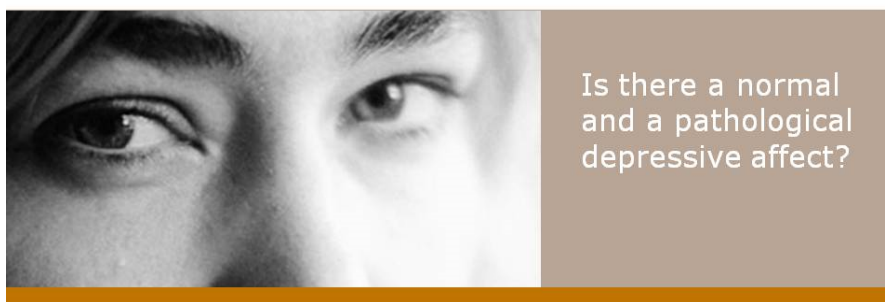


# Is there a normal and a pathological depressive affect?

Speech held by *Daniel Hell* at the 13<sup>th</sup> “Congresso Nacional de Psiquiatria” in Vilamoura, Portugal, on Jan. 25, 2018.

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Is there a normal  
and a pathological  
depressive affect?

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**Congresso Nacional de  
Psiquiatria**

**Vilamoura. 25.1.2018**

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In general, depressive or low mood is judged as a negative thing. It is often understood as a symptom of an illness. In psychiatry, it is one of several criteria for the diagnosis of a depressive episode, even one of the most important ones - as is well known.

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## Diagnostic criteria of depressive episodes (ICD 10)

red: affective symptoms

### A. Main symptoms

1. Depressive mood
2. Loss of interest and pleasure
3. Low energy or fatigability

However, it is not possible to diagnose a depressive episode only with affective symptoms.

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**Diagnostic criteria of depressive episodes  
(ICD 10)**

blue: cognitive symptoms  
black: somatic symptoms

B. Additional symptoms

1. Reduced concentration and attention
2. Reduced self-esteem and self-confidence
3. Ideas of guilt and unworthiness
4. Bleak and pessimistic views of the future
5. Suicidal thoughts and acts
6. Disturbed sleep
7. Diminished appetite

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Cognitive and somatic symptoms are necessary, too. Low mood does not prove that a person suffers from a depression. This is a possible clue for the assumption that a depressive affect is not always pathological. Even a very strong depressive affect must not be a symptom of depression or another illness. It has – as you know – to last for at least two weeks to be a symptom of depressive episodes. In the case of low mood, which is below the threshold of a depressive episode, it has e.g. to be present for years to fulfill the criteria of dysthymia.

Consequently, this poses some questions: What kind of functions has low mood? What makes it pathological? Is there a difference between normal and pathological depressive affects? Is it possible to differentiate between normal and pathological by measuring the strength or time course of low mood? Or is there a different quality of normal and pathological depressive affects?

At first I want to discuss some of the possible functions of low mood and its cognitive and behavioral consequences. Later on I will discuss reasons for low mood to possibly get dysfunctional. I will then present a model of depressive development which is based on a vicious circle, leading to a loss of functionality. I end my presentation with possible consequences of this concept for therapy.

## Agenda

- Possible functions of low mood
- Reasons for dysfunctionality
- Model of depressive development („from low mood to depression“)
- Some Consequences for Therapy

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Almost everybody experiences low mood in his or her lifetime, sometimes more, sometimes less. When you remember situations during which you felt bad, you usually think of frustrating situations, which you could not alter immediately. This could, for example, be a somatic illness, a chronic conflict, a strong blaming or an important loss, which not only made you sad, but first and foremost troubled you with low mood, a sense of emptiness.

Perhaps you can remember such a situation. I personally think of private and professional situations when it was impossible for me to avoid consuming and frustrating conflicts without risking heavy consequences for my life. Or I think of deceptions of own plans and goals or of being willingly or unwillingly blamed by persons. In all these cases I felt suppressed for a shorter or longer time, unmotivated, empty, sullen, moody, without energy and in the same time unquiet, sleep disturbed, constantly thinking about possible solutions. I was in a low mood or had a bad temper. On the long run, these moments had no bad effect. I had to pass through difficult life events without reacting uncontrollably, although the setting was very uncomfortable. Low mood was a strategy for avoiding risks. It helped with finding a better solution. A more risky or even a more manic reaction probably would have had a worse outcome for me.

I did not choose this type of reaction willingly. At least in the first moments I was directed or even somatically forced to do so. Evolutionary biologists speak of an inherited kind of reaction. You can find this type of reaction also in animals if higher organisms cannot escape a dangerous situation by flight or attack.

## Preservation reaction in animals and humans (Lowering the risk of further damage)

### Biological (animal experiments)

- Immobility
- Submission
- Passivity

**= Inhibition of actions**

### Psychological (human experience)

- Low mood
- Loss of interest
- Unrestful thinking

**= Low mood**

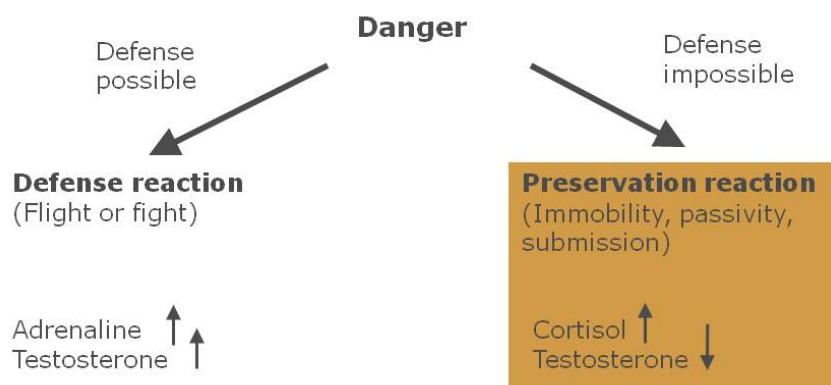
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They react with immobility, passivity, and social retreat or submission, probably to avoid further disadvantages. Some decades ago Henry and Stephens differentiated between a defense reaction and a preservation reaction.

## Inhibition of actions as a preservation reaction



Stress model by Henry and Stephens

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Preservation means to keep safe, what can be rescued in a situation of loss or defeat. Some study groups (like Shively et al., 1997) demonstrated that apes in experimentally caused loss of a dominant rank react with this behavior of passivity, immobility, and submission. This way they avoid exhaustion and expulsion of the group. In primates there is also a reaction which Price (1994, 2004) called “inverted flight”. It means that a primate does not fly or attack in a conflict with a stronger ape. He keeps quiet in a short distance from the dominant ape, but is also attentive to what happens and looks for a better outcome. We do not know how apes feel, but their reaction is similar to human behavior in low mood. Price (2004) interprets it as a “No threat message”, as an attempt, “to cut the losses” and “to leave the fight to another day”.

### Functions of the preservation reaction in animals (Price 2004)

- Peace appeal, „no threat message“
- Safeguarding, „cut the losses“
- Withdrawal, „leave the fight to another day“

Human reactions are more complex, among others because of our self-consciousness.

## Combination of social and psychomotor inhibition and mental restlessness in depressive episodes

- Loss of *exterior* drive (psychomotor inactivity and social immobility)
- *Interior* unrest (rumination)

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Besides the inhibition of psychomotor and social activity, there is also an activation of problem oriented thinking or mental agitation.

There are different hypotheses by evolutionary biologists to interpret depression:

## Alarming and protective functions of low mood (mild depressive affect)

- Psychic pain hypothesis (Thornhill, 1991, a.o.)
- Social risk reduction hypothesis (Allen 2006 a.o.)
- Analytical rumination hypothesis (Andrews and Thompson, 2009)
- Behavioral shutdown hypothesis (Gilbert, 2006 a.o.)

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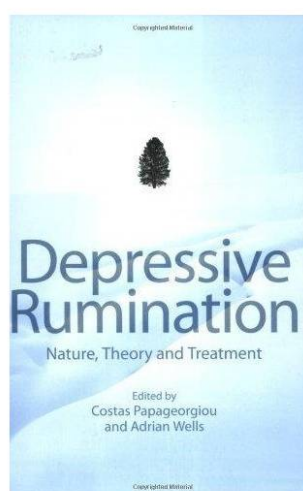
Some think that low mood or depressive affect is a kind of psychic pain and functions in the same way as an alarming message like somatic pain. It uses even the same brain mechanisms in the anterior cingulate cortex like somatic pain.

Others postulate that low mood helps to avoid exclusion from social groups. It is an adaptive response to being sensitive to social risks by inhibiting competitive behavior and by signaling some need for help.

Another hypothesis is the so-called analytical rumination hypothesis. Low mood increases the individual focus on a problem by inducing a helpful kind of analytical thinking. This happens by activation of the left ventrolateral prefrontal cortex, often seen in depression.

The behavioral shutdown hypothesis states that retreating and reducing psychomotor exposure is the best strategy if an organism is faced with more risk than reward from social activities.

All these hypotheses cannot explain the bigger risk of suicide and some other fundamental problems in more severe depression. To take an example: constant rumination – demonstrated by the studies of Noelen-Hoeksema – is a burden for many depressive patients, which does not lower distress and depressive symptoms but enhances them.



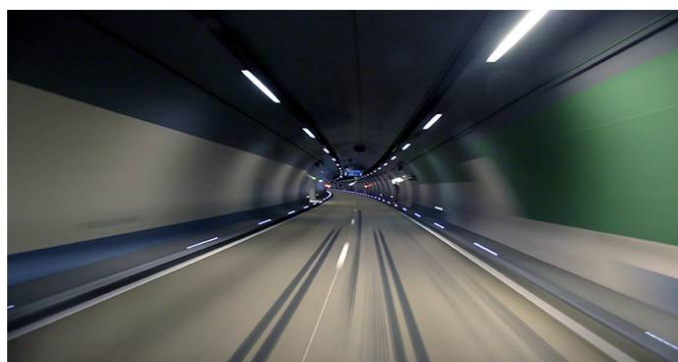


Low mood is not only functional in certain situations, it gets dysfunctional in others.

## Depression as a dysfunctional adaption (Hell, 1994, 2006, Nesse, 2005 a.o.)

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Low mood and the preservation reaction are important to understand depressive mechanisms (**as a kind of brake**)



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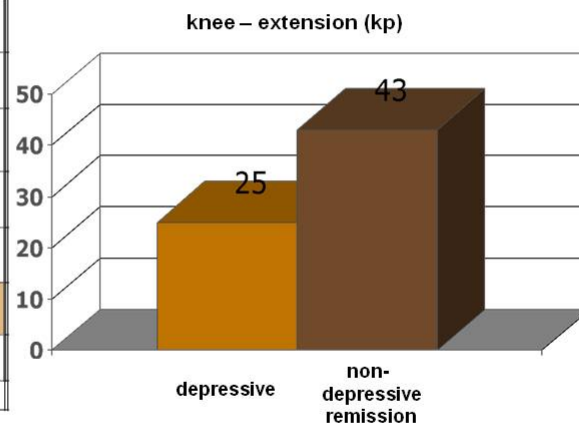
It can be a dysregulated adaption. But what is well expressed by these theories is the fact that low mood and the preservation reaction are important to understand depressive mechanisms. Its usefulness has something to do with its problems. Like anxiety disorders have something to do with the normal and physiologic anxiety, depression is better understood if the personal and social function of low mood is studied. Depression is very often a deficit, but a deficit that starts from a functional base. In my opinion, you have to understand the physiology of an affect before you can observe its disorder. Low mood is not sadness. It is not an intensive emotion, but a slowdown of social behavior, a kind of psychic brake. If you cannot release the brake, you have a big problem. But if you have no brake, you have another, not smaller problem.

In former years at the University Hospital of Psychiatry in Zurich I have studied the motor system and the mobility of a big and representative sample of depressive patients with some colleagues. We were able to show that muscular strength, for example the extensors of the knee, is lower in depressive state than after remission.



## Difference of the isometrical muscle power in depressive und non-depressive people

variable	p
shoulder-abduction	< .01
shoulder-rotation	n.s.
elbow-extension	< .05
grip-force	< .05
knee-extension	< .0001
hip-extension	< .01



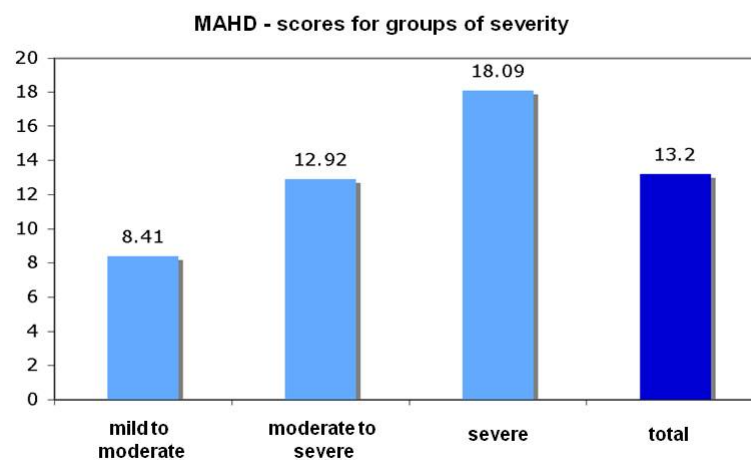
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We also found a highly significant correlation between loss of muscle strength and the extent of a depression.

## Difference of muscle power in groups of patients with mild, moderate and severe depression

(Hell and Weber 2009)



We could demonstrate that other psychomotor qualities are also lowered in depression: facial expression, gesture, voice, pace.

## Clinically observable changes in the psychomotor activity of depressive patients

(Studies at the PUK Zurich)

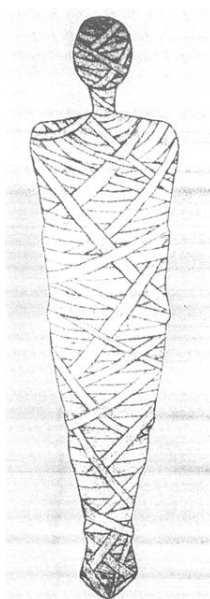
- Gesture, attitude: Inhibition (Weber and Hell 2008)
- Mimic, eye contact: Decrease (Kästner 1989, Endrass 2000, Braunschweig 2001)
- Language, voice: Extension of speaking time, monotony (Stassen et al. 1991, 1998, 2002)
- Muscle strength: Loss of strength (Bühler 1998, Bader et al. 1999)
- Gear: shortened stride length and walking distance (Bader and Hell, 1999)

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These results signalize that a behavioral shutdown is common in depression. What is soft and short in normal low mood can become a handicap in severe depression. In extremis, a patient can be paralyzed, as illustrated in a picture by a patient.



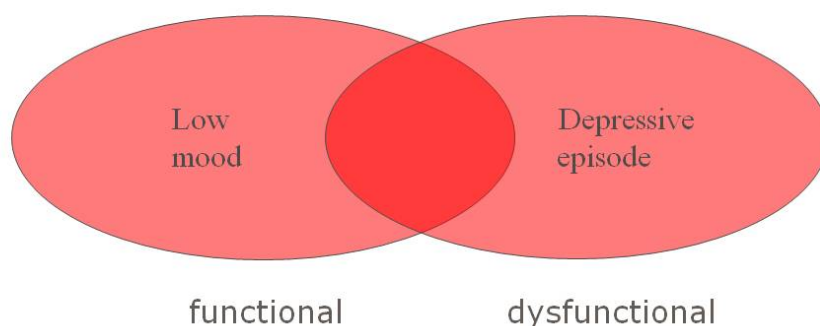
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The same is true for changes in thinking. The concentration on a seemingly unsolvable problem can help finding a solution later on. But it can be frustrating and endless.

I conclude that there is a potential development from functional low mood to dysfunctional depressive affect. But I think that there is no clear cut frontier between normal and pathological affect and that there are mixtures of functional and dysfunctional moods in mild and moderate depressions. That has some implications for therapy, which I will discuss at the end of my presentation.

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### Functional and dysfunctional depressive mood during depression

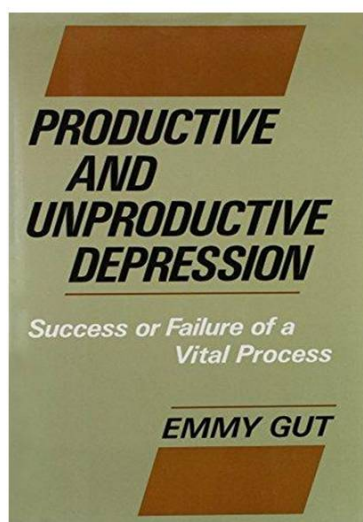


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The psychoanalyst Emmy Gut goes a step further in her book “Productive and unproductive depression”, with a foreword by John Bowlby. She describes that a more severe depression can be productive or useful, too, if a person becomes more introverted and gets more open for hidden or unconscious thoughts.



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With a foreword  
by John Bowlby

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I started by saying which constellations are typical for normal low mood. The epidemiologic studies on depression have found similar causes for more severe depressive affects. There are at least 4 main routes to depression, namely by somatic illness, severe losses, chronic exhaustion, and by blaming.

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## Main Causes of Depression

There are at least **four routes to depression**:

1. **Somatic illnesses** (e.g. deficits of hormones or vitamins, neurologic disorders)
2. **Severe losses** (family or professional) or chronic conflicts and isolation
3. **Chronic exhaustion** (e.g. severe burnout)
4. **Blaming** (e.g. mortification, mobbing)

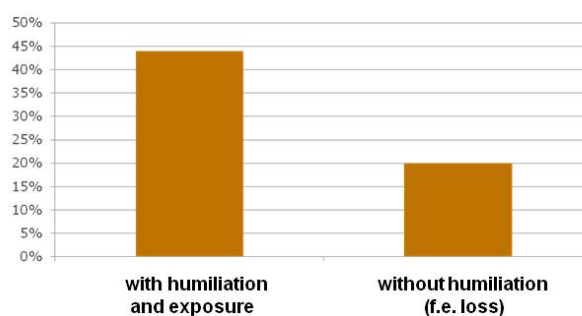
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The last factor is often underestimated as has been shown at first by Brown.

### Depression rate after stress



**Depression rate** in 130 women (with low self-esteem and low social support) **depending on type of stress** / Brown 2004

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Severe blaming has a more depressogenic effect in the course of a vulnerable population (left chart bar) than the death of a near relative (right bar). That makes it probable that depressions often start with functional low mood but end with dysfunctional affect reactions, something like “depression developing on depressive mood” or in short “depression on depression”. This is similar to the vicious circle of anxiety, a kind of “anxiety about anxiety”.

### „Depression on depression“ (vicious circle of low mood)

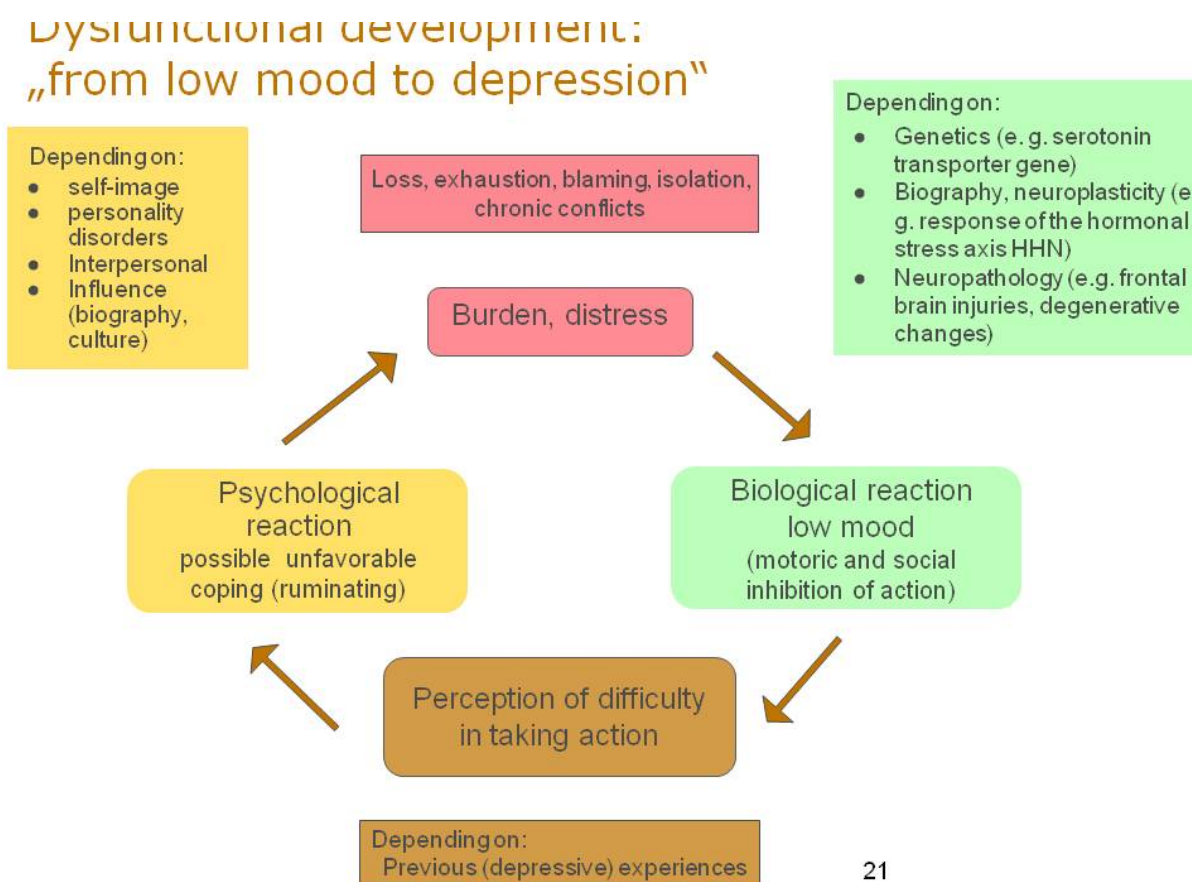
- Low mood protects from further social risks in an aversive situation by *inhibiting* psychomotor or social activity (likewise: **anxiety** protects from vital danger by *activation*)
- The risk of low mood is the development of a vicious circle between enduring inhibition and unfavorable coping strategies, leading to greater frustration and distress

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This dysfunctional development of depression can be illustrated by the following model (Hell, 2009):



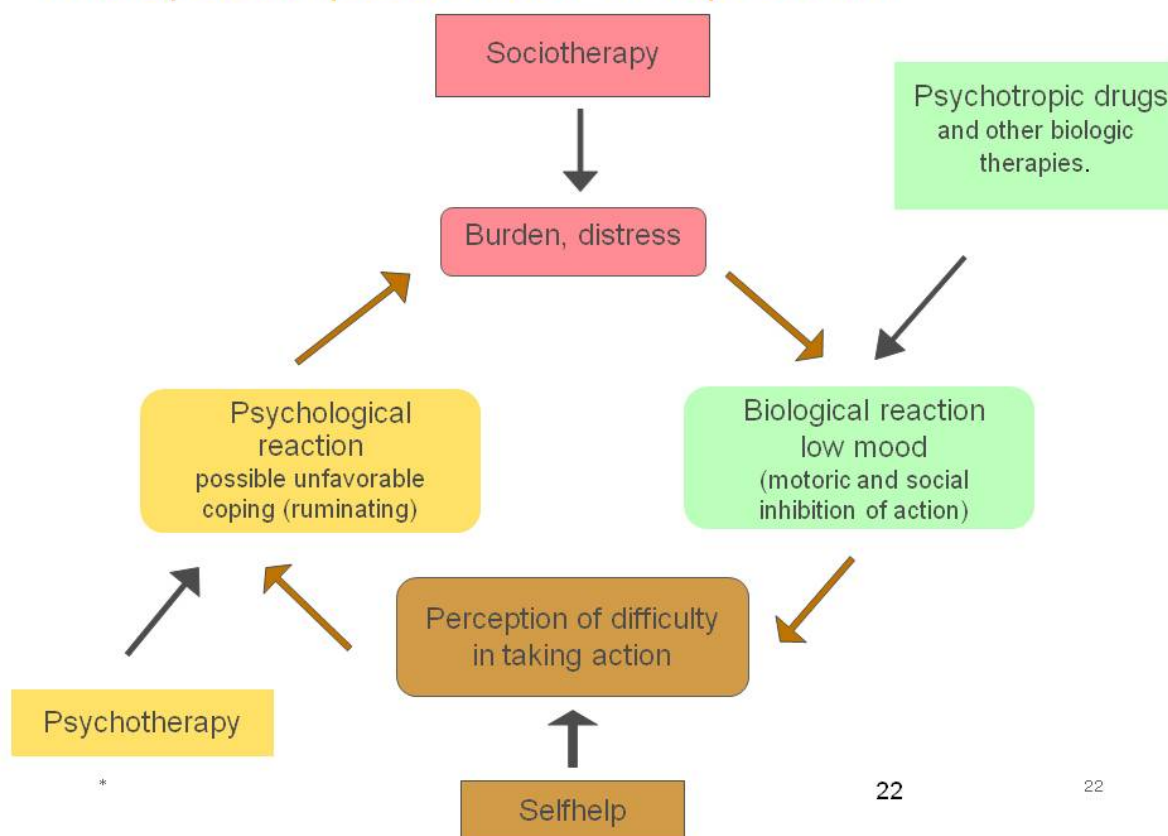
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It starts with a typical, already described distress, which is followed by a biologically anchored reaction of low mood. This is self-consciously experienced by men and women, but not in the same way by animals. The conscious perception leads to a psychological reaction. Dependent from earlier experiences and biographic influences, low mood is more or less accepted. There are favorable and unfavorable ways of coping. If low mood is evaluated as dangerous and destructive or if it is not compatible with the self-ideal, the risk of fighting unsuccessfully against the normal biological reaction will grow. To defeat in this struggle is very frustrating and enhances the distress. That makes it even more difficult to accept a now stronger depressive affect.

Psychotherapy tries in one or another manner to change the unfavorable coping with depressive affects. Interpersonal forms of therapy focus directly on stress making situations. Biologic therapy, especially medication, seeks to alter the neurophysiologic base of depressive affect. Each kind of therapy has its own rationale. Its success depends on the extent

or severity of the depression, on psychosocial factors, on the personality of the patient, and the relationship between patient and therapist.

## Therapeutic possibilities in depression



If the depressive affect still has some functional or protective effect, it should not be aggressively treated. In the guidelines of most European countries it is suggested to “wait watchfully” some time with special therapies in mild depressive episodes, because they are often reversible. If a person can feel intense sadness, it indicates that the depressive affect is not very strong. Therapy should not hinder this reaction.



## Therapeutical Consequences (According to guidelines from DGPPN)

Mild depressions (< 16 points on the Hamilton Depression Scale) are often reversible

Therefore, according to new European guidelines, in the case of mild depression, an accompanied medical surveillance for a few weeks is justifiable

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It seems important to me to differentiate in each case if a person suffers mainly from the stressing life event or from the following depressive affect and inhibition of actions, from the secondary problematic reactions of relatives or more seldom from somatic problems. In my opinion, therapy has to focus on what the patient is mainly stressed by. It is often the stressing life event, but in our success oriented and competitive society it is more and more the depressive inhibition of action itself that is unacceptable, sometimes combined with a narcissistic self-image which prevents a helpful coping. But that is another topic.

## What is the main burden for depressive people?

Depending on the patient:

- Triggering stressor (usually a loss, chronic conflicts or isolation)
- Consecutive inhibition of action
- Secondary reaction of the environment
- Somatic problems (pain, fatigue)

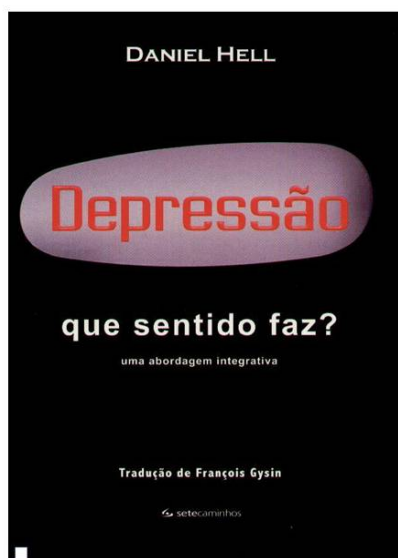
→ Different starting points for therapy

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If you are interested in these questions, you will find further material in the book “Depressão – que sentido faz? Uma abordagem integrativa”, translated by your colleague Francois Gysin.



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Thank you very much for your attention.



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