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Reference Style examples

Muse M & McGrath R. Training comparison among three professions prescribing psychoactive medications: Psychiatric nurse practitioners, physicians, and pharmacologically-trained psychologists. J Cl Psycho. 2010, 66, 1-8.

Norcross J & Goldfried M. Handbook of Psychotherapy Integration: Second Edition. 2003. New York: Oxford Univ Press.

Army Regulation 40-68, Medical Services, Clinical Quality Management (Revised). Issue Date: 22 May 2009, pgs 33 & 34. Department of the Army, Washington, DC: Author. Web link: http://www.army.mil/usapa/epubs/pdf/r40_68.pdf

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Prescriptive Authority for Psychologists: A Comparison between Nurse Practitioners and Physician Assistants

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Abstract

The Prescriptive authority for psychologists (RxP) movement has been viewed as a controversial issue for members of the medical community. The existing literature has focused primarily on psychologists' support and psychiatrists' opposition to psychologists gaining prescriptive privileges. The purpose of this study is to assess the views of physician assistants (PAs) and nurse practitioners (NPs) using a modified version of Sammons and colleagues (2000) survey. In this survey, benefits and liabilities of RxP were presented. Based on social identity theory and persuasion theory, we formulated three hypotheses. First, we hypothesized that NPs would support RxP. Secondly, we hypothesized that PAs would oppose RxP. Thirdly, we hypothesized that there would be significant differences in mean scores between the two groups.A total of 192 participants (119 NPs, 72 PAs, and 1 who did not report their profession) participated in this study. Using a Chi Square analysis, we found that both NPs and PAs endorsed the benefits of RxP. However, PAs reported more concern than NPs about the liabilities of granting prescriptive privileges in the areas of malpractice, over-prescription and under-prescription. These findings support hypothesis one and partially support hypothesis two. Using independent samples t-tests, we found significant differences in mean scores for the drawback items and total scores between nurse practitioners and physician assistants. These findings support hypothesis three. In sum, these results suggest that while both NPs and PAs support RxP, NPs provided greater support for psychologists obtaining prescriptive authority.

Keywords: *prescriptive authority, psychologists, prescribing professionals, social learning theory, theory of persuasion*

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Prescriptive Authority for Psychologists: A Comparison between Nurse Practitioners and Physician Assistants

The medical community has expressed mixed opinions about psychologists obtaining prescriptive authority (Fox et al., 2009). Sammons, Paige, and Levant (2003) reiterate that there is a need to expand the practice of mental health to better serve the needs of society. This can result in blurring the lines between the applied sciences and professional practitioners. Unfortunately, with the continuous change in medicine, there are notable gaps in healthcare.

According to the Association of American Medical Colleges (2012), there are 46,000 psychiatrists who are actively practicing. According to U.S. Bureau of Labor Statistics (2014), there is a shortage of psychiatrists. The number of new psychiatric graduates has steadily decreased between the years of 2000 and 2008. However, the need for psychiatric services continues to rise. It is estimated that the need for psychiatry services will continue to increase by 19 percent from 1995 to 2020. According to the Association of American Medical Colleges (2012), there needs to be an additional 2,600 psychiatrists in order to compensate for the shortage of mental health providers in "federally designated mental health" areas. This gap in psychiatric services makes the issue of prescriptive authority for psychologists central to our client's well-being.

In the early years, RxP faced opposition within and outside the psychological community (Sammons, 2010). For example, there was considerable debate regarding prescriptive authority when Hawaii proposed the first bill to allow psychologists prescriptive rights (Deleon, Fox & Graham, 1991). However, support among the psychological community has continued to rise since the 1980s (Kaplin & Dacunto, 2014).As a function of this support, in 1999, Guam became the first territory to grant prescriptive authority. This was followed by New Mexico in 2002 and Louisiana in 2004 (Kaplin & Dacunto, 2014). Ten years later, Illinois to became the third state to grant prescriptive privileges (American Psychological Association, 2014). Iowa became the fourth state to grant prescriptive privileges in 2016 (American Psychological Association, 2016).

The purpose of this research investigation is to examine the views of physician assistants and nurse practitioners in New York State about psychologists gaining prescriptive privileges. Psychological disorders have become one of the five most expensive conditions (Soni, 2009). The best treatment for psychological disorders is an integrated care model where the psychologist and primary care physician (PCP) and/or another prescribing professional work together to develop treatment plans (McGrath, 2010; McGrath & Sammons, 2011). According to McGrath et al. (2004), the struggle to gain prescriptive authority for psychologists has been a particularly rewarding experience due to the expression of support from non-psychiatric physicians.

In New York, both physician assistants and nurse practitioners are authorized to prescribe psychotropic medications. This could pave the way for psychologists to also obtain prescriptive authority. Social identity theory and persuasion theory serve as the theoretical framework for physician assistants' and nurse practitioners' attitudes on whether psychologists should be granted prescriptive authority.

Physician Assistants: Proponent or Opponent to RxP

It is reasonable to suggest that physician assistants are better understood as technical assistants to a medical doctor (MD), and would share the same values as their superordinate. With the majority of MDs often opposed, PAs are likely to view prescriptive authority with disapproval (Sammons, 2003). One possible reason for this opposition is that the prescriptive authority of psychologists can be seen as an actual threat to their profession.

Jackson and Smith (1999) found that self-identification with a group can significantly affect a person's attitudes and social behavior. Physician assistants define themselves as complementary to physicians; therefore, the physician guides their professional views (Fox et al., 2009). A physician assistant's training, when compared to a medical doctor, is far less extensive. They serve as the "eyes and hands" of a physician rather than a fully autonomous health care provider (Burns, Chemtob, Deleon, Welch, & Samuels, 1988). The physician assistant program consists of two years of medical and clinical coursework and a national certification examination (Burns et al., 1988). This understanding of a physician assistants' identity is beneficial when explaining the social identity theory because it allows for group cohesion, or the "we" concept of one's social identity.

Social identity is the "we" aspect of our self-concept (Myers, 2010; Tajfel, 1982). An individuals' social identity can be best understood when that person is asked to view themselves within a group. An in-group (or the subjective "us") refers to a group of people who share a sense of belonging or a feeling of common identity. An out-group (or the subjective "them"), on the other hand, is a group that people perceive as distinctively different from themselves. Below, we apply social identity theory to physician assistants as a foundation of our hypothesis that PAs will oppose RxP.

Social Identity Theory (SIT) and Physician Assistants

'Social identity' refers to an individual's self-concept in relation to his or her membership of social groups, which has been utilized in many situations within social sciences (Monrouxe, 2009). This concept was introduced by Tajfel and his colleagues (1979) who defined social identity theory as "that part of an individual's self concept which derives from one's knowledge of his membership in a social group (or groups) together with the value and emotional significance attached to that group member" (Tajfel, 1982, p.255).

This theory is associated with positive attitudes towards in-group members (*in-group favoritism*) and negative attitudes towards out-groups (*out-group derogation*) (Burford, 2012).It can explain how psychologists are perceivedas either an insider or an outsider within in the medical community. If one were to examine the professional training of psychologists relative to physician assistants and medical doctors, psychologists would be considered as outsiders.

Sherifproposed (1966), "Whenever individuals belonging to one group interact, collectively or individually, with another group or its members *in terms of their group identification,* we have an instance of intergroup behavior" (p.12). This characteristic of intergroup behavior becomes more important as the interpersonal extreme becomes more distant. There is a decrease in similarity of characteristics and behavior from the members of the out-group, perceived by the members of the in-group (Tajfel, 1982).

In-group and out-group members view information differently, which can lead to the more accurate recall of information obtained from in-group members and the greater valuing of in-group sources (Ostrom, Carpenter, Sedikides, & Li, 1993).It is generally agreed that identifying with a particular group can notably affect a person's social behavior (Jackson &Smith, 1999), which is why the social identity theory is so important, not only to RxP for psychologists, but from a social aspect as well. In other words, psychologists are likely to be viewed by PAs as the so-called 'out-group.' Accordingly, we hypothesize that PAs will oppose the movement for prescriptive authority.

The Theory of Persuasion and Physician Assistants

A second rationale for our hypothesis that PAs would not support psychologist's desire to obtain prescriptive authority is based on the theory of persuasion.The *theory of persuasion* was proposed by Petty, Cacioppo, and Goldman (1981) on their basis of attitude and

attitude change. They suggested “there are many variables affecting the motivation to process a message, but the idea of personal involvement is consistent with the view that increasing involvement enhances issue-relevant thinking” (p. 363).

Brock (1967) stated “beliefs are derived from thoughts about the communication; and these thoughts themselves are particularly a function of the amount of the objective information on either side of the case” (p.302). Here we see that content of the communication is a crucial component to the success not only from SIT, but also in the theory of persuasion.When a persuasive communication is on a topic of high personal relevance, attitude change would be conducted mostly by a thoughtful consideration of the issue-relevant arguments presented. On the other hand, when a message was on a topic of low personal relevance, it’s been believed that those everlasting features of the persuasion situation would be more influential (Petty et al., 1981).

Physician assistants are considered physician extenders, which permits them prescriptive authority under predetermined guidelines set forth by the supervising physician. In relation to the PAs personal involvement in RxP, one can further hypothesize that this dynamic will influence their attitudes. The ability to change one’s attitudes or beliefs through persuasion is not only an important quality to possess, but seems to be paramount in forming an attitude about psychologists obtaining prescriptive authority.

Persuasion is typically defined as “human communication that is designed to influence others by modifying their beliefs, values, or attitudes” (Simons, 1976, p. 21). O’Keefe (1990) noted that there are requirements for the sender, the means, and the recipient to consider something persuasive. First, persuasion involves a goal and the intent to achieve that goal from the message sender. Initially, communication is the means to achieve that goal. Lastly, the message receiver must have free will to make one’s own choices. Clearly persuasion is not accidental, nor is it forced. It is inherently communicational (O’Keefe, 1990).

Many theories are focused on shifts in attitude. As we previously stated, an attitude is a favorable or unfavorable evaluation reaction towards something or someone. It is rooted in one’s beliefs and brought out in one’s feelings or intended behavior (Myers, 2010). We have attitudes toward people, places, events, products, policies, ideas, and so forth (O’Keefe, 1990). Because attitudes are lasting, they are neither disappearing nor based on urges. Yet at the same time, attitudes are *learned*. As such, attitudes vary and change continually. The last and probably most important point is that attitudes are presumed to influence behavior. Attitude changes that occur via the second route or *peripheral route* occur because the person relates the attitude issue or object with positive or negative signals (Petty & Cacioppo, 1984).

One important consideration is the personal relevance with which one has within a given situation. Research has indicated that the level of personal involvement with an issue influences how the relevant arguments are considered (Petty, Cacioppo, & Goldman, 1981). Psychiatrists see prescriptive authority for psychologists as a potential threat to the survival of their profession. It should come as no surprise to find that they are intently opposed (McGrath & Sammons, 2011). Based on this research, we hypothesized that PAs, who have a similar vested interest in prescriptive authority, would not be proponents of RxP.

Nurse Practitioners: Proponent or Opponent to RxP

Since the mid-1960s, approximately 192,000 individuals have been licensed to practice as nurse practitioners (American Association of Nurse Practitioners, 2014). With that being said, despite being granted prescriptive authority, there is no evidence to suggest that nurse practitioners are any more involved in mental disorders, excluding those that have received specialty training (Muse & McGrath, 2010). Nurse practitioners have different

levels of autonomy in their prescriptive authority. Advanced practice psychiatric nurse practitioners can prescribe unaccompanied in 15 states, while 27 require collaboration with a physician, and eight require supervision by a physician (Feldman, Backman, Cuffel, Freisen, & McCabe, 2003). As one of the core mental health professions, it is vital that for nurse practitioners to be knowledgeable about mental health practices (Haber et al., 2004). There is also an understanding that nurse practitioners should incorporate psychosocial care. As stated in the National Organization of Nurse Practitioner Faculties (2002), NPs have a responsibility to evoke the patient's perspective and consult on their plan of care collaboratively.

It is crucial to note that psychologists who prepare to prescribe receive more than six times the instructional training in pharmacology than psychiatric nurse practitioners (Muse et al., 2009). Although psychologists have traditionally provided quality patient care without the knowledge and skills that are acquired as Advanced Health Assessment for Nurse Practitioners, changes in health care place more demands on psychologists to integrate within the general healthcare system (Folen, Keller, James, Porter, & Peterson, 1998). Thus, these changes give way to a more integrated health care team.

Social Identity Theory (SIT) and Nurse Practitioners

With the expansion of NPs scope of practice, they tend to be inclusive towards prescriptive privileges for psychologists. Collaboration between NPs and psychologists is preferred to supervision (McGrath et al., 2004; Wiggins, 2004). This in-group relationship between psychologists and NPs can distinguish the role of nurse practitioner from that of a physician assistant. Partnership between psychologists and nurse practitioners actually builds teamwork, mediates conflict, and offers support, and may lead to mutual respect and collaboration.

With any new identity or role, there are mixed feelings, which can initially create ambivalence or internal conflict (Perspectives in Psychiatric Care, 2004). Psychiatric nurse practitioners may be experience conflict when trying to synthesize their identities when shifting from a nurse to a nurse practitioner (Burford, 2012). This is an important concept when we examine a criticism expressed by some psychologists that granting prescriptive authority would result in a potential loss of professional identity (Kaplin & Dacunto, 2014).

Perhaps, this concern could be reduced when one looks at how prescriptive authority has impacted nurse practitioners. One finding is that NPs are not considered separate from the nursing community.Second, nurse practitioners tend to treat their patients from a holistic perspective.As such, prescribing psychologists are not likely to be viewed as separate to the psychological community.In addition, similar to the nursing community, psychologists tend to relate to clients from a holistic level.These similarities could lead to a shared identity between nurse practitioners and psychologists, which would result in NPs supporting prescriptive authority for psychologists. As previously discussed, PAs categorize intergroup behavior from psychologists consistent with an out-group. Here, we see NPs categorize psychologists behavior as 'in-group' behavior; making them most likely proponents of this social identity movement.

Theory of Persuasion for RxP and Nurse Practitioners

The persuasion routes we discussed earlier for PAs showed their view as more of the peripheral route. Nurse practitioners, on the other hand utilize a central route, meaning their attitude change results from a person's careful consideration of the arguments presented (Petty et al., 1981). Nurse practitioners have very little to lose by psychologists obtaining prescriptive authority. This should result in NPs showing greater support for RxP.

Keeping involved through collaborating amongst other medical professionals is what is expected of all nurse practitioners (NONFF, 2002). The more involving the issue, the more

motivated people would be to signify their right to hold their attitudes by counter-arguing opposing messages (Cialdini, Petty, & Cacioppo, 1981). This theory of normative influence allows for nurse practitioners and psychologists to form a shared cause due to their relatively similar identities within the medical field regarding RxP. As we know, groups with similar characteristics stick together. In this case, we can learn from this theory why both psychologists and nurse practitioners are on the same page in regards to prescriptive authority for psychologists.

Hypotheses

Our first hypothesis is that nurse practitioners will provide significant support for RxP. Our second hypothesis is that physician assistants will see RxP as a potential risk to their profession and they will not provide support for RxP. Our third hypothesis is that nurse practitioners will provide significantly greater support to RxP than physician assistants.

Methods

Participants

Prospective respondents were identified through the NPI registry, the Nurse Practitioner Association of New York (NPA), and utilization of the Greater New York Hospital Association membership directory. Emails, phone calls and faxes were also used to distribute this survey.

After receiving IRB approval, a total of 500 surveys were sent out to prospective participants. A total of 192 respondents expressed interest in participating in the study. One third of the participants were administered a hard copy of the survey. The remainder of participants responded via online channels.The total response rate was 30%. Confidentiality was assured and once the inclusion criteria were satisfied, informed consent was obtained.

The professional affiliation was as follows: (a) 72 respondents were Physician Assistants (PAs), (b) 119 were Nurse Practitioners (NPs), and (c) one did not report their profession. Fifty respondents were male, 138 were female, and four did not report their gender. In order to participate, the respondents were required to be licensed in the state of New York. A full summary of demographics are provided in Table 1.

Table 1

Demographics

	Frequency	Percent
Gender		
Male	50	26%
Female	138	71.9%
Missing	4	2.1
English as a First Language		
Yes	174	90.6%
No	11	5.7%
Missing	7	3.6%
Ethnicity		
Asian-American	12	6.3%
Latino	10	5.2%

African-American	8	4.2%
West-Indian	5	2.6%
White/Non-Hispanic	142	74%
Other	8	4.2%
Prefer Not to Answer	3	1.6%
Missing	4	2.1%
Occupation		
Nurse Practitioner	119	62%
Physician Assistant	72	37.5%
Missing	1	.5%
Years in Practice		
1-5	45	23.4%
6-10	34	17.7%
11-15	41	21.4%
16+	67	34.9%
Missing	5	2.6%
Income		
50,000-74,999	12	6.3%
75,000-99,999	78	40.6%
100,000-199,999	76	39.6%
200,000+	1	.5%
Prefer Not to Answer	21	10.9%
Missing	4	2.1%
Marital Status		
Single	33	17.2%
Married	123	64.1%
Divorced	18	9.4%
In a Relationship	10	5.2%
Prefer Not to Answer	3	1.6%
Missing	5	2.6%

Instruments

This study uses an abridged version of Sammons, Gorny, Zinner, and Allen's (2000) assessment of the benefits and drawbacks of psychologists obtaining prescriptive authority.The wording of the items was modified to be more inclusive to other prescribing professions.This 15-item scale evaluated seven benefits and eight drawbacks, which are

commonly presented as arguments for and against RxP (See Appendix A). This instrument was checked for reliability using Chronbach's Alpha (α = .863) and content validity by comparing it against Sammons and colleagues (2000) instrument. Together, this suggests the instrument is appropriate for use in this study. These items were converted into three scaled scores: (a) Benefit Mean Scores (Q1-5; Q13-14), (b) Drawback Mean Scores (Q6-12; Q15), and (c) Total Mean Scores (Q1-15 with the drawbacks reverse coded).

There was also a nine-item demographics questionnaire assessing age, gender, language, national status, income, years in the respective profession, ethnicity, marital status and occupation.These items were used as group variables to compare against the scores on Sammons et al.'s (2000) 15-item questionnaire. As noted above, the most important demographic factor is the respondent's occupation.

Procedures

Nurse Practitioner and Physician Assistant participants were sent an email invitation to participate in the study and respond to a 15-item survey to assess support for RxP. If this was ineffective, participants were called by phone and asked to participate to which we sent the survey via email or fax based on their preference. A follow up was required to make sure they received the survey and if they had indeed responded.

Results

The first hypothesis was whether nurse practitioners support RxP. Using a Chi Square analysis, NPs demonstrated significant support for the concepts that appropriately trained psychologists should be provided legal authority to prescribe psychotropic medication (68%), the acquisition of prescriptive privileges will enhance the ability of psychologists to more effectively treat certain clients/patients (80%), increase psychologist's scope of practice (82%), change psychologists professional identity (62%), lead to increased ability to care for underserved populations (77%), and increased ability to practice in a hospital setting (49%). All of these results were significant at $p < .01$.Significantly more NPs felt that prescriptive authority should be limited to doctoral-level licensed psychologists.These results were significant at $p < .05$ (see Table 2).

Table 2

Nurse Practitioners' Attitudes Regarding the Benefits of Psychologists Prescribing

Item	Agree	Neutral	Disagree
1. Appropriately trained Psychologists should be provided legal authority to prescribe psychotropic medication.	68%	14%	18% ***
2. Psychology training for prescriptive privileges should be limited to doctoral-level licensed providers.	45%	23%	32% *
3. The acquisition of prescriptive privileges will enhance the ability of psychologists to more effectively treat certain clients/ patients.	80%	6%	14% ***
4. The acquisition of prescriptive privileges would increase psychologist's scope of practice.	82%	12%	6% ***
5. The acquisition of prescriptive privileges would change psychologist's professional identity.	62%	18%	20% ***
13. The acquisition of prescriptive privileges would lead to increased ability to practice in a hospital setting.	49%	35%	16% ***
14. The acquisition of prescriptive privileges would lead to increased ability to care for underserved populations.	78%	13%	9% ***

*** *p*< .05 ** *p*< .01*** *p*< .001**

The items reflecting the drawbacks of granting prescriptive authority were reverse coded. Therefore, lower levels of agreement would indicate greater support for RxP. A minority of nurse practitioners felt that the acquisition of prescriptive privileges would lead to under-prescription (4%), over-prescription (21%), insufficient monitoring of medication (19%), inappropriate prescription (19%), suboptimal medication (14%), prescriptive privileges would lead to medication taking the place of psychotherapy (23%), and damaged relations between psychologists and psychiatrists (22%). All results were significant at $p < .001$ with the exception of the item suggesting prescriptive authority would result in damaged relations between psychologists and psychiatrists.This item was significant at $p < .05$. Nurse practitioners showed significant concern that prescriptive privileges would lead to an increase in malpractice rates (52%) and that it would change psychologists' professional identities (see Table 3).

Table 3

Nurse Practitioners' Attitudes Regarding the Drawbacks of Psychologists Prescribing

Item	Agree	Neutral	Disagree
6. The acquisition of prescriptive privileges would lead to an increase in malpractice rates.	52%	24%	24% ***
7. The acquisition of prescriptive privileges would lead to underprescription of medication by psychologists.	4%	29%	67% ***
8. The acquisition of prescriptive privileges would lead to overprescription of medication by psychologists.	21%	28%	51% ***
9. The acquisition of prescriptive privileges would lead to insufficient monitoring of medication.	19%	19%	62% ***
10. The acquisition of prescriptive privileges would lead to inappropriate prescription of medication.	19%	24%	57% ***
11. The acquisition of prescriptive privileges would lead to prescription of suboptimal medication.	14%	22%	64% ***
12. The acquisition of prescriptive privileges would lead to medication taking the place of psychotherapy	23%	21%	56% ***
15. The acquisition of prescriptive privileges would lead to damaged relations between psychologists and psychiatrists.	22%	34%	44% *

*** $p < .05$ ** $p < .01$ *** $p < .001$**

The second hypothesis that physician assistants will see RxP for psychologists as a potential risk to their profession and, as a result, not provide support for RxP was only partially supported. The majority of PAs also supported the concepts that appropriately trained psychologists should be provided legal authority to prescribe psychotropic medication (57%), the acquisition of prescriptive privileges will enhance the ability of psychologists to more effectively treat certain clients/patients (60%), increase psychologist's scope of practice (74%), change a psychologist's professional identity (58%), lead to increased ability to care for underserved populations (64%), and increased ability to practice in a hospital setting (54%). All results were significant at $p < .001$. Similar to nurse practitioners, the majority of PAs felt prescriptive authority should be restricted to doctoral-level licensed psychologists (53%).This item was significant at $p < .01$ (See Table 4). These results were inconsistent with our hypothesis.

Table 4

Physician Assistants Attitudes Regarding the Benefits of Psychologists Prescribing

Item	Agree	Neutral	Disagree
1. Appropriately trained Psychologists should be provided legal authority to prescribe psychotropic medication.	57%	13%	30%***
2. Psychology training for prescriptive privileges should be limited to doctoral-level licensed providers.	53%	22%	25%**
3. The acquisition of prescriptive privileges will enhance the ability of psychologists to more effectively treat certain clients/ patients.	60%	22%	18%***
4. The acquisition of prescriptive privileges would increase psychologist's scope of practice.	74%	18%	8%***
5. The acquisition of prescriptive privileges would change psychologist's professional identity.	58%	19%	23%***
13. The acquisition of prescriptive privileges would lead to increased ability to practice in a hospital setting.	54%	33%	13%***
14. The acquisition of prescriptive privileges would lead to increased ability to care for underserved populations.	64%	17%	19%***

*** $p < .05$ ** $p < .01$ *** $p < .001$**

Nevertheless, PAs were concerned with psychologists having increased malpractice rates (53%), under-prescription (14%), and over-prescription (38%).These items were significant at $p < .05$ or lower. Some PAs felt that the acquisition of prescriptive privileges would lead to insufficient monitoring of medication (42%), inappropriate prescription (35%), suboptimal medication (32%),prescriptive privileges would lead to medication taking the place of psychotherapy (35.5%), and damaged relations between psychologists and psychiatrists (43%). However, these results were non-significant (see Table 5).The significant items reflecting drawbacks of RxP were consistent with our hypothesis.Thus, our second hypothesis was partially supported.

Table 5

Physician Assistants Attitudes Regarding the Drawbacks of Psychologists Prescribing

Item	Agree	Neutral	Disagree
6. The acquisition of prescriptive privileges would lead to an increase in malpractice rates.	53%	25%	22%**
7. The acquisition of prescriptive privileges would lead to underprescription of medication by psychologists.	14%	36%	50%***
8. The acquisition of prescriptive privileges would lead to overprescription of medication by psychologists.	38%	43%	19%*
9. The acquisition of prescriptive privileges would lead to insufficient monitoring of medication.	42%	26%	32%
10. The acquisition of prescriptive privileges would lead to inappropriate prescription of medication.	35%	26%	39%
11. The acquisition of prescriptive privileges would lead to prescription of suboptimal medication.	32%	26%	42%
12. The acquisition of prescriptive privileges would lead to medication taking the place of psychotherapy	35.5%	29%	34.5%
15. The acquisition of prescriptive privileges would lead to damaged relations between psychologists and psychiatrists.	43%	32%	25%

*** $p < .05$ ** $p < .01$ *** $p < .001$**

The third hypothesis that nurse practitioners will provide significantly greater support to RxP than physician assistants was also partially supported. An independent samples t-test was conducted to compare RxP benefit items mean scores for nurse practitioners and physician assistants. There was a non-significant difference in scores for the nurse practitioners (M= 15.9, SD= 4.61) and physician assistants (M= 17.1, SD= 5.45) conditions t (182) = -1.602, p = .111. These results indicate that both groups agree with the benefits of RxP, though the difference is not significant.

An independent samples t-test was conducted to compare RxP drawback items mean scores for nurse practitioners and physician assistants. There was a significant difference in scores for the nurse practitioners (M= 26.8, SD= 6.07) and physician assistants (M= 23.3, SD= 5.88) conditions t (185) = 3.867, p = .001. These results indicate PAs, on average, expressed more concerns for the drawbacks (or liabilities) of psychologists obtaining prescriptive authority than their NP counterparts.

An independent samples t-test was conducted to compare RxP total scores for nurse practitioners and physician assistants. There was a non-significant difference in scores for the nurse practitioners (M= *36.8*, SD= 8.96) and physician assistants (M= 41.7, SD= 9.73) conditions t (179) = 4.874, p = .001.Nurse Practitioners, on average, report higher general support for RxP than physician assistants (see Table 6).

Table 6

Results of t-tests and Descriptive Statistics Benefits, Drawbacks, and Total RxP Scores

Outcome	Group							
	Nurse Practitioners			Physician Assistants				
	M	SD	n	M	SD	n	T	df
Benefits	15.91	4.61	113	17.11	5.45	71	-1.602	182
Drawbacks	26.79	6.07	116	23.29	5.88	71	3.867***	185
Total	36.88	8.96	111	41.75	9.73	70	-4.874***	179

*** *p*< .05 ** *p*< .01 *** *p*< .001**

Discussion

The results from the Chi Square analysis indicated that both PAs and NPs endorsed the benefits of RxP.No significant differences were found between the groups.However, PAs tend to express more concern about the drawbacks of RxP.This was found on both the Chi Square and t-test analyses.Lastly, NPs show greater general support for RxP.

These findings were consistent with the hypothesis that because NPs have a very similar identity as psychologists, they would support prescriptive authority for psychologists.As noted earlier in this article, Tajfel (1982) defined social identity as “the part of an individual’s self concept which derives from one’s knowledge of his membership in a social group (or groups) together with the value and emotional significance attached to that group member” (p.255). From the NPs strong endorsement of the benefits and disagreement with the drawbacks of RxP, we can see that nurse practitioners can identify with psychologists’ desire to expand their scope of practice. Moreover, NPs are permitted to prescribe psychotropic medication in all 50 states (Feldman et al., 2003). Therefore, the RxP movement would not interfere with NP legislation surrounding prescriptive authority or inhibit their existing scope of practice.As a result, NPs operate on the central route of persuasion (Petty et al., 1981); where attitude change would be conducted mostly by a thoughtful consideration of the issue-relevant arguments presented. Thus, it is not surprising that NPs reported high support in this study.

Physician Assistants, on the other hand, endorsed the benefits of RxP, but also endorsed concern about some liabilities of RxP. The social identity theory led us to believe that the idea of “us” versus “them” would make it harder for PAs to be objective in their responses. The results only partially support this hypothesis. If we were to examine the benefits alone, one could be led to believe that PAs have no concern about the RxP movement.This is counter-indicated by their endorsement of the liabilities. Physician Assistants can prescribe in all 50 states, but there tends to be greater restrictions on their prescriptive authority (Fox et al., 2009). This could lead to competing interests between RxP bills for psychologists and PAs.It would seem that they recognize that RxP could be a good thing for psychologists; however, due to their personal investment as noted by Sammons & McGrath (2011), they could not fully endorse RxP.

The theory of persuasion was effective because when a message is high in personal relevance, the quality of the issue relevant arguments in the message is an important factor of persuasion. Our research indicated that the level of personal involvement with an issue is one variable that influences which pertinent arguments will be considered (Petty et al., 1981). Although other factors can generate the persuasion theory as plausible such as the convenience sample in which we presented. In conclusion, nurse practitioners and physician assistants had a significant difference in RxP support. Nurse practitioners, on average, expressed more support for RxP than PAs.This is not surprising given the aforementioned theories.

One limitation to our study is that there is not an equal sample size amongst NPs and PAs. Also, we recruited participants using convenience sampling. This could introduce a non-response bias. More specifically, perhaps people who responded could be categorically different than those whom didn't respond. This impacts our ability to generalize these results (external validity) to the population at large.Future research on RxP could address some of the limitations previously mentioned as well as gather opinions from other groups that are not in question. Assessing the perspectives of primary care physicians and psychiatrists could lead to better understanding of RxP and its associated theories.

References

American Association of Nurse Practitioners. (2014). Nurse Practitioner Fact Sheet. Retrieved January 13, 2015 fromhttp://www.aanp.org/all-about-nps/np-fact-sheet

American Psychological Association (2014, June 25). APA lauds landmark Illinois law allowing psychologists to prescribe medications. Retrieved January 13, 2015 from http://www.apa.org/news/press/releases/2014/06/prescribe-medications.aspx

American Psychological Association (2016, May 27).Iowa becomes fourth state to allow psychologists to prescribe medications. Retrieved August 3, 2016 from http://www.apa.org/news/press/releases/2016/05/iowa.aspx

Apker, J., & Eggly, S. (2004). Communicating professional identity in medical socialization: considering the ideological discourse of morning report. *Qualitative Health Research, 14(3)*, 411-429.doi:10.1177/1049732303260577

Association of American Medical Colleges (2012). Center for Workforce Studies: Recent Studies and Reports on Physician Shortages in the US. Retrieved from https://www.aamc.org/download/100598/data/recentworkforcestudies.pdf

The Belmont Report. (1979). Ethical principles and guidelines for the protection of human subjects research. The National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research. Retrieved from http://www.hhs.gov/ohrp/humansubjects/guidance/belmont.html

Brock, T. C. (1967).Communication discrepancy and intent to persuade as determinants of counterargument production. *Journal of Experimental Social Psychology*, *3*(3), 296-309.doi:10.1016/0022-1031(67)90031-5

Burford, B. (2012). Group processes in medical education: Learning from social identity theory. *Medical Education, 46(2)*, 143-152.doi:10.1111/j.1365-2923.2011.04099.x

Burns, S. M., Chemtob, C. M., Deleon, P. H., Welch, B. L., & Samuels, R.M. (1988). Psychotropic medication: a new technique for psychology?.*Psychotherapy: Research, Practice, Training*, *25*(4), 508-515.10.1037/h0085376

Cialdini, R. B., Petty, R. E., &Cacioppo, J. T. (1981). Attitude and attitude change. *Annual Review of Psychology*, *32(1),* 357-404.doi:10.1146/annurev.ps.32.020181.002041

DeLeon, P. H., Fox, R. E., & Graham, S. R. (1991). Prescription privileges: psychology's next frontier? *American Psychologist, 46(4)*, 384-393.doi:10.1037/0003-066X.46.4.384

Epstein, R. M., &Hundert, E. M. (2002). Defining and assessing professional competence. *JAMA: Journal of the American Medical Association, 287(2),*226-235. doi:10.1001/jama.287.2.226

Feldman, S., Bachman, J., Cuffel, B., Friesen, B., & McCabe, J. (2003). Advanced practice psychiatric nurses as a treatment resource: Survey and analysis. *Administration and Policy in Mental Health, 30(6)*, 479-494.doi:10.1023/A:1025025600989

Folen, R. A., Kellar, M. A., James, L. C., Porter, R. I., & Peterson, D. R. (1998). Expanding the scope of clinical practice: the physical examination. *Professional Psychology: Research and Practice, 29(2)*, 155-159. doi:10.1037/0735-7028.29.2.155

Fox, R. E., DeLeon, P. H., Newman, R., Sammons, M. T., Dunivin, D. L., & Baker, D. C. (2009). Prescriptive authority and psychology: A status report. *American Psychologist*, *64*(4), 257-268.doi:10.1037/a0015938.

Haber, J., Toombs, F. C., Hamera, E., Hilyer, D., Limandri, J. B., Pagel, S., Staten, R. R., Zimmerman, L. M. (2004). Advanced practice psychiatric nurses: 2004 legislative update. *Journal of the American Psychiatric Nurses Association, 10,* 298-310.doi:10.1177/1078390304271860.

Jackson, J. W., & Smith, E. R. (1999). Conceptualizing social identity: A new framework and evidence for the impact of different dimensions. *Personality and Social Psychology Bulletin, 25(1)*, 120-135.doi:10.1177/0146167299025001010

Kaplin, D. & Dacunto, M. (2014) Understanding the benefits and drawbacks of allowing prescriptive authority to psychologists. *Archives of Medical Psychology*, *5*(1), 12-29.

McGrath, R. E. (2010). Prescriptive authority for psychologists. *Annual Review of Clinical Psychology*, *6*(1), 21-47.doi:10.1146/annurev-clinpsy-090209-151448

McGrath, R. E., & Sammons, M. (2011). Prescribing and primary care psychology: Complementary paths for professional psychology. Professional Psychology: *Research and Practice, 42(2),* 113-120. doi:10.1037/a0022649

McGrath, R. E., Wiggins, J. G., Sammons, M. T., Levant, R. F., Brown, A., & Stock, W. (2004). Professional issues in pharmacotherapy for Psychologists. *Professional Psychology: Research and Practice, 35(2)*, 158-163.doi:10.1037/0735-7028.35.2.158

Monrouxe, L. (2009). Negotiating professional identities: Dominant and contesting narratives in medical students' longitudinal audio diaries. *Current Narratives*, *1*, 41-59.

Muse, M. & McGrath, E. R. (2010). Training comparison among three professions prescribing psychoactive medications: psychiatric nurse practitioners, physicians, and pharmacologically trained psychologists. *Journal of Clinical Psychology, 66(1),* 97-103.doi:10.1002/jclp.20623

Myers, G. D. (2010). *Social Psychology* (10th ed.) New York: McGraw-Hill.

O'Keefe, D. J. (1990). Persuasion: theory and research. Thousand Oaks, CA US: Sage Publications, Inc.

Ostrom, T. M., Carpenter, S. L., Sedikides, C., & Li, F. (1993). Differential processing of in-group and out-group information. *Journal of Personality and Social Psychology,64(1)*, 21-34.doi:10.1037/0022-3514.64.1.21

Petty, R. E., Cacioppo, J. T., & Goldman, R. (1981). Personal involvement as a determinant of argument-based persuasion. *Journal of Personality and Social Psychology*, *41(5),* 847-855.doi:10.1037/0022-3514.41.5.847

Petty, R. E., & Cacioppo, J. T. (1984). The effects of involvement on responses to argument quantity and quality: Central and peripheral routes to persuasion. *Journal of Personality and Social Psychology*, *46(1),* 69-81.doi:10.1037/0022-3514.46.1.69

Sammons, M. T., Sexton, J. L., & Meredith, J. M. (1996). Basic science training in psychopharmacology: How much is enough? *.American Psychologist*, 51(3), 230-234.doi:10.1037/0003-066X.51.3.230

Sammons, M. T., Gorny, S. W., Zinner, E. S., & Allen, R. P. (2000). Prescriptive authority

for psychologists: A consensus of support. *Professional Psychology: Research and Practice.31*(6)*,* 604-609.doi:10.1037/0735-7028.31.6.604

Sammons, M. T., Paige, R., & Levant, R. F. (2003). Prescribing psychologists: the future. InSammons, M. T., Paige, R., Levant, R. F. (Eds.), Prescriptive authority for psychologists: a history and guide (pp. 191-212). Washington, DC US: *American Psychological Association*. doi:10.1037/10484-011

Sammons, M. T. (2010). The psychopharmacology demonstration project: What did it teach us, and where are we now? In R. E. McGrath, Moore, B. A. (Eds.). Pharmacotherapy for psychologists: prescribing and collaborative roles (pp. 49-67). Washington, DC US: *American Psychological Association*. doi:10.1037/12167-003

Soni, A. (2007). The five most costly conditions, 2000 and 2004 estimates for the U.S. civilian noninstitutionalized population. *Agency for Healthcare Research and Quality*. Rockville, Md.:Medical Expenditure Panel Survey

Tajfel H. (1982). Social psychology of intergroup relations*. Annual Review of Psychology, 33*(1), 1-39.doi:10.1146/annurev.ps.33.020182.000245

Turner, J. C., Brown, R. J., &Tajfel, H. (1979).Social comparison and group interest in ingroup favouritism. *European Journal of Social Psychology*, 9(2), 187-204.doi:10.1002/ejsp.2420090207

United Department of Labor. (2014). Occupational Employment Statistics. Retrieved from http://www.bls.gov/oes/current/oes291066.htm

Wiggins, G. J. (2004). Prescribing, professional identity, and costs. *Professional Psychology: Research and Practice. 35(2),* 148-150.doi:10.1037/0735-7028.35.2.148.

Addressing Cognitive Health: Problems and Solutions

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Abstract

The personal and societal impact of cognitive related health issues is immense, reducing the quality of life for millions of Americans, and costing billions of dollars annually in medical services. The etiology of cognitive impairment is diverse, impacting individuals across the lifespan, and leading to deficits in attention, working memory, inhibitory control, information processing, organization, and planning. Assessment, intervention, and prevention are key areas to help stave off the effects of cognitive decline. The use of valid and reliable psychological and neurophysiological assessment tools aid in the diagnosis and identification of individuals at risk of cognitive decline, selection of appropriate treatments, and evaluation of treatment effects. Emerging interventions such as cognitive brain training and neurofeedback are designed to improve deficits and functioning by directly exercising pre-existing cognitive skills/abilities and/or the underlying neural networks associated with those cognitive processes. Collectively, these interventions have been applied to participant samples with varying degrees of cognitive impairment (e.g. healthy controls, ADHD children, aging adults, etc.), demonstrating performance, clinical, and neurophysiological gains in laboratory settings. However, additional research is needed to further demonstrate the specificity, generalizability, and durability of training effects to support these interventions as frontline treatments for cognitive impairment. Technological advancements have allowed for the development of assessment tools, cognitive brain training games, and neurofeedback protocols that can be delivered through mobile devices, supervised remotely, and seamlessly integrated into existing daily behaviors or interventions. These options are cost effective and user friendly, but require additional research to determine their impact on compliance, generalizability, and efficacy.

Addressing Cognitive Health: Problems and Solutions

As the population of the United States is aging rapidly, the incidence of cognitive impairment is expected to rise. New focus on maintaining brain health has emerged in an effort to reduce age related decline, improve quality of life, and reduce the costs associated with cognitive impairment. However, cognitive impairment and the accompanying changes in mental, physical, and emotional functioning are not limited to a specific age group, disease process, or condition. Individuals with cognitive impairment may experience trouble with memory and learning new things or with concentrating and making decisions in everyday life. Impairment usually presents as difficulty in measurable cognitive skills - like poor memory, weak attention, difficulty concentrating, poor impulse control, and slower pro-

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cessing speed. These impairments create struggles with everyday functioning, learning, and problem solving and range in severity from being a minor nuisance to requiring direct care and supervision.

Impact of Cognitive Impairment

Cognitive health has significant implications on quality of life and economic costs to society (Stoddard, 2014). In 2009, the Centers for Disease Control (CDC) published data collected from 5 states (CA, FL, IA, LA, & MI) that participated in identifying the number of citizens living with some type of cognitive impairment (CDC, 2011). The report indicated the prevalence rate of adults aged 18-49 with some form of cognitive impairment ranged from a low of 4% in Iowa to a high of 8% in Michigan and California. Above the age of 50, the prevalence of cognitive impairment ranged from 9% in Iowa and Louisiana to 15% in Michigan. The Cornell University Employment and Disability Institute published a comprehensive report estimating the 2013 prevalence of cognitive impairment among people all ages in the U.S. at 5.0% or roughly 14.6 million individuals (Erickson, Lee, & von Schrader, 2014). The report also indicated lower rates of employment (11.5% vs. 56.8%), decreased annual earnings ($32,200 vs. $43,300), and higher rates of poverty (34.6% vs. 12.5%) among working-age individuals with cognitive disabilities compared to individuals with no disability. Finally, the estimated annual cost of medical treatment for adults with some type of cognitive impairment at approximately $42.5 billion (Courtney-Long, Carroll, & Zang, 2015) and the cost of "unpaid care" for cognitive impairment at approximately $144 billion (Alzheimer's Association, 2010).

While it is clear that the incidence of cognitive impairment increases with age (Ericson et al., 2014) a variety of etiologies have been identified. In the United States the estimated average annual number of traumatic brain injury (TBI) related emergency room visits is approximately 2.5 million (CDC, 2015). Many of these individuals experience cognitive deficits in attention, learning and memory, planning and decision-making, language and communication, reaction time, and reasoning and judgment. Moreover, many other medical conditions can lead to cognitive impairment. For example, cognitive impairment often accompanies brain tumor, stroke, multiple sclerosis, infections, Parkinson's disease, Huntington's disease, and exposure to neurotoxins (Fatemi & Clayton, 2008). Common psychiatric conditions like depression, generalized anxiety, bipolar disorder (Gualtier & Morgan, 2008), attention deficit hyperactivity disorder (ADHD; van Zomeren & Brouwer, 1994), and chronic stress (McEwen & Sapolsky, 1995) often present with some degree of cognitive impairment.

The previous sections highlight the personal and economic impact of cognitive impairment on society. As a result of these challenges, a focus on cognitive health has developed to assess and improve core cognitive skills and abilities. Greater emphasis has been placed on developing effective tools for assessment and intervention. Proper assessment can lead to a better understanding of cognitive weaknesses, and this understanding can lead to the development of more focused solutions.

Cognitive Components

The presence of cognitive health is viewed as the effective control over one's skills and abilities. This cognitive control has been referred to as executive control or functioning. These terms are used to explain how individuals use general ability to plan and direct activity from beginning to end. Generally, executive functions are considered as the collective input of more specific cognitive processes which include attention, working memory, impulse control, processing speed, and organization and planning. Given the importance of these key cognitive abilities, we will briefly describe each component and highlight its relevance below.

Attention. Attention is the primary glue that holds the cognitive processes together. At-

tention is generally described as the ability to start focusing, select important targets or elements, and avoid distraction from non-essential details. Attention is also responsible for sustaining focus and shifting focus as needed. Likewise, attention allows people to inhibit or ignore distractions from the environment around them and from their own internal thoughts and feelings that are not related to their present goals (Dosenbach & Peterson, 2009; Riccio, Reynolds, & Lowe, 2001). Researchers have identified a strong positive relationship between attention, working memory, and general intelligence (Ilkowska & Engle, 2010; Shipstead, Redick, Hicks, & Engle, 2012).

Working Memory. Working memory is generally considered as a complex interplay of systems that allows humans to manipulate and store information during active thinking, active learning, and active problem solving in everyday life. Working memory capacities are strongly related to general intelligence, attentional control, and processing speed (Redick, Unsworth, Kelly, & Engle, 2012; Shipstead, Harrison, & Engle, 2015; Shipstead, Lindsey, Marshall, & Engle, 2014). The most widely cited model of working memory was developed by Alan Baddeley and has been extensively researched and studied for over 40 years. Baddeley proposed that there are four independent components of working memory that work together and with other abilities like attention to allow the performance of complex human behavior (Baddeley, 2007). The model first describes the use of a phonological loop or the ability to rehearse verbally to ourselves. A complementary system, the visuospatial sketchpad, allows us to visually hold onto information to determine its properties and to verbally name the information. A buffer like or holding space allows for the integration of different types of information (as in verbally naming visual information) and the ability to work back and forth from long term memory. Finally, all of these functions operate under the control of a central processing system responsible for utilizing the attention controls towards task completion, while also maintaining the ability to remain flexible (Baddeley, 2007).

Inhibitory Control. Inhibitory control allows for the suppression of actions and the resistance to interference from irrelevant sources. Impaired inhibitory control is related to poor decision making and susceptibility to addiction (Khurana et al., 2012). Furthermore, poor impulse control has been implicated in underachievement (St-Clair-Thompson & Gathercole, 2006), ADHD (Barkley, 1997), and as a deficient component in a significant number of neuropsychiatric disorders (Pliszka, Carlson, & Swanson, 1999). Overall, the ability to stop responding when needed is a crucial factor for the successful execution of complex social, cognitive, and emotional functioning (Anderson, Jacobs, & Anderson, 2008).

Processing Speed. Processing speed is a measure of an individual's ability to easily perform simple repetitive cognitive tasks. Issues with processing speed are usually more evident when completing previously learned information or tasks than with new learning. Often, despite knowing how to perform a given task, individuals with slow processing find that their recall and thinking are sluggish and require more effort than do their peers. These struggles often lead to stress, anxiety, and rushing to complete tasks, which in turn can lead to frustration and more frequent errors. Research has consistently shown that processing speed is related to working memory and intelligence (Hale & Hale, 1996, Redick et al., 2012). Weak processing speed is common in ADHD and has also been shown to negatively impact reading comprehension and reading fluency (Jacobson et al., 2011). Poor processing speed can have a negative impact on obtained academic achievement, future employment outcomes, and late quality of life (Manard, Carabin, Jaspar, & Collette, 2014).

Organization and Planning. Effective organization and planning require the deployment of the above outlined skills to begin, organize, plan, and monitor various aspects of task completion. For example, task initiation is the ability to get started on something. Individuals who struggle with this skill often have issues with planning and prioritizing as well. With-

out having a plan for a task, it is hard to know how to start. Planning and prioritizing are the abilities to come up with the steps needed to reach a goal and to decide the order of importance. Organization is the ability to keep track of information. Finally, it is important to self-monitor ongoing performance. People that have difficulty with this process cannot tell if their strategies are working. Problems with organization and planning are often found in ADHD, learning disabilities, cognitive impairment, and many neuropsychiatric conditions (Barkley, 1997; Pliszka et al., 1999).

Cognitive Assessment

Given the importance of good cognitive health, cognitive tests and measures have been developed to provide an understanding of an individual's strengths and weaknesses in key skill areas of functioning. The costs of impairment underscore the need for reliable and valid assessments as key determinants in evaluating the state of an individual's cognitive health. Relevant questions range from: "Does someone need help?" and if so, "Is the intervention working and to what extent is a given person's time being used efficiently?". In this way, assessment can help balance the time spent training, against time not spent on other productive activities like reading, socializing, gardening, exercising, or engaging in many other activities shown to benefit cognitive health (Langenbahn, Ashman, Cantor, & Trott, 2013; Rabipour & Raz, 2012; Smith et al., 2010).

Psychological Assessments. Why is there so much emphasis on assessment? Clearly, good assessment is crucial to the identification of deficits, selection of appropriate treatments, and measuring improvement or lack thereof. A variety of assessment tools are available for the evaluation of cognitive function/dysfunction. These include brief screening measures (e.g. Modified Mini Mental Status Exam; Montreal Cognitive Assessment), self-report and third-party (parent, teacher, caregiver) behavioral rating scales (e.g. Conners-3; Functional Activities Questionnaire), intelligence test batteries that include subtests to assess for deficits in verbal comprehension, perceptual reasoning, working memory, and processing speed (e.g. Wechsler Intelligence Scale for Children, WISC-V; Wechsler Adult Intelligence Scale, WAIS-IV), and domain specific tests of executive function (e.g. Test of Variables of Attention, d2 Test of Attention, Stroop Task, Wisconsin Card Sorting Test, Trail-Making Test).

Neurophysiological Assessments. Electroencephalography (EEG) measures the electrical activity in the brain utilizing electrodes placed on the scalp at standardized locations according to the International 10-20 system (Klem, Lüders, Jasper, & Elger, 1999). These assessments can be conducted during a variety of tasks including resting state eyes-open and eyes-closed conditions, continuous performance tasks (CPT), math, reading, and working memory tasks. Quantitative EEG (QEEG) analysis, or the mathematical processes of EEG components, has led to the identification of signature brain patterns that are characteristics of attention deficits and cognitive impairment across the lifespan. For example, children with ADHD exhibit elevated theta power, reduced relative alpha and beta power, and elevated theta/alpha and theta/beta ratios over the frontal and central midline regions (Barry, Clarke, & Johnstone, 2003) compared to healthy peers.

Adults suffering with mild cognitive impairment (MCI) exhibit decreased delta and alpha power and enhanced theta power compared to a healthy control sample, and theta power commensurate to values observed in individuals with Alzheimer's disease (Jackson & Snyder, 2008). An event-related potential (ERPs) is the measure of a brain response that is time-locked to specific sensory, cognitive, or motor events. During the EEG recording, these responses can be evoked using specific protocols and averaged over several trials, allowing the administer/researcher to observe the cognitive operations that occur (within milliseconds) before and after the presentation of a stimulus or a behavioral response

(Woodman, 2010). A variety of ERP differences have been observed in children with ADHD compared to their healthy peers, with reduced posterior P300 amplitudes being the most robust (Barry, Johnstone, & Clarke, 2003). In adults, P300 latencies are prolonged in individuals with MCI compared to unaffected controls, but shortened for individuals with MCI compared to individuals with Alzheimer's disease (Howe, Bani-Fatemi, & De Luca, 2014).

Reliability and Validity. Oversight on the development of assessments has been crucial in stimulating properly constructed and useful tests, while maintaining the safety of the general population. As the use of tests increased, there became a need to develop common standards and guidelines for proper test construction and to demonstrate reliable and valid properties of tests performance. Reliability refers to the ability of a test to produce consistent and stable scores on a given measure over time and validity refers to the ability of a test to accurately measure what it claims to measure. In conjunction with solid reliability and validity, acceptable cognitive tests should include large national samples comprised of individuals from all geographic locations and population demographics. This way, individuals taking the measures can be sure they are comparing their performance to the national standard (American Educational Research Association, 2014). Multilayered oversight ensures that tests and measures that fail to meet the appropriate standards for general or clinical use are not formally promoted and can be removed and/or mitigated as sources of potential harm to consumers. To this end, the Buros Center for Testing publishes the *Mental Measurement Yearbook,* provides professional peer critiques of assessments and descriptive information to inform researchers and clinicians on the reliability and validity of a given measure (Carlson, Geisinger, & Jonson, 2014).

Interventions

The remediation of cognitive impairments has strong roots in medical rehabilitation, resulting in a diverse range of treatment approaches, each showing improvement in cognitive and social functioning and emotional control. While a thorough review of available interventions is beyond the scope of this paper, general approaches have ranged from structured pencil and paper activities to meditation, physical exercise, immersion with nature, and language and music training (reviews see, Langenbahn et al., 2013; Rabipour & Raz, 2012; Smith et al., 2010). Cognitive brain training and neurofeedback training are two emerging computer-based treatment interventions designed to improve cognitive functioning by actively exercising specific skills. Training is focused directly in the areas where basic but specific cognitive difficulties occur, with the goal of positively impacting functional and behavioral outcomes. Although the treatment goals are similar, each intervention approaches training in a different way, e.g., top-down vs. bottom-up approaches. The following sections provide a description of cognitive brain training and neurofeedback training, as well as a brief review of the current treatment research and limitations.

Cognitive Brain Training. Cognitive brain training utilizes a predominantly top-down intervention approach, i.e. targets performance/skill deficits observed in cognitive impairment. An individual actively engages in a task or activity that targets a specific pre-existing cognitive skill or ability (e.g. working memory, sustained attention). Through repetition, practice, and increasing challenge, the individual enhances the cognitive and neurophysiological processes required to effectively perform the task, leading to improved cognitive function and/or symptom reduction. On a cognitive level, increased awareness of the cognitive processes and strategies required for the task are hypothesized to generalize from the specific training task and environment to other settings and cognitive functions. On the biological level, skill repetition and practice are hypothesized to promote neuroplastic processes in the brain.

The efficacy of cognitive brain training, including attention, working memory, and auditory/sensory training, has been investigated in several systematic reviews and meta-analy-

ses for a variety of general health and psychiatric conditions. Post-training improvements on measures of working memory, processing speed, and cognitive function have been observed in healthy older adults (Kelly et al., 2014), producing small to moderate effect sizes for nonverbal memory, verbal memory, working memory, processing speed, and visuospatial skills compared to control conditions (Lampit, Hallock, & Valenzuela, 2014). In healthy younger adults, n-back cognitive training programs led to significant changes on measures of fluid intelligence, producing small effect sizes (Au et al., 2015). For the treatment of children and adolescents with ADHD, cognitive training produced medium effect size for the reduction of total ADHD and inattentive symptoms, and large effect sizes for improvements on visual and working memory (Cortese et al., 2015). For individuals at high risk of cognitive decline (MCI and dementia), training gave rise to improvements in attention, executive function, and memory, along with transfer and maintenance of improvement on psychological measures of depression (Coyle, Traynor & Solowji, 2015; García-Casal et al., 2016). Finally, cognitive training utilizing a mixed (verbal and visuospatial) memory approach produced a medium effect size on pre-posttest performance of verbal short-term memory in individuals with intellectual disabilities (Danielsson, Zottarel, Palmqvist, & Lanfranchi, 2016).

Neurofeedback Training. Neurofeedback training utilizes a predominantly bottom-up intervention approach, i.e. targets brain-based deficits associated with cognitive impairment. Neurofeedback is a specialized form of biofeedback in which the electrical activity of the brain is recorded from the scalp in a region of interest, filtered into the brainwave frequencies of interest, and linked to various forms of visual, auditory, and kinetic feedback displays in a software program (Hammond, 2011). In real-time, the person actively learns to modulate their brainwave activity (e.g. theta, alpha, beta) and feedback components in an a priori direction. Through operant conditioning, behavioral shaping, and reward (Sherlin et al., 2011) the individual enhances neurophysiological and cognitive processes, leading to improved function, performance, and/or symptom reduction. On the biological level, self-regulation of specific brainwave activity is hypothesized to promote neuroplastic processes and flexibility in the brain. On a cognitive level, increased awareness of the cognitive state and strategies required to produce specific brainwave activity are hypothesized to generalize from the training environment to other settings and tasks.

The efficacy of neurofeedback training has also been investigated for the treatment of a variety of conditions. In the treatment of ADHD, neurofeedback training has produced a variety of neurophysiological and behavioral outcomes. In the behavioral domain, neurofeedback has led to improvements in core symptoms of ADHD, with medium effect sizes for overall symptom reduction (Lofthouse, Arnold, Hersch, Hurt, & DeBeus, 2012; Micoulaud-Franchi et al., 2014), and medium (Micoulaud-Franchi et al., 2014) to large (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009) effect sizes on inattention and impulsivity. Neurofeedback led to enhanced improvement on WISC performance and absolute power reductions in delta, theta, alpha, and beta activity among children with learning disabilities (Fernández et al., 2003). Stroke survivors have demonstrated voluntary control of specific brain rhythms during training sessions, with enhancement of SMR activity leading to improvements in visuospatial short-term memory, and enhancement of individualized upper alpha activity leading to improved working memory performance, with comparable gains observed in healthy controls sample (Kober et al., 2015). Finally, neurofeedback training in healthy older adults has also led to improved verbal comprehension and enhanced left hemisphere absolute alpha and beta power following the down-training of theta activity (Fernández et al., 2008), and enhanced cognitive processing and executive function following peak alpha frequency training (Angelakis et al., 2007).

A Practical Example. While cognitive brain training and neurofeedback training have some similarities, there are important differences. As a practical example, think about going to the

gym to work with a personal trainer. The trainer guides you through exercises that work your body to build muscle and increase strength in targeted areas like legs, shoulders, etc. The exercises physically alter your body in a positive way so you can meet pre-established goals, e.g. increase squat max weight. However, if you don't have the muscles necessary to help you complete the task, it won't matter how many repetitions you do. Cognitive training is designed to improve pre-existing cognitive skills like working memory, attention, and impulse control by exercising/practicing an existing skill/task. Conversely, neurofeedback targets the underlying physiology of cognitive processes or impairment. This training is designed to change brain physiology through the operant conditioning of brainwave activity to develop and enhance the cognitive functions associated with and needed for task performance.

State of the Science

Despite the positive treatment outcomes reported above, several independent reviews and meta-analyses highlight methodological issues within the cognitive brain training and neurofeedback research. Additionally, the specificity, generalizability, and durability of treatment effects have been key points of debate (Melby-Lervag & Hulme, 2013; Noack, Lövdén, & Schmiedek, 2014; Shipstead, Redick, & Engle, 2010; Thibault, Lifshitz, Birbaumer, & Raz, 2015; Thibault, Lifshitz, & Raz, 2016). These issues and recommendations for future research are discussed below in greater detail.

Methodological Issues. Collectively, reviewers report that many cognitive brain training studies lack scientific rigor; citing problems with clearly defining target training measures, employing a limited number and type of outcome measures, lack of controlled designs and blinding procedures, small participant samples, and lack of replication and long-term follow-up (Boot & Kramer, 2014; Melby-Lervag & Hulme, 2013; Rabipour & Raz, 2012; Sonuga-Barke, Brandeis, Holtmann, & Cortese, 2014). Neurofeedback research has received similar criticisms, including use of small sample sizes, limited sample demographics, inappropriate use of statistical methods, lack of controlled design or reporting of concomitant treatments, lack of long-term treatment follow-up or monitoring of adverse events, and heterogeneity in protocol design, conditioning methods, and screening procedures (Lofthouse et al., 2012; Loo & Barkley, 2005; Loo & Makeig, 2012; Micoulaud-Franchi et al. 2015; Sonuga-Barke et al. 2013). Due to the large degree of overlap in reviewer criticisms, recommendations for methodological improvements are applicable to both training modalities. Recommendations include the (1) use of power analysis software to determine appropriate sample sizes, (2) use of randomized double-blinded placebo-controlled trials or alternative designs employing partial blinding, additive comparison, treatment dismantling, an interrupted time-series design, counterbalancing or condition crossover, and active or semi-active comparison groups, (3) and independent replication (Green, Strobach, & Schubert, 2014; Vollebregt, van Dongen-Boomsma, Slaats-Willemse, & Buitelaar, 2014).

Specificity. Specificity refers to the ability of an individual to demonstrate task specific learning or mastery that leads to improvement on task specific outcomes, often referred to as *near-transfer* effects. Task specificity has been well documented in cognitive brain training studies, with participants demonstrating improvements in attention, memory, and reasoning tasks following training of those specific abilities (Green & Bavelier, 2008). According to Gruzelier (2014b), specificity in neurofeedback research involves demonstration of frequency band specificity, topographical specificity, and outcome specificity. Review of frequency band specificity reveals mixed results, as the regulation of protocol specific training bands has been observed within and across sessions and at follow-up (Zuberer, Brandeis, & Drechsler, 2015; Gruzelier, 2014b), as well as non-specific changes observed in flanking frequency bands (Gruzelier, 2014b). Outcome specificity has been observed through the comparison of adjacent band protocols, slow and fast wave protocols, and different neurofeedback methods. For example, augmentation of upper alpha activity correlated

with gains in working memory and enhancement of gamma activity correlated with gains in fluid intelligence in healthy training samples (Gruzelier, 2014a), and theta/beta and slow cortical potential (SCP) training led to distinct EEG changes (decreased theta and enhanced alpha and CNV - respectively) that correlated with core symptom improvements in a sample of children with ADHD (Gevensleben et al., 2009).

To further demonstrate the specificity of cognitive brain training and neurofeedback protocols, future investigations should include (1) analysis of within and cross-session EEG regulation performance and assess the impact of self-regulation on clinical outcomes, (2) examination of training characteristics that impact learning and optimize outcomes including the task difficulty, thresholding, type of feedback, rate of reinforcement, and the intensity, frequency, and duration of training, (3) investigation of predictors, mediators, and moderators of training response including age, developmental stage, genetics, neuropsychological functioning, neurophysiological profile, disorder severity, adherence, and compliance and (4) assessment and control of nonspecific factors including motivation, expectancy, emotional state, and client-therapist interaction (Gevensleben, Rothenberger, Moll, & Heinrich, 2012; Green & Bavelier, 2008; Keshavan, Vinogradov, Rumsey, Sherrill, & Wagner, 2014; Micoulaud-Franchi et al., 2015; Strehl, 2014; Zuberer et al. 2015)

Generalizability. While cognitive brain training has repeatedly demonstrated task specificity or near-transfer effects, reviewers are highly critical of the evidence in support of the generalizability of effects from the training task to broader cognitive processes (Boot & Kramer, 2014; Noack et al., 2014; Rabipour & Raz, 2012; Shipstead et al., 2010). Generalizability or *far-transfer* of training effects impacts performance on tasks that are not of the same nature or appearance as the training task (Shipstead et al., 2010) and may be observed across multiple domains and contexts including sensory modality (e.g. visual, auditory), knowledge base, and physical (e.g. specific setting - lab, home, driving), temporal (e.g. time between training and observed effect), functional (e.g. task to general activities of daily living), and social context (Zelinski, 2014). In a meta-analysis, Au and colleagues (2015) reported that working memory training with an n-back task produced a small but significant effect on fluid intelligence in healthy young adults, demonstrating far-transfer. Similarly, evidence of far-transfer in multiple domains has been reported following executive-control, memory, dual-task performance, and complex task training in healthy aging adults (Karbach & Verhaeghen, 2014; Zelinski, 2009). In a review of working memory training research, Klingberg (2010) reported far-transfer to other cognitive tasks (Stroop, paced auditory serial addition task, continuous performance task, recall of nouns, and mathematical reasoning), and reasoning tasks (Raven colored progressive matrices and Bochumer Matrizen test) in patients with ADHD and stroke. Finally, a mounting body of evidence has demonstrated far-transfer with neurophysiological changes following cognitive brain training (Jaušovec & Jaušovec, 2012; Johnstone, Roodenrys, Philips, Watt, & Mantz, 2010; Klingberg, 2010; Olesen, Westerberg, & Klingberg, 2004; Patel, Spreng, & Turner, 2013; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005; Westerberg & Klingberg, 2007).

Evidence of generalizability and far-transfer have also been observed following neurofeedback. Among individuals with ADHD, neurofeedback training has led to performance improvements on a variety of cognitive assessments including the Test of Variables of Attention (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Kaiser & Othmer, 2000; Monastra, Monastra, & George, 2002; Rossiter, 2005; Rossiter & La Vaque, 1995), Counting Stroop (Lévesque, Beauregard, & Mensour, 2006), subtests of the Wechsler Intelligence Scale for Children-Revised (Lévesque et al., 2006; Strehl et al., 2006), d2 Test of Attention (Fuchs et al, 2003), and reaction time and reaction time variability tasks (Mayer Blume, Wyckoff, Brockmeier, & Strehl, 2016). In addition to core ADHD symptoms, be-

havioral changes have also been observed in the reduction of comorbid anxiety and depression (Mayer, Wyckoff, Schulz, & Strehl, 2012a), sleep related issues (Arns, Feddema, & Kenemans, 2014), and classroom and homework behaviors (Mayer, Wyckoff, & Strehl, 2012b). In the neurophysiological domain, changes in EEG activity have been observed within and across training sessions (Mayer et al., 2012b), pre-post intervention (Bakhshayesh, Hänsch, Wyschkon, Rezai, & Esser, 2011; Doehnert, Brandeis, Straub, Steinhausen, & Drechsler, 2008) and during ERP tasks (Arns, Drinkenburg, & Leon Kenemans, 2012; Bakhtdadze, Dzhanelidze, & Khachapuridze, 2011; Egner & Gruzelier, 2004; Mayer et al., 2012a; Wangler et al., 2011).

To promote generalization, researchers recommend that future cognitive brain training and neurofeedback protocols incorporate (1) complexity, novelty, and diversity in training designs to maximize ecological validity, (2) employ transfer trials that require the trainee to practice the training skill without direct feedback or in everyday life settings, (3) integrate training cues from the lab into everyday life situations and vise versa, and (4) promote recognition of situations in which the targeted training task, cognitive process, or brain state are required and assign between session "homework" exercises to practice newly acquired skills in those situations (Gevensleben et al., 2012; Moreau & Conway, 2014; Sonuga-Barke et al., 2014; Strehl, 2014; Vollebregt et al., 2014). To assess for transfer effects, study designs should also incorporate a variety of assessments that measure near-transfer and far-transfer effects, while controlling for test-retest effects, cognitive fatigue, carry-over effects, and the increased probability of Type I errors (Cortese et al., 2015; Green et al., 2014).

Durability. A limited number of cognitive brain training and neurofeedback studies have investigated the long-term persistence of training effects. Within ADHD research, cognitive brain training has led to persistent effects from 9 weeks to 6 month follow-up (Klingberg et al., 2005; Steiner, Frenette, Rene, Brennan, & Perrin, 2014; van der Oord et al., 2014). Similarly, neurofeedback studies have reported the maintenance or continued reduction of core ADHD symptoms, as well as the ability to demonstrate self-regulation skills from six months (Gevensleben et al., 2010; Leins et al., 2007) up to two years post-intervention (Gani, Birbaumer, & Strehl, 2008). Future investigations should include extended follow up periods to investigate the durability of the training effects, as well as potential for training to delay the progression of cognitive impairments (Coyle et al. 2015).

Discussion

The personal and societal impact of cognitive related health issues is immense, reducing the quality of life for millions of Americans, and costing billions of dollars annually in medical services. The etiology of cognitive impairment is diverse, impacting individuals across the lifespan, and leading to deficits in attention, working memory, inhibitory control, information processing, organization, and planning. Assessment, intervention, and prevention are key areas to help stave off the effects of cognitive decline. The use of valid and reliable psychological and neurophysiological assessment tools aid in the diagnosis and identification of individuals at risk of cognitive decline, selection of appropriate treatments, and evaluation of treatment effects. Emerging interventions such as cognitive brain training and neurofeedback are designed to improve deficits and functioning by directly exercising pre-existing cognitive skills/abilities and/or the underlying neural networks associated with those cognitive processes. Collectively, these interventions have been applied to participant samples with varying degrees of cognitive impairment (e.g. healthy controls, ADHD children, aging adults, etc.), demonstrating performance, clinical, and neurophysiological gains in laboratory settings. However, methodological limitations have been identified within this body of work. Additional research is needed to demonstrate the specificity, generalizability, and durability of training effects to support these interventions as frontline treatments for cognitive impairment.

Technological advancements have allowed for the development of assessment tools, cognitive brain training games, and neurofeedback protocols that can be delivered online or via mobile devices, administered and supervised remotely, and integrated into other interventions and activities of daily living. Computerized testing is an efficient and cost effective way to administer standardized cognitive, behavioral, and performance based assessments; decreasing the amount of time needed to administer and score pencil-paper measures, reducing publisher and clinician costs of printing/reordering pencil-paper measures every time an assessment is used, and replacing the need for highly skilled psychometrists. Computerized testing allows clinicians to administer broad baseline assessments to inform the development of adaptive cognitive brain training games that can be tailored to a user's individual cognitive strengths and weaknesses. Similarly, the development and validation of low cost user friendly wireless dry electrode systems (Wyckoff, Sherlin, Ford, & Dalke, 2015) permit clinicians to capture high quality EEG data to monitor neurophysiological treatment outcomes, inform the selection of standardized or individualized neurofeedback protocols, and facilitate training sessions. While these advancements will certainly reduce treatment barriers and enhance accessibility to cognitive brain training and neurofeedback interventions, additional research is needed to determine their impact on compliance, generalizability, and efficacy.

References

Alzheimer 's Association. (2010). *Alzheimer's disease facts & figures, 2010.* Retrieved from: https://www.alz.org/documents_custom/report_alzfactsfigures2010.pdf

American Educational Research Association. (2014). *Standards for educational and psychological testing*. Washington DC: American Educational and Psychological Testing Association.

Anderson, V., Jacobs, R., & Anderson, P. J. (2008). *Executive functions and the frontal lobes: A lifespan perspective*. New York, NY: Taylor and Francis.

Angelakis, E., Stathopoulou, S., Frymiare, J. L., Green, D. L., Lubar, J. F., & Kounios, J. (2007). EEG neurofeedback: A brief overview and an example of peak alpha frequency training for cognitive enhancement in the elderly. *Clinical Neuropsychologist, 21*(1), 110–129. doi:10.1080/13854040600744839

Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*(3), 180-9. doi:10.1177/155005940904000311

Arns, M., Drinkenburg, W., & Leon Kenemans, J. (2012). The effects of QEEG-informed neurofeedback in ADHD: An open-label pilot study. *Applied Psychophysiology and Biofeedback, 37*(3), 171-180. doi:10.1007/s10484-012-9191-4

Arns, M., Feddema, I., & Kenemans, J. L. (2014). Differential effects of theta/beta and SMR neurofeedback in ADHD on sleep onset latency. *Frontiers in Human Neuroscience, 8*, 1-10. doi:10.3389/fnhum.2014.01019

Au, J., Sheehan, E., Tsai, N., Duncan, G. J., Buschkuehl, M., & Jaeggi, S. M. (2015). Improving fluid intelligence with training on working memory: A meta-analysis. *Psychonomic Bulletin & Review, 22*(2), 366-377. doi:10.3758/s13423-014-0699-x

Baddeley, A. (2007). *Working memory thought and action*. Oxford: Oxford University Press.

Bakhshayesh, A. R., Hänsch, S., Wyschkon, A., Rezai, M. J., & Esser, G. (2011). Neurofeedback in ADHD: A single-blind randomized controlled trial. *European Child & Adolescent Psychiatry, 20*(9), 481–491. doi:10.1007/s00787-011-0208-y

Bakhtadze, S. Z., Dzhanelidze, M. T., & Khachapuridze, N.S., (2011). Changes in cognitive evoked potentials during non pharmacological treatment in children with attention deficit/hyperactivity disorder. *Georgian Medical News, 192*, 47-57.

Barkley, R. A. (1997). *ADHD and the nature of self control*. New York, NY: Guilford Press.

Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Quantitative and qualitative electroencephalography. *Clinical Neurophysiology, 114*(2), 171-183. doi:10.1016/S1388-2457(02)00362-0

Barry, R. J., Johnstone, S. J., & Clarke, A. R. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials. *Clinical Neurophysiology, 114*(2), 184-198. doi:10.1016/s1388-2457(02)00363-2

Boot, W. R., & Kramer, A. F. (2014). The brain-games conundrum: Does cognitive training really sharpen the mind? *Cerebrum*, 1-13. Retrieved from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4445580/

Carlson, J. F., Geisinger, K. F., & Jonson, J. L. (2014). *The nineteenth mental measurements yearbook*. Lincoln, NE: Buros Center for Testing.

Centers for Disease Control and Prevention. (2011). *Cognitive impairment: A call for action, now!* Retrieved from http://www.cdc.gov/aging/pdf/cognitive_impairment/cogimp_poilicy_final.pdf

Center for Disease Control and Prevention. (2015). *Report to Congress on traumatic brain injury in the United States: Epidemiology and rehabilitation*. National Center for Injury Prevention and Control; Division of Unintentional Injury Prevention. Atlanta, GA. Retrieved from: http://www.cdc.gov/traumaticbraininjury/pdf/TBI_Report_to_Congress_Epi_and_Rehab-a.pdf

Cortese, S., Ferrin, M., Brandeis, D., Buitelaar, J., Daley, D., Dittmann, R. W., … Sonuga-Barke, E. J. S. (2015). Cognitive training for attention-deficit/hyperactivity disorder: Meta-analysis of clinical and neuropsychological outcomes from randomized control trials. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(3), 164-174. doi:10.1016/j.jaac.2014.12.010

Courtney-Long, E. A., Carroll, D. D., & Zhang, Q. (2015). Prevalence of disability and disability type among adults, United States 2013. *Morbidity and Mortality Weekly Report, 64*(29), 777-782. doi:10.15585/mmwr.mm6429a2

Coyle, H., Traynor, V., & Solowij, N. (2015). Computerized and virtual reality cognitive training for individuals at high risk of cognitive decline: Systematic review of the literature. *American Journal of Geriatric Psychiatry, 23*(4), 335-359. doi:10.1016/j.jagp.2014.04.009

Danielsson, H., Zottarel, V., Palmqvist, & Lanfranchi, S. (2015). The effectiveness of working memory training with individuals with intellectual disabilities - a meta-analytic review. *Frontiers in Psychology, 6*(Article 1230), 1-10. doi:10.3389/fpsyg.2015.01230

Doehnert, M., Brandeis, D., Straub, M., Steinhausen, H.-C., & Drechsler, R.(2008). Slow cortical potential neurofeedback in attention deficit hyperactivity disorder: Is there neurophysiological evidence for specific effects? *Journal of Neural Transmission, 115*(10), 1445–1456. doi:10.1007/s00702-008- 0104-x

Dosenbach, N. U., & Petersen, S. E. (2009). Attentional networks. In *Encyclopedia of Neuroscience*, (pp. 655-660). doi:10.1016/b978-008045046-9.00204-7

Egner, T. & Gruzelier, J. H. (2004). EEG biofeedback of low beta band components: Frequency-specific effects on variables of attention and event-related brain potentials. *Clinical Neurophysiology, 115*(1), 131-139. doi:10.1016/s1388-2457(03)00353-5

Erickson, W., Lee, C., & von Schrader, S. (2014). *2013 Disability status report: United States.* Ithaca, NY: Cornell University Employment and Disability Institute (EDI). Available from: http://www.disabilitystatistics.org/StatusReports/2013-PDF/2013-StatusReport US.pdf

Fatemi, S. H., & Clayton, P. J. (2008). *The medical basis of psychiatry* (3rd ed.). Totowa, NJ: Humana Press.

Fernández, T., Becerra, J., Roca, M., Espino, M., Bahlke, M., Harmony, T.,.. . . Díaz-

Comas, L. (2008). Neurofeedback in healthy elderly humans with electroencephalographic risk of cognitive impairment. *Frontiers in Human Neuroscience. Conference Abstract: 10th International Conference on Cognitive Neuroscience.* doi:10.3389/conf.neuro.09.2009.01.173

Fernández, T., Herrera, W., Harmony, T., Díaz-Comas, L., Santiago, E., Sánchez, L., …Valdés, P. (2003). EEG and behavioral changes following neurofeedback treatment in learning disabled children. *Clinical Electroencephalography, 34*(3), 145–150. doi:10.1177/1550059403400308

Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for attention deficit/hyperactivity disorder in children: A comparison with methylphenidate. *Applied Psychophysiology and Biofeedback, 28(*1), 1–12. doi:10.1023/A:1022353731579

Gani, C., Birbaumer, N., & Strehl, U. (2008). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attention-deficit/hyperactivity disorder. *International Journal of Bioelectromagnetism,10*(4), 209–232. Retrieved from: http://ijbem.ws.hosei.ac.jp/volume10/number4/100402.pdf

García-Casal, J. A., Loizeau, A., Csipke, E., Franco-Martín, M., Perea-Bartolomé, V., & Orrell, M. (2016). Computer-based cognitive interventions for people living with dementia: A systematic literature review and meta-analysis. *Aging & Mental Health,* 1-14. doi:10.1080/13607863.2015.1132677

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., ... Heinrich, H. (2010). Neurofeedback training in children with ADHD: 6-month follow up of a randomised control trial. *European Child & Adolescent Psychiatry*, *19*(9), 715-724. doi:10.1007/s00787-010-0109-5

Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., ... Heinrich, H.(2009). Distinct EEG effects related to neurofeedback training in children with ADHD: A randomized control trial. *International Journal of Psychophysiology, 74*(2), 149-157. doi:10.1016/j.ijpsycho.2009.08.005

Gevensleben, H., Rothenberger, A., Moll, G., & Heinrich, H. (2012). Neurofeedback in children with ADHD: Validation and challenges. *Expert Review of Neurotherapeutics, 12*(4), 447-460. doi:10.1586/ern.12.22

Green, C.S., & Bavelier, D. (2008). Exercising your brain: A review of human brain plasticity and training-induced learning. *Psychology and Aging, 23*(4), 692-701. doi:10.1037/a0014345

Green, C.S., Strobach, T., & Schubert, T. (2014). On methodological standards in training and transfer experiments. *Psychological Research, 78*(6), 756-772. doi:10.1007/s00426-013-0535-3

Gruzelier, J. H., (2014a). EEG-neurofeedback for optimising performance. I: A review of cognitive and affective outcome in healthy participants. *Neuroscience and Biobehavioral Reviews, 44*, 124-141. doi:10.1016/j.neubiorev.2013.09.015

Gruzelier, J. H., (2014b). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience and Biobehavioral Reviews, 44*, 159-182. doi:10.1016/j.neubiorev.2014.03.015

Gualtier, C. T., & Morgan, D. W. (2008). The frequency of cognitive impairment in patients with anxiety, depression, and bipolar disorder: An unaccounted source of variance in clinical trials. *Journal of Clinical Psychiatry*, *69(7)*, 1122-1130. doi:10.4088/jcp.v69n0712

Hale, A. F., & Hale, S. (1996). Processing speed, working memory, and fluid intelligence: Evidence for a developmental cascade. *Psychological Science*, *7*(4), 237-241. doi:10.1111/j.1467-9280.1996.tb00366.x

Hammond, D. C. (2011). What is neurofeedback: An update. *Journal of Neurotherapy, 15*(4), 305-336. doi:10.1080/10874208.2001.623090

Howe, A. S., Bani-Fatemi, A., & De Luca, V. (2014). The clinical utility of the auditory

P300 latency subcomponent event-related potential in preclinical diagnosis of patients with mild cognitive impairment and Alzheimer's disease. *Brain and Cognition, 86*, 64-74. doi:10.1016/j.bandc.2014.01.015

Ilkowska, M., & Engle, R. W. (2010). Trait and state differences in working memory capacity. In A. Gruszka et al. (Ed.), *Handbook of individual differences in cognition: Attention, memory, and executive control.* (pp. 295-320). [The Springer Series on human Exceptionality]. doi:10.1007/978-1-4419-1210-7_18

Jackson, C. E., & Snyder, P. J. (2008). Electroencephalography and event-related potentials as biomarkers of mild cognitive impairment and mild Alzheimer's disease. *Alzheimer's & Dementia, 4*(1), S137-S143. doi:10.1016/j.jalz.2007.10.008

Jacobson, L. A., Ryan, M., Martin, R. B., Ewen, J., Mostofsky, S. H., Denckla, M. B., & Mahone, E. M. (2011). Verbal working memory influences processing speed and reading fluency in ADHD. *Child Neuropsychology*, *17*(3), 209-224. doi:10.1080/09297049.2010.532204

Jaušovec, N., & Jaušovec, K. (2012). Working memory training: Improving intelligence - changing brain activity. *Brain and Cognition, 79*(2), 96-106. doi:10.1016/j.bandc.2012.02.007

Johnstone, S. J., Roodenrys, S., Phillips, E., Watt, A. J., & Mantz, S. (2010). A pilot study of combined working memory and inhibition training for children with AD/HD. *Attention Deficit Hyperactivity Disorder, 2*(1), 31-42. doi:10.1007/s12402-009-0017-z.

Kaiser, D. A., & Othmer, S. (2000). Effect of neurofeedback on variables of attention in a large multi-center trial. *Journal of Neurotherapy, 4*(1), 5–15. doi:10.1300/j184v04n01_02

Karbach, J., & Verhaeghen, P. (2014). Making working memory work: A meta-analysis of executive-control and working memory training in older adults. *Psychological Science, 25*(11), 2027-2037. doi:10.1177/0956797614548725

Kelly, M. E., Loughrey, D., Lawlor, B. A., Robertson, I. H., Walsh, C., & Brennan, S. (2014). The impact of cognitive training and mental stimulation on cognitive and everyday functioning of healthy older adults: A systematic review and meta-analysis. *Aging Research Reviews, 15*, 28-43. doi:10.1016/j.arr.2014.02.004

Keshavan, M. S., Vinogradov, S., Rumsey, J., Sherrill, J., & Wagner, A. (2014). Cognitive training in mental disorders: Update and future directions. *American Journal of Psychiatry, 171*(5), 510-522. doi:10.1176/appi.ajp.2013.13081075

Khurana, A., Romer, D., Betancourt, L. M., Brodsky, N. L., Giannetta, J. M., & Hurt, H. (2012). Working memory ability predicts trajectories of early alcohol use in adolescents: The mediational role of impulsivity. *Addiction*, *108*(3), 506-515. doi:10.1111/add.12001

Klem, G. H., Lüders, H. O., Jasper, H. H., & Elger, C. (1999). The ten-twenty electrode system of the International Federation. The International Federation of Clinical Neurophysiology. *Electroencephalography & Clinical Neurophysiology, 52*, 3-6. Retrieved from: www.clinphjournal.com/pb/assets/raw/Health%20Advance/journals /clinph/Chapter1-1.pdf

Klingberg, T. (2010). Training and plasticity of working memory. *Trends in Cognitive Sciences, 14*(7), 317-324. doi:10.1016/j.tics.2010.05.002

Klingberg, T., Fernell, E., Olesen, P., Johnson, M., Gustafsson, P., Dahlstrom, L., ... Westerberg, H. (2005). Computerized training of working memory in children with ADHD - A randomized, controlled, trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*(2), 177-186. doi:10.1097/00004583-200502000-00010

Kober, S. E., Schweiger, D., Witte, M., Reichert, J. L., Grieshofer, P., Neuper, C., & Wood, G. (2015). Specific effects of EEG based neurofeedback training on memory functions in post-stroke victims. *Journal of NeuroEngineering and Rehabilitation, 12*(1), 107. doi:10.1186/s12984-015-0105-6

Lampit, A., Hallock, H., & Valenzuela, M. (2014). Computerized cognitive training in cog-

nitively healthy older adults: A systematic review and meta-analysis of effect modifiers. *PloS Medicine, 11*(11), e1001756. doi:10.1371/journal.pmed.1001756

Langenbahn, D. M., Ashman, T., Cantor, J., & Trott, C. (2013). An evidenced based review of cognitive rehabilitation in medical conditions affecting cognitive functions. *Archives of Physical Medicine and Rehabilitation*, *94*(2), 271-286. doi:10.1016/j.apmr.2012.09.011

Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and theta/beta protocols. *Applied Psychophysiology and Biofeedback, 32*(2), 73–88. doi:10.1007/s10484-007-9031-0

Lévesque, J., Beauregard, M., & Mensour, B. (2006). Effects of neurofeedback training on the neural substrates of selective attention in children with attention-deficit /hyperactivity disorder: A functional magnetic imaging study. *Neuroscience Letters, 394*(3), 216–221. doi:10.1016/j.neulet.2005.10.100

Lofthouse, N., Arnold, L. E., Hersch, S., Hurt, E., & DeBeus, R. (2011). A review of neurofeedback treatment for pediatric ADHD. *Journal of Attention Disorders, 16*(5), 351–372. doi:10.1177/1087054711427530

Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention-deficit/hyperactivity disorder. *Applied Neuropsychology, 12*(2), 64-76. doi:10.1207/s15324826an1202_2

Loo, S. K., & Makeig, S. (2012). Clinical utility of EEG in attention-deficit/hyperactivity disorder: A research update. *Neurotherapeutics, 9*(3), 569-587. doi:10.1007/s13311-012-0131-z

Manard, M., Carabin, D., Jaspar, M., & Collette, F. (2014, 8 January 2014). Age-related decline in cognitive control: The role of fluid intelligence and processing speed. *BioMed Central Neuroscience*, *15*(7), 1-23. doi:10.1186/1471-2202-15-7

Mayer, K., Blume, F., Wyckoff, S. N., Brockmeier, L. L., & Strehl, U. (2016). Neurofeedback of slow cortical potentials as a treatment for adults with attention deficit-/hyperactivity disorder. *Clinical Neurophysiology, 127*(2), 1374-1386. doi:10.1016/j.clinph.2015.11.013

Mayer, K., Wyckoff, S. N., Schulz, U., & Strehl, U. (2012a). Neurofeedback for adult attention-deficit/hyperactivity disorder: Investigation of slow cortical potential neurofeedback-Preliminary results. *Journal of Neurotherapy, 16*(1), 37-45. doi:10.1080/10874208.2012.650113

Mayer, K., Wyckoff, S. N., Strehl, U. (2012b). One size fits all? Slow cortical potentials neurofeedback: A review. *Journal of Attention Disorders, 17*(5), 393-409. doi:10.1177/1087054712468053

McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. *Current Opinion in Neurobiology, 5*(2), 205-216. doi: 10.1016/0959-4388(95)80028-x

Melby-Lervag, M., & Hulme, C. (2013). Is working memory training effective? A meta-analytic review. *Developmental Psychology*, *49*(2), 270-291. doi:10.1037/a0028228

Micoulaud-Franchi, J.-A., Geoffroy, P. A., Fond, G., Lopez, R., Bioulac, S., & Philip, P. (2014). EEG neurofeedback treatments in children with ADHD: An updated meta-analysis of randomized controlled trials. *Frontiers in Human Neuroscience, 8* (Article 906), 1-7. doi:10.3389/fnhum.2014.00906

Micoulaud-Franchi, J.-A, McGonigal, A., Lopez, R., Daudet, C., Kotwas, I., & Bartolomei, F. (2015). Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. *Clinical Neurophysiology, 45*(6), 423-433. doi:10.1016/j.neucli.2015.10.077

Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 27,* 231–249. doi: 10.1007/s00213-002-1205-0

Moreau, D., & Conway, A. R. A. (2014). The case for an ecological approach to cognitive training. *Trends in Cognitive Sciences, 18*(7), 334-336. doi:10.1016/j.tics.2014.03.009

Noack, H., Lövdén, M., & Schmiedek, F. (2014). On the validity and generality of transfer effects in cognitive training research. *Psychological Research*, *78*(6), 773-789. doi:10.1007/s00426-014-0564-6

Olesen P, Westerberg H, & Klingberg T. (2004). Increased prefrontal and parietal brain activity after training of working memory. *Nature Neuroscience 7*(1), 75–79. doi:10.1038/nn1165

Patel, R., Spreng, R. N., & Turner, G. R. (2013). Functional brain changes following cognitive and motor skills training: A quantitative meta-analysis. *Neurorehabilitation and Neural Repair, 27*(3), 187-199. doi:10.1177/1545968312461718

Pliszka, S. R., Carlson, C. L., & Swanson, J. M. (1999). *ADHD with comorbid disorders: Clinical assessment and management*. New York, NY: The Guilford Press.

Rabipour, S., & Raz, A. (2012). Training the brain: Fact and fad in cognitive and behavioral remediation. *Brain and Cognition*, *79*(2), 159-179. doi:10.1016/j.bandc.2012.02.006

Redick, T. S., Unsworth, N., Kelly, A. J., & Engle, R. W. (2012). Faster, smarter? Working memory capacity and perceptual speed in relation to fluid intelligence. *Journal of Cognitive Psychology, 24*(7), 844-854. doi:10.1080/20445911.2012.704359

Riccio, C. A., Reynolds, C. R., & Lowe, P. A. (2001). *Clinical applications of continuous performance tests: Measuring attention and impulsive responding in children and adults*. New York, United States of America: John Wiley & Sons, Inc.

Rossiter, T. (2005). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: Part II. Replication. *Applied Psychophysiology and Biofeedback, 29*(4), 233–243. doi: 10.1007/s10484-004-0383-4

Rossiter, T. R., & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. *Journal of Neurotherapy, 1*(1), 48–59. doi:10.1300/j184v01n01_07

Rueda, M. R., Rothbart, M. K., McCandliss, B. D., Saccomanno, L., & Posner, M. I. (2005). Training, maturation, and genetic influences on the development of executive attention. *Proceedings of the National Academy of Sciences of the United States of America, 102*(41), 14931–14936. doi:10.1073/pnas.0506897102

Sherlin, L., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy, 15*(4), 292-304. doi:10.1080/10874208.2011.623089

Shipstead, Z., Harrison, T. L., & Engle, R. W. (2015). Working memory capacity and the scope and control of attention. *Attention, Perception, & Psychophysiology*, *77*(6), 1863-1880. doi:10.3758/s13414-015-0899-0

Shipstead, Z., Lindsey, D. R., Marshall, R. L., & Engle, R. W. (2014). The mechanisms of working memory capacity: Primary memory, secondary memory, and attentional control. *Journal of Memory and Language*, *72*, 116-141. doi:10.1016/j.jml.2014.01.004

Shipstead, Z., Redick, T. S., & Engle, R. W. (2010). Does working memory training generalize? *Psychologica Belgica*, *50*(3-4), 245-276. doi:10.5334/pb-50-3-4-245

Shipstead, Z., Redick, T. S., Hicks, K. L., & Engle, R. W. (2012). The scope and control of attention as separate aspects of working memory. *Memory, 20*(6), 608-628. doi:10.1080/09658211.2012.691519

Smith, P. A., Blumenthal, J. M., Hoffman, B. M., Cooper, H., Strauman, T. A., Welsh-Bohmer, K., & Browndyke, J. (2010). Aerobic exercise and neurocognitive performance: A meta-analytic review of randomized controlled trials. *Psychosomatic Medicine*, *72*(3), 239-252. doi:10.1097/PSY.obo13e3181d14633

Sonuga-Barke, E. J., Brandeis, D., Cortese, S., Daley, D., Ferrin, M., Holtmann, M., … Sergeant, J. (2013). Nonpharmacological interventions for ADHD: systematic review

and meta-analyses of randomized controlled trials of dietary and psychological treatments. *American Journal of Psychiatry, 170*(3), 275-289. doi: 10.1176/appi.ajp.2012.12070991

Sonuga-Barke, E., Brandeis, D., Holtmann, M., & Cortese, S. (2014). Computer-based cognitive training for ADHD: A review of current evidence. *Child and Adolescent Psychiatric Clinics of North America, 23*(4), 807-824. doi:10.1016/j.chc.2014.05.009

St Clair-Thompson, H. L., & Gathercole, S. E. (2006). Executive functions and achievements in school: Shifting, updating, inhibition, and working memory. *The Quarterly Journal of Experimental Psychology*, *59*(4), 745-759. doi:10.1080/17470210500162854

Steiner, N. J., Frenette, E. C., Rene, K. M., Brennan, R. T., & Perrin, E. C. (2014). In-school neurofeedback training for ADHD: Sustained improvements from a randomized control trial. *Pediatrics, 133*(3), 483-492. doi:10.1542/peds.2013-2059

Stoddard, S. (2014). *2014 Disability statistics annual report*. Durham, NH: University of New Hampshire. Retrieved from University of New Hampshire Institute on Disability: www.iod.unh.edu

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience, 8*(Article 894), 1-8. doi:10.3389/fnhum.2014.00894

Strehl, U., Leins, U., Goth, G., Klinger, C., Hinterberger, T., & Birbaumer, N. (2006). Self-regulation of slow cortical potentials a new treatment for children with attention deficit hyperactivity disorder. *Pediatrics*, *118*(5), e1530-e1540. doi:10.1542/peds.2005-2478

Thibault, R. T., Lifshitz, M., Birbaumer, N., & Raz, A. (2015). Neurofeedback, self-regulation, and brain imaging; Clinical science and fad in the service of mental disorders. *Psychotherapy and Psychosomatics, 84*, 193-207. doi:10.1159/000371714

Thibault, R. T., Lifshitz, M., & Raz, A. (2016). The self-regulating brain and neurofeedback: Experimental science and clinical promise. *Cortex, 74*, 247-261. doi:10.1016/j.cortex.2015.10.024

van der Oord, S., Ponsioen, A. J., Geurts, H. M., Ten Brink, E. L., & Prins, P. J. (2014). A pilot study of the efficacy of a computerized executive functioning remediation training with game elements for children with ADHD in an outpatient setting: Outcome on parent- and teacher-rated executive functioning and ADHD behavior. *Journal of Attention Disorders, 18*(8), 699-712. doi:10.1177/1087054712453167.

van Zomeren, A. H., & Brouwer, W. H. (1994). Behavioral and social consequences of attentional deficits. In *Clinical Neuropsychology of Attention* (pp. 179-197). New York : Oxford University Press.

Vollebregt, M. A., van Dongen-Boomsma, M., Slaats-Willemse, D., & Buitelaar, J. K. (2014) What future research should bring to help resolving the debate about the efficacy of EEG-neurofeedback in children with ADHD. *Frontiers in Human Neuroscience, 8*(321), 1-6. doi:10.3389/fnhum.2014.00321

Wangler, S., Gevensleben, H., Albrecht, B., Studer, P., Rothenberger, A., Moll, G. H., & Heinrich, H. (2011). Neurofeedback in children with ADHD: Specific event-related potential findings of a randomized controlled trial. *Clinical Neurophysiology, 122*(5), 942-950. doi:10.1016/j.clinph.2010.06.036

Westerberg, H., & Klingberg, T. (2007). Changes in cortical activity after training of working memory. *Physiology & Behavior*, *92*(1-2), 186-192. doi:10.1016/j.physbeh.2007.05.041

Woodman, G. F. (2010). A brief introduction to the use of event-related potentials in studies of perception and attention. *Attention, Perception & Psychophysics, 72*(8), 2031–2046. doi:10.3758/app.72.8.2031

Wyckoff, S. N., Sherlin, L. H., Ford, N. L., & Dalke, D. (2015). Validation of a wireless dry electrode system for electroencephalography. *Journal of NeuroEngineering*, *12*(1),

95. http://dx.doi.org/10.1186/s12984-010089-2
Zelinski, E. M. (2009). Far transfer in cognitive training of older adults. *Restorative Neurology & Neuroscience, 27*(5), 455-471. doi:10.3233/RNN-2009-0495.
Zuberer, A., Brandeis, D., & Drechsler, R. (2015). Are treatment effects of neurofeedback training in children with ADHD related to the successful regulation of brain activity? A review on the learning of regulation of brain activity and a contribution to the discussion on specificity. *Frontiers in Human Neuroscience, 9*, 1-15. doi:10.3389/fnhum.2015.00135

NEUROPLASTICITY IN THE VORTEX OF ADOLESCENCE: Mind and Matter

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Abstract

With a focus on adolescence, this article discusses rewiring our brains through the mind-brain connection, cultivated through metabolic and social interventions. The fertile area of neuroplasticity examines the brain's inherent capacity to transform itself, together with manifold implications for health and adaptation. Neuroplasticity is also a *raison d'etre* of psychotherapeutic methods. Dedicated researchers transcended objection and controversy, to persuade the scientific community that the brain, like the rest of the body, enjoys plasticity throughout life—not just in childhood. Navigation across assessment and treatment paradigms presents neuroplasticity- from neuroscience, biochemical, genetic, and nutritional perspectives, in addition to psychosocial ones. Animal research and human clinical studies coalesce with mounting evidence of neuroplasticity. These studies also provide guidelines for expanded clinical applications. Case illustrations are briefly summarized, with implications for future directions in adolescent psychotherapy. Capitalizing on its tremendous energy flow and redirecting it constructively, adolescence is a prime neuroplastic stage, based on biological and psychological malleability.

So *doth* this conversation about neuroplasticity start:

For those who, like comedian Carol Burnett, view adolescence as
"just one big walking pimple," look at what sizzles inside.....

On the dark side of adolescence, William Shakespeare wrote:
"I would there were no age between ten and three-and-twenty, or that youth would sleep out the rest; for there is nothing in the between but getting wenches with child, wronging the anciently, stealing, fighting."

On its bright side, developmental psychologist G. Stanley Hall would retort,
"Adolescence is a new birth, for the higher and more completely human traits are now born."

Neuroplasticity (Greek: '*plastos*'= molded) is defined as the **reorganization of brain connectivity through experience**. In response to stimulation, nerve cells modify their activity. Neuron circuitry changes to sustain adaptation, learning, and general health. The term plasticity goes back to the work of two Italian neurologists: Eugenio Tanzi and Ernesto Lugaro. In 1893, **Tanzi** put forth a **neural hypothesis of learning and memory**. He proposed that **practice and experience promote neuronal growth** and shorten the tiny spatial gaps between functionally linked neurons, facilitating their interactions. Lugaro, meanwhile, drew upon his mentor Tanzi, and foresaw the chemical nature of central nervous system (CNS) synaptic transmission. Lugaro referred to nervous "conduction" and "transmission" as they are used today. In 1906, he **introduced** the term **plasticity** to the neurosciences. He understood that the anatomic-functional relations between neurons change adaptively for psychological maturation, learning, and functional recovery after

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brain damage[1]. In Spain, the father of neuroscience, Santiago Ramon y Cajal, used the term "neuroplasticity" to refer to non-pathological changes in the structure of adult brains[2]. Freud termed "mental plasticity" what he observed as an individual's ability to change through psychoanalysis[3].

The brain has an extraordinary ability to **modify its own structure and function** following changes elsewhere, in the body or in the external environment[4]. The brain's outer layer (cortex) is particularly able to make those adjustments. Plasticity underlies normal brain function such as our ability to learn and modify our behavior. It is strongest during childhood—explaining the fast learning abilities of children. A strong "second round" of neuroplasticity operates during adolescence. Thereafter, plasticity remains a significant lifelong property in the recovery from sensory-motor deprivation and brain injury (peripheral and central). Neuroplasticity is also implicated in alleviating chronic pain and the ability to use prosthetic devices such as robotic arms for paraplegics, or artificial hearing and vision devices for the deaf and the blind, respectively.

Neuroplasticity entails: neural **membrane** health, proper **neurotransmitter (NT)** levels, and **receptor** viability. It is influenced by the **immune system**, chronic **inflammation**, and **thoughts. Repeated reinforcement leads to strengthened neurocircuitry** patterns, as expressed through learned behavior. This quality manifests in the healing of psychiatric and neurodegenerative disorders, in humans and animal models. These dysregulations include: obsession, depression, compulsion, disorders triggered by psychosocial stress, and diseases such as Alzheimer's and Parkinson's. Recent research suggests **neuropathology** in such disorders is associated with a **loss of plasticity**.

The **old computer model of "fixed" neurons** depicted the brain having hard-wired connections. The adaptive, flexible software and information were distributed over a hard-wired network. Critical processes for brain maturation, such as neurogenesis, neuron migration and pruning were thought to stop at full development. Such model also neglected the links between physical illness, mental distress, and brain function or structure. In contrast, the **neuroplastic paradigm** drew support from the discovery of **neurogenesis in adult human brain.** Neurogenesis (neuron birth) is the process whereby neurons are generated from neural stem cells and progenitor cells. It is key to neural development. Most active prenatally, neurogenesis populates the growing brain with neurons. Hebb foreshadowed this model stating "strengthening synapses" without structural reorganization of cell assemblies. "Neurons that fire together stay together," he wrote[5], to characterize brain dynamics. Hebb proposed a "dual trace mechanism," whereby neuronal circuits that are interconnected and coactive change the efficacy of activated pathways in enduring ways. Inspired by the theories of psychologists and physiologists, Hebb took concepts from the molecular to systems level. His integration served as a springboard for research on learning and memory processes at all levels of brain function[6]. Over the last several years, studies found that stimulating neuronal communication through learning, psychotherapy, antidepressants, nutrition, or exercise enhances the growth of dendrites, axonal sprouting, and increases or strengthens synaptic connections.

Neural stem cells exist throughout life in the adult brain[7]**.** They create new neurons, astrocytes, and oligodendrocytes, just as in the developing brain. Stem cells were found in the **subventricular** zone (SVZ) near the caudate nucleus incorporated into the olfactory bulb[8,9,10,11,12]**.** Evidence exists from primates regarding neurogenesis in other ventricular areas and their incorporation into **cortical and subcortical** areas. Certain protein families, namely, Brain-Derived Neurotrophic Factor (**BDNF),** Vascular Endothelial Growth Factor (**VEGF),** and Insulin-like Growth Factor (**IGF), help regulate neurogenesis** through increased cell birth, maturation and survival. Those proteins also affect glial cells.

The **evidence base for neurogenesis** includes its links to structural plasticity[13]**,** axonal elongation and synaptic reorganization post injury[14,15,16] along with pre- and post- synaptic structural changes stemming from experience[17] at any age. Fetal tissue can be grafted into the adult intact brain. Further, even the damaged brain and spinal cord allow newly grafted cells to survive and differentiate[18]**.** Grafted cells for their part, receive and send connections. They also release NTs in a behavior-dependent manner[19]**.** Since **stem cells** also give rise to neurons, the damaged adult brain can potentially accept, if not participate in, the differentiation of developing cells into functional neurons.

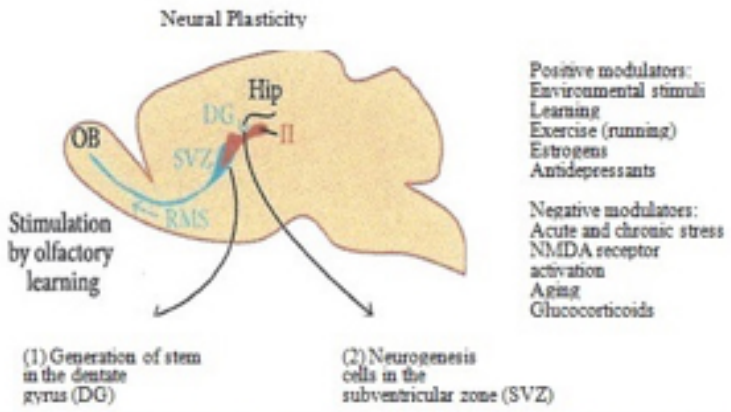

FIGURE 1: A schematic view on adult neurogenesis. (Fuchs E & Fluegge G, Adult Neuroplasticity: More than 40 years of research, Hindawi Publishing Corp., Neural Plasticity, vol 2014, Article ID 541870, 10 pages)

Neuroplasticity applies to neuron growth, synaptogenesis/synaptic pruning, and changes in the efficacy of existing synapses. These processes occur largely through **activity-dependent** mechanisms. Moreover, such plasticity is critical for **learning, memory, and recovery** as the brain constantly adjusts to environments or experiences. For example, cognitive training expands cortical areas, enhances neuronal organization, and neuronal dendrite branching, while increasing the connections and neuronal survival rates upon injury. Among preterm babies, impaired neuroplasticity may underlie cognitive and motor skills deficits, with lasting effects into adolescence[20]**.**

There are three types of neuroplasticity:

1) **Developmental**: immature brain cells being shaped by early life experiences;
2) **Activity Dependent**: from years of intensive practice of a skill (e.g. musical), learning and memory, or new connections emerging through experience, knowledge acquisition, and life experiences;
3) **Injury induced:** altered balance of brain activity due to trauma

Developing children have more neurons and synapses than adults but lose many during adolescent pruning[21]. Pruning enables further CNS adaptation: highly utilized neuronal networks are spared and become more interconnected. After pruning, the capacity to change decreases in certain areas; e.g. compare language learning pre- and post- adolescence. Moreover, a study of 43 children and adolescents (ages 8 to 14) compared brain scans with their math performance as they grew up. Researchers focused on the brain features that aid math learning. They identified three brain regions that predict improvement in math learning: (1) the posterior parietal cortex, (2) ventro-temporal occipital cortex, and (3) prefrontal cortex. Volume variations between individuals indicated that increased gray matter in those areas correlated with better math performance over time. But beyond those areas, the connection networks among them mattered most[22].

Activity-dependent plasticity reflects brain changes as a result of repeated practice over time. A study of the functional Magnetic Resonance Images (fMRI) from London cab and bus drivers showed they had a larger right posterior hippocampus (parietal lobe) than matched controls. The volume of that brain region correlated with years of experience/ proficiency[23]. In geriatrics, research showed that training reversed aging by 7 to 14 years with regards to visual concentration, reasoning and memory**.**

Post brain injury, physical, cognitive, and behavioral recovery continues for as many as five years, on a continuum. Cognitive rehabilitation supports plasticity, physiologically and/or structurally altering the brain. **Neural organization can be enhanced through specific training** rather than general experience, as demonstrated by neuropsychological testing and SPECT scan (Single-Photon Emission Computed Tomography) data; i.e., regional cerebral blood flow[24].

Factors affecting neuroplasticity can be grouped as **facilitators and suppressors of neurogenesis**. Facilitators are a motley group, including environmental enrichment, diet, exercise, learning, and antidepressants, including Lithium. In contrast, chronic stress, depression, and illness inhibit plasticity. Sensory and motor experiences can be either positive or negative in relation to plasticity.

Animal research documented acute and chronic stress effects on neuroplasticity. Both aversive (-) and rewarding (+) stress lead to hypothalamic-pituitary-adrenal (HPA) axis activation, with opposite outcomes on hippocampal plasticity, as measured by neurogenesis, dendritic branching, number of spine synapses, and so on. While chronic, unrelenting stress leads to deleterious effects, successful coping with intermittent and social stress is positively correlated with neuroplasticity[25,26]**.**

In humans, clinical psychiatric research illustrates neuroplasticity. For instance, in obsessive-compulsive disorder (OCD), dysfunctional brain network interactions may be due both to inefficiencies and dorsal anterior cingulate cortex (dACC) hyperactivity. Treatment approaches improve dACC modulation of cortical, striatal, and thalamic areas through interventions such as cognitive-behavioral therapy (CBT), mindfulness, and neurofeedback.

Adolescence presents enhanced **cognitive complexity and plasticity.** Along with a major learning spurt arrives vulnerability. Neuroimaging research (fMRI) documents **increased** c**onnectivity** among brain regions. Furthermore, the **balance among frontal** (executive-control) and **limbic/striatal** (emotional) systems changes to favor frontal influences. The **orbitofrontal cortex**, ruling motivation, is strengthened. The executive function is thus consolidated[27,28]**.** From a sociocultural angle, today's adolescents are highly influenced by digital technology (techno-social, internet), extended school learning, and the lack of practical training. The modern teenage brain thus has a **slower development of the pre-frontal cortex** than before due to a shift away from an apprenticeship mindset. Because of the information revolution, children assume adult roles later. Puberty tends to arrive earlier in most cultures, and teenage "weirdness" happens in the transitional period [29]**.**

During adolescence, changes in two brain control systems take place. One is the **limbic** system, which rules emotion and motivation. For example, a quiet 11-year-old turns into a restless, driven teenager, only to calm down again as an adult. Moreover, reckless adolescents overestimate rewards, such as the highs of a first love or a sports match[30]. Risky behavior is often tied to expected peer recognition. One study simulated a high-risk driving task while the adolescents were lying in fMRI brain-imaging machines. The brain reward system lit up more when the subjects thought a teenager was watching them. Hence, they took on greater risks[32,33]. The key motivator was a **social reward,** namely, peer respect**.** A

second brain control system that changes is the **prefrontal** cortex, which governs impulse inhibition, decision-making, long-term planning, and delay of gratification**.** This system evolves as the adolescent gathers and directs energy, becomes increasingly effective as childhood progresses, and develops further in adolescence and into adulthood through **learning from experience**. This is also a crucial stage for habit formation[34]**.**

Hunter-gatherer and farming societies typically educated their young through formal and informal **apprenticeship under adult supervision and protection**. In contrast, our highly technological societies offer adolescents fewer **opportunities to practice** basic living skills such as cooking, care-giving, or tending to the land[35]**.** Adolescents are inundated with academic courses, homework, and exams. Few of them take newspaper routes, baby-sitting jobs, or help out at home. But knowing physics and chemistry does not a soufflé make...

Earlier and contemporary cultures nurture different kinds of intelligence. While the former emphasized finely-honed and skill-specific expertise, the latter foster **broad-based, flexible** learning. Throughout most of our history, children started their internships when they were age seven. Today's youths go through **longer, protected immaturity and dependence,** with specialized training into their late twenties and early thirties**.** Although **advanced** schooling and knowledge beget higher IQs, a **higher IQ** also correlates with delayed frontal lobe development**.** Furthermore, adolescents nowadays experience a deep transformation in their **sense of social self,** encompassing belief systems, favorite music, fashion preferences, and group belonging. Their need for peer affiliation coexists with heightened sensitivity to being excluded or ostracized. This transition takes place in the context of intense activation of the medial pre-frontal cortex[36,37]**.**

A protracted adolescence impacts neuroplasticity since the **maturation of control systems depends on real world experiences** in real time. Hence, adolescents face the problem of developing an "accelerator" long before they can properly steer and brake. They have a high drive for sex, power and recognition, without the expertise and self-control it takes to avoid fallouts such as unwanted pregnancy or violence. More young people entering adulthood exhibit a **lack of direction and the inability to commit** to specific work or a particular love until well into their thirties. Since real world activities stimulate adolescent synaptic plasticity, it is crucial to offer adolescents real life "**apprenticeship"** opportunities, rather than overloading them with extracurricular activities and homework. Some examples are: supervised community-service programs (big brothers and sisters, seniors), events involving "Take your child to work," having college undergraduates watch and help scientists or scholars in addition to listening to their lectures, internships in science or government agencies, summer jobs carrying responsibilities alternated with travel, working on a farm, and summer camps.

Another factor affecting adolescent neuroplasticity is experimentation with recreational substances[38], marihuana and hashish being the most commonly used. Animal research on adolescent rats treated with tetra- hydrocannabinol (THC) from days 35 to 45 showed that THC pretreated rats performed worse than controls on tests of aversive and spatial memory using the passive avoidance and radial maze tasks. Structurally, THC pretreated rats established fewer synaptic contacts and/or less efficient synaptic hippocampus connections [39]. Among adolescent boys, marijuana use combined with high genetic risk for schizophrenia, was associated with lower cortical thickness, especially in areas dense with cannabinoid-1 receptors (CNR1)[40]. High CNR densities are found in associational cortical regions of the frontal and limbic regions. CNR binding sites are primarily found in the following areas:

1) forebrain regions associated with **higher cognitive** functions;
2) forebrain, midbrain and hindbrain areas associated with **movement control**;
3) hind-brain areas associated with control of **motor and sensory functions of the autonomous nervous system** (ANS).

Those structural brain changes from THC use thus impact behavior, learning, and self-control.

Adolescents often consume **alcoholic beverages**. In one longitudinal study, two to six MRIs (Magnetic Resonance Imaging) were done with children between ages 12 and 24. They found gray and white matter volume changes in 134 adolescents after 3 1/2 years. The follow up lasted 8 years. Among the 75 heavy drinkers in the group, there was decreased gray matter in cortical lateral frontal and temporal volume, and attenuated white matter growth of corpus callosum and pons. In late adolescence, there was diminished white matter growth, with its implications for neuronal connectivity and hence, overall mental function[41]**.**

During adolescence, the **second separation-individuation** takes place in the emotional/interpersonal arena[42]**.** Interestingly, **immune processes**, which set up boundaries between self- and non-self biologically are the strongest at this stage. Hence, adolescence can be physically the healthiest time of life. Mentally, it is a period of **learning to survive independently** in any environment, but also with exposure to **heightened risk.** Those physical and mental factors interact closely with family and life experiences. There are normal mood fluctuations and occasional poor judgment. But from there, several pathologies can also arise, such as anxiety disorders, bipolar disorders (BD), psychoses, and substance abuse. The neural circuitry in moodiness may differ from major depression (MDD) or bipolar disorder (BD). One also sees in adolescence, the rise of somatoform disorders involving pain (head, stomach, etc), fatigue, and eating/ nutrition. It is possible to assess brain networks by looking at connectivity, which depends on proper activation and communication among default neuron networks (DNN). Network engagement is determined through qEEG profiles in task-active states. Those profiles predict cognitive and behavioral deficits in complex behavioral phenotypes. A key **biomarker of emotional disorders is disconnection** among networks.

Epidemiological research showed that 21% of children (9-17 yrs) in the US are diagnosed with “mental illness.” Under this rubric were also included: suicide attempts, drug use, low performance, interpersonal issues, legal problems[43,44,45]. The most effective treatment approaches are multimodal, emphasizing prevention and early treatment. Biopsychosocial interventions normalize brain structure and function, correlating with mental/emotional and behavioral changes[46]. Both pharmacological and nonpharmaco-logical interventions with adolescents modify brain structure and function, per a research review of 50 studies (1966-2012) using structural MRI, diffusion tensor imaging (DTI), functional magnetic resonance imaging (fMRI), and magnetic resonance spectroscopy (MRS) in youth with selected psychiatric disorders including anorexia nervosa (AN), ADHD, autism, BD, depressive disorders, OCD, & schizophrenia. But the multi-factorial effects of medications on brain development remain largely unknown.

One AN study illustrates multimodal therapies, including medication (selective serotonin reuptake inhibitors - SSRIs), nutrition, a standard behavioral program to improve eating patterns and weight, and individual/group cognitive therapy. Differences between AN and controls were found in activation, especially at the temporal superior gyrus, which faded after treatment. Moreover, pre-treatment correlations between such activation and body mass were negative, while activation was positively correlated to depressive symptoms [47]**.**

Among psychotropic drugs effects, Lithium was found to normalize amygdala and hippocampal volumes and correlated with clinical improvement. Other mood stabilizers, stimulants, atypical antipsychotics (AAP), and anti-depressants (AD) balanced functional activation per neurocognitive tasks[48]. Sertraline (Zoloft) may alter brain structures differently in animals

and humans. Among depressed animals, this AD led to increased anterior cingulate cortex volume. In non-depressed animals, however, Sertraline was linked to decreased anterior cingulate and hippocampus volumes. These areas are interconnected with key functions such as memory, will, learning, spatial navigation, motivation, and emotion. On the other hand, among depressed humans, Sertraline administration correlated with reduced volumes of the cingulate cortex and hippocampus. Furthermore, Sertraline promoted neuron growth and connectivity[49]**.** SSRIs are also prescribed for disorders including bulimia, obsessive-compulsive disorder (OCD), and posttraumatic stress disorder (PTSD). In relation to those conditions, there are no studies on Sertraline's effect on brain volumes. AAPs are increasingly used with non-psychotic youths. While brain volume reduction secondary to such medication use may correlate with better connectivity and cognitive performance, the AAP effects during adolescent brain development and among non-psychotics remain unknown[50]**.** Much needs to be learned about psychotropic drug effects on brain plasticity[51]**.**

Nutrigenomics also has effects on neuroplasticity. One such example is a paraphilias case in a young male, complicated with Tourette's syndrome. He had responded to ropinorole (Requip, a non-ergoline, dopamine agonist [DA]) but because of an insurance infraction, it was discontinued (D/C). Knowledge of DA modulation led to try NAAT (nutrigenomic amino acid therapy). Within two weeks, the patient's behavior was normalized[52]**.** The writer has several adolescent cases from her practice to be discussed later within a holistic psychotherapeutic framework. In several instances, individualized free form amino acid formulae, diet and micronutrients sizably improved concentration, mood, school performance, and social interactions.

A mental/behavioral health focus on adolescence considers the brain as dynamic, flexible, and adaptable. Psychotherapeutic methods restore inner balance, and successful outcomes entail positive brain changes. Giants of neuroplasticity have recognized that the **mind changes the brain's messages to the rest of the body**. The mind is a major player in rehabilitation of serious physical conditions, from trauma to cardiovascular conditions and degenerative ones. The mind is also key to psychotherapy for emotional disorders.

Neuroplastic therapies address the brain's cellular health (inflammation) and specific wiring issues (e.g., diet and detoxification). In the area of **neurologic/behavioral rehabilitation**, several groups exemplify neuroplasticity at work: **Bach y Rita's** stroke rehab through daily brain stimulation[53]**,** and **Taub's Constrained Induced Movement Therapy (CI)**[54] for stroke patients, based on animal experiments. Taub's monkeys resumed using their deafferented arm if their good arm was constrained. There is also the **Feldenkrais method**[55], based on awareness through movement**.**

Of equal caliber are contributions from **De Tomatis'** auditory training (listening), which exposed patients to proper frequencies and thereby they attained *permanent* improvements in listening (brain) and voice[56]. Further, **Merzenich** created a program of **intensive** cognitive training for traumatic brain injury (TBI), and to optimize cognition among the aged. His TBI work with owl monkeys showed cortical reorganization after new areas took over a transected nerve or an amputated finger[57]. In the mid 1990s, **Ramachandran** explained "**phantom limb"** through homunculi rearrangement after cortical reorganization post injury. He demonstrated that somatosensory remapping took place by testing a 17 year old boy who had recently lost his left arm just above the elbow in a car crash. That boy felt the missing appendage. His injured brain rewired itself to receive input from alternative, healthy sources. Phantom sensation thus reflects neuroplastic changes in brain[58]**.**

Common to the above neuroplastic programs are essential components of the therapeutic process:

1) **NOVELTY,** which engages neuropeptides;
2) **SURPRISE**, a key to mind change. **Awareness** is used to differentiate one's brain maps and rewire the cortex;
3) **THINKING,** which can set hearts racing, hormones surging or, alternatively, it induces calmness.

Over time four major types/levels of neuroplastic change occur:

- **Immediate** (within minutes): after cognitive or physical exercise;
- **Synaptic** (days to weeks): new and more lasting connections (stronger, more efficient conduction along axons); e.g. better sleep, speech, balance, gait (begins to change pathology);
- **Neuronal** (after more than one month): throughout the neuron, consistent activation over 28 days allows neurons to produce new proteins & internal structures; e.g. ride a bike, improve vision;
- **Systemic** (months to years): consolidated new networks, the system functions fully and self-corrects.

Learning strengthens synapses when pre- and post-synaptic neurons are simultaneously active. Thus one cell's activity is more likely to cause the others to fire. For example, an "enriched" environment leads to improved behavior. Eric Kandel's work on memory, a prime example of neuroplasticity, won him the 2000 Nobel Prize for Medicine. In his study of the giant snail Aplysia, he described several steps in the formation of long-term memory, a critical one being **new protein synthesis** which, in turn, is necessary for establishing new synapses and synaptic connections[59]. Further, Kandel and his colleagues noted that experience during critical periods of development from birth into adolescence powerfully impacts neuronal circuits since the cellular and molecular conditions are optimal for plasticity. In addition, a long-term pattern of connectivity does not yet exist[60]**.** While this does not negate adult plasticity, it reasserts the notion of **adolescence as a timely period for resetting connections**.

For the treatment of mental disorders, two programs of mindfulness-based cognitive therapy exemplify neuroplasticity. First, Teasdale in Cambridge, UK[61], designed a program for depression involving two-hour sessions for 8 weeks. He outlined "triggering cues" resulting in depressive processing of emotion-laden thoughts. His program teaches to cultivate the "Impartial Spectator," mindfully aware. The therapist helps patients to encode in memory alternative thought patterns activated by same cues that tap into despairing states, also offering attention skills to disengage from depressive ideation. Focus on breathing is part of the process.

Second is the Schwartz/Gorbis program for OCD at University of California, Los Angeles (UCLA)[62]. They assessed 18 OCD Pts through PET and fMRI scans, and measured cerebral blood in three ***overactive*** areas that are interconnected, namely:

(1) **inferior prefrontal cortex** (detects something is wrong);
(2) **striatum** (motor response, thought/emotion);
(3) **anterior cingulate gyrus** ("gut-churning," doom if fail to act on compulsion).

Their program teaches patients to **reinterpret the environment and willfully change** patterned behavioral responses to disturbing feelings. In OCD, the caudate nucleus does not properly perform its gating role. Key neuroplastic changes through psychotherapy include:

- Calming the "worry circuit" enhances **caudate gating**;
- Mindful awareness increases **blood flow and function in the anterior cingulate**

cortex. This area mediates empathy, social awareness, intuition, compassion, and emotional regulation.

The mind-brain work of the UCLA group stands on **cognitive strategies** known as "**the 4 Rs**": re-labeling, re-attributing, re-focusing, and re-evaluation of the obsessive ideas. Self-directed, bi-directional processes thus alter brain function**. Mental energy expresses itself through the brain**, activating a given circuit preferentially. The psychotherapy programs described here are applicable to other disorders, and can also be adapted to adolescents.

From a **biochemical** perspective, neuroplasticity involves **brain signaling molecules** that participate **in axon targeting, neuron growth, and synapse maturation,** namely:

1. Nerve growth factor (**NGF**) (Levi-Montalcini)[63,64,65] **;**
2. Brain-Derived Neurotrophic Factor (**BDNF**)[66,67]**;**
3. **Neurotrophins 3 and 4**[68]**.**

BDNF is key in modulating synapse growth, promoting an increase in dendritic spine density, and enhancing synaptic receptor density. **BDNF, pre- and post-synaptic**, is a molecule of high interest here. Pre-synaptic BDNF signaling promotes NT release. Post-synaptic BDNF signaling enhances ion channel functions, including the -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor, the NMDA receptor, transient receptor potential cation channels, as well as sodium (Na) and K (potassium) channels. Moreover, **BDNF acts at excitatory and inhibitory synapses**. It may modulate both spontaneous and stimulated neuronal activity[69,70]. BDNF action on synapses occurs within seconds of its stimulation or application/ release. When neurons connect to form networks, BDNF provides a basis for turning contact between two neurons into a lasting "dance." **BDNF enhancement** is an essential, **modifiable factor of neural network formation and plasticity.** Different **ways to upregulate BDNF transcription include**: psychotherapy, voluntary physical exercise, intellectual stimulation, caloric restrictions, omega 3 DHA foods and supplements, glutamate, curcumin (in turmeric), antidepressants, and sleep. For the above reasons, BDNF is a target for future drug development[71,72]**.**

Among the micronutrients, **vitamin B12** (cobalamin, Cbl) as methylcobalamin partakes of BDNF transcription[73,74]**.** B12 is indispensable for proper **brain development, function**, and in metabolism. The molecular basis of its neurotrophic effects remains hypothetical in the absence of an efficient cell model to research the consequences of B12 cellular deficiency in neuronal cells. However, it is known that two enzymes are B12-dependent in mammalian cells:

1) mitochondrial enzyme l-methylmalonyl-CoA mutase, &
2) cytoplasmic homocysteine methyltransferase (methionine synthase).

Two direct effects of B12 deficiency are: the accumulation of methylmalonic acid (MMA) [pernicious anemia] and homocysteine (Hcy) [cardiovascular risk]. B12 has two active coenzyme forms, methylcobalamin (MeCbl) and adenosylcobalamin (AdCbl). A paradigm shift in B12 deficiency therapy led to the more widespread use of MeCbl. Both MeCbl & AdCbl have distinct metabolic roles. **MeCbl** is involved along **with folate in hematopoiesis and brain development** during childhood**. AdCbl** deficiency disturbs carbohydrate, fat and amino acid (AA) metabolism, **interfering with myelin formation.** For therapeutic purposes, a **combination of MeCbl and AdCbl** or hydroxocobalamin is desirable. The **sublingual** route compares with intramuscular (IM) applications for B12 deficiency. Dosing is individualized[75]**.**

A link between BDNF formation and SSRIs was proposed to explain the lag between the

onset of antidepressant (AD) therapy and patient improvement. Depressed patients showed a lack of cell division in the hippocampus. Those cells renewed their division just when patients noticed mood-shifting effects, about two weeks after starting SSRI therapy[76]. This finding challenges the notion that depression is a mere monoamine (MOA) imbalance. Researchers increasingly underscore the inhibition of neuronal growth by stress hormones as the source of depression, which sustains hypothalamic-pituitary-adrenal axis (HPA) overdrive. Energy generation through mitochondrial function must also be considered[77]. In addition, B12 deficiency was linked to a lack of response to antidepressant treatment[78]. From a **genetics** angle, **long-term neuroplasticity is mediated by altered gene expression,** as seen in **learning, memory, chronic stress**, and **drug abuse** effects on reward-related circuits. The process goes as follows: stimulation activates second messenger pathways, enhancing transcription factor activity at gene promoters. This in turn leads to the expression of new growth factors, ion channels, structural molecules, and other proteins altering the neuronal circuit. Repeated stimulation leads to more permanent **changes to transcription factors and chromatin structure,** selectively sensitizing or desensitizing a circuit. **Neuroplasticity** studies are uncovering molecular mechanisms underlying **long-term brain changes,** including the epigenetic modulation of mood disorders[79]**.**

Mental and behavioral health implications from all the above perspectives are **applicable to adolescents**[80]. Brain activation in emotional disturbances requires the consideration of sympathetic (SNS) vs parasympathetic (PNS) factors. Treatment aims at autonomic (ANS) balance, by modulating visceral, rational, and spiritual components. Successful, traditional psychotherapy is associated with brain changes[81]**.** Furthermore, meditation practices interwoven with psychotherapy approaches, **strengthen the anterior cingulate and calm the amygdala**[82]**.**

Certain correlations between mental states and the brain are well-established:

- **Anger shuts down communication to the prefrontal cortex** via the anterior cingulate. Under those conditions, emotion and fear determine behavior;
- **A consistent focus on spiritual values and goals** enhances blood flow to the frontal lobes and anterior cingulate, which in turn decreases activity in primitive emotional brain centers;
- **The anterior cingulate area mediates the perception of self, actions in relation to others, and the world** at large**.**

To revisit Hebb's statement, "Neurons that fire together, wire together": Mental states and activities that facilitate the formation of cell assemblies and foster connectivity are keys to maturation and mental health.

Mindfulness, for example, creates a context to experience **neuroplastic self-help**. The following instructions can be used with adolescents and adults, also adapted to children:

- **Sit comfortably** and relax;
- **Close your eyes;**
- Perform **abdominal breathing**, paying attention to your nostrils;
- **Notice the sensation** of cool air entering, and warm air exiting your nose;
- **Focus on each breath,** staying relaxed and alert;
- **Notice whenever your mind wanders;**
- **Return to the breath sensation;**
- You are increasingly able to **stay present** by steadily returning to the present moment.
- **Practice:** 10-15 minutes one to three times daily for a month, as needed. At the end of that period, reflect on the changes that this practice has brought to you.

Adolescent Case Examples:
Several adolescent cases and two children from the writer's pediatric population illustrate neuroplastic transformations within a holistic psychotherapeutic matrix. In all instances, adaptations from yoga and mindfulness were woven into treatment. The immense plasticity of childhood is already well-known, as two examples with children illustrate. In both cases, treating B12 deficiency provided a bridge to mental and behavioral progress. The first child was a 7 year old boy diagnosed with ADHD and dyslexia. His parents refused stimulants. He was adopted at birth from a single mother who had a heavy family history of alcoholism. This child's blood chemistries showed elevated MCV, a sign of pernicious anemia overlooked in prior nutritional counseling. Organic acids testing (Organix profile) confirmed a functional B12 deficit per elevated methylmalonate levels. He was given sublingual B12 (methyl- and hydrocobalamin), and within 48 hours, the classroom teacher noticed improved handwriting (control and dexterity). Brief psychotherapeutic interventions with mother and child over a three-month period, along with a homeopathic remedy, *Pulsatilla,* placed this child on a healing path, manageable in the classroom, eager and ready to learn.

A second pediatric case, significantly more complex, was that of a 10 year-old diagnosed with an Attention Deficit Hyperactivity Disorder (ADHD) by a psychiatrist and psychologist, and placed on Adderall. After a brief medication trial, the child stopped it because in his words, "I did not like the way it made me feel. That was not me." A hitherto excellent student, athletically inclined, he was now overeating, overweight, failing academically, and the recipient of excessive peer teasing in school. Neuropsychological testing and a quantitative electroencephalogram (qEEG) documented that this was a very bright child with attention and performance problems secondary to depression. Family dynamics significantly influenced his emotional state and mental functioning. From a metabolic angle, an Organic Acids profile revealed B12 deficiency, along with an altered gastrointestinal (GI) flora, and imbalanced neurotransmitter turnover (dopamine, norepinephrine, and serotonin). Nutritional interventions, including the use of a free-form amino acid base, addressed those imbalances. He was also given a multivitamin, EPA/DHA gels, and probiotics. Psychotherapy with a cognitive-interpersonal focus, addressed his negative self-image and interaction patterns involving his father and peers. He passed his classes, and transferred to a school with individualized teaching in small classes. Within one year, he was again at the top of his class. He also overcame difficulties triggered by conflicts between his parents that severely distorted the relationship to his father. Ten years later he is close to graduation from a prestigious university, and has thrived socially.

Several adolescent case summaries attest to profound neuroplastic changes. Fictitious names were assigned to protect their identity:

Case 1: Pete, a 14 year old boy diagnosed with bipolar disorder (BD) had this history:
- failed every outpatient and inpatient program;
- suffered untoward side effects from several psychotropics, among them priapism from trazodone (Desyrel) during a psychiatric hospitalization at age 10 (!);
- remained viciously aggressive towards his nuclear family and was entrenched in his school refusal.

Molecular nutrition testing (Individualized Optimum Nutrition [ION] Profile) showed impaired mitochondrial function, imbalanced neurotransmitter turnover (excessive Vanilmandelate [VMA] and deficient 5-Hydroxyindoleacetate [5-HIA]), and oxidative stress, consistent with findings on mood disorders, along with a disturbed microbiome (bacterial overgrowth and digestive issues).

Upon starting tasty lozenges of 100 mg CoQ100 daily, he mustered up the energy to attend a specialized public school for emotionally disturbed students. CoQ targeted mitochondrial health, indirectly benefiting NT production. He was given a customized micronutrient program, including free-form aminoacids and elemental lithium (lithium orotate) in the mix. Digestive health was further supported with an individualized diet, herbs, and probiotics. On retest, one and a half year later, several biomarkers had improved. Most strikingly, there was a significant increase in 5-HIA while VMA was now within normal limits. This corresponds to greater serotonin (SE) availability, which helps modulate other NTs. Interestingly, there was no specific intervention addressing SE production or reuptake but a metabolic pathway improvement.

Psychotherapy was used with positive results. Pete's father came alone to an initial interview, in which he challenged this writer to perform "a little miracle" with his son. Regular sessions with the mother alone (father was too busy) facilitated the discussion and understanding of Pete's communication and actions *vis a vis* the family. Plans for home behavior management were formulated. Periodically Pete himself had individual sessions that combined cognitive rearrangement, breathing methods, and body-mind awareness. There was also an examination of family dynamics, conflicts, and strategies for their resolution. Later in the process, Pete was able to reflect on his exceedingly self-critical attitude that bred inner distress and violent reactions.

The later introduction of homeopathy, *Tuberculinum bovinum,* catalyzed major progress. After the first year of treatment, favorable outcomes occurred in succession for Pete:

(a) he transitioned to a regular public school;
(b) became a star athlete, earned top academic honors; and
(c) earned an athletic scholarship to attend a top US university;
(d) successful college graduation.

As he entered young adulthood, Pete developed a significant romantic attachment. He has experienced a fulfilling career, created his own social network, and has been able to overcome "rough spots" on his own for the most part. The nuclear family, including his parents and two brothers, provided a safety net for Pete, and he remains close to them. He recently went back to the school for children with emotional disabilities, which he attended for one year, and gave an inspirational speech. Currently he has an excellent job and a serious romantic interest.

Case 2: Alice, a 19 year old college sophomore, was rushed home from her Ivy League college in a severely depressed state. Her father emphatically rejected drug treatment for his daughter. She was diagnosed as having a dysthymic disorder. Her crisis was precipitated by a mismatch with her academic advisor, who was harshly critical of Alice and did not seem to see her talents. To break up a cycle of insomnia and self-deprecation, psychodynamic psychotherapy with specific cognitive interventions was started, along with yoga breathing and relaxation methods for home practice. A homeopathic remedy, *Natrum muriaticum*, in successive potencies, addressed the chronic undercurrent of self-loathing, negativity, pessimism, and feelings of loss that had plagued her throughout high school. Basic nutritional interventions with diet and supplements enhanced Alice's energy and cleared her skin, since she had chronic acne that further impaired her self-esteem. She returned to college in the Fall, transformed, and continued to work on herself independently. She thrived academically and socially. For two years she was seen only during seasonal school breaks. During her year abroad in Europe, she met a young man with whom she developed a steady relationship that culminated in marriage upon graduation.

Case 3: Janette, an 18 year-old college freshman, developed panic attacks, aggravated before exams, when she went away to college. Underneath her fears and bulimia there was a profound separation anxiety disorder. She was placed on sertraline HCL in collaboration with her internist. Janette refused Clonazepam for her insomnia upon reading about its side effects, and was given a botanical, Passionflower, with equivalent benefits. Since she also suffered from bulimia, her diet was equally addressed. On campus, she attended a bulimia support group with meager results. Behavioral therapy for bulimia by a college counselor did not work either. Based on molecular nutrition lab tests, a metabolic bridge was built with a custom amino acid base and individualized nutrition. She stabilized and utilized well a program of in-depth psychodynamic psychotherapy with added cognitive techniques for self-help.

After 8 months, Janette reached a ceiling on antidepressant benefits. Although she was calmer and more focused at 50 mg qam, once sertraline was raised to 75 mg qam, she developed noticeable CNS (dizziness, tremors) and ANS side effects (sweating). At 100 mg those side effects became intolerable. Hence, the AD was tapered off. She remained on a combination of nutrients and homeopathy, in addition to regular psychotherapy. She made a romantic attachment, developed some friendships, and graduated college with high honors. The selected homeopathic remedy was *Thuja occidentalis* in various potencies. Her romantic relationship later broke up but she eventually worked through the loss. For three years Janette alternatively lived with roommates and with her parents. She also decided to postpone graduate school and secured a job. At that point, psychotherapy was terminated by mutual accord. However, her "identity diffusion" in the Eriksonian sense, remained within her. This case shows a gifted young person with a deeply conflicted personality, who will likely benefit from further psychotherapy once she is again receptive to it.

Case 4: Beth, a 15 year old girl, was truant from school, also experimented with drugs, alcohol, heavy smoking, and got into physical fights with other girls. She also suffered from disrupted sleep but neurological studies yielded negative results. She was thought to have an oppositional defiant disorder, with significant mood changes. Her mother wondered whether she would graduate from high school. Neuropsychological testing demonstrated ample talent to do so and to pursue a higher education. The therapeutic program included psychotherapy, basic dietary rearrangements, along with yoga breathing and relaxation methods. Despite a bumpy road, she finished high school at a small private school. She overcame an incident of being raped at a part-time summer job, and moved on valiantly. While she attended college overseas, Beth's transformation unfolded. After a rough first year, her grades improved, she found a romantic interest, studied harder, was followed up on a yearly basis during summer. She completed a master's degree, got married, had two children, and is working at a prestigious organization.

Case 5: Ed was a 13 year old boy with severe behavior, mood, and academic problems. He bore multiple diagnoses: ADHD, bipolar disorder, Tourette's syndrome, and PANDAS (Pediatric Autoimmune Neuropsychiatric Disorder, Alpha Streptococcus). Upon several psychiatric hospitalizations, he returned to school with untamable aggressiveness and restlessness. On the verge of failing academically, he was close to being expelled due to his unruly behavior, tantrums, and foul language. At intake, his psychotropic drug regimen included quetiapine, valproic acid, and a clonidine patch. Other medications that had been serially tried and discontinued at previous times. This youth also suffered from allergies and health challenges since birth. Ed attended a specialized school for bright children with learning disabilities and ADHD. Neuropsychological testing showed a gifted youth with attention and organization deficits—mild to moderate ADHD—that would complicate classroom participation but not interfere with learning. In fact, curiosity was one of his keynotes.

Because of the complex health history, there was a comprehensive nutritional evaluation followed by a therapeutic nutrition program. Psychotherapy began with hypnosis that helped him control his tics—"unexpectedly" for the family. Further on, the work included family relationships, supportive sessions for his mother, and a school-based social skills group. A homeopathic remedy, *Zincum metallicum*, in successive potencies, catalyzed Ed's treatment. Within months, his mental and physical health improved exponentially. His medications were tapered off in collaboration with the pediatrician. Not only did his school performance improved but he aced most subjects. One year later, at his own insistence and against the school staff's advice, Ed transferred to a most competitive public high school. There he made new friends, graduated with very high marks and was admitted to an excellent college. His multiple diagnoses faded in the background, with only periodic allergies remaining.

Case 6: Grace, a 17 year-old girl diagnosed with anorexia nervosa, resisted conventional treatments after hospitalization had enabled her to restore a minimum ambulatory health. However, she easily responded to yoga and biofeedback- assisted psychotherapy (temperature and skin conductance) for the normalization of vital signs, in combination with molecular nutrition and a homeopathic remedy, *Arsenicum album.* Psychotherapy combined cognitive methods with work on family dynamics, especially the mother-daughter dyad. She maintained weight during her senior year, graduated with honors, was admitted to one of the finest colleges in the US, and went on to study medicine. She became a fitness instructor on the side. Her academic and occupational choices enabled Grace to sublimate constructively a hitherto obsessive quest for health and correct a distorted body-image. Her family relationships improved, but peer socialization remained rather challenging. Grace declined a referral for continued psychotherapy at her college and maintained occasional contacts for three years, having "managed" emotionally on her own. Through physical fitness she functions well outwardly in spite of a rigid personality structure that calls for further interventions[83].

In view of the compelling treatment outcomes described above, one may ask: Can psychological therapies alone match the level of neuroplastic change promoted through a holistic approach?

The above youths presented with complicated disorders of the highest severity and significant chronicity, for which the standard care indicates combined psychotherapy and psychotropics. Medications created temporary bridges but did not shift the functional Gestalt. In certain instances, medications were poorly tolerated. Hence, the parents sought alternative interventions, especially in light of the limited evidence base for psychotropic drug treatment with adolescents. Mindfulness and yoga breathing techniques were interwoven with cognitive and dynamic psychotherapy to promote continuity between sessions and encourage self-regulation in daily life. Metabolic nutrition provided a solid substrate to the mind-brain-gut connection, and homeopathy catalyzed inner changes. In all combinations, the above cases illustrate **deep transformations throughout a turbulent stage of development**. Within a holistic psychotherapeutic matrix, those cases support a biopsychosocial and health-building model to harness neuroplasticity in adolescence, positively shaping personality, balancing mind-neuroendocrine immune (MNEI) connections, and facilitating the entry into adulthood.

Discussion:

Adolescence is associated with a shifting vortex of multiple pulls and contradictory currents that drive this stage, ultimately into an adult identity. Mind, body, and the social self metamorphose, emerging from the nuclear family. Each of these factors can be viewed as a vector carrying its own plastic force. Their reciprocal actions interact with genes to shape the individual manifesting in young adulthood.

In adolescence, the dawn of abstract thinking creates competing worlds with great appeal: fantasy/possibility *vs* reality, and mindlessness *vs* responsibility. Thereupon arise polarized constructs regarding basic needs and drives in a context of changing family relationships and an ever stronger focus on peers and the peer group. Experimentation and novelty attract. On a personal level, those "magnets" translate into infatuation, romance, and attachment. Alternatively, the adolescent may be caught up in seduction, deception, betrayal, disappointment, and hurt. Bullying in its various forms such as teasing, marginalization, and ostracism may produce scars into advanced adulthood.

Many factors make and shape the course of adolescence. Biologically, there are NTs and endocrine processes creating shifts that turn a child into an adult: maturation of the body and its functions, along with the capacity for reproduction[84]. But those variables do not a personality make. Habitual communication patterns and personal ties leave indelible marks, for better or worse. As Newberg wrote, "Words can change your brain"[85]**.** Words, as the meaning and connotation of language conveys, can alter thinking and change minds; hence, the reciprocal influences between people. The powers of suggestion in a vulnerable youth are enormous, at times placing that young person in an altered, trance-like state. Extreme examples are gang participation, and being subjected to physical abuse — sexual and/or aggressive, mental manipulation through mixed messages, most blatantly occurring in cults. Daily intra-familial communication leaves deep imprints on the young. **MNEI changes stemming from aberrant interpersonal experiences may carry over into adulthood**, complicating overall health and adaptation—extreme forms being found in PTSD and psychotic spectrum disorders. It is important to explore early history to get an accurate developmental picture. For early childhood depression causes changes in gray matter maturation later in childhood and in adolescence[86]. In schizophrenic disorders, delayed development of brain connectivity was found not only among the adolescent patients but also in their well siblings[87]**.** Effects from haloperidol taken in adolescence were also documented well into adulthood[88]**.**

Another biosocial phenomenon impacting adolescent brains today is the **widespread use of pharmacotherapy**, from medications with mood effects (acne drug Accutane, oral contraceptives) to psychotropic drugs themselves. Moreover, in many cases, polypharmacy is prescribed for their mental/emotional disturbances, as well as attention and academic deficits. Investigative reports point to a fallout from the current psychiatric practices, namely, growing ranks of chronically disabled individuals[89]. The suppression of unpleasant symptoms and appeasement without concomitant, therapeutic dialogue does not afford adolescents a fair chance to work through their inner conflicts- albeit intense, complex and annoying to those around them. Such treatments, minimizing psychotherapy, also curtail the acquisition of new skills and resourcefulness. Furthermore, the use of atypical antipsychotics (AAPs) during adolescence makes them more sensitive to other medications of that class in adulthood[90]**.**

Neuroplasticity permeates the adolescent's life. It is important to create a compassionate community around the adolescent. This means, across settings (family, school, peer groups), the stimulation of competence, openness to empathic connections, regular activity, food awareness, and fitness. Such infrastructure also reinforces peer friendships, strategic problem-solving, trust, fitness, nutrient dense meals, the measured use of computers and texting, discipline and responsibility. In order to promote mental flexibility and thinking skills, school programs need to consider the following issues: modulate rote learning, the quest for grades, and discourage bullying, while rewarding creativity, initiative, cooperation, and overall mastery.

A neuroplastic-friendly approach to adolescent psychotherapy balances boundary setting and self-expression, as a way to help the adolescent develop a positive identity. This has major consequences for adult functioning, including the development of healthy intimacy and a productive orientation. Those changes parallel the emergent dominance of the pre-frontal lobes over limbic structures, and the ability to balance thinking, feeling, and actions. Major life decisions and choices are made. Adolescence and young adulthood recap all prior developmental tasks in the Eriksonian sense, ushering in the lifelong evolution of identity, intimacy, and generativity, and the formation of interpersonal networks[91.91]. Today's globalization facilitates the cross-cultural spread of ideas and technologies through the media[93].

Several psychotherapies may work to support adolescent mind-brain transformation: a psychodynamic orientation, cognitive-behavioral methods (CBT), mindfulness-based, or experiential techniques and body-mind integration. Common to all is the impact of forming a relationship between patient and therapist. Regardless of technique, the awareness of interaction processes, in addition to intrapsychic ones, deserves discussion in the psychotherapeutic conversation. Such processes, in addition to the intrafamilial patterns and collegial ones deeply influence identity formation and shape neuroplastic changes.

Adolescence is considered a **sensitive period of brain development**[94]**.** Most available work covers the unfolding of early sensory, motor, and language abilities. But the teen years deserve equal scrutiny as they open "a second 'window of opportunity'" in connection with **memory, social stress, and drug use**[95]**.** Research with rodents, neuroimaging, and large-scale human behavior studies point to high neuroplasticity in adolescence. For example, adolescent rodent studies found significant effects from social isolation and reduced fear extinction, also suggesting that it is a critical period for the development of mental disturbance. Furthermore, the impact of sensitive periods is affected by sizeable individual differences. In animal and human studies, adolescent exposure to drugs such as cannabis may have deleterious effects on cognitive functions. Future work is needed, directly manipulating environmental input and comparing its impact on child, adolescent, and adult cohorts. Other major areas of interest in adolescent neuroplasticity pertain to chronic stress, adverse experience, exercise, nutrition, friendships, and psychotropic drug effects.

Chronic stress was found to have dual effects on learning and memory in adulthood**.** Acute stress can produce several cognitive impairments, but whether negative experiences continue to hinder individuals as they age is not as well understood. In contrast, chronic unpredictable stress during adolescence impacts learning and memory processes in adulthood. Using male Sprague Dawley rats, researchers found that adolescent stress affected adult cognitive abilities in a context-dependent way. Compared to rats reared without stress, adolescent-stressed rats showed enhanced reversal learning, which reflects behavioral flexibility. However, they showed no change in associative learning and reference memory abilities. Working memory, which underlies reasoning, mathematical skills, and reading comprehension, may be actually be enhanced by exposure to adolescent stress. However, when stressed adolescent animals were tested after a novel disturbance, they exhibited a five-fold decrease in working memory performance while unstressed rats continued to exhibit a linear learning curve. Maybe the unstressed rats had a "mind-brain reserve" that the stressed ones had already spent[96].

Studies of the hippocampus as a target of stress and stress hormones revealed structural plasticity in the adult brain. Recurrent stress produces shortening and debranching of dendrites in the CA3 region of the hippocampus and suppresses neurogenesis of dentate gyrus granule neurons. Both forms of structural remodeling of the hippocampus seem re-

versible. They are mediated by glucocorticoid hormones working in concert with excitatory amino acids (EAA) and N-methyl-D-aspartate (NMDA) receptors. The latter operate along with NTs such as SE and the GABA-BZD (gamma aminobutyric acid- benzodiazepine) system. Glucocorticoids, EAA, and NMDA receptors are also involved in neuronal damage and death in pyramidal neurons, secondary to seizures and ischemia. A similar mechanism may underlie **hippocampal damage from severe and prolonged psychosocial stress in adolescence.**

Magnetic resonance imaging has shown selective atrophy of the human hippocampus in a number of psychiatric disorders, accompanied by deficits in declarative, spatial, and contextual memory. Hence, it is essential to appreciate the role of hippocampal dysfunction in psychiatric illness. Therapeutically, one must discern a permanent loss of cells from reversible remodeling, in order to prevent or reverse deficits. While hippocampus remodeling may be only the tip of the iceberg, other brain areas may be impacted.

Exercise is a key factor of health throughout life. But **adolescence is particularly suited for physical activity** thanks to its leaps in physiological strength, endurance, and stamina. Physical activity awakens molecular and cellular processes enhancing neuroplasticity through growth factors. Specific examples include: neurotrophic factors (NFs), BDNF, insulin-like growth factor 1 (IGF-1), and nerve growth factor (NGF), among others. NFs are produced in central or peripheral tissues and released into the blood circulation. They cross the blood–brain barrier (BBB) and reach the brain. NFs activate a metabolic cascade with neurotrophic and neuroprotective effects. Once in the blood and through its high-affinity receptor, tyrosine kinase type 2 (TrkB), BDNF activates the cAMP response element-binding protein (CREB) transcription factor. CREB is responsible for the hippocampal expression of several genes required for learning and memory.

Adolescence is also a critical period for the emergence of psychiatric disorders, in which BDNF modulation plays a role. BDNF changes were studied in relation to psychotropic drugs and ECT. Low BDNF was found in the following disorders: depression, PTSD, schizophrenia, OCD, autism, bipolar disorder, addiction, and ADHD. Several antidepressants (AD), antipsychotics (typical and atypical), and euthymic drugs, as well as ECT were reported to enhance BDNF[97,98]. A Canadian study of adolescents examined psychiatric medication use beyond its BDNF beneficial effects. **Shared decision-making** was a positive factor in compliance with medication regimens since it enlisted the adolescents' cooperation. It also reinforced their ability to make decisions and assume responsibility for themselves. The **conversation about psychiatric drugs and their use may transform the developing adolescent's sense of self and social personhood**. That interpersonal process seems key to understanding the long-term effects of medications, including neuroplastic ones[99]**.**

A **school-based psychosocial program for adolescents** with neuroplastic benefits is "FRIENDS for Life."[100]**.** Two organizations, National Educational Psychological Service (NEPS) and the Social, Personal and Health Education Support Service (SPHE) implemented this program as a pilot project across 14 partner schools and 244 students. The World Health Organization (WHO) considers this to be the only **evidence-based program deemed effective for all levels of anxiety** (WHO 2004). Originating in Ireland, the program helps students handle worry, stress and change. Furthermore, it teaches necessary skills to decrease anxiety and foster resilience. Teachers applied the program in schools for anxiety prevention, in the classroom, with a small group, or with an individual student. The results showed anxiety reduction.

The word 'FRIENDS for Life' is an acronym that helps adolescents to remember the coping steps, namely:

F- Feeling Worried?

R - Relax & feel good

I- I can do it!

E - Explore solutions & coping step plans

N - Now reward yourself

D – Don't forget to practice

S - Stay cool!

The three main components in 'FRIENDS' stand on cognitive-behavioral therapy (CBT) principles:

(1) Learning/ Behavior: problem-solving, coping skills, gradual exposure to feared situations, self-reward for brave behavior, positive role models and support networks;

(2) Cognitive: positive self-talk ("green thoughts"), challenge negative self-talk ("red thoughts"), realistic self-assessment and reward;

(3) Physiological: awareness of internal body clues, relaxation, and self-regulation.

In the clinical setting, a CBT preventive program with adolescents yielded promising results[101].

In the epigenetics of adolescent neuroplasticity, **nutrigenomics** also figures prominently because the "second window" of adolescence provides a chance to remodel the mind-brain-gut connection and whole body systems functioning. Research has addressed the interfaces of genomics, cognitive science, and nutrition, without shedding light on the crossroads among those three disciplines. For example, one may ask about nutrition effects on the brain genome, or how someone's genetic make-up shapes their nutritional response with effects on cognitive performance. Puberty is one of the life stages in which epigenetic changes most often take place[102,103]**.**

During puberty, the growth spurt, roughly from 11 to 16 years of age, gives rise to **specific nutritional needs**. Monitoring nutrients such as iron and calcium is important. Adolescents are susceptible to iron deficiency anemia since their increased blood volume and muscle mass raise iron requirements in order to build up hemoglobin and myoglobin. For boys, the increase in lean body mass (LBM) is crucial. For girls, menarche leads to higher iron requirements. Since iron is essential for the transport of oxygen to the brain, a deficit produces fatigue and may lead to decreased mental performance. Iron-rich foods include lean meats and fish, beans, dark green vegetables, nuts and grains. Iron from animal foods (heme iron) is better absorbed than iron from non-animal sources. This places vegetarian adolescents at a disadvantage, although vitamin C intake (e.g. from citrus fruits, cherries, etc) helps absorb non-heme iron. In the case of calcium, the skeleton contains most of the

body's stores, and almost half of the adult skeletal mass is formed during adolescence. Dietary calcium is key for adolescence skeletal growth, and also for proper neurotransmission. Vitamin D and phosphorous support bone strength, and vitamin D acts as a neurohormone with effects on NT formation.

Dietary factors can affect multiple brain processes by regulating cellular energy, neurotransmitter pathways, synaptic transmission, membrane fluidity, and signal-transduction pathways. Examples of nutrients with significant impact on cognitive functioning are: **polyphenols**, which protect against neurotoxins and suppress neuro-inflammation, potentially enhancing memory and learning. A type of polyphenols, flavonoids impact long-term potentiation (LTP) by interacting with signaling pathways. Furthermore, flavonoids activate the extracellular signal-regulated kinase and the phosphatidylinositol-3 kinase/protein kinase B/Akt (Akt) signaling pathways. In turn, this activates the cyclic adenosine monophosphate response element binding protein, a transcription factor enhancing the expression of neurotrophins - e.g. BDNF—important in LTP and long-term memory. Flavonols, a type of flavonoid, comes from fruits, wine, beans, and cocoa. Generally, polyphenols are abundant in vegetables and fruit. Cross-sectional and longitudinal studies correlated a high intake of food-based flavonoids with improved cognition. Hence, the consumption of polyphenol-rich foods is highly desirable in adolescence. Translational work will shed light on the pharmacological potential of polyphenols regarding clinical conditions [104].

Another important nutrient class are essential fatty acids. For example, **omega-3 fatty acids** contribute building material to the brain, support intercellular signaling, and facilitate synaptic transmission[105]**].** In contrast, diets rich in sugar, saturated fats, or high fats increase oxidative stress and reduce synaptic plasticity. Further, being overweight correlates positively with poor academic performance. Eating breakfast daily supports cognitive performance. **Brain morphology** responds to specific stimuli during the lifespan, reflecting high plasticity during adolescence. The brain, moreover, accounts for a high percentage of the total metabolic rate. Nutrition can potentially influence functioning from moment to moment, in addition to modifying brain architecture[106]**.** Non-invasive imaging techniques have clearly demonstrated that simply thinking about food modulates neural activity in specific brain areas implicated in the cognitive control of appetitive behaviors, leading to physiological responses such as saliva and gastric acid secretion. In tandem with nutrition, exercise enhances brain functioning, and decreases the unhealthy effects of a high-fat diet.

During adolescence, healthy eating entails nutrient-dense, balanced meals, regular eating patterns, and snacks. Stress and emotional problems alter energy balance, and may lead to an inappropriate food intake. Infections, anxiety, menstruation, or skin problems can alter appetite. Emotional imbalances coexist with food faddism and slimming trends, or worse, may end as eating disorders (anorexia nervosa, bulimia). Diet templates have been put forth[107]**,** but nutrigenomics uses the **N of 1 approach** to optimizing each adolescent's potential. Assessment of the gut-brain connection through molecular laboratory tests and interventions through probiotics and nutrition represent a promising area for clinical work. Thus more direct data would exist on the links between nutrition, mental functioning, and neuroplasticity from this writer's MNEI perspective[77,81]**.**

Research on neuroplasticity and psychosocial genomics supports a **biopsychosocial approach** by shedding light on factors that shape neurobiology. The research discloses a complex interaction between genes and epigenetics into adulthood. With this knowledge, psychologists specializing in adolescents can enhance their effectiveness. Social cognitive theory and resilience theory exemplify ways to incorporate neuroscience findings. Nutritional awareness and enrichment also belong in the treatment equation[108,109,110,111,112,113,114]**.** Furthermore, a recent animal study suggested that psychotherapy benefited even geneti-

cally depressed rats, which conveys an encouraging message about the environment's importance in changing biomarkers for youths with a strong family history of depression[115].

Neuroplasticity entails a learning process that modifies biology and behavior, enlisting the adolescent's active participation. Interventions relying solely on metabolic nutrition and/or pharmacology may have a positive yet limited impact. Thus micronutrient supplementation must coexist with suitable diet practices. Participation in decision-making about medication, as shown in a Canadian study, favorably modifies treatment outcomes. Exercise and sports represent active ways of creating competence, with demonstrated neuroplastic benefits. Mind-body methods, from the Eastern traditions (yoga, tai chi, chi gong) or the West (Autogenic training, biofeedback) touch on the very thread that unites biological and psychological factors of health in the adolescent patient with mental/emotional and or behavioral problems. The preceding modalities need to be applied within the psychotherapeutic matrix, which focuses on mind-brain patterns affecting behavior[116].

The following issues require exploration and understanding in a unified context: adolescent's self-image and self-doubts, fears, hopes and dreams, grievances, yearnings, his/her relationships, daily diet, physical activity, and belief systems. Awareness of those patterns and substitution with more effective and mature ones, becomes "learning to learn" and creates room for developing individualized, metacognitive strategies that support the desired neuroplastic changes. Thus a therapeutic synergy emerges to sustain and reinforce healing from several vectors into one vortex promoting growth.

To sum up, **adolescence is a neuroplastic powerhouse** with engines readied for neurocorrective interventions. Because of its teleological drive towards maturity, this stage offers large scale opportunities for stimulating positive development. It is prime time to:

(1) identify dysfunctional habits/patterns of the body and mind through psychotherapy;

(2) adopt constructive ways of thinking, relating, and handling emotions as well as stressors; and

(3) shape lifestyle towards health and wellbeing.

A holistic treatment matrix includes: education, cognitive and interpersonal processes, nutrigenomics, exercise, meditational disciplines, and the judicious use of medication as needed. A productive psychotherapeutic relationship awakens the adolescent's courage and resolve to work through conflicts around dependency, autonomy, and identity. One must capitalize on the biologically-driven changes in cognition and brain functioning. Adolescence is a critical period to learn awareness, self-care, self-directedness, self-regulation, compassion, and responsibility. In addition, encouraging mastery, and the formation of positive affiliations through a balance of competition and cooperation further induce neuroplasticity—in class, the sports field, at home, or in psychotherapy.

The stakes are very high during adolescence. Two tenets of age-old wisdom merit final reflection:

"Good habits formed at youth make all the difference."
—Aristotle

"It takes courage to grow up and become who you really are."
—E.E. Cummings

References

1. Bonavita V. (2011). The Italian neurological schools of the twentieth century. *The Funct Neurol*, 26(2), 77-85.
2. Fuchs E. & Fluegge G. (2014). Adult Neuroplasticity: More than 40 years of research, *Neural Plasticity,* Hindawi Publishing Corp, 1-10.
3. Freud S, 1918/1955, An infantile neurosis, in Standard edition of Complete Psychological Works, vol 17, 116.
4. Doidge N. (2007). The Brain that Changes Itself: Stories of Personal Triumph from the Frontiers of Brain Science, Penguin Books.
5. Hebb DO. (1949) The Organization of Behavior. Wiley.
6. Haider B. (2008). Contributions of Yale neuroscience to Donald O. Hebb's Organization of Behavior, *J Biol Med.* 81(1):11–18.
7. Belzung C, Wigmore P (Eds.) (2013) Neurogenesis and Neural Plasticity: Current Topics in Behavioral Neuroscience, Volume 15, Springer] [Gage FH. (2002). Neurogenesis in the adult brain, *J Neuroscience*, 22(3): 612-613.
8. Reynolds BA, Weiss S (1992). Generation of neurons and astrocytes from isolated cells of the adult mammalian central nervous system. *Science,* 255*:*1707.
9. Richards LJ, Kilpatrick TJ, Bartlett PF *(*1992*)* De novo generation of neuronal cells from the adult mouse brain. *Proc Natl Acad Sci USA* 89: 8591–8595*.*
10. Palmer TD, Kuhn HG, Fisher LJ, Suhonen JO, Peterson DA, Suhr ST, Ray J. *(*1995*)* Survival and differentiation of adult neuronal progenitor cells transplanted to the adult brain. *Proc Natl Acad Sci USA* 92:11879–11883.
11. Palmer TD, Markakis EA, Willhoite AR, Safar F, Gage FH (1999). Fibroblast growth factor 2 activates a latent neurogenic program in neural stem cells from diverse regions of the adult CNS, *J Neurosci* 19*:*8487–8497.
12. Kondo T & Raff M. (2000). Oligodendrocyte precursor cells reprogrammed to become multipotential CNS stem cells. *Science* 289*:*1754–1757*.*
13. Altman J. (1962). Are neurons formed in the brains of adult mammals? *Science* 135*:*1127–1128*.*
14. Raisman G (1969) Neuronal plasticity in the septal nuclei of the adult rat. *Brain Res* 14:25–48.
15. Moore RY, Bjorklund A, & Stenevi U. (1971) Plastic changes in the adrenergic innervation of the rat septal area in response to denervation. *Brain Res* 33:13–35.
16. Lynch G, Deadwyler D, & Cotman CW. *(*1973*)* Post lesion axonal growth produces permanent functional connections. *Science* 180*:*1364–1366.
17. Greenough WT, West RW, & DeVoogd TJ 1978*.)* Postsynaptic plate perforations: changes with age and experience in the rat. *Science* 202*:*1096–1098.
18. Bjorklund A, Gage FH. *(*1985*)* Neural grafting in animal models of neurodegenerative diseases. In Nottebohm F (Ed). Hope for a New Neurology*, Annals of New York Academy of Science,* Vol. 457.
19. Dunnett SB & Bjorklund A. *(*1994*)* Functional neural transplantation, Raven.
20. Pascual-Leone A., Freitas C., Oberman L., Horvath J. C., Halko M., Eldaief M.; et al. (2011). "Characterizing brain cortical plasticity and network dynamics across the age-span in health and disease with TMS-EEG and TMS-fMRI". *Brain Topography*, 24: 302–315.
21. Rapoport JL & Gogtay N (2008). Brain Neuroplasticity in healthy, hyperactive, and psychotic children: Insights from Neuroimaging, *Neuropsychopharm*, 33:181-197.
22. Evans TM, Kochalka J, Ngoon TJ, Wu SS, Qin S, Battista C, & Menon V (2015) Brain Structural Integrity and Intrinsic Functional Connectivity Forecast 6 Year Longitudinal Growth in Children's Numerical Abilities, *J Neurosci,* 35(33):11743-11757*.*
23. Maguire EA, Woollett K, & Spiers HJ. (2006). London taxi drivers and bus drivers:

a structural MRI and neuropsychological analysis, *Hippocampus,* 16:1091-1101.
24. Herbet G, Maheu M, Costi E, Lafargue G, & Duffau H (2016). Mapping neuroplastic potential in brain-damaged patients, *Brain,* Mar;139(Pt 3):829-844.
25. Barha CK, Brummelte S, Lieblich SE, & Galea LA (20—). Chronic restraint stress in adolescence differentially influences hypothalamic-pituitary-adrenal axis function and adult hippocampal neurogenesis in male and female rats, *Hippocampus*, 21(11):1216F-1227.
26. Chaby E, Cavigelli SA, Hirrlinger AM, Lim J, Warg KM, & Braithwaite VA (2015). Chronic stress in adolescence impairs and improves learning and memory into adulthood, *Front Behav Neurosci*, 11(9):327-334.
27. Selemon LD (2013). A role for synaptic plasticity in the adolescent development of executive function, *Translational Psychiat*, 3:e238.
28. Timble C & Barbas H (2015). Pathways for emotions: specializations in the amygdalar, mediodorsal thalamic, and posterior orbitofrontal network, *J Neurosci,* 35(34):11976-11987.
29. Choudhury S & McKinney KA (2013). Digital media, the developing brain and the interpretive plasticity of neuroplasticity, *Transcult Psychiat,* 50(20):192-215.
30. Casey BJ, Jones RM, & Hare TA (2008) The adolescent brain, *Ann NY Acad Sci,* 1124:111-126.
31. Chein J, Albert D, O'Brien L, Uckert K, & Steinberg L (2011). Peers increase risk taking by enhancing activity in the brain's reward circuitry, *Dev Sci*, 14 (2):F1-F10.
32. Steinberg L. (2008). A social neuroscience perspective on adolescent risk-taking, *Dev Rev*, 28(1):78–106
33. Steinberg L (2015). Age of Opportunity: Lessons from the New Science of Adolescence, Harcourt Pub. Co., First Mariner Book.
34. Lally P, van Jaarsveld CHM, Potts HWW & Wardle J (2010). How are habits formed: Modelling habit formation in the real world, *European J Social Psychol*, 40(6): 998-1009.
35. Choudhury S (2010). Culturing the adolescent brain; what can neuroscience learn from anthropology? *SCAN*, 5:159-167.
36. Blakemore SJ. (2008).The social brain in adolescence, *Nature Reviews Neuroscience,* 9:267-277.
37. Choudhury S, Blakemore SJ, & Charman T (2006). Social cognitive development during adolescence, *Soc Cogn Affect Neurosci,* 1(3):165–174.
38. NIDA (2015). Understanding Drug Abuse and Addiction: What Science Says, 4: the reward pathway, 9-17.
39. Rubino T, Realini N, Braida D, Guidi S, Capurro V, Vigano D, Guidali C, Pinter M, Sala M, Bartesaghi R, & Parolaro D (2009). Changes in hippocampus morphology and neuroplasticity induced by adolescent THC treatment are associated with cognitive impairment in adulthood, *Hippocampus,* 19(8):763-772.
40. French , Gray C, Leonard G, Perron M, Pike GB, Richer L, Séguin JR, Veillette S, C. Evans J, Artiges E, Banaschewski T, Bokde AWL, Bromberg U, Bruehl R, Buchel C, Cattrell A, Conrod PJ, Flor H, Frouin H, Gallinat J, Garavan H, Gowland P, Heinz A, Lemaitre H, Martinot JL, Nees F, Papadopoulos Orfanos D, Pangelinan MM, Poustka L, Rietschel M, Smolka MN, Walter H, Whelan R, Timpson NJ, Schumann G, Smith GD, Pausova Z, & Paus T, Early Cannabis Use (2015). Polygenic Risk Score for Schizophrenia and Brain Maturation in Adolescence , *JAMA Psychiat,*72(10):1002-1011.
41. Cássila Kindler D, Fugita F, Davanso H, & Leite ML (2012). Alcohol Abuse, neurodevelopment and neuroplasticity in the adolescent, *Science & Cognition* (Neurociencias em Debate, Cencia e Cognicao, Brazil), Dec. 5.
42. Blos P (1967). The Second Individuation Process of Adolescence, *The psychoana-*

lytic study of the child, 22:162-186.
43. Cartl V, Hoven CW, Wasserman C, Chiesa F, Guffanti G, Sarchiapone MA, Apter A, Balasz J, Brunner R, Corcoran P, Cosman D, Haring C, Iosue M, Kaess M, Kahn JP, Keeley H, Postuvan V, Saiz P, Varnik A, Wasserman D (2014). A newly identified group of adolescents at "invisible" risk for psyhopathology and suicidal behavior: findings from the SEYLE study, *World Psychiatry* (Research Report), Feb 4, DOI: 10.1002/wps 20088.
44. Rowe SL, French RS, Henderson C, Ougrin D, Slade M, Moran P (2014) Help-seeking behavior and adolescent harm*:* A systematic review*, Australian & New Zealand J Psychiat* , 48(12):1083-1095.
45. McEwen BS (2000) Effects of adverse experiences for brain structure and function, Biol Psychiatry, 48(8):721-731.
46. Rogers JC and De Brito SA (2016). Cortical and Subcortical Gray Matter volume in youths with conduct problems, JAMA Psychiat, 73(1):64-72.
47. Popoli M, Gennarelli M, & Racagni G (2002). *Bipolar Disord*, Modulation of synaptic plasticity by stress and antidepressants, 4(3):166-182.
48. Cousins L, Goddyer IM (2015). Antidepressants and the adolescent brain, *J Psychopharmacol*, 29(5):545-555.
49. Willard SL, Uberseder B, Clark A, Daunais JB, Johnston WD, Neely D , Massey A, Williamson JD, Kraft RA, Bourland JD, Jones SR, & Shively CA. (2015). Long term sertraline effects on neural structures in depressed and nondepressed adult female nonhuman primates, *J Neuropharmacol,* 99:369-378.
50. Correll CU & Blader JC (2015). Antipsychotic use in youth without psychosis: a double-edged sword*, JAMA Psychiat*, Editorial, July 1:E1-E2.
51. Paulzen M, Veselinovic T, & Grunder G (2014). Effects of psychotropic drugs on brain plasticity in humans. *Restor Neurol Neurosci,* 32(1):163-81.
52. Mclaughlin T, Oscar-Berman M, Simpatico T, Giordano J, Jones S, & Barh D (2013). Hypothesizing repetitive paraphilia behavior of a medication refractive Tourette's syndrome patient having rapid clinical attenuation with KB220Z-utrigenomic aminoacid therapy (NAAT), *J Behav Addictions*, 2(2):117-124.
53. Bach- y-Rita P. (1972) Brain Mechanisms in Sensory Substitution, Academic Press.
54. Wolf Sl, Winstein CJ, Miller JP, Taub E, Uswatte G, Morris D, Giuliani C, Light KE, Nichols-Larsen D (2006). Effects of Constraint-Induced Movement Therapy on Upper Extremity Function 3 to 9 months after stroke: The EXCITE randomized clinical trial, *JAMA*, 296: 2095-2104.
55. Feldenkrais M (1977). Awareness through Movement, Harper & Row.
56. Gilmor TM (1999). The efficacy of the Tomatis Method for chldren with learning and communication disorders: a meta-analysis, *Intl J of Listening*, 13, 12-23.
57. Merzenich, MM, Nelson, RJ, Stryker, MP, Cynader, MS, Schoppmann, A & Zook, JM (1984). Somatosensory Cortical Map Changes Following Digit Amputation in Adult Monkeys, *J Comparative Neurol,* 224 (4):591–605.
58. Ramachandran, VS & Blakeslee, S (1998). Phantoms in the Brain. W. Morrow.
59. Kandel ER (2007). In Search of Memory: the Emergence of a New Science of Mind, W.W. Norton & Co.
60. Kandel ER, Schwartz JH, Jessell TM, Siegelnaum SA, & Hudspeth AJ (2013). Principles of Neural Science, Fifth Edition, McGraw Hill.
61. Kuyken W, Watkins E, Holden E, White K, Taylor RS, Byford S, Evans A, Radford S, Teasdale JD, & Dalgleish T. (2010) How does mindfulness-based cognitive therapy work? *Behav Res and Ther*, 48:1105-1112.
62. Schwartz JM & Begley S (2003). The Mind and the Brain: Neuroplasticity and the Power of Mental Force, HarperCollins.

63. Aloe L (2004). Rita L-M: The discovery of NGF and modern biology, *Trends in Cell Biol*, 14(7):395-399.
64. Aloe L. (2002) Rita Levi-Montalcini and the Discovery of NGF: Past and Present Studies, *Arch. Italiennes de Biol*, 141:65-83.
65. Levi-Montalcini R (1987).The Nerve Growth Factor: thirty-five years later, EMBO J. May, 6(5), 1145–1154. Errata, *The EMBO J* 6(9):2856.
66. Barde YA, Edgar D, Thoenen H. (1982) Purification of a new neurotrophic factor from mammalian brain. *EMBO J,* 1:549–553.
67. Autry AE & Monteggia LM. (2012). Brain-Derived Neurotrophic Factor and Neuropsychiatric Disorders, *Pharmacol Rev,* 64(2):238–258.
68. Hohn A, Leibrock J, Bailey K, Barde YA. (1990) Identification and characterization of a novel member of the nerve growth factor/brain-derived neurotrophic factor family. *Nature*, 344:339–341.
69. Schuman EM. (19990 Neurotrophin regulation of synaptic transmission, *Curr Opin Neurobiol*, 9:105-109.
70. Tyler WJ, Zhang XL, Hartman K, Winterer J, Muller W, Santon PK, & Pozzo-Miller L (2006), BDNF increases release probability and the size of a rapidly recycling vesicle pool within rat hippocampal excitatory synapses, *J Physiol,* 574:787-803.
71. Autry AE & Monteggia LM (2012). Brain-Derived Neurotrophic Factor and Neuropsychiatric Disorders, *Pharmacol Rev,* 64 (2):238-258.
72. Juan Pablo Lopez, Raymond Lim, Cristiana Cruceanu, Liam Crapper, Caroline Fasano, Benoit Labonte, Gilles Maussion, Jennie P Yang, Volodymyr Yerko, Erika Vigneault, Salah El Mestikawy, Naguib Mechawar, Paul Pavlidis, Gustavo Turecki (2014). miR-1202 is a primate-specific and brain-enriched microRNA involved in major depression and antidepressant treatment. *Nature Medicine,* DOI: 10.1038/nm.3582.
73. Lu H, Liu X, Deng Y, & Qing H (2013) DNA Methylation, a hand behind neurodegenerative diseases, *Front Aging Neurosci*, 5: 85.
74. Thakkar K & Billa G (2015). Treatment of vitamin B12 deficiency - Methylcobalamine? Cyanocobalamine? Hydroxocobalamin? - clearing the confusion, *Eur J Clin Nutr*, 69:1-2.
75. Aguilar F, Charrondiere U, Dusemund B, Galtier P, Gilbert J, Gott DM, Grilli S, Guertler R, Kass GEN, Koenig J, Lambre C, Larsen J-C, Leblanc J-C, Mortensen A, Parent-Massin D, Pratt I, Rietjens I, Stankovic I, Tobback P, Verguieva T, & Wotersen R. (2008) On 5'-deoxyadenosylcobalamin and methylcobalamin as sources for Vitamin B12 added as a nutritional substance in food supplements, *The EFSA* (European Food Safety Authority) *Journal*, 815:1-21.
76. Hyman SE (2006), Even chromatin gets the blues, *Nature Neurosci*, 9:465 - 466**.**
77. Galle SA (2014) Minding the mitochondria: from cell to psyche, *Arch. Med Psych*, 6:10-35.
78. Kate N, Grover S, & Agarwal M (2010*)* B12 deficiency lead to lack of treatment response to conventional antidepressants? *Psychiatry,* 7(11):42–44.
79. Archer T, Oscar-Berman M, Blum K, & Gold MS (2013) Epigenetic Modulation of Mood Disorders*, J Genet Syndr Gene Ther*, 4(120):1-25.
80. Kays JL, Hurley RA, & Taber KH, The dynamic brain: neuroplasticity and mental health, *J Neuropsychiat & Clin Neurosci,* 24 (2):118-124.
81. Galle SA (2015). AGE and RAGE: hazardous to the mind? A plea for translational work, *Arch Med Psych,* 7:6-18.
82. Murray G (2015). Do I have a self? (and other useful questions from Buddhist mindfulness), *Aust NZ J Psychiat*, 49(7):593-4.
83. Galle SA & Silber T (2007). Yoga therapy with anorexic adolescents: alternative or first line Approach?, Suppl. to *Intl J Yoga Ther*, First IAYT Symposium on Yoga

Therapy & Research, Los Angeles, CA, January 18-21.
84. Westbrook A (2016) Dopamine does double duty in motivating cognitive effort, *Neuron*, 89(4):695-710.
85. Newberg AB & Waldman, MR (2012). Words Can Change Your Brain: 12 Conversation Strategies to Build Trust, Resolve Conflict, and Increase Intimacy, Hudson Street Press.
86. Luby JL, Belden AC, Jackson JJ, Lessov-Schlaggar CN, Harms MP,Tillman R, Botteron K, Whalen D, Barch DM (2015), *JAMA Psychiat*, Dec. 16, online, e1-e8.
87. Zalesky A, Pantelis C, Cropley V, Fornito A, Cocchi L, McAdams H, Clasen L, Greenstein D, Rapoport JL, Gogtay N. (2015). Delayed Development of Brain Connectivity in Adolescents With Schizophrenia and Their Unaffected Siblings, *JAMA Psychiat*, 72(9):900-8.
88. Gao J & Li M (2014). Differential effects of intermittent versus continuous haloperidol treatment throughout adolescence on haloperidol sensitization and social behavior in adulthood, *Prog Neuropsychopharmacol Biol Psychiat*, 54:67-75.
89. Whitaker R (2010). Anatomy of an Epidemic: Magic Bullets, Psychiatric Drugs, and the Astonishing Rise of Mental Illness in America, Broadway Books, Random House.
90. Qiao J, Zhang Q, & Li M (2014). Long-term impacts of adolescent risperidone treatment on behavioral responsiveness to olanzapine and clozapine in adulthood, *Prog Neuropsychopharmacol Biol Psychiat*, 3(48):177-85.
91. Erikson EH (1968). Identity and the Life Cycle, Intl. Universities Press.
92. Erikson EH (1959). Identity, Youth and Crisis, WW Norton.
93. Jensen LA & Arnett JJ, (2012) Going Global: New pathways for adolescents and emerging adults in a changing world, *J Social* Issues, 68 (3):473-492.
94. Fuhrmann D, Knoll LJ, & Blakemore SJ (2015), Adolescence as a sensitive period of brain development, *Trends Cog Sci*, 19 (10):558-566.
95. Fuhrmann T & Holmbeck GN (2008). A Contextual-Moderator Analysis of Emotional Autonomy and Adjustment in Adolescence, *Child Develop*, 66 (3):793-811.
96. Chaby LE, Cavigelli SA, Hirrlinger AM, Lim J, Warg KM, & Braithwaite VA (2015). Chronic stress during adolescence impairs and improves learning and memory in adulthood, *Behav Neurosci*, 9 (237):1-11.
97. Balaratnasingam S, Janca A (2012). Brain Derived Neurotrophic Factor: a novel neurotrophin involved in psychiatric and neurological disorders. *Pharmacol Ther*, 134:116-124.
98. Pareja-Galeano H, Brioche T, Sanchis-Gomar F, Montal A, Jovani C, Martinez-Costa C, Gomez-Cabrera MC, & Vina J. (2013). Impact of exercise training on neuroplasticity-related growth factors in adolescents, *J Musculoskelet Neuronal Interact*, 13(3):368-371.
99. Choudhury S, McKinney KA, Kirmayer LJ (2015). Learning how to deal with feelings: Psychotropic medications as vehicles of socialization, Social Sci & Med, 30:1-9.
100. Rodgers A & Dunsmuir S (2015) A controlled evaluation of the “Friends for Life” emotional resilience program on overall anxiety levels, anxiety subtype levels, and school adjustment, *Child and Adol Mental Health,* 20(1):13-19.
101. Brent DA, Brunwasser SM, Hollon SD, Weersing VR, Clarke GN, Dickerson JF, Beardslee WR, Gladstone TRG, Porta G, Lynch FL, Iyengar S, & Garber J (2015). Effect of a Cognitive-Behavioral Prevention Program on Depression 6 Years After Implementation Among At-Risk AdolescentsA Randomized Clinical Trial, *JAMA Psychiat*, 72(11):1110-1118.
102. Kussman M, Krause L, & Siffert W (2010). Nutrigenomics: where are we with genetic and epigenetic markers for disposition and susceptibility? *Nutrition Reviews*, 68(1): Suppl, S38-S47.

103. Gomez-Pinilla F (2008). Brain foods: the effects of nutrients on brain function, *Nat Rev Neurosci*, 9(7):568-578.
104. Rathod R, Kale A, & Joshi S (2016) Novel insights into the effect of vitamin B12 and omega-3 fatty acids on brain function, *J Biomed Sci,* 23:17.
105. van der Wurff ISM, von Schacky C, Berge K, Zeegers MP , Kirschner PA, & de Groot, RHM (2016). Association between blood omega-3 index and Cognition in Typically Developing Dutch Adolescents, *Nutrients,* 8(13):20.
106. Benton D (2008). The influence of children's diet on their cognition and behavior, *Eur J Nutr*, 47 (Suppl. 3):25-37.
107. Galle SA (2010). Molecular nutrition: a missing link in pediatric pharmacotherapy? *The Tablet, APA Div. 55 Newsletter*, 11(3):68-73.
108. JG Fine & Sung C (2014). Neuroscience of child and adolescent health development. Special Section: The Integration of Counseling Psychology and Neuroscience, *J Counseling Psych*, 61(4):521-527.
109. Emmelkamp PMG, David D, Beckers T, Muirs P, Cuijpers P, Lutz W, Andersson G, Araya R, Banos Rivera RM, Barkham M, Berking M, Berger T, Botella C, Carlbring P, Colom F, Essau C, Hermans D, Hofman SG, Knappe S, Ollendick TH, Raes F, Rief W, Riper H, Van der Oord S, Vervliet V (2014). Advancing Psychotherapy and Evidence-Based Psychological Interventions, *Intl J of Methods in Psychiatric Res,* 23 (S1):58-91.
110. Cramer SC, Sur M, Dobkin Bh, O'Brien C, Sanger TD, Trojanowski JQ, Rumsey JM, Hicks R, Cameron J, Chen D, Chen WG, Cohen LG, deCharms C, Duffy CJ, Eden GF, Fetz EE, Filart R, Fruend M, Grant SJ, Haber S, Kalivas PW, Kolb B, Kramer AF, Lunch M, Mayberg HS, McQuillen PS, Nitkin R, Pascual-Leone A, Reuter-Lorenz P, Schiff N, Sharma A, Shekim L, Stryker M, Sullivan EV, Vinogradov S (2011). Harnessing neuroplasticity for clinical applications*, Brain*, 134(6):1591-609.
111. Simmons WK, Avery JA, Barcalow JC, Bodurka J, Drevets WC, & Bellgowan P (2013). Keeping the body in mind: insula functional organization and functional connectivity integrate interoceptive, exteroceptive, and emotional awareness, *Hum Brain Mapping,* 34(11):2944-58.
112. Kendler KS, Ohlsson H, Mezuk B, Sundquist JO, & Sunquist K (2016). Observed cognitive performance and deviation from familial cognitive aptitude at age 16 years and ages 18 to 20 years and risk for schizophrenia and bipolar illness in a Swedish national sample, *JAMA Psychiat,* 73(3): 185, doi: 10.1001.
113. Gilman JM, Curran MT, Calderon V, Schuster RM, & Eden Evins A (2016). Altered neural processing to social exclusion in young adult marijuana users. *Biol Psychiat: Cog Neurosci & Neuroimag*, 1(2):152.
114. Khundrakpam BS, Lewis JD, Zhao L, Chouinard-Decorte F, & Evans AC (2016). Brain connectivity in normally developing children and adolescents, *J Neuroimage,* 30, doi:10.1016/j. neuroimage. 03.062.
115. Mehta-Raghavan NS, S L Wert SL, Morley C, E N Graf EN, & Redei EE (2016). Nature and nurture: environmental influences on a genetic rat model of depression, *Translational Psychiat*, 6:1-10.
116. Galle SA (2015). The fire within: a perspective on pain, *AMP (Acad Med Psych) Newsletter*, 4(1): 3.

Scientifically Supported Treatments for Depressive Spectrum Disorders: Growth and Remediation, Medication, & Lifestyle Techniques

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Some have traced Medical Psychology to 1945 and Karl Heiser and the Connecticut Psychology licensure law[1]. I hold the Karl Heiser award given for national psychology advocacy, and revere him and the award, but he was not the father of Medical Psychology. To think that is an error and the sanitized American Psychological Association (APA) version. Medical psychology started with a neurologist turned psychologist (Freud) and other physicians turned psychologists (the Vienna Group) in the late 1800s. These physicians left medicine and the medical science training because there was more to health, brain development and repair, and relational and social life than bio-mechanistic construction. Freud, a neurologist, came to view the brain as a marvelous but autoplastic mechanism which could change by contrived and special experience. He became aware of methods to energize sections of the brain so that they could affect reorganization, inhibition, and more sophisticated expression. By 1916, shortly after Freud made his famous trip to the USA and the renounced psychological speeches, a physician by the name of Lydia Ross published a paper entitled "Medical Psychology" in which she warned against becoming so dominated by man's "animal nature" (physical) that the Medical Psychologist loses the capacity to integrate the physical, psychological, social, and life-style aspects of disease[2]. More recently the specialty of Medical Psychology (www.amphome.org), and the American College of Life-style Medicine (ACLM, see, http://www.lifestylemedicine.org/) have led these aged conceptualizations of health and positive and integrative approaches into the twenty-first century! Increasingly physicians in the ACLM (membership is physicians and psychologists) physicians are given continuing education on behavioral and cognitive interventions, nutrition, relational and stress management, substance abuse treatment, and other contextual aspects of controlling and preventing physician and psychological disease. They are following in the perception of Freud and the Vienna Group and leaving the body medicine and become much more psychologist in their orientation.

Recently, the US healthcare system has been weighed in the balance and found wanting and overly biomechanistic[3]. The Patient Protection and Affordable Care Act (ACA) has attempted to address this problem by moving us back to a more holistic and integrated care system of healthcare[4]. The modern healthcare team will consist of a psychologist, physicians, nurse practitioners, nurses, bachelor's psychology and social work case managers, and health educators. This is contrary to the traditional medical model mindset which views all health and healthcare from a simple bio-reductionistic or biomechanical philosophy. Still, the realities of the current body of research evidence, long-term migration of many of the more insightful and educated physician, and the scientific community points to a multi-dimensional and multi-pathway etiology of most serious diseases.

We now know that habit patterns and chronic behaviors, attitudes, stressors and their management, and lifestyle and recreational patterns are involved in the cause of most serious disease. Ornish and colleagues began to shock the bio-reductioists by reversing aspects of heart disease with life-style psychology[5]. Boll and colleagues later made it clear that early medicine was "mostly psychological" and that only recently became so allopathic, surgical, and dominated by medications[6]. Cummins, by 1968 was practicing medical

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psychology and proving that it was a clinical, programmatic, and economic success[7]. The science is now available that these psychological factors and Medical Psychology are just as important in the etiology and treatment and management of the major and most costly diseases as the biology of the person[8]! We also know more about the cost/benefit of palliative and allopathic interventions and that there is "no free lunch" (short-term and symptom solutions cost dramatically in the long run)[9].

The United States Healthcare System is Flawed

The four major factors negatively affecting health in the USA are: 1. The over prescription, wrong prescription, and misuse of medications, 2. Sedentary life-style and lack of proper exercise, nutrition, and outdoor activity, 3. Poor stress management, and 4. Immaturity/mental illness and related choices that effect 1-3 above). Unfortunately, the current Medical Industrial Complex is set up to capitalize upon these 4 factors rather than correct them. Primary care medicine, and primary care physicians (the future leaders of the emerging Affordable Care Act system) have abdicated their leadership role in "growth and curative interventions" and have opted for alliances with the pharmaceutical houses to provide economically rewarding, overly simplistic, and short-term effective allopathic solutions to healthcare symptoms. These entities have preyed upon human nature which tends to seek "over-simplification" of threat and anxiety provoking situations and conditions, denial as a defense, and ambivalence about major changes in one's self, lifestyle, and relationships. Thus, quick and symptom focused techniques such as medications, surgery for things that can be improved in other ways, and misinformation that over estimates the potential efficacy of the first two are offered and supported by the Medical Industrial Complex. They have, with the exception of non-mainstream groups like the American College of Lifestyle Medicine, given up on leading patients to long-term, non-medication (or certainly replacement of short-term medication), lifestyle, and maturation and self-management approaches to healthcare. They have not joined with Medical Psychologists and Lifestyle Specialty Physicians and included these disciplines in Primary Care Clinics and Community Hospitals. The Government has been dominated by the Medical Industrial Complex and has not developed adequate staffing and reimbursement standards and polices for these facilities.

Even with all the science indicating the need to broaden the staffing requirements in Primary Care Centers and Community Hospitals, the core staff membership in these health facilities central to the healthcare system, and Psychiatric Hospitals and Residential Care Centers, Nursing Homes have not been modernized in generations. These facilities and private practices have been the protected bastion of the scientifically and functionally archaic Medical Model and most generally admit to the staff only the medically trained provider. The belief that the provider trained in medical school is always the most qualified, highest qualified, or best leadership on a case is in opposition to much science, clinical experience, to documented disease origins and process, and to rational thought! The idea is spawned from an era when it was more rational and true because there was not the wealth of information, multidisciplinary sciences and scientists contributing to healthcare, and when there were no other qualified doctorate level healthcare disciplines and providers available. Still, to remain in an archaic perception of reality and not adapt to the emerging reality is "guild serving" at best, and the very definition of illusional or delusional at worse.

If health was conceptualized as maintaining a "biological homeostasis with drugs or scalpel" then the staffing and leadership of these facilities needed to rest on physicians and nurses. There was a time when we were this primitive, and this vision was true. However, the realities of evolving science and training available in the healthcare professions has rendered the perception as having utility only for physicians and their economic wellbeing. These medical providers generally have scant training or ability to conceptualize the complexities and expansive science related to complex behavioral and attitude and philo-

sophical modifications that are required to restore and maintain optimal health. Often, their behavioral and psychological training is at the level of a high-school teacher and they possess very superficial courses provided by a psychologist in medical school. Many medical practitioners and students resent the psychologist as the "expert teacher" and have lobbied to keep them out of medical faculties (possibly because this superior knowledge of the psychologist tends to dispel the "Halo Effect" of "the physician knows the most about everything" that has become the "Branding Core Concept" of the pharmaceutical houses as they co-opted physicians into serving as their "drug delivery system"; now over 60% of the addicts in the USA have a legal prescriber as their drug delivery system or in the supply chain of that system).

Over simplifications of diagnoses in the mental health and substance abuse area, superficial psychoeducation techniques inappropriately applied to the mentally ill (MI) and substance use disorders (SUDs) populations abound in the primary care system! Patients with MI are diagnosed as a "feeling" (depression, anxiety, anger control problems), or an impulse! This illustrates scant understanding of the complexity of diagnosing and treating mental disorders. Yet, we routinely put primary care physicians and nurses (many with two year degrees) in charge of a lifetime of care for many mental patients. The result is failure to stall the progression of the mental and addiction diseases, destruction of generations of families, loss of productivity and contribution to the economy and tax base, exacerbation and triggering of physical disease, unnecessary emergency department and hospital care, and huge long-term healthcare costs. Yet, few physicians, hospital administrators, healthcare policy wonks, Government science and regulatory bodies, and even psychologists are calling for significant rethinking of the staffing and program and leadership requirements in the Primary Care and Community Hospital Systems. These leaders are captured by a bygone past, strong guild resistance, and economic forces which reinforce them for putting up with the status quo! Thus, while they have the mantle and trappings of "leadership"-they are, in fact, "followers and visionless"!

Healthcare is becoming increasingly team and multi-disciplinary oriented. No one discipline can master the vast array of multidisciplinary sciences, and techniques. None can afford the practice time to be the meta-practitioner, or even to lead in all situations and with all diseases and patient populations. We simply must quit pretending and distorting reality and face the truth! In evaluating the level of distortion of reality in patients, doctors of psychology and psychiatry are taught a simple illustrative pneumonic, "Neurotics build castles in the sky, and psychotics attempt to live in them"! The "level of distortion and maintenance of an archaic construction, or attempting to live in a wholly non-extant one" must be evaluated in the USA healthcare system and its' driver-The Medical Industrial Complex!

While some physicians and nurses will resist this, and no one can doubt the conflict of interest and significant guild and financial motivation to maintain the passé medical model, scientifically oriented and rational physicians and nurses will and are embracing the multi-disciplinary and health improvement and change future. Many enlightened physicians enjoy the introduction of the psychologist and the psychologist's mid-level assistants (social workers, counselors, marital and family therapists, substance abuse counselors) into the Primary Care and Community Hospital System. These modern medical practitioners will move past the "medical model" to a more "holistic model" where they will partner with specialists in the psychological sciences who will treat the MI, SUDs, and life-style related patients in depth and meaningful and scientifically proven ways. This will allow these physicians and nurses to retain these patients, collaborate in long-term care of these patients, and the medical personnel and doctors of psychology and their supervised mid-level practitioners will cross fertilize and congeal the medical and psychological knowledge and abilities of each other.

These enlightened and more scientific physicians will be more willing to assist teams that have behavioral health experts and doctors with the time to significantly affect and manage MI and SUD patients. Everyone (the psychologist, the mid-level behavioral healthcare providers with a competent psychologist supervisor, the poorly geographically distributed psychiatrist who only has time to manage complex medicine [drug treatments] and physically disabled mental health cases, the general physician, the nurse, the facility administrator, the patient, the family, and the next generation of the family, the businesses dependent on the workforce, and the economic managers inside and outside of healthcare) stands to benefit by reorganizing the staffing and standards of the general healthcare system. The quality of care for many patient populations will improve! Arguments by the medical guild that "patients will be hurt" have not proven true in those areas of psychologist diagnosing, psychologists being on medical staffs, psychologists writing behavioral orders and making certain leadership decisions in hospitals, psychologists providing doctor's opinions in court cases, psychologist prescribing psychoactive medications, etc. Now over 3 million psychologist prescriptions in this country have resulted in no damage to patients, no malpractice suits that have been successful, and no loss of license or suspensions of prescribing psychologists. The science and healthcare experience dictates that if these changes are implemented the quality of the team's knowledge and ability will be enhanced, and long-term costs and side effects will be decreased.

In order to make this transition from the medical model to the holistic model we must avoid several dangers. The first is continuing to put physicians in charge of hospital and primary care staffs who resist change and have guild interests that supersede patient care interests. Of course these physicians will have prevalent financial, fraternal, and philosophical reinforcers to support their position. However, innovative "early adopters" and scientifically informed physicians will be the better choice and when placed in the leadership of facilities and programs will be able to capitalize on the available laws, reimbursement and incentive pay patterns, and competitive contracts and grants that will help them build multi-disciplinary and increasingly effective programs. Second, psychologists must realize that out of the 55 varieties of psychologists very few are trained to adequately do diagnosis, treatment of MI and SUDs and the medical and lifestyle aspects of physical illness! Most psychologists are simply not trained or equipped as "core healthcare providers"! Social psychologists, Educational Psychologists, Industrial Psychologists, General Psychologists, History and Systems Psychologists, and others are trained in their particular disciplines but are not required to have a singly graduate course in the diagnosis and treatment of mental disorders or of the psychological aspects of chronic medical and life-style related disorders. Some follow ethical practice and American Psychological "re-specialization guidelines" (generally one to two additional years of graduate clinical coursework and a one-year clinical internship). This dirty little secret in psychology has allowed many psychologists to practice in or around healthcare facilities will little training and ability that can distinguish them from mid-level behavioral health providers! The medical practitioner is almost never aware that PhDs in general psychology, social psychology, guidance and counseling, industrial psychology, and many other types of psychology degrees have little or no training in diagnoses and treatment of mental disorders and behavioral aspects of physical disorders! Actually, many physicians know so little about the diagnostic and clinical training of clinical psychologists that they voice that they are "shocked by how much they know and can do" after working closely with such doctors of psychology. It will be crucial that Hospital and Primary Care Center Chief Psychologists are well trained and capable of advanced diagnostic work, advanced psychotherapy, and to guide and supervise mid-level behavioral health workforces! The optimal Chief Psychologist would be a board certified Medical Psychologist with advanced training in clinical psychology and health psychology and medical foundations (psychopharmacology, biochemistry, anatomy and physiology, general pharmacology, pathophysiology, organic chemistry, etc.), and supervised precep-

torships or post-doctoral residencies in medical facilities or collaborative medical practice. The strength of future facilities and programs will rest on the quality of chiefs of medical staff and chief psychologists.

Modern healthcare facilities and modern psychologists will become involved in medical facilities. These doctors of psychology with clinical training will be consulting and helping to review historical electronic records and diagnostics, select or prescribing psychoactive medications, performing specialty diagnostics, and formulating holistic treatment plans based on a mental status interview, extensive psychosocial history. They will make informed diagnostic and treatment planning decisions and write orders based upon obtained collateral records, a battery of psychological testing, and collateral family interviews obtained in a variety of medical and psychological facilities. This will lead treatment teams to facilitate comprehensive treatment plans, and case configurations rapidly and in a meaningful way (many times electronically for virtual teams)[10, 11, 12]. Additionally, they will have to have good crisis intervention, short-term therapy, and mid-level practitioner supervision skills. They will have to respect and support their physician colleagues who have the arduous task of focusing on over 3,000 disorders while the behavioral health specialists have the luxury of focusing on about 100. They will have to be adroit at sensitively negotiating boundaries with physicians as colleagues and strong enough to refuse to be lumped with mid-level (in medicine or behavioral health) providers.

All primary care and facility practitioners and other specialist doctors will be pressed to cover larger volumes of patients who will increasingly have healthcare coverage and access to healthcare. They will do so in an increasingly capitalistic and price competitive environment where practitioners, facilities, and systems compete on a cost and product quality basis for area and specialty (diagnoses based) contracts. Thus, efficient multidisciplinary teams with both intervention, and prevention skills will be in demand. Physicians, nurses, and psychologists will eventually realize that by being open to each other and collaborating they will compete well with other systems and have much better chance of capturing greater market share (which translates into better income and job security for all). Telehealth skills will become a tool that most psychologists, especially in rural areas, will develop to be able to lead treatment plans and mid-level provider teams over a number of facilities and geographic locations. Increasingly, pharmacists will educate patients and may even become medication renewal prescribers (like many foreign countries where this has always been done safely and affordably). Substance abuse and dependency, devastating American worker productivities, family well-being, individual's health, and the economic picture in the healthcare system will become a primary component of primary care centers. These programs will require a doctor of psychology to direct and manage them in a way that doesn't add to the already burdened general physician's stress level. The psychologist will protect the busy physician from a loss of productivity, and from becoming embroiled in complex and time consuming behavioral and family interventions. This expansion of primary care centers and community hospitals will revolutionize and expand America's and educational institution's concept of what the role and mission, staffing, and management of primary care and community hospitals entails. Physicians, nurse practitioners, nurses, and psychologists will need to amass significant specialty skill in the area of the diagnosis and treatment of substance use disorders (SUDs)[11,12,13,14,15].

General physicians will become accustomed to multi-disciplinary team treatment and the luxury of not starting a psychoactive medication until a psychologist has worked up the patient, established an admitting diagnoses and recommended treatment plan (including medications, psychotherapies, medical studies and lab work that is indicated, and the formulation of a suggested multidisciplinary team), and have accepted that they do not have the training and experience to diagnose and plan for these disorders[16,17]. They will be-

come increasingly educated about the limited capacity of psychopharmacological techniques to treat mental disorders (http://truthindrugs.com/index.html). They have functioned with limited training in the areas of mental disorders, SUDs, prevention and life-style management, and family systems interventions and management. While a few enlightened physicians (Minuchen, Whitaker, Maltsby, Glasser, Beck in the early years and many more in recent years)[18,19,20,21,22], and the American College of Lifestyle Medicine (http://lifestylemedicine.org/) have pointed out for three generations the need for more psychological and holistic healthcare, primary care and biological psychiatry and the drug companies have veered at a substantial variance to this wisdom. Lifestyle physicians are highly motivated and interested in these concepts and post MD/DO (medical) training in nutrition, physical education, and psychology. They should join with psychologists to advocate for more rational Primary Care Center and Community Hospital Staffing Requirements. These physicians should lobby for standards that require at least one psychologist and life-style physician psychologist team in every healthcare delivery system. Only then, and with these two very special doctors who are dedicated to causing health instead of just controlling disease and disease discomfort become a dominant force in the healthcare system. Not until physicians and psychologists join forces and return medicine to its psychosocial roots will healthcare systems have the leadership and broadened vision and thrust that can capitalize on new and emerging healthcare science and control the nation's healthcare costs, respond appropriately to the new longevity, and ensure quality of life in an increasingly aging population.

The Dynamics Driving Change to the Holistic Model and Multi-disciplinary Healthcare:

The dynamics driving evolution from the medical to the holistic model of healthcare include new scientific findings, scarcity and demand and overall healthcare economics, and a more sophisticated public that is increasingly difficult to herd into short-term, high risk and cost, and unsubstantiated treatments. The American, and even the world, consumer has started to move beyond gullibility and halo effect interventions! They have figured out the marketing ploys and shaping and thanks to the web and ready access to information educate themselves and guard against them. Consumers have been liberated from dependency upon physicians for their science and healthcare education and information. They are often as informed or more informed related to the heuristic aspects and options of their specific disease state and related intervention and treatment plan options and risks than their physician or nurse. They are increasingly skeptical of those who are given artificial status to "think for them"! The modern patient has been so often misled, hoaxed, and inappropriately treated by politicians, employers, and the healthcare system that they are becoming realistically skeptical[23.] A new era of a skeptical, informed, educated, and assertive consumer is emerging! This is one of the advantages of mass societal education and accessible information systems.

A second dynamic is that fact that MI and SUDs are now recognized as very prevalent in all health centers[24]. The mental disorders are so prevalent in Primary Care Centers and Medical/surgical Hospitals that general physicians appear to welcome help with these complex specialty cases[25]. Mental disorders are estimated to account for 12% of the global burden of disease, and yet only a minority of persons affected receives basic treatment[26], and one out of five patients in primary care clinics have these disorders, and nearly one of 10 have substance use disorders[27,28]. The treatment of depression in primary care centers can be cost effective and can offset indirect medical costs[29]. Major depressive disorder has a very high incidence in medical facilities. It has been recently found to be associated with high medical utilization and more functional impairment than most chronic medical illnesses. Major depression is a common illness among persons in the community, in ambulatory medical clinics, and in inpatient medical care. Studies have estimated that major

depression occurs in 2%-4% of persons in the community, in 5%-10% of primary care patients, and 10%-14% of medical inpatients. The problem of sub-syndromal depression, which often manifests as serious depressive illness occurs in two to three times as many persons with depressive symptoms that fall short of major depression criteria. One-third to one-half of patients with major depression experience symptoms that persist over a 6-month to one-year period. The majority of longitudinal studies have determined that severity of initial depressive symptoms and the presence of a comorbid medical illness were predictors of persistence of depression[30].

A secondary dynamic to the recognition of the prevalence of MI and SUDs and their incidence in primary care systems is the data, and patient experience, related to how poorly general hospitals, and primary care system, and the medical model has performed in the identification, diagnosis, and treatment of these disorders. The way depression is treated in the United States demonstrates a disturbing and changing trend toward superficial palliative care. Between 1987 and 1997 most MI and SUDs became an outpatients condition and the focus was on medication interventions which decreased several symptoms and basically the healthcare system gave up on changing the brain in any meaningful or growth oriented way. During this time, the use of psychotherapy declined (replaced by antidepressant medications even through there was little evidence of any major efficacy on the long-term of these drugs)[31]. In 1987, 37.3 percent of outpatients treated for depression received antidepressant medications and by1997 the rate had doubled to 74.5 percent[32]. During the next decade from 1998 to 2007 the research documented a continued decline in the use of psychotherapy and stabilization in the use of antidepressants to treat depression even though the science presented above indicated that just the opposite would be pragmatic. The trend toward outpatient treatment for depression increased significantly from 2.37 per 100 persons in 1998 (6.48 million) to 2.88 per 100 persons in 2007 (8.69 million). The increase delivered this substandard and marginally scientifically founded treatment to the least educated and most vulnerable Americans. The largest proportionate increases in treatment included blacks, Medicare patients, and adults with less than a high school education. There was a significant decline in depression outpatients receiving psychotherapy, from 53.6 percent in 1998 to 43.1 percent in 2007. This, largely medication only approach caused expenditures for depression treatment increased at the national level, for medications, and for Medicare.

Another dynamic beaconing change from the medical model of healthcare is the underlying philosophy with started with hope and turned into religion rather than science! Much of the problem centers around the “myth that mental disorders are genetically or biologically caused”! No scientist of renown or respect any longer subscribes to this myth, articulating the more validated and reasonable multi-component etiology of these disorders. Still, the core hope of the medical model, and the bio-reductionistic philosophy was that “a biological or genetic anomaly would be found for every disease and this would lead to a mechanistic and easily delivered fix in the form of a mass produced, rapid delivery, and powerfully effective biological intervention.

Generally, allopathic medicine refers to “the broad category of medical practice that is sometimes called Western medicine, biomedicine, scientific medicine, or modern medicine[33]. The American healthcare system has evolved through a recent system of searching for chemical, surgical, humor balancing, and pathogen blocking efforts to eradicate illness and has finally come to realize that not all illnesses can best be approached in these ways. Certainly, many illnesses are influenced by these physiological and psychosocial states and modern scientists have come to believe in multi-pathway and multi-etiology illnesses which are the result of complex interplays of forces and effects on the human condition[34].

Recently, more advanced understandings of the auto-plasticity of the central nervous system and its' responsiveness to presses from the internal and external environment have made us realize that the brain indeed can and does change and with it personality, outlook, and capacity all change[35,36,37]. The work of Eric Kandel, Philip Goelet, and others at Columbia University and resulting in a Nobel Prize have determined that short-term memory is consolidated, put into permanent storage by growth of new synaptic connections that require intervening molecular genetic steps[38,39,]. Further, we now know a myriad of things, including drugs and medications that can mutate or act as negative autoplastic pressers on the brain[40]. This great brain of ours changes for better or worse based upon with whom we interact, how we treat it, which drugs we take, and what experiences impinge upon it.

It takes 4-5 years to change the mind of a high school student to change their personality (stylized way of perceiving reality and the self and others and formulating behavioral responses) and subcomponents related to the brain, role and function in a family and community and society, and related skill sets, and interpersonal skills and social tolerances to the point that they can function effectively in the classroom. We say,—We've made them a teacher (a professional mind)! It takes a three-year apprenticeship to fashion the expanded personality, skill set, and cultural expectations and brain connections to create an analytic plumber or carpenter. We say,—We made them a craftsman! It takes two years of premedical training and three to four years of professional school to create the brain, personality, acculturation, and vision and expectations of a physician. Because of the change in their brain we call them a doctor! It takes four years of college, four years of graduate school, and one year of internship to train the basic clinical psychologist, and another two years of graduate school and one year of residency to train the medical psychologist. We then call them doctor! All are examples of the neuroplasticity of the brain and the modifiability of the brain and its relatively enduring and characteristic ways of thinking, behaving, construing, and self-regulating we call personality.

The brain changes[41], and therefore personality can change. Brains, grow, make new connections, modify themselves, and change[42]. The mechanisms of how the brain changes and grows new neuronal connections by virtue of switching on and off genes in the neuron nucleus are becoming better understood. Experience is the essential component in setting this change in motion. The Nobel Prize winning scientist, Eric Kandel, indicates: —The fact that a gene must be switched on to form long-term memory shows clearly that genes are not simply determinants of behavior but are also responsive to environmental stimulation, such as learning[43]. Since the work of Kandel and others have become worldwide disseminated, no scientifically trained and well educated practitioner can doubt that focused and engineered experience, like psychotherapy, is a necessary component of any well-conceived healthcare delivery system. Finally, and most importantly, medication only approaches are not likely to be effective as standalone interventions in the treatment of brain driven illnesses[44].

We now know that childhood trauma and stress can increase a child's chances of everything from mental disorder, addiction, health problems, and dramatically decrease longevity and ability to heal from disease[45]. Even though managed care and its advocates have attempted to change psychotherapy into a short (6-12 session) psychoeducational intervention, there is ample evidence that it takes nearly 6 months of intensive treatment to make beginning changes and the longer you stay in treatment the more positive the outcome[46]. We have extant models for brain modification and recovery growth which summarize the auto-plasticity research, delineate the neuroscience of psychological therapies, and describe related techniques and procedures[47].

Even parents of generally considered normative children realize that augmentation strate-

gies related to grade achievement, selection test performance, and competition with others "change children's brains"! We now realize that the aging brain can have decline retarded with augmentation strategies (brain modification). We know emersion techniques are powerful changes of language, acculturation, and ethno-insight (brain change)! To give up on the brain, related personality, and related growth is a pessimism that healers simply can't afford! Yet, when we use bio-reductionistic, limited efficacy, and high risk/cost medication interventions for mental illness other than as one short-term technique and component of a more comprehensive brain change and growth program-we often succumb to pessimism and hopelessness! This pessimism is often witnessed in medically trained individuals who adopt the jaded philosophy that "the mentally ill never change and the best we can do is control symptoms"! Patients should run, not walk, from healers with such attitudes!

Practitioners of the future will have to explain to the patient how our psychoactive medications theoretically work (by disrupting normal neuronal functioning so that their acting out or in symptoms are lessened) and how our real goal is to capitalize on neurogenesis and neuroplasticity or growth to eventually get them off psychoactive drugs and thus remove the several risks of mutation of their central nervous system and organs and genetic switches in a way that can permanently change them negatively (side effects). Based on ethical and informed consent and malpractice risk management, the practitioner will review the science indicating that none of our psychoactive medications cure or permanently handle the symptoms of mental illness. We are responsible to fully inform them of the significant limits of medications like antidepressants and that the science shows that they do not work at all with over half of individuals and that they only work marginally better than placebo with the minority getting a positive effect. We must ethically and professionally inform them that neurogenesis and auto-plasticity offer a hopeful opportunity to change their brains, brain-based personality, and to modify the future of subsequent generations of their families[48,49,50,51,52]. The competent practitioner and healthcare team will be bound to explain that most cases of significant mental disorder have a significant and multigenerational family systems component and that the inclusion of the family and family therapy in the treatment plans is one of the most effective configurations of treatment.

One of the dynamics of decline of the family, Governments, and healthcare system is the loss of capacity to teach Self-Regulation (A Complex but Necessary Skill): Antidepressants as a Stand Alone Technique represents a Primitive and Inadequate Approach to the modification of the brain/personality/depression syndromes[53,54,56,56,57,58]! I, and psychophysiologists have written for years about the complexity of brain regulation and the emergence of that capacity to organize perception, understanding and insight, problem solving, goal directed behavior and strategies, learning and memory into realistic perception, choices, and self-management. In reality, humans have not one brain, but rather several brains that developed as specialized brains over the long period of evolution. These competing or collaborating brains require constant internal monitoring and switching among appropriate sectional expression or dominance and submission. The mature or healthy human being is practiced at regulating the many brains and brain systems and choosing and energizing and sustaining appropriate and goal directed behaviors. They have logged thousands of repetitions of self-regulation with guidance, correction, modeling, and fine tuning until new neural networks and connections exist or in the more prosaic languages of psychology objects are introjected, internalized, and solidified into object permanence. In other words, genetics and genetic transmission shaped by evolution guides the construction of basic underlying brains (or brain types). These brains have more or less basic underlying patterns of circuitry, lobe and sectional specialization, and neural connection patterns throughout the brain. For instance, genetics leads to a dominant brain/section functioning for emotions and impulses, one for vision, one for attention and recognition, and another for working memory and still another for conceptual memory, etc. and the tendency to

emphasize one brain over another, but experience and training define the way we choose to use and regulate this cacaughany of brains and their expression.

As the individual grows and interacts with the environment the autoplastic brains grow and establish interacting neuronal ties with different areas of the brain. Eventually, neurons that are fired frequently fire more quickly than others, some neuronal systems become auto-receptor oriented and act as breaks on neuronal systems, mirror neurons form and allow for modeling and vicarious learning and differentiation of the brain[50,51,52], and some areas of the brain become supersedant to others and act as integrating or output controlling and integration creating brains. In actuality, the human brain has demonstrated great capacity to learn and change given the proper motivation, feedback, and practice in virtually every paradigm that has been tested. A component of depression is global negativity, the view that who one is, what one has access to in regard to satisfying needs, and that others and the world is fixed in a negative schema, and this delusion of hopelessness and helplessness results in brain change! The depressed person no longer self-regulates or organizes brain area collaboration in a rational goal directed manner. The depressed person, in this philosophical position, sends biological messages to the brain which causes it to conserve energy and resources by giving up, not developing goals and strategies, lowering activation levels, and becoming cautiously disengaged! If we could find a pep pill (SSRI) that could overcome this philosophy and withdrawal, we would be blessed indeed! Still, this negative self-view, other-view, and world view, once solidified and fixed, protects the patient from "dashed hopes", "memories of failure (pain)", "embarrassing efforts during skill inadequacy/acquisition", and "seeming useless and unnecessary effort"! Thus, the secondary gain and related natural reinforcement can ossify the depressed philosophy and cause the patient to develop well known rational to defend and protect the hopeless and helpless schemas.

Should we really be so inane that we think a little pep pill will take care of this! Certainly, a little extra pep may punch up a HAM-D or other simple questionnaire two to four points (as the French say Walllagh! Or, as the South Americans would muse-Ago Mas?). But, have we really "treated the depressive syndrome and related perceptions and attitudes and philosophies underlying the brain change, or have we just put a little more pep into our depressed patient and congratulated ourselves far beyond our just deserts? To the informed we must appear completely narcissistic (to be so enthusiastically congratulating ourselves for such a puny drug intervention), out of touch with the total failure of faith, hope, and love (self and others) represented by this delusion of doom and pessimism! And, at least subconscious reinforcement of the giving up and pessimism by our cavalier and self-serving (because it is easy and not time consuming for us) delivery of a medication only technique! The small bump of hope and energy with a probable placebo effect surely runs its course and reinforces the pessimism when the patient's brain doesn't really change, upregulates and adapts to the "pep pill", and the syndrome doesn't change and relapse in inevitable! Then, the pessimistic physician and psychologist say, "You'll probably have to take this antidepressant for the rest of your life"! We create another "hope bump" with prescribing an augmented dose which also runs its course and results in feelings of failure and hopelessness. We eventually progress to the stage of being depressed with the job of treating rigidified depressive perception and ourselves?

Still, the elite psychotherapists understand the marvelous auto-plasticity of the brain. They comprehend that the epigenetic quality of the human brain, personality, and life's trajectory with regard to our ability to change is an essential! Depression cannot be treated by the pessimist whether they are using a drug delivery system, lobotomy, or are cynical philosopher. Growing awareness of the limitations of efficacy of psychoactive medications such as the rate of treatment response following first-line treatment with SSRIs has made us take stock of the im-

portance of the brain, personality, and course of human life cycles relative to change capacity rather than —symptom control and palliation. Even the top physician researchers are now realizing the importance of working with the patient's brain development and life trajectory and denaturing and damaging experience[18,19, 20,21,22,23].

Antidepressants:
The best example of "obsession with medications" to the exclusion of more comprehensive science is the SSRIs. The SSRI effect is moderate and really has nothing to do with positively changing the neural structure and connections in the CNS (actual and lasting brain modification in a meaningful way)[23]. Antidepressants work for a minority of only the most severe depressives and provide marginally significant effect over placebo which has an effect of similar magnitude[59]. Even though studies about antidepressant minority symptom control show that these drug companies imply that medication is the medical model's "first line" (preferred) solution to depression. In reality, and substantiated by a growing body of science, SSRIs literally work only marginally above placebo and in a minority of patients! If subjected to the requirement for powerful magnitude of effect and "scientifically validated treatments" in order to get approved for reimbursement "stand alone application of antidepressants would not be reimbursed" by most third party payers! Still tradition and the religion of antidepressants continues to make them one of the most prescribed medications! General physicians, having only antidepressants as an interventions for depressed patients and usually devoid of the diagnostic training to divine which type of depression being treated (Dysthymia, Major Depression and its subtypes, Cyclothymic Disorder, Bipolar Disorder and its many subtypes, Schizoaffective disorder and its subtypes, depressive episodes superimposed on Schizophrenic spectrum disorders, or brief depressive disorders in individuals suffering from Character disorders) are "put in charge of the treatment of most depressed patients in the US"! It has been estimated that 80 percent of antidepressant medications are prescribed by general physicians[60.] Still, general physicians are in charge of most of the diagnostic and treatment interventions for depression in the USA.

Remission rates (what drug companies and drug researchers call scores in the normal range on short and poor validity and reliability symptom check lists like the Ham-D) vary from a puny 30 to 45 percent[61]. The drug companies, and the average physician and psychologist are using antidepressants as titular one trick ponies. This occurs, even though most persons taking antidepressant medications will develop recurrent symptoms of depression while on the medication therapy[47,62], and most will return to depressive episodes within the year post treatment initiation[63]. This makes perfect sense only if you have given a "pep pill" and haven't really modified the global negativity and related non-goal and satiation oriented behaviors. Only one third of patients given antidepressants improve significantly. Placebo is just about as effective (non-chemical, or active component)[61], and only in the severe depressions (less than half of depressive illnesses) do we get really noteworthy effect. Thus, the "traditional first line and very expensive intervention of the primary care and medical establishment" may really work for less than 10% of total depressive illness. If your tire salesman, or butcher delivered a product that worked for you only 10% of the time you'd have real problems with that! Still physician referral sources and third party payers herd patients like chattel into these very marginally effective hands and approaches!

Thus science indicates resoundingly that "antidepressants cannot qualify as a stand-alone or first line treatment for depressive illness." As a medication only treatment they represent a marginally ethical and unscientific professional activity and at worse a collaborative (between prescribers, Government agencies, drug companies, and healthcare institutions) hoax[44,47,61,64]. Top practitioner and guideline groups, and now on backwater (never directly published on their web site) the FDA are on record supporting the idea that medication only

approaches‖ are not recognized as adequate or recommended for the treatment of most mental disorders.

Summary and Conclusions:

As the multi-pathway etiology of mental illness became apparent in the science driving the healthcare system and the auto-plasticity of the brain became firmly established old myths related to genetics as a universal cause of disease, the hopelessness of fixed gene determination, and health and science information exploded onto the internet, the old view of genes gave way to genes as switches and triggers influenced by environment[35] Recognition and management of mental health problems reduce the inappropriate use of medical and surgical care, thus reducing health care costs became one of the top primary care goals.[7,8,9,65]. Soon, the legal establishment will teach drug companies and prescribers that medication only approaches to the treatment of mental disorders is an inadequate and malpractice treatment plan (see: http://www.vaughns-1-pagers.com/medicine/index.htm). Innovative and early adopter physicians and healthcare facilities will include psychologists and their several mid-level behavioral health assistants in their hospitals and primary care systems. This will have great potential to cut long-term and catastrophic medical costs, increase system quality and efficacy, and improve the system's ability to contract in the emerging accountable care and competitive contracting era! Wise administrators will catch this vision and will hire a flexible and multi-disciplinary and holistic model chief medical officer and a compatible chief psychologist. Such innovative teams with a competent CEO (MBA or PhD) will drive the entrepreneurial healthcare systems of the future. They will have to be strong individuals who can redress the guild interests in the medical establishment, pharmaceutical houses, medical schools and psychology programs at universities, and in their workforces. Still the stakes and rewards will be very high!

Here are some strategic planning bullet points that might help such an innovative leadership trio!

1. Bust the myth of a single bio-mechanistic or genetic cause of mental illness and substance abuse with clearly stated program philosophies, plans for professional services, staffing and privileging plans that are truly multidisciplinary and allow all to practice to the extent of their licensure!
2. Gear up for team treatment, teamwork, and team system philosophies.
3. Always start with adequate diagnoses in MI and SUDs, and only then proceed to a comprehensive technique treatment plan (which may include both short-term-like medications, exercise, light therapy, etc., and long-term growth oriented therapies).
4. Put the most relevant doctor on staff in charge of the team (the team leader in some cases should be a physician where complex medical diagnoses complicate a depression, and a psychologist in others where the medical expertise is secondary in relevance to the psychological and family and life-style issues). Put the chief of psychology on the active medical staff so that teamwork and coordination emerges.
5. Force cross fertilization. Have mandatory medical and psychological education, outcome evaluations, post-case reviews and autopsies, and access to a library that helps encourage mastery of the science and related publications.
6. Understand how to hire an adequately trained chief psychologist that has a reasonable chance of succeeding in the highly complex, stressful, demanding medical facility environment and culture. Psychologists, and especially Medical Psychologists (see: http://www.amphome.org/) will be more versatile (trained extensively in advanced diagnostics, psychopharmacology, psychotherapy, and health and lifestyle counseling and interventions), cost effective, and primary care team compatible than psychiatrists and psychiatric nurses with very limited physical health and pharmacology training. General physicians, prescribing the psychoactive medications for almost two of every three patients with mental illness will need the specialty diagnostic, psychopharmacology, and follow-up monitoring and psychotherapy collaboration with psychologists to ensure quality of care, spread and cover their liability, and prevent excessive consumption of their primary care practice time.

7. Bust the myth that psychiatrists are the chief mental health diagnosticians and are the leadership in hospital units, mental health centers, and are necessary for leading the treatment of the most severely mentally ill. In reality psychiatrists are in such small numbers and limited geographical distribution that they could never assume or cover the leadership and treatment roles for the severely mentally ill. They generally do medication management and very little psychotherapy and advanced diagnostics, make a few minutes of rounds in psychiatric hospital units a day, and don't really do much more than approve decisions of much lesser trained staff (RNs and mid-level counselors). This situation creates a "sham leadership and quality of care" phenomena that is a false front, and the situation contributes to barriers to development of mental health hospital beds, day treatment programs, residential care beds, community mental health centers, and effective outpatient facilities. A senior psychologist diagnostician in collaboration with a well-trained general physician can more comprehensively diagnose mental and physical illness and devise a more accurate diagnosis and comprehensive treatment plan. Laws and rules should be modified to remove these barriers, utilize the doctor of psychology leadership workforce and geographic distribution and economic accessibility, and services could be expanded.
8. Remove the barriers to long-term psychotherapy and brain remodeling for the severely mentally ill and dually and multiply diagnosed patients. Even college faculty are given 4-5 years to remodel the brains of students, and the remolding of brain based personalities takes time and must additionally dissipate resistance and aberrant learning barriers.
9. Ensure that all mental health treatments of children and adolescents are required to start with diagnoses of the identified patient, individual family members that report symptoms, and the family system. Medication only and individual psychotherapy only approaches to these disorders should be prohibited and reimbursement barriers to comprehensive diagnoses and treatment of these families should be removed.
10. Bust the myth that appropriately post doctorally trained psychopharmacology trained psychologists and Board Certified Medical Psychologists (see www.amphome.org) are "unsafe diagnosticians and prescribers" and allow them to prescribe psychoactive medications for patients that they are treating with psychotherapy, family therapy, substance abuse therapies, neuropsychological deficits, and behavioral aspects of medical disorders. These psychologists have written millions of prescriptions and managed mentally ill patients in several states, all of the military, and in other organizations in the US and the world.

The problems in the mental health system related to the high costs of "medication only approaches" (higher long-term medical costs offsets, higher incarceration rates, higher emergency department use, limited personality and lifestyle change leading to lost productivity and potential, failure to change the multigenerational trajectory in dysfunctional families and repeated and amplification of the above costs, and ethical and credibility impoverishment in the mental health and healthcare system) are huge! The costs of turning the mental health system over to the few psychiatrists in the USA and their tendency to desert treatment and practice with largely "medication only approaches" has been dangerous, costly, and simple minded. To abdicate getting consistently and intimately involved with patients and families and in "hands on and fully involved" leadership roles in psychiatric hospitals, residential care centers, day treatment centers, and outpatient programs has reduced the quality of the system significantly. The failure of psychologists to speak up about these things and their willingness to collaborate and collude in the "hoax system" is embarrassing and disqualifying for many psychologists seeking leadership roles in the healthcare system. The failure of the Government to investigate the "fly by" (few minute) medical rounds provided by most psychiatrists on inpatient units, and the signature of stacks of treatment plans without any real understanding of the patient and their diagnoses and their course in the treatment center is shocking. The running 8-10 patients through (supposed 20 minute) medication checks and medical psychotherapy each hour in outpatient settings is to invest in either unscientific fantasy or delusion. To briefly interview the patients, fail to compile multi-generational psychosocial histories, and to find out what is really going on under the auspices of "psychiatric leadership" in the mental health sys-

tem is embarrassing. Any student of the mind and the personality understands that this is pure fictional diagnosis and pretense rather than actual scientific acumen and treatment!

The mental health tragedies related to acting out and ultimate imprisonment of the mentally ill, death by overdose, acting out and harming others, and mostly just giving up on the mental health and medical systems is a Great American Embarrassment! Well educated physicians and psychologists hear these high profile cases in the media and never raise the question "were they adequately diagnosed and in appropriate treatment"! These psychologist and physician doctors don't bother to challenge the media when they "tout taking a medication as if that is TREATMENT rather than simply a small technique that has no chance of changing the person"! Still, these are not hard things to know, figure out, or investigate. The fact that they continue is a testament that pretends and looks the other way.

References

1. Wiggins, J.G. (2013). Milestone Events in the History of Develoment of Medical Psychology. In Medical Psychology Practice and Policy Perspectives. Caccavale, J.(Editor). NAPPP Books, Los Angelas, California.
2. Ross, L. (1916). Medical Psychology. Point Loma, The Aryan Theosophical Press.
3. Ahn AC, Tewari M, Poon C-S, Phillips RS (2006) The Limits of Reductionism in Medicine: Could Systems Biology Offer an Alternative? PLoS Med 3(6): e208. doi:10.1371/journal.pmed.0030208
4. Patient Protection and Affordable Care Act (Public Law 111-148). Enacted 3/23/10.
5. Ornish, d., Brown, S. E.k Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990). Can lifestyle changes reverse coronary heart disease? Lanxet, 336, 129-133.
6. Boll, T. J.(2002). Introduction to the series. Boll, T. J., (Editor-in-Chief). Handbook of Clinical Psychology. Medical disorders and Behavioral Applications. Vol. 1, APA, Washington, DC.
7. Cummings, N. A., & Follette, W. T. (1968). Psychiatric services and medical utilization in a prepaid health plan setting: PartII Medical Care, 6, 31-41.
8. Caccavale, J. (Editor). Medical Psychology Practice and Policy Perspectives. NAPPP Books, Los Angelas, California.
9. Cummings, N. A., O'Donohue, W., & Ferguson, K. (Eds.) (2002). The Impact of Medical Cost Offset on Practice and Research: Making it work for you. Reno, NV: Context Press.
10. Morris, J. A. (Ed.) (1997). The rural psychologist in the hospital emergency room, Chapter 6, p. 81-96., in *Practicing Psychology in the Rural Setting: Hospital Privileges and Collaborative Care.* Washington, DC: American Psychological Association.
11. Morris, J. A. (2010). *Principles of Alcohol Detoxification for Collaboration Among Psychologists and Other Medical Professionals. The Archives of Medical Psychology, Nov., Vol. 1, Issue 2.*
12. Morris, J. A. (2013). Changes in Medicare Hospital and Healthcare Facility Rules Affecting Psychology. The Archives of Medical Psychology. Aug., Vol. 4, Issue 1.
13. Morris, J. A., & Lawson, W. (1998). Neuropsychological deficits in patients with alcohol and other psychoactive substance abuse and dependence: A pilot study. *Alcoholism Treatment Quarterly,* J:'TIJ.(4).
14. Morris, J. A., (1995). Alcohol and Other Drug Dependency Treatment: A Proposal for Integration with Primary Care. *Alcoholism Treatment Quarterly,* 13(3),4556.
15. Morris, 1. A. (1986). Interdisciplinary treatment of alcoholism. *Alcoholism Treatment Quarterly,* 3(4),125-131.
16. Kroenke, K., & Manglesdorff, D. (1989). Common symptoms in ambulatory care: Incidence, evaluation, therapy, and outcome. *American Journal of Medicine, 86*, pp. 262-266.

17. Kunen, S., Niederhauser, R., Smith, P., Morris, J. A., & Marx, B. (2005). Raced disparities in psychiatric rates in emergency departments. *Journal of Consulting and Clinical Psychology, 73* (1), pp. 116-126.
18. Healy, D. (1997). The anti-depressant era. Harvard University Press, Cambridge, Mass.
19. Healy, D. (2012). *Pharmageddon*. Berkeley: University of California Press.
20. Breggin, P. R. (2008). *Medication Madness: A Psychiatrist Exposes the Dangers of Mood-Altering Medications*. New York: St. Martin's Press. ISBN 978-0312363383.
21. Amen, D. G., Routh, L. C. (2003). Healing Anxiety and Depression. Penguin Group, New York.
22. Szasz, T. (2003). Ceremonial Chemistry: The Ritual Persecution of Drugs, Addicts, and Pushers (Revised Edition). Syracuse University Press, Syracuse NY. ISBN 13244-5160.
23. Morris, J. A., & Caccavale, J. L. (2013). Antidepressant Medication Claims are ineffective and misleading. In Medical Psychology Practice and Policy Perspectives, Caccavale, J. (Editor). NAPPP Books. LA, California.
24. Morris, J. A. (2013). The golden age of psychology: Serving the public health facilities need in the age of integrated care. In Medical Psychology Practice and Policy Perspectives, Caccavale, J. (Editor). NAPPP Books. LA, California.
25. Institute of Medicine (U.S.), National Academies of Science, Advising the Nation. Improving Health. *Improving the Quality of Health Care for Mental and Substance- Use Conditions: Quality Chasm Series.* http://www.nap.edu/catalog/11470.html. Pages 1-504,2006.
26. World Health Organization, (2001). "Legal Status of Traditional Medicine and Complementary/Alternative Medicine: A Worldwide Review" (PDF). *World Health Organization*. World Health Organization. 2001. http://whqlibdoc.who.int/hq/2001/WHO_EDM_TRM_2001.2.pdf. Retrieved 2007-09-12.
27. Grant BF, Dawson DA, Stinson FS, Chou SP, Dufour MC, Pickering RP. The 12month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991-1992 and 2001-2002. Drug Alcohol Depend. 2004;74:3,223-234.
28. Morris, J. A. (Ed.) (1997). The rural psychologist in the hospital emergency room, Chapter 6, p. 81-96., in *Practicing Psychology in the Rural Setting: Hospital Privileges and Collaborative Care.* Washington, DC: American Psychological Association.
29. Cost-effectiveness of depression case management in small practices. Gensichen, Jochen; Petersen, Juliana J.; von Korff, Michael; Heider, Dirk; Baron, Steffen; König, Jochem; Freytag, Antje; Krauth, Christian; Gerlach, Ferdinand M.; König, Hans-Helmut The British Journal of Psychiatry, Vol 202(6), Jun 2013, 441-446. doi: 10.1192/bjp.bp.112.118257.
30. Katon,W.; Schulberg,H. (1992). General hospital psychiatry, Volume 14, Issue 4, p.237-247.
31. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, Johnson (2008). Initial severity and antidepressant benefits: a meta -analysis of data submitted to the Food and Drug Administration. PLoS Med;5:e45.
32. Marcus, S. C. and Olfson, M. (2010, December). "National trends in the treatment for depression from 1998 to 2007." *Archives of General Psychiatry* 67(12), pp. 1265-1273.
33. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS (January 1996). "Evidence based medicine: what it is and what it isn't".*BMJ* 312 (7023): 71–2. doi:10.1136/bmj.312.7023.71. PMC 2349778.PMID 8555924.
34. Olfson, M. and Marcus, S. C. (2010, December). "National trends in outpatient psychotherapy." American *Journal of Psychiatry* 167(12), pp. 1456-1463.
35. Cappas, N. M., Andres-Hyman, R., & Davi, L. What Psychotherapists Can Begin to Learn from Neuroscience: Seven Principles of a Brain-Based Psychotherapy. Psychotherapy: Theory, Research, Practice, Training 1997 Copyright 2005 by the Educa-

tional Publishing Foundation 2005;42,(3):374–383.
36. Tarter, R. E., & Vanyukov, M. (1997). Alcoholism: A Developmental Disorder. In Marlatt, G. A., & VandenBos. Addictive Behaviors: Readings on Etiology, Prevention, and Treatment. American Psychological Association. P. 43-67.
37. Kandel ER, Schwartz, JH, & Jessell, TM. Principles of Neural Science. 2001 McGraw-Hill. New York. From Neuroscience: Seven Principles of a Brain-Based Psychotherapy. Psychotherapy: Theory, Research, Practice, Training 1997 Copyright 2005 by the Educational Publishing Foundation 2005;42,(3):374–383.
38. Kandel E.R. A new intellectual framework for psychiatry. Am J Psychi, 1998; 155, 457–469
39. Green, C. S., & Bravelier, D. (2008). Exercising Your Brain: A Review of Human BrainPlasticity and Training-Induced Learning. *Psychology and Aging.* Vol. 23, No. 4, 692-701.
40. Keller, T., & Just, M. (2009). Behavioral training improves connectivity and function in the brain. Neuron, Dec. 10.
41. Hirsch, J. B. (2010). The weight of being; Psychological perspectives on the existential moment. New Ideas in Psychology. Vol 28(1), 20-36. 16. Kandel, (2006). In Search of Memory: The Emergence of a New Science of Mind. W.W. Norton & Company, New York. A. P.276, B. P.261-276.
42. National Institute of Health, Public Access (2007). Brain research. Author manuscript, available in PMC 2007, April 26. Published in final edited form as: Brain Research 2007 February 23; 1134(10; 148-161.
43. Wei-Chung Allen Lee, Hayden Huang, Guoping Feng, Joshua R. Sanes, Emery N. Brown, Peter T. So, Elly Nedivi (2006). Dynamic Remodeling of Dendritic Arbors in GABAergic Interneurons of Adult Visual Cortex. Volume 4, Issue 2, e29.
44. Caccavale, J., Cummings, N., Morris, J., Reinhardt, D., Rubin, H., Wiggins, J. (2010b). Failure To Serve: *A White Paper on The Use of Medications As A First line Treatment And Misuse In Behavioral Interventions This report was prepared by:* The National Alliance of Professional Psychology Providers, www.nappp.org .
45. Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D., Spitz, A., Edwards, V., Koss, M.P., Marks, J. S (1998). Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ADCE) Study. *American journal of Preventive Medicine.* Vol. 14, Issue 4, p. 245-258.
46. Miklowitz, D. J., Otto, M. W., Frank, E., et al., (2007). Intensive psychological interventions enhances functioning in patients with bipolar depession: results from a 9-month randomized controlled trial. American Journal of Psychiatry; 164: 1340-7.
47. World Health Organization, (2009). Pharmacological treatment of mental disorders in primary health care.. *World Health Organization*. World Health Organization. WHO Library Cataloguing-in-Publication Data, ISBN 978 92 4 154769 7 (NLM classification: QV 77.2), http://www.who.int/mental_health/management/psychotropic/en/.
48. Purves & Lichtman, (1985). Principles of Neural Development. Sunderland, MA; Sinauer, Associates, Inc.
49. Folensbee, R. W. (2007). The Neuroscience of Psychological Therapies. Cambridge University Press, New York.
50. Iacoboni, M., Molnar-Szakacs, I., Gallese, V., Buccino, G., Mazziotta, J.C., Rizzolatti, G., 2005. Grasping the intentions of others with one's own mirror neuron system. PLoS Biol. 3, e79.
51. Arden, J. B. & Linford, L. (2009). Brain-Based Therapy with Adults: Evidence-Based Treatment for Everyday Practice. John Wiley & Sons, Inc. Hoboken, New Jersey.
52. Fahle, M., & Poggio, T. (2002). Perceptual learning. Cambridge: MIT Press.
53. Karni, A., Meyer, G., Rey-Hipolito, C., Jezzard, P., Adams, M. M., & Turner, R. (1998).

The acquisition of skilled motor performance: Fast and slow experience-driven changes in primary motor cortex. Proceedings of the National Academy of Science, 95, 861–868

54. Willis, S. L., Tennstedt, S. L., Marsiske, M., Ball, K., Elias, J., & Koepke, K. M. (2006). Long-term effects of cognitive training on everyday functional outcomes in older adults. Journal of the American Medical Association, 296(23), 2805–2814.
55. Ansari, A. & Osser, D. N. (2010). The Pharmacology Algorithm Project at the Harvard South Shore Program: An Update on Bipolar Depression. *Harvard Review of Psychiatry*. Bol. 18, Number 1.
56. Moncrieff, J. (2009). The myth of the chemical cure: A critique of psychiatric drug treatment. Palgrave Macmillan. New York.
57. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, Johnson (2008). Initial severity and antidepressant benefits: a meta -analysis of data submitted to the Food and Drug Administration. PLoS Med;5:e45.
58. Scott, T. (2006). America Fooled: The truth about antidepressants, antipsychotics, and how we've been deceived. Argo Publishing, LLC., Victoria, Texas. pp. 157-186.
59. Caccavale, J. L., Rubin, H. S., & Cummings, N. A. (2013). The need for science-based prescribing standards in mental health treatment. In Caccavale, J. (Editor), Medical Psychology Practice and Policy Perspectives. NAPPP Books. LA, California.
60. Lieberman, J. A., (2003). History of the use of antidepressants in primary care. Journal of Clinical Psychiatry 7, sppl. 7, 6-10.
61. Scott, T. (2006). America Fooled: The truth about antidepressants, antipsychotics, and how we've been deceived. Argo Publishing, LLC., Victoria, Texas. pp. 157-186
62. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, Johnson (2008). Initial severity and antidepressant benefits: a meta -analysis of data submitted to the Food and Drug Administration. PLoS Med;5:e45.
63. Caccavale, J., Cummings, N., Morris, J., Reinhardt, D., Rubin, H., Wiggins, J. (2010b). Failure To Serve: *A White Paper on The Use of Medications As A First line Treatment And Misuse In Behavioral Interventions This report was prepared by:* The National Alliance of Professional Psychology Providers, www.nappp.org .
64. Ansari, A. & Osser, D. N. (2010). The Pharmacology Algorithm Project at the Harvard South Shore Program: An Update on Bipolar Depression. *Harvard Review of Psychiatry*. Bol. 18, Number 1.
65. Sthal, S. M. (2008). Stahl's Essential Psychopharmacology: Neuroscientific Basis andPractical Applications. Cambridge University Press, New York. P. 177-194.