Poster Hospital PRACTICAL DESIGN GUIDELINES FOR CONCEPT AND DATA GRAPHICS ON A LARGE CANVAS

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Vancouver, Canada

Poster Hospital http://mkweb.bcgsc.ca/poster.design/

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POSTER CHILD OF SCIENCE

A poster is your first opportunity to organize and communicate your reasearch to members outside of your lab. It will help you to practise telling and "drawing" your science story and its design should be based on its concepts, themes and transitions.

Most posters are bad not because they are ugly (they are) but because they fail to present concisely what was done and, more importantly, why it was done. Most posters have too much on them. Less is more: get to the point, then stop.

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ALL SCIENCE DESERVES EXCELLENT EXPLANATIONS

Explain quickly and clearly. Motivate why the work was done-what is the cost of not doing it?

The poster is your prop. In most settings, you will be there to present it. Match its content to the story you will tell

Conneed total truly hulter ONLY YOU CAN STOP orrestonian at theread but und 17 algorithm POSTER DUMPSTER FIRES

Clip art, pie charts, bullet points, boxes around text, background fills and gradients. Only you can stop it.

Maintain good Gestalt-similar shapes and colors will form groups. Use them to encode real-world relationships and be on the lookout for unintended accidental groupings.

EVERYTHING IS IMPORTANT, BUT SOME THINGS ARE MORE IMPORTANT THAN OTHERS. Establish a visual hierarchy by emphasizing your hypothesis, conclusion and the key points that connect them. Relegate protocols, technical methods, and other minutias to the bottom of the poster. Always be mindful of what the reader needs to know to understand onough to ask insightful quastions and frontfloed this information.

"here". This that as a growthat a many which is even of peri-Don't by to be available therein or with - most afternate do cot manufact they's would the joiners contaction. In particular and in particular in the second se

Use figure titles to explain trends, not

merely to specify the axes.



A Don't tell the reader what is obvious: "a linear fit. to a scatter plot" is redundant. Don't tell me what I'm seeing --interpret the figure instead. Italicize variables in fit diagnostics and use shaded bands for confidence intervals. Highlight regions of interests with a solid color (or grey), not outlines.

Alone Genheit to askablish layout proportions and on out temphasian putters with from One or two much distributions has allocated, but has many will be the positive left, a lat-

USE SMALL MULTIPLES

Use ink sparingly to make compact figures legible-dense is not necessarily crowded. Explain an encoding once and reuse it. Create a visual key for complex encodings and choose graphical explanations over text.

Avoid visual complications that are not relevant-for example, color blending can create distracting intersections of color. Superimpose white outlines to emphasize shapes with an opaque fill.

> FORCE LINE BREAKS that split a sentence into noun phrases or offer a natural pause. such as all a comma or a period. Batarice layout by shortening sentences -- there are many ways to say something and some ways are easier to typeset. Do not let a template bully you into using a specific column width. Change proportions to suit content

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Look for opportunities to include key observations (-) and explanations in the figure-don't leave it to the main text, where it may be far from the graphic. Emphasize what guantities are important -- anticipate the reader's guestions and answer them.

Use hupberedtable german agentration in contribution, multiple areasi plape. A wall placed sorvitet or taket care convert literary or indicate the purpose of test in g. Interpret suggest a legerid.

MAINTAIN AND CONTROL PROPORTIONS

This poster is 16" x 12" (1152 × 864 pt), uses Helvetica Neue with a 5. 8, 13, 21, 34, 55 pt scale ladder and is legible on most screens.

Sans-serif is clearer than serif at small sizes and suitable for modest amounts of copy.

Keep line length short and hyphenate instead

inch Lassa When displayed hull-headst on a 27" 18/9 monitor, distances are preserved within 5%. points L 23.5...... minut.

A point is a unit of size used in typography Without a physical also they lose their mean ing, but can provide a helpful scale.



AVOID OBVIOUS HEADINGS such as "references". Citations can be set in a block of text, with bold numbers like this 1. R. Bringhurst, Elements of Typographical Style. 4th ed (2012) and 2. W. Strunk Jr., Elements of Style (1918). Liniess a specific obtation style is required, use a compact style that also includes the 5tle.

B TD ARROWS imply U. a relationship



TABULATE plots and test searclessly with a column or row for explanations thaticize test with care and look for unimendod itakes in subscripts.



of fully justifying.

mean, n = 5). Report P-values with effect sizes. or confidence intervals. A statistically significant observation isn't necessarily of biological interest.

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Categorical variables in bar charts do not need

an explicit axis. Specify sample sizes and what

error bars represent (e.g. standard error of

you are the steward of your science

Show your passion for the subject.

Be a good explainer.

Resist visual tropes, fluff and garnish.

design is a process

Thank you for your submissions!

My redesigns are not the "best and only" options. They're merely better options.

When I first look at a figure, I typically know what needs fixing but I don't always know how to fix it.

design is a set of choices

When you speak, you generally know

what / why / why now

you're saying something.

It's the same with design except time is replaced by space.

you're 90% of the tool

Good design is never due to software expertise.

Know your software enough to make your ideas possible.

Learning tool XYZ will not make you a better communicator.



emphasize data

make other elements visually subordinate









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conclude first and explain early

don't squirrel important information into the least accessible part of the poster

Exploring the impact of financial insecurity on adolescent health behaviors: How has COVID-19 influenced screen time, physical activity and diet quality?

lyoma Y. Edache¹, MSc; Mark Pitblado²; Sarah M. Hutchinson³, PhD and Louise C. Mâsse¹, PhD

School of Population and Public Health¹; Department of Microbiology & Immunology², Department of Pediatrics³, University of British Columbia

BACKGROUND

The COVID-19 pandemic disrupted Canadian families' daily routines and social interactions due to government-mandated physical distancing restrictions. Three out of 10 Canadians report that COVID-19 has negatively impacted their ability to meet financial obligations¹. Health behaviors have also been impacted as physical activity has decreased while screen time and food consumption have increased². Cumulatively, these disruptions have increased parent and adolescent emotional strain².

OBJECTIVES

 To investigate the link between financial security, parent and adolescent emotional wellbeing and adolescents' health behaviors during the COVID-19 pandemic.

METHODS

Analytical sample and measures

Parents and grade 7 student pairs (N=355) completed an online survey in May –June 2020, assessing family financial security, parent and teen emotional wellbeing (self-esteem, optimism, worry and depression) and teen health behaviors (screen time and physical activity).

Adolescents completed three 24-hour dietary recalls using the ASA24 platform. Dietary quality was computed using the Healthy Eating Index (HEI)³, which evaluates compliance of reported intake with national dietary recommendations.

Statistical analysis

Structural equation modelling was used to examine linear relationships using the Stata software (version 15.1).

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Weight, its, mean (50)	57 61 (11 20)
nei score, mean (50)	37.01 (11.20)
Screen time, hours/week, Mean (SD)	11.84 (7.60)
Physically active days/week, n (%)	10110-0010
0 days	31 (9.1)
1-3 days	120 (35.3)
4-6 days	118 (34.8)
Parent demograp	hic profile
Age, Years, Mean (SD)	45.95 (5.42)
Sex = Female, n (%)	285 (79.7)
Ethnicity (%)	
African American	5(1.4)
Caucasian	126 (36.3)
Chinese	93 (26.8)
Other	48 (13.9)
South Asian	51 (14.7)
South East Asian	24 (6.9)
Annual household income (%)	
\$100,000 or higher	169 (48.2)
\$80,000 to \$99,999	29 (8.3)
\$70,000 to \$79,999	16 (4.6)
\$50,000 to 569,999	28 (8.0)
Less than \$50,000	53 (15.1)
Marital status, n (%)	
Divorced	32 (9.1)
Married or common-law	301 (85.8)
Single	13 (3.7)
Prefer not to answer	5(1.4)
Education, n (%)	and the first
High school degree or less	27 (7.7)
Went to college	122 (34.8)
Bachelors degree or above	202 (57.5)
Employment (%)	3134125042
A homemaker	40 (11.4)
Currently not working	25 (7.4)

Employed for wages-full time

Employed for wages-part time

Self-employed

181 (51.7)

43 (12.3)

42 (12)

Table 1. Characteristics of participants (N=355)

KEY FINDINGS

IMPLICATIONS

Study results highlight the role of emotional wellbeing in the pathway through which financial security impacts adolescent health behaviors. As public policy addressing financial security may indirectly improve adolescent health behaviors, our findings will inform COVID-19 public health priorities — specifically, family-based efforts to support and promote adolescent health behaviors and emotional wellbeing.

- 27 Carrier, N., Sedovini, K., Leek, A., Hindrid, Y., Haldi, M., Hei, E. W., & Heimer, J. (2020). Nutrients, 12(8), 2352.
- Gueether, P. M., Reedy, J., & Kreth, Smith, S. M. (2008). Journal of the American Dieterlo Association, 108(11), 1896–1921.

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STATISTICAL ANALYSIS

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- 1 Statistics Canada's March 2020 Canadian Perspectives Survey Series
- 2 Carroll, N., Sadowski, A., Laila, A., Hruska, V., Nixon, M., Ma, D. W., & Haines, J. (2020), Nutrients, 12(8), 2352.
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Characteristics of participants (n = 355).

ADOLESCENT PROFILE				
age (years)		13.01	0.12	μ, σ
sex (female)		199	54	n, %
weight (lbs)		106.6	22.9	μ, σ
HEI score		57.6	11.2	μ, σ
screen time, hours/week		11.8	7.6	μ, σ
physically active days/week				n, %
0 days		31	9.1	
1-3 days		120	35	
4-6 days		118	35	
PARENT PROFILE				
age (vears)		45.9	5.4	μ, σ
sex (female)		285	80	n %
ethnicity	-	200	00	n %
African American		5	14	11, 70
Caucasian		126	36.3	
Chinese		93	26.8	
South Asian		51	14.7	
South East Asian		24	6.9	
other		48	13.9	
	_			
annual household income				n, %
≥ \$100,000		169	48.2	
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\$50,000 to \$69,999		28	8.0	
< \$50,000		55	15.1	
marital status				n, %
married or common-law		301	85.8	
divorced		32	9.1	
single		13	3.7	
prefer not to answer		5	1.4	
education				n. %
bachelors degree or above		202	57.5	
went to college		122	34.8	
high school degree or less		27	7.7	
employment				n, %
full-time wage		181	52	
part-time wage		43	12	
self-employed		42	12	
homemaker		40	11	
currently not working		26	7	

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Associations between financial security, emotional wellbeing and adolescent health behaviors.

Exploring the impact of fin influenced screen time, phy

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School of Population and Public Health¹; Depa

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Table 1. Characteristics of participants (N=355)

Adolescent demogra	phic profile
Age, Years, Mean (SD)	13.01 (0.12)
Sex = Female, n (%)	199 (54.0)
Weight, Ibs, Mean (SD)	106.56 (22.90)
HEI score, Mean (SD)	57.61 (11.20)
Screen time, hours/week, Mean (SD)	11.84 (7.60)
Physically active days/week, n (%)	
0 days	31 (9.1)
1-3 days	120 (35.3)
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Parent demograp	hic profile
Age, Years, Mean (SD)	45.95 (5.42)
Sex = Female, n (%)	285 (79.7)
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African American	5 (1.4)
Caucasian	126 (36.3)
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Less than \$50,000	53 (15.1)
Marital status, n (%)	
Divorced	32 (9.1)
Married or common-law	301 (85.8)
Single	13 (3.7)
Prefer not to answer	5 (1.4)
Education, n (%)	
High school degree or less	27 (7.7)
Went to college	122 (34.8)
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self-employed	42	12	
homemaker	40	11	
currently not working	26	7	

	Yes (%)	В	SE	Odds ratio	(95% CI)	Р
Are you concerned about Ne	wcastle disease? (n = 398)"					
Age	A 99					0.007
<24 years	5(17.2)	0	9 1	1.00	244 2	
25-34 years	10(28.6)	1.33	0.86	3.80	(0.8 - 27.6)	
35-44 years	22(36.1)	1.37	0.82	3.92	(0.9 - 26.9)	
45-54 years	42(43.8)	1.85	0.79	6.35	(1.7 - 42.0)	
55-64 years	36(50.0)	203	0.80	7.62	(1.9-51.5)	
+65 years	63 (60.0)	2.24	0.79	9.39	(2.4-62.4)	
State	* 351					0.042
SA/WA	27(31.8)	0	14	1.00		
NSW	58(47.5)	0.59	0.35	1.80	(0.9-3.6)	
QLD	45(47.4)	0.49	0.36	1.63	(0.8 - 3.3)	
TAS	15(33.3)	-0.23	0.45	0.79	(0.3 - 1.9)	
VIC	29(64.4)	1.04	0.45	2.82	(1.2-6.9)	
Do you keep a written recon	d of treatments given to your	birds? (n - 398)"			1.28	
Years owning poultry	N N 1991	22 X				0.006
1-5 years	19(51.4)	0	94	1.00	(7 44)	
6-15 years	21 (28.4)	-1.54	0.51	0.21	(0.1-0.6)	
16-29 years	33(45.2)	-0.75	0.49	0.47	(0.2 - 1.2)	
+30 years	70(33.2)	-1.39	0.45	0.25	(0.1-0.6)	
Sex	22. ISA				1.20	0.066
Female	38(44.2)	0	94	1.00	22 <u>4</u> 2	
Male	102(33.9)	-0.56	0.30	0.57	(0.3 - 1.0)	
Have you contacted a veteri	narian in the past 12 months	for the health of yo	ur birds?(n – 398)		138 II	
Years owning poultry	35 7		18 IV			0.006
1-5 years	17 (45.9)	0	84	1.00	2 4	
6-15 years	22(29.7)	-0.71	0.51	0.49	(0.2 - 1.3)	0.017
16-29 years	15(20.5)	-1.44	0.54	0.24	(0.1 - 0.7)	
+30 years	38(18.0)	-1.49	0.48	0.23	(0.1 - 0.6)	
Sex	2011 (Sec.)					
Female	34(39.5)	0	¥	1.00	249) 	
Male	54(17.9)	-0.79	0.33	0.46	(0.2 - 0.9)	
State	20 TEX					0.040
SA/WA	13(15.3)	0	<u> </u>	1.00	322	
NSW	37(30.3)	1.01	0.46	2,75	(1.1-6.6)	
OLD	17(17.9)	0.18	0.47	1.19	(0.5 - 3.0)	
TAS	11(24.4)	0.91	0.52	2.49	(0.9-7.1)	
VIC	14(31.1)	1.03	0.51	2.79	(1.0-7.8)	

* Number of exhibitors contributing to the regression model analysis.

	Р	Yes (%)	fβ	SEβ	OR	95% CI
Are you concerned		46.0				
about Newcastie atsease:						
Age (years)	0.007					
≤24 ‡		17.2				
25-34		28.6	1.33	0.86	3.8	0.8 - 27.6
35-44		36.1	1.37	0.82	3.9	0.9 - 26.9
45-54		43.8	1.85	0.79	6.3	1.7 - 42.0
55-64		50.0	203	0.80	7.6	1.9 - 51.5
≥65		60.0	2.24	0.79	9.4	2.4 - 62.4
State	0.007					
SA/WA		31.8				
NSW		47.5	0.59	0.35	1.8	0.9 - 3.6
QLD		47.4	0.49	0.36	1.6	0.8 - 3.3
TAS		33.3	-0.23	0.45	0.8	0.3 - 1.9
VIC		64.4	1.04	0.45	2.8	1.2 - 6.9
Do you keep a written record						
of treatments given to your birds?		35.2				
Years owning poultry	0.006					
1-5		51.4				
6-15		28.4	-1.54	0.51	0.2	0.1 - 0.6
16-29		45.2	-0.75	0.49	0.5	0.2 - 1.2
≥30		33.2	-1.39	0.45	0.2	0.1 - 0.6
Ser	0.066					
Female	0.000	44.2				
Male		33.9	-0.56	0.30	0.6	0.3 - 1.0
Have you contacted a veterinarian in the						
<i>past 12 months for the health of your birds?</i>		35.2				
Vars owning poultry	0.006					
1 5	0.000	45.0				
1-5 6 15		43.9	0.71	0.51	0.5	0 2 1 2
0-13		29.7	-0./1	0.51	0.5	0.2 - 1.3
>30		20.5	-1.44	0.54	0.2	0.1 - 0.7
230	0.017	10.0	-1.49	0.48	0.2	0.1 - 0.6
Sex	0.01/	20 5				
Female		39.5	0.70	0.22	0.5	0.2 0.0
Male		17.9	-0./9	0.33	0.5	0.2 - 0.9
State	0.040					
SA/WA		15.3				
NSW		30.3	1.01	0.46	2.8	1.1 - 6.6
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TAS		24.4	0.91	0.52	2.5	0.9 - 7.1
VIC		31.1	1.03	0.51	2.8	1.0 - 7.8

† Out of n = 398 survey participants.

 \ddagger For first factor level, β = 0, OR = 1, SE β and 95% CI are not defined.

Exploring the impact of financial insecurity on adolescent health behaviors: How has COVID-19 influenced screen time, physical activity and diet quality?

199

106.6

24

48

28

5

26 7

Iyoma Y. Edache¹, MSc Mark Pitblado² Sarah M. Hutchinson³, PhD Louise C. Mâsse¹, PhD

ADOLESCENT PROFILE

physically active days/week

PARENT PROFILE

African American

annual household income

\$80,000 to \$99,999

\$70,000 to \$79,999

\$50,000 to \$69,999

married or common-law

bachelors degree or above

high school degree or less

prefer not to answer

age (years)

sex (female)

weight (lbs)

0 days

1-3 days

4-6 days

age (years)

sex (female)

ethnicity

Caucasian

South Asian South East Asian

≥ \$100,000

< \$50,000

divorced

education

employment

went to college

full-time wage

part-time wage

currently not working

self-employed

homemaker

single

marital status

Chinese

other

¹School of Population and Public Health ²Department of Microbiology & Immunology ³Department of Pediatrics, University of British Columbia

OBJECTIVES

To investigate the link between financial security, parent and adolescent emotional wellbeing and adolescents' health behaviors during the COVID-19 pandemic.

IMPLICATIONS

Study results highlight the role of emotional wellbeing in the pathway through which financial security impacts adolescent health behaviors. As public policy addressing financial security may indirectly improve adolescent health behaviors, our findings will inform COVID-19 public health priorities - specifically, family-based efforts to support and promote adolescent health behaviors and emotional wellbeing.

BACKGROUND

The COVID-19 pandemic disrupted Canadian families' daily routines and social interactions due to government-mandated physical distancing restrictions. Three out of 10 Canadians report that COVID-19 has negatively impacted their ability to meet financial obligations¹. Health behaviors have also been impacted as physical activity has decreased while screen time and food consumption have increased2. Cumulatively, these disruptions have increased parent and adolescent emotional strain².

ANALYTICAL SAMPLE AND MEASURES

Parents and grade 7 student pairs (n = 355) completed an online survey in May -June 2020, assessing family financial security, parent and teen emotional wellbeing (self-esteem, optimism, worry and depression) and teen health behaviors (screen time and physical activity). Adolescents completed three 24-hour dietary recalls using the ASA24 platform. Dietary quality was computed using the Healthy Eating Index (HEI)³, which evaluates compliance of reported intake with national dietary recommendations.

STATISTICAL ANALYSIS

UBC

Structural equation modelling was used to examine linear relationships using the Stata software (version 15.1).

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- 2 Carroll, N., Sadowski, A., Laila, A., Hruska, V., Nixon, M., Ma, D. W., & Haines, J. (2020), Nutrients, 12(8), 2352.
- 3 Guenther, P. M., Reedy, J., & Krebs-Smith, S. M. (2008). Journal of the American Dietetic Association, 108(11), 1896-1901

THE UNIVERSITY OF BRITISH COLUMBIA

FINANCIAL SUPPORT amet, consectetuer adipiscing elit, sed diam nonummy nibh euismod tinci dunt ut laoreet dolore magna aliguam erat volutpat. Ut wisi enim.Lorem ipsum dolor sit amet, con sectetuer adipiscing elit, sed diam nonummy nibh euismod tincidunt ut laoreet dolore magna aliquar KIND THANKS TO amet, consectetuer adipiscin elit, sed diam nonummy nibh euismod tincidunt u laoreet dolore magna aliguam erat volutpat. Ut wisi enim.Lorem ipsum dolor sit amet, conse tetuer adipiscing elit, sed diam nonummy nibh eu ismod tincidunt ut laoreet dolore magna aliguan

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UBC

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THE UNIVERSITY OF BRITISH COLUMBIA

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Exploring the impact of finate How has COVID-19 influence

lyoma Y. Edache¹, MSc Mark Pitbla

¹School of Population and Public Health ²

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UBC

THE UNIVERSITY OF BRITISH COLUMBIA

plescent health behaviors: How has COVID-19 quality?

, Department of Pediatrics³, University of British Columbia

KEY FINDINGS

figure 1. Associations between financial security, emotional wellbeing and adolescent health behaviors.

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Study results highlight the role of emotional wellbeing in the pathway through which financial security impacts adolescent health behaviors. As public policy addressing financial security may indirectly improve adolescent health behaviors, our findings will inform COVID-19 public health priorities — specifically, family-based efforts to support and promote adolescent health behaviors and emotional wellbeing.

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no bullets

establish hierarchy using good text layout

Differentially-Expressed miRNAs in the Human Placenta

Nikita Telkar^{1,2,3}, Victor D. Martinez³, Victor Yuan¹, Magda E. Price^{1,2}, Brenda C. Minatel³, Erin A. Marshall³, Wendy P. Robinson^{1,2}, Wan L. Lam³

¹BC Children's Hospital Research Institute, Vancouver, British Columbia, Canada ²Department of Medical Genetics, University of British Columbia, Vancouver, British Columbia, Canada ³British Columbia Cancer Research Centre, 675 West 10th Ave, Vancouver, British Columbia, Canada

Introduction

- Placental gene regulation is crucial in the maintenance of a healthy pregnancy; aberrant expression can lead to severe complications in both mother and fetus¹.
- MicroRNAs (miRNAs) are known regulators of gene expression, and cause repression by destabilizing target mRNA molecules^{2,3}.
- Several factors are associated with changes in gene expression^{4,5}; however, these factors have not yet been fully elucidated in the placenta.

Question Does placental miRNA expression differ by inherent biological, and extrinsic technical variables?

Methods

30 placental samples were previously subjected to RNA-seq, enriching for small RNAs.

N = 30	Trimester 1 N = 5 (16.6%)	Trimester 2 N = 16 (53%)	Trimester 3 N = 9 (30%)
Neural Tube Defects	0/5	6/16	0/9
Sex	3F/2M	10 F/6 M	4F/5M

- 13,038 miRNAs were filtered at RPM of >=1 and if present in >=2 samples, giving 527 miRNAs.
- Relative Log Expression (RLE) normalization was applied, as it performs best for small sequences.

Results

Samples separate by trimester

- Principal Component Analysis showed that the samples distinctly separated by trimester (for PC1 vs PC2), especially for trimester 1 samples.
- Samples did not group by sex or condition status (control/NTD); however, this might be because the study was underpowered to detect an association.

Trimester and Flow Cell contribute to the most variance in the data

 Both trimester and sequencer flow cell were associated with PC1 explaining 48% of the variance in observed, followed by condition.

MiRNAs show variable expression across trimesters

 Linear regression was applied to identify differentially-expressed (DE) miRNAs, followed by multiple-testing correction using the Benjamini-Hochberg method at an FDR of 0.05.

miRNA ~Trimester + Sex + FlowCell

 6 miRNAs were commonly DE for trimester 1, 2, and term samples, of which two are novel miRNAs. All showed an overall negative correlation with trimester. No miRNAs were DE by sex or condition.

Comparison	Upregulated	Downregulated	Total
TI vs T2	0	161	161
TI vs Term	2	205	207
T2 vs Term	20	60	80

Conclusions

- 1. Placental gene regulation is influenced by numerous variables, both biological and technical.
- There still exist undiscovered, novel placental miRNAs; their characterization would provide better understanding of the human placenta and improved care of pregnant women and the fetus.

References

B. Cox, et al., Am J Obstet Gynecol. 213, S138–S151 (2015).
 J.-F. Mouilletet al., Am J Obstet Gynecol. 213, S163–S172 (2015).
 D. M. Morales-Prieto, et al., Journal of Reproductive Immunology. 97, 51–61 (2013).
 D. A. Hughes et al., Genome Biol. 16, 54 (2015).
 H. E. J. Yong, S.-Y. Chan, Hum Reprod Update. 26, 799–840 (2020).

Does placental miRNA expression differ by inherent biological and extrinsic technical variables? Yes, but it's more complicated than that.

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527/13,038 miRNAs were present in more than one sample with RPM ≥1. Relative Log Expression (RLE) normalization was applied, which performs best for small sequences.

SAMPLES SEPARATE BY TRIMESTER

Samples distinctly separate by trimester, especially for T1. Samples did not group by sex or condition status (control/NTD). This may be due to lack of power to detect this association.

P-value

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ink-heavy templates shout over data

explain the science, brand later (or never)

The placenta is not an asexual organ: Patterns of sex-specific autosomal DNA methylation

Amy M Inkster^{1,2}, Victor Yuan^{1,2}, Chaini Konwar^{1,2}, Alison M Matthews^{1,3}, Carolyn J Brown¹, Wendy P Robinson^{1,2}

Introduction

- · Sex differences exist in healthy pregnancy and certain adverse peritotal outcomes, and appeal to be partially mediated by the placenta.4
- The letus and placenta poistess the same sex chromosome complement, except in raie cases.
- Placental DNA methylation (DNAme) differs by see due to X-chromosome inactivation, but other Instores, such as letal sex harmones and autocomol ONAme likely contribute to placental we differences as well.

We hypothesize that sex specific patterns of DNA methylation exist, at autospinal loci in the human placenta, and may be related to sex specific placental function.

Aims

- 1. Identify autocomal DNAme signatures associated with biological sex
- 2. Validate robust patterns of sex-specific ONAme il independent delatets
- 3. Investigate relationships between other placental features and sex-specific DNAme

Samples & Methods

- Bumina 450K DNAme data from healthy normal placentas were obtained from publicly available datasets (>37 weeks gestation, no preeclampsia, no known chromosomal abbormalities).
- Sample sex was assessed with XY probes. Data were BMIC normalized and filtered to remove poor quality, non variable, non-specific,
- and XY probes in...=161,4085 Log-transformed M values at 324,104 autonoma CpG sites used in downstream analysis.

GGG Accession	Samples (n. % female)
05673375,05674738, 85675248,058300197, 65630857,036328827	847, (52%)
international landstation of	TERPHI
	and addressed

Genome-wide placental DNAme is not sex-specific

average DNAme at all autoscenal loci or at Aluor UNE1 planants.

· Linear modelling on AI-values was used to test for differential DNAme (DM) by sex at autocoreal Call sites, with Bayesianmaderated t-statistics.

Volcano plot of 124,104 autosomal Colls, 166 top DM loci satisfying a false discovery rate <0.01, and d8>0.10, where $\Delta\beta=\beta_{\rm max}-\beta_{\rm barrier}$. Adjusted for GA, ethnicity, and dataset.

Sex-specific differentially methylated sites are biologically interesting

- ARMGAP15 (chr2, higher female DNAme in gene body) imment in placental elements."
- ZNF300 (chr6, higher male DNAme near 155), higher expression in ternale placentas.¹

Sex-specific DNAme patterns validate in independent cohorts

- Samplies cluster by sea in two independent datasets QSE70453 and GSE11SSDB at top 166 DM loci, suggesting a robust sex effect at these loci. *Indicates p<2.2e-16 (rigClust2) and stable (michael) Allo-BD charter, 24
- Significant patterns of differential DNAme by sea validate at 90% of iper in CSE75453 and CSE115508 IV = 0.62, pr 2.2e-161

Autosomal DNAme signatures are

not sexually dimorphic

tamples fail across a continuum of sex when investigating DNAme patterns at top 166 DM loci.

- We found that this trend is not associated with: · Outlying autocomal DNAme I'll highly variable
 - probest
- Sex antionation errors
- Technical factors or batch effects. + DM loci proximity to sex-hormone binding sites. · Biological variables (genetic ancestry, gestational
- age, birthweight; maternal age).

This trend is significantly associated with: Increased average LINEL DNAme

* Positive or negative destations from population mean & chromersome ONAme.

Females with outlying X-linked DNAme relative to population trends have more male-like DNAme at top autosomal DM sites, and vice versa-

Conclusions

- Entruit sex-specific autosomal DNAme signatures exist in the human placenta.
- Placental autosomal DRAme patterns are continuous between the sexes, rather than discrete. This may be related to interactions between autosomal and X chromosomal locit other unmeasured biological factors may contribute (e.g. fetal or maternal sex harmotres)
- II. Sex-specific autosomal DNAme reflects sex specific function of the human placents and may provide insight into fetal health are disperitien. but is not sufficient to explain sea-specific outconves.

Acknowledgements

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THE PLACENTA IS NOT AN ASEXUAL ORGAN Patterns of sex-specific autosomal DNA methylation

Amy M Inkster1,2, Victor Yuan1,2, Chaini Konwar1,2, Allison M Matthews1,3, Carolyn J Brown2, Wendy P Robinson1,2

1BC Children's Hospital Research Institute Vancouver British Columbia Canada

²Department of Medical Genetics, University of British Columbia, Vancouver, British Columbia, Canada ³Department of Pathology and Laboratory Medicine, University of British Columbia, Vancouver, British Columbia. Canada

SEX-SPECIFIC PATTERNS OF DNA METHYLATION

Sex differences exist in healthy pregnancy and certain adverse perinatal outcomes, and appear to be partially mediated by the placenta¹.

The fetus and placenta possess the same sex chromosome complement, except in rare cases.

Placental DNA methylation (DNAme) differs by sex due to X chromosome inactivation, but other features, such as fetal sex hormones and autosomal DNAme likely contribute to placental sex differences as well.

We hypothesize that sex-specific patterns of DNA methylation exist at autosomal loci in the human placenta, and may be related to sex-specific placental function.

Genome-wide placental DNAme is not sex-specific

Specific autosomal CpG sites are differentially methylated by sex

DNAme :: ~ sex; + dataset; + GA; + ethnicity; + ɛ;;

324,104 autosomal CpGs. Adjusted for GA, ethnicity, and dataset

(R = 0.62, p< 2.2e-16).

Clifton 2010. Placenta 21:S33-S39. [2] Teschendorff et al. 2013. Bioinformatics 29:189-96. [3] Liu et al. 2008, J Am Stat Assoc 103:1281, [4] Suzuki & Shimodaira 2006, Bioinformatics 12:1540-42. [5] Kuleshov et al. 2016. Nucleic Acids Res gkw377

ROBUST SEX-SPECIFIC AUTOSOMAL DNAme SIGNATURES EXIST IN THE HUMAN PLACENTA.

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Sex-specific differentially methylated sites are biologically interesting

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Autosomal DNAme signatures are not sexually dimorphic

PC2 -0.05

P < 10-15

AU > 80

0.06

Samples fall across a continuum of sex when investigating DNAme patterns at top 166 DM loci.

from publicly available datasets (>37 weeks gestation, no preeclampsia, no chromosomal abnormalities). Sample sex was assessed with XY probes. Data were BMIQ normalized and filtered to remove poor quality. non-variable, non-specific, and XY probes (nfilt=161.408), Log-transformed M values at 324,104 autosomal CpG sites used in downstream analysis

Thank you to all patients and families who kindly donated samples, and members of the Robinson Lab, especially WPR, VY, MSP, and GDG for valuable feedback. This work was supported by the Department of Medical Genetics (UBC) and the Canadian Institutes of Health Research (ICT-163379.CIHR SVB-158613).

The placenta is not an asexual organ: Patterns of sex-specific autosomal DNA methylation

Amy M Inkster^{1,2}, Victor Yuan^{1,2}, Chaini Konwar^{1,3}, Allison M Matthews^{1,3}, Carolyn J Brown², Wendy P Robinson^{1,4}

Introduction

- Sex differences rotat in healthy programcy and certain adverse perinatal outcomes, and appear
- to be partially mediated by the placenta.1 The fetus and placenta possess the same sex chromosome complement, except in rare cases. Placental DNA methylation (DNAme) differs by sex due to X chromosome inactivation, but other
- features, such as fetal sex hormones and autosomal DNAme likely contribute to placental sex differences as well. We hypothesize that sex-specific patterns of DNA

methylation exist at autosomal loci in the human placenta, and may be related to sen-specific placental function.

Aims

- 1. Identify autosomal DNAme signatures associated with biological sex 2. Validate robust patterns of sex-specific DNAme
- in independent datasets 3. Investigate relationships between other
- placental features and sex-specific DNAme

Samples & Methods

- Illumina 450K DNAme data from healthy normal placentas were obtained from publicly available datasets i>37 weeks gestation, no preeclampsia, no known chromosomal abnormalities).
- Sample sex was assessed with XY probes, Data were BMIQ normalized and filtered to remove poor quality, tion-variable, non-specific, and XY probes (new=161,408).
- Log-transformed M values at 324,104 autosomal CpG sites used in downstream analysis.

GEO Accession	Samples (n. % female)	
GSE78875, GSE74738, GSE75348, GSE100197, GSE100857, GSE128827	141 (52%)	
STATE NO. 12 SHOULD BE REAL PROPERTY OF	0.0770	
Assistant and and and	an indui	

Genome-wide placental DNAme is not sex-specific

Specific autosomal CpG sites are differentially methylated by sex

· Linear modelling on M-values was used to test for differential DNAme (DM) by sex at autosomal CpG sites, with Bayesian-

moderated 5-statistics.

DNAme, " Sex, + Dataset, + GA, + Ethnicity, + z.,

08 00	-0	+58	=D26	+20%
<0.05	A715	3,947	196	
40.81	34,238	2,682	166	

Volcano plot of 334.104 autosomal CpGs. 366 top DM loct

satisfying a false discovery rate <0.01, and AB>0.10, where

 $\Delta\beta = \beta_{max} - \beta_{max}$ Adjusted for GA, ethnicity, and dataset.

Sex-specific differentially methylated sites

ARHGAP15 (chr2, higher female DNAme in gene body)

ZNF300 (chr6, higher male DNAme near 755), higher

are biologically interesting

present in placental excupres.⁹

expression in female placentas.⁴

14

Samples fail across a continuum of sex when

Autosomal DNAme signatures are

not sexually dimorphic

We found that this trend is not associated with:

- · Outlying autosomal DNAme (% highly variable
- Sea annotation errors
- · Technical factors or batch effects.
- · DM loci proximity to sex hormone binding sites.

· Positive or negative deviations from population

· Females with outlying X-linked DNAme relative to population trends have more male-like DNAme at

Conclusions

- 1. Robust sex-specific autosomal DNAme signatures exist in the human placenta.
- 2. Placental autosomal DNAme patterns are continuous between the sexes, rather than discrete. This may be related to interactions between autosomal and X chromosomal loci; other unmeasured biological factors may contribute (e.g. Fetal or maternal sex hormones).
- specific function of the human placents and may provide insight into fetal health sex disparities. but is not sufficient to explain sex specific. outcomes.

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References

Kei 2022, Marsena D.511.105, [2] Suchweiself et al. 2025, Ramilie W. (K) and a 2020, Annihue Anne 2021, [3] Such & Home Relightments 11 (2021-82 [1] Hybridize et al. 2020, Nucleic Relight Relightments.

- (probes)

- · Biological variables (genetic ancestry, gestational
- age, birthweight, maternal age).
- This trend is significantly associated with: Increased average LINE1 DNAme
- mean X chromosome DNAme.

top autosomal DM sites, and vice-versa.

- - 1. Sex-specific autosomal DNAme reflects sex-

· Significant patterns of differential ONAme by sex validate at

90% of loci in GSE70453 and GSE115508 (R = 0.62, p< 2.2e-16).

THE PLACENTA IS NOT AN ASEXUAL ORGAN Patterns of sex-specific autosomal DNA methylation

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We hypothesize that sex-specific patterns of DNA methylation exist at autosomal loci in the human placenta, and may be related to sex-specific placental function.

Genome-wide placental DNAme is not sex-specific

all autosomal loci (ns)	avg Alu DNAme	avg LINE1 DNAme

ROBUST SEX-SPECIFIC AUTOSOMAL DNAme SIGNATURES EXIST IN THE HUMAN PLACENTA.

Children's

Hospital

Placental autosomal DNAme patterns are continuous between the sexes, rather than discrete. This may be related to interactions between autosomal and X chromosomal loci; other unmeasured biological factors may contribute (e.g. Fetal or maternal sex hormones). Lorem ipsum dolor sit amet, consectetuer adipiscing elit, sed diam nonummy nibh eu-

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Sex-specific autosomal DNAme reflects sex-specific function of the human placenta and may provide insight into fetal health sex disparities, but is not sufficient to explain sex-specific outcomes. Lorem ipsum dolor sit amet, consectetuer adipiscing elit, sed diam nonummy nibh euismod tincidunt ut laoreet dolore magna aliquam erat volutpat. Ut wisi enim ad minim

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ARHGAP15 (chr2) ZNF300 (chr6) present in placental exomes higher expression in female placentas

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Specific autosomal CpG sites are differentially methylated by sex				
$DNAme_{ij} \sim sex_i + dataset_i + GA_i + ethnicity_i + \varepsilon_{ij}$				

324,104 autosomal CpGs. Adjusted for GA, ethnicity, and datase

Sex-specific DNAme

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Samples cluster by sex in two inde-

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Cluster stable (pvclust) at AU > 80.

Significant patterns of differential DNAme by sex validate at 90% of

loci in GSE70453 and GSE115508

(R = 0.62, p<2.2e-16).

pendent datasets at top 166 DM

FDR	Δβ	> 0	> 5%	> 10%	> 209
< 0.05		24,715	2,942	166	4
< 0.01		14,108	2,682	166	4

- Positive or negative deviations from population mean X chromosome DNAme

This trend is not associated with - Outlying autosomal DNAme (% highly variable probes)

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GSE70

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- Sex-specific autosomal DNAme reflects sexspecific function of the human placenta and may provide insight into fetal health sex disparities, but is not sufficient to explain sex-specific outcomes.

Samples & Methods

GEO Accession Samples (*n*, % female) GSE73375, GSE74738, GSE75248, GSE100197, GSE100857, GSE128827 (341, 51%) GSE70453 (validation) (72, 47%) GSE115508 (validation) (44, 45%)

Illumina 450K DNAme data from healthy normal placentas were obtained from publicly available datasets (>37 weeks gestation, no preeclampsia, no known chromosomal abnormalities). Sample sex was assessed with XY probes. Data were BMIQ normalized and filtered to remove poor quality, non-variable, non-specific, and XY probes (nfilt=161,408). Log-transformed M values at 324,104 autosomal CpG sites used in downstream analysis.

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Genome-wide placental DNAme is

not sex-specific

 There were no significant differences by sex in average DNAme at all autosomal loci or at Alu or LINE1 elements.

Genome-wide placental DNAme is not sex-specific

Sex-specific differentially methylated sites are biologically interesting

- ARHGAP15 (chr2, higher female DNAme in gene body) present in placental exosomes.⁵
- ZNF300 (chr6, higher male DNAme near TSS), higher expression in female placentas.⁵

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 Linear modelling on M-values was used to test for differential DNAme (DM) by sex at autosomal CpG sites, with Bayesianmoderated t-statistics.

DNAme,, " Sex, + Dataset, + GA, + Ethnicity, + E,

• Volcano plot of 324,104 autosomal CpGs, 166 top DM loci satisfying a false discovery rate <0.01, and $\Delta\beta$ >0.10, where $\Delta\beta = \beta_{maile} - \beta_{lemaile}$ Adjusted for GA, ethnicity, and dataset.

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Sex-specific DNAme patterns validate in independent cohorts

- Samples cluster by sex in two independent datasets GSE70453 and GSE115508 at top 166 DM loci, suggesting a robust sex effect at these loci. *Indicates p<2.2e-16 (sigClust2) and stable (pvclust) AU>80 cluster.¹⁴
- Significant patterns of differential DNAme by sex validate at 2014 of loci in GSE30463 and GSE315508 (8 = 0.62, no. 2, 2n. 16)

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Sex-specific DNAme patterns validate in independent cohorts

Samples cluster by sex in two independent datasets at top 166 DM loci, suggesting a robust sex effect at these loci. P-value from sigClust2. Cluster stable (pvclust) at AU > 80. Significant patterns of differential DNAme by sex validate at 90% of loci in GSE70453 and GSE115508 (R = 0.62, p < 2.2e-16).

 $p < 10^{-15}$

AU > 80

arrange and rearrange your puzzle pieces

use color for key themes — and for nothing else

Genetic Influences on the Severity of Ethanol-Induced **Cell Death in the Developing Prenatal Brain**

Kristina Balce¹, Emilie Théberge¹, Kristen Hamre², Daniel Goldowitz¹

¹Centre for Molecular Medicine and Therapeutics, Dept. of Medical Genetics, The University of British Columbia, Vancouver, BC ²Dept. of Anatomy and Neurobiology, The University of Tennessee Health Science Center, Memphis, TN

BACKGROUND

- Fetal Alcohol Spectrum Disorder (FASD) is caused by fetal exposure to alcohol consumed by mothers during pregnancy¹
- Most common preventable cause of developmental disability in Canada; over 3000 Canadian newborns diagnosed annually2
- Characterized by abnormal brain development, cognitive/learning deficits, behavioural issues, and/or specific patterns of physical defects³

Figure 1. Typical craniofacial defects of full blown Fetal Alcohol Spectrum Disorder (FASUL known as Fetal Alcohol Synchrome (FASL

- Research in mouse models implicates ethanol-induced apoptosis (i.e. programmed cell death) as one process contributing to disruption in early brain development⁴
- Severity of ethanol's effects appears to vary depending on genetic background; how and which genes are involved in susceptibility or resistance to alcohol remain largely unknown
- Genetic influences may be important for screening, prevention, and therapeutic treatment of FASD

OBJECTIVES

- Demonstrate genetic differences in vulnerability to the apoptotic ÷ effects of prenatal alcohol exposure
- Use Quantitative Trait Locus (QTL) analysis to identify genes ÷ involved in susceptibility or resistance to ethanol-induced apoptosis in the developing brain

METHODS

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4) Sectioning & Mounting

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Figure 4. Quantitative Trait Locus (27U) Interval mapping of ethanol-induced apoptosis in the brainstein, 34) Whole percent (7D, mail: The s-axis represents chromosome: 1–15, & # and their physical maps in megalianes. The y-axis and the blue line indicate the Nei-Pond ratio statistic CNU, which reports the strength of association between construms in generative and the phenotype (i.e. ethanol-induced apportunit). The red and gray horizontal lines respectively must be significant (p. = 0.05) and suggestive (p. = 0.03) itersholds. Peaks that much these thresholds indicate genome regions containing candidate genes that may be implicated in othered-relaced appropriate (Arta) of Maps of the suggestive (Arta) on chromosome 4 (LR).

13.510) and chromosome 14 (LRS + 15.655) were expanded to analyze candidate genes and ongle nucleotide polymorphytom (SNPs, yellow peaks on the x-axis) at each local.

CONCLUSIONS & FUTURE DIRECTIONS

- * There is significant variation in vulnerability to ethanol-induced apoptosis between BXD strains, suggesting that genetic differences influence the severity of the effects of prenatal alcohol exposure
- Suggestive QTLs on chromosomes 4 and 14 were identified
- BXD strains with the CS7BL/6J genetic background at these QTLs were more susceptible to ethanol-induced apoptosis
- Candidate genes at these two QTLs may play an important role in vulnerability; are potential future targets for prenatal screening and therapeutic intervention of FASD

RESULTS – Genetic Differences in Vulnerability to Ethanol-Induced Apoptosis

Figure 2. Representative images of the developing bitainstem from (A) susceptible and (B) resistant mouse BKD strains, treated with ethenol on embryonic day 9 (E9.0). Apoptotic calls were labelled using the terminal deconnucleation transferance stuff# sids-end labeling [TUNTL] assay. Cell madei of nonapositotic colls were counterstained with methyl green. The rest arrow industry a greater amount of cell ideath in the susceptible strain.

Figure 3. Differences in witherability to ethanol-induced apoptosis in the mouse (800 panel. The worder of apoptoria, (TUNE), positive) only or mm¹ was measured in incluse embryos treated with ethanol (DDH, red) and a maltone-devican (MD), blue) sugar control at embryonic day 9 (DLD) Between strains, there was a significant difference (p = 0.001) in apoptous means after FIOH treatment, tast no significant difference (p = 0.084) after MD treatment. Comparisons between treatments within strain reported agrifuant differences (a = 0.01) in apoptious means in the CS/RL/Az 8XD 51, 8XD 96, and BXD 100 strates, indicating that these strains show the highest vulnerability to ethanor-induced apoptosis

RESULTS – Quantitative Trait Locus (QTL) Analysis for Candidate Genes

Figure 5. The influence of the C578L/IU (IBG) and DBA/2 (ISD) alleles on valverability to ethenol-induced apoptosis at the suggestive CPL intervals on chromosomes 4 and 14, 0x0 mice were grouped based on presence of the B4 or D2 allele at each G71, interval, and the average number of ethanof-induced apoptatic calls were compared internation of BE alleles is associated with significantly higher issueptibility to etheriof-induced spontrom (p < 0.01).

Table 1. Summary of select candidate genes located in the chromosome 4 and 14 CITL intervals, and relevant biological processes described in Gene Ontology (GO, www.geneontology.org).

qu	Candidate Gerre	DOI BIOMATICAL WHERE
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ACKNOWLEDGEMENTS & REFERENCES

The authors wish to thank Aria Shokoshi, Mike Wu and Julia Boyle for assistance in tissue processing, sectioning, an staining for this project, and Rob Williams for assistance in QTL data analysis.

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Genetic Influences on the Severity of Ethanol-induced **Cell Death in the Developing Prenatal Brain**

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Fetal Alcohol Spectrum Disorder

Fetal Alcohol Spectrum Disorder (FASD) is caused by fetal exposure to alcohol consumed by mothers during pregnancy [1]. It is the most common preventable

TUNEL AND APOPTOTIC CELLS PER MM²

20

in the susceptible strain.

RESISTANT BXD39

Images of the developing brainstem from a susceptible and resistant mouse BXD strain, treated with ethanol on embryonic day 9 (E9.0). Apoptotic cells were labelled using the terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL) assay. Cell nuclei of nonapoptotic cells were counterstained with

methyl green. The arrow indicates a greater amount of cell death

6,000

4.000

2,000

FtOH

T MD

C57BL/6JDBA/2J BXD2

cause of developmental disability in Canada - over 3000 Canadian newborns diagnosed annually [2]. FASD is characterized by abnormal brain development, cognitive/learning deficits, behavioural issues, and/or specific patterns of physical defects.

Research in mouse models implicates ethanol-induced apoptosis (i.e. programmed cell death) as one process contributing to disruption in early brain development [4]. Severity of ethanol's effects appears to vary depending on genetic background; how and which genes are involved in susceptibility or resistance to alcohol remain largely unknown. Genetic influences may be important for screening, prevention, and therapeutic treatment of FASD.

BXD51 SUSCEPTIBLE

increased apoptosis

Objectives

Demonstrate genetic differences in vulnerability to the apoptotic effects of prenatal alcohol exposure. Use Quantitative Trait Locus (QTL) analysis to identify genes involved in susceptibility or resistance to ethanol-induced apoptosis in the developing brain.

Conclusions

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in the brainstem

apoptosis)

50 100 150 Mb

effect

18 94 significant p = 0.05I BS

10.60

p = 0.63

There is significant variation in vulnerability to ethanol-induced apoptosis between BXD strains, suggesting that genetic differences influence the severity of the effects of prenatal alcohol exposure.

Suggestive QTLs on chromosomes 4 and 14 were identified BXD strains with the C57BL/6J genetic background at these QTLs were more susceptible to ethanol-induced apoptosis.

Candidate genes at these two QTLs may play an important role in vulnerability; are potential future targets for prenatal screening and therapeutic intervention of FASD.

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Figure 1. Typical craniofacial defects of full blown Fetal Alcohol Spectrum Disorder (FASD), known as Fetal Alcohol Syndrome (FAS).

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BACKGROUND

- Fetal Alcohol Spectrum Disorder (FASD) is caused by fetal exposure to alcohol consumed by mothers during pregnancy¹
- Most common preventable cause of developmental disability in Canada; over 3000 Canadian newborns diagnosed annually2
- Characterized by abnormal brain development, cognitive/learning deficits, behavioural issues, and/or specific patterns of physical defects³

Figure 1. Typical craniofacial defects of full blown Fetal Alcohol Spectrum Disorder (FASU), known as Fetal Alcohol Syndrome (FAS).

- Research in mouse models implicates ethanol-induced apoptosis (i.e. programmed cell death) as one process contributing to disruption in early brain development⁴
- Severity of ethanol's effects appears to vary depending on genetic background; how and which genes are involved in susceptibility or resistance to alcohol remain largely unknown
- Genetic influences may be important for screening, prevention, and therapeutic treatment of FASD

OBJECTIVES

- Demonstrate genetic differences in vulnerability to the apoptotic ÷ effects of prenatal alcohol exposure
- Use Quantitative Trait Locus (QTL) analysis to identify genes. involved in susceptibility or resistance to ethanol-induced apoptosis in the developing brain

CONCLUSIONS & FUTURE DIRECTIONS

- There is significant variation in vulnerability to ethanol-induced apoptosis between BXD strains, suggesting that genetic differences influence the severity of the effects of prenatal alcohol exposure
- Suggestive QTLs on chromosomes 4 and 14 were identified \mathbf{r}
- BXD strains with the CS7BL/6J genetic background at these QTLs were * more susceptible to ethanol-induced apoptosis
- Candidate genes at these two QTLs may play an important role in * vulnerability; are potential future targets for prenatal screening and therapeutic Intervention of FASD

Genetic Influences on the Severity of Ethanol-induced Cell Death in the Developing Prenatal Brain

Kristina Balce¹, Emilie Théberge¹, Kristen Hamre², Daniel Goldowitz¹

¹Centre for Molecular Medicine and Therapeutics, Dept. of Medical Genetics, The University of British Columbia, Vancouver, BC ²Dept. of Anatomy and Neurobiology, The University of Tennessee Health Science Center, Memphis, TN

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1) Mating

Male and female mice of the same strain were mated during a 4 hour period.

4) Sectioning & Mounting

Paraffin embedded embryos were sectioned using a microtome at 8µm, and sections were mounted onto glass slides.

5) TUNEL Staining

Apoptotic cells in the brainstem were labelled using the terminal dUTP nick-end labeling (TUNEL) assay and counted.

6) QTL Analysis

Quantitative Trait Locus (QTL) analysis was done using GeneNetwork (www.genenetwork.org), and candidate genes were identified.

2) Treatment

At day 9 of pregnancy (E9.0), pregnant dames were treated twice, 2 hours apart with either ethanol (EtOH, 5.9g/kg), or an isocaloric maltose-dextran (MD) sugar control.

3) Harvest

7 hours after treatment, embryos were collected from dames and embedded in paraffin wax.

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Figure 2. Representative images of the developing brainstem from (A) susceptible and (B) resistant mouse BXD strains, treated with ethanol on embryonic day 9 (E9.0). Apoptotic cells were labelled using the terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL) assay. Cell nuclei of nonapoptotic cells were counterstained with methyl green. The red arrow indicates a greater amount of cell death in the susceptible strain.

Figure 3. Differences in vulnerability to ethanol-induced apoptosis in the mouse BXD panel. The number of apoptotic (TUNEL positive) cells per mm² was measured in mouse embryos treated with ethanol (EtOH, red) and a maltose-dextran (MD, blue) sugar control at embryonic day 9 (£9.0). Between strains, there was a significant difference (p < 0.001) in apoptosis means after EtOH treatment, but no significant difference (p = 0.084) after MD treatment. Comparisons between treatments within strain reported significant differences (p < 0.05) in apoptosis means in the CS7BL/6J, BXD 96, and BXD 100 strains, indicating that these strains show the highest vulnerability to ethanol-induced apoptosis.

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Images of the developing brainstem from a susceptible and resistant mouse BXD strain, treated with ethanol on embryonic day 9 (E9.0). Apoptotic cells were labelled using the terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL) assay. Cell nuclei of nonapoptotic cells were counterstained with methyl green. The arrow indicates a greater amount of cell death in the susceptible strain.

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Figure 5. The influence of the C578L/6J (B6) and D8A/2 (D2) alleles on vulnerability to ethanol-induced apoptosis at the suggestive QTL intervals on chromosomes 4 and 14. BXD mice were grouped based on presence of the B6 or D2 allele at each QTL interval, and the average number of ethanol-induced apoptotic cells were compared. Inheritance of B6 alleles is associated with significantly higher susceptibility to ethanol-induced apoptosis (p < 0.01).

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Figure 4. Quantitative Trait Locus (QTL) Interval mapping of ethanol-induced apoptosis in the brainstem. (A) Whole genome QTL map. The x-axis represents chromosomes 1–19, & X and their physical maps in megabases. The y-axis and the blue line indicate the likelihood ratio statistic (LRS), which reports the strength of association between variations in genotype and the phenotype (i.e. ethanol-induced apoptosis). The red and gray horizontal lines respectively mark the significant (p = 0.05) and suggestive (p = 0.63) thresholds. Peaks that reach these thresholds indicate genome regions containing candidate genes that may be implicated in ethanol-induced apoptosis. (B, C) Maps of the suggestive QTLs on chromosome 4 (LRS = 13.510) and chromosome 14 (LRS = 15.655) were expanded to analyze candidate genes and single nucleotide polymorphisms (SNPs, yellow peaks on the x-axis) at each locus.

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Chr 4 QTL interval

QTLs were more susceptible to ethanol-induced apoptosis.

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Robert Bringhurst

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The Visual Display of Quantitative Information

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Gordon Andrews (designer) Gazelle chair (c. 1950) designed, 1957 manufactured plywood, aluminium, wool 74.0 x 48.0 x 55.0 cm Museum of Applied Arts and Sciences, Sydney Purchased, 1989 (89/499)