Our intention is to have in-person meetings going forward. For the time being, we will hold the City Committee Meetings, Plan Commission, Council and most others at the Community Room at 933 Michigan Avenue. This in-person location will meet the legal requirement for our open meetings.

We will have a virtual option available, but the technology for the hybrid style meeting may not be reliable all of the time.

Members

- Mayor Wiza
- Alderperson Kneebone
- Commissioner Arntsen
- Commissioner Beacom
- Commissioner Rice
- Commissioner Schade Stroik
- Commissioner Schuler

MINUTES

CITY PLAN COMMISSION

Date and Time:	January 5, 2026 6:00 PM	**Location:**	Community Room 933 Michigan Avenue, Stevens Point, WI OR Zoom Teleconferencing Meeting ID: 822 1741 7977 Passcode: 531976 By Computer: https://us02web.zoom.us/j/82217417977?pwd=ze7arGObLm6M By Phone: +1-312-626-6799 (US Chicago)

Opening Section:

1. Roll Call

   - Meeting called to order at 6:00PM.
   - Members present: Mayor Wiza, Alder Kneebone, Commissioners Arntsen, Beacom, Rice, Schade-Stroik, Schuler

Discussion and Possible Action on:

2. Report of the December 1, 2025 meeting of the City Plan Commission.

   - Background: Report of the December 1, 2025 meeting of the City Plan Commission included in the agenda packet.
   - Motion: Arntsen moves approval of the report.
   - Second: Schade-Stroik seconds the motion.
   - Vote: Unanimous approval.

3. Public Hearing and action on a request from Trent Kastenschmidt, representing Kwik Trip, Inc., for a conditional use permit to operate a gas station on the properties located at 3383

Hoover Road (Parcel ID 281230802301117) and 5278 County Road HH (Parcel ID 281230802301118), consistent with Ch. 23.02(4)(e)(4)(c)(4)(e).

- Background: Associate Planner & Zoning Administrator Kuhn provides background regarding the request. Director Lemke provides additional information regarding conditional uses for groundwater protection zones and the staff-recommended conditions.
- Discussion:
    - Rice asks how the applicant may have been included in the creation of the conditions. Kuhn answers, providing that Director Lemke, Kuhn, and the applicant worked diligently to refine the conditions.
- Public Hearing:
    - Christine Mechenich, 4416 Second St, asks if the spill response plan will receive its own public hearing. Chris provides an additional handout for commissioners to review and requests additional conditional uses.
    - Janet Smith, 520 Elm Court, Village of Whiting, speaks in opposition of the request, and urges stricter conditions if approved.
    - DeeAnn Donahue, 3395 Howard Ave, shares a handout with the commission and speaks in opposition to the request.
    - Deb Zaske, 5222 Forest Circle S, speaks in opposition to the request, citing concerns about traffic congestion and pedestrian safety.
    - Alicia Skrenes, 5316 Forest Circle, speaks in opposition to the request, stating that there is no documented need for the gas station.
    - John Jaeger, 3217 Olympia Ave, speaks in opposition to the request.
    - Donna Mott, 5324 Forest Circle N, speaks in opposition to the request, citing studies on the impacts of gas stations, and requesting additional conditions.
    - Jon Mott, 5324 Forest Circle N, speaks in opposition to the request citing noise and operational impacts, and requesting additional conditions.
    - Savannah Hebior, 5272 Forest Circle S, shares the importance of vegetated buffers in connection with the request.
    - Michael Corrigan, 3271 Tulip Lane, speaks in opposition to the request noting the intensity of the proposed use and adjacency to the residential neighborhood.
    - Amberle Schwartz, 1336 Birchwood Lane, Town of Linwood, requests that the commissioners consider if the staff-recommended conditions are enough.
    - Eric Renfert, 3349 Orchid Lane, shares specific recommendations to the listed staff-recommended conditions, and claims that the conditional use permit is for all acreage to be developed.
    - Harold Wolfgram, 114 W Fifth Street, Marshfield, legal counsel the developer, refers the commission to the staff report.
    - Joe Donahue, 3395 Howard Avenue, speaks in opposition to the request.
    - Lynn Markham, 302 Georgia Street N, speaks in opposition to the request.
    - Scott Tiegan, Vice President of Kwik Trip stores, shares tax and workforce impacts of the project.
    - Kevin Nestigen, Environmental Manager with Kwik Trip, shares that the conditions presented are the most strict of all other locations and shares general details about environmental protections and spill response plans.

    - Kevin Lutz, 504 Autumn Court, Village of Whiting, reminds the commission about County input and response to the plan and speaks in opposition to the request.
    - Nancy Eggleston, 2410 Enterprise Court, Village of Plover, speaks in opposition to the request.
    - Dale Steinmetz, Alder for District 6, speaks in support of the request.
    - Elizabeth Trzebiatowski, 3262 Sunflower Road, speaks in opposition to the request.
    - Justin Frahm, JSD, engineer and planner working on the project, shares information regarding the traffic impact analysis, lighting plan, landscaping plan, DNR recommendations, and forthcoming certified survey map.
    - Victor Baeten, owner of the Parcel, speaks on the project.
    - Chris Tiffany, 4808 Nicolet Avenue, speaks in support of the request.
    - BJ Hermsen, 5295 Rose Pass, speaks in opposition of the request.
    - Trevor Roark, 601 Washington Ave, speaks in opposition of the request.
    - Michael Corrigan, 3271 Tulip Lane, again speaks in opposition of the request.
    - Savannah Hebior, 5272 Forest Circle S, again speaks in opposition of the request citing concerns about representation.
    - Amberle Schwartz, Portage County Supervisor District 19, shares prospective revisions to the County groundwater protection ordinance.
    - Christine Mechenich, 4416 Second St speaks again on the request.
    - Eric Renfert, 3349 Orchid Lane, again cites concerns about the administrative report.
- Discussion:
    - Kuhn and Director Lemke respond to questions and statements presented in the public hearing and recommend clarifications and modifications to conditions provided in the request; Mayor addresses the commission.
    - Schuler asks Atty Beveridge to clarify how the commission is to discern what is substantial evidence; Beveridge answers.
    - Kuhn asks the applicant to share their environmental protections letter referenced in the administrative report. Kevin Nestigen shares the details of the letter with the commission.
    - Schade-Stroik asks for clarification on groundwater protection jurisdiction.
    - Beacom comments that there is enough evidence and discussion to vote on the request tonight.
    - Schuler asks for clarification on when the spill response plan could be provided, as well as the details on emergency management.
    - Kneebone shares that they cannot vote in favor of the request given the risk and unknown of road redesign approvals; Kuhn responds that the County would review the request following City review.
    - Chief Moody shares details regarding the spills that local responders have responded to throughout the City.
- Motion: Beacom moves to postpone action until the next meeting and that details regarding the applicant's spill response plan and monitoring plans be shared with the commission, and for the commission to receive and review information provided by the public.
- Second: Schade-Stroik seconds.
- Discussion:

    - Wiza requests modifications for several conditions previously referenced by Director Lemke and noted by Kuhn.
    - Arntsen requests that spill response and monitoring plans be reviewed and approved by designated professionals.
- Vote: Unanimous approval.

4. Public Hearing and action on a request from Trent Kastenschmidt, representing Kwik Trip, Inc., for a conditional use permit to operate a car wash on the properties located at 3383 Hoover Road (Parcel ID 281230802301117) and 5278 County Road HH (Parcel ID 281230802301118), consistent with Ch. 23.02(2)(d)(3)(k).

    - Background: Given the vote to postpone action on agenda item #3, no discussion or action is taken on agenda items #4 and #5. Meeting is recessed at 9:38PM, and reconvened at 9:43PM at agenda item #6.

5. Public Hearing and action on a request from Trent Kastenschmidt, representing Kwik Trip, Inc., for a conditional use permit to construct an off-premise sign on the property located at 3383 Hoover Road (Parcel ID 281230802301117), consistent with Ch. 23.02(2)(a)(3)(j).

    - Background: Given the vote to postpone action on agenda item #3, no discussion or action is taken on agenda items #4 and #5. Meeting is recessed at 9:38PM, and reconvened at 9:43PM at agenda item #6.

6. A request from Bill Pritchard, representing the Elizabeth Revocable Trust, for a conceptual subdivision plat review for a proposed subdivision on an unaddressed parcel off of West Zinda Drive (Parcel ID 281240831401241).

    - Background: Kuhn provides background information on the conceptual subdivision plat review prior to the conventional review and approval process. Kuhn shares that the parcels would meet the standards of R2.
    - Discussion:
        - Arntsen provides input that there may be unique geological features on or near the property and should be acknowledged in some way. Mr. Pritchard notes the features are on the north side of the adjacent railroad tracks.
        - Schuler asks for clarification if there are any rules about buildable area on the parcel; Kuhn answers. The designer of the proposed subdivision responds that
        - Arntsen asks if there will be any wetland fill for the project; the designer of the proposed subdivision responds that there will
        - Nick Proulx, representative of Ahlstrom, shares that Ahlstrom's pipeline crosses several parcels in the subdivision but is in support of the request and will provide coordinates of the pipe to prevent conflicts.
        - Jackie Schimke, 440 W Trillium Court, shares concern about traffic impact generated by the proposed subdivision citing pedestrian safety on West River Drive, conflict with the railroad crossing, and potential environmental impact.

- Kneebone asks Chief Moody if the cul de sacs will support emergency service vehicles.
- Rice acknowledges and commends Pritchard for addressing density concerns that were raised during the original subdivision plat review. Beacom echoes the comments.

7. April - November 2025 Monthly Reports.

    - Background: Monthly reports are included in the agenda packet.

8. Director's Report.

    - Background: Director’s report is included in the agenda packet.

Closing Section:

9. Adjournment

    - Meeting adjourned at 10:00PM.

PLEASE TAKE NOTICE that any person who has special needs while attending these meetings or needs agenda materials for these meetings should contact the City Clerk as soon as possible to ensure that a reasonable accommodation can be made. The City Clerk can be reached by telephone at (715) 346-1569 or by mail at 1515 Strongs Avenue, Stevens Point, WI 54481.

Maps further defining the above area(s) may be obtained from the City of Stevens Point Department of Community Development, 1515 Strongs Avenue, Stevens Point, WI 54481, or by calling (715) 346-1567, during normal business hours.

PLEASE TAKE FURTHER NOTICE that a quorum of the Common Council may be in attendance at this meeting.

SIGN IN SHEET

PLEASE PRINT LEGIBLY

FAILURE TO PRINT LEGIBLY MAY RESULT IN MISS SPELLED NAMES ON OFFICAL RECORD OF MEETING MINUTES.

NAME:	ADDRESS:
Victor R. Baeten	5936 Seminole Centre Ct, Suite 100 Fitchburg, WI 53711
Lynn Markham	302 Georgia N, SP
Harold C. Wolfgram	Legal Counsel 114 W. 5th Street Marshfield WI.
Christine Mechenich	4416 2nd St Stevens Point
Savannah Hebior	5272 Forest Cir S Stevens Point
Chris Tiffany	4808 Nicolet Ave St Pt
JAY FISCHER	5299 FOREST Circle South St. Pt.
Michael Corrigan	3171 Tulip Ln
George Lars Kedrowski	5286 forest circle So
Jeff & Amy Brojan	5355 Clover Ct, Stevens Point
Scott Teigen	Kwik Trip
BRIAN J GUZMAN	5288 CROCUS CT STPT WI
Nicholas Pawlik	3101 Matthias Ln St. Pt.
Deb Zaske	5223 Forest Cir. S. St. Pt.
LeAnn Tilden	3032 Channel Drive St. Point
Alicia Skrenes	5316 forest Cir. Stevens Point

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NAME:	ADDRESS:
Madeline Stockbridge	5315 Forest Circle Stevens Point
Donna Mott	5324 Forest Circle SP
Bill Pritchard	Green Bay
Dexter Kattchenny	1831 RED OAK PLOVER
Jon Mott	5324 Forest Cir SP
Kevin Lutz	504 AUTUMN CT.
DAVID Powell	Kwik TRIP
JUSTIN C. FRAHM	SEH PROFESSIONAL SERVICES, INC. 1402 STONE RIDGE DR. STE 4, WESTON, WI
DeeAnn Donahue	3395 Howard
JOHN & TARA JAEGER	3217 OLYMPIA AVE
Janet Smith	520 Elm Ct Plover
Joe Donahue	3395 Howard
Mike Schinke	440 W Trillium Ct.
Jackie Schinke	" " " "
Kevin Nestingen	Kwik Trip
Tom Goldschmidt	Teton Drive

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PLEASE PRINT LEGIBLY

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NAME:	ADDRESS:
Dale Steinmetz	1833 Halstad Drive Stevens Point
Eric Renfert	3349 Orchid Ln, Stev Pt
Lynette Kvatek	216 W. Pleasant St. S.P.
Ginger Keymer	1816 Lincoln / Dist #3 Alder
Deborah Grall	4500 Pleasant View Dr
Brian Grall	4500 Pleasant View Dr.
JOHN ZAPPA	3232 NICOLET CT
GERI ZAPPA	" "
Brian Zimmerman	3356 Iris Lane - SPC
Scot Barton	3225 Nicolet Ct Stevens Pt
ELIZABETH TRZEBIATOWSKI	3262 SUNFLOWER RD
DAN BUSS	1271 CROCUS COURT
Stacy Kizewski	5279 Forest Cir S Stevens Pt
Scott Trzebiatowski	3262 SUNFLOWER RD

PLEASE RETURN TO SECRETARY, THANK YOU.

Agenda

Agenda Packet

Minutes

5. Special Meeting Attendance Report...

No Attachment File

6. Chair Schwartz - Ad Hoc Advisory C...

No Attachment File

7. T-2025-001 - Proposal to amend the ...

Combined Files

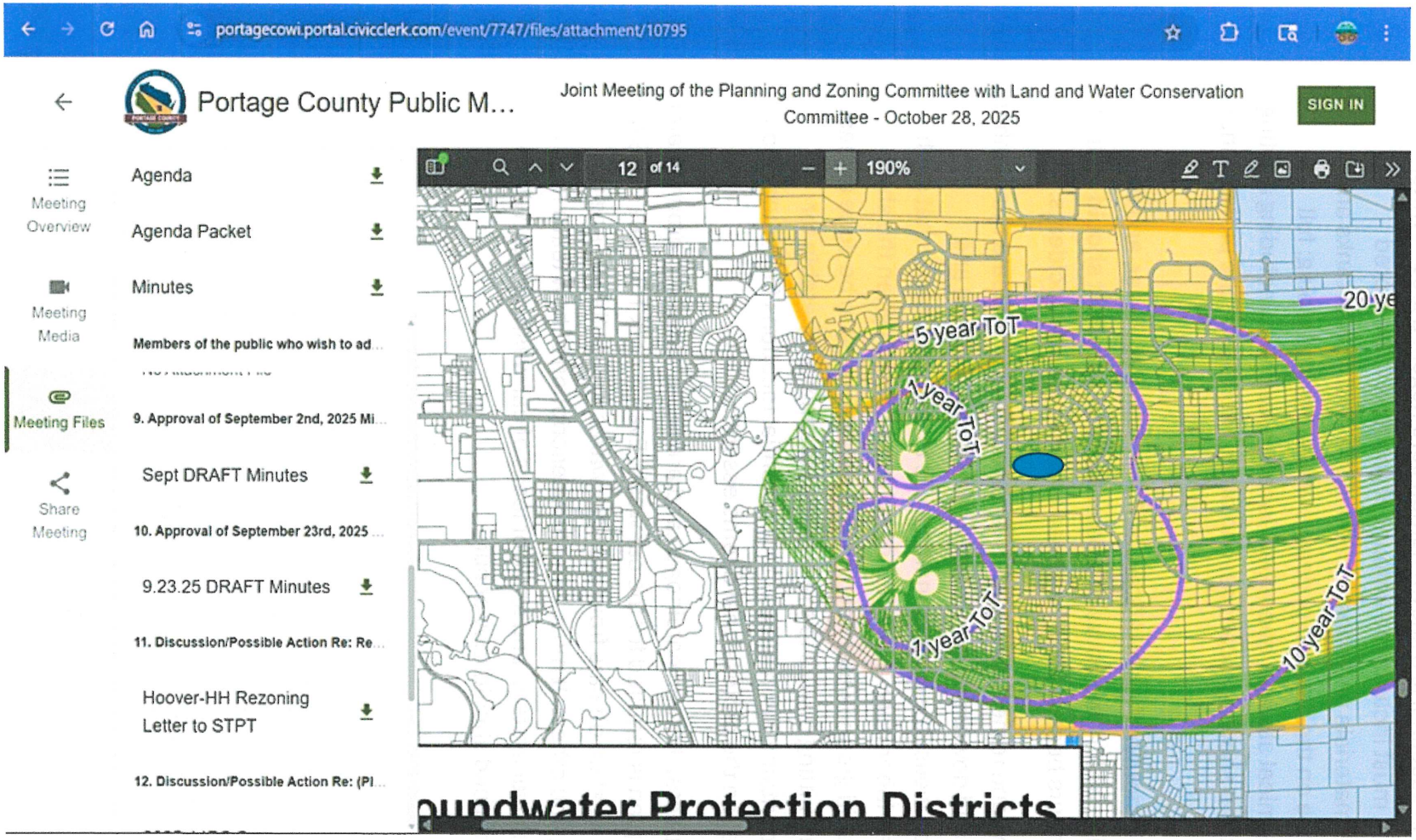
8. Portage County Groundwater Mana...

Combined Files

Members of the public who wish to ad...

No Attachment File

All the water pumped from the Whiting wells originated as rainfall somewhere in the area outlined in green toward the bottom of the map. The recharge area for the wells goes northeast and ends in the glacial moraine north of Polonia. At its farthest extent, water takes 100 years from the time it enters the groundwater to reach the Whiting wells. The green lines represent the flow paths of the water underground.



This is a closeup of the same map that zooms in closer to the wells, which are the pinkish circles. I've added a blue oval to show the location of the parcel. It is in between the one and five year time of travel lines on the map. That is, a drop of water that soaks into the ground to become groundwater will reach the wells in somewhere between one and five years.

In a 1988 report, the Wisconsin Geological and Natural History Survey estimated that groundwater flow within the recharge area toward the Whiting well field is 1,070 ft per year (0.20 miles per year). That estimate would put the time of travel from the parcel to the wells at approximately 1.5 years.

Three separate plans, prepared by qualified professionals and reviewed by other qualified professionals of the city's choosing, must be submitted and subjected to public comment before the permit is approved.

1. Because benzene, found in gasoline, is a known human carcinogen, a groundwater protection plan is required. It must include the amount of gasoline to be stored underground; details on the materials and construction of the double wall tanks and piping; any available evidence on the lifespan of double wall tanks and piping; full disclosure and limits on chemical storage, quantities, location, and use, including a listing of chemicals on site and a map showing locations; and other information the professional deems essential.

It must also include a plan to notify officials in Stevens Point, Whiting, Plover, and the Wisconsin DNR Spills Hotline within two hours of a detected leak or spill. It should restrict fuel tank filling to daytime hours because this has been found to reduce accidents.

2. A groundwater monitoring and remediation plan is required. It must indicate the number and locations of upstream and downstream monitoring wells; the depth(s) and construction details for such wells; the contaminants to be tested; provisions that such testing be done by a certified laboratory; a monitoring schedule, which given the short distance to the wells and rapid groundwater flow in the area, may need to be done at least monthly; a timetable for submitting results to the city after testing; a timetable for remediation to begin after contamination is detected; potential locations for remediation systems; and other information the professional deems essential.

3. Because PFAS are a suspected human carcinogen and are highly mobile in groundwater, a spill and leak prevention plan is required for the car wash. Even if Kwik Trip uses PFAS free products, they may still be washed off cars from past car waxes and polishes, engine degreasers and lubricants, and others. It must include a description of how leaks from the car wash piping to the city sewer system, and spills or leaks of car wash sludge, will be prevented, detected or contained should they occur.

Resources:
1995 Boundary Agreement between the town of Plover and the city of Stevens Point- the first planned annexation in the state of Wisconsin. The city committed to protecting the Whiting wells, as follows: "Because the Boundary Adjustment Area lies within the A and B Zones of the Village of Whiting's municipal wellfield, the Stevens Point Wellhead Protection Ordinance will be used to protect Whiting's municipal wellfield and recharge area by restricting and regulating new development within the Boundary Adjustment Area."
https://mds.wi.gov/View/Documents?Type=BoundaryAgreementFile&ID=10634, page 32.

"The purpose of this Wellhead Protection Ordinance is to institute land use regulations and restrictions to protect the municipal water supplies of the Villages of Amherst, Junction City, Plover and Whiting, and the City of Stevens Point, and to promote the public health, safety and general welfare of the residents of Portage County."
Stevens Point Zoning Code, chapter 23, page 102.
https://stevenspoint.com/DocumentCenter/View/769/Chapter-23---Zoning--Floodplain

Groundwater Protection Districts for Municipal Recharge Areas in Stevens Point, Whiting and Plover. Draft Update October 2, 2025.
https://portagecowi.portal.civicclerk.com/event/7747/files/attachment/10795

Wellhead-Protection Districts in Wisconsin: An Analysis and Test Applications
https://wgnhs.wisc.edu/catalog/publication/000551/resource/sr10

Village of Whiting Wellhead Protection Ordinance
https://storage.googleapis.com/juniper-media-library/414/2025/11/Municipal-Code-Chapter-31-Wellhead-Protection.pdf

Rapid Response Inquiry: PFAS contamination in commercial car wash wastewater & groundwater
Download pdf from https://www.pprc.org/post/pfas-contamination-in-commercial-car-wash-wastewater-groundwater

Portage County Groundwater Management Plan (page 37, costs of municipal water treatment or replacement)
https://www.co.portage.wi.gov/DocumentCenter/View/6135/2025-Groundwater-Management-Plan?bidId=

By Christine Mechanich to Stevens Point Plan Commission
1/5/26

This request is submitted on behalf of the following Village Green residents:

Resident Name	Street Address	Contact
Jeff & Karen Rautio	3352 Howard Ave	rautiojk@charter.net
Pete, Deb, Anissa Zaske	5222 Forest Circle S	pdzaske@tds.net
Scott & Michele Janz	3331 Orchid	puttnut@charter.net
Song Cheng & Sheng Yang	5340 Clover Ct	Song.cheng@gmail.com
Bill & Madeline Stockbridge	5315 Forest Circle N	billmadstockbridge@att.net
William & Lenore Kirby	3258 Howard Ave	Lkirby39@gmail.com
Jane Tibbetts	5324 Clover Ct	Jane4nature@yahoo.com
Laura & Nathan Wahn	5248 Forest Cir S	cauley.laura114@gmail.com

This request is submitted on behalf of the following Village Green residents:

Resident Name	Street Address	Contact
Savannah & Chris Hebior	5272 Forest Cir S	Savannah.hebior@gmail.com
Ginger & Dean Nelson	5278 Forest Cir S	Bgnelson8@gmail.com
BJ Hermsen	5295 Rose Pass	bpscguy@hotmail.com
Stephanie Johnson	3372 Iris Ln	sbtaz@charter.net
Ryan & Jessica McCaffrey	3249 Sunflower Rd	ryan@kprbrokers.com
Jeff & Barb Johnson	5346 Clover Ct	Barb.johnson3@yahoo.com
Suzanne Fee	5278 Crocus Ct	Ssfee15@gmail.com
Sam & Mary Schroeder	3363 Iris Ln	schroederurzer@gmail.com
Brian & Mary Guzman	5288 Crocus Ct	Bmguz85@gmail.com
David & Mary Lamb	3323 Orchid Ln	dandmlamb@charter.net
Joe & DeeAnn Donahue	3395 Howard Ave	d.donahue@charter.net
Jeff & Amy Brogan	5355 Clover Ct	buckyjumparound@gmail.com
Wiliam Christiansen	3284 Orchid Ln	Cps1432@charter.net
Gary & Lois Kedrowski	5236 Forest Circle S	Kedrowski3@gmail.com
Annette & Ben Hultman	5360 Lily Ct	Annette.hultman@gmail.com
Donna & Jon Mott	5324 Forest Cir	dlmott@hotmail.com
Alicia & Eric Skrenes	5316 Forest Cir	aliciaskrenes@gmail.com
Danielle & Jake Warta	5271 Forest Cir S	dannyann989@gmail.com

Executive Summary
Conditional Use Permit Requests – Village Green Neighborhood

We recognize that the property has been rezoned to B-4 commercial. The conditional use permit process is therefore the City's primary remaining tool to ensure that these high-intensity uses comply with the Purpose and Intent of the Stevens Point Zoning Code (§23.01(4)) and are not injurious to nearby residential properties, groundwater resources, or public safety (§23.01(19)).

The Plan Commission previously raised concerns about groundwater protection, neighborhood compatibility, and the difficulty of regulating future uses once approval is granted. The following requests are designed to directly address those concerns in a manner that is measurable, reasonable, enforceable, and consistent with City authority.

Requested Conditions (Summary)

1. Groundwater & Protection of Human Health
Require a groundwater protection plan to be submitted and approved as part of the CUP, not after approval.
Require full disclosure and limits on chemical storage, quantities, location, and use including a list of chemicals on site and a map showing locations.
Require spill and leak notification within two hours to Stevens Point, Whiting, Plover, and the DNR groundwater section manager.
Restrict fuel tank filling to daytime hours because this has been found to reduce accidents and speed up response time in the event of a spill.
Require air-quality analysis or monitoring for benzene and related compounds.
Why it matters: Groundwater impacts are irreversible. Benzene, found in gasoline, is classified by EPA as a known human carcinogen for all routes of exposure. Prevention must occur before approval, not after construction.
2. Traffic & Pedestrian Safety
Require a post-opening traffic study (6–12 months) with mandatory mitigation if failures occur.
Prohibit residential cut-through traffic, including deliveries.
Install traffic-calming measures and pedestrian safety enhancements, subject to City engineering approval.
Why it matters: Real-world traffic impacts often differ from projections and must be corrected if unsafe.
3. Lighting & Noise Controls
Require dark-sky compliant, downward-facing lighting, oriented away from homes.
Dim lighting during overnight hours.
Limit car wash operation to 9:00 p.m., consistent with other City locations.
Prohibit outdoor amplified audio.
Require car wash doors to remain closed and dryers oriented away from residences.

Why it matters: Light and noise impacts are ongoing quality-of-life issues that are difficult to correct later.

4. Landscaping & Buffering

Require a minimum 50-foot vegetative buffer along all residential edges that abut lots 1, 2 and 3 (see map for location designation). Mature height of buffer should be 40-60 feet.
Require multi-layer, year-round vegetation with defined planting densities.
Maintain planting densities over time. Replant if needed.
Prohibit chemical treatments in buffer areas.
Prohibit buffer reduction without Plan Commission and Council approval.

Why it matters: Buffers are functional environmental infrastructure, not decoration.

5. Future Phases & Use Creep

Limit the CUP to Lot 1 and Lot 2 of Phase 1 only, as shown on page 6 of the CUP staff report.
Explicitly prohibit automatic or implied approvals for future phases.
Require all future phases or intensity increases to return to the Plan Commission and Council.
Require cumulative impact review for future requests.

Why it matters: Once granted, CUPs can unintentionally become blank checks unless clearly limited.

6. Enforcement

All conditions must be enforceable, run with the land, and apply to all future owners and tenants.
Noncompliance must allow for enforcement, modification, or revocation under §23.01(19).

In Closing

These requests do not seek to block development. They seek to ensure that development proceeds **responsibly, transparently, and in alignment with Stevens Point's zoning intent, groundwater stewardship, and neighborhood compatibility standards.**
The conditional use permit process exists precisely for this purpose. We respectfully ask the Plan Commission to use it fully.

Conditional Use Permit Requirements for Consideration

Village Green Neighborhood – Hoover Road & County Road HH

We respectfully submit the following comments and requested conditions for the record regarding the conditional use permit applications for a gas station, car wash, and off-premise sign proposed at 3383 Hoover Road and 5278 Portage County HH.

These requested conditions are intended to ensure that the proposed uses meet the City's conditional use standards by not being injurious to neighboring property, not impairing the orderly development of surrounding land, and remaining compatible with adjacent residential neighborhoods, consistent with the Purpose and Intent of the Stevens Point Zoning Code (§23.01(4)) and the City's authority to impose site-specific conditions on conditional uses (§23.01(19)).

Groundwater and Wellhead Protection

The Purpose and Intent section of the Stevens Point Zoning Code states that zoning regulations are intended to promote public health, safety, and welfare; protect water, sanitation, and drainage; conserve natural resources; stabilize property values; and prevent land uses that create hazards or dangers (§23.01(4)). The City's conditional use provisions further authorize the Plan Commission to impose conditions necessary to prevent harm and mitigate impacts associated with high-intensity uses (§23.01(19)).

Given the proximity of this site to sensitive groundwater resources and established residential neighborhoods, the following conditions are respectfully requested.

Requested Conditions

1. **Groundwater Protection Plan Required Prior to Permit Approval**

Pursuant to the City's obligation to protect public health and natural resources and its authority to require site-specific documentation for conditional uses (§23.01(19)), a comprehensive groundwater protection plan shall be submitted, reviewed, and approved as part of the conditional use permit application itself. Approval of the permit shall not be granted contingent upon a future or post-approval submittal. The Plan Commission must have the opportunity to evaluate the adequacy of groundwater safeguards before determining that the use is appropriate at this location.

2. **Chemical Storage Disclosure and Limitations**

The applicant shall submit a complete inventory of all chemicals proposed to be stored, handled, or used on site, including but not limited to fuels, car wash chemicals, cleaning agents, lubricants, and additives. Storage or use of any chemicals not expressly identified and approved shall be prohibited without additional Plan Commission review and approval (§23.01(4), §23.01(19)).

3. **Air Quality Monitoring for Benzene and Volatile Organic Compounds**

To protect public health and welfare (§23.01(4)), baseline and ongoing air quality monitoring for benzene and other fuel-related volatile organic compounds shall be required or, alternatively, the applicant shall demonstrate through third-party analysis that air quality impacts will not adversely affect adjacent residential areas. A mitigation plan identifying action thresholds and corrective measures shall be submitted, with results made available to the City upon request.

4. **Daytime Fuel Tank Filling Requirement**

Fuel tank filling operations shall be restricted to daytime hours only to reduce spill risk, ensure visibility, and allow immediate response in the event of a release, as a reasonable operational condition authorized under §23.01(19).

5. **Mandatory Spill and Leak Notification Protocol**

Any spill, leak, or release of fuel or hazardous materials, regardless of quantity, shall trigger immediate notification to:

Village of Whiting

Village of Plover

Wisconsin Department of Natural Resources – Groundwater Section Manager

Traffic, Pedestrian Safety, and Neighborhood Access

Pursuant to §23.01(4) and §23.01(19), the following traffic-related conditions are requested to protect public safety and neighborhood access.

1. **Post-Opening Traffic Impact Study with Mandatory Mitigation**

A traffic impact study shall be conducted 6–12 months after operations begin to evaluate actual traffic volumes, turning movements, speeds, and cut-through impacts. If any intersections, street segments, or pedestrian crossings operate at unacceptable levels, the land owner shall implement additional mitigation measures as approved by the City.

2. **Prohibition of Residential Cut-Through Traffic**

Clear, permanent signage shall prohibit residential cut-through traffic, including commercial deliveries. Delivery routing policies shall reflect this restriction.

3. **Stop Control to Discourage Cut-Through Traffic**

Two stop signs shall be installed at Forest Circle South and Iris Lane, subject to approval by the City's traffic engineer, to slow traffic and discourage neighborhood cut-through.

Lighting and Noise

Pursuant to §23.01(4), §23.01(19), and applicable provisions of the City's Noise Control Ordinance (Chapter 15), the following conditions are requested.

Lighting Controls

1. **Dark-Sky Compliant Lighting**

All exterior lighting shall use fully shielded, downward-facing fixtures to minimize glare, light trespass, and sky glow. Fixture design and placement shall be reviewed during site plan approval.

2. **Lighting Orientation Away from Residential Areas**

Canopy lighting, illuminated signage, neon elements, and internally lit signs shall be oriented away from residential properties.

3. **Reduced Lighting During Overnight Hours**

Exterior lighting shall be dimmed during overnight hours, except where minimal lighting is required for safety and security.

4. **Signage Limitations**

Digital, animated, or changing-message signs shall be prohibited. Any future modifications to signage illumination shall require Plan Commission approval.

Noise Controls

1. **Car Wash Hours of Operation**

The car wash shall close no later than 9:00 p.m., consistent with other Kwik Trip locations in the City. Any extension of hours shall require Plan Commission and Common Council approval (§23.01(19)).

2. **Operational Noise Restrictions**

Deliveries, trash pickup, and maintenance activities shall be limited to daytime hours.

3. **Car Wash Noise Mitigation**

Car wash doors shall remain closed during operation, and dryer equipment shall be oriented toward County HH and away from residences.

Landscaping and Buffering

Landscaping in this location shall be treated as functional environmental infrastructure, not merely decorative screening.

1. **Minimum Buffer Width and Design**

A minimum 50-foot vegetative buffer shall be required between commercial uses and residential properties, consisting of canopy trees, mid-story shrubs, and groundcover designed for year-round effectiveness.

The required minimum 50-foot vegetative buffer shall be designed as a layered cross-section, measured horizontally from the commercial property line toward the residential property.

From commercial side to residential side:

a. Outer Canopy Zone (Approx. 20 feet wide)

Large canopy trees forming a continuous overhead screen

Primary functions: noise attenuation, air filtration, visual screening, and interception of airborne pollutants

b. Mid-Story Shrub Zone (Approx. 20 feet wide)

Dense evergreen and deciduous shrubs

Primary functions: year-round visual screening, headlight shielding, noise absorption

c. Groundcover & Infiltration Zone (Approx. 10 feet wide)

Native grasses, sedges, low shrubs, and infiltration-friendly plantings

Primary functions: stormwater infiltration, groundwater filtration, erosion control

2. **Minimum Planting Densities and Performance**

Canopy trees: 1 per 30 linear feet

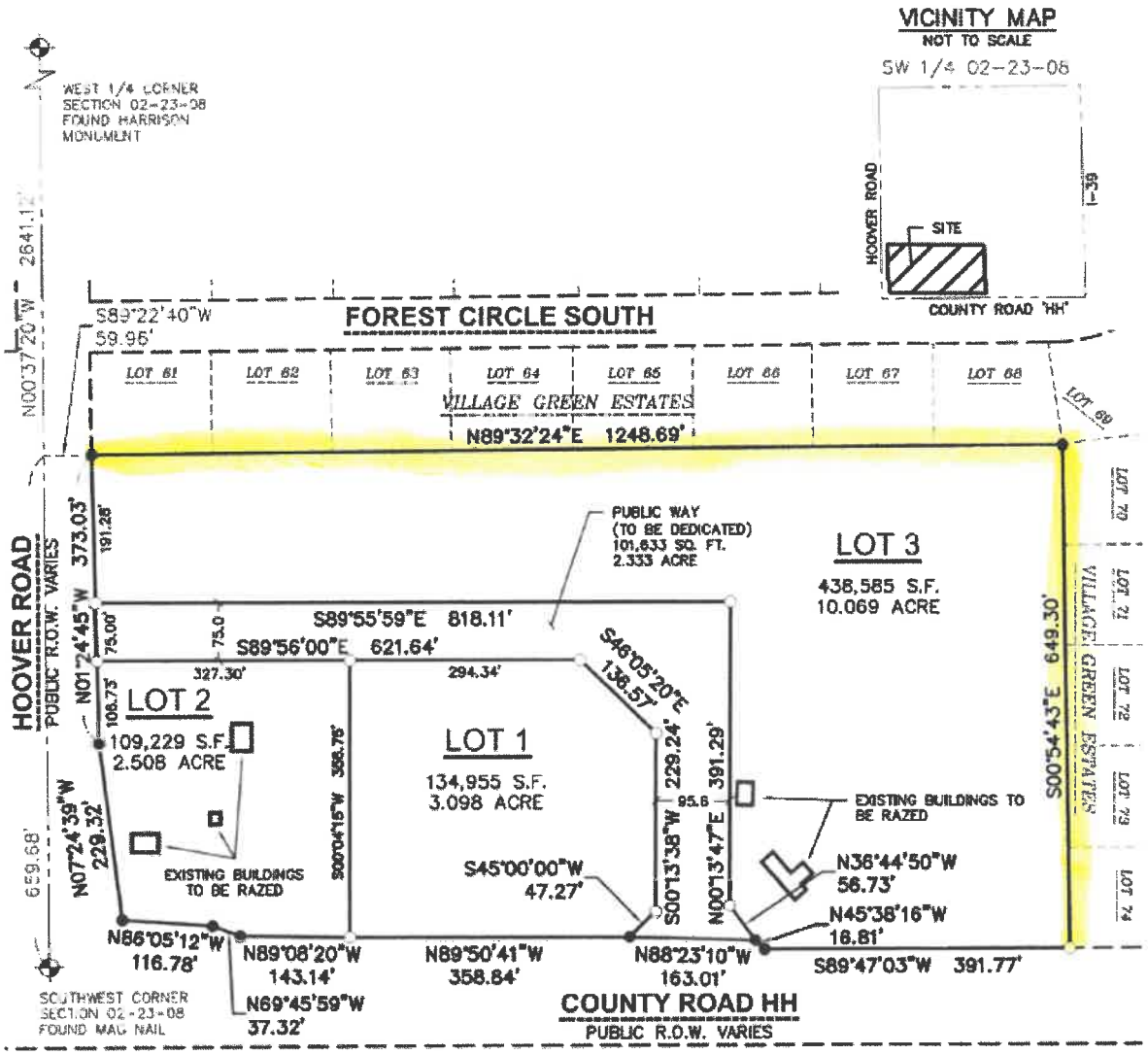
Evergreen trees: 1 per 20 linear feet along residential edges

Shrubs: 1 per 6 linear feet (minimum 60% evergreen)

Groundcover: minimum 80% vegetative coverage

The buffer shall achieve at least 70% year-round visual opacity within three years and at maturity be at a height of 40-60 feet, or an equivalent standard approved by the Plan Commission.

Below is a diagram of the land proposal indicating with yellow where the buffer needs to be:



Proposed Land Division

3. **Maintenance and Replacement**

All buffer vegetation and fencing shall be maintained by the property owner. Dead or failing elements shall be promptly replaced. Maintain planting densities overtime, replanting if needed.

4. **No Chemical Maintenance**

No pesticides, herbicides, or chemical treatments shall be used for routine buffer maintenance.

5. **Protection Against Reduction**

Buffers shall not be reduced or removed without Plan Commission and Common Council approval.

Future Phases, Scope Creep, and Limitations of Approval

1. **Phase-Specific Approval**

This conditional use permit shall apply only to Lot 1 and Lot 2 of Phase 1 as identified in the certified survey map.

2. **No Automatic or Implied Approvals**

Approval shall not be construed as approval of future phases, uses, or expansions.

3. **Future Review Required**

Any future phase, building, or increase in intensity shall require separate Plan Commission and Common Council approval (§23.01(19)).

4. **Cumulative Impact Review**

Future applications shall be evaluated for cumulative impacts to traffic, groundwater, lighting, noise, and neighborhood compatibility (§23.01(4)).

Enforcement and Applicability

All conditions of approval shall be enforceable conditions of the conditional use permit. Failure to comply shall constitute grounds for enforcement action, permit modification, or revocation pursuant to §23.01(19) and applicable City ordinances. All conditions shall run with the land and bind all future owners, tenants, and operators.

Closing Statement

These conditions are reasonable, necessary, and consistent with the City's zoning authority. They ensure that approval of the proposed uses does not compromise public health, groundwater protection, or neighborhood compatibility, and that development proceeds responsibly and transparently over time.

These requests represent the collective voice of many residents who will live with the long-term impacts of this development. While residents may hold different views on how and where growth should occur, there is strong agreement on the need for enforceable safeguards that protect groundwater, neighborhood safety, and quality of life.

We respectfully ask the Plan Commission to give this collective input meaningful weight as part of its decision.

Submitted on behalf of Village Green neighborhood residents (individual resident names and contact information are attached for the record).

packet from DeeAnn Donahue
3395 Howard Ave.

I have been a resident of Village Green Estates since 1994. In December 1995, the Town of Plover and Stevens Point adopted a Cooperative Boundary Adjustment Plan, which covered the annexation of Village Green. The primary purpose of the plan was to protect Whiting's Wellhead, as many of the 225 residences' wells tested were high in nitrates and many had failing septic systems.

Within this plan, it stated that in 1993 due to the concern for Whiting's water supply, all the surrounding communities and Portage County adopted Wellhead Protection Ordinances, which new development is to abide by.

So as you can see, protecting the wellheads has always been a priority for all surrounding municipalities. It is even more important today, as we see so many areas struggling with polluted water. And as Mayor Wiza so eloquently stated at the August 4 Plan Commission meeting, at 1:16:26, "Once you pee in a pool, you can't unpee. That's important. Protecting our groundwater is paramount." This new development is less than ~~a mile~~ 1500 ft from Whiting's Wellhead. This is "paramount" and extra groundwater and human health protections need to be in place.

Reviewing Kwik Trip's

Conditional Use Permit Standard 1. The establishment, maintenance, or operation of the use will not be detrimental to, or endanger the public health, safety, morals, comfort, or general welfare.

Is this conditional use permit standard met? No

Explanation why this CUP standard is not met: Stevens Point's Zoning Code Chapter 23.02(4)(e)(1) states "Certain land use practices and activities can seriously threaten or degrade groundwater quality. The purpose of this Wellhead Protection Ordinance is to institute land use regulations and restrictions to protect the municipal water supplies of the Villages of Amherst, Junction City, Plover and **Whiting**, and the City of Stevens Point, and to promote the public health, safety and general welfare of the residents of Portage County." It's very peculiar that the staff report does **not** include this, as it is in **OUR** zoning code. The 18-acre parcel is in the City's wellhead protection district requiring a conditional use for gas stations, and Whiting's wellhead protection district **prohibiting** gas stations per it's Wellhead Protection Ordinance 31.05(4)(i).

Public health will be endangered by this development. Gasoline contains benzene. According to a report from the American Cancer Society, people are exposed to benzene through gasoline fumes and automobile exhaust. Exposure to it is linked to a higher risk of cancer, particularly leukemia and other blood cell cancers.

Groundwater is also vulnerable. A report to the legislature from Wisconsin Groundwater Coordinating Council states identifying and monitoring known sources of volatile organic compounds (VOCs) is key to the protection of drinking water. As of June 2025, the DNR monitors over 700 landfills, tracks 21,000 leaking tanks, and has recorded about 46,000 spills. VOCs are chemicals that evaporate when exposed to air—including gasoline. Long-term exposure to VOCs may cause cancer, liver damage, spasms, and impaired speech, hearing, and vision.

The Bureau for Remediation and Redevelopment Tracking System (BRRTS) is a database that reports potential contamination and clean up activities. Since January 1, 2015, there has been

11,409 gasoline spills in Wisconsin. The following were specifically at Kwik Trips in Wisconsin, some in Portage County. Many of the spills were by people pumping gas and not paying attention losing 1 to 50 gallons of gas on the pavement. Some of these led to groundwater and soil contamination taking two years to clean up, and then not 100% clean. For other spills, Kwik Trip has had to provide assessment of vapor intrusion pathways. Another spill was due to a delivery truck with a leak in its saddle tank. A 50-gallon spill happened due to a transformer rupture causing a pump to not shut off. What do these examples mean? Human error causes groundwater contamination. Can't eliminate human error. Equipment fails. It's not if but when. Will double-walled tanks and piping leak? Is there evidence from monitored installations that they won't leak in 50 years? How often are tanks replaced? What is state-of-the art today is not tomorrow.

So per this evidence, Kwik Trip has not met the conditional use permit standard.

The staff report also contains items needing additional consideration. 7 states "All monitoring shall be continuous monitoring, meaning that an alarm shall sound **OR** proper authorities shall be immediately notified if a leak is detected". I think staff may want to proof read this. How do you know the monitoring system works? Where is the evidence it will work for 50 years? There is no way to literally see if underground tanks are leaking.

12, 20, and 23 have or will be discussed. I agree—how can a plan be evaluated when it does not yet exist?

Remediation of groundwater contamination will be difficult after it has traveled. There are many buildings in the way as the pollutants move towards the wellhead making cleanup complicated. Imagine a leak happens the day after monitoring is completed. It will be three months before the next monitoring, and then Kwik Trip is given an additional 2 months to delineate the spill if it is detected. Contaminates can travel far in 5 months. Then there is additional time for a plan to be enacted. What incentive does Kwik Trip have to respond quickly to a spill?

The Purpose and Intent section of the Stevens Point Zoning Code states "Zoning regulations are intended to promote public health, safety, and welfare; protect water, sanitation, and drainage; conserve natural resources; stabilize property values; and prevent land uses that create hazards or dangers (23.01(4)). The City's conditional use provisions further authorize the Plan Commission to impose conditions necessary to prevent harm and mitigate impacts associated with high-intensity uses (23.01(19)).

Given the proximity of this site to sensitive groundwater resources and established residential neighborhoods, the following conditions are respectfully requested.

Requested Conditions – all refer to 23.01(4) and 23.01(19)

1. **Groundwater Protection Plan Required Prior to Permit Approval**

Pursuant to the City's obligation to protect public health and natural resources and its authority to require site-specific documentation for conditional uses (23.01(19), a comprehensive groundwater protection plan shall be submitted, reviewed, and approved as part of the conditional use permit. Approval of the permit shall not be granted contingent upon a future or post-approval submittal. The Plan Commission must have the opportunity to evaluate the

adequacy of groundwater safeguards before determining that the use is appropriate at this location.

2. Chemical Storage Disclosure and Limitations

The applicant shall provide full disclosure and limits on chemical storage, quantities, location, and use, including a listing of chemicals on site and a map showing locations. Storage or use of any chemicals not expressly identified and approved shall be prohibited without additional Plan Commission review and approval (23.01(4), 23.01(19).

3. Air Quality Monitoring for Benzene and Volatile Organic Compounds

To protect public health and welfare (23.01(4)), baseline and ongoing air quality monitoring for benzene and other related volatile organic compounds shall be required or, alternatively, the applicant shall demonstrate through third-party analysis that air quality impacts will not adversely affect adjacent residential areas. A mitigation plan identifying action thresholds and corrective measures shall be submitted, with results made available to the City upon request. This matters because groundwater impacts are irreversible. Benzene is classified by EPA as a know human carcinogen for all routes of exposure. Prevention must occur before approval, not after construction.

4. Daytime Fuel Tank Filling Requirement

Fuel tank filling operations shall be restricted to daytime hours because this has been found to reduce accidents and ensures better visibility and immediate response in the event of a spill. This is a reasonable operational condition authorized under 23.01(19).

5. Mandatory Spill and Leak Notification Protocol

Require spill and leak notification within 2 hours to Stevens Point, Whiting, Plover, and DNR–Groundwater Section Manager.

RESOLUTION APPROVING THE BOUNDARY ADJUSTMENT PLAN AND AGREEMENT BETWEEN THE CITY OF STEVENS POINT AND THE TOWN OF PLOVER

WHEREAS, the City of Stevens Point and the Town of Plover desire to take advantage of Wisconsin State Statute 66.023 to jointly develop a Boundary Adjustment Plan and Agreement; and

WHEREAS, the City of Stevens Point and the Town of Plover passed initial Authorizing Resolutions signifying their intent to develop a cooperative Boundary Adjustment Plan and Agreement; and

WHEREAS, the Boundary Adjustment Plan and Agreement will accomplish a coordinated, adjusted and harmonious development of the territory covered by the Agreement, of which a legal description is herein attached, which will, in accordance with existing and future needs, best promote public health, safety, morals, order, convenience, prosperity and the general welfare, as well as the efficiency and economy in the process of development; and

WHEREAS, the Boundary Adjustment Plan and Agreement identifies boundary changes that are to occur and the conditions that will trigger the boundary changes, how services will be provided to the area and by whom, and a Land Use Plan that will be used to guide development; and

WHEREAS, the Boundary Adjustment Plan and Agreement has been prepared in accordance with Section 66.023 of the Wisconsin State Statutes; and

WHEREAS, a joint Public Hearing was held on September 13, 1995, in accordance with Section 66.023(4)(b) of the Wisconsin Statutes to solicit input on the Boundary Adjustment Plan and Agreement and comments were received and considered in the preparation of the final Plan and Agreement; and

WHEREAS, the City Finance Committee, Plan Commission and Public Works Committee have recommended adoption of the Boundary Adjustment Agreement to the Common Council, now therefore be it

RESOLVED, that the Common Council of the City of Stevens Point adopts and approves the final version of the Cooperative Boundary Adjustment Plan Between The City of Stevens Point And The Town of Plover. Portage County. Wisconsin, dated November 28, 1995, which is incorporated herein by reference and submits the Plan and Agreement to the Department of Administration for review in accordance with Section 66.023(4)(s) of the Wisconsin Statutes.

APPROVED: _____
Gary Wescott, Mayor

ATTEST: _____
Barbara Kranig, City Clerk

Dated: December 18, 1995
Passed: December 18, 1995

SECTION 1.0 INTRODUCTION AND PURPOSE OF THE COOPERATIVE BOUNDARY ADJUSTMENT PLAN

SECTION 1.1 INTRODUCTION

On June 8, 1994 the City of Stevens Point and the Town of Plover, in conjunction with the Portage County Planning and Zoning Department, held a joint public informational meeting to explore the feasibility of an intergovernmental agreement between the City and Town. This agreement would utilize the recently passed state statute regarding boundary changes pursuant to an approved cooperative plan (State Statute 66.023), to provide municipal sewer and water to Town of Plover residents that are located north of County Trunk Highway (CTH) HH and west of United States Highway (USH) 51 with municipal boundaries adjusted from the Town of Plover to the City of Stevens Point effective ten years after sanitary sewer and water are installed.

At this meeting, which was attended by approximately 150 people, consensus was reached to pursue a Cooperative Boundary Adjustment Plan, as outlined by State Statute 66.023, between the City of Stevens Point and the Town of Plover. In addition, the Portage County Planning and Zoning Department was requested and agreed to serve as a neutral third party and assist in the preparation of the Cooperative Boundary Adjustment Plan.

The City of Stevens Point and the Town of Plover propose to develop a Cooperative Boundary Adjustment Plan, as authorized by Wisconsin State Statute 66.023. This Statute entitles the City of Stevens Point and the Town of Plover to set boundaries between themselves because they are parties to the Cooperative Boundary Adjustment Plan and Agreement.

Figure 1 illustrates that the territory affected by the Cooperative Boundary Adjustment Plan currently lies within the Town of Plover. Once conditions set forth in the Plan have been satisfied, this area will become part of the City of Stevens Point. Neighboring jurisdictions include the Villages of Plover and Whiting.

SECTION 1.2 PURPOSE

The purpose of the Cooperative Boundary Adjustment Plan is outlined by State Statute 66.023(3)(b), which requires that the Plan guide and accomplish "a coordinated, adjusted and harmonious development of the territory covered by the Plan which will, in accordance with existing and future needs, best promote public health, safety, morals, order, convenience, prosperity or the general welfare, as well as efficiency and economy in the process of development."

The Cooperative Boundary Adjustment Plan requires that specified boundary changes occur during the planning period and identifies dates by which such changes shall occur. The specified boundary changes are subject to the occurrence of conditions set forth in the Plan. When completed, the Cooperative Boundary Adjustment Plan will be submitted to the Department of Administration for review. The Plan also requires that a contact person who is empowered to speak for all the participating municipalities be identified. The following has been identified as the contact person:

Daniel R. Mahoney, Associate Planner
Portage County Planning and Zoning Department
1516 Church Street
Stevens Point, WI 54481

7. The City of Stevens Point shall administer and enforce the following Permits and Ordinances within the Boundary Adjustment Area:

   a. **City of Stevens Point Subdivision Ordinance**

   b. **City of Stevens Point Zoning Ordinance**

   c. **City of Stevens Point Wellhead Protection Ordinance**

   d. **City of Stevens Point Building Permit**

   e. **City of Stevens Point Sign Permit**

   f. **City of Stevens Point Well Permit**

   g. **City of Stevens Point Sewer/Water Codes** (City charges to those who live within the Boundary Adjustment Area will follow City policies).

8. The City of Stevens Point shall assume any and all assessments for sanitary sewer and water service to City owned property.

B. SERVICES TO BE PROVIDED BY THE TOWN OF PLOVER FOR THE DURATION OF THE BOUNDARY ADJUSTMENT AGREEMENT

To ensure that adequate provision is made for the delivery of necessary municipal services to the Boundary Adjustment Area, the Town of Plover shall provide the following services and assume the following responsibilities:

1. The Town of Plover shall establish a Utility District for the express purpose of providing sanitary sewer and water in accordance with the Boundary Adjustment Agreement. Such Utility District, or portions thereof, shall dissolve upon boundary adjustment from the Town of Plover to the City of Stevens Point.

2. Assessment and billing procedures for the Phase 1 and 2 Sanitary Sewer and Water Extension Projects shall be determined by the Town of Plover Board, in accordance with utility district requirements.

3. Services currently provided to residents who live within the Boundary Adjustment Area by the Town of Plover, including police, fire, recycling/garbage pickup, and road maintenance, will continue to be provided according to Town of Plover servicing provisions.

   As such, police protection will be provided to the Boundary Adjustment Area by the Portage County Sheriff's Department. This service will continue to be funded through the Portage County tax levy, therefore, there is no Town of Plover tax levy impact.

   The Town of Plover shall continue to contract with the Village of Plover for fire protection services for lands located within the Boundary Adjustment Area until municipal boundaries are adjusted from the Town of Plover to the City of Stevens Point. This service will be funded through the Town's tax levy. The Town of Plover, in its sole discretion, maintains the right to renegotiate a new fire protection agreement with any other entity.

The County's Subdivision Ordinance provides that no land may be approved for subdivision which is found to be unsuitable for the proposed use. Under this provision, a number of subdivision proposals throughout Portage County have been denied by the County due to the potential for land use conflicts and health problems related to adjacent, intensive agricultural activities. A variety of other requirements may be applicable to a proposed land division including: groundwater testing/monitoring, driveway separation standards, parkland dedication, design standards for lots and construction standards for roads.

C. PORTAGE COUNTY FLOODPLAIN ZONING ORDINANCE

Floodplain development in Portage County is regulated in accordance with Wisconsin Administrative Code NR116 and the County's Floodplain Zoning Ordinance. These regulations allow certain types of construction within the floodfringe under specific conditions, but prohibit new construction within the floodway. The Floodplain Ordinance adds layers of regulations to the underlying zoning districts, and are referred to as "overlay" districts. That part of the Boundary Adjustment Area that lies within and along McDill Pond is subject to the County's Floodplain Zoning Ordinance.

D. PORTAGE COUNTY SHORELAND ZONING ORDINANCE

Portage County's Shoreland Zoning Ordinance regulates the use of certain wetlands throughout the County, as mandated by Wisconsin Administrative Code NR115. This Ordinance regulates only those wetlands within 300 feet of navigable rivers and streams (or to the landward side of the floodplain), and within 1,000 feet of navigable lakes, ponds and flowages. The U.S. Army Corps of Engineers also regulates the use of wetlands, under Federal authority. That part of the Boundary Adjustment Area that lies along McDill Pond is subject to the County's Shoreland Zoning Ordinance.

E. PORTAGE COUNTY WELLHEAD PROTECTION ORDINANCE

A portion of the Boundary Adjustment Area, as shown in Figure 9, is subject to the Portage County Wellhead Protection Ordinance. The Wellhead Protection Ordinance was adopted by the Portage County Board of Supervisors in February 1993. The purpose of this ordinance is to protect municipal water supplies and promote the public health, safety and general welfare of the residents of Portage County.

Lands within the wells cone of depression (denoted as "A" in Figure 9) are subject to the most stringent land use and development restrictions because of close proximity to municipal wells and the corresponding high threat of contamination. Permitted uses in Groundwater Protection Overlay District A include parks and playgrounds, wildlife areas, nonmotorized trails, sewered residential development (subject to maximum lawn areas), unsewered residential development on lots of record (also subject to maximum lawn areas) and certain agricultural activities. Certain commercial uses served by municipal sanitary sewer may be allowed as special exceptions.

A secondary portion of the municipal well recharge area that is protected is the land which lies within the five year time of travel zone, upgradient from the Whiting municipal wells (denoted as "B" in Figure 9). Land use restrictions in Groundwater Protection Overlay District B are less restrictive than in District A because of longer flow times and a greater potential for remediation, dilution and attenuation. Permitted uses in District B include all uses listed as permitted in District A, plus agricultural activities, above ground petroleum product storage tanks up to 660 gallons, basement heating fuel storage tanks, certain commercial and industrial uses served by municipal sewer and unsewered single family residential uses (subject to maximum lawn areas). Nurseries, cemeteries, salt storage facilities and recycling facilities may be allowed as special exceptions.

SECTION 3.3 EXISTING ENVIRONMENTAL CONDITIONS

A. TOPOGRAPHY

As shown in Figure 4, the topography of the Boundary Adjustment Area ranges from 1075 feet along the shoreline of McDill Pond to 1095 feet above sea level at the boundary's eastern edge. The land generally drains from east to west, toward McDill Pond. This generally flat topography presents no significant development limitations, other than potential surface drainage problems. The sharpest changes in topography within the Boundary Adjustment Area occur along the shoreland of McDill Pond. Because a 100 foot setback will be required for any building construction adjacent to McDill Pond, the slopes adjacent to the pond should not be adversely affected.

B. GEOLOGY

The Boundary Adjustment Area is located in the Central Wisconsin Sand Plain geographical province. This area is characterized by a relatively thick layer of highly permeable glacial sediments overlying impermeable rock. Depth to bedrock in the Village Green Subdivision ranges from 65 to 85 feet. The glacial material consists primarily of outwash sands and gravel and tends to be quite uniform in composition. These outwash sands and gravel serve as a groundwater aquifer. This aquifer provides an abundant supply of water as evidenced by the Village of Whiting's municipal well, which is located adjacent to the Boundary Adjustment Area, and the multitude of private wells that serve existing residences in the Boundary Adjustment Area.

In the Boundary Adjustment Area, a large percentage of the land is comprised of single family residences that are located on lots averaging 18,000 square feet. Highly permeable sandy soils generally have low organic matter content, which allows organic wastes and fertilizers to enter the groundwater. Once organic wastes and fertilizers move past the root zone, where organisms and roots are able to absorb them, there is little chance of decay. These materials then move through the aquifer to a discharge point, which may be a surface water body such as McDill Pond or water supply wells.

C. GROUNDWATER

Currently, residents within the Boundary Adjustment Area rely exclusively on groundwater as a source of safe drinking water. Water for domestic use must be free of bacteria and viruses and meet other aspects of safe drinking water standards. Some components, such as high iron (which creates a bad taste), may lower the aesthetic quality of water, but do not make it unsafe to drink.

Nitrates have received the most public attention, in terms of groundwater quality issues. Nitrates occur naturally as the decomposition by-product of organic materials and are usually found in small quantities, i.e. less than 2 milligrams per liter (mg/l). Nitrates over 2 mg/l generally indicate human impact, such as septic system effluent, agricultural and residential fertilizers, and disposal of domestic animal wastes. The public health enforcement standard for nitrates is 10 mg/l, which is considered unsafe for infants less than 6 months in age. In addition, some local lending institutions have considered nitrates greater than 10 mg/l to be a basis for denying residential loan applications.

Groundwater quality is of specific concern within the Boundary Adjustment Area. A study, initiated in 1987 (Subdivision Impacts On Groundwater Quality), attempted to quantify the impacts of unsewered subdivisions on groundwater quality in the Central Sands Area of Wisconsin. One of the two subdivisions studied lies within the Boundary Adjustment Area (Village Green Subdivision). The report concluded that subdivisions on sandy soils do impact groundwater quality, with nitrate-N levels exceeding 10 mg/l. Chloride, phosphorous, sodium and limited organic chemicals were also found in elevated concentrations downgradient of the subdivision. The report also documented elevated background nitrate levels, which is probably the result of intensive agricultural activity to the east.

A review of private well water tests within the Boundary adjustment Area reveals that water quality is of significant concern. Between 1990 and 1994, approximately 25 percent of the 225 residences located within the Boundary Adjustment Area had their water tested. Of the wells tested, 44 percent had nitrates that exceeded 10 mg/l. This result is more than double the countywide average (20 percent of private wells tested for nitrates exceed 10 mg/l) of wells tested that had nitrates that exceeded the health standard of 10mg/l. In addition, another 37 percent of the wells tested had nitrate levels between 5 and 10 mg/l.

In addition to private well water quality, residents of this area have also been concerned about failing septic systems. These two factors have been instrumental in some residents seeking annexation to the City of Stevens Point, where sanitary sewer and water are available. The City's current policy, however, is to require annexation before providing sanitary sewer and water service. Where sewer and water have been in close proximity to those seeking it, individual or small scale annexations have taken place. An example would be the recent annexations along Alder Street.

Groundwater protection is also a concern to the Village of Whiting, because its municipal wells are located adjacent to the Boundary Adjustment Agreement Area. The high density of private septic systems within the Agreement Area, coupled with intensive agricultural activities to the east, has elevated nitrates in the Village of Whiting's municipal water supply and forced the Village to install a costly denitrification system. Two of the Village's municipal wells have nitrate levels that currently exceed 20 mg/l (prior to the denitrification process).

To protect their municipal water supplies, communities have, in conjunction with Portage County, adopted Wellhead Protection Ordinances. These ordinances institute land use regulations and restrictions to protect municipal water supplies and wellfields. The Boundary Adjustment Area lies within the A and B Zones (cone of depression and 5 year time of travel, respectively) of the Village of Whiting's municipal wellfield. New development within the Boundary Adjustment Area must be served by sanitary sewer and water, according to the City of Stevens Point and Portage County Wellhead Protection Ordinances. These Ordinances, however, do not place restrictions on existing development. The approximately 225 residences, therefore, are not subject to the requirements of the Stevens Point or Portage County Wellhead Protection Ordinances.

D. SOILS

There are four soil types found in the Boundary Adjustment Area (Figure 5), including Alluvial land, wet; Plainfield loamy sand, 0-2 percent slopes; Plainfield loamy sand, 2-6 percent slopes; and Richford loamy sand, 0-2 percent slopes. Plainfield loamy sand, 0-2 percent slopes, is the most abundant soil type. The Alluvial soils are found along McDill Pond. The soils found in the Boundary Adjustment Area are sandy in nature and present few limitations to development, except they are characterized as rapidly permeable and constitute a "moderate" limitation for on-site sanitary systems because of the danger of contaminating groundwater supplies. The possible pollution of groundwater from the

Stevens Point
Zoning Code
Chapter 23.02(4)

use shall be set back from the lot lines a distance that equals or exceeds the height of the tower.

b) Any new wireless communication tower that is constructed on or substantially modified on a parcel that is adjacent to a parcel that allows for a single-family residence as a permitted use shall be set back from the lot lines a distance that equals or exceeds the height of the tower.

c) Any new wireless communication tower that is constructed on or substantially modified on a parcel that identifies a single-family residence as a conditional or prohibited use shall be set back from lot lines a distance equal to the setbacks of a principal building/structure pursuant to the underlying zoning district development standards. Setbacks may be reduced to a lesser specified agent if the applicant submits a report stamped by a Wisconsin Registered Professional Engineer that certifies that the wireless communication tower is designed and engineered to collapse upon failure within the lesser specified distance unless the City has and provides the applicant with substantial evidence that the engineering certification is flawed.

e) Groundwater Protection Overlay District

1) Purpose and Authority

Purpose. The residents of Stevens Point and Portage County, whether served by private wells or municipal supplies, depend exclusively on groundwater for a safe drinking water supply. Certain land use practices and activities can seriously threaten or degrade groundwater quality. The purpose of this Wellhead Protection Ordinance is to institute land use regulations and restrictions to protect the municipal water supplies of the Villages of Amherst, Junction City, Plover and Whiting, and the City of Stevens Point, and to promote the public health, safety and general welfare of the residents of Portage County.

Authority. Statutory authority of the City to enact these regulations was established by the Wisconsin Legislature in 1983, Wisconsin Act 410 (effective May 11, 1984), which specifically added groundwater protection to the statutory authorization for municipal planning and zoning to protect public health, safety and welfare.

a) Application of Regulations

The regulations specified in this Groundwater Protection Ordinance shall apply to the incorporated and unincorporated areas of the City of Stevens Point and Portage County that lie within the recharge areas for municipal water supply wells as a Groundwater Protection Overlay District and are in addition to the requirements in the underlying zoning district. If there is a conflict between this Ordinance and the Zoning Ordinance, the more restrictive provision shall apply.

The Groundwater Protection Overlay District is divided into three districts: District A, District B, and District C. All district boundaries are intended to end or begin at road centerlines, railways, surface water features, or tax parcel lines. The boundaries of the districts are shown upon maps of the City of Stevens Point, being designated as the "Groundwater Protection Districts for Municipal Recharge Areas in and around Stevens Point" as adopted or amended by the Common Council.

b) Definitions

1) Animal Confinement Facilities - Locations of confinement of livestock at a density exceeding three animal units per acre.
2) Animal Units - A unit of measure used to determine the total number of single animal types or combination of animal types, as specified in s. NR 243.11, that are at an animal feeding operation.
3) Aquifer - A saturated, permeable geologic formation that contains and will yield significant quantities of water.
4) Cone of Depression - The area around a municipal well, in which the water level has been lowered at least one tenth of a foot by pumping the well.
5) Farm Plan – A written document which details the conduct and management of agricultural activities on a farm utilizing best management practices and applicable Federal, State, and local standards based on the potential contamination of groundwater. A farm plan must be approved by the Land and Water Conservation section of the Planning and Zoning Department.
6) Hazardous Chemical – Chemicals identified as hazardous by CFR 40 Part 302
7) Ten Year Time of Travel (TOT) - The Ten-Year TOT is the recharge area upgradient of the cone of depression, the outer boundary of which it is determined or estimated that groundwater will take ten years to reach a pumping well.
8) Twenty Year Time of Travel (TOT) - The Twenty-Year TOT is the recharge area upgradient of the cone of depression, the outer boundary of which it is determined or estimated that groundwater will take twenty years to reach a pumping well.
9) POWTS - Private On-Site Wastewater Treatment System or POWTS, has the meaning given under s. 145.01(12), Wis. Stats.
10) Toxic - When ingested or absorbed, is harmful or fatal to living organisms.
11) Recharge Area - Area in which water reaches the zone of saturation by surface infiltration and encompasses all areas or features that supply groundwater recharge to a well.
12) Wellfield - A piece of land used primarily for the purpose of locating wells to supply a municipal water system.

2) Groundwater Protection Overlay District A

a) Intent

District A of the groundwater protection overlay district is the area of land within the cone of depression as shown on the groundwater protection overlay district maps. These lands are subject to the most stringent land use and development restrictions because of close proximity to the wells and therefore greatest threat of contamination.

b) Permitted Uses

The following uses are permitted uses within Groundwater Protection Overlay District A, provided that such uses are subject to minimum separation distances of chapter NR 811.12(5)(d) Wisconsin Administrative Code. Uses not listed here are considered prohibited uses unless a determination of similarity to a permitted or special exception use (based on potential for groundwater contamination) is made by the Planning Commission and/or Common Council.

1) Parks and playgrounds, provided there are no POWTS dispersal components or fuel storage tank facilities
2) Wildlife areas, natural and woodland areas
3) Nonmotorized trails, such as biking, skiing, nature and fitness trails
4) Sewered residential developments
5) Unsewered (single family) residential development only on existing lots of record on the effective date of this Ordinance.
6) Agricultural activities, except those listed as prohibited.
7) Above ground liquid propane tanks, 1000 gallons or less, accessory to a permitted or special exception use

c) Conditional Uses

The following uses may be allowed as special exceptions within Groundwater Protection Overlay District A, provided that such uses are subject to minimum separation distances of chapter NR 811.12(5)(d) Wisconsin Administrative Code.

1) Commercial uses served by municipal sanitary sewer except those listed as prohibited.

d) Prohibited Uses

The following uses are prohibited uses within the Groundwater Protection Overlay District A. These uses are prohibited based on the high probability that the activities routinely associated with these uses (storage, use, and handling of potential pollutants) will cause groundwater contamination.

1) Above or below ground hydrocarbon, petroleum, or hazardous chemical bulk storage tanks
2) Animal confinement facilities
3) Any manufacturing or industrial use
4) Any commercial use where hazardous chemicals are an integral part of such use including but not limited to:
    a) Bulk fertilizer and/or pesticide facilities
    b) Dry cleaning businesses
    c) Electroplating facilities
    d) Exterminating businesses
    e) Gas stations
    f) Paint and coating manufacturing
    g) Printing and duplicating businesses
    h) Radioactive or toxic materials storage and waste facilities
5) Asphalt and concrete products manufacturing
6) Automobile, truck, agricultural implement and equipment, municipal, small engine and other vehicle repair of a commercial nature
7) Bus or truck terminals
8) Cemeteries
9) Junk yards or auto salvage yards
10) Landfills or waste disposal facilities
11) Nonmetallic mining
12) Manure and animal waste storage facilities
13) Recycling facilities
14) Salt or deicing material storage
15) Septage and/or sewage sludge spreading
16) Slaughterhouses
17) Spray wastewater facilities
18) Wastewater treatment or disposal facilities that are commercial, industrial, or municipal

3) Groundwater Protection Overlay District B

a) Intent

District B of the groundwater protection overlay district is the area of land which contributes water to the municipal well starting at the line which delineates the cone of depression and ends at the line delineating the ten-year time of travel. Land use regulations within Groundwater Protection Overlay District B are less restrictive than in Overlay District A because of longer flow times and a greater potential for remediation, dilution and attenuation. Agricultural Best Management Practices (BMP) minimizing use of pesticides and fertilizers are strongly encouraged.

b) Permitted Uses

Village of Whiting

CHAPTER 31
WELLHEAD PROTECTION

WELLHEAD PROTECTION

Sec. 31.01 Purpose and Authority

The residents of the Village of Whiting depend exclusively on groundwater for a safe drinking water supply. Certain land use practices and activities can seriously threaten or degrade groundwater quality. The purpose of this Wellhead Protection Ordinance is to institute land use regulations and restrictions to protect the Village's municipal water supply and well fields, and to promote the public health, safety, and general welfare of the residents of the Village of Whiting.

Statutory authority of the Village to enact these regulations was established by the Wisconsin Legislature in 1983, Wisconsin Act 410 (effective date May 11, 1984), which specifically added groundwater protection to the statutory authorization for municipal planning and zoning to protect public health, safety and welfare.

Sec. 31.02 Application of Regulations

The regulations specified in this Wellhead Protection Ordinance shall apply only within the Village of Whiting corporate limits, to all lands that lie within 1,500 feet of the Whiting well field (Cone of Depression) and within the five year Time of Travel (TOT) of the well field.

Sec. 31.03 Definitions

1) AQUIFER. A saturated, permeable, geologic formation that contains and will yield significant quantities of water.
2) CONE OF DEPRESSION. The cone-shaped area around a well, in which the eater level has been, lowered 0.1 of a foot by pumping of the well. The Wisconsin Geological and Natural History Survey Special Report No. 10, entitled "Wellhead-Protection Districts in Wisconsin: An Analysis and Test Applications", identified the Cone of Depression for the Village of Whiting to be a radius of 1,422 feet. For ease of determination and to protect the entire Village of Whiting well field, the Cone of Depression or Groundwater Protection Overlay District A has been established as being 1,500 feet from the property boundaries of the Whiting well field.
3) FIVE YEAR TIME OF TRAVEL (TOT). The Five Year TOT is the recharge area up gradient of the cone of depression, the outer boundary of which it is determined or estimated that groundwater and potential contaminants will take five years to reach a pumping well(s). The Five Year TOT for the Village of Whiting well filed is established as a distance of one (1) mile up gradient from the ownership boundaries of the well field, based on groundwater flow rates calculated for the Whiting recharge area as discussed in the Wisconsin Geological and Natural History Survey Special Report No. 10.
4) NATURAL VEGETATION. Includes native trees, shrubs, prairie species, and non-native plants, excluding lawns.
5) NET LOT AREA. Determined by subtracting the area of the building footprint, driveways, patios, pools and other impervious surface from the total lot area.
6) RECHARGE AREA. Area in which water reaches the zone of saturation by surface infiltration and encompasses all areas or features that supply groundwater recharge to a well.
7) WELL FIELD. A piece of land used primarily for the purpose of locating wells to supply a municipal water system.

Sec. 31.04 Groundwater Protection Overlay District A - Cone Of Depression

1) INTENT. The primary area of the Whiting recharge area to be protected is the land within 1,500 feet of the boundary of the Whiting well field, known as the Cone of Depression. These lands are subject to the most stringent land use and development restrictions because of their close proximity to the well field and the corresponding high threat of contamination.
2) PERMITTED USES. The following uses are permitted uses within Groundwater Protection Overlay District. Uses not listed are to be considered prohibited uses.
   a. Parks provided there is no on-site waste disposal or fuel storage tank facilities associated within this use.
   b. Playgrounds.
   c. Wildlife areas.

d. Nonmotorized trails, such as biking, skiing, nature and fitness trails.

3) PROHIBITED USES. The following uses are prohibited uses within the Groundwater Protection overlay District. Uses not listed are not considered permitted uses, unless specifically listed above under Permitted Uses.
   a. Sewered or unsewered residential uses.
   b. On-site private sewage systems.
   c. Underground storage tanks of any size.
   d. Basement storage tanks.
   e. Agricultural activities.
   f. Pesticide and/or fertilizer storage and use.
   g. Septage and/or sludge spreading.
   h. Animal waste landspreading.
   i. Animal waste facilities.
   j. Animal confinement.
   k. Gas stations.
   l. Vehicle repair establishments, including auto body repair.
   m. Printing and duplicating businesses.
   n. Any manufacturing or industrial businesses.
   o. Bus or truck terminals.
   p. Repair shops.
   q. Landfills or waste disposal facilities.
   r. Wastewater treatment facilities.
   s. Spray wastewater facilities.
   t. Junk yards or auto salvage yards.
   u. Bulk fertilizer and/or pesticide facilities.
   v. Asphalt products manufacturing.
   w. Dry cleaning businesses.
   x. Salt storage.
   y. Electroplating facilities.
   z. Exterminating businesses.
   aa. Paint and coating manufacturing.
   bb. Hazardous and/or toxic materials storage.
   cc. Toxic and hazardous waste facilities
   dd. Radioactive waste facilities.

Sec. 31.05 Groundwater Protection Overlay District B - 5 Year Time Of Travel

1) INTENT. A secondary area of the Whiting recharge area to be protected is the land which lies within the five year groundwater travel zone up gradient from the Whiting well field. The Five Year Time of Travel (TOT) for the Whiting well field is established at one (1) mile from the boundaries of the Whiting well field, based on an average rate of groundwater flow of 1,070 feet per year in the Whiting District B are less restrictive than in overlay District A because of longer flow time and a greater opportunity for containment, dilution and attenuation potential.
2) PERMITTED USES. The following uses are permitted within the Groundwater Protection Overlay District B. Uses not listed are to be considered prohibited uses.
   a. All uses listed as permitted uses in Groundwater Protection Overlay District A.
   b. Sewered residential uses.
   c. Above ground storage tanks (660 gallons).
   d. Basement storage tanks.
   e. Sewered commercial and/or industrial uses, except those listed as prohibited uses in Prohibited Uses.
3) PERFORMANCE STANDARDS. The following standards and requirements shall apply to all uses permitted within the Groundwater Protection Overlay District B.

a. All residential, commercial and industrial uses are allowed only 25% of the net lot area to be maintained in manicured lawn or grass. Seventy-five percent of the net lot area shall be retained or developed into natural vegetative cover not requiring the use of pesticides or fertilization after the initial establishment.
b. All above ground liquid petroleum storage tanks shall be approved leak proof containment equal to 125% of the tank volume.

4) PROHIBITED USES. The following uses are prohibited uses within Groundwater Protection Overlay District. Uses not listed are not considered permitted uses unless specifically listed above under Permitted Uses.
   a. Underground storage tanks of any size.
   b. On-site private sewage systems.
   c. Agricultural activities.
   d. Pesticide and/or fertilizer storage and use.
   e. Septage and/or sludge spreading.
   f. Animal waste landspreading.
   g. Animal waste facilities.
   h. Animal confinement facilities.
   i. Gas stations.
   j. Vehicle repair establishments, including auto body repair.
   k. Printing and duplicating.
   l. Bus or truck terminals.
   m. Repair shops.
   n. Landfills.
   o. Wastewater treatment facilities.
   p. Spray wastewater facilities.
   q. Junkyards or auto salvage yards.
   r. Bulk fertilizer and pesticide facilities.
   s. Asphalt products manufacturing.
   t. Dry cleaning facilities.
   u. Salt storage.
   v. Electroplating.
   w. Exterminating shops.
   x. Pain and coating manufacturing.
   y. Hazardous and toxic materials storage and use.
   z. Hazardous and toxic waste facilities.
   aa. Radioactive waste facilities.
   bb. Tire and battery services.
   cc. Garage and vehicular towing.
   dd. Public and municipal maintenance garages.

Sec. 31.06 Enforcement and Penalties

1) VIOLATIONS. It shall be unlawful to construct or use any structure, land or water in violation of any of the provisions of this Chapter. In case of any violation, the Village Board, Plan Commission, Building Inspector, or any person who would be specifically damaged by such violation, may institute appropriate action or proceedings to enjoin a violation of this Chapter.
2) PENALTIES. Any person, firm, or corporation who fails to comply with the provisions of this chapter of this chapter shall upon conviction thereof, forfeit not less than One Hundred ($100.00) Dollars nor more than Five Hundred ($500.00) Dollars, plus the costs of prosecution for each violation, and in default of payment thereof, but not exceeding Thirty (30) Days, or in the alternative, shall have such costs added to their real property as a lien against the property. Each say a violation exists or continues shall constitute a separate offense.

What is benzene?

Benzene is a colorless, flammable liquid with a sweet odor. It evaporates quickly when exposed to air. Benzene is formed from natural processes, such as volcanoes and forest fires, but most people are exposed to benzene through human activities.

Benzene is one of the 20 most widely used chemicals in the United States. It is used mainly to make other chemicals, including plastics, resins, lubricants, rubbers, dyes, detergents, drugs, and pesticides. In the past it was also commonly used as an industrial solvent (a substance that can dissolve or extract other substances) and as a gasoline additive, but these uses have been greatly reduced in recent decades.

Benzene is also a natural part of crude oil and gasoline (and therefore motor vehicle exhaust), as well as cigarette smoke.

How are people exposed to benzene?

People are exposed mainly by breathing in air containing benzene. Benzene can also be absorbed through the skin during contact with a source such as gasoline, but because liquid benzene evaporates quickly, this is less common.

People can be exposed to benzene:

- At work
- In the environment
- When using some consumer products

The highest levels of exposure typically have been in the workplace, although high-level exposures have decreased greatly over the last several decades due to federal and state regulations.

Workplace exposures

Workers in industries that make or use benzene can be exposed to this chemical. These include the rubber industry, oil refineries, chemical plants, shoe manufacturers, and gasoline-related industries. Other people who may be exposed to benzene at work include steel workers, printers, lab technicians, gas station employees, and firefighters.

Federal regulations limit exposure to benzene in the workplace (see below).

Community exposures

People can be exposed to benzene from gasoline fumes, automobile exhaust, emissions from some factories, and wastewater from certain industries. Benzene is commonly found in air, but the levels in most places are usually very low. Levels of benzene can be higher in enclosed spaces with unventilated fumes from gasoline, glues, solvents, paints, and art supplies. Areas with heavy traffic, gas stations, and areas near industrial sources may also have higher air levels.

Cigarette smoke (either from smoking yourself or from secondhand smoke) accounts for about half of the exposure to benzene in the United States. Benzene levels in rooms containing tobacco smoke can be many times higher than normal.

People can also be exposed to benzene in contaminated drinking water and some foods (although the levels are usually very low).

Does benzene cause cancer?

Exposure to benzene has been linked with a higher risk of cancer, particularly leukemia and other cancers of blood cells.

What do studies show?

Researchers try to determine if a substance causes cancer using 2 main types of studies:

- **Studies in people** (epidemiologic studies)
- **Lab studies** (studies done using lab animals or cells in lab dishes)

Often neither type of study provides conclusive evidence on its own, so researchers usually look at both human and lab-based studies when trying to figure out if something causes cancer.

Studies in people

Rates of leukemia, particularly acute myeloid leukemia (AML), have been found to be higher in studies of workers exposed to high levels of benzene, such as those in the chemical, shoemaking, and oil refining industries.

Some studies have also suggested links to childhood leukemia (particularly AML) as well as acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), and other blood-related cancers (such as multiple myeloma and non-Hodgkin lymphoma) in adults. However, the evidence is not as strong for these cancers.

There is much less evidence linking benzene to any other type of cancer.

Lab studies

When inhaled or swallowed, benzene has been found to cause different types of tumors in lab animals such as rats and mice. These results support the finding of an excess risk of leukemia in humans. However, most studies in people have not found an increased risk of cancers other than leukemia among those with higher exposures.

Benzene has been shown to cause chromosome changes in bone marrow cells in the lab. (The bone marrow is where new blood cells are made.) Such changes are commonly found in human leukemia cells.

What expert agencies say

Several national and international agencies study substances in the environment to determine if they can cause cancer. (A substance that causes cancer or helps cancer grow is called a **carcinogen**.) **The American Cancer Society looks to these organizations to evaluate the risks based on the available evidence.**

Based on animal and human evidence, several expert agencies have evaluated the cancer-causing potential of benzene.

The **International Agency for Research on Cancer (IARC)** is part of the World Health Organization (WHO). One of its goals is to identify causes of cancer. IARC classifies benzene as "carcinogenic to humans," based on sufficient evidence that it causes acute myeloid leukemia (AML). IARC also notes that benzene exposure has been linked with acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), multiple myeloma, and non-Hodgkin lymphoma.

The US **National Toxicology Program (NTP)** is an interagency program that includes the National Institutes of Health (NIH), the Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA). The NTP has classified benzene as "known to be a human carcinogen."

The US **Environmental Protection Agency (EPA)** maintains the Integrated Risk Information System (IRIS), an electronic database of human health effects from exposure to various substances in the environment. The EPA classifies benzene as a known human carcinogen.

(For more information on the classification systems used by these agencies, see Determining if Something Is a Carcinogen and Known and Probable Human Carcinogens.)

Does benzene cause any other health problems?

Benzene is a potentially dangerous chemical. High levels of exposure can cause both short-term and long-term health effects.

Short-term effects

Breathing in high doses of benzene can affect the nervous system, which can lead to drowsiness, dizziness, headaches, tremors, confusion, and/or unconsciousness. Eating foods or drinking fluids contaminated with high levels of benzene can cause vomiting, stomach irritation, dizziness, sleepiness, convulsions, and rapid heart rate. In extreme cases, inhaling or swallowing very high levels of benzene can be deadly.

Exposure to benzene liquid or vapor can irritate the skin, eyes, and throat. If benzene touches the skin, it can result in redness and blisters.

Long-term effects

Long-term exposure to benzene mainly harms the bone marrow, the soft, inner parts of bones where new blood cells are made. This can result in:

- Anemia (a low red blood cell count), which can cause a person to feel weak and tired.
- A low white blood cell count, which can lower the body's ability to fight infections and might even be life-threatening.
- A low blood platelet count, which can lead to excess bruising and bleeding.

There is also some evidence that long-term exposure to benzene might harm reproductive organs. Some women who have breathed in high levels of benzene for many months have had irregular menstrual periods and ovary shrinkage, but it is not known for sure if benzene caused these effects. It is not known if benzene exposure affects fertility in men.

Are benzene levels regulated?

Several US government agencies regulate benzene levels and exposures.

The Occupational Safety & Health Administration (OSHA) is the federal agency responsible for health and safety regulations in most workplaces. OSHA limits exposure to benzene in the air in most workplaces to 1 ppm (part per million) during an average workday and a maximum of 5 ppm over any 15-minute period. When working at potentially higher exposure levels, OSHA requires employers to provide personal protective equipment such as respirators.

The EPA limits the percentage of benzene allowed in gasoline to a yearly average of 0.62% by volume (with a maximum of 1.3%).

The EPA limits concentrations of benzene in drinking water to 5 ppb (parts per billion). Some states may have lower limits. Likewise, the US Food and Drug Administration (FDA) sets a limit of 5 ppb in bottled water.

The Consumer Product Safety Commission (CPSC) considers any product containing 5% or more by weight of benzene to be hazardous, requiring special labeling.

Can I avoid or limit my exposure to benzene?

It might not be possible to avoid benzene completely. But if you are concerned about benzene, you can limit your exposure in several ways .

Stay away from cigarette smoke. If you smoke, it's important to try to quit. It's also important to avoid secondhand smoke. Cigarette smoke is a major source of exposure to benzene, as well as some other harmful chemicals.

Pump gas carefully and use gas stations with vapor recovery systems that capture the fumes. Avoid skin contact with gasoline.

When possible, **limit the time you spend near idling car engines.** This can help lower your exposure to exhaust fumes, which contain benzene (as well as other potentially harmful chemicals).

Use common sense around any chemicals that might contain benzene. Limit or avoid exposure to fumes from solvents, paints, and art supplies, especially in unventilated spaces.

If you are exposed to benzene at your workplace, **talk to your employer about limiting your exposure** through process changes (such as replacing the benzene with another solvent or enclosing the benzene source) or by using personal protective equipment. If needed, you can also contact the Occupational Safety & Health Administration (OSHA), which can provide more information or assistance.

What should I do if I've been exposed to benzene?

For short-term exposure to high levels of benzene, the Centers for Disease Control and Prevention (CDC) recommends getting away from the source of benzene, removing any clothing that may have benzene on it, washing exposed areas with soap and water, and getting medical care as soon as possible.

If you think you may have been exposed to benzene over a long period of time, talk to a doctor. Benzene can be measured in the blood or breath, and breakdown products of benzene can be measured in urine. These tests can only detect recent exposures to benzene. They can't reliably show how much benzene you've been exposed to, nor can they predict possible health effects.

Written by References

VOLATILE ORGANIC COMPOUNDS (VOCs)

Key Takeaways

Continuing to identify and monitor known sources of VOCs is key to the protection of drinking water.

1. Thousands of wells have been sampled for VOCs and about 60 different VOCs have been found in Wisconsin groundwater, but only 34 currently have established health-based NR 140 groundwater quality standards.

2. Sources of VOCs in Wisconsin groundwater include landfills, underground storage tanks, and hazardous spills. As of June 2025, the DNR monitors over 700 landfills, tracks 21,000 leaking tanks, oversees 10,000 groundwater-monitoring facilities, and has recorded about 46,000 spills, some contributing to VOC contamination.

A critical role of GCC agencies is identifying and monitoring all known sources of VOCs, not only landfills. GCC member agencies continue to work on multiple initiatives related to reducing Volatile Organic Compounds (VOCs) in groundwater (see *DHS*, *DNR*, *DATCP*).

For actions to address Volatile Organic Compounds (VOCs) contamination in groundwater, see the *Recommendations Section*.

What are Volatile Organic Compounds (VOCs)?

Volatile Organic Compounds (VOCs) are a group of common industrial and household chemicals that evaporate, or volatilize, when exposed to air. Examples of products containing VOCs include gasoline and industrial solvents, paints, paint thinners, air fresheners and household products such as spot and stain removers. Chemical names for the VOCs in these products include benzene,

Collection of household products containing VOCs including paints, stains, and paint thinners. Photo: Tom Murphy VII

Trichloroethylene (TCE), toluene and vinyl chloride, among others. Improper handling or disposal of VOCs is often the reason why they occur in groundwater.

What are the Human Health Concerns?

Health risks vary depending on the VOC. Short-term exposure to high concentrations of many VOCs can cause nausea, dizziness, anemia, fatigue or other health problems. Long-term exposure to some VOCs may cause cancer, liver damage, spasms, and impaired speech, hearing and vision. For more on the health effects of specific VOCs, see the Wisconsin Department of Health Services (DHS) website.

How Widespread are VOCs in Wisconsin?

Thousands of wells have been sampled for VOCs and about 60 different VOCs have been found in Wisconsin groundwater. While about 60 different VOCs have been found, only 34 currently have established health based groundwater quality standards in ch. NR 140 (groundwater *WI NR 140.10*), or public drinking water system maximum contaminant levels (MCLs) in ch. NR 809 (*WI NR 809.24*). The main sources of VOCs in Wisconsin groundwater are landfills, leaking underground storage tanks (LUSTs), and a variety of facilities that use VOCs in their regular operations, including gas stations, bulk petroleum and pipeline facilities, plating facilities, dry cleaners and other industrial facilities. DNR currently tracks about 700 current or former landfills, 21,000 LUSTs and 10,000 other facilities which are required to monitor groundwater. The DNR also tracks approximately 46,000 spills, some of which are also sources of VOCs. Given how common potential sources of VOCs are, these substances are more frequently found in groundwater near urban industrial and commercial areas. However, exceedances of groundwater standards for VOCs have been reported in every county in the state.

Installation of a compacted clay and geotextile liner at a landfill site in Wisconsin.

How is VOCs Contamination Trending Over Time?

Continuing to identify and monitor known sources of VOCs is key to continued protection of drinking water. Each year, several hundred contaminated sites, some of which involve VOCs, are reported to the DNR. And each year, cleanup begins at another several hundred sites. Continuing to track and respond to this ongoing issue remains an important objective for GCC agencies.

Early studies by the DNR and DHS in the late 1980s and early 1990s focused on VOC contamination from landfills, specifically from those without linings to protect

groundwater from leachate. DNR scientists found that VOCs contaminated groundwater at 60% of unlined industrial landfills and 80% of unlined municipal solid waste landfills[1,2].

Further review of monitoring data showed that while VOC levels typically decrease following the closure of unlined landfills, concentrations remain high and do not always show continued improvement within a reasonable period of time[3].

In the late 1990s, this knowledge raised concerns since increasing numbers of residential developments were located close to old, closed landfills. In 1999, the DNR and DHS designed targeted sampling of private wells near old, closed landfills to investigate and address the problem. For wells where VOCs were detected above drinking water standards, residents were given health advisories not to drink water and the DNR took follow-up measures at the nearby landfills. Much more stringent engineering standards have guided the design of modern landfills (those built after the 1980s), so these have a much better record in terms of limiting VOC contamination. Older landfills, however, continue to remain a concern[4].

Drilling to monitor for VOCs near a Wisconsin landfill. Photo: DNR

A critical role of GCC agencies is identifying and monitoring all known sources of VOCs.

- The Department of Agriculture and Consumer Protection (DATCP) keeps track of all underground storage tanks (USTs) with a capacity of 60 gallons or greater; this registry has identified over 180,000 USTs since 1991.

- Hazardous waste treatment, storage and disposal facilities must be licensed by the DNR and are subject to corrective action authorities in the event of spills or releases. The DNR's Bureau for Remediation and Redevelopment oversees investigation or remediation at 133 Resource Conservation and Recovery Act (RCRA) 2020 corrective action sites.

- More broadly, the Hazardous Substance Spill Law requires immediate notification to the DNR when any hazardous spills or discharges occur and requires that all necessary actions be pursued to restore the environment to the extent practicable. The spills program also develops outreach materials to help reduce the number and magnitude of spills and provide guidance for responding to spills. Outreach material topics addressed include spills from home fuel oil tanks, releases from illegal methamphetamine labs, and

mercury spills, all of which can lead to significant environmental impacts if not properly addressed.

Further Reading

- *DHS resources for contaminants in drinking water*
- *DNR overview of VOCs in private drinking water wells*
- *DNR map of open and closed contaminated sites*
- *DNR database of contaminated soil and groundwater*
- *DHS overview of vapor intrusion*
- *USGS report on VOCs in the nation's groundwater and drinking water wells*

References

1. Friedman, M.A. 1988. Volatile Organic Compounds in Groundwater and Leachate at Wisconsin Landfills. Wisconsin groundwater management practice monitoring project, DNR-004. Available at *https://search.library.wisc.edu/digital/AHLPFVKRI2TMVD87*
2. Battista, J.R. and J.P. Connelly. 1989. VOC Contamination at Selected Wisconsin Landfills - Sampling Results and Policy Implications. Wisconsin groundwater management practice monitoring project, DNR-005. Available at *https://search.library.wisc.edu/digital/APD2NNXATSJIRR8N*
3. Battista, J.R. and J.P. Connelly. 1994. VOCs at Wisconsin landfills: recent findings. In: Proceedings of the 17th International Madison Waste Conference, Madison, WI, pp. 67–86
4. U. S. Department of Human and Health Services. 2006. Private Well Impacts from Wisconsin's Old Landfills. Public Health Report. Available at *http://www.atsdr.cdc.gov/HAC/pha/Wisconsin's%20Old%20Landfill/WellImpacts-WisconsinOldLandfills021306.pdf*

another 6/14/16 Stanley transformer rupture. Pump never shut off. 50 gal. spill.

REMEDIATION AND REDEVELOPMENT DATABASE - BRRTS

The Wisconsin Department of Natural Resources (DNR) maintains a searchable database of information on activities related to property assessments and investigations, contamination, cleanup or redevelopment activities.

Records associated with the Activity may be available below. Records from the DNR's historical paper file are included in the Site File as they are digitized. All other activity records are in the Actions and Documents section. Records that are confidential, attorney-client privileged or sensitive in nature are not always included. Additional documents may be available through an open records request submitted to the program contact listed at the bottom of this webpage.

If additional Activities, documents or other details are present at this location, they may be accessed from the Location Details button below.

ACTIVITY DETAILS

O

Activity Type	Status	Jurisdiction	DNR Region	County
SPILL	CLOSED	DNR RR	NORTHERN	BARRON

Location Name		Address	Municipality
DAVID ALDERMAN EZ STOP	L	220 W KNAPP ST	RICE LAKE

PLSS Description	Latitude (WGS84)	Longitude (WGS84)		
			G	RR SITES MAP

Additional Location Description

SADDLE TANK LEAK FROM DELIVERY TRUCK ON TO CONCRETE

Acres	Facility ID	PECFA Number	EPA ID	Start Date	End Date
UNKNOWN	603083580	NONE		2019-02-13	2019-02-14

C

Above Ground Petrol Tank	Dry Cleaner	EPA NPL Site	PECFA Funds Eligible
No	No	No	No

PFAS	ROW Impact	Sediments	WI DOT Site	Underground Petrol Tank
No	No	No	No	No

A

File	Document Category	Date	Action Code	Name	Comment
		2019-02-14	11	Spill Activity Closed	
		2019-02-13	1	Spill Incident Occurred	
		2019-02-13	5	Notification of Hazardous Substance Spill	

S

SERTS Spill ID	Spill Occurred Date/Time	Spill Reported Date/Time
20190213N003-1	2019-02-13 1420	2019-02-13 1618

Description of Incident/General Comments

02-14-2019 MR: EMAIL FROM RP CONTACT. ALL INFORMATION IS COMPLETE AND CORRECT IN INITIAL REPORT.

Type of Location

GAS OR SERVICE STATION/GARAGE/AUTO DEALER/REPAIR SHOP

Spill Cause

OVERFILL (STORAGE TANK, VEHICLE OR EQUIPMENT)

Description of Cause or Additional Details

DELIVERY TRUCK HAD A SMALL LEAK IN THE SADDLE TANK.

Description of Environmental Impact

ALL SPILLED ON CONCRETE.

Description of Cleanup

STORE STAFF AND FIRE DEPARTMENT APPLIED ABSORBENT MATERIAL. ALL WAS CONTAINED AND CLEANED UP.

S

Action	Comment
Cleanup Method - Absorbent	

R

The hazardous substance(s) reported as present or previously present at an Activity. Other hazardous substances may be present that have not been identified. Substances can be added or removed through ongoing investigations.

Reported Substance(s)	Comments	Type	Amt Released	Units
Diesel Fuel		Petroleum	15	Gal

F

REMEDIATION AND REDEVELOPMENT DATABASE - BRRTS

The Wisconsin Department of Natural Resources (DNR) maintains a searchable database of information on activities related to property assessments and investigations, contamination, cleanup or redevelopment activities.

Records associated with the Activity may be available below. Records from the DNR's historical paper file are included in the Site File as they are digitized. All other activity records are in the Actions and Documents section. Records that are confidential, attorney-client privileged or sensitive in nature are not always included. Additional documents may be available through an open records request submitted to the program contact listed at the bottom of this webpage.

If additional Activities, documents or other details are present at this location, they may be accessed from the Location Details button below.

ACTIVITY DETAILS

0 [redacted] KWIK TRIP SPILL

Activity Type	Status	Jurisdiction	DNR Region	County
SPILL	CLOSED	DNR RR	WEST CNTRL	PORTAGE

Location Name		Address	Municipality
KWIK TRIP #336	L [redacted]	1900 POST RD	PLOVER

PLSS Description	Latitude (WGS84)	Longitude (WGS84)	
[redacted]			G [redacted] RR SITES MAP

Acres	Facility ID	PECFA Number	EPA ID	Start Date	End Date
UNKNOWN	750098030	NONE		2021-10-27	2021-10-29

C [redacted]

Above Ground Petrol Tank	Dry Cleaner	EPA NPL Site	PECFA Funds Eligible
No	No	No	No

PFAS	ROW Impact	Sediments	WI DOT Site	Underground Petrol Tank
No	No	No	No	No

A [redacted]

File	Document Category	Date	Action Code	Name	Comment
		2021-10-29	11	Spill Activity Closed	
		2021-10-28	5	Notification of Hazardous Substance Spill	
		2021-10-27	1	Spill Incident Occurred	

S [redacted]

SERTS Spill ID	Spill Occurred Date/Time	Spill Reported Date/Time
20211028WC50-1	2021-10-27 1600	2021-10-28 1100

Description of Incident/General Comments

1320HRS - CW LOCKMAN CONTACTS JS TO DISCUSS REPORT. LOCKMAN WILL CONTACT REPORTING PARTY AND DRIVE BY SITE. JS WILL REACH OUT TO STORE TO INQUIRE AND DISCUSS.

Type of Location

GAS OR SERVICE STATION/GARAGE/AUTO DEALER/REPAIR SHOP

Spill Cause

HUMAN ERROR

Description of Cause or Additional Details

GAS SPILLED FROM TANKER WHEN FILLING THE UNDERGROUND TANK YESTERDAY AROUND 4PM AT THE KWIK TRIP ON POST RD. IT WAS LEAKING THE WHOLE TIME WHEN FILLING. RP IS WORRIED THIS IS HAPPENING AT EVERY GAS STATION THIS TANKER FILLS AT

Description of Environmental Impact

ENVIRONMENTAL IMPACT UNKNOWN AT THIS TIME.

Description of Cleanup

CLEAN-UP PROGRESS UNKNOWN OR CLEAN-UP NOT STARTED.

R [redacted]

The hazardous substance(s) reported as present or previously present at an Activity. Other hazardous substances may be present that have not been identified. Substances can be added or removed through ongoing investigations.

Reported Substance(s)	Comments	Type	Amt Released	Units
Gasoline - Unleaded and Leaded		Petroleum		

F [redacted]

Program Specialist	HAYLEY SCHNAE (https://apps.dnr.wi.gov/staffdir/ContactSearchResultsExt.aspx?cno=61354&cSrc=EMPLOYEE)	hayley.schnae@wisconsin.gov (mailTo:hayley.schnae@wisconsin.gov?subject=BRRTS Activity 04-50-5[redacted]680)

BRRTS data comes from various sources, both internal and external to the DNR. There may be omissions and errors in the data and delays in updating new information.

REMEDIATION AND REDEVELOPMENT DATABASE - BRRTS

The Wisconsin Department of Natural Resources (DNR) maintains a searchable database of information on activities related to property assessments and investigations, contamination, cleanup or redevelopment activities.

Records associated with the Activity may be available below. Records from the DNR's historical paper file are included in the Site File as they are digitized. All other activity records are in the Actions and Documents section. Records that are confidential, attorney-client privileged or sensitive in nature are not always included. Additional documents may be available through an open records request submitted to the program contact listed at the bottom of this webpage.

If additional Activities, documents or other details are present at this location, they may be accessed from the Location Details button below.

ACTIVITY DETAILS

O

Activity Type	Status	Jurisdiction	DNR Region	County
SPILL	CLOSED	DNR RR	WEST CNTRL	PORTAGE

Location Name		Address	Municipality
KWIK TRIP # 183.	L	3258 CHURCH ST	STEVENS POINT

PLSS Description	Latitude (WGS84)	Longitude (WGS84)		
NW 1/4 of the SW 1/4 of Sec 04, T23N, R08E	44.5041153	-89.5671323	G	RR SITES MAP

Acres	Facility ID	PECFA Number	EPA ID	Start Date	End Date
UNKNOWN		NONE		2025-01-24	2025-02-10

C

Above Ground Petrol Tank	Dry Cleaner	EPA NPL Site	PECFA Funds Eligible
No	No	No	No

PFAS	ROW Impact	Sediments	WI DOT Site	Underground Petrol Tank
No	No	No	No	Yes

S

File	Description	File Name	Size (MB)
(/rrbotw/download-document?docSeqNo=310096&sender=activity)	SITE FILE DOCUMENTATION FOR ACTIVITY.	20250124WC50-1_SITE_FILE.PDF	5.3

A

File	Document Category	Date	Action Code	Name	Comment
		2025-02-10	11	Spill Activity Closed	
		2025-01-24	1	Spill Incident Occurred	
		2025-01-24	5	Notification of Hazardous Substance Spill	

S

SERTS Spill ID	Spill Occurred Date/Time	Spill Reported Date/Time
20250124WC50-1	2025-01-24 1500	2025-01-24 1511

Description of Incident/General Comments

1530HRS - JS SPOKE WITH KWIK TRIP REP BATZEL. REI WILL BE RESPONDING.

Type of Location

GAS OR SERVICE STATION/GARAGE/AUTO DEALER/REPAIR SHOP

Spill Cause

HUMAN ERROR

Description of Cause or Additional Details

CUSTOMER SPILLED 20 GALLONS WHILE FILLING UP CAR; CUSTOMER NAME CURRENTLY UNKNOWN

Description of Environmental Impact

5-10 GALLONS WENT INTO STORM DRAIN, THE REST IS ON THE PAVEMENT

Description of Cleanup

CLEAN-UP PROGRESS UNKNOWN OR CLEAN-UP NOT STARTED.
KWIK TRIP PERSONNEL USED ABSORBAENT TO STOP ROUGHLY HALF THE SPILL FROM REACHING THE STORM SEWER

S

Action	Comment
Storm Sewer Cleaned Out	
Cleanup Method - Absorbent	

R

The hazardous substance(s) reported as present or previously present at an Activity. Other hazardous substances may be present that have not been identified. Substances can be added or removed through ongoing investigations.

4/29/19 - Clear continue Obligatio

State of Wisconsin
DEPARTMENT OF NATURAL RESOURCES
473 Griffith Ave.
Wisconsin Rapids WI 54494

Scott Walker, Governor
Cathy Stepp, Secretary
Telephone 608-266-2621
Toll Free 1-888-936-7463
TTY Access via relay - 711

January 20, 2017

Kwik Trip

Attn: Troy Batzel

1626 Oak Street

La Crosse WI 54601

Subject: Reported Contamination at Kwik Trip Store #921
1610 State Road 13, Friendship, WI
DNR BRRTS Activity # 03-01-578658
DNR FID #701058050

Dear Mr. Batzel:

On January 4, 2017, Mark Gretebeck of Braun Intertec, on behalf of Kwik Trip, notified the Department of Natural Resources (DNR) that petroleum contamination had been detected at the site described above.

Based on the information that has been submitted to the DNR regarding this site, we believe you are responsible for investigating and restoring the environment at the above-described site under Section 292.11, Wisconsin Statutes, known as the hazardous substances spill law.

This letter describes the legal responsibilities of a person who is responsible under section 292.11, Wis. Stats., explains what you need to do to investigate and clean up the contamination, and provides you with information about cleanups, environmental consultants, possible financial assistance, and working cooperatively with the DNR or the Department of Agriculture, Trade and Consumer Protection (DATCP).

Legal Responsibilities:

Your legal responsibilities are defined both in statute and in administrative codes. The hazardous substances spill law, Section 292.11 (3) Wisconsin Statutes, states:

- RESPONSIBILITY. A person who possesses or controls a hazardous substance which is discharged or who causes the discharge of a hazardous substance shall take the actions necessary to restore the environment to the extent practicable and minimize the harmful effects from the discharge to the air, lands, or waters of the state.

Wisconsin Administrative Code chapters NR 700 through NR 754 establish requirements for emergency and interim actions, public information, site investigations, design and operation of remedial action systems, and case closure. Wisconsin Administrative Code chapter NR 140 establishes groundwater standards for contaminants that reach groundwater.

Steps to Take:

The longer contamination is left in the environment, the farther it can spread and the more it may cost to clean up. Quick action may lessen damage to your property and neighboring properties and reduce your costs in investigating and cleaning up the contamination. To ensure that your cleanup complies with Wisconsin's laws and administrative codes, you should hire a professional environmental consultant who understands what needs to be done. The following information provides the timeframes and required steps to take. Unless otherwise approved by DNR in writing you must complete the work by the timeframes specified.

1. Within the next **30 days,** by February 20, 2017, you should submit written verification (such as a letter from the consultant) that you have hired an environmental consultant. If you do not take action within this time frame, the DNR may initiate enforcement action against you.

2. Within **60 days,** by March 20, 2017, you must submit a work plan for completing the investigation. The work plan must comply with the requirements in the NR 700 Wis. Adm. Code rule series and should adhere to current DNR technical guidance documents.

3. You must initiate the site investigation within 90 days of submitting the site investigation work plan. You may proceed with the field investigation upon DNR notification to proceed. If the DNR has not responded within 30 days from submittal of the work plan, you are required to proceed with the field investigation. If a fee for DNR review has been submitted, the field investigation must begin within 60 days after receiving DNR approval.

4. Within 60 days after completion of the field investigation and receipt of the laboratory data, you must submit a Site Investigation Report to the DNR or other agency with administrative authority. For sites with agrichemicals contamination, your case will be transferred to the Department of Agriculture, Trade and Consumer Protection for oversight.

5. Within 60 days after submitting the Site Investigation Report, you must submit a remedial actions options report (RAOR). The RAOR shall include an evaluation of Green and Sustainable Remediation opportunities as required by s. NR 722.09 (2m), Wis. Adm. Code.

Sites where discharges to the environment have been reported are entered into the Bureau for Remediation and Redevelopment Tracking System ("BRRTS"), a version of which appears on the DNR's internet site. You may view the information related to your site at any time (http://dnr.wi.gov/botw/SetUpBasicSearchForm.do) and use the feedback system to alert us to any errors in the data.

If you want a formal written response from the department on a specific submittal, please be aware that a review fee is required in accordance with ch. NR 749, Wis. Adm. Code. If a fee is not submitted with your reports, you must complete the site investigation and cleanup to maintain your compliance with the spills law and chapters NR 700 through NR754. **The timeframes specified above are required by rule, so do not delay the investigation of your site.** We have provided detailed technical guidance to environmental consultants. Your consultant is expected to know our technical procedures and administrative rules and should be able to answer your questions on meeting cleanup requirements.

All correspondence regarding this site should be sent to:

Dee Lance
Remediation and Redevelopment Program
Wisconsin Department of Natural Resources
473 Griffith Avenue

Wisconsin Rapids WI 54494
Dee.Lance@wisconsin.gov

Unless otherwise directed, submit one paper copy and one electronic copy of plans and reports. To speed processing, correspondence should reference the BRRTS and FID numbers (if assigned) shown at the top of this letter.

Site Investigation and Vapor Pathway Analysis

As you develop the site investigation work plan, we want to remind you to include an assessment of the vapor intrusion pathway. Chapter NR 716, Wisconsin Administrative Code outlines the requirements for investigation of contamination in the environment. Specifically, s. NR 716.11(3) (a) requires that the field investigation determine the "nature, degree and extent, both areal and vertical, of the hazardous substances or environmental pollution in all affected media". In addition, section NR 716.11(5) (g) and (h) contains the specific requirements for evaluating the presence of vapors in the sub-surface as well as in indoor air.

You will need to include documentation with the Site Investigation Report that explains how the assessment was done. If the vapor pathway is being ruled out, then the report needs to provide the appropriate justification for reaching this conclusion. If the pathway cannot be ruled out, then investigation and, if appropriate, remedial action must be taken to address the risk presented prior to submitting the site for closure. The DNR has developed guidance to help responsible parties and their consultants comply with the requirements described above. The guidance includes a detailed explanation of how to assess the vapor intrusion pathway and provides criteria which identify when an investigation is necessary. The guidance is available at: http://dnr.wi.gov/files/PDF/pubs/rr/RR800.pdf.

Additional Information for Site Owners:
We encourage you to visit our website at http://dnr.wi.gov/topic/Brownfields/, where you can find information on selecting a consultant, financial assistance and understanding the cleanup process. You will also find information there about liability clarification letters, post-cleanup liability and more.

If you have questions, call me at 715-421-7862 for more information or visit the RR web site at the address above.

Thank you for your cooperation.

Sincerely,

Dee Lance

Dee Lance
Hydrogeologist
Remediation & Redevelopment Program

cc: Mark Gretebeck, Braun Intertec

State of Wisconsin
DEPARTMENT OF NATURAL RESOURCES
2501 Golf Course Rd.
Ashland WI 54806-3505

Tony Evers, Governor
Preston D. Cole, Secretary
Telephone 608-266-2621
Toll Free 1-888-936-7463
TTY Access via relay - 711

November 30, 2022

MR JASON POWELL
KWIK TRIP INC
PO BOX 2107
LACROSSE WI 54602

KT REAL ESTATE HOLDINGS
1626 OAK ST
LA CROSSE WI 54602

KEEP THIS LEGAL DOCUMENT WITH YOUR PROPERTY RECORDS

SUBJECT: Case Closure with Continuing Obligations
Kwik Trip 426, 220 West Knapp Street, Rice Lake, Wisconsin
DNR BRRTS #03-03-589890, FID #603083580

Dear Mr. Powell:

The Wisconsin Department of Natural Resources (DNR) is pleased to inform you that the Kwik Trip #426 case identified above met the requirements of Wisconsin Administrative (Wis. Admin.) Code chs. NR 700 to 799 for case closure with continuing obligations (COs). COs are legal requirements to address potential exposure to remaining contamination. No further investigation or remediation is required at this time for the reported hazardous substance discharge and/or environmental pollution.

However, you, future property owners and occupants of the property must comply with the COs as explained in this letter, which may include maintaining certain features and notifying the DNR and obtaining approval before taking specific actions. You must provide this letter and all enclosures to anyone who purchases, rents or leases this property from you.

This case closure decision is issued under Wis. Admin. Code chs. NR 700 to 799 and is based on information received by the DNR to date. The DNR reviewed the closure request for compliance with state laws and standards and determined the case closure request met the notification requirements of Wis. Admin. Code ch. NR 725, the response action goals of Wis. Admin. Code § NR 726.05(4), and the case closure criteria of Wis. Admin. Code §§ NR 726.05, 726.09 and 726.11, and Wis. Admin. Code ch. NR 140.

The Kwik Trip #426 site was investigated for a discharge of hazardous substances and/or environmental pollution detected during a Tank System Site Assessment (TSSA) conducted on July 7, 2022. The hazardous substance discharge was from a petroleum storage tank system's dispensers and associated piping located beneath and immediately adjacent to the existing canopy. Case closure is granted for the petroleum contaminants as documented in the case file. The original TSSA and additional *TSSA with No Further Action Request* (July 29, 2022) addressed soil and groundwater contamination at the site.

PRINTED ON RECYCLED PAPER

State of Wisconsin
Department of Natural Resources
PO Box 7921, Madison WI 53707-7921
dnr.wi.gov

Notification For Hazardous Substance Discharge (Non-Emergency Only)

Form 4400-225 (R 06/17)

Emergency Discharges / Spills should be reported via the 24-Hour Hotline: 1-800-943-0003

Notice: Hazardous substance discharges must be reported immediately according to s. 292.11 Wis. Stats. Non-emergency hazardous substance discharges may be reported by telefaxing or e-mailing a completed report to the Department, or calling or visiting a Department office in person. If you choose to notify the Department by telefax or by email, you should use this form to be sure that all necessary information is included. However, use of this form is not mandatory. Under s. 292.99, Wis. Stats., the penalty for violating the reporting requirements of ch. 292 Wis. Stats., shall be no less than $10 nor more than $5000 for each violation. Each day of continued violation is a separate offense. It is not the Department's intention to use any personally identifiable information from this form for any purpose other than program administration. However, information submitted on this form may also be made available to requesters under Wisconsin's Open Records Law (ss. 19.31 – 19.39, Wis. Stats.).

Confirmatory laboratory data should be included with this form, to assist the DNR in processing this Hazardous Substance Release Notification.

Complete this form. **TYPE or PRINT LEGIBLY.** NOTIFY appropriate DNR region (see next page) **IMMEDIATELY** upon discovery of a potential release from **(check one)**:

- (●) Underground Petroleum Storage Tank System (additional information may be required for Item 6 below)
- ( ) Aboveground Petroleum Storage Tank System
- ( ) Dry Cleaner Facility
- ( ) Other - Describe: _____

ATTN DNR: **R & R Program Associate** Date DNR Notified: 11/26/2018

1. Discharge Reported By

Name	Firm	Phone Number (include area code)
Andrew Delforge	REI Engineering, Inc.	(715) 675-9784

Mailing Address	Email
4080 North 20th Avenue, Wausau, WI 54401	adelforge@reiengineering.com

2. Site Information

Name of site at which discharge occurred. Include local name of site/business, not responsible party name, unless a residence/vacant property.

Kwik Trip #1060

Location: Include street address, not PO Box. If no street address, describe as precisely as possible, i.e., 1/4 mile NW of CTHs 60 & 123 on E side of CTH 60.

1610-1620 Lawrence Drive

Municipality: (City, Village, Township) Specify municipality in which the site is located, not mailing address/city.

City of DePere

County	Legal Description:	WTM:
Brown	SW ¼ of SW ¼ Section 29 , Town 23 N, Range 20 (●) E ( ) W	X 670596 Y 441554

3. Responsible Party (RP) and/or RP Representative

Responsible Party Name: Business or owner name that is responsible for cleanup. If more than one, list all. Attach additional pages as necessary.

Kwik Trip, Inc.

☐ A local governmental unit claiming an exemption from state Spill Law and Solid Waste Management responsibilities for the discharge being reported, per Wis. Stat. §§ 292.11(9)(e) and 292.23, should: 1) check this box; 2) review DNR publication RR-055; and 3) provide documentation to DNR that demonstrates compliance with the statutory requirements of the liability exemptions. Local governmental units may also request a fee-based liability clarification letter from DNR by using DNR Form 4400-237.

Contact Person Name (if different)	Phone Number	Email
Troy Batzel	(608) 793-6283	tbatzel@kwiktrip.com

Mailing Address	City	State	ZIP Code
1626 Oak Street	LaCrosse	WI	54603

Responsible Party Name: Business or owner name that is responsible for cleanup. If more than one, list all. Attach additional pages as necessary.

Contact Person Name (if different)	Phone Number	Email

Mailing Address	City	State	ZIP Code

(continued)

Notification For Hazardous Substance Discharge (Non-Emergency Only)

4. Hazardous Substance Information

Identify hazardous substance discharged (check all that apply):

- ☐ VOCs
  - ☐ PCE
  - ☐ TCE
  - ☐ Other Chlorinated
  - ☐ Diesel
  - ☐ Fuel Oil
  - ☒ Gasoline
  - ☐ Hydraulic Oil
  - ☐ Jet Fuel
- *(VOCs continued)*
  - ☐ Mineral Oil
  - ☐ Waste Oil
  - ☐ Petroleum-Unknown Type
- ☐ PAHs
- ☐ PCBs
- ☐ Cyanide
- ☐ Leachate
- ☐ Manure
- ☐ Metals
  - ☐ Arsenic
  - ☐ Chromium
  - ☐ Lead
  - ☐ Other: _____
- ☐ Pesticides: _____
- ☐ Fertilizer: _____
- ☐ RCRA Hazardous Waste: _____
- ☐ Other: _____
- ☐ Unknown

5. Impacts to the Environment Information

Enter "K" for known/confirmed or "P" for potential for all that apply.

- ___ Air Contamination
- ___ Co-mingled (Petroleum & Non-Petroleum)
- ___ Contamination in Fractured Bedrock
- ___ Contamination Within 1 Meter of Bedrock
- ___ Contaminated Private Well
- ___ Contaminated Public Well
- ___ Contamination in Right of Way
- ___ Fire Explosion Threat
- ___ Free Product
- K Groundwater Contamination
- ___ Off-Site Contamination
- ___ Sanitary Sewer Contamination
- ___ Storm Sewer Contamination
- ___ Sediment Contamination
- P Soil Contamination
- ___ Soil Gas Contamination
- ___ Sub-slab Vapor Contamination
- ___ Surface Water Contamination
- ___ Within 100 ft of Private Well
- ___ Within 1000 ft of Public Well

Other (specify): _____

Contamination was discovered as a result of:

☐ Tank closure assessment Date _____ ☒ Site assessment Date 07/17/2018 ☐ Other - Describe: _____ Date _____

Lab results: ☐ Lab results will be faxed upon receipt ☒ Lab results are attached

Additional Comments: Include a brief description of immediate actions taken to halt the release and contain or cleanup hazardous substances that have been discharged.
Contamination appears to be limited to water in UST basin backfill

6. Federal Energy Act Requirements (Section 9002(d) of the Solid Waste Disposal Act (SWDA))

For all confirmed releases from USTs occurring after 9/30/2007 please provide the following information:

☐ Does not apply.

Source

- ☐ Tank
- ☐ Piping
- ☐ Dispenser
- ☐ Submersible Turbine Pump
- ☐ Delivery Problem
- ☒ Other (specify): Unknown

Cause

- ☐ Spill
- ☐ Overfill
- ☐ Corrosion
- ☐ Physical or Mechanical Damage
- ☐ Installation Problem
- ☐ Other (does not fit any of above)
- ☒ Unknown

Contact information to report non-emergency releases in DNR's five regions are as follows:

Northeast Region (FAX: 920-662-5413); Attention -- R&R Program Associate: DNRRRNER@wisconsin.gov
Brown, Calumet, Door, Fond du Lac **(except City of Waupun - see South Central Region)**, Green Lake, Kewaunee, Manitowoc, Marinette, Marquette, Menominee, Oconto, Outagamie, Shawano, Sheboygan, Waupaca, Waushara, Winnebago counties

Northern Region (FAX: 715-623-6773); Attention -- R&R Program Associate: DNRRRNOR@wisconsin.gov
Ashland, Barron, Bayfield, Burnett, Douglas, Forest, Florence, Iron, Langlade, Lincoln, Oneida, Polk, Price, Rusk, Sawyer, Taylor, Vilas, Washburn counties

South Central Region (FAX: 608-273-5610); Attention -- R&R Program Associate: DNRRRSCR@wisconsin.gov
Columbia, Dane, Dodge, Fond du Lac **(City of Waupun only)**, Grant, Green, Iowa, Jefferson, Lafayette, Richland, Rock, Sauk, Walworth counties

Southeast Region (FAX: 414-263-8550); Attention -- R&R Program Associate: DNRRRSER@wisconsin.gov
Kenosha, Milwaukee, Ozaukee, Racine, Washington, Waukesha counties

REPORT OF THE COUNCIL ON SCIENCE AND PUBLIC HEALTH

CSAPH Report 2-A-16

Subject: Human and Environmental Effects of Light Emitting Diode (LED) Community Lighting

Presented by: Louis J. Kraus, MD, Chair

Referred to: Reference Committee E
(Theodore Zanker, MD, Chair)

INTRODUCTION

With the advent of highly efficient and bright light emitting diode (LED) lighting, strong economic arguments exist to overhaul the street lighting of U.S. roadways.[1-3] Valid and compelling reasons driving the conversion from conventional lighting include the inherent energy efficiency and longer lamp life of LED lighting, leading to savings in energy use and reduced operating costs, including taxes and maintenance, as well as lower air pollution burden from reduced reliance on fossil-based carbon fuels.

Not all LED light is optimal, however, when used as street lighting. Improper design of the lighting fixture can result in glare, creating a road hazard condition.[4,5] LED lighting also is available in various color correlated temperatures. Many early designs of white LED lighting generated a color spectrum with excessive blue wavelength. This feature further contributes to disability glare, i.e., visual impairment due to stray light, as blue wavelengths are associated with more scattering in the human eye, and sufficiently intense blue spectrum damages retinas.[6,7] The excessive blue spectrum also is environmentally disruptive for many nocturnal species. Accordingly, significant human and environmental concerns are associated with short wavelength (blue) LED emission. Currently, approximately 10% of existing U.S. street lighting has been converted to solid state LED technology, with efforts underway to accelerate this conversion. The Council is undertaking this report to assist in advising communities on selecting among LED lighting options in order to minimize potentially harmful human health and environmental effects.

METHODS

English language reports published between 2005 and 2016 were selected from a search of the PubMed and Google Scholar databases using the MeSH terms "light," "lighting methods," "color," "photic stimulation," and "adverse effects," in combination with "circadian rhythm/physiology/radiation effects," "radiation dosage/effects," "sleep/physiology," "ecosystem," "environment," and "environmental monitoring." Additional searches using the text terms "LED" and "community," "street," and "roadway lighting" were conducted. Additional information and perspective were supplied by recognized experts in the field.

ADVANTGAGES AND DISADVANAGES OF LED STREET LIGHTS

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Action of the AMA House of Delegates 2016 Annual Meeting: Council on Science and Public Health Report 2 Recommendations Adopted and Remainder of Report Filed.

The main reason for converting to LED street lighting is energy efficiency; LED lighting can reduce energy consumption by up to 50% compared with conventional high pressure sodium (HPS) lighting. LED lighting has no warm up requirement with a rapid "turn on and off" at full intensity. In the event of a power outage, LED lights can turn on instantly when power is restored, as opposed to sodium-based lighting requiring prolonged warm up periods. LED lighting also has the inherent capability to be dimmed or tuned, so that during off peak usage times (e.g., 1 to 5 AM), further energy savings can be achieved by reducing illumination levels. LED lighting also has a much longer lifetime (15 to 20 years, or 50,000 hours), reducing maintenance costs by decreasing the frequency of fixture or bulb replacement. That lifespan exceeds that of conventional HPS lighting by 2-4 times. Also, LED lighting has no mercury or lead, and does not release any toxic substances if damaged, unlike mercury or HPS lighting. The light output is very consistent across cold or warm temperature gradients. LED lights also do not require any internal reflectors or glass covers, allowing higher efficiency as well, if designed properly.[8,9]

Despite the benefits of LED lighting, some potential disadvantages are apparent. The initial cost is higher than conventional lighting; several years of energy savings may be required to recoup that initial expense.[10] The spectral characteristics of LED lighting also can be problematic. LED lighting is inherently narrow bandwidth, with "white" being obtained by adding phosphor coating layers to a high energy (such as blue) LED. These phosphor layers can wear with time leading to a higher spectral response than was designed or intended. Manufacturers address this problem with more resistant coatings, blocking filters, or use of lower color temperature LEDs. With proper design, higher spectral responses can be minimized. LED lighting does not tend to abruptly "burn out," rather it dims slowly over many years. An LED fixture generally needs to be replaced after it has dimmed by 30% from initial specifications, usually after about 15 to 20 years.[1,11]

Depending on the design, a large amount blue light is emitted from some LEDs that appear white to the naked eye. The excess blue and green emissions from some LEDs lead to increased light pollution, as these wavelengths scatter more within the eye and have detrimental environmental and glare effects. LED's light emissions are characterized by their correlated color temperature (CCT) index.[12,13] The first generation of LED outdoor lighting and units that are still widely being installed are "4000K" LED units. This nomenclature (Kelvin scale) reflects the equivalent color of a heated metal object to that temperature. The LEDs are cool to the touch and the nomenclature has nothing to do with the operating temperature of the LED itself. By comparison, the CCT associated with daylight light levels is equivalent to 6500K, and high pressure sodium lighting (the current standard) has a CCT of 2100K. Twenty-nine percent of the spectrum of 4000K LED lighting is emitted as blue light, which the human eye perceives as a harsh white color. Due to the point-source nature of LED lighting, studies have shown that this intense blue point source leads to discomfort and disability glare.[14]

More recently engineered LED lighting is now available at 3000K or lower. At 3000K, the human eye still perceives the light as "white," but it is slightly warmer in tone, and has about 21% of its emission in the blue-appearing part of the spectrum. This emission is still very blue for the nighttime environment, but is a significant improvement over the 4000K lighting because it reduces discomfort and disability glare. Because of different coatings, the energy efficiency of 3000K lighting is only 3% less than 4000K, but the light is more pleasing to humans and has less of an impact on wildlife.

Glare

Disability glare is defined by the Department of Transportation (DOT) as the following:

"Disability glare occurs when the introduction of stray light into the eye reduces the ability to resolve spatial detail. It is an objective impairment in visual performance."

Classic models of this type of glare attribute the deleterious effects to intraocular light scatter in the eye. Scattering produces a veiling luminance over the retina, which effectively reduces the contrast of stimulus images formed on the retina. The disabling effect of the veiling luminance has serious implications for nighttime driving visibility.[15]

Although LED lighting is cost efficient and inherently directional, it paradoxically can lead to worse glare than conventional lighting. This glare can be greatly minimized by proper lighting design and engineering. Glare can be magnified by improper color temperature of the LED, such as blue-rich LED lighting. LEDs are very intense point sources that cause vision discomfort when viewed by the human eye, especially by older drivers. This effect is magnified by higher color temperature LEDs, because blue light scatters more within the human eye, leading to increased disability glare.[16]

In addition to disability glare and its impact on drivers, many residents are unhappy with bright LED lights. In many localities where 4000K and higher lighting has been installed, community complaints of glare and a "prison atmosphere" by the high intensity blue-rich lighting are common. Residents in Seattle, WA have demanded shielding, complaining they need heavy drapes to be comfortable in their own homes at night.[17] Residents in Davis, CA demanded and succeeded in getting a complete replacement of the originally installed 4000K LED lights with the 3000K version throughout the town at great expense.[18] In Cambridge, MA, 4000K lighting with dimming controls was installed to mitigate the harsh blue-rich lighting late at night. Even in places with a high level of ambient nighttime lighting, such as Queens in New York City, many complaints were made about the harshness and glare from 4000K lighting.[19] In contrast, 3000K lighting has been much better received by citizens in general.

Unshielded LED Lighting

Unshielded LED lighting causes significant discomfort from glare. A French government report published in 2013 stated that due to the point source nature of LED lighting, the luminance level of unshielded LED lighting is sufficiently high to cause visual discomfort regardless of the position, as long as it is in the field of vision. As the emission surfaces of LEDs are highly concentrated point sources, the luminance of each individual source easily exceeds the level of visual discomfort, in some cases by a factor of 1000.[17]

Discomfort and disability glare can decrease visual acuity, decreasing safety and creating a road hazard. Various testing measures have been devised to determine and quantify the level of glare and vision impairment by poorly designed LED lighting.[20] Lighting installations are typically tested by measuring foot-candles per square meter on the ground. This is useful for determining the efficiency and evenness of lighting installations. This method, however, does not take into account the human biological response to the point source. It is well known that unshielded light sources cause pupillary constriction, leading to worse nighttime vision between lighting fixtures and causing a "veil of illuminance" beyond the lighting fixture. This leads to worse vision than if the light never existed at all, defeating the purpose of the lighting fixture. Ideally LED lighting installations should be tested in real life scenarios with effects on visual acuity evaluated in order to ascertain the best designs for public safety.

Proper Shielding

With any LED lighting, proper attention should be paid to the design and engineering features. LED lighting is inherently a bright point source and can cause eye fatigue and disability glare if it is allowed to directly shine into human eyes from roadway lighting. This is mitigated by proper design, shielding and installation ensuring that no light shines above 80 degrees from the horizontal. Proper shielding also should be used to prevent light trespass into homes alongside the road, a common cause of citizen complaints. Unlike current HPS street lighting, LEDs have the ability to be controlled electronically and dimmed from a central location. Providing this additional control increases the installation cost, but may be worthwhile because it increases long term energy savings and minimizes detrimental human and environmental lighting effects. In environmentally sensitive or rural areas where wildlife can be especially affected (e.g., near national parks or bio-rich zones where nocturnal animals need such protection), strong consideration should be made for lower emission LEDs (e.g., 3000K or lower lighting with effective shielding). Strong consideration also should be given to the use of filters to block blue wavelengths (as used in Hawaii), or to the use of inherent amber LEDs, such as those deployed in Quebec. Blue light scatters more widely (the reason the daytime sky is "blue"), and unshielded blue-rich lighting that travels along the horizontal plane increases glare and dramatically increases the nighttime sky glow caused by excessive light pollution.

POTENTIAL HEALTH EFFECTS OF "WHITE" LED STREET LIGHTING

Much has been learned over the past decade about the potential adverse health effects of electric light exposure, particularly at night.[21-25] The core concern is disruption of circadian rhythmicity. With waning ambient light, and in the absence of electric lighting, humans begin the transition to nighttime physiology at about dusk; melatonin blood concentrations rise, body temperature drops, sleepiness grows, and hunger abates, along with several other responses.

A number of controlled laboratory studies have shown delays in the normal transition to nighttime physiology from evening exposure to tablet computer screens, backlit e-readers, and room light typical of residential settings.[26-28] These effects are wavelength and intensity dependent, implicating bright, short wavelength (blue) electric light sources as disrupting transition. These effects are not seen with dimmer, longer wavelength light (as from wood fires or low wattage incandescent bulbs). In human studies, a short-term detriment in sleep quality has been observed after exposure to short wavelength light before bedtime. Although data are still emerging, some evidence supports a long-term increase in the risk for cancer, diabetes, cardiovascular disease and obesity from chronic sleep disruption or shiftwork and associated with exposure to brighter light sources in the evening or night.[25,29]

Electric lights differ in terms of their circadian impact.[30] Understanding the neuroscience of circadian light perception can help optimize the design of electric lighting to minimize circadian disruption and improve visual effectiveness. White LED streetlights are currently being marketed to cities and towns throughout the country in the name of energy efficiency and long term cost savings, but such lights have a spectrum containing a strong spike at the wavelength that most effectively suppresses melatonin during the night. It is estimated that a "white" LED lamp is at least 5 times more powerful in influencing circadian physiology than a high pressure sodium light based on melatonin suppression.[31] Recent large surveys found that brighter residential nighttime lighting is associated with reduced sleep time, dissatisfaction with sleep quality, nighttime awakenings, excessive sleepiness, impaired daytime functioning, and obesity.[29,32] Thus, white LED street lighting patterns also could contribute to the risk of chronic disease in the populations of cities in which they have been installed. Measurements at street level from white LED street lamps are needed to more accurately assess the potential circadian impact of evening/nighttime exposure to these lights.

ENVIRONMENTAL EFFECTS OF LED LIGHTING

The detrimental effects of inefficient lighting are not limited to humans; 60% of animals are nocturnal and are potentially adversely affected by exposure to nighttime electrical lighting. Many birds navigate by the moon and star reflections at night; excessive nighttime lighting can lead to reflections on glass high rise towers and other objects, leading to confusion, collisions and death.[33] Many insects need a dark environment to procreate, the most obvious example being lightning bugs that cannot "see" each other when light pollution is pronounced. Other environmentally beneficial insects are attracted to blue-rich lighting, circling under them until they are exhausted and die.[34,35] Unshielded lighting on beach areas has led to a massive drop in turtle populations as hatchlings are disoriented by electrical light and sky glow, preventing them from reaching the water safely.[35-37] Excessive outdoor lighting diverts the hatchlings inland to their demise. Even bridge lighting that is "too blue" has been shown to inhibit upstream migration of certain fish species such as salmon returning to spawn. One such overly lit bridge in Washington State now is shut off during salmon spawning season.

Recognizing the detrimental effects of light pollution on nocturnal species, U.S. national parks have adopted best lighting practices and now require minimal and shielded lighting. Light pollution along the borders of national parks leads to detrimental effects on the local bio-environment. For example, the glow of Miami, FL extends throughout the Everglades National Park. Proper shielding and proper color temperature of the lighting installations can greatly minimize these types of harmful effects on our environment.

CONCLUSION

Current AMA Policy supports efforts to reduce light pollution. Specific to street lighting, Policy H-135.932 supports the implementation of technologies to reduce glare from roadway lighting. Thus, the Council recommends that communities considering conversion to energy efficient LED street lighting use lower CCT lights that will minimize potential health and environmental effects. The Council previously reviewed the adverse health effects of nighttime lighting, and concluded that pervasive use of nighttime lighting disrupts various biological processes, creating potentially harmful health effects related to disability glare and sleep disturbance.[25]

RECOMMENDATIONS

The Council on Science and Public Health recommends that the following statements be adopted, and the remainder of the report filed.

1. That our American Medical Association (AMA) support the proper conversion to community-based Light Emitting Diode (LED) lighting, which reduces energy consumption and decreases the use of fossil fuels. (New HOD Policy)

2. That our AMA encourage minimizing and controlling blue-rich environmental lighting by using the lowest emission of blue light possible to reduce glare. (New HOD Policy)

3. That our AMA encourage the use of 3000K or lower lighting for outdoor installations such as roadways. All LED lighting should be properly shielded to minimize glare and detrimental human and environmental effects, and consideration should be given to utilize the ability of LED lighting to be dimmed for off-peak time periods. (New HOD Policy)

Fiscal Note: Less than $500

REFERENCES

1. Municipal Solid State Street Lighting Consortium. http://www1.eere.energy.gov/buildings/ssl/consortium.html. Accessed April 4, 2016.

2. Illuminating Engineering Society RP-8 – Guide to Roadway Lighting. http://www.ies.org/? 2014. Accessed April 4, 2016.

3. LED Lighting Facts–A Program of the United States Department of Energy. http://www.lightingfacts.com. Accessed April 5, 2016.

4. Lin Y, Liu Y, Sun Y, Zhu X, Lai J, Heynderickz I. Model predicting discomfort glare caused by LED road lights. *Opt Express*. 2014;22(15):18056-71.

5. Gibbons RB, Edwards CJ. A review of disability and discomfort glare research and future direction. 18th Biennial TRB Visibility Symposium, College Station TX, United States, April 17-19, 2007.

6. Shang YM, Wang GS, Sliney D, Yang CH, Lee LL. White light–emitting diodes (LEDs) at domestic lighting levels and retinal injury in a rat model. *Environ Health Perspect.* 2014:122(3):269-76.

7. Lougheed T. Hidden blue hazard? LED lighting and retinal damage in rats, *Environ Health Perspect*. 2014;122(3):A81.

8. A Municipal Guide for Converting to LED Street Lighting, (http://www1.eere.energy.gov/buildings/ssl/consortium.html) 10/13/2013.

9. In depth: Advantages of LED Lighting. http://energy.ltgovernors.com/in-depth-advantages-of-led-lighting.html. Accessed April 5, 2016.

10. Silverman H. How LED Streetlights Work. HowStuffWorks.com. June 22, 2009. http://science.howstuffworks.com/environmental/green-tech/sustainable/led-streetlight.htm. Accessed April 7, 2016.

11. Jin H, Jin S, Chen L, Cen S, Yuan K. Research on the lighting performance of LED street lights with different color temperatures. *IEEE Photonics Journal*. 2015;24(6):975-78. http://ieeexplore.ieee.org/stamp/stamp.jsp?arnumber=7328247. Accessed April 7, 2016.

12. Morris N. LED there be light. Nick Morris predicts a bright future for LEDs. *Electrooptics.com*. http://www.electrooptics.com/features/junjul06/junjul06leds.html. Accessed April 7, 2016.

13. Mills MP. The LED illumination revolution. *Forbes Magazine*. February 27, 2008. http://www.forbes.com/2008/02/27/incandescent-led-cfl-pf-guru_in_mm_0227energy_inl.html. Accessed April 5, 2016.

14. Opinion of the French Agency for Food, Environmental and Occupational Health & Safety, October 19, 2010. https://web.archive.org/web/20140429161553/http:/www.anses.fr/Documents/AP2008sa0408EN.pdf

15. U.S. Department of Transportation, Federal Highway Administration, 2005.

16. Sweater-Hickcox K, Narendran N, Bullough JD, Freyssinier JP. Effect of different coloured luminous surrounds on LED discomfort glare perception. *Lighting Research Technology*. 2013;45(4):464-75. http://lrt.sagepub.com/content/45/4/464. Accessed April 5, 2016.

17. Scigliano E. Seattle's new LED-lit streets Blinded by the lights. *Crosscut*. March 18, 2013. http://crosscut.com/2013/03/streetlights-seattle-led/. Accessed April 6, 2016.

18. Davis will spend $350,000 to replace LED lights after neighbor complaints. CBS Local, Sacramento;October 21, 2014. http://sacramento.suntimes.com/sac-news/7/138/6000/davis-will-spend-350000-to-replace-led-lights-after-neighbor-complaints.

19. Chaban M. LED streetlights in Brooklyn are saving energy but exhausting residents. *NY Times*; March 23, 2015. http://www.nytimes.com/2015/03/24/nyregion/new-led-streetlights-shine-too-brightly-for-some-in-brooklyn.html?_r=0. Accessed April 5, 2016.

20. Vos JJ. On the cause of disability glare and its dependence on glare angle, age and ocular pigmentation. *Clin Exp Optom*. 2003;86(6):363-70.

21. Stevens RG, Brainard GC, Blask DE, Lockley, SW, Motta, ME. Breast cancer and circadian disruption from electric lighting in the modern world. *CA Cancer J Clin*. 2014;64:207-18.

22. Evans JA, Davidson AJ. Health consequences of circadian disruption in humans and animal models. *Prog Mol Biol Transl Sci*. 2013;119:283-323.

23. Wright KP Jr, McHill AW, Birks BR, Griffin BR, Rusterholz T, Chinoy ED. Entrainment of the human circadian clock to the natural light-dark cycle. *Curr Biol*. 2013;23:1554-8.

24. Energy Savings Estimates of Light Emitting Diodes in Niche Lighting Applications. Building Technologies Program, Office of Energy Efficiency and Renewable Energy, U.S. Department of Energy. January 2011. http://apps1.eere.energy.gov/buildings/publications/pdfs/ssl/nichefinalreport_january2011.pdf. Accessed April 7, 2016.

25. Council on Science and Public Health Report 4. Light pollution. Adverse effects of nighttime lighting. American Medical Association, Annual Meeting, Chicago, IL. 2012.

26. Cajochen C, Frey S, Anders D, et al. Evening exposure to a light-emitting diodes (LED)-backlit computer screen affects circadian physiology and cognitive performance. *J Appl Physiol*. 2011;110:1432-8.

27. Chang AM, Aeschbach D, Duffy JF, Czeisler CA. Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. *Proc Natl Acad Sci USA*. 2015;112:1232-7.

28. Gooley JJ, Chamberlain K, Smith KA, et al. Exposure to room light before bedtime suppresses melatonin onset and shortens melatonin duration in humans. *J Clin Endocrinol Metab.* 2011;96:E463-72.

29. Koo YS, Song JY, Joo EY, et al. Outdoor artificial light at night, obesity, and sleep health: Cross-sectional analysis in the KoGES study. *Chronobiol Int.* 2016;33(3):301-14.

30. Lucas RJ, Peirson SN, Berson DM, et al. Measuring and using light in the melanopsin age. *Trends Neurosci.* 2014;37:1-9.

31. Falchi F, Cinzano P, Elvidge CD, Keith DM, Haim A. Limiting the impact of light pollution on human health, environment and stellar visibility. *J Environ Manage.* 2011;92:2714-22.

32. Ohayon M, Milesi C. Sleep deprivation/insomnia and exposure to street lights in the American general population. American Academy of Neurology Annual Meeting. April 15-21, 2016. Vancouver, BC.

33. Pawson SM, Bader MK. Led lighting increases the ecological impact of light pollution irrespective of color temperature. *Ecological Applications.* 2014;24:1561-68.

34. Gaston K, Davies T, Bennie J, Hopkins J. Reducing the ecological consequences of night-time light pollution: Options and developments. *J Appl Ecol.* 2012;49(6):1256–66.

35. Salmon M. Protecting sea turtles from artificial night lighting at Florida's oceanic beaches. In-Rich C, Longcore T (eds.). *Ecological Consequences of Artificial Night Lighting.* 2006:141-68. Island Press, Washington, DC.

36. Rusenko KW, Mann JL, Albury R, Moriarty JE, Carter HL. Is the wavelength of city glow getting shorter? Parks with no beachfront lights record adult aversion and hatchling disorientations in 2004. Kalb H, Rohde A, Gayheart K, Shanker, K, compilers. 2008. *Proceedings of the Twenty-fifth Annual Symposium on Sea Turtle Biology and Conservation,* NOAA Technical Memorandum NMFS-SEFSC-582, 204pp. http://www.nmfs.noaa.gov/pr/pdfs/species/turtlesymposium2005.pdf

37. Rusenko KW, Newman R, Mott C, et al. Using GIS to determine the effect of sky glow on nesting sea turtles over a ten year period. Jones TT, Wallace BP, compilers. 2012. *Proceedings of the Thirty-first Annual Symposium on Sea Turtle Biology and Conservation.* NOAA Technical Memorandum NOAA NMFS-SEFSC-631:32p.

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Stormwater Best Management Practice

Vegetated Buffers

Minimum Measure: Construction Site Stormwater Runoff Control
Subcategory: Sediment Control

Description

Vegetated buffers are areas of natural, existing or established vegetation that protect the water quality of neighboring areas and waterbodies during construction. Buffer zones provide an area where stormwater can permeate the soil and replenish the groundwater (WES, 2008). They also slow the flow of stormwater, which helps to filter sediment, decrease soil erosion and prevent streambank collapse.

Applicability

Vegetated buffers are applicable in most areas able to support vegetation. They are most effective and beneficial on floodplains, near wetlands, along streambanks and on unstable slopes. Local requirements or a construction general permit may require natural vegetated buffers based on a site's proximity to waterbodies or if a site discharges to a sensitive water, such as impaired waters, exceptional waters or wetlands.

Siting and Design Considerations

When siting vegetated buffers, design engineers should first identify existing and proposed natural buffer zones on a site map (MDE et al., 2011). Prior to construction, construction staff should mark clearing limits to keep all construction activities out of natural buffer zones and limit damage to vegetation (Washington Department of Ecology, 2019).

It is important to not overwhelm vegetated buffers with fast, erosive, and/or concentrated flows. If upstream flowpaths generate concentrated flows, design engineers should incorporate other practices such as sediment traps or check dams to moderate discharges onto buffers. Design engineers can also use level spreaders upstream of buffers to reestablish sheet flow conditions.

Additional siting and design considerations include:

Using a vegetated buffer along the perimeter of a construction site can deter sediment from moving off site.
Photo Credit: Steven Chase for USEPA

- Preserving natural, existing or established vegetation in clumps, block or strips.
- Preserving natural, existing or established vegetation on unstable, steep slopes.
- Making sure slopes are shallow enough to allow establishment of vegetation.
- Making sure soils are not compacted.
- Where possible, intermixing layers of vegetation (native vegetation in particular), including grasses, deciduous and evergreen shrubs, and understory and overstory trees.

Limitations

Adequate land should be available for a vegetated buffer. If land costs are high, a buffer zone may not be the most cost-effective practice. Vegetated buffers work well with sheet flow, but they are not appropriate for mitigating concentrated stormwater flows. In addition, construction staff should maintain adequate vegetative cover to keep buffers effective.

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Maintenance Considerations

Keeping buffer vegetation healthy requires routine maintenance. Maintenance needs depend on vegetation species, soil type and climatic conditions. Maintenance can include weed and pest control, mowing, fertilizing, liming, irrigating, and pruning. Inspection and maintenance are most important during buffer installation. Following establishment, vegetated buffers only require routine maintenance and periodic inspections. Construction staff should inspect them after heavy rainfall and at least once a year. Inspections should focus on encroachment, erosion, vegetation density, evidence of concentrated flows, and any damage from foot or vehicular traffic.

Effectiveness

The effectiveness of vegetated buffers depends on buffer width, buffer slope, vegetation type, soil conditions and geographic location. For 50-foot natural buffers in combination with perimeter controls, the EPA's Construction General Permit Appendix G reports sediment removal efficiencies ranging from approximately 25 to 90 percent.

Cost Considerations

A vegetated buffer can be a low-cost practice when there is adequate area for preserving natural, existing or established vegetation. Establishing a vegetated buffer includes the cost of clearing, plants or seeding, and maintenance.

Additional Information

Additional information on related practices and the Phase II MS4 program can be found at EPA's National Menu of Best Management Practices (BMPs) for Stormwater website

References

Maryland Department of the Environment (MDE), Natural Resources Conservation Service, & Maryland Association of Soil Conservation Districts. (2011). *2011 Maryland standards and specifications for soil erosion and sediment control.* Maryland Department of the Environment.

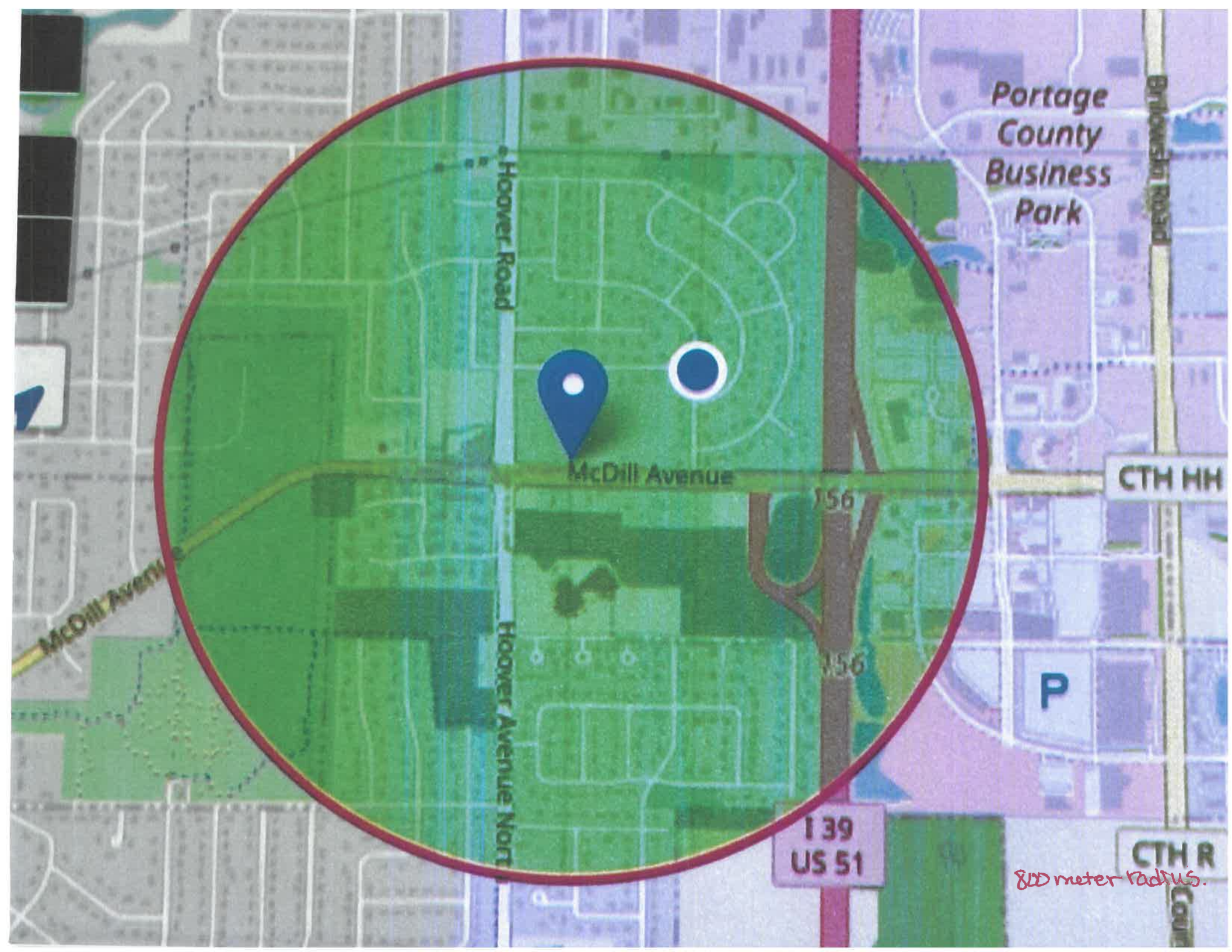
U.S. Environmental Protection Agency (U.S. EPA). (2017). *2017 Construction general permit (CGP) (as modified).*

Washington Department of Ecology. (2019). *2019 Stormwater management manual for western Washington.*

Water Environment Services (WES). (2008). *Erosion prevention and sediment control: Planning and design manual.*

Disclaimer

This fact sheet is intended to be used for informational purposes only. These examples and references are not intended to be comprehensive and do not preclude the use of other technically sound practices. State or local requirements may apply.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Vent Pipe Emissions from Storage Tanks at Gas Stations: Implications for Setback Distances

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Abstract

At gas stations, fuel vapors are released into the atmosphere from storage tanks through vent pipes. Little is known about when releases occur, their magnitude, and their potential health consequences. Our goals were to quantify vent pipe releases and examine exceedance of short-term exposure limits to benzene around gas stations. At two US gas stations, we measured volumetric vent pipe flow rates and pressure in the storage tank headspace at high temporal resolution for approximately three weeks. Based on the measured vent emission and meteorological data, we performed air dispersion modeling to obtain hourly atmospheric benzene levels. For the two gas stations, average vent emission factors were 0.17 and 0.21 kg of gasoline per 1,000 L dispensed. Modeling suggests that at one gas station, a 1-hour Reference Exposure Level (REL) for benzene for the general population (8 ppb) was exceeded only closer than 50 m from the station's center. At the other gas station, the REL was exceeded on two different days and up to 160 m from the center, likely due to non-compliant bulk fuel deliveries. A minimum risk level for intermediate duration (>14–364 days) benzene exposure (6 ppb) was exceeded at the elevation of the vent pipe opening up to 7 and 8 m from the two gas stations. Recorded vent emission factors were more than 10 times higher than estimates used to derive setback distances for gas stations. Setback distances should be revisited to address temporal variability and pollution controls in vent emissions.

1. INTRODUCTION

In the US, approximately 143 billion gal (541 billion L) of gasoline were dispensed in 2016 at gas stations[1] resulting in release of unburned fuel to the environment in the form of vapor or liquid.[2] This is a public health concern, as unburned fuel chemicals such as benzene, toluene, ethyl-benzene, and xylenes (BTEX) are harmful to humans.[3] Benzene is of special

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Competing financial interest declaration: TT directs a company (ARID), which develops technologies for reducing fuel emissions from gasoline-handling operations. AMR, BAM and MH have no conflicts of interests to declare.

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concern because it is causally associated with different types of cancer.[4] Truck drivers delivering gasoline and workers dispensing fuel have among the highest exposures to fuel releases.[4] However, people living near or working in retail at gas stations, and children in schools and on playgrounds can also be exposed, with distance to the gas stations significantly affecting exposure levels.[5–8] A meta-analysis[9] of three case-control studies[10–12] suggests that childhood leukemia is associated with residential proximity to gas stations.

Sources of unburned fuel releases at gas stations include leaks from storage tanks, accidental spills from the nozzles of gas dispensers,[13–15] fugitive vapor emissions through leaky pipes and fittings, vehicle tank vapor releases when refueling, and leaky hoses, all of which can contribute to subsurface and air pollution.[2] Routine fuel releases also occur through vent pipes of fuel storage tanks but are less noticeable because the pipes are typically tall, e.g., 4 m. These vent pipes are put in place to equilibrate pressures in the tanks and can be located as close as a few meters from residential buildings in dense urban settings (Figure 1).

Unburned fuel can be released from storage tanks into the environment through "working" and "breathing" losses.[16] A working loss occurs when liquid is pumped into or out of a tank. For a storage tank, this can happen when it is refilled from a tanker truck or when fuel is dispensed to refuel vehicles[17] if the pressure in the storage tank exceeds the relief pressure of the pressure/vacuum (P/V) valve.[18] P/V valve threshold pressures are typically set to around +3 and −8 inches of water column (iwc) (7.5 and −20 hPa). However, P/V valves are not always used, particularly in cold climates, as valves may fail under cold weather conditions.[17]

Breathing losses occur when no liquid is pumped into or out of a tank because of vapor expansion and contraction due to temperature and barometric pressure changes or because pressure in the storage tank may increase when fuel in the tank evaporates.[16,18] Although delayed or redirected by the P/V valve, breathing emissions can be significant and represent an environmental and health concern.[16]

Stage I vapor recovery systems, put in place to prevent working losses while delivering fuel to a station, collect the vapors displaced while loading a storage tank, redirecting them into the delivery truck. Stage II vapor recovery systems minimize working losses while delivering gas from the storage tank to the customer's car. During Stage II vapor recovery, gasoline vapors can be released through the vent pipe, if the sum of the flow rates of the returned volume and of the fuel evaporating within the storage tank is greater than the volume of liquid gasoline dispensed.[17] We refer to this scenario as pressure while dispensing (PWD). In theory, a properly designed Stage II vapor recovery system should not have working losses, although in practice this is not typically the case.[19]

Regulations on setback distances for gas stations are based on lifetime cancer risk estimates. Several studies have assessed benzene cancer risk near gas stations.[20–25] Based on cancer risk estimations, the California Air Resources Board (CARB) recommended that schools, day cares, and other sensitive land uses should not be located within 300 ft (91 m) of a large gas station (defined as a facility with an annual sales volume of 3.6 million gallons = 13.6

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million L or greater).[26] This CARB recommendation has not been adopted by all US states, and within states setback distances can depend on local government. Notably, CARB regulations do not account for short term exposure limits and health effects. An important limitation of existing regulations is the use of average gasoline emission rates estimated in the 90's that do not consider excursions.[27]

The main objective of this study is to evaluate fuel vapor releases through vent pipes of storage tanks at gas stations based on vent emission measurements conducted at two gas stations in the US in 2009 and 2015, including the characterization of excursions at a high temporal resolution (~minutes) and meteorological conditions at an hourly temporal resolution. In addition, we performed hourly simulations of atmospheric transport of emitted fuel vapors to inform regulations on setback distances between gas stations and adjacent sensitive land uses by comparing modeled benzene concentrations to four 60-min benzene exposure limits: an acute Reference Exposure Level (REL) for infrequent (once per month or less) exposure[28] and Emergency Response Planning Guidelines ERPG-1, ERPG-2 and ERPG-3.[29] Finally we compared simulated benzene levels to a Minimal Risk Level (MRL) for benzene for intermediate exposure duration (14 to 364 days)[30] because that duration window includes our duration of data collection. See Table 1 for the various benzene exposure limits and issuing agencies.

2. METHODS

Although we provide SI unit conversions, we report some measures in English engineering units (ft, gal, and lb) as regulatory agencies such as CARB use these units.

2.1. Sites

Data for this study were obtained from vent release measurements conducted at two gas stations as part of technical assistance to the gas stations to quantify fuel vapor losses through the vent pipes of their storage tanks. A motivation for conducting the measurements was to perform a cost-benefit analysis to compare the economic losses due to the lost fuel versus the cost of technologies that reduce the emissions. The exact location of the two gas stations is not revealed for confidentiality reasons. The gas station managers and staff who authorized the collection and analysis of these data have not been involved in the current manuscript.

The first gas station, "GS-MW," was located in the US Midwest and is a 24-hour operation. The study was conducted from December 2014 to January 2015 for 20 full days, and fuel sales $\dot{V}_{sales}$ were about 450,000 gal (1.7 million L) per month. Fuel deliveries to the gas station usually took place during the nighttime. The second gas station, "GS-NW," was located on the US Northwest coast and closed at night. Hours of operation were between 6:00 am and 9:30 pm on weekdays and between 7 am and 7 pm on weekends. That study was conducted in October 2009 for 18 full days, and fuel sales were $\dot{V}_{sales}$ ~700,000 gal (2.6 million L) per month.

Both gas stations are considered to be high-volume, because they dispense more than 3.6 million gallons of gasoline (both regular and premium) per year,[26] and fuel was stored in

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underground storage tanks (USTs), which is typical in the US. Both gas stations had Stage II vapor recovery installed using the vacuum-assist method. In that method, gasoline vapors, which would be ejected into the atmosphere as a working loss during refueling of customer vehicle tanks, are collected at the vehicle/nozzle interface by a vacuum pump. The recovered vapors are then directed via a coaxial hose back into the combined storage tank ullage (head space) of the gas station. Stage I vapor recovery was also used at both gas stations during fuel deliveries. Both sites had a 3-inch diameter (7.5 cm) single above-grade vent pipe with below-grade manifold that connected the vent lines from several USTs; the cracking pressures of the P/V valves were set to +3 and −8 iwc (+7.5 and −20 hPa).

2.2. Vent Emission Measurements

To quantify evaporative fuel releases through the vent pipe of a storage tank, the volumetric flow of the mixture of gasoline vapor and air was measured in the vent pipe. A dry gas diaphragm flow meter (American Meter Company, Model AC-250) was used. For each cubic foot (28 L) of gas flowing through the meter, a digital pulse was generated. Every minute, the number of pulses was read out and stored together with date and time on a data logger. Gas flow meters were obtained from a distributor calibrated and equipped with temperature compensation and a pulse meter.

To determine the time-dependent volumetric flow rate $Q(t)$ of the gasoline vapor/air mixture through the vent pipe, the time series of measured flow volumes were integrated over an averaging period (15 or 60 minutes) and divided by the duration of that period. I.e., $Q(t)$ is given by the number of pulses registered by the gas flow meter in a time window multiplied by 1 cubic foot and divided by the averaging time. The 15-minute averaging time was chosen to visualize time-dependent data, while the 60-minute averaging time was chosen because air pollution simulations were performed at that resolution.

Gas pressure p in the ullage of the storage tank was measured to assess vent emission patterns. For instance, releases can occur when the pressure exceeds the cracking pressure of the P/V valve in the vent pipe (the dry gas flow meter was fitted with a P/V valve on the outlet). Pressure was measured with a differential pressure sensor (Cerabar PMC 41, Endress +Hauser) every 4 seconds, and 2-minute average values were stored. The sensor range was scaled from −15 to +15 iwc (−37 to +37 hPa), with a full scale accuracy of 0.20%. We also obtained 15- and 60-minute averaged tank pressure data $p(t)$ where averages represent the means of the 2-minute average pressure measurements taken during each time window.

2.3. Descriptive Analysis

For the 60-minute flow rate, we calculated medians and inter quartile ranges (IQRs). To illustrate diurnal fluctuations in vapor emissions, we created box plots for the 60-minute flow rate distribution that occurred during each hour of the day. Spearman correlation coefficients between the time series for pressure and flow rate were calculated to evaluate whether pressure can be used to infer vent emissions.

To estimate the mass flow rate of gasoline $\dot{m}_{gas}$ that is released through the vent pipe in the form of a mixture of gasoline vapors and fresh air, we assumed, following the protocol of a

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study by the California Air Pollution Control Officers Association (CAPCOA) that assessed risks from fuel emissions from gas station (Appendix D-2[27]), that the density of gasoline vapors in this mixture is given by $\rho_{gas}^{(v)} = 0.3 \times 65$ lb / 379 ft^3 = 0.824 kg/m^3, i.e., the molar percentages of gasoline and air were 30% and 70%, respectively. Then the volumetric flow rate Q can be converted into a mass flow rate of the vaporized gasoline:

$$\dot{m}_{gas} = \rho_{gas}^{(v)} Q \quad \text{(Eq. 1)}$$

To arrive at vent emission factors, we first calculated the mean volumetric flow rate $\bar{Q}$, and then the mean mass flow rate $\bar{\dot{m}}_{gas} = \rho_{gas}^{(v)} \bar{Q}$. From the latter, one can calculate the vent emission factor

$$\mathrm{EF}_{vent} = \bar{\dot{m}}_{gas} / \dot{V}_{\mathrm{sales}} \quad \text{(Eq. 2)}$$

For EF_{vent}, CARB uses units of pounds of emitted gasoline vapors (also called total organic gases (TOG)) per 1,000 gallons dispensed, or more briefly lb/kgal where kgal stands for kilogallons.

As we were not able to measure benzene levels in the tank ullage, we assumed like the CAPCOA study (Section C) that the density of the mixture of gasoline vapors and fresh air was $\rho_{mix}^{(v)} = 1.05$ lb/ft^3 = 1.682 kg/m^3 and that the emitted gasoline vapor/air mixture contained 0.3% of benzene by weight.[27] Therefore, the mass flow rate of benzene through the vent pipe was estimated as follows:

$$\dot{m}_{benz} = 0.003\, \rho_{mix}^{(v)} Q \quad \text{(Eq. 3)}$$

2.4. Air Pollution Modeling

We used the AERMOD Modeling System developed by the US Environmental Protection Agency (EPA) to model the dispersion of benzene vapors released into the environment through vent pipes of fuel storage tanks and from other sources.[31] AERMOD simulates atmospheric pollutant transport at a 1-hour temporal resolution. 3D polar grids were created with the gas station in the origin and potential receptors at different radial distances (up to 170 meters) and angles (10° increments). The grids were placed at the ground level (z = 0 m), in the breathing zone (z = 2 m), and at the 2nd floor level (z = 4 m) where the vent pipe emissions were assumed to occur. The topography was simplified for modeling purposes consistent with the CAPCOA study,[27] i.e., the terrain was assumed to be flat with no buildings present. Vent pipe emissions were modeled as a capped point source. Chemical reactions of benzene were not modeled, as residence times of atmospheric benzene are on the order of hours or even days,[32] i.e. much longer than the travel time of benzene vapors across the 340-m diameter model domain.

For the period of time when vent emission measurements were made, we obtained meteorological data at a 1-hour temporal resolution that are representative for the geographic

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locations of the two gas stations. Table SI-1 provides descriptive statistics of that data. The time series were used in AERMOD to model the transport of benzene in the temporally varying turbulent atmosphere. We also used the 1-hour average time series of benzene emission rates (Eq. 3) as an input into AERMOD.

To evaluate at each grid point whether OEHHA's acute REL or AIHA's ERPG levels were exceeded at least once, we determined maximum 1-hour average benzene concentrations that were simulated for about three weeks. To evaluate how often the OEHHA REL was exceeded at each grid point in the breathing zone, we created plots indicating the number of exceedances and the day when the maximum benzene level was observed.

To facilitate comparison to published benzene measurements around gas stations, we determined for each simulated radial distance from a gas station the mean of the average concentrations simulated for each ten degree increment on the radius around the gas station.

3. RESULTS: VENT RELEASES

3.1 Times Series of Tank Pressure and Flow Rate

Figure 2 shows the time-series data for the volumetric flow rate Q of the gasoline vapor/air mixture through the vent pipe and tank pressure p that we collected at the two gas stations. At GS-MW, little vapor was typically released in the late night and in the very early morning, while releases were generally much higher during the daytime and evenings, presumably when more fuel was dispensed (Figure 2a). Occasionally, no vapor releases occurred for several hours. While we do not have access to time of fuel delivery records, field visits indicate that time periods with no releases coincide with fuel deliveries. For instance, fuel delivery likely occurred on January 6 at 7 pm (see Figure 3a; an amplification of data shown in Figure 2a). As a result, the UST pressure dropped by about 10 hPa, far below the cracking pressure of the P/V valve. The decreased gas pressure in the ullage increased until the cracking pressure of the P/V valve was reached. A very small vapor release (~2 L/min) was observed briefly on the next day at 2 am. The vapor flow rate becomes relatively large again, ~12 L/min, only after 6 am, i.e., 11 hours after fuel delivery.

Figure 3b amplifies a major vapor release at GS-MW. The UST pressure significantly exceeded the cracking pressure of the P/V valve and rose rapidly up to 37 hPa, which coincides with vapors being released at a high flow rate (15-min average) of about 470 L/min.

At GS-NW, vapor releases followed a quite different pattern (Figure 2b). Contrary to GS-MW, vapor releases occurred in a cyclical pattern, and tended to be higher in the late night and in the very early morning when the gas station was closed.

3.2. Statistics of Vapor Emissions

The average volumetric flow rate $\bar{Q}$ through the vent pipe for the entire period of time during which measurements were taken was $\bar{Q}$ = 7.9 L/min for GS-MW and $\bar{Q}$ = 15.4 L/min for GS-NW, which is consistent with the higher sales volume $\dot{V}_{sales}$ of GS-NW. These emissions consist of a mixture of gasoline vapors and air. Using Equation (1), the volumetric

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flow rates were converted into average mass flow rates of gasoline: $\overline{\dot{m}}_{gas}$ = 0.39 kg/hr for GS-MW and $\overline{\dot{m}}_{gas}$ = 0.76 kg/hr for GS-NW. Using Eq. (2), we determined a vent emission factor EF_{vent} = 0.17 kg per 1,000 L = 1.4 lb/kgal for GS-MW and EF_{vent} = 0.21 kg per 1,000 L = 1.7 lb/kgal for GS-NW.

The medians (IQRs) for the 60-minute averaged flow rate Q (L/min) were 6.1 (1.9, 10.9) for GS-MW and 16.0 (12.7, 18.4) for GS-NW. For GS-MW, the mean is larger than the median, indicating a more skewed distribution of flow rates when compared to GS-NW. Also the first quartile is much lower than the median for GS-MW, indicating that there are periods of time during which little emissions occurred. Conversely, GS-NW was releasing emissions more consistently.

Figure 4a shows boxplots illustrating the distribution of flow rate Q for each hour of the day at GS-MW. Less vapor was released between 10 pm and 4 am, even though the gas station was in operation, albeit at lower activity levels. The flow rate Q at GS-NW (Figure 4b) had fewer outliers, and the highest outlier was an order of magnitude lower than the highest one at GS-MW. Emissions were highest between 1 and 3 am, when the gas station was closed.

The Spearman correlation coefficients between tank pressure p and vent flow rate Q were r = 0.58 for GS-MW and r = 0.85 for GS-NW. Thus, vent releases are moderately and strongly correlated with tank pressure, respectively. Table 2 summarizes statistical properties of vent emissions at the two gas stations.

4. RESULTS: AIR POLLUTION MODELING

4.1. Emission Sources and Rates

Vent pipe emissions of benzene were modeled at a 1-hour temporal resolution as described in Section 2.4. However, they are not the sole source of gasoline emissions at gas stations. Accidental spills from nozzles regularly occur near the dispensers, "refueling losses" can occur when gasoline vapors are released from the vehicle tank during refueling due to the rising liquid levels in the tanks, fuel vapors are released from permeable dispensing hoses, and "fugitive" or leakage emissions occur with driving force derived from storage tank pressure. In Section A of Supporting Material, we detail how these other emission sources were modeled. Table 3 summarizes estimated mean emission rates. Note that the vent pipe losses are much greater than other losses.

4.2. Predicted Benzene Levels

Figure 5 shows for both gas stations and at each grid point the maximum 1-hour average benzene concentration observed during the simulated periods in time. Benzene levels depend significantly on elevation within a 50-meter radius around the centers of the gas stations. Close to the centers of the gas stations, benzene levels are higher at the 4-m elevation and at ground level due to vent pipe emissions, which represent the largest emission source (Table 3). Further than 50 m away from the center, the vertical concentration differences become less obvious due to dispersion causing vertical mixing of benzene vapors.

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At GS-MW, the 1-hour acute REL of 26 μg/m^3 was exceeded 160 m away from the center of the gas station, at the location (x = 158 m, y = 28 m) both at ground level and in the breathing zone. At grid points with a distance greater than 50 m from the center of the gas station, the REL was exceeded at most once (Figure SI-1a). However, the exceedance at different grid points did not occur on the same day (Figure SI-1b). Within the 20 days during the measurement campaign, exceedances occurred on the 4th and 13th of January.

At GS-NW, the furthest REL exceedance occurred at 50 m from the center of the gas station at the grid point (x = −38 m, y = 32 m) as shown in Figure SI-2a. At a distance of 40 m, the REL was exceeded three times at one grid point (260° angle), and at 35 m four times at two grid points (250° and 260° angles) (Figure SI-2b). At a distance of 20 m, the REL was exceeded at 30 (out of 36) grid points, and on nine different days.

Average benzene levels are shown in Figure 6 for both gas stations. The MRL is exceeded at the elevation of the vent pipe opening, z = 4 m, up to 7 m away from for GS-MW and up to 8 m from GS-NW. Figure 7 shows the average benzene concentration as a function of distance at an elevation of 2 m. Close to the center, benzene levels first increase and then decrease.

5. DISCUSSION

5.1. Vent Emission Factors

We present unique data on vent emissions from USTs at two gas stations. Emissions can be compared to vent losses assumed by CAPCOA.[27] For a gas station with Stage I and II vapor recovery technology and a P/V valve on the vent pipe of the UST (Scenario 6B), the CAPCOA study assumed loading losses of 0.084 and breathing losses of 0.025 lb/kgal dispensed. The total loss of gasoline through the vent pipe is the sum of the two and amounts to a vent emission factor EF_{vent} = 0.109 lb/kgal. Based on actual measurements in two fully functioning US gas stations, we obtained EF_{vent} values of 1.4 lb/kgal for GS-MW and 1.7 lb/kgal for GS-NW, more than one order of magnitude higher than the CAPCOA estimate. While the difference between our measurements and the CAPCOA estimates may appear surprising, it is important to consider that the CAPCOA estimates are based on relatively few measurements and some unsupported assumptions,[33] particularly with regard to uncontrolled emissions due to equipment failures or defects (Appendix A-5[27]).

5.2. Pressure Measurements

Tank ullage pressure p was moderately to strongly positively correlated with vent flow rate Q, likely because exceedance of the cracking pressure of the P/V valve causes a vent release. Thus pressure measurements can be used to infer vent releases. Real-time detection of equipment failures and leaks via so-called in-station diagnostics systems is based on our observed correlations between p and Q.

5.3. Diurnal Fluctuations in Vent Emissions

Diurnal vent emissions were quite different at the two gas stations. At GS-MW, a 24-hour operation, vent emissions were high during the daytime, presumably due to PWD. Emissions

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ceased at night, likely because less gasoline was dispensed and fuel deliveries with relatively cool product were frequent. Evaporative losses could also have been lower at night because the cooler delivered fuel would cause slight contraction of the liquid phase with corresponding growth in the ullage volume while at the same time lowering the vapor pressure of gasoline in the UST.

At GS-NW, vent pipe releases occurred most of the time, during the daytime when fuel was dispensed (PWD) and at night when the gas station was closed. Vent releases were higher when the gas station was closed, suggesting that during the day-time Stage II vapor recovery resulted in the injection of vapors into the storage tank that were not completely equilibrated with the liquid gasoline. During night-time, the gradual equilibration of unsaturated air in the ullage of the UST with gasoline vapors could then have caused exceedance of the cracking pressure of the P/V valve and consequently vapor release. It seems counterintuitive that less nighttime emissions occurred at the gas station where fuel was dispensed. However, while fuel is being dispensed, the outgoing liquid creates additional ullage volume, and depending on excess air ingestion rate, a negative pressure could result that lowers vent pipe emissions.

Dispensing fuel to customer vehicles and the associated Stage II vapor recovery system interact with vent emissions and can even cause vent emission during PWD, because the vacuum-assist method can negatively interfere with Onboard Refueling Vapor Recovery (ORVR) installed in customer vehicles.[34] However, Stage II vapor recovery is not obsolete. It can be used in conjunction with ORVR to minimize exposure of gas station customers and workers to benzene due to working losses,[35] particularly when customer vehicles are not equipped with ORVR (e.g., older vehicles, boats, motorcycles) or small volume gasoline containers are refueled. Enhanced Stage II vapor recovery technology can significantly reduce vapor emissions both at the nozzle and from UST vent pipes.[36]

5.4. Fuel Deliveries and Accidental Vent Releases

Based on observations and interpretation of time series of the tank pressure data, it is likely that the peak vent emissions (e.g., Figure 3b) were partly due to non-compliant bulk fuel drops where the Stage I vapor recovery system either was not correctly hooked up by the delivery driver or to hardware problems with piping and/or valves. This conjecture is consistent with typical US storage tank volumes (~10,000 to 30,000 gal). Assuming that Phase I vapor recovery did not work at all and that 10,000 gal (~38,000 L) of fuel were delivered, the working loss (volume of gasoline vapor/air mixture released to the atmosphere through the vent pipe) is 38,000 L. It is also reasonable to assume that delivery lasted less than one hour. According to Table 2, the maximum hourly flow rate through the vent pipe was 250 L/min at GS-MW, which would result in a maximum cumulative vapor release of 15,000 L within this hour. The measured maximum cumulative release underestimates the assumed working loss of 38,000 L. This could be due to a fuel delivery, which involved dropping fuel from multiple compartments of a tanker truck, with the vapor return hose not being correctly hooked up for only some of the emptied compartments.

At GS-MW, UST pressure decreased after fuel delivery (causing vent emissions to cease for several hours) during the climatic conditions prevalent during the observation period,

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behavior not observed at GS-NW. In practice, it is possible to observe both positive and negative pressure excursions, even during the same fuel delivery (when multiple fuel compartments of tanker trucks are unloaded), when Stage I vapor recovery is in place (personal observation by TT).

5.5. Exceedance of 1-hour Exposure Limits

AERMOD air pollution modeling suggests that at GS-MW the 1-hour acute REL was exceeded at one grid point 160 m (525 ft) from the center of the gas station once in 20 days (Figure 5). This distance is larger than the 300-ft (91 m) setback distance recommended by CARB for a large gasoline dispensing facility.[26] Assuming the gas station's fence line is less than 225 ft (69 m) from its center (where the vent pipe was assumed to be located), our study shows that sensitive land uses at a distance further than 300 feet from the fence line of the gas station would represent a health concern despite compliance with the CARB guidelines because of non-compliance with the acute REL.

At any location further than 50 m from the gas station's center, the REL was exceeded at most once during the 20-day measurement campaign (Figure SI-1a). However, exceedance occurred at several locations, and on two different days (Figure SI-1b). E.g., at a distance of 120 m from the center, the REL was exceeded at three grid points, and the number of grid points increased with closer proximity to the gas station. This suggests that it was not just a single worst-case scenario or a single accidental vapor release that led to REL exceedance; rather exceedance may occur more frequently than is anticipated. Prevalent wind directions during the measurement campaign explained the directional patterns of exceedances (see the wind rose in Figure SI-3a).

At GS-NW, despite its higher sales volume, the REL was exceeded only closer than 50 m from the gas station's center. However, exceedance occurred much more frequently (Figure SI-2), likely because of the higher sales volume of GS-NW. Again, the wind rose for GS-NW (Figure SI-3b) explains spatial patterns of REL exceedance.

None of AIHA's three ERPG levels were exceeded, meaning that individuals, except perhaps sensitive members of the public, would not have experienced more than mild, transient adverse health effects.

5.6. Average Benzene Levels

The initial increase in average benzene levels when moving away from the gas stations' centers (Figure 7) is likely due to the vent emissions (at 4 m) which represent the largest benzene source, and which require a certain transport distance until they reach the 2-m level through dispersion. Further away from the gas station, benzene levels are higher for GS-NW than for GS-MW likely because of the higher sales volume of GS-NW. However, close to the center, benzene levels are higher at GS-MW. This can be attributed to the higher wind speeds at GS-NW (Table SI-1), which result in greater initial dilution of emitted pollutants in the incoming airstream and also in greater subsequent pollutant dispersion.

Modeled average benzene concentrations are generally lower (~10 $\mu g/m^3$ or less) than those measured in the surroundings of gas stations, likely because our simulations do not account

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for traffic-related air pollution (TRAP). For instance, a study published by the Canadian petroleum industry found average benzene concentrations of 146 and 461 ppb (466 and 1,473 $\mu g/m^3$) at the gas station property boundary in summer and winter, respectively,[37] values orders of magnitudes higher than ours. A South Korean study examined outdoor and indoor benzene concentrations at numerous residences within 30 m and between 60 and 100 m of gas stations and found median outdoor benzene concentrations of 9.9 and 6.0 $\mu g/m^3$, respectively,[7] while we simulated benzene levels on the order of 1 $\mu g/m^3$ (Figure 7). In a study on atmospheric BTEX levels in an urban area in Iran, the three highest BTEX levels were measured near gas stations (~150 m away); the measured benzene levels (64±36, 31±28, 52±26 $\mu g/m^3$) were again much higher than ours simulated at that distance, likely due to TRAP. Our modeled average benzene levels at a distance of about 50 m are on the same order as background benzene levels of 1.0 $\mu g/m^3$ that were measured in 2010 in the National Air Toxics Trend Sites (NATTS) network of 27 stations located in most major urban areas in the US.[38] However, our modeled levels at a distance of 170 m were 0.07 at GS-MW and 0.12 at GS-NW, a non-negligible addition to urban background levels.

At both gas stations, the MRL was exceeded at the level of the vent pipe opening in the vicinity of the gas stations, up to 7 m away from the vent pipe at GS-MW and 8 m at GS-NW. Therefore there might be an appreciable risk of adverse noncancer health effects for individuals living at the 2nd-floor level relatively close to high-volume gas stations such as GS-MW and GS-NW.

5.7. Limitations

A limitation of our study is that data were collected only in fall and winter. Results cannot be easily extrapolated to other seasons, because vent pipe emissions are seasonally dependent, e.g., due to seasonally dependent gasoline formulations and meteorological conditions. However, modeled exceedance of the OEHHA acute REL in the winter season is already of concern, because that REL was developed for once per month or less exposures.

Another limitation is that we did not directly measure benzene levels in the vent pipe, and instead made assumptions about vapor composition that were also made in the CAPCOA study[27] of gas station emissions. In practice it may be difficult to obtain permission from gas station owners to measure benzene levels directly.

In part because we did not want to reveal the locations of the gas stations, we did not use site-specific topography information in the air dispersion modeling and instead assumed flat terrain. While this simplification results in less accurate air pollution predictions for the two sites, using a "generic" gas station is perhaps more representative of other gas station sites, and is consistent with an approach used in a previous study.[27]

Finally, our study did not predict benzene levels in indoor environments. Even though indoor air pollution levels may substantially differ from outdoor levels due to indoor sources (e.g., smoking, photocopying),[39] our study can still inform exposure levels in indoor environments as outdoor sources may be the main contributors to indoor air pollution, e.g., in buildings situated in urban areas and close to industrial zones or streets with heavy traffic.[40] This is

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relevant to workers and customers in C-stores or other fast-food/gasoline station combination facilities.

6. CONCLUSIONS

Our study is to the best of our knowledge the first one to (1) report hourly vent emission data for gasoline storage tanks in the peer-reviewed literature and (2) use these data in hourly simulations of atmospheric benzene vapor transport. This allowed us to examine potential exceedance of short-term exposure limits for benzene. Prior studies including CAPCOA's[27] could not do so as average emission rates were used (only meteorological data was used at an hourly resolution).

Our findings support the need to revisit setback distances for gas stations, which are based on more than 2-decade old estimates of vent emissions.[33] Also, CARB setback distances are based on a binary decision, related to whether the gasoline sales volume $\dot{V}_{sales}$ is more than 3.6 million gal per year. Our data support, however, that setback distances should be a continuous function of sales volume $\dot{V}_{sales}$ and also include the type of controls installed at the facility. Setback distances should also address health outcomes other than cancer. OEHHA's acute REL for benzene could be used to inform setback distances as it accounts for non-cancer adverse health effects of benzene and its metabolites.[41] ATSDR's MRL could also be considered since it is a health-based limit.

We note that CARB recommended their setback distances in 2005, presumably assuming pollution prevention technology yielding a 90% reduction in benzene emissions.[26] Since then, CARB further promoted use of second-generation vapor recovery technology (Enhanced Vapor Recovery, EVR) to reduce emissions further. EVR includes technology that is supposed to prevent fuel vapors in overpressurized tanks from being expelled into the atmosphere.[42] To that end, "bladder tanks" have been proposed, into which the gasoline vapor/air mixture is directed as the pressure in the combined ullage space of the storage tank increases and from which the mixture is redirected into the fuel storage tanks if the ullage pressure becomes negative (when fuel is dispensed). The challenge with such a system is to ensure that the bladder tank capacity is not exceeded by the fuel evaporation rate. Alternatively, fuel vapor release can be reduced by processing the fuel/air mixture through either a semi-permeable membrane which selectively exhausts clean air and returns enriched fuel vapor[43] or an activated carbon filter which adsorbs hydrocarbons (and water vapor) and exhausts air into the atmosphere, or by combusting the fuel/air mixture which would otherwise be released through the P/V valve. Therefore, current CARB setback distances might be adequate for gas stations in California but less so for the other 49 US states, and other countries—depending on pollution prevention technology requirements.

The larger areal extent of modeled REL exceedance at GS-MW is due to "accidental" releases of gasoline vapors. Even though regulations appear generally not to be driven by accidental releases, at GS-NW such releases likely led on two different days to REL exceedances at distances beyond CARB's recommended setback distances. Policies should address accidental fuel vapor releases that depending on pollution prevention technology

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(here Stage I vapor recovery) and its proper functioning can occur on a frequent basis (twice at GS-MW within about three weeks).

In future work, potential exceedance of other shorter-term exposure limits should be examined, e.g., the 15-minute short-term exposure limits (STELs) and the 8-hour time-weighted averages (TWAs) used for occupational exposures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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REFERENCES

1. EIA. U.S. Product Supplied of Finished Motor Gasoline: U.S. Energy Information Administration; 2017 Available from: http://www.eia.gov/dnav/pet/hist/LeafHandler.ashx?n=pet&s=mgfupus1&f=m.
2. Hilpert M, Mora BA, Ni J, Rule AM, Nachman KE. Hydrocarbon Release During Fuel Storage and Transfer at Gas Stations: Environmental and Health Effects. Current Environmental Health Reports. 2015;2(4):412–22. doi: 10.1007/s40572-015-0074-8. [PubMed: 26435043]
3. ATSDR. Interaction Profile for: Benzene, Toluene, Ethylbenzene, and Xylenes (BTEX). Agency for Toxic Substances and Disease Registry, 2004.
4. IARC. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 100F 2012 [12 24, 2017]. Available from: http://monographs.iarc.fr/ENG/Monographs/vol100F/.
5. Terres IMM, Minarro MD, Ferradas EG, Caracena AB, Rico JB. Assessing the impact of petrol stations on their immediate surroundings. Journal of Environmental Management. 2010;91(12):2754–62. doi: 10.1016/j.jenvman.2010.08.009. [PubMed: 20810207]
6. Jo WK, Oh JW. Exposure to methyl tertiary butyl ether and benzene in close proximity to service stations. Journal of the Air & Waste Management Association. 2001;51(8):1122–8. doi: 10.1080/10473289.2001.10464339. [PubMed: 11518287]
7. Jo WK, Moon KC. Housewives' exposure to volatile organic compounds relative to proximity to roadside service stations. Atmospheric Environment. 1999;33(18):2921–8. doi: 10.1016/s1352-2310(99)00097-7.
8. Hajizadeh Y, Mokhtari M, Faraji M, Mohammadi A, Nemati S, Ghanbari R, Abdolahnejad A, Fard RF, Nikoonahad A, Jafari N, Miri M. Trends of BTEX in the central urban area of Iran: A preliminary study of photochemical ozone pollution and health risk assessment. Atmospheric Pollution Research. 2018;9(2):220–9.
9. Infante PF. Residential Proximity to Gasoline Stations and Risk of Childhood Leukemia. American Journal of Epidemiology. 2017;185(1):1–4. [PubMed: 27923798]
10. Steffen C, Auclerc MF, Auvrignon A, Baruchel A, Kebaili K, Lambilliotte A, Leverger G, Sommelet D, Vilmer E, Hemon D, Clavel J. Acute childhood leukaemia and environmental exposure to potential sources of benzene and other hydrocarbons; a case-control study. Occupational and Environmental Medicine. 2004;61(9):773–8. doi: 10.1136/oem.2003.010868. [PubMed: 15317919]
11. Brosselin P, Rudant J, Orsi L, Leverger G, Baruchel A, Bertrand Y, Nelken B, Robert A, Michel G, Margueritte G, Perel Y, Mechinaud F, Bordigoni P, Hemon D, Clavel J. Acute childhood leukaemia and residence next to petrol stations and automotive repair garages: the ESCALE study (SFCE). Occupational and Environmental Medicine. 2009;66(9):598–606. [PubMed: 19213757]

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12. Harrison RM, Leung PL, Somervaille L, Smith R, Gilman E. Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. Occupational and Environmental Medicine. 1999;56(11):774–80. [PubMed: 10658564]
13. Hilpert M, Breysse PN. Infiltration and evaporation of small hydrocarbon spills at gas stations. Journal of Contaminant Hydrology. 2014;170:39–52. [PubMed: 25444115]
14. Mora BA, Hilpert M. Differences in Infiltration and Evaporation of Diesel and Gasoline Droplets Spilled onto Concrete Pavement. Sustainability. 2017;9(7). doi: 10.3390/su9071271.
15. Morgester JJ, Fricker RL, Jordan GH. Comparison of spill frequencies and amounts at vapor recovery and conventional service stations in California. Journal of the Air & Waste Management Association. 1992;42(3):284–9.
16. Yerushalmi L, Rastan S. Evaporative Losses from Retail Gasoline Outlets and Their Potential Impact on Ambient and Indoor Air Quality In: Li A, Zhu Y, Li Y, editors. Proceedings of the 8th International Symposium on Heating, Ventilation and Air Conditioning: Volume 1: Indoor and Outdoor Environment. Berlin, Heidelberg: Springer Berlin Heidelberg; 2014 p. 13–21.
17. Statistics Canada. Gasoline Evaporative Losses from Retail Gasoline Outlets Across Canada: Environment Accounts and Statistics Analytical and Technical Paper Series; 2009 Available from: http://www.statcan.gc.ca/pub/16-001-m/2012015/part-partie1-eng.htm.
18. EPA. Transportation and Marketing of Petroleum Liquids. Environmental Protection Agency, 2008 AP 42, Volume I, Chapter V: Petroleum Industry.
19. McEntire BR. Performance of Balance Vapor Recovery Systems at Gasoline Dispensing Facilities. San Diego Air Pollution Control District, 2000.
20. Atabi F, Mirzahosseini SA. GIS-based assessment of cancer risk due to benzene in Tehran ambient air. Int J Occup Med Environ Health. 2013;26(5):770–9. Epub 2014/01/28. doi: 10.2478/s13382-013-0157-4. [PubMed: 24464541]
21. Correa SM, Arbilla G, Marques MRC, Oliveira KMPG. The impact of BTEX emissions from gas stations into the atmosphere. Atmospheric Pollution Research. 2012;3(2):163–9.
22. Cruz L, Alves L, Santos A, Esteves M, Gomes Í, Nunes L. Assessment of BTEX Concentrations in Air Ambient of Gas Stations Using Passive Sampling and the Health Risks for Workers. Journal of Environmental Protection. 2007;8:12–25.
23. Edokpolo B, Yu QJ, Connell D. Health risk characterization for exposure to benzene in service stations and petroleum refineries environments using human adverse response data. Toxicology Reports. 2015;2:917–27. [PubMed: 28962430]
24. Edokpolo B, Yu QJ, Connell D. Health Risk Assessment of Ambient Air Concentrations of Benzene, Toluene and Xylene (BTX) in Service Station Environments. International Journal of Environmental Research and Public Health. 2014;11(6):6354–74. [PubMed: 24945191]
25. Karakitsios SP, Delis VK, Kassomenos PA, Pilidis GA. Contribution to ambient benzene concentrations in the vicinity of petrol stations: Estimation of the associated health risk. Atmospheric Environment. 2007;41(9):1889–902.
26. CalEPA/CARB. Air Quality and Land Use Handbook: A Community Health Perspective: California Environmental Protection Agency & California Air Resources Board; 2005.
27. CAPCOA. Gasoline Service Station Industrywide Risk Assessment Guidelines. Toxics Committee of the California Air Pollution Control Officers Association (CAPCOA), 1997.
28. WHO. WHO Guidelines for Indoor Air Quality: Selected Pollutants: Geneva: World Health Organization; 2010.
29. AIHA. 2016 ERPG/WEEL Handbook. Current ERPG® Values (2016): American Industrial Hygiene Association; 2016.
30. ATSDR. Minimal Risk Levels (MRLs): Agency for Toxic Substances and Disease Registry; 2018 [5 24, 2018]. Available from: https://www.atsdr.cdc.gov/mrls/index.asp.
31. Cimorelli AJ, Perry SG, Venkatram A, Weil JC, Paine RJ, Wilson RB, Lee RF, Peters WD, Brode RW. AERMOD: A Dispersion Model for Industrial Source Applications. Part I: General Model Formulation and Boundary Layer Characterization. Journal of Applied Meteorology. 2005;44(5):682–93.

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32. ATSDR. Toxicological Profile for Benzene. Agency for Toxic Substances and Disease Registry, 2007 CAS#: 71-43-2.
33. Aerovironment I. Underground Storage Tank Vent Line Emissions form Retail Gasoline Outlets. Prepared for WSPA, 1994 AV-FR-92-01-204R2.
34. EPA. Stage II Vapor Recovery Systems Issues Paper. U.S. EPA. Office of Air Quality Planning and Standards Emissions Monitoring and Analysis Division. Emissions Factors and Policy Applications Group (D243–02), 2004.
35. Cruz-Nunez X, Hernandez-Solis JM, Ruiz-Suarez LG. Evaluation of vapor recovery systems efficiency and personal exposure in service stations in Mexico City. Science of the Total Environment. 2003;309(1–3):59–68. doi: 10.1016/s0048-9697(03)00048-2. [PubMed: 12798092]
36. CARB. Revised Emission Factors for Gasoline Marketing Operations at California Gasoline Dispensing Facilities. California Air Resources Board, Monitoring and Laboratory Division, 2013.
37. Akland GG. Exposure of the general population to gasoline. Environ Health Perspect. 1993;101 Suppl 6:27–32. Epub 1993/12/01.
38. Strum M, Scheffe R. National review of ambient air toxics observations. Journal of the Air & Waste Management Association (1995). 2016;66(2):120–33. Epub 2015/08/01. doi: 10.1080/10962247.2015.1076538. [PubMed: 26230369]
39. El-Hashemy MA, Ali HM. Characterization of BTEX group of VOCs and inhalation risks in indoor microenvironments at small enterprises. Science of The Total Environment. 2018;645:974–83. [PubMed: 30248884]
40. Jones AP. Indoor air quality and health. Atmospheric Environment. 1999;33(28):4535–64.
41. Budroe J Notice of Adoption of Revised Reference Exposure Levels for Benzene: Office of Environmental Health Hazard Assessment (California, US); 2014 Available from: https://oehha.ca.gov/air/crnr/notice-adoption-revised-reference-exposure-levels-benzene.
42. CARB. Public Workshop to Discuss: Overpressure Conditions at Gasoline Dispensing Facilities Equipped with Underground Storage Tanks and Phase II Enhanced Vapor Recovery including In-Station Diagnostic Systems. 12 12–13, 2017 Diamond Bar & Sacramento, CA California Air Resources Board; 2017. Available from: https://www.arb.ca.gov/vapor/op/wrkshps/dec2017op_vr_pres.pdf.
43. Semenova SI. Polymer membranes for hydrocarbon separation and removal. Journal of Membrane Science. 2004;231(1–2):189–207.
44. EPA. Gasoline Mobile Source Air Toxics 2017. Available from: https://www.epa.gov/gasoline-standards/gasoline-mobile-source-air-toxics.
45. EPA. Air Quality: Widespread Use for Onboard Refueling Vapor Recovery and Stage II Waiver2012;Federal Register 40 CFR 51:28772–82.

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Figure 1:
The three vent pipes (enclosed by the red ellipse) on the right side of the convenience store of a gas station are less than 10 m away from the residential building.

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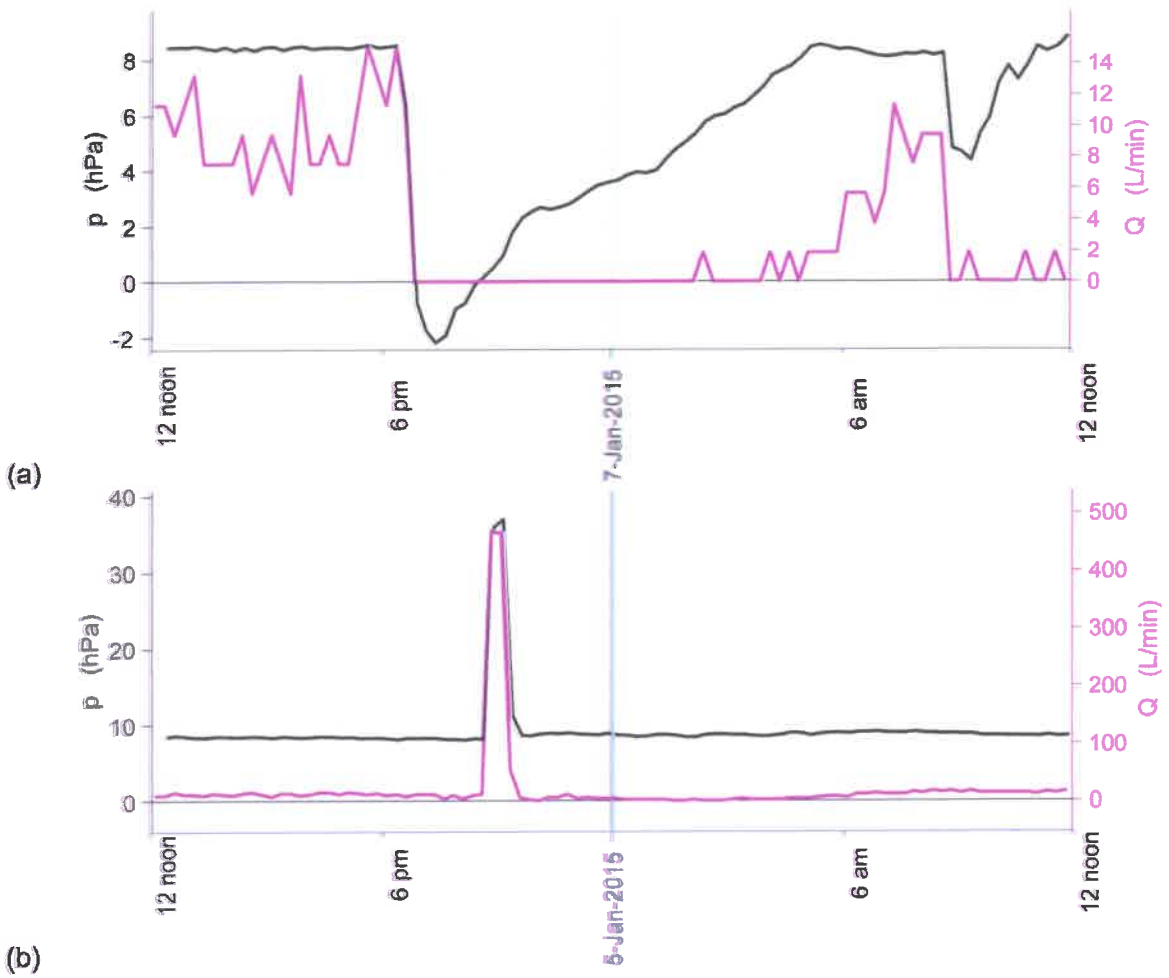

Figure 3:

Amplifications of time series data (15-minute averages) for GS-MW. (a) Tank pressure *p* became negative after fuel delivery. As a result, vent emission ceased for several hours. (b) A major vapor release (burst) likely occurred when the cracking pressure of the P/V valve was significantly exceeded at around 9 pm during a non-compliant bulk fuel delivery.

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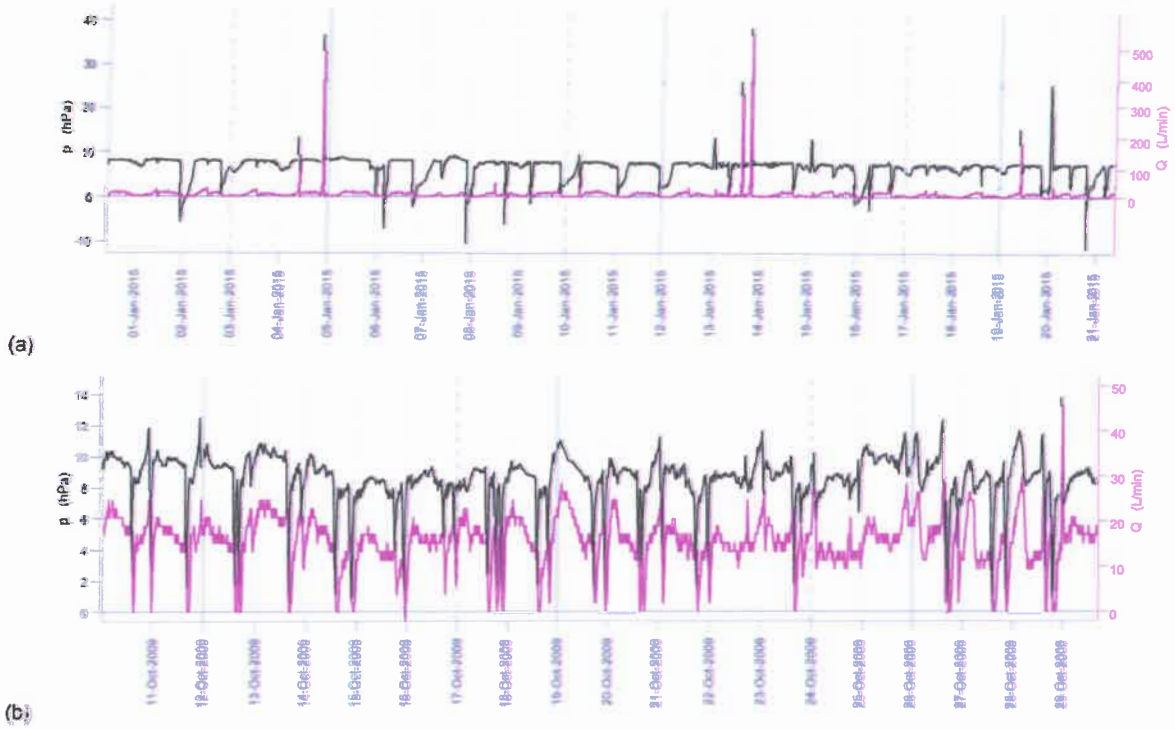

Figure 2:

Time series of ullage pressure p (left ordinate) and volumetric flow rate Q (right ordinate) for (a) GS-MW and (b) GS-NW. Horizontal tick marks indicate midnights. The vertical dashed and thick solid gray lines enclose weekends.

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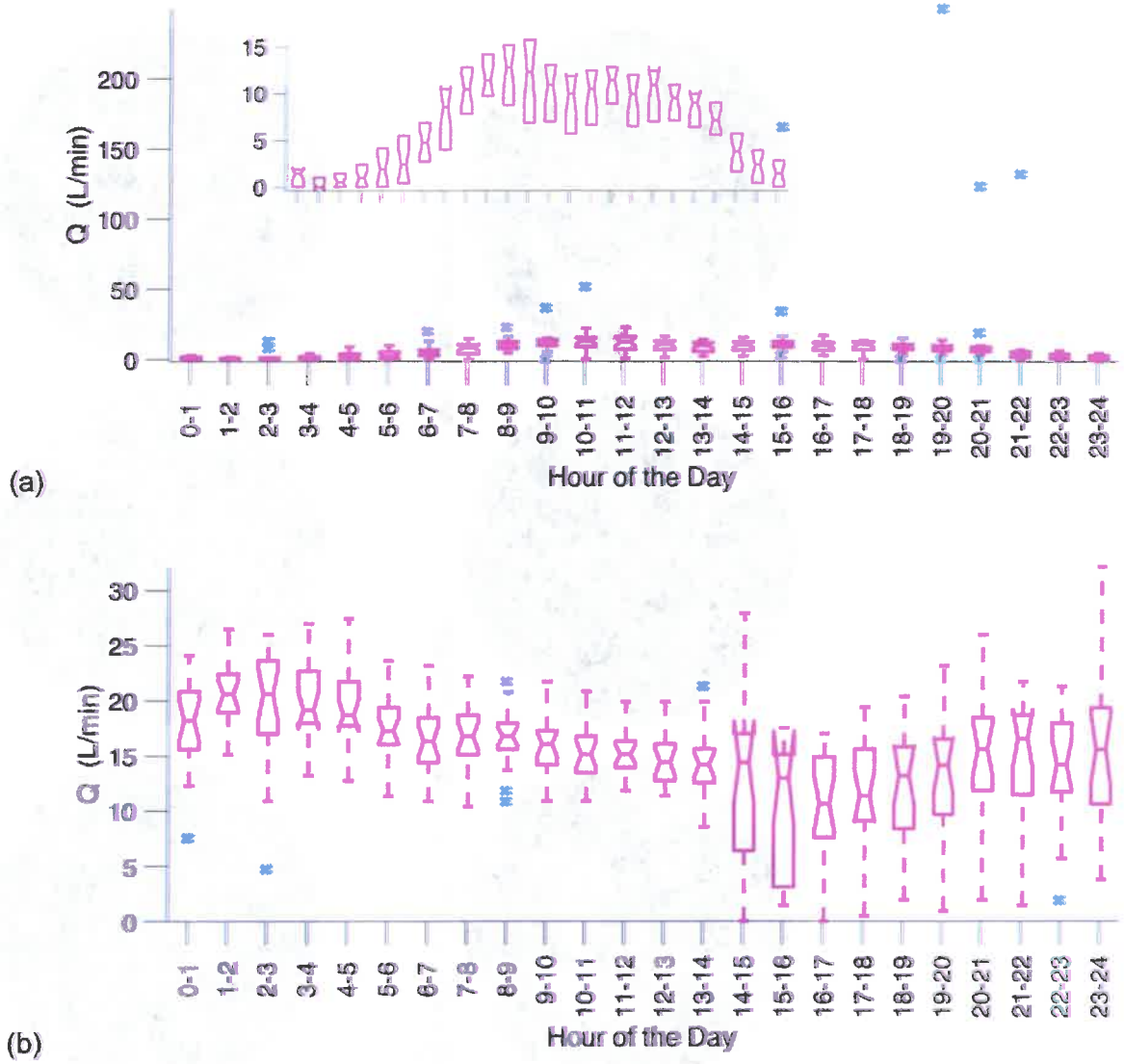

Figure 4:

Distribution of vent emissions Q observed for each hour of the day at (a) GS-MW [insert shows the IQRs of Q] and (b) GS-NW gas stations. In (a), outliers make it difficult to recognize variations in median hourly emissions. We therefore plotted in the inset only the IQRs. Boxes indicate median and IQR, whiskers values within 1.5 the IQR, and asterisks outliers.

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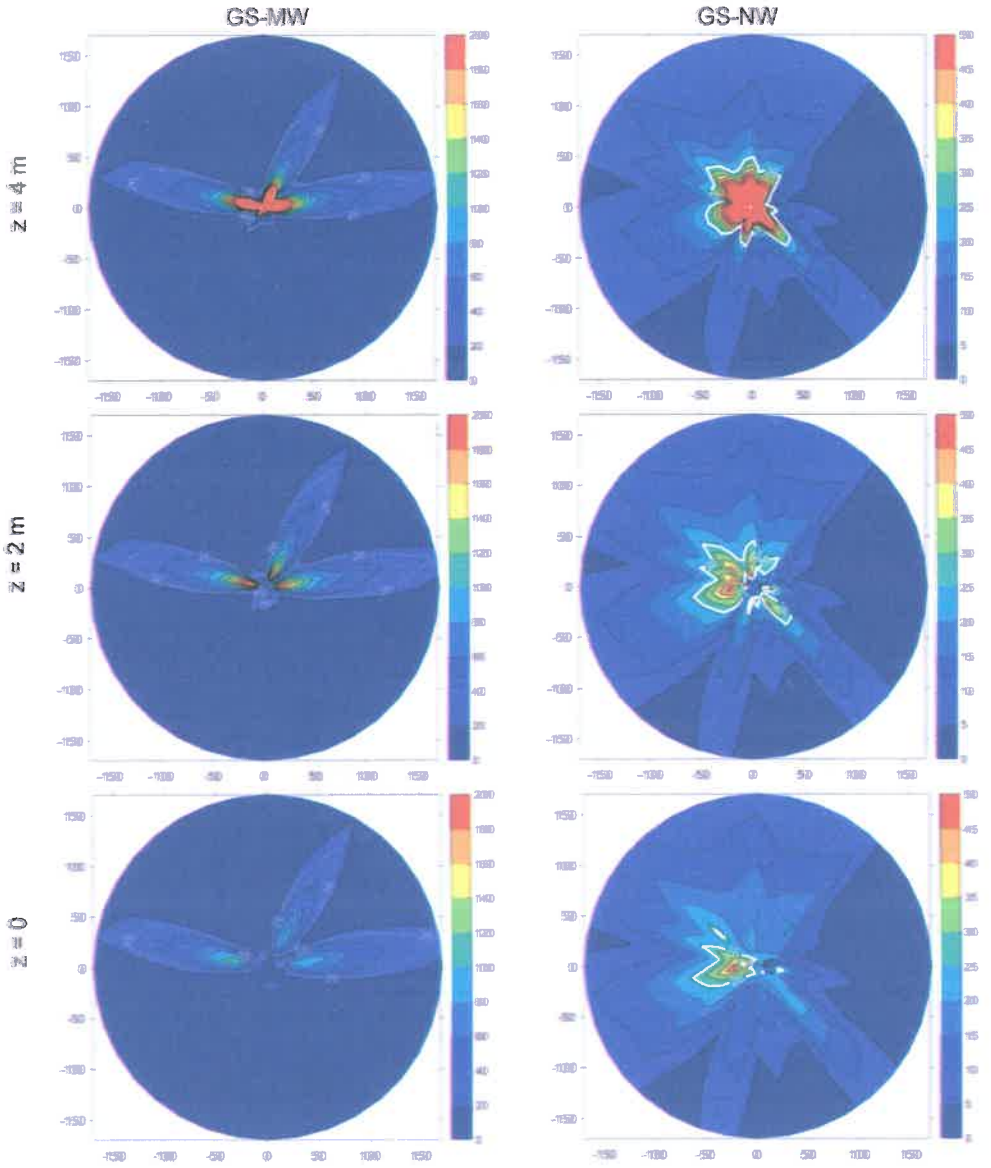

Figure 5:

Modeled maximum benzene concentrations for GS-MW and GS-NW at three different elevations *z*. The *x*- and *y*-axes indicate horizontal coordinates in meters. The color indicates benzene levels in units of $\mu g/m^3$. Left column: time series of benzene emission rates were used. Right column: average benzene emission rate was used in the modeling. The white isoline indicates OEHHA 's acute REL of 26 $\mu g/m^3$ = 8 ppb.

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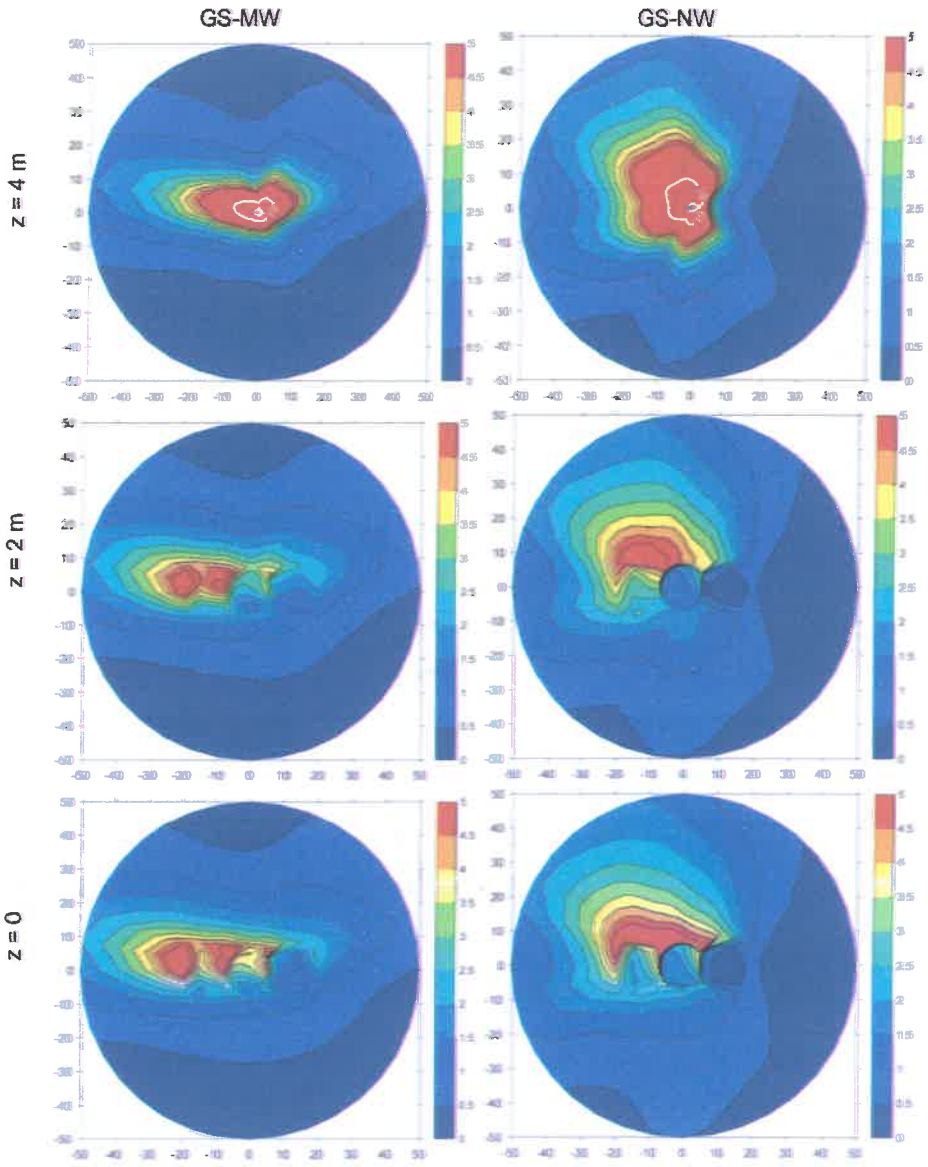

Figure 6:

Modeled average benzene concentrations for GS-MW and GS-NW at three different elevations *z*. The *x*- and *y*-axes indicate horizontal coordinates in meters. The color indicates benzene levels in $\mu g/m^3$ and the white isoline the MRL of 19 $\mu g/m^3$ = 6 ppb.

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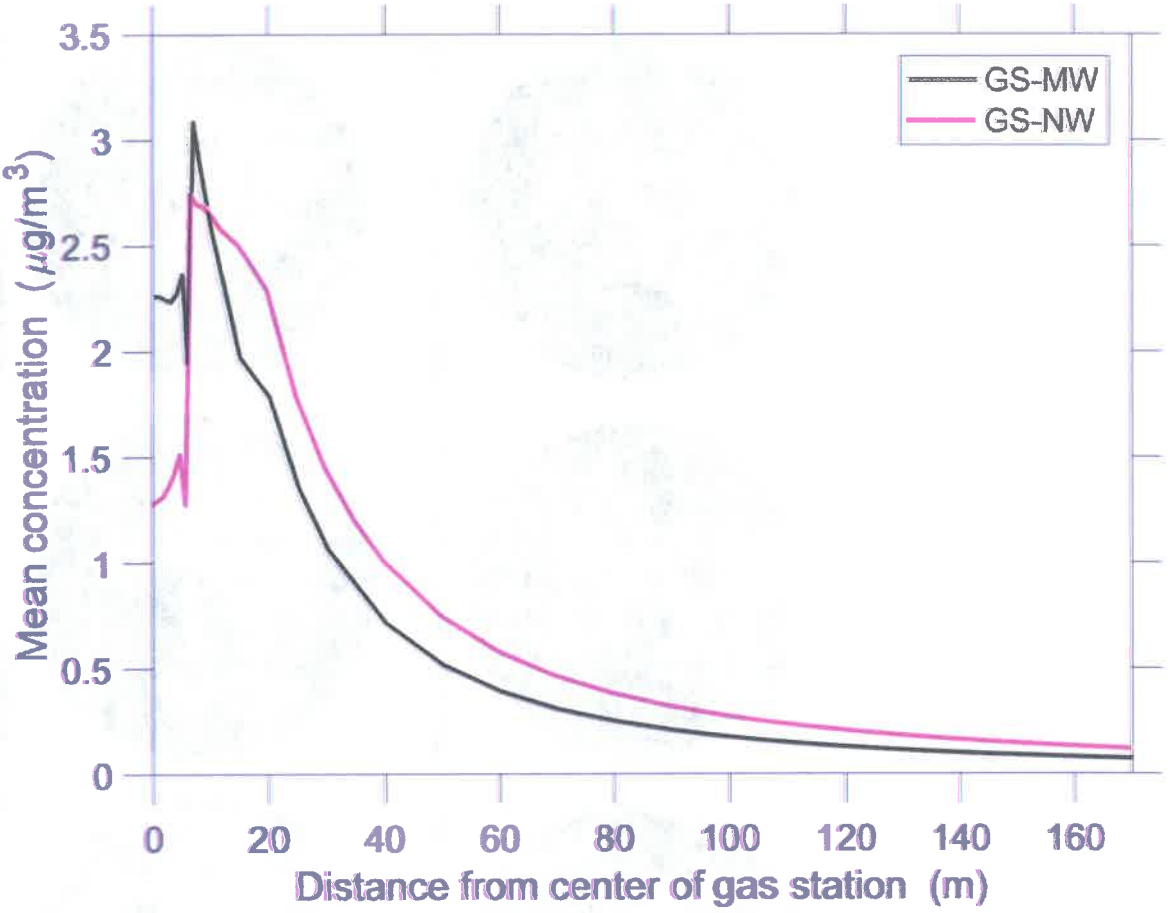

Figure 7:
Mean benzene concentrations as a function of distance from the center of the gas stations.

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Table 1:

Benzene exposure limits, to which we compared simulation results. For unit conversion, we assumed a temperature of 25°C, i.e., 1 ppm = 3,194 μg/m^3.[27]

Agency	Name	Value (ppb)	Value (μg/m^3)	Exposure duration
California Office of Environmental Health Hazard Assessment (OEHHA)	REL	8	26	1 hour
American Industrial Hygiene Association (AIHA)	ERPG-1	50	159,700	1 hour
AIHA	ERPG-2	150	479,100	1 hour
AIHA	ERPG-3	1,000	3,194,000	1 hour
Agency for Toxic Substances and Disease Registry (ATSDR)	MRL	6	19	14 to 364 days

ERPG = Emergency Response Planning Guidelines. The primary focus of ERPGs is to provide guidelines for short-term exposures to airborne concentrations of acutely toxic, high-priority chemicals.

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Table 2:

Summary of gas station characteristics and vent emissions.

	GS-MW	GS-NW	Units
Sales volume $\dot{V}_{sales}$	450,000	700,000	gal/month
Volumetric flow rates (of gasoline vapor/air mixture)			
Mean $\bar{Q}$	7.9	15.4	L/min
Median (IQR) of 60-min average	6.1 (1.9, 10.9)	16.0 (12.7, 18.4)	L/min
Maximum of 60-min average	250	32.1	L/min
Vent emission factor EF_{vent}	1.4	1.7	lb/kgal
Mass flow rates of gasoline (w/o air)			
Mean $\bar{\dot{m}}_{gas}$	0.39	0.76	kg/hr
Maximum of 60-min average	12.3	1.6	kg/hr
Correlation coefficient			
between Q and p	0.58	0.85	-

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Table 3:

Mean benzene emission rates $\dot{m}_{benz}$ for the two gas stations.

Emission source	Benzene emissions (mg/s)	
Gas station	GS-MW	GS-NW
Vent pipe	0.80	1.55
Spillage	0.39	0.65
Refueling	0.41	0.69
Hose permeation	0.06	0.10
Total	1.67	2.90

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CANCER

Residential proximity to petrol stations and risk of childhood leukemia

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Abstract

Petrol stations emit benzene and other contaminants that have been associated with an increased risk of childhood leukemia. We carried out a population-based case-control study in two provinces in Northern Italy. We enrolled 182 cases of childhood leukemia diagnosed during 1998–2019 and 726 age- and sex-matched population controls. We geocoded the addresses of child residences and 790 petrol stations located in the study area. We estimated leukemia risk according to distance from petrol stations within a 1000 m buffer and amount of supplied fuel within a buffer of 250 m from the child's residence. We used conditional logistic regression models to approximate risk ratios (RRs) and 95% confidence intervals (CIs) for associations of interest, adjusted for potential confounders. We also modeled non-linear associations using restricted cubic splines. In secondary analyses, we restricted to acute lymphoblastic leukemia (ALL) cases and stratifed by age (<5 and ≥5 years). Compared with children who lived≥1000 m from a petrol station, the RR was 2.2 (95% CI 0.5–9.4) for children living<50 m from nearest petrol station. Associations were stronger for the ALL subtype (RR=2.9, 95% CI 0.6–13.4) and among older children (age≥5 years: RR=4.4, 95% CI 0.6–34.1; age<5 years: RR=1.6, 95% CI 0.1–19.4). Risk of leukemia was also greater (RR=1.6, 95% CI 0.7–3.3) among the most exposed participants when assigning exposure categories based on petrol stations located within 250 m of the child's residence and total amount of gasoline delivered by the stations. Overall, residence within close proximity to a petrol station, especially one with more intense refueling activity, was associated with an increased risk of childhood leukemia, though associations were imprecise.

Keywords Childhood leukemia · Benzene · Petrol station · Case-control study · Refueling activity

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Abbreviations

AIC	Akaike Information Criterion
AIEOP	Associazione Italiana Ematologia Oncologia Pediatrica
ARPAE	Regional Agency for Environmental Protection and Energy
CI	Confidence interval
GIS	Geographical Information System
ICD-9	International Classification of Diseases, 9th Edition
μT	MicroTesla
RR	Relative risk

Introduction

Leukemia is the most common cancer among children. Childhood leukemia accounts for approximately 27% of all childhood cancers in the United States and 30-35% in Europe and Asia (e.g., Ireland, France, Germany, China) [1]. Acute lymphoblastic leukemia (ALL) is the most commonly diagnosed childhood cancer worldwide [2] and is more frequent in European and Hispanic populations [3]. According to data from populations covered by high-quality cancer registries, the incidence of ALL has been estimated at 46.4 per 1 million children (i.e., one third of all new diagnoses) [3].

Though epidemiological studies have identified several potential genetic and environmental risk factors for childhood leukemia, uncertainties still surround their causes, the presence of dose-response relations, and the presence of thresholds for environmental contaminants to increase disease risk [4–16]. Air pollution generated by motorized traffic and industrial sources is one of these putative risk factors [17]. The International Agency for Research on Cancer (IARC) classified outdoor air pollution and particulate matter from outdoor air pollution as carcinogenic to humans (IARC Group 1), based on sufficient evidence of carcinogenicity in humans and experimental animals and strong mechanistic evidence. Among traffic pollutants, there is benzene, a designated Group I carcinogen by the IARC that is associated with adult leukemia and lymphomas [18–20], and there is consistent evidence indicating that benzene exposure increases the risk of childhood leukemia [21]. A harmful effect of benzene is still conceivable even at the much lower air pollution levels documented in western countries during the last decades [22].

In urban areas, the highest concentrations of atmospheric pollutants have been found near high-traffic roads. However, since pollutants are emitted during fuel refueling activities and petrol leaks, petrol stations are also considered potential sources of exposure to air pollutants, such as 1,3-butadiene and in particular benzene [23, 24]. Primary petrol compounds enter the air of gas stations due to high evaporation of gasoline. A recent study evaluated the concentration of these compounds in the ambient air of gas stations [23] and found benzene concentrations ranged from about 1 to more than 5 ppm, higher than the recommended exposure limit, 0.1 ppm time weighted average, by The National Institute for Occupational Safety and Health [25, 26].

Some studies have assessed the contribution of petrol refueling stations to the pollutants concentrations in their wider vicinity, finding higher levels of pollutants not only close to the pumps, but also throughout the whole service station area and some distance beyond (50–100 m) [24, 27–30], depending on station fueling activity and meteorological conditions [29, 31]. To our knowledge, however, no studies have considered the station's activity when estimating effects of exposure on disease risk, despite the levels of pollutant concentrations around stations being strongly dependent on the amount of fuel delivered [31, 32].

We assessed the extent to which residential exposure to gasoline service stations was associated with risk of childhood leukemia in Northern Italy, by considering both distance from the stations located within proximity of the residence and gas station activity. We also critically summarized existing epidemiologic evidence on the risk of childhood leukemia following long-term exposure to gasoline service stations, updating a previous meta-analysis on this topic [33].

Methods

Case and control selection

Following the Ethics Committee approval [14, 16, 34], we have identified all newly-diagnosed cases of childhood leukemia (ICD-9 codes 204–208) in the 0–14 aged population of Modena and Reggio Emilia, two provinces in Northern Italy (population around 1,200,000), from 1998 to 2019. Details for case and control identification have been described elsewhere in detail [35]. Briefly, we identified the cases through the Italian hospital-based registry of childhood malignancies, available from the Italian Association of Pediatric Emato-Oncology (AIEOP) and capturing all cancer cases arising in Italy [36]. The referent population included four children for each case, matched on sex, year of birth, and province of residence during the year of diagnosis, randomly selected among those enrolled in the National Health Service directory of the Modena and Reggio Emilia provinces, where all residents are compulsorily registered.

We collected data on residential address at time of diagnosis for cases from the AIEOP databased, and in the corresponding year for their matched controls using the historical population database of the National Health Service. We geocoded the home buildings within a Geographical Information System (GIS) using Arc-GIS software (version 9.2, ESRI, Redlands, CA 2006). The satellite coordinates of the residences were retrieved using methodology based on an official geocoding database made available by the Modena and Reggio Emilia Province and, for addresses not included in the database, through the Google Earth App or direct *in loco* measurement using a portable GPS device (GPSmap 60CSx, Garmin Int. Corp., Olathe, KS) [16, 34, 35]. To identify petrol stations, we obtained information from the Trade Observatory of the Emilia-Romagna Region about all the 859 facilities located in the territory of Modena and

Reggio Emilia provinces. All stations were georeferenced in the GIS through Google Earth or *in loco* measurements, focusing on the exact position of the pumps whenever possible, or to the geometric center of the station area.

Exposure to petrol station

We assessed exposure to petrol stations in two ways. First, petrol station exposure was expressed as distance from home to the nearest petrol station divided into categories with the following cut points: 50, 200, 500, 1000 and over 1000 m.

Secondly, we estimated the activity of each station in terms of fuel sold over an entire year, and we assessed child exposure to all stations located up to 1000 m around the residences. For this purpose, through record-linkage with the database provided by the Trade Observatory of the Emilia-Romagna Region, we retrieved the average daily quantity (L/day) of fuel sold in each petrol station in the year of leukemia diagnosis for cases and their matched controls. The total average daily quantity of fuel supplied by all the petrol stations located within 250 m buffer around residence was computed for each study participant. For this analysis, we then categorized petrol station exposure based on the amount of supplied fuel as follows: 0 = no petrol stations within the 250 m-buffer; 1 = fuel supply until 149 L/day within the 250 m-buffer; 2 = fuel supply ≥ 150 until 999 L/day within the 250 m-buffer; and 3 = fuel supply ≥ 1000 L/day within the 250 m-buffer.

Confounders

We considered several potential confounders in multivariable analysis. We modeled outdoor air concentration of PM_{10} at the residence of each child, using the CAlifornia LINE Source Dispersion Model, version 4 (CALINE4 - Sacramento CA, Dept. of transportation, Division of New Technology and Research, 1989), a line source air quality model, based on vehicular traffic flow parameters and meteorological data [34]. We also modeled the magnetic fields generated by the 132 + kV power lines possibly located close to the child's residence in the study territory using a methodology previously validated and explained in detail [16, 37]. We also included as adjustment factor to the multivariable analysis the presence of indoor transformer stations in the building of residence, using information about high-voltage power line net and transformer stations in the two study provinces made available by the Emilia-Romagna Regional Agency for Environmental Prevention and Energy (ARPAE). We then determined the urban and arable crop by calculating the percentage of the land use type in proximity to each geocoded home, based on a circular area with a radius of 100 m around the residence of each child according to the Land Use Map 2014 for both Modena and Reggio Emilia provinces [14, 38, 39].

We also collected information on socio-demographic variables maternal age and ethnicity using birth certificate data provided by the Local Health Authorities of Modena and Reggio Emilia, and parental annual income for the index year by the Italian Revenue Agency of the Ministry of Finance.

Data analysis

We estimated the risk ratio (RR) of childhood leukemia in relation to categorical distance (categorized as < 20, 50 - <200, 200 - <500, 500 - <1000 and ≥ 1000 m) to the nearest petrol station by computing the disease odds ratio and its 95% confidence interval (CI) using conditional logistic regression models, with matching for age, sex and province of residence. In the multivariable models, we included as potential confounders the following variables: modeled PM_{10} concentrations, calculated ELF-MF (categorized as < 0.1, 0.1 - <0.2, 0.2 - <0.4 and ≥ 0.4 μT) [16], presence of electric transformer rooms near the building of residence (categorized as < 5 m, ≥ 5 - <10 m, ≥ 10 - <20 m, - ≥20 m), percentage of urban area providing information related to the type of neighborhood within the 100 m-buffer around the residence [35], and percentage of arable crops within the 100 m-buffer around the residence (continuous) as related to pesticide exposure [14]. In addition, in a subgroup analysis for participants having additional information available, we added maternal ethnicity (categorized as white, Black or Asian), father income, and maternal age at delivery (continuous). We also conducted subgroup analyses by child's age of diagnosis (< 5 and ≥ 5 years), and restricted to ALL, the only cancer subtype with sufficient numbers for meaningful analyses. We used restricted cubic splines to model the shape of the association. We selected the number of knots using the Akaike Information Criterion (AIC) and the knot-placement method [40] to assess the association between residential distance from the nearest petrol station and RR of leukemia through a nonlinear model based on restricted cubic splines, using three knots at fixed distances (50, 200, and 500 m) and ≥ 1000 m as the reference.

Updated systematic review and meta-analysis

We performed a systematic literature search (PROSPERO registration no. CRD42023402919) using online databases PubMed/MEDLINE, Web of Science and EMBASE from inception up to April 3, 2023 according the PRISMA guidelines [41]. We also used citation chasing methods namely backward and forward reference list scanning to retrieve additional eligible papers. Two authors (TF and MV) performed the screening of title/abstract and then of the full-text

with the help of a third author (MM) to solve disagreement. According PECOS (population, exposure, comparison, outcome and study design) statement, we searched all observational studies that have investigated the risk of childhood leukemia in relation to exposure to petrol station using either proximity of children residence or modelled exposure. We used keywords related to 'petrol' or 'gasoline station' and 'childhood' or 'infant leukemia'. Details of literature search are reported in Supplemental Table S1. We assessed the risk of bias (RoB) of included studies using the Newcastle - Ottawa quality assessment scale (NOS). Details of criteria for study evaluation are reported in Supplemental Table S2. Two authors independently performed the RoB assessment (MM and TF), with discrepancies solved based on the review of a third author (MV). We carried out a highest versus lowest exposure meta-analysis of all eligible studies using a random-effects model, and we performed stratified analysis according exposure assessment method (i.e., using questionnaires or georeferencing data). Finally, we assessed potential for publication bias using a funnel plot and Egger's test.

Results

The study enrolled 183 incident cases of childhood leukemia, of which 148 were cases of lymphoblastic leukemia ALL, and 732 matched controls. We excluded one case (alongside the respective controls) and two additional eligible controls due to missing residential information. The final analysis included 182 cases (98 males and 84 females) and 726 age-and sex-matched population controls. The average age at diagnosis was 6.2 years (standard deviation: 3.9), with corresponding median value of 5.7 years (interquartile range-IQR: 3.0–9.0). The median values (IQR) for cases and controls of the adjustment variables were: fuel supply (L/day) within the 1000 m-buffer = 804 (0-2454) and 868 (0-2255); PM_{10} ($\mu g/m^3$) = 4.9 (2.4–8.3) and 4.6 (2.0-7.8); urban area within the 100 m-buffer around the residence = 0.6 (0.4–0.8) and 0.6 (0.4–0.8); arable crops within the 100 m-buffer around the residence = 0.0 (0.0-0.1) and 0.0 (0.0-0.1), respectively. Distribution for ELF-MF was categorized as follows: <0.1 μT for 180 cases and 725 controls; 0.1 - <0.2 μT for 0 cases and 1 control; 0.2 - <0.4 μT for 1 case and 0 controls and ≥0.4 μT 1 case and 0 controls. Electric transformer rooms near the building of residence were categorized for cases and controls as follows: ≥20 m for 178 cases and 709 controls; ≥10 and < 20 m for 2 cases and 10 controls; ≥5 and < 10 m for 1 case and 4 controls; <5 m for 1 case and 3 controls. Data on the distribution of cases and controls by residential proximity to the nearest petrol station and by exposure category are reported in Table 1.

RRs for leukemia risk according to residential proximity to the nearest petrol station are shown in Table 2. Compared to those living ≥ 1000 m, RRs for children living < 50 m were 2.3 (95% CI 0.5–10.0) and 2.2 (95% CI 0.5–9.4) in crude and adjusted analyses, respectively. Corresponding RRs were stronger in analyses confined to ALL cases: 2.8 (95% CI 0.6–13.2) and 2.9 (95% CI 0.6–13.4), respectively.

The age-stratified results are presented in Table 2. Focusing on children whose residence was extremely close to petrol station (< 50 m), we found an increased risk among older children (age ≥ 5 years) of 4.3 (95% CI 0.6–32.4) compared with 1.2 (95% CI 0.1–12.2) among younger children

Table 1 Distribution of study population (cases and controls) by distance from residence to the nearest petrol station and category of exposure to air pollutants emissions from the nearby station

	All leukemias			Acute lymphoblastic leukemia (ALL)		
	All subjects	<5 years	≥5 years	All subjects	<5 years	≥5 years
Distance to the nearest petrol station, meters						
≥1000 (Referent)	48/185	23/83	25/102	38/145	19/63	19/82
500–< 1000	53/214	22/93	31/121	44/177	17/72	27/105
200–< 500	58/242	25/113	33/129	47/202	20/94	27/108
50–< 200	20/80	12/40	8/40	16/62	9/33	7/29
<50	3/5	1/3	2/2	3/4	1/2	2/2
All subjects	182/726	83/332	99/394	148/590	66/264	82/326
Class of exposure[a]						
0 (Referent)	146/608	64/278	82/330	118/497	51/219	67/278
1	4/12	2/6	2/6	4/11	2/5	2/6
2	21/76	14/34	7/42	18/59	11/28	7/31
3	11/30	3/14	8/16	8/23	2/12	6/11
All subjects	182/726	83/332	99/394	148/590	66/264	82/326

[a]Exposure category: 0 = no petrol stations within 250 m-buffer; 1 = fuel supply until 149 L/day within 250 m-buffer; 2 = fuel supply ≥ 150 until 999 L/day within 250 m-buffer; 3 = fuel supply ≥ 1000 L/day within 250 m-buffer

Table 2 Distance from residence to the nearest petrol station and risk of childhood leukemia

Distance to the nearest petrol station, m	All subjects RR (95% CI)	Age < 5 years RR (95% CI)	Age ≥ 5 years RR (95% CI)
Bivariate model[a]			
All leukemias			
≥ 1000 (Referent)	1.0	1.0	1.0
500–< 1000	1.0 (0.6–1.6)	0.9 (0.4–1.6)	1.1 (0.6–2.0)
200–< 500	0.9 (0.6–1.4)	0.8 (0.4–1.6)	1.1 (0.6–2.0)
50–< 200	1.0 (0.5–1.8)	1.1 (0.5–2.4)	0.9 (0.4–2.1)
< 50	2.3 (0.5–10.0)	1.2 (0.1–12.2)	4.3 (0.6–32.4)
ALL			
≥ 1000 (Referent)	1.0	1.0	1.0
500–< 1000	1.0 (0.6–1.6)	0.7 (0.3–1.6)	1.2 (0.6–2.3)
200–< 500	0.9 (0.6–1.5)	0.7 (0.4–1.5)	1.2 (0.6–2.3)
50–< 200	1.0 (0.5–2.0)	0.9 (0.4–2.3)	1.2 (0.4–3.1)
< 50	2.8 (0.6–13.2)	1.6 (0.1–18.8)	4.7 (0.6–36.0)
Multivariable model[b]			
All leukemias			
≥ 1000 (Referent)	1.0	1.0	1.0
500–< 1000	0.9 (0.6–1.5)	0.8 (0.4–1.6)	1.1 (0.6–2.1)
200–< 500	0.9 (0.5–1.5)	0.8 (0.4–1.7)	1.1 (0.5–2.2)
50–< 200	1.0 (0.5–1.9)	1.0 (0.4–2.5)	1.0 (0.4–2.7)
< 50	2.2 (0.5–9.4)	1.1 (0.1–11.4)	3.6 (0.5–27.6)
ALL			
≥ 1000 (Referent)	1.0	1.0	1.0
500–< 1000	1.0 (0.6–1.7)	0.7 (0.3–1.6)	1.3 (0.6–2.7)
200–< 500	1.0 (0.6–1.7)	0.7 (0.3–1.7)	1.4 (0.6–2.9)
50–< 200	1.2 (0.5–2.5)	0.9 (0.3–2.5)	1.7 (0.6–5.0)
< 50	2.9 (0.6–13.4)	1.6 (0.1–19.4)	4.4 (0.6–34.1)

ALL acute lymphoblastic leukemia

[a]Matched on sex, age and province of residence

[b]Adjusted for fuel supply within the 1000 m-buffer, PM_{10}, ELF-MF from high-voltage power lines, indoor transformer stations, urban area and arable crops

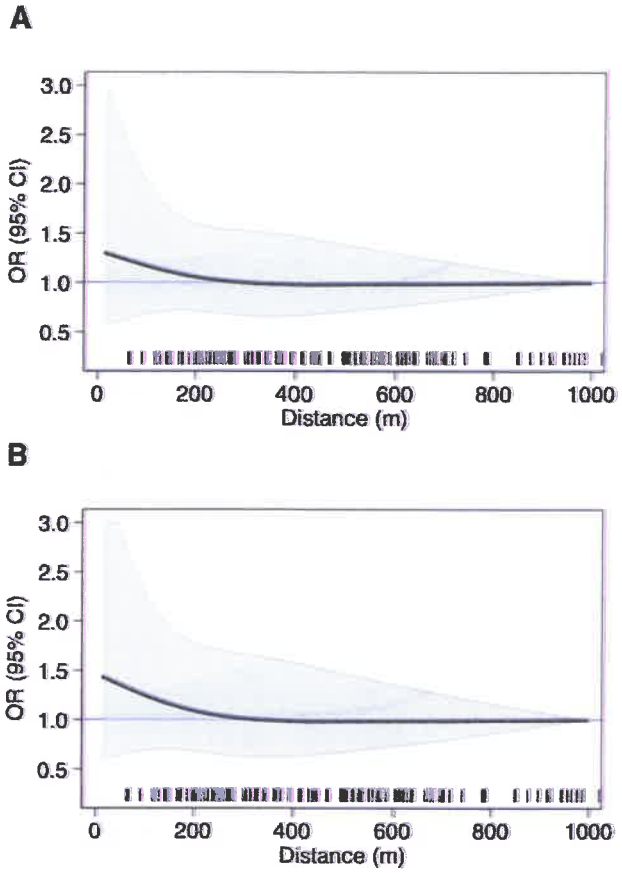

Fig. 1 Spline regression analysis assessing the risk ratio of childhood leukemia according to distance of child's residence from the closest petrol station. Restricted cubic spline with three knots at fixed distances (50, 200, and 500 m). **A** Bivariate model; **B** Multivariable model

(age < 5 years) in the bivariate model. When limiting the analysis to ALL cases in the bivariate model, RRs were 1.6 (95% CI 0.1–18.8) among younger children and 4.7 (95% CI 0.6–36.0) among older children. In multivariable analysis, we found relatively similar RRs for all leukemia cases and as well as ALL cases (Table 2).

In spline regression analyses for overall leukemia (Fig. 1) and ALL (Fig. 2), residential proximity to a petrol station was positively associated with leukemia risk only within close proximity of the station (< 50 m): RR = 1.4 (95% CI 0.6–2.9).

When we considered all the petrol stations located within 250 m of the child's residence, as well as the total amount of gasoline sold by the station the year before the index year, RRs for leukemia were 1.6 (95% CI 0.8–3.2) and 1.6 (95% CI 0.7–3.3) in bivariate and multivariable analyses, respectively, for the highest category of exposure (Table 3), with no indication of monotonic relation across the exposure categories. Corresponding RR estimates for ALL were 1.5 (0.6–3.5) and 1.5 (0.6–3.8). Older children (age ≥ 5 years) living inside buffer of 250 m with petrol stations that have sold more than 1000 L/day had a higher risk of leukemia overall (bivariate and multivariable models: RR = 2.1, 95% CI 0.8–5.1 and RR = 2.4, 95% CI 0.9–6.1, respectively), and of ALL (RR = 2.4, 95% CI 0.8–7.2 in the bivariate model and RR = 3.4, 95% CI 1.0-11.1 in the multivariable model). In the remaining categories of exposure, there was no clear association, neither evidence of dose-response trends. Sensitivity analyses among subjects with complete data on demographic variables, specifically maternal ethnicity and paternal income, showed similar though less precise results (Supplemental Table S3) when compared with the overall analysis and the analysis among the same subgroup without adjusting for these additional variables.

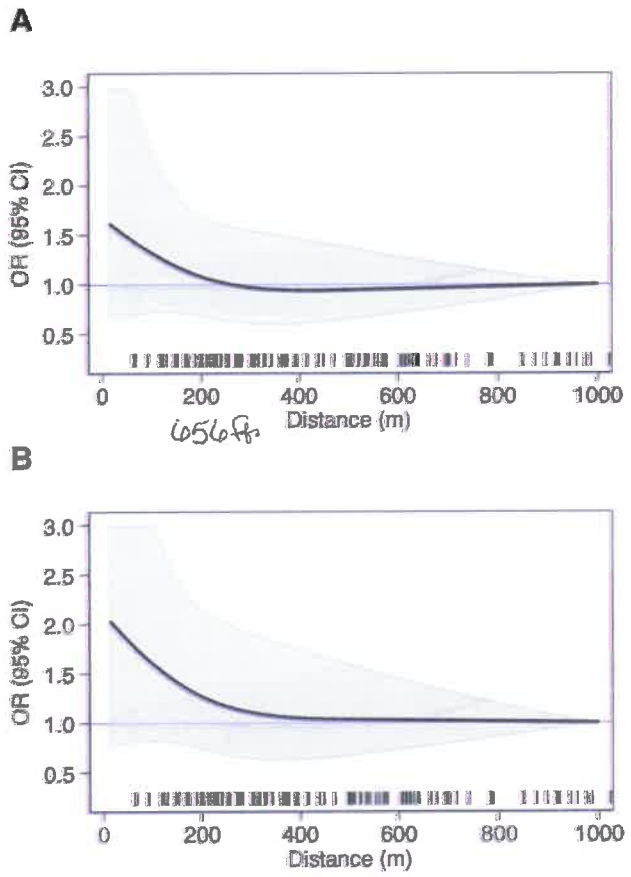

Fig. 2 Spline regression analysis assessing the risk ratio of childhood acute lymphoblastic leukemia (ALL) according to distance of child's residence from petrol station. Restricted cubic spline with three knots at fixed distances (50, 200, and 500 m). **A** Bivariate model; **B** Multivariable model

Table 3 Class of exposure within a 250-meter buffer from petrol stations considering gasoline distributed, and risk of childhood leukemia

Class of exposure	All subjects RR (95% CI)	<5 years RR (95% CI)	≥5 years RR (95% CI)
Bivariate model[a]			
All leukemias			
0 (Referent)	1.0	1.0	1.0
1	1.4 (0.4–4.3)	1.6 (0.3–8.4)	1.2 (0.2–6.2)
2	1.2 (0.7–1.9)	1.8 (0.9–3.6)	0.7 (0.3–1.6)
3	1.6 (0.8–3.2)	0.9 (0.2–3.2)	2.1 (0.8–5.1)
ALL			
0 (Referent)	1.0	1.0	1.0
1	1.5 (0.5–4.8)	2.0 (0.4–11.0)	1.3 (0.3–6.4)
2	1.3 (0.7–2.3)	1.7 (0.8–3.8)	1.0 (0.4–2.4)
3	1.5 (0.6–3.5)	0.6 (0.1–3.0)	2.4 (0.8–7.2)
Multivariable model[b]			
All leukemias			
0 (Referent)	1.0	1.0	1.0
1	1.4 (0.4–4.4)	1.5 (0.3–7.9)	1.4 (0.3–7.5)
2	1.2 (0.7–2.1)	2.0 (1.0–4.1)	0.7 (0.3–1.7)
3	1.6 (0.7–3.3)	0.8 (0.2–3.1)	2.4 (0.9–6.1)
ALL			
0 (Referent)	1.0	1.0	1.0
1	1.6 (0.5–5.0)	1.8 (0.3–10.1)	1.5 (0.3–8.1)
2	1.4 (0.8–2.5)	2.0 (0.9–4.5)	1.0 (0.4–2.6)
3	1.5 (0.6–3.8)	0.6 (0.1–3.0)	3.4 (1.0–11.1)

ALL acute lymphoblastic leukemia

[a]Matched on sex, age and province of residence

[b]Adjusted for PM_{10}, ELF-MF from high-voltage power lines, indoor transformer stations, urban area and arable crops

[c]Class of exposure: 0 = no petrol stations within the 250 m-buffer; 1 = fuel supply until 149 L/day within the 250 m-buffer; 2 = fuel supply ≥ 150 until 999 L/day within the 250 m-buffer; 3 = fuel supply ≥ 1000 L/day within the 250 m-buffer

In the systematic review and meta-analysis, our literature search retrieved 31 records after removal of duplicates. We then excluded 20 studies after title/abstract screening and an additional six studies after full-text evaluation as they assessed benzene exposure during parental occupation only (n = 1), petrol station exposure was not evaluated (n = 2), or were conference abstract (n = 1) or commentaries (n = 2) (Supplemental Figure S2). We retrieved one additional study [42] through citation chasing, leading to a total of six studies for analysis not including the present study. Characteristics of retrieved studies are reported in Table 4. The ages of the study populations for all in the range 0–14 years, with dates of diagnosis ranging from 1985 to 2019. All studies had a case-control design, including a case-cohort study [33]. Three studies investigated exposure to petrol station using questionnaire [42–44], although one study assessed exposure to both petrol station and car repair garage [43]. One study validated questionnaire-based exposure using georeferencing data [44]. The three remaining studies used georeferencing data for exposure assessment [33, 45, 46], one in particular through evaluation of petrol station density (number of stations per km^2). Results of the bias assessment are reported in Supplemental Table S4.

Meta-analysis of the results of the aforementioned six studies plus those generated by the present study are reported in Fig. 3, showing a summary RR of 1.66 (95% CI 1.14–2.41). Analyses stratified by modality of exposure assessment yielded similar results, with lower precision for studies based on questionnaire data (Supplemental Figure S3). Sensitivity analysis excluding the study assessing also proximity of car repair garages showed consistent results, yielding a summary RR of 1.50 (95% CI 1.05–2.15) (Supplemental Figure S4). The sensitivity analysis restricted to four "high-quality" studies (NOS score ≥ 8) still showed an elevated disease risk with RR = 1.80 (95% CI 1.37–2.38)

Table 4 Characteristics of studies included in the systematic review

Reference	Design	Region	Cases/non cases	Age (years)	Diagnosis	Assessment	Risk estimate	Adjusting factors
Abdul Rahman 2008 [42]	Case-control	Klang Valley, Malaysia	128/128	<15	2001–2007 acute leukemia	Questionnaire: distance of residence at the time of diagnosis from a petrol station ≤1 km vs. >1 km	OR: 0.84 (95% CI 0.50–1.41)	Crude
Brosselin 2009 [44]	Case-control	France	765/1681	<15	2003–2004 acute leukemia	Questionnaire: Ever (vs. never) lived in proximity (not described in detail) to a petrol station and/or automotive repair garage Data validated using georeferencing data	Repair garage: OR: 1.4 (95% CI 1.0–2.0) Petrol station: OR: 1.9 (95% CI 1.2–3.0) Any: OR: 1.6 (95% CI: 1.2–2.2) Any by period: Childhood: OR: 1.3 (95% CI 0.9–1.9) During pregnancy: OR 1.4 (95% CI 1.0–2.1) Both: OR: 1.7 (95% CI: 0.9–3.4)	Age, sex, number of children < 15 years living in the household, and stratification variables
					ALL		Repair garage: OR: 1.5 (95% CI: 0.9–2.3) Petrol station: OR: 2.0 (95% CI: 1.0–4.0) Any: OR: 1.6 (95% CI 1.9–2.3) Both: OR: 1.8 (95% CI 0.9–3.5)	
					AML		Repair garage: OR: 0.8 (95% CI: 0.2–2.5) Petrol station: OR: 2.5 (95% CI: 0.7–8.8) Any: OR: 1.1 (95% CI 0.5–2.5) Both: OR: 0.8 (95% CI 0.1–6.2)	
Harrison 1999 [46]	Case-control	West Midlands, UK	130/251	0–15	1990–1994 acute leukemia	Georeferencing data: petrol station proximity (≤100 m vs. >100 m)	OR: 1.99 (95% CI 0.73–5.43) IR: 1.48 (95% CI 0.65–2.93)	Crude

Table 4 (continued)

Reference	Design	Region	Cases/non cases	Age (years)	Diagnosis	Assessment	Risk estimate	Adjusting factors
Mazzei 2022 [33]	Case-cohort	Swiss	1880/18,800	0–15	1985–2015	Georeferencing data: petrol station distance < 50 m vs. ≥500 m	OR: 1.08 (95% CI 0.46–2.51)	Age- and sex-matched. Adjusted for NO_2, distance to the nearest highway, socio-economic position of the immediate neighborhood area, degree of urbanization of the municipality of residence, and years of existence of a general cantonal cancer registry
Steffen 2004 [43]	Case-control	Nancy, Lille, Lyon and Paris, France	280/285	0–14	1995–1999 acute leukemia	Face-to-face interview: vicinity (< 50 m for traffic) of dwellings neighboring including petrol station or repair garage	Childhood: OR: 4.0 (95% CI 1.5–10.3) During pregnancy: OR: 2.2 (95% CI 0.9–5.7)	Age, sex,center, and ethnic origin
					ALL		Childhood: OR: 7.7 (95% CI 1.7–34.3)	
					AML		Childhood: OR: 3.6 (95% CI 1.3–9.9)	
Weng 2009 [45]	Case-control	Taiwan	729/729	0–14	1996–2006 acute leukemia	Petrol station density (n/km^2) in tertiles: T1: ≤0.149 (median 0.065); T2: 0.150–0.395 (0.225); T3: 0.399–2.692 (0.585)	T2 - OR: 1.45 (95% CI 1.06–1.98) T3 - OR: 1.91 (95% CI 1.29–2.82)	Sex, year of birth, year of death, and urbanization level
This study	Case-control	Italy	182/726	0–14	1998–2019 acute leukemia	Georeferencing data: petrol station proximity (≤50 m vs. >1000 m) Modeling using also fuel supply data divided in four categories	Proximity 2.2 (95% CI 0.5–9.4) Modeling 1.6 (95% CI 0.7–3.3)	Age- and sex-matched. Adjusted for PM_{10}, ELF-MF from high-voltage power lines, indoor transformer stations, urban area and arable crop
			148/590		ALL		Proximity 2.9 (95% CI 0.6–13.4) Modeling 1.5 (95% CI 0.6–3.8)	

(Supplemental Figure S5). The funnel plot showed some evidence of publication bias (Supplemental Figure S6).

Discussion

In this study, we examined childhood leukemia risk in relation to proximity to petrol stations. We observed an increased leukemia risk, though imprecise, when assessing exposure as distance from residence (< 50 m) to the nearest petrol station, while the excess risk associated with the intensity of activity of all stations located < 250 m from the residence was not as large. In both analyses no dose-response relation emerged, since a clear excess risk occurred only in the highest exposure category, suggesting the occurrence of a threshold of exposure to the fugitive chemical emissions from fuel pumps, heightening disease risk, mainly based on the distance from the station. This suggests a far higher relevance of close residential proximity to the gas station when compared with 'moderate' proximity and with the overall station refueling activity, in terms of increasing childhood leukemia risk.

While there are no previous studies based on the station activity, to the best of our knowledge, our results on residential distance from petrol station are consistent with three of the four studies carried out on the same topic. In a UK study [46] that estimated childhood cancer risks in relation to proximity to main roads and petrol stations, a slight increase in leukemia risk was found within 100 m from a petrol station (OR = 1.5, 95% CI 0.6–2.9). In a hospital-based case-control study in France [43], residence close to a petrol station or a repair garage during childhood was strongly associated with excess risk of childhood leukemia (OR = 4.0, 95% CI 1.5 to 10.3). This association was even stronger for acute non-lymphocytic leukemia (OR = 7.7, 95% CI 1.7 to 34.3), and was not altered by adjustment for potential confounding factors. In 2009, Brosselin et al. [44] reported results of a large national registry-based case-control study ESCALE in France (2003–2004) indicating a strong positive association between living in a residence adjoining of a garage or petrol station and acute childhood leukemia. However, these studies differed from ours regarding exposure definitions and assessment: while we classified children living within 50 m of a petrol station in the highest exposure category, the English study used a 100 m threshold and the French studies considered only children living close to a gas station, without specifying the exact distance, also assessing exposure through interviews with the children's mothers and thus potentially affected by recall bias [43, 44]. In a case-control study carried out in Malaysia [42], and based on data collected through questionnaires, no association emerged, though the cut point used to refine residential exposure was quite large, not comparable to that used in the other studies including our one and likely inadequate to detect any association (≤ 1 km vs. >1 km from a petrol station).

An additional case-control study [45] revealed an exposure-response relation between petrol station density (per square kilometer), as a marker of traffic-related air pollution, and the risk of leukemia in young children. A nationwide case-control study carried out in Switzerland (during 1985–2015) found evidence of an increased risk of childhood cancer (all diagnoses combined) among children living in close vicinity (< 50 m vs. ≥500 m) of petrol stations [33].

A relevant distinct feature of the present analysis is the integration of data on petrol station activity along with its distance from the child's residence. There is clear evidence from studies of gas station workers that their exposure to air pollutants is positively related to the volume of refueling in petrol stations, as well as the confinement of pollutants in semi-closed spaces of the work place [25, 47–49].

Our results showed a direct relation between residing in close proximity to a petrol station and risk of leukemia, both based on fixed cut points of distance from the stations and modeling exposure through a combination of distance and gas station activity. Associations were strongest for ALL cases in older children (age ≥ 5 years), with a two-fold increased risk for children in the category of major exposure (< 50 m) and a four-fold increased risk among children diagnosed after 5 years. The latter finding might be ascribed to a higher cumulative exposure among older children, due to both their age and to their tendency to spend more time outside. Such excess risk may persist up to 250 m from the gas station also depending on their activity, a plausible finding given the results of air monitoring studies [31, 50].

In this study, we assessed exposure without requiring any active participation by study participants and their families, nor by petrol station personnel, thus avoiding selection and information bias, as individual participation was not needed. We also carried out the exposure assessment in a blinded manner with reference to the case and control status of the participants. A potential limitation of our study is that information on the activity of each petrol station was available during 1998–2017 only. Since our study includes children with diagnoses that occurred from 1998 to 2019, we decided to consider, for the last two years 2018-19 and for each station, the fuel supply corresponding to the most recent year available: 2017. Residual or unmeasured confounding could have also been possible, and some demographic characteristics were unavailable for many study participants as well as complete information about medical imaging procedures [4]. A sensitivity analysis limited to study participants accounting for all potential confounders measured yielded similar results to the analysis carried out without such more comprehensive adjustment, both in this subgroup and in the entire study population, suggesting that the demographic factors for which we lacked complete information were not a major

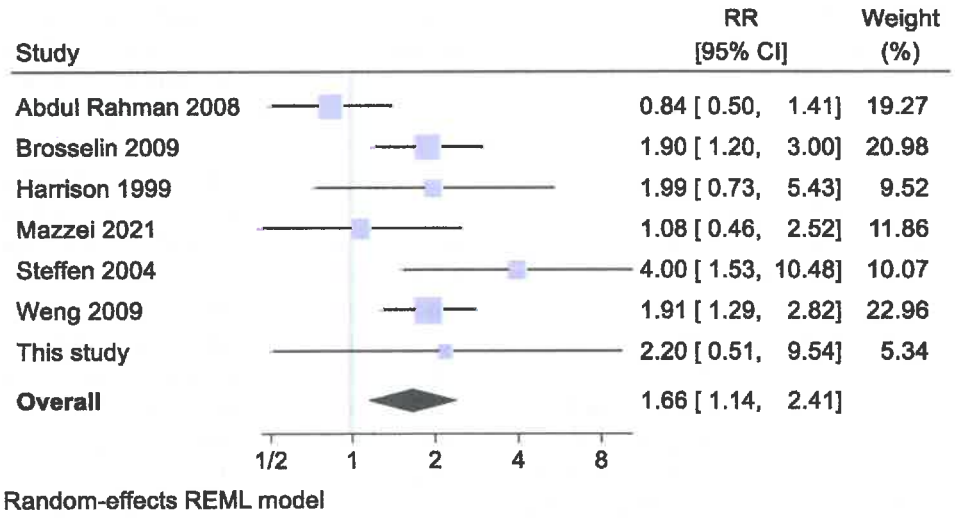

Fig. 3 Forest-plot of the meta-analysis of the association between petrol station exposure and childhood leukemia risk. The area of each grey square is proportional to the inverse of the variance of the estimated log risk ratio (RR) and horizontal lines represent their 95% confidence intervals (Cis). The black diamond represents the combined RR using the random-effects restricted maximum likelihood (REML) model. The solid vertical line represents RR = 1

source of confounding. However, we may not have collected and controlled for all relevant confounders of the associations (e.g., using paternal income as a proxy for household income, or lacking information about exposure to ionizing radiation for diagnostic purposes), and therefore some effect of residual confounding in biasing our results could not be entirely ruled out [51]. We also acknowledge the statistical instability of our risk estimates, due to the very limited number of exposed children, a limitation suggesting caution in interpreting our results, though being consistent with the results from the other comparable studies as reflected by the pooled estimates of the meta-analysis. Finally, we acknowledge the potential for exposure misclassification associated with lack of historical residential stability or time spent at home (e.g., a substantial part of the daytime hours may have spent at a different residence (e.g., grandparents' home, school, or day care). However, while we could not comprehensively assess residential mobility or time spent at one's residence since the study design did not include a direct contact with children's families, residential stability of the study participants was likely to be high (> 70% for all children and > 82% for children less than 5 years) based on data previously ascertained in subgroup of the study population [14, 16, 34].

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10654-023-01009-0.

Authors contribution MV and CM conceived the study. Material preparation and data collection were performed by MM, CM, TF, AB, GP, MC and SC. Data analyses were performed by MM, CM, TF and MV. The first draft of the manuscript was written by MM and CM; and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Declarations

Competing interests LAW is a consultant for AbbVie Inc. and the Gates Foundation, and has received in-kind donations for primary data collection in PRESTO from Swiss Precision Diagnostics and Kindara.com. All other authors declare that they have no competing interests.

Ethics approval This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Modena Ethics Committee (approval number 1103/2020/OSS/AUO/MO).

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References

1. Namayandeh SM, Khazaei Z, Lari Najafi M, Goodarzi E, Moslem A. GLOBAL leukemia in children 0–14 statistics 2018, incidence and mortality and human development index (HDI): GLOBOCAN sources and methods. Asian Pac J Cancer Prev. 2020;21(5):1487–94. https://doi.org/10.31557/APJCP.2020.21.5.1487.
2. Sun Y, Long S, Liu W. Observation of the molecular genetics among children with acute lymphoblastic leukemia: a retrospective study based on the SEER database. Med (Baltim). 2020;99(21):e20009. https://doi.org/10.1097/MD.0000000000020009.

3. Steliarova-Foucher E, Colombet M, Ries LAG, et al. International incidence of childhood cancer, 2001-10: a population-based registry study. Lancet Oncol. 2017;18(6):719–31. https://doi.org/10.1016/S1470-2045(17)30186-9.
4. Onyije FM, Olsson A, Baaken D, et al. Environmental risk factors for childhood acute lymphoblastic leukemia: an umbrella review. Cancers (Basel). 2022;14(2):382. https://doi.org/10.3390/cancers14020382.
5. Karalexi MA, Tagkas CF, Markozannes G, et al. Exposure to pesticides and childhood leukemia risk: a systematic review and meta-analysis. Environ Pollut. 2021;285:117376. https://doi.org/10.1016/j.envpol.2021.117376.
6. Mazzei-Abba A, Folly CL, Kreis C, et al. External background ionizing radiation and childhood cancer: update of a nationwide cohort analysis. J Environ Radioact. 2021;238–239:106734. https://doi.org/10.1016/j.jenvrad.2021.106734.
7. Lupatsch JE, Kreis C, Konstantinoudis G, Ansari M, Kuehni CE, Spycher BD. Birth characteristics and childhood leukemia in Switzerland: a register-based case-control study. Cancer Causes Control. 2021;32(7):713–23. https://doi.org/10.1007/s10552-021-01423-3.
8. Van Maele-Fabry G, Gamet-Payrastre L, Lison D. Household exposure to pesticides and risk of leukemia in children and adolescents: updated systematic review and meta-analysis. Int J Hyg Environ Health. 2019;222(1):49–67. https://doi.org/10.1016/j.ijheh.2018.08.004.
9. Kreis C, Doessegger E, Lupatsch JE, Spycher BD. Space-time clustering of childhood cancers: a systematic review and pooled analysis. Eur J Epidemiol. 2019;34(1):9–21. https://doi.org/10.1007/s10654-018-0456-y.
10. Amoon AT, Crespi CM, Ahlbom A, et al. Proximity to overhead power lines and childhood leukaemia: an international pooled analysis. Br J Cancer. 2018;119(3):364–73. https://doi.org/10.1038/s41416-018-0097-7.
11. Schuz J, Erdmann F. Environmental exposure and risk of childhood leukemia: an overview. Arch Med Res. 2016;47(8):607–14. https://doi.org/10.1016/j.arcmed.2016.11.017.
12. Metayer C, Petridou E, Arangure JM, et al. Parental tobacco smoking and acute myeloid leukemia: the Childhood Leukemia International Consortium. Am J Epidemiol. 2016;184(4):261–73. https://doi.org/10.1093/aje/kww018.
13. Wiemels J. Perspectives on the causes of childhood leukemia. Chem Biol Interact. 2012;196(3):59–67. https://doi.org/10.1016/j.cbi.2012.01.007.
14. Malagoli C, Costanzini S, Heck JE, et al. Passive exposure to agricultural pesticides and risk of childhood leukemia in an italian community. Int J Hyg Environ Health. 2016;219(8):742–8. https://doi.org/10.1016/j.ijheh.2016.09.015.
15. Pedersen C, Johansen C, Schuz J, Olsen JH, Raaschou-Nielsen O. Residential exposure to extremely low-frequency magnetic fields and risk of childhood leukaemia, CNS tumour and lymphoma in Denmark. Br J Cancer. 2015;113(9):1370–4. https://doi.org/10.1038/bjc.2015.365.
16. Malagoli C, Fabbi S, Teggi S, et al. Risk of hematological malignancies associated with magnetic fields exposure from power lines: a case-control study in two municipalities of northern Italy. Environ Health. 2010;9:16. https://doi.org/10.1186/1476-069X-9-16.
17. Kreis C, Heritier H, Scheinemann K, et al. Childhood cancer and traffic-related air pollution in Switzerland: a nationwide census-based cohort study. Environ Int. 2022;166:107380. https://doi.org/10.1016/j.envint.2022.107380.
18. IARC, Benzene. IARC Monographs on the evaluation of carcinogenic risks to humans. Volume 120. France: Lion; 2018.
19. Talbott EO, Xu X, Youk AO, Rager JR, Stragand JA, Malek AM. Risk of leukemia as a result of community exposure to gasoline vapors: a follow-up study. Environ Res. 2011;111(4):597–602. https://doi.org/10.1016/j.envres.2011.03.009.
20. Goldstein BD. Benzene as a cause of lymphoproliferative disorders. Chem Biol Interact. 2010;184(1–2):147–50. https://doi.org/10.1016/j.cbi.2009.12.021.
21. Filippini T, Hatch EE, Rothman KJ, et al. Association between outdoor air pollution and childhood leukemia: a systematic review and dose-response meta-analysis. Environ Health Perspect. 2019;127(4):46002. https://doi.org/10.1289/EHP4381.
22. Ghahremanloo M, Lops Y, Choi Y, Mousavinezhad S. Impact of the COVID-19 outbreak on air pollution levels in East Asia. Sci Total Environ. 2021;754:142226. https://doi.org/10.1016/j.scitotenv.2020.142226.
23. Allahabady A, Yousefi Z, Tahamtan RAM, Sharif ZP. Measurement of BTEX (benzene, toluene, ethylbenzene and xylene) concentration at gas stations. Environ Health Eng Manag. 2022;9(1):23–31. https://doi.org/10.34172/Ehem.2022.04.
24. Jo WK, Oh JW. Exposure to methyl tertiary butyl ether and benzene in close proximity to service stations. J Air Waste Manag Assoc. 2001;51(8):1122–8. https://doi.org/10.1080/10473289.2001.10464339.
25. Chaiklieng S, Suggaravetsiri P, Autrup H. Risk Assessment on benzene exposure among gasoline station workers. Int J Environ Res Public Health. 2019;16(14):2545. https://doi.org/10.3390/ijerph16142545.
26. The National Institute for Occupational Safety and Health (NIOSH). Immediately Dangerous to Life or Health concentrations (IDLH), Benzene. 1994.
27. Duarte-Davidson R, Courage C, Rushton L, Levy L. Benzene in the environment: an assessment of the potential risks to the health of the population. Occup Environ Med. 2001;58(1):2–13. https://doi.org/10.1136/oem.58.1.2.
28. Gonzalez-Flesca N, Vardoulakis S, Cicolella A. BTX concentrations near a stage II implemented petrol station. Environ Sci Pollut Res Int. 2002;9(3):169–74. https://doi.org/10.1007/BF02987484.
29. Sairat T, Homwuttiwong S, Homwutthiwong K, Ongwandee M. Investigation of gasoline distributions within petrol stations: spatial and seasonal concentrations, sources, mitigation measures, and occupationally exposed symptoms. Environ Sci Pollut Res Int. 2015;22(18):13870–80. https://doi.org/10.1007/s11356-015-4615-3.
30. Uren S, Report. SSE/AQ/1085: a pilot study to assess benzene concentration in the vicinity of petrol stations: Department for Environmental Food & Rural Affairs - Air Quality Division. London, UK Division DotE-AQ;1996. Report No.: SSE/AQ/1085.
31. Karakitsios SPD, Kassomenos VK, Pilidis PA. Contribution to ambient benzene concentrations in the vicinity of petrol stations: estimation of the associated health risk. Atmos Environ. 2007;41:1889–902. https://doi.org/10.1016/j.atmosenv.2006.10.052.
32. Sarigiannis DA, Karakitsios SP, Gotti A, Papaloukas CL, Kassomenos PA, Pilidis GA. Bayesian algorithm implementation in a real time exposure assessment model on benzene with calculation of associated cancer risks. Sens (Basel). 2009;9(2):731–55. https://doi.org/10.3390/s90200731.
33. Mazzei A, Konstantinoudis G, Kreis C, et al. Childhood cancer and residential proximity to petrol stations: a nationwide registry-based case-control study in Switzerland and an updated meta-analysis. Int Arch Occup Environ Health. 2022;95(5):927–38. https://doi.org/10.1007/s00420-021-01767-y.
34. Vinceti M, Rothman KJ, Crespi CM, et al. Leukemia risk in children exposed to benzene and PM(10) from vehicular traffic: a case-control study in an italian population. Eur J Epidemiol. 2012. https://doi.org/10.1007/s10654-012-9727-1.

35. Malagoli C, Malavolti M, Costanzini S, et al. Increased incidence of childhood leukemia in urban areas: a population-based case-control study. Epidemiol Prev. 2015;39(4 Suppl 1):102–7.
36. Ferrari A, Dama E, Pession A, et al. Adolescents with cancer in Italy: entry into the national cooperative paediatric oncology group AIEOP trials. Eur J Cancer. 2009;45(3):328–34. https://doi.org/10.1016/j.ejca.2008.12.003.
37. Andreuccetti D. Manuale programma CAMPI version 4.1. Firenze: Consiglio Nazionale delle Ricerche; 2002.
38. Vinceti M, Filippini T, Violi F, et al. Pesticide exposure assessed through agricultural crop proximity and risk of amyotrophic lateral sclerosis. Environ Health. 2017;16(1):91. https://doi.org/10.1186/s12940-017-0297-2.
39. Costanzini S, Teggi S, Bigi A, et al. Atmospheric dispersion modelling and spatial analysis to evaluate population exposure to pesticides from farming processes. Atmosphere. 2018;9(2):38. https://doi.org/10.3390/atmos9020038.
40. Harrell FE. Regression modeling strategies with applications to linear models, logistic regression, and survival analysis. 1 edition ed: Springer Cham; 2001.
41. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. J Clin Epidemiol. 2021;134:178–89. https://doi.org/10.1016/j.jclinepi.2021.03.001.
42. Abdul Rahman HI, Shah SA, Alias H, Ibrahim HM. A case-control study on the association between environmental factors and the occurrence of acute leukemia among children in Klang Valley, Malaysia. Asian Pac J Cancer Prev. 2008;9(4):649–52.
43. Steffen C, Auclerc MF, Auvrignon A, et al. Acute childhood leukaemia and environmental exposure to potential sources of benzene and other hydrocarbons; a case-control study. Occup Environ Med. 2004;61(9):773–8. https://doi.org/10.1136/oem.2003.010868.
44. Brosselin P, Rudant J, Orsi L, et al. Acute childhood leukaemia and residence next to petrol stations and automotive repair garages: the ESCALE study (SFCE). Occup Environ Med. 2009;66(9):598–606. https://doi.org/10.1136/oem.2008.042432.
45. Weng HH, Tsai SS, Chiu HF, Wu TN, Yang CY. Childhood leukemia and traffic air pollution in Taiwan: petrol station density as an indicator. J Toxicol Environ Health A. 2009;72(2):83–7. https://doi.org/10.1080/15287390802477338.
46. Harrison RM, Leung PL, Somervaille L, Smith R, Gilman E. Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. Occup Environ Med. 1999;56(11):774–80. https://doi.org/10.1136/oem.56.11.774.
47. Dehghani M, Fazlzadeh M, Sorooshian A, et al. Characteristics and health effects of BTEX in a hot spot for urban pollution. Ecotoxicol Environ Saf. 2018;155:133–43. https://doi.org/10.1016/j.ecoenv.2018.02.065.
48. Geraldino BR, Nunes RFN, Gomes JB, et al. Evaluation of exposure to toluene and xylene in gasoline station workers. Adv Prev Med. 2021;2021:5553633. https://doi.org/10.1155/2021/5553633.
49. Tongsantia U, Chaiklieng S, Suggaravetsiri P, Andajani S, Autrup H. Factors affecting adverse health effects of gasoline station workers. Int J Environ Res Public Health. 2021;18(19):10014. https://doi.org/10.3390/ijerph181910014.
50. Kwon J, Weisel CP, Turpin BJ, et al. Source proximity and outdoor-residential VOC concentrations: results from the RIOPA study. Environ Sci Technol. 2006;40(13):4074–82. https://doi.org/10.1021/es051828u.
51. Marinacci C, Spadea T, Biggeri A, Demaria M, Caiazzo A, Costa G. The role of individual and contextual socioeconomic circumstances on mortality: analysis of time variations in a city of north west Italy. J Epidemiol Community Health. 2004;58(3):199–207. https://doi.org/10.1136/jech.2003.014928.

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Benzene-induced Cancers: Abridged History and Occupational Health Impact

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Abstract

Benzene-induced cancer in humans was first reported in the late 1920s. Carcinogenesis findings in animals were not reported conclusively until 1979. Industry exploited this "discrepancy" to discredit the use of animal bioassays as surrogates for human exposure experience. The cardinal reason for the delay between first recognizing leukemia in humans and sought-after neoplasia in animals centers on poor design and conduct of experimental studies. The first evidence of carcinogenicity in animals manifested as malignant tumors of the zymbal glands (sebaceous glands in the ear canal) of rats, and industry attempted to discount this as being irrelevant to humans, as this organ is vestigial and not present per se in humans. Nonetheless, shortly thereafter benzene was shown to be carcinogenic to multiple organ sites in both sexes of multiple strains and multiple species of laboratory animals exposed via various routes. This paper presents a condensed history of the benzene bioassay story with mention of benzene-associated human cancers.

Keywords

benzene; carcinogenicity; industry influence; history; bioassay; public health; occupational safety; primary prevention

Further to Peter Infante's excellent investigative exposé of the truth behind some of the benzene industry's malpractices and abuses (e.g., withholding incriminating data) and resultant OSHA standard-setting issues,[1] there were similar shenanigans surrounding the experimental findings from benzene-exposed animals. Following a series of early, albeit patently inadequate, bioassay experiments on benzene—too few animals, lack of control animals, low and short-term exposures, incomplete pathology often looking only for leukemias[2]—the more modern animal bioassay data clearly confirm and extend the possible cancer hazards of worker and consumer exposures to benzene. To complement the feature by Infante,[1] a brief history of benzene bioassays is given, accompanied by mention of human cancer findings.

CARCINOGENESIS BIOASSAYS

Experimental chemical carcinogenesis bioassays are designed and carried out to identify potential carcinogenic hazards and likely effects for humans.[3] Carcinogenesis results in rodents—mainly rats and mice—have been shown to be consistent and reliable indicators of human cancer risks. All known human carcinogens that have been evaluated in animal bioassays are also correlatively carcinogenic. Further, of the nearly 100 recognized human carcinogens, about one third were shown first to be carcinogenic in experimental animals and subsequently in humans.[4] Hence, for chemicals discovered to be carcinogenic to

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laboratory animals, prudent public health policy suggests strongly that eliminating or minimizing exposures to these carcinogens would reduce environmentally and in particular occupationally associated cancers. Today, this primary prevention strategy serves unfortunately only as a dim beacon of better times when the health of workers was more prominent than profit. This has evolved into what is now referred to as the precautionary principle,[7–9] which poses to act earlier in the available albeit scanty data stream to initiate health safety strategies and proactive occupational and public health measures, despite possible scientific uncertainties.[10]

IARC BENZENE EVALUATIONS

Over the years, the International Agency for Research on Cancer (IARC) has evaluated benzene on three occasions: 1974,[11] 1982,[12] and 1987.[13] In 1974,[11] the IARC decided "The data reported do not permit the conclusion that carcinogenic activity has been demonstrated" based on "Benzene has been tested only in mice by subcutaneous injection and skin application." Regarding human carcinogenicity data, IARC noted "It is established that exposure to commercial benzene or benzene-containing mixtures may result in damage to the haematopoietic system. A relationship between such exposure and the development of leukaemia is suggested by many case reports, and this suggestion is strengthened by a case–control study from Japan."

In 1982[12] IARC evaluated benzene as having "sufficient evidence that benzene is carcinogenic to man," but the available animal data on benzene as only "limited evidence of carcinogenicity in experimental animals." This animal-data conclusion was based on:

> Benzene has been tested in rats by intragastric administration and inhalation exposure, and in mice by skin application, inhalation exposure and subcutaneous injection. Oral administration to rats resulted in an increase in the incidence of Zymbal-gland carcinomas. Anaemia, lymphocytopenia and bone-marrow hyperplasia and an increased incidence of lymphoid tumours occurred in male mice exposed by inhalation to benzene; in similar inhalation studies with another strain of mice and with rats there was no evidence of a leukaemic response. Experiments involving skin application or subcutaneous injection of benzene did not produce evidence of carcinogenicity, but most of these experiments were inadequate.

The last time IARC evaluated benzene was in 1987,[13] with evidence of carcinogenicity considered sufficient both for humans and for animals. In humans the evidence was based on increases in leukemia in benzene workers.[14,15] In animals, according to IARC

> Benzene was tested for carcinogenicity in mice and rats by several routes of administration. Following its oral administration at several dose levels, it induced neoplasms at multiple sites in males and females of both species. After mice were exposed to benzene by inhalation, a tendency towards induction of lymphoid neoplasms was observed. Exposure of rats by inhalation increased the incidence of neoplasms, mainly carcinomas, at various sites. Skin application or subcutaneous injection of benzene to mice did not produce evidence of carcinogenicity, but most of the experiments were inadequate for evaluation. In a mouse-lung tumour bioassay by intraperitoneal injection, an increase in the incidence of lung adenomas was observed in males.

Since the 1987 IARC evaluation, more confirmatory epidemiologic information has become available,[16,17] with multiple myeloma,[18] lung cancer, and non-Hodgkin's lymphoma[19–23] now clearly attributable to benzene exposures. Risks of acute myeloid leukemia and other malignant and nonmalignant hematopoietic disorders associated with benzene exposure in China are consistent with known benzene exposures, hematotoxicity, and cancer risks,

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extending evidence for hematopoietic cancer risks to levels substantially lower than previously established.[24] From global public health and occupational perspectives, perhaps IARC should consider updating its 20-year-old benzene and cancer evaluation. Granted the human and animal data are considered already to be at the highest level of concern, new cancer sites have been discovered and the carcinogenesis correlations between animals and humans are mechanistically worth re-examining.

MALTONI AND CHEMICAL BIOASSAYS

From his early cocarcinogenicity studies of croton oil on rabbit skin[25] and DMBA on hamster skin,[26] through seminal bioassays of vinyl chloride,[27–29] gasoline products,[30,31] including benzene and MTBE,[32,33] and formaldehyde,[34] up to consumer chemicals such as ethyl alcohol,[35] one can justifiably track the early history of chemical carcinogenesis by following Cesare Maltoni and his colleagues' dedicated work on identifying occupational carcinogens[36–38] for developing public health and occupational standards and polices of primary prevention of human cancers.[39]

BENZENE BIOASSAYS

Following Maltoni and his colleagues' seminal finding of the carcinogenicity of benzene to laboratory animals in 1979,[40] they and others more definitively elucidated the carcinogenesis of benzene in a series of papers[41–47] using their unique bioassay exposure design with various experimental protocols.[36–38]

Near the beginning of Maltoni's efforts evaluating benzene for carcinogenesis in animals the National Toxicology Program,[3,48–55] created by David Rall,[56,57] also embarked on unraveling the enigma of why benzene appeared to be an exception (along with arsenic[58–64]) to the mammalian carcinogen paradigm[2,65–67]; that is, a chemical known to cause cancer in humans had not been found to do so similarly in animals.* In this vein, I remember being contacted persistently by industry to learn of early results of our bioassays on benzene. This was a bit amusing, because we already knew that benzene caused cancers (leukemias) in humans, and I wondered why the concentrated interest of industry in our animal findings. Calls came at least weekly from the chemical and petroleum industries. Finally one frequent caller told me that if the bioassays were negative then the industry would have some mammalian biologic means to better challenge the human epidemiologic findings, which industry was already confronting. If our bioassay were clearly negative this would help bolster their argument that other chemical exposures and workplace circumstances (confounders) would lend credence to their benzene-is-not-the-culprit rationale. In keeping with the NTP's open policy I responded with new pathology information as it came available, reminding the inquisitors that until the data were peer-reviewed in public session, our findings could only be considered preliminary. When our pancarcinogenesis findings confirmed and complemented Maltoni's, industry was seemingly taken aback, and momentarily puzzled regarding their next strategy.[68–70]

Carcinogenesis results of 21 mutually tested chemicals including benzene were compared between the Ramazzini Foundation and the NTP, finding remarkable concordance of overall results, and identifying a combined total of 13 target sites of benzene-induced carcinogenesis in animals[67] (Table 1).

*Both of these "exceptions" have been appropriately debunked, solidifying the inexorable correlation that all known human carcinogens that have been tested in laboratory animals are likewise carcinogenic.[3,71–77]

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In 1928, Delore and Borgomano[78] reported the first human case of leukemia associated with benzene exposure.[2,65,78] Prior to 1928, of course, benzene was known to cause "benzene poisoning," a sequela typically involving bone marrow damage. Chronic benzene poisoning among workers leads to various blood disorders such as leukopenia, agranulocytosis, anemia, pancytopenia, aplastic anemia, myelodysplastic syndrome (MDS; preleukemia), and leukemias.[24,79–81] More recently, occupational exposures to benzene have been causatively linked with multiple myeloma,[18] non-Hodgkin's lymphoma,[23] acute[82,83] and chronic[84–87] lymphocytic leukemia, chronic myelogenous leukemia,[82,83] and at lower exposures,[24,84,87] with some indications for lung cancer.[19–21] All of these and additional target sites have been identified in animals as well.[2,47,65,67] Another prime use of chemical carcinogenesis results allows the identification of other potential presumptive target sites that may be added to or looked for distinctly in epidemiologic investigations.

Speculatively, perhaps other sites of human carcinogenicity have not been either looked for or seen in benzene-exposed workers because death from leukemia is relatively rapid after onset and diagnosis. This could also be because available benzene cohorts individually have been small, except for the one in China. Humans showing the later-aged or latency-occurring lung cancers likely escaped developing leukemias.[13,16,19–21] This concept of competing risks of cancer is shown experimentally quite nicely with the potent carcinogen 1,3-butadiene: as exposure concentrations are lowered different tumor patterns become manifest.[88,89] As with benzene or other chemicals, early lethal tumors such as lymphocytic lymphomas or leukemias often reduce the number of animals at risk for expressing later-developing and -occurring neoplasms at other sites. The same, of course, pertains to humans exposed to different exposure levels, patterns, and durations.

Before, at, and subsequent to Maltoni's first reports of clear evidence of benzene carcinogenicity in laboratory animals (and arsenic as mentioned above), intense industry propaganda and pressure attempted to discount long-term animal bioassays as being irrelevant to human risk identification; this strategy certainly had much to do with stifling evidence of benzene carcinogenicity, and extended to many other economically important chemicals showing positive cancer findings in animals as well.[3,69] For some years before the benzene issue, industry and others had mounted a strenuous effort to dismiss the value of bioassays in a concerted global attempt to continue unabated marketing and use of chemicals shown to cause cancers in laboratory animals, and not yet examined epidemiologically.[90]

To justify its basic premise, industry seized and campaigned on the then-notion that arsenic and benzene were both considered to be carcinogenic to humans and yet had not been shown to cause cancer in laboratory animals. Now that these two temporarily, albeit historically non-concordant chemicals have been tested adequately in animals and shown to be classic and multifarious carcinogens (benzene[2,47,65,67]; arsenic[57–64]), this once-dynamic duo touted by industry for vested purposes no longer serves its needs (see footnote on page 214). Now industry uses other arguments such as those based on threshold differences,[91] "non-genotoxic" carcinogens,[92,93] mechanisms or "modes-of-action" being unique to animals and not relevant to humans,[91–100] even hormesis,[101,102] cell proliferation,[92,192–109] inflammation and general toxicity,[110–112] hormonal mediation,[113–115] mouse liver tumors,[116] and benign tumors,[117] to name a few obfuscatory issues industry has latched onto to cloud bioassay results and impede or derail regulatory actions.

Additionally, Maltoni's first findings of benzene cancer induction in animals[40,41] were heavily disputed by industry because the organ affected by cancer was the zymbal gland (located in the inner ear canal), which humans have as a vestige. This issue of zymbal glands has been addressed and debunked, along with other so-called "rodent-unique" organs susceptible to benzene-induced carcinogenesis.[118] Fortunately, this issue became moot

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given the plethora of tumors and tumor types and organ sites seen in the benzene studies. The other issue making the benzene-is-safe argument less tenable, as with arsenic, was that benzene was already long known to be carcinogenic to humans. If this had not been the case, the battle for more stringent and better worker protection and reduction of occupational standards for acceptable exposure levels would have been even more difficult. As it was, lowering of the occupation exposure standard took ten years longer than anticipated because of adverse decisions issued by the U.S. Supreme Court.[119]

We witness this animal-only argument every single time a chemical of some economic importance is found to cause cancer in laboratory animals in the absence of epidemiologic data. What we never encounter is industry's questioning or disputing the many "negative" bioassay results on big-volume chemicals.

BENZENE METABOLITES

Mammalian metabolism of benzene is complex, with multiple pathways and diverse metabolites. Despite abundant research, neither the most active carcinogenic metabolite(s) nor a detailed well-accepted mechanism(s) of carcinogenicity of benzene is known with even a modicum of certainty.[2,120,121] Tsutsui et al.,[120] for example, studied benzene- and key metabolite-induced cell transformation, gene mutations, chromosome aberrations, aneuploidy, sister chromatid exchanges, and unscheduled DNA synthesis in Syrian hamster embryo cells. They found an array of effects for these endpoints but no one metabolite of benzene consistently drove the results. Another way to shed light on those is to investigate carcinogenicity of individual metabolites. Fortunately, several of the most abundant or long-lasting metabolites (or those that could be gotten in sufficient quantity) have been tested for carcinogenic activity: catechol, hydroquinone, and phenol. The designs and findings for these carcinogenicity studies are summarized.

Catechol

Naturally occurring in fruits and vegetables, present in cigarette smoke, and an industrial chemical, catechol is used to make insecticides, perfumes, drugs, and polymerization inhibitors. Catechol has been used as an antiseptic, in photography, and in dyestuffs.

Catechol has been shown to have strong promoting activity in mice, and alone induces forestomach hyperplasia, generally a few papillomas of the forestomach (non-glandular), and adenomatous hyperplasia and adenocarcinomas of the glandular stomach in near all rats.[122–126] Administered with known carcinogens, catechol typically increased the occurrence of initiator-targeted tumors of the forestomach and stomach, tongue, and esophagus, but did not enhance their occurrence in liver, urinary bladder, or thyroid.[122–126] Thus, catechol exhibited strong cancer-promotion activity.

Hydroquinone

Used an antioxidant in the rubber industry, as a developing agent in photography, and as an intermediate in the manufacture of rubber and food antioxidants and monomer inhibitors, hydro-quinone products are also used as depigmenting agents to lighten skin. Hydroquinone in deionized water was given by gavage for two years to groups of rats and mice of each sex, five days per week at 0, 25 (rats), 50, or 100 (mice) mg/kg.[127,128] Nephropathy was common among the rats, with hyperplasia of the renal pelvic transitional epithelium and renal cortical cysts increased in male rats. In mice, thyroid follicular cell hyperplasia was increased (males: 9% vs 28% and 35%; females: 24% vs 85% and 82%). Increases of anisokaryosis, multinucleated hepatocytes, and basophilic foci occurred in the livers of male mice.[127,128]

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Regarding carcinogenic responses, renal tubular cell hyperplasia was seen in two top-dose male rats, with dose-related increases in renal tubular cell adenomas in 0% vs 7% and 15%. Mononuclear cell leukemia in female rats was increased: 16% vs 27% and 40%. In low-dose male mice liver tumors were marginally elevated: 36% vs 54% & 45%, whereas in female mice liver tumors were intensified in both hydroquinone groups: 5% vs 29% and 24%.[127,128]

Hydroquinone made available to rats and mice of both sexes at 0.8% in the diet for two years induced renal tubular cell hyperplasia as well as adenomas, mainly in males of both species, and was associated with chronic nephropathy in rats.[129] Also, epithelial hyperplasia of renal papilla was increased in male rats. Hepatocellular adenoma was enhanced in male mice. Squamous-cell hyperplasia of the forestomach epithelium was higher in mice of both sexes given hydroquinone, but no increase in tumor development was observed.[129]

Thus, hydroquinone caused kidney tumors in male (and possibly in female) rats and mice, leukemia in female rats, thyroid follicular cell hyperplasia in mice, and liver tumors in male and female mice.[127–130] Overall, there is clear evidence that hydroquinone causes cancer in laboratory animals.

Phenol

Approximately 90% of phenol is used in the manufacture of phenolic (phenol formaldehyde) resins, caprolactam, bisphenol A, alkyl phenol, and adipic acid. The remainder is used to produce an assortment of products: salicylic acid, phenacetin, dyes, metal cleaners, disinfectants, antiseptics, photographic chemicals, wood preservatives (pentachlorophenol), paints, paint and varnish removers, and agricultural chemicals (2,4-D and parathion).[131]

For two years rats and mice of each sex were given drinking water containing 0, 2,500, or 5,000 ppm phenol. The only carcinogenic response was increases in leukemia, 36% vs 62% and 50%, that may have been associated with the phenol administration.[131,132] Even though the NTP considered an association with administration of phenol was not established, the incidences in both exposure groups were greater than in controls and the low dose showed a statistically significant effect. No other carcinogenic response was observed in rats or mice. Other low dose increases in male rats included C-cell tumors of the thyroid (8% vs 14% and 2%), adrenal gland pheochromocytomas (26% vs 44% and 18%), and interstitial-cell tumors of the testes (88% vs 98% and 94%).[131,132]

In other studies, phenol given orally with benzo(a)-pyrene produced sixfold increases in malignant tumors of the forestomach over BaP given alone.[112] Phenol also promoted mouse skin carcinogenesis in two-stage protocols.[133]

Scientific evidence indicates that multiple mechanisms are likely to contribute to benzene-induced leukemias and cancers in other target organs; whether these include individual or co-mechanisms for the individual metabolites remains to be ascertained. (Reasonably straightforwardly, this of course tends to represent universal thinking in chemical carcinogenesis, and leads to the notion that unique chemicals or classes of chemicals induce cancers by "different" mechanisms.) Increasing information lends further credence that metabolites of benzene are primarily responsible for its carcinogenic activity.[2,120,121,134–137] Phenol, hydroquinone, and catechol are the major metabolites of benzene in mammals, established in analyses of human urine,[135] and have been tested for long-term carcinogenicity. Phenol, to a lesser extent, and hydroquinone are associated individually with inducing leukemia in animals, and we might opine in humans as well. One wonders what would be the result(s) if the two chemicals were tested together; that is, whether these findings would be more or less potent than those for benzene or either of these metabolites

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alone. Catechol causes forestomach and stomach tumors in animals, whereas benzene causes forestomach tumors but does not cause stomach tumors. Some of the other carcinogenic effects of benzene may be due to combinations of the metabolites or to others not yet evaluated for carcinogenic activity.

At the same time, carcinogenic concordance in target sites between animals and humans need not be sacrosanct. Typically in animals there are more tumor sites identified simply because more pathology is done on animals than on humans. One suspects that if all organs were evaluated in humans when people died of "old age" or with cancers other organs would be found to be neoplastic as well. Meanwhile, epidemiology might best broaden the organ scope for future studies.

CONCLUSIONS

The clear findings of cancers in animals resulting from exposures to benzene (and to arsenic), and to all other known human carcinogens that have been tested in animals, confirm and validate once again the value of long-term animal bioassays for identifying potential cancer risks to humans.[3,5,67–77,138–173] Virtual acknowledgement of this led industry to new strategies to deny bioassay results: posing that mechanisms (or "modes of action") of carcinogenesis in animals are unique and hence not relevant to humans. Interestingly most of these claims are based on supposition and not data regarding either the exact mechanism in animals or the lack thereof in humans. A key to reducing damage from all carcinogens, whether identified in animals or in humans or in both mammalian species, centers on reducing exposures.

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References

1. Infante PF. The past suppression of industry knowledge of the toxicity of benzene to humans and potential bias in future benzene research. Int J Occup Environ Health. 2006; 12:268–72. [PubMed: 16967835]
2. Huff J, Haseman JK, DeMarini DM, et al. Multiple-site carcinogenicity of benzene in Fischer 344 rats and B6C3F1 mice. Environ Health Perspect. 1989; 82:125–63. [PubMed: 2676495]
3. Huff, J. Value, validity, and historical development of carcino-genesis studies for predicting and confirming carcinogenic risks to humans. In: Kitchin, KT., editor. Carcinogenicity Testing, Predicting, & Interpreting Chemical Effects. Marcel Dekker; New York: 1999. p. 21-123.Chapter 2
4. Tomatis L. The predictive value of rodent carcinogenicity tests in the evaluation of human risks. Annu Rev Pharmacol Toxicol. 1979; 19:511–30. [PubMed: 378109]
5. Huff J. Chemicals and cancer in humans: first evidence in experimental animals. Environ Health Perspect. 1993; 100:201–10. [PubMed: 8354167]
6. IARC. Preamble 9-31. Formaldehyde, 2-butoxyethanol and 1-tert-butoxypropan-2-ol. IARC Managr Eval Carcinog Risks Hum. Dec.2006 88:1–478.
7. Grandjean P, Gailar JC, Gee D, et al. Implications of the precautionary principle in research and policy-making. Am J Ind Med. 2004; 45:382–5. [PubMed: 15029572]
8. Tickner JA. Precautionary principle encourages policies that protect human health and the environment in the face of uncertain risks. Public Health Rep. 2002; 117:493–7. [PubMed: 12576528]
9. Stirling A. Risk, precaution and science: towards a more constructive policy debate. Talking point on the precautionary principle. EMBO Rep. 2007; 8:309–15.

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10. Ozonoff D. On being careful what we wish for: some difficulties with operationalizing the precautionary principle. Int J Occup Med. Environ Health. 2004; 17:35–41. [PubMed: 15212204]
11. IARC. Benzene. IARC Monogr Eval Carcinog Risk Chem Hum. 1974; 7:203–21.
12. IARC. Benzene. IARC Monogr Eval Carcinog Risk Chem Hum. 1982; 29:93–148. 391–8. [PubMed: 6957390]
13. IARC. Benzene. IARC Monogr Eval Carcinog Risk Chem Hum. 1987; (suppl 7):120–2.
14. Infante PF, Rinsky RA, Wagoner JK, Young RJ. Leukaemia in benzene workers. Lancet. 1977; 9;2(8028):76–8.
15. Infante PF, White MC. Projections of leukemia risk associated with occupational exposure to benzene. Am J Ind Med. 1985; 7(5-6):403–13. [PubMed: 3890530]
16. NTP RoC. 11th Report on Carcinogens. National Toxicology Program, Research Triangle Park, NC 27709; 2004. Benzene. http://ntp-server.niehs.nih.gov/ntp/roc/eleventh/ profiles/s019benz.pdf
17. ATSDR. Agency for Toxic Substances and Disease Registry, 1825 Century Blvd. Atlanta, GA: Sep. 2005 Toxicological Profile for Benzene. Draft for Public Comment30345http://www.atsdr.cdc.gov/toxprofiles/tp3.html
18. Infante PF. Benzene exposure and multiple myeloma: a detailed meta-analysis of benzene cohort studies. Ann NY Acad Sci. 2006; 1076:90–109. [PubMed: 17119195]
19. Yin SN, Hayes RB, Linet MS, et al. A cohort study of cancer among benzene-exposed workers in China: overall results. Am J Ind Med. 1996; 29:227–35. [PubMed: 8833775]
20. Yin SN, Hayes RB, Linet MS, et al. An expanded cohort study of cancer among benzene-exposed workers in China. Benzene Study Group. Environ Health Perspect. 1996; 104(suppl 6):1339–41. [PubMed: 9118917]
21. Hayes RB, Yin SN, Dosemeci M, et al. Mortality among benzene-exposed workers in China. Environ Health Perspect. 1996; 104(suppl 6):1349–52. [PubMed: 9118919]
22. Hayes RB, Yin SN, Dosemeci M, et al. Benzene and the dose-related incidence of hematologic neoplasms in China. Chinese Academy of Preventive Medicine—National Cancer Institute Benzene Study Group. J Natl Cancer Inst. 1997; 89:1065–71. [PubMed: 9230889]
23. Smith MT, Jones RM, Smith AH. Benzene exposure and risk of non-Hodgkin lymphoma. Cancer Epidemiol Biomarkers Prev. 2007; 16:385–91. [PubMed: 17337645]
24. Hayes RB, Songnian Y, Dosemeci M, Linet M. Benzene and lymphohematopoietic malignancies in humans. Am J Ind. Med. 2001; 40:117–26. [PubMed: 11494338]
25. Parmeggiani A, Prodi G, Maltoni C. [Incorporation of radioactive sulfate in sulfurated polysaccharides of rabbit skin during treatment with cocarcinogenic substance (Croton oil)]. Boll Soc Ital Biol Sper. 1957; 33:496–9. [In Italian]. [PubMed: 13479563]
26. Maltoni C, Prodi G. [Cutaneous oncogenesis in *Cricetus auratus* (hamster) by 9,10-dimethyl-1,2-benzanthracene.]. Boll Soc Ital Biol Sper. 1957; 33:506–7. [In Italian]. [PubMed: 13479566]
27. Maltoni C, Lefemine G. Carcinogenicity bioassays of vinyl chlo-ride. I. Research plan and early results. Environ Res. 1974; 387:405.
28. Warren H, Huff J. Health effects of vinyl chloride monomer: an annotated literature collection. Environ Health Perspect. 1975; 11:251–319. [PubMed: 1100368]
29. Infante PF. Oncogenic and mutagenic risks in communities with polyvinyl chloride production facilities. Ann NY Acad Sci. 1976; 271:49–57. [PubMed: 1069539]
30. Maltoni C. Biomedical research as a science for development: the case of gasoline. Ramazzini Lecture, 1995. Ann NY Acad Sci. 1997; 837:1–14. [PubMed: 9472328]
31. Maltoni C, Ciliberti A, Pinto C, Soffritti M, Belpoggi F, Menarini L. Results of long-term experimental carcinogenicity studies of the effects of gasoline, correlated fuels, and major gasoline aromatics on rats. Ann NY Acad Sci. 1997; 837:15–52. [PubMed: 9472329]
32. Belpoggi F, Soffritti M, Maltoni C. Methyl-tertiary-butyl ether (MTBE)—a gasoline additive—causes testicular and lymphohaematopoietic cancers in rats. Toxicol Ind Health. 1995; 11:119–49. [PubMed: 7491630]
33. Belpoggi F, Soffritti M, Filippini F, Maltoni C. Results of long-term experimental studies on the carcinogenicity of methyl tert-butyl ether. Ann NY Acad Sci. 1997; 837:77–95. [PubMed: 9472331]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

34. Soffritti M, Maltoni C, Maffei F, Biagi R. Formaldehyde: an experimental multipotential carcinogen. Toxicol Ind Health. 1989; 5:699–730. [PubMed: 2815102]
35. Soffritti M, Belpoggi F, Cevolani D, Guarino M, Padovani M, Maltoni C. Results of long-term experimental studies on the carcinogenicity of methyl alcohol and ethyl alcohol in rats. Ann NY Acad Sci. 2002; 982:46–69. [PubMed: 12562628]
36. Maltoni C. The contribution of experimental (animal) studies to the control of industrial carcinogenesis. Appl Occup Environ Hyg. 1995; 10:749–60.
37. Maltoni C, Soffritti M, Belpoggi F. The scientific and methodological bases of experimental studies for detecting and quantifying carcinogenic risks. Ann NY Acad Sci. 1999; 895:10–26. [PubMed: 10676406]
38. Soffritti M, Belpoggi F, Minardi F, Maltoni C. Ramazzini Foundation cancer program: history and major projects, life-span carcinogenicity bioassay design, chemicals studied, and results. Ann NY Acad Sci. 2002; 982:26–45. [PubMed: 12562627]
39. Mehlman MA. Carcinogenic effects of benzene: Cesare Maltoni's contributions. Ann NY Acad Sci. 2002; 982:137–48. [PubMed: 12562633]
40. Maltoni C, Scarnato C. First experimental demonstration of the carcinogenic effects of benzene; long-term bioassays on Sprague-Dawley rats by oral administration. Med Lav. 1979; 70:352–7. [PubMed: 554913]
41. Maltoni C, Cotti G, Valgimigli L, Mandrioli A. Zymbal gland carcinomas in rats following exposure to benzene by inhalation. Am J Ind Med. 1982; 3:11–6. [PubMed: 7124739]
42. Maltoni C, Conti B, Scarnato C. Squamous cell carcinomas of the oral cavity in Sprague-Dawley rats, following exposure to benzene by ingestion. First experimental demonstration. Med Lav. 1982; 73:441–5. [PubMed: 7177031]
43. Maltoni C, Cotti G, Valgimigli L, Mandrioli A. Hepatocarcinomas in Sprague-Dawley rats, following exposure to benzene by inhalation. First experimental demonstration. Med Lav. 1982; 73:446–50. [PubMed: 7177032]
44. Maltoni C, Conti B, Cotti G. Benzene: a multipotential carcinogen. Results of long-term bioassays performed at the Bologna Institute of Oncology. Am J Ind Med. 1983; 589:630.
45. Maltoni C, Conti B, Cotti G, Belpoggi F. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: current results and ongoing research. Am J Ind Med. 1985; 7(5-6): 415–46. [PubMed: 4003403]
46. Maltoni C, Conti B, Perino G, Di Maio V. Further evidence of benzene carcinogenicity. Results on Wistar rats and Swiss mice treated by ingestion. Ann NY Acad Sci. 1988; 534:412–26. [PubMed: 3389671]
47. Maltoni C, Ciliberti A, Cotti G, Conti B, Belpoggi F. Benzene, an experimental multipotential carcinogen: results of the longterm bioassays performed at the Bologna Institute of Oncology. Environ Health Perspect. 1989; 82:109–24. [PubMed: 2792037]
48. Huff J. Carcinogenesis bioassay results from the National Toxicology Program. Environ Health Perspect. 1982; 45:185–98. [PubMed: 7140693]
49. Huff J, Moore JA. Carcinogenesis studies design and experimental data interpretation/evaluation at the National Toxicology Program. Prog Clin Biol Res. 1984; 141:43–64. [PubMed: 6718388]
50. Chhabra RS, Huff JE, Schwetz BS, Selkirk J. An overview of prechronic and chronic toxicity/ carcinogenicity experimental study designs and criteria used by the National Toxicology Program. Environ Health Perspect. 1990; 86:313–21. [PubMed: 2205492]
51. Huff J, Haseman J. Long-term chemical carcinogenesis experiments for identifying potential human cancer hazards: collective database of the National Cancer Institute and National Toxicology Program (1976–1991). Environ Health Perspect. 1991; 96:23–31. [PubMed: 1820269]
52. Huff J. Design strategies, results and evaluations of long-term chemical carcinogenesis studies. Scand J Work Environ Health. 1992; 18(suppl 1):31–7. [PubMed: 1411375]
53. Huff J. A historical perspective on the classification developed and used for chemical carcinogens by the National Toxicology Program during 1983–1992. Scand J Work Environ Health. 1992; 18(suppl 1):74–82. [PubMed: 1411383]
54. Haseman J, Melnick R, Tomatis L, Huff J. Carcinogenesis bioassays: study duration and biological relevance. Food Chem Toxicol. 2001; 39:739–44. [PubMed: 11397520]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

55. Bucher JR. The National Toxicology Program rodent bioassay: designs, interpretations, and scientific contributions. Ann NY Acad Sci. 2002; 982:198–207. [PubMed: 12562638]
56. Huff J. The legacy of David Platt Rall. Scientific, environmental, public health, and regulatory contributions. Eur J Oncol. 2000; 5:85–100.
57. Huff J. David Rall and the national toxicology program. Environ Health Perspect. 2005; 113:A152. [PubMed: 15743701]
58. Hammons AS, Lewis EB, Braunstein HM, Huff J. Arsenic: Potential hazards of environmental exposures. Environmental Chemicals: Human and Animal Health Proceedings IV. 1976:75–93.
59. Chan P, Huff J. Arsenic carcinogenesis in animals and in humans: mechanistic, experimental, and epidemiological evidence. Environ Carcinog Ecotox Revs. 1997; C15:83–122.
60. Huff J, Waalkes M, Chan P. Arsenic—evidence of carcinogenicity in animals. Environ Health Perspect. 1998; 106:A582–A583. [PubMed: 10207116]
61. Huff J, Chan P, Nyska A. Is the human carcinogen arsenic carcinogenic to laboratory animals? Toxicol Sci. 2000; 55:17–23. [PubMed: 10788555]
62. Waalkes MP, Ward JM, Liu J, Diwan BA. Transplacental carcinogenicity of inorganic arsenic in the drinking water: induction of hepatic, ovarian, pulmonary, and adrenal tumors in mice. Toxicol Appl Pharmacol. 2003; 186:7–17. [PubMed: 12583988]
63. Waalkes MP, Ward JM, Diwan BA. Induction of tumors of the liver, lung, ovary and adrenal in adult mice after brief maternal gestational exposure to inorganic arsenic: promotional effects of postnatal phorbol ester exposure on hepatic and pulmonary, but not dermal cancers. Carcinogenesis. 2004; 25:133–41. [PubMed: 14514661]
64. Waalkes MP, Liu J, Ward JM, Diwan BA. Mechanisms underlying arsenic carcinogenesis: hypersensitivity of mice exposed to inorganic arsenic during gestation. Toxicology. 2004; 198:31–8. [PubMed: 15138027]
65. Huff J. National Toxicology Program. NTP Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Natl Toxicol Program Tech Rep Ser. 1986; 289:1–277. [PubMed: 12748714]
66. Huff J, Eastin W, Roycroft J, Eustis SL, Haseman JK. Carcino-genesis studies of benzene, methyl benzene, and dimethyl benzenes. Ann NY Acad Sci. 1988; 534:427–40. [PubMed: 3389672]
67. Huff J. Chemicals studied and evaluated in long-term carcino-genesis bioassays by both the Ramazzini Foundation and the National Toxicology Program: in tribute to Cesare Maltoni and David Rall. Ann NY Acad Sci. 2002; 982:208–30. [PubMed: 12562639]
68. Breilh J, Jefer C Branco, Castelman BI, et al. Texaco and its consultants. Int J Occup Environ Health. 2005; 11:217–20. [PubMed: 15875903]
69. Huff J. Industry influence on occupational and environmental public health. Int J Occup Environ Health. 2007; 13:107–17. [PubMed: 17427355]
70. Bailar JC 3rd. How to distort the scientific record without actually lying: truth, and the arts of science. Eur J Oncol. 2006; 11:217–24.
71. Tomatis L, Aitio A, Wilbourn J, Shuker L. Human carcinogens so far identified. Jpn J Cancer Res. 1989; 80:795–807. [PubMed: 2513295]
72. Huff, J. Chemicals causally associated with cancers in humans and in laboratory animals: a perfect concordance. In: Waalkes, MP.; Ward, JM., editors. Carcinogen-esis. Raven Press; New York: 1994. p. 25-37.Chapter 2
73. Huff, J. Chemically associated respiratory carcinogenesis in rodents and in humans. In: Waalkes, MP.; Ward, JM., editors. Carcinogenesis. Raven Press; New York: 1994. p. 199-214.Chapter 7
74. Huff J. Animal and human carcinogens. Environ Health Perspect. 1999; 107:A341–2. [PubMed: 10405250]
75. Tomatis L, Huff J, Hertz-Picciotto I, et al. Avoided and avoidable risks of cancer. Carcinogenesis. 1997; 18:97–105. [PubMed: 9054595]
76. Tomatis L. The identification of human carcinogens and primary prevention of cancer. Mutat Res. 2000; 462:407–21. [PubMed: 11523541]
77. Tomatis L, Melnick RL, Haseman J, Barrett JC, Huff J. Alleged misconceptions' distort perceptions of environmental cancer risks. FASEB J. 2001; 15:195–203. [PubMed: 11149907]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

78. Delore P, Borgomano C. Leucemie aique au cours de l'intoxication benzenique. Sur l'origine toxique de certaines leucemies aigues et leurs relations avec les anemies graves. J Med Lyon. 1928; 9:227–33. [In French].
79. Li G, Yin S. Progress of epidemiological and molecular epidemiological studies on benzene in China. Ann NY Acad Sci. 2006; 1076:800–9. [PubMed: 17119257]
80. Kuang S, Liang W. Clinical analysis of 43 cases of chronic benzene poisoning. Chem Biol Interact. 2005; 153-154:129–35. [PubMed: 15935809]
81. Landrigan PJ. Benzene and blood: one hundred years of evidence. Am J Ind Med. 1996; 29:225–6. [PubMed: 8833774]
82. Shu XO, Gao YT, Brinton LA, et al. A population-based case–control study of childhood leukemia in Shanghai. Cancer. 1988; 62:635–44. [PubMed: 3164642]
83. Adegoke OJ, Blair A, Shu XO, et al. Occupational history and exposure and the risk of adult leukemia in Shanghai. Ann Epidemiol. 2003; 13:485–94. [PubMed: 12932623]
84. Glass DC, Gray CN, Jolley DJ, et al. Leukemia risk associated with low-level benzene exposure. Epidemiology. 2003; 14:569–77. [PubMed: 14501272]
85. Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR. The health watch case–control study of leukemia and benzene: the story so far. Ann NY Acad Sci. Sep.2006 1076:80–9. [PubMed: 17119194]
86. Glass DC, Sim MR, Fritschi L, Gray CN, Jolley DJ, Gibbons C. Leukemia risk and relevant benzene exposure period—Re: follow-up time on risk estimates. Am J Ind Med. 2002; 41:481–9.2004; 45:222–3.
87. Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR. Health watch exposure estimates: do they underestimate benzene exposure? Chem Biol Interact. 2005; 153-154:23–32. [PubMed: 15935797]
88. Huff J, Melnick RL, Solleveld HA, Haseman JK, Powers M, Miller RA. Multiple organ carcinogenicity of 1,3-butadiene in B6C3F1 mice after 60 weeks of inhalation exposure. Science. 1985; 227(4686):548–9. [PubMed: 3966163]
89. Melnick RL, Huff J, Chou BJ, Miller RA. Carcinogenicity of 1,3-butadiene in C57BL/6 × C3H F1 mice at low exposure concentrations. Cancer Res. 1990; 50:6592–9. [PubMed: 2208121]
90. Melnick RL, Kamel F, Huff J. Declaring chemicals "not carcinogenic to humans" requires validation, not speculation. Environ Health Perspect. 2003; 111:A203–4. [PubMed: 12676636]
91. Haseman JK. An alternative perspective: a critical evaluation of the Waddell threshold extrapolation model in chemical carcinogenesis. Toxicol Pathol. 2003; 31:468–70. [PubMed: 14692613]
92. Huff J. Mechanisms, chemical carcinogenesis, and risk assessment: cell proliferation and cancer. Am J Ind Med. 1995; 27:293–300. [PubMed: 7755018]
93. Melnick RL, Kohn MC, Portier CJ. Implications for risk assessment of suggested nongenotoxic mechanisms of chemical carcinogenesis. Environ Health Perspect. 1996; 104(suppl 1):123–34. [PubMed: 8722116]
94. Barrett JC, Huff J. Cellular and molecular mechanisms of chemically induced renal carcinogenesis. Ren Fail. 1991; 13:211–25. [PubMed: 1780490]
95. Melnick RL. An alternative hypothesis on the role of chemically induced protein droplet (alpha 2u-globulin) nephropathy in renal carcinogenesis. Regul Toxicol Pharmacol. 1992; 16:111–25. [PubMed: 1279759]
96. Melnick RL. Critique does not validate assumptions in the model on alpha 2u-globulin and renal carcinogenesis. Regul Toxicol Pharmacol. 1993; 18:365–8. [PubMed: 7506437]
97. Huff J. Alpha-2u-globulin nephropathy, posed mechanisms, and white ravens. Environ Health Perspect. 1996; 104:1264–7. [PubMed: 10736606]
98. Melnick RL, Kohn MC, Huff J. Weight of evidence versus weight of speculation to evaluate the alpha2u-globulin hypothesis. Environ Health Perspect. 1997; 105:904–6. [PubMed: 9341100]
99. Melnick RL. Is peroxisome proliferation an obligatory precursor step in the carcinogenicity of di(2-ethylhexyl)phthalate (DEHP)? Environ Health Perspect. 2001; 109:437–42. [PubMed: 11401753]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

100. Melnick RL. The IARC evaluation of di(2-ethylhexyl)phthalate (DEHP): a flawed decision based on an untested hypothesis. Int J Occup Environ Health. 2002; 8:284–6. [PubMed: 12358086]
101. Thayer KA, Melnick R, Burns K, Davis D, Huff J. Fundamental flaws of hormesis for public health decisions. Environ Health Perspect. 2005; 113:1271–6. [PubMed: 16203233]
102. Thayer KA, Melnick R, Huff J, Burns K, Davis D. Hormesis: a new religion? Environ Health Perspect. 2006; 114:A632–3. [PubMed: 17107829]
103. Melnick RL. Does chemically induced hepatocyte proliferation predict liver carcinogenesis? FASEB J. 1992; 6:2698–706. [PubMed: 1612294]
104. Melnick RL, Huff J, Barrett JC, Maronpot RR, Lucier G, Portier CJ. Cell proliferation and chemical carcinogenesis: a symposium overview. Mol Carcinog. 1993; 7:135–8. [PubMed: 8489710]
105. Melnick RL, Huff J. Liver carcinogenesis is not a predicted out come of chemically induced hepatocyte proliferation. Toxicol Ind Health. 1993; 9:415–38. [PubMed: 8367884]
106. Ward JM, Uno H, Kurata Y, Weghorst CM, Jang JJ. Cell proliferation not associated with carcinogenesis in rodents and humans. Environ Health Perspect. 1993; 101(suppl 5):125–35. [PubMed: 8013399]
107. Tomatis L. Cell proliferation and carcinogenesis: a brief history and current view based on an IARC workshop report. International Agency for Research on Cancer. Environ Health Perspect. 1993; 101(suppl 5):149–51. [PubMed: 8013403]
108. Farber E. Cell proliferation as a major risk factor for cancer: a concept of doubtful validity. Cancer Res. 1995; 55:3759–62. [PubMed: 7641190]
109. Farber E. Cell proliferation is not a major risk factor for cancer. Mod Pathol. 1996; 9:606. [PubMed: 8782195]
110. Hoel DG, Haseman JK, Hogan MD, Huff J, McConnell EE. The impact of toxicity on carcinogenicity studies: implications for risk assessment. Carcinogenesis. 1988; 9:2045–52. [PubMed: 3052903]
111. Huff J. Chemical toxicity and chemical carcinogenesis. Is there a causal connection? A comparative morphological evaluation of 1500 experiments. IARC Sci Publ. 1992; (116):437–75. [PubMed: 1428093]
112. Huff J. Absence of morphologic correlation between chemical toxicity and chemical carcinogenesis. Environ Health Perspect. 1993; 101(suppl 5):45–53. [PubMed: 8013424]
113. Huff J, Boyd J, Barrett JC. Cellular and molecular mechanisms of hormonal carcinogenesis: environmental influences. Prog Clin Biol Res. 1996; 394:i–xix. 1–479.
114. Huff J, Boyd J, Barrett JC. Hormonal carcinogenesis and environmental influences: background and overview. Prog Clin Biol Res. 1996; 394:3–23. [PubMed: 8778803]
115. Huff J. Chemically induced cancers in hormonal organs of laboratory animals and of humans. Prog Clin Biol Res. 1996; 394:77–102. [PubMed: 8778812]
116. Maronpot RR, Haseman JK, Boorman GA, Eustis SE, Rao GN, Huff J. Liver lesions in B6C3F1 mice: the National Toxicology Program, experience and position. Arch Toxicol. 1987; 10(suppl): 10–26.
117. Huff J, Eustis SL, Haseman JK. Occurrence and relevance of chemically induced benign neoplasms in long-term carcinogenicity studies. Cancer Metastasis Rev. 1989; 8:1–22. Erratum in: Cancer Metastasis Rev 1989;8:281. [PubMed: 2667783]
118. Huff J. Applicability to humans of "rodent-specific sites" of chemical carcinogenicity: tumors of the forestomach and of the harderian, preputial, and zymbal glands induced by benzene. J Occup Med Toxicol. 1992; 1:109–41.
119. White MC, Infante PF, Walker B Jr. Occupational exposure to benzene: a review of carcinogenic and related health effects following the U.S. Supreme Court decision. Am J Ind Med. 1980; 1:233–43. [PubMed: 7044110]
120. Tsutsui T, Hayashi N, Maizumi H, Huff J, Barrett JC. Benzene-, catechol-, hydroquinone- and phenol-induced cell transformation, gene mutations, chromosome aberrations, aneuploidy, sister chromatid exchanges and unscheduled DNA synthesis in Syrian hamster embryo cells. Mutat Res. 1997; 373:113–23. [PubMed: 9015160]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

121. Whysner J, Reddy MV, Ross PM, Mohan M, Lax EA. Genotoxicity of benzene and its metabolites. Mutat Res. 2004; (566):99–130. [PubMed: 15164977]

122. Hirose M, Fukushima S, Shirai T, et al. Stomach carcinogenicity of caffeic acid, sesamol and catechol in rats and mice. Jpn J Cancer Res. 1990; 81:207–12. [PubMed: 2112522]

123. Hirose M, Fukushima S, Tanaka H, Asakawa E, Takahashi S, Ito N. Carcinogenicity of catechol in F344 rats and B6C3F1 mice. Carcinogenesis. 1993; 14:525–9. [PubMed: 8453730]

124. IARC. Catechol. IARC Monogr Eval Carcinog Risks Hum. 1999; 71(Pt 2):433–51. [PubMed: 10476456]

125. Hirose M, Hakoi K, Takahashi S, et al. Sequential morphological and biological changes in the glandular stomach induced by oral administration of catechol to male F344 rats. Toxicol Pathol. 1999; 27:448–55. [PubMed: 10485826]

126. Hagiwara A, Takesada Y, Tanaka H, et al. Dose-dependent induction of glandular stomach preneoplastic and neoplastic lesions in male F344 rats treated with catechol chronically. Toxicol Pathol. 2001; 29:180–6. [PubMed: 11421485]

127. Kari F. National Toxicology Program. NTP Toxicology and Carcinogenesis Studies of Hydroquinone (CAS No. 123-31-9) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Natl Toxicol Program Tech Rep Ser. Oct.1989 366:1–248. [PubMed: 12692638]

128. Kari FW, Bucher J, Eustis SL, Haseman JK, Huff J. Toxicity and carcinogenicity of hydroquinone in F344/N rats and B6C3F1 mice. Food Chem Toxicol. 1992; 30:737–47. [PubMed: 1365401]

129. Shibata MA, Hirose M, Tanaka H, Asakawa E, Shirai T, Ito N. Induction of renal cell tumors in rats and mice, and enhancement of hepatocellular tumor development in mice after long-term hydroquinone treatment. Jpn J Cancer Res. 1991; 82:1211–9. [PubMed: 1752780]

130. IARC. Hydroquinone. IARC Monogr Eval Carcinog Risks Hum. 1999; 71(Pt 2):691–719. [PubMed: 10476468]

131. Huff J. National Toxicology Program. Bioassay of Phenol for Possible Carcinogenicity (CAS No. 108-95-2). Natl Toxicol Program Tech Rep Ser. Aug.1980 203:1–123. [PubMed: 12778176]

132. Huff J. Carcinogenesis results on seven amines, two phenols, and one diisocyanate used in plastics and synthetic elastomers. Prog Clin Biol Res. 1984; 141:347–63. [PubMed: 6326159]

133. IARC. Phenol. IARC Monogr Eval Carcinog Risks Hum. 1999; 71(Pt 2):749–68. [PubMed: 10476471]

134. Gopalakrishna R, Chen ZH, Gundimeda U. Tobacco smoke tumor promoters, catechol and hydroquinone, induce oxidative regulation of protein kinase C and influence invasion and metastasis of lung carcinoma cells. Proc Natl Acad Sci USA. 1994; 91:12233–7. [PubMed: 7991611]

135. Rothman N, Bechtold WE, Yin SN, et al. Urinary excretion of phenol, catechol, hydroquinone, and muconic acid by workers occupationally exposed to benzene. Occup Environ Med. 1998; 55:705–11. [PubMed: 9930093]

136. Golding BT, Watson WP. Possible mechanisms of carcinogenesis after exposure to benzene. IARC Sci Publ. 1999; 150:75–88. [PubMed: 10626210]

137. Lau SS, Monks TJ, Everitt JI, Kleymenova E, Walker CL. Carcinogenicity of a nephrotoxic metabolite of the "nongenotoxic" carcinogen hydroquinone. Chem Res Toxicol. 2001; 14:25–33. [PubMed: 11170505]

138. Rall DP. The role of laboratory animal studies in estimating carcinogenic risks for man. IARC Sci Publ. 1979; 25:179–89. [PubMed: 457152]

139. Huff, J.; Moore, J.; Rall, D. The National Toxicology Program and preventive oncology. In: Estrin, N.; Estrin, N., editors. The Cosmetic Industry: Scientific and Regulatory Foundations. Marcel Dekker; New York: 1984. p. 647-76.

140. Rall DP. Relevance of animal experiments to humans. Environ Health Perspect. 1979; 32:297–30. [PubMed: 120250]

141. Rall DP, Hogan MD, Huff JE, Schwetz BA, Tennant RW. Alternatives to using human experience in assessing health risks. Annu Rev Public Health. 1987; 8:355–85. [PubMed: 3555527]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

142. Haseman JK, Huff JE, Zeiger E, McConnell EE. Comparative results of 327 chemical carcinogenicity studies. Environ Health Perspect. 1987; 74:229–35. [PubMed: 3691430]
143. Huff J, McConnell EE, Haseman JK, et al. Carcinogenesis studies: results of 398 experiments on 104 chemicals from the U.S. National Toxicology Program. Ann NY Acad Sci. 1988; 534:1–30. [PubMed: 3291703]
144. Rall DP. Laboratory animal toxicity and carcinogenesis testing. Underlying concepts, advantages and constraints. Ann NY Acad Sci. 1988; 534:78–83. [PubMed: 3291727]
145. Rall DP. Carcinogens in our environment. IARC Sci Publ. 1990; 104:233–9. [PubMed: 2228121]
146. Huff J, Haseman J, Rall D. Scientific concepts, value, and significance of chemical carcinogenesis studies. Annu Rev Pharmacol Toxicol. 1991; 31:621–52. [PubMed: 2064387]
147. Huff J, Hoel D. Perspective and overview of the concepts and value of hazard identification as the initial phase of risk assessment for cancer and human health. Scand J Work Environ Health. 1992; 18(suppl 1):83–9. [PubMed: 1411384]
148. Huff, J.; Rall, DP. Relevance to humans of carcinogenesis results from laboratory animal toxicology studies. In: Last, JM.; Wallace, RB., editors. Maxcy–Rosenau–Last's Public Health & Preventive Medicine. 13th ed. Appleton & Lange; Norwalk, CT: 1992. p. 433-440.p. 453-457.p. 1257
149. Fung VA, Huff J, Weisburger EK, Hoel DG. Predictive strategies for selecting 379 NCI/NTP chemicals evaluated for carcinogenic potential: scientific and public health impact. Fundam Appl Toxicol. 1993; 20:413–36. Erratum in: Fundam Appl Toxicol 1993;21:402. [PubMed: 8314458]
150. Huff J. Issues and controversies surrounding qualitative strategies for identifying and forecasting cancer causing agents in the human environment. Pharmacol Toxicol. 1993; 72(suppl 1):12–27. [PubMed: 8474975]
151. Rall DP. Shoe-leather epidemiology—the footpads of mice and rats: animal tests in assessment of occupational risks. Mt Sinai J Med. 1994; 61:504–8. [PubMed: 7838166]
152. Fung VA, Barrett JC, Huff J. The carcinogenesis bioassay in perspective: application in identifying human cancer hazards. Environ Health Perspect. 1995; 103:680–3. [PubMed: 7588478]
153. Rall DP. Can laboratory animal carcinogenicity studies predict cancer in exposed children? Environ Health Perspect. 1995; 103(suppl 6):173–5. [PubMed: 8549469]
154. Huff J, Weisburger E, Fung VA. Multicomponent criteria for predicting carcinogenicity: dataset of 30 NTP chemicals. Environ Health Perspect. 1996; 104(suppl 5):1105–12. [PubMed: 8933061]
155. Huff, J. Carcinogenesis results in animals predict cancer risks to humans. In: Wallace, RB., editor. Maxcy–Rosenau–Last's Public Health & Preventive Medicine. 14th ed. Appleton & Lange; Norwalk, CT: 1998. p. 543-550.p. 567-569.Chapter 23
156. Huff J. Long-term chemical carcinogenesis bioassays predict human cancer hazards. Issues, controversies, and uncertainties. Ann NY Acad Sci. 1999; 895:56–79. [PubMed: 10676409]
157. Rall DP. Laboratory animal tests and human cancer. Drug Metab Rev. 2000; 32:119–28. [PubMed: 10774768]
158. Tomatis L. Etiologic evidence and primary prevention of cancer. Drug Metab Rev. 2000; 32:129–37. [PubMed: 10774769]
159. Tomatis L, Huff J. Evolution of cancer etiology and primary prevention. Environ Health Perspect. 2001; 109:A458–60. [PubMed: 11675275]
160. Huff J. IARC monographs, industry influence, and upgrading, downgrading, and under-grading chemicals: a personal point of view. International Agency for Research on Cancer. Int J Occup Environ Health. 2002; 8:249–70. Erratum in: Int J Occup Environ Health. 2003;9:84. [PubMed: 12358081]
161. Tomatis, L.; Huff, J. Evolution of research on cancer etiology. In: Coleman, WB.; Tsongalis, GJ., editors. The Molecular Basis of Human Cancer: Genomic Instability and Molecular Mutation in Neoplastic Transformation. Humana Press Inc.; Totowa, NJ: 2002. p. 189-201.Chapter 9
162. Johnson FM, Huff J. Bioassay bashing is bad science. Environ Health Perspect. 2002; 110:A736–7. [PubMed: 12460809]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

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163. Melnick RL. Carcinogenicity and mechanistic insights on the behavior of epoxides and epoxide-forming chemicals. Ann NY Acad Sci. 2002; 982:177–89. [PubMed: 12562636]

164. Tomatis L. Primary prevention protects public health. Ann NY Acad Sci. 2002; 982:190–7. [PubMed: 12562637]

165. Maronpot RR, Flake G, Huff J. Relevance of animal carcino-genesis findings to human cancer predictions and prevention. Toxicol Pathol. 2004; 32(suppl 1):40–8. [PubMed: 15209402]

166. Huff J, Melnick R, Tomatis L, LaDou J, Teitelbaum D. Trichloroethylene and cancers in humans. Toxicology. 2004; 197:185–7. [PubMed: 15003328]

167. Melnick RL, Huff J. Testing toxic pesticides in humans: health risks with no health benefits. Environ Health Perspect. 2004; 112:A459–61. [PubMed: 15175190]

168. Tomatis L. Primary prevention of cancer in relation to science, sociocultural trends and economic pressures. Scand J Work Environ Health. 2005; 31:227–32. [PubMed: 15999576]

169. Melnick RL. A Daubert motion: a legal strategy to exclude essential scientific evidence in toxic tort litigation. Am J Public Health. 2005; 95(suppl 1):S30–4. [PubMed: 16030335]

170. Tomatis L. Experimental chemical carcinogenesis: fundamental and predictive rôle in protecting human health in the 1930s–1970s. Eur J Oncol. 2006; 11:5–13.

171. Tomatis L. Role of experimental and epidemiological evidence of carcinogenicity in the primary prevention of cancer. Ann Ist Super Sanita. 2006; 42:113–7. [PubMed: 17033130]

172. Huff J, Melnick RM, Gold LS, Slone TH, Manley NB, Ames BN. What are the real causes of cancer? An opinionated book review of "Misconceptions about the Causes of Cancer". Int J Occup Environ Health. 2006; 12:81–6.

173. Tomatis L. Identification of carcinogenic agents and primary prevention of cancer. Ann NY Acad Sci. 2006; 1076:1–14. [PubMed: 17119190]

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TABLE 1

Benzene: Organ/Tissue site Tumors Identified in Studies from the Ramazzini Foundation and the National Toxicology Program* in Seven Experiments Using Three Strains of Rats and Three Strains of Mice

	Sprague-Dawley Rats (Gavage)	Sprague-Dawley Rats (Inhalation)	Wistar Rats (Gavage)	Fisher* Rats (Gavage)	Swiss Mice (Gavage)	RF/J Mice (Gavage)	B6C3F1* Mice (Gavage)	
Zymbal gland	+	+	+	+	+	−	+	6
Mammary gland	[+]	[+]	−	−	+	+	+	5
Oral	+	+	+	+	−	−	−	4
Lung	−	−	−	−	+	+	+	3
Nasal cavities	+	[+]	+	−	−	−	−	3
Lymphoma	[+]	−	−	−	−	+	+	3
Liver	+	[+]	−	−	−	−	+	3
Forestomach	+	−	−	−	−	−	[+]	2
Skin	+	−	−	+	−	−	−	2
Uterus	−	−	−	−	−	−	+	1
Ovary	−	−	−	−	−	−	+	1
Harderian	−	−	−	−	−	−	+	1
Preputial gland	−	−	−	−	−	−	+	1
All malignant	+	+	+	+	+	+	+	7
Total sites	9	6	4	4	4	4	11	

+ = positive carcinogenic response; [+] = marginally increased carcinogenic response; − = no significant carcinogenic activity; sites listed in order of prevalence of responses per organ/tissue.

*The two strains utilized in the NTP studies.

Source: Huff.[56]

Wisconsin Statutes include the following language about conditional uses for cities from Act 67.

62.23(7)(de) Conditional use permits.

1. In this paragraph:

a. "Conditional use" means a use allowed under a conditional use permit, special exception, or other special zoning permission issued by a city, but does not include a variance.

b. "Substantial evidence" means facts and information, other than merely personal preferences or speculation, directly pertaining to the requirements and conditions an applicant must meet to obtain a conditional use permit and that reasonable persons would accept in support of a conclusion. (Substantial evidence needs to be provided in hard copy to provide to the Plan Commission and becomes part of the legal record.)

2.

a. If an applicant for a conditional use permit meets or agrees to meet all of the requirements and conditions specified in the city ordinance or those imposed by the city zoning board, the city shall grant the conditional use permit. Any condition imposed must be related to the purpose of the ordinance and be based on substantial evidence.

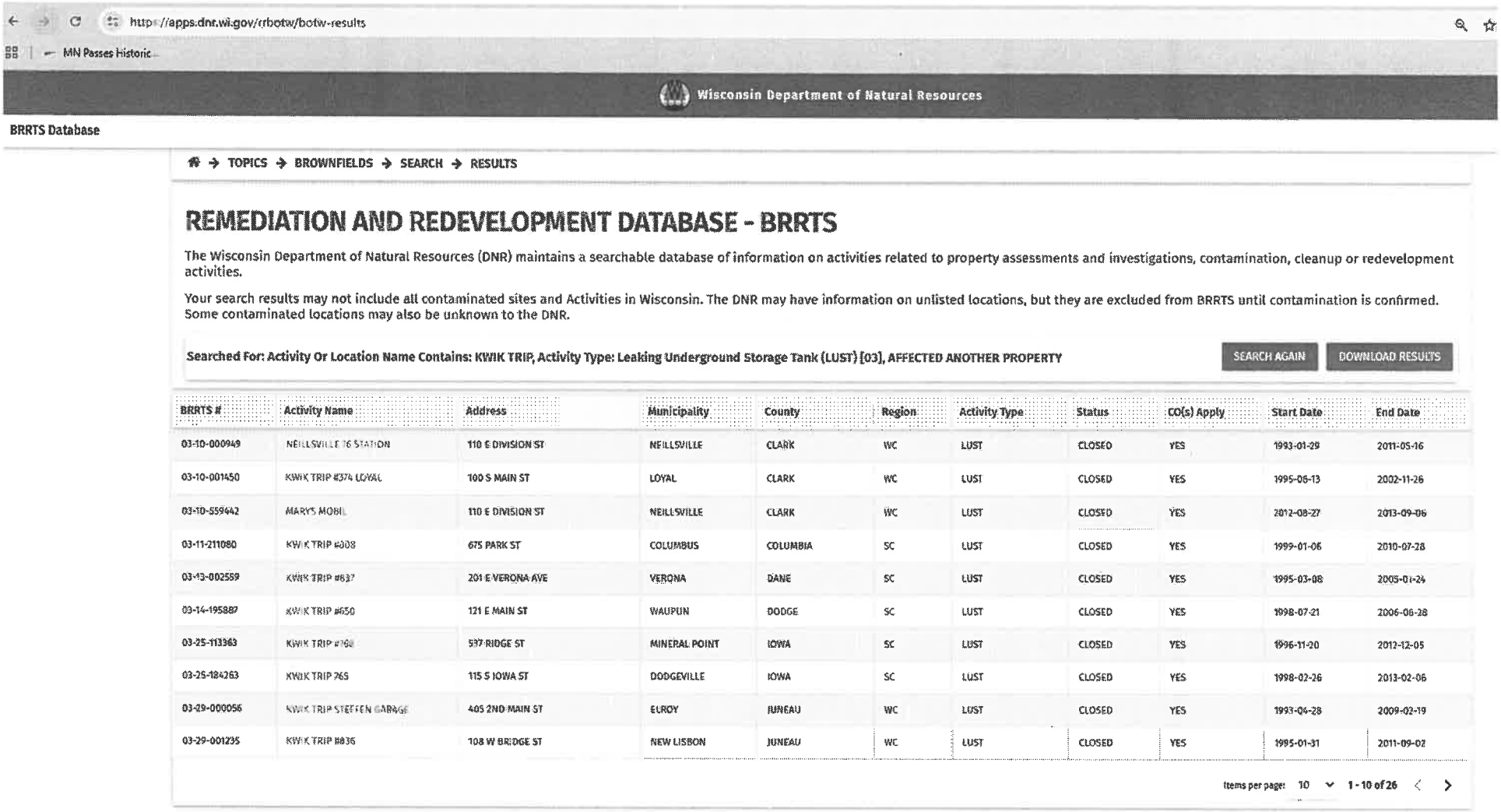
b. The requirements and conditions described under subd. 2. a. must be reasonable and, to the extent practicable, measurable and may include conditions such as the permit's duration, transfer, or renewal. The applicant must demonstrate that the application and all requirements and conditions established by the city relating to the conditional use are or shall be satisfied, both of which must be supported by substantial evidence. The city's decision to approve or deny the permit must be supported by substantial evidence.

3. Upon receipt of a conditional use permit application, and following publication in the city of a class 2 notice under ch. 985, the city shall hold a public hearing on the application.

4. Once granted, a conditional use permit shall remain in effect as long as the conditions upon which the permit was issued are followed, but the city may impose conditions such as the permit's duration, transfer, or renewal, in addition to any other conditions specified in the zoning ordinance or by the city zoning board.

5. If a city denies a person's conditional use permit application, the person may appeal the decision to the circuit court under the procedures contained in par. (e) 10. a., or if the decision is on an application for an approval, as defined in s. 781.10 (1) (a), under the procedures described in par. (e) 10. b.

Searched for Leaking Underground Storage Tanks (LUST) with a location name of Kwik Trip and affected another property: 26 sites



http://apps.dnr.wi.gov/rrbotw/botw-results

MN Passes Historic

Wisconsin Department of Natural Resources

BRRTS Database

TOPICS → BROWNFIELDS → SEARCH → RESULTS

REMEDIATION AND REDEVELOPMENT DATABASE - BRRTS

The Wisconsin Department of Natural Resources (DNR) maintains a searchable database of information on activities related to property assessments and investigations, contamination, cleanup or redevelopment activities.

Your search results may not include all contaminated sites and Activities in Wisconsin. The DNR may have information on unlisted locations, but they are excluded from BRRTS until contamination is confirmed. Some contaminated locations may also be unknown to the DNR.

Searched For: Activity Or Location Name Contains: KWIK TRIP, Activity Type: Leaking Underground Storage Tank (LUST) [03], AFFECTED ANOTHER PROPERTY

SEARCH AGAIN | DOWNLOAD RESULTS

BRRTS #	Activity Name	Address	Municipality	County	Region	Activity Type	Status	CO(s) Apply	Start Date	End Date
03-10-000949	NEILLSVILLE 76 STATION	110 E DIVISION ST	NEILLSVILLE	CLARK	WC	LUST	CLOSED	YES	1993-01-29	2011-05-16
03-10-001450	KWIK TRIP #374 LOYAL	100 S MAIN ST	LOYAL	CLARK	WC	LUST	CLOSED	YES	1995-06-13	2002-11-26
03-10-559442	MARYS MOBIL	110 E DIVISION ST	NEILLSVILLE	CLARK	WC	LUST	CLOSED	YES	2012-08-27	2013-09-06
03-11-211080	KWIK TRIP #308	675 PARK ST	COLUMBUS	COLUMBIA	SC	LUST	CLOSED	YES	1999-01-06	2010-07-28
03-13-002559	KWIK TRIP #637	201 E VERONA AVE	VERONA	DANE	SC	LUST	CLOSED	YES	1995-03-08	2005-01-24
03-14-195887	KWIK TRIP #650	121 E MAIN ST	WAUPUN	DODGE	SC	LUST	CLOSED	YES	1998-07-21	2006-06-28
03-25-113363	KWIK TRIP #768	537 RIDGE ST	MINERAL POINT	IOWA	SC	LUST	CLOSED	YES	1996-11-20	2012-12-05
03-25-184263	KWIK TRIP 265	115 S IOWA ST	DODGEVILLE	IOWA	SC	LUST	CLOSED	YES	1998-02-26	2013-02-06
03-29-000056	KWIK TRIP STEFFEN GARAGE	405 2ND MAIN ST	ELROY	JUNEAU	WC	LUST	CLOSED	YES	1993-06-28	2009-02-19
03-29-001235	KWIK TRIP #836	108 W BRIDGE ST	NEW LISBON	JUNEAU	WC	LUST	CLOSED	YES	1995-01-31	2011-09-02

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BRRTS data comes from various sources, both internal and external to the DNR. There may be omissions and errors in the data and delays in updating new information.

Wisconsin Bureau for Remediation and Redevelopment Tracking System (BRRTS) https://dnr.wisconsin.gov/topic/Brownfields/botw.html

Searched for Leaking Underground Storage Tanks (LUST) with a location name of Kwik Trip: 269 sites

Wisconsin Department of Natural Resources

BRRTS Database

TOPICS → BROWNFIELDS → SEARCH → RESULTS

REMEDIATION AND REDEVELOPMENT DATABASE - BRRTS

The Wisconsin Department of Natural Resources (DNR) maintains a searchable database of information on activities related to property assessments and investigations, contamination, cleanup or redevelopment activities.

Your search results may not include all contaminated sites and Activities in Wisconsin. The DNR may have information on unlisted locations, but they are excluded from BRRTS until contamination is confirmed. Some contaminated locations may also be unknown to the DNR.

Searched For: Activity Or Location Name Contains: KWIK TRIP, Activity Type: Leaking Underground Storage Tank (LUST) [03]

SEARCH AGAIN | DOWNLOAD RESULTS

BRRTS #	Activity Name	Address	Municipality	County	Region	Activity Type	Status	CO(s) Apply	Start Date	End Date
03-01-000278	KWIK TRIP #316	302 MAIN ST	ADAMS	ADAMS	WC	LUST	CLOSED	YES	1990-04-30	2003-07-18
03-01-000532	KWIK TRIP #774 ADAMS	160 S MAIN	ADAMS	ADAMS	WC	LUST	CLOSED	YES	1991-01-02	2000-01-10
03-01-578658	KWIK TRIP STORE #921	1610 STH 13	PRESTON	ADAMS	WC	LUST	CLOSED	YES	2017-01-06	2019-04-29
03-02-000724	MIDLAND TOWN MART - KWIK TRIP 163 CAR WASH	109 6TH ST E	ASHLAND	ASHLAND	NO	LUST	CLOSED	YES	1994-01-11	2003-02-04
03-02-000975	QUEARM OIL CO - KWIK TRIP #163	105 6TH ST W	ASHLAND	ASHLAND	NO	LUST	CLOSED	YES	1995-11-14	2003-02-28
03-02-579250	MIDLAND TOWN MART - KWIK TRIP 163 CAR WASH	109 6TH ST E	ASHLAND	ASHLAND	NO	LUST	CLOSED	YES	2017-04-20	2018-09-28
03-02-579510	COCA COLA BOTTLING PLANT (FMR) KWIK TRIP 110	1800 LAKESHORE DR W	ASHLAND	ASHLAND	NO	LUST	CLOSED	NO	2017-06-05	2017-11-27
03-03-539603	KWIK TRIP #602	424 2ND ST	CHETEK	BARRON	NO	LUST	CLOSED	NO	2004-12-30	2005-02-03
03-03-543961	KWIK TRIP STORE #749	1821 S MAIN ST	RICE LAKE	BARRON	NO	LUST	CLOSED	YES	2005-09-09	2006-08-21
03-03-553309	ROMAINE QUINN PROPERTIES	1821 S MAIN ST	RICE LAKE	BARRON	NO	LUST	CLOSED	YES	2009-01-22	2010-12-15

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Benzene

From EPA at https://www.epa.gov/sites/default/files/2016-09/documents/benzene.pdf

71-43-2

Hazard Summary

Benzene is found in the air from emissions from burning coal and oil, gasoline service stations, and motor vehicle exhaust. Acute (short-term) inhalation exposure of humans to benzene may cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract irritation, and, at high levels, unconsciousness. Chronic (long-term) inhalation exposure has caused various disorders in the blood, including reduced numbers of red blood cells and aplastic anemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing fetus have been observed in animal tests. Increased incidence of leukemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene. EPA has classified benzene as known human carcinogen for all routes of exposure.

Please Note: The main sources of information for this fact sheet are the Agency for Toxic Substances and Disease Registry's (ATSDR's) Toxicological Profile for Benzene (1) and EPA's Integrated Risk Information System (IRIS) (4), which contains information on the health effects of benzene including the unit cancer risk for inhalation exposure.

Uses

- Benzene is used as a constituent in motor fuels; as a solvent for fats, waxes, resins, oils, inks, paints, plastics, and rubber; in the extraction of oils from seeds and nuts; and in photogravure printing. It is also used as a chemical intermediate. Benzene is also used in the manufacture of detergents, explosives, pharmaceuticals, and dyestuffs. (1,2,6)

Sources and Potential Exposure

- Individuals employed in industries that manufacture or use benzene may be exposed to the highest levels of benzene. (1)
- Benzene is found in emissions from burning coal and oil, motor vehicle exhaust, and evaporation from gasoline service stations and in industrial solvents. These sources contribute to elevated levels of benzene in the ambient air, which may subsequently be breathed by the public. (1)
- Tobacco smoke contains benzene and accounts for nearly half the national exposure to benzene. (1)
- Individuals may also be exposed to benzene by consuming contaminated water. (1)

Assessing Personal Exposure

- Measurement of benzene in an individual's breath or blood or the measurement of breakdown products in the urine (phenol) can estimate personal exposure. However, the tests must be done shortly after exposure and are not helpful for measuring low levels of benzene. (1)

Health Hazard Information

Acute Effects:

- Coexposure to benzene with ethanol (e.g., alcoholic beverages) can increase benzene toxicity in humans. (1)

- Neurological symptoms of inhalation exposure to benzene include drowsiness, dizziness, headaches, and unconsciousness in humans. Ingestion of large amounts of benzene may result in vomiting, dizziness, and convulsions in humans. (1)
- Exposure to liquid and vapor may irritate the skin, eyes, and upper respiratory tract in humans. Redness and blisters may result from dermal exposure to benzene. (1,2)
- Animal studies show neurologic, immunologic, and hematologic effects from inhalation and oral exposure to benzene. (1)
- Tests involving acute exposure of rats, mice, rabbits, and guinea pigs have demonstrated benzene to have low acute toxicity from inhalation, moderate acute toxicity from ingestion, and low or moderate acute toxicity from dermal exposure. (3)
- The reference concentration for benzene is 0.03 mg/m3 based on hematological effects in humans. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive groups) that is likely to be without appreciable risk deleterious noncancer effects over a lifetime. (4)

Chronic Effects (Noncancer):

- Chronic inhalation of certain levels of benzene causes disorders in the blood in humans. Benzene specifically affects bone marrow (the tissues that produce blood cells). Aplastic anemia (a risk factor for acute nonlymphocytic leukemia), excessive bleeding, and damage to the immune system (by changes in blood levels of antibodies and loss of white blood cells) may develop. (1)
- In animals, chronic inhalation and oral exposure to benzene produces the same effects as seen in humans. (1)
- Benzene causes both structural and numerical chromosomal aberrations in humans. (1)
- EPA has established an oral Reference Dose (RfD) for benzene of 0.004 milligrams per kilogram per day (mg/kg/d) based on hematological effects in humans. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk, but rather a reference point to gauge the potential for effects. At exposures increasingly greater than the RfD, the potential for adverse health effects increases. Lifetime exposure above the RfD does not imply that an adverse health effect would necessarily occur. (4)
- EPA has established a Reference Concentration (RfC) of 0.03 milligrams per cubic meter (0.03 mg/m3) for benzene based on hematological effects in humans. The RfC is an inhalation exposure concentration at or below which adverse health effects are not likely to occur. It is not a direct estimator of risk, but rather a reference point to gauge the potential for effects. At lifetime exposures increasingly greater than the reference exposure level, the potential for adverse health effects increases. (4)

Reproductive/Developmental Effects:

- There is some evidence from human epidemiological studies of reproductive and developmental toxicity of benzene, however the data do not provide conclusive evidence of a link between exposure and effect. (4) Animal studies have provided limited evidence that exposure to benzene may affect reproductive organs, however these effects were only observed at exposure levels over the maximum tolerated dose. (4)
- Adverse effects on the fetus, including low birth weight, delayed bone formation, and bone marrow damage, have been observed where pregnant animals were exposed to benzene by inhalation.(4)

Cancer Risk:

- Increased incidence of leukemia (cancer of the tissues that form white blood cells) has been observed in humans occupationally exposed to benzene. (1,4)
- EPA has classified benzene as a Group A, known human carcinogen. (4)
- EPA uses mathematical models, based on human and animal studies,to estimate the probability of a person developing cancer from breathing air containing a specified concentration of a chemical. EPA calculated a range of 2.2×10^{-6} to 7.8×10^{-6} as the increase in the lifetime risk of an individual who is continuously exposed to 1 µg/m3 of benzene in the air over their lifetime.

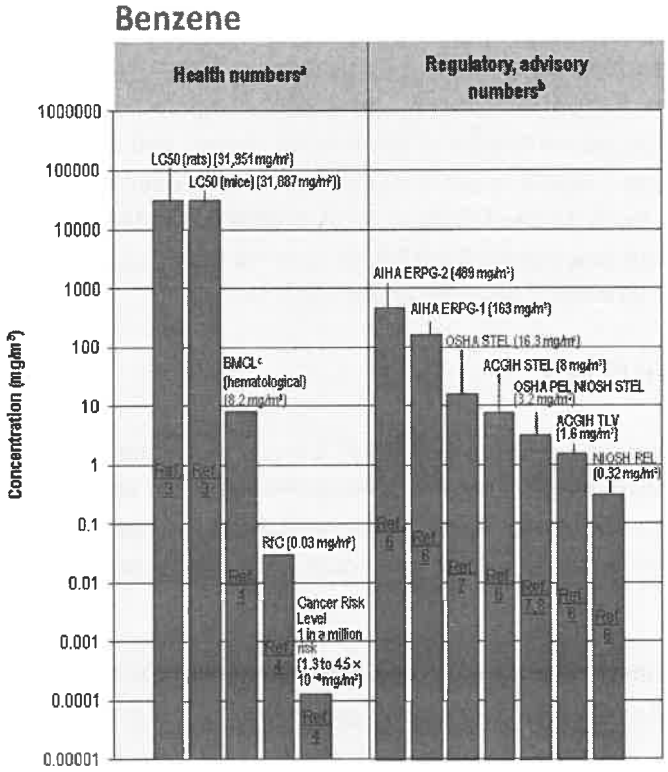
- EPA estimates that, if an individual were to continuously breathe the air containing benzene at an average of 0.13 to 0.45 µg/m^3 ($1.3x10^{-4}$ to $4.5x^{-4}$ mg/m^3) over his or her entire lifetime, that person would theoretically have no more than a one-in-a-million increased chance of developing cancer as a direct result of continuously breathing air containing this chemical. Similarly, EPA estimates that continuously breathing air containing 1.3 to 4.5 µg/m^3 ($1.3x10^{-3}$ to $4.5x10^{-3}$ mg/m^3) would result in not greater than a one-in-a-hundred thousand increased chance of developing cancer, and air containing 13 to 45 µg/m^3 (1.3×10^{-2} to 4.5×10^{-2} mg/m^3) would result in not greater than a one-in-ten thousand increased chance of developing cancer. For a detailed discussion of confidence in the potency estimates, please see IRIS.(4)
- EPA has calculated an oral cancer slope factor ranging from 1.5×10^{-2} to 5.5×10^{-2} $(mg/kg/d)^{-1}$ that is an extrapolation from inhalation dose-response data. (4)

Physical Properties

- The chemical formula for benzene is C_6H_6, and it has a molecular weight of 78.11 g/mol. 4) Benzene occurs as a volatile, colorless, highly flammable liquid that dissolves easily in water. (1,7)
- Benzene has a sweet odor with an ASTDR reported odor threshold of 1.5 ppm (5 mg/m^3).
- The vapor pressure for benzene is 95.2 mm Hg at 25 °C, and it has a log octanol/water partition coefficient (log Kow) of 2.13. (1)

Conversion Factors (only for the gaseous form):
To convert concentrations in air (at 25°C) from ppm to mg/m^3: mg/m^3 = (ppm) × (molecular weight of the compound)/(24.45). For benzene: 1 ppm = 3.19 mg/m^3. To convert concentrations in air from µg/m^3 to mg/m^3: mg/m^3 = (µg/m^3) × (1 mg/1,000 µg).

Health Data from Inhalation Exposure



ACGIH STEL--American Conference of Governmental and Industrial Hygienists' short-term exposure limit.
ACGIH TLV--American Conference of Governmental and Industrial Hygienists' threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.
AIHA ERPG--American Industrial Hygiene Association's emergency response planning guidelines. ERPG 1 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor; ERPG 2 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed up to one hour without experiencing or developing irreversible or other serious health effects that could impair their abilities to take protective action. The American Industrial Hygiene Association's detection and recognition odor thresholds for benzene are 61 ppm and 97 ppm, respectively.
LC_{50} (Lethal Concentration$_{50}$)--A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.
NIOSH REL--National Institute of Occupational Safety and Health's recommended exposure limit; NIOSH-recommended exposure limit for an 8- or 10-h time-weighted-average exposure and/or ceiling.
NIOSH STEL--NIOSH's short term exposure limit; NIOSH recommended exposure limit for a 15-minute period.
OSHA PEL--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.
OSHA STEL--Occupational Safety and Health Administration's short-term exposure limit.
The health and regulatory values cited in this graph were obtained in April 2009.
[a] Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.
[b] Regulatory numbers are values that have been incorporated in Government regulations, while advisory numbers

are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH, ACGIH, and AIHA numbers are advisory.

[c] The BMCL (statistical lower confidence limit on the concentration at the benchmark concentration, which is the concentration producing a specified change in a response rate that is considered a critical effect) was used as the point of departure for the RfC derivation. The BMCL for benzene is for hematological effects (reduction in absolute lymphocyte count) in humans (4).

Summary created in April 1992, updated in January 2000 and January 2012.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Benzene. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. 2007.
2. M. Sittig. Handbook of Toxic and Hazardous Chemicals and Carcinogens. 2nd ed. Noyes Publications, Park Ridge, NJ. 1985.
3. U.S. Department of Health and Human Services. Registry of Toxic Effects of Chemical Substances (RTECS, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.
4. U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Benzene. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 2009.
5. California Environmental Protection Agency(CalEPA). Air Toxics Hot Spots Program Risk Assessment Guidelines: Part III. Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels. SRP Draft. Office of Environmental Health Hazard Assessment, Berkeley, CA. 1999.
6. The Merck Index.An Encyclopedia of Chemicals, Drugs, and Biologicals. 11th ed. Ed. S. Budavari. Merck and Co. Inc., Rahway, NJ. 1989.
7. American Conference of Governmental Industrial Hygienists (ACGIH). 1999 TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents. Biological Exposure Indices. Cincinnati, OH. 1999.
8. 8. Occupational Safety and Health Administration (OSHA). Occupational Safety and Health Standards, Toxic and Hazardous Substances. Code of Federal Regulations. 29 CFR 1910.1000. 1998.
9. National Institute for Occupational Safety and Health (NIOSH). Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention. Cincinnati, OH. 1997.