Psych 3102
Introduction to Behavior Genetics

Lecture 25
Health psychology
- stress and cardiovascular risk
- obesity and eating disorders

Health psychology = behavioral medicine

- the role of behavior in promoting health and preventing and treating disease
- new areas of study in behavior genetics:

    stress → cardiovascular risk

    body weight → obesity
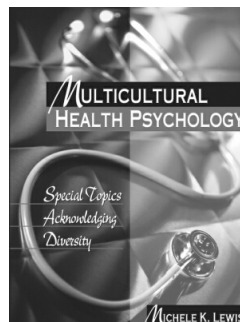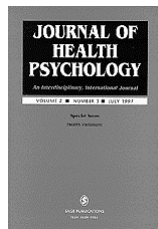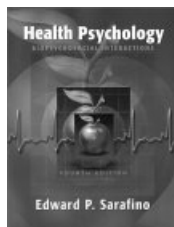
    additive behaviors   smoking, alcoholism

Stress and cardiovascular risk

cardiovascular disease leading cause of death in USA, in both
males and females

- individual reaction to stress may play a role in risk for cardiovascular disease

   large stress reaction associated with increase in cardiovascular disease

- reactions to stress have been shown to have a genetic component:

10 twin studies , varied age groups, mix of males and females, mixture of stressors (video games, reaction-time tests, color-word tests, mental math, speech tasks, mirror-drawing)

- stress reaction measured in terms of heart rate and blood pressure changes

- moderate gene influences: heritabilities   HR 30-50%

   BP 60-70%

- no shared environment

- reactions to stress outside lab much more difficult to measure over long term
   – people chose how and where to live
   – some avoid stress, some seek it out
   – gene influence likely to be present at all levels of choice

   Identifying contributing genes

   Using methods to locate QTLs –   +10 genes involved it blood pressure

   One QTL involved in reactivity of blood pressure to stress:

   alpha-1-antitrypsin gene   - product protects against inflammation
                                            - smoking/emphysema risk also

   rare single-gene effect: familial hypercholesterolemia

TABLE 2

Frequently Cited Randomized Trials of Behavioral Medicine Interventions in Cardiology*

NUMBER OF PATIENTS/TYPE OF DISORDER/REFERENCE	INTERVENTION	LENGTH OF FOLLOW-UP	OUTCOME MEASURE	FINDING	
				TREATMENT	CONTROL
117 patients with mild essential hypertension[70]	12 sessions, 45 minutes twice weekly, of breathing–relaxation training and biofeedback	1 year	Patients whose systolic or diastolic blood pressure decreased by >10% from baseline (%)	66%	32%[†]
48 patients with mixed coronary heart disease[71]	Lifestyle program of diet, exercise, stress management, smoking cessation, and group psychological support	5 year	Cardiac hospitalizations per patient	0.82	2.2[†]
			Any cardiac events per patient (MI, PTCA, CABG, cardiac hospitalization, and death)	0.89	2.3[†]
585 patients with myocardial infarction[72]	Scheduled interaction between case managers and patients after discharge: 14 nurses initiated telephone contacts; progress reports mailed to patients; and 4 individual nurse sessions of exercise testing, diet–drug therapy for hyperlipidemia, and smoking cessation	6 months after MI	Smokers who quit 2 months after MI (nonsmoking status was biochemically confirmed) (%)	70%	53%[‡]
			Functional capacity measured by stress tests in resting METS (higher score = better)	9.3 METS	8.4 METS[†]
52 patients with mixed coronary artery disease[73]	3 weekly groups of pain management and relaxation training, cognitive reframing, and problem solving	1 month	Weekly chest pain frequency (range 0 to > 5 times/day)	1 less episode/week	0.5 more/week[†]

TABLE 1

Frequently Cited Studies of Behavioral Medicine Interventions in Cancer Patients

NUMBER OF PATIENTS/TYPE OF DISORDER/REFERENCE	INTERVENTION	LENGTH OF FOLLOW-UP	OUTCOME MEASURE	FINDING	
				TREATMENT	CONTROL
Randomized trials					
86 patients with metastatic breast cancer[41]	Fifty-two 90-minute group sessions of coping, emotional expression, relaxation training, psychological support	10 years	Mean survival (SD)	36.6 months (37.6)	18.9 months (10.8)[†]
235 patients with metastatic breast cancer[56]	Fifty-two 90-minute group sessions of emotional expression, coping, psychological support	6 years	Median survival (NS)	17.9 months	17.6 months
		1 year	Mean TMD score (SD)*	Baseline, 35.8 (39.6)	Baseline, 27.6 (28.2)
					Change, 9.7 (24.6)[†]
66 patients with melanoma with anxiety and depression[54]	Six 90-minute group sessions consisting of health education, problem solving, stress management, psychological support	6 months	Mean TMD score (SD)*		Baseline, 44.46 (21.89)
					Change, 5.84[†]
Observational study					
6 patients with mixed cancer diagnoses with anticipatory emesis[55]	Three to five 30-minute individual hypnosis sessions before chemotherapy	Chemotherapy session	Anticipatory emesis	Change, −1.8 (31.7)	38/69 (55%) sessions with anticipatory emesis[‡]

*Derived from Profile of Mood States. Total Mood Disturbance (TMD) scores range from 0 to 232; higher scores = lower mood. Although there are no norms for the TMD score, the reduction for the treatment group probably represents a clinical reduction in overall distress, with no such improvement noted for the control group.
[†]$P < 0.01$.
[‡]$P < 0.05$.

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Eating disorders

- severe disturbances in eating behavior

anorexia nervosa (AN)   extreme dieting, avoidance of food,
                   fear of weight gain, extremely low body weight
     females : highest mortality among psychiatric disorders

bulimia nervosa (BN)   binge eating followed by vomiting /
              purging, not necessarily accompanied by low weight
Onset: late adolescence, early adulthood, mostly females
Prevalence: AN 1 – 2% females   0.1 – 0.2% males

- familial, frequently comorbid with mood and anxiety disorders
       AN and MDE genetic correlation = 0.58
    34% of genetic variance is common between AN, MDE

First twin study 1991   anorexia
    Concordances: MZ = 59%
                    DZ = 8%
- clear genetic influence   heritability ~ 58% e^2 = 42%

First studies on bulimia indicated no gene influence
    MZ = DZ concordance ~37%
Recent studies show bulimia has been diagnosed with very
    low reliability (kappa = 0.28)

 - with more reliable measures, recent family and twin studies
    indicate stronger genetic influence

- several candidates genes found for both disorders

Body weight and obesity

- animal studies indicate genetic component for tendency towards certain body mass

  can selectively breed for fat mass/ muscle mass

Health problems caused by high body weight/obesity

"..obesity is probably the second leading preventable cause of death in the US ."
  Manson, 1999

High body mass
- increases risk for Type II diabetes, heart disease and cancer
1/3 of cancer deaths are related to diet and inactivity (American Cancer Society)

- contributes more to health-care costs than either smoking or problem drinking

- reaching obesity has same effect on chronic health conditions as aging from 30 to 50

Sturm (2000)   financial burden of obesity

Survey of 10,000 households, 1997-98 , 18 - 65 year-olds
- all effects compared with non-drinking, non-smoking, healthy weight members

obesity:

  36% increase in hospital/outpatient spending

  77% increase in medication costs

smoking:

  21% increase in health services

  28% increase in medication costs

problem drinking:

  10% increase in health services

  decrease in medication costs

Science   'Special issue' on Obesity Feb, 2003

- identified obesity as .." the great public health irony of the 21st Century
  - hundreds of millions of people across the world lack adequate food and suffer deficiency diseases
  - hundreds of millions in other parts of the world overeat to the point of increasing their risk of diet-related chronic diseases

- problems associated with obesity divert scarce resources away from food security in poorer countries to take care of people with preventable heart disease and diabetes

- food is overproduced in richer nations
  - USA   food supply provides 3800 kcals/person/day
  - = 2 times that required by most adults, given their lifestyles

- supplying food is 'big business' in richer nations
  - large adjustments to the US economy would have to be made if people ate more healthily
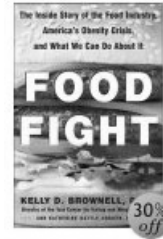
Prevalence of overweight and obesity

Worldwide   +1 billion adults are overweight
            300 million are clinically obese

UK   obesity rate has tripled in 20 years
      2/3 of adults are overweight

USA   20 states have obesity rate of 15 - 19%
       29 states have obesity rate of 20 - 24%
       1 state has rate of +25%

France   obesity rate if 8%   but rising

Measurement of body mass

- body mass index (BMI)

$$BMI = \frac{\text{weight in kg}}{\text{height in m}^2} \qquad \frac{\text{wt(lbs) x 703}}{\text{ht(ins)}^2}$$

<u>BMI</u>

10 -12	Dead
16.7	Kate Moss
26.5	mean, US adults
25	'overweight' ← US government guidelines
~28	morbidity increases
30	obese ←
40	'morbidly obese'
43.7	average sumo wrestler
45	Chris Farley when he died

6' 185lbs
5'5'' 150lbs

5'5'' 180lbs
6' 220lbs

Food intake

- based on self-reports, the obese do not eat more than people with healthier weights:

  correlation between self-reported food intake and obesity = -0.16

- when food intake is actually measured

  correlation = 0.56

Twin correlations for food intake: MZ > DZ
  - indication of genetic influence

- several genes influencing appetite have been located

Family, twin and adoption studies on body weight

- indicate genetic influence on body weight

Relationship   Correlations for body weight

Relationship	Correlations for body weight	
MZ	0.82	reared apart = 0.72
DZ	0.43	
Siblings	0.34	
Adoptive sibs (non-bio)	0.01	
Parent/offspring	0.26	
Parent/adopted-away offspring	0.23	
AdoptiveParent/offspring	0.00	
Spouses	0.13	

- very little shared environment
- heritability ~ 70%   additive genes
- non-shared environment

similar results for BMI and skin-fold thickness

- most variation for body mass seems to come from genes not the environment
- to maintain a healthy body weight, each person will have to be eating /exercising to different extents depending on their genetic tendencies

Gene influence could act at all possible levels, including interacting with the environment:

      BMR   - internal, physiological controls

      appetite control   - hormonal / brain interaction

      tendency to be active / exercise

      personality   - will-power to change, attitudes to what constitutes overweight /obesity

      attitudes to eating

Developmental aspects

longitudinal twin studies:

Correlations   at birth: MZ = DZ = 0.6 – 0.7 $h^2 = 0$

                        at 1 year: MZ = 0.87

                                 DZ =   0.58 $h^2 = 60\%$

- birthweight is NOT a good indicator of future weight
- no genetic variation for birthweight indicated
- best predictor of future weight is rapid growth of body fat around age 6
  – earlier spurt is correlated with obesity in adulthood

  most genes contribute to continuity – lifestyle changes to maintain healthy weight also have to be continuous

Identifying genes for obesity

- 'obese' gene in mice   1950's   recessive allele→ obesity
- gene cloned in 1994
- product identified as leptin

  - a hormone that decreases

  appetite and increases energy use in mice → thin mouse
- leptin receptor gene active in mouse brain
- same hormone found in humans, gene for leptin chr 2

  - little/no variation for it is found

  - leptin receptor now being studied
- several genes influencing levels of leptin now being investigated
- many other genes found to have influence, replication of studies needed