

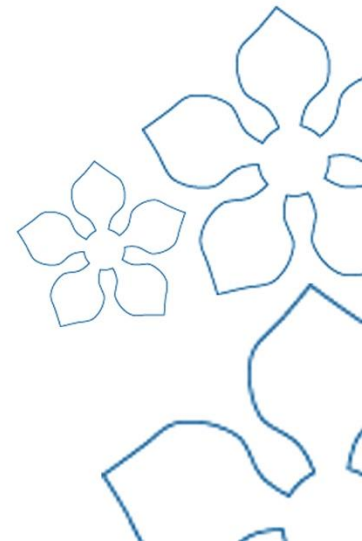
MSc Applied Social and Health Psychology
Applied Research, Ethics, and Analysis (AREA)

Multiple Regression

Dr. Kate Williams



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ESSENTIAL READING!

- Pallant, J. SPSS Survival Manual.
- Brace, N., Kemp, R., & Snelgar, R. SPSS for Psychologists.
- Coolican, H. Research Methods and Statistics in Psychology.
- Relevant chapters on Multiple Regression



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Multiple Regression design

- Used to explore the relationship between one DV and a number of IVs (or predictor variables).
 - Standard
 - Hierarchical (Enters in Blocks)
 - Stepwise (to be avoided! – adds/deletes predictors based on strength)
- Ideal for investigation of complex real-life research questions
 - Less used in lab-based experimental designs
 - Ideal for longitudinal prospective designs
- Often used to test how “good” a theory is
 - To what extent do the variable identified by a theory explain a given behaviour
 - Can add additional variables/new element to theory and test this



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Why do Multiple Regression?

- Research questions:
 - Which of a **range** of variables are most strongly related to a specific variable of interest?
 - Do these variables do better than chance or simple mean scores at **predicting** scores on a variable of interest?
 - How might these variables **combine** to predict a variable?
- Multiple variables:
 - Could just carry out many simple correlations
 - Provide several Bivariate associations (2 variables correlated)
 - Still only a non-causal correlation produced
 - Will not indicate our overall success with all variables used



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Why do Multiple Regression?

- Important extension of 'regression' over 'correlation':
 - **Combined** association effect of the predictor variables
 - **Multiple** regression
 - Treating the predictors (separate variables) as a combined variable in relation to the dependent variable
 - How 'associated' they are as a whole
 - How 'predictive' of the DV they are as a whole
 - (NB: Will also look at each in isolation)



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Assumptions

- Continuous data
- Normal distribution of data
- Linear relationships between IVs and DVs
- Homogeneity of variance
- Collinearity between IVs



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How do you choose which variables to use as your “predictors” or IVs in a regression design?



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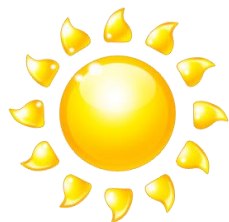
Multiple Regression

- Regression can be used to analyse a more 'causal' relation between variables
- Theory suggests a dependent variable and independent (or explanatory) variable(s)
 - Can also use more than one explanatory variable (IV)
- Regression analyses amount of change in the dependent variable based on change in the explanatory/independent variables
- Can be used to make **predictions** for the DV based on scores for the IV



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- Ice-cream sales example
 - What guided selection of IV's?



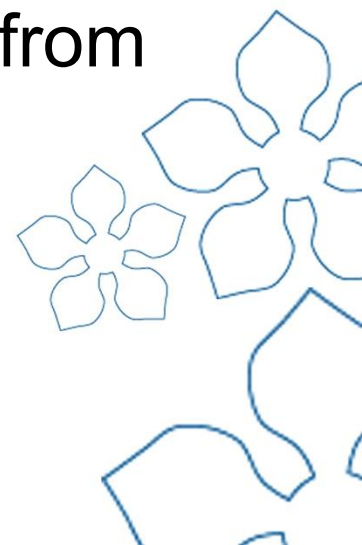
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- Ice-cream sales example

- Was this “empirically driven good science?”
- Better approach if funded research project?
- Would you actually expect a “good fit” from this model?



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– What could predict level of poverty?

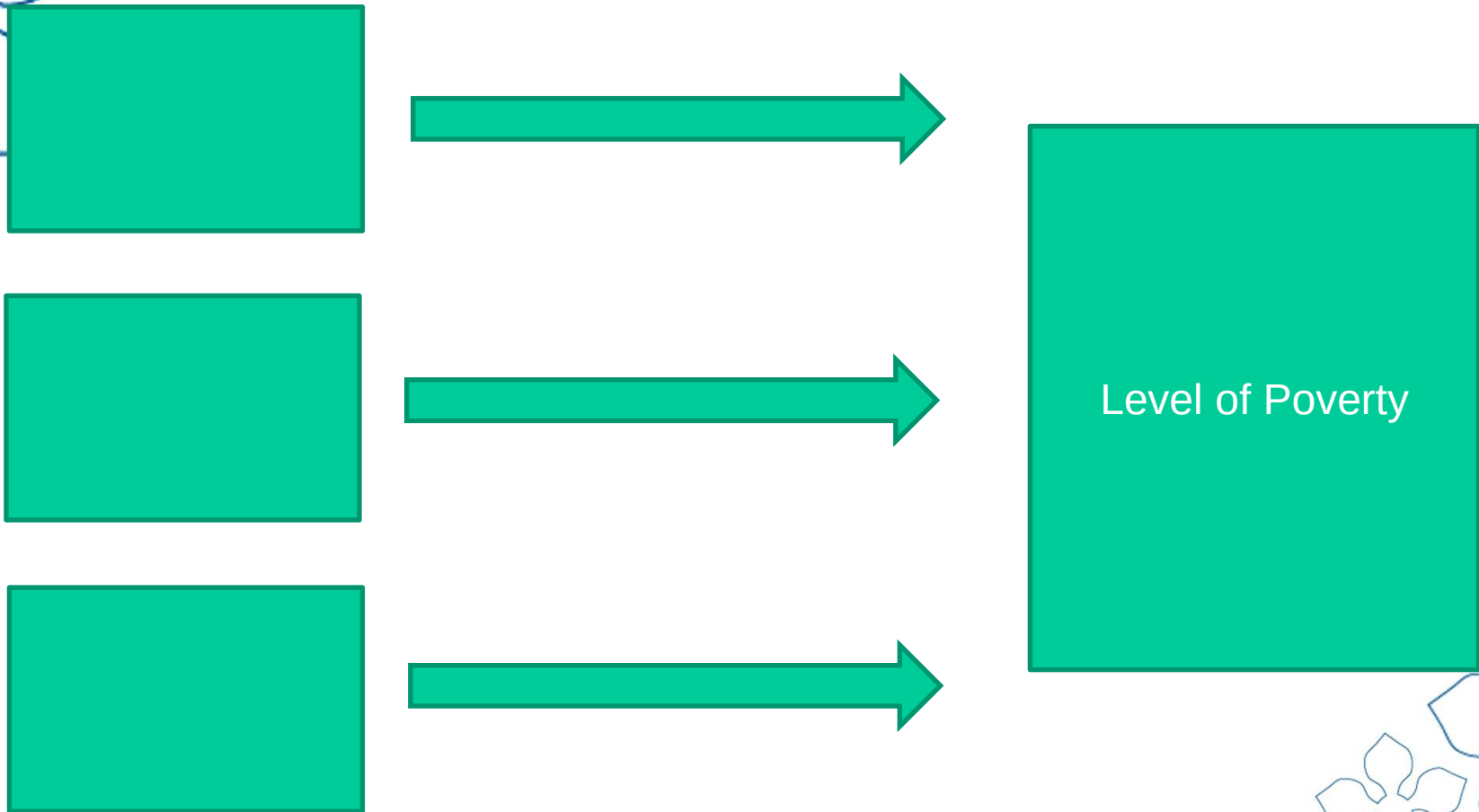


Level of Poverty



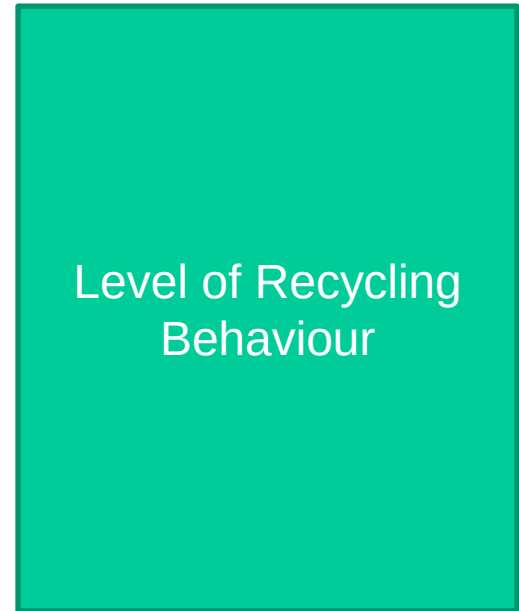
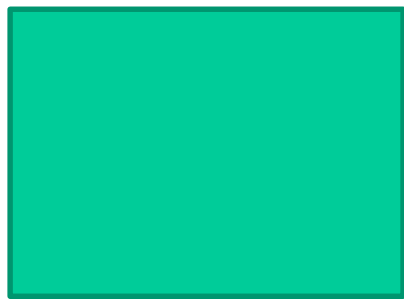
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– What should guide selection of predictors?

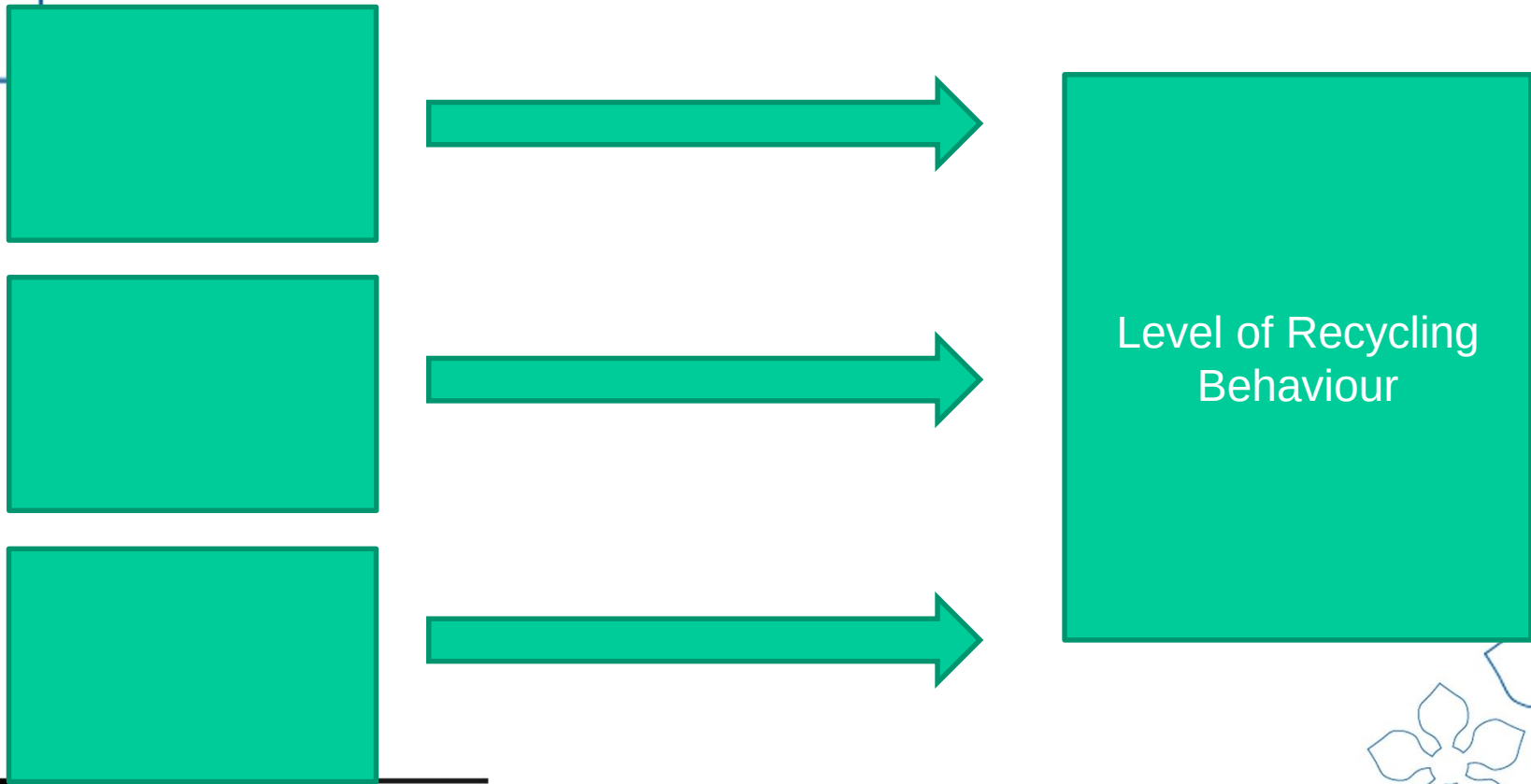


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– Predicting recycling behaviour



- Predicting recycling behaviour
- What should guide selection of predictors?



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Multiple Regression

- Multiple Regression statistics of interest:
 - Multiple regression indicates how good a 'fit' all the predictors are in explaining the DV
 - Can assess the degree to which the DV can be predicted from the set of predictors
 - R = multiple correlation coefficient
 - (between 0 and 1)
 - The greater R value, the less residual values
 - As with r , of more use can be R^2
 - The % of variance in the DV that 'model' explains
 - Adjusted R^2 - adjusted for sample size
 - Beta values represent amount of contribution (how good a fit) for each of the independent variables



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British Psychosocial Oncology Society Conference 2013

Predicting psychological responses to cancer genetic risk assessment using the self- regulation model of illness perception

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Background: understanding responses to genetic risk

Self-regulation model (Leventhal et al., 1992)

- People are “common-sense scientists” or active information processors.
- When faced with a health threat, form a cognitive representation and emotional response based upon our beliefs about:
 - Identity (label, signs & symptoms)
 - Causes (genetic, psychological, environmental)
 - Consequences (e.g., emotional, financial, physical)
 - Timeline (acute, chronic, cyclical)
 - Curability/controllability

Leventhal's Self Regulation Model

- Illness representations acquired through:
 - Media
 - Personal experience
 - Family & Friends
- May be inaccurate!
- Influence preventive behaviour and coping responses:
 - Seeking medical help (over or under-use)
 - Adherence to treatment (e.g., screening, medication)
 - Emotional reactions to symptoms (e.g., avoidance, hypervigilance)
 - Engagement in self-care behaviour (e.g., BSE, healthy lifestyle)

Study aim:

SRM suggests poorer psychological outcomes in those who believe that:

- being at genetic risk of cancer has serious emotional, physical or financial consequences
- little they can do to control their chances of developing cancer
- little faith in screening or surgery controlling their chances of developing cancer
- do not understand the genetic risks of cancer

Aim:

- ▶ To explore whether it is possible to predict psychological outcomes in individuals awaiting genetic risk information through measuring participants' beliefs about implications of being at genetic risk of cancer

Methods

- Longitudinal questionnaire data from a larger trial dataset (Phelps, Bennett, Brain, Hood & Murray, 2012).
- Participants undergoing early stages of cancer genetic risk assessment through the Cancer Genetics Service for Wales.
- 331 individuals undergoing genetic risk assessment analysed at baseline/upon referral; 201 at follow-up (one-month later).

Current Study: Measures

- ❑ Cancer specific distress

(Impact of Event Scale-revised, Horowitz et al., 1979)

- ❑ Negative affect

(Positive and Negative Affect Schedule, Watson et al. 1988)

- ❑ Positive affect

(Positive and Negative Affect Schedule, Watson et al. 1988)

- ❑ Trait optimism

(Life Orientation Test, Scheier & Carver, 1984)

Current Study: Measures

- **Illness Perceptions Questionnaire-revised (Moss-Morris et al., 2002) adapted for potentially being at genetic risk:**
 - Cognitive Illness representations sub-scales:
 - Identity – removed from current study
 - Consequences
 - Curability/controllability
 - Timeline - removed from current study
 - Illness coherence
 - Causal Beliefs (list of 18 potential causes of cancer factor analysed)
 - Emotional representation sub-scale – not used for current study

Adapted IPQ-r illness representations sub-scales

We are interested in your own personal views about how you think you would feel if you were found to be at increased risk of cancer....

	Scale range	No. of items	Example item
Consequences	6-30	6	Being at increased risk would be a serious issue for me
Personal control	6-30	6	There is a lot that I could do to control my chances of getting cancer
Illness Coherence	3-15	3	I have a clear picture or understanding of the genetic risks of cancer
Treatment control	4-20	4	Screening could help control my chances of getting cancer

Principal Components Analysis on IPQ-R causal factors: beliefs about the possible causes of cancer

Causal Factors/sub-scales	Causal factors comprising sub-scale	α
Psychological Attributions	Emotional state, family problems, overwork, stress or worry, mental attitude, my personality	.87
Risk Factors	Diet/eating habits, alcohol, smoking, my own behaviour, ageing, pollution	.71
Physical/Biological Harm	A germ or virus, faulty immune system, accident or injury	.54
Poor medical care in past	Single item	
Chance or bad luck	Single item	
Heredity	Single item	
<i>Acknowledgement: Lancaster, Brain & Phelps (2011)</i>		

Do illness beliefs predict psychological outcomes over time?

Multiple regression on those completing time 1 and time 2 assessments (n=201)

- Step one:
 - Illness representations (personal control, treatment control, illness coherence, consequences):
 - Causal beliefs (six items)
- Step two:
 - Illness representations
 - Causal beliefs
 - Baseline distress/PA/NA
 - Trait optimism

- Illness representations example:
- Do illness representations predict distress in people undergoing cancer genetic risk assessment?
- Illness representations (based on SRM)
 - personal control
 - treatment control
 - illness coherence
 - Consequences

Model Summary^b

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.501 ^a	.251	.233	13.852	.251	13.982	4	167	.000

a. Predictors: (Constant), t1 treatment control , t1 illness coherence, t1 consequences, t1 personal control

b. Dependent Variable: toties2

ANOVA^a

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	10731.942	4	2682.986	13.982	.000 ^b
	Residual	32045.619	167	191.890		
	Total	42777.561	171			

a. Dependent Variable: toties2

b. Predictors: (Constant), t1 treatment control , t1 illness coherence, t1 consequences, t1 personal control

Adjusted R Square: Accounting for sample size (1.0 = 100% explained)

Model explains 23.3% of variance for the DV (in this example)

ANOVA: is the model significantly better than simply making predictions using the mean score for the DV ($p < 0.05$ if significantly better)

Coefficients^a

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
	B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
(Constant)	-19.230	8.754		-2.197	.029					
1 t1 consequences	1.775	.253	.487	7.020	.000	.496	.477	.470	.932	1.073
t1 personal control	.153	.301	.039	.510	.611	.007	.039	.034	.782	1.278
t1 illness coherence	-.344	.402	-.059	-.856	.393	-.143	-.066	-.057	.953	1.049
t1 treatment control	.055	.491	.009	.112	.911	.078	.009	.007	.765	1.308

a. Dependent Variable: toties2

Values for the individual predictor variables:

Beta values: how much change in DV observed with change in IV (in Standard Deviations)

Higher the Beta score = more of a predictor for the dependent variable (better fit)

Note: can be a minus value (negative correlation)

p-value (sig.) also shown for each predictor variable

Predicting cancer-specific distress at Time 2

		β	Adjusted R^2	R^2 change	F
Model 1	Consequences	.46**	.21		5.17**
	Personal control	.02			
	Treatment control	.01			
	Illness coherence	-.03			
	Psychological attributions	.05			
	Risk factors	-.04			
	Harm/Neglect	-.00			
	Hereditary	.04			
	Chance/bad luck	.04			
	Poor medical care in past	.08			
Model 2	Consequences	.23**	.51	.29	22.52**
	Personal control	-.02			
	Treatment control	.03			
	Illness coherence	-.00			
	Psychological attributions	.06			
	Risk factors	.03			
	Harm/Neglect	-.01			
	Hereditary	-.04			
	Chance/bad luck	.01			
	Poor medical care in past	.04			
	Baseline distress	.46**			
	Baseline positive affect	-.06			
	Baseline negative affect	.22*			
	Trait optimism	-.06			

Stronger beliefs in the serious consequences of being found to be at increased risk of cancer significantly predicted higher levels of cancer specific distress at follow-up

Predicting Negative Affect at Time 2

		β	Adjusted R^2	R^2 change	F
Model 1	Consequences	.45**	.27		6.62**
	Personal control	.08			
	Treatment control	.03			
	Illness coherence	-.07			
	Psychological attributions	.00			
	Risk factors	-.14			
	Harm/Neglect	.09			
	Hereditary	.12			
	Chance/bad luck	.08			
	Poor medical care in past	.11			
Model 2	Consequences	.22**	.53	.26	21.75**
	Personal control	.01			
	Treatment control	.03			
	Illness coherence	-.05			
	Psychological attributions	.00			
	Risk factors	-.06			
	Harm/Neglect	.03			
	Hereditary	.02			
	Chance/bad luck	-.01			
	Poor medical care in past	.08			
	Baseline distress	.13			
	Baseline positive affect	-.08			
	Baseline negative affect	.51**			
	Trait optimism	-.02			

Stronger beliefs in the serious consequences of being found to be at increased risk of cancer significantly predicted higher levels of Negative Affect at follow-up

Predicting Positive Affect at Time 2

		β	Adjusted R^2	R^2 change	F
Model 1	Consequences	.09	.10		2.69*
	Personal control	-.09			
	Treatment control	.31**			
	Illness coherence	.03			
	Psychological attributions	-.02			
	Risk factors	.05			
	Harm/Neglect	.07			
	Hereditary	.13			
	Chance/bad luck	-.11			
	Poor medical care in past	.03			
Model 2	Consequences	.13	.43	.32	21.65**
	Personal control	-.07			
	Treatment control	.15*			
	Illness coherence	-.03			
	Psychological attributions	-.05			
	Risk factors	.05			
	Harm/Neglect	.03			
	Hereditary	.09			
	Chance/bad luck	-.07			
	Poor medical care in past	.04			
	Baseline distress	-.02			
	Baseline positive affect	.60**			
	Baseline negative affect	-.10			
	Trait optimism	-.02			

Stronger beliefs in screening/surgery helping reduce chances of getting cancer significantly predicted higher levels of positive affect at follow-up

Conclusions 1 (*the key findings bit*)

- Illness representations better able to predict negative psychological responses to the assessment process at both baseline and follow-up than positive outcomes.
- Whilst the model for positive affect only explained a small amount of the variance in positive affect at Time 2, an interesting finding was that stronger beliefs about screening/surgical options being able to control chances of developing cancer was a significant predictor of positive affect.
- Strongest predictors of all emotional responses at follow-up remain baseline levels of emotion and distress.

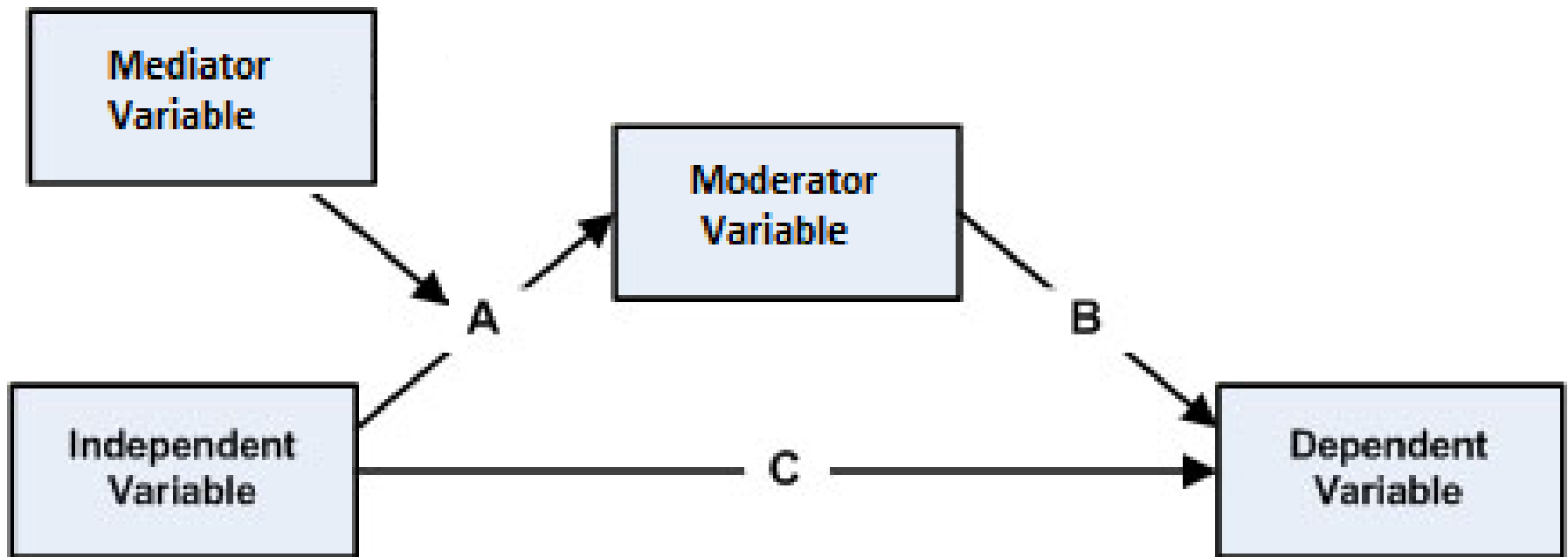
Conclusions 2 (*the implications bit*)

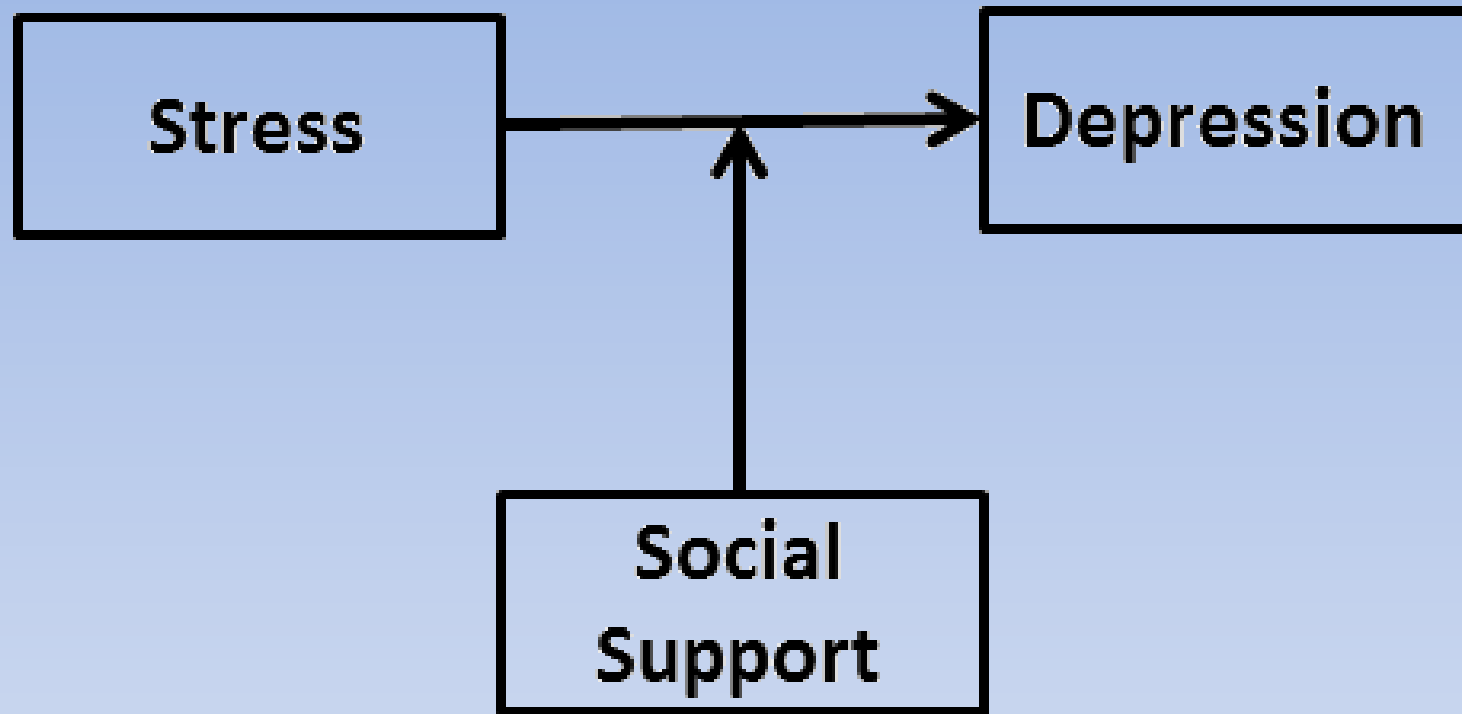
- Cancer genetic risk assessment process has the potential to change individuals' illness perceptions
- Post-risk differences in illness coherence may reflect differences in mechanisms by which individuals are informed.
- May also influence people's decision-making processes regarding genetic testing or risk-reducing options such as screening and surgery.
- Important for those involved in patient care to have an understanding of these psychological processes.
- Potential role of interventions aimed at changing health-related cognitions.

Moderation V Mediation

Moderation V Mediation

- A moderator variable influences the strength of a relationship between two other variables (affects the direction and/or strength of the relation between predictor variable and a criterion variable)
- A mediator variable explains the relationship between two other variables (accounts for relation between predictor and criterion)





The relationship between stress and depression may differ in strength at different levels of social support.

Moderation V Mediation

E.g., relationship between social class (SES) and frequency of breast self-exams (BSE).

- Age might be a moderator variable, in that the relation between SES and BSE could be stronger for older women and less strong or non-existent for younger women.
- Education might be a mediator variable in that it explains why there is a relation between SES and BSE. When you remove the effect of education, the relation between SES and BSE disappears.

https://www.youtube.com/watch?v=p7XmOCu_d4c