually.[82]

The large percentages of select and premier players and goalies might have resulted from Coach Griffin having more contact with these groups than with other soccer players. Nonetheless, we do not feel that additional response by the health department is needed at this time to further explore whether select and premier players or goalkeepers are at increased risk for cancer given the very low O/E ratios among select and premier players and among goalies from the coach's list.

About a third of the participants were unable to list all practice fields for some levels of play. Additionally, because names of fields can change, we might have missed some fields that players had in common. Thus, the number of fields or the number of participants playing at the same field might be greater than reported. Nonetheless, the diversity of practice fields argues against implicating any particular field in this potential cancer cluster.

Of the 24 respondents who provided detailed information on type of artificial turf, 22 noted fields with crumb rubber infill for some portions of soccer practice or competitive games. A few of the reports might have been coded to crumb rubber incorrectly, due to the interviewer classifying all reports of "little black balls" as crumb rubber. Other surfaces, such as those with coated sand infill, might look similar. We expect this happened rarely and does not significantly affect results. That most players had exposure to crumb rubber through

soccer is not surprising given the ubiquity of crumb rubber infill. All of these 24 participants also played on grass fields and several played on other types of artificial turf.

Although most play was outdoors on grass or dirt fields, participants who played a lot of soccer could still have substantial amounts of time on indoor artificial turf. For example, participants who played year round might play outdoors for three seasons and indoors during the winter. If the seasons were of equal length, overall, about 25 percent of their play would be on indoor artificial turf. This average masks the two to three months of play exclusively on indoor artificial turf that could occur for several years. Likewise, participants could play almost entirely outdoors for years and then play for several years exclusively indoors. Playing outdoors for six years and indoors for two years, for example, averages to 25 percent of time indoors. This average masks the two years of 100 percent indoor play. Thus, while the averages provide a summary description of the types of surfaces on which participants played, researchers designing studies to measure exposures might need more detailed information.

Limitations of information in interviews. Limitations on information collected during the interviews resulted from the interview form, itself; the information collection process; respondents' difficulty in recalling details of soccer play and other information; and potential overrepresentation of females, people from King County, select and premier players and goalkeepers among those interviewed.

The interview form was not formally pilot-tested, but rather evolved over the first nine interviews. Thus, some of the first nine interviews did not include all of the information collected later, such as specific types of artificial surfaces and percent of play on indoor artificial surfaces. Additionally, the questionnaire did not cover the full array of risk factors for cancer. For example, we did not ask about potential exposures to the developing fetus even though many of those are known to affect one's risk of developing leukemia. We included some of the risk factors for leukemia and lymphoma, but we did not include risk factors that might have contributed to the development of other types of cancer. Thus, while few people reported known risk factors for leukemia and lymphoma, we might have missed other known risk factors, as well as risk factors for other types of cancer.

The interview was conducted as a paper and pencil survey and not a computer-assisted survey. With a computer-assisted survey, potential reporting inconsistencies—such as dates that resulted in unusual ages for grades in school—could possibly have been identified during the interview and corrected as needed. In a few instances, the interviewer inadvertently skipped questions. Computer-assisted interviews reduce the potential for skipping questions.

Respondent difficulty in recalling details of soccer play and other factors likely resulted in some inaccuracies in reporting. Minor inaccuracies that did not represent an extreme response would not have affected ranges of responses and most likely would not have large impacts on medians.

Finally, the overrepresentation of females, King County residents, select and premier players and goalies among those interviewed limits the ability to generalize information on soccer play obtained in the interviews to soccer players in general.

Conclusions and Recommendations

Our investigation was not designed to determine if soccer players in general were at increased risk of cancer due to exposures from crumb rubber in artificial turf. Rather, its purpose was to explore whether the information from Coach Griffin's list warranted further public health response.

This investigation did not find increased cancer among the soccer players on the coach's list compared to what would be expected based on rates of cancer among Washington residents of the same ages. This finding is true for all soccer players on the coach's list, as well as soccer players on the list at the WYS-defined select and premier levels, and goalkeepers on the list. The variety of fields and residences suggests that no specific field or geographic residence is problematic in terms of soccer players getting cancer.

In addition, the currently available research on the health effects of artificial turf does not suggest that artificial turf presents a significant public health risk. Assurances of safety, however, are limited by lack of adequate information on potential toxicity and exposure. The Washington State Department of Health will continue to monitor new research on health and environmental impacts of crumb rubber.

Thus, the Washington State Department of Health recommends that people who enjoy soccer continue to play irrespective of the type of field surface.

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Appendix A: Project Team

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Appendix B: Methods for Calculating Cancer Rates

Definition of types of cancer

Incidence. The Washington State Cancer Registry (WSCR) collects information on newly diagnosed cancers among Washington residents even if the individuals are diagnosed and treated out-of-state. The North American Association of Central Cancer Registries (NAACCR) and the Centers for Disease Control and Prevention (CDC) National Program of Cancer Registries review the WSCR incidence data annually for quality and completeness. The national program also conducts in-depth audits of WSCR on a regular basis. WSCR continues to be recognized by both organizations for the completeness (more than 95 percent complete) and quality of its data. The cancer registry uses the International Classification of Disease for Oncology Third Edition (ICD-O-3) primary site and histology codes information to determine types and subtypes of cancer, as does the National Surveillance, Epidemiology and End Results (SEER). We used SEER classifications to group types and subtypes of cancer into categories of leukemia, non-Hodgkin and Hodgkin lymphoma (http://seer.cancer.gov/siterecode/icdo3_dwhoheme/index.html).

The all cancers combined category included in situ and invasive cancers. In situ cancers have not invaded or penetrated surrounding tissues. The "in-situ" designation does not apply to leukemia, non-Hodgkin and Hodgkin lymphoma.

Cancer Type	International Classification of Diseases for Oncology Third Edition Codes
All cancers combined	C00.0 - C80.9, all histology codes
Leukemia	All sites with histology 9733, 9742, 9800-9810, 9820, 9826, 9831-9836, 9838-9948, 9963, 9964; for sites C420, C421 and C424 only, histology 9823, 9827, 9837, or 9811-9818
Non-Hodgkin lymphoma	All sites with histology 9590-9597, 9670-9729, 9735, 9737, 9738; and except for sites C420, C421 and C424 all other sites with histology 9823, 9827, 9837, or 9811-9818
Hodgkin lymphoma	All primary sites with histology codes 9650-9667

Death. The Washington State Department of Health, Center for Health Statistics collects information on causes of death for all Washington residents including those who die out of state. According to the National Center for Health Statistics, more than 99 percent of all deaths occurring in the United States are registered in the death certificate system. Accuracy of reporting specific causes of death varies since classification of disease conditions is a medical-legal opinion subject to the best information available to the physician, medical examiner, or coroner certifying the cause of death. Causes of death in Washington and nationally are coded to the International Classification of Diseases 9th Revision for deaths during 1980–1998 and 10th Revision for deaths beginning in 1999. This change in coding did not have substantively affect classification of deaths due to cancer. Following SEER standards, we used the following ICD-9 and ICD-10 codes. (http://seer.cancer.gov/codrecode/1969+_d04162012/index.html).

	International Classification of Diseases	
Cancer Type	**Revision 9**	**Revision 10**
All cancers combined	140-208, 238.6	C00-C97
Leukemia	202.4, 203.1, 204.0-208.9	C90.1, C91-C95
Non-Hodgkin lymphoma	200.0-200.8, 202.0-202.2, 202.8-202.9	C82-C85, C96.3
Hodgkin lymphoma	201.0-201.9	C81

Methods

Data sources

- Washington State cancer incidence data: Washington State Department of Health, Washington State Cancer Registry dataset released in January 2016
- Washington State mortality data: Washington State Department of Health, Center for Health Statistics Vital Registration System Annual Statistical Files, Washington State Deaths 1980–2013 issued August 2014.
- Washington State population denominators: Washington State Office of Financial Management, Forecasting Division. Estimates of Age, Sex, Race, and Hispanic Origin by 2010 Census: County. Population estimates for 1992-1999 released March 2013; U.S. Census counts and intercensal estimates for 2000-2010 released February 2013; estimates for 2011-2013 released June 2016
- National data: Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database version 8.3.2, National Cancer Institute, DCCPS, Surveillance Research Program, Surveillance Systems Branch, public use file released April 2016

Age Adjusted Rates. SEER data are available by five-year age groups. To compare Washington and national rates, we focused on ages five to 24 years because these ages are the closest to the six- to 24-year age group in the case definition. Within ages five to 24, we computed age-adjusted incidence and mortality rates using the direct method of age adjustment. Age-adjustment allows us to compare rates among groups with different age distributions. It helps us to understand whether there are differences among groups independent of differences in numbers of people at older or younger ages. This is important when looking at cancer rates because even within the five to 24-year-old age group, cancer rates are higher for people of some ages than for people at other ages. Consistent with the age-adjustment procedures used by the National Cancer Institute we used the age distributions from the United States 2000 standard population shown below.

2000 US Standard Population Proportions

age group	proportion
5 - 9	0.072532
10 - 14	0.073032
15 - 19	0.072168
20 - 24	0.066478

For the national age-adjusted incidence rates for 2009–2013 combined and for trends from 1992–2013, we used the National Cancer Institute's 13 SEER regions. For the 1992–2013 trend analysis, the SEER*Stat software provided incidence data from the 13 regions. We used the same 13 SEER regions to compute national incidence rates for 2009–2013 combined. For the national listing of the most frequently diagnosed cancers among people ages five to 24 years, we used incidence data from 18 SEER regions, because SEER*Stat provides frequencies only for the 18 SEER regions.

We used a z-test statistic to test for differences in rates in Washington and the United States. If the probability of the differences between Washington and the United States were more than five percent ($Z \geq 1.96$), we considered Washington and the United States to be statistically significantly different.

Changes in Incidence Rates Over Time. We used Joinpoint software, version 4.2.0.2, developed by the National Cancer Institute to test for changes over time.[1] This software calculates the annual percent change (APC). Following the method described by Ries et al.[2] to interpret findings from the Joinpoint analyses, we describe rates as level over time when the APC is not statistically significantly different from zero ($p \geq 0.05$). For statistically significant trends ($p<0.05$), the increase or decrease is described as slight if the APC is less than 1 percent, steady if the APC is between 1 to 3.9 percent and sharp if the APC is greater than or equal to 4 percent.

[1.] Joinpoint Regression Program, Version 4.2.0.2, released June 2015; Statistical Research and Applications Branch, National Cancer Institute.)

[2.] Ries LAG, Wingo PA, Miller BF, Miller DS, Howe HI et al. The annual report to the nation on the status of cancer, 1973-1997, with a special section on colorectal cancer. Cancer, 2000, 88:2398-2424.

Appendix C. Observed and expected cancers for soccer cohort

Appendix C
Observed and expected cancers for soccer cohort

Washington State Department of Health
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This documents the calculation of the expected number of cancers from the soccer cohort, and displays those results, along with the observed numbers and the observed/expected ratios.

The basic case definition is a cancer diagnosis from 2002–2015 among persons age 6–24 at diagnosis, who have a history of playing soccer in Washingon State. We assume a minimum 0.4 year latency; therefore cases must have started playing soccer at least 0.4 years before the date of diagnosis. We consider 4 variations of this case definition: all cancers, Hodgkin lymphoma, non-Hodgkin lymphoma, and leukemia.

The person-years-at-risk consists of all the years spent by people in which they would be considered a case if they had a cancer diagnosis. According to this case definition, the person-years at risk include all the years that soccer players spend that meet these 3 conditions: they play soccer or have played in the past, and began playing at least 0.4 years before they are considered to be at risk; the time period for diagnosis is 2002–2015; and they are age 6–24 at diagnosis. Some examples: if Jack started playing soccer at age 10 in 1996, he would become eligible to be a case in 2002 when he was 16 years old. He would be eligible for 9 years, until he turned 25, so he contributes 9 person-years at risk. The case definition requires only a history of playing soccer, so Jack contributes 9 person-years even if he stopped playing soccer in 1997. If Mary started playing soccer at age 6 in mid-2011, she would be eligible to become a case from 0.4 years after she started until the end of 2015, so she contributes 4.1 person-years at risk.

To make a definitive calculation of person-years at risk, we would need a roster of everyone who played soccer from 1983 to the present. What we have are counts of players from 2000–2015 by age, for approximately ages 6–19 (Table 2), and counts of the total number of players for years 1983–1999. Therefore, we need to make several approximations in order to calculate person-years.

Here is a list of the approximations and/or assumptions made:

- The age distribution of youth soccer players during 1983–1999 was the same as in the year 2000.
- There is some player turnover at ages 7–15, meaning that some players stop playing at the end of each year, and each year sees some new players who have never enrolled before. I assume 10% turnover each year, defined like this: if the previous year had fewer players than the current year (for example, as the age 7 enrollment in 2006 is less than the age 8 enrollment in 2007), then I assume that 90% of the previous year's players have returned, and the difference is made up with new players. If the previous year had more players (for example, as the age 10 enrollment in 2006 is more than the age 11 enrollment in 2007), then I assume that the current year's players are 90% returnees and 10% new players. There is a big drop in the number of players at age 16, and I assumed that every player who was enrolled at age 16 and older had been enrolled in the past. (This means that enrollment figures for players age 16 and older are not needed for calculating person-years at risk.)
- Follow-up ends at the end of 2015. This provides 9 months lag for case ascertainment (as I write this in September 2016). This also means that people diagnosed in 2016 do not meet the case definition and are not considered as cases in the computations.

The player registration spreadsheet (Table 2) shows the number of players of each age registered in each year. The age displayed in the table is the age the player was when he or she registered that

year. Players who registered in April are listed under their age in April; players who registered in August are listed under their age in August. Therefore, players who are the same age may be listed as different ages in the spreadsheet if some registered in April, some registered in August, and their birthdays fall between April and August. My understanding is that most players register in August. Among players who registed in August, the average month at which they became their registration age was February. For example, players who registered as 9 year olds in August could have turned 9 anytime between the previous August and the current August, with the average month being February. Similarly, among players who registered in April, the average month at which they became their registration age was November of the previous year. If 80% of players registered in August, then among both groups of players, the average month at which they turned their registration age was January of the registration year. Among players registering for the first time, the average month of registration (and average month during which exposure begins) is July.

Although I have approximated player turnover to be 10% at ages 6–15, I also computed person-years at risk under an assumption of no turnover (defined like this: if the current year's enrollment is less than the previous year's enrollment, then all players are assumed to be returning players). This provided a lower bound on the effect of this assumption on person-years (and an upper bound on its effect on O/E).

We can partition the years-at-risk calculation into 4 types of players:

1. Players who started playing before 2002 and reached age 25 between 2002 and the end of 2015. For example, consider players who started at age 8 in 1995. The average such player turned 8 in mid-January 1995, and turned 25 in mid-January 2012. They entered the at-risk cohort on January 1, 2002, and spent an average of 10 years and 0.5 month at risk. Their years at-risk are computed as the (year they turn 25) - 2002 + 0.5 month. The year they turn 25 is computed as 25 - (age at registration) + (year they began playing). Therefore, their average years-at-risk are 25 - (age at registration) + (year they began playing) - 2002 + 0.5 month.

2. Players who began playing in 2002 or later and reached age 25 by the end of 2015. For example, consider players who began at age 14 in 2004. The average date at which these players begin exposure is mid-July 2004, and after a 0.4 year latency period, the average date at which they become eligible to be a case is mid-December 2004. The average date at which they reach their 25th birthday is mid-January 2015. Their average years-at-risk is 10 years and 1 month each. The average years-at-risk for players of this type is 24.5 - (the age at which they began playing + 0.4).

3. Players who began playing before 2002 and have not reached age 25 by the end of 2015. These players have spent the entire period January 2002–December 2015 at risk, and the years-at-risk for each of them is 14 years.

4. Players who began playing in 2002 or later and have not reached age 25. For example, consider players who started at age 8 in 2005. After a 0.4 year latency, these players enter the at-risk state in mid-December 2005, and have spent the entire period since then at risk, so the years-at-risk for each of them is 10 years and 0.5 months (to the end of December 2015). The years-at-risk for players of this type is 2015 - (year they began playing + 0.4) + 5.5 months.

The way to identify these players is like this:

1. Players who started playing before 2002 and reached age 25 between 2002 and the end of 2015. These players meet three conditions:
   (a) (year began playing) $\leq$ 2001
   (b) 25 - (age began playing) $\leq$ 2015 - (year began playing)

Table 1: Washington Youth Soccer player counts, 1983–2010.

Year	Boys	Girls	Total
1983	51,613	25,806	77,419
1984	54,234	18,551	72,785
1985	52,162	17,241	69,403
1986	50,568	18,688	69,256
1987	51,323	19,959	71,282
1988	56,632	18,465	75,097
1989	55,408	21,548	76,956
1990	55,415	21,550	76,965
1991	56,529	22,493	79,022
1992	58,343	22,691	81,039
1993	62,624	23,163	85,787
1994	67,674	25,030	92,704
1995	73,938	28,754	102,692
1996	79,532	29,416	108,948
1997	83,516	32,479	115,995
1998	86,274	33,550	119,824
1999	90,558	35,217	125,775
2000	89,280	34,720	124,000
2001	87,840	34,160	122,000
2002	87,644	34,083	121,727
2003	89,518	34,812	124,330
2004	87,610	34,070	121,680
2005	87,550	34,048	121,598
2006	88,637	34,470	123,107
2007	90,734	35,286	126,020
2008	92,260	35,879	128,139
2009	87,824	34,154	121,978
2010	82,020	31,898	113,918

   (c) 25 - (age began playing) $\geq$ 2002 - (year began playing)

2. Players who began playing in 2002 or later and reached age 25 by the end of 2015. These players meet these two conditions:

   (a) (year began playing) $\geq$ 2002

   (b) 25 - (age began playing) $\leq$ 2015 - (year began playing)

3. Players who began playing before 2002 and have not reached age 25 by the end of 2015. These players meet these two conditions:

   (a) (year began playing) $\leq$ 2001

   (b) 25 - (age began playing) $>$ 2015 - (year began playing)

4. Players who began playing in 2002 or later and have not reached age 25 by the end of 2015. These players meet these two conditions:

   (a) (year began playing) $\geq$ 2002

   (b) 25 - (age began playing) $>$ 2015 - (year began playing)

Table 2: Washington Youth Soccer player registration counts, 2000–2015. The figures for each age are the players who were that age at registration, regardless of when they registered during the year. For example, players who turned 9 in June would be listed as 8-year-olds if they registered in April, and as 9-year-olds if they registered in August.

	Age														
Year	6	7	8	9	10	11	12	13	14	15	16	17	18	19	total
2000	2,875	3,325	11,074	13,291	13,273	11,518	12,159	13,087	16,521	14,477	4,001	3,514	2,985	1,900	124,000
2001	2,965	3,146	10,984	12,548	12,483	12,471	11,247	13,547	15,749	14,912	5,016	2,987	2,100	1,845	122,000
2002	3,000	3,254	11,158	12,086	11,487	13,894	12,197	13,824	13,581	13,547	4,899	3,994	2,550	2,256	121,727
2003	3,125	3,987	12,158	13,548	13,477	13,145	13,548	14,867	12,194	13,270	4,112	3,154	2,147	1,598	124,330
2004	2,918	3,319	11,485	12,094	11,981	14,110	12,954	14,581	12,185	13,178	4,174	3,554	3,000	2,147	121,680
2005	2,704	3,548	12,846	11,184	12,421	12,549	13,197	14,061	13,154	14,547	3,489	3,154	2,899	1,845	121,598
2006	3,200	3,600	11,547	13,197	13,731	13,816	13,146	12,136	13,297	13,258	4,100	3,612	2,530	1,937	123,107
2007	3,197	4,254	12,931	13,491	13,968	12,714	13,955	13,147	12,056	13,844	4,011	3,098	3,254	2,100	126,020
2008	2,994	3,559	13,125	13,998	12,194	14,228	13,939	13,121	12,009	12,446	5,009	4,111	3,954	2,512	127,199
2009	2,165	3,356	13,844	12,156	12,669	13,995	12,191	13,458	12,556	12,199	4,154	3,566	3,121	2,548	121,978
2010	1,944	2,211	12,354	11,451	11,258	12,946	12,147	12,595	11,963	12,458	4,448	3,118	3,147	1,878	113,918
2011	2,547	2,966	13,144	11,556	12,846	11,981	11,846	13,411	12,263	13,194	4,158	3,178	3,558	2,154	118,802
2012	1,468	2,584	12,548	10,139	11,886	10,548	10,107	11,669	12,118	12,158	3,945	2,658	2,471	1,487	105,786
2013	2,191	2,348	12,114	11,244	11,369	11,174	11,214	10,340	10,247	11,334	2,582	2,174	2,131	1,564	102,026
2014	2,451	2,945	11,478	10,954	10,474	10,558	11,114	10,228	10,897	10,284	2,489	2,548	2,698	1,984	101,102
2015	2,945	2,115	9,118	10,941	9,876	10,119	10,087	10,369	10,147	9,200	2,189	2,948	2,964	1,568	94,586
TOTAL	42,689	50,517	191,908	193,878	195,393	199,766	195,048	204,441	200,937	204,306	62,776	51,368	45,509	31,323	NA

To calculate the total person-years at risk, I used Washington Youth Soccer (WYS) enrollment figures to estimate the number of players who enroll for the first time each year. We have been given enrollment figures by age only back to 2000. For years 1983–1999, I assumed the age distribution was the same as in 2000, but I scaled the number of players per year of age so that the total for each year matches the total enrollment reported in each year in *The History Book* (see Table 1).

To compute person-years, first, I created a matrix with rows for each year 1983–2015 and columns for each age 6–15. For each cell in the matrix, I computed the estimated number of players who began playing soccer in that year and at that age (I called this the `newplayers` matrix). Next, I prepared a similar matrix and assigned to each cell a code of 1 to 4 defining which of the 4 types of players described above were represented by that cell (cells that corresponded to none of those 4 definitions could not contribute person-years and received a code of 0) (I called this the `playerType` matrix). Then I applied the one of the 4 formulae descibed above for computing years-at-risk to each cell of the `newplayers` matrix, by using the codes in the `playerType` matrix. This resulted in a matrix with the each cell having the person-years contributed by the players defined by that cell. For example, consider the cell for players who began playing soccer at the age of 7 in 2004. After applying the 10% turnover assumption, there are an estimated 506 such players. These players are of type 4 (players who began playing in 2002 or later and have not reached age 25 by the end of 2015). The person-years-at-risk for each player of type 4 is given by 2015 - (year they began playing + 0.4) + 5.5 months, which computes to 11.0583 years per player. So the 506 players who began playing in 2004 at age 7 contributed a total of 5,595.5 person-years.

By these calculations, assuming 10% turnover, the total person-years-at-risk is 4,977,989.

Assuming no turnover

I repeated the calculations assuming no turnover. Here, if the enrollment for the current year exceeds that for the previous year, then the excess players were counted as new players. Otherwise, there were no new players.

Under the assumption of no turnover, the total person-years-at-risk is 3,479,355. This is much different from the figure for 10% turnover, and indicates that the calculation is sensitive to this assumption.

Person-years by age

I computed the person-years-at-risk by individual year of age, for ages 6–24, to help compute the expected numbers of cancers. The `personYears` matrix has the person-years that each cell, which is defined by the year and age at which a player started playing, accumulated. I took the entries in each cell of the `personYears` matrix and distributed them over the years of age that the players defined by that cell were at risk. The details of how person-years were distributed by age depend on the player type category, as described below. The average time at which players are assumed to have started playing is mid-July of the year they began playing.

1. Type 1 players donate half a month of their person-years to their first year of eligibility, and a full year to all their later years of eligibility. For example, players who started at age 6 in 1985 turned 23 in mid-January 2002, so they are at risk from Jan 1 to Jan 15 when they are 22, then for 2 full years until they turn 25 in mid-January 2004.

2. Type 2 players give 0.1 years of their person-years to the year in which they began playing, and a full year to each year until they turn 25. For example, players who started at age 14 in

Table 3: Person-years-at-risk by age, for all the players in the at-risk population.

Age	person-years
6	3,808
7	38,027
8	62,085
9	182,461
10	206,872
11	232,697
12	258,931
13	278,909
14	306,197
15	325,518
16	349,008
17	351,334
18	349,839
19	348,832
20	345,256
21	342,759
22	338,210
23	332,832
24	325,111

2002 are at risk for 0.1 years at age 14, from about mid-December 2002 to the following mid-January 2003, then for 10 full years until they turn 25 in mid-January 2013.

3. Type 3 players give half a month of person-years to their first year of eligibility, 11.5 months (mid-January to end of December) to their last, and a full year to each of the years in between.

4. Type 4 players give 0.1 years of their person-years to the year in which they began playing, 11.5 months (mid-January to end of December) to their last year of eligibility, and a full year to each of the years in between.

Compute cancer rates

I computed Washington State cancer rates by single year of age, for ages 6–24, for each of the groupings in the case definition (all cancers, non-Hodgkin lymphoma, Hodgkin lymphoma, and leukemia).

I used the 2002–2013 WSCR data and the 2002–2013 OFM population files to compute cancer rates. The rates for each single year of age 6–24 are in Table 4.

Expected numbers of cancers

To compute the expected numbers of cancers in the soccer player cohort, I applied the age-specific rates to the person-years-at-risk, then summed across all ages (see Table 5).

Table 4: Age-specific cancer rates among Washington residents, 2002–2013. The rates are diagnoses per 100,000 population per year.

	cancer rates per 100,000 population per year			
age	all cancers	leukemia	Hodgkin lymphoma	non-Hodgkin lymphoma
6	12.14	4.11	0.4894	0.881
7	13.31	3.82	0.0979	1.370
8	11.35	3.62	0.3915	1.077
9	9.49	2.64	0.1958	0.979
10	11.06	2.17	0.6618	1.229
11	11.35	3.03	0.6618	0.756
12	14.65	3.97	1.3236	0.662
13	14.37	2.74	1.3236	1.607
14	17.11	3.31	1.5127	1.702
15	19.29	2.85	2.2959	1.929
16	20.94	2.39	2.2959	1.469
17	25.25	2.76	3.2142	1.745
18	28.47	2.66	3.3979	1.469
19	29.48	2.39	4.2244	2.020
20	38.31	2.72	3.9944	2.088
21	39.04	2.45	4.9930	2.905
22	45.03	2.36	3.6313	2.451
23	46.57	1.36	4.6299	2.179
24	53.56	2.45	6.0824	2.542

Table 5: Expected numbers of cancers diagnosed from January 1, 2002 to December 31, 2015 among Washington residents age 6–24 who have a history of playing organized soccer.

cancer type	expected cases
All cancer	1,384
Leukemia	131
Hodgkin lymphoma	147
Non-Hodgkin lymphoma	89

Observed/Expected ratios

There are 55 cases on the case master list (including 2 people with 2 cancer diagnoses each). Of those, 51 have had the diagnosis of cancer confirmed by DOH. Of those confirmed, 49 were diagnosed between 2002 and 2015. Of those, 43 played soccer. Of those, 28 were between 6 and 24 years old at diagnosis (see Table 6).

I computed observed/expected ratios for the age groups 6–9, 10–14, 15–19, and 20–24 for all cancers, along with the observed/expected ratios for all ages combined for all cancers and for the three cancer types listed in the case definition: leukemia, non-Hodgkin lymphoma, and Hodgkin lymphoma.

The observed cancer cases that fit the case definition are tabulated in Table 8. The numbers are from the spreadsheet "SoccerPlayerCaseMasterList.xlsx." One person who fit the case definition had 2 cancer diagnoses; both are counted.

The expected numbers of all cancers by age group are shown in Table 7.

Table 6: Number of cases and their eligibility status.

condition	Yes	No	Unknown
Age 6-24	36	16	3
Diagnosis confirmed	51	4	0
Played soccer	49	6	0
Total reported cases	55	.	.
Total eligible cases	28	.	.

Table 7: Expected cancers by age group, for all cancer types diagnosed between January 2002 and December 2015, in people age 6–24 at diagnosis, and who have a history of playing organized soccer.

Age group	expected cancers
06-09	29.9
10-14	179.7
15-19	427.0
20-24	747.5
Total	1,384.1

To compute observed/expected ratios, I just divided the observed numbers by the expected numbers of cancers (see Table 8). I computed 95% exact Poisson confidence intervals for the observed to expected ratio.

Goalkeepers

I computed the expected numbers of cancers for players who have a history of playing the goalkeeper position. Players are generally not assigned to be goalkeepers until they play on '10 and

Table 8: Observed to expected ratios for cancer by age group and by type, in the soccer players cohort.

Age group	observed cancers	expected cancers	observed/ expected	95% CI for O/E lower	upper
06-09	1	30	0.033	0.0008	0.19
10-14	12	180	0.067	0.0345	0.12
15-19	8	427	0.019	0.0081	0.04
20-24	7	747	0.009	0.0038	0.02
Cancer type	**observed cancers**	**expected cancers**	**observed/ expected**	**95% CI for O/E lower**	**upper**
All cancers	28	1,384	0.020	0.013	0.029
Leukemia	6	131	0.046	0.017	0.099
Hodgkin lymphoma	5	147	0.034	0.011	0.079
Non-Hodgkin lymphoma	6	89	0.067	0.025	0.146

Table 9: Washington Youth Soccer goalkeeper estimates, 2000–2015. The figures for each age are the number of goalkeepers estimated to play on teams for that age and under.

	Age									
Year	10	11	12	13	14	15	16	17	18	19
2000	2,172	1,595	1,368	1,472	1,239	1,086	300	264	224	142
2001	2,043	1,727	1,265	1,524	1,181	1,118	376	224	158	138
2002	1,880	1,924	1,372	1,555	1,019	1,016	367	300	191	169
2003	2,205	1,820	1,524	1,673	915	995	308	237	161	120
2004	1,961	1,954	1,457	1,640	914	988	313	267	225	161
2005	2,033	1,738	1,485	1,582	987	1,091	262	237	217	138
2006	2,247	1,913	1,479	1,365	997	994	308	271	190	145
2007	2,286	1,760	1,570	1,479	904	1,038	301	232	244	158
2008	1,995	1,970	1,568	1,476	901	933	376	308	297	188
2009	2,073	1,938	1,371	1,514	942	915	312	267	234	191
2010	1,842	1,793	1,367	1,417	897	934	334	234	236	141
2011	2,102	1,659	1,333	1,509	920	990	312	238	267	162
2012	1,945	1,460	1,137	1,313	909	912	296	199	185	112
2013	1,860	1,547	1,262	1,163	769	850	194	163	160	117
2014	1,714	1,462	1,250	1,151	817	771	187	191	202	149
2015	1,616	1,401	1,135	1,167	761	690	164	221	222	118

under' or older teams. Therefore, for this computation, the case definition is a person diagnosed with cancer between 2002 and the present, who was age 9–24 at diagnosis, and has a history of playing the goalkeeper position in organized soccer in Washington State. The person-years at risk are computed as all the years spent by people such that they would meet the case definition if they were diagnosed with cancer.

The goalkeeper calculations do not incorporate a latency period, since it is assumed that most goalkeepers played organized soccer for at least a short time before officially becoming goalkeepers.

To calculate the total person-years at risk, I used the estimated number of goalkeepers (Table 9) to estimate the number of players who play goalkeeper for the first time each year. We have been given estimates only back to 2000. For 1983–1999, I assumed that the age distribution was the same as in 2000, but I scaled the number of players so that the total number of players was the same as the total reported in each year in *The History Book*. The estimated number of players declines a lot at the age '16 and under' team, and I assume that all goalkeepers on the '16 and under' or older teams have played goalkeeper before.

By these calculations, assuming 10% turnover, the total person-years-at-risk is 527,076.

Person-years by age

I computed the person-years-at-risk among goalkeepers by individual year of age, for ages 10–24, to help compute the expected numbers of cancers.

Expected numbers of cancers

To compute the expected numbers of cancers in the goalkeeper cohort, I applied the age-specific rates to the person-years-at-risk, then summed across all ages (see Table 11).

Table 10: Person-years-at-risk by age, for all the goalkeepers in the at-risk population.

Age	person-years
10	17,076
11	31,681
12	33,962
13	36,584
14	38,826
15	40,251
16	41,175
17	40,882
18	40,337
19	39,438
20	38,446
21	37,659
22	36,882
23	35,778
24	34,724

Table 11: Expected numbers of cancers diagnosed from January 1, 2002 to August 31, 2015 among Washington residents age 10–24 who have a history of playing the goalkeeper position in organized soccer.

cancer type	expected cases
All cancer	153.48
Leukemia	14.42
Hodgkin lymphoma	16.59
Non-Hodgkin lymphoma	9.87

Observed/Expected ratios

To ascertain goalkeeper status, I used both the data reported by Coach Griffin, and the data obtained by interviewing the cases. If a case was interviewed, I used that information, unless it was missing. Otherwise, I used the information from the coach.

To compute observed/expected ratios, I just divided the observed numbers by the expected numbers of cancers (see Table 12). I computed 95% exact Poisson confidence intervals for the observed to expected ratio.

Table 12: Observed to expected ratios for cancer by type, in the goalkeepers cohort.

Cancer type	observed cancers	expected cancers	observed/ expected	95% CI for O/E lower	upper
All cancers	14	153.5	0.091	0.050	0.15
Leukemia	3	14.4	0.208	0.043	0.61
Hodgkin lymphoma	4	16.6	0.241	0.066	0.62
Non-Hodgkin lymphoma	2	9.9	0.203	0.025	0.73

Table 13: Person-years-at-risk by age, for elite players.

Age	person-years
6	0
7	0
8	3,002
9	32,095
10	41,378
11	46,898
12	51,825
13	56,791
14	62,386
15	66,815
16	72,277
17	72,768
18	72,591
19	72,463
20	71,692
21	71,238
22	70,286
23	69,262
24	67,729

Table 14: Expected numbers of cancers diagnosed from January 1, 2002 to August 31, 2015 among Washington residents age 7–24 who have a history of playing elite organized soccer.

cancer type	expected cases
All cancer	283.6
Leukemia	26.2
Hodgkin lymphoma	30.4
Non-Hodgkin lymphoma	18.2

Expected cancers among elite players

The spreadsheet `Player Distribution - Will Holden Model Updated 9-24.xlsx` has estimates of the numbers of "premier" and "select" players by age for 2013–2014. Together, these two categories comprise the elite players. Elite players are of particular interest because they play more months of the year than recreational players, and are more likely to play on crumb rubber fields (because they play during seasons with poor weather).

I computed the proportion of elite players by age, and applied that proportion to the cohort of players I used for computing expected cancers among all players. Then I computed the person-years at risk and expected cancers as before.

By these calculations, assuming 10% turnover, the total person-years-at-risk is 1,001,496.

Table 15: Observed to expected ratios for cancer by type, among elite soccer players.

Cancer type	observed cancers	expected cancers	observed/ expected	95% CI for O/E lower	upper
All cancers	15	284	0.053	0.030	0.087
Leukemia	3	26	0.114	0.024	0.334
Hodgkin lymphoma	4	30	0.132	0.036	0.337
Non-Hodgkin lymphoma	4	18	0.220	0.060	0.563

Compute observed to expected ratios for elite players

I used the case interview data to count the number of elite players among the cases.

To compute observed/expected ratios, I divided the observed numbers by the expected numbers of cancers (see Table 15). I computed 95% exact Poisson confidence intervals for the observed to expected ratio.

References

1. R Core Team. *R: A Language and Environment for Statistical Computing.* R Foundation for Statistical Computing, Vienna, Austria, 2015. Http://www.R-project.org, accessed January 26, 2016.

Appendix D: Interview Questionnaire

Washington State Department of Health ID#:_____
Revised 2016

Version 4.3

Soccer Cancer Cluster Investigation Initial Inquiry Survey

Consent Form

You have been invited to take part in a survey about soccer playing and a possible association with your (child's) cancer diagnosis. The Washington State Department of Health is conducting interviews as part of a public health investigation. The University of Washington soccer coach, Amy Griffin, gave us your name and contact information. Your participation will take 30-45 minutes. Questions include asking about your (child's) health history (and cancer diagnosis), and detailed information about playing soccer. You may find it difficult to discuss your (child's) diagnosis and health history, but there are no other risks to participation.

You can choose not to participate. There will be no bad effects from this decision; it will not affect the healthcare or services you or your family receives.

If you choose to participate in this survey, you can stop at any time and decline to answer any specific questions.

Your responses will be kept confidential and your (child's) identity will remain private. Survey forms are kept in a locked file cabinet and the information is entered onto a computer file on a secure Department of Health server with limited access. The information you provide may be shared with other investigators, but without information that could identify you (your child).

There is no payment for answering the survey, but your participation is helpful in our investigation. Any report of this research that is made available to the public will not include your (child's) name or any other information by which you (your child) could be identified. If you have questions, you can contact WA State Epidemiologist Cathy Wasserman at 1-800-525-0127.

Do you wish to continue with the survey? ☐ **Yes** ☐ **No**

If respondent does not want to participate, thank them for their time and verify that they have Washington State Department of Health contact information for questions or concerns.

Washington State Department of Health ID#:_____
Revised 2016

Section 1: Interviewer Information (Questions 1-4 to be completed before interview.)

1. Patient id: _____

2. Date Interview Completed:__/___/_____
MM DD YYYY

3. Interviewer Information Name: _____ Agency or Organization: _____

4. Respondent was: 1 Self 2 Parent 3 Spouse 4 Other (Specify):_____

5. Before this interview, has a local, state, or federal public health representative interviewed you about your (child's) illness? If so, how many times?
: ☐ None ☐ Once ☐ Twice ☐ Other _____ ☐ Unknown

Section 2: Demographic Data: I'd like to begin by asking a few questions about yourself (your child). (Can fill in information from the Washington State Cancer Registry for questions 6 and 7 and ask for verification.)

6. Date of Birth: _____/_____/_____
MM DD YYYY

7. Sex: 1 Male 2 Female

8. Are you (your child) Hispanic or Latino oirigin: 1 Yes 0 No 9 Unknown

9. How would you describe your (child's) race ? 1 White 2 Black/African American 3 American Indian/Alaska Native 4 Asian 5 Native Hawaiian/Pacific Islander 6 Other _____

Section 3: Clinical Information: Now I have a few questions about your (your child's) cancer(s) diagnosis.

10. From the Washington State Cancer Registry, I understand that you (your child) was diagnoses with [**Check all that apply and add diagnosis dates.]**

1. Leukemia—no subtype
2. Acute lymphoid leukemia (ALL)
3. Acute myeloid leukemia (AML)
4. Chronic lymphoid leukemia (CLL)
5. Chronic myeloid leukemia (CML)
6. Leukemia—other (specify: _____)

7. Non-Hodgkin's lymphoma—no subtype
8. NHL--diffuse B-cell
9. NHL—B-cell
10. NHL—T-cell
11. NHL—Other (specify: _____)

12. Hodgkin's Lymphoma—no subtype
13. Classic Hodgkin's Lymphoma
14. HL—nodular-sclerosis
15. HL—mixed-cellularity
16. HL—lymphocytic-rich
17. HL—lymphocyte-depleted
18. HL—nodular lymphocyte-predominant
19. HL—Other (specify: _____)

20. Other (specify _____)

11. Is this correct? 1 yes 2 no If no, note errors

12. Have you (has your child) been diagnosed with any additional types of cancer? 1 yes 0 no

If yes, collect following information

Type of cancer: _____ Diagnosis date _ _ / _ _ _ _
MM YYYY

Facility of diagnosis: _____ (hospital/clinic [city, state])

Health care provider: _____ (who made diagnosis)

Type of cancer: _____ Diagnosis date _ _ / _ _ _ _
MM YYYY

Facility of diagnosis: _____ (hospital/clinic [city, state])

Health care provider: _____ (who made diagnosis)

Type of cancer: _____ Diagnosis date _ _ / _ _ _ __
MM YYYY

Facility of diagnosis: _____ (hospital/clinic [city, state])

Health care provider: _____ (who made diagnosis)

13. Have you (has your child) ever been diagnosed with any autoimmune disease? 1 Yes 0 No 9 Unknown
    If yes, what was the condition and date of diagnosis? _____

14. Have you (has your child) ever been diagnosed with any other chronic condition? 1 Yes 0 No 9 Unknown
    If yes, what was the condition and date of diagnosis? _____

Section 4: <u>Personal Background Information</u>: Now, I have a few questions about your (your child's) personal and family health history.

15. Not counting CT or CAT scans that you (your child's) had in diagnosing your (child's) (first) cancer, did you (your child) ever have a CT or CAT scan? 1 Yes 0 No 9 Unknown
16.
    If yes, number of scans? ____ date of first scan? _ _ / _ _ _ _
    MM YYYY

17. Before your (child's) (first) cancer diagnosis, did you (your child) ever smoke cigarettes?:
    ☐ Yes 0 No 9 Unknown

    If yes, were you (your child) smoking at the time of diagnosis?: 1 Yes 2 No (former smoker) 9 Unknown

18. Were any of your (child's) biological brothers, sisters or parents diagnosed with cancer? 1 Yes 0 No 9 Unknown

If no, do you (does your child) have brothers or sisters? 1 Yes 0 No

If yes, collect type of cancer, diagnosis date, age at diagnosis and relation to case

			1 Mom 2 Dad 3 Sis 4 Bro
Type	MM/YYYY (diagnosis)	Age at dx	Relation
			1 Mom 2 Dad 3 Sis 4 Bro
Type	MM/YYYY (diagnosis)	Age at dx	Relation
			1 Mom 2 Dad 3 Sis 4 Bro
Type	MM/YYYY (diagnosis)	Age at dx	Relation

19. **Prior** to your (child's) (first) cancer diagnosis, Were you (your child) ever diagnosed with the following:?

1 Yes 0 No 9 Unknown Hepatitis B
1 Yes 0 No 9 Unknown Human Immunodeficiency virus (HIV)
1 Yes 0 No 9 Unknown Infectious mononucleosis or Epstein-Barr virus (also known as human herpesvirus 4)

Section 5: Residence: Now, I have a question about where you (your child) lived up to the time of diagnosis. You can start with birth and work forward or with diagnosis and work backward, whichever is easiest for you.

20. Residence 1

_ _ / _ _ _ _ to _ _ / _ _ _ _ City/town_____ State abbr. _____
MM YYYY MM YYYY

21. Did you (your child) live anywhere else?

If answered **no**, skip to section 6
If answered **yes**, complete boxes below for previous residences. Use additional residence sheet if needed.

_ _ / _ _ _ _ to _ _ / _ _ _ _ City/town_____ State abbr. _____
MM YYYY MM YYYY

_ _ / _ _ _ _ to _ _ / _ _ _ _ City/town_____ State abbr. _____
MM YYYY MM YYYY

_ _ / _ _ _ _ to _ _ / _ _ _ _ City/town_____ State abbr. _____
MM YYYY MM YYYY

_ _ / _ _ _ _ to _ _ / _ _ _ _ City/town_____ State abbr. _____
MM YYYY MM YYYY

Section 6: Soccer Related Play:

Now, I have several questions about your (your child's) history of playing soccer up to the time of your (child's) (most recent) cancer diagnosis.

NOTE: See appendix for definition of different skill levels of soccer play

22. Before your (child's) (most recent) diagnosis, did you (your child) play for a school team [**check all levels that apply**]?
    ☐ Middle School team ☐ High School Team ☐ College Team ☐ College Other

23. Before your (child's) (most recent) diagnosis did you (your child) play for a club team? If yes, **check all levels that apply**
    ☐ Recreational ☐ Select ☐ Premier/Elite ☐ Adult Recreational ☐ Semi Pro/Pro

24. For each level of play identified in the previous questions, I will ask more specific details. You can start with your (your child's) level of play at your (his/her) (most recent) diagnosis and work backwards or start at your (his/her) earliest play and work forwards, whichever is easiest for you. (Use 1 box for each school, club or league at each level.)

Level of Play: _____ Years : _ _ _ _ to _ _ _ _

School, club or league name: _____ City_____ State ___

Season: fall 1 Yes 0 No # months/year_____

winter 1 Yes 0 No # months/year_____

spring 1 Yes 0 No # months/year_____

summer 1 Yes 0 No # months/year_____

Did you (your child) play goalie? 1 yes % time _____ 0 Never

Name/location of practice field(s): _____ City _____ State ____

Name/location of practice field(s): _____ _____ City _____ State ____

Did you (your child) **practice** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Did you (your child) **play matches** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Section 6: Soccer Related Play Continued

Level of Play: _____ Years : _ _ _ _ to _ _ _ _

School, club or league name: _____ City_____ State ___

Season: fall 1 Yes 0 No # months/year_____

winter 1 Yes 0 No # months/year_____

spring 1 Yes 0 No # months/year_____

summer 1 Yes 0 No # months/year_____

Did you (your child) play goalie? 1 yes % time _____ 0 Never

Name/location of practice field(s): _____ City _____ State ____

Name/location of practice field(s): _____ _____ City _____ State ____

Did you (your child) **practice** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Did you (your child) **play matches** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Level of Play: _____ Years : _ _ _ _ to _ _ _ _

School, club or league name: _____ City_____ State ___

Season: fall 1 Yes 0 No # months/year_____

winter 1 Yes 0 No # months/year_____

spring 1 Yes 0 No # months/year_____

summer 1 Yes 0 No # months/year_____

Did you (your child) play goalie? 1 yes % time _____ 0 Never

Name/location of practice field(s): _____ City _____ State ____

Name/location of practice field(s): _____ _____ City _____ State ___

Did you (your child) **practice** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Did you (your child) **play matches** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Section 6: Soccer Related Play Continued

Level of Play: _____ Years : _ _ _ _ to _ _ _ _

School, club or league name: _____ City_____ State ___

Season: fall 1 Yes 0 No # months/year_____

winter 1 Yes 0 No # months/year_____

spring 1 Yes 0 No # months/year_____

summer 1 Yes 0 No # months/year_____

Did you (your child) play goalie? 1 yes % time _____ 0 Never

Name/location of practice field(s): _____ City _____ State ____

Name/location of practice field(s): _____ _____ City _____ State ____

Did you (your child) **practice** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Did you (your child) **play matches** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Level of Play: _____ Years : _ _ _ _ to _ _ _ _

School, club or league name: _____ City_____ State ___

Season: fall 1 Yes 0 No # months/year_____

winter 1 Yes 0 No # months/year_____

spring 1 Yes 0 No # months/year_____

summer 1 Yes 0 No # months/year_____

Did you (your child) play goalie? 1 yes % time _____ 0 Never

Name/location of practice field(s): _____ City _____ State ____

Name/location of practice field(s): _____ _____ City _____ State ___

Did you (your child) **practice** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Did you (your child) **play matches** on artificial turf? 0 No 9 Unsure/Don't know

Yes 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

If yes, state as a percentage (%) or season(s) _____ Percentage indoors _____

Section 7 : Sports played *other* than soccer:

Now, I have some questions about your (child's) history of playing sports other than soccer before your (child's) most recent cancer diagnosis. We are interested in sports that you (your child) played at least 12 times a year.

25. What sports did you (your child) play [may list more than one]? _____

26. Were any of these sports played on artificial turf? 1 Yes 2 No 3 Unknown
    For each sport played on artificial; turf collect the following

Sport: _____ percent on artificial turf _____ turf type 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

Field name, city, state_____ Dates _ _ _ _ to _ _ _ _
Y Y Y Y Y Y Y Y

Sport: _____ percent on artificial turf _____ turf type 1 crumb rubber 2 astroturf 3 red rock 4 other 5 unknown

Field name, city, state_____ Dates _ _ _ _ to _ _ _ _
Y Y Y Y Y Y Y Y

27. Is there any other information that we did not ask about that you want to share (e.g. something relating to your cancer diagnosis)?

In the unlikely event that the Washington State Department of Health would like to contact you again, can we call you at the number I called today?

☐ **Yes** ☐ **No**

It will take a while to collect and analyze the information and develop a report. We hope to have a completed report by the end of the year. Are you interested in being kept updated: ☐ **Yes** ☐ **No**

If yes, collect current mailing address

If you have questions or further information you want to share, you can contact me or WA State epidemiologist Cathy Wasserman whose contact information was in the original letter you received. (If asked for Cathy's contact give: 1-800-525-0127.

Thank you again for your time and willingness to participate.

APPENDIX:

Definitions

Recreational (soccer): Typically practices begin in the summer with play starting in the fall months with the season complete by the end of the calendar year. Coaches are required to play all players 50% of the games and are not chosen to participate based on soccer ability. Teams sometimes do 1 or 2 tournaments in the summer as training for the season starting the first weekend in September.

Select (soccer): In between recreational and premier soccer in terms of commitment and competition level. Select level players often participate in other sports. Attendance is not as strict and players typically paly 7-9 months of the year. Select teams often participate in 2 or 3 summer tournaments and then play their regular season in the fall, which can lead to tournament play in January and February each year.

Premier/Elite (soccer): Highest level of play with the most commitment from players, parents, and coaches. Premier players generally focus on soccer as their number 1 sport interest with many only playing soccer. Premier teams practice and play year round (which includes numerous tournaments, both indoor and outdoor soccer leagues).

Appendix E. Computations for Elements of History of Soccer Play

The interviewer used a semi-structured approach to obtain information on soccer play. This approach allowed participants to report information in the way they most easily remembered it, rather than requiring them to make computations during the interview. For example, participants often remembered ages or grades in school, rather than calendar years, of playing Washington Youth Soccer (WYS)-defined recreational, select or premier soccer. As another example, some participants reported percentages of play on artificial turf at each field and number of years of play at that field for a given WYS-defined level, rather than providing an overall percentage of play on artificial turf. Thus, as needed, for each type of play, the interviewer assigned ages or years of beginning play and number of years played. For each type of play, interviewer also computed as needed the average number of months of play per year and the average percentages of time spent playing goalkeeper, playing on grass or dirt, outdoor artificial turf and indoor artificial turf.

Assigning ages and years of play. While many interview responses required assigning ages at beginning play and years of play, in some instances, these assignments required using one or more of the following conventions:

- Children enter kindergarten at age five.
- The soccer season begins in late August or early September.
- A player needed to be a given age by September 15 of a given year. For example, a person who reported starting soccer play at age 10 and was born September 15 or later of 2000 would be assigned a starting year of 2011, while a person who began play at 10 and was born before September 15, 2000 would have a starting year of 2010. Conversely, a person born before September 15, 2000 who reported beginning play in 2010 would be given a starting age of 10, while a player born September 15 or later 2000 would be given a starting age of nine.
- For WYS-defined levels of play and adult recreational play, we used “school years” or calendar years to calculate the number of years of play depending on the number of seasons played and whether the starting year for the next level was the same as the ending year of the previous level.
- Several participants reported starting and ending years for a given category of play, grades, ages or total years of play that resulted in unrealistic scenarios such as starting kindergarten at age three. In these instances, we adjusted the starting or ending years or the total years of play working backward from the most recent play. We used this approach, because reports of the most recent play were likely to be the most accurate especially when the most recent play occurred at the time of cancer diagnosis and was, thus, associated with specific dates, ages, and often specific grades in school.

These conventions were used only when we did not have other information. For example, if a participant provided calendar years and grades in school such that a child would have begun kindergarten at age four, we did not adjust that information or use the convention that children begin kindergarten at age five. Likewise, if someone reported starting to play soccer

in January, we did not use the convention that the soccer season starts in late August or September.

Computing averages. To describe a specific category of soccer play (such as, recreational, select, or premier) for each participant, we developed weighted averages for participants who reported differing percentages of time playing goalkeeper, playing on artificial turf, or playing indoors or who reported playing different numbers of seasons per year over time for a given category of play. The weights for percentages were most often weeks of play computed from the reported seasons or months of play provided in the interviews. For example, a three-month season was considered 13 weeks; a two and a half month season was counted as 10 weeks.

The following computations illustrate computing a weighted average for someone who played on artificial turf on a select team 25 percent of the time for two years when they played for nine months a year and then 95 percent of the time for three years when they played for ten months a year:

- 9 months/year for 2 years = 78 weeks (39 weeks/year x 2 years)
- 10 months/year for 3 years = 129 weeks (43 weeks/year x 3 years)
- Weighted average = [(78 x .25) + (129 x .95)] / (78 + 129) x 100 = 68.6

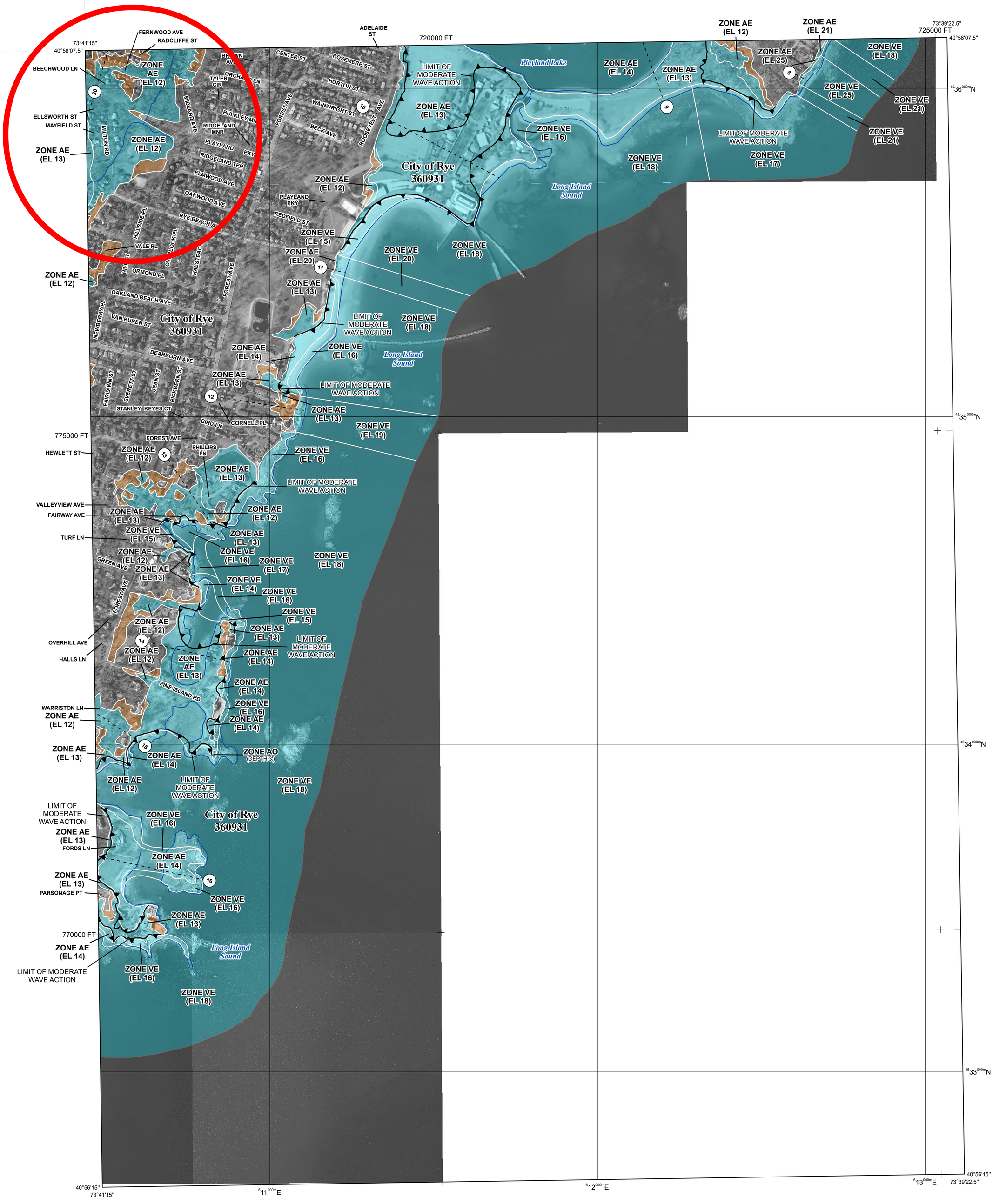
We then classified percentages in five percentage point increments. Thus, 68.6 was classified as “65 to less than 70 percent.”

For number of months of play per year, we rounded to the nearest half. For example, we rounded an average of 3.3 months of play per year to 3.5 months. For participants with minimal missing data, we used median values to replace the missing information related to soccer play for all calculations except percent of time spent playing goalkeeper beyond the recreational level. After the recreational level, most players played goalie most of the time or almost never. Thus, the median was unlikely to represent the actual time spent playing. This affected one player whose information about the percentage of time playing goalie was not included in the overall statistics.

Stantec

Appendix:

J. FEMA Flood Map



FLOOD HAZARD INFORMATION

SEE FIS REPORT FOR DETAILED LEGEND AND INDEX MAP FOR FIRM PANEL LAYOUT

THE INFORMATION DEPICTED ON THIS MAP AND SUPPORTING DOCUMENTATION ARE ALSO AVAILABLE IN DIGITAL FORMAT AT

HTTP://MSC.FEMA.GOV

SPECIAL FLOOD HAZARD AREAS	Without Base Flood Elevation (BFE) *Zone A,V, A99*
	With BFE or Depth *Zone AE, AO, AH, VE, AR*
	Regulatory Floodway
OTHER AREAS OF FLOOD HAZARD	0.2% Annual Chance Flood Hazard, Areas of 1% Annual Chance Flood with average depth less than one foot or with drainage areas of less than one square mile *Zone X*
	Future Conditions 1% Annual Chance Flood Hazard *Zone X*
	Area with Reduced Flood Risk due to Levee See Notes *Zone X*
OTHER AREAS	NO SCREEN Area of Minimal Flood Hazard *Zone X*
	Area of Undetermined Flood Hazard *Zone D*
GENERAL STRUCTURES	Channel, Culvert, or Storm Sewer
	Levee, Dike, or Floodwall
	E 18.2 17.5 Cross Sections with 1% Annual Chance Water Surface Elevation (BFE)
	8 Coastal Transect
	Coastal Transect Baseline
	Profile Baseline
	Hydrographic Feature
	513 Base Flood Elevation Line (BFE)
OTHER FEATURES	Limit of Study
	Jurisdiction Boundary

NOTES TO USERS

For information and questions about this map, available products associated with this FIRM including historic versions of this FIRM, how to order products or the National Flood Insurance Program in general, please call the FEMA Map Information eXchange at 1-877-FEMA-MAP (1-877-336-2627) or visit the FEMA Map Service Center website at http://msc.fema.gov. Available products may include previously issued Letters of Map Change, a Flood Insurance Study Report, and/or digital versions of this map. Many of these products can be ordered or obtained directly from the website. Users may determine the current map date for each FIRM panel by visiting the FEMA Map Service Center website or by calling the FEMA Map Information eXchange.

Communities annexing land on adjacent FIRM panels must obtain a current copy of the adjacent panel as well as the current FIRM Index. These may be ordered directly from the Map Service Center at the number listed above.

For community and countywide map dates refer to the Flood Insurance Study report for this jurisdiction.

To determine if flood insurance is available in this community, contact your Insurance agent or call the National Flood Insurance Program at 1-800-638-6620.

Base map information shown on this FIRM was provided in digital format by New York State Cyber and Critical Infrastructure. This information was derived from digital orthophotography at a 0.5 foot ground resolution from imagery flown in April 2013.

LIMIT OF MODERATE WAVE ACTION: Zone AE has been divided by a Limit of Moderate Wave Action (LiMWA). The LiMWA represents the approximate landward limit of the 1.5-foot breaking wave. The effects of wave hazards between the VE Zone and the LiMWA (or between the shoreline and the LiMWA for areas where VE Zones are not identified) will be similar to, but less severe than those in the VE Zone.

Limit of Moderate Wave Action (LiMWA)

SCALE

N

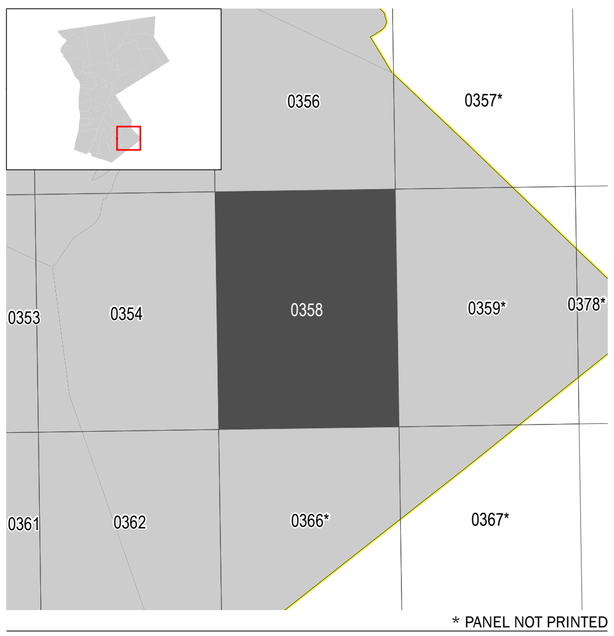
Map Projection:
Universal Transverse Mercator Zone 18N; North American Datum 1983; GRS_1980 Spheroid; North American Vertical Datum of 1988

1 inch = 500 feet 1:6,000

0 500 1,000 2,000 Feet

0 125 250 500 Meters

PANEL LOCATOR



* PANEL NOT PRINTED

FEMA

National Flood Insurance Program

NATIONAL FLOOD INSURANCE PROGRAM
FLOOD INSURANCE RATE MAP

WESTCHESTER COUNTY, NEW YORK
All Jurisdictions

PANEL **358** OF **426**

FEMA

Panel Contains:

COMMUNITY	NUMBER	PANEL	SUFFIX
RYE, CITY OF	360931	0358	G

PRELIMINARY
DECEMBER 8, 2014

VERSION NUMBER
2.2.2.1

MAP NUMBER
36119C0358G

MAP REVISED