·理论与史·

从动物到人类，社会缓冲效应如何促进心理健康？*

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摘　要　社会关系能够有效缓解应激反应对个体的负面影响，这一现象被称为社会缓冲效应。通过回顾现有动物和人类研究，归纳总结了实验室中常用的社会缓冲效应研究范式与社会支持的主要来源，并进一步探讨了动物模型中社会缓冲效应的持续性与物种广泛性、人类研究中社会支持类型和性别对社会缓冲效应的影响以及该效应的神经生理机制。未来的研究应从发生媒介、研究方法和研究精度等角度深入探讨社会缓冲效应的生理机制。同时，应关注准社会关系对社会缓冲的作用，并进一步探讨社会缓冲效应在精神障碍干预与治疗中的实际应用价值。

关键词　社会缓冲效应　社会支持　压力　下丘脑–垂体–肾上腺轴　神经生理机制

1 引言

在人类漫长的生命史中，人通过与他人联结抵御外界侵害，维护生命安全，这种与他人保持联结的基因便得以保留和延续。马克思提出“人的本质在其现实性上是一切社会关系的总和”，强调了人的社会属性。作为“社会人”，个体通过与他人的社会互动建立社会关系，进一步发展出情感联系和归属感，这种状态被称为社会联结（social connectedness）（Lee & Robbins, 1995）。社会联结能够为个体提供社会支持（social support），从而缓解压力对其造成的负面影响，促进身心健康（Schachter, 1959），这一过程被称为社会缓冲效应（social buffering effect）。

缓冲效应模型认为，社会支持能在个体面临压力时，通过减弱压力对身心健康的负面影响而起到缓冲作用（Cohen & Wills, 1985）。具体来说，社会缓冲效应主要和“社会关系”与“压力”有关。压力是个体在面对威胁性或挑战性情境时所产生的一系列心理与生理反应，既包括情绪与认知层面的负担，也包含如疼痛等基于生理机制的威胁性感受。因此，后续学者提出了疼痛的社会缓冲模型，即社会支持可以通过促进个体采用认知重评、注意力转移等应对策略，有效缓解疼痛体验（Che et al., 2018a）。从神经机制的角度来说，压力可通过激活下丘脑 - 垂体 - 肾上腺（hypothalamic-pituitary-adrenocortical, HPA）轴和交感神经系统（sympathetic nervous system, SNS）引发个体行为和生理反应，而社会关系所提供的社会支持能够缓冲生理反应，减少压力对个体的负面影响（Che et al., 2018a, 2018b; Gunnar, 2017; Kiyokawa & Hennessy, 2018）。

基于上述模型，本文通过梳理相关的动物和人类研究，探讨个体如何利用从社会关系中感知或获得的社会支持来缓解压力反应。对此问题的考察不仅能够加深对社会缓冲效应的理解，还能为促进普通人心理健康以及合理运用社会缓冲效应对心理异常人群进行干预治疗提供理论依据。

2　社会缓冲效应的动物研究

2.1　常用范式

大多数关于社会缓冲的动物研究都在实验室中进行，研究对象常为啮齿类动物（如小鼠、大鼠、

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草原田鼠等）和非人类灵长类动物（如黑猩猩、猴等），仅有少数研究是在自然条件下进行（Wittig et al., 2016; Young et al., 2014），本文主要聚焦于实验室中的研究。

常用的实验室压力源包括暴露于新环境、固定不动、电击以及与电击相关的条件性刺激等，基本的实验室程序包括暴露型（exposure-type）和庇护型（housing-type）两种（Kiyokawa & Hennessy, 2018），其区别主要在于社会支持提供者是否与被试动物同时经历压力情境。暴露型程序是让被试动物和其伴侣一起暴露在压力源中，减少被试动物的压力反应。例如，在 Kiyokawa 和 Takeuchi （2017）的研究中，与独自面对应激源的大鼠相比，与其他大鼠共同暴露在压力源里的被试大鼠表现出较少的冻结行为（freezing）。庇护型程序则是让被试动物经历压力事件后与伴侣或同类接触，伴侣或同类动物通常会给予被试动物安慰行为（如梳毛），被试动物的压力反应会得到缓解（Wilson, 2021）。

2.2 社会缓冲效应的提供者

根据 Kiyokawa （2018）的观点，社会缓冲的类型可被划分为“母婴缓冲（maternal buffering）”、“伴侣缓冲（mate buffering）”和“同类缓冲（conspecific buffering）”，分别描述在不同的生命阶段中，来自母亲、伴侣（有性关系）以及同种族同性或异性（无性关系）的社会支持对应激反应的缓冲作用。

在生命的早期，主要关注的是母婴缓冲。当幼崽暴露于新环境时，皮质酮水平会升高，而母亲在场能够有效减少皮质酮水平增加，从而减轻压力反应（Hennessy et al., 2006）。类似的结果也在非人类灵长类动物的研究中得到了验证（Avellaneda & Kamenetzky, 2021）。随着幼崽的成长，母亲的社会缓冲效应开始减弱，伴侣和同类成为缓冲的重要来源（Sullivan & Perry, 2015）。在伴侣缓冲和同类缓冲中，无论是被试动物经历压力刺激后再次回到自己的伴侣 / 同类身边，还是和自己的伴侣 / 同类一同接受压力刺激，都能够发现被试动物的焦虑样行为减少（Burkett et al., 2016; Chun et al., 2022; Donovan et al., 2018）、皮质醇水平下降（Cavanaugh et al., 2016;Pereira & Barros, 2021）、HPA 轴激活被抑制（Kiyokawa et al., 2014; Ishii et al., 2016）等。

然而，不同类型的社会缓冲在神经机制作用上存在差异。在生命早期，母亲对幼崽压力的缓冲主要是通过阻断去甲肾上腺素（norepinephrine, NE）向下丘脑室旁核（paraventricular nucleus of hypothalamus, PVN）的释放达到抑制 HPA 轴的作用（Sanchez et al., 2015, Sullivan & Perry, 2015）。随着幼崽的成长，伴侣和同类的缓冲效应主要与腹内侧前额叶皮层（ventromedial prefrontal cortex, vmPFC）和前扣带回皮层（anterior cingulate cortex, ACC）有关，前额叶可能会与杏仁核和终纹床核（bed nucleus of the stria terminalis, BNST）相互关联，通过影响孤束核（Nucleus of the solitary tract, NTS）与 PVN 形成神经通路（Sullivan & Perry, 2015），从而抑制 HPA 轴激活。

2.3 社会缓冲效应的持久性

在以往研究中，研究者多关注社会支持对压力的即时缓冲。但近期研究表明，社会缓冲是具有持久性的。例如，在 Mikami 等人（2016）的研究发现，接受社会缓冲的 Wistar 大鼠在恐惧消退训练后的回忆测试中表现出冻结行为减少，而 Wall 等人（2024）不仅发现同伴的社会缓冲可抑制经历恐惧学习大鼠的恐惧反应，还发现社会缓冲的效果在同伴离开后仍会持续。

从神经机制的角度解读，下丘脑和杏仁核参与促进了社会缓冲效应的持久性。研究者发现，有社会缓冲的 Wistar 大鼠在回忆测试阶段出现下丘脑室旁核和外侧杏仁核中的 Fos 表达减少（Mikami et al., 2016）。经历社会缓冲的被试大鼠，其下丘脑至中央杏仁核投射的催产素（oxytocin, OT）信号会减少其压力反应，并且这样的缓冲作用在没有同类陪伴后的 24 小时后仍然存在（Hegoburu et al., 2024）。但是否还有其他脑区参与促成社会缓冲效应的持久性，仍需要更多的研究解答。但总体而言，有关社会缓冲效应的追踪研究仍然较少，社会缓冲效应的持久性目前只在急性压力中得到验证，该作用是否同样适用于慢性压力反应，尚需进一步研究和探讨。

2.4 社会缓冲效应于社会性动物中的广泛性

近来，除了啮齿类动物和非人类灵长类动物以外，许多研究也将目光放在其他的社会性动物（social animal）身上。例如，在美新亮丽鲷（Neolamprologus pulcher）和斑马鱼（zebrafish）身上发现了与啮齿类、非人类灵长类社会缓冲效应相同的神经递质变化（Culbert et al., 2019, 皮质醇；Wilson et al., 2024, OT）。除此之外，研究者还在鸟类（Edgar et al., 2015）、猪（Kanitz et al., 2016）、马（Ricci-Bonot et al, 2021）和牛（Nogues et al., 2023）等动物身上

都发现了社会缓冲效应，这说明了社会缓冲普遍存在于社会性动物之中，对此的考察可以探讨社会缓冲效应于不同物种间的一般性和特异性，为人类社会缓冲效应提供更多思考。

3 社会缓冲效应的人类研究

3.1 常用范式

在以人类为被试的社会缓冲研究中，大多采用生理性痛觉（电击痛、热痛和冷痛）作为实验室压力源。实验过程通常是在被试经历疼痛的时候提供社会支持，会发现社会支持能够降低被试对于疼痛的感知（Che et al., 2021; Luo et al., 2020; Reddan et al., 2020; von Mohr et al., 2018），并减轻被试对与疼痛有关的条件性刺激的恐惧反应（Dou et al., 2022; Hornstein et al., 2016）。

此外，也有实验将社会压力作为被试的压力源，一般是通过特里尔社会压力测试（Trier Social Stress Test, TSST）或是社会疼痛（如社会排斥）来引发被试的社会压力，再向被试提供社会支持。在这些研究中可以发现，无论社会支持的提供者是真人还是虚拟人，他们所提供的肢体接触、言语鼓励、以及鼓励性的表情（如微笑）等等都能缓冲被试的应激反应（Kothgassner et al., 2019; von Mohr et al., 2017）。

3.2 社会缓冲效应的提供者

和动物相同，人类社会缓冲类型也可以分为母婴缓冲、伴侣缓冲和同类缓冲三种类型。随着生命体的不断发展，不同生命阶段能为个体提供有效社会缓冲的社会伙伴是不同的。

对于婴儿和幼儿期的孩子，主要抚养者（大多数为父母）是社会缓冲的主要来源，父母在场能够缓解儿童的压力反应（Gunnar & Hostinar, 2015）。但也有研究指出，只有父母被儿童视为安全的存在时，才能够产生社会缓冲效应，如果抚养者对儿童来说并不安全，反而会增加其压力反应（Jacobvitz & Reisz, 2019）。随着个体青春期的来临和发展，亲子之间的缓冲功能逐渐减弱（Hostinar et al., 2015），友谊等其他社会关系逐渐开始占据重要地位，为个体提供缓冲作用（Hoferichter et al., 2024）。然而，也有研究指出，朋友的存在有时反而会增加青少年的皮质醇水平（Doom et al., 2017），可能是因为青少年正处于心理敏感的时期，容易被同伴压力影响。

对于成年人社会缓冲的研究主要聚焦在浪漫关系（romantic relationship）中。在这些研究里，社会支持的提供方式可分为主动支持和被动支持两类。主动支持是指伴侣在现场通过触觉（Goldstein et al, 2018; Reddan et al., 2020; Shamay-Tsoory & Eisenberger, 2021; von Mohr et al, 2018）、视觉（Eisenberger et al., 2011; Hornstein & Eisenberger, 2018）、听觉（Dou et al., 2022; Mazza et al., 2023）等方式传递社会支持。在涉及主动支持的研究中，多数提供的是触觉支持，如拥抱、按摩、牵手和抚摸等，而少数提供的是视觉、听觉支持，如观看伴侣的照片、听伴侣的声音或支持性言语。而被动支持是指被试的伴侣并不提供直接肢体或感官接触，而是通过伴侣在场陪伴（Duschek et al., 2019; Krahé et al., 2015）或是由被试想象伴侣为其提供社会支持（Jakubiak & Feeney, 2019），从而缓解被试压力。除了浪漫关系，朋友和兄弟姐妹（Brumbaugh, 2017），甚至是社会连结较为薄弱的陌生人（Qi et al., 2020, 2021）同样能为成年人提供社会支持，表明社会缓冲效应不只发生在浪漫关系之间。

3.3 社会支持类型和性别对社会缓冲的影响

先前的一项元分析指出，社会缓冲效应受到支持来源与支持形式的共同影响（Che et al., 2018b）。具体而言，虽然来自陌生人的社会支持可在一定程度上降低被试的生理唤醒水平，且某些特定形式的支持（如言语支持）不仅有助于降低唤醒反应，也能减轻疼痛感知，但来自亲密关系个体的社会支持则展现出更为显著的缓冲效应。亲密关系中的社会支持不仅降低了生理唤醒水平，还能够激活多个与情绪调节、压力控制及疼痛相关的脑区。

此外，性别也是一个很重要的影响因素。当个体接受的是恋人的主动触摸时，无论男女都表现出生理压力减小（Ditzen et al., 2019）和情感状态的改善（Debrot et al., 2017）。但是当社会支持的提供对象是陌生人时，会发现性别差异。例如，对于女性被试而言，同性陌生人的触摸能够缓冲其压力反应（Schirmer et al., 2022），但异性陌生人的触摸会造成产生更大的生理压力；而男性的表现则与之相反（Debrot et al., 2024）。然而，如果只是被动支持，或者是间接接触的主动支持，如使用刷子模拟人类触摸（Krahé et al., 2016），陌生人的性别因素对社会缓冲效应的影响则有所降低。因此，性别对缓冲效应的影响，也会受到社会支持方式的调节。

4 社会缓冲效应的神经生理机制

在动物模型和人类研究中已经发现，社会缓冲效应是通过减弱以自主神经系统（autonomic nervous system, ANS）和 HPA 轴为主的多个应激系统的相关活动而发生的（邢小莉 等, 2016; Ali et al., 2020; Sullivan & Perry, 2015）。HPA 轴是一种神经内分泌反馈系统，包括下丘脑、脑垂体和肾上腺。当机体面对压力时，下丘脑室旁核（PVN）释放促肾上腺皮质激素释放激素（corticotropin-releasing hormone, CRH, 或称 corticotropin-releasing factor, CRF），CRH 刺激垂体分泌促肾上腺皮质激素（adrenocorticotropic hormone, ACTH），最终导致肾上腺皮质释放糖皮质激素皮质醇（cortisol, CT, 人类和非人类灵长类动物）或皮质酮（corticosterone, CORT, 啮齿类动物）。而糖皮质激素通过负反馈机制抑制 PVN 神经元活性，可分为两条抑制路径。一条路径是由中继中心，如环绕 PVN 的局部脑区、BNST 和多个下丘脑核团投射并抑制 PVN 的神经元活性；另一条路径则是由来自前额叶皮层和海马体谷氨酸能投射，通过增加中继中心 GABA 能的活性，来抑制 PVN 神经元的激活，调节糖皮质激素的产生，最终实现压力调节，维持体内平衡（Heck & Handa, 2019）。

而社会缓冲效应的发生，主要通过调节神经肽（如催产素、加压素等）的分泌，并激活或抑制与压力反应相关的脑区（如杏仁核、前额叶皮层和海马体等），从而抑制 HPA 轴和自主神经系统的活动，实现对压力的缓冲。因此，对社会缓冲效应神经机制的研究核心在于探讨与压力相关的神经肽、大脑皮层、边缘系统与 HPA 轴和 ANS 之间的相互作用。基于以往研究，本综述提出以下社会缓冲效应模型（图 1），并在下文对模型中提到的神经肽和脑区进行详细论述。

4.1 神经肽

4.1.1 催产素

催产素（OT）是一种神经肽，能够影响中枢神经、自主神经、免疫和生殖系统（Carter et al., 2020），对整个生命周期的健康至关重要。研究表明，OT 可以在应对压力刺激和与支持性同类共处的环境中，下调 HPA 活性（Burkett et al., 2016; Riem et al., 2020），在促进社会缓冲效应的过程中发挥重要作用。在动物研究中发现，OT 可以通过抑制 CRH/CRF 来调节 HPA 轴（Jamieson et al., 2017, Jurek et al., 2015），也可以通过影响杏仁核、脑岛等与恐惧或依恋相关的脑区来影响 HPA 轴，从而缓解压力反应（Hegoburu et al., 2024; Herpertz et al.,2019; Johnson & Young, 2015; Kuppusamy et al., 2021）。而在人类研究中发现，社会缓冲效应与 OT 受体基因的单核苷酸多态性相关（Sicorello et al., 2020）。并且，有研究表明，情感触摸（通过 C- 触觉神经传递）能够

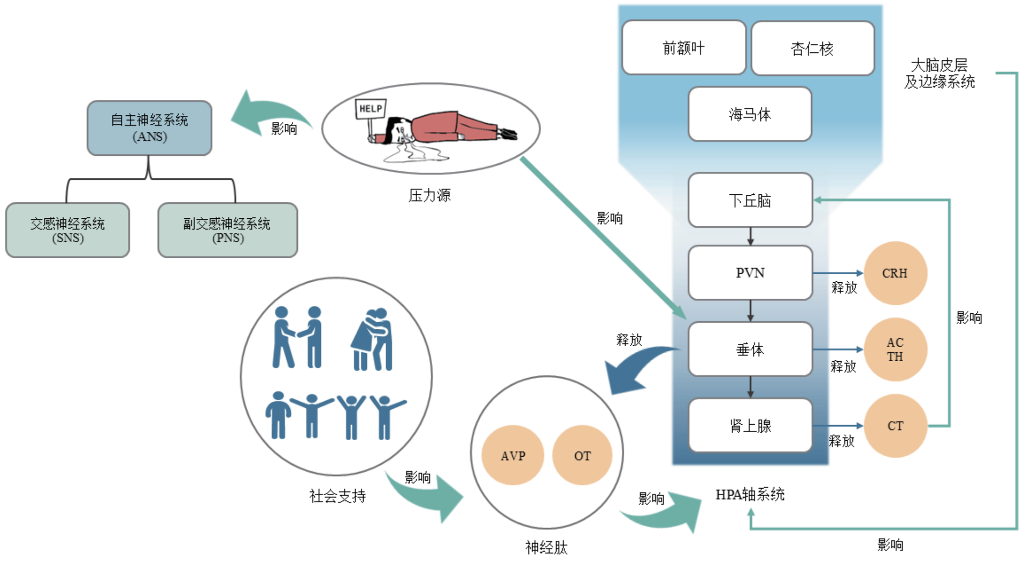

图 1 社会缓冲效应神经机制

注：PVN：下丘脑室旁核；CRH：促肾上腺皮质激素释放激素；ACTH：促肾上腺皮质激素；OT：催产素；VP：加压素；CT：皮质醇

促进个体 OT 的分泌（Walker et al., 2017），从而降低 HPA 轴活性（Uvnäs-Moberg et al., 2020）。除了机体自身分泌的 OT 以外，外源性 OT（通常由鼻腔给药，又称为鼻内催产素，intranasal oxytocin，inOT）也能够增强社会支持对个体的压力缓冲作用（Riem et al., 2020）。

4.1.2 加压素

加压素（vasopressin, VP）又称为抗利尿激素（ADH）或精氨酸加压素（AVP），它通常与 OT 一同进行研究，因为它们在结构上相似，功能上拮抗。例如，在中央杏仁核中，OT 与 VP 的受体区域通过抑制性 GABA 神经元投射相连，OT 通过激活 GABA 神经元抑制恐惧反应，而 VP 则会触发恐惧反应（Huber et al., 2005）。然而，VP 在社会缓冲效应中的作用仍不明确。尽管已有研究表明，VP 能够促进人类女性之间的亲社会行为（Chen et al., 2016），但尚无直接研究探讨 VP 在社会缓冲效应中的具体作用。但鉴于 VP 与 OT 在功能上具有相反的调节作用，而 OT 已被广泛证明能够通过提升其浓度来缓解个体的压力反应，因此可以推测，抑制 VP 浓度或许也具有类似的压力缓冲效应。

4.2 大脑皮层及边缘系统

4.2.1 前额叶

前额叶是涉及调节压力反应和社会缓冲效应的关键脑区。在动物研究中发现，社会支持有助于恐惧消退记忆的巩固，而这一过程涉及 vmPFC 的参与（Raineki et al., 2019）。在人类研究中发现，当被试面临恐惧刺激时，伴侣作为安全信号，能激活 vmPFC，并抑制与疼痛相关的前扣带皮层激活（Eisenberger et al., 2011; Hornstein et al., 2024）。Mulej Bratec 等人（2020）探讨了社会缓冲效应调节厌恶情绪的神经基础，发现社会支持能够减少应激引发的眶额叶皮层（orbitofrontal cortex, OFC）、vmPFC 和背外侧前额叶皮层（dorsolateral prefrontal cortex, DLPFC）活动，并抑制杏仁核和下丘脑反应。

4.2.2 杏仁核

杏仁核在识别负面情绪、恐惧和威胁信号等方面发挥重要作用。杏仁核的基底外侧和内侧核在应对心理压力源时，会优先激活 HPA 轴（Segal, 2016; Ulrich-Lai & Herman, 2009）。杏仁核与神经递质的关系紧密，如外源性 OT 可以减少杏仁核激活（Kanat et al., 2015, Kirsch et al., 2005）、抑制杏仁核对情绪刺激的反应来降低威胁反应（Jones et al., 2017）。Roberts 等人（2022）发现，杏仁核对恐惧面孔反应增加的同时也会增强个体皮质醇反应。除了自身分泌或给药以外，社会支持也能够促进 OT 释放从而抑制杏仁核激活。在动物研究中发现，社会缓冲能够抑制由条件刺激 CS 引发的外侧杏仁核激活（Fuzzo et al., 2015），也能够增加下丘脑至中央杏仁核投射的 OT 信号来减少大鼠的恐惧（Hegoburu et al., 2024）。而在人类研究中发现，比起陌生人的图像，依恋对象的图像能够减少厌恶刺激引发的左侧杏仁核激活（Hornstein et al., 2024）。

4.2.3 海马体

海马体除了和记忆有关以外，还与压力恢复有关。海马体具有密集的糖皮质激素受体，能够参与糖皮质激素的反馈抑制（Chang & Yu, 2019; Sandi, 2013），从而抑制 HPA 轴活动，维持机体激素水平稳态。海马体还会通过影响其他脑区或神经核团以此参与 HPA 轴调节。例如，在 Cole 等人（2022）的研究发现，海马体通过 BNST 对 PVN 的 CRF 释放细胞进行抑制性调节，从而抑制 HPA 轴活动以减轻急性压力反应。已有研究表明，高水平的皮质醇会导致海马体神经元减少或体积缩小（Brown et al., 2015），而社会关系能够缓解压力反应对海马体的负面影响。动物研究发现，社会互动或许是通过调节 OT 水平来调控皮质类固醇的分泌，从而维持海马体体积（Kalman & Keay, 2017）。在人类研究中，一项纵向研究表明，未遭受童年虐待的成年人，其海马体体积与社会支持呈正相关（Förster et al., 2021）。这些发现提示，海马体在社会缓冲效应的发生和维持中发挥了重要作用。

4.3 社会缓冲效应中神经肽与大脑皮层之间的协同

根据前文所述，社会缓冲效应的实现依赖于神经肽（尤其是 OT）与前额叶皮层及边缘系统等关键脑区之间的协同作用。大量研究表明，OT 是介导社会缓冲效应的核心激素之一。首先，CRH 的释放能够激活 HPA 轴和 ANS（Calleja-Agius et al., 2024），而 OT 能够通过抑制 CRH 的分泌从而抑制 ACTH 和 CT 的释放，进而抑制 HPA 轴的激活，实现社会缓冲。其次，在有社会支持的情境下，OT 的释放可以抑制内侧前额叶皮层（medial prefrontal cortex, mPFC）的锥体神经元活动，从而抑制恐惧记忆的形成及恐惧行为的表达，这或许是 OT 促进社会缓冲效应的神经机制之一（Jung et al., 2021）。最后，在海马体和杏仁核中广泛表达 OT 受体，外源性 OT 能够

减少这些区域的压力活动，从而促进社会缓冲的发生（Love, 2018）。综上所述，神经肽与大脑皮层及边缘系统之间形成了一个功能整合的社会缓冲网络，使个体在社会支持的背景下能够更有效地抑制生理应激反应，增强心理与生理的适应能力。

5 未来展望

本文综述了社会缓冲效应的相关研究，重点梳理了动物研究和人类研究中的发现，特别是不同社会关系和社会支持类型在缓解压力反应中的作用，并总结了与社会缓冲效应相关的神经生理机制。未来的研究可在现有成果的基础上进一步探讨缓冲效应的发生机制，具体可在以下几个方面展开：

5.1 社会缓冲效应发生的生理机制

现有关于社会缓冲效应的研究大多集中于描述这一现象，但对其发生机制的探讨仍显不足。在发生媒介上，尽管已有研究表明社会缓冲效应可以通过触觉、视觉、听觉等方式实现，但对嗅觉的关注相对较少。而动物研究已证实，嗅觉在缓解压力反应中发挥着关键作用（Kiyokawa et al., 2009, 2012）。因此，未来研究可进一步探讨嗅觉在人类社会缓冲效应中的潜在作用，以期为理解该效应的发生机制提供新的视角。在研究方法上，目前关于社会缓冲效应的研究多采用静态、预设的社会互动模式，缺乏动态的、更加生态化的互动情境。近年来，自然情境下的实验设计逐渐兴起，这种方法能够提高研究的生态效度，并为深入理解社会缓冲效应的发生与发展提供更多信息。然而，这种方法对实验设计和数据统计分析提出了更高的要求，未来的研究可以在此领域进一步探索。在研究精度方面，动物研究能够在更微观的神经元层面考察社会缓冲效应的发生，而在人类研究中，大多数神经生理研究采用无创的电生理或脑成像技术，神经元水平的神经活动与社会缓冲效应的关系尚未明了。未来的研究可以考虑采用颅内脑电（intracranial electroencephology, iEEG）等技术，进一步探讨在神经元水平的神经机制。

5.2 准社会关系的缓冲效应

除了人与人之间的关系外，人类还能和媒体角色或者图书影视作品中的虚拟角色建立起情感联结，称为准社会关系（parasocial relationships, Hoffner & Bond, 2022）。近年来，随着人工智能（Artificial Intelligence, AI）技术尤其是生成式 AI 的快速发展，人与 AI 之间的关系正成为一种新型关系。近期的研究表明，AI 伴侣（如虚拟社交 AI、社交机器人、社交智能体等）能够提供情感支持，从而提升个体的幸福感（Obaigbena et al., 2024），减轻个体压力（Meng & Dai, 2021）。因此，未来的研究可关注人类与实体或虚拟生成式 AI 伴侣之间能否建立情感联结，并探讨这种联结能否促进缓冲效应的发生。但是，与 AI 的过度互动可能加剧社交焦虑患者的依赖性，进而导致回避人际交往（Hu et al., 2023）。因此，未来的研究不仅需要评估 AI 在缓解个体心理压力方面的有效性，还应关注 AI 如何在缓解个体压力的基础上帮助个体重建和维持真实的社会关系，以促进其社会适应能力和心理健康的全面恢复。

跨物种之间可能也存在缓冲效应。有研究者提出，不同物种之间存在“共文化”（co-culture）现象（Sueur & Huffman, 2024），他们通过共享空间和直接或间接互动，从而实现文化互动、学习与合作行为。因此，不同物种间构建的社会网络或许也可起到社会缓冲作用。在动物研究中，跨物种的缓冲效应还很少或没有被探测到，但有研究表明，与实验大鼠同种但不同品系的大鼠也能为其提供社会缓冲（Nakamura et al., 2016）。相比之下，人类 - 动物跨物种研究较为丰富。有研究者提出，宠物社会支持可以作为其他形式社会支持的补充（McConnell et al., 2011）。并且，有研究表明，宠物提供的支持能够有效抑制 HPA 轴激活（Reilly et al., 2024）。这表明宠物提供的支持可能与人类社会支持引发相同的神经回路，未来研究也可从此角度进一步探讨。

5.3 社会缓冲效应在心理障碍干预中的应用

作为社会心理干预疗法之一，社会支持在常规精神障碍的治疗中发挥着重要作用。例如，社会支持不仅能缓解 PTSD 患者因创伤事件导致的心理症状（Wang et al., 2021），对青少年的抑郁和焦虑相关临床表现（如自杀意念和行为）也具有显著保护作用（Scardera et al., 2020）。实验室研究表明，社会缓冲效应能够降低个体对条件刺激的恐惧反应，并促进对条件刺激的恐惧消退（Dou et al., 2022; Hornstein et al., 2016; Pan et al., 2024）。在生理机制上，社会支持能够促进多种神经肽的分泌，而这些神经肽有助于缓解个体的焦虑和抑郁症状（Zhang et al., 2023）。尽管这些研究提供了重要的理论支持，但社会缓冲效应在心理障碍干预实践中的应用仍相对有限。未来研究可尝试将社会缓冲效应引入系统

脱敏疗法，用于促进焦虑症 / 恐怖症患者的恐惧消退，或将其整合进认知行为疗法的标准治疗程序。此外，近年来已有研究开始探索物理治疗手段（如无创神经调控技术等）对难治性抑郁症、成瘾等心理障碍的干预效果（Ekhtiari et al., 2019; Zhao et al., 2024），未来研究也可以进一步探讨在社会支持背景下实施神经调控，评估社会支持是否具有增强治疗效果的潜力。

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From Animals to Humans, How Does Social Buffering Effect Facilitate Mental Health?

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Abstract Throughout human evolution, individuals have developed a tendency to form connections with others in order to counter external threats and ensure their survival. These social connections provide individuals with social support, and the phenomenon in which social support buffers the effects of stress and promotes physical and mental well-being is referred to as the social buffering effect. In this study, we systematically reviewed relevant animal and human studies, exploring how individuals perceive or receive social support through social relationships to mitigate stress.

Most animal studies on social buffering are conducted in laboratories, mainly using rodents and non-human primates, with common stressors such as novel environments, restraint, and electric shocks. These studies often employ two main paradigms: (a) exposure-type, in which both the subject and a partner experience the stressor together, and (b) housing-type, in which the subject receives support from a partner after exposure to stress. Social buffering can be maternal, mate, or conspecific. Maternal buffering is most effective early in life, primarily by inhibiting the release of norepinephrine to the paraventricular nucleus of the hypothalamus, thereby suppressing the HPA axis. As the offspring mature, the social buffering effect of the mother diminishes, and partners and peers become important sources of buffering, which is mainly associated with the ventromedial prefrontal cortex (vmPFC) and the anterior cingulate cortex (ACC). Recent research suggests that the effects of social buffering can persist, with findings indicating that neural mechanisms in the hypothalamus and amygdala contribute to this prolonged effect. Social buffering has also been observed in various other social species, including fish, birds, pigs, and cattle, highlighting its broad applicability.

In human studies on social buffering, common stressors include pain (e.g., electric, heat, or cold) and social stress (e.g., the Trier Social Stress Test or social exclusion). Social support, whether provided by real or virtual figures, has been shown to reduce pain perception and alleviate fear responses. The sources of social buffering vary by life stage. In infancy and childhood, parents provide the main buffering effect. As individuals reach adolescence, peer relationships, including friendships, become more significant, though sometimes these relationships can increase stress due to peer pressure. In adulthood, social buffering in romantic relationships is studied, with support provided either actively (e.g., physical touch, visual or auditory support) or passively (e.g., partner presence or imagined support). Interestingly, social buffering extends beyond romantic relationships, with friends, siblings, and even strangers offering support. The type of social support and gender differences play a role in buffering. While strangers can provide social support, support from close relationships tends to be more effective. Moreover, women benefit more from same-gender stranger touch, while men respond better to opposite-gender touch. However, these gender differences diminish when support is passive or indirect.

Social buffering effects are primarily mediated by reducing the activity of stress systems like the autonomic nervous system (ANS) and HPA axis. The HPA axis involves the hypothalamus, pituitary, and adrenal glands, with cortisol (or corticosterone in rodents) being released in response to stress. Social buffering modulates this system by regulating neuropeptides (e.g., oxytocin, vasopressin) and activating or inhibiting brain regions related to stress (e.g., amygdala, prefrontal cortex, hippocampus), thus reducing stress responses. Oxytocin plays a key role by downregulating HPA activity and affecting regions related to fear and attachment. In contrast, vasopressin, which antagonizes oxytocin, may contribute to stress responses, though its precise role in social buffering is less clear. The prefrontal cortex is involved in regulating stress responses, with social support activating this region and reducing fear-related activity. The amygdala, crucial for processing threat and fear, is also influenced by social buffering, where oxytocin release inhibits its activation, reducing threat responses. Lastly, the hippocampus, involved in memory and stress recovery, helps regulate the HPA axis and is protected from stress effects through social support. These neurobiological mechanisms illustrate how social buffering operates to mitigate stress and maintain physiological balance. In summary, the social buffering effect relies on the coordinated interaction between neuropeptides and key brain regions such as the prefrontal cortex and the limbic system, with oxytocin (OT) playing a central role.

Future research should further explore the physiological mechanisms of social buffering, such as olfactory cues, and employ more dynamic, real-world experimental designs. Additionally, the impact of parasocial relationships with virtual or AI companions on stress relief needs further exploration, while cross-species buffering, particularly between humans and pets, could reveal shared neurobiological responses. Finally, the social buffering effect plays a role in the intervention and treatment of psychological disorders. It may be incorporated into systematic desensitization therapy, integrated into standard cognitive behavioral therapy protocols, or combined with physical interventions, such as non-invasive.

Key words social buffering effect, social support, stress, hypothalamic-pituitary-adrenocortical axis, neurophysiological mechanism