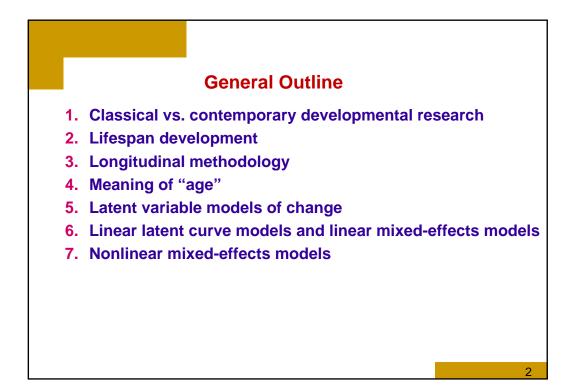


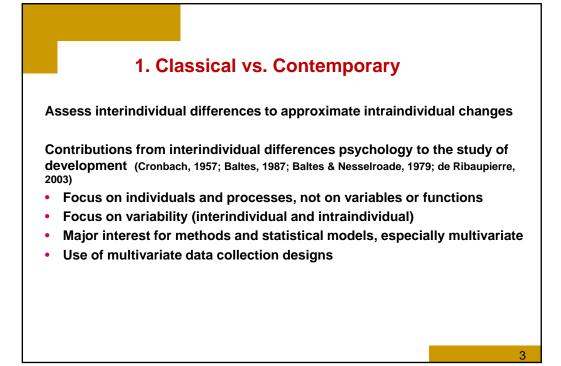
Cognitive development across the lifespan: Conceptual, methodological and analytical challenges of a lifespan approach

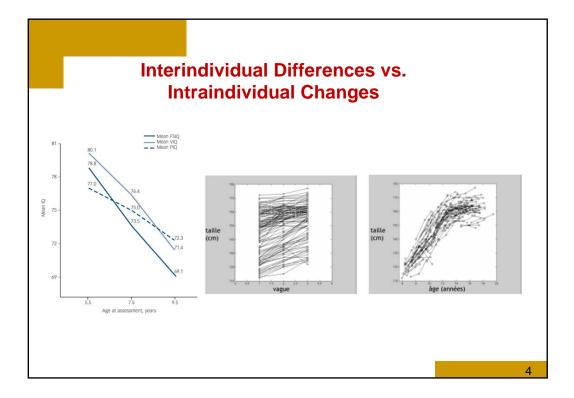
Part 1

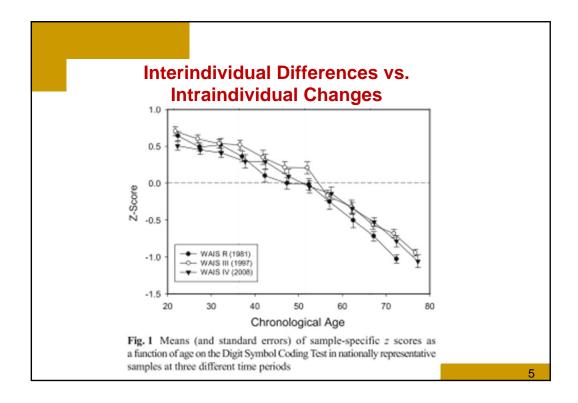
Paolo Ghisletta University of Geneva ISSBD Workshop, Sept 3 2015

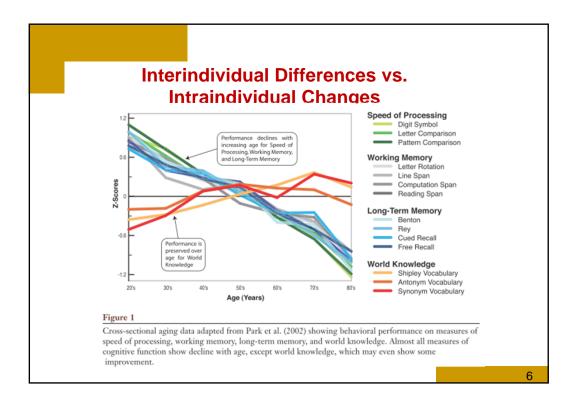


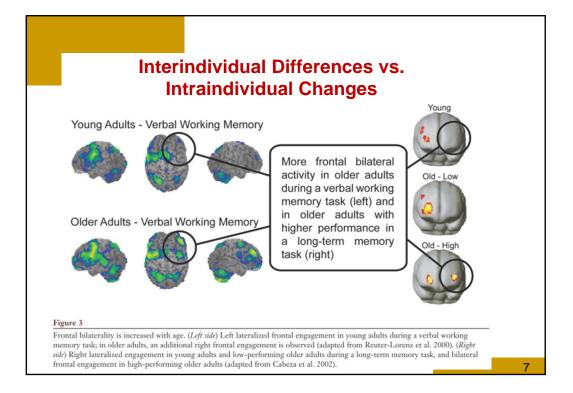


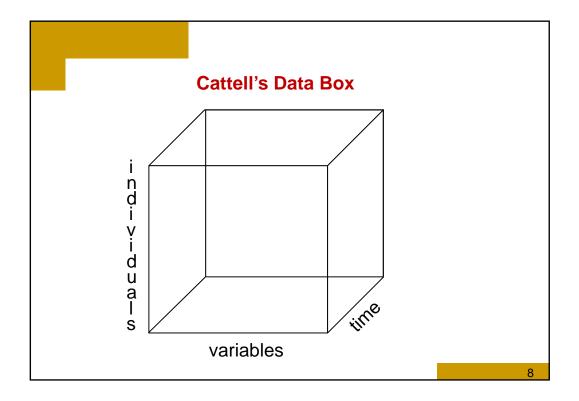


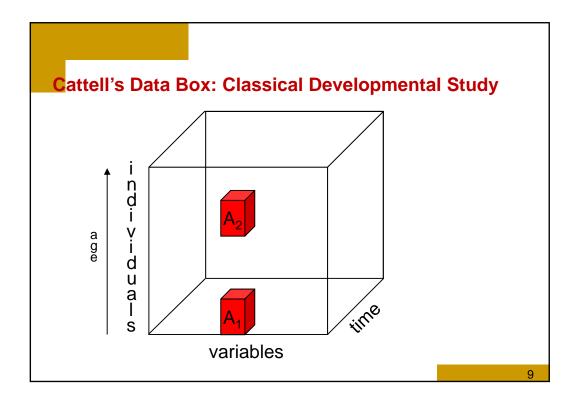


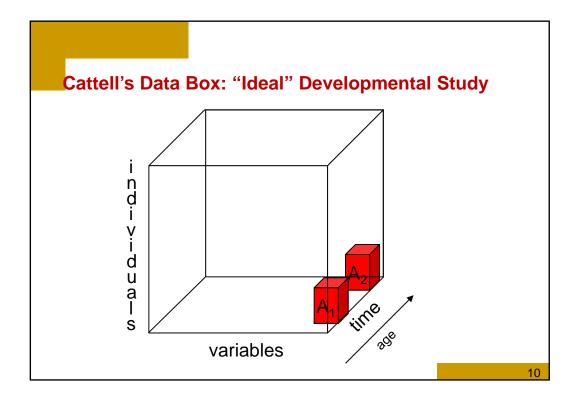


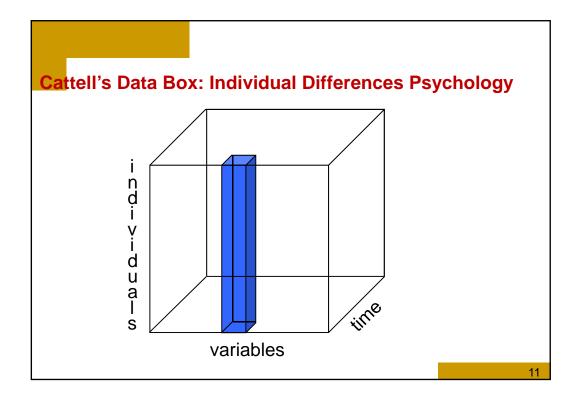


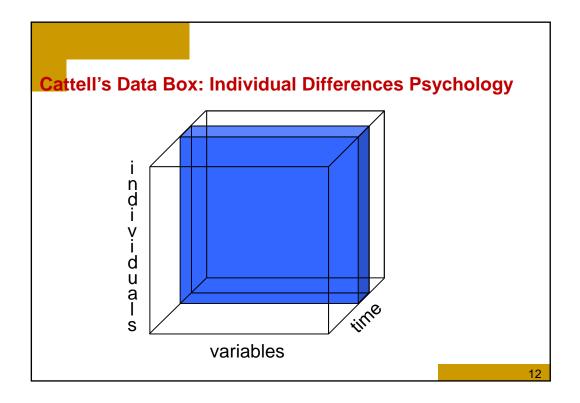


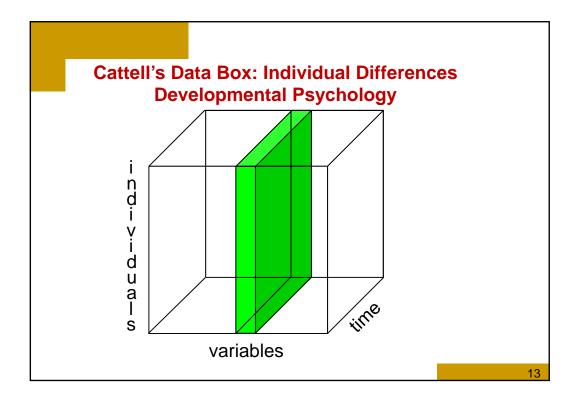


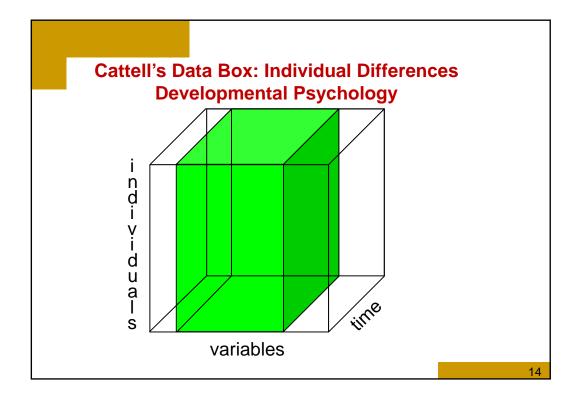












1. Classical vs. Contemporary: Conclusions

Cognitive aging is the field that most successfully implements methodological convergence (Schaie, Baltes)

French-speaking individual-differences psychologists in child development (Reuchlin, Lautrey, de Ribaupierre)

Developmental studies necessitate longitudinal methodologies

Methodological expertise is required in developmental psychology

The lifespan approach best reunites individual-difference approach and developmental psychology. Deep comprehension of the age variable and of longitudinal methodologies is necessary.

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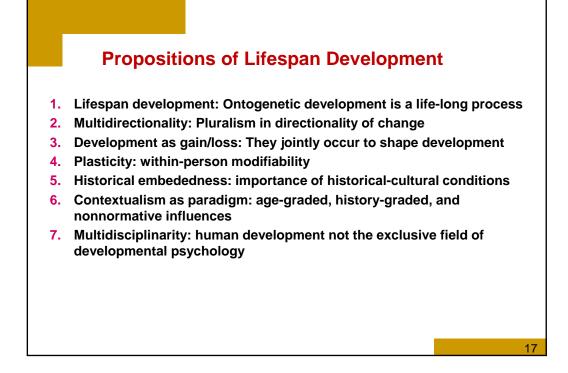
2. Lifespan Development

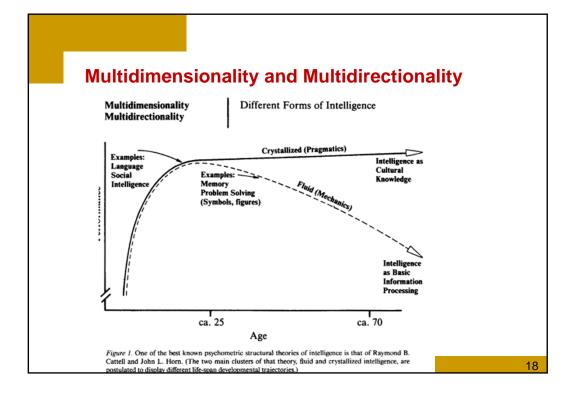
Baltes, P. B. (1987). Theoretical propositions of life-span developmental psychology :On the dynamics between growth and decline. *Developmental Psychology, 23*, 611-626

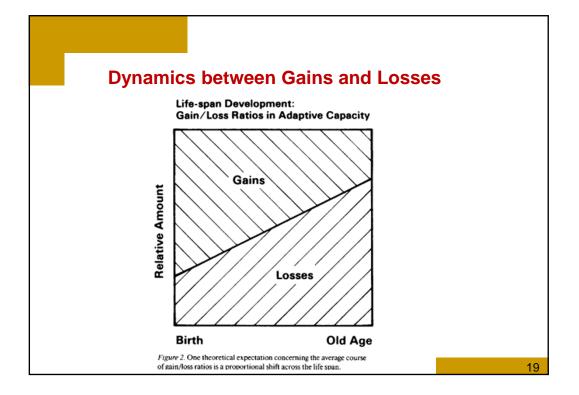
"Life-span developmental psychology involves the study of constancy and change in behavior throughout the life course (ontogenesis), from conception to death. The goal is to obtain knowledge about general principles of life-long development, about interindividual differences and similarities in development, as well as about the degree and conditions of individual plasticity or modifiability of development." (p. 611)

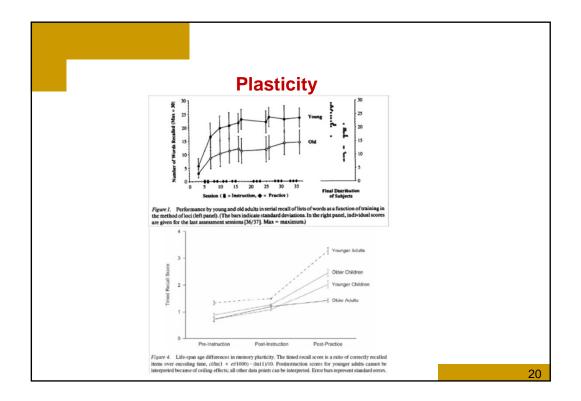
"The most general orientation toward this subject matter is simply to view behavioral development as a lifelong process." (p. 612)

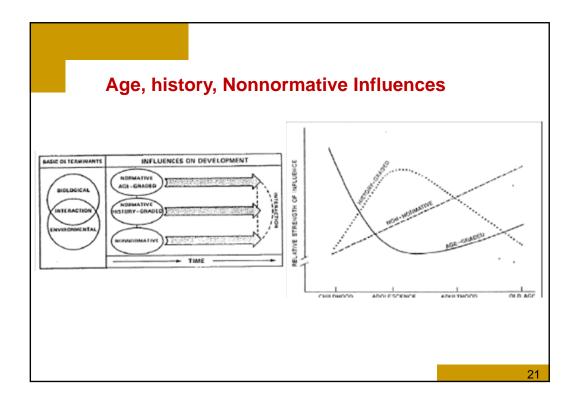
For many researchers, the life-span orientation entails several prototypical beliefs that, in their weighting and coordination, form a family of perspectives that together specify a coherent metatheoretical view on the nature of development. (p. 612) 16







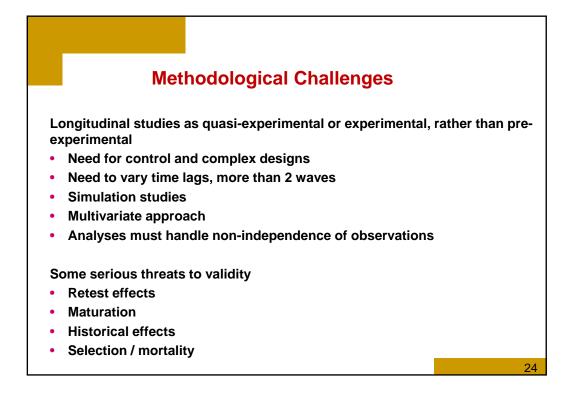


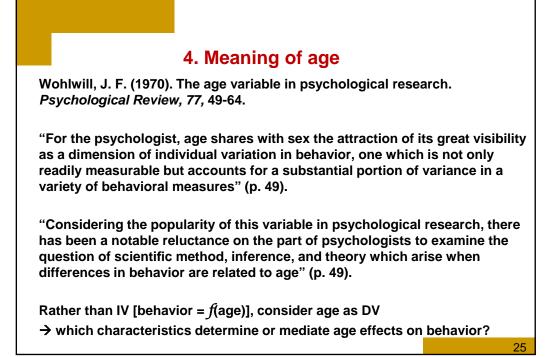


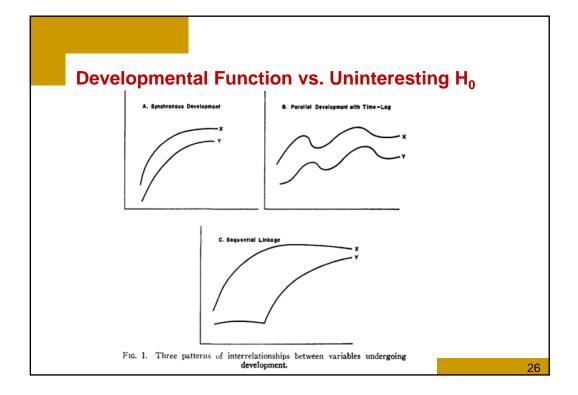
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Five Objectives

- 1. Direct identification of intraindividual change
- 2. Direct identification of interindividual differences (similarity) in intraindividual change
- 3. Analysis of interrelationships in behavioral change
- 4. Analysis of causes (determinants) of intraindividual change
- 5. Analysis of causes (determinants) of interindividual differences in intraindividual change







Sliwinski et al. (2003)

Sliwinski, M. J., Hofer, S. M., Hall, C., Buschke, H., & Lipton, R. B. (2003). Modeling memory decline in older adults : The importance of preclinical dementia, attrition, and chronological age. *Psychology and Aging, 18*, 658-671.

This longitudinal study examined memory loss in a sample of 391 initially nondemented older adults. Analyses decomposed observed memory loss into decline associated with preclinical dementia, study attrition, terminal decline, and chronological age. Measuring memory as a function of only chronological age failed to provide an adequate representation of cognitive change. Disease progression accounted for virtually all of the memory loss in the 25% of the sample that developed diagnosable dementia. In the remainder of the sample, both chronological age and study attrition contributed to observed memory loss. These results suggest that much of memory loss in aging adults may be attributable to the progression of preclinical dementia and other nonnormative aging processes that are not captured by chronological age.

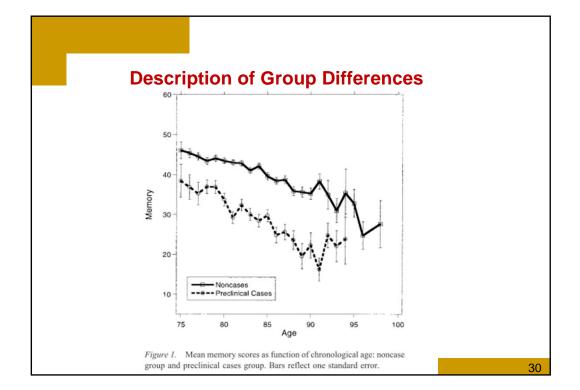
Sliwinski et al. (2003)

We argue here that representing cognition as a function of chronological age can obscure identification and modeling of important causes of cognitive change, such as the progression of preclinical dementia. Alternative representations of cognitive change are required to understand such nonnormative influences on cognition. (p. 658)

Wohwill (1970, p. 49) has argued that chronological age "be incorporated into the dependent variable in developmental studies" by defining it in terms of parameters of functional change. Modern approaches to longitudinal data analyses (i.e., multilevel modeling) have in some sense met Wohlwill's demand. (p. 658)

Sam	ple	Data
Juni		Pulu

Person	Wave	Age (years)	Disease progression (years)	Memory score
1	1	67	-3.25	100.00
1	2	68	-2.25	88.50
1	3	69	-1.25	74.00
1	4	70	-0.25	56.50
1	5	71	0.75	36.00
1	6	72	1.75	12.50
2	1	55	-5.00	100.00
2	2	56	-4.00	93.50
2	3	57	-3.00	84.00
2	4	58	-2.00	71.50
2	5	59	-1.00	56.00
2	6	60	0.00	37.50



Statistical Modeling

Table 1 Models of Intraindividual Change

Model	Construct	Measurement (within-person) model	Structural (between-persons) mode
1	Chronological aging	$y_{ij} = \pi_{0j} + \pi_{1j} Age_{ij} + \pi_{2j} Age_{ij}^2 + r_{ij}$	$\pi_{0j} = \beta_{00} + U_{0j} \pi_{1j} = \beta_{10} + U_{1j} = 0$
la	Chronological aging	$y_{ij} = \pi_{0j} + \pi_{1j} Age_{ij} + \pi_{2j} Age_{ij}^2 + r_{ij}$	$\begin{aligned} \pi_{2j} &= \beta_{20} + U_{2j}^{*} \\ \pi_{0j} &= \beta_{00} + \beta_{01}(\text{PreDx}) + U_{0j} \\ \pi_{1j} &= \beta_{10} + \beta_{11}(\text{PreDx}) + U_{1j} \\ \pi_{2j} &= \beta_{20} + \beta_{21}(\text{PreDx}) + U_{2j} \end{aligned}$
2	Dementia	$y_{ij} = \pi_{0j} + \pi_{1j} \text{ToD} \mathbf{x}_{ij} + \pi_{2j} \text{ToD} \mathbf{x}_{ij}^2$ $+ r_{ij}$	$\pi_{2j} = \beta_{20} + \beta_{21}(\text{TEDX}) + \delta_{2j}$ $\pi_{0j} = \beta_{00} + U_{0j}$ $\pi_{1j} = \beta_{10} + U_{1j}$
2a	Aging + dementia	$y_{ij} = \pi_{0j} + \pi_{1j} \operatorname{Age}_{ij} + \pi_{2j} \operatorname{Age}_{ij}^2 + \\ \pi_{3j} \operatorname{ToDx}_{ij} + \pi_{4j} \operatorname{ToDx}_{ij}^2 + r_{ij}$	$\begin{aligned} \pi_{2j} &= \beta_{20} + U_{2j} \\ \pi_{0j} &= \beta_{00} + U_{0j^{\flat}} \\ \pi_{3j} &= \beta_{30} + U_{3j} \end{aligned}$

Results for Entire Sample

Table 4

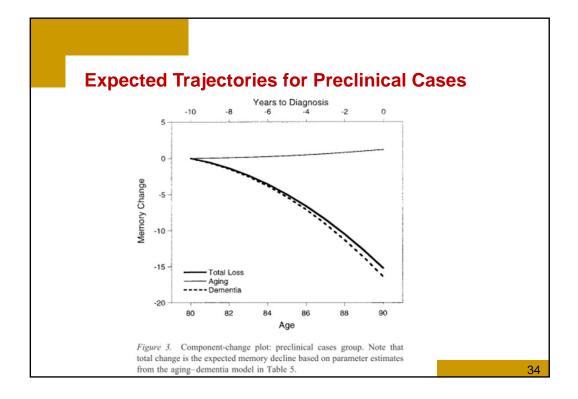
Fixed and Random Effects: Pooled Noncase and Preclinical Case Groups (N = 391)

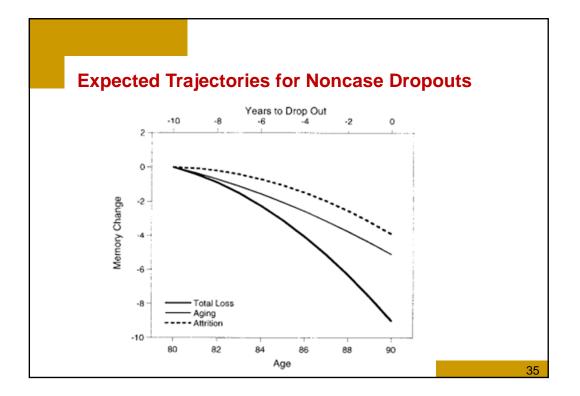
	Mo	del 1	Model 1a			
Fixed effect	Coefficient	95% CI	Coefficient	95% CI		
Intercept, β_{00}	36.76	35.63, 37.88	39.93	38.75, 41.09		
PreDx, β_{01}			-11.88	-14.25, -9.62		
Linear age, β_{10}	-1.00	-1.18, -0.84	-0.88	-1.07, -0.69		
PreDx, B ₁₁			-0.26	-0.66, 0.13		
Quadratic age, β_{20}	-0.031	-0.05, -0.01	-0.03	-0.06, -0.01		
PreDx, β_{21}			0.020	-0.04, 0.08		
-2 log-likelihood	-10,056.30		9,953.70			
BIC	10,107.30		10,026.50			

Note. Linear age = age - 85; quadratic age = $(age - 85)^2$. CI = confidence interval; PreDx = prediagnosis (1 if preclinical dementia, 0 otherwise); BIC = Bayesian Information Criterion.

Results for Preclinical Cases

		odel 1	Me	odel 2	Model 2a		
Fixed effect	Coefficient	95% CI	Coefficient	95% CI	Coefficient	95% CI	
intercept	27.99	25.72, 30.26	34.67	32.85, 36.50	34.83	32.38, 37.28	
inear age	-1.21	-1.62, -0.79			0.12	-0.33, 0.57	
Quadratic age	-0.03	-0.08, 0.03			0.02	028, 0.07	
Linear ToDx			-1.58	-1.30, -1.85	-1.65	-1.27, -2.03	
Quadratic ToDx			-0.11	-0.17, -0.06	-0.12	-0.17, -0.06	
-2 log-likelihood	-2,289.20		-2,236.50		-2,231.20		
BIC	2,329.60		2,271.10		2,283.30		
	ge – 85; quadratic ag			linear ToDx = years to		atic ToDx = (y	





Gerstorf et al. (2013)

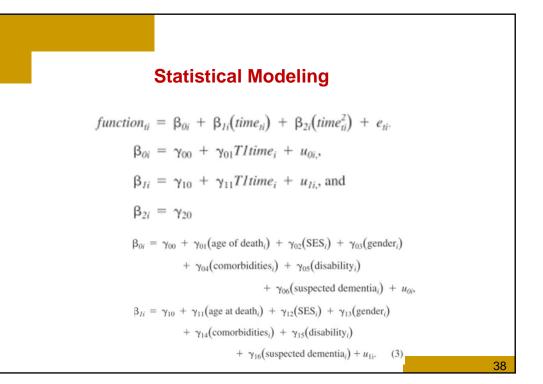
Gerstorf, D., Ram, N., Lindenberger, U., & Smith, J. (2013). Age and time-to-death trajectories of change in indicators of cognitive, sensory, physical, health, social, and self-related functions. *Developmental Psychology*, *49*, 1805-1821.

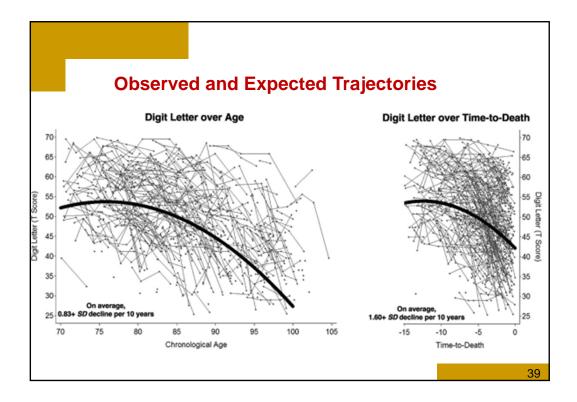
Relatively little is known about what aspects of late-life functioning are prone to "attacks" from mortality-related processes and what factors contribute to end-of-life decline. We use longitudinal data from the Berlin Aging Study to examine the multidimensional nature of late-life change. To broadly represent central characteristics of individual functioning, we selected six domains and well-established indicators thereof from the measurement battery of the BASE. We pursue two sets of goals. First, we apply growth models to characterize late-life change trajectories in key indicators of cognitive, sensory, physical, health, social, and self-related functions across chronological age and time-to-death to determine whether mortality-related processes do indeed generalize across multiple domains of function. Second, we explore the role of sociodemographic characteristics and proxies of pathologies as correlates of mortality-related decline.

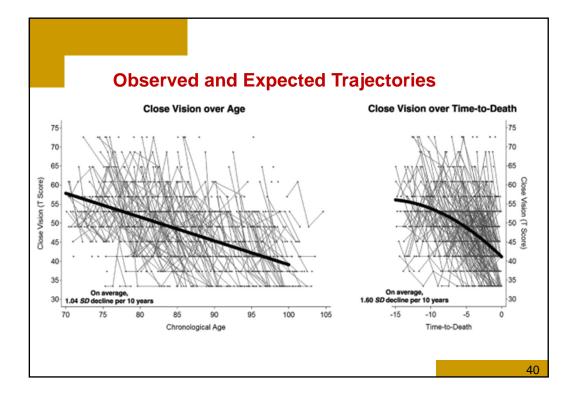
Descriptive Data

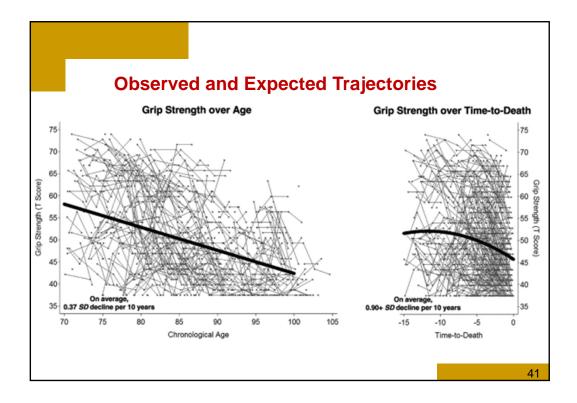
Table 1 Descriptive Statistics for the Cognitive Indicator (Digit Letter) Over Age and Time-to-Death

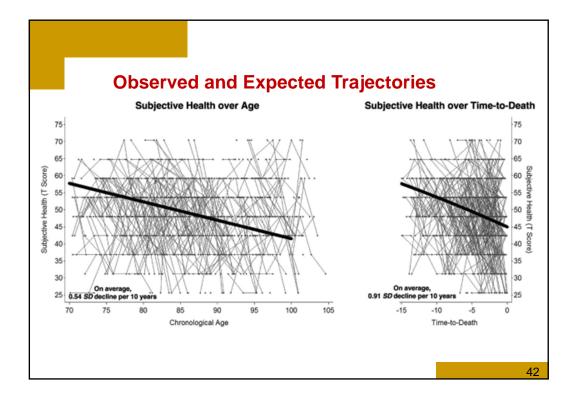
		Chrone	ological age		Time-to-death					
	Age	п	Estimate	SE	Years before death	п	Estimate	SE		
Between-person variance			53.42	7.93			5.49	0.61		
Within-person variance			7.93	0.46			7.53	0.42		
ICC			.87				.42			
			М	SD			М	SD		
	70	5	51.05	7.80	16	2	51.46	8.69		
	71	9	58.55	6.80	15	11	59.96	6.35		
	72	15	58.26	7.14	14	14	55.04	9.26		
	73	13	54.69	10.43	13	24	55.08	9.41		
	74	27	56.12	7.85	12	27	57.63	7.95		
	75	31	55.98	7.49	11	27	56.83	6.55		
	76	32	56.56	8.24	10	41	57.29	6.85		
	77	25	55.23	8.68	9	42	54.43	9.36		
	78	38	54.65	8.32	8	50	53.32	8.84		
	79	41	54.45	8.16	7	63	53.72	8,56		
	80	46	52.61	9.26	6	72	51.20	10.65		
	81	39	52.83	8.62	5	93	50.44	9.32		
	82	45	53.54	9.34	4	114	48.22	9.44		
	83	41	51.60	7.72	3	116	47.41	8.96		
	84	40	52.92	8.90	2	129	45.98	10.52		
	85	57	52.25	9.63	1	124	45.90	9.96		
	86	45	48.42	8.67	0	40	44.92	10.29		
	87	42	49.44	11.15						

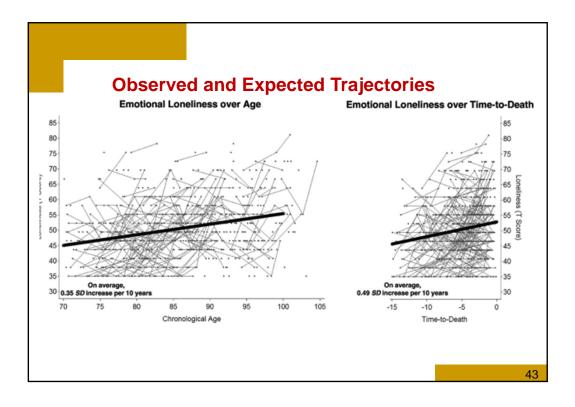


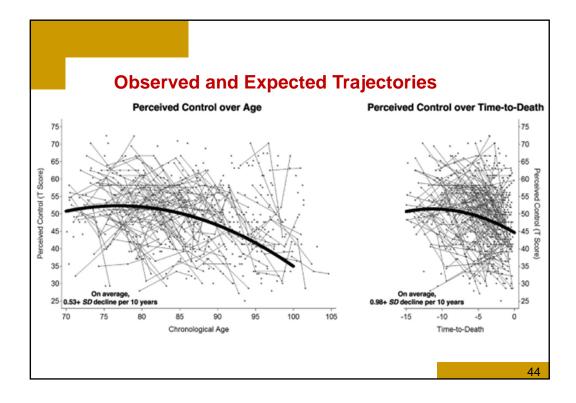






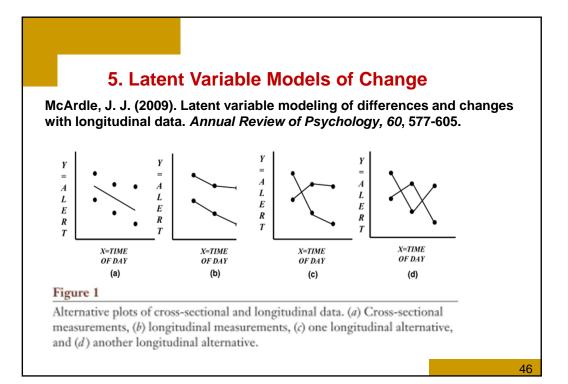


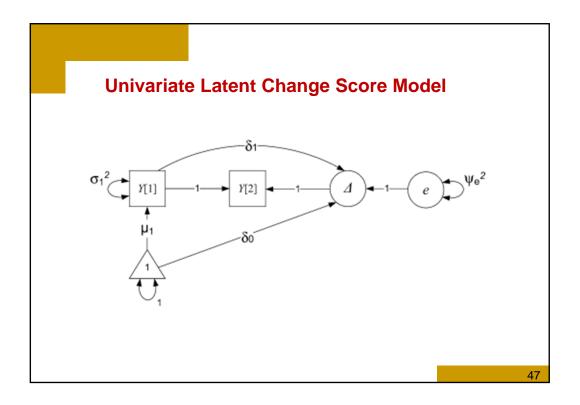




Role of Covariates

Variable	Cognitive: Digit Letter Estimate (SE)	Sensory: Close vision Estimate (SE)	Physical: Grip strength Estimate (SE)	Health: Self-rated health Estimate (SE)	Social: Loneliness Estimate (SE)	Self: Perceived control Estimate (SE)
Fixed effects						
Intercept, Yoo	46.13" (0.40)	44.95* (0.39)	48.08* (0.27)	47.43* (0.44)	51.66* (0.50)	47.50* (0.50)
Time-to-death, γ_{10}	$-1.56^{\circ}(0.11)$	$-1.46^{*}(0.14)$	$-0.84^{*}(0.10)$	$-0.86^{\circ}(0.16)$	0.46" (0.17)	$-0.79^{*}(0.18)$
Time-to-death ² y ₂₀	$-0.07^{*}(0.01)$	$-0.05^{*}(0.02)$	$-0.03^{*}(0.01)$	$-0.04^{\circ}(0.02)$	0.00 (0.01)	-0.03(0.02)
Age at death, γ_{01}	-0.45* (0.06)	-0.34* (0.06)	$-0.39^{*}(0.04)$	0.09 (0.06)	0.22° (0.07)	$-0.20^{*}(0.07)$
SES, y ₀₂	0.25* (0.04)	0.09* (0.04)	0.07* (0.03)	0.08 (0.04)	$-0.14^{\circ}(0.05)$	-0.02(0.05)
Women, yor	0.49 (0.81)	-1.44(0.78)	$-9.72^{*}(0.56)$	$-1.87^{*}(0.90)$	1.65* (0.99)	$-2.74^{*}(0.99)$
Comorbidities, You	-0.05(0.04)	-0.05(0.04)	$-0.09^{*}(0.03)$	$-0.09^{*}(0.04)$	0.11* (0.05)	0.01 (0.05)
Disability, yos	$-2.98^{*}(0.85)$	-1.52(0.82)	$-1.66^{\circ}(0.59)$	-0.81(0.95)	0.80 (1.05)	$-4.46^{*}(1.05)$
Sus. dementia, You	$-6.68^{*}(0.84)$	-0.99(0.81)	$-0.85^{*}(0.58)$	1.23 (0.93)	0.67 (1.03)	1.31 (1.03)
Age at Death \times Time-to-Death, γ_{II}	$-0.03^{*}(0.01)$	-0.01(0.01)	0.02* (0.01)	-0.01(0.01)	0.00 (0.01)	-0.02(0.01)
SES \times Time-to-Death, γ_{12}	0.00 (0.01)	$-0.02^{*}(0.01)$	0.00 (0.01)	-0.01(0.01)	0.01 (0.01)	-0.01(0.01)
Women \times Time-to-Death, γ_{I3}	-0.04(0.14)	-0.01(0.17)	0.49* (0.11)	0.19 (0.16)	-0.22(0.16)	0.15 (0.17)
Comorbidities \times Time-to-Death, γ_{14}	0.00 (0.01)	0.00 (0.01)	$-0.01^{*}(0.00)$	0.01 (0.01)	-0.01(0.01)	-0.01 (0.01)
Disability \times Time-to-Death, γ_{15}	$-0.32^{*}(0.15)$	-0.18(0.19)	$-0.29^{*}(0.12)$	0.07 (0.18)	0.24 (0.17)	-0.04(0.18)
Sus. Dementia × Time-to-Death, Y16	$-0.59^{*}(0.23)$	0.26 (0.18)	0.04 (0.11)	0.37* (0.17)	0.01 (0.17)	0.57* (0.18)
Sus. Dementia \times Time-to-Death ² , γ_{17}	$-0.06^{+}(0.02)$	_	_	_		_
Random effects						
Intercept, $\sigma_{\mu 0}^2$	47.33* (4.31)	36.69" (4.41)	17.19* (2.18)	45.84* (4.92)	63.40° (7.45)	59.38* (7.59)
Time-to-death, σ_{ul}^2	0.34* (0.11)	0.62* (0.15)	0.01 (0.00)	0	0.07 (0.14)	0.03 (0.18)
Cov. Intercept \times Time-to-Death, $\sigma_{\mu 0 \mu l}$	1.56* (0.54)	2.72* (0.69)	-0.37(0.26)	a	1.42 (0.98)	0.53 (1.00)
Explained variance (between person)						
In intercept, $\sigma_{\mu0}^2$.48	.28	.72	.13	.15	.14
In time-to-death, $\sigma_{\mu J}^2$.38	.26	.94	a	a	a
Residual, σ_e^2	11.47* (0.83)	26.89* (1.80)	17.47* (0.97)	44.75* (2.51)	28.36* (2.87)	32.05* (3.53)
Note. Ns range between 404 (Digit Letter vision). Unstandardized estimates and stand not included because many of these were pr intercept = Covariance intercept. Dashes ir ⁸ For model convergence, variance of time-	ard errors are pre- eviously found to idicate that effect	sented. Intercept i be nonsignificant. was not estimated	s located at 2 year SES = socioecon I.	s prior to death. Leve omic status; Sus. Dem	1 2 versions of tim entia = Suspected	ne-to-death were

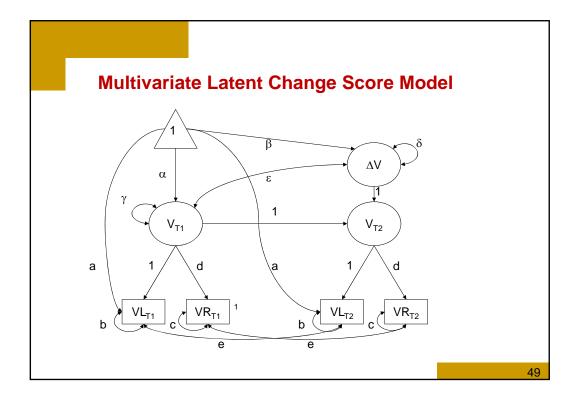


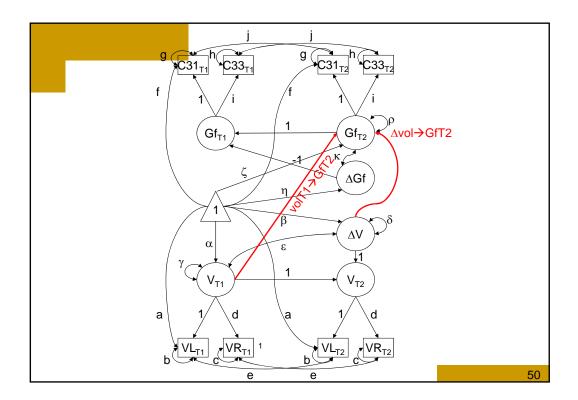


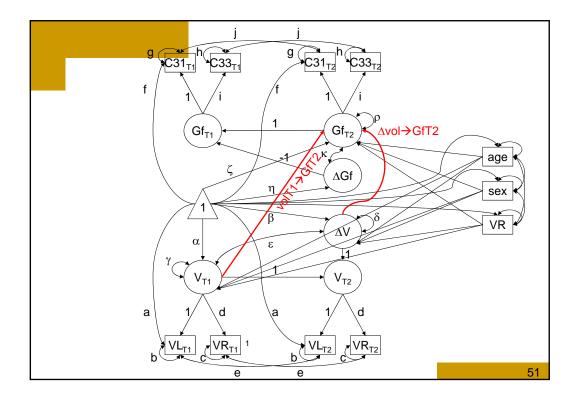
Raz et al. (2008)

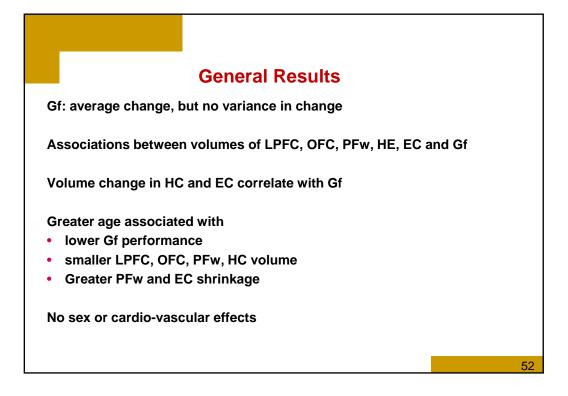
Raz, N., Lindenberger, U., Ghisletta, P., Rodrigue, K. M., Kennedy, K. M., & Acker, J. D. (2008). Neuroanatomical correlates of fluid intelligence in healthy adults and persons with vascular risk factors. Cerebral Cortex, 18, 718-726.

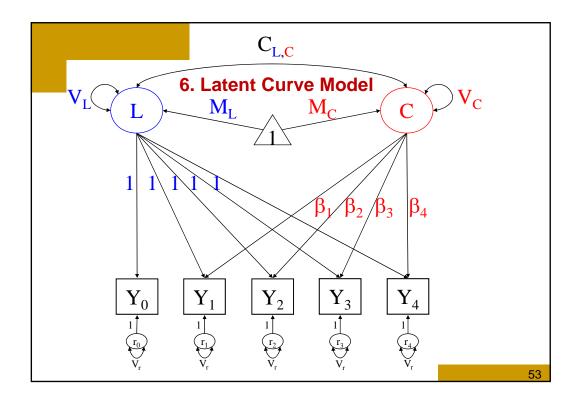
The main objective of this study was to examine the effects of regional brain changes on cognitive decline and the modifying influence of vascular risk (VR) factors. We present latent difference score analyses of associations among 5-year changes in 12 regional brain volumes and age-sensitive cognitive functions in 87 adults (32 with identifiable VR factors). We found reliable individual differences in volume change for 11 of the 12 brain regions but not in the cognitive measures that showed average longitudinal decline. Thus, associations between rates of change in fluid intelligence and brain volumes could not be assessed. We observed, however, that lower levels of fluid intelligence were associated with smaller prefrontal and hippocampal volumes. Lower fluid intelligence scores were also linked to greater longitudinal shrinkage of the entorhinal cortex (EC). After accounting for the effects of age, sex, and VR factors, the orbitofrontal cortex and the prefrontal white matter (PFw) volumes as well as the 5-year change in the EC volume predicted fluid intelligence level. VR was independently associated with smaller prefrontal volumes and lower fluid intelligence.

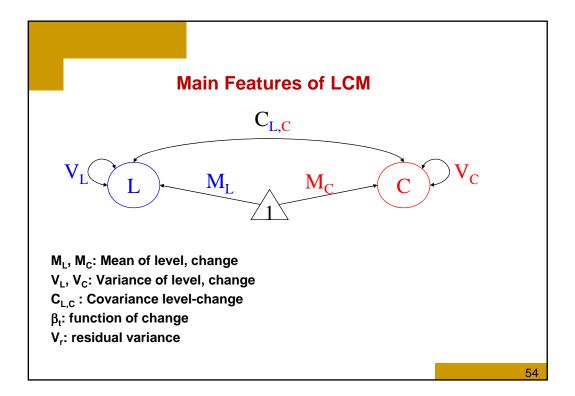












Latent Curve Model / Linear Mixed Effects Model

Under several conditions, the two approaches are equivalent.

Generally speaking, the LCM approach is more flexible, allows more extensions, but also requires a greater understanding.

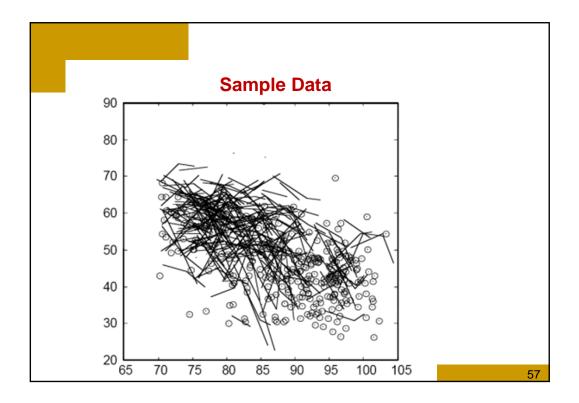
Computationally speaking, the LMEM approach is more efficient (especially with sparse data)

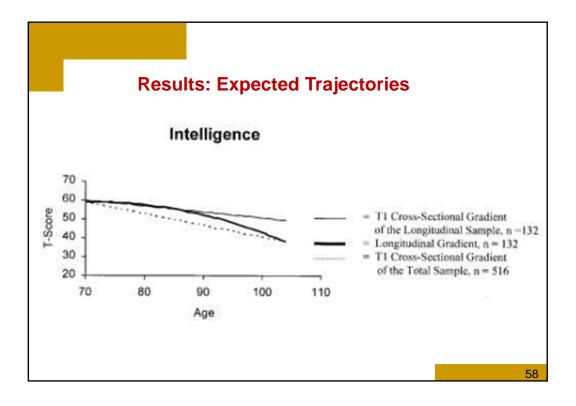
Singer et al. (2003)

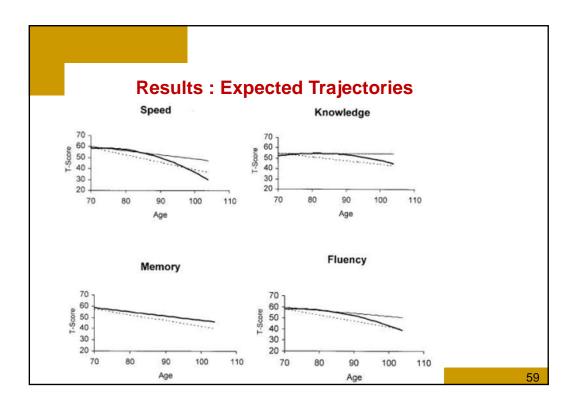
Singer, T., Verhaeghen, P., Ghisletta, P., Lindenberger, U., & Baltes, P. B. (2003). The fate of cognition in very old age : Six-year longitudinal findings in the Berlin Aging Study (BASE). Psychology and Aging, 18, 318-331.

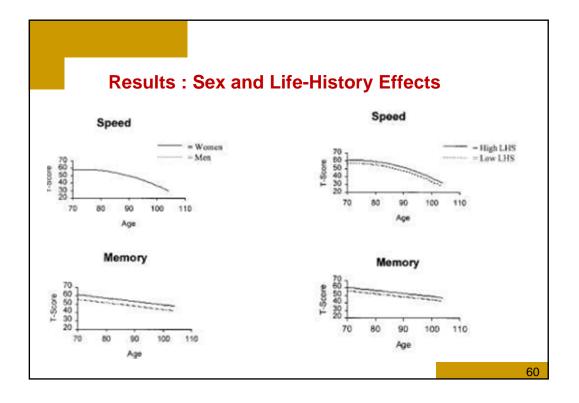
The authors report full-information longitudinal age gradients in 4 intellectual abilities on the basis of 6-year longitudinal changes in 132 individuals (mean age at T 1 = 78.27, age range = 70–100) from the Berlin Aging Study. Relative to the crosssectional parent sample (N=516, mean age at T 1 = 84.92 years), this sample was positively selected because of differential mortality and experimental attrition. Perceptual speed, memory, and fluency declined with age. In contrast, knowledge remained stable up to age 90, with evidence for decline thereafter. Age gradients were more negative in old old (n = 66, mean age at T 1 = 83.04) than in old (n = 66, mean age at T 1 = 73.77) participants. Rates of decline did not differ reliably between men and women or between participants with high versus low life-history status. They conclude that intellectual development after age 70 varies by distance to death, age, and intellectual ability domain.

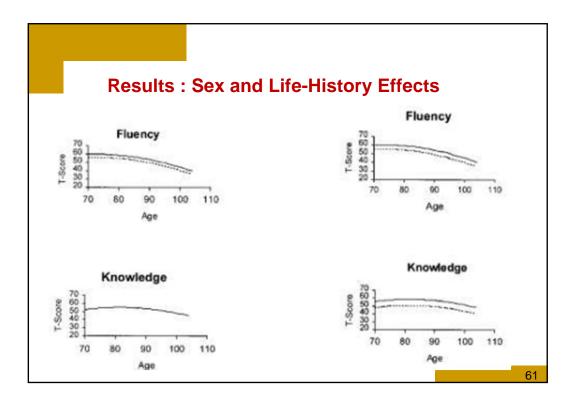
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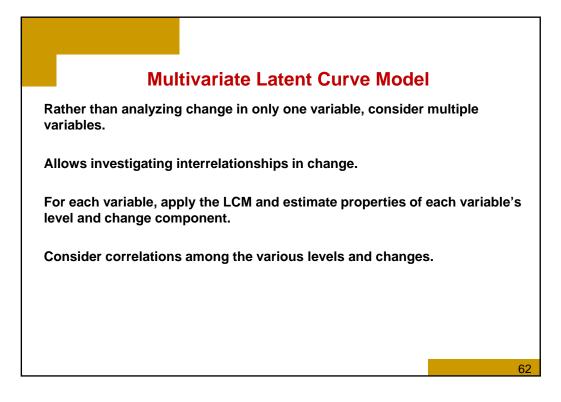












Stoolmiller (1994)

Stoolmiller, M. (1994). Antisocial behavior, delinquent peer association, and unsupervised wandering for boys : Growth and change from childhood to early adolescence. *Multivariate Behavioral Research, 29*, 263-288.

Latent growth curve analysis was used to study individual differences in initial status and growth rates of antisocial behavior, delinquent peer association, and unsupervised wandering during the transition from childhood to early adolescence for a sample of 206, primarily working-class, European-American boys. All three constructs showed significant individual differences in initial status at Grade 4 and growth rates from Grade 4 to Grade 8. Wandering and delinquent peer association showed positive mean trends. Linear growth curves adequately described growth for delinquent peer association and antisocial behavior. Growth on wandering was linear up to Grade 7 and then showed positive acceleration from Grade 7 to Grade 8. All three constructs were highly related at the initial assessment point. Individual differences in growth rates were highly correlated on all three constructs. The findings were discussed in terms of the trait-confluence model for peer influence on antisocial behavior.

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Descriptive Statistics of the Data

Table 1

Univariate Descriptives Statistics for Constructs

						Cons	truct						
	D	elinque	ent Pee	ers		Antis	ocial			Wan	dering		
		Gra	ade		_	Grade				Grade			
	4	6	7	8	4	6	7	8	4	6	7	8	
Mean	.33	.37	.40	.42	.37	.40	.40	.39	.29	.35	.40	.50	
Variance	.11	.13	.14	.16	.06	.07	.07	.07	.04	.07	.07	.11	
Skewness	1.23	.89	.87	.82	.81	.74	.90	.94	.76	.92	.94	1.01	
Kurtosis	1.00	.02	05	45	.35	.22	.62	.57	.96	.63	1.30	.82	
N	204	203	203	202	203	200	203	201	204	203	202	203	

Descriptive Statistics of the Data

Table 2 Bivariate Correlations for Constructs

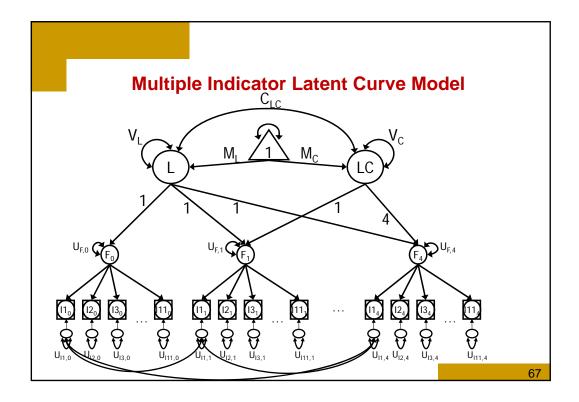
						Cons	truct					
	D	elinque	ent Pee	rs		Antis	social			Wan	dering	
		Gr	ade		_	Gr	ade		_	G	ade	
	4	6	7	8	4	6	7	8	4	6	7	8
Delinquent	1.00											
Peers	.49	1.00										
	.50	.60	1.00									
	.46	.61	.68	1.00								
Antisocial	.59	.52	.47	.45	1.00							
	.45	.64	.55	.58	.64	1.00						
	.47	.58	.65	.60	.64	.75	1.00					
	.42	.50	.54	.64	.59	.69	.75	1.00				
Wandering	.40	.36	.26	.26	.46	.41	.32	.27	1.00			
	.34	.44	.37	.40	.43	.54	.45	.44	.41	1.00		
	.34	.40	.45	.45	.43	.44	.45	.38	.37	.52	1.00	
	.28	.34	.44	.40	.36	.40	.43	.41	.31	.51	.54	1.00
		10	A0,00	1.0.0.0	_	1000	1.585	22.0				

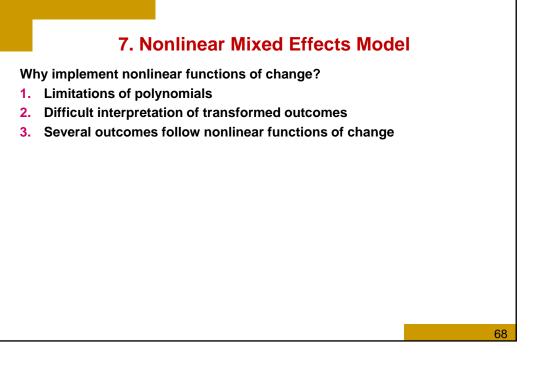
Results: Interrelationships of levels and changes

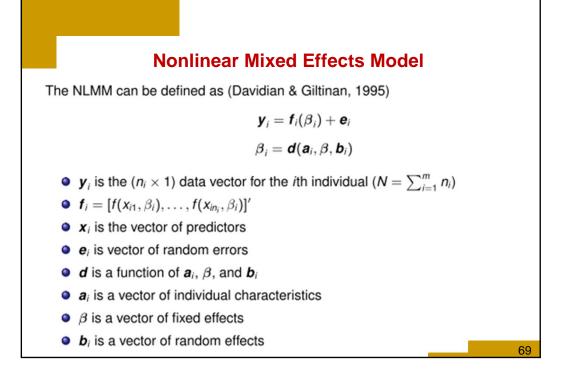
Multivariate Growth Model of Antisocial, Delinquent Peers and Wandering

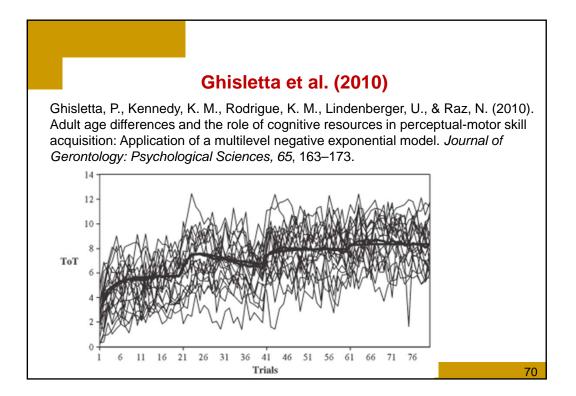
Correlati	ons Among	Compone	ents of Gro	owth		
Delinquent Peers intercept	1.00					
Antisocial intercept	.84	1.00				
Wandering intercept	.78	.83	1.00			
Delinquent Peers slope	.00	.09	18	1.00		
Antisocial slope	09	.00	36	.73	1.00	
Wandering slope/shape	.08	.14	.00	.63	.55	1.00
	Time-Specif	ic Correla	ations			
Antisocial 4th, Delinquent Peers	4th	24				
Antisocial 6th, Delinquent Peers	6th	27				
Antisocial 7th, Delinquent Peers	7th	28				
Antisocial 8th, Delinquent Peers	8th	22				
$\chi^2(58) = 44.2$	2 n = 900	RRN =	060 CF	/ = 1 000		

Table 4









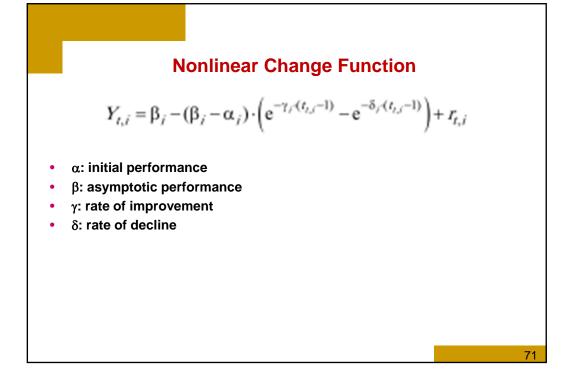


	Table 1. Para	meter Estimates a	and Standard Err	Res		Control for Age and	Cognitive Resou	rces		
		Fixed	effects		Random effects					
Block	α	γ	δ	β	α	γ	δ	β		
	2.855 (0.225) 4.430 (0.489) 7.448 (0.258) 8.282 (0.233)	0.336 (0.029) 0.525 (0.137) 0.297 (0.046) 0.228 (0.042)	0.100 (0.025)	5.759 (0.230) 6.461 (0.248) 7.946 (0.242) 8.362 (0.233)	2.863 (0.489) 10.222 (9.903) 4.169 (0.679) 3.473 (0.574)	0.070 (0.019) 0.031 (0.454) 1.269 (0.816) 48.065 (162.757)	0.034 (0.038) —	3.806 (0.55) 3.638 (0.846 4.125 (0.60) 3.992 (0.61)		
re preser orameter	8.282 (0.233) The fit indices of inted with point esti- rs of the negative	0.228 (0.042) this model were χ ² imates and, in parer exponential function	n. Italicized numb	8.362 (0.233) 2) = 5,630.108, RM rors. α (initial perfo ers refer to statistic	3.473 (0.574) SEA = .088 (90% Cl ormance), γ (acquisit	48.065 (162.757) = [.085092]), SRMB ion rate), δ (decline rat parameter estimates. R	e), and β (final perf	3.992 (0.6 .828. Paramet ormance) are		

Results	
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Table 4. Correlations Among Learning Parameters in Analyses Without or With Predictors in Lower or Upper Diagonal, Respectively

	Block 1			Block 2				Block 3			Block 4		
	α_1	71	βι	α.2	γ2	δ2	β2	α3	γ3	β3	α.4	γ4	β4
(e.)	-	196	.747º	.475	.082	.030	.839 ^a	.646 ^a	041	.642ª	.651º	122	.619 ^a
71	119	_	306°	670 ^b	.721 ^b	.808	169	166	153	169	172	.353b	135
β	.801ª	247 ⁿ	-	.457	032	122	.980°	.837ª	083	.830ª	.793*	130	.700ª
12	.576	555	.566"	_	.186	881	.517	.391	375	.394	.343	159	.217
12	.393	-2.361	.084	1.419	_	.227	.318	147	120	.065	062	102	134
52	.069	.691 ^b	049	714	-1.273	_	221	.141	.356	029	.185	.247	.133
32	.834°	074	.9399	.502	.026	.101	_	1.028 ^a	128	.943*	1.009°	078	.875 ^a
23	.707ª	129	.870°	.541	333	.135	.991ª	_	045	.866ª	.959	119	.782 ^a
13	.039	.170	024	324	125	.295	066	016	_	122	.027	.567 ^b	003
β3	.711*	141	.867ª	.513b	.192	.026	.926ª	.903ª	090	_	.892ª	125	.877ª
2.4	.706*	173	.825*	.511	.299	.075	.918 ^a	.945ª	.019	.902*	_	019	.882ª
4	060	.388 ^b	092	145	569	.288	010	092	.570 ^b	114	094		269
β.4	.6850	132	.7690	.394	167	.095	.864 ^a	.844 ^a	016	.9091	.9120	267	

Notes: α (initial performance), γ (acquisition rate), δ (decline rate), and β (final performance) are the parameters of the negative exponential function. Indices refer to the block.

*Statistically significant correlations at the p = .01 level. *Correlations that resulted statistically significant but that must be ignored because they are not defined (given that at least one of the two variables being correlated had no variance.

			Resu	lte		
	•		Nesu	11.5		
-	1. 0 D	An and the state of		Level A - 2 - d - D - P	den and the transfer of	
1;	able 3. Regression Weig	ghts (and SEs) of the Co		les and Age in the Predi	ction of the Learning C	omponents in
			Each Block			
	LS	CS	SJS	SR	WCST	Age
ε,	0.004 (0.018)	0.008 (0.014)	0.165 (0.123)	0.142ª (0.060)	0.006 (0.010)	-0.006 (0.012)
	0.003 (0.004)	0.000 (0.003)	0.000 (0.024)	0.007 (0.010)	-0.002 (0.002)	0.006ª (0.003
1	-0.004 (0.002)	0.002 (0.014)	0.157 (0.129)	0.1579 (0.057)	0.000 (0.010)	-0.024 (0.013
2	-0.089 (0.146)	0.088 (0.126)	0.007 (0.469)	0.005 (0.235)	-0.002 (0.061)	-0.065 (0.107
2	-0.029 (0.017)	0.028 (0.013) ^b	-0.017 (0.110)	-0.010 (0.047)	0.013 (0.009)	-0.018 (0.012
2	0.007 (0.006)	-0.006 (0.004)	0.006 (0.029)	0.012 (0.013)	-0.003 (0.003)	0.005 (0.005)
2	0.023 (0.021)	-0.023 (0.015)	0.158 (0.144)	0.167a (0.063)	-0.016 (0.012)	-0.013 (0.014)
13	0.014 (0.020)	-0.011 (0.015)	0.125 (0.138)	0.149º (0.059)	-0.010 (0.011)	-0.034ª (0.014
3	-0.008 (0.017)	0.003 (0.012)	-0.019 (0.113)	0.073 (0.052)	0.006 (0.009)	0.002 (0.011)
9	0.008 (0.020)	-0.005 (0.014)	0.204 (0.133)	0.116º (0.058)	-0.006 (0.011)	-0.028ª (0.013
	0.020 (0.018)	0.004 (0.014)	0.175 (0.122)	0.080 (0.055)	0.010 (0.010)	-0.042a (0.012
ta:	-0.037 (0.084)	-0.025 (0.060)	-0.194 (0.529)	0.392 (0.514)	-0.029 (0.056)	0.053 (0.080)
4.		-0.001 (0.014)	0.280 ^a (0.127)	0.074 (0.057)	-0.002 (0.010)	-0.033ª (0.013

*Statistically significant parameters at the p = .01 level. *An effect that resulted statistically significant but that must be ignored because it is not defined (given that in the previous model the dependent variable of this effect had no variance).

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8. Conclusions

- Lifespan developmental psychology has come a long way
- Many theoretical concepts of lifespan psychology have been operationalized with advanced methodologies and statistical models
- Many theoretical, methodological, and statistical advances have taken place, mutually feeding each other
- Nevertheless, lifespan developmental psychology has still a long way to go!!
- There is no need to be a sect! Any developmental field can become lifespan – actually, any field of research in psychology!

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• Go for it!

