1	tafii250	bind	tbp	experiment				
This sa DNA l	ame region of <mark>TAFII250</mark> binds binding by TFIID .	s to TBP and represses its i	nteraction with TATA boxes,	thereby decreasing				
http://v	www.jbc.org/cgi/content/full/2	276/27/25582						
2	proteasome	degrade	p53	no experiment				
As we ubiqui and Ya	ll as directly blocking p53 acti tinated and degraded by the p1 asuda, 1999).	ivity in the nucleus, MDM2 roteasome (Haupt et al., 19	2 induces <mark>p53</mark> export to the cy 97 ; Kubbutat et al., 1997 ; Ro	toplasm where <mark>p53</mark> is oth et al., 1998 ; Honda				
<u>http://e</u>	emboj.oupjournals.org/cgi/con	tent/full/18/16/4424						
3 creb depend on pka no exper								
Howev depend	ver, the mutant seems also to a dent phosphorylation of CREI	ffect other <mark>p38</mark> -dependent : <mark>3</mark> .	activities (e.g. p62TCF activa	tion) and the PKA-				
http://e	emboj.oupjournals.org/cgi/con	tent/full/16/5/1009						
4	hairy	interact	groucho	no experiment				
Thus, i the two	it has been suggested that Hain p proteins are components of a	ry interacts with both dCtl common corepressor com	BP and <mark>Groucho</mark> , thereby raisi plex (9).	ing the possibility that				
http://v	www.pnas.org/cgi/content/full	/96/2/535						
5	p53	interact	cbp	experiment				
In sum http://l	mary, we have identified mult inkinghub.elsevier.com/retriev	tiple FKLF2 interacting do ve/pii/S0006291X0200842	mains and a new <mark>p53</mark> interacti <u>2</u>	ng domain of <mark>CBP</mark> .				
6	cbp	interact	creb	experiment				
In this extent http://v	regard, our demonstration tha of this association is correlate www.jbc.org/cgi/content/full/2	t CBP interacts with CREI d with the ALAS promoter 278/4/2317	under basal conditions in He activity provides a strong sup	pG2 cells and that the port for our hypothesis				
7	ctbp	interact	e1a	experiment				
variety http://v 8 Consec	v of cellular transcriptional re www.jbc.org/cgi/content/full/2 akt quently, although we do not kn	epressors through variation 277/41/38755 lead to now at present how TNFal tinocutes, a possible explore	s of a motif related to -PXDL jnk pha is produced in the absence	S experiment e of normal NF-				
expres	sion can lead to the activation	of <mark>JNK</mark> .	ation might be that Art mino					
<u>nup.//</u>	www.jbc.org/cgi/content/1uii/2	nhognhogylata	ask 2hoto					
9 A a DT	aKi EN inactivatas Akt/DKD (21)	phosphorylate	gsk-Speta	no experiment				
AST 1 Akt/P	KB (7).), it activates GSK-Speta,	which is phosphorylated and h	nactivated by				
http://1	ncb.asm.org/cgi/content/full/2	3/17/6139						
10	p53	interact	cbp	experiment				
B, wile	d-type p53 interacts with CBP							
http://v	www.jbc.org/cgi/content/full/2	274/20/13760						
11	hh	regulate	ci	no experiment				
Nonetl the ran activat	heless, the dependence of emb ge of Hh action (Chen and St or activity of <mark>Ci</mark> .	ryonic wg expression on C ruhl, 1996) allow us to attr	i activator function and the re ribute an essential in vivo role	ble of Ptc in restricting to the <mark>Hh</mark> -regulated				
http://v	www.cell.com/cgi/content/full	/96/6/819						
12	pka	regulate	creb	no experiment				
CREB kinase et al., 1	phosphorylation is regulated (CaMK) (Matthews et al., 19 1999).	by <mark>PKA</mark> and by other prote 994), and protein kinase C	in kinases such as Ca2+/calm C (PKC) (Muthusamy and Lei	odulin-dependent den, 1998 ; Roberson				
http://j	pet.aspetjournals.org/cgi/conte	ent/full/301/1/66						
13	catenin	bind	tcf	no experiment				

Upon activation, -catenin binds nuclear TCF and may recruit the basal transcriptional complex to the promoter possibly via the TATA binding protein TBP [Hecht et al., 1999] or the TBP-associating protein TIP49 [Bauer et al., 1998].

<u> http://l</u>	inkinghub.elsevier.com/retriev	ve/pii/S0012160603003051		
14	ck2	phosphorylate	tbp	no experiment
The av	vailable evidence suggests that ated by CK2 is not known.	CK2 phosphorylation of T	BP activates TFIIIB , althoug	gh the step in initiation
http://v	www.cell.com/cgi/content/full	/106/5/575		
15	calmodulin	bind	fusion proteins	experiment
After v	washing, <mark>calmodulin</mark> bound to	<mark>fusion proteins</mark> was eluted	l and resolved by <mark>SDS</mark> -PAGI	Ξ.
http://v	www.jbc.org/cgi/content/full/2	275/51/39846		
16	ftz	interact	ftz-f1	no experiment
In Dro of <mark>Ftz</mark>	sophilids, loss of the YPWM 1 with <mark>Ftz-F1</mark> such that the fund	motif abolished this compet ction of Dm-Ftz was devote	ition for cofactors, allowing a entirely to segmentation.	for exclusive interaction
http://v	www.current-biology.com/cgi/	/content/full/11/18/1403		
17	ctbp	bind	e1a	no experiment
The bi	nding of <mark>CtBP</mark> to <mark>E1A</mark> repress	ses CR1-dependent transcri	ptional activation and tumori	genesis (21, 34).
http://v	www.jbc.org/cgi/content/full/2	274/16/11334		
18	abd-a	repress	ubx	experiment
Interes posteri	stingly, 69B=> <mark>Abd-A</mark> represse for expansion of Scr expressio	es <mark>Ubx</mark> in ectodermal tissue n in the VMS (Fig. 5C).	es (Table 1) and appears to p	roduce a slight
http://l	inkinghub.elsevier.com/retriev	ve/pii/S092547730100301X		
19	pka	phosphorylate	gsk-3beta	no experiment
Althou severa previo	igh phosphorylation and inacti l cell lineages (32, 33, 57-62) usly reported.	vation of GSK-3beta by Pl , whether this inactivation le	KA, PKB/Akt, and PKC have eads to free <i>beta</i> -cat accumul	e been demonstrated in ation has not been
http://v	www.jbc.org/cgi/content/full/2	<u>278/2/1380</u>		
			•	
20	hh	induce	ci	no experiment
20 It wou concer extents	hh Id be important, therefore, to r ntrations of Hh, and to determi s.	induce neasure the levels of Ci pho ine if distinct thresholds of	ci osphorylation in cells stimula th induce dephosphorylation	no experiment ted with different n of <mark>Ci</mark> to different
20 It wou concer extents http://v	hh Id be important, therefore, to r ntrations of Hh, and to determi s. www.genesdev.org/cgi/conten	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315	ci osphorylation in cells stimula Hh induce dephosphorylation	no experiment ted with different a of Ci to different
20 It wou concer extents http://v 21	hh Id be important, therefore, to r ntrations of Hh, and to determi s. www.genesdev.org/cgi/conten creb	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on	ci osphorylation in cells stimula th induce dephosphorylation mapk	no experiment ted with different n of Ci to different no experiment
20 It wou concer extents http://v 21 Concu MAPI depend	hh Id be important, therefore, to r intrations of Hh, and to determine s. www.genesdev.org/cgi/contern creb rrent increases in Ca2+ and cA is signal, in addition to the pre- dent activation of CREB (Imp	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998).	ci osphorylation in cells stimula Hh induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways	no experiment ted with different n of Ci to different no experiment rolonged phospho- s in the MAPK-
20 It wou concer extents http://v 21 Concu MAPH depend http://v	hh Id be important, therefore, to r intrations of Hh, and to determing www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on MP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways	no experiment ted with different n of Ci to different no experiment rolonged phospho- i in the MAPK-
20 It wou concer extents http://v 21 Concu MAPH depend http://v 22	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA is signal, in addition to the predent activation of CREB (Imp www.jneurosci.org/cgi/content pka	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate	ci osphorylation in cells stimula Hh induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways ci	no experiment ted with different n of Ci to different no experiment rolonged phospho- s in the MAPK- no experiment
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The in duplica al., 199	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content Creb rrent increases in Ca2+ and cA signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka plication that PKA may there ation mutations inactivates the 95).	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pro- rative role of these pathways ci s supported by the fact that ar Jiang and Struhl, 1995 ; Lepa	no experiment ted with different n of Ci to different no experiment rolonged phospho- in the MAPK- no experiment nother of the wing age et al., 1995 ; Li et
20 It wou concer extents http:/// 21 Concu MAPH depend http:/// 22 The in duplica al., 199 http://d	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka application that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (tent/full/17/13/3505	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pri- rative role of these pathways ci s supported by the fact that an Jiang and Struhl, 1995 ; Lepa	no experiment ted with different n of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The in duplica al., 199 http://d 23	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA signal, in addition to the pre- ident activation of CREB (Imp www.jneurosci.org/cgi/content pka uplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (tent/full/17/13/3505 inhibit	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pro- rative role of these pathways ci s supported by the fact that an Jiang and Struhl, 1995 ; Lepa ci	no experiment ted with different of Ci to different no experiment rolonged phospho- s in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment
20 It wou concer extents http:/// 21 Concu MAPI depend http:// 22 The in duplica al., 199 http:// 23 The re 1997)	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA is signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka mplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/con hh pressor form is generated by p	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is e catalytic subunit of PKA (tent/full/17/13/3505 inhibit proteolytic cleavage of Ci, w	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways ci s supported by the fact that an Jiang and Struhl, 1995 ; Lepa ci vhich is inhibited by Hh signa	no experiment ted with different n of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al.
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The in duplica al., 199 http://d 23 The re 1997) http://v	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA signal, in addition to the pred dent activation of CREB (Imp www.jneurosci.org/cgi/content pka nplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh pressor form is generated by p www.genesdev.org/cgi/content	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing in catalytic subunit of PKA (tent/full/17/13/3505 inhibit proteolytic cleavage of Ci, w	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pro- rative role of these pathways ci s supported by the fact that and Jiang and Struhl, 1995 ; Lepa ci which is inhibited by Hh signal	no experiment ted with different of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al.
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The im duplica al., 199 http://v 23 The re 1997) http://v 24	hh Id be important, therefore, to r intrations of Hh, and to determin is. www.genesdev.org/cgi/content Creb rrent increases in Ca2+ and cA K signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content plication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh pressor form is generated by p www.genesdev.org/cgi/content ela	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on MP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (tent/full/17/13/3505 inhibit proteolytic cleavage of Ci, w	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pro- rative role of these pathways ci s supported by the fact that and Jiang and Struhl, 1995 ; Lepa ci thich is inhibited by Hh signation ctbp	no experiment ted with different of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al.
20 It wou concer extents http://v 21 Concu MAPH depend http://v 22 The in duplica al., 199 http://v 23 The re 1997) http://v 24 We ne	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka uplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh pressor form is generated by p www.genesdev.org/cgi/content e1a xt asked whether bona fide ac	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (tent/full/17/13/3505 inhibit proteolytic cleavage of Ci, w t/full/17/2/282 interact etylation of E1A disturbed	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways ci s supported by the fact that an Jiang and Struhl, 1995 ; Lepa ci thich is inhibited by Hh signa ctbp its direct interaction with Ctl	no experiment ted with different n of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al. experiment BP.
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The in duplica al., 199 http://v 23 The re 1997) http://v 24 We ne	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content Creb rrent increases in Ca2+ and cA is signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka nplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh pressor form is generated by p www.genesdev.org/cgi/content ela xt asked whether bona fide ac www.jbc.org/cgi/content/full/2	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on AMP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is catalytic subunit of PKA (tent/full/17/13/3505 inhibit proteolytic cleavage of Ci, w t/full/17/2/282 interact etylation of E1A disturbed 277/41/38755	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pro- rative role of these pathways ci s supported by the fact that ar Jiang and Struhl, 1995 ; Lepa ci thich is inhibited by Hh signa ctbp its direct interaction with Ctl	no experiment ted with different of Ci to different no experiment rolonged phospho- in the MAPK- nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al. experiment BP.
20 It wou concer extents http://v 21 Concu MAPI depend http://v 22 The in duplica al., 199 http://v 23 The re 1997) http://v 24 We ne http://v 25	hh Id be important, therefore, to r intrations of Hh, and to determin s. www.genesdev.org/cgi/content creb rrent increases in Ca2+ and cA K signal, in addition to the pre- dent activation of CREB (Imp www.jneurosci.org/cgi/content pka nplication that PKA may there ation mutations inactivates the 95). emboj.oupjournals.org/cgi/content hh pressor form is generated by p www.genesdev.org/cgi/content e1a xt asked whether bona fide ac www.jbc.org/cgi/content/full/2 e1a	induce neasure the levels of Ci pho ine if distinct thresholds of t/full/16/18/2315 depend on MP thus may be particular viously demonstrated coope ey et al., 1998). t/full/21/18/7053 regulate by regulate Ci processing is e catalytic subunit of PKA (tent/full/17/13/3505 inhibit oroteolytic cleavage of Ci, w t/full/17/2/282 interact etylation of E1A disturbed t77/41/38755 bind	ci psphorylation in cells stimula th induce dephosphorylation mapk ly effective in generating a pr rative role of these pathways ci s supported by the fact that an Jiang and Struhl, 1995 ; Lepa ci thich is inhibited by Hh signa ctbp its direct interaction with Ctl ctbp	no experiment ted with different of Ci to different no experiment rolonged phospho- in the MAPK- no experiment nother of the wing age et al., 1995 ; Li et no experiment aling (Aza-Blanc et al. experiment BP.

profiles.			
http://mcb.asm.org/cgi/content/full/2	22/15/5296		_
26 pka	mediate	gsk-3	experiment
In addition to the PI3K-PKB/Akt m directly mediate GSK-3 phosphoryl.	nodule, we and others have ation and inactivation (21,	demonstrated that cAMP-depe 44).	ndent <mark>PKA</mark> can
http://mcb.asm.org/cgi/content/full/2	22/7/2099		
27 erk	phosphorylate	ser	no experiment
Since it is known that the Tyr -phosp phosphorylation by ERK regulates t ERK activity.	bhorylation of STATs is require transactivating potential	uired for DNA binding, where [25], we also looked for any	eas <mark>Ser</mark> - effect of SMase on
http://linkinghub.elsevier.com/retrie	ve/pii/S0014579301029775		
28 p300/cbp	interact	e1a	no experiment
R UPPERT et al. 1993), and p300/(TFIIB (O GRYZKO et al. 1996 ;	CBP interacts with CREB ,	<mark>E1A</mark> , PCAF, c-jun, c-fos, c-N	lyb, MyoD , and
http://www.genetics.org/cgi/content/	/full/148/1/251		
29 akt	phosphorylate	gsk-3beta	no experiment
However, several molecules have be to phosphorylate GSK-3beta on the	en reported to affect GSK- regulatory serine 9 residue	<mark>3beta kinase</mark> activity (34), wi	hile only <mark>Akt</mark> is known
http://www.jbc.org/cgi/content/full/2	274/34/23858		
30 catenin	regulate	cadherin	experiment
-catenin, which mediates the associated adhesion.	ation of <mark>cadherin</mark> with <mark>acti</mark> i	n filaments, is an important re	gulator of <mark>cadherin</mark>
http://www.neuron.org/cgi/content/f	<u>ull/35/1/91</u>		
31 pka	activate	creb	experiment
The N-terminal 1098 amino acids of CREB-mediated transcription to the	CBP were found to contain same extent seen with full-	n sufficient information to enh length CBP.	ance PKA-activated
http://www.jbc.org/cgi/content/full/2	271/45/28138		
32 ctbp	bind	e1a	experiment
CtBP bound to E1A on the plate wa <i>goats</i> , diluted 1:5000; Amersham Ph against goat IgG (1:1000 Santa Crut	s determined by incubation narmacia Biotech) followed z).	with an antibody against GST by horseradish peroxidase-l	C (antibody raised in inked antibody)
http://www.jbc.org/cgi/content/full/2	273/33/20867		
33 hairy	interact	groucho	no experiment
Hairy interacts with a second ubiqu	itous corepressor protein , htent/full/20/9/2246	Groucho (Paroush et al. 1994	4).
34 groucho	bind	hairv	experiment
B-C dCtBP and Grouche can bind	Hairy simultaneously in vi	itro	experiment
http://www.jbc.org/cgi/content/full/2	275/48/37628		
35 e1a	bind	p300/cbp	experiment
E1A binds to Rb and p300/CBP .			
http://www.jbc.org/cgi/content/full/2	274/40/28716		
36 cbp	interact	creb	no experiment
This region of CBP is known to dire (52).	ectly interact with multiple t	ranscription factors, such as	CREB and c-Myb
http://www.jbc.org/cgi/content/full/2	274/12/8143		
37 pka	induce	creb	no experiment
Model of PKA -induced CREB phose	sphorylation mediated by th	e PKC/ERK-dependent pathy	vay.
http://www.jbc.org/cgi/content/full/2	276/15/11487		
38 e1a	bind	ctbp	no experiment
The Lys-239 acetylation site is locat	ed adjacent to the sequence	e Pro-Leu-Asp-Leu-Ser (PLD	LS), which has been

shown	to be responsible for the bind	ing of E1A to CtBP (29).							
http://v	www.pnas.org/cgi/content/full	/97/26/14323							
39	akt	phosphorylate	gsk-3	no experiment					
Regulation of p21Cip1 by GSK-3 In various cell types, AKT phosphorylates and inhibits the kinase activity of GSK-3 (11), which regulates the proteasomal degradation of cyclin D (13).									
http://v	www.jbc.org/cgi/content/full/2	277/12/9684							
40	jnk	phosphorylate	p53	experiment					
(C) Laminar shear stress of 12 dynes/cm2 increases p53 phosphorylation by JNK.									
http://www.pnas.org/cgi/content/full/97/17/9385									
41	pka	regulate	creb	experiment					
Indeed to 3 h) CREE	l, CREB remained heavily pho , underscoring the potential in B by PKA (Fig. 5C and D).	osphorylated throughout the aportance of chromatin loca	e attenuation phase in A-CR lization for stimulus-appropri	EB-expressing cells (1 ate regulation of					
<u>http://</u> 1	mcb.asm.org/cgi/content/full/2	20/5/1596							
42	e1a	inhibit	p53	experiment					
Inhibit <mark>E1A</mark> .	tion of NQO1 activity induces	Mdm-2-independent degra	adation of $p53$ that is inhibited	d by LT, p14ARF, and					
http://v	www.pnas.org/cgi/content/full	/99/20/13125							
43	erk	lead to	creb	experiment					
Althou provid of cell	igh the MAPK pathway has be e evidence here that persistent growth.	een shown to function in th ERK activation can also lo 278/13/11138	e stimulation of cellular prolif ead to suppression of CREB a	feration (37), we activity and inhibition					
	nlze	inhihit	orly)	no ovnorimont					
Activa 1996 ·	рка ited <mark>PKA</mark> then inhibits <mark>ERK2</mark> , Aubry et al. 1997)	possibly acting via the sma	all GTP-binding protein RA	S (Knetsch et al.,					
<u>http://v</u>	www.molbiolcell.org/cgi/conte	ent/full/9/12/3521							
45	akt	phosphorylate	creb	no experiment					
The re that C (Shieh	levance of the phosphorylation REB regulates the expression et al. 1998 ; Tao et al. 1998).	n of CREB by Akt to apop genes critical for survival	tosis is also still unclear, althous such as the gene encoding the	ough there is evidence cytokine BDNF					
http://	www.genesdev.org/cgi/conten	t/full/13/22/2905							
46	pkc	phosphorylate	gsk-3	no experiment					
Instea <mark>GSK-</mark>	d of assessing site-specific pho 3 by PKC with [gamma-32]A	osphorylation of <mark>GSK-3</mark> , Go TP.	bode et al. examined the gener	ral phosphorylation of					
<u>http://1</u>	mcb.asm.org/cgi/content/full/2	<u>2/1/2099</u>	-						
47	gsk-3	phosphorylate	creb	no experiment					
GSK- CREE	boost of the second sec	at Ser-129) is reported to ha liated activation of PEPCK	ave a stimulatory effect on CF Cexpression (48).	EB activity (42), and					
<u>http://e</u>	diabetes.diabetesjournals.org/c	g1/content/full/50/5/937							
48	pkc	activate	erk	no experiment					
The fo ERK	ormer is more likely because R activation (54), whereas Gq/1	hoA, which is activated by 1-mediated PKC activation	G12/13, is not considered to a can activate ERK by several	be directly linked to I mechanisms (55).					
nup://	www.jbc.org/cgi/content/full/2		1477	•					
49	teashirt	bind	armadillo	experiment					
GALL of Win	ET, A., A. ERKNER, B. CHAngless signaling in <i>Drosophila</i>	ARROUX, L. FASANO, an by <mark>Teashirt</mark> binding to <mark>Ar</mark> (611/155/4/1725	d S. KERRIDGE, 1998 Trunk <mark>madillo</mark> .	s-specific modulation					
nttp://	www.genetics.org/cgi/content/	1011/155/4/1/25		•					
50	tafii250	bind	tbp	experiment					
(B) Le	evels of in vitro-expressed TA	FII250 bound to GST, TBI	e, or Rb, as detected by Weste	ern analysis					

(monoclonal anti-hTAFII250 antibody) of nonradioactive, duplicate kinase reactions performed concurrently with

those s	hown in panel A.			
<u>http://r</u>	ncb.asm.org/cgi/content/full/1	9/1/846		
51	creb	depend on	pka	experiment
These depend transcr	data suggest a revised model f lent phosphorylation of CREI iption.	for cAMP regulation of CR and Rap1-ERK stimulation	EB in which the co-ordinate a on of a downstream target are	action of both PKA- e required for full
http://v	www.jbc.org/cgi/content/full/2	275/44/34433		
52	pkc	regulate	mapk	no experiment
Althou wall in kinases	gh it is known that the PKC -r tegrity in the absence of an ex s in the mating and IG pathwa	regulated MAPK cascade h sternal stimulus (9), it is cu sys perform specialized fund (96/22/12679	as a basal vegetative function rrently thought that Stell and ctions only in response to their	that maintains cell d the downstream ir respective stimuli.
53	chn	interact	creb	experiment
CBP P interac	Potentiates Basal and Inducible tions with AP-1, C/EBP, CR	e IL-6 Gene Expression V EB, or NF-kappaB in basa	We further analyzed the relati l or p65-driven IL-6 gene ex	ve contribution of CBP pression.
http://v	www.jbc.org/cgi/content/full/2	274/45/32091		
54	cbp	interact	tbp	no experiment
Howev 69).	ver, CBP interacts stably with	TBP in coimmunoprecipita	ation reactions and in vitro bir	nding reactions (1, 63,
http://r	ncb.asm.org/cgi/content/full/1	<u>9/3/161/</u>	4	•
55	p53	interact	sp1	no experiment
It is po access	to the promoter (7) .	Spl and prevents other tran	scriptional activators, such as	s p300 , impeding their
http://r	nmbr.asm.org/cgi/content/full	<u>/66/3/407</u>		•
56	akt	inactivate	gsk-3	no experiment
GSK-3 enhanc	is a kinase phosphorylated at	nd inactivated by Akt/PKB	(7) that phosphorylates nucl	ear cyclin D1,
http://r	nch asm org/cgi/content/full/?	23/17/6139		
57	erk	activate	ink	experiment
B don	inant negative SEK-1 prever	ts the activation of JNK ac	tivity by ERK	
http://v	www.jbc.org/cgi/content/full/2	273/41/26722	······	
58	calmodulin	bind	fusion proteins	experiment
As a co	ontrol, we show <mark>calmodulin</mark> a	lso binds specifically to fus	ion proteins that contain IQ	sites (Fig. 8 C).
http://v	www.jcb.org/cgi/content/full/1	42/3/711		
59	dfd	regulate	pb	experiment
Regula	tion of pb expression by Dfd	and Scr		•
<u> http://l</u>	inkinghub.elsevier.com/retriev	ve/pii/S092547730100301X	<u> </u>	
60	ck2	phosphorylate	p53	experiment
(C) <mark>C</mark>	K2 phosphorylates Thr155 of	<mark>p53</mark> .		
http://e	emboj.oupjournals.org/cgi/con	tent/full/22/6/1302		
61	pkc	activate	erk	experiment
To det	ermine whether PKC activate	s <mark>Erk</mark> through <mark>Ras</mark> or by di	rectly activating Raf and inde	ependently of Ras, we
blocke	d Ras activation by either a do	ominant negative mutant F	kas (N17-Ras) or a farnesyl t	transferase inhibitor.
<u>nup://v</u>	www.jbc.org/cgi/content/full/2	<u>:</u>	au-l	
02	сор	interact	creb	experiment
In the operation of the second	case of UKEB , UBP interacts horylated KID domain, and to porvlation-independent manner	with CREB via two region the C/H2 domain of CBP i r	is; the KIX domain binds to the interacts with the b-ZIP region	n of CREB in a
http://x	www.ibc.org/cgi/content/full/2	273/44/29098		
63	ahd_a	renress	uby	no experiment

Since Abd-A also represses Ubx in the posterior midgut (Bienz and Tremml, 1988), it is conceivable that Abd-A could be this Tsh-recruiting partner.

<u>http://</u>	emboj.oupjournals.org/cgi/con	tent/full/20/1/137		
64	jnk	phosphorylate	p53	experiment
deltaN irradia	IEKK1 effect on JNK phosphoten photon photon photon (Fig. 2 A).	orylation of <mark>p53</mark> was compa	arable to that seen with the kir	nase purified from UV-
http://	www.pnas.org/cgi/content/full	<u>/95/18/10541</u>		
65	dfd	regulate	pb	no experiment
It has embry	been shown that <mark>Dfd</mark> and <mark>Scr</mark> progenesis (Rusch and Kaufman	positively regulate pb and a n, 2000).	re required for its correct dep	loyment during
<u>http://</u>	dev.biologists.org/cgi/content/	full/128/14/2803		
66	gsk-3beta	depend on	akt	experiment
Under <mark>GSK-</mark>	physiological conditions, insu 3beta at Ser9.	ilin inhibits glycogenolysis	by promoting the Akt-depend	lent phosphorylation of
<u>http://</u>	www.sciencemag.org/cgi/cont	ent/full/300/5625/1574		
67	dctbp	interact	ttk69	experiment
We fo repres	und that deletion of the putative sion, suggesting that it is dispe	ve dCtBP-interacting region ensable.	1 of <mark>Ttk69</mark> (Wen et al., 2000)	has little effect on
<u>http://</u>	linkinghub.elsevier.com/retriev	ve/pii/S0925477302001831		
68	mapk	phosphorylate	mbp	experiment
A pos phosp	itive control sample contained horylation of MBP by the activ	1.0 mug of <mark>MBP</mark> as substravated MAPK.	ite and is shown in lane 1, den	nonstrating
<u>http://</u>	www.jbc.org/cgi/content/full/2	275/48/38032		
69	cbp	bind	e1a	no experiment
Cotrar transa	nsfection of an expression vect ctivation or on the inhibition o	or for <mark>CBP</mark> , which can also f transactivation by <mark>E1A</mark> (F	bind E1A, did not have any e ig. 4 B).	effect on DBP
<u>http://</u>	www.jbc.org/cgi/content/full/2	274/25/17643		
70	hth	interact	exd	no experiment
Rieck	hof et al. (1997) showed that H	ITH and MEIS1 can direct	ly interact with EXD in vitro.	
<u>http://</u>	linkinghub.elsevier.com/retriev	ve/pii/S0925477399003160		
71	cbp	interact	p53	no experiment
CBP i into G	is known to interact with $p53$ to 1 (22, 35).	o potentiate the transcriptio	nal activation functions of p5.	3 in inhibiting entry
http://	mcb.asm.org/cgi/content/full/1	9/4/2515		
72	p53	interact	spl	experiment
These region	data indicate that the physical that contains the DNA bindin	interactions of p53 with S Ig domain of Sp1 .	are possibly mediated by th	e 610-702 amino acid
<u>http://</u>	www.1bc.org/cg1/content/full/2	<u>276/31/29116</u>	-	
73	creb	depend on	pka	no experiment
PKA- regula	dependent phosphorylation of tion by the cAMP pathway (4	CREB on Ser133 is a critic 7).	al step involved in the transcr	riptional gene
http://	www.jbc.org/cgi/content/full/2	<u>275/43/33379</u>		
74	pkc	regulate	mapk	experiment
Regula regula adeno	ation of MAPK Activity by Pl te gene expression during kera virus encoding PKCdelta or P	KCdelta and PKC To 1d tinocyte differentiation, we PKC .	entify the mechanism whereby transfected keratinocytes wit	h empty <i>adenovirus</i> or
<u>http://</u>	www.jbc.org/cgi/content/full/2	277/35/31753		
75	mapk	phosphorylate	mbp	no experiment

However, although both MAPK and DOA phosphorylate intact MBP, three peptides phosphorylated by MAPK (the sequences from MBP, tyrosine hydroxylase, and the epidermal growth factor receptor phosphorylated by MAPK) were not phosphorylated by DOA, suggesting that the phosphorylation site of DOA is different from that

of <mark>MA</mark>	APK.							
http://	www.jbc.org/cgi/content/full/2	271/44/27299						
76	akt	inhibit	gsk-3	no experiment				
Inhibi	tion of <mark>GSK-3</mark> by <mark>Akt</mark> inhibits	apoptosis and promotes ce	ll survival (22, 26).					
http://	www.jbc.org/cgi/content/full/2	277/23/20927						
77	tfiid	bind	tbp	no experiment				
A logi TAFI	cal explanation for these obser 1145p, and that the histone-lik	vations is that additional T te TAFIIs are more importation	FIID subunits bind to TBP in ant for TFIID structure.	the absence of				
http://	www.jbc.org/cgi/content/full/2	275/23/17391						
78 proteasome degrade p53 no exp								
It has cytopl	been shown that Hdm2-media asm (17, 18).	ted p53 degradation occurs	through a proteasome-depen	ident pathway in the				
http://	www.pnas.org/cgi/content/full	<u>/96/6/3077</u>						
79	sp1	interact	target dna	experiment				
In sun sequer vitro c	n, these results demonstrate that nee, although there was no clear conditions.	tt SNURF and Sp1 may int ar cooperative effect by SN	eract concomitantly with the s URF on the DNA binding of	same target DNA Sp1 under these in				
<u>nup.//</u>		.10/20/23033	a bar	- 4				
80	creb	Interact	сор	experiment				
D, pre	sence of two domains in CRE. www.jbc.org/cgi/content/full/2	B that interact with \bigcirc BP . 73/44/29098						
81	akt	inhibit	gsk-3	no experiment				
There phosp growt	are reciprocal relationships be horylation and overexpression h factor (BDNF) (Mai et al., 2	tween these pathways, as A of GSK-3 prevents CREB 2002).	kt activation inhibits GSK-3 phosphorylation induced by b	through serine (9) prain-derived nerve				
<u>http://</u>	www.neuron.org/cgi/content/fi	<u>ull/38/2/157</u>						
82	pka	phosphorylate	creb	experiment				
The te	mporal association between ki horylate CREB in the <i>hippoca</i>	nase activity and CREB pl mpus and the cortex, respe-	nosphorylation suggests that P ctively, during seizure activity	P <mark>KA</mark> and CaMK y.				
<u>http://</u>	www.jbc.org/cgi/content/full/2	<u>.71/24/14214</u>						
83	akt	inactivate	gsk-3	no experiment				
GSK- inactiv	3 is a direct substrate for PKB vates GSK-3 in vivo (43, 44).	Akt in vitro (42), and the	e is evidence that PKB/Akt p	hosphorylates and				
http://	www.jbc.org/cgi/content/full/2	274/40/28279						
84	hairy	interact	dctbp	no experiment				
<mark>dCtB</mark> 1998) doma	P was identified in interaction in These repressor proteins int ins located near their C-termin	studies with Knirps, Snail, eract with dCtBP through al ends.	and <mark>Hairy</mark> (Nibu et al., 1998) CtBP binding motifs located v	b ; Poortinga et al., within the repressor				
<u>http://</u>	www.molecule.org/cgi/content	t/full/9/2/213						
85	pkc	regulate	mapk	experiment				
Furthe plasm	ermore, the regulation of the M a membrane through their asso	APK cascade by both Ras ciation with c-Raf-1.	and PKC is intimately linked	, converging at the				
http://	www.jbc.org/cgi/content/full/2	276/31/29079						
86	ela	interact	dctbp	experiment				
It appo NIH 3	ears that interaction of E1A wi T3 cells induced weak transcri	th dCtBP may expose this ptional activation.	activation function since coex	pression of E1A in				
http://	www.molecule.org/cgi/content	t/full/9/2/213						
87	mapk	phosphorylate	mbp	experiment				
To vis	ualize the phosphorylation of	MBP by active MAPK , 3x	kinase buffer containing 50 <i>n</i>	<i>uM</i> [gamma-32]ATP				

88	ci	depend on	hh	experiment						
More i accum	More intriguingly, posterior degradation of <mark>Ci</mark> depends on <mark>Hh</mark> signaling, as removal of Smo results in the accumulation of high levels of Ci155.									
http://v	www.genesdev.org/cgi/conten	t/full/16/18/2315								
89	ctbp	bind	e1a	no experiment						
Moreo promo	over, the CtBP-binding sequen ter by fusion with a heterologo	ce of <mark>E1A</mark> is a potent repre ous DNA-binding domain	ssor of RNA transcription wh (23).	en targeted to a						
http://v	www.jbc.org/cgi/content/full/2	273/39/25388								
90 pkc phosphorylate gsk-3 experime										
Phospl	horylation of <mark>GSK-3</mark> by <mark>PKC</mark>									
http://l	inkinghub.elsevier.com/retriev	ve/pii/S0014579300012345	_							
91	cbp	enhance	creb	no experiment						
Overez antibo 1994)	xpression of CBP enhances C dies that block formation of a	REB -mediated transcription CREB-CBP complex inhib	n (Kwok et al. 1994), and mi bits CREB-mediated transcrip	croinjection of tion (Arias et al.						
<u>nttp://v</u>	www.neuron.org/cgi/content/1	<u>un/22/4/799</u>	6							
92 Colu	caimodulin	Dina Durifical factor	Tusion proteins	experiment						
Calmo NumA	deltaN165 were resolved usin	g 10% SDS-PAGE.	ceins MBP-NumAdeltaC218 a	and MBP-						
<u>nttp://v</u>	www.jbc.org/cgi/content/full/2	11/22/19/35	(⁰ ** 1							
93		bind	tina	no experiment						
bindin indicat	g of TBP to the intact TFIID ting that TBP makes contacts	complex is resistant to salt with additional TAF subun	concentrations above 0. concentrations above 1 M pot its.	tassium acetate (6,7),						
http://v	www.jbc.org/cgi/content/full/2	<u>.75/23/17391</u>								
94	akt	inhibit	gsk-3beta	no experiment						
Intrigu <mark>GSK-</mark>	iingly, the effector of PI3K sig 3beta (Cross et al., 1995 ; Pap	gnaling is protein kinase B (and Cooper, 1998).	(or <mark>Akt</mark>), which is known to in	nhibit the activity of						
<u>http://v</u>	www.jneurosci.org/cgi/content	t/full/23/22/8125								
95	tsh	stimulate	pka	no experiment						
TSH thereby	timulates not only cAMP accu y ensuring that cAMP elevatio	Imulation but also PKA-de n is transient (39).	pendent increases in phosphore	diesterase activity,						
<u>http://1</u>	mcb.asm.org/cgi/content/full/2	<u>1/6/1921</u>								
96	pka	regulate	creb	experiment						
[In new http://v	w window] PKA regulates the www.molecule.org/cgi/content	activity of another widely a t/full/1/5/661	studied inducible transcription	on factor, <mark>CREB</mark> .						
97	cbp	activate	creb	no experiment						
Compa related identit CREB	arison of the amino acid seque I protein p300 , which like CB y in this domain (16, 35), and B -mediated transcription.	nce found within amino aci P activates CREB-mediate thus this same region of p3	ids 227-460 of CBP to the sar ed transcription, shows that the 300 is also likely to participate	ne region of the CBP ese proteins share 68% es in activation of						
nttp://v	www.jbc.org/cgi/content/full/2	./1/45/28138		• •						
98	pka	phosphorylate	gsk-3	experiment						
To fur <mark>PKA</mark> ,	ther examine our hypothesis th we examined GSK-3 phospho	hat Wnt pathway mediates brylation and inactivation by	the activation of proglucagon y <mark>PKA</mark> in the intestinal endoci	gene transcription by rine cell lines.						
nttp://v	www.jbc.org/cgi/content/full/2	18/2/1380	12 1 ·							
99 LDD	lab proteins		lim domains	experiment						
LDB [proteins bind <mark>El Ni domains</mark> o	1 LIVI-HD (and nuclear LI	ini-only) proteins.							
100	ho	intorra et	guov sha							

Hairy and Runt proteins interact with Groucho through their C-terminal WRPW or WRPY motifs (1, 12), whereas Dorsal binds Groucho through its Rel homology domain (52) and Tcf interacts with Groucho through an HMG domain that also mediates its interaction with CBP (46). http://mcb.asm.org/cgi/content/full/21/17/5935 101 inhibit mae yan experiment However, because MAE inhibits YAN-mediated transcriptional repression, we expect that, in the absence of signaling, not all **YAN** will be bound to **MAE**. http://dev.biologists.org/cgi/content/full/130/5/845 102 ctbp interact e1a no experiment

CtBP interacts with the **E1A**-related sequence PXDLS (Poortinga et al., 1998), whereas **Groucho** binds to the C-terminal WRPW sequence.

http://emboj.oupjournals.org/cgi/content/full/18/12/3392

103crebinteractcbpno experimentShaywitz et al. (15) demonstrated that the magnitude of transcription activated by CREB is dependent on the
strength of the interaction of CREB with CBP.CREBCREB

http://www.jbc.org/cgi/content/full/276/44/40721

104	pka	lead to	creb	experiment	

In conclusion, we have found that intracellular Ca2+ release induced by **PKA** leads to **CREB** phosphorylation via a **PKC**- and **ERK**-dependent mechanism, most likely involving **Rap1** activation.

http://www.jbc.org/cgi/content/full/276/15/11487

105	hairy	interact	groucho	experiment				
The relative values of hete collecteridese estivity for each interaction of Heim with dCtBD or Creater (a, a)								

The relative values of **beta-galactosidase** activity for each interaction of **Hairy** with **dCtBP** or **Groucho** (as a control) are listed on the right.

http://emboj.oupjournals.org/cgi/content/full/17/7/2067

106 p53		interact	cbp	no experiment	
Maraa	war both CDED and n52 aan	interact directly with CDD	and phase hard and CDEP a	adjotant rearry itmant of	

Moreover, both CREB and p53 can interact directly with CBP, and phosphorylated CREB mediates recruitment of CBP to p53-responsive promoters through direct interaction with p53 (13).

http://www.jbc.org/cgi/content/full/278/13/11138

107	07 cbp		NOT activate			creb			experiment			
	• • • • • • • •		0 11 1		1	, ·		11			<u></u>	

This is consistent with the idea that the full-length **CBP** molecule is not normally a potent activator of basal **CREB** activity.

http://www.jbc.org/cgi/content/full/271/30/17746

108	gsk-3	phosphorylate	catenin	experiment		
Thus, v	Thus, we investigated whether this kinase also serves as a priming kinase for -catenin phosphorylation by GSK-3.					
http://l	inkinghub.elsevier.com/retriev	ve/pii/S0006-291X(02)0048	35-0			

109 pka phosphorylate fusion proteins experiment

Fusion proteins were phosphorylated in vitro by **PKA** and/or **PKC**, resolved by **SDS**-PAGE, and visualized by autoradiography.

http://www.jbc.org/cgi/content/full/272/8/5157

110	scr	activate	pb	no experiment
In the	Duaganhila amhrua Dfd and	San activate the expression	of nh in the maxillary and lab	vial accoments

In the *Drosophila* embryo, **Dfd** and **Scr** activate the expression of **pb** in the maxillary and labial segments, respectively

http://www.sciencemag.org/cgi/content/full/298/5591/97

111	cbp	bind	creb	experiment

Since **CBP** binds only the phosphorylated form of **CREB**, and the synergism we have observed between the proteins bound at the **LSR** and **CREB** is observed only under **PKA**-stimulating condition and can be competed by overexpression of KID, it is interesting to speculate that **CBP** may be involved in this synergistic response. http://www.jbc.org/cgi/content/full/270/14/8225

112	n53	interact	cbp	no experiment
	500	Internet	en p	no enperimente

In addition, a double-point mutation in the activation domain 1 or the activation domain 2, which abolishes the transcriptional activity of **p53**, also abolishes the interaction of **p53** with **CBP** in vitro (21, 23).

http://w	ww.jbc.org/cgi/content/full/2	<u>78/19/17557</u>				
113	paxillin	NOT bind	gst	experiment		
<mark>Paxilliı</mark> signalir	n failed to bind GST alone or ng molecules.	the SH2 domain of Grb2	that belongs to the family of S	SH2/SH3-containing		
http://w	ww.jcb.org/cgi/content/full/1	<u>48/5/957</u>				
114	creb	depend on	erk	no experiment		
Further CA1 of (Robers	Furthermore, it has been shown recently that both PKA and PKC are upstream regulators of ERK/MAPK in area CA1 of the <i>hippocampus</i> and that PKA-mediated CREB phosphorylation depends on ERK/MAPK activation (Roberson et al., 1999).					
http://w	ww.jneurosci.org/cgi/content	/full/20/21/8177				
115	pka	mediate	creb	experiment		
The present findings demonstrate that substitution of a negatively charged residue, Asp, at Ser of the transcriptional activation domain of CREB greatly reduces PKA-mediated activation of CREB.						
http://w	ww.jbc.org/cgi/content/full/2	<u>70/13/7041</u>				
116	gsk-3	phosphorylate	catenin	no experiment		
Withou degrade	t stimulation by Wnt ligand , ed by proteasome .	free - <mark>catenin</mark> is phosphory	lated by GSK-3, then ubiquit	inated and rapidly		
<u>http://li</u>	nkinghub.elsevier.com/retriev	<u>/e/pii/S0925443900000284</u>				
117	erk2	phosphorylate	mbp	experiment		
The act ERK2,	ivity of the immunoprecipitat ERK6 , p38 and ERK5 , and	ed MAPKs was determine GST-ATF2(96) for SAPK	d by measuring the phosphory 4.	lation of <mark>MBP</mark> for		
http://w	ww.jbc.org/cgi/content/full/2	<u>74/47/33287</u>				
118	mapk	phosphorylate	cbp	no experiment		
Supporting this hypothesis, it has been reported that the C-terminal transactivation domain of CBP was phosphorylated by MAPK in vitro (49), and we now demonstrate that the C-terminal transactivation domain of p300 can be phosphorylated by MAPK in a similar manner (Fig. 4 E).						
phospho p300 ca	an be phosphorylated by MAI	24 9), and we now demonstra 2 K in a similar manner (Fig 78/16/14012	g. 4 E).	ctivation domain of		
phospho p300 ca http://w	an be phosphorylated by MAPK in vitro (www.jbc.org/cgi/content/full/2	⁴⁹), and we now demonstra ¹ K in a similar manner (Fig <u>78/16/14013</u> <u>phosphorylata</u>	g. 4 E).	ctivation domain of		
p10sph p300 ca http://w 119 These c http://w	an be phosphorylated by MAPK in vitro (an be phosphorylated by MAI www.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re www.jbc.org/cgi/content/full/2	49), and we now demonstra PK in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337	fusion proteins hosphorylation of the fusion	experiment		
p105ph p300 ca http://w 119 These c http://w 120	an be phosphorylated by MAPK in vitto (an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re ww.jbc.org/cgi/content/full/2 e1a	49), and we now demonstra PK in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337 bind	the that the C-terminal transaction of the fusion proteins the fusion of	experiment experiment proteins		
phospho p300 ca http://w 119 These c http://w 120 Binding	an be phosphorylated by MAPK in vitro (A an be phosphorylated by MAI www.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re www.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran	49), and we now demonstrapy (and we now demonstrapy), and we now demonstrapy (and we have a similar manner (Fig 78/16/14013) (and the second secon	te that the C-terminal transaction of the fusion proteins hosphorylation of the fusion	experiment proteins. experiment		
phosphi p300 ca http://w 119 These c http://w 120 Binding http://w	an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re ww.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran	49), and we now demonstra ^{PK} in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337 bind scriptional activation medi 73/44/29098	the that the C-terminal transaction of the fusion proteins hosphorylation of the fusion of the fusio	experiment proteins. experiment		
http://w 119 These c http://w 120 Binding http://w 121	an be phosphorylated by MAPK in vitro (4 an be phosphorylated by MAI www.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re www.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran www.jbc.org/cgi/content/full/2 proteasome	49), and we now demonstra ^{PK} in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337 bind scriptional activation medi 73/44/29098 degrade	te that the C-terminal transaction g. 4 E). fusion proteins hosphorylation of the fusion proteins cbp ated by CBP.	experiment proteins. experiment no experiment		
nosphi p300 cz http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteas	an be phosphorylated by MAPK in vitro (4 an be phosphorylated by MAI www.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re www.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran www.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deacc inate the same lysine residues some machinery (27, 30, 31), otained.	49), and we now demonstrapy (and we now demonstrapy) (and we now demonstrapy) (and we now demonstrapy) (blue) (and the serial science) (and the serial science) (and the serial science) (blue) (bl	the that the C-terminal transaction g. 4 E). fusion proteins hosphorylation of the fusion protection cbp ated by CBP. p53 lysines residues would allow by to p53 degradation through th p53 ubiquitination to its degr	experiment proteins. experiment no experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet		
http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteas been oc http://w	an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re- ww.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran ww.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deace- inate the same lysine residues some machinery (27, 30, 31), otained.	49), and we now demonstra ^{PK} in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337 bind scriptional activation medi 73/44/29098 degrade etylation at the C-terminal of p53 (52), thus leading although evidence linking 77/34/30838	the that the C-terminal transaction of the fusion proteins hosphorylation of the fusion of the fusio	experiment proteins. experiment no experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet		
http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti protease been oc http://w 122	an be phosphorylated by MAPK in vitto (an be phosphorylated by MAI www.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re- ww.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran www.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deace inate the same lysine residues some machinery (27, 30, 31), otained. www.jbc.org/cgi/content/full/2 e1a	49), and we now demonstra ¹ K in a similar manner (Fig 78/16/14013 phosphorylate esults obtained with PKA p 75/8/5337 bind scriptional activation medi 73/44/29098 degrade etylation at the C-terminal of p53 (52), thus leading although evidence linking 77/34/30838 interact	the that the C-terminal transaction g. 4 E). fusion proteins hosphorylation of the fusion protection cbp ated by CBP. p53 lysines residues would allow protection p53 degradation through the p53 ubiquitination to its degrees cbp	experiment proteins. experiment experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet experiment		
nosphi p300 cz http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteas been ob http://w 122 Regard http://w	an be phosphorylated by MAPK in vitro (4 an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re ww.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran ww.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deacc inate the same lysine residues some machinery (27, 30, 31), otained. ww.jbc.org/cgi/content/full/2 e1a less of p53, E1A can interact ww.jbc.org/cgi/content/full/2	49), and we now demonstraces (19), and we now demonstraces (19), and we now demonstraces (19), and (19)	the that the C-terminal transaction is a set of the fusion proteins is a set of the fusion of the fu	experiment proteins. experiment experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet experiment ptional activation.		
nosphi p300 ca http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteas been ob http://w 122 Regard http://w 123	an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re- ww.jbc.org/cgi/content/full/2 e1a g of E1A to CBP inhibits tran ww.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deace inate the same lysine residues some machinery (27, 30, 31), otained. ww.jbc.org/cgi/content/full/2 e1a less of p53, E1A can interact ww.jbc.org/cgi/content/full/2 pka	49), and we now demonstract ¹ K in a similar manner (Fig. 78/16/14013 phosphorylate esults obtained with PKA pr. 75/8/5337 bind scriptional activation medir 73/44/29098 degrade etylation at the C-terminal of p53 (52), thus leading athough evidence linking 77/34/30838 interact with CBP and/or with X, w. 73/28/17303 activate	the that the C-terminal transaction g. 4 E). fusion proteins hosphorylation of the fusion protection cbp ated by CBP. p53 lysines residues would allow 1 to p53 degradation through th p53 ubiquitination to its degr cbp which leads to modest transcription creb	experiment proteins. experiment experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet experiment ptional activation.		
nosphi p300 cz http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteas been ot http://w 122 Regard http://w 123 Since P binding comple	an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re- ww.jbc.org/cgi/content/full/2 ela g of E1A to CBP inhibits tran ww.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deace inate the same lysine residues some machinery (27, 30, 31), otained. ww.jbc.org/cgi/content/full/2 ela less of p53, E1A can interact ww.jbc.org/cgi/content/full/2 pka CA phosphorylation of CRE g protein and p300, we sugger mentary interactions with these orylation-dependent conformation	49), and we now demonstration of the phosphorylate (14) (14) (14) (14) (14) (14) (14) (14)	te that the C-terminal transaction of the fusion proteins hosphorylation of the fusion of the previous would allow of p53 degradation through th p53 ubiquitination to its degradation through the previous of the transcriptional co of CREB occurs by the produing the previously proposed of DNA binding affinity.	experiment proteins. experiment experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet experiment ptional activation. experiment -activators CREB- action of specific, mechanisms of a		
nosphi p300 cz http://w 119 These c http://w 120 Binding http://w 121 Inhibiti ubiquiti proteast been ot http://w 122 Regard http://w 123 Since P binding comple phosphi	an be phosphorylated by MAI ww.jbc.org/cgi/content/full/2 pka lata confirm the sequencing re- ww.jbc.org/cgi/content/full/2 ela g of E1A to CBP inhibits tran ww.jbc.org/cgi/content/full/2 proteasome on of p53 acetylation or deace- inate the same lysine residues some machinery (27, 30, 31), otained. ww.jbc.org/cgi/content/full/2 ela less of p53, E1A can interact ww.jbc.org/cgi/content/full/2 pka VAPK phosphorylation of CRE g protein and p300, we sugger mentary interactions with the orylation-dependent conformation ww.jbc.org/cgi/content/full/2	49), and we now demonstraces in a similar manner (Fig. 78/16/14013) Phosphorylate esults obtained with PKA pressults obtained with PKA pressults obtained with PKA pressults obtained activation media 73/44/29098 degrade etylation at the C-terminal of p53 (52), thus leading although evidence linking 77/34/30838 interact with CBP and/or with X, wrest 73/28/17303 activate B results in its specific bimest that the PKA activation se proteins, rather than throational change or increased 71/23/13716	the that the C-terminal transaction is a set of the fusion proteins is a set of the fusion of the fu	experiment proteins. experiment experiment MDM2 to efficiently e ubiquitin-mediated adation has not yet experiment ptional activation. experiment -activators CREB- action of specific, nechanisms of a		

Our data demonstrate that phosphorylation of **CREB** by **PKA** does not alter the DNA binding affinity for either the canonical SSCRE or the non-canonical TATCRE.

http://v	www.jbc.org/cgi/content/full/2	271/23/13716		
125	nuclear protein	bind	sp1	experiment
A supe the mE	ershift assay was performed to EPCR gene could be shifted by	test whether the binding of an SP1 antibody (Fig. 5).	nuclear protein to the <mark>SP1</mark> s	ite (GGGAGGGG) of
http://v	www.jbc.org/cgi/content/full/2	<u>275/17/12481</u>		
126	dctbp	bind	hairy	experiment
B-C, <mark>d</mark>	ICtBP and Groucho can bind	Hairy simultaneously in vi	tro.	
http://v	www.jbc.org/cgi/content/full/2	275/48/37628		
127	akt	inactivate	gsk-3	no experiment
Additi dephos	onally, <mark>Akt</mark> phosphorylates an sphorylated, active GS in the c	d inactivates GSK-3 (21), cell (4, 10).	which may increase the amou	nt of
http://v	www.jbc.org/cgi/content/full/2	.74/39/27497		
128	pka	phosphorylate	gsk-3beta	experiment
One is that Al inhibit	that PKA directly phosphoryl KAP220 and PKA inhibit PP ing the GSK-3beta activity.	ates and inhibits GSK-3be 1 co-operatively, thereby er	ta efficiently in the AKAP22 hancing the phosphorylation	0 complex; the other is of <mark>GSK-3beta</mark> and
http://v	www.jbc.org/cgi/content/full/2	<u>.77/40/36955</u>	• •	•
129	ркс	mediate	jnk	experiment
Interes activat http://v	stingly, on coexpression of PK tion could be observed (Fig. 6) www.jbc.org/cgi/content/full/2	Cmu with constitutively ac , indicating a negative feed <u>777/8/6490</u>	tive PKC A/E , a reduction of back regulation of PKCmu or	PKC -mediated JNK PKC kinase activity.
130	ctbp	bind	e1a	no experiment
To det bindin E1a oi	ermine specifically whether th g activity, we mutated residue ncoprotein (20).	e P- X-D-L-R motif (residues DL to AS, which has bee	ues 23-27) of MITR was resp n shown previously to abolish	onsible for all <mark>CtBP</mark> - a <mark>CtBP</mark> binding to the
http://v	www.jbc.org/cgi/content/full/2	.76/1/35		
131	ctbp	interact	ela	no experiment
Althou presun preven	igh the H315Q mutation was probably other repressor protein ably other repressor protein at the interaction of GST-E1a	broposed (16) to prevent th s containing CtBP-recruitn with CtBP in vitro (Fig. 4 b	e interaction of CtBP with the nent motifs, we found that this o).	e E1a C terminus and s mutation did not
<u>nup://v</u>	www.pnas.org/cgi/content/ruii	100/8/4368	11	•
132	Cl	mediate	nn	experiment
I hese	findings suggest that sr expres	$\frac{1}{6}$ $\frac{11}{6}$	is activated by Ci-mediated I	th signaling.
<u>nup://v</u>	www.molecule.org/cgl/content	/Tull/6/1/203		•
				experiment
C, the	iuii-length isoform of Paxo bi	has to both the PD and the	HD but not to the TAD in vit	ro.
<u>nup.///</u>	www.jbc.org/cgi/content/101/2	••••••		
134			gsk-Sbeta	no experiment
Recent and at	rial natriuretic factor express	sion (29, 47).	Akt induces cardiomyocyte	hypertrophy in vitro
<u>125</u>	www.jbc.org/cgi/content/101/2	.11/25/22890		-
135 T1		repress		experiment
1 nus, correla	UDX FKE -associated nucleoso ates with PC binding and repre-	omes appear to be targeted t ession of Ubx. ent/full/298/5595/1039	by E(Z)-mediated H3-K27 me	emylation, which
124	w w w.selencemag.org/egi/com	611/1011/2/0/3393/1039		
1 10	owl-2	nhosnhowdata	mhn	ovnoniment
The	erk2	phosphorylate	mbp	experiment
The ex was as	erk2 stent of phosphorylation of MI sumed as 100%.	phosphorylate 3P by ERK2 as a result of a	mbp activation by MEK1 from unt	experiment reated mitotic extract
The ex was as http://v	erk2 atent of phosphorylation of MI isumed as 100%. www.cell.com/cgi/content/full nuclear proteins	phosphorylate 3P by ERK2 as a result of a /92/2/183 bind	mbp activation by MEK1 from unt	experiment reated mitotic extract

Gel mobility shift analysis of nucles	ar proteins bound to <mark>E1</mark> .				
http://linkinghub.elsevier.com/retrie	eve/pii/S0167478102002282				
138 pka	activate	akt	no experiment		
However, not only was the activation phosphorylation at serine 473 of Al	n of <mark>Akt</mark> by cAMP and <mark>PK.</mark> <mark>ct</mark> (44).	only minor, cAMP rather ir	hibited		
http://www.jbc.org/cgi/content/full/	276/16/12864				
139 pka	mediate	creb	experiment		
To determine whether the induction hypoxia on CREB phosphorylation	of CREB phosphorylation in PKA-deficient PC12 ce	was mediated by PKA, we te lls (123.7) (28).	sted the effect of		
http://www.jbc.org/cgi/content/full/	<u>273/31/19834</u>				
140 hh	control	ci	experiment		
Models Illustrating how Ci[rep] and Ci[act] Are Controlled by Hh and how These Transcriptional Activities of Ci Shape the Dpp Morphogen Source					
http://www.cell.com/cgi/content/ful	<u>1/96/6/819</u>				
141 erk	activate	creb	no experiment		
Several studies have demonstrated t kinase Rsk2	hat MAPK/ <mark>ERK</mark> can also a	ctivate CREB via phosphory	lation of the CREB		
http://www.jneurosci.org/cgi/conter	nt/full/20/12/4563				
142 hth	interact	exd	no experiment		
Hth contains a homeodomain and in binding specificity.	nteracts directly with Exd, s	uggesting that Hth also contr	ibutes to Hox DNA		
http://www.developmentalcell.com/	cgi/content/full/3/4/487				
143 e1a	activate	p53	no experiment		
encoded by the alternate reading fra of p53 by E1A and Myc (23).	me of the p16INK4 gene (s	ee above), has recently been i	mplicated in activation		
http://www.sciencemag.org/cgi/con	tent/full/281/5381/1317				
144 cbp	inhibit	tcf	no experiment		
The mechanism by which CBP inhi specific lysine residue in the armad	bits TCF function is not ent lillo binding domain of TCF	irely clear, but <mark>CBP</mark> has been	shown to acetylate a		
http://linkinghub.elsevier.com/retrie	ve/pii/S0167488999001585				
145 erk	activate	creb	no experiment		
The full expression of L-LTP and I Erk , suggesting that the activation of plasticity.	J- LTP-associated CRE- me of <mark>CREB</mark> by <mark>Erk</mark> plays a cri	diated gene expression requir tical role in the formation of l	es the activation of ong-lasting neuronal		
http://linkinghub.elsevier.com/retrie	ve/pii/S0736574800000940				
146 hox	interact	pbx	experiment		
These findings demonstrate that into PBX). To explain these results, we prequired for association with coactiv	eraction of HOX with PBX propose a model whereby phy vators and corepressors, resp	is required for the TSA responses to the test of	nse of pML(5xHOX- OX and PBX is		
http://mcb.asm.org/cgi/content/full/.	20/22/8623	•	•		
14/ creb	depend on	pka	experiment		
Neuregulin-induced phosphorylation	n of CREB is dependent on	PKA in Sol8 myotubes.			
http://linkinghub.elsevier.com/retrie	eve/pii/S0006291X03006600				
148 cbp	interact	ela	experiment		
This region is involved in the specific transcription factors (MyoD, E2F	c interaction of CBP not or , c-Fos, TFIIB) as well as w	ily with EIA, but also with a rith the P/CAF protein.	variety of specific		
http://nar.oupjournals.org/cgi/conter	nt/1u11/30/15/3312				
149 pkc	activate	erk	experiment		
The phosphorylation of p85 is not d hyperosmosis-induced ERK activat	ownstream to the activation ion by PKC depletion or by	of PKC or ERK-2 , since the inhibition of MEK leaves th	prevention of the e tyrosine		

phospł	norylation of p85 intact.			
<u>http://v</u>	www.jbc.org/cgi/content/full/2	272/26/16670		
150	mapk	activate	transcription factor	experiment
The re the FG	sults of the present study sugg F signaling cascade in early i	est that HrEts is the trans nductive events in <i>ascidian</i>	cription factor that is likely a embryos.	ctivated by MAPK in
http://l	inkinghub.elsevier.com/retriev	ve/pii/S0012160603002462	<u>K</u>	
151	fusion proteins	NOT bind	gst	experiment
In ligated ata no compo cytopl	nd blots, GST-syntenin FL (f ot shown) fusion proteins fail sed of GST and the C-termina asmic domain.	Full-length syntenin) and C ed to bind to GST itself, to al deletions (C9 , C21 , C30,	C31) or the F(C30)A mutant	uence starting at M92; or to <mark>fusion proteins</mark> of the syndecan-2
<u>152</u>	ser	regulate	nh	experiment
Regula	sci ation of nh expression by Dfd	and Ser	po	experiment
http://l	inkinghub elsevier com/retriev	ve/nii/S0925477301003013	X	
153	n53	interact	sn1	evneriment
Howev direct	ver, p53, which interacts with binding to the promoter region	Sp1 with a similar affinity, 1.	is able to activate EGFR pro	noter activity through
http://v	www.jbc.org/cgi/content/full/2	276/45/41717		
154	p53	bind	consensus dna	experiment
We we	ere interested in determining w new DNA recognition elemen	hether CR2aa2055-2150 cd t.	ould form a ternary complex v	vith <mark>p53</mark> bound to its
<u>http://v</u>	www.jbc.org/cgi/content/full/2	<u>277/11/9054</u>		-
155	yan	bind	mae	experiment
amoun http://c	t of YAN present is comparable view.biologists.org/cgi/content/	full/130/5/845	12, lanes 1 and 3).	
130 Tudaad	ркс Т соЦаности etimolotica	pnospnorylate	crep	no experiment
reauire	es MAPK activation (206).	induces CKEB phosphory	ation through a PKC -depende	nt pathway that also
http://t	biochem.annualreviews.org/cg	i/content/full/68/0/821		
157	ela	bind	n300/cbn	no experiment
It is the respon	ought that binding of E1A to t sive gene(s).	the p300/CBP inactivates the	he p300/CBP complex and rep	presses the p300/CBP-
http://v	www.jbc.org/cgi/content/full/2	272/10/6101		
158	ela	activate	р53	experiment
<mark>E1A</mark> a death.	ctivates p53, which selectively	y induces target genes such	n as p21WAF1/CIP1 but not N	1dm2, leading to cell
http://r	ncb.asm.org/cgi/content/full/2	20/15/5554		
159	dfd	activate	pb	no experiment
In the respect	Drosophila embryo, <mark>Dfd</mark> and s tively	Scr activate the expression	of pb in the maxillary and lab	ial segments,
http://v	www.sciencemag.org/cgi/cont	ent/full/298/5591/97		
160	akt	phosphorylate	gsk-3	no experiment
This co with w results of insu	onclusion is based on the obse ortmannin or LY 294002 (9, in the inhibition of GSK-3 ac lin on GSK-3beta (18); and	rvations that insulin-induce 17); coexpression of GSK - tivity (10); expression of a Akt phosphorylates and ina	ed inhibition of GSK-3 is bloc 3beta with wild-type or const a dominant-negative mutant of activates both GSK-3 isoforms	ked in cells treated itutively active Akt Akt blocks the effect is in vitro (9).
nttp://v	www.jbc.org/cgi/content/full/2		1 (1	-
161	knirps	interact	dctbp	no experiment

dCtBP was identified in interaction studies with Knirps, *Snail*, and Hairy (Nibu et al., 1998b ; Poortinga et al., 1998) These **repressor proteins** interact with **dCtBP** through **CtBP** binding motifs located within the **repressor domains** located near their C-terminal ends.

http://www.molecule.org/cgi/content/full/9/2/213

<u>mup.//w</u>	ww.molecule.org/cgl/content	/1u1/ <i>9/2/213</i>				
162	transportin	bind	m9	experiment		
Even though transportin can bind an M9 signal and the BIB domain simultaneously, it appears unlikely that transportin normally would import the two substrates at the same time: import of the trimeric M9-transportin -BIB complex is apparently much less efficient than import of, for example, an M9-transportin complex (not shown).						
http://er	mboj.oupjournals.org/cgi/con	tent/full/17/15/4491				
163	transcription factor	bind	pre	experiment		
A yeast proxima	A <i>yeast</i> one-hybrid system was used to clone a transcription factor that binds to the PRE sequences in the proximal promoter of the NPT2 gene .					
http://w	ww.jbc.org/cgi/content/full/2	274/40/28256				
164	ci	mediate	hh	no experiment		
This hig through	ghly economic process is furt Ci in <i>Drosophila</i> .	her exemplified by the find	ing that most, if not all, Hh si	gnaling is mediated		
http://w	ww.genesdev.org/cgi/content	t/full/15/23/3059				
165	pkc	mediate	jnk	no experiment		
PKC ar in trans	nd PI3K mediate FRK and J activation of the AP1 transcri	NK activity, respectively, v principal structure of the second structure of th	which in turn phosphorylate	Fos and Jun, resulting		
http://w	ww.brjpharmacol.org/cgi/cor	ntent/full/139/2/191				
166	ckii	phosphorylate	ser	experiment		
The cle	avage site of caspase 8 is loc	ated exactly between Thr a	and <mark>Ser</mark> residues phosphorylat	ed by CKI and <mark>CKII</mark> .		
http://w	ww.molecule.org/cgi/content	t/full/8/3/601				
167	pkc	regulate	mapk	no experiment		
Howeve	er, <mark>PKC</mark> is an upstream regul	ator of the <mark>MAPK</mark> pathway	v (37, 38).			
http://w	ww.jbc.org/cgi/content/full/2	276/48/45320				
168	erk	depend on	pka	no experiment		
Schema	tic model of FSH-stimulated	, PKA-dependent activatio	n of <mark>ERK</mark> in granulosa cells.			
http://w	ww.jbc.org/cgi/content/full/2	278/9/7167				
169	sp1	NOT interact	cbp	no experiment		
The rest either p	ults showed that ZBP-89 inte 300 or CBP as reported prev	eracts with p300 but not <mark>CE</mark> iously (15).	BP (Fig. 8 A), whereas Sp1 did	l not interact with		
http://w	ww.jbc.org/cgi/content/full/2	275/39/30725				
170	pkc	regulate	mapk	experiment		
These r	esults indicate that PKC regu	lates fibronectin-induced	MAPK activation at a step up	stream of Raf-1 .		
http://w	ww.jbc.org/cgi/content/full/2	274/15/10571				
171	jnk	phosphorylate	transcription factor	no experiment		
The aut	hors then explored the function of c-Jun, a typical t	onal consequence of this in ranscription factor target	teraction on JNK3 activation ed by the <mark>JNK</mark> signaling pathy	by measuring vay.		
http://w	ww.sciencemag.org/cgi/conte	ent/full/290/5496/1515				
172	mapk	mediate	yan	no experiment		
Baker e phospho	et al. (Baker et al., 2001) show orylation of <mark>YAN</mark> , leading to	wed that the binding of ED inactivation of <mark>YAN</mark> functi	L/MAE to YAN is required for on as a repressor of Ets targe	or MAPK-mediated t genes.		
http://de	ev.biologists.org/cgi/content/	full/130/17/4085				
173	erk2	phosphorylate	mbp	no experiment		
Under i displays	nitial rate conditions, the pho s a specificity constant among	sphorylation of <mark>MBP</mark> by <mark>E</mark> g the highest of all known I	<mark>RK2</mark> occurs at a single site (T E RK2 substrates (kcat/ K m	hr97) (10), which = 2.4 muM 1 s 1) (6).		
http://www.jbc.org/cgi/content/full/276/44/40817						

174	cbp	interact	p53	experiment
CBP in	nteracts with FKLF2 and p53	through distinct domains.		
<u>http://l</u>	inkinghub.elsevier.com/retriev	ve/pii/S0006291X02008422	<u>2</u>	
175	tcf	bind	groucho	no experiment
It is the Wg tan http://c	ought that in the absence of W rget genes (Cavallo et al., 199 lev.biologists.org/cgi/content/	' g signaling, <mark>TCF</mark> bound to 8 ; Yang et al., 2000). <u>full/129/14/3393</u>	Groucho acts as a transcrip	tional repressor of
176	tafii250	bind	gst	experiment
(B) Le (mono those s	vels of in vitro-expressed TAI clonal anti-hTAFII250 antiboo hown in panel A.	FII250 bound to GST, TBI dy) of nonradioactive, dupl	P, or Rb , as detected by West icate kinase reactions perform	ern analysis ed concurrently with
http://r	ncb.asm.org/cgi/content/full/1	<u>9/1/846</u>		
177	nuclear proteins	bind	sp1	experiment
Cooper OSM s	ration of nuclear proteins bin signaling may recruit addition	iding to SP1 and Ets DNA al factors that interact with	elements is also needed for m sequences downstream from	aximal expression, and +1 to +47.
http://v	www.jbc.org/cgi/content/full/2	<u>.73/9/5211</u>		
178	abdominal-b	regulate	empty spiracles	experiment
JONES segmen	S, B. and W. MCGINNIS, 199 nt identity function.	3 The regulation of empty	<mark>spiracles</mark> by <mark>Abdominal-B</mark> n	nediates an abdominal
http://v	www.genetics.org/cgi/content/	full/162/1/189		
179	pkc	inactivate	gsk-3	no experiment
Indeed (6,9).	, it has been reported that Wn	t proteins induce inactivati	ion of <mark>GSK-3</mark> through a <mark>PKC</mark>	-dependent mechanism
http://r	ncb.asm.org/cgi/content/full/2	2/7/2099		
180	akt	phosphorylate	gsk-3beta	no experiment
Subsec	quently <mark>Akt</mark> can phosphorylate	e Ser9 of <mark>GSK-3beta</mark> (6).		
http://v	www.jbc.org/cgi/content/full/2	276/40/37436		
181	cbp	interact	ela	no experiment
T		n adenoviral <mark>E1A</mark> onconrot	ain(AA)	
These http://y	www.jbc.org/cgi/content/full/2	277/22/20011	cm (++).	
These http://w 182	www.jbc.org/cgi/content/full/2	<u>177/22/20011</u>	lim domains	no experiment
These http://w 182 Combi predict the ind	Idb ned with the biochemical data the presence of molecules while lividual members of the Islet-1	bind bind that only the first LIM do that only the first LIM do the would bind to their sec family, may modulate spe	lim domains main binds to Ldb (Jurata et ond LIM domains and, by ac ecific functions of them.	no experiment al., 1996), our results eting cooperatively with
These http://w 182 Combi predict the ind http://w	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-1 www.neuron.org/cgi/content/full/2	that only the first LIM do that only the first LIM do that only the first cheir sec family, may modulate spe all/30/2/423	lim domains main binds to Ldb (Jurata et ond L1M domains and, by ac ecific functions of them.	no experiment al., 1996), our results tting cooperatively with
These http://w 182 Combi predict the ind http://w 183	Idb ned with the biochemical data the presence of molecules while www.neuron.org/cgi/content/full/2 www.neuron.org/cgi/content/full/2 nuclear protein	that only the first LIM do that only the first LIM do that only the first sec family, may modulate spe all/30/2/423 bind	lim domains main binds to Ldb (Jurata et ond LM domains and, by ac ecific functions of them. sp1	no experiment al., 1996), our results eting cooperatively with experiment
These http://w 182 Combi predict the ind http://w 183 Effect	Idb ned with the biochemical data the presence of molecules whi www.neuron.org/cgi/content/fit nuclear protein of EGF on nuclear protein b	that only the first LIM do ich would bind to their sec family, may modulate spe all/30/2/423 bind inding to an Sp1-binding c	lim domains main binds to Ldb (Jurata et ond LAM domaine and, by acception functions of them. sp1 onsensus sequence in lympho	no experiment al., 1996), our results sting cooperatively with experiment blastoid cells.
These http://v 182 Combiner predict the ind http://v 183 Effect http://v	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-1 www.neuron.org/cgi/content/fi nuclear protein of EGF on nuclear protein b www.jbc.org/cgi/content/full/2	that only the first LIM do that only the first LIM do that only the first LIM do the would bind to their sec family, may modulate spe all/30/2/423 bind inding to an Sp1-binding c t76/12/8884	lim domains main binds to Ldb (Jurata et ond L W domains and, by ac ecific functions of them. sp1 onsensus sequence in lympho	no experiment al., 1996), our results ting cooperatively with experiment blastoid cells.
Thesehttp://v182Combipredictthe indhttp://v183Effecthttp://v184	Idb Idb ned with the biochemical data the presence of molecules wh lividual members of the Islet-1 www.neuron.org/cgi/content/ft nuclear protein of EGF on nuclear protein b www.jbc.org/cgi/content/full/2 creb	that only the first LIM do that only the first LIM do that only the first LIM do the would bind to their sec family, may modulate spe all/30/2/423 bind inding to an Sp1-binding c 76/12/8884 interact	lim domains main binds to Ldb (Jurata et ond Ldd domains and, by accepting functions of them. sp1 onsensus sequence in lympho cbp	no experiment al., 1996), our results eting cooperatively with experiment blastoid cells. experiment
These http://v 182 Combi predict the ind http://v 183 Effect http://v 184 Since v constru CREB	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-1 www.neuron.org/cgi/content/ful of EGF on nuclear protein of EGF on nuclear protein by ww.jbc.org/cgi/content/full/2 Creb we found that both CREB and uct containing a mutant P-Lin binding site (346M).	indentify the first LIM do inch would bind to their sec inch would bind to their sec inch would bind to their sec inding, may modulate spe ull/30/2/423 bind inding to an Sp1-binding c :76/12/8884 interact P-Lim interact with CBP, n-binding site (346W Lin	lim domains main binds to Ldb (Jurata et ond E-W) domains and, by accepting functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct containing a mut), or a 346 construct containing a mut a set of the set of th	no experiment al., 1996), our results eting cooperatively with experiment blastoid cells. experiment uct (346W), a 346 ntaining a mutant
These http://w 182 Combi predict the ind http://w 183 Effect http://w 184 Since w constru CREB	Idb ned with the biochemical data the presence of molecules while www.neuron.org/cgi/content/fill/2 www.neuron.org/cgi/content/fill/2 of EGF on nuclear protein of EGF on nuclear protein by www.jbc.org/cgi/content/full/2 creb we found that both CREB and act containing a mutant P-Ling binding site (346M). www.jbc.org/cgi/content/full/2	that only the first LIM do ich would bind to their sec ich would bind to their sec i family, may modulate spe all/30/2/423 bind inding to an Sp1-binding c 76/12/8884 interact P-Lim interact with CBP, n-binding site (346W Lin 275/43/33365	lim domains main binds to Ldb (Jurata et ond Lth domains and, by accepting functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct contained and by accepting and by a	no experiment al., 1996), our results eting cooperatively with experiment blastoid cells. experiment uct (346W), a 346 ntaining a mutant
These http://v 182 Combi predict the ind http://v 183 Effect http://v 184 Since v constru CREB http://v	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-1 www.neuron.org/cgi/content/fm of EGF on nuclear protein of EGF on nuclear protein by www.jbc.org/cgi/content/full/2 creb we found that both CREB and act containing a mutant P-Lir B-binding site (346M). www.jbc.org/cgi/content/full/2 pka	addition that performed a set of the sec of th	lim domains main binds to Ldb (Jurata et ond LAN domains) and, by accepting functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct con	no experiment al., 1996), our results ting cooperatively with experiment blastoid cells. experiment act (346W), a 346 ntaining a mutant experiment
These http://v 182 Combi predict the ind http://v 183 Effect http://v 184 Since v constru CREB http://v 185 Transfe reporte recept	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-1 www.neuron.org/cgi/content/ful of EGF on nuclear protein of EGF on nuclear protein b www.jbc.org/cgi/content/full/2 Creb we found that both CREB and act containing a mutant P-Lin binding site (346M). www.jbc.org/cgi/content/full/2 pka ection of cells with either PKI er activity, strongly suggesting ors in Rat1 fibroblasts is med	addition (finite party of ecoprote and composite compos	lim domains main binds to Ldb (Jurata et ond LW chroning and, by accepting functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct conta mut), or a 346 construct conta mut), or a sp1 ha1 adrenergic receptor-sting transcription induced by alp sphorylation of CREB.	no experiment al., 1996), our results etting cooperatively with experiment blastoid cells. experiment uct (346W), a 346 ntaining a mutant experiment mulated CRE-CAT hal adrenergic
These http://v 182 Combi predict the ind http://v 183 Effect http://v 184 Since v constru CREB http://v 185 Transfe reporter recept http://v	Idb ned with the biochemical data the presence of molecules whi ividual members of the Islet-I www.neuron.org/cgi/content/full/2 Inclear protein of EGF on nuclear protein b www.jbc.org/cgi/content/full/2 Creb we found that both CREB and act containing a mutant P-Lir binding site (346M). www.jbc.org/cgi/content/full/2 Dka fection of cells with either PKI er activity, strongly suggesting ors in Rat1 fibroblasts is med www.jbc.org/cgi/content/full/2	the construction of t	lim domains main binds to Ldb (Jurata et ond LAA domains and, by acception functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct contained and, or a 346 construct contained and the sector of the sector o	no experiment al., 1996), our results etting cooperatively with experiment blastoid cells. experiment act (346W), a 346 ataining a mutant experiment mulated CRE-CAT ha1 adrenergic
These http://v 182 Combi predict the ind http://v 183 Effect http://v 184 Since v constru CREB http://v 185 Transfo reporter recept http://v	Idb ned with the biochemical data the presence of molecules while ividual members of the Islet-1 www.neuron.org/cgi/content/full/2 nuclear protein of EGF on nuclear protein b www.jbc.org/cgi/content/full/2 creb we found that both CREB and act containing a mutant P-Ling b-binding site (346M). www.jbc.org/cgi/content/full/2 pka ection of cells with either PKI er activity, strongly suggesting ors in Rat1 fibroblasts is med www.jbc.org/cgi/content/full/2	the second state of t	lim domains main binds to Ldb (Jurata et ond LOV chroning and, by accepting functions of them. sp1 onsensus sequence in lympho cbp we utilized a 346 WT construct contained and the sector of them. nutlized a 346 wT construct contained and the sector of the s	no experiment al., 1996), our results etting cooperatively with experiment blastoid cells. experiment uct (346W), a 346 ntaining a mutant experiment mulated CRE-CAT ha1 adrenergic

<u>http://l</u>	biochem.annualreviews.org/cg	i/content/full/68/0/821		
187	akt	phosphorylate	creb	experiment
We ca	n assume that the phosphoryla	tion of <mark>CREB</mark> by activated	Akt may be long-lasting.	
<u>http://l</u>	inkinghub.elsevier.com/retriev	ve/pii/S0006899302024745		
188	teashirt	bind	armadillo	experiment
GALL of <mark>Wi</mark> l	ET, A., A. ERKNER, B. CHAngless signaling in <i>Drosophila</i>	RROUX, L. FASANO, an by <mark>Teashirt</mark> binding to <mark>Ar</mark>	d S. KERRIDGE, 1998 Trunk <mark>madillo</mark> .	-specific modulation
http://v	www.genetics.org/cgi/content/	full/153/1/319		
189	e1a	inhibit	p53	experiment
In sum inhibit	nmary, we conclude that adeno ion of <mark>p53</mark> activity by <mark>E1A</mark> .	viral oncogene E1A inhibi	ts p73 -mediated transcription	in a manner similar to
<u>http://v</u>	www.jbc.org/cgi/content/full/2	278/20/18313		
190	pka	phosphorylate	cbp	experiment
Our re to activ	sults likewise suggest that CB vate transcription in response t	P phosphorylation by PKA to increased intracellular cA	is the signal transduction step MP.	p required for HOXD4
101	integregi/content/101/2	donord on	nko	no ovneniment
171	jiik oduction of Fos and Jun is me	diated by MAD binage act	PKC	FDK and INK is
depend	dent on PKC and phosphoino	sitide-3-kinase (PI3K) res	pectively (Yang et al., 1996;	Huang et al., 1998 ;
<u>102</u>		henu/1011/139/2/191		
192		Dind		experiment
NS5A	inhibits p53 and TBP binding	to their consensus DNA p	robes	
http://l	linkinghub.elsevier.com/retriev	<u>/e/p11/8016/488902003154</u>		•
193	erk		рка	experiment
throug	h its direct phosphorylation by activation by ERK (Fig. 8), su	$\frac{1}{2}$ ERK , could be uncovered ch as inhibition of COX-2 a	by ablating various stages of and PLA2.	the pathway that led to
http://1	molpharm.aspetjournals.org/cg	gi/content/full/60/5/1100	-	•
194	p53	bind	sp1	experiment
As sho	own in Fig. 5A, elevated bindin	ng of p53 to Sp1 resulted in	a decrease in Sp1-associated	I HDACI.
http://1	mcb.asm.org/cgi/content/full/2	3/8/2669		•
195	mapk	phosphorylate	mbp	experiment
Kineti	c parameters of MBP phospho	orylation by MAPK at diffe	rent pH values (2K)	
http://l	linkinghub.elsevier.com/retriev	ve/p11/S016/483899002232		•
196	creb	depend on	erk	experiment
Phosp	horylation of <i>Elk</i> -1 and CREB	is dependent on ERK afte	r glutamate stimulation.	
http://1	mcb.asm.org/cgi/content/full/1	<u>9/1/136</u>		•
197	mapk	phosphorylate	yan	experiment
We de nuclea MAPI	monstrate that the molecular n r export and define a novel rol K phosphorylation of YAN.	nechanism underlying down e in this context for MAE,	nregulation of <mark>YAN</mark> involves a co-factor previously implic	CRM1 -mediated ated in facilitating
http://o	dev.biologists.org/cgi/content/	full/130/5/845		
198	transportin	mediate	m9	experiment
Note the panels	hat transportin-mediated M9 with the control without trans	import in the absence of Rasportin).	an and energy is significant (c	ompare corresponding
http://v	www.current-biology.com/cgi/	/content/full/9/1/47		
199	pka	phosphorylate	fusion proteins	experiment
Phospl muM	horylation of <mark>fusion proteins</mark> l ATP.	by PKA was performed in 2	20 mM HEPES , pH 7.0, 10 n	nM MgCl2, and 250
http://v	www.jneurosci.org/cgi/content	t/full/19/12/4748		

200pkcstimulatemapkno experimentOnce activated, PKC stimulatesMAPK activity via a poorly understood mechanism involving the activation of Raf
kinase(13, 14).

http://www.jbc.org/cgi/content/full/271/3/1266