

# حافظهٔ اجتماعی

## مغز، ذهن و جامعه

تاگاشی تسوکیورا، ساتوشی اومدا (ویراستار)

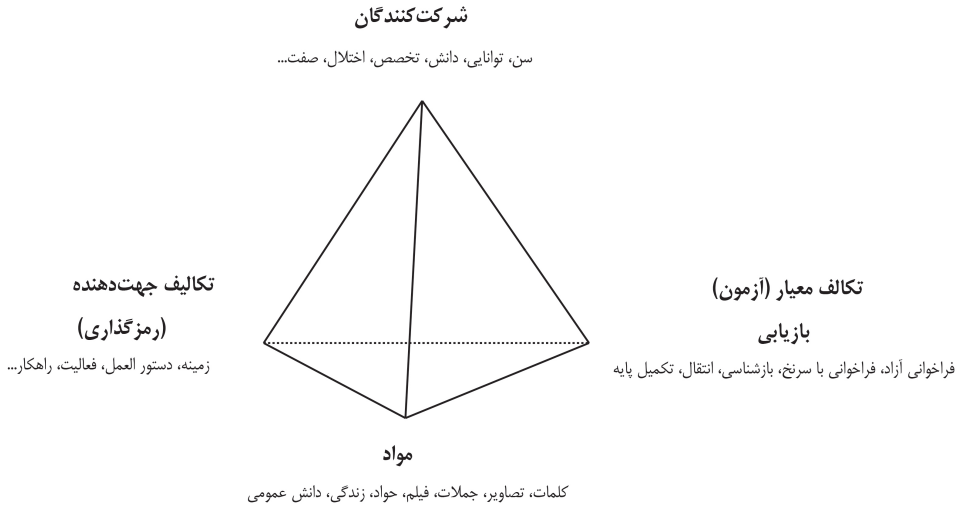
دکتر سوسن علیزاده فرد

مقدمه دکتر حسین زارع

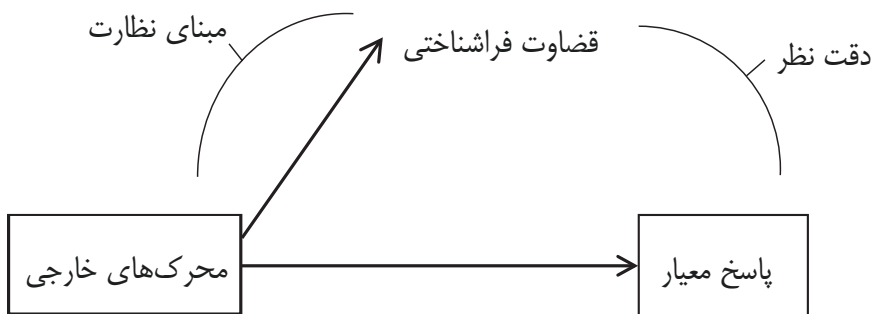


مؤسسه  
انتشارات  
بعثت

## منابع فصل اول



**شکل ۱،۱. نسخه اصلاح شده مدل چندوجهی جنکینز برای آزمایش های حافظه  
(جنکینز و همکاران؛ و رودیگر، ۲۰۰۸)**



**شکل ۲،۱. طرح بازنمایی تمایز بین تأثیر یک متغیر مستقل بر بزرگنمایی قضاوت نظارتی فراشناختی (مبتنی بر نظارت) در مقابل اندازه ارتباط بین قضاوت و عملکرد معیار (دقت قضاوت) (نلسون، ۱۹۹۶)**

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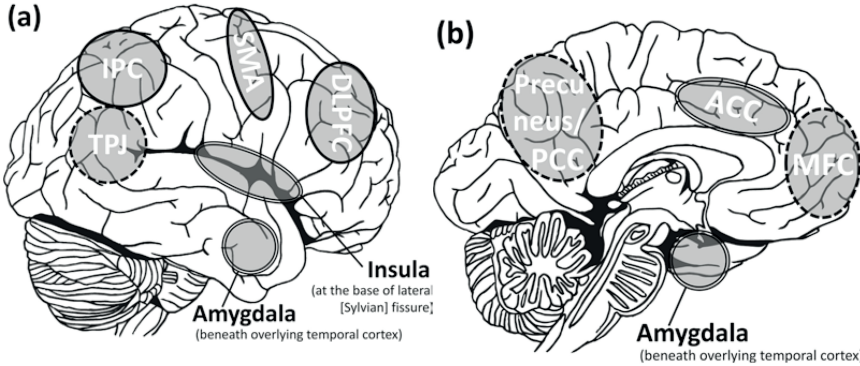
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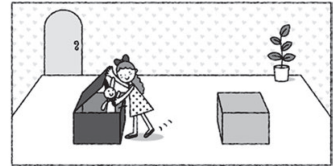
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## منابع فصل سوم



شکل ۱، ۳. الف) سطح جانبی و ب) سطح داخلی مغز. بیضی‌ها با خط ممتد مناطق اصلی تشکیل‌دهنده شبکه پیشانی-اهیانهای (FPN) را نشان می‌دهند؛ بیضی‌ها با خط چین مناطقی را نشان می‌دهند که برای شبکه حالت پیش فرض (DMN) و شبکه شناخت اجتماعی، مشترکند؛ بیضی‌ها با خط دوتایی مناطق اصلی تشکیل‌دهنده شبکه برجسته را نشان می‌دهند که در جابه‌جایی بین FPN و DMN نقش دارد.

سالی اسباب‌بازی خود را در جعبه قرمزی گذاشته و برای بازی بیرون می‌رود.



در غیاب او، دختر دیگری به نام آن، اسباب‌بازی را از جعبه قرمز به جعبه سبز جابه‌جا می‌کند و سپس برای بازی بیرون می‌رود.



سالی بازمی‌گردد و اسباب‌بازی‌اش را می‌خواهد. فکر می‌کنید اولین جایی که به دنبال آن خواهد گشت کجاست؟



شکل ۲، ۳. تصویری از تکلیف باورهای نادرست سالی - آن<sup>۱</sup> که برای ارزیابی نظریه ذهن کودکان استفاده می‌شود (تصاویر از مای‌هارا ۲۰۱۴، بازسازی شده از انتشارات دانشگاه توکیو)

1. Sally-Anne false-belief task

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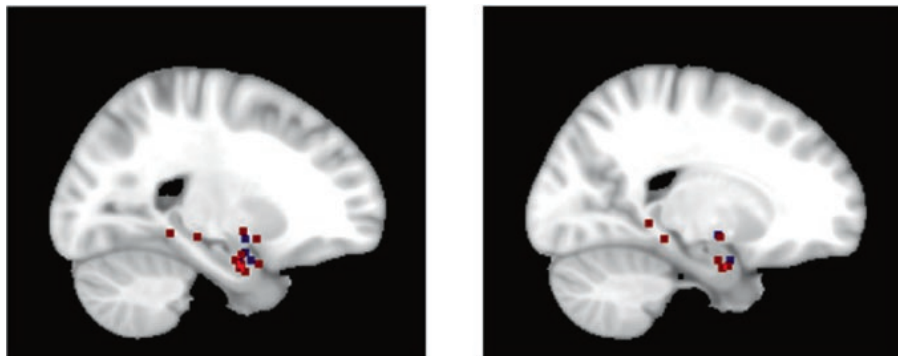
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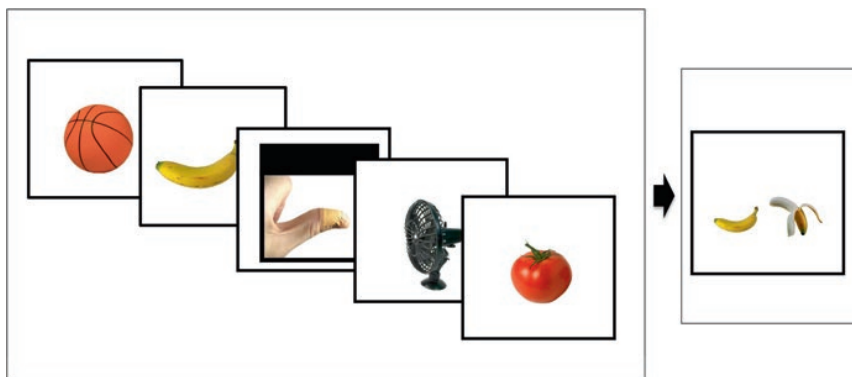
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## منابع فصل چهارم



شکل ۱,۴. همپوشانی در فعالیت لوب گیجگاهی میانی (الف) چپ و (ب) راست، در طول رمزگذاری (قرمز) و بازیابی (آبی) محرک‌های هیجانی در مقایسه با محرک‌های خنثی (مطالعاتی که برای ایجاد این شکل استفاده شده، در منابع با علامت ستاره \* مشخص شده‌اند).



شکل ۲,۴. تصویری نمادین از یک کوشش در مرحله رمزگذاری و آزمون حافظه؛ پژوهش ساکاکي و همکاران (۲۰۱۴)

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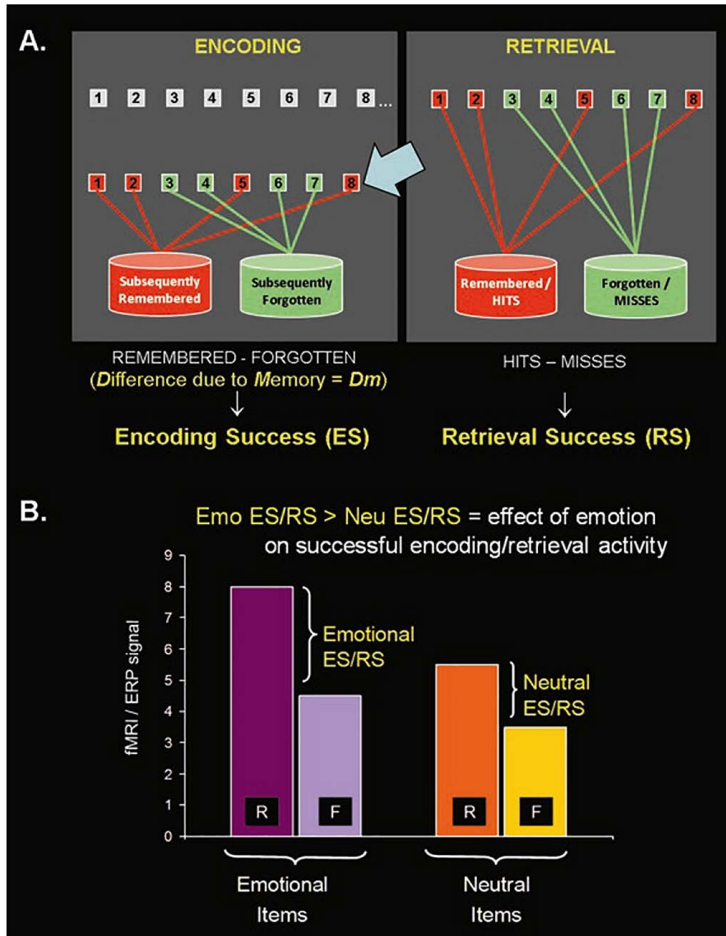
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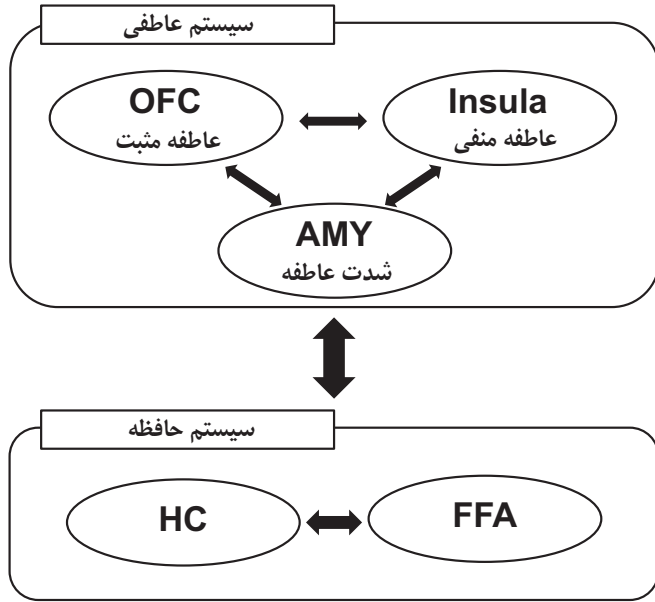
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منابع فصل پنجم

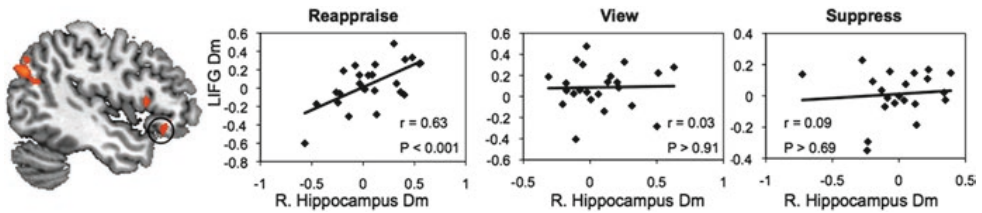


شکل ۵.۱. شیوه حافظه متعاقب (SMP) - سنجش تأثیر هیجان‌ها بر فعالیت موفقیت رمزگذاری (ES) و موفقیت بازیابی (RS). (الف) روند کلی دخیل در SMP. (ب) نمودار مقایسه‌هایی که شناسایی مناطق مغزی حساس به ES و RS هیجانی را امکان‌پذیر می‌کند. R مواردی که بعداً به خاطر آورده می‌شوند، F موارد فراموش شده بعدی، fMRI، تصویربرداری تشدید مغناطیسی عملکردی، ERP، پتانسیل وابسته به رویداد.

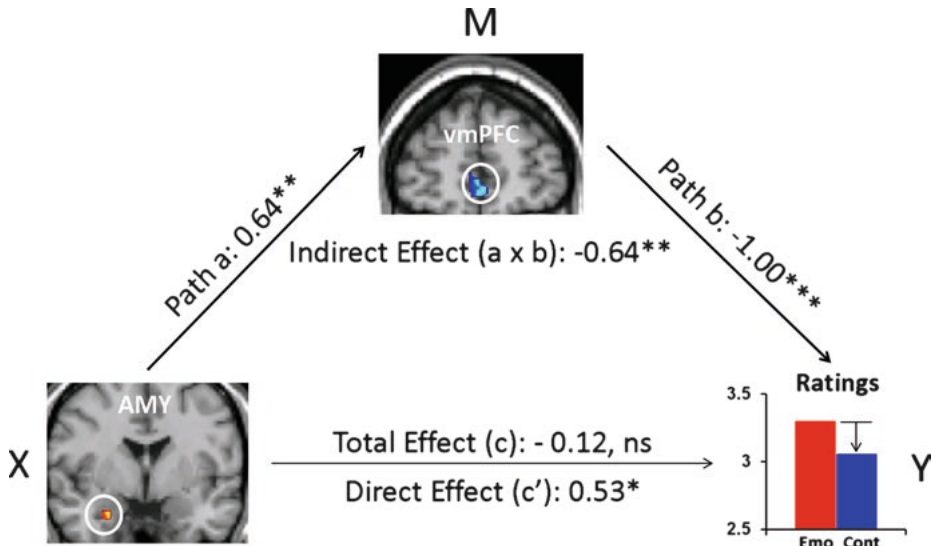




شکل ۲،۵. یک مدل فرضی از سازوکارهای عصبی زمینه‌ساز تأثیر سیگنال‌های هیجانی مثبتی بر چهره بر حافظه چهره‌ها. آمیگدال - آمیگدال، OFC - قشر اوربیتوفرونتال، INS - قشر اینسولار، HIP - هیپوکامپ، FFA - ناحیه دوکی شکل چهره (بازتولید شده و با کسب اجازه از تسوکورا، ۲۰۱۲).

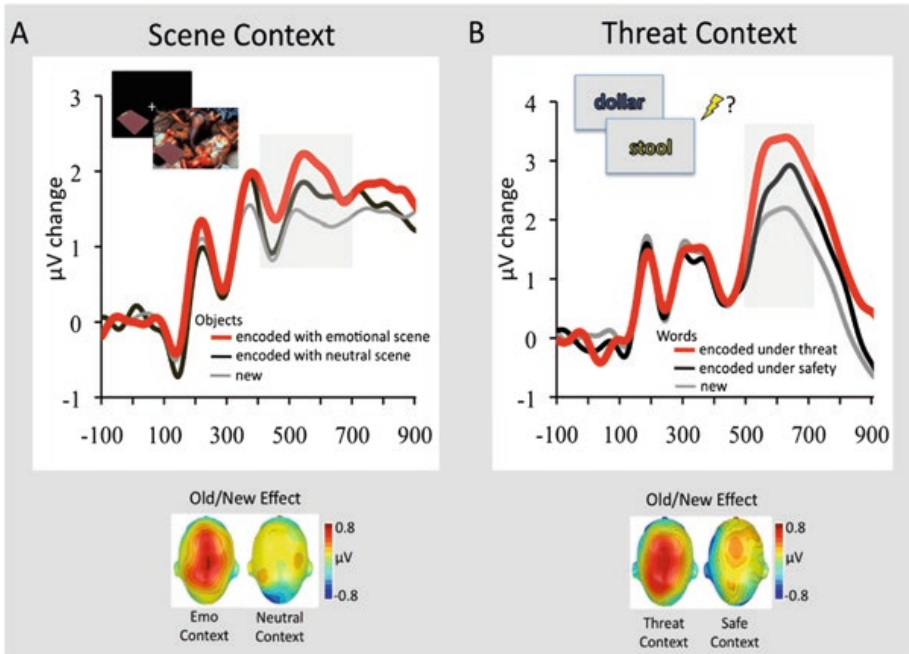


شکل ۳،۵. هم‌بستگی مثبت قوی‌تر بین شکنج پیشانی تحتانی چپ (IFG) و هیپوکامپ (هیپوکامپ) در زمان استفاده از ارزیابی مجدد، در مقایسه با مشاهده غیرفعال و سرکوب، ارزیابی مجدد با افزایش هم‌بستگی IFG-هیپوکامپ برای فعالیت وابسته به حافظه (اثر Dm) به‌عنوان تابعی از سازوکارهای تنظیم هیجان (ER) مورد استفاده در طول رمزگذاری همراه بود. Dm - تفاوت ناشی از حافظه (بازتولید و اجازه از هایس و همکاران، ۲۰۱۰)



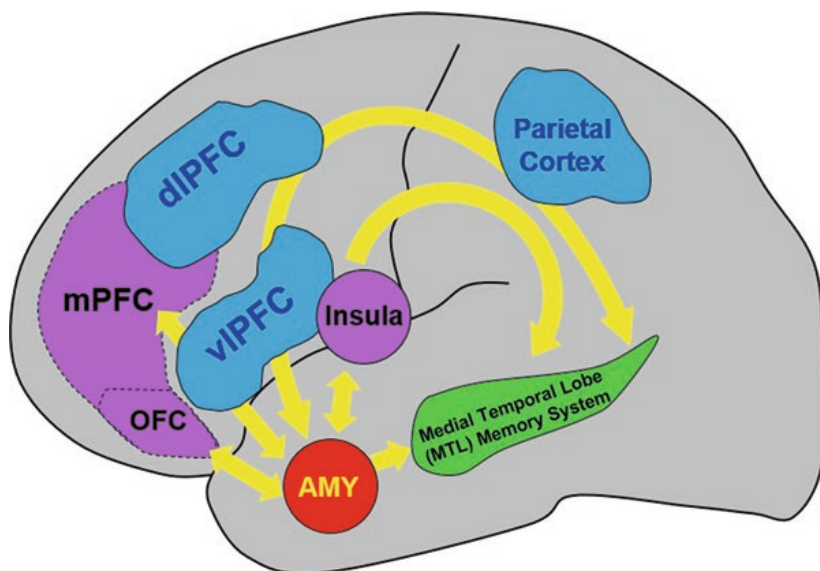
شکل ۴،۵. PFC شکمی میانی زمانی که تمرکز از جنبه‌های هیجانی یادآوری‌های زندگی‌نامه‌ای دور می‌شود، بین آمیگدال و نمرات هیجانی میانجی می‌شود. تجزیه و تحلیل میانجی زمانی که تمرکز بر جنبه‌های غیرهیجانی خاطرات شخصی بود یک اثر میانجی منفی معنادار ( $p=0.009$ ) مربوط به PFC شکمی میانی (vmPFC) بر رابطه بین آمیگدال و رتبه‌بندی هیجانی شناسایی کرد، و در زمان کنترل برای تأثیر vmPFC، اثر مستقیم مثبت معناداری ( $p=0.03$ ) بین آمیگدال و رتبه‌بندی هیجانی نشان داد (مسیر c، کنترل X به Y برای M). برای هر مسیر ضرایب استاندارد شده و معناداری که با ستاره نشان داده شده، گزارش شده است.

ns؛ \* $p<0.05$ ؛ \*\* $p<0.01$ ؛ \*\*\* $p<0.001$



شکل ۵. اثر ERP وابسته به بازیابی (اثر قدیم/ جدید) مربوط به محرک‌هایی که قبلاً با زمینه‌های هیجانی و خنثی مرتبط بودند. (الف) شکل موج‌های ERP متوسط بزرگ در یک خوشهٔ حسی مرکزی- آهیانه‌ای برای اشیائی که به‌درستی بازشناسی شده‌اند، که در زمینهٔ یک صحنه پس‌زمینهٔ هیجانی (خط قرمز)، یا صحنه پس‌زمینهٔ خنثی (خط سیاه) و اشیای جدیدی که به‌درستی طبقه‌بندی شده‌اند (خط خاکستری) رمزگذاری شده‌اند. توالی رمزگذاری این آزمایش در قسمت بالا سمت چپ نشان داده شده است. در این آزمایش ۱۴۴ شیء در ۱۴۴ صحنهٔ پس‌زمینه (۴۸ صحنهٔ دلپذیر، ۴۸ صحنهٔ خنثی و ۴۸ صحنهٔ ناخوشایند) ارائه شدند. برای جلوگیری از رقابت مستقیم بین پس‌زمینه‌های هیجانی و اشیای خنثی، ابتدا اشیای نمایش داده شده و به‌دنبال آن صحنهٔ پس‌زمینه ارائه شد. برای تسهیل پیوند حافظه، از شرکت‌کنندگان خواسته شد تصور کنند شیء بخشی از صحنه است. نمودار پایین توپوگرافی‌های پوست سر مربوط به اختلاف ERP (قدیمی منهای جدید؛ ۴۰۰- ۷۰۰ میلی‌ثانیه) را نشان می‌دهد که به تفکیک برای اشیائی که با صحنه‌های هیجانی یا خنثی جفت شده‌اند به کار می‌رود.

(ب) شکل موج‌های ERP متوسط بزرگ در یک خوشهٔ حسی مرکزی- آهیانه‌ای برای کلماتی که به‌درستی به خاطر آورده می‌شوند که با رنگ فونتی رمزگذاری شده‌اند (توالی رمزگذاری بالا سمت چپ را ببینید) که نشانهٔ تهدید شوک (خط قرمز) یا ایمنی (خط سیاه) است و کلمات جدیدی که به‌درستی طبقه‌بندی شده است (خط خاکستری). نمودار پایین توپوگرافی‌های پوست سر مربوط به اختلاف ERP (قدیمی منهای جدید؛ ۵۰۰- ۷۰۰ میلی‌ثانیه) را نشان می‌دهد که به تفکیک برای اشیائی که تحت تهدید یا ایمنی رمزگذاری شده‌اند، به کار می‌رود.



شکل ۶،۵. نمودار خلاصه‌ای از مناطق‌های عصبی اثر هیجان‌ها بر تقویت حافظه که از مطالعات تصویربرداری مغز حاصل شده است. دو سازوکار اصلی دخیل در تأثیر هیجان‌ها بر تقویت حافظه شناسایی شده است: یکی بر اساس لوب گیجگاهی میانی (MTL)؛ (سیستم حافظه MTL و آمیگدال = هیپوکامپ و قشر پاراهیبوکامپ مرتبط) و دیگری نیز شامل مناطق غیر MTL مانند قشر پیش‌پیشانی میانی و پشتی/شکمی جانبی (به ترتیب mPFC و DIPFC/vIPFC) در میان دیگران (مانند قشر آهیانه). مناطق حافظه MTL و آمیگدال از طریق سازوکارهای عصبی-هورمونی مستقیم/خودکار تعامل می‌کنند که در اثر هیجان‌ها بر تقویت حافظه مشارکت دارند (سازوکار پایین به بالا)، درحالی‌که PFC بخشی از سازوکاری است که با ارتقای فرایندهای سازوکارک، معنایی، حافظه‌کاری و توجه (سازوکار بالا به پایین)، مشارکت غیرمستقیم/با واسطه‌ای در خاطرات هیجانی دارد. علاوه بر این، بررسی حافظه هیجانی برای جنبه‌های اجتماعی، مشارکت خاص خوشایندی سایر مناطق مغزی را که در تقویت حافظه هیجانی در زمینه اجتماعی نقش دارند، شناسایی کرد- یعنی حافظه برای اطلاعات مرتبط اجتماعی، برای مواردی با مفاهیم مثبت شامل فعالیت در OFC میانی و MTL و تعاملات بین آن‌ها است و تعاملات بین اینسولا و MTL برای مواردی با مفاهیم منفی است. در نهایت، بررسی تأثیر تنظیم هیجان بر حافظه هیجانی، روابط دو سویه بین مناطق PFC و MTL مربوط به سازوکارهای خاص تنظیم هیجانی را شناسایی کرد، که شامل PFC جانبی/میانی در مدولاسیون بالا به پایین، سازوکارهای آمیگدال MTL - در رمزگذاری و بازیابی حافظه هیجانی و سیگنال‌دهی آمیگدال به PFC میانی است که نیاز به اعمال کنترل بر محرک‌های هیجانی دارد، که به کاهش کلی تجربه هیجانی در طول بازیابی زندگینامه‌ای منجر می‌شود (برگرفته و کسب اجازه از دنوکووا و همکاران، ۲۰۱۵؛ دولکاس و همکاران، ۲۰۱۱، ۲۰۱۲).

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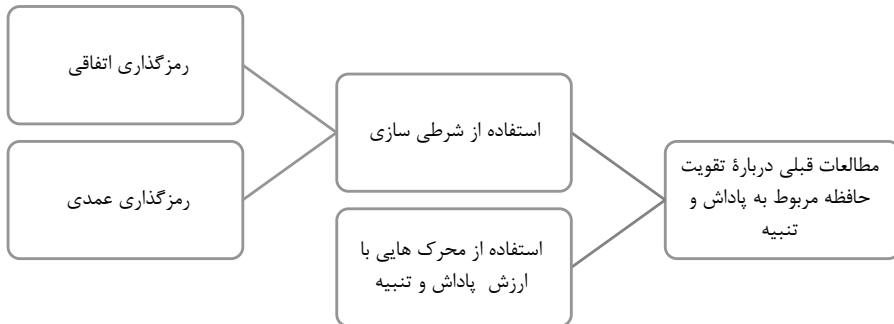


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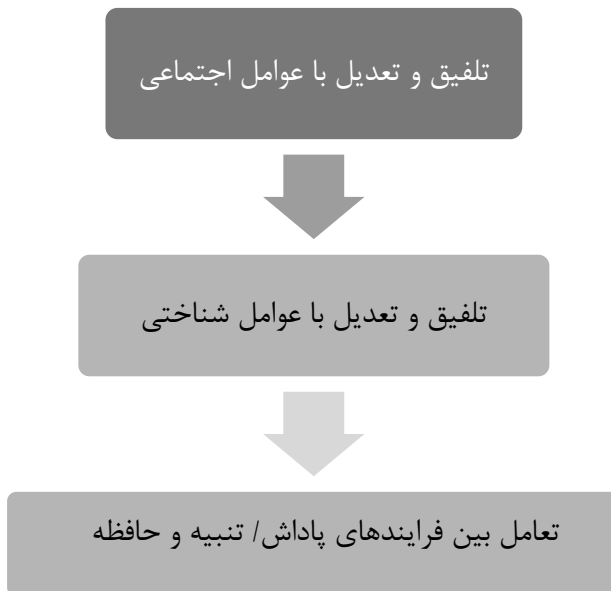
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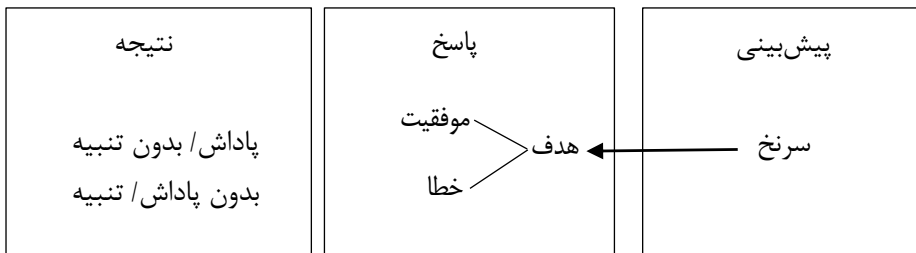
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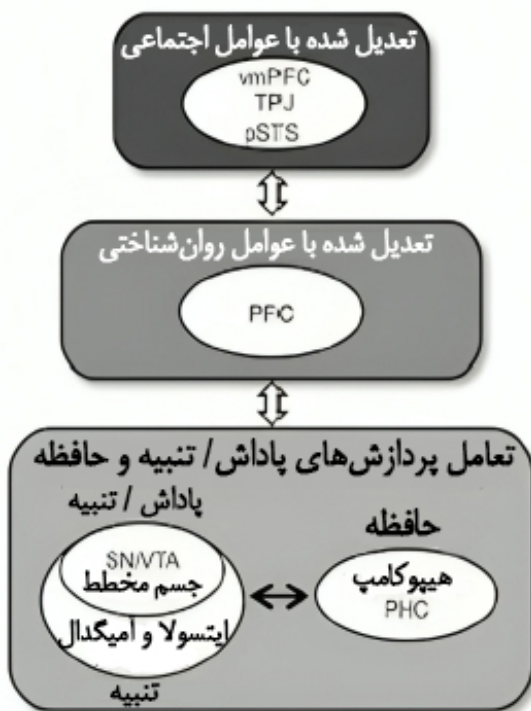
شکل ۱,۶. طبقه‌بندی مطالعات قبلی درباره تقویت حافظه وابسته به پاداش یا تنبیه



شکل ۲,۶. ساختار سلسله‌مراتبی سه لایه مربوط به تقویت حافظه با پاداش یا تنبیه در شرطی‌سازی



شکل ۳,۶. روند کلی تکلیف تأخیر مشوق پولی (MID)



شکل ۴,۶. مدل فرضی سازوکارهای عصبی زیربنای ساختار سلسله مراتبی سه لایه تقویت حافظه توسط پاداش/تنبیه در شرطی سازی

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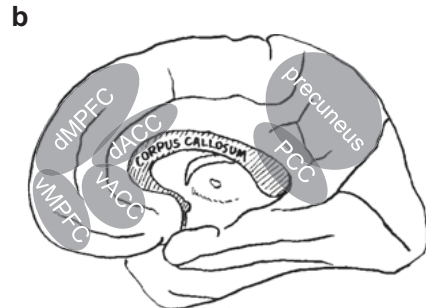
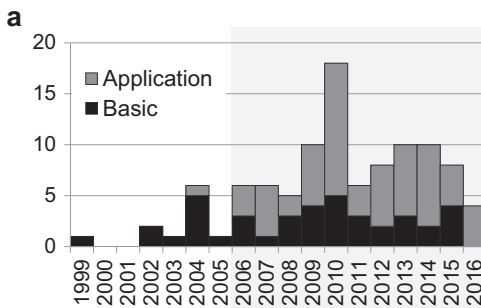
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## منابع فصل هفتم

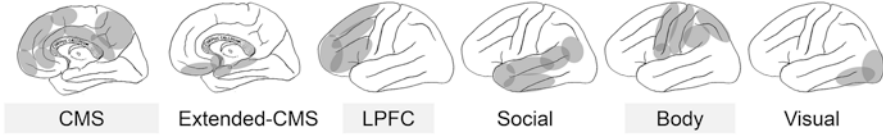


شکل ۱،۷. وظیفه ارزیابی صفات خود (STE). (الف) از آزمودنی‌ها خواسته می‌شود قضاوت کنند که آیا صفت ارائه‌شده توصیفی از خودشان است یا نه، یا درجه توصیفی بودن آن را ارزیابی کنند. با تغییرات کمی در دستورالعمل‌های تکلیف، می‌توان تکالیف کنترلی مختلفی ایجاد نمود، از جمله (ب) تکلیف انتساب اجتماعی در مورد فردی آشنا، و انواع مختلفی از تکالیف پایه، مانند (ج) وظیفه معنایی (یا ارزشی) و (د) وظیفه ساختاری (یا ادراکی).



شکل ۲،۷. مطالعاتی که از تکلیف STE و ساختارهای خط میانی قشر مغز (CMS) استفاده می‌کنند (a) تعداد مقالات منتشرشده تا اواسط سال ۲۰۱۶ که فعالیت CMS را در طول تکلیف STE گزارش کرده‌اند. و در مجموع، ۱۰۲ مطالعه (۴۰ مطالعه پایه و ۶۲ مطالعه کاربردی) شناسایی شد (برای اطلاع از جزئیات بیشتر، به متن مربوط به روش مورد استفاده برای فراتحلیل مراجعه کنید). افزایش در تعداد مطالعات با دوره‌ای هم‌زمان است که اصل CMS تأثیرگذار بود (پس‌زمینه سایه‌دار؛ برای جزئیات بیشتر متن را ببینید). (b) تشریح آناتومیکی متداول CMS. vMPFC قشر پیش‌پیشانی شکمی میانی، dMPFC قشر پیش‌پیشانی خلفی میانی، vACC بخش شکمی قشر سینگولیت قدامی، dACC بخش پشتی قشر سینگولیت قدامی، PCC قشر سینگولیت خلفی.

جدول ۱,۷ انواع فعالیت در ارزیابی صفت خود، تکلیف STE



Paper	label	vMIPFC	dMPFC	vACC	vACC	MCC	SMA	dACC	PCC	precuneus	alms	amygdala	OFC	MTL	IFG	MFG	SFG	IFG	TP	VLTC	dLTC	AG	TPJ	preCG	postCG	SMG	IFG/SP_L	plns	mOC	IOC	pVTC	
Pfeifer07jocn19_1323	Yng+	•	•	•					•																							
Pfeifer09ChildDev80_1016	Yng+	•	•	•																												
Bedford12BMCPsci12_106	Scz-	•	•	•																												
Bedford12BMCPsci12_106	Scz+	•	•	•																												
Holt11BioIPsci69_415	Scz+								•	•																						
McAdams14scan9_12	Ref+								•	•																						
Ray09ChildDev80_1232	Mem+	•	•	•	•	•					•					•																
Ray10scan5_318	Sif+	•	•	•																												
Ruby09NrbioAging30_1637	Age-	•	•	•							•																					
Schneider12DevCgNsci2_277	Scz-	•	•	•																												
Schneider12DevCgNsci2_277	Scz-	•	•	•																												
Schneider12DevCgNsci2_277	Scz-	•	•	•																												
Pfeifer09ChildDev80_1016	Yng+	•	•	•																			•									
Bedford12BMCPsci12_106	Scz-	•	•	•																												
Bedford12BMCPsci12_106	Scz-	•	•	•																												
Blackwood04PscMed34_591	Scz-					•																										
Blackwood04PscMed34_591	Scz+									•																						
D'Argembeau07jocn19_935	Ref+					•																										
Holt11BioIPsci69_415	Scz-	•	•	•																												
Lieberman04JPrsScPsc87_421	Sif-	•	•	•																												
Lieberman04JPrsScPsc87_421	Sif+	•	•	•																												
Lieberman04JPrsScPsc87_421	Sif-	•	•	•																												
Ma14scan9_73	Sif-				•																											
Ma14scan9_73	Sif+				•																											
Ma14scan9_73	Sif-				•																											
Macrae04cc14_647	Sif+	•	•	•																												
Macrae04cc14_647	Sif-					•																										
McAdams14scan9_12	Ann-										•																					
McAdams14scan9_12	Ann+						•																									
McAdams14scan9_12	Ann-						•																									
McAdams14scan9_12	Ref+										•																					
McAdams14scan9_12	Ref+										•																					
McAdams14scan9_12	Ref-						•																									
Moran06jocn18_1	Val+				•																											
Moran06jocn18_1	Sif+	•	•	•		•						•				•																
Moran09SocNsci4_197	Sif+	•	•	•																												
Ochsner05ni28_797	Ref-																															
Pfeifer07jocn19_1323	Yng-	•	•	•																												
Pfeifer09ChildDev80_1016	Yng+	•	•	•																												
Pfeifer09ChildDev80_1016	Fem+	•	•	•																												
Pfeifer13jns33_7415	Yng-	•	•	•																												
Pfeifer13JAutismDevDisord43_272	Asd-	•	•	•																												
Ruby09NrbioAging30_1637	Alz-	•	•	•																												
Sarsam13POne8_e78844	Dep+	•	•	•																												
Tan15PlosOne10_e0138737	Scz-																															
Veroude14scan9_513	Fem-																															
Yang12nsy50_1267	Adp+																															
Hoefler15POne10_e136027	Val+																															
Schmitz06nsy44_762	Tbi+																															
Yoshimura14scan9_487	Dep-	•	•	•																												
McAdams14scan9_12	Ref+																															
D'Argembeau12cc22_659	Sif+	•	•	•																												
Ma14scan9_1360	Sif-	•	•	•																												
Pfeifer13jns33_7415	Yng+	•	•	•																												
Zhu12Hypo22_1540	Mem+																															
Hughes13jocn25_613	Adp+	•	•	•																												
Hoefler15POne10_e136027	Adp-	•	•	•																												
Lieberman04JPrsScPsc87_421	Sif-	•	•	•																												
Ma14cc24_2421	Val-																															
Colton13FrmHumNsci7_537	Age+																															
Ochsner05ni28_797	Ref+																															
Lieberman04JPrsScPsc87_421	Sif+	•	•	•																												
Pauly14scan9_1779	Scz-																															
Lieberman04JPrsScPsc87_421	Sif+	•	•	•																												



Paper	label	vAMPFC	dMPFC	vACC	dACC	SMA	MCC	PCC	precuneus	alms	amygdala	OFC	MTL	IFG	MFG	SFG	aPFC	TP	VLTc	dLTC	TPJ	AG	preCG	postCG	SMG	IFS/IFL	plns	mOC	IOC	pVTC
Goldin12FrmHumNsci6_295	Sad-	●							●					●	●										●					
Lieberman04JPrsScPsc087_421	Sif-													●	●															
McAdams14scan9_12	Ann+													●	●															
Pfeifer13AutismDevDisord43_272	Asd+								●					●	●						●			●						
Schmitz06nsy44_762	Tbi-													●	●															
Yang16SRep6_20274	Adp+													●	●		●													
Bedford12BMCPsci12_106	Scz+		●											●	●															
Benoit10ni50_1340	Sif+	●												●	●															
Kim16PlosOne11_e0149554	Adp-		●											●	●															
Lemogne09scan4_305	Dep+		●											●	●															
Ma14scan9_73	Sif+								●					●	●															
Ma14scan9_1360	Sif-		●											●	●															
McAdams14scan9_12	Ref-				●									●	●															
Chiao09hbm30_2813	Ctx+		●					●	●					●	●					●	●							●		
Ma14scan9_1360	Sif+													●	●											●				
Ma14scan9_1360	Sif+								●					●	●										●					
D'Argembeau08scan3_244	Now+		●					●						●	●															
Goldin09JcgnPsc0Thr23_242	Sad+		●											●	●															
Goldin12FrmHumNsci6_295	Sad-	●	●								●	●		●	●															
Gutches07SocNsci2_117	Age+		●											●	●															
Lieberman04JPrsScPsc087_421	Sif-				●									●	●															
Lieberman04JPrsScPsc087_421	Sif+				●									●	●															
Ma14cc24_2421	Val-		●		●					●				●	●															
Ma14cc24_2421	Val-		●		●					●				●	●															
Macrae04cc14_647	Mem+	●	●										●	●																
Rameson10ni50_701	Sif+	●	●	●			●	●	●				●	●																
Ochsner05ni28_797	Ref+													●	●					●	●						●			
Fossati03AmJPsych160_1938	Val+													●	●					●	●	●					●			
Pauly14scan9_1779	Scz-													●	●					●	●									
Yang16SRep6_20274	Adp+													●	●					●	●									
Zhu12Hippo22_1540	Mem+		●							●				●	●					●	●									
D'Argembeau12cc22_659	Fam+	●	●							●				●	●					●	●					●				
Lieberman04JPrsScPsc087_421	Sif+		●				●	●	●					●	●					●	●					●				
Ma14scan9_73	Sif+								●					●	●															
Pfeifer09ChildDev80_1016	Yng-							●						●	●															
Veroude14scan9_513	Ref+							●						●	●															
Veroude14scan9_513	Fem-							●						●	●															
Murphy10SczRes116_252	Scz-							●						●	●															
Zhu12Hippo22_1540	Mem+		●							●				●	●					●	●									
Chiao09hbm30_2813	Ctx-													●	●															
Meffert13FrmHumNsci7_46	Val-													●	●									●	●		●			
McAdams14scan9_12	Ref-		●											●	●															
Meffert13FrmHumNsci7_46	Val+							●						●	●										●	●		●		
Ochsner05ni28_797	Ref-								●					●	●															
Bedford12BMCPsci12_106	Scz-													●	●															
Chen15CultBrain3_39	Adp+	●					●	●						●	●					●	●				●	●				
Goldin12FrmHumNsci6_295	Sad-													●	●															
Kim16PlosOne11_e0149554	Adp+							●						●	●															
Ma14scan9_1360	Sif+													●	●											●				
Pauly14scan9_1779	Scz+													●	●									●						
Ruby09NrbioAging30_1637	Alz+													●	●															
Kircher02npla40_683	Sif+							●	●					●	●															
Moran06jocn18_1	Sif+	●												●	●															
Gutches10nsy48_211	Age+		●		●				●					●	●					●	●									
Bedford12BMCPsci12_106	Scz+			●										●	●															
Goldin09JcgnPsc0Thr23_242	Sad-								●					●	●															
Gutches07SocNsci2_117	Age+					●								●	●															
Kim16PlosOne11_e0149554	Val+							●						●	●															
D'Argembeau12cc22_659	Sif+	●	●						●					●	●															
Chen15CultBrain3_39	Adp+													●	●															
Colton13FrmHumNsci7_537	Age+							●	●					●	●															
Chiao09hbm30_2813	Fam+			●				●					●	●						●	●									
Bedford12BMCPsci12_106	Scz-													●	●															
Bradley16DevCogNsci19_87	Dep-													●	●															
Veroude14scan9_513	Ref-													●	●															
Zhu12Hippo22_1540	Mem+													●	●															
Colton13FrmHumNsci7_537	Age+							●	●					●	●															
Bradley16DevCogNsci19_87	Dep+							●	●					●	●															
Ma14cc24_2421	Val+							●	●					●	●															
Chiao10jocn22_1	Fam-				●		●							●	●															
Yang16SRep6_20274	Adp+													●	●															
Chiao09hbm30_2813	Fam+													●	●															
Chiao10jocn22_1	Fam+													●	●															
Goldin12FrmHumNsci6_295	Sad+		●	●										●	●															
Zhu12Hippo22_1540	Sif+		●											●	●															

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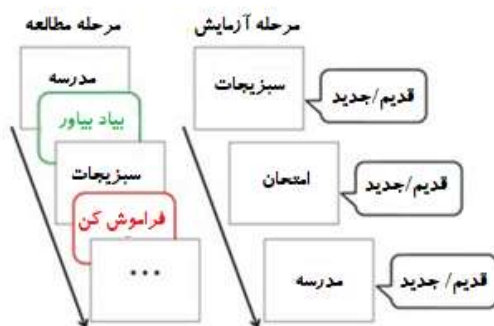
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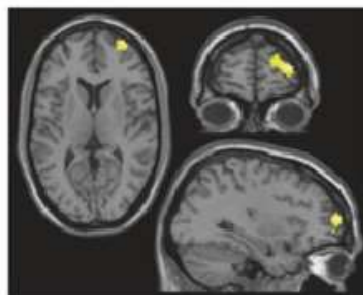
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## منابع فصل هشتم

الف) رویکرد فراموشی هدایت‌شده با روش موردی



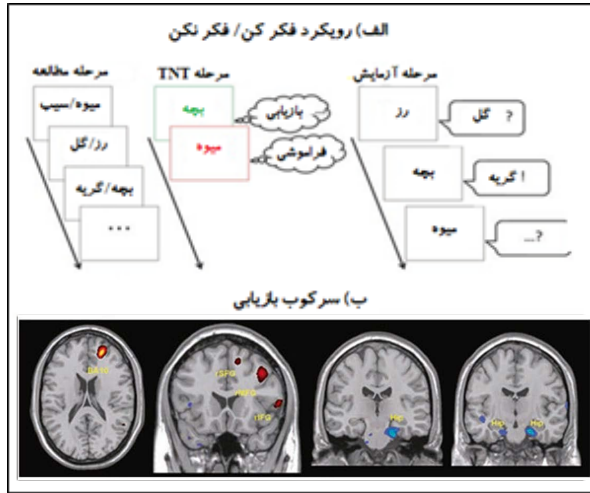
ب) فراموشی هدایت‌شده موفق



شکل ۸، ۱. نمونه‌ای از رویکردهای فراموشی هدایت‌شده تجربی و هم‌بستگی‌های عصبی زیربنایی

الف) رویکرد فراموشی هدایت‌شده با روش موردی. در این رویکرد که سرکوب ارادی در رمزگذاری را می‌سنجد، شرکت‌کنندگان موردها را یک‌به‌یک مطالعه می‌کنند، و هر مورد با یک دستورالعمل «فراموش کردن» یا «به خاطر سپردن» دنبال می‌شود. در ادامه حافظه شرکت‌کنندگان برای تمام موردها آزمایش می‌شوند.

ب) نقشه فعال‌سازی مربوط به یک مطالعه fMRI اخیر مربوط به فراموشی هدایت‌شده با روش موردی. نواحی نشان داده شده است که در رابطه با فراموشی عمدی در مقایسه با فراموشی اتفاقی به‌طور معناداری فعال‌ترند (موردهایی که باید فراموش شوند و با موفقیت فراموش می‌شوند در مقابل موردهایی که باید به خاطر سپرده شوند و تصادفاً فراموش شده‌اند). فراموشی هدایت‌شده موفق (در مقایسه با فراموشی اتفاقی) با افزایش فعال‌سازی در شکنج پیشانی میانی راست یعنی قشر پیش‌پیشانی خلفی جانبی (DLPFC) همراه است.



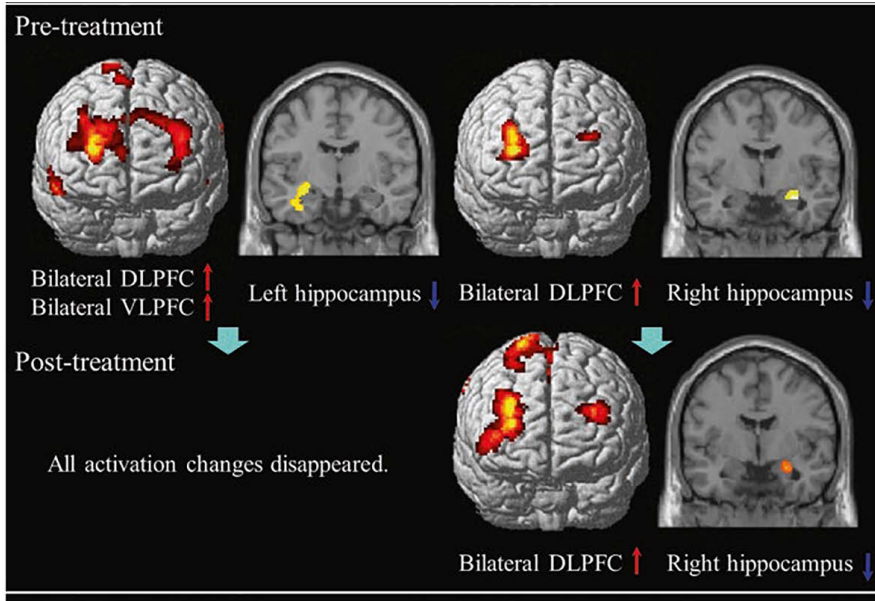
شکل ۲،۸. نمونه‌ای از رویکردهای سرکوب بازایی آزمایشی و هم‌بستگی‌های عصبی زیربنایی

الف) رویکرد فکرکن / فکر نکن (TNT). در این رویکرد، که سرکوب ارادی در بازایی را می‌سنجد، شرکت‌کنندگان در ابتدا جفت‌های نشانه-هدف را مطالعه می‌کنند. در ادامه شرکت‌کنندگان تحت مرحله TNT قرار می‌گیرند، که در آن نشانه‌های یادآوری یک‌به‌یک به آن‌ها ارائه شده و از آن‌ها خواسته می‌شود تا هدفی را بازایی (فکرکن) یا فراموش (فکر نکن) کنند که با رنگ‌های سبز یا قرمز نشان داده می‌شوند. حافظه شرکت‌کنندگان برای تمام جفت‌ها آزمایش می‌شوند.

ب) نقشه‌فعال‌سازی مربوط به یک مطالعه fMRI در مورد سرکوب بازایی با استفاده از رویکرد TNT (اصلاح‌شده با اجازه دوپ و همکاران، ۲۰۰۷). مناطقی که در آزمون‌های فکرکن در مقایسه با آزمون‌های فکرکن نمایش داده شدند که به‌طور معناداری فعال‌تر (قرمز) یا غیرفعال (آبی) بودند. سرکوب بازایی با افزایش فعال‌سازی در مناطق پیش‌پیشانی راست (شامل شکنج پیشانی فوقانی راست، میانی و تحتانی و ناحیه ۱۰ برودمن) و غیرفعال‌سازی در هیپوکامپ دوطرفه همراه بود.

جدول ۱،۸. دوره فراموشی و تغییرات آن با درمان در بیماران ۱ و ۲ با تشخیص فراموشی تجزیه‌ای

بیمار ۲	بیمار ۱	فراموشی پس‌گستر
یک دوره حدوداً ۳۵ ساله از زمان فارغ‌التحصیلی از دبیرستان تا زمان بروز مشکل	یک دوره ۴/۵ ساله از زمان نیم‌سال آخر دوران تحصیل در دانشگاه و ۴ سال بعد از آن به‌عنوان تاجر	دوره فراموشی پیش از درمان
↓	↓	درمان: مصاحبه تحت بیهوشی
هیچ خاطره‌ای بازایی نشد.	تمام خاطرات از ۴ سال اول دوره فراموشی تقریباً به‌طور کامل بهبود یافت. نیم‌سال آخر دوره فراموشی بهبود نیافت.	پس از درمان



شکل ۳,۸. تغییرات فعال‌سازی مربوط به فراموشی تجزیه‌ای از یک مطالعه fMRI مرتبط با تکلیف (اصلاح شده با اجازه از کیکوچی و همکاران، ۲۰۱۰).

تغییرات فعال‌سازی معنادار در پاسخ به محرک‌های «غیرقابل تشخیص» در مقابل محرک‌های «قابل تشخیص»، که به‌طور کامل با تغییرات فعال‌سازی معنادار در پاسخ به محرک‌های «غیرقابل تشخیص» در مقابل محرک‌های «کنترل» پوشانده شده‌اند، نشان داده شده است. پیش از درمان، بیماران ۱ و ۲ مبتلا به فراموشی تجزیه‌ای، الگوهای تغییر یافته‌ای از فعال‌سازی مغزی را نشان دادند که در آن مناطق پیش‌پیشانی فعال‌تر و مناطق هیپوکامپ غیرفعال‌تر بودند. پس از درمان، تمام تغییرات فعال‌سازی در بیمار ۱ که تقریباً تمام خاطرات از دست‌رفته خود را بازیابی کرد، ناپدید شدند. در مقابل در بیمار ۲ که خاطرات از دست‌رفته خود را بازیابی نکرد، الگوی تغییر یافته‌ی فعال‌سازی‌های مغزی تقریباً بدون تغییر باقی ماند. DLPFC قشر پیش‌پیشانی خلفی جانبی، VLPFC قشر پیش‌پیشانی شکمی جانبی.

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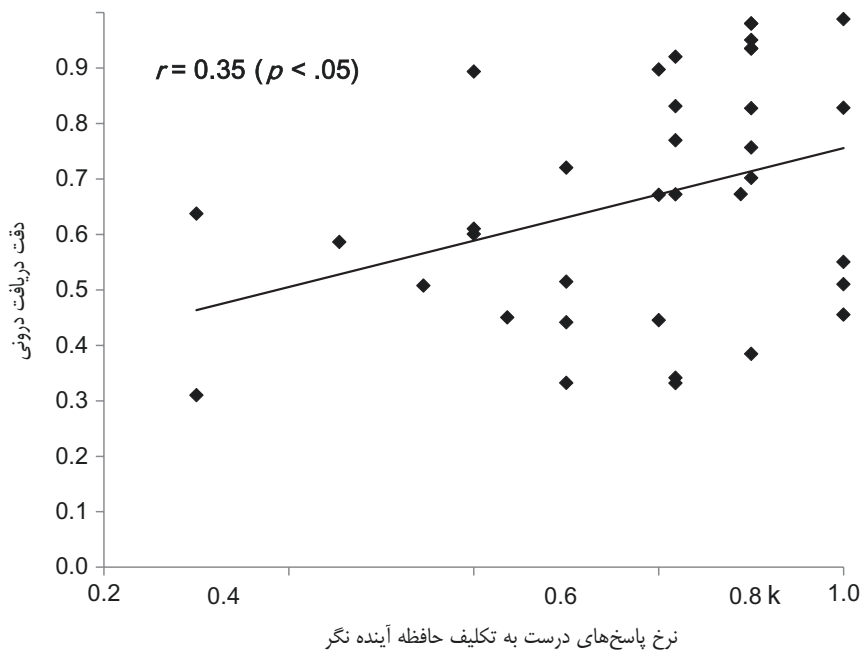


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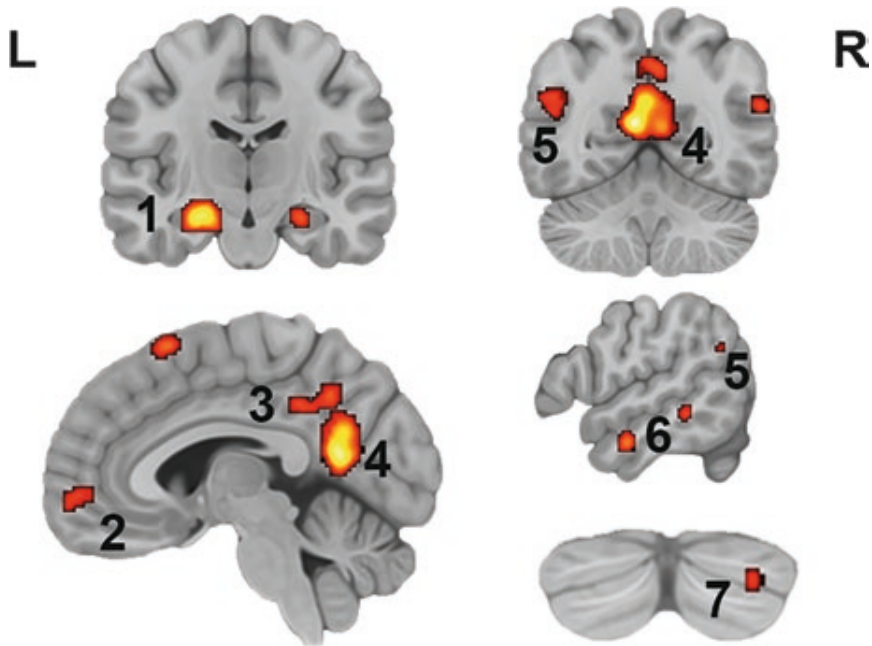


شکل ۹،۱. همبستگی مثبت بین عملکرد حافظه آینده‌نگر (PM) و دقت دریافت درونی شرکت‌کنندگانی که عملکرد PM بالاتری نشان دادند، می‌توانستند ضربان قلب خود را نیز دقیق‌تر احساس کنند (نویسندگان اومدا و همکاران (۲۰۱۶) اجازه استفاده از این شکل را بر اساس داده‌های خود دادند).

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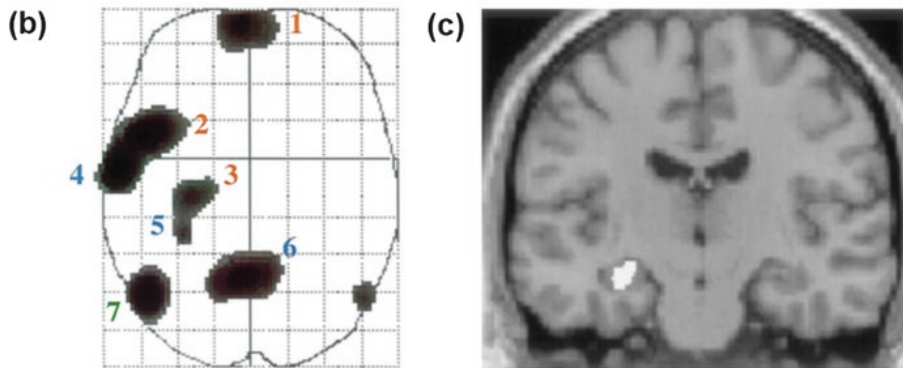


شکل ۱۰.۱. شبکه بازبایی حافظه سرگذشتی (خاطرات سرگذشتی). فراتحلیل تخمین احتمال فعال سازی از ۳۲ مطالعه نشان داد که شبکه مغزی به طور پایایی با بازبایی خاطرات سرگذشتی خاص مرتبط است. این شبکه شامل (۱) هیپوکامپ دوطرفه، (۲) قشر پیش پیشانی میانی، (۳) سینگولیت خلفی، (۴) قشر رترواسپنلیال و پره کونئوس، (۵) شکنج زاویه‌ای دوطرفه، (۶) قطب گیجگاهی و (۷) مخچه راست خلفی است (برگرفته از آدیس و همکاران، ۲۰۱۶).

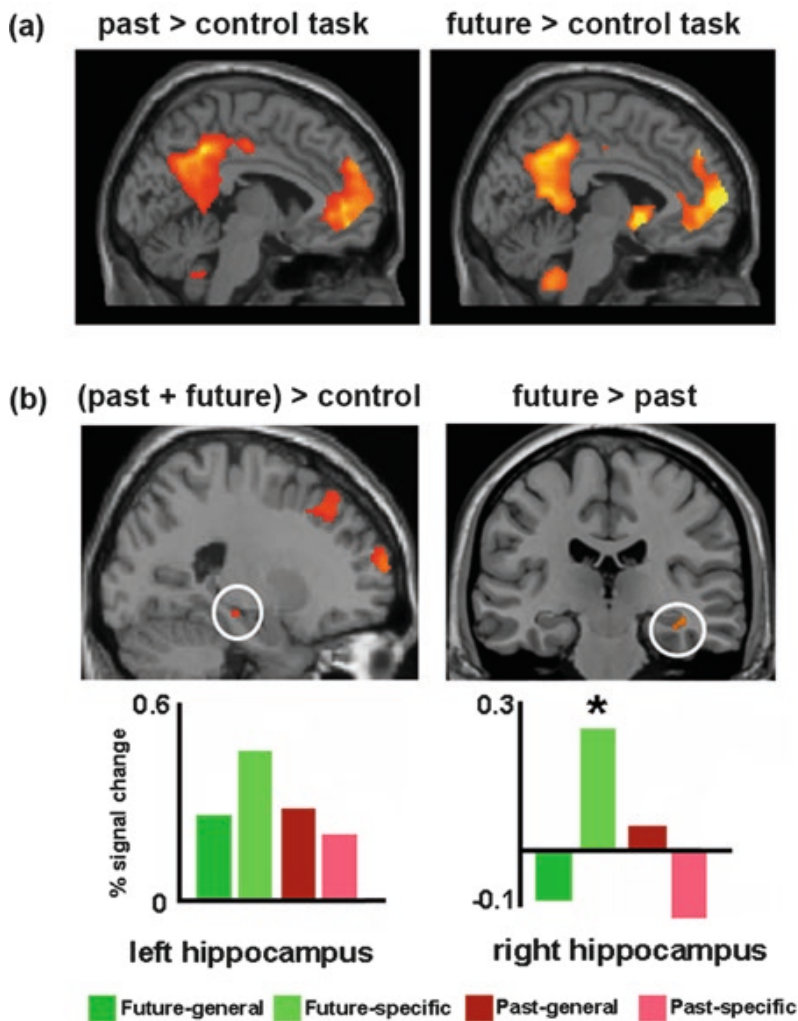


(a)

Memory Type	Temporally Specific?	Personally Relevant?	Example
Episodic AM	+	+	"You were Mike's best man at his wedding"
Semantic AM	-	+	"Ray is the youngest of your brothers"
Public event	+	-	"Windsor Castle was damaged by a fire"
Semantic facts	-	-	"The people of Holland are known as Dutch"
Control task	-	+	"While yet your therefore still about this"
Control task	-	-	"Ago rather he yet if because"



شکل ۲، ۱۰. شبکه بازبایی حافظه سرگذشتی میانی و جانبی چپ، که با تکلیف بازشناسی زندگی‌نامه‌ای نشان داده شده است. (a) شرایط تکلیف بازشناسی زندگی‌نامه‌ای برحسب اختصاصی بودن زمانی و ارتباط شخصی متفاوت است. (b) مغز شیشه‌ای به‌وضوح نشان می‌دهد که فعال‌سازی در طول بازشناسی حافظه سرگذشتی عمدتاً میانی و جانبی چپ است، چنان‌که در موارد زیر دیده می‌شود: (۱) قشر پیش‌پیشانی میانی، (۲) قطب گیجگاهی چپ، (۳) هیپوکامپ چپ، (۴) شکنج گیجگاهی میانی قدامی جانبی چپ، (۵) شکنج پاراهیپوکامپ چپ، (۶) قشر آهیانه‌ای میانی / سینگولیت خلفی، و (۷) اتصال گیجگاهی- آهیانه‌ای. (c) با قراردادن نتایج بر روی یک تصویر MRI، افزایش فعالیت در هیپوکامپ چپ در زمان بازبایی حافظه سرگذشتی زمانی خاص و مرتبط با شخص (یعنی حافظه سرگذشتی رویدادی نسبت به حافظه سرگذشتی معنایی، رویدادهای عمومی و حافظه معنایی عمومی) دیده می‌شود.



شکل ۳،۱۰. فعال‌سازی مرتبط با به خاطر آوردن گذشته و تصور آینده

(a) نقش مشترک شبکه‌ی بازیابی خاطرات سرگذشتی هنگام به خاطر آوردن رویدادهای گذشته و تصور رویدادهای آینده (مربوط به یک تکلیف کنترل معنایی / دیداری-فضایی). (b) فعالیت مشترک (چپ) و افتراقی (راست) هیپوکامپ در طول شبیه‌سازی رویدادهای گذشته و آینده کلی و خاص. در شبیه‌سازی رویدادهای آینده خاص، فعالیت افتراقی هیپوکامپ مشخص بود (برگرفته از آدیس و همکاران، ۲۰۰۷ و ۲۰۱۱).

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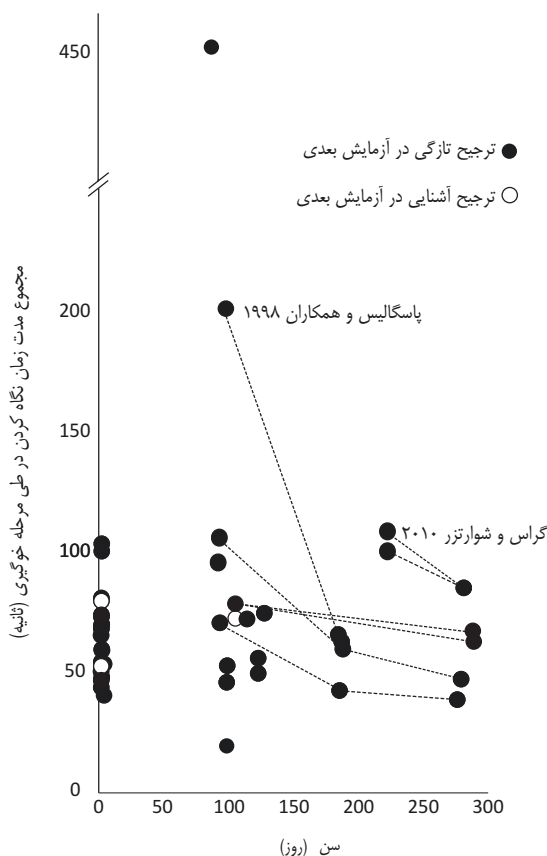
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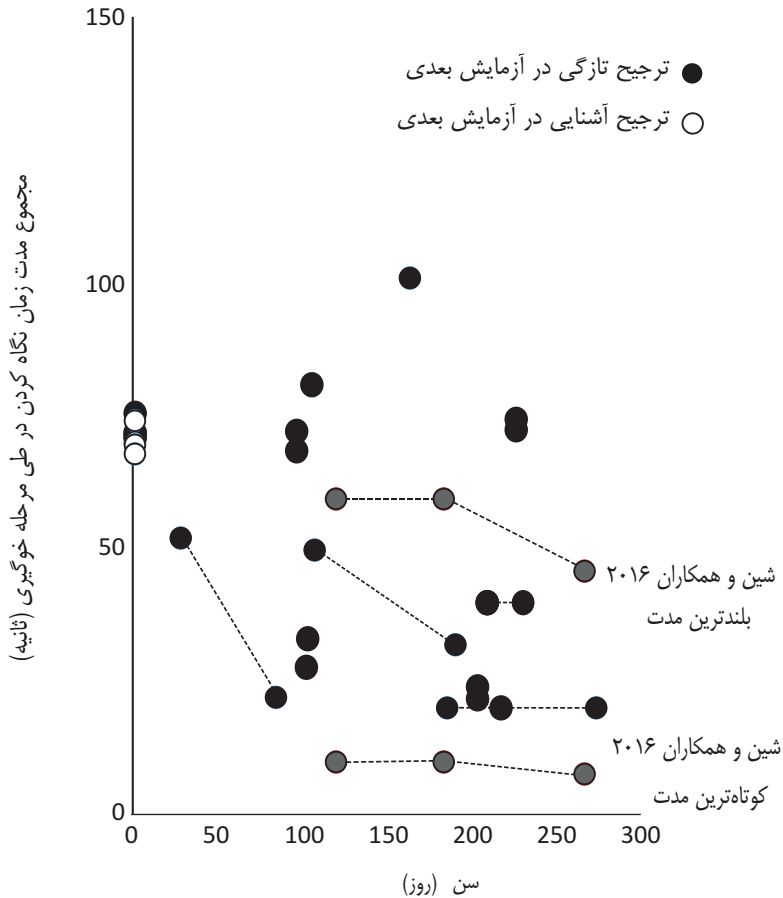
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## منابع فصل یازدهم



**نمودار ۱,۱۱.** مجموع مدت‌زمان نگاه کردن در طول مرحله‌ی خوگیری برای گروه‌های سنی در تحقیقات ذکرشده در جدول ۱,۱۱ دایره‌ی پرشده نمایانگر نقطه‌ی داده‌ی گروهی است که در مرحله‌ی آزمایش بعدی، موجب ترجیح تازگی شده است. دایره‌ی پر نشده نقطه‌ی داده‌ی گروهی را نشان می‌دهد که سبب ترجیح آشنایی در مرحله‌ی آزمایش بعدی شده است. نقاط داده که با خط‌چین به هم مرتبط شده‌اند، داده‌های گروه‌های سنی مختلف در تحقیقات مشابه را بیان می‌کنند. این نقاط داده‌ی مرتبط در گروه‌های سنی، بیان‌کننده‌ی کاهش مدت‌زمان نگاه کردن برای گروه‌های سنی بالاتر است. کاهش مدت‌زمان نگاه کردن نوزادان بزرگ‌تر در مرحله‌ی خوگیری مطابق و هم‌سو با یافته‌های تحقیقاتی است که دوره‌ی تحولی خوگیری به چهره در نوزادان را بررسی کرده‌اند (به‌طور مثال، کلمبو و همکاران، ۲۰۰۴).



**نمودار ۲،۱۱.** مجموع مدت زمان نگاه کردن طی مرحله آشنایی برای هر گروه سنی در بررسی‌های درج شده در جدول ۲،۱۱ دایره پُر شده نمایانگر نقطه داده گروهی است که در مرحله آزمایش بعدی، موجب تارجیح تازگی شده است. دایره پُر نشده نقطه داده گروهی را نشان می‌دهد که سبب تارجیح آشنایی در مرحله آزمایش بعدی شده است. نقاط داده که با خط چین به هم مرتبط شده‌اند، داده‌های گروه‌های سنی مختلف در تحقیق مشابه را بیان می‌کنند. نقاط داده در دایره‌های خاکستری در هر گروه سنی، کوتاه‌ترین تا بلندترین مدت زمان نگاه کردن آشنایی در تحقیق شین و همکاران را نشان می‌دهد (۲۰۱۶).

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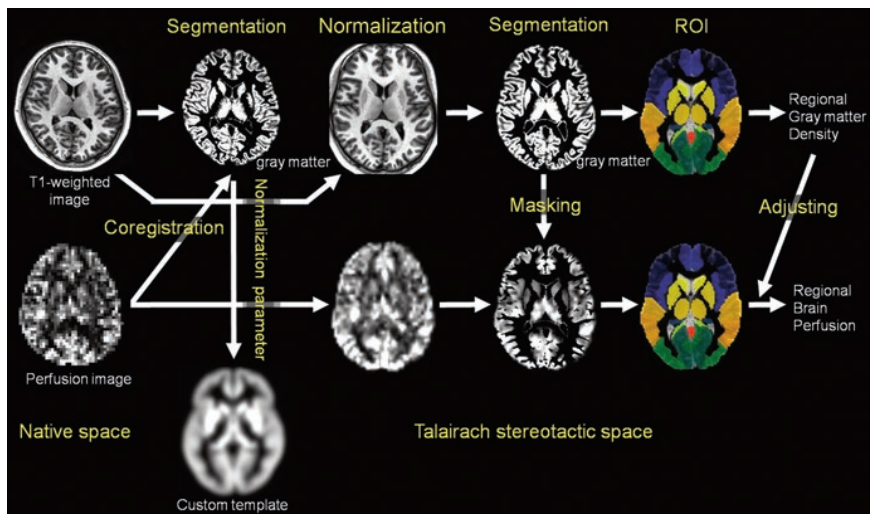
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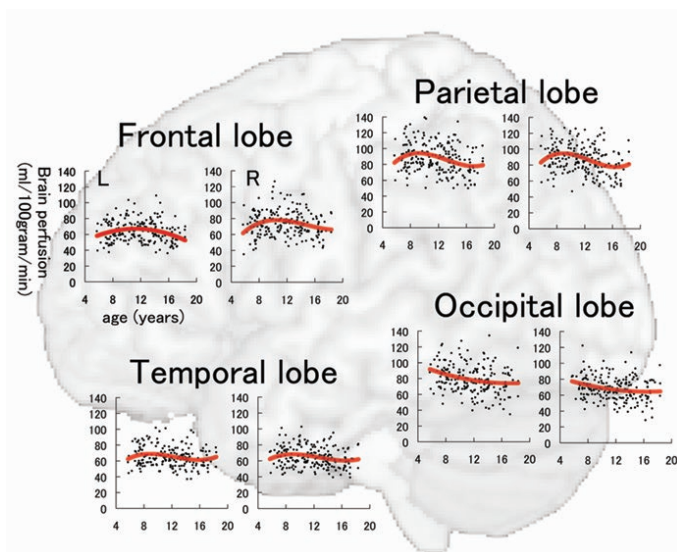
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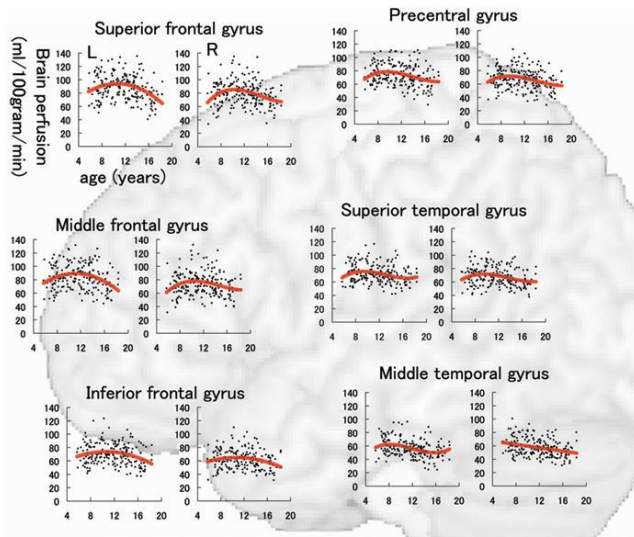
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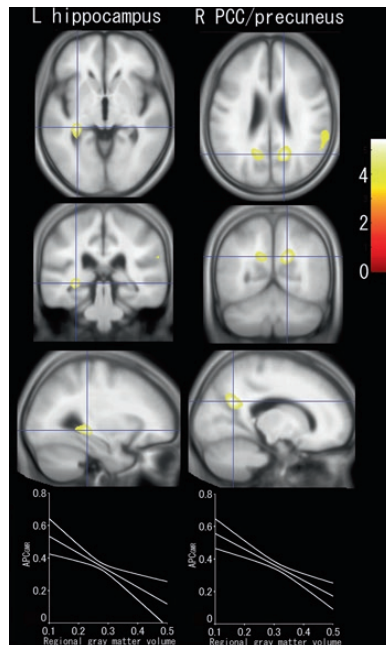
شکل ۱،۱۲. طرح کلی از تجزیه و تحلیل تصاویر



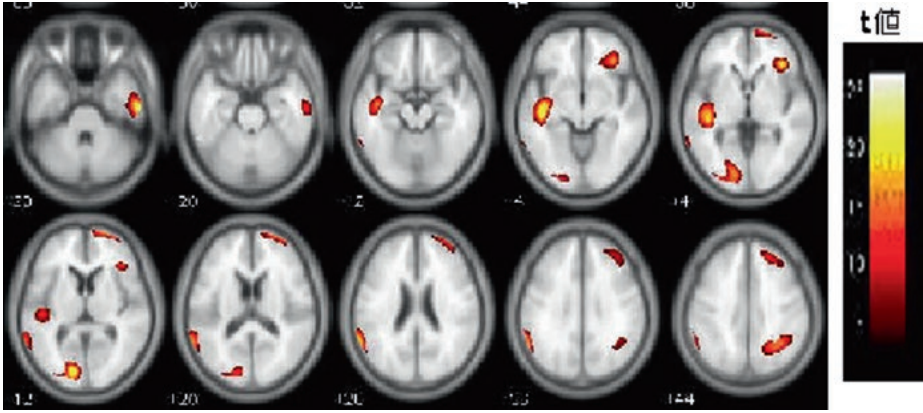
شکل ۲،۱۲. ارتباط بین پرفیوژن مغزی، تطابق تراکم ماده خاکستری و سن در لوب پیشانی، لوب آهیانه، لوب پس سری و لوب گیجگاهی در هر نیمکره



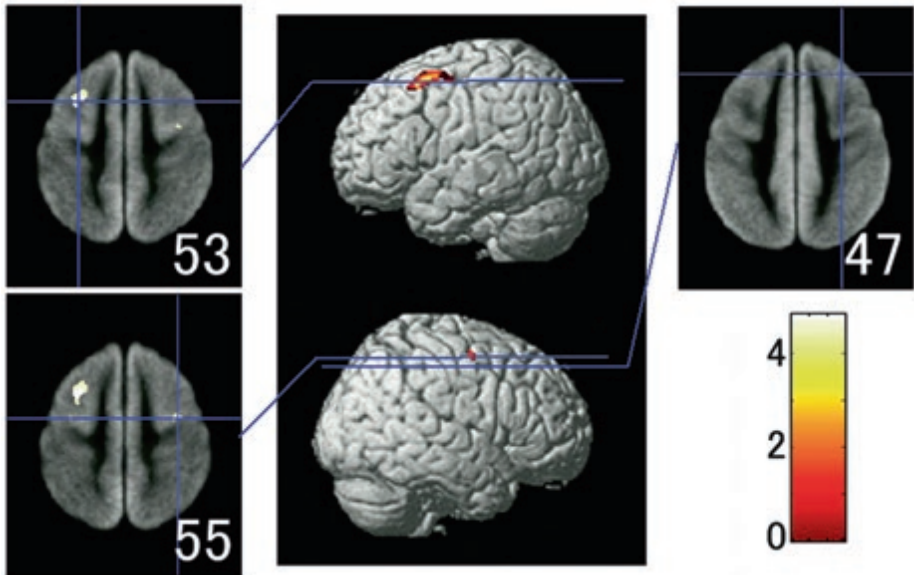
شکل ۳، ۱۲. ارتباط بین پرفیوژن مغزی، تطابق برای تراکم ماده خاکستری و سن در شکنج پیش مرکزی، شکنج پیشانی فوقانی، شکنج پیشانی میانی، شکنج پیشانی تحتانی، شکنج گیجگاهی فوقانی و شکنج گیجگاهی میانی



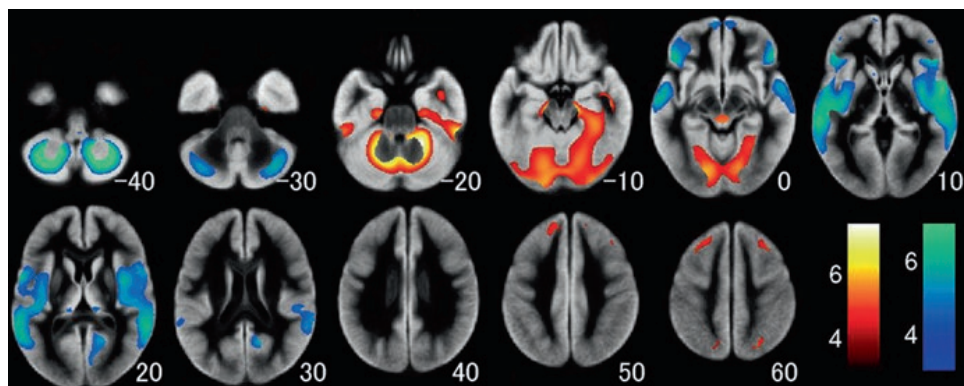
شکل ۴، ۱۲. مناطق ماده خاکستری که نمایانگر ارتباط منفی مهم با درصد تغییر سالانه نسبت ماده خاکستری (APCGMR) تنظیم شده برای سن، جنس و حجم درون مجموعه‌ای است



شکل ۵،۱۲. مناطق مغزی که ارتباط معکوس بین حجم ماده خاکستری و فشار سیستولیک خون را نشان می‌دهند



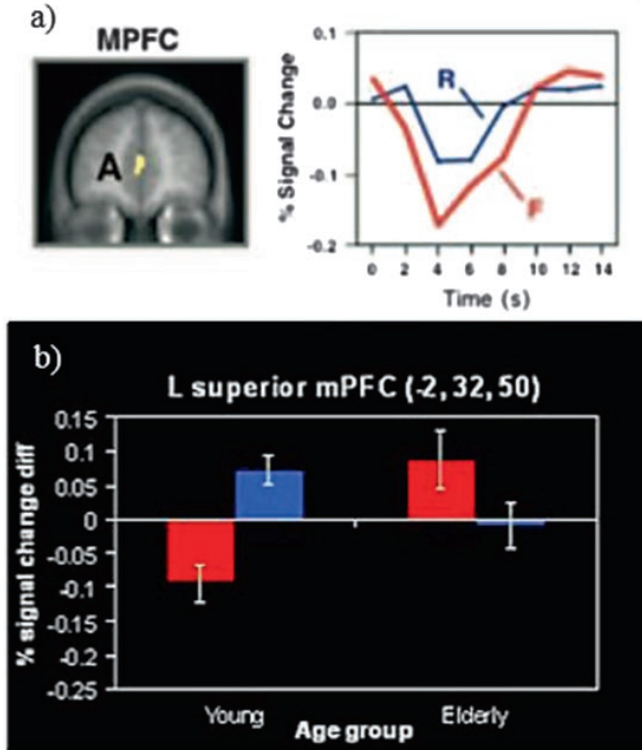
شکل ۶،۱۲. مناطق مغزی که ارتباط منفی بین ماده خاکستری و حجم مصرف الکل در طول زندگی را نشان می‌دهند



شکل ۷، ۱۲. مناطق مغزی که رابطه بین حجم ماده خاکستری و شاخص توده بدنی (BMI) را نشان می‌دهد. رنگ قرمز و آبی به ترتیب نمایانگر روابط منفی و مثبت هستند



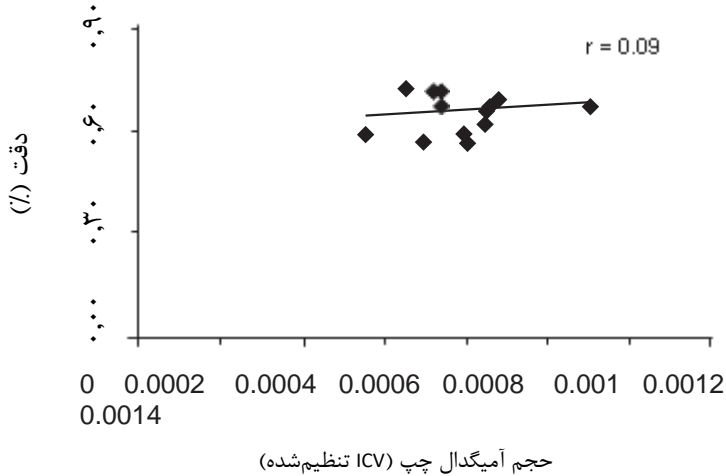
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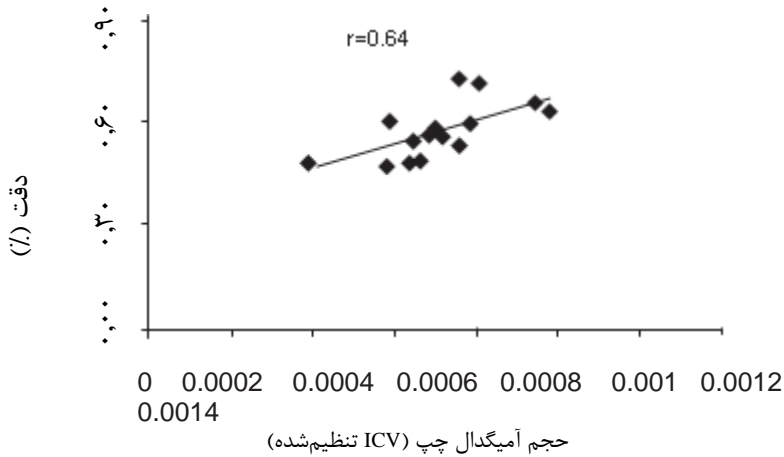
شکل ۱۳. تفکر درباره خود، مناطق عصبی مختلفی را فعال می‌کند که از مناطق مربوط به تفکر درباره دیگران قابل تفکیک هستند.

(الف) در پژوهشی شرکت‌کنندگان مشخص کردند که آیا ویژگی‌های شخصیتی خاصی، توصیف‌کننده خود آن‌ها است یا خیر. در این حال فعال‌سازی مغز با استفاده از fMRI اندازه‌گیری شد. تضادها بر اساس اینکه آیا هر کلمه به خاطر سپرده شده یا فراموش شده است و اینکه آیا این ویژگی‌ها خود توصیفی در نظر گرفته شده است یا خیر، ایجاد شد. در قشر پیش‌پیشانی میانی و در حین خودقضاوتی برای کلماتی که بعداً به یاد می‌آیند (R) در مقابل کلمات فراموش شده (F) فعال‌سازی رو به افزایشی مشاهده می‌شود (با اقتباس ماکرا و همکاران ۲۰۰۴ و با مجوز). (ب) وقتی قضاوت درباره خود و دیگران به‌طور هم‌زمان انجام می‌شود، فعال‌سازی‌ها به تغییر رویکرد مرتبط با سن از الگوهای قبلی منجر می‌شود. ارزیابی پارامترها برای قشر پیش‌پیشانی بالایی چپ، حوزه کاری افزایش‌یافته برای افراد جوان‌تر در خصوص موارد خودارجاعی که بعداً فراموش شده‌اند و در مقابل، برای سالمندان در موارد مربوط به خودی که متعاقباً به یاد می‌آیند و اثرات فراموشی برای موارد ارجاع به دیگران را نشان می‌دهد (با اقتباس از گوچس و همکاران ۲۰۱۰ و با مجوز).

## افراد جوان تر



## سالمندان

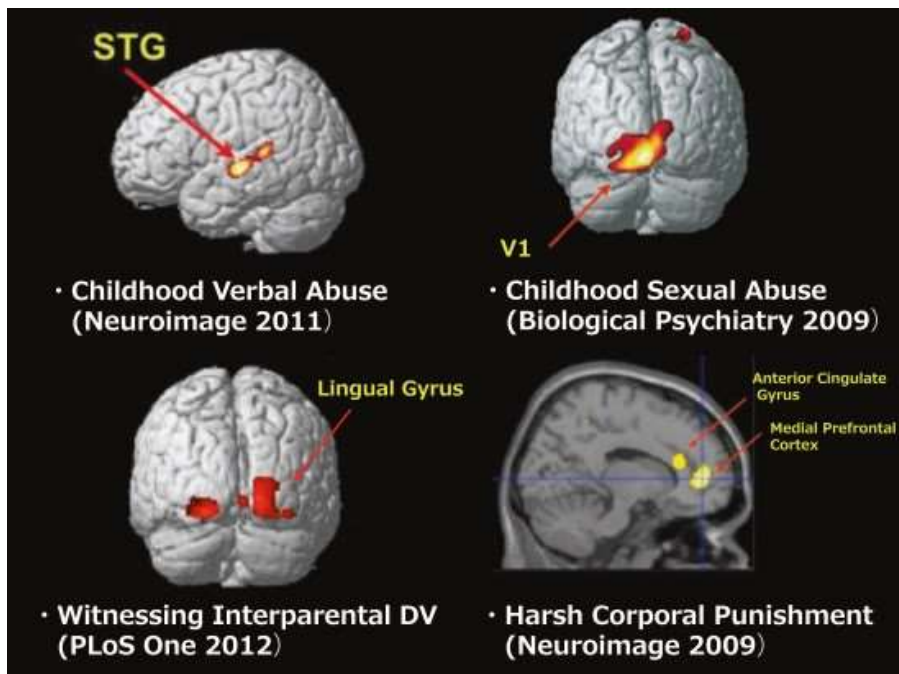


**نمودار ۲،۱۳.** پژوهش قبلی نشان داده است یکپارچگی ساختاری مناطق که در شکل گیری برداشت نقش دارند، به عملکرد حافظه متعاقب حاوی اطلاعات اجتماعی مربوط می شود. تصویربرداری های ام آر آی ساختاری افراد جوان و میانسال که در حال انجام تکالیف مربوط به برداشت های مبتنی بر رفتار و آزمون حافظه متعاقب بودند، نشان داد که میزان حجم آمیگدال چپ بیشتر، می تواند حافظه مربوط به برداشت کلی افزایش یافته را تنها در سالمندان پیش بینی می کند (اقتباس از کسیدی و همکاران (۲۰۱۲) ب) با مجوز).

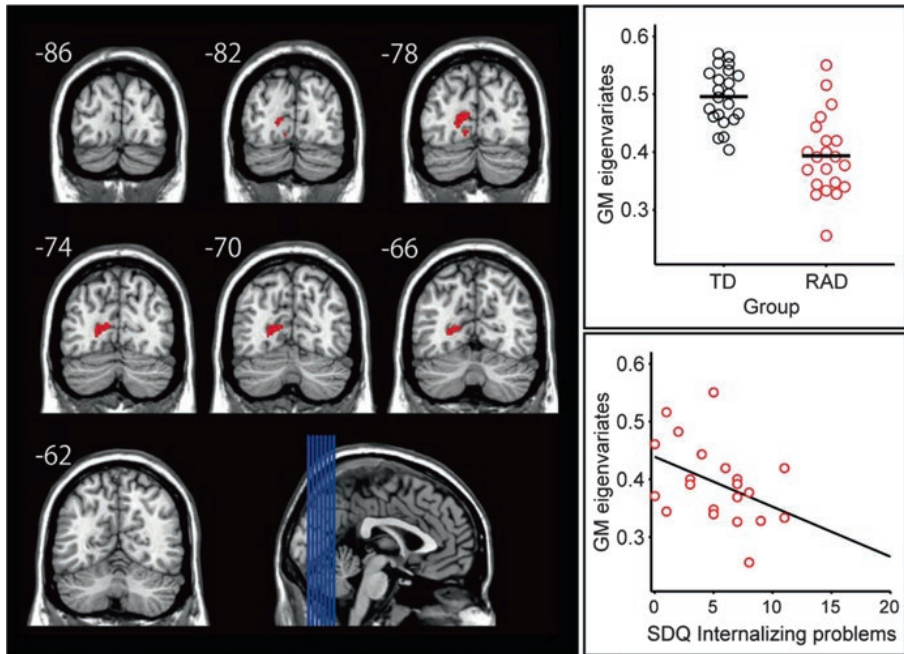
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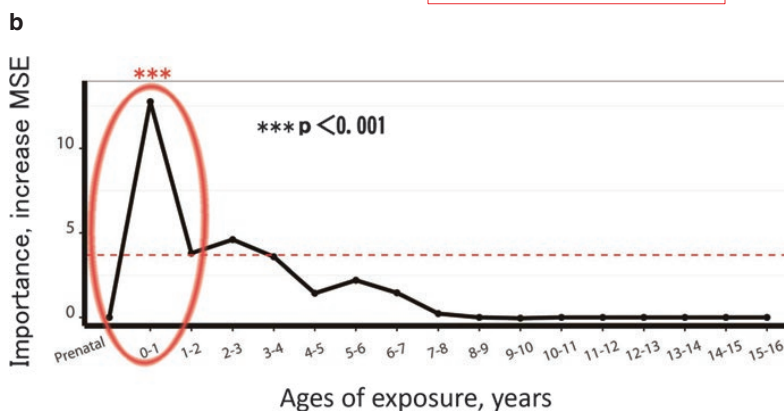
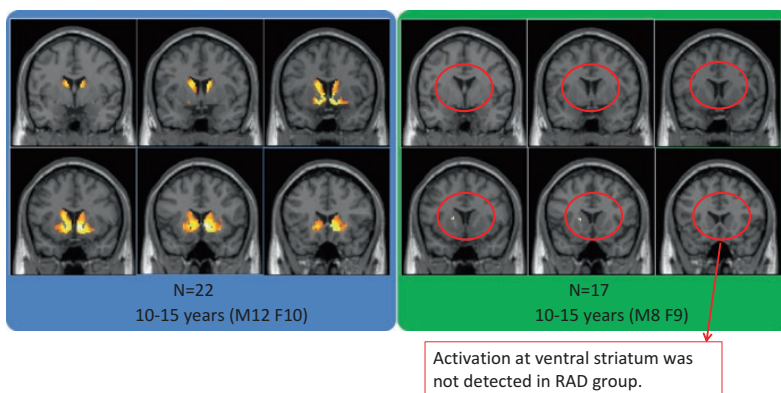


شکل ۱۴، ۱. تفاوت‌های بسیار در قشر مغزی بین افرادی که مورد بدرفتاری قرار گرفته‌اند و افراد گروه کنترل با استفاده از مورفومتری مبتنی بر وکسل دیده می‌شود (تومودا و همکاران ۲۰۰۹ الف، ب، ۲۰۱۱ و ۲۰۱۲). مشخصاً در افرادی که مورد آزار کلامی والدین بودند، تراکم ماده خاکستری (GM) در شکنج گیجگاهی فوقانی بالاتر بود (STG؛ شکل بالا سمت چپ)، در افرادی که در دوران کودکی مورد آزار جنسی قرار گرفته بودند، تراکم ماده خاکستری (GM) به‌طور قابل توجهی در قشرهای ارتباطی بینایی (شکل بالا سمت راست) و بینایی اولیه (V1) راست و چپ کمتر ارزیابی شد. در افرادی که شاهد خشونت خانگی بین والدینی بودند، تراکم ماده خاکستری (GM) به‌طور معنی‌داری در شکنج زبانی راست کمتر (شکل پایین سمت چپ) و تراکم ماده خاکستری (GM) مشخصاً در افرادی که مورد تنبیه بدنی شدید بودند، در قشر پیش‌پیشانی میانی سمت راست و شکنج کمربندی قدامی راست (شکل پایین سمت راست) کمتر ارزیابی شد.

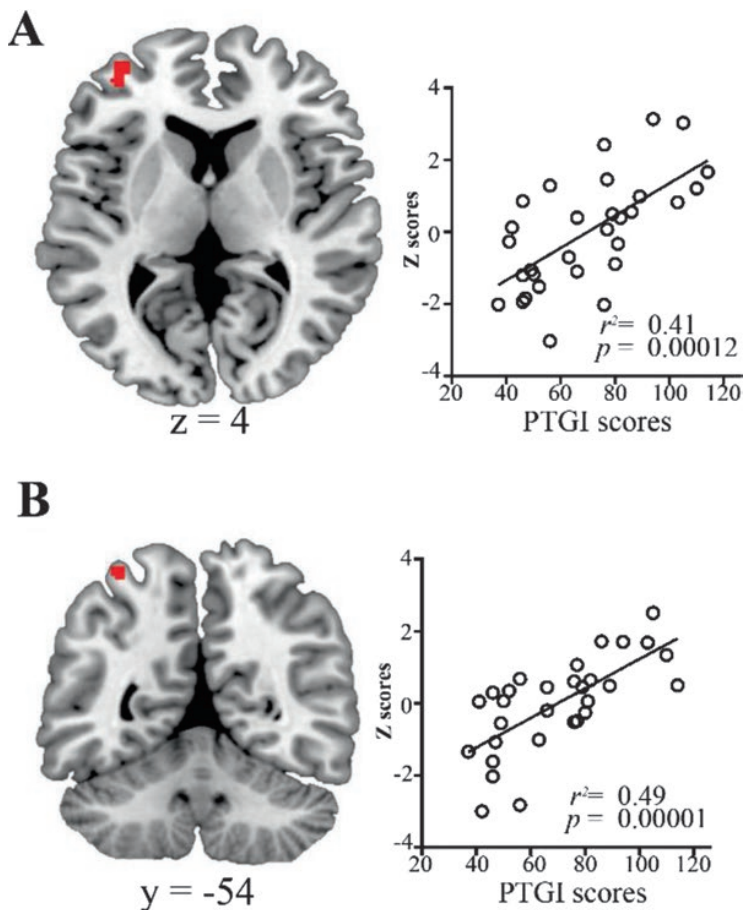


**شکل ۱۴.** تفاوت‌های ساختاری در میزان ماده خاکستری منطقه‌ای بین گروه‌هایی با رشد معمولی (TD) و اختلال دلبستگی واکنشی (RAD) (شیمادا و همکاران ۲۰۱۵). گروه اختلال دلبستگی واکنشی، میزان ماده خاکستری کاهش‌یافته مهمی را در قشر بینایی اولیه چپ (BA17) در مقایسه با گروه معمولی ( $FWE, p = 0.038$ ) سطح خوشه‌ای اصلاح‌شده) نشان داد. مقیاس‌های رنگی نمایانگر مقادیر  $t$  هستند. مقیاس‌های علائم روان‌پزشکی برای مشکلات درونی‌سازی پرسش‌نامه SDQ ( $\beta = -0.96, t = -3.86, p < 0.05$ ) پیش‌بینی‌کننده مهمی برای ارزیابی





شکل ۳، ۱۴. نقشه‌های پارامتری آماری پاداش پولی زیاد (HMR) منهای بدون پاداش پولی (NMR)] در افراد TD و بیماران مبتلا به اختلال دلبستگی واکنشی (تاکیدگویی و همکاران ۲۰۱۵). (الف) نقشه‌های پارامتری آماری مواجهه با چالش پاداش پولی زیاد در TD، اختلال دلبستگی واکنشی و TD منهای اختلال دلبستگی واکنشی (تاکیدگویی و همکاران ۲۰۱۵). سمت راست (R) و سمت چپ (L) و محور عرض‌ها (مختصات MNI) نشان داده شده است. آستانه تحمل تجزیه و تحلیل در  $P < 0.001$  سطح و کسل و  $P < 0.05$  با اصلاح FWE برای مقایسه‌های متعدد در سطح خوشه‌ای کل مغز تنظیم شد. هریک از شش قسمت مینا از گروه TD ( $n = 22$ ) ۱۲ مرد و ۱۰ زن، ۱۰-۱۵ سال) و گروه اختلال دلبستگی واکنشی ( $n = 17$ ) ۸ مرد و ۹ زن، ۱۰-۱۵ ساله) نشان داده شده است. (ب) حداکثر حساسیت پیرامون سن مواجهه (حداکثر اهمیت سن مواجهه، صرف‌نظر از نوع آن) در اختلال دلبستگی واکنشی (تاکیدگویی و همکاران ۲۰۱۵). یافته‌های رگرسیون جنگل تصادفی با درختان مشروط، اهمیت مواجهه با بدرقتاری اولیه از بدو تولد تا ۱۵ سالگی و با توجه به ارزیابی‌های مقایسه‌ای پاداش پولی زیاد (پاداش پولی زیاد منهای NMR) برای (الف) جسم مخطط راست و (ب) جسم مخطط چپ را نشان داد. این اهمیت، همان‌طور که با افزایش خطای مجذور میانگین، پس از حذف مؤثر هر سن از مدل با جایگشت نشان داده می‌شود، با تنزل در تناسب نمایش داده می‌شود. خط افقی خطچین، سطح معنی‌داری برای مقادیر بااهمیت متغیر نشان می‌دهد.



شکل ۱۴، ۴. یافته‌های تحلیل رگرسیون چندگانه میان شبکه‌ی اجرایی مرکزی و نمرات پرسش‌نامه‌ی رشد پس از سانحه (PTGI) (فوجیساوا و همکاران ۲۰۱۵). مناطق مغزی نشان‌دهنده‌ی ارتباط مستقیم بین نمرات پرسش‌نامه‌ی رشد پس از سانحه و توانایی فعالیت شبکه‌ی اجرایی مرکزی است که توسط تحلیل رگرسیون چندگانه مشخص می‌شود. نمودارهای پراکنده‌ی ارتباط میان نمرات پرسش‌نامه‌ی رشد پس از سانحه و توانایی فعالیت شبکه‌ی اجرایی مرکزی را نشان می‌دهد. (الف) قشر پیش‌پیشانی قدامی؛ (ب) لوبول ناحیه‌ی فوقانی آهیانه. آستانه‌ی آماری برای اختلاف‌ها، سطح و کسل  $p < 0.001$  اصلاح‌نشده برای ارتفاع و سطح خوشه‌ای  $P < 0.05$  برای مقایسه‌های چندگانه بود.

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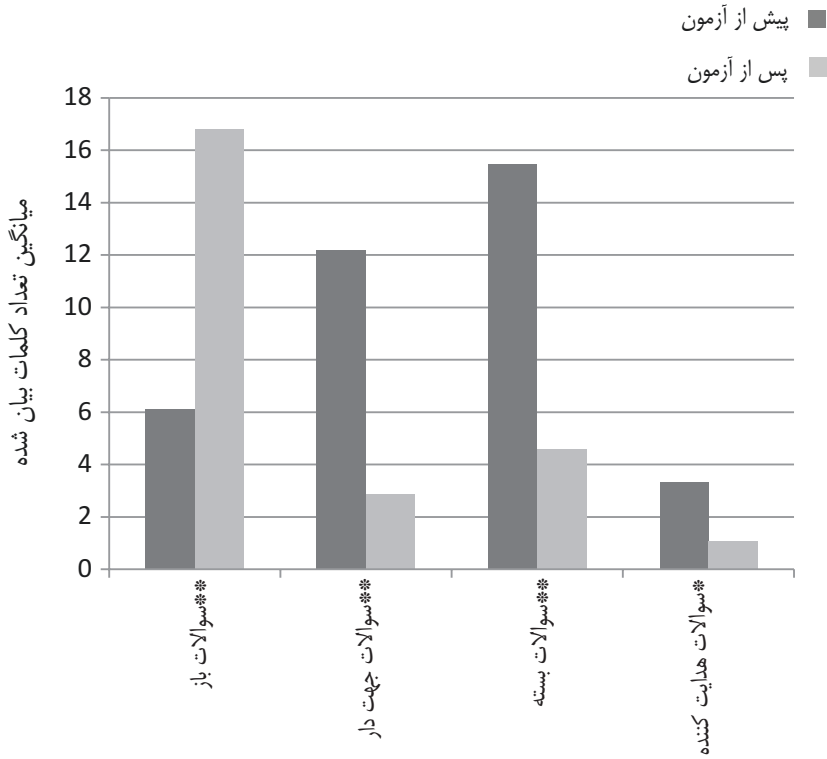


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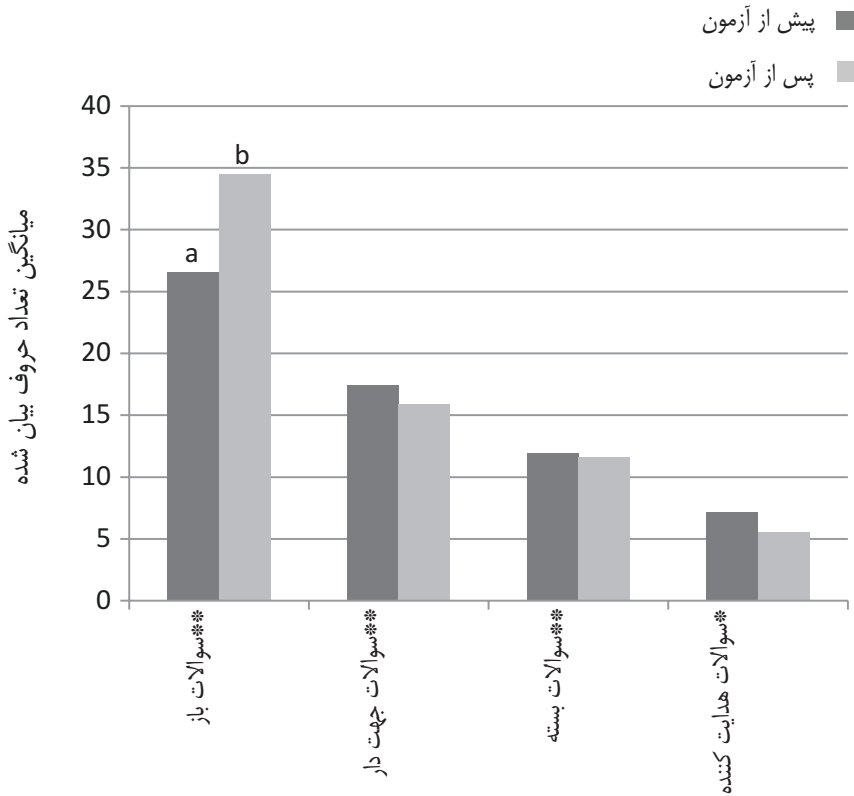
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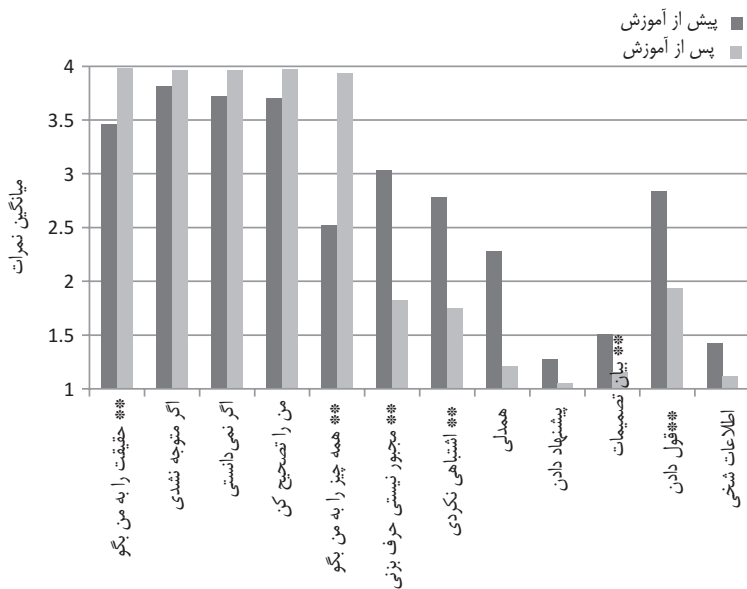
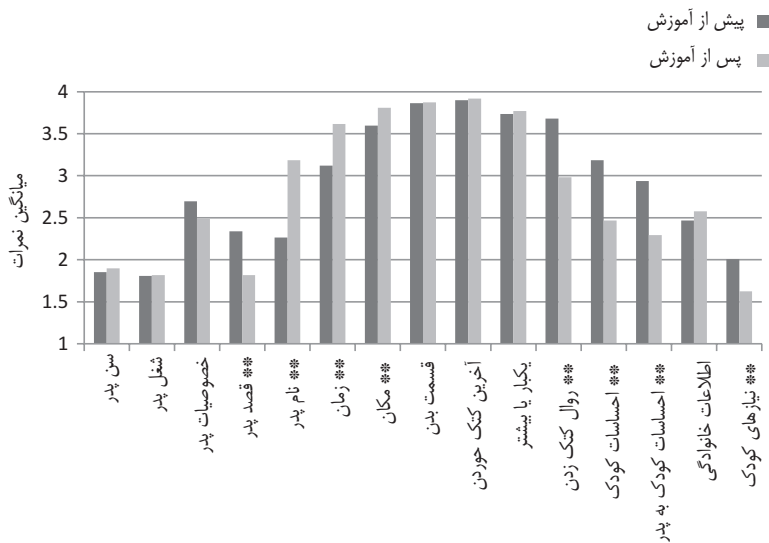
## منابع فصل هفدهم



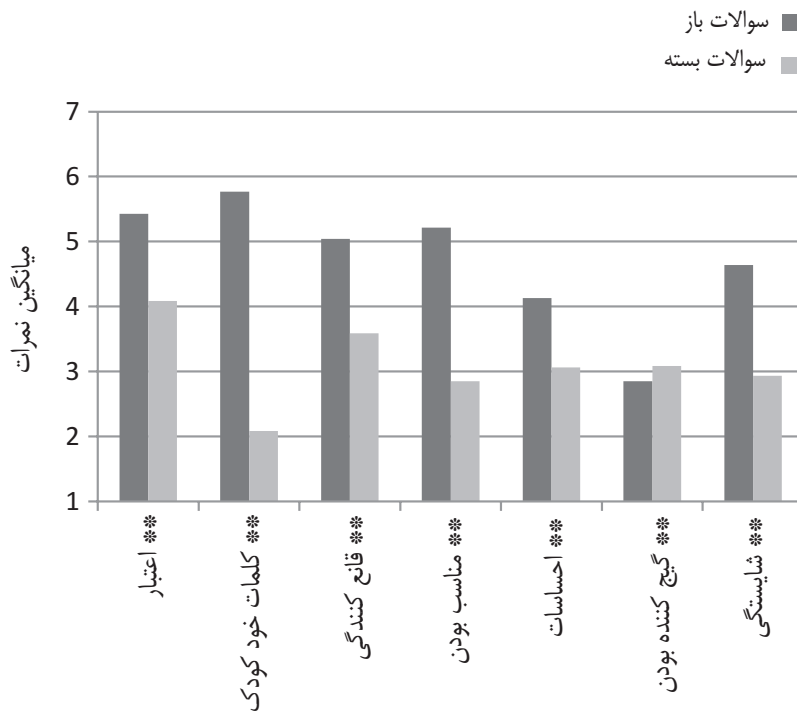
شکل ۱۷، ۱. میانگین میزان گفتار در هر نوع سؤال قبل و بعد از آموزش  
 (\* $p < 0.05$ ); (\*\* $p < 0.01$ )



شکل ۲،۱۷. میانگین تعداد حروف به دست آمده توسط هر نوع سؤال (پایین) در قبل و بعد از آموزش (الف) قبل از آموزش، سوالات باز و جهت دار اطلاعات بیشتری را نسبت به سوالات هدایت کننده به دست آوردند ( $p < 0.01$ ). سوالات جهت دار اطلاعات بیشتری نسبت به سوالات بسته به دست آوردند ( $p < 0.01$ ). سوالات بسته اطلاعات بیشتری نسبت به سوالات هدایت کننده به دست آوردند ( $p < 0.01$ ) (ب) پس از آموزش، سوالات باز اطلاعات بیشتری را نسبت به سوالات جهت دار ( $p < 0.05$ )، سوالات بسته و سوالات هدایت کننده ( $p < 0.05$ ) به دست آورد. سوالات جهت دار اطلاعات بیشتری نسبت به سوالات بسته و هدایت کننده به دست آوردند ( $p < 0.01$ ). سوالات بسته اطلاعات بیشتری نسبت به سوالات هدایت کننده به دست آوردند ( $p < 0.01$ ).



شکل ۱۷، ۳. دیدگاه حرفه‌ای‌ها درباره اطلاعاتی که باید جمع‌آوری شود (بالا) و اطلاعاتی که باید بیان شود (پایین)  
 \*\*  $p < 0.01$



شکل ۴، ۱۷. ارزیابی شهادت کودک در وضعیت سوالات باز و سوالات بسته

$p < 0.01$  \*\*

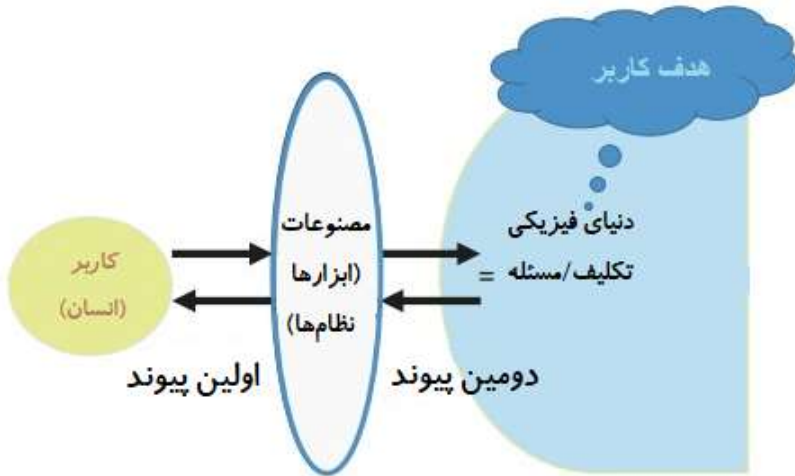
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## منابع فصل هجدهم



شکل ۱،۱۸. مدل مهندسی شناختی: نظریه پیوند دوگانه (سایکی ۱۹۸۸)

۳. فراشناخت دو نگرش‌ها / راهبردها و تنظیمات هدف  
- شامل عوامل فرهنگی/اجتماعی  
- مؤثر نگه داشتن خود به عنوان هدف اجتماعی

۲. کمبود دانش / مدل خای ذهنی  
- به خصوص فقدان مفهوم اطلاعات

۱. تغییر/کاهش در عملکردهای شناختی  
- به ویژه کاهش بازداری یا مهار و/ کاهش سرعت

۰. کاهش عملکردهای ادراکی / فیزیکی  
- تأثیر بر شناخت از طریق تلاش بیشتر

شکل ۲،۱۸. منابع فرضی مشکلات پیری شناختی در استفاده از تجهیزات فناوری اطلاعات: یک مدل چهارلایه (هارادا ۲۰۰۹)

اصل جامع طراحی:  
مشکلات برای همه افراد مشترک است  
یا طراحی بد برای همه بد است

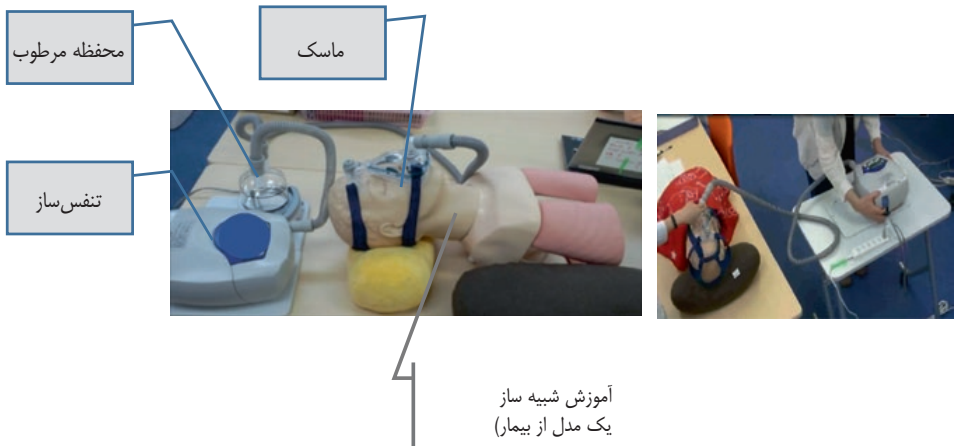
سالمدان نسبت به شناسایی قابل استفاده بودن و  
مشکلات طراحی حساس هستند



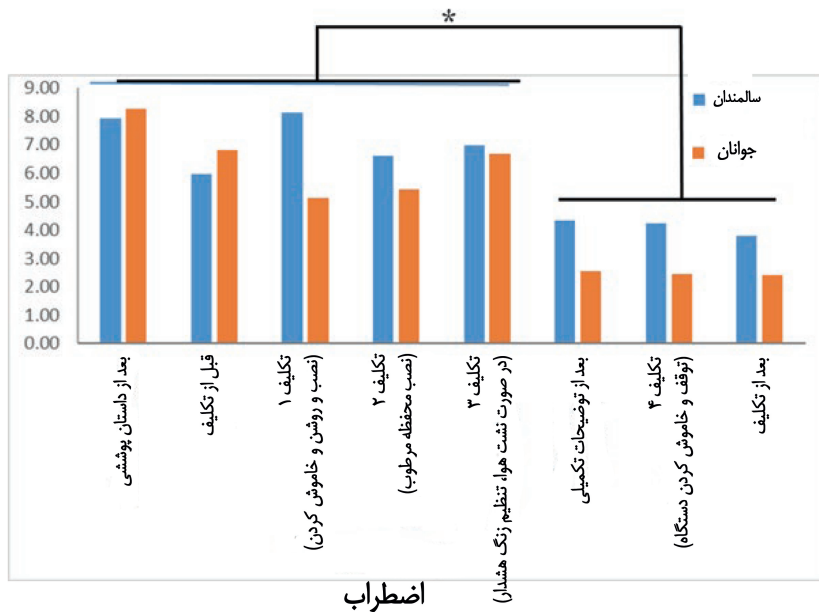
بزرگسالان جوان تر می‌توانند به تنهایی بر مشکلات  
طراحی لبه کنند  
(با دانش خود با تجربیات پرخطا):  
افراد مسن نمی‌توانند مشکلات را حل کنند

سؤال اساسی این است:  
قابلیت استفاده چیست؟  
و پیری شناختی چه معنایی دارد؟

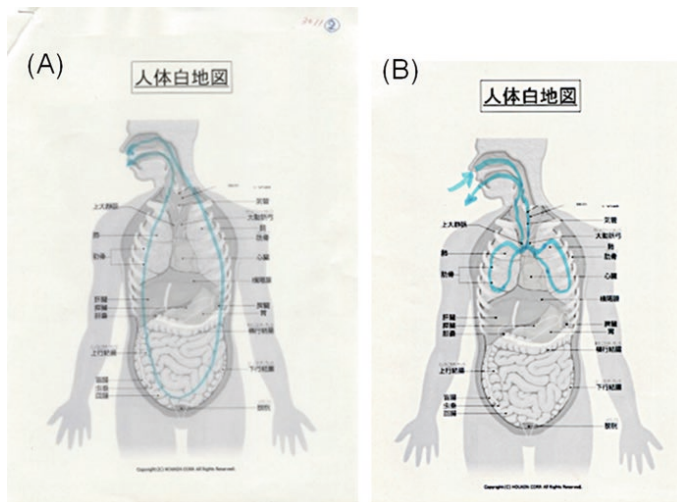
شکل ۳,۱۸. جوانب دوگانه سالمندی و قابلیت استفاده



شکل ۴,۱۸. صحنه آزمایش قابلیت استفاده یک دستگاه تنفسی از نوع NPPV (تهویه با فشار مثبت غیرتهاجمی)



شکل ۱۸، ۵. ارزیابی ذهنی اضطراب: هیچ اختلاف سنی وجود ندارد و شرکت کنندگان فقط پس از تکمیل همه تکالیف، اضطرابشان کاهش یافت (هارادا و اویچی ۲۰۱۴)



شکل ۱۸، ۶. نمونه‌هایی از ترسیم (خطوط آبی روی نمودار) در حین توضیح «تنفس». (الف) هوا به سمت «شکم» پایین می‌رود، (ب) الگوی معمول نقاشی بزرگسالان جوان‌تر

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