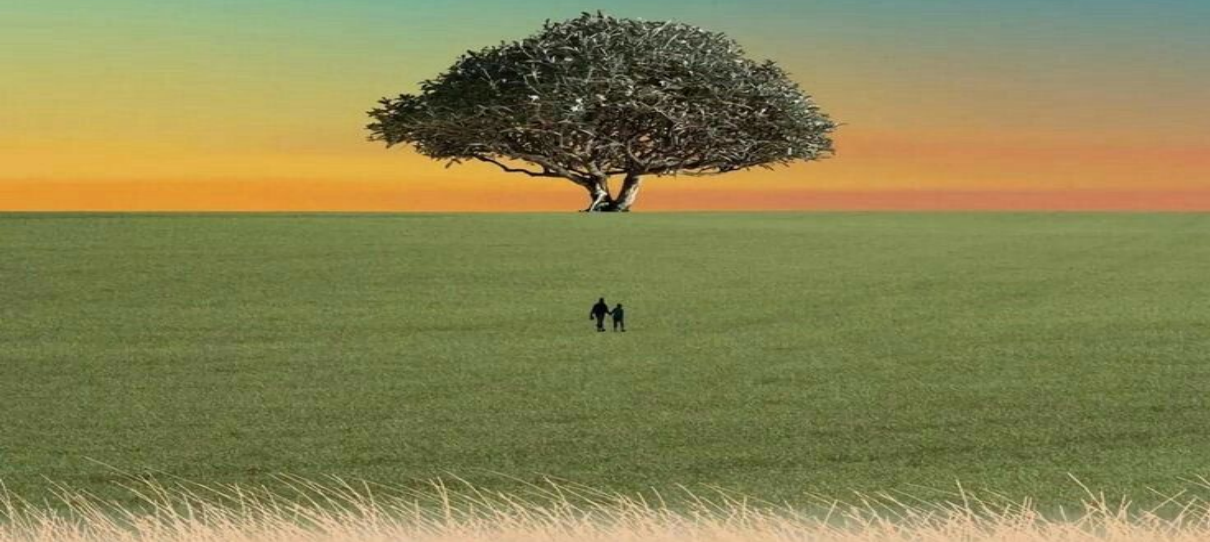
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"Bu kitapta yer alan bölümlerde kullanılan kaynakların, görüşlerin, bulguların, sonuçların, tablo, şekil, resim ve her türlü içeriğin sorumluluğu yazar veya yazarlarına ait olup ulusal ve uluslararası telif haklarına konu olabilecek mali ve hukuki sorumluluk da yazarlara aittir."

THE FcR RECEPTOR'S ROLE IN CALVES' PASSIVE IMMUNITY

Fatma ATLI[1]

Sezgin ŞENTÜRK[2]

1. INTRODUCTION

The immune system is vital to life and shields animals from microbial invasion. Although fetal calves can react to foreign invaders early in pregnancy, the placenta and the mother's immune system offer the fetal calf extra protection. According to Lemley et al., (2021), the ruminant placenta is characterized as cotyledonary morphologically and as syndesmochorial histologically. The cow's syndesmochorial placenta separates the fetal and maternal blood supply by forming a syncytium between the fetal trophectoderm and the maternal endometrium (Arthur, 1996). Immunoglobulin transmission in utero is prevented by this division of the mother's and fetus' blood supply (Gaspers, 2018). Calves that are born agammaglobulinemic—that is, with little to no immunoglobulins in circulation—are referred to as immuno-naïve because the transfer of immunoglobulins during pregnancy is prevented (Weaver et al., 2000). Despite being immuno-naïve while still in utero, the fetus's diverse innate and adaptive immune defenses develop as well (Barrington and Parish, 2001). The calf can react to a range of antigens by the time parturition occurs and

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it is born, but not as fully as when it is fully developed (Gaspers, 2018).

The first month of birth is the most crucial time for raising dairy heifers because of the increased risk of diseases and mortality (Svensson et al., 2006). Calves primarily experience gastrointestinal illnesses during this time due to infectious pathogens such as Cryptosporidium parvum, rotavirus, coronavirus, and Escherichia coli (McGuirk, 2008). According to Curtis et al. (1988), and van der Fels-Klerx et al. (2002), calves that survive diarrhea typically exhibit slower growth rates and are more prone to other illnesses, particularly bovine respiratory disease complex, which may have long-term detrimental effects on their performance during the first lactation. There are no established defenses against illnesses in newborn calves. Due to the large size of cow antibodies and the placental structure (syndesmochorial placenta), which prevents antibodies from being transferred from mother to fetus, calves are born with low levels of immunoglobulin and are not protected against disease agents until they are given enough colostrum. Passive immunity is the type of immunity that develops when a calf consumes colostrum (Fischer-Tlustos et al., 2021).

Passive immunity ends when maternal antibodies are destroyed, which takes 3-4 weeks (Lora et al., 2018).

One of the key elements influencing colostrum management is calf health and survival because calves are born almost without antibodies (Godden, 2008). Calf serum immunoglobulin concentrations, which indicate the success of colostral transfer, have been demonstrated to be an important indicator of preweaning morbidity and mortality. According to Perino et al., (1997), calves with low blood IgG concentrations (less than 8 mg/ml) 24 hours after delivery have a 3.2–9.5 times

higher risk of getting sick and a 5.4 times higher risk of dying before weaning.

Bovine rotaviruses (BRV) and bovine coronaviruses (BCV) cause diarrhea-characterized infections in newborns and usually subclinical infections in adult cattle (Vega and Parreño, 2014). Seroepidemiological studies (Burgu, 1995; Duman and Aycan, 2010) have shown that subclinical infection rates with these agents are quite high in adult cattle and therefore adult cattle have varying levels of immunity. Cattle carrying BRV and BCV-specific antibodies play a crucial part in shielding the infant from infection with the antibodies they transfer to their calves through colostrum and milk (Şentürk, 2018). The antibody titer transferred to calves born from naturally infected mothers varies according to elements like the time of infection of the mother, the level of antigenic stimulation, etc. It is also known that serum antibody levels are generally not very high in naturally infected adult cattle and therefore the antibodies transferred to the newborn calf through milk and colostrum are often insufficient to protect the newborn from infection (Neto et al., 2004).

The percentage of live-born calves raised to puberty effectively determines how profitable a dairy business can be. Despite significant progress in animal breeding, perinatal mortality rates are still high, especially on Holstein dairy farms, and vary from 2.4% to 9.7%. To reduce this gap, a number of rearing stage-related factors are being investigated (Gulliksen et al., 2009).

There are various techniques for the buildup of proteins and immune cells to infiltrate the cow mammary gland. Although the mammary epithelium itself does not synthesis immunoglobulins, the cow mammary gland actively controls the content of different immunoglobulins in colostrum, mostly IgG and to a lesser extent IgA (Stelwagen et al., 2009). According to

Stelwagen et al. (2009), the vast majority of immunoglobulins enter cells via a selective receptor-mediated pathway. Mayer et al. (2005) shown that the mammary epithelial cell (MEC) contains a particular IgG receptor called neonatal Fc receptor (FcRN), which is involved in the active transport of IgG into the lactating cow mammary gland. Intramammary plasma cells produce the IgA present in bovine colostrum (Gaspers, 2018). These immunoglobulins can be made in situ by intramammary plasma cells or obtained from blood. Chemokines facilitate the movement of these plasma cells through the circulation to the mammary gland (Wilson and Butcher, 2004). The receptor for polymeric immunoglobulin (pIgR), which is expressed in the mucosal epithelium, facilitates the passage of IgA across the MEC. IgA and a secretory component of pIgR are released into the alveolar lumen when pIgR is cleaved on the uppermost portion of the MEC (Apodaca et al., 1994).

This article summarizes the current understanding of the metabolism of FcR in passive immunity in newborn calves and highlights the components that require additional investigation to gain a deeper comprehension of how this system functions in these species and to benefit economically from it.

2. THE IMPORTANCE OF PASSIVE IMMUNITY IN NEWBORN CALVES

The transmission of passive immunity (TPI) through colostrum is one of these elements, which is crucial for the newborn calves' survival and well-being (Parreño et al., 2010). The immunoglobulin (Ig) concentration of the colostrum, the time of postpartum colostrum harvesting, the time between parturition and first ingestion, the volume given, the mode of administration, and bacterial contamination are some of the variables that can affect the effectiveness of TPI (Rodas et al., 2025). These

variables may also contribute to the failure of passive immunity transfer (FTPI) (Cordle et al., 1991).

When a newborn does not absorb enough immunoglobulins, FTPI happens (Şentürk, 2018). In addition to higher rates of morbidity and mortality, this syndrome has been linked to delayed puberty, lower weaning weights, and slower development rates (Guilloteau et al., 2009). IgG concentration is the main determinant of the quality of colostrum, which is a rich source of immunoglobulins and nutrients (Rodas et al., 2025). IgG concentration can be measured directly to evaluate this quality, or Brix values can be used indirectly by refractometry. High-quality colostrum has more than 50 mg/mL of IgG, or a reading above 21%; low-quality colostrum has fewer than 30 mg/mL of IgG, or a level below 20% (Yamanaka et al., 2003).

Newborns receive vital protection from the passive transfer of the mother's immunity. It is also known, though, that neonates are first exposed to an extensive array of antigens after birth (Silva et al., 2017). Neonates are therefore more likely to die following infection with common pathogens unless sufficient maternal immune support is given. Thus, maternal immunologic support can be a vital component of a newborn's survival (Parreño et al., 2010).

In order to protect the body from dangerous infections, the immune system has developed extremely intricate recognition, reaction, elimination, and memory pathways (Guilloteau et al., 2009). Heightened susceptibility to infectious illnesses, together with a rise in morbidity and mortality from them, might occur from a breakdown in these pathways or from their ineffective operation (Barrington & Parish, 2001). Additionally, the immune system works to guarantee tolerance to commensal bacteria, food, and additional environmental elements, in addition to "self." Inflammatory disorders may potentially arise from a malfunction

in the tolerogenic pathways. But tolerance is an ongoing process that starts at a very young age, even during pregnancy (Battersby & Gibbons, 2013).

Newborns are vitally shielded by maternal immunity's passive transmission. Newborn animals must distinguish between food antigens and pathogens in a hostile microbial environment, even if their immune systems are fully capable of developing primary immune responses against infection in mammals and birds (Bailey et al., 2005). This is important throughout this formative period of life in addition to throughout adulthood since the response patterns that are "learned" during this time remain relevant (van Keulen et al., 2021). The "antigenic immaturity" of the newborn immune system is caused by the antigenic inexperience and the predominance of suppression factors throughout fetal development. This happens under the "protective umbrella" of passive maternal antibodies and immune cells transferred from mother to child by various processes depending on the species because developing a primary immune response takes time and effort (Barrington & Parish, 2001).

Newborns are initially exposed to an extensive array of antigens after birth. Commensal microbiota acquired after birth aids in gut homeostasis establishment, immune system maturation, and modulation. The young animal experiences two crucial times when it is most exposed to novel antigens: right after birth and during weaning. In both situations, a new diet and the colonization of new bacterial strains and species can result in a significant shift in the antigenic makeup of the intestinal contents (Bailey et al., 2005). Several major immune responses may not be triggered quickly or powerfully enough to stop pathogen proliferation in response to certain antigens, which frequently result in mixed infections and consequences.

3. THE ROLE OF COLOSTRUM IMMUNOGLOBULINS IN IMMUNITY IN CALVES

Neonatal survival is significantly (although temporarily) aided by maternal immune support. Effective methods to ensure the passive transfer of mucosal and systemic immunity from the mother to her offsprings have been created through evolution. As previously stated, it should be understood that in order to prevent unwanted, excessive, and destructive inflammatory and allergic reactions, it is necessary to prevent the immunological response from being triggered against common environmental antigens that are harmless, such as food antigens and specific commensals (bacteria) (M‟Rabet et al., 2008). Consequently, the prevention of sickness and the development of healthy animals depend on the effective absorption of colostrum.

Colostrum contains a range of cells and immunomodulatory and antimicrobial components, such as antibodies (Chatthaa et al., 2013). The mammary gland uses an Fc-receptor-dependent process to concentrate maternal immunoglobulins from the mother's blood into colostrum. Colostrum contains lymphocytes from the common mucosal immune system that move to the gland of the breast (Salmon et al., 2009).

Fetuses of polygastric and monogastric ungulates (ruminants, horses, and pigs) belonging to the order Artiodactyla are born nearly agammaglobulinemic and acquire little to no maternal antibodies via the placenta (Nguyen et al., 2007). These newborns' intestines can transmit immunoglobulins and other macromolecules into the bloodstream through a non-specific route when they consume colostrum. After birth, this process lasts for a short duration, typically 24 to 36 hours. Immunoglobulins only work locally if the intestine undergoes a severe alteration

known as gut closure, which stops them from being absorbed from the intestines (Barrington & Parish, 2001).

Colostrum immunoglobulins are absorbed by pinocytosis, which transports them through the enterocytes. All kinds of immunoglobulins, along with other proteins and macromolecules, appear to be non-discriminately permeable to the enterocytes of newborn calves (Mayer et al., 2002)). Immunoglobulins enter the thoracic duct and the systemic circulation after passing through the glycocalyx on the enterocyte's apical membrane and into their exocytosis into the basolateral membrane the lacteals and intestinal capillaries. (Tizard, 2013). The fact that other protein macromolecule quantities and enzyme activities, including γ-glutamyltransferase (GGT), increase following colostrum intake has been used to support the non-selectivity of this process (Thompson and Pauli, 1981). But Goldstein et al. (1979) found clathrin molecules on the enterocytic microvilli in the calf's ileum and jejunum close to the IgG molecules, which could be proof of a receptor-mediated transport mechanism. The existence of coated pits in vesicles is implied by the presence of clathrin molecules. Additionally, Mayer et al. (2002) discovered that the apical side of enterocytes contains the FcRN transporter protein in neonatal ruminants. Similarly, IgG is transported into the lactating cow mammary gland by the particular IgG receptor FcRN in the MEC (Mayer et al., 2005). Whole leukocytes are shown to pass from the intestinal lumen through the enterocyte and into circulation by Reber et al. (2006). They showed that colostrum contains the cytokines Cd43 and Cd11, which are essential for leukocyte transendothelial migration and aid in the migration through the enterocyte and into the newborn circulation.

Immunoglobulins (Igs) in a particular immune response are released after vaccination or infection. Because they neutralize or eradicate the infection and its harmful byproducts,

they are protective. IgG, IgA, and IgM class antibodies are the most prevalent kinds present in the sera of immunological competent donors. Serum contains very little amounts of together, IgD and IgE make up less than 1% of serum immunoglobulin. The most prevalent isotype in blood and extravascular space, IgG is essential for mediating immunity, despite IgA being the primary antibody linked to the gut (Waldmann and Strober, 1969). The fact that IgG has a lengthy half-life in the bloodstream and that maternal immunity depends on IgG transfer from mother to infant or neonate emphasizes the significance of this isotype.

There are species-specific variations in how humoral immunity is acquired in animals through the transmission of IgG from mother to child. In rodents, maternal IgG is transferred from milk to the fetus postnatally through the neonatal small intestine, while in primates and rabbits, all maternal IgG is transferred through the placenta during fetal life (Ober et al., 2004). IgG and all other macromolecules are obtained from colostrum in ungulates twelve to eighteen hours after birth (Butler and Kehrli, 2005). Despite the fact that maternal immune transport varies in mammals, plasma cells must continuously secrete IgG and shield it from rapid removal in order to maintain a consistent serum IgG level. The longest-surviving of all plasma proteins, IgG was protected against quick breakdown by the existence of a comparable or identical receptor, was deduced by Brambell in 1958 after he described a hypothetical saturable receptor system involved in the maternal IgG transport (Brambell et al., 1958). It was eventually demonstrated that the Brambell receptor (FcRB) mediates both IgG's defense against catabolism and the transmission of IgG during antenatal and/or neonatal periods, where it is expressed as FcRn (neonatal Fc receptor) (Roopenian and Akilesh, 2007). The idea that FcRn also binds albumin and extends the half-lives of these two crucial serum proteins by preventing them from being broken down intracellularly by

endothelial cells is supported by recent results (Anderson et al., 2006).

IgG is the most common immunoglobulin isotype in the blood and extravascular space in animals. It is the primary immunoglobulin isotype that is passed from the mother to her offsprings and plays an important part in mediating immunity (Waldmann & Strober, 1969). In actuality, active IgG transfer from mother to baby or neonate is necessary for maternal passive immunity to a number of pathogenic pathogens (Baintner, 2007).

4. THE NEONATAL Fc RECEPTOR, OR FcRn

The receptor that transmits maternal immunoglobulins from mother to offspring through the neonatal intestine is called FcRn, and it was initially discovered in rodents (Rodewald, 1976). The intestinal brush border's FcRn molecules bind ingested IgGs from the mother's milk and move them through enterocytes to the newborn's systemic circulation (Rodewald and Kraehenbuhl, 1984). According to Simister and Mostov (1989), the functional a heterodimer is the expression of the molecule made up of two subunits: the beta 2-microglobulin (β2m) and an integral membrane α-chain that is similar to molecules of MHC class I.

Contact residues in the IgG Fc component's CH2 and CH3 domains, the α1 and α2 domains of FcRn, and a single contact site in β2m, are necessary for IgG to bind to FcRn (Vaughn et al., 1997). This pH-dependent mechanism exhibits poor or no binding when the pH is neutral (pH > 7.0). and high-affinity binding at pH ≤ 6.5, which is an acidic pH (Vaughn and Bjorkman, 1998). In certain internal vesicles (like early endosomes) and in certain situations on the cell surface (like duodenal enterocytes), this pH specificity guarantees precise binding.

The receptors for B cells (BCR), T cells (TCR), and Fc cells (FcR) are among the receptors that immune system cells use to recognize antigens. FcR recognizes the Fc component of antibodies rather than antigens. Despite this, antibody-FcR complexes act as membrane receptors for antigen without any preset specificity (Daëron, 1997). Antibodies do give antigen specificity to a range of cells, the majority of which lack antigen recognition structures, when they bind to FcR. Under the right circumstances, many FcR use antigen receptors and the same transduction pathways to initiate cell responses, and they share activation patterns with BCR and TCR. Additionally, FcR possesses biological characteristics that antigen receptors do not (Vaughn et al., 1997).

All antibody classes have FcR: FcγR binds IgG, FcαR binds IgA, FcϵR binds IgE, FcμR binds IgM, and FcδR binds IgD (Raghavan and Bjorkman, 1996). Using immunoglobulins and receptors altered by site-directed mutagenesis, relationships between the Fc and FcR component of antibodies have been thoroughly studied. Both membrane receptors and soluble molecules are produced by proteolysis of membrane receptors or alternative splicing of FcR transcripts. Soluble FcR can exhibit a variety of biological functions and maintain its affinity for immunoglobulins (Daëron, 1997).

The Fc region of immunoglobulins is bound by cell surface molecules called FcRs. Every member of the family may identify one possibly a few isotypes of the same immunoglobulin. There are two functional classes into which FcRs can be divided. When antibody–antigen complexes bind to a particular class of receptors on the exterior of effector cells, several biological reactions are triggered. Fcγ receptors for IgG (FcγRI, FcγRII, FcγRIII, and the bovine Fcγ2R), Fcε receptors for IgE (FcεRI, FcεRII), and Fcα receptors for IgA (FcαRI) are some examples of these. Immunoglobulins are transported across epithelial surfaces

by the other receptor classes, which include neonatal IgG transporter (FcRn) and poly IgA receptor (pIgR) (Kacskovics, 2004).

The majority of livestock species have antibody-dependent maternal immunity, which is primarily characterized in ruminants and solely mediated by colostral immunoglobulins. According to Kacskovics et al., (2000), the presence of bovine FcRn transcripts in several organs, including the mammary gland, indicates their role in IgG transcytosis. Actually, transcytosis within clathrin-coated vesicles is how FcRn mediates active transport within cells. According to Raghavan et al., (1995), the transport is bidirectional and pH-dependent, exhibiting high-affinity binding occurs at acidic pH (pH: 6.5) and weak or nonexistent binding occurs at neutral pH (pH: 7.0). This pH specificity guarantees appropriate release in bloodstream circulation (pH: 7.4) as well as specific binding in endosomes and on specific cell types' surfaces (duodenal enterocytes, placental tissue).

FcRn transports IgG from the cell surface to the recycling endosome, and exocytosis can happen via two different pathways, according to a recent study that described in full how FcRn recycles in real time from the endosome to the cell surface: i) whether the vesicle holding the FcRn fully fuses with the plasma membrane and transports the FcRn to the cell surface, as shown by traditional models of membrane receptor exocytosis. (Ober et al., 2004). ii) Kiss-and-run exocytosis, in which the secretory vesicle is structurally intact and only partially fuses with the plasma membrane in repeated cycles. Despite this, it is still possible for FcRn to leave the vesicle and combine with other cell-surface elements. (Baintner, 2007).

In ruminants, such as horses and pigs, the maternal immune transport is only mediated via colostral intake in the brief

postnatal period due to the ungulate placenta's complicated shape and lack of FcRn expression. Antibody transmission from the mother to the offspring in these species is also a dual process of immunoglobulins´ active transport: i) a receptor-mediated process that enriches the mother's lactogenic secretion with isotype-specific immunoglobulins, ii) a non-selective process that absorbs these immunoglobulins by the neonate, and iii) a receptor-mediated process that recycles IgG back into the intestine (Cervenak and Kacskovics, 2009).

5. WHAT EFFECT DOES FcR HAVE ON CELLS' REACTİON TO IMMUNE COMPLEXES?

Many studies have been conducted in recent years on how FcR causes cells to react to immune complexes and what influences the specificity of cellular responses. Research on FcR gradually expanded into cells, revealing links to additional transduction pathways (Ober et al., 2004). Experiments on in vitro reconstitution also produced important advancements. Last but not least, genetically altered mice with an inactivated FcR gene have enabled evaluation of the in vivo significance of in vitro experiment findings. These can be separated into two main categories from a functional perspective: FcR that can activate cells and FcR that cannot. Intracytoplasmic activation motifs, which resemble the signal transduction components of the BCR and TCR, are present in FcR that can activate cells (Baintner, 2007).

These motifs are now known as tyrosine-based activation motifs of immunoreceptors (ITAMs) and are made up of a twice-repeated YxxL sequence that surrounds seven variable residues. There are two kinds of FcR with ITAMs (Kacskovics, 2004). The majority of FcR are of the first type, which are multichain receptors made up of one or two intracytoplasmic signal

transduction subunits regions where ITAM are found, together with a ligand-binding FcRα subunit. The second kind of FcR consists of two human-specific, IgG receptors with single chains that are closely linked known as FcγRIIA and FcγRIIC. Between the two YxxL sequences, they have a single ITAM with 12 residues (rather than 7) (Daëron, 1997).

ITAM is absent from FcR that do not cause cell activation. They fall into two major types as well. The first class of FcR belongs to a group of IgG receptors with a single chain known as FcγRIIB. The intracytoplasmic domain of these receptors has a pattern that prevents cell activation by receptors that might cause cell activation (Nimmerjahn et al., 2005). One YxL sequence, known as the tyrosine-based inhibitory motif of immunoreceptors (ITIM), is present in this motif. The second class of FcR neither promotes nor prevents cell activation. They participate in IgG transcytosis via epithelia. They are the polymeric IgA and IgM receptor (pIgR) and the neonatal FcR for IgG (FcRn). FcγRIIIB, a human IgG receptor lacking ITAM, does not have the potential to trigger on its own but aids in cell signaling by joining forces with other FcR (Lee et al., 2007).

6. INTERACTION OF FcR WITH IMMUNOGLOBULINS

Immune defense is significantly influenced by immunoglobulin Fc domain receptors. Mammalian receptors fall into two distinct functional classes. Immunoglobulins are transported to their primary sites of action by one class of receptors through epithelial tissues (Monteiro and Van De Winkel, 2003). This class includes the neonatal Fc receptor (FcRn), which transports immunoglobulin G (IgG), and the polymeric immunoglobulin receptor (pIgR), which transports immunoglobulin A (IgA) and immunoglobulin M (IgM). When

antibody-antigen complexes attach to another type of receptors found on effector cell surfaces, other biological reactions are triggered. The most well-characterized of these are the immunoglobulin E (IgE) receptors (FcεR) and the IgG receptors (FcγR). The biological reactions that are triggered include phagocytosis, cell-mediated cytotoxicity that is dependent on antibodies, the release of inflammatory mediators, and the control of lymphocyte differentiation and proliferation (Ravetch and Bolland, 2001).

Two copies of the variable Fab region, which houses the antigen binding site, and the relatively constant Fc region, which interacts with effector molecules including Fc receptors (FcRs) and complement proteins, make up immunoglobulin (Ig) molecules (Lee et al., 2007). By (a) enabling the customized delivery of antibody molecules to the body's necessary locations, (b) creating the vital connection between effector responses and Igs' binding of antigens like inflammation and the control of antibody production, and (c) regulating the lifespan of Ig molecules in serum, the Fc domain of Ig molecules' receptors create vital components of immune defense (Monteiro and Van De Winkel, 2003).

7. FcRn'S FUNCTION IN RUMINANT COLOSTRUM IgG DEPOSITION

Livestock animals have a selective concentration of colostral IgG that is 10–40 times higher than plasma levels. Ruminants with specifically enriched IgG1 (colostrum contains 40–50 and roughly 3 mg/ml of IgG1 and IgG2, respectively, while their plasma concentration is nearly equal, i.e., about 9–11 mg/ml) and a significant decrease in maternal plasma IgG1 in the month before parturition have been the best candidates for this phenomenon (Chattha et al., 2013). Days after calving, this

transport stops parallel to the neonates' gut closure, resulting in milk IgG concentrations that are less than 1% of colostrum levels. It has previously been demonstrated that IgG1 preferentially binds to the mammary epithelial cells close to parturition (Barrington and Parish, 2001), and that these cells stain prepartum with anti-IgG1 serum. It has been hypothesized that FcRn could be involved in this process because to its selectivity and the fact that only it can transcytose IgG through epithelial cells (Lee et al., 2007).

According to Mayer et al. (2005), the FcRn is crucial for the transfer of IgG during colostrum synthesis in ruminants, as evidenced by the clear shift in distribution before and after parturition. The fact that distinct haplotypes of the bovine FcRn (bFcRn) genes—heavy chain and beta 2-microglobulin—were linked to the serum IgG level in newborn calves further supported this theory (Clawson et al., 2004). Whether FcRn mediates the transfer of IgG from plasma to milk or colostrum is still unknown, though. Recently, transgenic mice that overexpress bFcRn in their nursing mammary glands were developed in an effort to address this discrepancy. These animals' elevated serum and milk IgG levels indicate that the lactating mammary gland's over-expressed bFcRn contributes to IgG protection, extending the animal's longevity (Ravetch and Bolland, 2001). However, there was no discernible accumulation of either injected bovine IgG or endogenous mouse IgG in the transgenic animals' milk, indicating that over-expression of bFcRn causes IgG to remain in serum rather than accumulate in milk. In other words, bovine IgG1 binds weaker to the bFcRn and so gets easier to the colostrum, but IgG2 binds stronger to this receptor and is therefore recycled to circulation more efficiently. This finding validated the rodent model that was previously mentioned (Monteiro and Van De Winkel, 2003). It is noteworthy that ruminant IgG2's half-life is longer, which indicates a greater binding capacity to the FcRn.

However, we cannot rule out the possibility that the bovine FcRn does not function in mice in the same manner as it does in cows or that there is another, as-yet-undiscovered IgG1 receptor. particularly in a function that is known to be highly diverse between species (Cervenak and Kacskovics, 2009).

Among livestock animals, the majority of studies have focused on cattle this FcRn function to date. The half-life of exogenously injected human IgG was approximately 33 days in calves, which is roughly twice as long as its counterpart in cows. This implies that FcRn plays a role in maintaining cattle's IgG homeostasis. It was also demonstrated that bFcRn binds human IgG much stronger than it binds to bovine IgG (Kacskovics et al., 2004). In other transgenic models recently created, the function of the bFcRn in controlling the IgG half-life was also investigated (Clawson et al., 2004).

Consequently, more investigation is advised to clarify the ways in which FcR influences calves' immunological and metabolic responses. In order to determine the permanence of maternal effects over time, it is also advised that the components of the calf be examined later in life.

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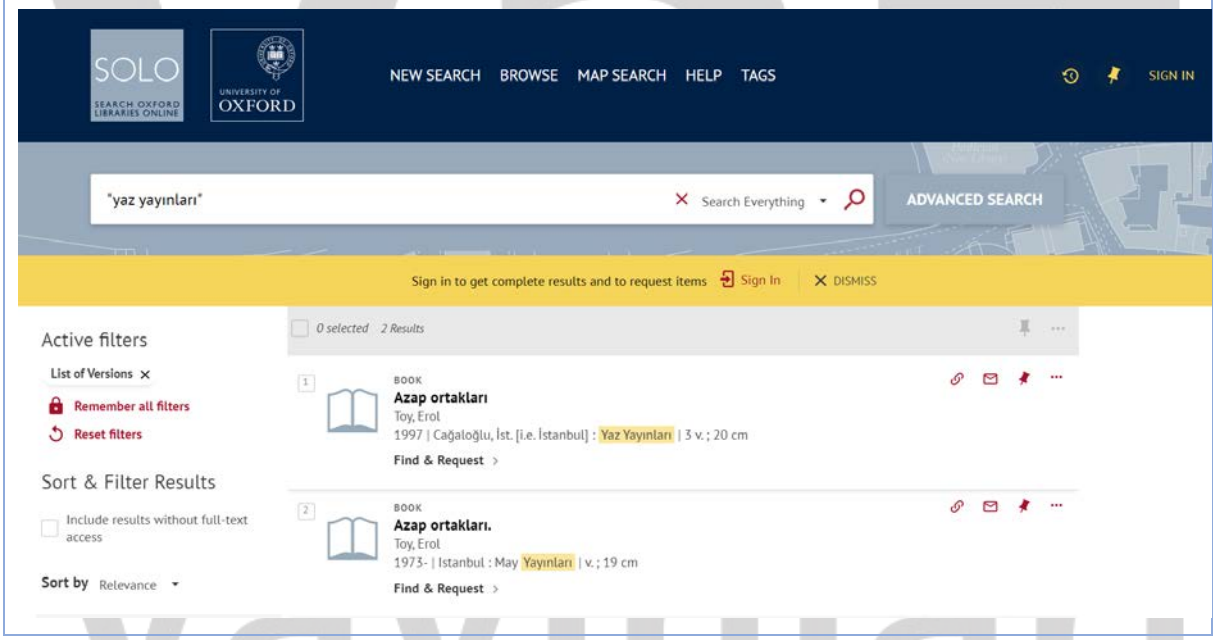
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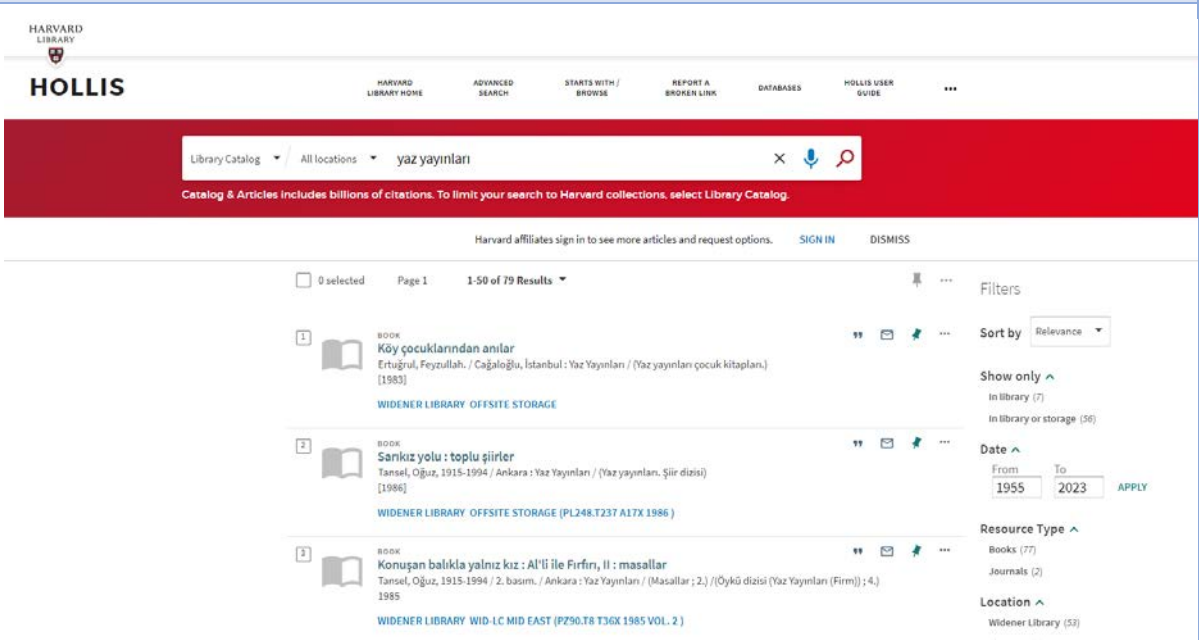
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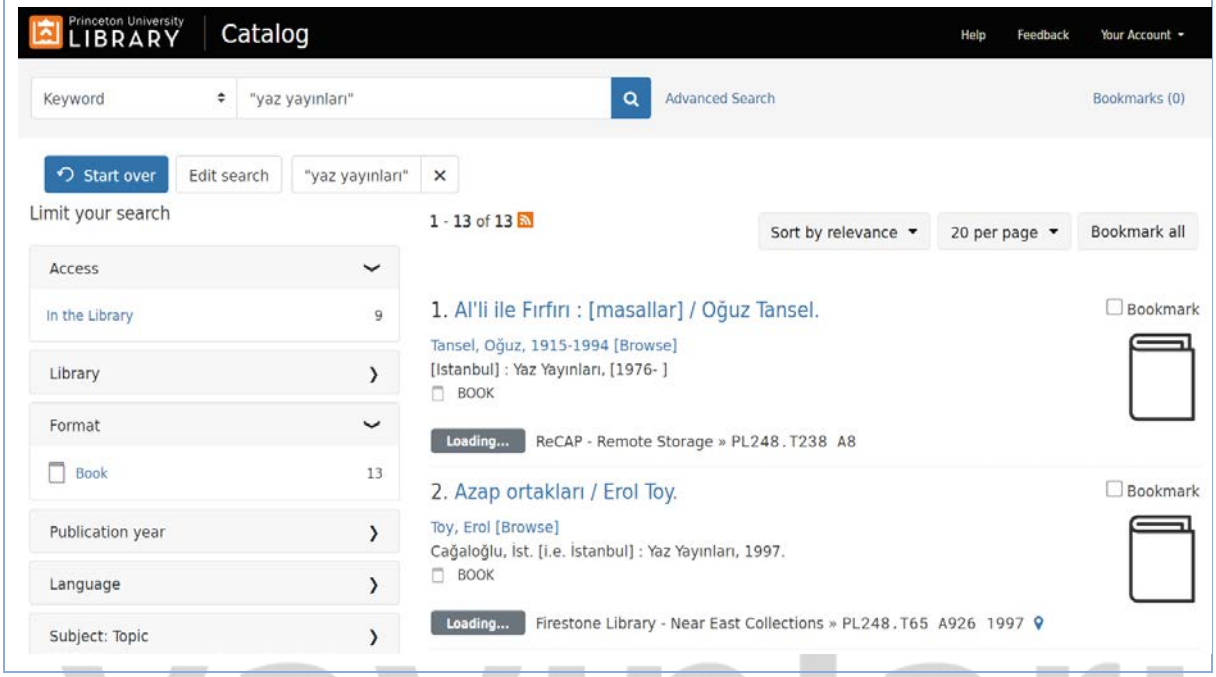
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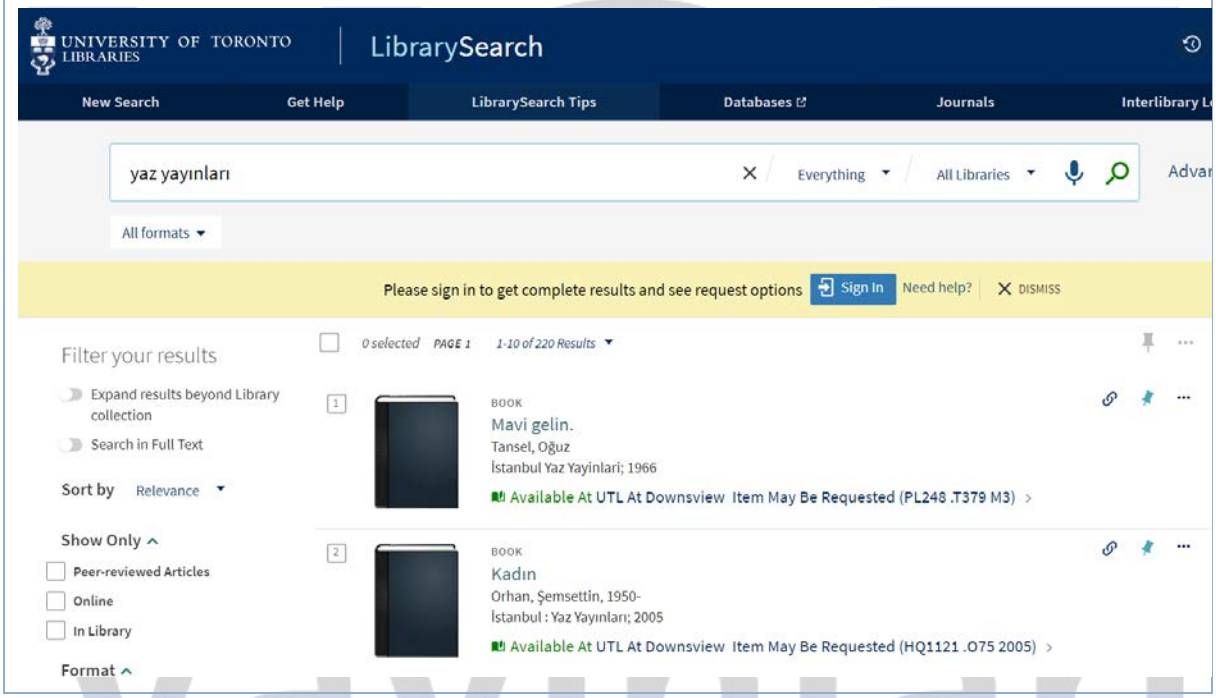
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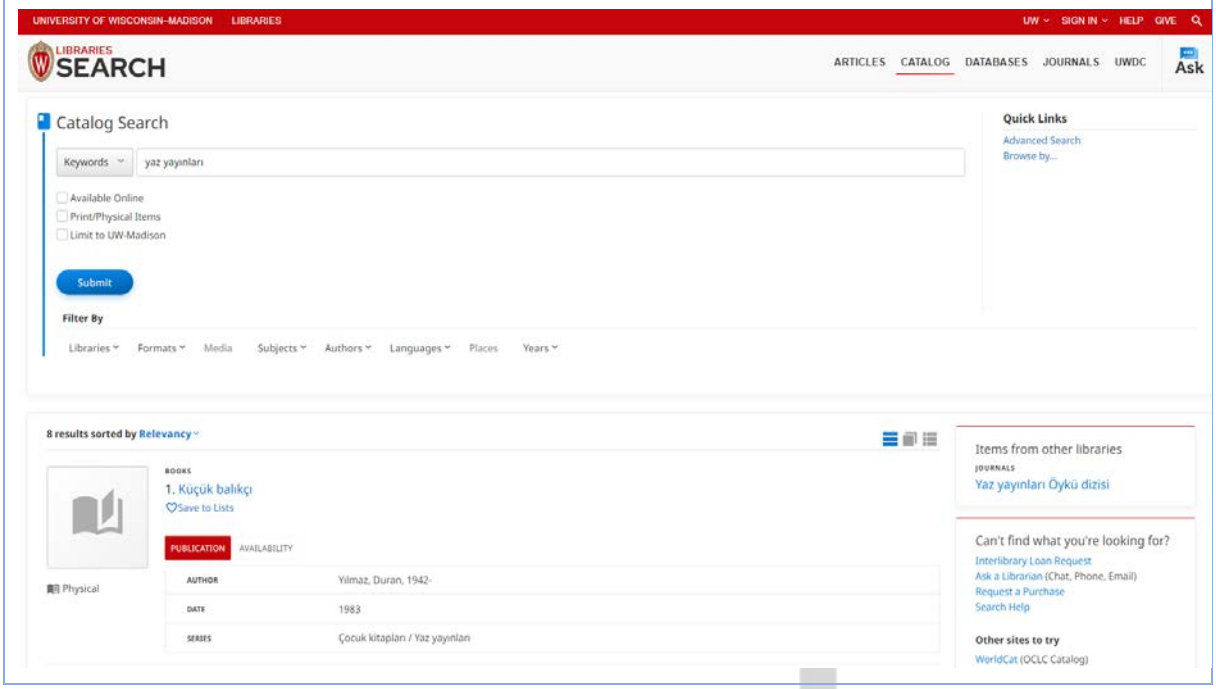
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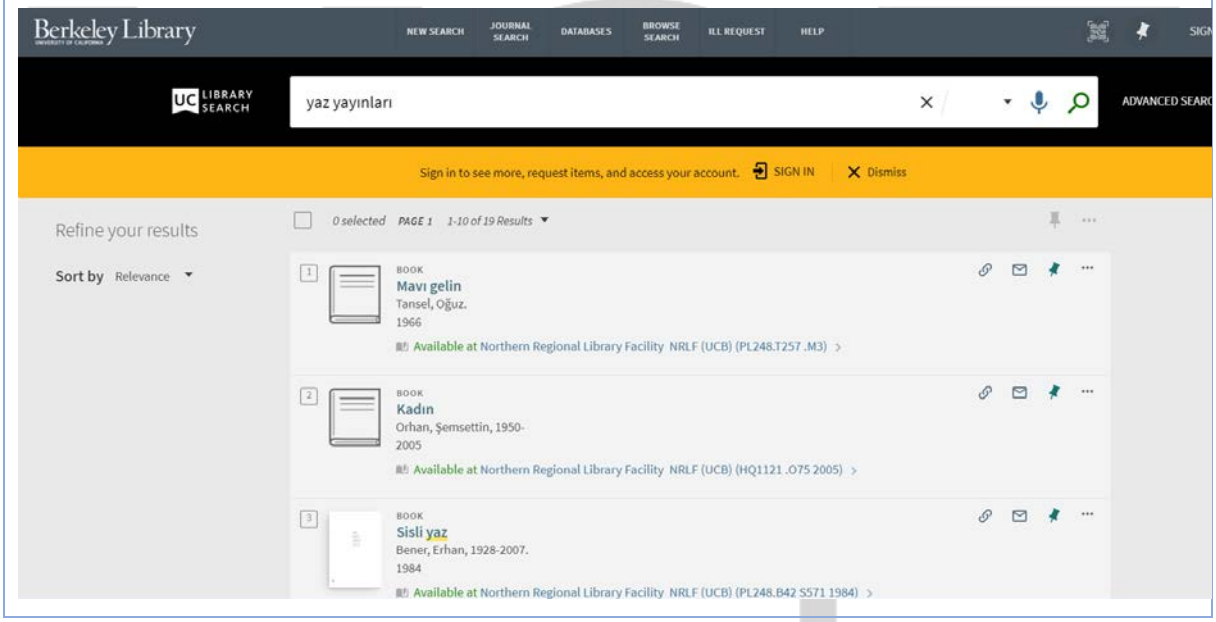
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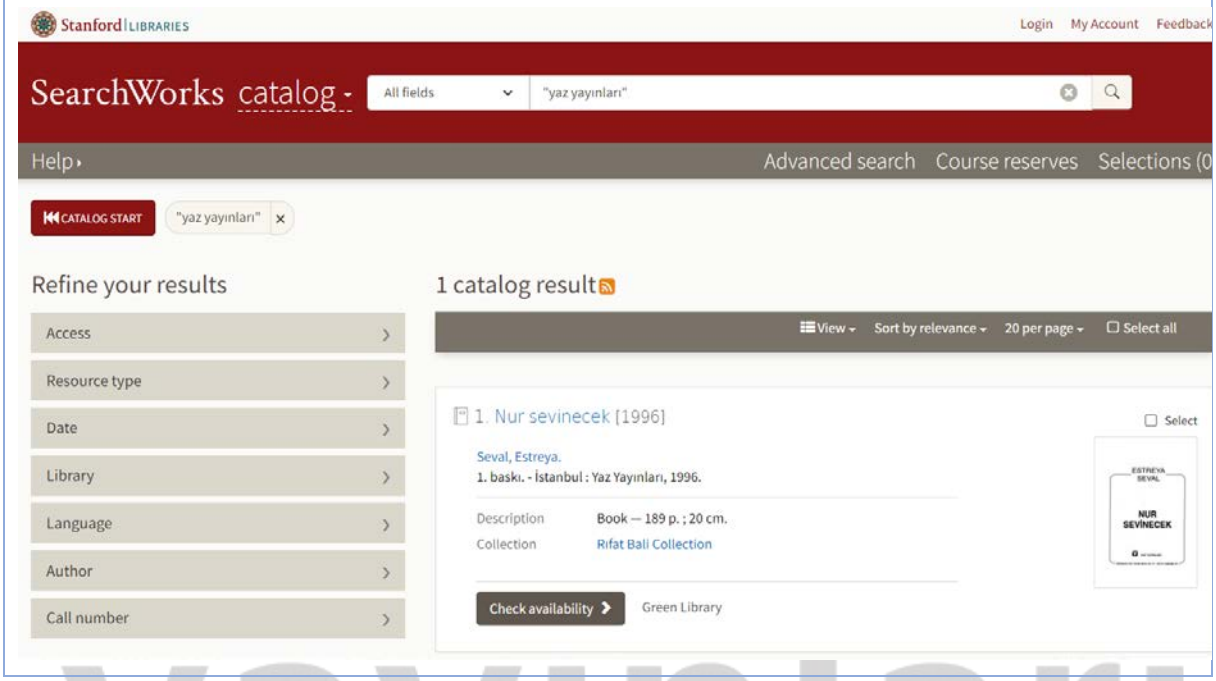
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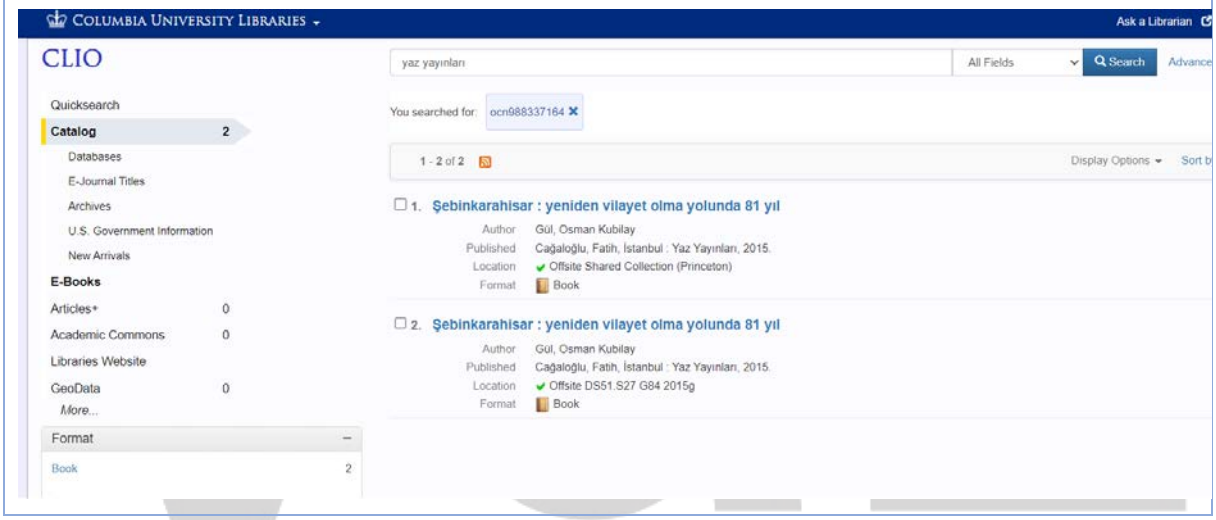
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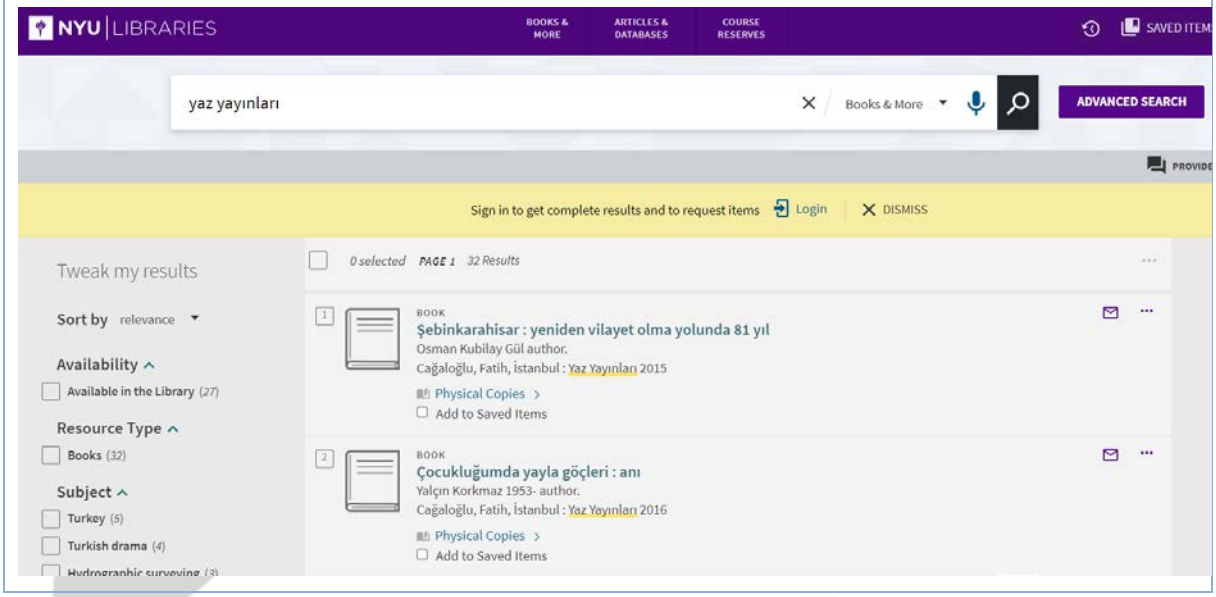
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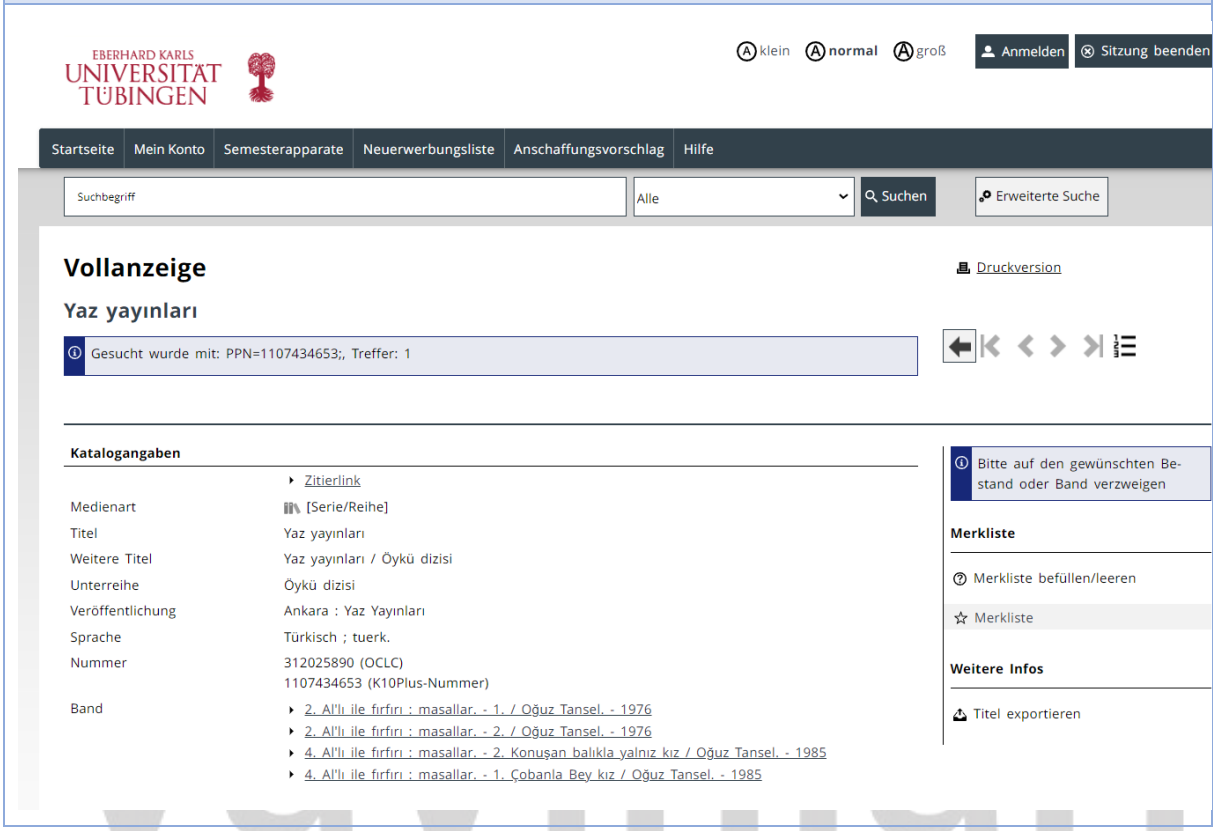
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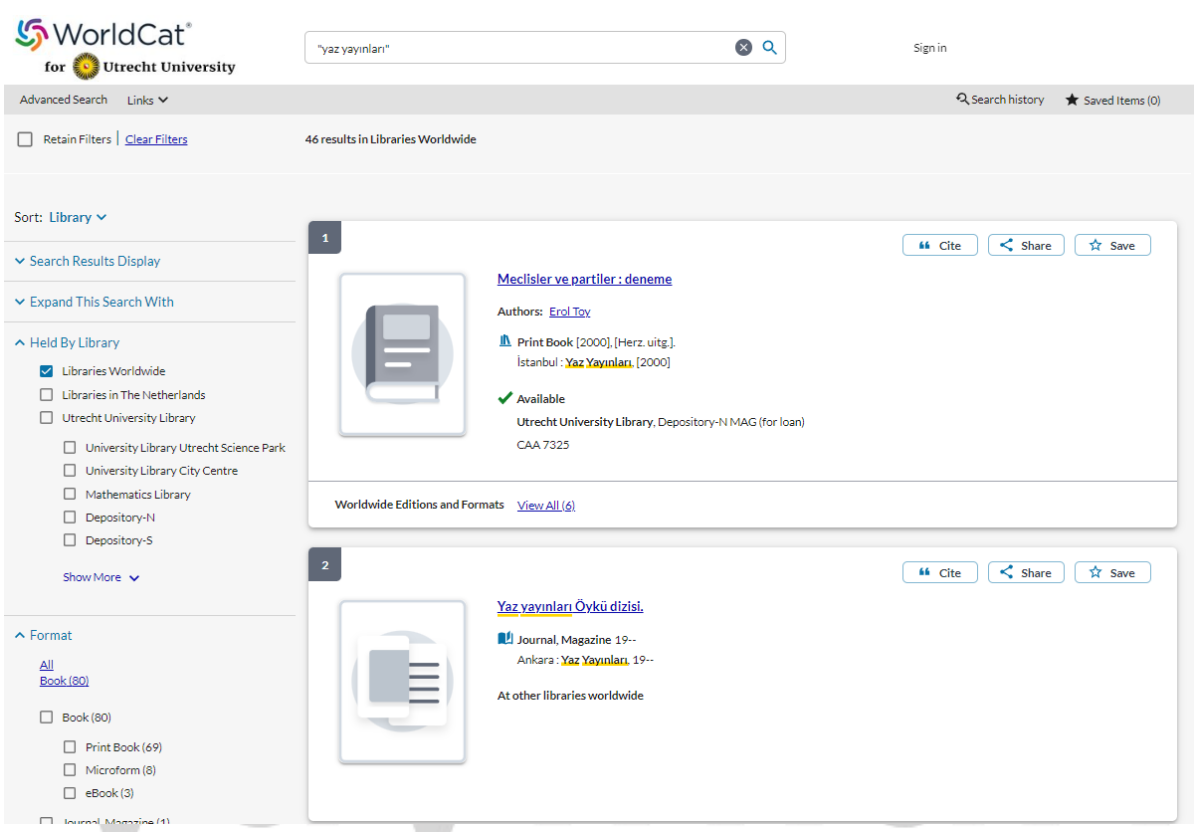
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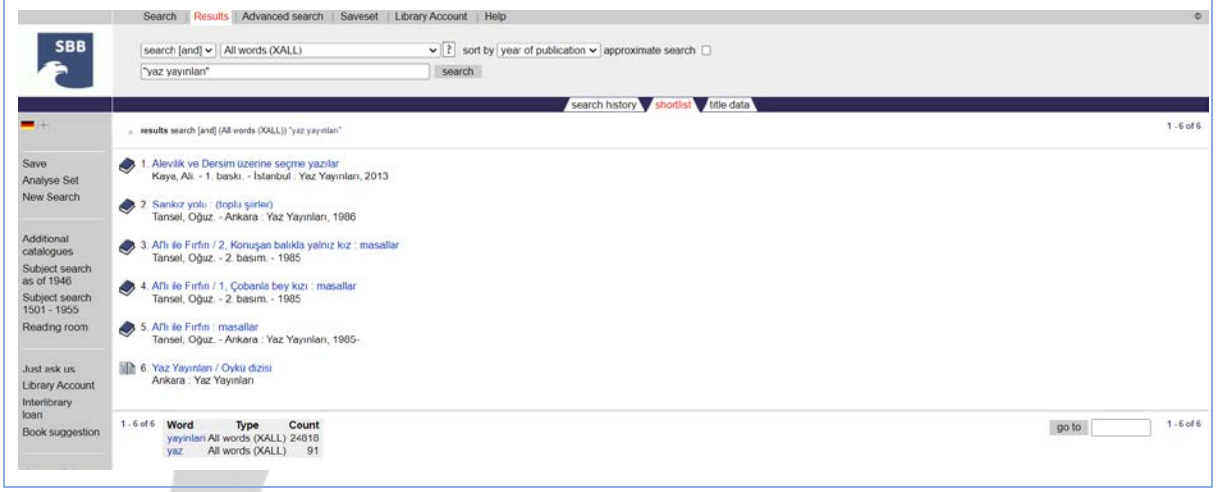
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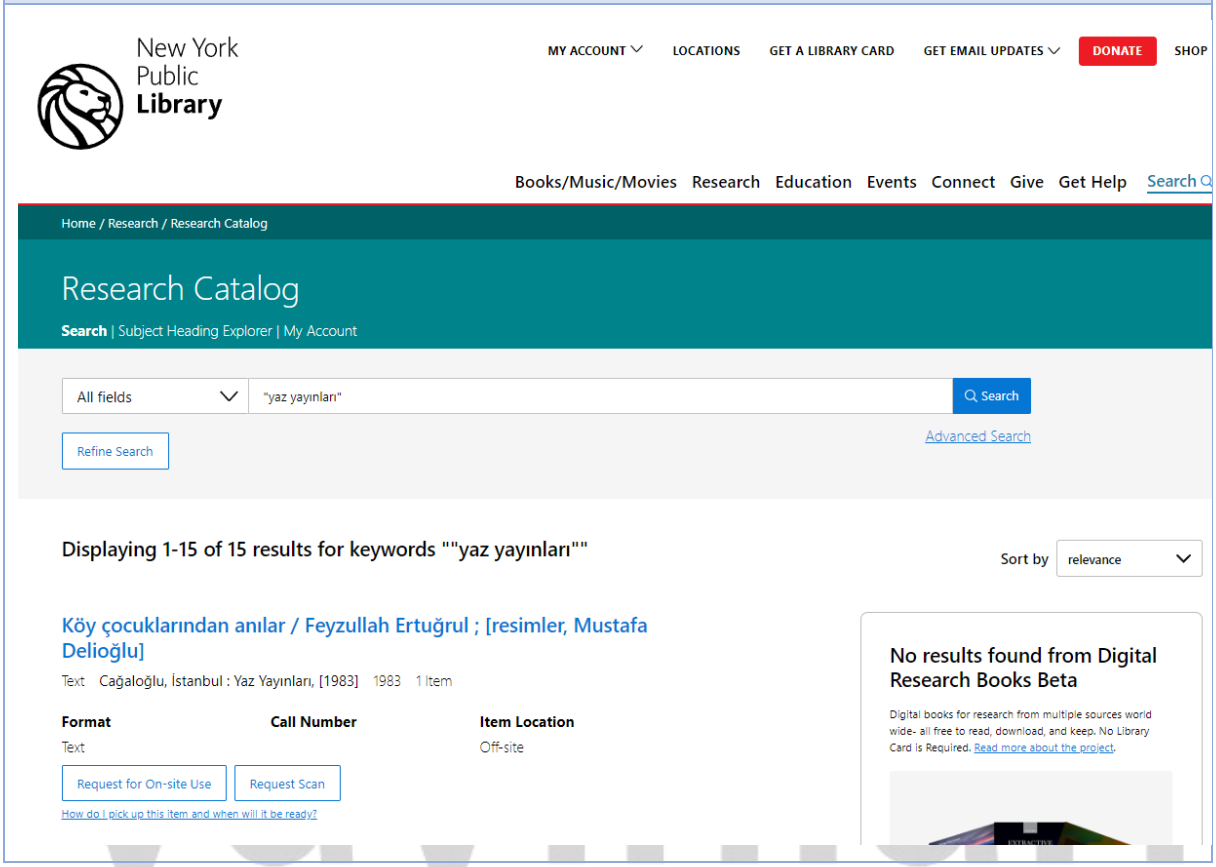
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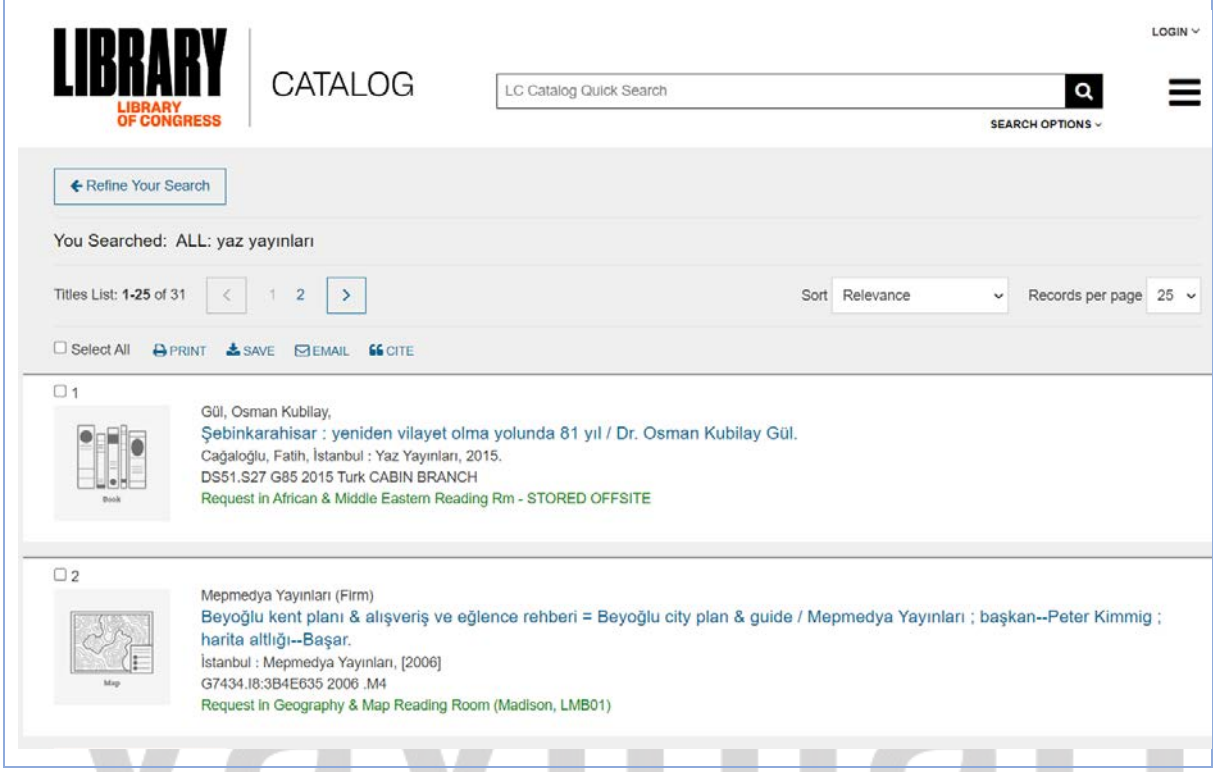
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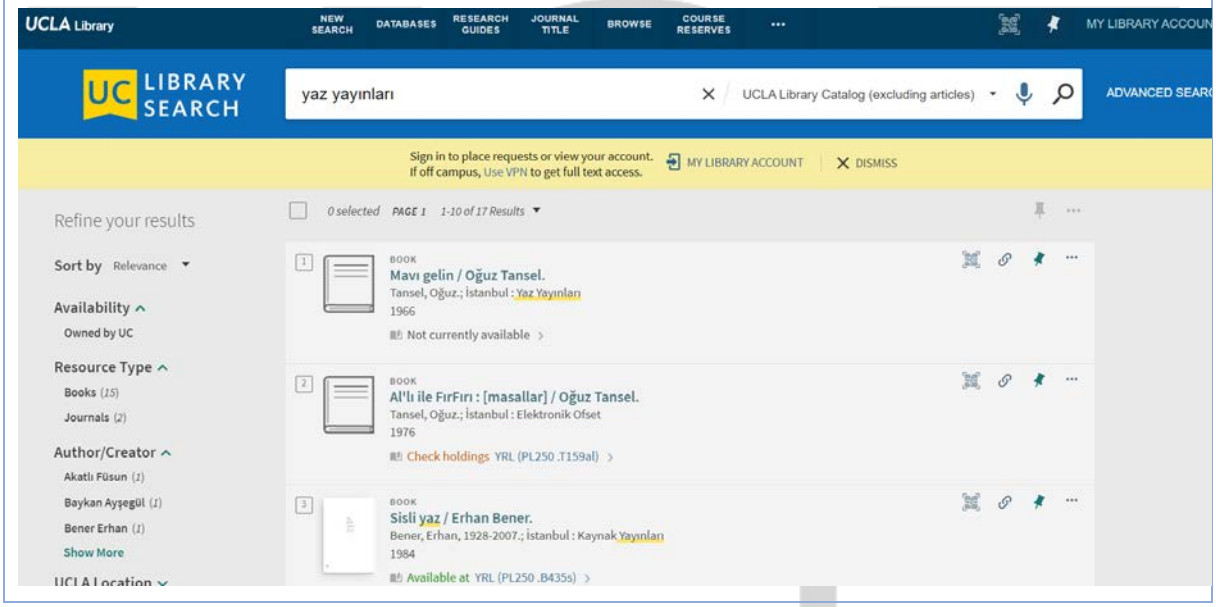
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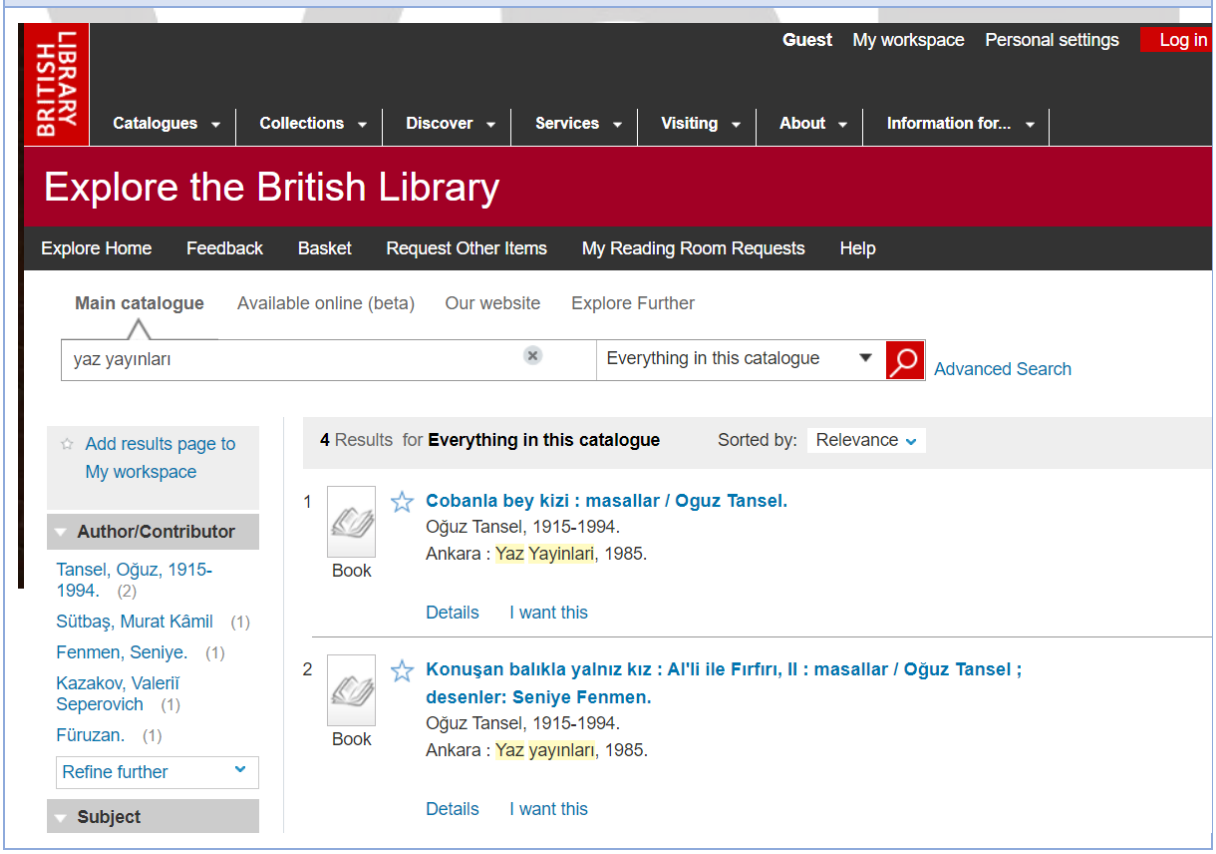
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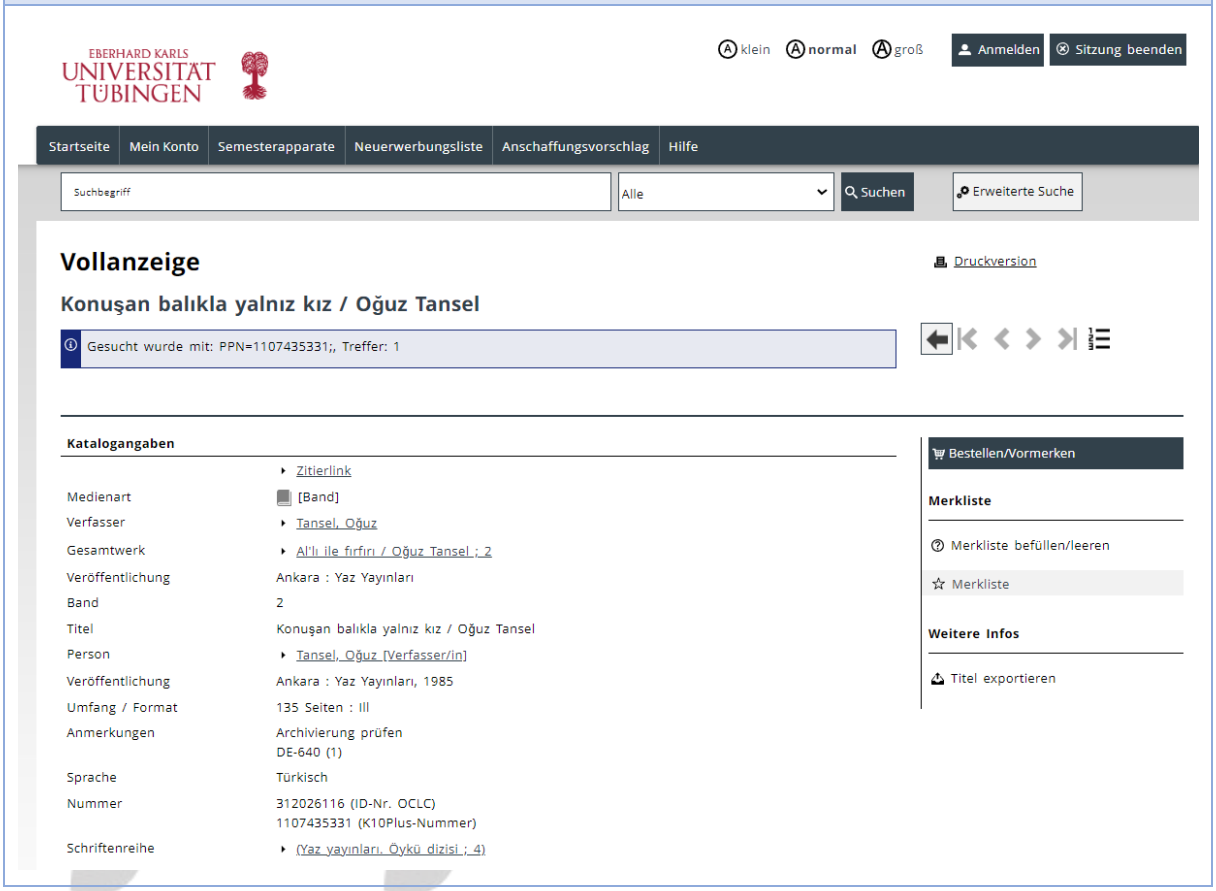
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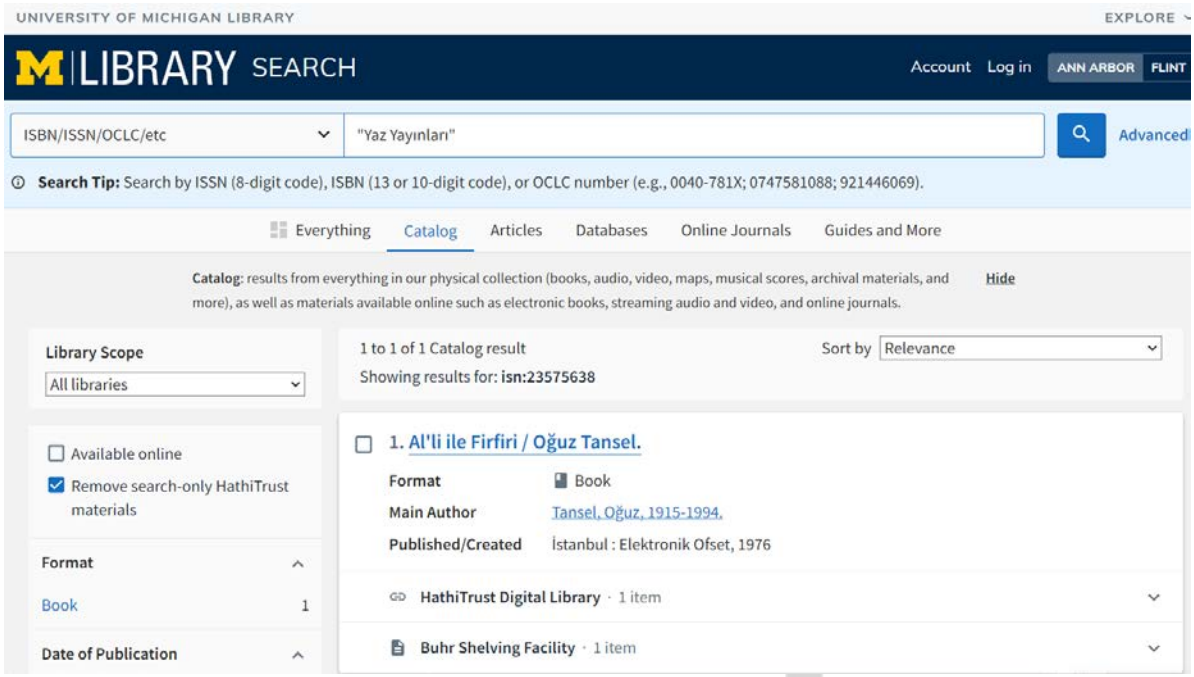
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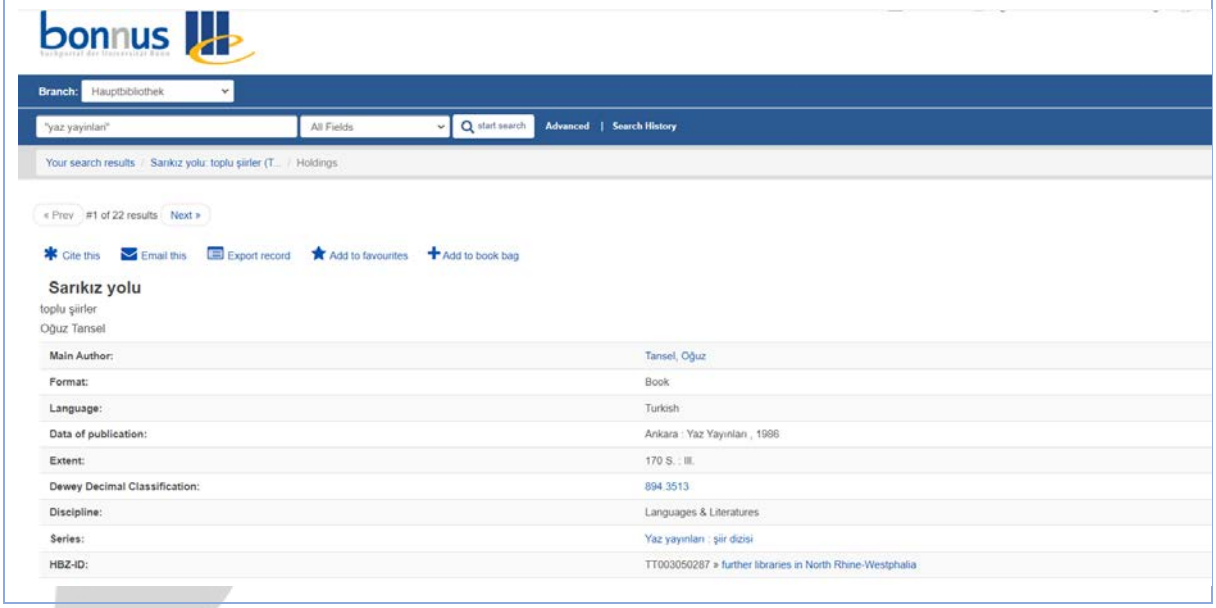
UNIVERSITY OF MICHIGAN

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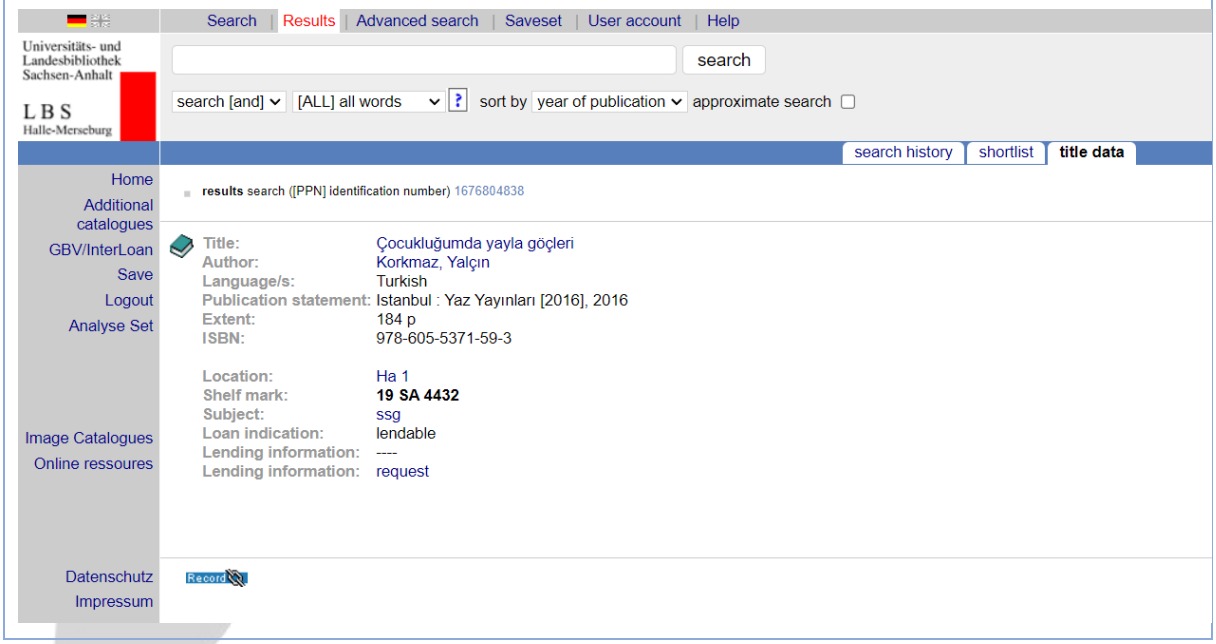
UNİVERSITÉ DE STRASBOURG

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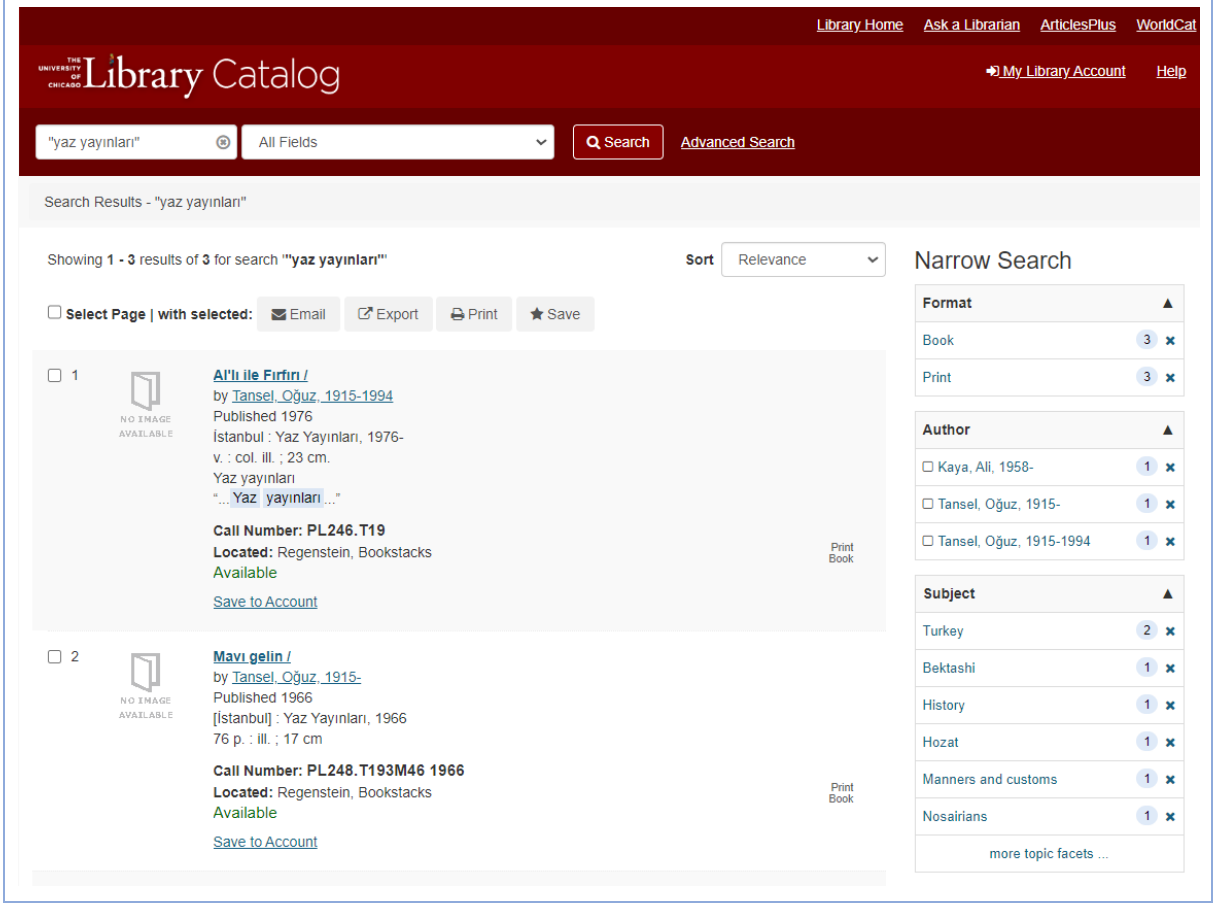
UNIVERSITÄTS- UND LANDESBIBLIOTHEK SACHSEN-ANHALT / ZENTRALE

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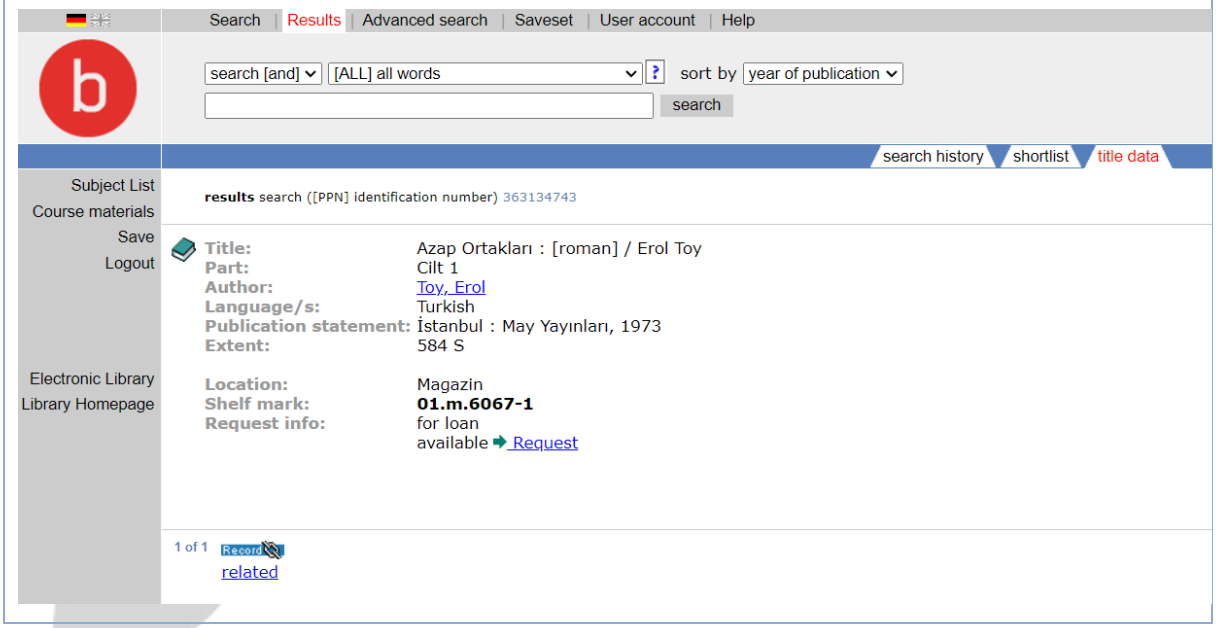
THE UNIVERSITY OF CHICAGO

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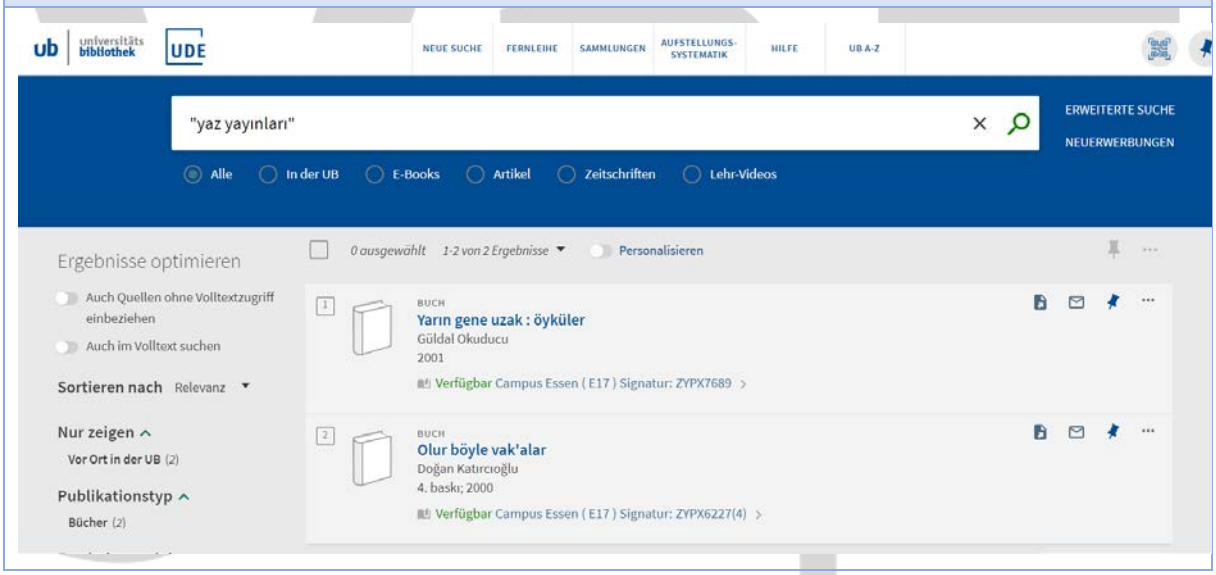
STAATS- UND UNIVERSİTÄTSBIBLIOTHEK BREMEN

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DUISBURG-ESSEN

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YALE UNIVERSITY

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