Mood and Food

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Objectives:
Identify effective methods for the practical application of concepts related to improving the delivery of services for persons with developmental disabilities

Notes:
Introduction

- Present from early life.
- Usually improve as the child grows older.
- However, entail impairments that continue through adult life.
- There is a strong genetic component.
- M>F.

Theories of DD

1. Genetic: Abnormalities pre-determined
   - about 1 : 300 children are born with spontaneous genetic mutations associated with rare developmental disorders.
2. Environment disrupts normal development (stress in early childhood)
   - DD caused by early childhood significant trauma. DD in traumatized children = 71% in adults.
   - Even small stresses can accumulate to result in emotional, behavioral, or social disorders in children
3. Combination of environmental and genetic factors.

Outline

- Introduction
- Autism Spectrum Disorder
- ADHD
- Reward pathway and food
- Food addiction and mood
- Vitamin deficiencies

Mood and Food

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As this population ages, they have poor eating habits, limited food preparation skills, lack nutritional knowledge, and have low levels of physical activity.

More of those suffering from developmental disabilities have been transitioned from institutions to community-based living. Therefore, food skill programs must be developed and implemented for this population.

Teaching nutrition and food skills to this population, will allow them to be more independent as adults and provide them with knowledge, skills, and self-confidence to make healthy meals.

Individual level

- At the individual level, attitudes, knowledge, and skills can influence health behaviors.
- It is important to address poor eating patterns, safety concerns, and low transferability of the skills.
- It is found that those in this population often do not consume the recommended amount of fruit and vegetables and their nutrient intake tends to be high in saturated fat and simple carbohydrates.
- Nutrition education and food skill programs must be developed.
- Building self-efficacy is also very important.

Interpersonal level

- Better training strategies for the support staff including general skill building and role playing, modeling, reinforcement, and corrective feedback being important.
- Setting goals.
- Social relationships are very important to establish.

Organization level

- Addressing factors in the private, public, and non-profit sectors to which the individual belongs.
- Staff training is very important.
- There is found to be limited awareness of safe food handling practices and nutrition knowledge of the staff in the homes for this population.
- Registered dietitians are strongly supported by literature.

Community and public policy levels

- Personal support services.
- Home-delivered meals.
- Home health care.
- Financial and legal assistance.

How does this apply to those with Developmental Disabilities?

- There has been little research done in this field specifically geared toward children.
- There is a disparity in health care between those suffering with developmental disability and the general population.
- People with developmental disabilities appear to be equally and in some cases more affected by obesity.
Why are individuals with DD more susceptible to obesity?
- Highly sedentary
- Low levels of physical fitness
- Poor eating habits
- Fewer opportunities for teaching healthy habits and supporting them
- Family environment
- Media and marketing environment

Prevention and Treatment of Obesity in children with DD
- The following need to be addressed in order to help this epidemic:
  - The cognitive, behavioral, and physical factors that are linked with ID
  - The role of parents and other family members in teaching and reinforcing healthy habits
  - The environment we live in that makes it difficult to make healthy choices which creates challenges to independent living for young adults with ID

ASD: Definition
- DSM-5 definition of Autism Spectrum Disorder (ASD)- abnormalities in social communication and interaction, as well as the occurrence of repetitive, restricted patterns of behavior or activities.
- ASD represents a single continuum of impairments with a varying degree of severity.

ASD: Hypothesis
- Hypotheses:
  - genetic origin
  - mitochondrial dysfunction
  - parental age
  - environmental agents (malleable, sometimes preventable):
    - exposure to environmental toxins in a fetal life and during the immediate neonatal period,
    - nutritional deficiencies. Many of the ASD children are picky eaters, they exhibit sensitivities to foods, or have selective eating behaviors. Children with autism had more limited food repertoires, and this led to an inadequate intake of nutrients.
  - MIA (Maternal Immune Activation theory) by infections during pregnancy,
  - Hormonal: fetal testosterone levels
- Interaction between genetic and environmental factors with oxidative stress as a potential mechanism linking the two
- Understanding these factors:
  - can address ASD prevalence
  - improve treatment opportunities

ASD: Nutrients on Mental DO
- Evidence demonstrates an interface between nutrients and:
  - schizophrenia spectrum,
  - depressive/anxiety disorders,
  - eating disorders,
  - neurocognitive disorders,
  - neurodevelopmental disorders such as ASD and ADHD
The first strong evidence for the impact of nutrition on the occurrence and development of diseases was described at the turn of the 18th/19th centuries. It represents selected milestones in the field of knowledge connected with nutritional strategies and their influence on mental disorders.

ASD: History of impact of Nutrition

- The diet of autistic children is not balanced.
- Nutritional deficits of folate, vitamin C, vitamin B1, B2, B6, and B12 and vitamin A.
- Low in many minerals, essential fatty acids, and amino acids.
- Those deficiencies result in metabolic, digestive, immune, and neurological problems (vision, speech, attention, and socialization weaknesses).
- Nutritional supplementation leads to behavioral and cognitive improvement.
- Children with ASD often suffer from impaired methylation, decreased glutathione, and oxidative stress.
- Supplementation with vitamin methyl-B12, folinic acid, and trimethylglycine is beneficial.
- Supplementation with Magnesium and vitamin B6 reduce symptoms of hyperexcitability, improve speech/communication, social interaction, and stereotypic behavior.

ASD: NUTRITIONAL INTERVENTIONS

- Can significantly help some ASD patients.
- Probiotics, digestive enzymes, vitamins, minerals, amino acids, specialty supplements.

ASD: AMINO ACIDS

- The levels of 14 organic acid compounds in ASD children are elevated.
- High Homocysteine (Hcy) level contributes to neuronal damage, associated with aggression/irritability.
- Supplementation of vitamin B6, B12, and folate lowers the levels of Hcy in blood.
- Glutamate (associated with aggression/irritability) is increased, and Glutamine is decreased in ASD children.
- Other amino acids which are increased in ASD children are:
  - Taurine
  - Isoleucine
  - Aspartic acid.

ASD: VITAMINS

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**ASD: GLUTEN AND CASEIN**
- Some ASD children suffer from deficiency of digestive enzymes transforming gluten and casein to amino acids.
- Increased gut permeability enables leaking gluten and casein into the blood stream, and passing the brain-blood barrier.
- In the brain they directly regulate signal transduction, causing disruption to the operation of the nervous system.
- In some patients elimination of gluten/casein from the diet results in disappearance of the symptoms of ASD.

**ASD: KETOGENIC DIET**
- The ketogenic diet is a high-fat, adequate-protein, low-carbohydrate diet.
- It forces the body to burn fats rather than carbohydrates.
- Normally, the carbohydrates are converted into glucose, which fuel brain functions.
- When carbohydrates are deficient, the liver converts fat into fatty acids and ketone bodies leading to ketosis.
- The ketone bodies pass into the brain and replace glucose as an energy source.

**ASD: Ketogenic Diet**
- KD contains a 4:1 ratio of Fat to Protein and Carbohydrates by excluding high-carbohydrate foods, while increasing high-fat food (nuts, cream and butter).
- Metabolic benefits of the KD are not solely due to increased fat.
- High-fat, sufficient-carbohydrate (i.e. non-ketogenic) diet worsens ASD core behaviors.
- Metabolic changes in blood within 2 days, behavioral effects at least 1 week.

**ASD: KD- Key Mechanisms**
1. **improve mitochondrial function**
   - In the gestational valproic acid model, KD treatment normalized dysfunctions in mitochondrial respiration
2. **reduce inflammation**
   - Low density of carbohydrates and limited protein forces nervous tissue to rely on ketone bodies for energy. Ketone bodies reduce inflammation. KD has neuroprotective and disease-modifying effects.
   - KD reduces core ASD symptoms subsequent to MIA (Maternal Immune Activation). MIA-acute inflammatory event during the first 2 trimesters - a physiological response to infection that increases the risk of persistent autistic behaviors in the offspring.
   - MIA-induced inflammation triggers an increase of proinflammatory factors and activates immune cells bathing the fetus in proinflammatory compounds and antibodies and increasing the likelihood of the child developing ASD.
3. **increases adenosine**
   - KD increases brain adenosine levels and signaling; it shows an inverse relationship between adenosine and symptoms of ASD.
   - Abnormalities in purine metabolism are common, and a purinergic treatment is effective in alleviating symptoms.

**ASD: KETOGENIC DIET**
- Improve:
  - core symptoms of ASD
  - symptoms of comorbidity of epileptic seizures
  - sociability
  - reduces self-directed repetitive behavior
  - normalizes play behavior in the gestational valproic acid model
Definition of ADHD

Attention deficit-hyperactivity disorder (ADHD) is a neurobehavioral disorder exhibited by difficulty in maintaining attention, as well as hyperactivity and impulsive behavior.

ADHD: CAUSES

- Combination of environmental and genetic factors.
- Neurobiology (some parts of the brain are smaller in children with ADHD).
- Genetics (ADHD tends to run in families).
- Environmental factors: 20-30%
  - can influence the severity of the disorder
  - modulate gene activation and deactivation (by epigenetic effect)
  - low socioeconomic class,
  - foster placement,
  - family dysfunction.
- Pregnancy-related factors:
  - low birth weight,
  - delivery complications,
  - prematurity,
  - dysmaturity,
  - prenatal alcohol and smoking exposure.

ADHD: CAUSES (cont)

- A study conducted by researchers at South Hampton University in the UK and published in The Lancet in 2007 found a definitive link between children’s ingestion of many commonly used artificial food colors, the preservative sodium benzoate and hyperactivity.
- The British Government took actions to “Food manufacturers are being encouraged to voluntarily phase out the use of most artificial food colors by the end of 2009”.
- However, in 2009 the EFSA in the UK re-evaluated the data and determined that “the available scientific evidence does not substantiate a link between the color additives and behavioral effects” for any of the dyes.
- The U.S. FDA did not make changes following the publication of the Southampton study, but following a citizen petition in 2008, the FDA reviewed the available evidence, and still made no changes.

ADHD: Artificial Food Colors (AFC)

- Food coloring, or color additive, is any dye, coloring matter, or other substance that imparts color when it is added to food or drink.
- Color additives are used in food for many reasons including:
  - To make food more attractive, appealing, appetizing, and informative.
  - Allow consumers to identify products on sight, like candy flavors or medicine dosages.
- 1882: German food regulations stipulated the exclusion of dangerous minerals such as arsenic, copper, chromium, lead, and zinc, which were frequently used as ingredients or colorants.
- 1906: In the United States, the Pure Food and Drug Act reduced the permitted list of synthetic colors from 700 down to seven.
- The seven dyes initially approved were:
  1. Ponceau 3R (FD&C Red No. 1),
  2. Amaranth (FD&C Red No. 2),
  3. Erythrosine (FD&C Red No. 3),
  4. Indigo Base (FD&C Blue No. 2),
  5. Light Green 8G (FD&C Green No. 2),
  6. Orange 1 (FD&C Orange No. 1),
  7. D&C Orange (FD&C Dye No. 3).
Four main categories of plant pigments to color food:
1. Carotenoids (E160, E161, E164)
2. Chlorophyllin (E140, E141)
3. Anthocyanins (E163)
4. Betanin (E162)

Other colorants or specialized derivatives of these core groups include:
- Annatto (E160b), a reddish-orange dye made from the seed of the achiote
- Caramel coloring (E150a-d), made from caramelized sugar
- Carmine (E120), a red dye derived from the cochineal insect, Dactylopius coccus
- Elderberry juice (E163)
- Lycopene (E160d)
- Paprika (E160c)
- Turmeric (E100)

Currently permitted by FDA in food:
- No. 1 – Brilliant Blue FCF, E133 (blue shade)
- No. 2 – Indigotin, E132 (indigo shade)
- No. 3 – Fast Green FCF, E143 (turquoise shade)
- No. 4 – Erythrosine, E127 (pink shade, commonly used in glacé cherries)
- No. 5 – Allura Red AC, E129 (red shade)
- No. 6 – Tartrazine, E102 (yellow shade)
- No. 7 – Sunset Yellow FCF, E110 (orange shade)

Meta-analytic review of 6 types of non-pharmacologic interventions:
- dietary (“Few Food Diet”, elimination of artificial food colors, and FFAs supplementation)
- psychological (cognitive training, neurofeedback, and behavioral interventions)
- Statistically significant effect for supplementation with Omega3/Omega6 FFA and elimination of artificial food colorings (for food sensitive patients).

Researchers from the Netherlands put 50 children with ADHD on a “restricted elimination diet” which was tailored to the preferences of each child.

Restricted Elimination Diet consists of foods with the least possible risk of allergic reaction – a combination of rice, meat, vegetables, pears and water.

A second group of 50 children’s parents were given advice on healthy eating and asked to keep a diary of everything their child ate.

The behavior of 78% of the 41 children who completed the five-week restricted diet phase improved, while the behavior of those who were not on a special diet remained the same.

The researchers concluded that dietary intervention should be considered in all children with ADHD if their parents are willing to follow a diagnostic restricted elimination diet for a 5-week period.

Systematic review of 52 studies: 20 with avoiding food elements and 32 with increase of food elements

Elimination diets and supplementation with fish oil are the most promising dietary interventions in reducing ADHD symptoms.

ADHD patients with subclinical Zinc deficiency may benefit from supplementation.
Multimodal treatments work best and involve a combination of biological and non-biological including nutritional approaches.

Large variety of foods and food components can provoke or exacerbate behavioral responses, though not every child responds to the same products in a similar manner.

Nutritional approaches are efficacious, safe and low-cost therapy that works by modulating immune system activity, and improving comorbid conditions.

Food can engage similar brain reward pathways as the drugs of abuse

Can result from casual eating or compulsive eating that leads to eventual addiction

In human and rodent studies, dysregulated brain reward pathways may contribute to increased intake of palatable foods leading to obesity

Overall increase in tasty and energy-rich foods that is independent of stress-induced hyperphagia or hypophagia

Food cravings are also present

Food is a natural reward

Food consumption leads to dopamine production

This activates the reward and pleasure centers of the brain

Which in turn leads to repetition of eating a particular food in order to experience this positive feeling of gratification

This repetitive behavior of food intake activates the reward pathways of the brain that override the signals of satiety and hunger

Overeating and obesity are the result of this

Mood: characterized by physiological arousal in the absence of a stimulus

Emotions: short-term affective responses to a reinforcing stimuli

Anger and joy are shown to have the strongest influence on appetite and food choice

Stress can effect feeding behavior

In some people, there will be increased intake and in others intake will be decreased

- This depends on the type of external or physiological stressors

Chronic stress can lead to increased consumption of palatable and rewarding food that leads to obesity OR a diminished appetite which leads to weight loss
Anxiety and depression often lead to increased consumption of food leading to overeating and obesity. People suffering from depression, usually show preference to “comfort foods” that help alleviate their negative feelings. However long term, can lead to consumption of calorically rich foods ultimately leading to obesity which in turn promotes vulnerability to depression and anxiety.

Findings show that prolonged high-fat foods leads to negative emotional states, increased stress sensitivity, and altered basal corticosterone levels. Altered serum cortisol level, HPA axis, and food intake have been associated with depression. Glucocorticoids regulate reward and emotional processes through their receptors in the midbrain and limbic circuits.

Vitamins
- Vitamin B12
- Folic acid
- Vitamin D
- Thiamine
- Niacin
- Calcium

Vitamin B12
- Cobalamin
- Water soluble vitamin
- Key role in the brain and nervous system
- Affects DNA synthesis, fatty acid/amino acid metabolism, formation of red blood cells
- Essential nutrient that cannot be produced in the body
- Found in: meat, fish, dairy products

Vitamin B12 Deficiency
- Can cause severe and irreversible damage to the brain and nervous system
- Symptoms include: Fatigue, lethargy, depression, poor memory, headaches, cognitive impairment, weakness, peripheral neuropathy
- This damage can be more severe in elderly due to less ability to absorb
  - Can even lead to symptoms of mania and psychosis
- Other clinical manifestations: hyperpigmentation, vitiligo, jaundice, anemia, thrombocytopenia

Psychiatric symptoms of Vitamin B12 deficiency
- Psychosis
- Depression
- Mania
- Cognitive impairment
- Delirium
**Folic acid**
- One of the B vitamins
- Essential to the body in order to make DNA, RNA, and for amino acid metabolism
- Required for the synthesis of SAMe (S-Adenosyl Methionine) which is needed for the synthesis of key neurotransmitters required in mood regulation
- Not produced by humans
- Found in: Dark green leafy vegetables, fruits, beans, nuts, dairy products, avocados, liver, spinach
- Recommended daily intake: 400 micrograms
- Signs and symptoms of deficiency manifest after 4 months

**Folate deficiency**
- Very common in excessive alcohol use and pregnancy
- Pregnant women are recommended to increase their daily intake of folate due to the risk of neural tube defects
- Clinical manifestations: megaloblastic anemia, glossitis, nausea and vomiting, diarrhea, thrombocytopenia, angular stomatitis

**Psychiatric symptoms of Folate deficiency**
- Depression
- Cognitive decline
  - Impairment in attention, visual spatial memory, abstract reasoning

**Thiamine**
- Vitamin B1
- Essential nutrient
- Found in food and used as a dietary supplement
- Needed for the metabolism of carbohydrates
- Found in: whole grains, meat, and fish

**Thiamine Deficiency**
- Beriberi, Wernicke-Korsakoff syndrome, optic neuropathy
- Symptoms early in the disease:
  - Fatigue
  - Irritability
  - Poor memory
  - Sleep disturbances
  - Chest pain
  - Anorexia
  - Abdominal pain
  - Constipation

**Psychiatric symptoms of Thiamine deficiency**
- Peripheral neuropathy
- Pain
- Paresthesias
- Degeneration of the myelin
- Wernicke’s Encephalopathy:
  - Triad = Ophthalmoplegia, Ataxia, and Confusion
**Vitamin D**
- Fat soluble
- Responsible for maintaining normal blood levels of calcium and phosphorus
  - Helps the body absorb calcium to maintain strong bones
- Most important form in humans:
  - Vitamin D3 = Cholecalciferol
  - Vitamin D2 = Ergocalciferol
- Sources of Vitamin D: sunlight, fish, eggs, fortified milk
  - Even just 10 minutes of sun exposure a day helps!

**Risk factors associated with Vitamin D deficiency**
- Age > 65
- Insufficient sunlight
- Breastfeeding
- Dark skin
- Malabsorption diseases
- Obesity
- Use of certain medications that alter vitamin D metabolism (anticonvulsants and glucocorticoids)
- Hepatobiliary disease
- Renal disease

**Vitamin D Deficiency**
- Rickets
  - Childhood disease
  - Soft, weak, deformed long bones
  - Found in lower income countries
- Osteomalacia
  - Adult disease
  - Softening of the bones that leads to bending of the spine, bow legs, proximal muscle weakness, bone fragility
  - Increased risk of fractures

**How does Vitamin D effect the brain?**
- Region specific expression of Vitamin D receptors in the cingulate cortex, thalamus, cerebellum, amygdala, and hippocampus
- Vitamin D regulates the expression of tyrosine hydroxylase which is the rate limiting enzyme in the synthesis of dopamine, norepinephrine, and epinephrine
- Vitamin D has an important role in the CNS

**Psychiatric symptoms of Vitamin D deficiency**
- Depression
- Seasonal Affective Disorder
- Psychosis
  - Increased risk of psychotic symptoms and schizophrenia
- Cognitive dysfunction
  - Memory and orientation impacted

**Niacin**
- Vitamin B3 or Nicotinic Acid
- Essential nutrient
- Precursor of nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP)
  - NAD is important for catabolism of fat, carbohydrates, protein, and alcohol
  - NAD also helps with cell signaling and DNA repair
  - NADP is important for fatty acid and cholesterol synthesis
- Found in a variety of whole and processed foods
**Niacin Deficiency**
- Pellagra
  - 4 D’s = Dermatitis, Diarrhea, Dementia, and Death
- Psychiatric symptoms:
  - Anxiety
  - Depression
  - Irritability
  - Poor concentration
  - Fatigue
  - Restlessness

**Calcium**
- Essential element
- Important component of bone and teeth
- Calcium carbonate and Calcium citrate are the forms of dietary supplementation
- Found in: dairy products (milk and cheese), seaweeds, nuts and seeds (almonds, pistachios, hazelnut), soy beans, figs, quinoa, orange juice

**Hypocalcemia**
- Low level of plasma calcium concentration below 8.8 mg/dL
- Common causes: hypoparathyroidism and vitamin D deficiency
  - Other causes include kidney failure, pancreatitis, rhabdomyolysis
- Treatment: supplementation

**Signs and symptoms of Hypocalcemia**
- Confusion
- Seizures
- Petechiae
- Tetany
- Weakness
- Hyperactive reflexes
- Laryngospasms
- Cardiac arrhythmias → prolonged Q-T interval
  - EKG

**Psychiatric symptoms seen in Hypocalcemia**
- Confusion
- Behavioral changes
- Psychosis
- Depression
- Irritability

**Hypercalcemia**
- High levels of serum calcium measured to be greater than 10.4 mg/dL
- Commonly caused by hyperparathyroidism and as a result of excessive bone resorption
  - Medications such as Lithium and Hydrochlorothiazide can also increase calcium levels
- Treatment: IV fluids, furosemide, bisphosphonates and calcitonin, and treating the underlying cause
Signs and symptoms of Hypercalce mia

- Abdominal pain
- Bone pain
- Nausea/vomiting
- Kidney stones
- Anemia
- Confusion
- Weakness
- Pruritus
- Cardiac arrhythmia \(\rightarrow\) shortened Q-t interval
  - EKG

Psychiatric symptoms seen in Hypercalce mia

- Depression
- Anxiety
- Cognitive deficits
- Memory issues
- Insomnia

References